Primary Management in General, Vascular and Thoracic Surgery

A Practical Approach

Daniele Bissacco
Alberto M. Settembrini
Andrea Mazzari
Editors

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Preface

A specialist doctor is a man who knows more and more about less and less.

Dr. William J. Mayo

Fudit equum magno tellus percussa tridenti.

Virgilio, Georgiche 1 1-42

These are two phrases that strike us to the heart.

Mayo is the founder of one of the most important hospitals in the world, and his phrase is a motto for all doctors who have married their work following the deep meaning of the word "doctor" by taking care of all patients.

Virgil's phrase must be translated into medical words considering that the teacher must prepare young surgeons to learn and make their own the lessons received from the experience of the elders.

Medicine is a field where changes are inevitable, but changes are not always evolutions. To be good doctors, it is imperative to know the biology and processes of the body's biological behavior and, from there, try to identify changes and evolutions in therapies and treatments.

Indeed, in surgery it is essential to know the difference between the basics and the evolution of techniques.

The basics of surgery are the cornerstones of surgical growth for surgeons, but all good doctors and surgeons must keep in mind that the first approach to the patient is the correct diagnosis and management.

Why? The question can be answered by tracing its historical development over the centuries to identify the fundamental steps that depend closely on the evolution of thought. Why? The basis of surgery is not the hands but the head.

The decision-making process is the first and sometimes the most important condition to be addressed before treatment.

Our book is intended to be a concise but comprehensive guide for quick consultation by our younger colleagues on various clinical situations.

This book is for young surgeons by young surgeons. SPIGC (Italian Polyspecialistic Society of Young Surgeons) has endorsed this publication because who can be a better advisor to a young surgeon than another young surgeon who is more experienced, more skilled, and knowledgeable in a specific field.

Practical advice is given for all stages of management, and all chapters are rich in references and illustrations to provide additional reading to those who want more in-depth coverage.

This is the reason why the authors have written very practical and up-to-date chapters on different but frequent pathologies of the most critical surgical fields: general, thoracic, and vascular surgery.

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Historical Overview

1

E. Botteri, F. Bongiovanni, R. L. Meniconi, and E. Grespi

1.1 New Science

Understanding the origins of 'new' scientific knowledge, understanding what and who took the first steps towards breaking free from superstition, supernatural, rigid and immutable beliefs, is a considerable cultural step and one which brings full enjoyment of what science can offer us both as individuals and as members of the scientific community. Above all, intellectual effort calls for a critical mind, in order not to simply accept what is transmitted to us. Thinking can and should be remodelled by everyone, according to well-accepted methods to make it ever more detailed and in keeping with the reality we wish to convey. It requires intellectual honesty, as well as specialised training, and once conclusions have been reached about any piece of work, the results should be conveyed to everyone involved. While 'our' centuries-old science has managed to overcome superstition, an anti-scientific attitude has remained on the side-lines of human thought, threatening its very foundations whenever an immediate explanation to a phenomenon cannot be provided. Science takes time and requires patience, a sound mind and method.

Let us not forget that many of the western world's values and perceptions are inspired by the scientific method and the resulting technical progress. The 'Scientific

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Method', its concepts and its dissemination underpinned the veritable knowledge revolution of the seventeenth century. The Scientific Method represents 'the rules of the game' and covers three elements in its scope:

1. LOGIC and its *inductive* methods (the effects are observed to confirm the causes, according to a process of subsequent reasoning) and *deductive* methods (from the cause, the effects are hypothesised, according to a process of prior reasoning).
2. Systematic application of OBSERVATION, not for mere description purposes, but rather in order to inspire EXPERIMENTATION. Every hypothesis must go hand in hand with repetition and reproducible tests in order for it to be affirmed.
3. Speaking the same language: the language of MATHEMATICS. Galileo Galilei claimed that 'the universe is written in the language of mathematics'. Used in Plato's era as the language of nature, especially in geometry, to avoid being misled by pure sensory experiences.

The main advocates of this development were three scientists closely linked to one another by the knowledge that 'we have been too inefficient thus far', Galileo Galilei, Renè Descartes and Francis Bacon.

Nevertheless, New Science figured little, at least to begin with, in the world in which it emerged. Inevitably, new institutions were needed; composed of people involved with specific scientific fields, supported by patrons or corporations with the aim of facilitating communication and debate in the scientific community—the SCIENTIFIC SOCIETIES were born.

1606 saw the launch of Rome's Lincean Academy, quickly followed by the Accademia del Cimento (1657), Paris' Academy of Sciences (1666) and the Royal Society (1660). In the latter, the influence of the new cultural climate was so strong that the fine arts, rhetoric, metaphysics and theology were excluded from the Articles of Association (without, however, undermining their prominence). The importance of communicating ideas was supported by their own dissemination means and journals such as the Philosophical Transaction, Acta Eruditorum and the Journal de Savants.

At all levels, scientific research products should therefore be made accessible, employing the correct language for the target audience.

1.2 Young People in Science

When we speak about young people in science, particularly about the contribution, young people have made, make and will make, is not only a matter of age. Being young implies a new mental approach and boundless physical endurance. One could wonder who was the first 'young surgeon' to have both a young mind and a youthful age. There is no doubt as to the answer: Giovanni Battista Morgagni (Forlì 1682-Padua 1771). He studied under Valsalva in Bologna before moving to the Republic of Venice and finally settling down in Padua in 1711, when he was called to the second chair of theoretical medicine.

One of his many accomplishments was the sacrilegious method of 'looking into a body' according to a new perspective that began to weaken the old assumption that diseases were linked to an imbalance of humours (humourism), opening the door to the idea of diseased organs and of symptoms being the 'cry of the suffering organs'. From Morgagni we can draw inspiration for the times in which we are about to move away from clinical practice, touching upon research and experimentation: curiosity, critical analysis, accepting findings which are in contradiction with previous hypotheses (indestructible unless proved otherwise) and dissemination capacity. Morgagni's work marked the beginning of pathology as we know it today: the result of systematic observation and experimentation.

When Morgagni was aged only 22, he was named president of the Accademia degli Inquieti, which managed to make reforms, drawing on the experience of the Accademia del Cimento, by bringing to the fore investigative enquiries and consultations, and relegating theoretical debates to a marginal role. The turning point came in 1705 when he gave a reading of the first volume of his publication, *Adversaria Anatomica.* The publication of all of these papers when Morgagni was only 24, gave him instant international recognition as an anatomist.

Another positively larger-than-life young man, and probably for this reason, highly creative and steadfast, was Thomas Fogarty (1934). We all know his name thanks to his famous catheter for embolectomy which we use in our operating theatres, but probably only a few people are aware that the commercialisation and widespread use of this instrument began when Fogarty was only 29 years old. During the years spent at the Good Samaritan Hospital (where he worked as a medical instrument maintenance technician) he met Dr. Jack Cranley—his main mentor—and the man who inspired him to study medicine. The fact of being present during a number of surgical procedures, meant that he witnessed the death and suffering of a several patients suffering from acute artery ischemia. After resolving various technical difficulties, he managed to develop his instrument in 1960, but no one acknowledged the significance of the idea. Only a few years later, as he was finishing his specialisation at Oregon University, did he successfully put forward his idea to the cardiac surgeon, Al Star, who helped him to obtain the final patent in 1969. From this instrument came the idea in 1965 for the design of the first angioplasty balloon.

Of course, Gianbattista and Thomas are only two well-known examples of what a young person driven by interest and passion can achieve. Unquestionably, the history of surgery is brimming with contributions by young, motivated men and women to whom we should express our gratitude each day and from whom we should draw inspiration.

Part I

Surgical Instruments and Materials

Surgical Instruments and Materials in General Surgery

2

Andrea Mazzari, Pasquina M. C. Tomaiuolo, and Roberto Luca Meniconi

2.1 Cut and Dissect Instruments

Dissection of tissues can be done with scalpel, scissors, (Fig. 2.1) or through the use of energy such as ultrasonic devices, laser, and radiofrequency. Conventionally, scalpels have been used to make surgical incisions by manually cutting through tissue using a sharp blade. Scalpel consists of a blade and handle and is usually used for initial incision. Different blades are marked with a number.

Scissors are used for cutting tissue, suture, or for dissection. Scissors can be straight or curved, and may be used for cutting heavy or fine structures.

Since its introduction in the early part of the twentieth century, electrosurgery has been used as an alternative tool for creating incisions [1, 2]. Two different surgical effects can be achieved with electrosurgery, namely cutting and coagulating. In the cutting mode, a continuous current rapidly produces extreme heat causing intracellular water to boil and cells to explode into steam. As a result, the heat produced in the cells dries up the tissue but is not intense enough to evaporate intracellular water. The coagulating mode results in a greater degree of thermal damage and necrosis of adjacent tissues.

Electrosurgery can be performed using either monopolar or bipolar energy in conjunction with a specialized instrument.

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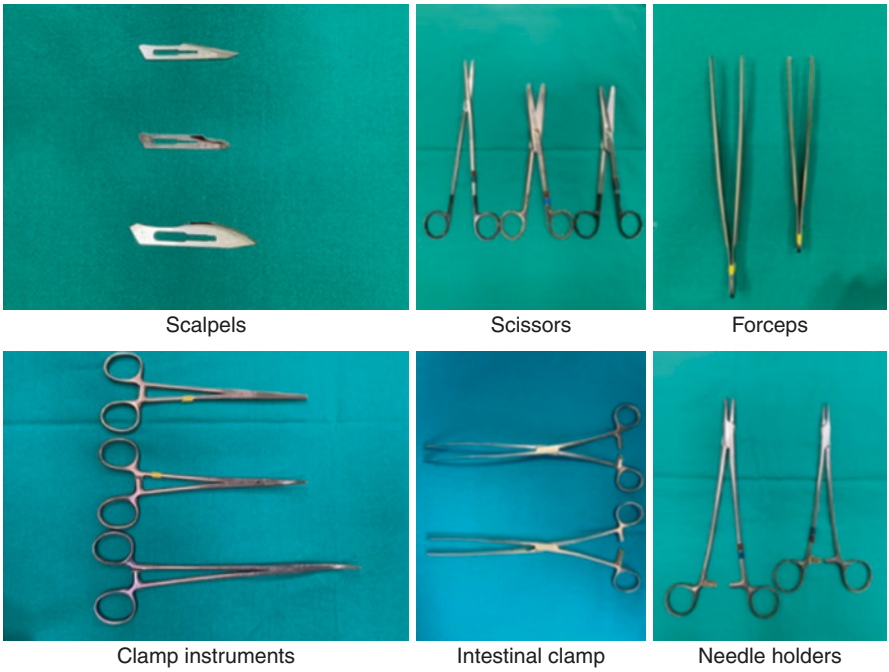


Fig. 2.1 Open surgery—surgical instruments

2.1.1 Monopolar Electrosurgery

Monopolar electrosurgery can be used for several modalities including cut, blend, desiccation, and fulguration. Using a pencil instrument, the active electrode is placed in the entry site and can be used to cut tissue and coagulate bleeding. The return electrode pad is attached to the patient, so the electrical current flows from the generator to the electrode through the target tissue, to the patient return pad and back to the generator. Monopolar electrosurgery is the most commonly used because of its versatility and effectiveness.

2.1.2 Bipolar Electrosurgery

Bipolar electrosurgery uses lower voltages so less energy is required. But, because it has limited ability to cut and coagulate large bleeding areas, it is more ideally used for those procedures where tissues can be easily grabbed on both sides by the forceps electrode. Electrosurgical current in the patient is restricted to just the tissue between the arms of the forceps electrode. With bipolar electrosurgery, the risk of patient burns is reduced significantly. In the most common techniques, the surgeon uses forceps that are connected to the electrosurgical generator. The current moves

through the tissue that is held between the forceps. Because the path of the electrical current is confined to the tissue between the two electrodes, it can be used in patients with electrical implanted devices, to prevent a short-circuit or misfire.

2.1.3 Ultrasonic Energy

Ultrasonic dissection technology works by generating a high-frequency ultrasound producing three main effects:

1. Cavitation/tissue fragmentation (and dissection) caused by cellular destruction secondary to intracellular fluid evaporation.
2. Cooptation/coagulation: caused by conversion of ultrasonic energy into a localized heat, this has been reported to reach from 60 °C to 100 °C. Denaturation of collagen in the walls of hollow structures can result in the occlusion or sealing of the lumen.
3. Cutting which is achieved by the "sharp" blade mode.

It has been reported that with ultrasonic energy, there is a minimal lateral spread of vibration current in the surrounding tissues minimizing the risk of injury compared with monopolar electrocautery.

2.1.4 Radiofrequency Energy

The main functionality of radiofrequency energy is to seal and coagulate. Hemostasis is obtained by melting the collagen and elastin in the vessel walls; in clinical practice this technology can seal blood vessels up to 7 mm in diameter.

2.2 Grasp and Hold Instruments

Forceps are the grasping instruments that allow the surgeon to manipulate tissue, to facilitate dissection or suturing by holding the edges of tissue (Fig. 2.1). They have multiple uses other than holding skin when suturing: extracting needles, passing ligatures to other instruments around vessels, grasping vessels to apply diathermy. They could have various forms and designs and are probably the most commonly used instrument. Forceps can be toothed (serrated) or non-toothed at the tip. Tissue forceps are non-toothed and used for fine handling; DeBakey forceps are used for atraumatic dissection of soft tissues and vessels; Adson forceps are toothed at the tip and are used for skin closure; Bonney forceps are used for holding thick tissue, like during fascial closure.

It is strongly recommended to use intestinal forceps when manipulating the bowel to avoid incidental enterotomies especially in emergency setting, when the intestinal wall could be frailty for edema due to peritonitis.

2.3 Clamp Instruments

Clamping instruments are used to constrict tissue, structures, and vessels. They could be curved on flat, on the side or straight. There are specifically designed clamps for delicate tissue especially in vascular and in gastrointestinal surgery. The jaws of these instruments are designed to prevent tissue damage: the intestinal clamps have flexible and long jaws that allow a proper occlusion of the bowel but prevent any damage (Fig. 2.1).

Hemostatic clamps are used to occlude vessels prior to ligating or to provide hemostasis.

2.4 Suture Instruments

Needle holder (Fig. 2.1) and forceps are used for handsewn suture with absorbable or not absorbable suture materials. Needles come in many shapes and cutting edges for various applications. Sutures are available in sizes between 5 and 11/0. Higher numbers indicate larger suture diameter (e.g., 2 is larger than 1), and more zeros indicate smaller suture diameter (e.g., 4/0 is smaller than 3/0, or 0). There are two main types of suture: braided and non-braided (or monofilament); the second one can be absorbable and non-absorbable. Additionally, suture can be made with natural or synthetic materials.

2.5 Suction Instruments

Suction devices are attached to a vacuum source via suction tubing and are used to remove blood and body fluid; they have various designs and "tips" attached to a form of handle to allow suctioning of everything from small wounds to large abdominal wounds.

2.6 Surgical Staplers and Clips

Surgical stapling devices have changed gastrointestinal surgery; the first one was built in 1908 by Humer Hultl. Nowadays they are used both for open and laparoscopic surgery. Surgical staplers can seal tissue through metal clips that allows the correct vascularization of the tissues. They are used to perform intestinal anastomoses making theme quicker and easier, and for vascular ligations. Linear staplers with a double or triple layer allows a safe closure of the tissue, but they do not have a cutting system. Linear cutter creates a linear cut and immediately staples both free

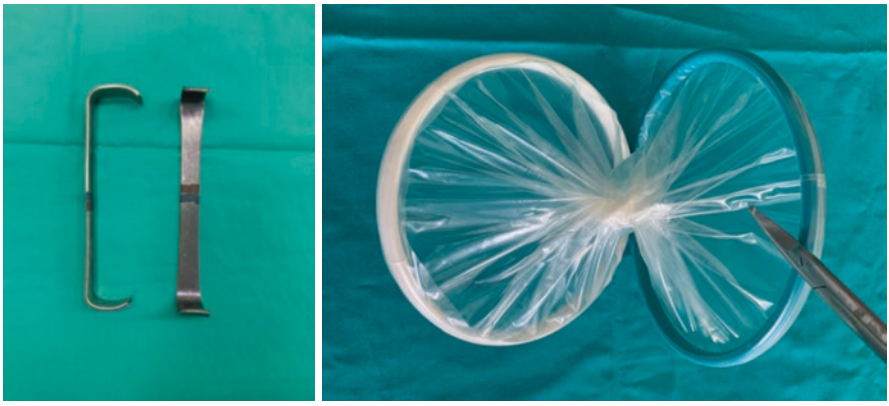
edges. Circular staplers are cylindrical in shape with a cone at the tip that can be removable: they are used to perform end-to-end or side-to-end anastomosis in colorectal and esophageal surgery.

Construction of intestinal anastomosis is a hallmark of surgical training; surgical staplers facilitate the surgeon but there is no evidence that demonstrate any superiority of the stapled over handsewn anastomosis [3].

Clips used in the ligation of vessels may be in metal, polymeric or absorbable material.

2.7 Retractors

Retractors are the instruments designed to expose tissue and organs during the surgical procedure, they could be hand-held or self-retained, and have a large variety of sizes. An optimal view is mandatory for a safe procedure. The hand retractors could be single or double ended and usually have a comfortable design. The self-retaining retractors have multiple blades for different tissues and depth, in most cases can be attached directly to the table to guarantee more stability in open large abdominal surgery. Disposable self-retractors are useful to prevent surgical site infection, especially in emergency setting when the surgical field is often contaminated [4] (Fig. 2.2).



Hand-held retractor Disposable self-retractor

Fig. 2.2 Retractors

2.8 Laparoscopic Surgery

Laparoscopic surgery has emerged over the past two decades as the surgical approach of choice in the treatment of many digestive disorders. Laparoscopy has its role in the management of abdominal surgical emergencies since it provides the same benefits: less postoperative pain and shorter length of hospital stay when compared to laparotomy [5, 6]. In emergency surgery, the laparoscopic approach provides a better view of the abdominal cavity, giving the opportunity for a precise diagnosis and, at the same time, a definitive treatment. However, its role in the management of acute abdomen is strongly influenced by the laparoscopic skills of the surgeon.

Most of the instruments are similar to those used in open surgery, adapted to fit through ports placed through the skin. The camera, connected to light source through a fiber-optic cable, magnifies the image and is connected to a monitor that can be viewed by the surgical team. Camera vision can provide different angle of vision, the most used are 0° and 30°. The instruments are inserted into the abdomen between trocar and ports with different diameter (3 mm, 5 mm, 10–12 mm) and length. Trocars can be bladed or not bladed; all the trocars have a seal which maintain pneumoperitoneum during the surgical procedure. Laparoscopic instruments are similar to open and are 30–33 cm long (Fig. 2.3). Their main parts are handles with or without ratchet device, rotator that allow a full rotation device and the operative inner that can be easily assembled. The working part as in open surgery is used to dissect, aspirate, grasp, retract, cut, suture, and cauterize tissue.

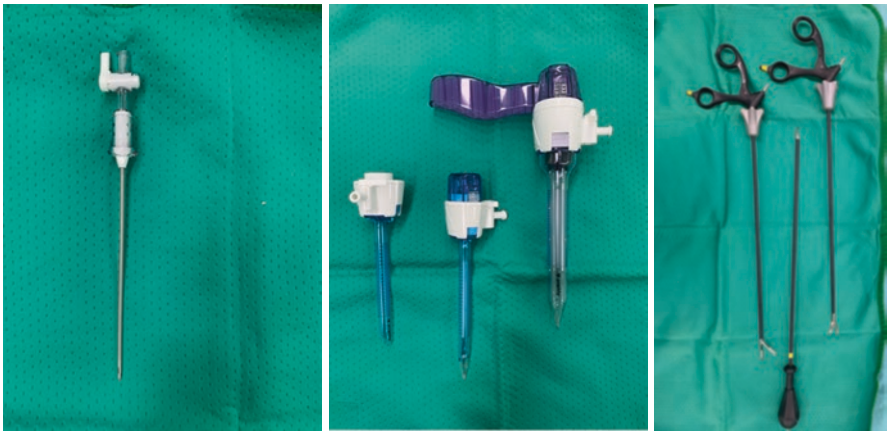


Fig. 2.3 Laparoscopic surgery—surgical instruments

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Surgical Instruments and Materials in Thoracic Surgery

3

Valeria Musso and Francesco Damarco

This chapter briefly describes the most used surgical instruments and devices in thoracic surgery, in an emergency setting. In addition we focused on the most common procedures performed in emergency.

3.1 Thoracentesis

Thoracentesis is often used in the Emergency Room as a both diagnostic and therapeutic procedure in patients presenting with pleural effusion. There are several commercial pre-packaged kits available, containing the necessary items to perform a thoracentesis (Fig. 3.1). In general, standard set consists of:

- Protective equipment: sterile gloves, eye protection, face mask.
- Field preparation: sterile drapes, skin sterilizing fluid (chlorhexidine or povidone-iodine solution).
- Ultrasonography (optional): Convex or linear probe with a sterile sheath cover. The use of ultrasonography to identify the appropriate location for the procedure or for direct guidance improves the safety of thoracentesis.
- Local anesthetic agent (1–2% lidocaine), 21 or 22-gauge needle, and a syringe for injection.
- Thoracentesis needle: the most commonly used is an 8-Charriere (1 Ch = 1/3 mm) over-the-needle catheter; otherwise Seldinger technique can be employed

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Fig. 3.1 Pre-packaged set for thoracentesis, including needles, three-way stopcock, and a drainage bag

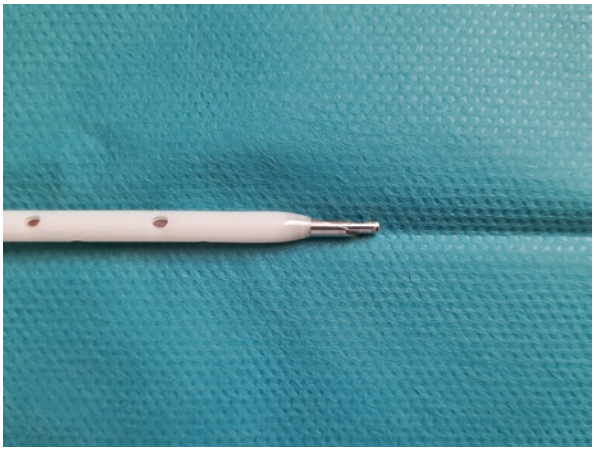

Fig. 3.2 Detail of a Veress needle

(requiring a wire, dilator, and a 6–14 Ch catheter); some kits contain a Veress needle (Fig. 3.2).

- Three-way stopcock, a 30–50 ml syringe, drainage bag.
- Sterile containers for pleural fluid collection.

3.2 Tube Thoracostomy

The insertion of a chest tube is a simple procedure, and yet, severe complications may arise if not performed properly. Different characteristics of chest tube are summarized in Table 3.1.

The indications and technique will be discussed further in the text (Chap. 7 "Chest: surgical anatomy and general consideration in emergency settings"; Chap. 9 "Pulmonary and thoracic emergencies").

Box 3.1 Preparation for chest tube insertion

- Obtain informed consent (unless it is an emergency)
- Monitor vital parameters
- Large-bore intravenous line
- Exams (if possible): ECG, blood tests, chest X-rays, CT scan in case of trauma, ultrasound for guidance.
- Adequate analgesia, consider mild sedation

In case of small-bore chest tubes, the drainage can be inserted with the Seldinger technique or using the pre-packaged kit.

The instruments required to insert a chest tube are listed below:

- Protective equipment: sterile gloves, eye protection, face mask.
- Field preparation: sterile drapes, skin sterilizing fluid (chlorhexidine or povidone-iodine solution).
- Ultrasonography (optional): Convex or linear probe with a sterile sheath cover. The use of ultrasonography to identify the appropriate location for the procedure or for direct guidance improves the efficacy of chest tube placing, especially in case of pleural effusion.
- Local anesthetic agent (1–2% lidocaine), 21- or 22-gauge needle and a syringe for injection.
- Scalpel with a n°10 or 11 blade.
- Gauze sponges.
- Straight- and curved-blade Mayo scissors.
- Chest tube: the diameter ranges from 8 to 36 Ch.
- Clamp.
- Closed system drain (including water) and tubing.
- Needle holder.
- Non-adsorbable 0 or 1-0 suture.

Table 3.1 Chest tube characteristics

Chest tube characteristic	
Material	Silicone; polyvinyl chloride
Caliber	From 8Ch to 36Ch
Shape	Straight; angled; Pigtail
Other	With internal, external, or no mandril

Box 3.2 Type of drainage systems

- Unidirectional flutter valves (Heimlich): The distal portion can be connected to a bag or a suction device, or left open. It is useful in an outpatient setting.
- Traditional water seal and drainage bottle (Bulau): A one-bottle system allows for fluid and air drainage; a two- or three-bottle system is required for suction.
- Disposable drainage systems: There are many types of devices available, working as one-, two-, or three-bottle systems; they can be used with portable suction units.
- Digital drainage systems: These devices constantly record the presence of air-leaks and fluid output; they integrate suction units.

3.3 Thoracotomy

Thoracic trauma, related to both blunt and penetrating mechanism of injury, can determine devastating damages with an elevated morbidity and mortality. The initial evaluation in case of trauma of the thoracic cavity should always be based on an Advanced Trauma Life Support (ATLS) program. In selected patients and clinical circumstances, resuscitative thoracotomy (or emergency department thoracotomy in general) can be performed to pursue damage control such as to temporize hemorrhage or decompress cardiac tamponade.

Box 3.3 Initial assessment for severe thoracic trauma

- Monitor vital parameters
- At least one large-bore intravenous access
- Endotracheal tube
- Nasogastric tube

A sterile tray with the equipment to perform thoracotomy should be available at all times in the trauma room. Together with sterile drapes, sterilizing fluid, gloves, and other protective equipment, the set should include the following instruments:

- Scalpel holder and blades; electrocautery.
- Gauze sponges and a suction unit.
- Tissue forceps: Quénu forceps, long DeBakey tissue forceps; grasping forceps: Duval, Lovelace lung grasping forceps.
- Allison lung retractor.
- Scissors: long curved Mayo scissors, long curved Metzenbaum scissors.

- Rib spreader: Burford-Finochietto, Tuffier; sternal retractor; Farabeuf retractors.
- Giertz-Shoemaker rib shears.
- Sternal saw (e.g., Gigli, oscillating); Lebsche sternal chisel.
- Mallet.
- Aortic clamps, angled and curved: Fogarty aortic cross-clamp, DeBakey aorta clamp; hemostatic clamps: Satinsky clamp. A Foley catheter may also be of aid to control the hemorrhage.
- Bailey rib contractor.
- Needle holder: DeBakey, Ryder.
- Sutures.

3.4 Video-assisted Thoracic Surgery (VATS)

As VATS surgery becomes increasingly employed, even in the emergency setting, surgeons are required to know the basic instrumentation to perform thoracoscopic procedures. In general, the instruments are thinner than those used for open surgery, their fulcrum is located at the distal end, and they have a curved shape.

Instead of a rib spreader, trocars or wound retractors are used to improve exposure, causing less pain and reducing the incidence of rib fractures.

A thoracoscope is used for visualization: 0° and 30° scopes are the most frequently used.

The VATS surgery set (Fig. 3.3) usually consists of non-traumatic graspers, dissecting forceps, and a dissecting suction tube. For dissection an electrocautery hook, an ultrasonic dissector-coagulator, or bipolar electrocautery devices can be

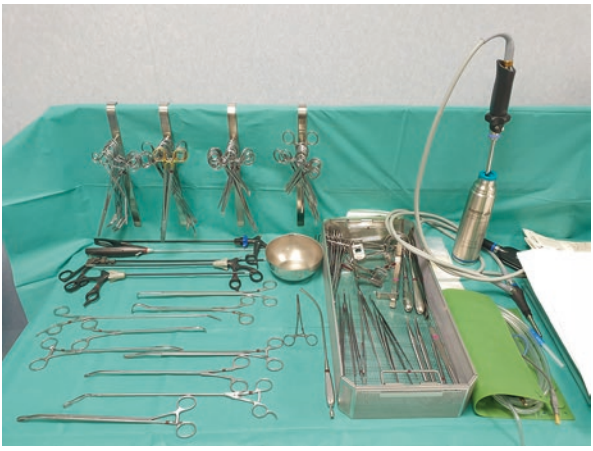

Fig. 3.3 Video-assisted thoracic surgery set

used. Endoscopic stapling devices are fundamental to safely divide lung parenchyma, vessels, and bronchi, providing adequate hemostasis and aerostasis.

Further Reading

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Surgical Instruments and Materials in Vascular Surgery

4

Ilenia D'Alessio and Matteo Marone

4.1 Introduction

In the vascular emergency setting, the main objective is to stop the bleeding and/or re-establish blood flow to a specific anatomic district.

To achieve this, the emergency physician and/or vascular surgeon has access to multiple kinds of surgical and endovascular materials, which can be used according to patient characteristics and prompt availability.

To facilitate the lecture of this chapter, we have decided to divide surgical instruments and materials in a vascular and an endovascular section, even if they are often used together during hybrid procedures.

4.1.1 Open Surgery

The basic techniques and exposure of single vessels will be discussed in the following chapters. Here below we will describe the general principles of vascular surgery.

1. The basic concept is to approach and expose the vessel using the most direct and easier route possible, avoiding iatrogenic damages. Anatomic landmarks, location of pulses, and/or both are typically used to guide the initial skin incision.

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2. Once the sheath is reached, it is incised and the vessel's adventitia is held and retracted in one direction.
3. The exposure of the vessel is carried out close to the edge of the blood vessel wall.
4. Once the vessel is isolated, it can be circumferentially dissected and encircled using vessel loops.
5. Blood vessel control can be achieved in different ways: using vascular clamps, balloon occlusion, vessel loops, tourniquets, internal occluders or also, especially during emergency procedures, using direct finger compression.
6. Before interrupting blood flow (approximately 3–5 min), the patient must be adequately anti-coagulated (unfractionated heparin at a dose of 75–100 Ui/kg intravenously). The Activated Clotting Time (ACT) can be measured 180 s after the heparin administrations and it should reach values of more than 250 s. It is useful to monitor the ACT values during the procedure [1, 2].

To perform these basic steps, the surgeon needs the following instruments:

- *Clamps* (Fig. 4.1)

  They are surgical instruments used to occlude a vessel from outside, without damaging it, they are considered atraumatic for vessels' wall. Clamps allow the surgeon to open the vessel (perform an arteriotomy) and work inside the vessel itself.

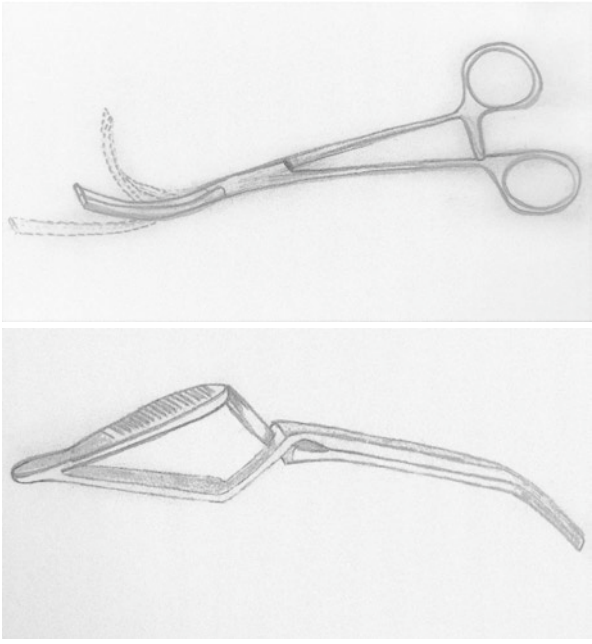

Fig. 4.1 Examples of vascular clamp (first image represents a DeBakey clamp, the second image represents a Dietrich camp)

It is important to clamp the artery opposing the healthy wall to the plaque (if present) to avoid the plaque rupture.

Clamps are usually divided according to vessels' size: large, medium, and small vessels or into totally occluding and partially occluding [2].

Below you can find the most common vascular clamps:

- DeBakey aortic clamp (supraceliac, infrarenal aorta)
- Fogarty aortic clamp (infrarenal aorta, aortic grafts, calcified aorta)
- DeBakey peripheral vascular clamp (iliac arteries and common carotid arteries)
- Satinsky clamp (aorta; vena cava)
- Cooley Derra Clamp (graft limbs)
- Dietrich bulldog (small vessels)

- *Needle Holders, Forceps, and Scissors*

  Needle holders are devices created to handle needles in a precise way, reducing the risk of injury for the operator itself. The choice of needle holder is dictated by the size of the needle and by the surgeon preferences. The Mayo-Hegar needle holder is used with large needles, while Castroviejo needle holders are typically used with fine vascular needles.

  The forceps used during vascular procedures typically have fine, non-crushing jaws, exemplified by the DeBakey forceps. They are useful because it allows the surgeon to touch the vessels without provoking important injuries to the arterial or venous wall.

  Metzenbaum and Church scissors are used for the dissection of blood vessels. Special Potts scissors with various angulations are used to enlarge and shape arteriotomies and venotomies (Fig. 4.2) [1].

- *Suture*

  They are traditionally divided in absorbable/non-absorbable and mono/multifilament. In vascular surgery the favorite suture materials are non-absorbable monofilament sutures due to their non-traumatic action on the vessel itself.

  Non-absorbable monofilament sutures are used for vascular anastomoses and repairs. Vascular sutures are usually double-armed with a needle on each end to allow continuous suturing in both directions.

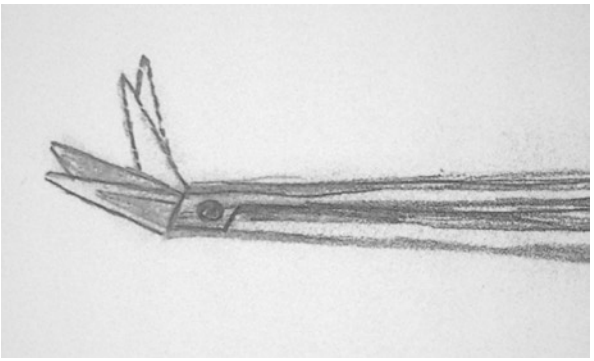

Fig. 4.2 Potts scissors

Table 4.1 Fogarty catheter classification based on color code, French size, and balloon diameter

Color code	French size (Fr)	Balloon diameter (mm)	Target vessel
Violet	2	4	Foot and hand vessels
Green	3	5	Leg and forearm vessels
Red	4	9	Popliteal artery, superficial and common femoral artery, external iliac artery, brachial artery, subclavian artery
White	5	11	Popliteal artery, superficial and common femoral artery, external and common iliac artery, brachial artery, subclavian artery
Blue	6	13	External and common iliac artery, aorta
Yellow	7	14	External and common iliac artery, aorta

Commonly used monofilament sutures include: polypropylene, polybutester, and polytetrafluoroethylene (PTFE).

PTFE sutures are quite recent and they were developed to minimize the needle hole bleeding that is often seen when polypropylene sutures are used with PTFE grafts or patches. They are designed so that there is minimal difference in the diameters of the needle and the suture [2].

In an emergency setting, it is useful to know the existence of the:

- *Fogarty catheter*®, it is a hollow tube with an inflatable balloon attached to its tip. The catheter is inserted into the blood vessel through a clot. The balloon is then inflated to extract the clot from the vessel. It is available in different lengths and sizes, and is often color coded by size (Table 4.1). They can be also used to clamp the target vessel from the inside (inflating the balloon) and this is useful especially if the wall of the vessel is extremely damaged.
- *Aortic Balloons occlusion (see endovascular section)*

4.1.2 Endovascular Surgery

The goal of this section is to provide a "step-by-step" guide to obtain a safe vascular access in an emergency setting.

Basic endovascular skills are integral part of vascular and trauma patient care and often offer rapid solutions to complex clinical problems (i.e., clamp a broken aorta using aortic balloons occlusion). Moreover, nowadays, endovascular procedures are the mainstream of treatment during the non-operative management of many traumatic injuries like: splenic injuries, hepatic injuries, and pelvic injuries [3].

In the previous section (see open surgery) we have analyzed the basic techniques to obtain the surgical exposure of a vessel, now we'll list the principles of percutaneous access:

1. Choose the more appropriate puncture site according to the planned procedure.
2. Pick an access site that is far enough from the lesion so the sheath may be placed without encountering the lesion itself.

3. Feel the artery intended for puncture and palpate anatomic landmarks.
4. Use ultrasound guidance to visualize the artery and its relationship to anatomic landmarks.
5. If you encounter a problem, hold pressure for a few minutes and start again. It's rare to have any significant damage to the access artery from the needle alone [3].

Usually to obtain a percutaneous access you'll need to: a sterile swab, a scalpel, a hemostat, a percutaneous entry needle, a syringe with local anesthetic, and a guidewire. These instruments are required to perform the Seldinger technique, also known as Seldinger wire technique, a surgical procedure to obtain safe access to blood vessels. Using ultrasounds to locate the vessel and to choose the ideal puncture side will help you avoiding failure and access-related complications (Fig. 4.3) [4].

The main steps of the procedures are the following:

1. Inject the anesthetic into the subcutaneous tissue that surrounds the area designated for the puncture, remember to inject the solution also close to the artery in order to avoid pain stimulations during the procedures.
2. The entry needle approaches the artery at a 45° angle (Fig. 4.3a).
3. A pulsate backbleeding indicates that the needle tip is in the artery (Fig. 4.3a).
4. While the non-dominant hand holds the needle, the dominant hand retrieves the guidewire, straightens its tip, and inserts it into the needle hub (Fig. 4.3b).
5. When the guidewire is parked far in the target artery, the needle is retrieved and a coaxial dilator system is advanced over the guidewire (Fig. 4.3c, d).
6. The starter guide and the inner trocar are removed, and, at this point it is possible introduce the desired wire for the case (Fig. 4.3e) [3].

In some cases it is mandatory a surgical exposure of the vessel, especially in case of diffuse atheromasia, in this case we talk about hybrid approach.

To perform these basic steps the surgeon needs:

- *Entry needle:*

  It is a sharp hollow needle (usually 18 gauge) usually between 2 and 3.5 inches in length whose beveled tip is placed into the anterior wall of the artery. These kinds of needles can accommodate a 0.035 inch guidewire. However, fine needles (21 gauge) can be used to access to small vessel like the radial and pedidial artery.
- *Guidewire*:

  The guidewire you introduce initially must be stiff enough to serve as an initial rail for sheath, its tip must be atraumatic, so that if plaque is encountered, it is not disrupted. The initial guidewire must be inserted into the vessel for a sufficient length to not have unintentional displacement. Usually access is performed with 0.035-inch diameter guidewires using, e.g., Starter Guidewire. There are many types of guidewires and they are classified according to their length, the shape of the tip, their material and their diameters. The main diameters used are 0.035 inch, 0.018 inch, and 0.014 inch.

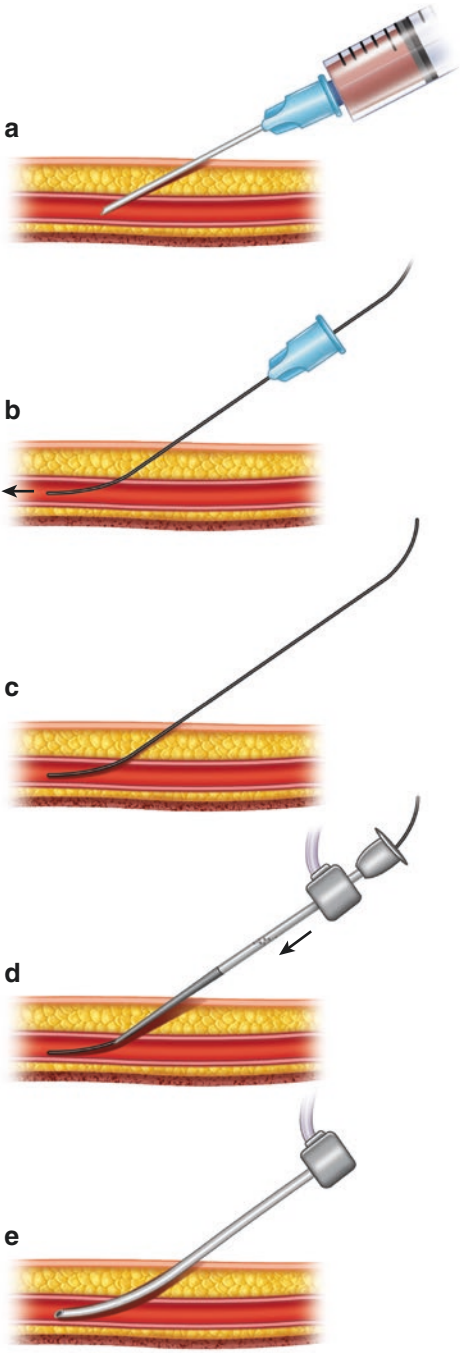

Fig. 4.3 Seldinger technique

- *Sheath:* The sheath placement permits a safe and hemostatic access. The main role of the sheath is to provide protection of the access site from the irregular edges of endovascular devices, because it reduces the friction encountered at the access site when manipulating a selective catheter into a branch. An access sheath has one way hemostatic valve, a dilator to stiffen it during placement, and a side arm port that is used for the administration of medication or contrast. The sheath is advanced always with its dilator to avoid damages by the hollow sheath tip. Sheaths are sized according to the largest diameter catheter the sheath will accept [3, 4].

  The physician has available a wide variety of guides, sheets, and catheters. A deeper treatise is beyond the purpose of this chapter, if the reader is interested we suggest to look at an endovascular handbook.

  Once gained the access to the vessel, there are several diagnostic and therapeutic options.

  The physician may need to perform an angiography to detect the source of a bleeding, and he/she can use a variety of coils, plugs, glues, and hemostatic devices to embolize a vessel or even put stents (covered or uncovered) to complete the procedure.

  More detailed:
- *Coils and Plugs*:

  The coils and plugs are vascular embolization devices that occlude vessels' lumen. Usually they are used in an emergency setting to control hemorrhage. For instance, in a blunt splenic injury with a grading less than III instead of a splenectomy the surgeon can perform an endovascular ligation of the splenic artery occluding it with coil and plug. As a result, the spleen is partially excluded from the arterial system and the bleeding stops.
- *Stents*: The stents are metallic scaffolding used to dilate an occluded vessel. They are specially designed mesh metal tubes that are inserted into the body in a collapsed state on a catheter. They can be divided into balloon expandable and self-expandable. Balloon expandable stents have a high radial force and they can be used to dilate thigh strictures that are not really flexible. In contrast, self-expandable stents have a good flexibility and they can navigate really well in a vessel but their radial force is weak.
- *Angioplasty balloon*: The angioplasty balloon is a small balloon inserted into an atherosclerotic vessel and inflated to crush and disrupt the atherosclerotic plaque and re-establish the patency of the lumen. They come in different size: there are big balloon used for the aorta and little balloon used for the coronary arteries. In emergency setting they can be used to internally clamp a vessel or to treat a dissection flap [3, 5, 6].
- *Stent graft*: The stent graft is self- or balloon expandable stent, covered with a polytetrafluoroethylene or woven Dacron layer that makes them impermeable to blood. They are useful to exclude vascular aneurysm from the blood flow, in different parts of the body and they come in different size and shape according to their purposes. Ruptured vessels can be treated in an endovascular fashion using a stent graft to cover the vascular injury and stop the bleeding. However, espe-

cially if we speak about penetrating trauma, these kinds of procedures have a great risk of postoperative device infection [3, 5].

- *REBOA*™:

  It is the acronyms for Resuscitative Endovascular Balloon Occlusion of the Aorta. They are balloon designed to be inserted (percutaneously or with a femoral artery cut-down) without guidewires in the body of in-extremis patients in order to occlude the aorta from inside. As a consequence these balloons can stop the bleeding and at the same time they help centralizing the blood flow to noble organs such as the brain, the heart, and the lungs. The REBOA™ can be used not only in a hospital setting but also in a prehospital and street setting. The scenario where it is mostly used is blunt trauma with non-compressible hemorrhages like: pelvic fractures, solid organ injuries, mesenteric injuries. The main drawback of the device is that it is often inserted in a chaotic environment with no guidewire and without fluoroscopy and this can result in a wrong positioning of the device and in vascular injuries. In addition, once the balloon is inflated it creates a state of total vascular occlusion downstream and this can create severe ischemic damage especially to kidney, bowel, liver, and biliary tract [7, 8].

4.1.3 Hybrid Surgery

Nowadays, it has become far more common the use of hybrid theater especially in tertiary hospitals. A hybrid suite is an operating theater integrated with diagnostic devices like mobile C arms. These rooms allow the surgeon the possibility to perform open procedures and to complete them using endovascular technique. A clear example of the utility of these places can be seen thinking of peripheral bypass. The vascular surgeon can perform the bypass in a comfortable setting and at the end of the procedure can perform double subtraction angiography (DSA) and percutaneous balloon angioplasty as in a normal angio-suit.

In first world countries hybrid room is used also as shock room in big trauma centers. For trauma purpose this setting offer important advantage like the possibility to perform an active resuscitation while the vascular surgeon performs an angiography using a vascular access. This allows to minimize the time between the arrival of the patient in the emergency room and the diagnostic and treatment phase. These rooms can allow the vascular and trauma surgeon to perform also computed tomography scan on the resuscitation table, giving important advantages especially in patients with traumatic brain injuries.

The drawbacks of this kind of setting are that these rooms are expensive, they need specifically trained professionals to function, and they cannot be set up in every hospital.

Key Messages

- Knowledge of materials and techniques allows the best and safest vascular approach for the injured vessel.
- Before going to the operating room and start a procedure check which materials are available and if you are familiar with them.
- Ask for help to a senior consultant if you are not familiar with a particular device.
- Consider every time the possibility of endovascular procedures.
- Hybrid procedures are becoming the mainstream of treatment of trauma and vascular patients.
- Infections can occur after endovascular procedures for trauma and a close follow-up is needed.
- Be ready with a backup plan because endovascular devices can fail.
- Be open to any technological advancement.

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Part II

Neck

Supra-aortic Trunks Emergency Conditions

5

Enza Lucia Castronovo, Daniele Bissacco, D'Oria Mario, Alberto M. Settembrini, and Sirignano Pasqualino

5.1 Introduction

Emergency supra-aortic trunks (SATs) disease includes a group of urgent onset conditions involving the origin of the common carotid arteries, subclavian arteries, internal carotid arteries (ICAs), and vertebral arteries. These pathologies represent one of the main causes of death and disability in the occidental population and require prevention and rapid management when symptomatic. Among SATs pathologies, ICA and/or vertebral stenosis or occlusion due to atherosclerosis or other factors are the most frequent and fearsome conditions, being responsible for the majority of SATs' urgencies. In particular, ICA plaque is considered one of the main causes of ischemic stroke, one of the most cause of death in the USA and Europe. Subclavian arteries rarely lead to urgent conditions, although their acute occlusion may provoke vertebral insufficiency and/or ischemic complications of the upper limbs.

The aim of this chapter is to provide clinical and instrumental information to better recognize and diagnose SATs emergent/urgent conditions, to describe the best first management and treatment choice for each scenario, particularly in case of cerebrovascular complications related to ICA stenosis or occlusion.

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5.2 Anatomy of Supra-aortic Trunks

The term "supra-aortic trunks" (SAT) includes the vessels (brachiocephalic trunk [BCT], left common carotid artery [LCCA], and left subclavian artery [LSA]) arising from the aortic arch (Fig. 5.1). BCT also called anonymous artery is 5–7 cm long and arises from the aortic arch and goes up the upper mediastinum to the right sternoclavicular joint, where it divides into its final branches: the right subclavian artery and the right common carotid artery. ACL arises from the aortic arch near BTC. It originates on the left side of the trachea. Its mediastinal course is more or less vertical, so it travels like the RCCA in the lateral sulcus of the neck. The origin of LSA is usually approximately 1 cm distant from that of LCCA. Its mediastinal course is vertical and leads it to the base of the left supraclavicular cavity. The common carotids have a vertical course in the lateral margin of the neck up to the mandibular angle where the ICA and the external carotid artery (ECA) originate at the level of C4. They run inside the vascular fascia with the jugular vein and the vagus nerve along the neck. Then, the ICA enters the skull through the external opening of the carotid canal and divides into seven segments depending on the areas crossed. Along its course, the ICA divides into its two terminals called the anterior and middle cerebral arteries.

The ICA supplies the brain including the eyes, while the external carotid the face, scalp, skull, and meninges.

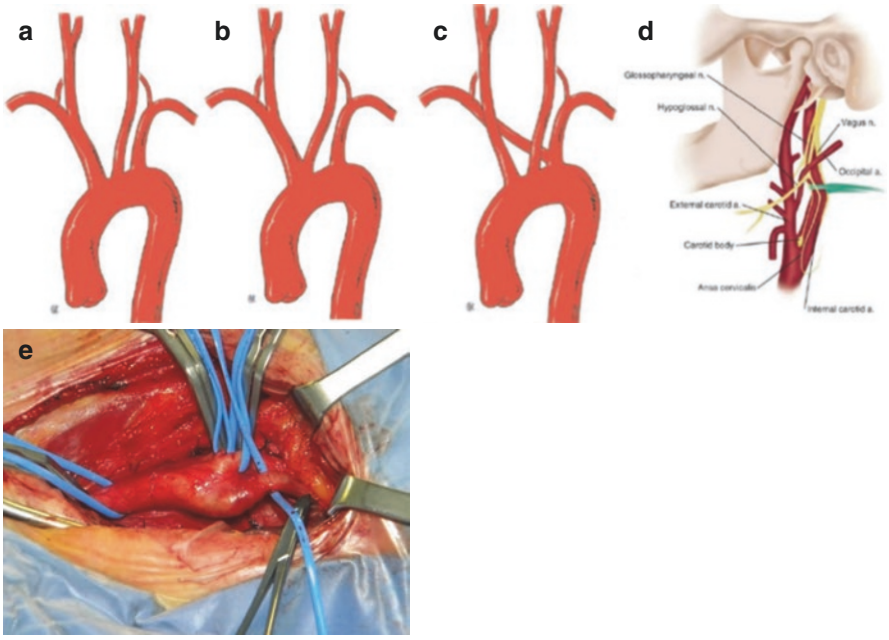

Fig. 5.1 Anatomy of supra-aortic trunks. (**a**) Normal anatomy. (**b**) Bovine arch. (**c**) Lusoria subclavian artery. (**d**) Anatomy of common, internal and external carotid arteries. (**e**) Intra-operative carotid anatomy. From: Rutherford's Vascular Surgery and Endovascular Therapy, E-Book. Elsevier Health Sciences (2018)

5.3 Stroke

Stroke causes 1.1 million deaths annually in Europe and 5.5 million in the USA making it the second commonest cause of death. Furthermore, more than half of stroke survivors experience complex disability for one or more aspects in everyday activities [1, 2]. The World Health Organization (WHO) has defined stroke as focal, occasionally global, loss of neurological function lasting >24 h (or leading to death) and that it has a vascular etiology. The Stroke Council of the American Heart Association/American Stroke Association defined transient ischemic attack (TIA): "A transient episode of neurological dysfunction caused by focal cerebral, spinal cord or retinal ischemia, without acute infarction" [3, 4]. Silent infarction is defined as "imaging or neuropathological evidence of cerebral/retinal infarction without a history of acute neurological dysfunction attributable to the lesion" [5]. TIA can be further classified in other subgroups: crescendo TIAs and "stroke-in-evolution." Crescendo TIAs are multiple TIAs within a short time period, with full recovery in between. The exact number and/or frequency has never been defined, but at least three events in 7 days would seem reasonable. "Stroke-in-evolution" is a fluctuating deficit (never fully back to normal) or a progressively worsening neurological deficit.

Stroke can be also classified as hemorrhagic (8–18%) or ischemic (72–87%), depending on its main cause [4, 6].

The main causes of ischemic stroke are thromboembolism from the internal carotid artery (ICA) or middle cerebral artery (MCA) territory (25%), small vessel intracranial disease (25%), cardiac embolism (20%), other specified rarer causes (5%), and unknown causes despite investigation (25%) [1, 7].

5.4 Internal Carotid Artery Stenosis

Stenosis of ICA is defined as the presence of a plaque—lipidic, fibrotic, calcific, or mixed—that narrow the ICA lumen. Carotid artery atherosclerosis represents the major clinical risk factors for ischemic stroke. Overall, about 10–15% of all strokes follow thromboembolism from a previously asymptomatic ICA stenosis >50% [1].

5.4.1 Etiology

The main risk factors for developing carotid stenosis are the same as for atherosclerotic and other arterial disease. Selective screening for asymptomatic carotid stenoses may be considered in patients with multiple vascular risk factors [1].

5.4.2 Asymptomatic and Symptomatic Patients

Patients with carotid stenosis have an overall increased risk of cardiovascular events. Extracranial carotid stenosis usually occurs without symptoms until the stroke begins.

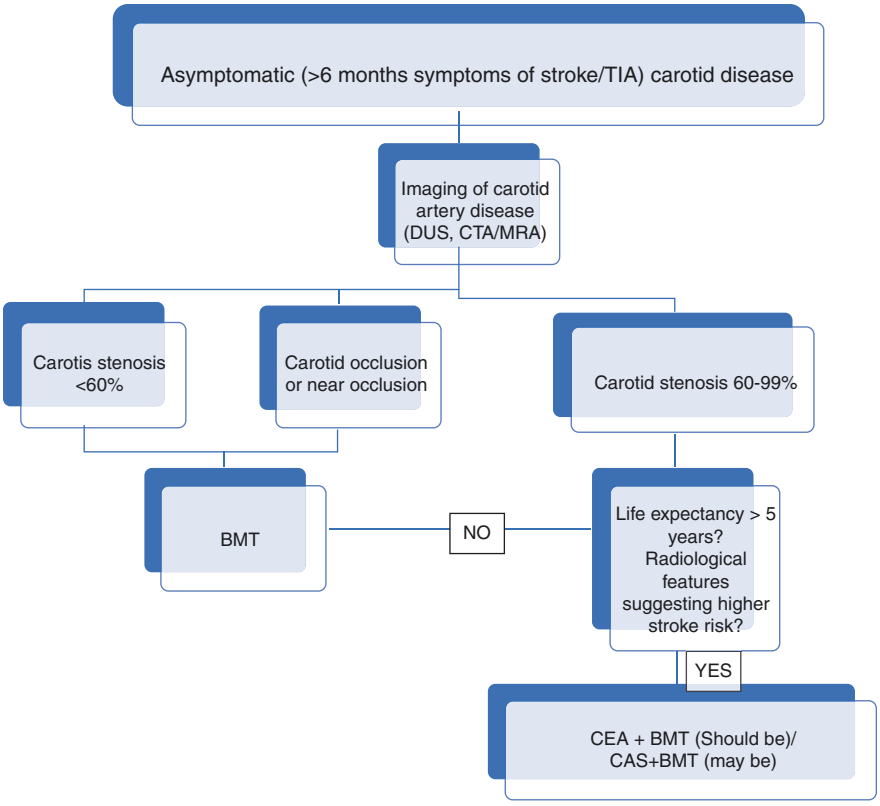

Fig. 5.2 Management of asymptomatic carotid stenosis

The presence of "significant" (degree of stenosis 60–99%) carotid bifurcation disease is a target for surgery. They are not an emergent/urgent condition and can be managed by their medical doctor. Figure 5.2 shows the management of asymptomatic patients. In patients with neurological symptoms (symptomatic patients) the presence of carotid bifurcation disease should be detected, while its absence should lead to a search for another source of symptoms. Symptomatic patients always represent an emergent/urgent condition and must be promptly managed in the emergency room. Figure 5.3 shows the flowchart to management of symptomatic patients.

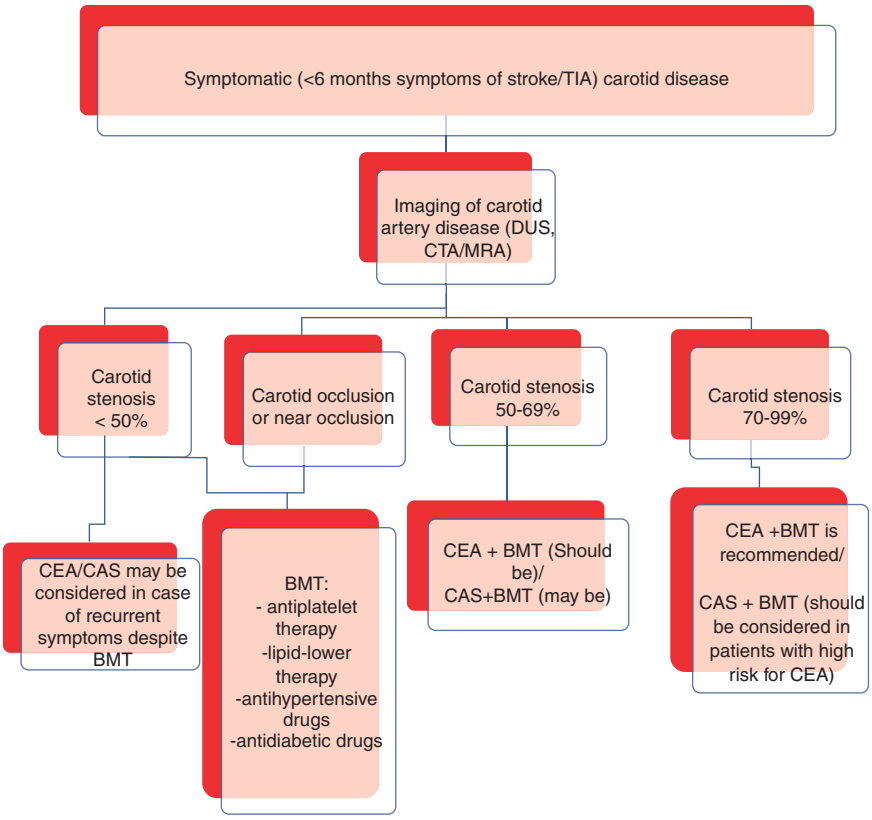

Fig. 5.3 Management of symptomatic carotid stenosis

5.4.3 Clinical Presentation

Symptoms related to stroke or TIA conditions depend on the extent and area affected by the ischemia. For this reason, there are symptoms related to the carotid territory (brain hemisphere, parietal area, temporal or frontal area) and symptoms related to the vertebro-basilar territory (see paragraph 7) (occipital area, brain stem, cerebellum). Carotid territory symptoms include:

- Hemi-sensory impairment, as numbness, paresthesia of face, arm, leg or all the soma.
- Hemimotor deficits (weakness of face/arm/leg, or limb clumsiness).
- Higher cortical dysfunction (dysphasia/aphasia, visuospatial problems).
- Amaurosis Fugax (transient monocular blindness): transient impairment or loss of vision in one eye.
- Retinal infarction (analogous to stroke): permanent visual loss.

- Ocular ischemia syndrome is nearly always associated with severe extracranial ICA stenotic/occlusive disease, although if collateralization via the circle of Willis is extremely poor, it can occur in patients with 50% stenoses [1].

5.4.4 Diagnosis

Color Duplex ultrasound (CDUS) scan is recommended as a first-line method for identifying and assessing the extent and severity of extracranial carotid stenosis [1]. When diagnosis is uncertain or when considering treatment, the estimate of Duplex ultrasound stenosis should be confirmed by computed tomographic angiography (CTA) or magnetic resonance angiography (MRA). Catheter angiography is now rarely required unless there are discrepancies on non-invasive imaging [1].

5.4.5 Treatment

In patients with asymptomatic carotid stenosis, secondary prevention with control of risk factors and the **best medical therapy (BMT)** (antiplatelet therapy, lipid-lowering therapy, antihypertensive treatment, and strict glycemic control in diabetic patients) are mandatory. In addition to BMT, the treatment options for treating carotid stenosis are carotid endarterectomy (CEA) and carotid stent (CAS). European guidelines suggest CEA should be considered in patients with asymptomatic 60–99% stenosis and imaging characteristics that may be associated with an increased risk, provided documented perioperative stroke/death rates are <3% of late ipsilateral stroke and the patient's life expectancy exceeds 5 years. Carotid stenting may be considered in selected asymptomatic patients who have been deemed by the multidisciplinary team to be "high risk for surgery": clinically significant cardiac disease (congestive heart failure, abnormal stress test, or need for open-heart surgery); severe pulmonary disease; contralateral carotid occlusion; contralateral laryngeal nerve palsy; previous radical neck surgery, cervical radiation therapy; recurrent stenosis after CEA and age >80 years [1]. The American Heart Association has repeatedly advised that only "highly selected" asymptomatic patients should undergo CEA but never defined what "highly selected" means [1].

In patients with symptomatic carotid disease, the goal of treatment is tertiary prevention to reduce the risk of recurrent TIA or stroke. Following an ischemic stroke, the risk for a recurrent ischemic stroke is 2% at 7 days, 4% at 30 days, 12% at 1 year, and 29% at 5 years. The risk of death after an initial ischemic stroke is 7% at 7 days, 14% at 30 days, 27% at 1 year, and 53% at 5 years [2].

Figure 5.3 shows the algorithm detailing the management of symptomatic carotid stenosis.

The risk of recurrent stroke is highest in the first few days/weeks after symptom onset. It's important to start mono or dual antiplatelet therapy (DAPT) early [1].

In patients with carotid stenosis 50–99% CEA or CAS are recommended. At the 2-year follow-up, there was a highly significant reduction in ipsilateral stroke incidence (9% vs. 26%) in patients who underwent CEA [2].

According to European guidelines in patients with recent (<6 months) symptomatic 70–99% carotid stenosis CEA is recommended, while it should be performed in patients who have 50–69% carotid stenoses and who are aged >70 years [1]. CAS may be considered in patients who have 50–69% carotid symptomatic stenoses, according to American Heart Association and Society of Vascular Surgery [2].

In the scientific community, there is still an active controversy about the ideal timing of intervention.

There is increasing evidence that CEA confers maximum benefit if performed <14 days, after 48 h had elapsed, each of the national audits showed that CEA could be performed within 3–7 days and 14 days with low procedural risks [1]. Performing CAS in the early time period after symptom onset is controversial and the literature contains conflicting data [1, 2]. When revascularization is not recommended? Revascularization should be deferred in patients with 50–99% stenoses who suffer a disabling stroke (modified Rankin score ≥ 3), whose area of infarction exceeds one-third of the ipsilateral middle cerebral artery territory, or who have altered consciousness/drowsiness, to minimize the risks of postoperative parenchymal hemorrhage. Patients with 50–99% stenoses who present with stroke-in-evolution or crescendo transient ischemic attacks should be considered for urgent carotid endarterectomy, preferably <24 h [1].

What to do when patients underwent intravenous thrombolysis? Early carotid endarterectomy (within 14 days) should be considered after intravenous thrombolysis in symptomatic patients if they make a rapid neurological recovery (Rankin 0–2), the area of infarction is less than one-third of the ipsilateral middle cerebral artery territory, a previously occluded middle cerebral artery mainstem has recanalized, there is a 50–99% carotid stenosis and no evidence of parenchymal hemorrhage or significant brain edema [1].

5.4.5.1 Complication Following Carotid Intervention

Complication after carotid surgery can be classified as local or systemic, early or late. Figure 5.4 shows the management of complication following carotid intervention.

- Stroke: emergency extracranial and intracranial CTA should be performed in patients who experience early stroke.
- Renal dysfunction: CAS can cause contrast-induced nephropathy. Adequate fluid resuscitation is recommended to avoiding acute kidney injury.
- Neck hematoma: If complicated by stridor or tracheal deviation, immediate evacuation is mandatory [1].
- Cranial nerve injury: It is the most common neurologic complication of CEA. The cranial nerve injuries can be managed expectantly.
- Prosthetic patch infection.

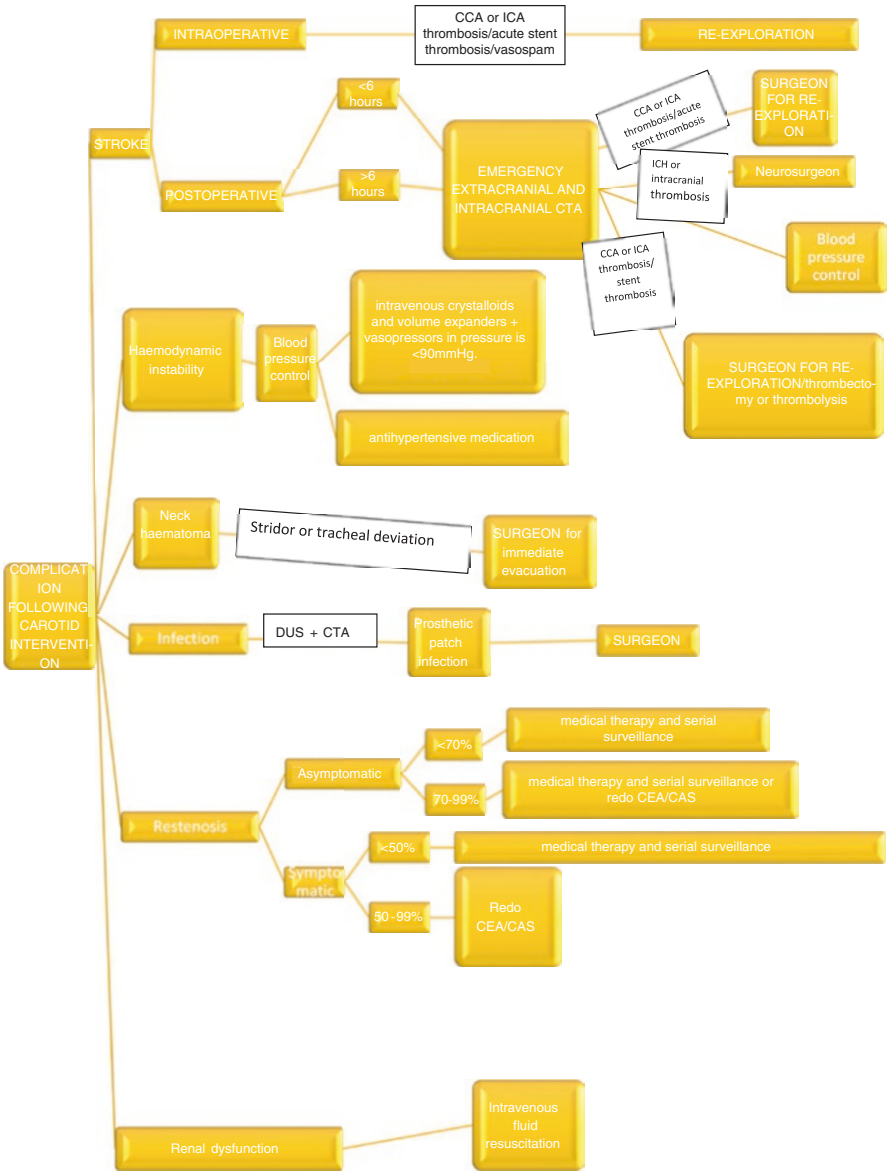

Fig. 5.4 Management of complication following carotid interventions. *CCA* common carotid artery, *ICA* internal carotid artery, *ICH* intracerebral haemorrhage, *DUS* doppler ultrasound, *CTA* computed tomography angiography, *CEA* carotid endarterectomy, *CAS* carotid artery stenting

- Restenosis can occur asymptomatically or symptomatically. For asymptomatic restenosis >70% serial surveillance and medical therapy is recommended; CEA or CAS may be considered for 70–99% stenosis following MDT review. For symptomatic restenosis 50–99% redo CEA/CAS should be considered, while symptomatic <50% restenosis should be treated medically [1].

5.5 Carotid Artery Dissection

Carotid artery dissection is a relatively uncommon pathology that can be manifest spontaneously without a clear etiology. Cervical carotid artery dissections are responsible for only 2% of all ischemic strokes but account for 10–20% of strokes in young and middle-aged patients [2].

5.5.1 Etiology

The etiology is unclear. It is documented that atherosclerosis and other known risk factors for vascular disease are usually absent in patients with spontaneous carotid artery dissection. Fibromuscular dysplasia, Ehlers–Danlos syndrome, cystic medial necrosis, Marfan syndrome, autosomal dominant polycystic kidney disease, and osteogenesis imperfecta type I have been implicated [2, 8]. Spontaneous carotid artery dissection may manifest as an extension of the aortic type A dissection.

5.5.2 Clinical Presentation

The most common initial symptom in patients with spontaneous dissection of the carotid artery is headache, partial unilateral Horner syndrome without facial anhidrosis and hemispheric symptoms. Patients with cervical carotid artery dissection also suffer from neck pain, amaurosis fugax, anisocoria, pulsatile tinnitus, and cranial nerve palsy (cranial nerves IX and XII). The prognosis after stroke caused by dissection is worse than the prognosis after stroke caused by atherosclerosis [2].

5.5.3 Diagnosis

DUS is the first-line diagnostic methods but CTA and angiography are the gold standards.

5.5.4 Treatment

Medical therapy is focalized on antithrombotic therapy to reduce stroke recurrence and clinical worsening. Indications for surgical or endovascular treatment of acute carotid artery dissection are fluctuating or deteriorating clinical neurologic symptoms despite medical treatment. Indications for surgery after 6 months of medical treatment are persistent high-grade stenosis and a new or persistent aneurysm greater than twice the diameter of the normal internal carotid segment [2]. The treatment available are surgical or endovascular approach. There are no randomized trials comparing open and endovascular treatment of patients with acute or late sequelae of carotid dissection, and no clear indications regarding when either approach might be preferred [2].

Figure 5.5 shows the management of patient with recent stroke.

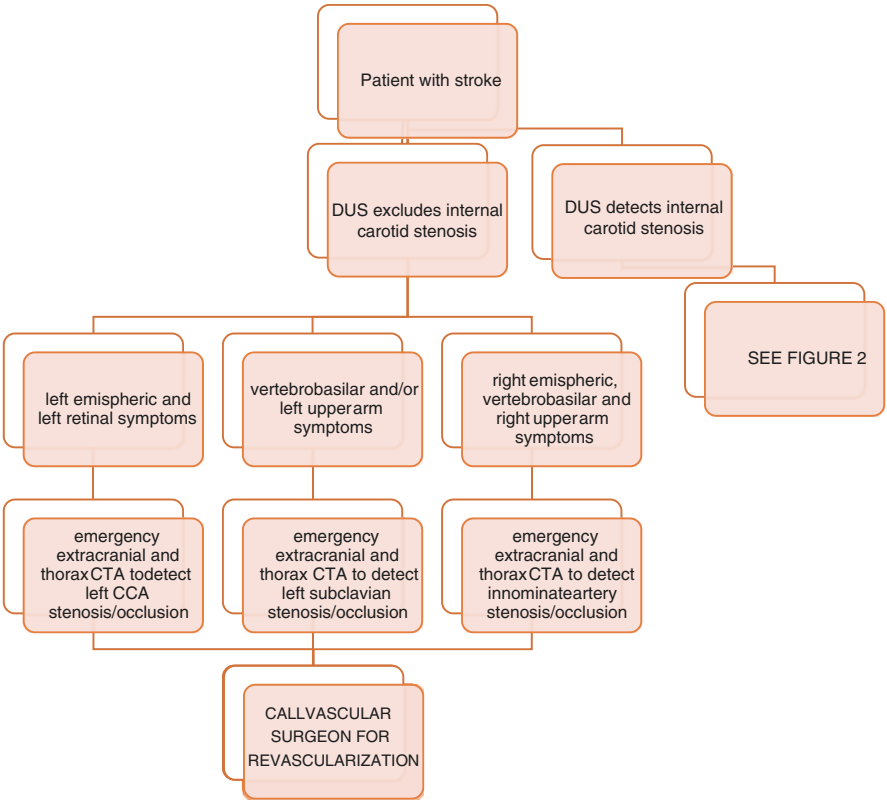

Fig. 5.5 Management of patients with stroke symptoms

5.6 Vertebral Artery Disease

Vertebrobasilar ischemia is less common than ischemic episodes related to internal carotid artery disease, approximately 25% of all ischemic strokes do occur in the distribution of the posterior brain circulation [2].

5.6.1 Etiology

The causes of vertebrobasilar stroke/TIA include low-flow hemodynamic ischemia, cardioembolism, large artery thromboembolism, small artery disease and dissection; atherosclerosis of the vertebral or basilar arteries accounts for 20–25% of strokes [1, 2].

The surgical anatomy of the paired vertebral arteries has traditionally been divided into four segments: segment V1 includes the origin of the vertebral artery as it arises from the subclavian artery to the point at which it enters the C6 transverse

process. This is the most common site of stenosis due to atherosclerotic lesions. Segment V2 includes the segment of the artery buried deep within the intertransversarium muscle and the cervical transverse processes of C6 to C2. This site is unfrequently involved by atherosclerotic lesions but it is a frequent site for the development of traumatic or spontaneous arteriovenous fistulas and pseudoaneurysms secondary to trauma or dissection. Segment V3 includes the segment of the vertebral artery that extends from the top of the transverse process of C2 to the atlanto-occipital membrane at the base of the skull. The most common problems at the V3 level are related to trauma and arterial dissection. Segment V4 includes the intracranial, intradural portion of the vertebral artery beginning at the atlanto-occipital membrane and terminating as the two paired vertebral arteries that converge to form the basilar artery. It is infrequently affected by atherosclerosis but is prone to arteriovenous fistula formation and aneurysmal degeneration [2].

5.6.2 Clinical Presentation

Vertebrobasilar symptoms include vertigo, ataxia, eye movement disorders, bilateral limb weakness, complete visual loss (cortical blindness), hemianopia, unilateral weakness or numbness, dizziness, dysarthria, headache, and nausea/vomiting [1].

5.6.3 Diagnosis

DUS is the first-line imaging method. DUS must be followed by contrast enhanced magnetic resonance angiography (CEMRA) or CTA [1].

5.6.4 Treatment

Asymptomatic vertebral artery atherosclerotic lesions should not be treated by open or endovascular interventions. BMT is recommended.

When vertebrobasilar symptoms occur, BMT plus vertebral artery revascularization is recommended early after symptoms appear for 50–99% extracranial vertebral artery stenosis. In fact, stenoses of the vertebral arteries are associated with high early recurrence stroke rates. Surgical approaches to lesions at the vertebral artery origin include transposition to the ipsilateral CCA, vertebral artery reimplantation, and vein bypass grafting from the subclavian artery. Few data are available for endovascular treatment [9]. Figure 5.6 shows management of vertebrobasilar symptoms onset.

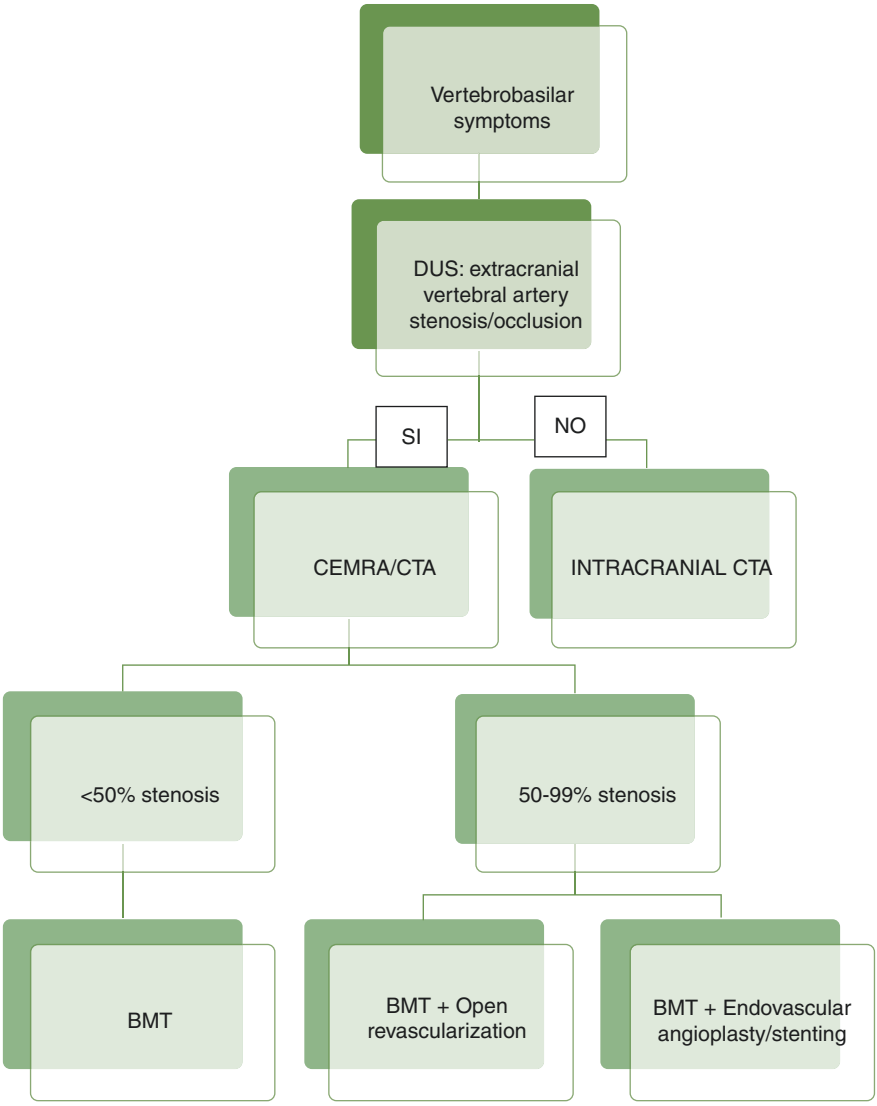

Fig. 5.6 Management of patient with vertebro-basilar symptoms

5.7 Vascular Injuries to the Cervical Vessels

Traumatic injuries to the cervical vessels are relatively uncommon and constitute only about 5–10% of all vascular injuries. The patients are mostly young, and despite the low incidence, mortality and morbidity are between 5% and 40%, and persistent neurological consequences are reported in up to 80% of patients [10].

5.7.1 Etiology

The most common mechanism is penetrating injuries. Blunt is thought to be less than 0.5% of all blunt traumas to the body, but recent reports indicate that many blunt vascular injuries go undetected [2].

The common carotid is the most frequently injured major artery. The internal carotid artery is involved in more than 90% of blunt injuries, most commonly its distal parts. Injuries to the vertebral artery are less common because they are well protected by osseous structures with an incidence of 0.20–0.77% of all trauma admissions [2, 10]. Injuries to the thoracic outlet are extremely lethal. Prehospital mortality is 50–80%, and of those who survive transport, 15% die during treatment [2].

5.7.2 Clinical Presentation

In case of penetrating trauma, bleeding is the most frequent sign. In a penetrating trauma there are "hard signs" and "soft signs" of vascular injury. Hard signs include shock, refractory hypotension, pulsatile bleeding, bruit, enlarging hematoma, and loss of pulse. Soft signs include history of bleeding at the scene of injury, stable hematoma, nerve injury, proximity of injury track, and unequal upper extremity blood pressure measurements [2]. In blunt carotid injuries, headache and/or cervical pain are the most common symptoms, followed by symptoms indicating cerebral or retinal ischemia due to embolization or thrombotic occlusion. A frequent type of symptom is a typical transient ischemic attack but complete stroke or amaurosis fugax also occurs. Horner's syndrome may be present if superficial cervical ganglion lesions coexist. In cases of vertebral artery blunt injuries vertebrobasilar ischemia may be manifested with dizziness, vertigo, nausea, tinnitus, dysarthria, dysphagia, ataxia, visual deficits, and hoarseness.

Secondary damage to the aerodigestive tract with dyspnea or stridor (for example, compression of the airways by a large expanding hematoma) may be associated [10].

Difficulty swallowing, vocal cord paresis, hoarseness, inability to shrug, and weakness may be present in case of cranial nerve injuries.

5.7.3 Diagnosis

The diagnosis of penetrating neck injury is usually obvious because bleeding is present. However, it is important to note the extent of the trauma. In fact, mild external signs of trauma can be associated with severe vascular injury or expanding hematomas. The location of penetrating cervical injuries are generally divided into three different zones. Zone I extends inferiorly from 1 cm above the manubrium to include the thoracic outlet; zone II extends from the upper limit of zone I to the

angle of the mandible; and zone III is between the angle of the mandible and the base of the skull. Zone II is the most commonly injured (47%), followed by zone III (19%) and zone I (18%) [2]. In zone II traumas if patients are stable and asymptomatic angiography, duplex ultrasound, and CT may be useful to rule out vascular injuries. Exploration and the possibility of obtaining control are much easier in zone II injuries. Injuries not penetrating the platysma need no further vascular evaluation. In stable patients with zones I and III trauma CTA or DSA is always indicated. All patients with neck trauma should have plain radiographs of the chest to rule out occult hemothoraces, pneumothoraces, or aortic arch injury [10].

In patients admitted to ER for blunt neck trauma, it is important to perform a careful neurological examination to obtain a baseline for later comparisons. In fact half of the patients develop neurological symptoms within 24 h [10].

DUS can be considered as a screening method but in case of a negative study CTA or digital subtraction angiography (DSA) is recommended. However, DSA has several limitations such as invasiveness that carries a risk of stroke (<1.0%) and cost [2].

5.7.4 Treatment

In the emergency scenario, the airway and breathing have priority, followed by the control of bleeding in case of penetrating trauma. Patients with shock, active brisk bleeding, rapidly expanding hematoma and those with neurological deficit or severe airway obstruction should be transported to the operating room for immediate exploration and treatment. Repair is recommended in all patients with penetrating carotid injuries and the patient has no major neurological symptoms. For minor injuries to the carotid artery, including those with small but adherent intimal flaps, defects, or pseudoaneurysms <5 mm in size, repair is recommended in symptomatic patients. If the patient is asymptomatic and there is no ongoing active bleeding, a conservative approach with antithrombotic therapy and early follow-up imaging has proven to be a safe alternative [2, 10].

In blunt trauma for asymptomatic injuries, including dissection, antithrombotic therapy is usually the only treatment needed and is indicated to prevent thrombosis of the injured segment and/or embolization from it.

Endovascular therapy has primarily been reserved for evolving dissections that are surgically inaccessible, pseudoaneurysms that persist or enlarge after antithrombotic treatment, or patients with worsening neurologic symptoms on medical therapy [2]. Based on the location of these injuries, endovascular therapy offers several advantages over open repair. However injuries that appear to be easily accessible during surgery and are symptomatic should be considered for repair while injuries located at the base of the skull, the only option for treatment may be endovascular exclusion [2, 10].

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Upper Airways Tract in Emergency Settings

6

Diotti Cristina and Francesco Damarco

6.1 Hemoptysis

Hemoptysis is defined as the expectoration of blood from the airways; it should be distinguished from hemoptoe, defined as coughing of sputum with blood.

Its annual incidence is around 0.1% in out-patients, representing 0.2% of causes of hospitalization; 10% of all episodes of hemoptysis occurs in patients with chronic lung diseases [1]. It generally affects adults aged around 60–70, although in rare cases it can occur in children.

Hemoptysis can be classified as mild (<20 mL in 24 h), moderate (20–600 mL in 24 h), and massive (>600 mL in 24 h) depending on the amount of blood.

More than 90% of episodes are self-limiting, but hemoptysis can be a life-threatening condition requiring an immediate intervention due to gas exchange impairment and asphyxia.

Blood in the airways can cause respiratory failure due to impaired gas exchange: since tracheobronchial dead space volume is around 150–200 mL, a bleeding volume >100 mL can lead to severe airway obstruction and cause asphyxia [2].

However, life-threatening conditions may occur even with a smaller amount of blood, in case of reduced coughing reflex or previous impaired lung function.

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6.1.1 Etiology

Bronchial arteries originate from the systemic circulation, generally from the descending thoracic aorta more commonly between T5 and T6, but aberrant bronchial vessels may arise from other systemic arteries (e.g., aortic arch, subclavian artery, internal mammary artery).

Some conditions (Table 6.1), such as chronic inflammation, neoplastic diseases, or hypoxic vasoconstriction, lead to the release of angiogenetic factors that promote vascular proliferation: in these cases bronchial arteries become thin and fragile and may rupture under systemic pressure, causing airway bleeding. In more than 90% of cases, bleeding arises from bronchial circulation; in only 5% of cases it originates from pulmonary vessels [1, 3, 4].

Tuberculosis is the most common cause of hemoptysis worldwide [5], but in about 40% of cases, the underlying cause of hemoptysis cannot be identified and remains unknown.

6.1.2 Clinical Presentation

In patients presenting with blood expectoration, an accurate medical history should be taken to define the amount of expectorated blood, the duration of symptoms, previous episodes, smoking history, active medications, and underlying diseases (lung, cardiac, malignancies, etc.). Hemoptysis should be distinguished from other causes of bleeding from the gastroenteric tract (hematemesis) or the upper respiratory tract (mouth, gums, nose, and pharynx).

A quick initial clinical evaluation based on patient's vital parameters and oxygenation is aimed at detecting any life-threatening situation, in particular an impaired gas exchange, associated with cyanosis, dyspnea/tachypnea, and potential loss of consciousness. In these cases, an immediate intervention is mandatory to restore gas exchange, clear the blood form the airways, and avoid death.

Considering the different causes of hemoptysis, the physical examination can show signs of pulmonary (e.g., altered breath murmurs, wheezes, crackles, pleural friction) or cardiovascular disease (e.g., heart murmur in case of mitral stenosis, hepatomegaly, and superior vena cava syndrome).

Table 6.1 Main causes of hemoptysis

Airway or lung infections (mainly tuberculosis and aspergillosis, that may be also associated with vascular erosion causing massive bleeding)
Inflammatory airway or lung diseases (e.g., COPD)
Bronchial primitive malignancies or metastatic diseases
Bronchiectasis, cystic fibrosis
Increased pulmonary venous pressure (e.g., pulmonary edema, mitral stenosis)
Pulmonary artery embolism
Iatrogenic causes (e.g., anticoagulation therapies, transbronchial biopsies, foreign bodies, surgical lung resections)
Other causes (e.g., vasculitis, vascular malformations, pulmonary hypertension, hemosiderosis, NSCLC treatment with bevacizumab)

6.1.3 Diagnosis

Once defined the grade of hemoptysis and managed possible life-threatening situations, diagnostic procedures include:

- Blood tests with complete blood count (CBC), coagulation tests, biochemistry and inflammation parameters.
- Chest X-ray: can show lung or airway lesions/masses, atelectasis, air bronchograms, pleural effusion, cardiomegaly.
- Multislice contrast-enhanced CT scan with CT angiography is useful in case of doubt, massive or recurrent hemoptysis. This procedure can localize the site and cause of hemoptysis, reveal active spots of bleeding and help plan a possible embolization. Bronchial systemic arteries are considered pathological if >2 mm in diameter and have a tortuous pathway.
- Bronchoscopy: useful in case of active bleeding, it can be performed with flexible or rigid instruments with both diagnostic (microbiological/cytological/histological samplings) and therapeutic purposes. Flexible bronchoscopy can be performed at bedside, it is easier to handle and enables an accurate inspection of the tracheobronchial tree; rigid bronchoscopy should be performed by highly trained specialists in the operating room under general anesthesia and does not allow the exploration of segmental bronchi.

6.1.4 Treatment

In case of mild or moderate self-limiting hemoptysis, conservative treatment and management of the underlying disease is the treatment of choice. Tranexamic acid (1–1.5 g bid or tid) can be administered orally [6] and further investigations on the etiology may be performed in outpatient setting.

In case of active bleeding with stable vital parameters, hospitalization is recommended and general indications for treatment include:

- Lateral decubitus with bleeding site down, if known, to protect airways and avoid aspiration into the non-affected lung.
- Monitor vital signs and get an IV access.
- Antifibrinolytics agents may reduce duration and volume of bleeding, with a low short-term risk of thromboembolism [6].
- Empiric broad-spectrum antibiotic therapy can be administered if an infective cause is suspected.
- Bronchoscopy may be able to remove the blood, to identify the site, and to stop the source of bleeding. Endobronchial instillation of vasoconstrictive agents, cold saline solution, laser photocoagulation, or argon plasma electrocoagulation are also useful. Temporary occlusion of the segmental bronchus with a balloon catheter is suitable in case of peripheral bleeding.
- Angiographic bronchial artery embolization (BAE) can be performed to stop the bleeding using metallic spirals or liquid agents. It is a safe non-surgical proce-

dure with high success rates. The main side effects of BAE are chest pain end transient dysphagia; serious complications include spinal cord ischemia and transverse myelitis if arteries supplying the spine are accidentally involved in the embolization.

- Surgery is indicated in case of necrotizing tumors, cavernous tuberculosis or aspergillomas, unsuccessful BAE or in rare cases of traumatic/iatrogenic events of bleeding. The procedure should be performed as an elective surgery, after stopping the bleeding and planning the extent and the feasibility of the lung resection.
- Emergency surgical treatment is reserved to extreme cases of uncontrolled hemoptysis and is associated with a high mortality rate (up to 50%) due to the patient's critical conditions related to hemodynamic and respiratory instability.

Algorithm 6.1 Algorithm for diagnosis and treatment of hemoptysis.

Box 6.1 Massive hemoptysis [3]
Life-threatening hemoptysis may be defined as any hemoptysis with:

- >100 mL in 24 h
- Abnormal gas exchange/airway obstruction
- Hemodynamic instability [4]

If the patient cannot eliminate the blood from the airways, is unconscious and in severe respiratory failure:

- immediate orotracheal intubation with a large diameter tube (8–9 mm) to protect the airways and ventilate the patient. Selective bronchial intubation of the non-affected side can be performed with endotracheal tube, bronchial blockers, or Fogarty catheters.
- Patient should be monitored in the ICU.
- Blood and volume supply can be administered in case of hypovolemia.
- Flexible bronchoscopy through the tube or rigid bronchoscopy in combination with flexible bronchoscopy should be performed to maintain ventilation, remove blood and clots, identify the bleeding source, and obtain hemostasis.
- Investigate the causes and treat the underlying disease.
- Once stabilized the patient, in case of persistent bleeding, BAE or surgical resection may be considered as alternative therapeutic options.

6.2 Airway Obstruction

Airway obstruction is a potential life-threatening obstacle to the airflow and to ventilation due to narrowing or occlusion of the airways.

It may be classified as acute or chronic, and be partial or complete, depending on how much caliber of the airway is obstructed. Acute obstruction can be fatal in a few minutes, compromising ventilation and causing asphyxia, and requires an immediate intervention.

6.2.1 Etiology

Common causes of airway obstruction are foreign bodies, benign or malignant endotracheal lesions, tracheal stenosis occurring after prolonged intubation or tracheostomy, edema, infections and abscesses, blunt or penetrating traumas and airway compression from extraluminal lesions (ab-extrinseco compression). Some anatomic variants can contribute to the obstruction.

6.2.2 Clinical Presentation

Clinical presentation varies according to the site and the degree of obstruction. Chronic or partial airway obstruction is a non-deferrable urgency, presenting with dyspnea, accessory respiratory muscle use, laryngeal stridor, abnormal breath sounds, and cough.

Acute airway obstruction is an emergency setting, associated with breathlessness, acute respiratory distress, cyanosis, altered consciousness, and agitation. Often, the history of the event cannot be told by the patient and it may be reported by bystanders or family members. A rapid clinical evaluation should include vital parameters detection and exploration of the upper airways (nose, throat, pharynx, head, and neck) and should be focused on identification of the possible causes of obstruction.

Resuscitation and airway protection must be promptly carried out: the equipment should include devices for nasotracheal or endotracheal intubation and surgical airway supplies.

6.2.3 Diagnosis

The exploration of the upper airways can be performed using nasal speculum, a laryngoscope or via direct visualization. Other diagnostic tools (imaging techniques, endoscopic procedures) should not delay the intervention in case of acute distress and may be helpful to identify the etiology once the patient is stable.

Imaging techniques include head-neck and chest X-ray, CT scan, and MRI: in particular, CT scan is rapid, widely available and safe and provides information on airway structure and diameter and possible causes of obstruction.

Examination should identify the type and size of the obstruction, in order to plan the most effective intervention.

6.2.4 Treatment

In case of acute obstruction, airways need to be secured by the members of the emergency staff involved. High-flow oxygen supply can temporarily increase airway patency with positive pressure and improve oxygenation during airway intervention.

Procedure to secure the airways in acute life-threatening situations includes:

- Awake fiberoptic intubation (AFOI) allows airways intubation maintaining spontaneous breathing and avoiding the obstructing. This technique is useful in case of supraglottic or glottic obstructions. Temporary complete obstruction of the airways caused by the scope should be considered.
- Awake videolaryngoscopy is useful in case of laryngeal obstruction, is more familiar with most anesthetists and it has same advantages of AFOI.
- Awake tracheostomy via percutaneous or surgical technique is the gold standard management in acute upper airways obstruction but it can be difficult to perform without sedation in critical situations.

6.2.5 Main Causes of Airway Obstruction

6.2.5.1 Foreign Bodies' Inhalation

Foreign body (FB) inhalation is a common event in children, but many cases in adults and elderly age are reported, mainly associated to dementia, alcohol consumption, or poor dentition. In the USA, the mortality rate in kids <4 years old is about 7%. Most frequently inhaled objects are food (peak incidence 1–2 years of age), coins, small toys, seeds, nuts, and balloons, but in adults also medical instruments, dental prostheses, and bones are common.

Especially in children, the patient is unable to explain what is happening, the event of inhalation may be not immediately recognized, and the aspirated object can escape detection of adults.

The aspirated object can cause various degrees of airway obstruction depending on location and shape: partial occlusion can cause chronic symptoms, while complete occlusion of the airway leads to sudden death. In case of complete obstruction, the FB lying in the larynx or trachea causes acute choking and asphyxia: in this scenario, an immediate intervention at the scene with the Heimlich maneuver, back blows, chest or abdominal thrusts can be life-saving.

If the FB obstruct the vocal cords and prevents endotracheal intubation, an emergency surgical or percutaneous airway is necessary: tracheostomy or needle cricothyroidotomy is preferred in children (the latter is avoided in children <8 years old).

If the FB is peripheral to the carina, the location in the bronchial tree depends on the size of the object and the endobronchial structure: in children <15 years, left and right mainstem bronchi have similar angulation and FB are found almost equally in the two sides. Growing up, the right mainstem bronchus becomes linear and FB are more frequently located on this side [7].

When the object lies in a lobar or sublobar bronchus, it causes inflammation, edema, and granulation reaction that can make the identification and removal of the FB harder. In particular, vegetables may swell in hours or days, thus worsening the obstruction: some organic materials, such as peanuts, release acid particles that can induce inflammation, edema, and erosion. The procedure of removal may be complicated by bleeding and mediastinitis or bronchial fistulae may occur.

Typical signs and symptoms in the acute phase include cough and air trapping and local emphysema distal to the obstruction due to a mechanism that prevents air outflow. In the chronic phase recurrent infections (post-obstructive pneumonia, lung abscesses) atelectasis, bronchiectasis, and hypoxic vasoconstriction are the most common clinical signs. Rates of serious complications arise up to 2.5 times if the diagnosis is >24 h after the inhalation. In case of non-specific symptoms, the obstruction can be misdiagnosed and interpreted as asthma or chronic bronchitis.

Diagnostic tools include:

- Chest X-ray: Less than 20% of FB are radio-opaque, but some indirect signs of FB inhalation, such as air trapping, monolateral hyperinflation, obstructive emphysema, mediastinal shift, lobar atelectasis, or pneumomediastinum may be recognized.
- CT scan allows the identification of the obstruction in the tracheobronchial tree and localized areas of air trapping or atelectasis.
- Rigid bronchoscopy is the gold standard for the airway protection, diagnosis and treatment of FB inhalation. Flexible bronchoscopy alone should not be performed because of the high risk of dislocation of the FB during the procedure [8].

A clinical observation of 24–48 h is recommended after the extraction in order to manage possible complications, such as re-expansion pulmonary edema, inflammation, hemoptysis, fever, up to ventilatory failure requiring ICU monitoring. In some cases, repeated endoscopic procedures may be necessary.

6.2.5.2 Tracheal Stenosis

Tracheal stenosis often presents as a circumferential lesion caused by tissue reaction after prolonged intubation, injuries, or radiation therapy. In rare cases it may be congenital, when abnormal cartilages alter tracheal structure.

Clinical presentation includes characteristic inspiratory laryngeal stridor, in addition to typical symptoms of airway occlusion.

Diagnostic evaluation should comprehend:

- Imaging tests (chest X-ray, CT scan with 3D reconstructions, MRI) provide information about the location and the extension of the stenosis and allow an adequate planning for invasive procedures.
- Flexible bronchoscopy is the gold standard procedure for the diagnosis of tracheal stenosis. It allows to evaluate the caliber of residual lumen and to plan the proper treatment [9].

Treatment options include:

- Observation in case of mild stenosis without relevant symptoms. Especially in children, mild stenosis tends to improve with growing.
- Non-severe stenosis can be treated endoscopically using a balloon dilation procedure, with or without laser radial cutting of the scar tissue. Recurrence of scar tissue can be avoided with topical instillation of steroids during the procedure [9].
- In cases of severe or recurrent stenosis, tracheal surgery is the treatment of choice. If few tracheal rings are involved, the resection of the stenotic tract and reconstruction by direct termino-terminal anastomosis can be performed; in case of long-segment tracheal stenosis, a slide tracheoplasty is required [10].

6.2.5.3 Malignant Airway Obstruction

Airway obstruction can be a manifestation of advanced lung/tracheobronchial cancer or a metastatic disease that causes a mechanical obstruction to the airflow. Its clinical presentation is usually late in the course of the disease and can be a cause of death.

Malignant airway obstruction can result from a direct invasion of the tracheobronchial lumen (intrinsic obstruction) or a compression from an extraluminal growing disease (ab-extrinseco compression). In some advanced cases, both conditions may contribute to the obstruction.

The most common causes of malignant airway obstruction are bronchogenic carcinomas (usually squamous cell lung cancer), or adjacent malignancies invading the airways (esophageal, laryngeal thyroid). Primitive tracheal tumors are rare (about 0.6% of all respiratory tract malignancies) and mainly represented by squamous cell, adenoid cystic and mucoepidermoid carcinoma [11].

Endoluminal lesion from metastatic diseases are uncommon and can occur mainly in case of breast, renal colorectal cancer and melanoma.

About 20–30% of lung cancer patients may develop symptoms related to central airway obstruction and invasion of tracheobronchial lumen (dyspnea, atelectasis, localized wheezing, post-obstructive pneumonia, and hemoptysis) that can be difficult to manage in advanced oncological patients.

In case of suspected malignant airway obstruction, the past medical and oncological history should be carefully investigated. Generally, airway obstruction occurs in patients with a known, late-stage oncological disease, but sometimes airway obstruction is the first manifestation of the disease. After the acute management of airway obstruction, an adequate diagnostic workup should be assessed.

If chest X-ray is not diagnostic, chest CT scan has a higher sensitivity (up to 98%) in detecting the type, the length, and the diameter of the obstruction and the characteristics of the distal airways in order to plan the proper treatment.

Both flexible and rigid bronchoscopies are the procedure of choice for the diagnosis and treatment of malignant obstruction: the lesion and the distal airways can be directly visualized and tissue specimens can be collected for histological analysis.

Flexible bronchoscopy should be performed carefully in order to avoid bleeding or other serious complications leading to complete obstruction of the airways.

In urgent situation with severe respiratory distress, the priority is to secure the airways: in these patients, an immediate endotracheal intubation is required and, if possible, a rigid bronchoscopy performed by a skilled interventional pulmonology team. If the equipment is not available, the patient should be transferred as soon as possible to a specialized center.

Once stabilized the patient and defined the type of the obstruction, the treatment depends on the extent and the stage of the malignancy and patient's life expectancy.

Unfortunately, radical surgical treatment is often not viable in advanced malignant diseases and the management is focused on palliation of symptoms and quality of life improvement.

In these cases, interventional bronchoscopy is the treatment of choice [12] and includes:

- Mechanical debulking in case of intrinsic obstruction, using rigid bronchoscope to core the lesion, or endoscopic forceps to remove obstructive tissue, or microdebriders.
- Balloon dilation or bronchoplasty involving the use of increasingly larger balloons endoluminally can be performed both for intrinsic and extrinsic obstructions.
- Thermal ablation using YAG laser, argon-plasma, electrocautery, cryotherapy.
- Airway stent placement (silicone and/or metallic), more efficient in extrinsic compressions.
- Brachytherapy allowing endobronchial delivery of radiations via the placement of radioactive substances using a flexible bronchoscope.

These interventional procedures are usually performed via rigid bronchoscopy, that provides a better airway control in case of complications and allows ventilation during the procedure.

6.3 Tracheobronchial Injuries

Tracheobronchial injuries (TBI) are an uncommon but potentially life-threatening condition. They can be caused by penetrating or blunt traumas, or may be iatrogenic injuries occurring during endotracheal intubation, endoscopic and surgical procedures.

6.3.1 Etiology

Traumatic injuries may be caused by penetrating objects or by gunshots; knives usually cause lesions to the cervical trachea, though gunshot injuries can occur anywhere in the tracheobronchial tree.

Blunt traumas are often related to high-impact events on the chest that cause forces of traction on the tracheobronchial tree or to direct hits on the neck. Most blunt injuries involve the distal trachea and the right main bronchus [13].

Morbidity and mortality associated with traumatic tracheobronchial injures are elevated, also due to serious concomitant traumas on other injured organs (facial and head traumas, pulmonary contusions, intra-abdominal injuries, orthopedic fractures): patients with multiple traumas should be managed in a high-volume trauma center.

Iatrogenic injuries may be a complication of endotracheal intubation (especially with double lumen tubes), tracheostomy, intrathoracic, esophageal, laryngeal and neck surgery, or of endoscopic procedures (rigid bronchoscopy, EBUS, EUS). Some anatomical variants including congenital abnormalities (tracheal diverticula, Mounier-Kuhn syndrome) or distortions caused by neoplasms may be a risk factor for iatrogenic damages.

6.3.2 Clinical Presentation

TBI should be early treated due to the potentially severe complications related to a delayed diagnosis. Hoarseness, dysphonia, dysphagia, and stridor are often associated with a cervical trauma.

Subcutaneous emphysema, hemoptysis, pneumothorax, pneumomediastinum, and air leakage from a chest penetrating trauma are related to a deeper TBI.

Tracheobronchial traumatic injuries are often associated with other major traumas: in these cases some symptoms can be hidden and TBI can be misdiagnosed. If TBI is not promptly recognized, granulation tissue and healing processes can mask the lesion and late manifestations may occur: typical late findings are pneumonia, bronchiectasis, atelectasis, and abscesses due to tracheobronchial stenosis.

6.3.3 Diagnosis

Chest X-ray is a useful diagnostic tool and can show pneumothorax, pneumomediastinum, pneumopericardium, subcutaneous emphysema, mediastinal shift, tracheal deviation, and deep cervical emphysema. Complete separation of the mainstem bronchus may be associated with atelectasis and “fallen lung sign of Kumpe” (appearance of collapsed lung away from the mediastinum when the bronchus is completely detached while the vascular structures are intact) [14].

High resolution CT scan helps identifying the site and the extent of the lesion, as well as other major injuries to intrathoracic structures. However, CT scan is

contraindicated in hemodynamically unstable patients which require an immediate intervention.

Bronchoscopy is the gold standard procedure to directly identify the injury and provides information about the site and the extent of the lesion. In case of suspected esophageal involvement, esophagus should be investigated using EGDS or oral contrast imaging.

6.3.4 Management

Securing the airways is the first step in managing TBI, especially if associated with other major traumas: patients in acute respiratory distress should be immediately intubated under flexible bronchoscopy, if available. In case of open cervical traumas or craniofacial traumas in which orotracheal intubation is difficult, tracheostomy may be necessary to protect the airways. If the upper trachea is completely transected, intubation is challenging and the distal portion of the trachea should be palpated into the mediastinum and directly intubated from the cervical wound.

Intubation is preferably performed with single lumen tube; in some cases a long single lumen tube can be used in a mainstem bronchus to ensure single lung ventilation. Once secured the airways and stabilized the patient, the treatment is planned on extent, position, and type of TBI.

For superficial injuries, in clinically stable patients in spontaneous breathing without O_2 support conservative treatment with endoscopic regular follow-up is the preferable choice: injuries up to 4 cm can be treated conservatively [15].

In case of lesions of the tracheobronchial mucosa with mediastinal emphysema, antibiotic prophylactic therapy is still debated and the treatment of choice should be individualized according to clinical presentation and to the extent of the injury.

For complete tracheobronchial injuries involving mediastinum, esophagus, and soft tissues, a multidisciplinary evaluation is needed to determine the best therapeutic approach: both surgical and endoscopic treatments should be evaluated.

Emergency surgical intervention is required in patients with progressive deterioration of general conditions, air leak and failure in lung re-expansion despite chest drainage, increasing subcutaneous emphysema and pneumomediastinum, esophageal prolapse into the tracheal lumen and ineffective mechanical ventilation. Primary surgical repair is also indicated in iatrogenic injuries.

Surgical approach should be planned according to the position of the injury and the presence of other organs damaged [16]:

- If the lesion is in the proximal trachea (one/two cranial thirds), surgical repair can be performed via cervicotomy with or without incision of the manubrium.
- For distal tracheal lesions (up to 2 cm above the carina) or for bronchial lesions, right or left thoracotomy is the preferred surgical approach.
- Simple lacerations can be repaired using interrupted absorbable sutures.
- In case of more complex lesions, tracheobronchial resection and reconstruction with end-to-end anastomosis is preferable.

An alternative to surgical treatment is endoscopic placement of self-expandable stents, that mechanically cover the injury and promote healing processes and can be removed after 4–6 weeks.

A regular bronchoscopic follow-up should be planned after surgical repair of TBI, since one of the most common complications is tracheal stenosis.

6.4 Pneumomediastinum

Pneumomediastinum is a clinical condition associated with the presence of air in the mediastinum. It is a rare situation and can be related to an underlying cause (e.g., blunt or penetrating chest trauma, infections, iatrogenic injuries) or can occur spontaneously without triggering factors.

6.4.1 Etiology

Pneumomediastinum can occur as a complication of surgical or endoscopic procedures, after an intense Valsalva maneuver, vomiting or other causes of esophageal rupture (Boerhaave syndrome, caustic ingestion), increase in intra-abdominal pressure (pneumomediastinum during childbirth is known as Hamman's syndrome), and rapid ascend in scuba diving. It also may occur after a penetrating or blunt chest trauma causing airways injury. Asthma, COPD and other chronic lung diseases, tobacco and recreational drugs use are described as inducing factors. When a cause cannot be identified, pneumomediastinum is defined as spontaneous.

Possible explanations for pneumomediastinum are:

- Increase in alveolar pressure that can cause ruptures in alveolar structures and subsequent air leakage that reaches the mediastinum.
- Increase in mediastinal pressure that cause air passage in the mediastinal tissues, subjected to a negative intrathoracic pressure.

Although it is rare, it is more common in young, tall, and thin men and patients suffering from asthma or COPD [17].

6.4.2 Clinical Presentation

The main reported symptom is acute and sudden retrosternal chest pain referring to the neck or the back, often misdiagnosed as spontaneous pneumothorax or heart diseases.

Sometimes it arises following a causative factor such as cough, vomiting, and Valsalva maneuver.

Other common symptoms are coughing, dyspnea, neck pain, hoarseness, dysphagia, rhinolalia, and subcutaneous emphysema involving chest, neck, and face. Tachypnea and tachycardia may also occur.

Even if not frequent, Hamman's sign is a specific sound audible on the precordium.

In some cases, pneumomediastinum can be asymptomatic and the diagnosis is only radiological.

6.4.3 Diagnosis

Imaging diagnostic tools include:

- Chest X-ray shows air in the mediastinal borders making the mediastinal pleura visible. It can reveal an associated pneumothorax or pneumopericardium. Typical radiological findings are "thymic sail" (superelevated triangular-shaped inferior thymic margin due to air, common in children), "ring sign" (due to the air around pulmonary artery), double bronchial wall, and air near the diaphragm or the spine [17].
- High resolution CT scan provides information about the extent of the pneumomediastinum and about the possible causes.
- Endoscopic procedures (bronchoscopy, EGD) are not routinely required in case of spontaneous pneumomediastinum and should be performed only if an underlying cause related to esophageal or tracheobronchial injury is suspected.

6.4.4 Management

In most cases, spontaneous pneumomediastinum is a self-limiting condition and does not require specific treatment: 24 h in-hospital observation and a symptomatic therapy with analgesics and antitussives are recommended following diagnosis. Once excluded possible underlying diseases and if the clinical conditions are stable, the patient can be discharged with indication of bed rest. Air in the mediastinal space is slowly reabsorbed by surrounding tissues and conservative treatment is usually effective.

In case of associated pneumothorax, chest tube placement can be needed to avoid air accumulation in the pleural and mediastinal space.

In rare cases, air leakage can continue causing cardiac tamponade and mediastinal shift: in these cases, an emergent intervention for decompression via VATS or thoracotomy is required.

Recurrences of pneumomediastinum are extremely rare and long-term follow-up is usually unnecessary; in case of recurrence, more detailed investigation should be assessed in order to define and treat a possible misdiagnosed underlying cause.

Algorithm 6.2 Algorithm for diagnosis and treatment of pneumomediastinum.

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Multidisciplinary Surgical Consensus on Neck Emergencies

7

Emanuela Fuccillo and Marco Giovenzana

7.1 General Approach to Neck Emergencies

1. Firstly, evaluate:
   (a) **airway patency assessment and breathing adequacy**
   (b) **possible vascular complications**
   (c) **possible involvement of other anatomical regions** (e.g., expansion of an infective process, involvement of the spine due to neck trauma...)
2. Secondly, define if the patient can be considered "stable" or "unstable."

→ If the patient is unstable, the anesthetist-resuscitator and the ENT specialist should be alerted immediately to achieve an early protection of the airways.

Below we present some of the signs and symptoms in the context of neck pathologies that can guide the clinical path in defining the patient's condition (Table 7.1):

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Table 7.1 Main clinical signs and symptoms of relevance to determine the degree of impairment

Suggestive of severe impairment	Suggestive of mild impairment (possibility of changing the framework in a shon time)	Suggestive of initial/ suspected impairment
- Absence of vital signs - Loss of consciousness - Unrecognizable respiratory movements - Asphyxia - Peripheral vasoconstriction - Massive bleeding GCS score ↓	- Tirage / comage / stridor - Dyspnea - Bradypnea - Confusion - Sweating - Tachycardia - Abnormal chest movements - Bruising - Subcutaneous emphysema - Cervical swelling	- Anamnesis of clysphagia, dysphonia, respiratory distress without compromisation of vital functions - Signs and symptoms of ENT interest in association with ongoing infectious / inflammatory signs / recent trauma/ new onset swelling / neck and or tracheal pain / known head and neck malignancy

7.1.1 Airway Patency Assessment

The evaluation of airway patency and respiratory exchanges can take place through indirect signs, which every doctor is required to know (refer to the dedicated chapter of this book) or through a direct evaluation, which may include fiberoptic laryngoscopy [1], bronchoscopy and completed by a radiological study (in emergency department we refer in almost all cases to a neck and/or chest CT with and without contrast) (Fig. 7.1).

In all cases of airway compromise, an anesthetist-resuscitator must be alerted for prompt airway intubation: airway safety must precede any other type of examination. In all cases in which there is the suspected or confirmed difficulty of intubation (e.g., expansive mass which compresses the glottis), the ENT team is required for an emergency tracheotomy. If the patient is stable and if the ENT specialist is available, the execution of a flexible fiberoptic laryngoscopy [1] represents an indispensable tool for diagnostic completion. If the ENT specialist is not available, the execution of an urgent CT without and with contrast material represents the first and most complete and useful radiological evaluation during neck emergency [2].

REMEMBER: In adults, tumors represent the main cause of laryngeal dyspnea; in a smaller number of cases, it is instead related to functional, neurological, infectious-inflammatory, traumatic causes or by inhalation of foreign bodies. During the emergency practice, on the contrary, inhalation of foreign bodies, tumors and infections represent the main causes of access to an emergency department.

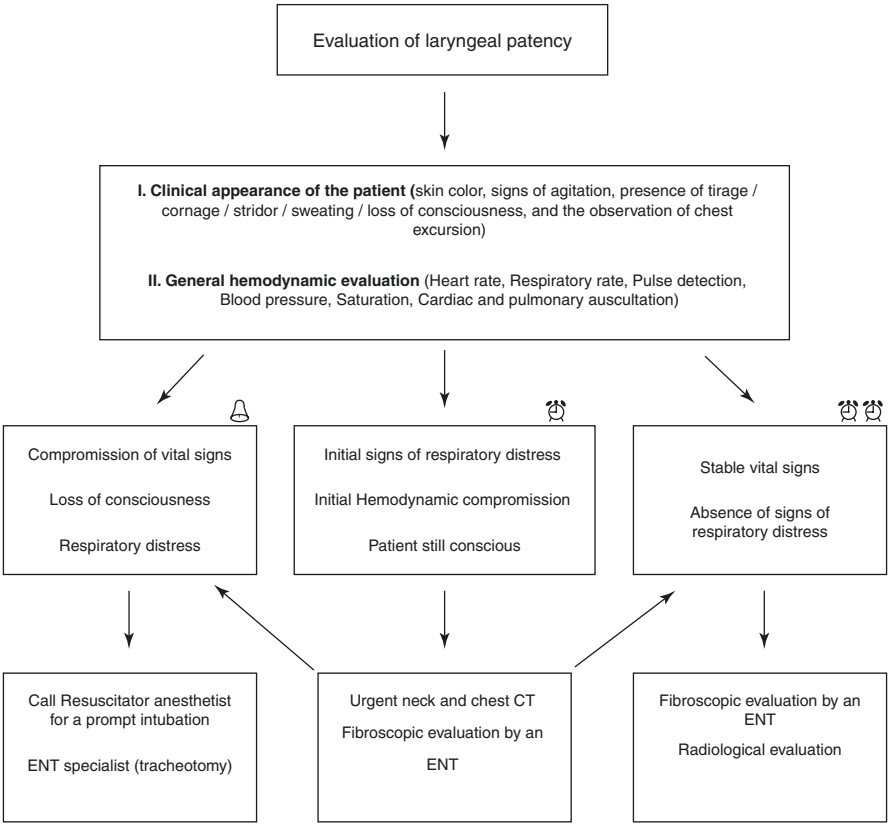

Fig. 7.1 Evaluation of laryngeal patency—Flowchart

7.1.2 Evaluation of Bleeding

When possible, the anamnestic evaluation is essential to raise a better diagnostic suspicion.

- The first action required is to evaluate if the patient is "stable" or "unstable."

The assessment of the "volume status," or the potential entity of the bleeding, can also be made considering the parameters reported in Table 7.2.

Table 7.2 Evaluation parameters of potential blood and water losses

Degree of severity	Estimated blood loss	Clinical appearance
Mild	0 – 20%	Pallor Cold
Moderate	20 – 40%	Oliguria Anuria
Strict	> 40%	Agitation Anxiety Restlessness loss of consciousness Coma

If the patient is "stable," we can proceed with radiological evaluation through an angio-CT and/or a neck and chest CT with and without contrast material (depending on the suspicion). If the patient is hemodynamically unstable, the first necessity is to stop the bleeding through exploration and surgical hemostasis, which requires securing the airway through oro-tracheal intubation, or a surgical tracheotomy in cases where it is not possible to intubate the patient (e.g., a patient with a fracture that does not allow hyperextension of the neck or with expansive masses that do not allow the correct visualization of the glottic plane), it is essential to contact the ENT team to perform an urgent tracheotomy. Secondly, evaluate an adequate restore of volumes.

7.1.3 Evaluation of a Possible Involvement of the Surrounding Tissues

1. Anamnesis (e.g., spinal injury, tooth infection).
2. In case of suspicion of involvement of surrounding tissues and if the patient is stable, imaging should always be planned. The CT scan with and without contrast material represents the best examination to evaluate the possible progression of inflammatory/infective/neoplastic pathologies or to localize possible trauma/presence of foreign bodies. In selected cases, the CT scan will be completed with an MRI that—however—is usually difficult to be performed in the emergency regime. If a traumatic vascular lesion is suspected → an angiographic assessment should be performed to evaluate the need for an immediate therapeutic intervention

7.2 Etiologies of Neck Emergencies

7.2.1 Traumatic Neck Emergencies

When possible, *collect an accurate anamnesis* to understand the mechanism of trauma (Table 7.3) [3].

Table 7.3 Main possible mechanisms of neck trauma distinguishing between extrinsic and intrinsic forces

"EXTRINSIC" FORCES	"INTRINSIC" FORCES
Blunt traumas (sports or motor vehicle accidents)	Iatrogenic procedures
Penetrating traumas (gunshot wounds, stab wounds)	Ingestion/Inhalation of foreign bodies
Hanging and strangulation traumas	Caustics

7.2.2 Remember

7.2.2.1 Extrinsic Forces

1. When the platysma is overcome, there is a greater risk of involvement of vital structures [4].
2. The sternocleidomastoid muscle separates the neck into two different triangles; the anterior triangle contains larynx, trachea, pharynx, esophagus, and major vascular structures, while the posterior triangle contains muscles, the spinal accessory nerve, and the spinal column.
3. In case of strangulation and/or hanging, we must consider also possible hypoxic suffering of the brain and/or head tissues.
4. Vascular injuries should be always suspected and investigated with radiological images.
5. Avoid any type of stress and/or traction in case of suspicion of infringement of the spine.

7.2.2.2 Intrinsic Forces

Foreign Bodies 

In case of ingestion of foreign bodies, patients may present with choking abscesses, most of which occur from the casual inhalation of food fragments. If the foreign body obstructs the laryngeal lumen in its entirety and/or oropharyngeal space, the patient can also die quickly from asphyxiation; if it is not possible to carry out quick ejection maneuvers, the urgent execution of an endoscopy is essential, with instruments equipped with an operating channel or with available surgical instruments for the extraction of foreign bodies, possibly by "4 hands" procedure (one operator guarantees the vision while the second operator proceeds with the extraction).

7.2.2.3 Caustics

Find out about the *type of caustic* and the *amount ingested.*

Endoscopy should often be repeated within 24 h.

7.2.2.4 Non-traumatic Neck Emergencies

Non-traumatic emergencies of the neck can occur in healthy patients and/or as a possible complication of already known malignant conditions. Among the possible non-traumatic etiopathogenesis, the main ones are therefore represented by infective and/or inflammatory, congenital, hemorrhagic, malignant diseases, and complications during systemic pathology [5].

7.2.3 Infections

The presence of a previous malignant pathology and/or of any condition of immunocompromission (e.g., diabetes, HIV, chronic steroid intake) indicates that the patient requires closer surveillance. Along with systemic symptoms, characterized by fever, fatigue, and alteration of blood tests, patients often report pain at the site of infection. In most cases, neck infections are due to spreading at the level of the neck spaces [6, 7].

7.2.4 Malignant

A neck mass or adenopathy should always be investigated in subjects with a history of malignant disease. If possible, it is necessary to request clinical documentation or collect thorough anamnestic information. When there is a suspected or proven airway or vascular compromise, the first step to do is to ensure patient stability. In these cases, a laryngoscopic evaluation is often indispensable and is possibly followed by dedicated imaging.

7.2.5 Emergencies Occurring in Patients with Tracheostomy Tube

Knowledge of the main components of the tracheostomy tube represents the *essential condition to deal with* possible emergencies in patients with tracheal cannula. We recommend you turn to the in-depth texts before being caught unprepared!

→ firstly: Why was a tracheostomy performed on the patient?? Was the procedure surgical or percutaneous?

- The surgical timing is important because stomal infections and bleeding are the most common complications during the first days, while percutaneous approach could be complicated by posterior tracheal wall lesion (more often if compared

to surgical tracheostomy). Moreover, percutaneous approach is less complicated by surgical site infections and postsurgical bleeding [8].
- Secondly, distinguish between the type of emergency.

Below we present some of the main possible emergencies occurring in patients with tracheostomy tube.

7.2.5.1 Obstruction

Often due to mucus or clotted blood.

First to do → remove the inner cannula and check the patency of the tracheal cannula through a suction tube. If the obstruction persists an urgent endoscopic examination should be performed to role out the presence of plugs/other obstructions.

7.2.5.2 Bleeding

If the cannula has a cuff → inflate the cuff to protect the airway.

If the bleeding is massive (early postoperative or in case of patients with relapsing malignant disease/presence of metastases with involvement of vessels) → surgical inspection and hemostasis (alert an anesthetist-resuscitator for performing control of bleeding in the operating room).

If the bleeding is superficial/mild and the parameters are stable try to manage it with local measures such as packing and alert ENT to perform an endoscopic examination.

7.2.5.3 Infection

- Severe tracheal infections are rare and often require a surgical debridement [8], other minor infections normally do not appear in an emergency regime and they are usually managed by using antibiotics and realizing chronic wound medications.

7.2.5.4 Dislodgment

First days after tracheostomy it could be due to a "false tract" (cannula is not properly lodged), which should be revealed by an endoscopic evaluation. If possible, an attempt and adequate accommodation through the use of a guide (for example, a tube changer) or surgical instruments that can keep in maintaining the tracheostomy lumen open (e.g., nasal speculum, Laborde Tracheal Dilator).

If replacement is not possible, consider an urgent oro-tracheal intubation in order to stabilize patient and wait for the ENT to perform a surgical inspection.

7.2.5.5 Tracheal stenosis

It often occurs in patients with a long-term tracheostomy tube.

→ Flexible bronchoscopy:

1. If the patient is stable → evaluate the tracheal stenosis by considering different possible therapeutic approach based on the dimension of the stenosis (e.g., surgical/endoscopical).
2. If the patient is unstable → urgent surgical treatment.

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Part III

Chest

Chest: Surgical Anatomy and General Consideration in Emergency Settings

8

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In order to manage and perform interventional and surgical procedures in the emergency settings, it is mandatory to know the anatomy of the chest wall. This chapter briefly reviews the anatomy of the thorax, focusing on the most important aspects to be considered when performing an invasive procedure in the emergency setting.

8.1 Chest Wall Surface Landmarks

There are several surface landmarks which should be taken in consideration when placing a thoracic incision, especially in an urgent context. Depending on the clinical situation, the thoracic incision must provide access to pulmonary hilum, trachea, and great thoracic vessels. Knowing the level of the diaphragm is also necessary to perform a tube thoracostomy or a thoracentesis.

The skeletal structures (sternum, costae, scapula, and vertebral column) of the chest provide readily palpable surgical landmarks. The upper margins of the thorax are bounded by the suprasternal notch above the sternum, the 1st ribs, and T1 posteriorly. The inferior limits of the thoracic cavity, separating it from the abdominal cavity, are formed by the sternal xiphoid process, the 7th to 10th costal cartilages, the 11th ribs, the arch of the 12th rib, and T12. The following vertical lines (Figs. 8.1 and 8.2) are commonly used to establish the correct site for incisions:

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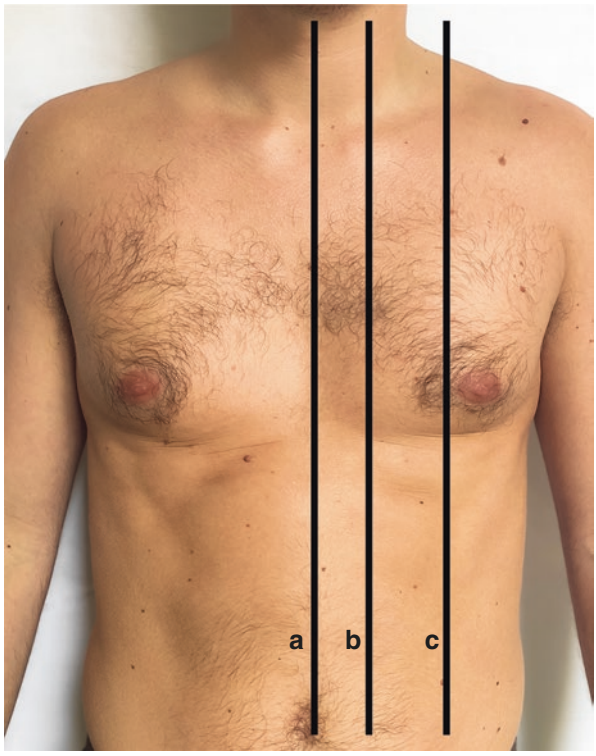

Fig. 8.1 Anterior chest wall lines: (**a**) midsternal line; (**b**) parasternal line; (**c**) midclavicular line

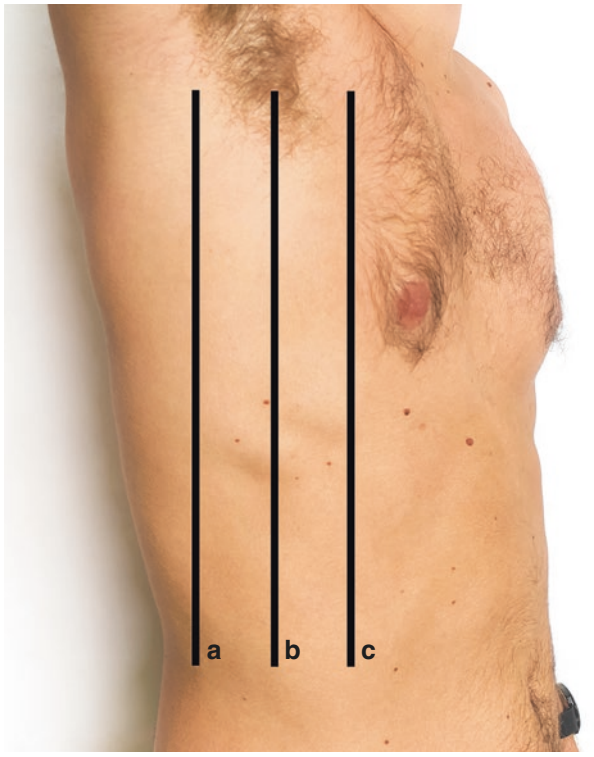

Fig. 8.2 Axillary lines: (**a**) posterior axillary line; (**b**) midaxillary line; (**c**) anterior axillary line

Table 8.1 Surface landmarks of the chest wall

Surface landmark	Underlying structure
Sternal manubrium	Aortic arch, left brachiocephalic vein
Angle of Louis/T4	Tracheal bifurcation; azygos arch to SVC junction (right); right to left lymphatic duct crossing; left recurrent laryngeal nerve
Sternal body, III-VI costal cartilages	Heart
T4/T5 posteriorly, 4th rib laterally, III intercostal space anteriorly	Right pulmonary horizontal fissure
T4/T5 posteriorly, V right intercostal space laterally, 6th rib anteriorly	Right pulmonary oblique fissure
T4 posteriorly, 5th rib and V left intercostal space laterally, 6th rib anteriorly	Left pulmonary oblique fissure
Tip of the scapula	Seventh intercostal space

- midsternal line, running down the middle of the sternum
- parasternal line, running along the lateral margins of the sternum
- midclavicular line, passing through the middle of the clavicles
- anterior axillary, midaxillary, and posterior axillary line, passing through the anterior axillary fold, the apex of the axilla and the posterior limit of the axilla, respectively

A transverse prominence called the angle of Louis (or sternal angle) can be appreciated at the T4 vertebral level, marking the manubriosternal junction and the second pair of costal cartilages. Table 8.1 reports the most important surface landmarks and the underlying structures. The nipple usually marks the 4th intercostal space in men, lateral to the midclavicular line, while in women its position varies depending on the size and shape of mammary glands. The xiphoid process lies at the T9 vertebral level. Posteriorly, the tip of the scapula lies on the 7th intercostal space, but when the patients lies in lateral position with the shoulder in about 100° of flexion, the tip of the scapula marks the 5th intercostal space.

8.2 Muscles of the Chest Wall

Understanding the anatomy of the chest wall musculature is essential in order to perform any surgical procedure, especially in an emergency setting. The anterior portion of the chest wall musculature is formed by the pectoralis major muscles, which originate from the sternum, the clavicle, and the costal cartilages (2nd to 6th); its insertion is located on the bicipital groove of the humerus, and its lower margins form the anterior form of the axilla. The pectoralis minor muscles lie deeper to the pectoralis major: they arise from the anterior surfaces of the ribs (2nd to 5th), forming a tendon which inserts onto the coracoid process of the scapula. These two muscles are both inspiratory muscles. The antero-inferior portion of the chest wall is also covered by fibers of the rectus abdominis muscle and the abdominal external oblique muscle: they are both expiratory muscles.

The latero-posterior aspect of the thorax is more complex, as it is formed by three different muscle layers: we will only discuss the most important ones. The superficial back muscles originate from the vertebral column and insert to the shoulder, thus being responsible for movements of the upper limb. From external to deep, these are the trapezius, the latissimus dorsi, the rhomboids, and the serratus anterior:

- Trapezius: It originates from the occipital bone and the spinous process of the C7 and all the thoracic vertebrae. Its insertion is located on the lateral portion of the clavicle, the acromion process, and the spine of the scapula.
- Latissimus dorsi: It is the largest muscle of the thorax. Its aponeurosis originates from the spinous process of T6-T12, the lumbodorsal fascia, the iliac crest and the external surface of the lower 3–4 ribs. This insertion lies in the intertubercular sulcus of the humerus, also forming the posterior fold of the axilla.
- Rhomboids: The rhomboid minor arises from the spinous processes of C7-T1 and attaches medially on the spine of the scapula; the rhomboid major arises immediately caudally originating from T2-T5 and attaching to the medial border of the scapula. In some cases, the two rhomboids can be fused.
- Serratus anterior muscle: It originates from the surface of the first 8 ribs on the lateral side of the chest and inserts along the anterior aspect and the medial border of the scapula.

A thinning of the chest wall muscles in the posterior chest wall at the level of the 6th and 7th rib is described as the triangle of auscultation: Its borders are formed by the horizontal fibers of the latissimus dorsi inferiorly, the vertebral border of the scapula laterally and superiorly, and the lateral margin of the trapezius medially and superiorly. This triangle allows for a better auscultation and is also used as a surface marker for surgical approaches and regional nerve blockade for pain relief.

Box 8.1 Surgical access: Thoracotomy

In case of emergency, the anterolateral approach with the patients lying supine is usually the preferred one. If necessary, the access to the mediastinum can be improved by extending the incision across the sternum, as the so-called "hemi-clamshell." In such case, the internal mammary vessels should be ligated.

- The incision is usually made throughout the 4th or 5th intercostal space, curving along the submammary crease (and retracting the mammary gland cranially in female patients) from the parasternal line to the posterior axillary line. The subcutaneous tissue is divided deep to the pectoralis fascia.
- The pectoralis major is divided at the 4th intercostal space; laterally, the serratus muscle is divided.
- The intercostal muscles are divided and the pleural space is entered bluntly from the lateral portion of the incision, right along the upper margin of the rib below, in order to avoid damaging the neurovascular intercostal bundle. This must be done with caution so as not to injure the lung, especially in case of adhesions.
- The remaining intercostal space is cut open using electrocautery.
- A Finochietto retractor is used to held the thoracotomy open at right angles.

Box 8.2 Surgical access: Median sternotomy
The patient is supine and his arms are tucked.

- The skin incision begins at the suprasternal notch and runs across the midsternal line right beyond the xiphoid process. The subcutaneous tissue is incised until reaching the pectoral fascia.
- The cranial portion of the manubrium is exposed by retracting the cranial portion of the incision, and the crossing jugular tributary veins are identified. The interclavicular ligament is divided.
- The retrosternal space is opened bluntly cranially and caudally.
- Cautery can be used to further mark the midsternal line; palpation of the intercostal spaces bilaterally can also be of aid in order to identify the midline.
- Lungs are deflated by disconnecting the ventilator before dividing the sternum using a reciprocating saw. Bleeding from the bone and the periosteum is immediately controlled using cautery, gauze packing, and bone wax (or biodissolvable bone hemostats).
- A sternal spreader is placed to obtain appropriate exposure.

8.3 Anatomy of the Intercostal Space

Proceeding from superficially to the deeper layer, the intercostal space is covered by 3 intercostal muscles (external, inner, and innermost); the fibers of the external and inner intercostal muscles run in opposed directions. The innermost intercostal muscles' fibers run vertically along the intercostal space.

On the lower margin of each rib, between the innermost fascia and the inner intercostal muscles, lies the neurovascular bundle, which is formed, from upper to lower, by the intercostal vein, artery, and nerve.

Notably, intercostal muscles can be used as a muscle flap for the protection of tracheal, bronchial, or esophageal anastomosis.

Box 8.3 Chest tube placement
The indications for thoracostomy tube placement are several, and they will be partially discussed in Chap. 11 "Pulmonary and thoracic emergencies." It is also important to select the appropriate diameter and type of tube (≥16 Ch) or catheter (≤14 Ch) depending on the patient and the condition requiring pleural drainage. The instruments required for chest tube placing are listed in Chap. 2 "Surgical instruments and materials in thoracic surgery." The patient can be positioned supine, in a semi-recumbent position (semi-Fowler or Fowler) or in a lateral recumbent position, depending on the patient's clinical condition and the operator's preference. Imaging guided (CT, US) thoracostomy improves the safety and efficacy of the procedure.

- Chest tubes are usually placed in the so-called "triangle of safety," which is defined anteriorly by the lateral edge of the pectoralis major, superiorly by the base of the axilla, posteriorly by the anterior margin of the latissimus dorsi, and inferiorly by a horizontal line at the level of the nipple.

  In urgent setting, some authors prefer to insert chest tube in the second intercostal space on the anterior midclavicular line in case of pneumothorax, and on the 4th to 6th intercostal space laterally for pleural fluid drainage.
- After preparing the sterile field, inject with local anesthetic at the level of the skin incision at the upper margin of the lower rib in order to avoid the neurovascular bundle. The needle should be kept perpendicular to the chest wall, aspirating at each advance of the needle so as to avoid intravascular injection, until air or fluid is aspirated, thus confirming the correct site for chest tube placement.
- After incising the skin, the previously anesthetized subcutaneous tissue and the muscular layers are bluntly dissecting using curved Mayo scissors or a Kelly clamp.
- The Mayo scissors are then pushed through the parietal pleura and then opened in order to widen the defect as required, depending on the diameter of the tube.
- The tube is then inserted following the same path: the tip of the catheter is usually directed to the pulmonary apex in case of pneumothorax, and to the pulmonary bases in case of pleural effusion.
- Once in the correct position, the tube is secured using a non-absorbable suture.

8.4 Anatomy of the Pleura

The pleura is a serous membrane covering the lungs (visceral pleura) and the inner part of the chest wall (parietal pleura), excluding the pulmonary hilum. Inferiorly, the continuation of the visceral and parietal pleurae forms the pulmonary ligament. The parietal pleura covers the internal aspects of the thoracic wall, thus separating the pleural cavity from the mediastinum. The parietal pleura is usually divided in the following portions: the dome of the pleura, the costal, the mediastinal, and the diaphragmatic part. The pleural space between the two pleurae is normally collapsed and contains about 10 ml of pleural fluid, which helps the sliding between the two pleural surfaces during respiration.

8.5 Anatomy of the Lungs

The lungs are located in the pleural space in the chest. The right lung is composed of three lobes: upper, medium, and lower lobe. The left lung only has two lobes, the upper and the lower. The lower lobe is separated from the upper and middle one by

the oblique (major) fissure, which is also found on the left lung. The horizontal fissure separates the upper and middle lobe. Every lobe can be divided into bronchopulmonary segments, which are supplied by their own bronchus and vascular structures.

The pulmonary hilum is composed of the bronchus, the pulmonary artery, and the superior and inferior pulmonary vein; these structures however are located in different positions in the left and right hilum. On both sides, the phrenic nerve and the vagus nerve, together with their vascular bundles, run on the anterior and posterior border of the hilum, respectively.

On the right side, the hilum is encircled by the azygos vein, the superior and the inferior vena cava. On the left side, the aortic arch passes above the superior aspect of the hilum, while the descending aorta lies behind the hilum and the vagus nerve. The esophagus runs vertically in the mediastinum behind the right and left hila.

The bronchial arteries originate mostly from the anterolateral portion of the aorta or its branches, or from the intercostal arteries (usually on the right side). They run along the posterior wall of the bronchi.

8.6 Anatomy of the Mediastinum

The mediastinum is the central region of the thoracic cavity located/situated between the two lungs. Conventionally it is divided into superior, anterior, middle, and posterior compartments. The superior compartment contains the upper portion of several thoracic structures: esophagus, great vessels, trachea, thymus. The anterior compartment's margins are formed by the posterior portion of the sternum anteriorly and the pericardium posteriorly: the rest of the thymus is located in this compartment. The middle portion of the mediastinum contains the pericardium, the intrapericardiac vessels, the heart, and the main carina. The esophagus, the thoracic duct, and the descending aorta form the posterior compartment.

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9 Acute Aortic Syndromes and Thoracic Aortic Aneurysms: From Diagnosis to Treatment

Andrea Xodo, Andrea Gallo, Paolo Magagna, and Mario D'Oria

9.1 Introduction

Acute aortic dissection (AD), the most common form of acute aortic syndrome (AAS), is a relatively rare but highly lethal disease: population-based studies suggest that the incidence of acute AD ranges from 3.5 to 6.0 cases per 100,000 person-years [1]. Risk factors for the development of AAS mainly reflect conditions associated with increased wall shear stress (hypertension and physical trauma), with chronic or acute exposure of the aorta to elevated intraluminal pressures, and/or abnormalities of the aortic media (syndromic and non-syndromic genetic diseases, including Marfan and Ehlers-Danlos syndromes, annuloaortic ectasia, and bicuspid aortic valve) [2]. In the classical definition, AD requires a tear in the intimal aortic wall, that is commonly preceded by medial wall degeneration or medial cystic necrosis. The blood passes through the tear that separates the intima from the media or from the adventitia, creating a dual channel that comprises a "false lumen" (FL) and a "true lumen" (TL). In addition to AD, two other distinct conditions require further description. Intramural hematoma (IMH), in contrast to AD, lacks an

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identifiable communication between the two lumens: different mechanisms for etiology have been proposed for this disease, including microscopic tears in the intima and rupture of the vasa vasorum. Penetrating atherosclerotic ulcer (PAU) is instead defined as an atherosclerotic lesion that penetrates the internal lamina of the aortic wall, also known with the term of "ulcer-like projections."

All these mechanisms that weaken the medial layers of the aorta, may lead to increased tension on the aortic wall, which can induce aortic dilation with consequent aneurysm formation, frank acute AD from IMH or PAU, or aortic rupture. Furthermore, AD can present with complications in the acute phase that requires expedite treatment.

Descending thoracic aortic aneurysms (TAAs), similar to other aortic aneurysmal disease, rarely manifest with symptoms and about 95% of the patients are asymptomatic at time of diagnosis [3]. Nevertheless, TAAs can lead to catastrophic complications, mainly rupture with hemorrhagic shock, why they are termed "silent killers." Blunt thoracic aortic injury (BTAI) is also sometimes included in the spectrum of AAS, but will not be discussed in this chapter.

Indications for the elective treatment of these pathologic entities when asymptomatic (usually within the context of incidental findings during imaging examinations performed for other medical or surgical reasons) are largely dependent on their size, etiology, and morphology. They will not be discussed further in this chapter, which will focus on the presentation, diagnosis, and management of AAS/TAA in the urgent/emergent setting. This chapter will focus on those AAS and TAA that require immediate attention from the clinician and prompt referral to cardiovascular specialists. The need for immediate treatment is usually dictated by the presence of symptoms and severity of presentation but may be also prompted or exacerbated when large aortic diameters, rapid size growth, or other alarming radiologic signs are encountered.

9.2 Anatomical Classification

The majority of TAAs are degenerative (i.e., atherosclerotic) in nature, while other causes include traumatic, mycotic, or related to vascular genetic disorders; aneurysmal evolution is indeed included among late aortic events of patients with AD.

Historically, classification of AAS was based on the anatomic location of the intimal entry tear that results in a separation of the aortic layers, and also on the longitudinal extension of the dissection flap. There are two main schemes that have been classically used to classify AD; the first one, the original "DeBakey classification" was described in 1965 and is based upon the site of origin of the dissection and its distal extension:

- Type 1 originates in the ascending aorta and to at least the aortic arch.
- Type 2 originates in and is limited to the ascending aorta.
- Type 3 begins in the descending aorta and extends distally above (type 3A) or below (type 3B) the diaphragm.

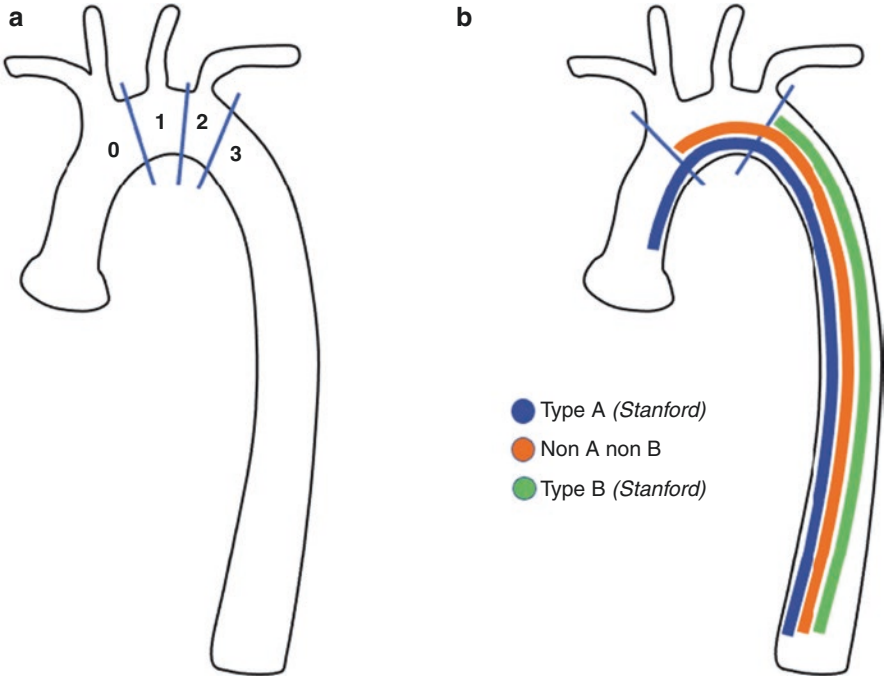

Fig. 9.1 (**a**) Ishimaru Classification. (**b**) Stanford Classification

The more widely adopted "Stanford system" instead classifies dissections into two types, based on whether the ascending or descending part of the aorta is involved (Fig. 9.1):

- Type A aortic dissection (TAAD) involves the ascending aorta, regardless of the site of the primary intimal tear.
- Type B aortic dissection (TBAD) originates distal to the left subclavian artery (LSA) and only involves the descending aorta [4].

A new classification has recently been proposed, with the aim of describing more precisely the involvement of the aortic arch. In this classification, the distinction between type A and type B is given by the entry tear zone (according to the classification of Ishimaru), and type A is represented only by an entrance door in zone 0 [5, 6].

TAAs are localized dilatations in the descending thoracic aorta, secondary to weakening and subsequent expansion of the aortic wall. When all aneurysms of the thoracic aorta are considered, those of the ascending aorta are the most common (40%), while aneurysms of the descending thoracic aorta account for 35% of TAAs, and aortic arch aneurysms (15%) or thoraco-abdominal aortic aneurysms (10%)

account for smaller percentages [7]. The descending thoracic aorta can be divided into three sections:

- Extent A: distal of the LSA to the 6th thoracic intercostal space.
- Extent B: 6th to the 12th intercostal space.
- Extent C: the entire descending aorta from LSA to the 12th intercostal space.

9.3 Temporal Classification

The temporal classification of the AD originates from the studies of Hirst and colleagues, who observed a markedly higher mortality, for both type A and type B dissections, in the first 14 days after the onset of the disease [8]. Based on these observations, the authors defined the AD as acute in the first 14 days from the symptom onset and as chronic as >14 days from symptom onset.

However, Hirst's observations date back to the 1950s, and since then huge improvements have emerged in diagnostic imaging, medical treatment, and surgical therapy, mainly owing to the endovascular innovation over the past 25 years. Thoracic endovascular aortic repair (TEVAR), in particular, has revolutionized the management and treatment of AD; due to this reason, more recent documents classify patients with AD into hyperacute (<24 h), acute (1–14 days), sub-acute (15–90 days), and chronic (>90 days), relating to the time of onset of complications requiring intervention [5, 9, 10].

9.4 Clinical Presentation

The pivotal symptom in the whole spectrum of AAS is acute thoracic pain, usually localized between the scapula and described as stabbing, ripping, or fluctuating; it is present in the majority of patients, with a severe initial intensity.

Pain usually reflects the entry tear site and may migrate in relation to the extent of the AD: its radiation to the neck, throat, teeth, or jaw may indicate an ascending aorta involvement; conversely, back pain or abdominal pain may herald a descending aorta localization.

Coronary malperfusion associated with TAAD is rare but when it occurs it may be fatal to the patient, as a result of AMI or fatal arrhythmia; in these cases, the onset symptomatology may include chest pain or cardiac arrest. In a large series of 464 patients, the majority of patients complained of chest pain (72.7%): anterior chest pain was typical in patients with TAAD, whereas patients with TBAD more often experienced pain in the back and abdomen [11].

A thorough clinical examination has a crucial role in patient with suspected AAS: assessment of arterial pulses should involve radial, brachial, femoral, and distal pulses, in addition to abdominal aorta palpation. The absence of a peripheral pulse, associated with clinical signs of ischemia, could suggest a malperfusion, that occurs in up to 40% of complicated TBAD [12].

Signs attributable to connective tissue diseases (e.g., Marfan syndrome) will be sought during the medical examination. Aortic regurgitation should be suspected in case of diastolic murmur; if dissection causes bleeding into the pericardium, distant heart sounds secondary to pericardial effusion may be noted, with signs and symptoms of cardiac tamponade (Beck's triad).

Neurologic deficits might be identified on presentation, as partial or complete loss of sensory or motor function; they can be caused by persistent or transient ischemic stroke, spinal cord ischemia, and branch vessel malperfusion.

The initial clinical presentation of AAS has major implication for both patient management and outcomes: this spectrum of conditions, in particular acute AD, may be classified as "uncomplicated," "high risk," and "complicated." Uncomplicated acute AD is defined for those patients without evidence of rupture or end-organ malperfusion, while complicated TBAD refers to malperfusion syndrome involving visceral, renal or extremity ischemia, rupture or impending rupture. The "high-risk" category includes clinical and radiological signs which were devised to identify those patients at increased risk of worse prognosis into a complicated AAS.

TAAs are usually asymptomatic and their diagnosis is often incidental. By contrast, when symptomatic, they usually cause severe pain or a feeling of fullness in the chest. Rupture of TAAs is a life-threatening condition that usually presents with massive hemothorax accompanied by dyspnea, syncope, and hemorrhagic shock. In much rare instances, the symptoms may include signs of distal embolism related to a mural thrombus or signs of compression of adjacent structures (trachea, bronchus, esophagus), causing dyspnea, bronchospasm, cough, hemoptysis, dysphagia, or hematemesis in case of aorto-esophageal/bronchial fistula [13].

9.5 Diagnosis

The urgent approach to the AAS/TAA (Fig. 9.2) requires optimization of resources within a short timeframe. The role of a quick yet exhaustive physical examination in patients with suspected AAS/TAA has been emphasized in the previous paragraphs. Obviously, acquiring a focused medical history in these patients helps to guide the diagnostic process: details that are particularly helpful to assess will include past medical history (hypertension, heart disease), family history (sudden death or aortic diseases), and chronic medications, which should all be assessed upon admission of the patient(s).

Patients presenting to the emergency department with typical chest pain should undergo an electrocardiogram (ECG) and serum troponin determination, to rule out acute myocardial infarction (AMI). It is important to note, however, that the ECG can be abnormal in up to 70% of patients with ascending aortic dissection. If the diagnosis of myocardial infarction is doubtful, other diagnostic studies should be performed promptly to rule out aortic dissection. A chest X-ray might be helpful to rule out alternative diagnoses provided it does not delay prompt referral of patients, and may show an enlargement of the upper mediastinal shadow or a pleural effusion. However, it should be borne in mind that up to 10–15% of patients with acute

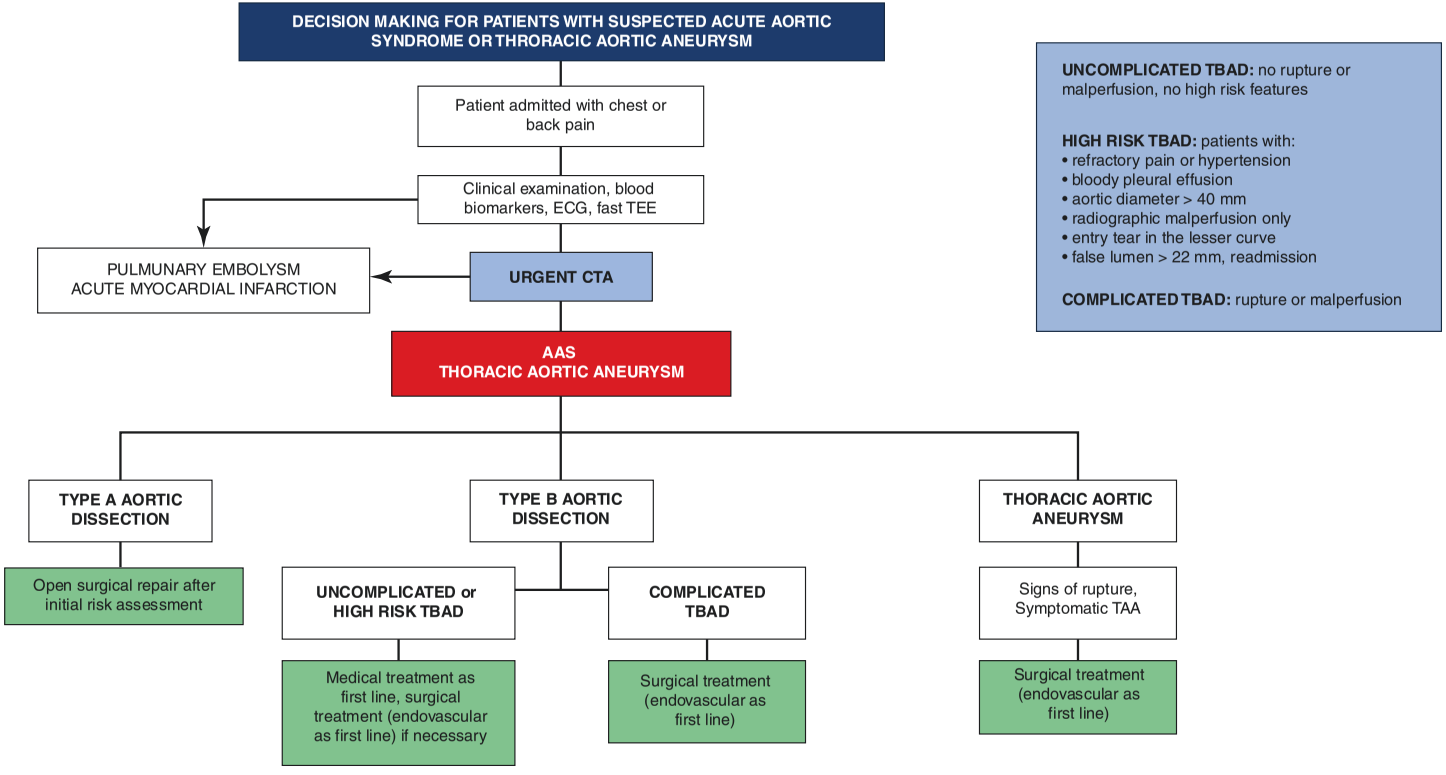

Fig. 9.2 Flowchart for decision-making and initial management of acute aortic syndromes

AD will have normal findings on chest radiography [14]. Therefore, while abnormality noted on ECG and chest X-ray might guide the initial assessment, they lack enough sensitivity or specificity to either exclude or confirm the diagnosis of AAS in patients presenting with typical symptoms, and a high index of clinical suspicion remains paramount.

D-dimer is probably the most widely available diagnostic biomarker for pulmonary embolism; several investigators have demonstrated that a cut-off level of 500 ng/mL is highly sensitive to rule out classical acute AD within the first 6 h of symptom onset and highest in the first hour [15, 16]. Nevertheless, it should be noted that low D-dimer levels may be found in patients with IMH, PAU, and/or AD with thrombosis of the false lumen (FL); at the same time, it may be elevated in many other disorders, including AMI complicated by mural thrombus and acute pulmonary embolism. In summary, high D-dimer level largely decreases the possibility of acute AD (although it cannot be used as a stand-alone indicator for its diagnosis), while low D-dimer rates moderately increase the possibility of acute AD [16].

Transthoracic echocardiography (TTE) is widely used in thoracic emergencies: a TTE focusing on the aortic valve complex, the ascending aorta, and the function of the left and right heart can provide useful information for time-sensitive clinical decision-making. However, although TTE is an excellent tool for quickly detecting life-threatening complications of acute AD, such as aortic regurgitation, pericardial effusion or cardiac tamponade, and wall movement abnormalities, its accuracy for AD remains relatively low (sensitivity range from 77 to 80%, specificity from 93 to 96%) [17]; therefore, a negative TTE does not rule out acute AD [18]; however, TTE is strongly recommended as an initial imaging investigation.

Transesophageal echocardiography has a limited role in urgency; however, it is an easy and useful tool in the operating theatre to guide the correct placement of the stent graft in case of TBAD.

Current diagnostic techniques for both AAS and TAAs focus on the use of computed tomography angiography (CTA). CTA is the most widely used modality worldwide, due to its cost and easy accessibility, providing meaningful anatomical information about the aorta and surrounding structures. It also allows surgeons to collect details necessary to plan and carry out interventions when needed. Ideally, the CTA should include the entire aorta, from the supra-aortic trunks to the femoral vessels. Using CTA, the morphologic characteristics of the entire aorta can be studied, in particular its diameters, the presence of intraluminal thrombus, the presence of IMH/PAU or calcifications, the state of peripheral vessels and of the main branches (including signs of malperfusion), as well as secondary evidence of end-organ damage or impending aortic rupture. Nevertheless, CTA may have several limitations, in particular for patients with history of allergic reactions to iodinated contrast media or for those with end-stage renal disease, for whom iodinated contrast loading could cause contrast-induced nephropathy. Despite these issues, CTA is an established imaging technique for diagnosing AAS in urgency and emergency settings, and last-generation multidetector CT scanners have improved the sensitivity and specificity of diagnosis for aortic disease, enabling rapid acquisition of

images with minimal contrast load. The electrocardiographic gated technique has virtually eliminated the pulsation artifacts in the ascending aorta, allowing a substantially better assessment of subtle aortic abnormalities, previously underestimated by uncontrolled CTA. While CTA remains the cornerstone for the initial diagnosis of patients with AAS, magnetic resonance imaging (MRI) may play a role during follow-up for identification of complications following a previous AAS, in order to reduce the overall burden of radiation exposure and iodinated contrast [19].

9.6 Primary Management

The concept of primary management of AAS should encompass those conditions where there is a high risk of imminent aortic rupture or frank complications have already arisen, allowing early identification of patients at such risk, rapid institution of medical treatment, prompt execution of CTA from the neck to the groin, strategic centralization to high-volume centers, and urgent/emergent aortic repair when indicated with post-procedure care in dedicated intensive care units.

9.6.1 Initial Management in Case of Stable and Uncomplicated Patients

In all patients with acute AD, PAU, and IMH, blood pressure (BP) and heart rate control are recommended in order to decrease the aortic wall stress and to limit the extension of the dissection, thereby reducing the risk of end-organ damage and aortic rupture.

Since uncontrolled hypertension associated with acute AD is considered a hypertensive emergency, systolic blood pressure should be immediately kept within 120 and 100 mmHg, with heart rate <60 bpm. In this scenario, intra-venous drugs with short half-life are preferred: International guidelines recommend the association of a beta-blocker, as esmolol, and a vasodilator such as sodium nitroprusside or nitroglycerine or nicardipine. Alternatives are beta-blockers as labetalol or metoprolol but with the downside of a longer half-life compared with esmolol (Table 9.1) [20]. Medical therapy is almost important for prevention: all hypertensive patients with aortic dilatation, whether associated with Marfan syndrome, bicuspid aortic valve disease, or not, should have their BP controlled.

Analgesia is important to obtain adequate control of pain, with use of opiates when indicated and continuous monitoring in intensive care or coronary care units.

Initial management of AAS could involve high-intensity serial imaging in the acute phase of TBAD, IMH, and PAU, to identify complicated cases for early intervention and selection of patients at high risk of disease progression.

Table 9.1 Drugs indicated for blood pressure and heart rate control in acute AD

Group of medications	Drug	Onset of action	Duration of action	Dose	Contraindications	Most relevant side effects
Beta-blockers	Esmolol	1–2 min	10–30 min	0.5–1 mg/kg as i.v. bolus; 50–300 lg/kg/min as i.v. infusion	2nd–3rd degree AV block, systolic heart failure, asthma, bradycardia	Bradycardia, Bronchoconstriction
	Metoprolol	1–2 min	5–8 h	2.5–5 mg i.v. bolus over 2 min—may be repeated every 5 min to a max. dose of 15 mg		
	Labetalol	5–10 min	3–6 h	0.25–0.5 mg/kg i.v. bolus; 2–4 mg/min infusion until goal is reached, thereafter 5–20 mg/h		
Nitrovasodilators	Sodium nitroprusside	Immediate	1–2 min	0.3–10 lg/kg/min i.v. infusion, increase by 0.5 lg/kg/min every 5 min until target BP values	Liver/kidney failure, severe aortic stenosis	Headache, reflex Tachycardia, cyanide intoxication for nitroprusside
	Nitroglycerine	1–5 min	3–5 min	5–200 lg/min i.v. infusion, 5 lg/min increase every 5 min		
Dihydropyridine class of calcium channel blockers (vasodilators)	Nicardipine	5–15 min	30–40 min	5–15 mg/h i.v. infusion, starting dose 5 mg/h, increase every 15–30 min with 2.5 mg until goal BP, thereafter decrease to 3 mg/h		

9.6.2 Initial Management in Case of Unstable and Complicated Patients

Patients presenting with circulatory shock should be immediately taken to the operating room after a diagnostic CTA scan. The management of hemorrhagic shock is crucial for the patient's outcome, with the aim to restore organ perfusion and systolic blood pressure range through aggressive fluid replacement. However, infusing large volumes of cold fluid causes dilutional and hypothermic coagulopathy; these factors, added with acidosis, may lead to increased bleeding [21].

In case of aortic rupture, an alternative to the "normotensive resuscitation" is represented by the "permissive" hypotension resuscitation: the aim of this strategy is to obtain a target systolic blood pressure of 50 mmHg to 100 mmHg, preventing blood loss and reducing the clot disruption caused by the rapid increase in systolic blood pressure. The use of this technique also avoids the dilution of coagulation factors, platelets, and fibrinogen, thereby reducing the decrease in temperature which may inhibit the enzymatic activity involved in the function of platelets and coagulation factor [22].

Along with replacing blood volume, massive-transfusion protocols with a minimalized imbalance in plasma, platelet, and red-cell units improve survival through the optimization of hemostasis. Moreover, for patients taking any anticoagulant medications is important to administer pharmacologic adjuncts to reverse anticoagulation [23].

The presence of cardiac tamponade should prompt urgent aortic repair; this condition is the most common cause of death in patients with TAAD and tamponade-induced hypotension, associated with aortic rupture, has been identified as a major risk factor for peri-operative mortality in these patients. Pre-operative pericardial drainage may be an effective temporizing strategy in selected patients with TAAD complicated by cardiac tamponade.

9.7 Principles of Surgical Treatment

9.7.1 Type A Aortic Dissection

The goal of surgery in the case of a TAAD is to avoid rupture of the ascending aorta, cardiac tamponade, insufficiency of the aortic valve, or coronary artery dissection resulting in AMI. The treatment of TAAD is therefore usually of cardiac surgery relevance, with the replacement of the ascending aorta; nevertheless, the aortic arch and the supra-aortic branches are often involved and a hemiarch or an entire aortic arch may require a more extensive approach, also combining the principles of open arch surgery and endovascular descending thoracic aorta repair by "elephant trunk" or "frozen elephant trunk" technique, although aortic arch surgery under emergency conditions carries much higher rates of peri-operative morbidity and mortality, based on the extent and location of the intimal tear and on the age of the patient. The aortic valve can also be replaced in case of insufficiency, using either a composite

valvulated graft (with a mechanical or a biologic valve); this procedure has been described by Dr. Bentall and Dr. De Bono. There are two widely used techniques for valve-sparing root replacement: aortic root reimplantation (David procedure) and aortic root remodeling (Yacoub procedure) [24]. The presence of IMH at the AsAO level is considered, due to its possible evolution, such as a TAAD and so treated immediately.

9.7.2 Type B Aortic Dissection

The main aims of acute repair when treating a type B aortic dissection are to cover the primary entry tear, to decrease the blood pressure in the FL while re-establishing adequate pressures in the TL, to prevent the extension of the dissection and to promote the FL thrombosis, inducing a positive aortic remodeling and resolve end-organ malperfusion (Fig. 9.3) when clinically relevant.

Thoracic endovascular aortic repair (TEVAR) is the currently accepted method of treatment in complicated or high-risk TBAD, with open repair considered only in case of particular contraindications to TEVAR [25]. Despite the vulnerability and fragility of the aorta during the hyperacute phase, associated with high mortality and morbidity rates, several studies have suggested that more favorable remodeling may be observed in case of early interventions.

Although coverage of the descending thoracic aorta with stent grafts is usually limited to the least extent possible in the acute phase, to reduce the risk of post-procedural spinal cord ischemia (SCI), peri-operative adjuncts (e.g., cerebrospinal fluid drainage) may be considered to decrease this risk, although their use may be troublesome in critical patients. Also, TEVAR often requires deployment of the

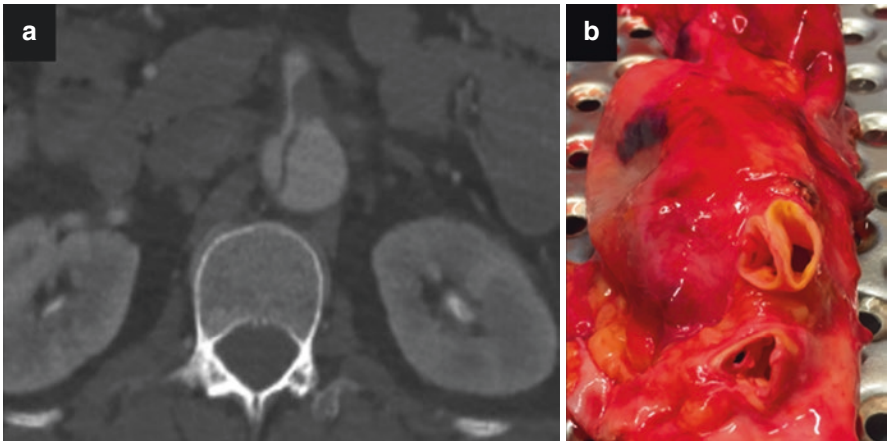

Fig. 9.3 (**a**) Superior mesenteric artery dissection, associated with a narrow compression of the true lumen, with a "radiological sign" of malperfusion. (**b**) Autopsy detail of a visceral vessel dissection, with a clear distinction between the two lumens

endograft across or proximally to the origin of the LSA to obtain an adequate proximal seal; intentional coverage of the LSA appears feasible in the urgent scenario, carrying a low risk of stroke, SCI, or left arm ischemia. However, LSA preservation is recommended for long aortic coverage (>200 mm) in order to decrease the risk of SCI, and in other selected circumstances (e.g., dominant left vertebral artery, patent bypass from the LIMA, or functional hemodialysis access). Different techniques and approaches are in use to ensure LSA revascularization, including open surgical procedures (bypass or transposition) or endovascular tools (chimney grafts, in situ fenestration or dedicated custom-made or off-the-shelf endografts) [26, 27]. Late complications of TEVAR in case of TBAD may include poor remodeling of the aortic wall, which may lead to aneurysmal degeneration over a number of years, and development of stent-graft induced new entry tears (SINE), which are usually caused by excessive stent-graft oversizing (a common technical issues with TEVAR for dissection as opposed to TEVAR for aneurysms) and can be a significant source for secondary interventions during follow-up.

9.7.3 Penetrating Aortic Ulcer and Intramural Hematoma

Patients presenting with uncomplicated IMH (Fig. 9.4) and PAU are primarily treated by medical therapy alone, with intensive care monitoring as appropriate, in similar fashion to those presenting with TBAD. However, patients with a PAU that initially measures >20 mm in diameter or >10 mm in depth might have a higher risk of disease progression and could be considered candidates for early endovascular repair. The role of endovascular treatment of IMH is still debated; however, the

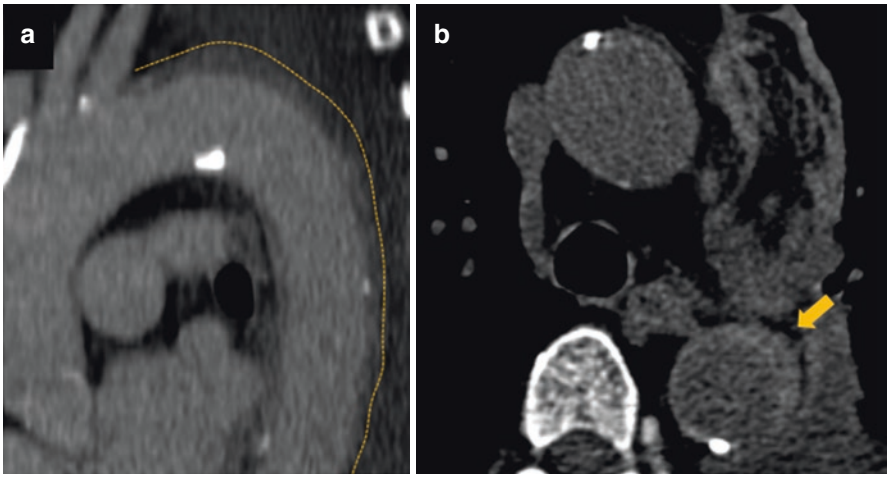

Fig. 9.4 (**a**, **b**) Acute IMH appear as shown by the orange arrow as focal, crescentic, high-attenuating regions of eccentrically thickened aortic wall on non-contrast CT

treatment is recommended in case of expansion of the hematoma, progression to frank AD, associated PAU, or intimal tear disruption [28, 29].

9.7.4 Thoracic Aortic Aneurysm

Prior to the endovascular era, open repair was the gold standard also for descending thoracic aortic aneurysms, requiring thoracotomy, aortic cross-clamping, aneurysm resection, and aortic replacement with a prosthetic graft. Owing to the advancements of endovascular techniques and technologies over the last two decades, TEVAR currently allows a minimally invasive procedure yet highly effective procedure compared with open surgical repair, decreasing operative time and blood loss. An initial diameter of 60 mm carries an annual risk of rupture of 10%, while intervention in TAAs below 55 mm may not afford a survival benefit. Indications for elective treatment exist for patients with TAAs >56–59 mm, reserving open surgical repair (OSR) treatment for fit patients considered unsuitable for TEVAR. Rupture of TAAs (Fig. 9.5) is an emergent condition regardless of the hemodynamic stability of the patient, with high associated mortality rates. TEVAR has showed favorable outcomes as compared to open repair. For the procedure to be successful and durable, adequate proximal and distal sealing zones are required to avoid development of type 1A or 1B endoleaks, thereby obtaining a complete exclusion of the aneurysm. Chest drainage may also be required to relieve respiratory function.

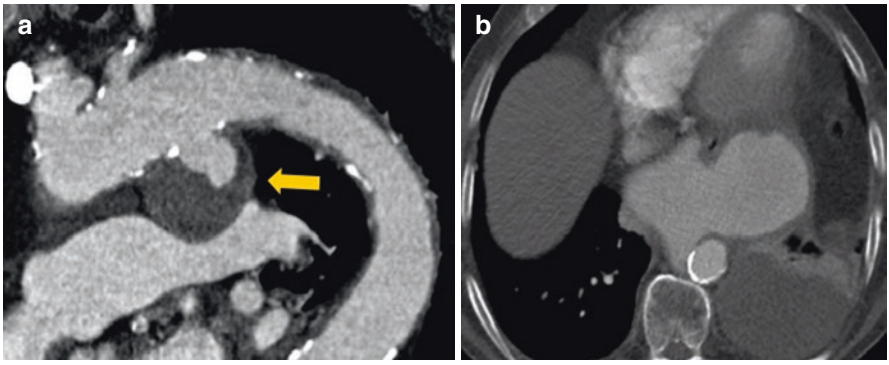

Fig. 9.5 (**a**) Large PAU in the inner curve of the aortic arch (orange arrow). (**b**) Large ruptured TAA

9.8 Conclusions

AAS are fatal cardiovascular emergencies that require complex treatment by several specialists referred in high-volume centers. In this chapter these pathologies, including TAAs, have been examined, from diagnosis to their initial treatment. Due to their peculiarity, blunt traumatic injuries of the descending thoracic aorta (which are mainly located at the aortic isthmus) and pseudoaneurysms have not been taken into consideration, although they share many similar aspects in terms of presentation, diagnosis, and management with the pathological entities discussed in this chapter.

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Pulmonary and Thoracic Emergencies

10

Francesco Damarco

Thoracic disorders are one of the most common reasons for presentation to the emergency department. It is due to various conditions that involve several organs, ranging from benign to life threatening.

Pulmonary diseases can present with pain, dyspnea, and respiratory failure but these symptoms are not always specific for a certain clinical situation (Algorithm 10.1).

Differential diagnoses in chest pain are:

- Cardiac (angina, acute myocardial infarction, pericarditis).
- Vascular (aortic dissection, pulmonary embolism).
- Thoracic (pneumothorax, pleural effusion, hemothorax, empyema, chylothorax, trauma with rib/sternal fractures, flail chest, pneumonia/pleuritis, superior vena cava syndrome).

Box 10.1 Key investigationMonitoring of vital parameters

- History: characteristics of the patient's pain (site, type, quality, onset, duration, precipitating factors), risk factors, medical history
- Physical evaluation: chest pain, dyspnea, respiratory failure, sweating, breathlessness, cough, hemoptysis, nausea and vomiting, fever, trauma
- Exams: ECG, blood test with biomarkers, ABG, chest X-rays, CT scan in case of trauma, ultrasound

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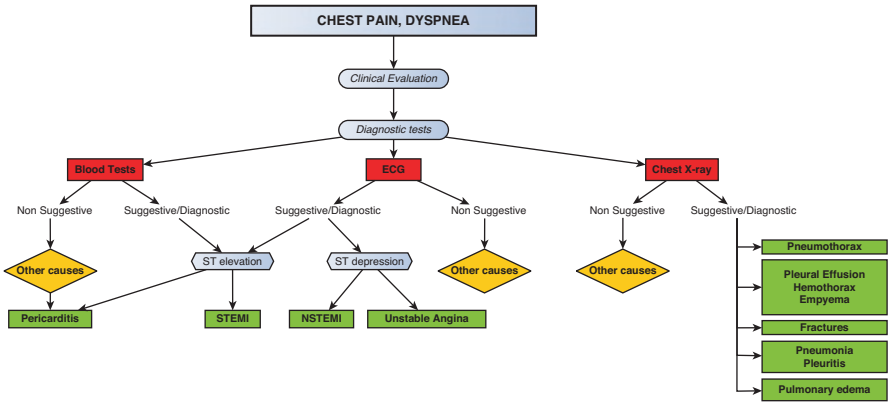
Algorithm 10.1 Simplified flowchart for patients with chest pain.



10.1 Pneumothorax

Pneumothorax is defined as the presence of air in the pleural space resulting in lung collapse (Fig. 10.1).

Depending on etiology it can be classified in spontaneous, primary (*PSP*) or secondary to underlying lung disease (*SSP*), and non-spontaneous [1, 2].

10.1.1 Etiology

10.1.1.1 Primary Spontaneous Pneumothorax (PSP)

PSP occurs in apparently normal lung in absence of external circumstances. It seems to be due to small subpleural blebs or bullae disruption, but the underlying pathogenic mechanism is not fully understood. It is more common in young and tall men between the ages of 10 and 30. The incidence ranges from 7.4 to 37 per 100,000 population per year [3, 4]. Smoking is a significant risk factor. PSP typically occurs at rest and it can be associated with chest pain, and dyspnea [5].

Small and asymptomatic first episode requires observation only followed by discharge. In case of first large and symptomatic episode, the patient should be treated air evacuation using aspiration or by placing a chest tube. A definite surgical treatment is normally proposed after a second episode, or in case of professional risks (e.g., airline personnel). Recurrence rate is estimated to be approximately 32%, with most occurring within the first year [6]. This finding increases exponentially with following pneumothorax episodes.

10.1.1.2 Secondary Spontaneous Pneumothorax (SSP)

SSP is secondary to underlying disease. It presents male preponderance with a peak of incidence after 55 years. Any lung disease can lead to pneumothorax, but the

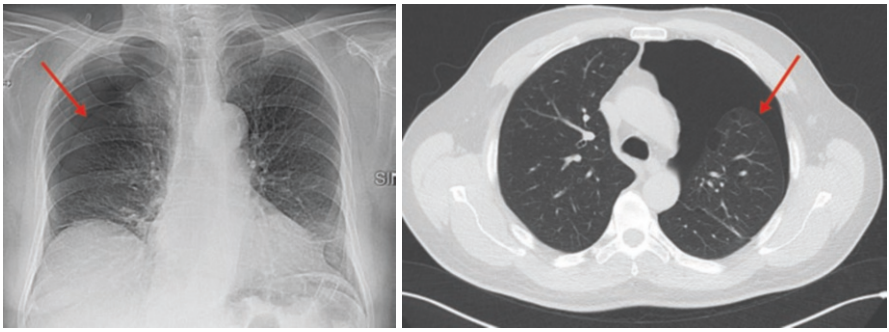


Fig. 10.1 Right- and left-sided spontaneous pneumothorax. The arrow indicates the edge of the collapsed lung

most commonly related are: COPD/emphysema (50–70%), cystic fibrosis (3–4%), cancer (16%), necrotizing lung infections (e.g. Pneumocystis jirovecii; Bacterial pneumoniae; Tuberculosis), HIV, thoracic endometriosis, genetic disorders (e.g., Marfan syndrome; Ehlers-Danlos syndrome; homocystinuria; Birt–Hogg–Dubé syndrome), cystic disorders (e.g., lymphangioleiomyomatosis; Langerhans cell histiocytosis) [2].

In SSP, dyspnea is usually more severe and requires treatment procedures.

Box 10.2 Catamenial pneumothorax

Catamenial pneumothorax defines a recurrent lung collapse in women of reproductive age in conjunction with . Pneumothorax usually occurs right-sided, within 72 h before or after the start of menstruation with combination of chest pain, dyspnea, and hemoptysis. The etiology is not fully understood but it seems to be due to the presence of ectopic endometrial tissue into the thoracic cavity. can be presumed by recurrence rates of pneumothorax in a woman of reproductive age with endometriosis. Thoracic surgical procedures are used for confirmation. Surgical treatment with potential diaphragmatic plication, pleurodesis, or pleurectomy is the treatment of choice. Hormonal treatment helps to prevent recurrences of catamenial and/or endometriosis-related pneumothorax [7].

10.1.1.3 Non-spontaneous Pneumothorax

Non-iatrogenic traumatic pneumothorax should be suspected in all patients undergoing significant trauma and it is due to blunt or more commonly penetrating thoracic trauma.

With non-penetrating injury, air enters the pleural space if the visceral pleura is lacerated secondary to a rib fracture or dislocation. In addition, abrupt chest compression increases the alveolar pressure, causing alveolar rupture.

With penetrating trauma (e.g., impalement on a foreign body, stab wounds, gunshot wounds) air can access the pleural space directly through the chest wall or from the tracheobronchial tree if the visceral pleura is torn.

Iatrogenic pneumothorax is a traumatic pneumothorax that develops as a complication of an invasive procedure. Mechanical ventilation (7%), pacemaker insertion, subclavian vein catheterization (22%), thoracentesis (20%), thoracic needle aspiration/biopsy (24%), and transbronchial lung biopsy are the most common causes of iatrogenic pneumothorax [8].

10.1.2 Signs and Symptoms

Pneumothorax is typically characterized by sudden onset, chest pain radiated to the scapula, dyspnea, and breathlessness. Especially in PSP, symptoms may be minimal or absent.

Clinical examination includes hyper-resonant percussion and reduced/absent breath sounds on the affected side. Hypoxia, acute respiratory distress, tachypnea, and hypotension are usually present in tension pneumothorax.

The imaging size of pneumothorax does not always correspond to the severity of the symptoms.

10.1.3 Diagnosis

Erect chest X-ray is the gold standard for the detection of pneumothorax. In some cases, expiratory film can help to identify pneumothorax. CT scan is not a primary diagnostic imaging, but it is generally used in trauma setting or for complex or uncertain cases. Bedside ultrasound can be useful in trauma setting and critically ill patients.

Pneumothorax is classified in "small" or "large" sized depending on whether the distance between the lung margin and the chest wall is <2 cm or ≥2 cm: ACCP guidelines measure the distance from the lung apex to the cupola, while BTS guidelines estimate the intrapleural distance at the level of the hilum.

10.1.4 Management and Treatment [9, 10]

Invasive approach is highly variable depending on the different institutions, but all patients with pneumothorax must receive care with attention to respiratory stabilization. In the following evaluation, you will need to decide if the air should be removed from the pleural space and what method to use (Algorithm 10.2).

In non-tension pneumothorax patients are clinically stable and a conservative management is often appropriate. Instead, tension pneumothorax is a medical

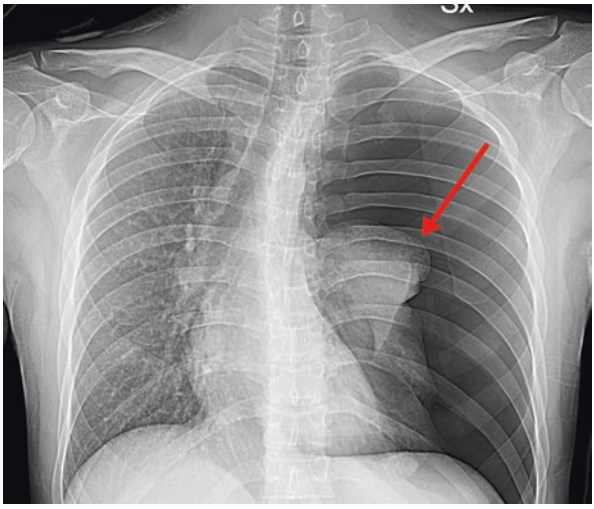


Fig. 10.2 A massive left-sided tension pneumothorax. The arrow indicates the edge of the collapsed lung.

emergency requiring urgent measures with prompt decompression: it occurs when the air enters into the pleural space but is not able to fully exit due to a one-way valve mechanism (Fig. 10.2). It leads to a gradual increase of intrapleural pressure, displace of mediastinal structures, and a compromised cardiopulmonary function. Tension pneumothorax does not require radiological confirmation [11, 12].

Box 10.3 Oxygen tips
Supplemental administration of oxygen in pneumothorax accelerates the rate of pleural air absorption in clinical and experimental situations. Use high concentration oxygen in all patients if not contraindicated by the underlying disease [13].

For PSP [9, 10]:

- Clinically stable patients with detected small volume pneumothorax should be observed and discharged in case of stability at the following chest X-ray. Indication to return in the event of worsening breathlessness.
- Selected asymptomatic patients with a large pneumothorax may be managed by observation alone.
- Breathless patients associated with any size of pneumothorax should undergo invasive approach.
  - BTS guidelines propose needle aspiration (14–16G) as first choice option. If aspiration is unsuccessful, insertion of a small-bore (<14Ch) chest drain is recommended.

  - ACCP guidelines suggest insertion of a small-bore (<14Ch) catheter or moderate-sized (16–22Ch) chest tube.
  - Moderate/large (16–28Ch) chest drain is required in unstable patients with large pneumothorax.

For SPP [9, 10]:

- Guidelines recommend hospitalization for secondary spontaneous pneumothorax.
- Use small-bore (<14Ch) chest drain for stable patients with small/large pneumothorax.
- For unstable patients guidelines suggest moderate/large (16-28Ch) chest drain.
- Referral to a thoracic surgeon for recurrence prevention.

For Traumatic pneumothorax:

- Traumatic non-iatrogenic pneumothorax is usually managed with the insertion of a large bore chest tube (28–32Ch) because chest trauma is often associated with hemothorax or could require mechanical ventilation.
- Observation and following discharge in clinically stable patients with detected small volume iatrogenic pneumothorax.
- In breathlessness patients or large iatrogenic pneumothorax, needle aspiration or insertion of a small-bore chest drain may be required.

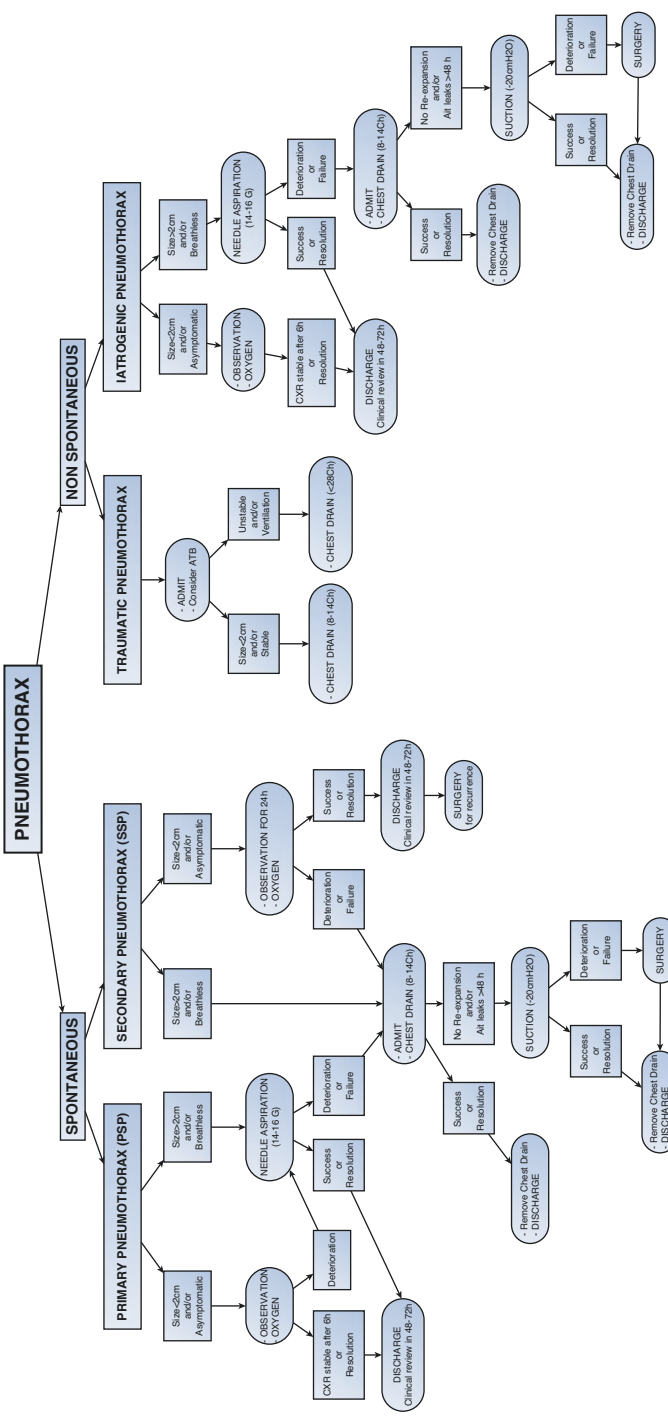
For Tension pneumothorax:

- Immediate decompression by placing a large bore (14–16G) needle in the second intercostal space at the midclavicular line or in the fifth intercostal space at the anterior axillary line.
- Insert a chest drain following needle decompression.

For any patient [9, 10]:

- In case of air leak (>48 h) or incomplete re-expansion, suction may be appropriate.
- In case of prolonged air leaks (>5 days) or failure of lung re-expansion, referral to a thoracic surgeon for definitive treatment (bleb/bullectomy combined with pleural abrasion/pleurectomy/chemical pleurodesis).

Algorithm 10.2 Flowchart for management of pneumothorax.



10.2 Pleural Effusion

Pleural effusion is defined as an abnormal deposition of fluid in the pleural space. It occurs, unilaterally or bilaterally, in several disease processes.

10.2.1 Etiology

Various pathogenic mechanisms lead to fluid accumulation by increasing the rate of pleural fluid formation or by reducing the fluid absorption (Table 10.1). Around 1.5 million people each year in the United States develop pleural effusion, with an approximate 1.1 to 1.3 million resulting from nonmalignant causes (NMPE) [8].

Malignant pleural effusion (MPE) accounts for 15–35% of all pleural effusions, and the most involved tumors are lung, breast cancers and lymphomas.

It is important to distinguish if the patient has a transudative (typically caused by increased hydrostatic pressures, decreased oncotic pressure, increased negative intrapleural pressure, or fluid migration through the diaphragm) or exudative (due to increased capillary permeability, inflammatory processes or obstruction of pleural lymphatic drainage) pleural effusion according to their protein content.

10.2.2 Signs and Symptoms

Pleural effusion is typically characterized by dyspnea, chest pain, cough, malaise, and fever.

Chest pain is frequently worsened by deep inspiration or movements.

Cough is usually dry and non-productive.

Dyspnea is not always proportional to the amount of the pleural effusion. Small amounts of effusion can be asymptomatic, even though patients with MPE more frequently present with dyspnea.

Clinical examination includes attenuated/absent tactile fremitus, dull or flat percussion, and reduced/absent breath sounds on the affected side.

Table 10.1 Main differential diagnoses of pleural effusion

Main differential diagnoses of pleural effusion	
Transudative pleural effusion	Exudative pleural effusion
Congestive heart failure	Neoplastic diseases
Cirrhosis	Infectious diseases
Nephrotic syndrome	Iatrogenic or trauma (hemothorax; chylothorax)
Hypoalbuminemia	Chylothorax
Superior vena cava obstruction	Pulmonary embolization
Peritoneal dialysis	Collagen vascular diseases
Atelectasis	Lymphatic abnormalities

Bilateral effusion is typically present in heart failure. The loculation of pleural space occurs more frequently in intense pleural inflammation such as parapneumonic effusion, empyema, and hemothorax.

10.2.3 Diagnosis

Erect chest X-ray is the first test in the detection of pleural effusion (Fig. 10.3). The posteroanterior view shows blunting of the lateral costophrenic angle when effusion exceeds 200 mL, while in the lateral view the posterior costophrenic angle is obliterated when fluid is over 50 mL.

Thoracic ultrasound is highly sensitive for pleural effusion and detects small amount of fluid (<50 mL). It provides an accurate characterization of effusion, the identification of potential loculation, the evaluation of pleural thickening and it allows to define the appropriate site for thoracentesis/chest tube placement [14].

The use of ultrasound for guidance reduces the risks of complication during invasive procedures (4% vs 9.5%) [15, 16].

CT scan yields excellent identification of pleural thickening, pleural or lung masses, and detection of loculated fluid amount.

FDG-PET is useful for differentiating benign and malignant effusion.

Thoracentesis plays a diagnostic and therapeutic role at the same time: it helps to relieve the dyspnea in symptomatic patients and allows to collect pleural fluid for further tests. The first step in the diagnosis of pleural effusion is the distinction between transudative and exudative analyzing the levels of protein and lactate

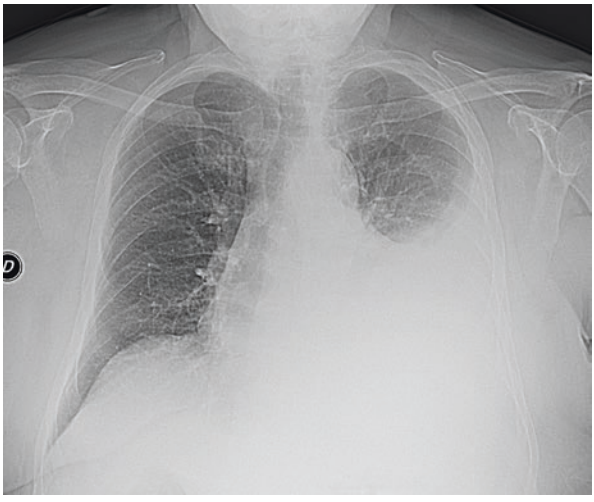


Fig. 10.3 A large left-sided pleural effusion on chest X-ray

dehydrogenase in the pleural fluid and serum according to Light's criteria [17]. A pleural effusion is likely exudative if at least one of the following exists:

- The ratio of pleural fluid protein to serum protein is greater than 0.5.
- The ratio of pleural fluid LDH and serum LDH is greater than 0.6.
- Pleural fluid LDH is greater than 0.6 or 2/3 times the normal upper limit for serum. Different laboratories have different values for the upper limit of serum LDH, but examples include 200 and 300 IU/L.

It is also possible to perform biochemical, cytological, and microbiological analysis of pleural fluid as shown in Table 10.2.

Furthermore, appearance and smell of pleural effusion provide additional information. Turbid pleural fluid can indicate increased cellular or lipid contents (empyema, chylothorax). For example, reddish effusion suggests blood presence in pleural fluid (malignant disease, trauma, hemothorax), while a brownish color denotes a long-standing process.

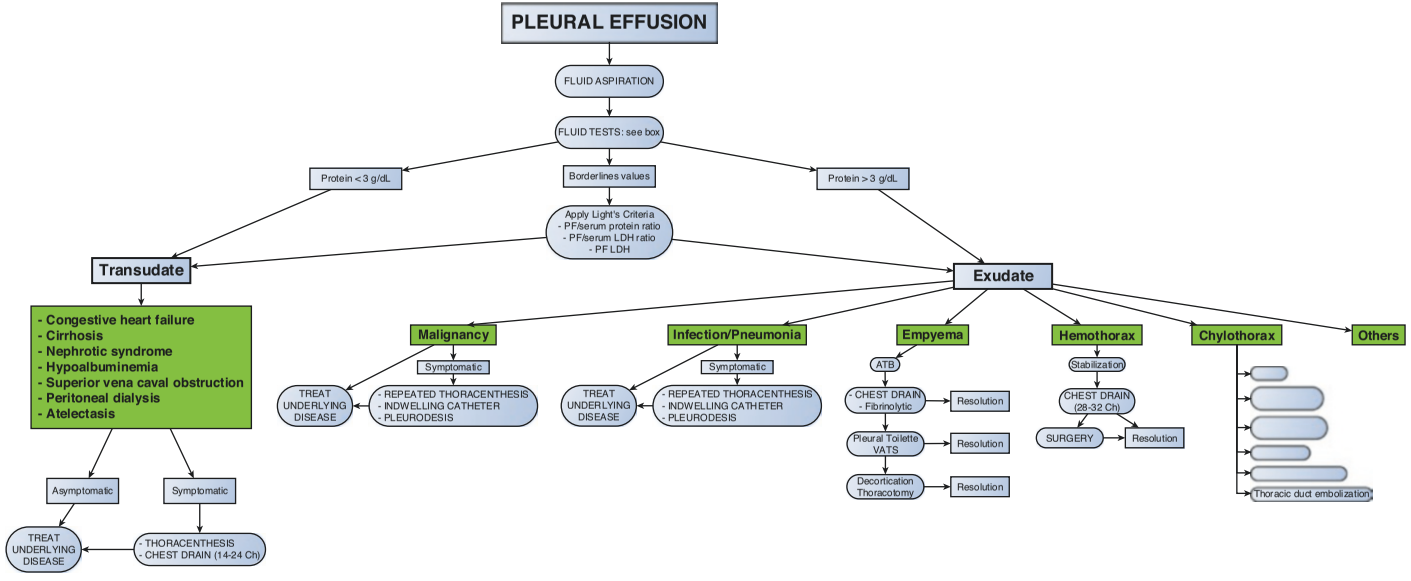
Table 10.2 Routine pleural fluid tests for pleural effusion [14]

Routine pleural fluid tests for pleural effusion		
	Value	Suggested diagnosis
Adenosine deaminase	>40 U/L	TBC (>90%); empyema (60%); complicated parapneumonic effusion (30%)
Amylase	>upper limit of normal	Malignancy; esophageal rupture
Culture	±	Infection
Cytology	±	Malignancy
Flow cytometry	±	Lymphoma
Glucose	<60 mg/dL	Empyema; TBC (20%); malignancy
Hematocrit fluid/blood ratio	≥0.5	Hemothorax
LDH	$> \frac{2}{3}$ upper limits of normal for serum LDH	Any exudative effusion
Lymphocytes	>50%	Malignancy; TBC; Pulmonary embolism
Neutrophils	>50%	Parapneumonic effusion
NT-pro-BNP	>1500 pg/mL	Heart failure
pH	<7.20	Complicated parapneumonic effusion; empyema; malignancy; esophageal rupture
Protein	>3 g/dL	Any exudative effusion
RBC count	>100,000/mm^3	Trauma; malignancy; parapneumonic effusion; pulmonary embolism
Triglycerides	>110 mg/dL	Chylothorax
WBC count	>10,000/mm^3	Empyema

10.2.4 Management and Treatment

- Asymptomatic patients with transudative effusion do not routinely require invasive pleural procedures.
- Symptomatic NMPE are normally responsive to drainage and treatment depending on the etiology of the primary disorder. A drainage technique involves US-guided needle thoracentesis or chest tube placement. BTS guidelines suggest to drain less than 1500 mL in one sitting with thoracentesis to reduce the risk of re-expansion pulmonary edema [18]. In case of chest tube placement, ERS/EACTS prefer large bore chest tubes (20–24 Ch) while other authors suggest similar outcomes with smaller diameter [19]. A significant percentage of patients present recurrence of pleural effusion: in these situations it is mandatory to repeat thoracentesis, maximize primary therapy, and re-evaluate the patients to exclude pleural infections, non-expendable lung, malignancy, or other diagnoses. Symptomatic patients with recurrence of nonmalignant pleural effusion despite of maximized medical therapy and repeated thoracentesis or chest placement are candidates for indwelling pleural catheter (IPC) and/or pleurodesis using a chemical sclerosant or manual abrasion.
- MPE usually represents advanced disease and average survival is around 3–12 months, therefore invasive treatments are intended to relieve symptoms [20]. The risk of recurrence should be considered so, after initial drainage, a definitive intervention with indwelling pleural catheter and/or talc pleurodesis is mandatory [16]. Talc is the most effective agent for chemical pleurodesis: talc poudrage is associated with a lower recurrence rate than talc slurry, but surgical procedures are more invasive and present more potential complications. IPC is also a valid option in the management of malignant trapped lung, where patients would not benefit from chemical pleurodesis.

Algorithm 10.3 Simplified flowchart for diagnosis and management of pleural effusion.



10.2.5 Hemothorax

Hemothorax is defined as the collection of blood in the pleural space (Fig. 10.4); approximately 300,000 cases are estimated every year in the United States.

Bleeding may arise from lung parenchyma, chest wall, pleura, mediastinum, great vessels, diaphragm, and abdomen. It is classified into three major groups, depending on the etiology.

Traumatic hemothorax is the most common, due to blunt or penetrating injuries to the chest. Blunt thoracic trauma occurs more frequently with an overall mortality of 9.4%, which is significantly increased in case of penetrating chest injuries [21].

Hemothorax may also be the result of *spontaneous* causes (spontaneous pneumothorax, infectious processes, coagulopathies, and malignancies) or *iatrogenic* causes (most commonly during thoracentesis, central venous catheterization, tube thoracostomy, and thoracotomy).

Hemothorax has respiratory and hemodynamic implication with potential development of hypoventilation, hypovolemia, and decreased cardiac output: rapid evaluation and intervention are crucial.

Clinical findings of hemothorax are broad and may often mimic other conditions. Signs and symptoms are dyspnea, tachypnea, hypoxia, hypotension, reduced/absent tactile fremitus, dull or flat percussion, and reduced/absent breath sounds on the affected side. Always check for signs of chest contusion, injury, ecchymosis, tenderness, paradoxical chest wall movements, and "seat belt sign."

Chest X-ray is still the initial diagnostic approach, but ultrasound is fast and more portable. It allows to determine quality and quantity of effusion and it has higher sensitivity especially in FAST protocols. After this assessment, CT can be performed to evaluate further chest detail.

All patients with hemothorax must receive care with particular attention to cardiorespiratory stabilization. Minimal blood collection (<300–400 mL) in the pleural space usually requires only conservative management with observation and

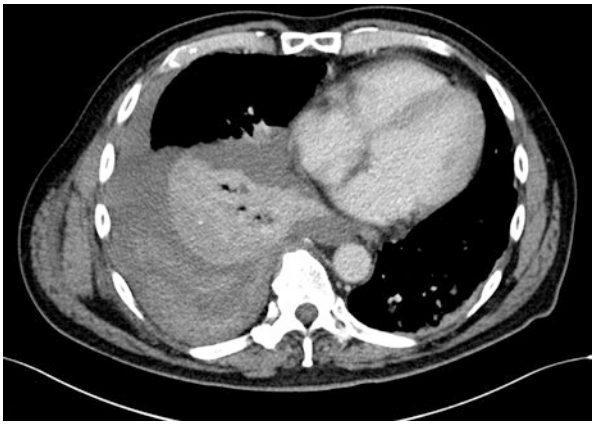


Fig. 10.4 CT scan shows a massive right-sided hemothorax

frequent imaging. Moderate and massive hemothoraces should be evacuated with a large bore chest tube (28–32 Ch): always check for the fluid output.

Consult a cardiothoracic specialist if: >1500 mL of blood drainage in 24 h; 500 mL/h for 2–4 consecutive hours after tube placement; cardiac tamponade; chest wall or great vessel injury.

Antibiotic treatment reduces the rate of infectious complications.

Retained hemothorax after evacuation is associated with significant risks for developing late complications such as empyema and fibrothorax, so in this situation all patients should undergo pleural toilette, debridement, and decortication via video-assisted thoracic surgery (VATS) or thoracotomy [22].

10.2.6 Empyema

Empyema is defined as pus in the pleural space. In the United States, the incidence is approximately 32,000 cases per year. It is mainly caused by pulmonary infection as community- and hospital-acquired pneumonia (CAP and HAP, 55–75%), but it can also occur as a result of thoracic-esophageal surgery (12–21%), thoracic trauma (3–6%), pneumothorax, hemothorax, and lung abscess (9–15%).

Risk factors for the development of empyema are diabetes mellitus, malignancy, and immunodeficiency; morbidity and mortality of pleural empyema remain high especially in these already vulnerable patients.

Aerobic gram-positive and gram-negative bacteria are predominant in community-acquired empyema (Streptococcus species, E. coli, K. pneumoniae), MRSA and gram-negative (Pseudomonas, Enterobacteriaceae) are more frequently related to hospital-acquired infection, while S. aureus is commonly seen in traumatic and surgical settings.

Microorganisms reach the pleural space through the damaged endothelium and inflammatory mediators: pathogenic material promotes fibrin depositions and suprainfection. These events led to septation with loculated effusion and progression to entrapped lung [23].

Patients with empyema typically present chest pain, fever, sputum production, and cough often associated with fatigue. On physical examination crackles, dull or flat percussion, and reduced/absent breath sounds on the affected side may be present. Laboratory tests reveal elevated infection parameters but the confirmation of certain infection on the effusion is low.

Chest X-ray is probably the first imaging test, but ultrasound allows to determine quality and quantity of effusion, to estimate potential fluid septations or loculations and to guide chest tube placement or thoracentesis. Contrast-enhanced chest CT is the gold standard (Fig. 10.5): thickening of visceral and parietal pleura with separation by a pleural fluid is pathognomonic for empyema ("split pleural sign").

Thoracentesis is a useful diagnostic tool and it allows for fluid evaluation. A turbid-purulent fluid, a pH <7.2, polymorphonucleocyte predominance, low glucose, and LDH >1000 UI/L effusion support the diagnosis of empyema and require surgical drainage.

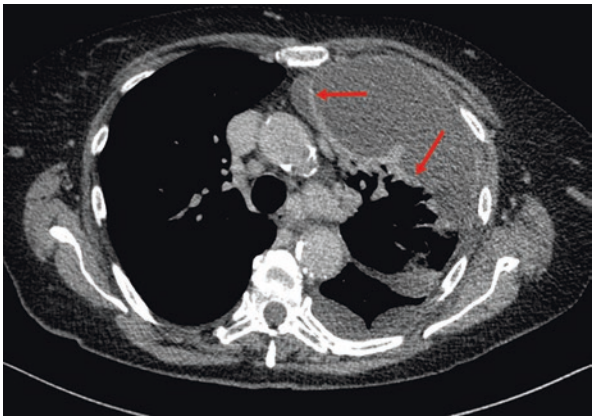


Fig. 10.5 A massive left-sided empyema. The arrow indicates the "split pleural sign"

The American Thoracic Society classified empyema in three stages as a sequence of events, characterized by different therapeutic indications:

- The exudative phase with fluid accumulation (2–5 days from the onset of pneumonia).
- The fibrinopurulent phase with the fluid colonization and the progression of infection leading to loculations (5–10 days after onset).
- The organized phase in which infection progression leads to fibrosis and entrapped lung.

At the onset of empyema, antimicrobial therapy is the gold standard treatment. The recommended treatments for CAP are third- or fourth-generation cephalosporin plus metronidazole or ampicillin with a beta-lactamase inhibitor. In case of HAP or traumatic and surgical empyema, coverage of MRSA and Pseudomonas is essential by adding vancomycin, cefepime, and metronidazole or piperacillin-tazobactam.

If disease progresses, medium-large bore chest tube insertion (16–28 Ch) with evacuation of effusion is required and usually sufficient in 75% of the cases. The adjunctive instillation of fibrinolytic agents helps to debride septation facilitating the exit of the pleural fluid.

When sepsis persists and the lung is not properly unfolded in the pleural cavity, surgical intervention is mandatory. Minimally invasive approach can be proposed for the fibrinopurulent phase to perform pleural toilette, while pleural decortication usually requires thoracotomy [24].

10.2.7 Chylothorax

Chylothorax is the accumulation of chyle in the pleural cavity due to rupture or obstruction of the thoracic duct or its branches. It can be classified as traumatic 50% (complication of surgery, blunt or penetrating injury), non-traumatic 44% (malignancy, infection, congenital disorder, and other conditions), and idiopathic 6%.

The effusion is typically turbid and milky, and the amount varies widely up to >1000 mL per 24 h.

Clinical features depend on the rate of chyle loss as well as the etiology. Symptoms can follow pleural effusion expression: dyspnea, cough, and eventually chest pain.

The loss of protein, fats, vitamins, and ions can result in malnutrition, weight loss, muscle wasting, and electrolyte disorder, especially in chronic conditions.

On physical examination dull or flat percussion and reduced/absent breath sounds may be present on the affected side.

Investigation of chylothorax begins with the confirmation of the diagnosis by fluid analysis and ends with the discovery of the etiology. The pleural fluid is very rich in large chain fatty acids and lymphocytes.

Definitive management of chylothorax depends on the cause and it can include dietary therapy, use of somatostatin/octreotide, thoracentesis or chest tube insertion, pleurodesis, thoracic duct ligation or embolization [25].

10.3 Trauma: Rib Fractures, Sternal Fractures, Diaphragm Injury

Traumas are one of the main causes of mortality worldwide. Specifically, thoracic traumas represent the leading cause of mortality along with vertebral and cranial traumas.

The outcome is determined by the severity of the trauma: the first assessment should evaluate circulation and airways considering respiratory and hemodynamics parameters. Major life-threatening conditions to consider after thoracic traumas are massive hemothorax, tension and open pneumothorax, flail chest, respiratory obstruction, air embolism, and cardiac tamponade. Nevertheless, emergency sternotomies and thoracotomies are required in 1–2% of cases [26].

Frequent thoracic site injured by a trauma is the chest wall, followed by pleura and lung parenchyma.

10.3.1 Rib Fractures

Rib fractures are the most common type of injury (35–40%), mainly located along 4th–9th ribs along the mid-axillary line (Fig. 10.6). Due to the support of the scapula, shoulders, and clavicle, fractures of the first and second rib are rare and imply high-energy trauma.

The majority of rib fractures are associated with blunt trauma (e.g., motor vehicle crashes, falls), but they can also result from penetrating injury, severe coughing, bone metastasis, or athletic activities.

Typical symptoms are point tenderness and pain, exacerbated by coughing, deep breathing, and movement.

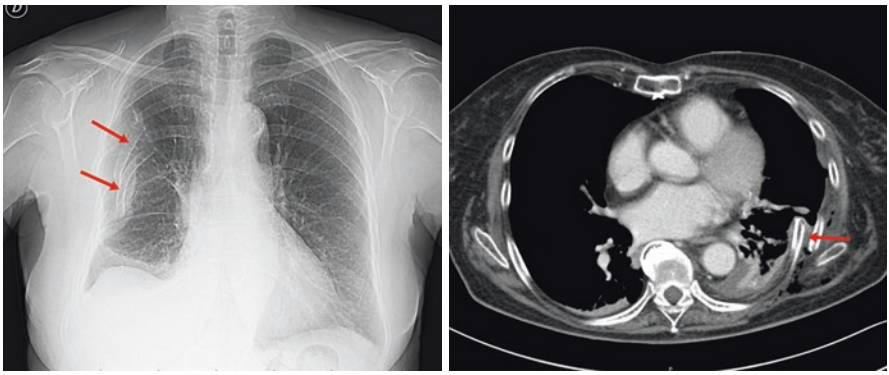


Fig. 10.6 Multiple rib fractures as indicated by the arrows

Ecchymosis and bone crepitus may be present on physical examination.

Several complications are related with rib fractures such as hemothorax, pleural effusion, pneumothorax, pneumonia, ARDS, and organ injury (e.g., pleural, lung, cardiac, bronchial, liver, spleen damage): therefore, it is important to identify potential associated problems.

Fractures of ribs 1st–3rd may be associated with mediastinal and great vessels injury; fractures from 9th to 12th can result in intra-abdominal lesions. Displaced rib fractures increase the risk of injury to the intercostal blood vessels and to the lung parenchyma.

Chest X-ray is the first diagnostic tool but it can be falsely negative. Chest CT is more specific and it allows to identify even small alterations in cortical bone and quantify possible organ damages.

Optimal treatment depends on type and extension of rib fracture. A conservative management based on pain control and pulmonary rehabilitation is the standard of care for non-displaced fractures with a good prognostic outcome. Adequate pain alleviation using nonsteroidal anti-inflammatory drugs, opioids, or intercostal nerve blocks is essential to avoid complications as well as incentive spirometry to prevent atelectasis. The pain can persist for several days after the injury, but most rib fractures fix within 6–12 weeks.

Patients are able to return to work within a few days, but for heavy labor or sport at least 3 weeks off are recommended.

Indication to surgical fixation aims to prevent potential complications, but there is no consensus. Stabilization is typically proposed in selected patients when rib stumps are severely displaced or in case of flail chest: bicortical screws, Kirschner's wires, Judet's struts, titanium bar, and intramedullary rods or splints are the most used devices.

Flail Chest is the paradoxical movement of the chest wall that occurs during breathing when three or more consecutive ribs are fractured in two places. This floating segment of rib sections and soft tissues moves independently from chest wall, outward during expiration and inward during inspiration, and it is usually

associated with underlying pneumothorax, pulmonary contusion, and/or hemothorax.

Pain, low chest wall expansion, increase in respiratory work, and the paradoxical movement with mediastinal shift can result in respiratory and hemodynamic impairment.

Diagnosis is most often clinical with confirmation of the paradoxical motion during breathing. Crepitus of the flail segment on palpation and chest X-ray can help confirm the clinical situation.

The treatment of flail chest is based on supportive care, strong respiratory physiotherapy, and analgesic pain medications. Mechanical ventilation may be necessary in unstable patients with severe pulmonary dysfunction. As mentioned above, corrective surgery can be proposed to reduce risk of pneumonia, morbidity and mortality or in non-ventilable patient.

10.3.2 Sternal Fractures

Sternal fractures usually result from blunt anterior chest-wall trauma, deceleration injuries, athletic injuries, and falls (incidence 3–6.8%). They can occur in isolated or associated injuries, but the prognosis is mostly excellent. Fractures are more prevalent in older patients and females.

The most common site of the fractures is located in the body and in the sternal manubrium, oblique or transverse.

Typical signs and symptoms are acute sternal pain and point tenderness, exacerbated by coughing, deep breathing, and movement. Ecchymosis, bone crepitus, and eventual step deformity may be present on physical examination.

Chest X-ray is the gold standard investigation: lateral projection usually allows to diagnose displacement or dislocation, while antero-posterior view helps to detect eventual further injuries (Fig. 10.7).

Chest CT is suitable to determine secondary trauma.

Patients with sternal fractures should be assessed according to ATLS guidelines, as well as all chest trauma.

ECG and cardiac enzymes generally suffice for stable patients with isolated sternal fractures.

Echocardiography, continuous monitoring, and ICU admission for unstable patients with intrathoracic injuries, especially if myocardial contusion is suspected.

Routine hospital admission is warranted in high-impact trauma, major comorbidities, inadequate pain control, or severely displaced fractures [27].

Pain control and pulmonary rehabilitation are the treatment of choice for isolated sternal fractures. They will recover spontaneously in around 10–12 weeks.

Complications can involve painful pseudoarthrosis and overlap deformities. Therefore, surgical fixation is requested in selected cases of non-union or chronic sternal pain. Transverse and oblique fractures of the sternal body generally require

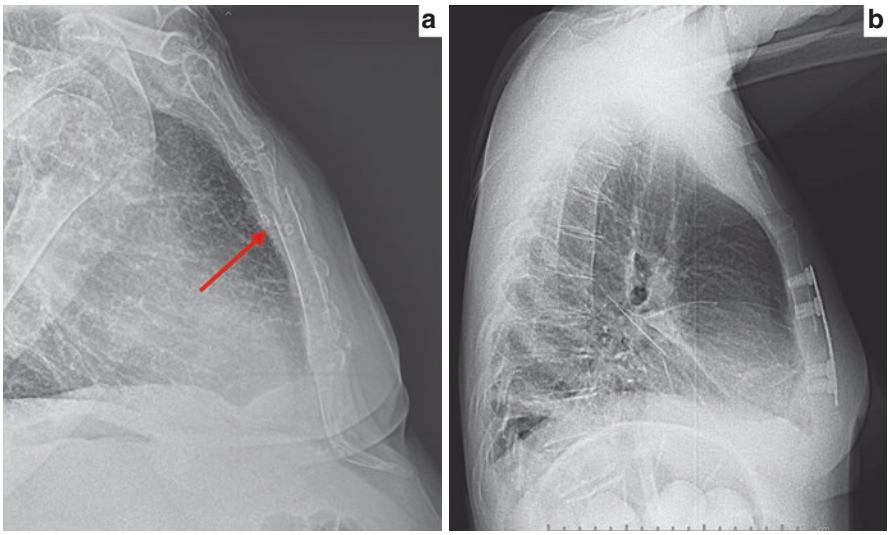


Fig. 10.7 (**a**) The arrow indicates the sternal fracture; (**b**) Example of metal bar placement for sternal fracture

longitudinal fixation with metallic prosthesis. Longitudinal, sternocostal split or oblique fractures of sternal manubrium are stabilized using transversal plate or bar from rib to rib [28].

10.3.3 Diaphragm Injuries

Diaphragm injuries are a rare complication following blunt and penetrating trauma (0.8–15%).

Two-thirds of all diaphragm injuries are associated with blunt traumas because of a sudden increase in intra-abdominal pressure. On the right side diaphragm is well protected by the liver, therefore injuries occur most commonly on the left side.

As a consequence of the muscular defect, abdominal organs may herniate into the chest leading to inappropriate ventilation, obstruction, or rupture of the abdominal viscera.

Signs can be either thoracic or abdominal and they include flat lung percussion, reduced/absent breath sounds, and auscultation of bowel sounds in the chest. Dyspnea, respiratory distress, chest/abdominal pain, nausea and vomiting due to obstruction of the viscera may arise depending on timing and severity of the clinical situation.

In an acute setting, diaphragm injury is hard to diagnose. Most of the time chest X-ray can show diaphragm elevation, atelectasis, blunting of the costophrenic sinus, and visceral herniation with diffuse gas bubbles.

Focal constriction of the abdominal organs through the diaphragm defect, called "collar sign," is another suggestive sign.

When in doubt, abnormal positioning of nasogastric tube can support the diagnosis but CT scanning allows to confirm not only diaphragm damages but also other potential concomitant injures.

In the acute phase, the main treatments are based on ATLS guidelines. In case of prompt diagnosis, reduction of visceral herniation and repairing of diaphragm defects under laparotomy or laparoscopy is the gold standard for hemodynamically stable patients. In case of delayed confirmation, video-assisted thoracoscopic surgery and thoracotomy are usually required to help lysis and visceral reduction [29, 30].

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Upper Gastrointestinal Tract Acute Conditions

11

Marco Giovenzana, Beatrice Giuliani, and Nicolò Maria Mariani

11.1 General Approach to Upper Gastrointestinal Bleeding

Firstly, assess the patient's hemodynamic stability, and proceed with resuscitation if necessary. Secondly, diagnostic studies, usually endoscopy, follow with the aim of diagnosis, and treatment of the specific disorder, when possible [1, 2].

11.1.1 Assessment of Hemodynamic Stability

The initial evaluation consists of a history of symptoms, physical examination, and laboratory tests. The aim is to evaluate the severity of the bleed and identify potential sources of the bleed.

- Patients with acute upper gastrointestinal (GI) bleeding commonly present with hematemesis and/or melena, if there have been warnings in the previous days. Symptoms that suggest a severe bleeding are orthostatic dizziness, confusion, angina, tachycardia, and cold extremities.
- The physical examination is a fundamental part of the evaluation of hemodynamic stability. The first assessment is to determine whether there are any signs of bleeding in the stool (proctorragy or melena).
  - If the patient is undergoing martial therapy and reports black, tarry stool, the presence of melena cannot be defined with certainty. In this case perform hydrogen peroxide test: H_2O_2 generates foam in the presence of blood.

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If the patient is unstable, it is essential to immediately stabilize the patient according to the common accepted guidelines: placement of at least two peripheral venous accesses of large caliber, administration of O_2 therapy, placement of a bladder catheter, and especially volemic adjustment while waiting for blood tests, on the basis of which, in case of anemia, you can possibly set up a transfusion therapy with blood derivatives. In order to know more quickly the state of anemia of the patient, the execution of an arterial blood gases can be useful, as its result is almost immediate.

The placement of a nasogastric tube is still debated, between those who are in favor of its placement, in order to quantify the blood loss and to avoid possible pneumonia ab ingestis, and those who are against, not considering the maneuver of any obvious benefit, as well as possible source of recurrence or new bleeding.

In our experience, it is a usual practice because it allows immediate evaluation of the bleeding characteristic. If ongoing bleeding, we perform a gastric lavage, better with cold water and ice, with the goal of vasoconstricting, reducing the bleeding, and flush the stomach of clots so as to facilitate subsequent intervention by the endoscopist. In any case, remember that blood spillage can cause anemia and melena with falsely negative nasogastric stagnation.

To facilitate endoscopy and decrease the risk of aspiration in patient with current hematemesis or altered metal status/respiratory, elective endotracheal intubation can be taken into consideration.

– Always monitor ABCDE parameters: clinical status, vital signs, cardiac rhythm, urine output, nasogastric output.

- Laboratory tests should include a complete blood count, liver and coagulation studies, and serum chemistries. Moreover, serial electrocardiograms and cardiac enzymes are suggested in patients who are at increased risk for a myocardial infarction.

  False positive:

  The initial hemoglobin value may be falsely normal, because the patient is losing whole blood.

  False negative:

  Typically, after 24 h or more, the hemoglobin level will decline because the fluid administration during volemic resuscitation dilutes the blood.

  – Importance of vital parameters, i.e., hypotension, anuria, tachycardia, unless the patient is on beta-blocker therapy.
- Administration of proton pump inhibitor (PPI), prokinetics, and vasoactive drugs is indicated, while there is no evidence for tranexamic acid in the treatment of upper GI bleeding.

11.1.2 Diagnostic Studies

Upper endoscopy is the diagnostic and therapeutic gold standard procedure for acute upper GI bleeding.

Early endoscopy (within 12–24 h) is recommended for most patients with acute upper GI bleeding, while in the case of a stable patient, the endoscopic procedure

can be performed in deferred urgency; however, if the patient remains unstable, early endoscopic examination can significantly improve survival [3].

In emergency, in acute esophageal variceal hemorrhages as bridging therapy while waiting for the endoscopic procedure, the positioning of a Sengstaken-Blakemore probe represents a save-life procedure [4]. It consists of two Foley balloons at its caudal end: a more distal one, useful for anchoring beyond the cardia, and an oblong one, more proximal, to ensure a progressive pressure at the distal esophageal level, offering a control of bleeding by compression of the varices. If it is used, remember to proceed to a progressive and not too rapid increase in the inflation pressure of the esophageal balloon, in order to limit any episodes of emesis and to cause iatrogenic injury of the distal esophagus.

Other diagnostic tests for acute upper GI bleeding may include CT angiography (CTA) and angiography, which can report active bleeding. In patients with melena and a negative upper endoscopy, a colonoscopy can be performed unless an alternative source of the bleeding has been identified.

11.2 Etiologies of Upper Gastrointestinal Bleeding and Therapeutic Approach

It's important to investigate whether there have been previous episodes of upper GI bleeding, since almost 60% of patients whith a history of an upper GI hemorrhage, bleed from the same lesion. Moreover, analyze any important comorbid conditions in the patient's past medical history that may lead to upper GI bleeding or may influence the clinical management.

11.2.1 Esophagus

Esophageal varices represent the leading cause of esophageal bleeding, followed by bleeding from Mallory-Weiss lesions secondary to reflux disease.

Endoscopic hemostasis is the therapy of choice, in particular in an urgent/emergency context, as it allows to perform an injection and/or mechanical hemostasis through the positioning of clips.

Surgical treatment, challenging both for the urgency and for the difficulty in reaching and controlling the distal esophagus, is not advisable as a treatment of first choice and would in any case require a resective approach, not easy to perform from a technical point of view.

11.2.2 Stomach

Gastric bleeding is usually secondary to chronic erosive gastric disease or an ulcer. In most cases it is a slow and constant bleeding, which is the reason why the patient presents to the general practitioner or to the emergency room complaining of melena; in rare cases in which the bleeding is massive, you can also see episodes of hematemesis.

The therapy consists of endoscopic hemostasis by injection of adrenaline or placement of metal clips, or radiological embolization. The surgical approach represents the last choice and consists of gastric resection or ulcer suturing.

- False negative: Dieulafoy's lesion, a vascular anomaly with a tortuous artery in the thickness of the submucosal tonaca that can be the cause of even massive gastrointestinal bleeding, is difficult to diagnose endoscopically because of the massive presence of blood, the size of the lesion, and the intermittent nature of the bleeding.

11.2.3 Duodenum

Bleeding secondary to duodenal ulcer. Unlike gastric bleeding, which, if massive, may give hematemesis, bleeding from a duodenal ulcer is more often an intermittent or tassel-like bleed and therefore the patient's primary presenting symptom in the emergency department will be melena, lipothymia, and fatigue, rather than pain.

11.3 General Approach to Upper Gastrointestinal Perforation

Clinical manifestations of upper gastrointestinal perforation depend on the nature of the substances released (gas, succus entericus, bile, blood), as well as the ability of the surrounding tissues to contain those contents.

Full-thickness injury and subsequent perforation of the upper gastrointestinal tract can be caused by different etiologies, including:

- Instrumentation (e.g., endoscopy, instillation of contrast, cautery application during surgery)
- Trauma (blunt or penetrating)
- Medications, other ingestions, foreign body
- Vomiting
- Neoplasms

11.3.1 Assessment of Hemodynamic Stability

A detailed history is important in evaluating patients with neck, chest, and abdominal pain. Physical examination should include vital signs, a complete examination of the abdomen, chest, and neck. Vital signs may initially be normal or reveal mild tachycardia or hypothermia. As the inflammatory response progresses, fever and other signs of sepsis may develop.

Palpation of the neck and chest should search for signs of subcutaneous emphysema and auscultation and percussion of the chest for signs of effusion. Abdominal

examination may initially be relatively normal or reveal only mild focal tenderness, as with tamponade or retroperitoneal perforations.

11.3.2 Diagnostic Studies

The presence of air outside the gastrointestinal tract in the abdomen (i.e., pneumoperitoneum) or mediastinum (i.e., pneumomediastinum), or complications associated with perforation leads to the diagnosis.

The diagnostic evaluation in mild symptomatic patients often begins with upright radiographs of the chest and abdomen, while the first diagnostic approach in severe acute conditions is computed tomography (CT).

- In our experience, when there is a suspicion of perforation, we perform a CT scan with oral administration of Gastrografin, as it confirms the original hypothesis and allows us to see the site of the perforation and its extent (tamponade or diffuse perforation). This information is critical to the decision of subsequent management.

11.4 Etiologies of Upper Gastrointestinal Perforation and Therapeutic Approach

The past medical history should include questioning about prior attacks of abdominal or chest pain, recent upper-GI procedures (placement of nasogastric tube, endoscopy, surgery), malignancy, abdominal trauma, ingested foreign bodies (e.g., fish or chicken bone ingestion), and medical conditions (e.g., peptic disease), with particular attention to medication that predispose to gastrointestinal perforation (e.g., NSAIDs).

11.4.1 Esophagus

Perforation of the esophagus is more often iatrogenic (endoscopy, surgery) followed by non-iatrogenic penetrating or blunt traumatism. Other causes may include tumors, foreign body or caustic ingestion, pneumatic injury, vomiting or, more rarely, it is spontaneous (Boerhaave's syndrome).

From the therapeutic point of view, it is necessary to keep the patient fasting, setting an adequate infusion therapy, a correct analgesic therapy and administering proton pump inhibitors intravenously; a broad-spectrum antibiotic therapy is advisable in order to limit possible septic complications (mediastinitis, pleurisy). An infectiological consultation could help in the choice of the most appropriate drug.

In case of esophageal perforation, given its particularity and the difficulty of its management, it is mandatory to alert the surgical team immediately for the possible intervention [5].

11.4.2 Stomach

Gastric perforation is mainly caused by a peptic ulcer which, whether or not it has previously bled, causes full-thickness damage to the gastric wall, creating a continuous solution between the intragastric acid environment and the abdominal cavity. The presenting symptom is stabbing pain in the epigastrium and radiating to the left hypochondrium, sometimes to the back. Among the prodromes, melena may be present if there has been continuous bleeding in the days preceding the actual perforation. A history of non-specific pain and subsequent continued NSAID therapy may also be a warning sign of perforation.

- Remember that in case of a tamponade ulcer, the abdomen may not be peritonitic.

Other causes include iatrogenic (endoscopy, surgery) or non-iatrogenic trauma, ingested foreign bodies, and neoplasm (particularly during chemotherapy).

The perforated tamponated gastric ulcer can be managed conservatively: the setting of an adequate infusion therapy and with proton pump inhibitors, together with a broad-spectrum antibiotic therapy is essential. In case of open perforation in the abdomen, peritonism, hemodynamic instability, or sepsis, it is essential to alert the surgical team for exploratory treatment in an emergency. The best surgical approach, if anesthesiologically permissive, is laparoscopic.

11.4.3 Duodenum

Perforation is less frequent and is more often related to endoscopic or surgical procedures than to the ulcer itself or to the perforation of a duodenal diverticulum, usually located between the second and third portion of the viscera.

- If the patient is hemodynamically stable, given the largely retroperitoneal position of the duodenum, these types of perforations are often managed conservatively.
- In general, in case of upper-GI perforation, the hemodynamic stability of the patient, the septic status, and the extent of the perforation itself guide the therapeutic choice:
  - Diffuse perforation (e.g., peritonitis) and instability/septicemia: surgical intervention.
  - Localized, tamponade perforation and hemodynamic stability: multimodal or step-by-step approach (endoscopist-interventional radiology and surgeon).

11.5 General Approach to Upper Gastrointestinal Ingestion of Foreign Bodies and Food Impactions

Foreign body ingestion and food impactions are not uncommon event that occur accidentally in adults.

11.5.1 Assessment of Hemodynamic Stability

The symptomatology is characterized by acute onset of dysphagia, foreign body sensation, mostly retrosternal, refusal to eat, wheezing, hypersalivation, and pain.

It's always essential to investigate the type of foreign body, the time of ingestion, and the presence and type of ongoing symptoms. In fact, foreign bodies represent a rather dangerous event, both for their nature and for their shape and size: there are objects of spheroidal shape and therefore not dangerous, but with a possible harmful content, such as a battery, or objects of non-toxic material, such as a toothpick, but with a sharp shape and therefore potentially harmful to the hollow viscera of the gastro-enteric tract.

Physical examination should also include an inspection of the oro- and hypopharynx, neck, chest, and abdomen to identify patients with esophageal obstruction (drooling and inability to swallow liquids), or perforation.

11.5.2 Diagnostic Studies

From a diagnostic point of view, radiographic localization and identification of foreign bodies is valuable in guiding management.

Chest-abdomen X-ray can confirm the size, shape, number, and location of a radio-opaque foreign body while food bolus, thin metal objects, wood, plastic, glass, and bones are not readily seen. CT is therefore a second-level diagnostic study, indicated but not mandatory for all cases of foreign body ingestion.

11.5.2.1 Esophagus

In an emergency context the only examination that may have a value is the CT scan, initially even without contrast medium: it can document the distension of the esophagus upstream of the foreign body/bolus, the size, the possible presence of mineralized material and free periesophageal air in case of perforation; the use of contrast could possibly facilitate the recognition of structures, such as the esophageal wall, with respect to foreign material.

11.5.2.2 Stomach

In asymptomatic patients, the priority is to establish the characteristics of the object by performing a chest-abdomen X-ray in order to document the position of the same; If the examination is not diriment for the location of the foreign body or shows possible complications (e.g., intra-abdominal free air), an abdominal CT scan with contrast medium is mandatory.

11.6 Etiologies and Therapeutic Approach

The approach to management is guided by the initial evaluation.

11.6.1 Esophagus

If the patient is compliant and the bolus consists of material of soft consistency (rice, pasta, bread, meat, fruit, vegetables), the "push" method can be used, which allows, through the positioning of a nasogastric tube, the progression of the bolus into the stomach by mechanical push. This maneuver, we reiterate, must be performed only when there is adequate certainty of the nature of the bolus and without forcing the push if the probe should not progress beyond the obstacle, in order to avoid any esophageal injury. Some authors also suggest the use of coke [6] in an attempt to mobilize the bolus, although this technique is certainly less effective and has a high failure rate. In case the bolus is made of material potentially harmful to the bowel, such as a bone, the endoscopist's intervention is essential for its removal [7]; the urgency of the procedure is related in particular to the symptoms and to the possible detection of complications on imaging, if performed.

- Emergent endoscopy (2–6 h) is indicated in patients with any of the following:
  - Complete esophageal obstruction as evidenced by drooling and an inability to handle oral secretions
  - Disk batteries in the esophagus
  - Sharp-pointed objects in the esophagus
- Urgent endoscopy (within 24 h): Any other foreign body should be retrieved within the 24 h following the ingestion, because the risk of complications dramatically increases with time.

11.6.2 Stomach and Proximal Duodenum

A foreign body potentially harmful, by shape and/or nature, which is located in the stomach or proximal duodenum, should be removed as soon as possible, in order to limit the early progression of the same along the intestinal tract, an event that would make surgery the only possible treatment of removal.

Foreign bodies that should be removed are definitely sharp metal objects (e.g., sewing needles), old batteries, and razor blades; all other foreign bodies should be considered potentially harmful and each case should be discussed with the reference endoscopist specialist. This evidence lies in the fact that potentially non-injurious objects (medium-sized coin, new generation batteries, dental prostheses) may not lead to a perforation of the digestive tract but could still be obstructive at any level of the jejunum-ileum but especially at the ileo-cecal valve; in this case it may be necessary to perform a colonoscopy or, if ineffective, surgery (enterotomy and removal of the foreign object).

- Food and objects: Therapeutic management is endoscopic, except on rare occasions, such as:
  - Bolus without bones or liches
  - Perforation (see above)
  - Object no longer detectable or removable endoscopically
- Remember that therapeutic choice is also time-related.

11.7 Ingestion of Caustics

The ingestion of caustics is usually due to a voluntary gesture performed for the purpose of self-harm even if, in some cases, the event may occur involuntarily [8]. The first thing to verify is the nature of the product and the likely amount ingested. These data, together with the objective examination, are fundamental to be able to identify the therapy and the eventual necessary investigations. Caustic substances can be divided into three groups: strong acids (e.g., hydrochloric, sulfuric, nitric, phosphoric, hydrofluoric acids), strong alkalis (e.g., ammonia, sodium carbonate, sodium hydroxide, potassium hydroxide), and oxidizing agents (e.g., peroxides, potassium permanganate). The most frequent and severe lesions for the esophagus are due to basic caustic substances (bleach) and for the stomach to acids (button batteries). The greatest injuries are localized to the antrum due to pyloric spasm.

Once the patient has been evaluated, it is essential to contact the Poison Centre, active 7/24, and communicate all the data collected. It is then the specialist who recommends the optimal therapy to be administered and the timing for the endoscopic examination. On the other hand, it is always useful to perform a CT scan of the neck, chest, and abdomen with contrast medium to evaluate the integrity of the potentially damaged viscera, the detection of extraluminal air, collections, or additional alterations.

- Do not place a nasogastric tube by yourself! Some combinations of caustics can damage the mucosa which, with the placement of nasogastric tube, would risk perforation. Always follow the instructions of the Poison Centre.

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Multidisciplinary Surgical Consensus on Chest Emergencies

12

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Clinical case: A 58-year-old male patient, with an history of alcohol abuse, current smoker under antipsychotic therapy, appeared in emergency department for chest pain after vomiting. Cardiological emergencies were immediately excluded (EKG and blood tests were negative). The CT scan with oral contrast showed pneumomediastinum and extravasation of contrast from distal esophagus: a collection of fluid with air-fluid levels was described in the posterior mediastinum, at the level of the right inferior pulmonary vein. At the abdominal scan, there was evidence of pneumoperitoneum at the esophageal-gastric passage and at the small gastric curvature, with extravasation of free contrast.

Considering the clinical deterioration and the radiological findings, the patient was taken to the operating room: a right posterolateral thoracotomy was performed through the sixth space access. Samples of pleural fluid were collected for culture examination. After opening the mediastinal pleura, the esophagus was isolated below the inferior pulmonary vein. Using the endoscope, a 4 cm long laceration was identified immediately above the cardia, and the breach was sutured directly. Indocyanine green test was performed, with evidence of good visceral esophageal vascularization. An intercostal muscle flap was used to buttress the suture and a

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drainage tube was placed in the mediastinum. Methylene blue was administered through the nasogastric tube to check for leakage, and an abdominal drainage tube was placed in the supramesocolic lodge after washing it thoroughly; the last step was the packing of the jejunostomy.

On the seventh postoperative day, due to the evidence on the CT scan of two fistulas in the distal third of the esophagus, a prosthesis was placed endoscopically. The postoperative course was characterized by the onset of infectious, respiratory, and renal complications; enteral nutrition was reintroduced 1 month after surgery, with no complications.

12.1 Spontaneous Esophageal Injuries

Esophageal injuries (EI) are an uncommon but potentially lethal event. A multidisciplinary approach involving all the relevant actors (thoracic and general surgeons, endoscopists, anesthesiologists, and radiologists) has proven to reach better outcomes.

EI are characterized by transmural disruptions of the esophageal wall. The leakage of esophageal contents into the surrounding structures leads to local and systemic inflammation, and increases the risk of sepsis [1].

Overall incidence is unclear but esophageal perforation presents high mortality rate ranging from 4% to 40%. The outcome depends on several factors, but essentially on early diagnosis and proper treatment.

Esophageal injuries are mainly iatrogenic (e.g., naso-enteric tube placement, endoscopic exams, surgical procedures), traumatic (e.g., blunt or penetrating trauma), or spontaneous (e.g., Boerhaave syndrome) [2].

Spontaneous rupture, *or* Boerhaave syndrome from the name of the doctor who first described it in 1729, represents 15% of all injuries, estimated in 3.1 cases per 1,000,000 per year [3].

It most commonly occurs in connection with vomiting, as a consequence of an increase in intraluminal esophageal pressure, and less frequently with childbirth, or seizure [4].

It appears usually among patients aged 50–70 years and chronic alcohol consumption is a risk factor.

Although injuries can occur along the entire esophagus, the perforation is most commonly observed in the left posterior aspect of the distal esophagus [5].

Esophageal rupture may be followed by serious complications such as mediastinitis, abscess formation, and multiple organ dysfunction. It is therefore important to obtain a proper picture of the clinical condition.

EI can be misdiagnosed as cardio-pericardial (e.g., myocardial infarction, pericarditis, acute aortic syndrome, pulmonary embolism), respiratory (e.g., spontaneous pneumothorax, pneumonia), or abdominal pathological conditions (e.g., perforated peptic ulcer).

It is a rare life-threatening condition and requires urgent diagnosis and treatment: patient outcomes are directly related to the timing.

12.1.1 Signs and Symptoms

Retrosternal chest pain is the main symptom (70%) and it may radiate to the left shoulder or to the back depending on the injury's location. Dyspnea and vomiting are very frequent (25%) [5].

Mackler's triad, including vomiting, subcutaneous emphysema, and thoracic pain, is pathognomonic for esophageal perforation but unfortunately is uncommon [6].

Atypical symptoms such as tachypnea, tachycardia, odynophagia, hypotension, restlessness, and cyanosis may also be present.

Pneumomediastinum can be a significant associated clinical finding with the characteristic Hamman's sign, a clicking or crunching sound synchronous with the heartbeat, which can be heard over the precordium.

- Cervical perforation: neck pain, dysphagia, or dysphonia.
- Intrathoracic perforation: sepsis may rapidly develop.
- Intra-abdominal perforation: epigastric pain with eventual irradiation to the shoulder, back pain, and an inability to lie supine or possible eventual acute (surgical) abdomen.

12.1.2 Diagnosis

Laboratory findings may be not specific, and Boerhaave syndrome is often diagnosed incidentally during the evaluation of a chest pain [6].

Chest X-ray usually shows signs of pneumothorax, pneumomediastinum, or pleural effusion.

Esophagogram with water-soluble contrast (e.g., Gastrografin®) can help to confirm the diagnosis and to locate the perforation.

CT scan is extremely effective in detecting perforations. Typical findings include esophageal wall edema and thickening, periesophageal fluid with or without gas bubbles, mediastinal widening, and air and fluid in the pleural spaces or retroperitoneum. The ingestion of Gastrografin® allows to estimate the esophageal perforations, their extension, and to guide any subsequent therapeutic interventions [5] (Fig. 12.1).

EGDS is not early recommended for diagnosis, since it may aggravate the rupture increasing the air and the esophageal contents in the mediastinum and pleural space.

12.1.3 Thoracic Treatments

The management of Boerhaave syndrome is still an unsolved problem, and the different therapeutic approaches depend on the esophageal injuries and the patient's conditions at the time of presentation [7] (Table 12.1).

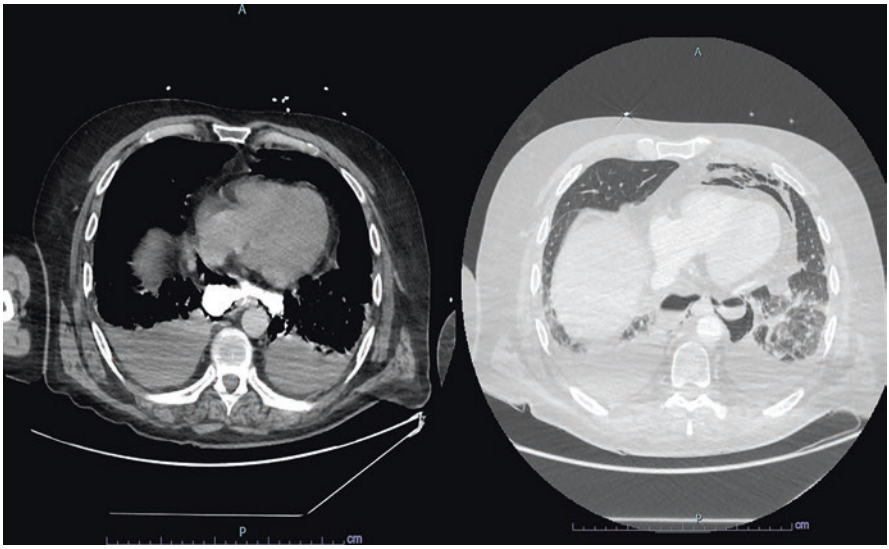

Fig. 12.1 Left distal esophageal rupture associated with pleural effusion, periesophageal fluid, and pneumomediastinum. Gastrografin® helps to locate the perforation

Table 12.1 Esophageal rupture management

Medical management:
Avoidance of all oral intake
Nutritional support, typically parenteral
Intravenous broad-spectrum antibiotics
Intravenous proton pump inhibitor
Drainage of fluid collections/debridement of infected and necrotic tissue, if present
Surgery:
Patients who are not candidates for or who fail conservative management require surgical approach

Surgery is the most widely used approach and should be performed within 24 h from the onset of symptoms: beyond this timing, mortality exceeds 56% and increases with the passing of the hours [8].

Proper management in esophageal injuries includes fluid replacement, nutritional support, control of sepsis with antibiotics, drainage, and primary repair of the perforation.

Conservative treatments should be applied only in selected patients because in esophageal perforations it is difficult to treat the infection.

Surgical procedures and primary repair remain the gold standard therapy: they range from simple drainage, to debridement, repair of the perforation, or esophagectomy. The specific procedure depends on etiology and site of perforation, time from diagnosis, and surgeon's experience.

Chest or mediastinal drainage helps to control the sepsis (the positioning technique of a chest tube has already been discussed in Chap. 8 "Chest: surgical anatomy and general consideration in emergency settings"), but it is often not enough.

Early diagnosis and primary repair play a key role in patient survival: it was traditionally performed using open thoracotomy, but the use of minimally invasive approach is progressively increasing [7].

Irrigation of pleural space, drainage and debridement, direct suture of the esophageal mucous membrane with the muscularis propria and adventitia are the most common procedures.

Soft tissue coverage with intercostal muscle flap or omental patch increases the tightness of the sutures.

In some cases, esophagectomy is required [9].

The general principles for the management of an intra-abdominal esophageal perforation include a careful dissection to isolate the esophagus without damaging vital structures, removal of debris and devitalized tissues, and debridement of the area of perforation. Laparotomy is usually the preferred approach.

Following primary suture repair, the hiatus is closed posteriorly with interrupted silk sutures. A Dor or a Nissen (fundoplication) is used to buttress the site of repair depending on the site of perforation and patients' preoperative history of swallowing dysfunction. Finally, drainages are placed near the site of repair, and a feeding jejunostomy tube can be placed for postoperative alimentation.

In the postoperative period, attention must be paid to nutritional support and decompression of the first intestinal tract. The most common postoperative complication is the leakage from the suture, which occurs in up to 40% of the operated patients: to decrease that chance it is necessary to cover the site of perforation through appropriate vascularized tissue. Moreover, the use of indocyanine green appears to be an option to verify the vascularization of the flaps.

Endoscopic treatments should be considered in patients with extensive underlying comorbidities who are unlikely to tolerate surgery. They include esophageal stent placement or endoscopic vacuum-assisted closure (EVAC) in order to stimulate granulation tissue and healing by second intent.

12.2 Conclusion

Boerhaave syndrome is rare, and there is limited evidence to guide management.

For this reason, a multidisciplinary approach among general surgeon, thoracic surgeon, and endoscopist is essential to ensure the best treatment for the patient.

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Part IV

Abdomen

Abdomen: Surgical Anatomy and General Consideration in Emergency Settings

13

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13.1 Basic Surgical Abdominal Anatomy

The abdominal cavity has a complex and intricate anatomy. A physician must know in which area of the abdomen every major structure is located to understand the clinical presentation of abdominal pathologies and/or in trauma situations to estimate which organs are most likely injured. The general surgeon, especially in emergency situations, uses this knowledge to execute the most advantageous surgical approach for a particular situation [1].

13.1.1 Surface Anatomy of the Abdomen and Abdominal Wall

13.1.1.1 Boundaries

Superior:

- Xiphoid process
- Costal cartilages of the 7th–10th ribs

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Inferior:

- Pubic bone and pubic symphysis
- Inguinal ligaments

Lateral:

- Superior: inferior aspect of the 10th rib
- Inferior: iliac crest

Landmarks

1 Umbilicus
2 Linea alba: fibrous junction of the right and left rectus muscles that runs down the midline of the abdomen
3 Semilunar lines: lateral borders of the rectus abdominis muscles.
4 Anterior superior iliac spine
5 Pubic symphysis
6 Inguinal grooves

13.1.1.2 Lines and Planes

- Right and left midclavicular lines
- Subcostal plane
- Transtubercular, or intertubercular, plane

13.1.1.3 Regions of the Abdomen

- Right and left hypochondria
- Epigastrium
- Right and left lateral abdominal regions or flanks
- Umbilical
- Right and left inguinal regions or iliac fossae
- Hypogastrium

13.1.1.4 Abdominal Wall

Layers:

- Skin
- Superficial fascia (subcutaneous tissue): Superficial fatty layer (Camper's fascia) and deeper membranous layer (Scarpa's fascia)
- Anterior fascial layer: Anterior rectus sheath (medial) and external abdominal oblique fascia (lateral)
- Abdominal muscles: Rectus abdominis–External abdominal oblique–Internal abdominal oblique–Transversus abdominis
- Posterior fascial layer: Posterior rectus sheath (medial; ends at the arcuate line midway between umbilicus and pubic symphysis) and transversalis fascia (lateral)
- Peritoneum

13.1.1.5 Inguinal Canals

Boundaries:

- Superior: inferior edge of the internal oblique and transverse abdominal muscles
- Inferior: inguinal ligament (continuation of the external abdominal oblique aponeurosis)
- Anterior: aponeurosis of the abdominal external oblique muscle
- Posterior: transversalis fascia and interfoveolar ligament

Course:

- The inguinal canal is approximately 4 cm long, situated in the lower anterior abdominal wall, connecting its outer and inner layers above the inguinal ligament (superior anterior iliac spine to pubic tubercle).
- Runs from an upper lateral to an inferior medial direction.
- *Inner orifice/deep inguinal ring*: evagination of the transversalis fascia (surrounding the spermatic cord as the internal spermatic fascia).
- *External orifice/superficial inguinal ring*: fissure in the aponeurosis of the abdominal external oblique muscle.

13.2 Initial Assessment of the Patient with Abdominal Pain

Abdominal pain is the most common reason for a visit to the emergency department (ED) and is the presenting issue in a high percentage of medicolegal actions against both general and pediatric physicians. The modern physician should be humbled by the fact that, despite diagnostic and therapeutic advances (computed tomography [CT], ultrasonography, and laparoscopy), the misdiagnosis rate of the most common surgical emergency, acute appendicitis, has changed little over time.

13.2.1 Assessment of the Patient's Pain [2]

The classic PQRST mnemonic for a complete pain history is as follows:

- P3—Positional, palliating, and provoking factors
- Q—Quality
- R3—Region, radiation, referral
- S—Severity
- T3—Temporal factors (time and mode of onset, progression, previous episodes)

This mnemonic will help to ensure a thorough history, but rigidly following the above sequence does not allow for a smooth patient interview, so the authors prefer to ask the patient where they feel the pain (location), what kind of pain it is (character), when and how it began (onset), how bad it is (intensity), and where else they feel it, what makes it worse or better, how it has changed over time, and whether they have ever had it before.

Embryology determines where a patient will "feel" visceral pain, which is generally perceived in the midline because afferent impulses from visceral organs are poorly localized.

Clinicians should seek to distinguish between the dull, poorly localized, aching, or gnawing pain generated by viscerally innervated organs, compared with the characteristically "sharp," more defined and localized somatic pain caused by irritation of the parietal peritoneum or other somatically innervated structures. Somatic pain is transmitted via the spinal nerves from the parietal peritoneum or mesodermal structures of the abdominal wall. Noxious stimuli to the parietal peritoneum may be inflammatory or chemical in nature (e.g., blood, infected peritoneal fluid, and gastric contents).

Acute-onset pain, especially if severe, should prompt immediate concern about a potential intra-abdominal catastrophe. The foremost consideration would be a vascular emergency such as a ruptured abdominal aortic aneurysm (AAA) or aortic dissection. Other considerations for pain of acute onset include a perforated ulcer, volvulus, mesenteric ischemia, and torsion; however, these conditions may also occur without an acute onset.

The neural pathways give rise to predictable patterns of referred pain and radiation. Kehr's sign is a classic example where diaphragmatic irritation, usually from free intraperitoneal blood, causes shoulder pain.

Persistent worsening pain is worrisome, while pain that is improving is typically favorable. Serious causes of abdominal pain generally present early in their course; however, delays in presentation can occur, especially in the elderly. Certain patterns of progression can be diagnostic, such as the migration of pain in appendicitis where the initial distention of the appendix causes a periumbilical visceral pain that shifts to the right lower quadrant once the inflammatory process is detected by the somatic sensors of the parietal peritoneum. Although labeled "colic," gallbladder pain is generally not paroxysmal, and it almost never lasts less than 1 h, with an average of 5–16 h duration, and ranging up to 24 h. Small bowel obstruction typically progresses from an intermittent ("colicky") pain to more constant pain when distention occurs. One would only expect somatic pain (arising from transmural ischemia or perforation contiguous to the parietal peritoneum) late in the course of a bowel obstruction.

Gastrointestinal and urinary symptoms are the primary focus; however, it is important to ask about fever and cardiopulmonary symptoms. Associated symptoms should be placed in the clinical context, including the patient's age and the current point in the course of the illness.

With appendicitis, most physicians expect the patient to report anorexia. However, pooling of the literature indicates that, while this is a discriminatory symptom, it is only present in 68% of patients with appendicitis. The report of this symptom decreases to 20–44% in elderly patients with appendicitis.

Vomiting may occur in almost any abdominal disease.

While diarrhea is a frequent accompaniment of more benign abdominal conditions, its presence alone should never rule out serious disease. For example, diarrhea is common with mesenteric ischemia and is frequently reported in conditions such as appendicitis.

Many genitourinary tract diseases can present with abdominal pain. Conversely, any inflammatory process contiguous to the genitourinary tract (including appendicitis, cholecystitis, pancreatitis, or any inflammatory process involving bowel) may result in both pyuria and dysuria.

13.2.2 Physical Examination

The general appearance of the patient is noted first. An "ill-appearing" patient with abdominal pain is always of great concern given the variety of potentially lethal underlying causes. On the other hand, especially in the elderly, the clinician must not be misled by the well appearing patient who may still have serious underlying disease. The clinician should take note of the patient's position, spontaneous movements, respiratory pattern, and facial expression.

Take care of:

- Vital signs
- Abdominal examination (inspection, auscultation, percussion, and palpation)
- Test for peritoneal irritation

Determining the presence or absence of peritonitis is a primary objective of the abdominal examination; unfortunately, the methods for detecting it are often inaccurate. Traditional rebound testing is performed by gentle depression of the abdominal wall for approximately 15–30 s with sudden release. The patient is asked whether the pain was greater with downward pressure or with release. Despite limitations, the test was one of the most useful in a meta-analysis of articles investigating the diagnosis of appendicitis in children.

13.2.3 Approach to the Unstable Patient [3]

On occasion, a patient with acute abdominal pain will present in extremis. The ill-appearing patient with abdominal pain requires immediate attention. This is particularly so in the elderly, as the overall mortality rate for all older patients with acute abdominal pain ranges from 11% to 14%, and those presenting in an unstable fashion have an even poorer prognosis.

The usual sequence of resuscitation is applied to the unstable abdominal pain patient with airway control achieved as necessary. Hypotension requires the parallel process of treatment and an early assessment for life-threatening conditions requiring emergent surgical intervention. Hypotension from blood and fluid loss from the gastrointestinal tract is usually apparent from the history coupled with a digital rectal examination. If this evidence is lacking in the patient with abdominal pain, there needs to be early consideration of third spacing, which can cause enormous fluid shifts into the bowel lumen or peritoneal space in bowel obstruction or other intestinal catastrophes. Bedside ultrasonography is an extremely useful diagnostic adjunct in such patients. In the older patient, hypotension should prompt an

immediate search for an abdominal aortic aneurysm, immediately followed by sonographic evaluation of the inferior vena cava for intravascular volume status, and sonography of the heart, pleural, and peritoneal spaces to exclude massive effusions or evidence of massive pulmonary embolus. Bedside echocardiography will also identify severe global myocardial depression as a cardiogenic cause of shock. In the younger patient, a large amount of free fluid detected by ultrasound in an unstable patient is most commonly due to rupture of an ectopic pregnancy, spleen, or hemorrhagic ovarian cyst. An immediate urine pregnancy test will be the first step in distinguishing these.

The proper place for the unstable patient with an acute abdominal aortic aneurysm is the operating room or, in some centers, the interventional suite for emergency aortic stent placement. Attempts to obtain CT imaging may cause fatal delays in definitive treatment. With a high clinical index of suspicion (if possible, supported by emergency bedside ultrasonography), most patients sent directly to surgery will be found to have an acute AAA, and nearly all others will have an alternative diagnosis that still needs operative intervention.

13.2.4 Diagnostic Studies

Appropriate diagnostic testing is covered in the respective chapters for specific entities; however, it must be emphasized that there are significant limitations of imaging and laboratory studies in the evaluation of acute abdominal pain and all diagnostic tests have a false-negative rate. If the history and physical examination leads to a high pre-test probability of a disease, a negative test cannot exclude the diagnosis. For example, the total leukocyte count can be normal in the face of serious infection such as appendicitis or cholecystitis. CT is frequently used in evaluation of the patient with abdominal pain. Clinicians are enamored with the recent advances in the technology that have allowed for improved image resolution and shorter acquisition times along with coronal and three-dimensional reconstruction. However, it remains an imperfect test for conditions such as appendicitis and may add little to the clinical assessment.

Plain abdominal radiographs are of limited utility in the evaluation of acute abdominal pain. Although they may be helpful (free intraperitoneal air, calcified aortic aneurysm, air fluid levels in obstruction), other diagnostic studies are almost always indicated or perform better as the initial testing. If plain radiographs are utilized, the limitations must be appreciated. For example, a standard upright film will not demonstrate free air in up to 40% of patients with a perforated ulcer.

The oft repeated axiom of “treat the patient, not the test” certainly applies in the patient with acute abdominal pain. An unexpected negative test result should prompt a reassessment of the patient and consideration for observation and repeat examination for disease progression. Whenever the diagnosis is in question, serial examination as an inpatient in an observation unit or in the ED is a sound strategy. When a patient is discharged home after an evaluation for abdominal pain, the authors recommend instructions to return if the pain worsens, new vomiting or fever occurs, or

if the pain persists beyond 8–12 h. Such instructions are targeted at ensuring the return of a patient who has progressed from an early appendicitis or small bowel obstruction, the two most common surgical entities erroneously discharged from an ED.

13.3 Some Useful Considerations in Emergency Setting

13.3.1 Timing of Surgery

Emergency surgery is required for many patients suffering from trauma, acute (surgical) disease process, or surgical complications. However, not all emergencies are equal. Some need surgery as soon as possible, for example, patients with major intra-abdominal hemorrhage or vascular compromise associated with bowel ischemia. Patients with generalized peritonitis might benefit from a short period for stabilizing the physiology as long as antimicrobial treatment is promptly started, and the delay does not exceed a couple of hours. The acceptable delay for patients where prolonged delay might lead to generalized peritonitis and poorer outcome (acute appendicitis) or more invasive surgical treatment and prolonged hospital stay (acute cholecystitis), respectively, is more controversial, and the trends seem to go in opposing directions. Nonoperative management with antibiotic treatment for acute uncomplicated appendicitis is gaining more favor, whereas early (laparoscopic) cholecystectomy for acute cholecystitis or even symptomatic cholelithiasis is supported by several recent studies. Most surgeons would agree that patients with non-strangulated small bowel obstructions or infected pancreatic necrosis (unless in septic shock) do not need to be operated on in the middle of the night, and the same is true for many other abdominal emergencies. Finally, there are many nonelective procedures that are performed by emergency surgery teams that are not true emergencies such as changing dressings in open abdomen patients or performing tracheostomies for patients from intensive care or acute neurology units. Prioritizing emergency operations by urgency and using some form of categorization into different groups is becoming more common. The so-called traffic light color coding system has been used at the Helsinki University hospital for a decade. It consists of three categories *coded red (surgery as soon as possible), orange (surgery within 24 h), and yellow (surgery within 48 h)*. Only patients with red code are operated on at nighttime. The majority of emergency surgery is performed during the daytime (three designated operation tables for emergency surgery) or during the evening shift ending at 22.00 h. Several studies show that in most patients with a surgical emergency, an operation performed as soon as possible is beneficial from a medical point of view, as it reduces complications and length of hospital stay. It saves hospital resources (every day spent waiting in a surgical ward for an emergency operation is a wasted day), and patients appreciate not having to wait for surgery longer than necessary. However, nighttime surgery is expensive and might not be as safe as surgery performed during regular hours. The solution to the dilemma could include the following components at least. Emergency surgery should be seen as an equal to

elective surgery, thereby guaranteeing sufficient daytime operating room capacity, and should probably be separated to an independent "production line" not affected by unexpected delays in elective procedures requiring substitute personnel from the emergency surgery teams. Accumulation of patients waiting for emergency surgery should be minimized by a flexible system that permits adjustments to the inevitable day-to-day variation in patient numbers [4].

13.3.2 Role of Laparoscopy in Emergency Setting

Abdominal emergencies can also be operated on through the laparoscopic approach: the approach can be diagnostic laparoscopy, surgery assisted by laparoscopy or laparotomy directed according to the findings of the laparoscopy. The general contraindications refer above all to the state of haemodynamic instability of the patient and to seriously ill patients (ASA IV). In the absence of any specific counterindications for the specific laparoscopic procedure to be carried out, many abdominal diseases requiring emergency surgery can be performed with the laparoscopic approach. The most frequent indications are appendicitis, acute cholecystitis, gastroduodenal perforation, occlusion of the small intestine, and some abdominal traumas. With a correct selection of patients and the appropriate experience of the surgeon, the results are excellent and better than open surgery (less infection of the wound, complications, hospital stay, and postoperative pain). A detailed explanation is given of the basic aspects of the surgical technique in the most frequent procedures of emergency laparoscopy [5].

13.3.3 Final Consideration

Patients undergoing emergency abdominal surgery managed by high volume surgeons have better survival outcomes. These findings contribute to the ongoing discussion regarding configuration of emergency surgery services and emphasize the need for effective clinical governance regarding observed variation in outcomes within and between institutions [6].

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Acute Abdominal Aorta and Visceral Vessel Disease

14

Davide Esposito and Elena Giacomelli

14.1 Introduction

Data from the National Hospital Ambulatory Medical Care Survey of 2006 (NHAMCS) reports that abdominal pain is the most common specific principal reason given by adult patients for visiting the Emergency Department after chest pain [1].

The evaluation of a patient presenting at the emergency department with acute abdominal aorta or visceral vessel disease is very often difficult since the clinical diagnosis is made elusive by the non-specificity of the symptoms, such as abdominal pain, back pain, or hypo/hypertension.

14.2 Acute Abdominal Aorta

Acute disorders of the abdominal aorta include a range of conditions which could be potentially lethal and require prompt recognition and management. In this scenario, imaging plays a crucial role in rendering it possible to make a precise diagnosis. Computed tomography (CT) represents the first-line imaging approach, with magnetic resonance imaging (MRI) as an alternative method for stable patients who have a contraindication to iodinated contrast [2].

Acute abdominal aortic syndrome comprises unstable or ruptured aneurysm, penetrating atherosclerotic ulcer, intramural hematoma (IMH) and dissection, but also more unusual conditions such as inflammation, infection, traumatic injury,

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fistulization, and occlusion of the abdominal aorta, all of which could likely be catastrophic if misdiagnosed.

14.2.1 Abdominal Aortic Aneurysm

An aneurysm is defined as a dilation, more or less circumscribed, of the caliber of an artery [3]. The current definition of abdominal aortic aneurysm (AAA) is based on the measurement of the diameter of the abdominal aorta: an aneurysm is considered a diameter greater than or equal to 3 cm [4]. The prevalence of AAA increases with age and is most common in men [5]. The segment of aorta most commonly involved is the infrarenal.

The most threatening complication of an AAA is rupture, with mortality rates ranging from 70% to 94% [6]. The risk of aneurysm rupture increases with AAA diameter, aneurysm expansion rate, smoking, and uncontrolled hypertension [7]. Rupture usually presents with a classic clinical manifestation consisting of pain (abdominal, back or flank), hypotension, and a pulsatile mass.

The suspicion of an aneurysm rupture must be investigated using CT imaging. The primary imaging findings of AAA rupture include a retroperitoneal hematoma extending directly from the aneurysm and active extravasation of contrast material (Fig. 14.1a). In case of impending or contained rupture, the findings may be much more subtle, including perianeurysmal soft tissue stranding.

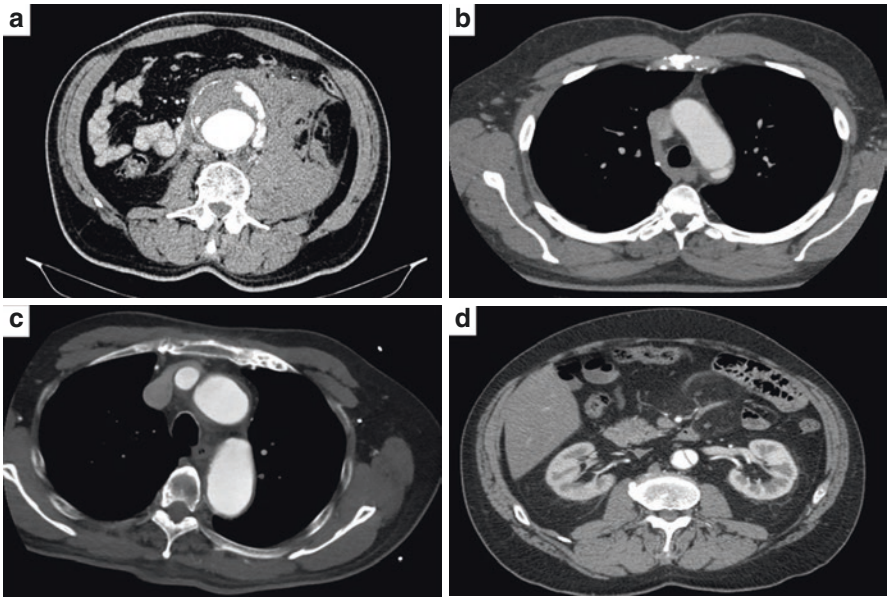

Fig. 14.1 CT images of ruptured abdominal aortic aneurysm (**a**), penetrating atherosclerotic ulcer (**b**), intramural hematoma (**c**), aortic dissection (**d**)

Treatment of an acute or impending rupture can be both endovascular aneurysm repair (EVAR) and open surgery.

BOX Signs of acute or impending rupture of aneurysms of the abdominal aorta:

Clinical: pain (abdominal, back or flank), hypotension, pulsatile mass.
Diagnostic: retroperitoneal hematoma, active contrast extravasation, perianeurysmal stranding.

14.2.2 Penetrating Atherosclerotic Ulcer

The term penetrating atherosclerotic ulcer (PAU) describes an ulcerating atherosclerotic lesion that penetrates the intima and progresses through the internal elastic lamina into the media [8]. It is most common in older patients and results from preexisting atherosclerotic disease most frequently in the middle and distal thirds of the thoracic aorta. Typically, patients present with acute intense chest pain, often described as tearing, ripping, migrating, or pulsating [9].

PAU may resolve spontaneously, but can progress to an enlarging IMH, dissection, subadventitial pseudoaneurysm, or rupture.

On imaging, PAU presents as a focal outpouching of the aortic lumen with associated hematoma in the aortic media (Fig. 14.1b).

If the pathology involves the ascending aorta, early/urgent or emergent surgical intervention is recommended [10]; when it involves the descending aorta, if asymptomatic, aggressive (antihypertensive) medical therapy in combination with close clinical and radiographic follow-up is recommended [11], if symptomatic or with signs of progression, endovascular stent-grafting (TEVAR) should be the treatment of choice [10].

BOX Signs of penetrating atherosclerotic ulcer:

Clinical: acute intense chest pain, described as tearing, ripping, migrating, or pulsating.
Diagnostic: focal outpouching of the aortic lumen with associated hematoma in the aortic media.

14.2.3 Intramural Hematoma

IMH is caused by spontaneous rupture of the vasa vasorum into the aortic media with resultant weakening of the aortic wall. Other theories describing the pathogenesis include thrombosis of a dissection lumen, microscopic intimal tears, progression from a PAU, and traumatic medial injury [8]. Clinical features of IMH are chest pain radiating to the back and hypertension.

On CT and MR imaging, IMH appears as an eccentric, crescent-shaped collection of blood in the aortic wall (Fig. 14.1c).

If an IMH involves the ascending aorta, surgical treatment is offered to prevent rupture and progression to classic aortic dissection. Conservative management is indicated for an IMH of the descending aorta.

BOX Signs of intramural hematoma:

Clinical: chest pain radiating to the back and hypertension.
Diagnostic: eccentric, crescent-shaped collection of blood in the aortic wall.

14.2.4 Aortic Dissection

Aortic dissection occurs when blood enters the medial layer of the aortic wall through a tear or penetrating ulcer in the intima and tracks longitudinally along with the media, forming a second blood-filled channel (false lumen) within the vessel wall.

The most common risk factor for aortic dissection is uncontrolled hypertension, even if there are other characteristic conditions associated with its development such as Marfan syndrome, bicuspid aortopathy, vasculitis, cocaine use, and pregnancy [12].

Clinical manifestations depend on the location of the dissection: when occurring proximally in the abdominal aorta, it may involve the mesenteric and/or renal arteries, leading in some cases to end-organ ischemia and causing affected patients to present with abdominal and/or flank pain; when distally, it may occlude the iliac and/or femoral arteries, leading to lower extremity ischemia; patients could also present with paraplegia if the artery of Adamkiewicz is involved.

Abdominal aortic dissection appears on imaging studies as an intimal flap dividing the aorta into true and false lumens. The true lumen typically is smaller than the false lumen, is surrounded by calcifications when present, and enhances more rapidly than the false lumen (Fig. 14.1d).

Treatment options include aggressive blood pressure control with beta-blockers as they reduce both blood pressure and also heart rate, or immediate surgical repair depending on the extension of the pathology.

BOX Signs of aortic dissection:

Clinical: if mesenteric and/or renal arteries involved, patients could present with abdominal and/or flank pain; if iliac and/or femoral arteries involved, lower extremity ischemia.
Diagnostic: intimal flap dividing the aorta into true and false lumens.

14.3 Abdominal Visceral Vessel Disease

Abdominal visceral vessel diseases are time-sensitive conditions which put perfusion of critical organs at risk, leading to the potential for ischemia, infarction, and translocation of enteric microbes, bacteremia, and sepsis.

In visceral artery conditions, blood flow through these arteries becomes reduced or blocked. Most often, the narrowing or blockage is caused by thrombosis; more rarely, visceral artery disease involves aneurysms formation or is secondary to abdominal traumas.

14.3.1 Mesenteric Ischemia

Mesenteric ischemia refers to vascular compromise of the bowel and its mesentery that in the acute setting has a very high mortality if not treated promptly.

It presents clinically as a severe abdominal pain that is disproportionate to examination findings and that responds poorly to analgesia.

Mesenteric ischemia can be classified into acute (most common) or chronic and specifically, in the acute setting, it could be determined by either arterial thrombosis/embolism, venous thrombosis, or non-occlusive mesenteric ischemia (NOMI).

CT is now the investigation of choice for patients with suspected intestinal ischemia, by virtue of its capacity to volumetrically assess the whole abdomen in multiple vascular phases and to diagnose alternative causes of acute abdominal pain. Common CT imaging features result from the bowel wall necrosis and perforation and include: pneumatosis intestinalis (gas in intestinal wall), pneumatosis portalis (gas in the portal vein or in mesenteric vein), pneumoperitoneum (perforation of the bowel), submucosal hemorrhage and free fluid in the abdomen [13].

In general, treatment is surgical and depends on the severity and extension of the ischemia, with the need of bowel viability assessment and eventual necrotic tissue resection, along with endovascular thrombolysis/thrombectomy when needed.

BOX Signs of mesenteric ischemia:

Clinical: severe abdominal pain that is disproportionate to examination findings and that responds poorly to analgesia.

Diagnostic: pneumatosis intestinalis, pneumatosis portalis, pneumoperitoneum, submucosal hemorrhage and free fluid in the abdomen.

14.3.2 Visceral Artery Aneurysms

Visceral artery aneurysms, which include renal and splanchnic lesions, are quite rare and usually asymptomatic. According to a large case series, 95% of the visceral artery aneurysms are detected during routine investigation into unrelated abdominal symptoms, with splenic and hepatic representing the most common types; aneurysms that rupture are typically greater than 2 cm, so this is often considered the threshold for repair in patients with asymptomatic disease [14].

Most of the times these aneurysms go asymptomatic until the time of rupture, which is a rare occurrence commonly presenting with abdominal pain.

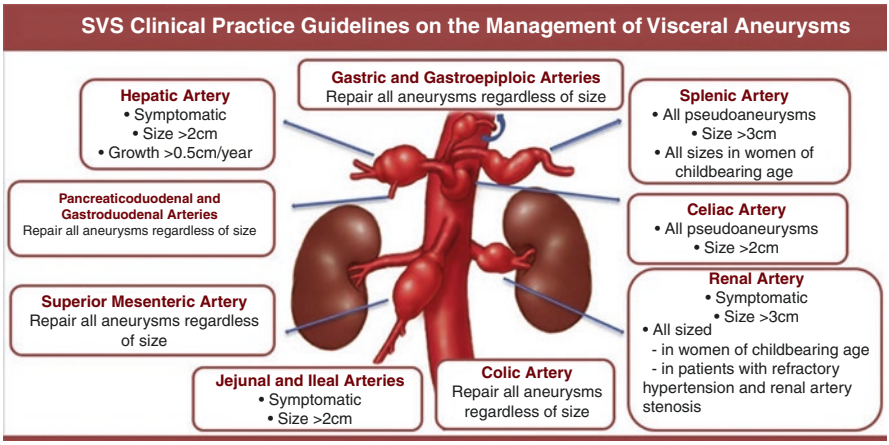

Fig. 14.2 Schematic representation of SVS Clinical Practice Guidelines on the Management of Visceral Aneurysms [15]

Focusing on splenic artery aneurysms, there is an increased prevalence of this disease in women, particularly those who are multiparous, so a high index of suspicion must be taken in such scenarios.

Hepatic artery aneurysms instead are more common in men and are typically associated to vascular diseases such as fibromuscular dysplasia and polyarteritis nodosa; unruptured hepatic artery aneurysms could cause symptoms secondary to compression on the biliary tree.

Renal artery aneurysms are not infrequently bilateral and mostly of saccular type occurring prevalently at the bifurcation of the main renal artery or first-order branch.

Visceral vessel aneurysms appear on CT imaging as contrast-filled outpouching in the course of the artery.

Treatment recommendations vary according to the different types of visceral artery involved and are schematically reassumed in Fig. 14.2 [15].

BOX Signs of visceral artery aneurysms:

Clinical: most of the times asymptomatic, unless nearby compression or rupture.
Diagnostic: contrast-filled outpouching in the course of the artery.

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Acute Abdomen and Acute Abdominal Conditions

15

Emanuele Botteri, Gianmaria Casoni Pattacini, Alessio Giordano, and Francesca Ratti

15.1 Acute Calculus Cholecystitis

15.1.1 Introduction

The estimated overall prevalence of gallstones is 10–15% in the general population, with some differences across countries. Between 20 and 40% of patients with gallstones will develop gallstone-related complications, with an incidence of 1–3% annually; acute calculus cholecystitis (ACC) is the first clinical presentation in 10–15% of the cases [1, 2]. In 95% of cases ACC is caused by gallstones, while in the remaining 5% of cases it can be associated with cardiovascular disorders, following trauma or severe burns, following abdominal or cardiac surgery, in prolonged fasting typical of critically ill patients, in severe immunodeficiencies, in elderly and diabetic patients [3].

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15.1.2 Diagnostic Criteria for ACC

The useful features for the diagnosis of ACC are:

- History and clinical examination: fever, right upper quadrant pain or tenderness, vomiting or food intolerance; Murphy's sign
- Laboratory tests: elevated C-reactive protein, elevated white blood cell count
- Imaging: signs suggestive of gallbladder inflammation

The Tokyo Guidelines 2013 and 2018 (TG13/TG18) [4, 5] defined an algorithm that allows to better define a suspected or certain diagnosis of acute cholecystitis following 3 diagnostic criteria (Table 15.1).

15.1.3 Which Initial Imaging Technique Should Be Used in Case of a Suspected Diagnosis of ACC?

The generally accepted imaging findings of acute cholecystitis are thickening of the gallbladder wall (≥4 mm), enlargement of the gallbladder (long axis ≥8 cm, short axis ≥4 cm), gallstones or retained debris, fluid accumulation around the gallbladder, and linear shadows in the fatty tissue around the gallbladder [6]. Abdominal US should be the first method of diagnostic imaging used for acute cholecystitis. However, as a causative stone in the gallbladder or bile duct may not always be clearly identifiable on abdominal US and the diagnosis of gangrenous cholecystitis may be difficult [7], it is also recommended that contrast-enhanced CT or MRI be performed if required [8].

15.1.4 Stratification of the Risk and Severity of ACC

The TG13 confirmed by TG18 made it possible to classify ACC according to 3 degrees of severity (mild, moderate, and severe) taking into consideration specific biohumoral and clinical parameters (Table 15.2).

Table 15.1 TG18/TG13 diagnostic criteria for acute cholecystitis [4, 5]

A. **Local signs of inflammation, etc.**
(1) Murphy's sign, (2) RUQ mass/pain/tenderness
B. **Systemic signs of inflammation, etc.**
(1) Fever, (2) elevated CRP, (3) elevated WBC count
C. **Imaging findings**
Imaging findings characteristic of acute cholecystitis
Suspected diagnosis: one item in A + one item in B
Definite diagnosis: one item in A + one item in B + C

Table 15.2 TG ACC severity grading [9]

Grade I Mild	Grade II Moderate	Grade III Severe
Patient with no signs of organ dysfunction and with mild inflammation of the gallbladder	Associated with one of the following conditions: 1. WBC >18,000/mm^3 2. Palpable gallbladder in right hypochondrium 3. Duration of symptoms >72 h 4. Marked local inflammation (gangrenous cholecystitis, liver abscess, biliary peritonitis, cholecystitis emphysematous)	Associated with dysfunction of one of the following bodies: 1. Cardiovascular dysfunction: hypotension that requires dopamine ≥5 μg/kg/min or other vasoactive amines 2. Neurological dysfunction: decrease in the level of consciousness 3. Respiratory dysfunction: PaO_2/FiO_2<300 4. Renal dysfunction: Oliguria, Creatinine >2.0 mg / dl 5. Hepatic dysfunction: PT-INR >1.5 6. Blood dysfunctions: PLT <100,000/mm^3

15.1.5 When ACC Was Associated Common Bile Duct Stones: Which Tools to Use for Suspicion and Diagnosis at Presentation?

Choledocholithiasis, i.e., the presence of common bile duct stones (CBDS), is reported to occur in 10% to 20% of gallstone cases, with lower incidence, ranging from 5 to 15%, in case of ACC [10]. In order to assess the risk for CBDS, WSES guidelines [11] suggest to perform liver function tests (LFTs), including ALT, AST, bilirubin, ALP, GGT, and abdominal US in all patients with ACC. The visualization of a stone in the common bile duct at transabdominal US was a predictor of CBDS in patients with ACC while an increased diameter of common bile duct was an indirect sign of stone presence but was not sufficient to identify ACC patients with CBDS. The American Society of Gastrointestinal Endoscopy and the Society of American of Gastrointestinal Endoscopic Surgeons combined the published validated clinical scores and proposed a risk stratification of CBDS in three different classes, defined as follows: low risk (<10%), moderate risk (10–50%), and high risk (>50%) of CBDS [12] (Tables 15.3 and 15.4). This proposed classification has clear clinical implications: patients with a low risk of CBDS should be operated without further investigation; patients with moderate risk should be evaluated with a second-level examination, either preoperatively with endoscopic US (EUS) or magnetic resonance cholangiopancreatography (MRCP) or intraoperatively with laparoscopic US

Table 15.3 Predictive CBDS factors [12]

Very strong factors	Strong factors	Moderate factors
Evidence of stones in CBDS during ultrasound of the abdomen	• Common bile duct diameter> 6 mm • Total bilirubin >4 mg/dl • Direct bilirubin >1.8–4 mg/dl	• Abnormal liver tests • Age >55 years • Clinical pancreatitis

Table 15.4 Risk classes for CBDS [12]

High risk	Moderate risk	Low risk
Very strong factors presence	Strong and moderate factors presence	No predictive factors

(LUS) or intraoperative cholangiography (IOC), in order to select patients who need stone removal; finally, according to local expertise, laparoscopic transcystic CBD exploration is a valuable option. Patients with high risk of CBDS should undergo pre- or intra- or postoperative endoscopic retrograde cholangiopancreatography (ERCP) depending on local expertise and availability.

CBDS could be removed with several techniques and a variation of timing: preoperative ERCP with sphincterotomy, intraoperative ERCP with sphincterotomy, laparoscopic or open common bile duct exploration, postoperative ERCP with sphincterotomy. A systematic review assessed the differences between these techniques [13]. No differences in terms of morbidity, mortality, and success rate were reported. Therefore, these techniques can be considered suitable options, depending on local expertise and availability.

15.1.6 Surgical Treatment of ACC

The laparoscopic cholecystectomy was recommended as the first-line treatment for patients with ACC [6, 7]. The laparoscopic approach should always be attempted except in cases of absolute anesthetic contraindications or severe hemodynamic instability.

Laparoscopic cholecystectomy in acute cholecystitis was associated with a reduction in mortality rates, infectious complications of the surgical site and pneumonia and a clear reduction in the average postoperative hospital stay, when compared to the traditional open approach [14–16]. The subtotal cholecystectomy is an option when the critical view of safety cannot be obtained. It is preferable to perform a cholecystectomy as soon as possible, after patient admission, preferably within 72 h of the onset of symptoms [4, 5] or in any case within 7 days from hospital admission and within 10 days from the onset of symptoms.

15.1.7 Alternative Treatment for Patients with ACC: Observation and Techniques for Gallbladder Drainage

A RCT with long-term follow-up of 14 years showed that about 30% of patients treated conservatively developed recurrent gallstone-related complications and 60% of patients had undergone cholecystectomy subsequently [17]. TG13 on ACC [6, 7] considered percutaneous transhepatic gallbladder drainage (PTGBD) as mandatory for patients with severe-grade ACC and also suggested its use in the moderate grade if conservative treatment fails. The revised TG18, based on recent studies, proposed that severe-grade cholecystitis, under certain strict criteria, may be treated with laparoscopic cholecystectomy [5, 18]. Gallbladder drainage decompresses the infected bile or pus in the gallbladder, removing the infected collection without removing the gallbladder. Gallbladder drainage may be an option in patients who failed conservative management after a variable time of 24–48 h and who present with strict contraindications for surgery.

15.1.8 Antibiotic Therapy

Biliary penetration of different antibiotics (indicated as the ratio of bile-to-serum concentrations) are listed in Table 15.5 [19], suggested by 2020 WSES update guidelines.

Table 15.5 Antibiotics biliary penetration

Good penetration efficiency antibiotics Bile/Serum (≥5)	Low penetration efficiency antibiotics Bile/Serum (<5)
Piperacillin/Tazobactam	Cefotaxime
Tigecycline	Meropenem
Ciprofloxacin	Ceftazidime
Ampicillin/Sulbactam	Vancomycin
Ceftriaxone	Amikacin
Levofloxacin	Gentamicin
Penicillin G	Cefepime
Amoxicillin/Clavulanate	Imipenem

15.2 Acute Colonic Diverticulitis

15.2.1 Introduction

Acute left-sided colonic diverticulosis is common in Western countries with its prevalence increasing throughout the world, which is likely due to changes in lifestyle [20]. Although left-sided colonic diverticulosis remains more common among elderly patients, a dramatic rise of its incidence has been seen in younger age groups in recent years [21]. Recent evidence suggests that lifetime risk of developing acute left-sided colonic diverticulitis (ALCD) is about 4% among patients with diverticulosis [22].

15.2.2 Which Classification Should Be Used in Patients with ALCD?

A proposal for a CT-guided classification of ALCD was published in 2015 by the WSES acute diverticulitis working group [23] and confirmed by recent update of this guideline [24]. It may guide clinicians in the management of acute diverticulitis and may be universally accepted for day-to-day practice (Table 15.6).

15.2.3 The Diagnosis of ALCD

In patients with suspected ALCD, a complete assessment of the patients using clinical history, signs, laboratorial inflammation markers, and radiological findings was performed. Clinical findings of patients having ALCD include acute pain or tenderness in the left lower quadrant that may be associated with increased inflammatory markers, including C-reactive protein (CRP) and white blood cell count (WBC). CRP has been identified as a useful biomarker of inflammation, and it may be useful in the prediction of the clinical severity of acute diverticulitis as demonstrated by several recent studies [25]. CT is the gold standard for both the diagnosis and the staging of patients with ALCD due to its excellent sensitivity and specificity [26].

Table 15.6 WSES acute diverticulitis classification [24]

Uncomplicated acute diverticulitis	Complicated acute diverticulitis
Stage 0: Diverticula, thickening of the wall, increased density of the pericolic fat	• **Stage 1a:** Pericolic air bubbles or small amount of pericolic fluid without abscess (within 5 cm from inflammed bowel segment) • **Stage 1b:** Abscess ≤4 cm
	• **Stage 2a**: Abscess >4 cm • **Stage 2b:** Distant gas (>5 cm from inflammed bowel segment)
	Stage 3: Diffuse fluid without distant free gas
	Stage 4: Diffuse fluid with distant free gas

CT scan can also rule out other diagnoses such as ovarian pathology, or leaking aortic or iliac aneurysm. CT findings in patients with ALCD may include diverticulosis with associated colon wall thickening, fat stranding, phlegmon, extraluminal gas, abscess formation, or intra-abdominal free fluid CT criteria may also be used to determine the grade of severity and may drive treatment planning of patients [27]. US is a real-time dynamic examination with wide availability and easy accessibility [28]. Its limitations include operator dependency, poor assessment in obese patients, and difficulty in the detection of free gas and deeply located abscesses [29].

15.2.4 The Nonoperative Treatment

15.2.4.1 Stage 0: Uncomplicated Acute Diverticulitis

Uncomplicated acute diverticulitis is defined as localized diverticular inflammation without any abscess or perforation. In recent years, several studies demonstrated that antimicrobial treatment was not superior to withholding antibiotic therapy, in terms of clinical resolution, in patients with mild unperforated diverticulitis [30]. The current consensus is that uncomplicated acute diverticulitis may be a self-limiting condition in which local host defenses can manage the inflammation without antibiotics in immunocompetent patients. In this context, antibiotics are not necessary in the treatment of uncomplicated disease [24]. If antibiotic therapy is necessary, oral administration of antibiotics may be equally as effective as intravenous administration. An expeditious switch from intravenous to oral may allow a rapid patient discharge [31]. Patients with uncomplicated acute diverticulitis symptoms without significant comorbidities, who are able to take fluids orally and manage themselves at home, can be treated as outpatients. They should be re-evaluated within 7 days from the time of the diagnosis. However, if the clinical condition deteriorates, re-evaluation should be carried out earlier. Patients with significant comorbidities and unable to take fluids orally should be treated in hospital with intravenous fluids.

15.2.4.2 Stage 1 and 2: Locally Complicated Acute Diverticulitis

Approximately 15–20% of patients admitted with acute diverticulitis have an abscess on CT scan [32]. In presence of pericolic air bubbles, small amount of pericolic fluid without abscess (within 5 cm from inflammed bowel segment) (Stage 1a) and in presence of abscess (Stage 1b or 2a) the treatment required was always antibiotic therapy. If the abscess is limited in size (Stage 1b), systemic antibiotic therapy alone is considered safe and effective in removing the abscess and solving acute inflammation with a pooled failure rate of 20% and a mortality rate of 0.6% [33]. The size of 4–5 cm may be a reasonable limit between antibiotic treatment alone, versus percutaneous drainage combined with antibiotic treatment in the management of diverticular abscesses [34]. A high suspicion for surgical control of the septic source should be maintained and a surgical treatment should be performed if the patient shows a worsening of inflammatory signs or the abscess does not reduce with medical therapy.

15.2.4.3 Stage 2b: The Role of Nonoperative Treatment

Although most patients hospitalized for acute diverticulitis can be managed by nonoperative treatment, up to 25% may require urgent operative intervention [35]. Highly selected group of patients at this stage may be treated by conservative treatment. However, it may be associated with a significant failure rate (57–60%) and a careful clinical and CT monitoring is mandatory [36]. Moreover, nearly 60% patients with distant intraperitoneal gas were primarily treated by surgery.

15.2.5 Operative Treatment

15.2.5.1 Stage 3 and 4

The recent update WSES guidelines [24] recommend Hartmann's Procedure (HP) for managing diffuse peritonitis in critically ill patients and in selected patients with multiple comorbidities. Whereas in clinically stable patients with no comorbidities suggest primary resection with anastomosis with or without a diverting stoma. The same authors suggest to perform an emergency laparoscopic sigmoidectomy only if technical skills and equipment are available. In fact, laparoscopic sigmoidectomy for diverticulitis had initially been confined to the elective setting. A damage control surgical strategy may be useful for patients in physiological extremis from abdominal sepsis [37]. The initial surgery focuses on control of the sepsis, and a subsequent operation deals with the anatomical restoration of the gastrointestinal tract, after a period of physiological resuscitation. Laparoscopic lavage and drainage can potentially avoid a stoma in patients with diffuse peritonitis. It consists of the laparoscopic aspiration of pus followed by abdominal lavage and the placement of abdominal drains, which remain for many days after the procedure. However, it cannot be considered the first-line treatment in patients with diverticular peritonitis, as confirmed by the last WSES guidelines [24].

15.2.6 The Planning Elective Resection in Cases of Acute Diverticulitis Treated Nonoperatively

Currently, the decision to perform an elective resection after one or more episodes of AD should be undertaken on a case-by-case basis, considering risk factors, complications, age, and severity of episodes as well as the patient's personal circumstances and comorbidities (e.g., immunosuppressed patients). In particular the WSES suggest planning of an elective sigmoid resection after a single episode of ALCD treated conservatively in high-risk patients, such as immunocompromised patients [24].

15.2.7 Antibiotic Therapy

The most common organisms that cause diverticulitis are *E. coli*, *K. pneumoniae*, and *B. fragilis*; thus, empiric therapy should at a minimum cover these organisms. Empiric therapy should also be guided by the severity of illness of the patient and

the extent of disease. Antibiotic therapy plays an important role in the management of complicated acute diverticulitis. Typically, it is an empiric antibiotic treatment. The regimen should depend on the severity of infection, the pathogens presumed to be involved, and the risk factors indicative of major resistance patterns. Patients who have signs of sepsis beyond 5 to 7 days of adequate antibiotic treatment warrant aggressive diagnostic investigation in search of a reservoir of infection. For patients with complicated diverticulitis with an abscess, fistula, obstruction, or perforation, four therapy with cefazolin, cefuroxime, or ceftriaxone, all plus metronidazole or ampicillin/sulbactam alone or ertapenem can be used. For patients with complicated diverticulitis associated with sepsis, consider broader coverage for Enterobacteriaceae and Pseudomonas with piperacillin/tazobactam or cefepime plus metronidazole. For patients with severe penicillin allergies, consider fluoroquinolone or aztreonam-based regimens, making sure to include anaerobic coverage unless moxifloxacin is used. Moxifloxacin has anaerobic activity; thus, addition of metronidazole is not needed when using this agent. Vancomycin should be added to ciprofloxacin and aztreonam in patients presenting with sepsis as these agents do not have Gram-positive activity to cover streptococci or enterococci.

15.3 Sigmoid Volvulus

15.3.1 Introduction

The term "volvulus" identifies the torsion of a segment of the gastrointestinal tract (from Latin "volvēre," meaning "to roll or twist"). The incidence of SV varies worldwide. High incidence has been reported in regions such as Latin America, Africa, Eastern Europe, Scandinavia, Russia, Middle East, Pakistan, and India, where this condition has been defined as endemic [38]. In these regions, sigmoid volvulus accounts for 20 to 54% of intestinal obstructions as opposed to low-incidence areas such as North America, Western Europe, and Australia, where SV accounts for 3–5% of intestinal obstructions [39].

15.3.2 Diagnosis

Common investigations include abdominal X-ray, contrast enema, or a CT scan, while endoscopy serves as a diagnostic and therapeutic technique and will be discussed in detail later in this chapter. Diagnostic accuracy of abdominal X-ray ranges from 57 to 90% [40]. Classic radiological findings include a markedly distended ahaustral sigmoid loop, that appears as a "coffee bean" (also known as "bent inner tube sign") with its apex projected under the left hemidiaphragm, which has a high specificity, although it is not always present [41]. Other highly specific radiological signs are the convergence of three radiopaque lines to the left inferior quadrant (Frimann-Dahl sign), and the identification of the sigmoid colon above the transverse colon. CT scan, albeit seldom required for diagnosis, has a high accuracy in detecting SV [42], with a sensitivity of 100% and a specificity >90%, showing a

whirled sigmoid pedicle, and is currently the preferred diagnostic study modality due to its rapidity and availability. Moreover, it can detect intestinal pneumatosis that appears as air bubbles within the bowel wall as a sign of sigmoid ischemia [43]. Endoscopic findings include a mucosal twisting with obstruction at the rectosigmoid junction.

15.3.3 Management

15.3.3.1 Nonoperative Treatment

Recently, the American Society for Gastrointestinal Endoscopy (ASGE) produced practice guidelines on the role of endoscopy in the management of colonic volvulus [44].

Patients with signs of complicated SV (peritonitis or perforation), recurrent volvulus, or unsuccessful nonoperative management are not eligible for endoscopic decompression and should be immediately referred for surgical management [45].

For patients who are eligible for nonoperative management, endoscopic detention represents the procedure of choice for SV decompression [46].

Endoscopic reduction is successful in 40–90% of cases [47, 48]. After successful detorsion of SV, elective surgical treatment should be strongly considered during the index admission if the patient can tolerate it after adequate preparation, since recurrence rates as high as 86% have been reported [48] and emergency surgery is associated with a considerable mortality risk.

15.3.4 Operative Treatment

15.3.4.1 Urgent Setting

Two possible scenarios represent an absolute indication for urgent surgical management: endoscopic management failure or the impossibility to perform it; suspicion or evidence of colonic ischemia, perforation or clinical findings suggestive of peritonitis and septic shock [46].

Surgical resection of the involved colonic segment is generally recommended, since simple detorsion carries a high mortality and recurrence risk [46].

The decision to perform a Hartmann's procedure, or a primary colorectal anastomosis with or without proximal diversion, is currently controversial.

Currently available data does not support the use of one technique over the other, and the decision on which approach to adopt highly depends on the surgeon's judgement. Nonetheless, several factors should be taken into account when deciding to perform an anastomosis, including patient's hemodynamic status, septic status, comorbidities, medications, functional status, intraoperative findings, and tissues viability.

15.3.4.2 Elective Setting

If nonoperative management is successful and the patient's surgical risk is reasonable, current consensus is that surgical intervention should be performed within 2 days of SV reduction or within the index admission [46].

Among the number of procedures that have been proposed in this setting (including Hartmann's resection, resection and anastomosis, detorsion alone, detorsion and colopexy and percutaneous colostomy), resection of the redundant sigmoid colon with primary anastomosis seems to have the best results in terms of preventing recurrence [45, 46].

15.4 Acute Appendicitis

15.4.1 Introduction

In young people, acute appendicitis (AA) is one of the most common cause of surgical acute abdomen. Lifetime risk of AA is 8.6% for male and 6.9% for women [49]. The peak of incidence is between 10 and 20 years old. Every year about 50,000 of appendectomies are performed in the UK and 300,000 in the USA. In our country the annual incidence of AA is 0.2%. The mortality for AA is 0.3% for uncomplicated conditions, but this percentage rises up to 1.7% in the complicated forms. AA can hide the presence of malignancies and often the onset symptoms are in 50% of appendiceal neoplasms, in 40% of cecum colon cancers and in 15% of whole colon cancers AA represents the onset condition [50].

15.4.2 Classification

There are several classifications for AA, but the most recent is proposed by Gomes et al. [51] and it is based on intraoperative findings:

- *Non-complicated Acute Appendicitis:*
  - Grade 0: Normal Looking Appendix (Endoappendicitis – Periappendicitis)
  - Grade 1: Inflamed Appendix (Hyperemia, edema with or without fibrin, without or little pericolic fluid)
- *Complicated Acute Appendicitis:*
  - Grade 2: Necrosis A Segmental (without or little pericolic fluid)
    B Base Necrosis (without or little pericolic fluid)
  - Grade 3: Inflammatory tumor A Phlegmon
    B Abscess <5 cm
    C Abscess >5 cm without peritoneal free air
  - Grade 4: Perforated diffuse peritonitis with or without peritoneal free air

15.4.3 Diagnosis and Indication

Symptoms and Sign [52]*:*

- Right Iliac Fossa (RIF) pain 91.2% (looking for: *Rovsing's sign, Psoas' sign, Obturator sign*)
- RIF tenderness or RIF rebound tenderness 69.9%
- Diffuse rebound tenderness or muscular defense 11.7%
- Vomiting 42%
- Fever 24.7%
- White Blood Cell (WBC) count >10,000 GB 24.7%
- CRP >10 mg/l 46.1%

15.4.4 Diagnostic Scores (Tables 15.7 and 15.8)

15.4.5 Therapy

The "gold standard" for the treatment of AA is appendectomy. If experienced team and equipment are present, laparoscopic appendectomy (LA) should represent the first choice since it offers advantages in terms of less pain, reduced LOS, lower occurrence of surgical site infection (SSI), earlier return to work, and overall costs. LA shows clear benefit in the treatment of obese or elderly patients and those with comorbidities. Several data from literature found LA more beneficial and cost-effective than open surgery also for complicated AA [52]. Nonoperative management with antibiotic therapy can be successful in selected patients in order to avoid surgery. This approach must be proposed only to patients with low risk of complicated AA, often at first admission for RIF pain. The patients should receive initial intravenous antibiotics with subsequent conversion to oral treatment. The clinical monitoring should continue until the symptoms disappear. The risk of recurrence is up to 38%.

Table 15.7 Alvarado Score [53]

Alvarado score	
Feature	Score
Migration of pain	1
Anorexia	1
Nausea	1
Tenderness in right lower quadrant	2
Rebound pain	1
Elevated temperature	1
Leukocytosis	2
Shift of white blood cell count to the left	1
Total	10

<5 Sensibility, exclusion of AA 99% (IC 95%, 97–99%)

Table 15.8 Appendicitis Inflammatory Response (AIR) [54]

Variables	Score
Vomiting	1
Right iliac fossa pain	1
White blood cell count	
• 10.000–14.000 /dl	1
• ≥15.000/dl	2
CRP	
• 10–49 g/L	1
• ≥50 g/L	2
Polymorphonuclear leukocytes	
• 70–84%	1
• ≥85%	2
Pyrexal (>38.5 °C)	1
Rebound tenderness or guarding	
• Light	1
• Medium	2
• Strong	3

<4 Sensibility, exclusion of AA 96% >8 specificity, diagnosis of AA 99%

15.4.6 Tips and Tricks for Laparoscopic Appendectomy

15.4.6.1 Patients Position

Supine, general anesthesia.

First operator on the left side.

15.4.6.2 Trocars Position

- Hasson's trocar umbilical
- 5 mm Left iliac fossa
- 5 mm–10 mm suprapubic

15.4.6.3 Diagnostic Laparoscopy

- Abdominal fluid (ascites, purulent, fecaloid)
- Gallbladder disease
- Gynecological disease
- Meckel diverticulum
- Other?

15.4.6.4 Mesoappendix Dissection

Monopolar electrocoagulation and bipolar energy are the most cost-effective techniques.

High energy devices can be used without clear advantages.

15.4.6.5 Stapler vs Endoloop for Stump Closure

The choice should be individualized on the basis of cecum and appendicular stump condition. If it were possible to use both, endoloops might be preferred for lowering the costs but operative time maybe longer.

15.4.6.6 Vesical Catheter

It should be placed after general anesthesia and remove before the awakening. Its role is to protect the bladder during the insertion of suprapubic trocar.

15.4.6.7 Drainage

The choice should be individualized based on intraoperative findings.

15.4.6.8 SILA (Single Incision Laparoscopic Appendectomy)

Only for cosmetic intent in center with adequate experience.

Inclusion criteria for SILA:

- Normal weight female
- Uncomplicated appendicitis
- Without risk factor for incisional hernia (diabetes, smoke, umbilical hernia, midline diastasis)

15.5 Acute Presentation of Abdominal Wall Disease

15.5.1 Epidemiology and Clinical Presentation

The wall abdominal diseases are traditionally divided into three categories:

- Primitive: arising from a weakness of abdominal wall
- Recurrent: occurring after surgery for a primitive hernia
- Incisional hernia: occurring along the course of a surgical scar

Lifetime risk of groin hernia occurrence is 27–43% for male and 3–6% for female [55]. The reasons that lead a patient to emergency ward are:

- Swelling and pain localized in the hernia area
- Irreducibility
- Irreducibility with pain (suspicion of incarceration)
- Occlusion

15.5.2 Diagnosis

15.5.2.1 Blood Exam

- Complete blood count: leukocytosis
- Lactate and CRP: high level

- Hepatic function
- Kidney function
- Coagulation

15.5.2.2 Radiological Exams

- US in the hernia area and abdominal
- Abdominal and chest RX
- Enhanced CT scan with contrast

If surgeon is involved in urgent consultation for abdominal wall disease, there will be three ways he can take:

- Complete resolution of the problem and refer the patient to elective surgery. The reduction maneuverers are successful. The pain is restored and the laboratory test or radiological finding is normal.
- Resolution of the problem and refer the patient to deferred urgency. The reduction maneuverers are successful but the pain is still present. Probably the patients have had other similar episodes in their life. The laboratory test or radiological finding is not normal and may show inflammation pattern or abdominal effusion.
- Failure of conservative approach and emergency surgery. The reduction maneuverers are not successful. This approach must be chosen in the presence of a high suspect of bowl ischemia or confirmed by CT scan.

15.5.3 Surgery for Hernias

The steps during emergency surgery for hernia are:

- Isolation of the hernia content
- Check of the viability of the hernia content with possible resection (bowel or omentum)
- Reduction of the hernia content
- Defect closure (with or without mesh repair)

15.5.4 Surgery for Urgent Groin Hernia

In surgical ward, the first attempt worth making is manual reduction.

This procedure could cause excruciating pain then the aid of a mild sedation can increase the success rate. In the absence of intestinal ischemic suffering, the treatment of choice is anterior approach with mesh repair. The viability of the content can be evaluated intraoperatively through a small incision in the peritoneal sac. This maneuver allows the collection of peritoneal fluid or the resection of a necrotic tract of omentum. If the effusion is purulent or fecal and if there is high suspect of bowel involvement, the abdominal cavity must be controlled by laparoscopy or laparotomy. The main contraindications to diagnostic laparoscopy are related to the

patient's hemodynamic status, the bowel distension, and the low laparoscopic experience of the surgical team. In case of clear fecal contamination, the mesh repair must be avoided.

15.5.5 Incisional and Other Midline Hernias

The urgent treatment of midline hernias including umbilical ones rely on the same advices of groin hernias. The isolation of hernia content evaluating the viability, its liberation from the crawler followed by the reduction in abdominal cavity and the defect repair are the main phases of the surgery. The incision can be used for the diagnostic laparotomy with the possibility of intestinal resection or other required procedure.

Incisional hernia can be challenge due to the scar of previous surgery. The fascial weakness almost always requires prosthetic repair. Incisional hernia often has a visceral involvement and, in urgent setting, the risk of visceral damage is high. Moreover, at the end of the reduction of the content it is impossible to proceed with a direct closure of the defect. In these cases, it is indicated the use of a biologic mesh to restore the abdominal wall.

15.6 Anorectal Emergencies

15.6.1 Introduction

The term "anorectal emergencies" refers to anorectal disorders presenting with some alarming symptoms such as acute anal pain and bleeding that might require an immediate management. They are represented by: thrombosed external hemorrhoids, thrombosed or strangulated internal hemorrhoids, anorectal abscess, rectal prolapse, anal fissure, rectal bleeding. Diagnosis of any of anorectal emergencies must include a physical examination that should include inspection of perianal tissues, anorectal digital examination, and anoscopy when available based on the patient's symptoms and pain [56].

15.6.2 Acute Thrombosed External Hemorrhoids

Classic symptoms of presentation are acute anal pain with a newly enlarged or tender blush lump at the anal verge. Some patients can describe a recent history of constipation or prolonged straining.

The management of this acute condition can be conservative or surgical depending on patient's symptoms.

The conservative treatment includes anti-inflammatory analgesics, phlebotonics, warm size bath and drugs avoiding constipation. The excision of thrombosed external hemorrhoid with surgical removal of the clot is reserved in patients with severe pain within 48–72 h from the onset of symptoms [57].

15.6.3 Thrombosed Internal Hemorrhoids

Internal hemorrhoid may become strangulated and thrombosed when prolapsed part is left protruded until vascular compromise. The management consists in manual reduction of the masses to relieve patient's pain then an urgent hemorrhoidectomy [58].

15.6.4 Rectal Bleeding

Rectal bleeding is a symptom that can represent different types of pathology of gastrointestinal tract like hemorrhoids, anal fissure, IBD, and rectal neoplasm. The patient history and the physical examination are necessary to differentiate the possible reason of the bleeding. The blood may be spotted on the toilet paper or could be on the toilette. Usually it is characterized by a painless passage of bright-red blood during bowel movements. In patients with rectal bleeding, an anoscopy and colonoscopy are mandatory to rule out the pathology that cause the bleeding [59].

15.6.5 Anal Fissure

Painful defecation with or without passage of red blood is a typical symptom of this condition. The patient can describe that the pain may last from minutes to hours after defecation. Patients are basically pain-free between bowel movement. During the anoscopy is visible a small linear laceration of the anoderm (acute anal fissure). For those who experience a long history of painful defecation a chronic linear laceration of the anoderm is visible, with hypertrophic anal papilla and enlarged perianal skin tag (chronic anal fissure). For acute anal fissure a conservative management is the treatment of choice that includes adequate pain control, stool softeners, topical nitrate and topical calcium channel blocker. For patient with chronic anal fissure, a botulin injection or a lateral anal sphincterotomy remains the treatment of choice [60].

15.6.6 Anorectal Abscess

The anorectal abscess is one of the most frequent anorectal emergencies in the ED. They usually originate from an infected anal gland located in the anal mucosa. The abscess can reach the inter-sphincteric area, supra-elevator space, perianal region, deep post-anal space, and ischiorectal fossa. Most of the abscess can be diagnosed with a careful history and physical examination that must include anorectal digital examination. Patient usually refer anal pain, fever, and the presence of a tender mass. The fluctuation of the abscess can't be evident. An endoanal ultrasonography, a CT scan, or MRI of the pelvis may add some additional information on the extension and exact location of the abscess and help to make the correct decision

on the management. The gold standard treatment is an adequate drainage of the abscess that can be done in ED with some sedation or in operating room, depending on the exact location of the abscess and surgeon's experience. An expert surgeon on colorectal disease may provide a definitive treatment of anal fistula but this type of approach is still in debate [61].

15.6.7 Rectal Prolapse

Classical signs of rectal prolapse are protruding full-thickness rectal wall with concentric rings of mucosa. It is important to differentiate between rectal prolapse or prolapsed internal hemorrhoid because of the different types of management needed.

Clinical and physical examination of anal region are mandatory. An irreducible rectal prolapse is quite rare but can happen.

For strangulated irreducible rectal prolapse an emergent surgery with rectosigmoidectomy is the treatment of choice. For all those rectal prolapse that can de reduce a conservative treatment and elective surgery can be scheduled [62].

15.6.8 Bowel Obstruction

15.6.8.1 Introduction

A complete history, laboratory tests, and physical examination must be done for all patients attending to the emergency department with abdominal pain. It is very important to know about when the abdominal pain has begun, the type of pain and when was the last pass of gas/defecation. A history of previous abdominal surgery or episode of obstruction or presence of diverticula or rectal bleeding are important news to know in order to think about the possibility of a bowel obstruction. The main cause of bowel obstruction can be differentiated between small bowel obstruction and large bowel obstruction. For small bowel obstruction the main causes are adhesions and hernias, the remaining are malignancies, carcinomatosis, endometriosis, IBD, foreign bodies, and bezoars. For large bowel obstruction, the main causes are malignancies, diverticular stenosis, and volvulus (Fig. 15.1).

15.6.9 Diagnosis

The initial radiological examination on the guide of the clinical presentation is represented by standard abdominal X-ray and ultrasound. On the basis of this exam, a CT scan can be performed to better understand the level of the obstruction. Supportive treatment with hydration, anti-emetics, and bowel rest must be done. A nasogastric suction is useful for initial diagnosis and treatment. A CV must be insert. Low arterial blood pH and high lactic acid level may be useful in the diagnosis of intestinal ischemia. In case of small bowel obstruction for adhesions, a water-soluble contrast administration can be performed. It is a feasible NOM with low

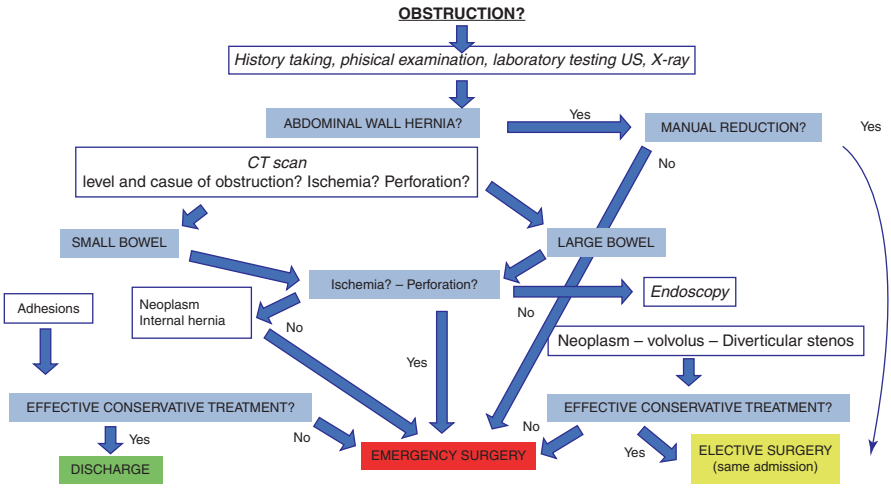

Fig. 15.1 Management strategy of bowel obstruction (for about 90% of causes)

morbidity and mortality. Colonoscopy is limited to the large bowel obstruction. To minimize the burden of ionizing radiation in children and pregnant women, magnetic resonance imaging is a valid alternative examination to computed tomography scan for bowel obstruction [63].

15.6.10 Therapy

15.6.10.1 Conservative Treatment

NOM is safe and useful for all small bowel obstruction caused by adhesions. Water soluble administration is useful. Evidence are lacking but for many authors 72 h of duration is safe and appropriate. In case of hernia a manual reduction has to be attempted. In case of unsuccessful reduction emergency surgery is needed. Diverticular obstruction can be solved with NOM. Sigmoid volvulus may benefit of endoscopic detorsion. In case of colonic necrosis, immediate surgery is needed. In case of left colon cancer obstruction, a self-expanding stent as bridge to surgery in centers with adequate expertise must be preferred to a diverting stoma [64].

15.6.10.2 Surgery

For abdominal wall complicated hernia, surgery is the treatment of choice. A prosthetic repair is mandatory. Diagnostic laparoscopy is useful to assess the bowel viability after reduction. Adhesiolysis for small bowel obstruction can be performed laparoscopically or by open surgery. In case of small bowel tumors, resection and anastomosis following oncological principles must be done. For large bowel obstruction, caused by a sigmoid volvulus, surgery is necessary in case of multiple episodes or ischemia and perforation. For large bowel tumors, surgery is needed when a "bridge to surgery" is not possible [60, 61].

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Abdominal Emergencies Requiring a Multidisciplinary Approach

16

Andrea Mazzari, Pasquina M. C. Tomaiuolo, Alessio Giordano, Roberto Luca Meniconi, and Alberto M. Settembrini

16.1 Aortoenteric Fistula Clinical Case

A 77-year-old Caucasian man consulted his general practitioner concerning a 2-week history of worsening weakness and was referred to the emergency department to detect an abdominal bruit. At arrival, the patient was pale, apyretic, eupneic, conscious, and oriented. Physical examination confirmed a mesogastric bruit associated with an abdominal pulsatile mass. Melanic stool was found on rectal examination. No history of previous surgery. At the blood tests, leukocytosis, mild anemia, and alteration of the renal function were revealed. An urgent esophagogastroduodenoscopy (EGD) was arranged, but after a sudden drop in the patient's blood pressure, and enhanced abdominal CT was obtained first. The examination revealed a 63 mm thick-walled infrarenal aortic aneurysm associated with distension of the small bowel loops by fluid content.

The patient was immediately transferred to the operating theatre, and a significant amount of fresh blood (400 mL) was aspirated through the nasogastric tube during anesthetic preparation. The laparotomy disclosed an aneurysm of inflammatory appearance, adherent to the fourth portion of the duodenum and surrounded by

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a small retroperitoneal hematoma. Following aortic and iliac cross-clamping, dissection of the aneurysmatic aortic wall from the duodenum was performed and a 1 cm aorto-duodenal fistula was found upon removal of a fresh clot. The peritoneal cavity was irrigated with rifampicin, the duodenal wall was sutured, and the aorta was repaired by silver-impregnated prosthetic interposition. The aneurysmatic wall was closed above the prosthesis and a pedicle of omentum was interposed between the aortic wall and the duodenum.

Blood cultures were negative for bacterial or fungal infection; therefore, a wide-spectrum antibiotic therapy with imipenem was administered for 30 days. On post-operative day 15, the patient was transferred to a rehabilitation center and discharged 2 weeks later.

16.1.1 Introduction

Aortoenteric fistula (AEF) is encountered in urgent diseases; it is a communication between the aorta and the bowel, initially described by Sir Astley Cooper in 1829 [1], caused by erosion between aortic and gastrointestinal wall. AEF can be primary or secondary.

Primary aortoenteric fistula (PAEF) is a rare condition that develops between the native aorta and gastrointestinal tract, usually in the presence of an abdominal aortic aneurysm (AAA).

Secondary aortoenteric fistula (SAEF) occurs if a previous aortic reconstruction has been performed, usually at the level of the anastomosis where the friction is high. The prevalence of PAEF is estimated to be between 0.04% and 0.07% based on autopsy studies [2, 3].

More frequent location of fistulas is the third portion of the duodenum, which is involved in more than 50% of cases [4].

Even though the diagnosis is challenging, prompt management is mandatory to avoid the patient's death: insidious episodes of gastrointestinal (GI) bleeding are frequently under-diagnosed until the occurrence of a massive hemorrhage. If left untreated, the overall mortality rate is almost 100% [4].

16.1.2 Pathogenesis

A PAEF occurs without prior aortic surgery, and aortic aneurysms are the most common etiology; however, other causes can be gastrointestinal malignancies, septic aortitis, tuberculosis, and radiation therapy.

SAEF is correlated in the most part with a graft infection, and its incidence is lower following endovascular aortic aneurysm repair (EVAR) but much higher after emergent open repair of ruptured AAA due to the increased likelihood of bacterial contamination of the graft and bowel trauma during hurried surgical dissection.

After *open repair* the infection can be the beginning of the contamination of the graft into the bowel or the mechanical erosion can cause the detachment of the proximal anastomosis with the development of a pseudoaneurysm or the direct

involvement of the third or fourth portion of the duodenum (which is extraperitoneal!): this explains why after a surgical AAA repair it is mandatory to cover with the residual AAA sac or greater omentum the graft and the anastomosis.

After *EVAR*, an AEF can be related to mechanical failure of the stent-graft: rupture or migration of the device in the aneurysmatic sac and constant pressurization of it.

The median interval from the original operation to the development of AEF is around 2–4 years [5].

16.1.3 Clinical Presentation

The classic triad of symptoms of AEF as GI bleeding, sepsis, and abdominal pain is rarely found. The clinical presentation has a broad spectrum of symptoms varying from malaise to sudden hematemesis or melena or chronic anemia associated with fever. GI bleeding is the most common initial symptom and occurs in nearly 70% of patients; other symptoms reported are back pain and fever; sometimes a minor bleeding, usually self-limiting (Herald sign), can precede a massive gastrointestinal bleeding [6].

16.1.3.1 Diagnosis

In the Emergency Room, a patient, after collection of medical history (if possible to know previous aortic surgery), should be subjected to angio-CT that can reveal communication between the intestine and aorta or air bubbles around the aorta peculiar impregnation of the bowel (Fig. 16.1).

Extravasation of contrast in intestinal lumen is rare but pathognomonic of aortoenteric fistula. Other not specific signs include effacement of periaortic fat, thickening of bowel wall close to the aorta.

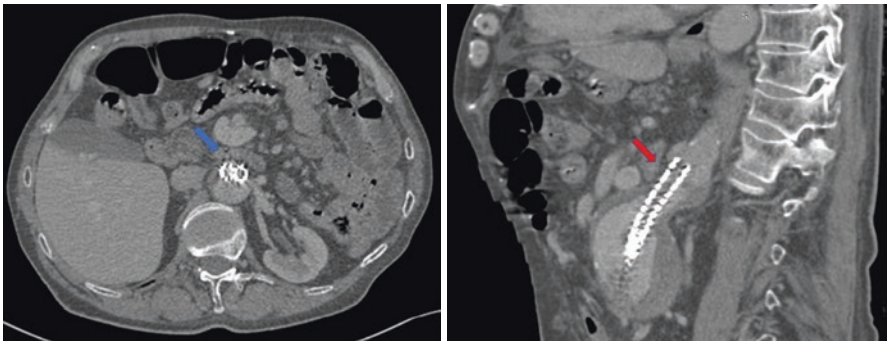

Fig. 16.1 CT axial (blue arrow) and sagittal (red arrow) view of air bubbles into aortic endograft after endovascular aneurysm repair. It is a CT sign of contamination between GI tract and aortic aneurysm

However, signs of suspected graft infection can be signs of AEF: perigraft gas collections, increase in perigraft soft tissue or fluid, pseudoaneurysm, disappearance of the continuous calcific aneurysm wrapping around the graft [7].

A second step test, if the patient is stable, is the esophagogastroduodenoscopy (EGD) that has low diagnostic accuracy: it could provide a more detailed look of duodenum (a trick is to use a longer endoscope) and visualize an AEF, sometimes with the underlying aortic graft material, and may also show active bleeding, as well as an adherent clot within the bowel. Only 25–50% of AEF are discovered by endoscopy due to the difficulty of visualizing the duodenum [8].

16.1.4 Decision-Making

The appropriate management of patients with massive bleeding due to aortoenteric fistula is still a significant challenge in the surgical setting. Timely recognition of hemorrhagic shock and subsequent treatment by evidence-based recommendations are crucial to patient outcome.

Treatment modalities include:

- Patient airway control, maintaining adequate ventilation and oxygenation, secure venous access with two large-bore cannulas.
- Avoid or correct hypothermia, acidosis, hypocalcemia (maintain Ca^{2+} >1.1 mmol/L).
- Crystalloid infusions (<3 L in the first 6 h).
- Systolic blood pressure of 80–90 mmHg achieved.
- Start massive infusion protocol as soon as possible.
- Obtain laboratory measures: full blood count, PT, fibrinogen, calcium, lactate, BE, pH.
- Tranexamic acid administrated and prothrombin complex in patients pre-treated with warfarin or NAO.
- Fibrinogen maintained at 1.5–2 g/dl.
- Platelets maintained at $>100 \times 10^9$/L.
- Target Hb level 7–9 g/dl achieved.

In unstable patients, decision-making must be rapid. Emergency surgical exploration in a patient with GI hemorrhage is the correct approach. The goal is to maintain the patient's life, so laparotomy should be the first approach, and then the surgeon has to take into account limb preservation.

Appropriate preparation of the patient provides to start broad-spectrum gram-positive and gram-negative antibiotic therapy.

16.1.5 Vascular Management

When a surgeon faces an AEF, he should keep in mind the hazards of going into infected fields, choosing the right abdominal approach, removing the graft, and revascularizing the limbs through two techniques: in situ or extra-anatomic revascularization.

When the surgical approach is decided, the surgeon should consider facing a hostile abdomen with a conglomerate of bowel loops that adheres to the retroperitoneum. The abdominal approach can be through a midline incision or a retroperitoneal incision.

For the in situ revascularization, autologous grafts (such as the saphenous vein, the superficial femoral vein, the endarterectomized superficial femoral artery) and biological conduits of arterial allografts or bovine pericardium grafts can be safely used in the infected fields; silver-impregnated or other impregnated grafts can be alternatively used but with higher re-infection risk (Fig. 16.2).

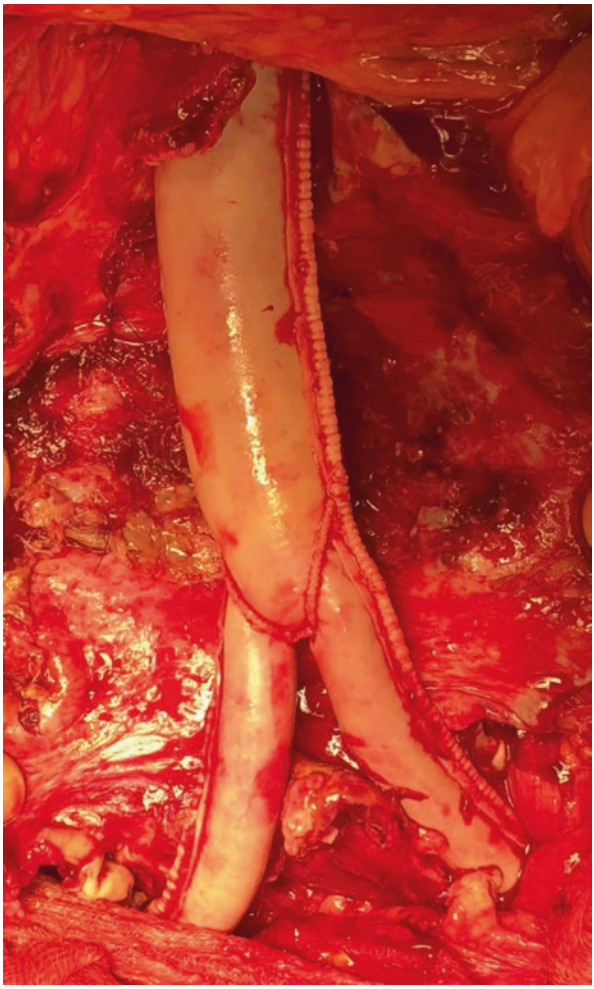

Fig. 16.2 In situ revascularization with biological graft in bovine pericardium after AEF removal

For extra-anatomic revascularization, prosthetic materials can be considered out of the infected field: in our opinion, silver-impregnated or antibiotics (rifampin or tobramycin) bonded grafts reduce the risk of graft infection. After aortic stump closure, extra-anatomic revascularization provides an axillo-femoral or bifemoral bypass or other less frequent revascularization techniques from the thoracic aorta to the limbs.

In case of hemodynamic instability, a staged approach with endovascular treatment of the bleeding by deploying an endograft in the aorta can be chosen. This is a bridge procedure before the total removal of infected grafts. A trick could be to use an infrarenal fixation endograft to reduce the challenge of suprarenal cross-clamping during its removal.

16.1.6 Bowel Reconstruction

The management of bowel defects depends on the site and the size of the fistula. Laparotomy is required with a midline or subcostal incision in aorto-duodenal fistula when bowel defect is less than 3–4 cm and the intestinal wall is not involved in massive inflammatory process, a duodenorraphy can be performed (Fig. 16.3). The reconstruction should be seromuscular in transverse direction with absorbable suture; it is mandatory to avoid tension above the suture line to prevent leakage. A small, half-circle needle is placed through the edge of the defect, approximately 0.5 to 1.0 cm from the edge of the perforation. Three or four sutures are placed and are then tied to close the duodenal defect; the omental patch is placed on the tied sutures.

When the bowel defect is more extensive than 3–4 cm a duodenal resection with duodenojejunal anastomosis is the preferred option. A right-sided visceral mobilization and intestinal derotation maneuver (Cattell-Braasch-Valdoni maneuver) will completely expose the duodenal-jejunal junction and its mesentery. The proximal jejunum was transected approximately 5–10 cm distal to the ligament of Treitz, and

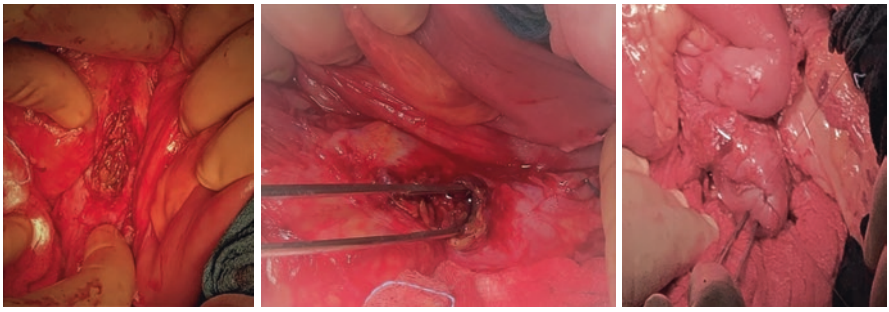

Fig. 16.3 From left to right, evidence of duodenal lesion of the AEF and duodenorraphy

the mesentery was sequentially ligated and divided. Dissection continued proximally to involve the fourth and third portions of the duodenum; an end-to-end duodenojejunal anastomosis is performed. To avoid injury of the ampulla of Vater, a cholecystectomy is systematically performed and a Fogarty catheter is placed in the duodenum through the cystic duct. A feeding jejunostomy can be helpful for nutritional support in the postoperative period.

The volume of gastric and pancreatic-biliary secretions can be decreased from the duodenal suture lines by routinely using an anterograde drainage including a nasogastric tube located in the second portion of the duodenum and a transcystic biliary drain. Other techniques can be used to divert the digestive enzymes from repairs. A triple-tube decompression, including a nasogastric tube or gastrostomy and two jejunostomies, one retrograde for duodenal decompression and one anterograde for enteral nutrition is provided.

When AEF occurs in jejunum ileum, sigmoid colon, ascending/descending colon, a midline incision is the preferred option; a bowel resection should be considered in case of large defect with thickened intestinal wall due to edema or inflammation. A primary anastomosis should be performed, there is no difference between handsewn or stapled anastomosis, it depends on surgeon preferences and experience.

Omentoplasty is useful to exclude the digestive suture or anastomosis from the graft to reduce septic complications.

16.1.7 Postoperative Time and Long-Term Follow-up

Postoperatively, these patients are frequently debilitated and have at least an element of systemic sepsis. Multisystem support in the intensive care setting is required, and a multidisciplinary team approach that includes specialists in intensive care, infectious disease, and nutrition offers the best chance of survival.

Even if antibiotics are continued, late infections can occur; therefore, these patients should be periodically reevaluated.

16.1.8 Take-home Messages (Fig. 16.4)

1. Endovascular is only as a bridge procedure in acute bleeding.
2. Graft removal and flow restoration is the treatment of choice.
3. In situ reconstruction only with biologic grafts or extra-anatomical revascularization is better.
4. Omentoplasty or myoplasty is mandatory to protect the aortic stump.
5. Single stitches reinforced by autologous pledgets to close the aorta.
6. Size and site of bowel defect is crucial for bowel reconstruction.
7. Suture closure of intestinal wall when defect is smaller than 3–4 cm.

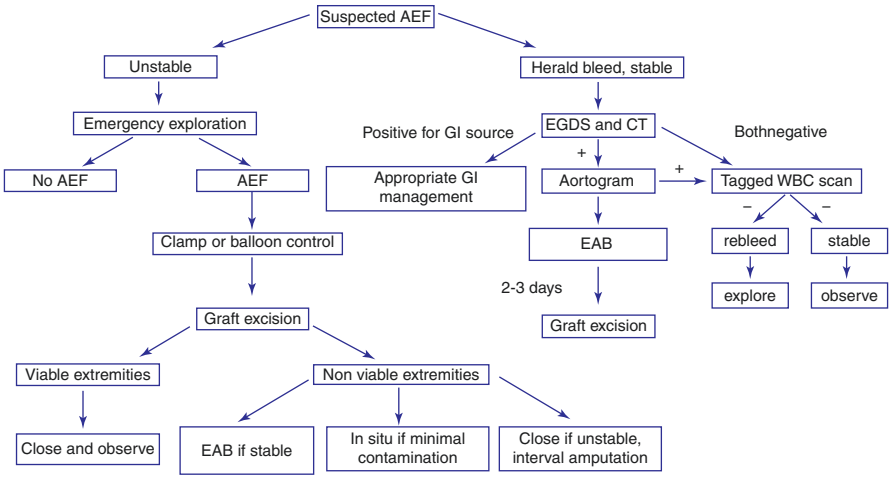

Fig. 16.4 Therapeutic algorithm in AEF

8. Intestinal resection when bowel defect larger is than 3–4 cm or malacic intestinal wall.
9. Decompression of the bowel by a second nasogastric tube over the Treitz or by jejunostomy.

16.2 Acute and Chronic Mesenteric Ischemia Clinical Case

An 81-year-old female was referred to the emergency department due to abdominal pain, nausea, and vomiting with a history of atrial fibrillation. Physical examination confirmed abdominal bruit associated with diffuse peritonitis. ECG demonstrated an acute atrial fibrillation, and blood tests showed leukocytosis with mild anemia. An abdominal CT scan revealed thrombotic occlusion of the superior mesenteric artery associated to small bowel and right colon abnormal distension, with intraparietal air in the intestinal wall. The patient was immediately transferred to operative theatre for an urgent laparotomy. At laparotomy all the small bowel, the right and transverse colon were dilatated, intestinal wall appeared thickened with associated edema and clinical sign of ischemia. A retrograde access to SMA was performed that confirmed thrombotic occlusion of the artery, revascularization was obtained by a Fogarty catheter. A damage control surgery with closure of the abdomen with negative pressure was preferred by the general surgeon to perform a second look to avoid an intestinal resection and anastomosis in critical condition. On Postoperative Day (POD) 2, the patient was again referred to the operative theatre, all the colon showed a good vascularization, a resection of 80 cm of small bowel was performed due to segmental ischemia. An intestinal anastomosis with double stapling technique was done and the abdomen was definitively closed. The postoperative course was uneventful except for a pulmonary complication treated by antibiotic therapy. The patient was discharged in 22 POD.

16.2.1 Introduction

Acute and chronic mesenteric ischemia (AMI and CMI) is a syndrome caused by inadequate blood flow through the mesenteric vessels with the high risk of gangrene development of bowel wall.

AMI is when symptoms occur in the first hours up to 24–48 h and duration is less than 2 weeks; subacute when a new symptomatic condition over chronic symptoms between 2 to 4 weeks.

CMI happens when mesenteric circulation fails to provide the postprandial response that is required to supply oxygen for the metabolic processes.

The overall prevalence of AMI is 0.1% of all hospital admissions; venous thrombosis is found in approximately 0.001% of patients who undergo exploratory laparotomy. CMI accounts <2% of all admissions for gastrointestinal conditions and median age of 65 years old (40 to 90 years) and female to male ratio is 3–4:1 [9, 10].

16.2.2 Mesenteric Collateral Patterns

To know the collateral pattern of mesenteric arterial and venous circulation is essential to understand the pathogenesis and to have a correct approach to the pathology.

The gastroduodenal and pancreaticoduodenal arteries provide collateralization between the CT and SMA; the marginal artery of Drummond and the arc of Riolan connect the left colic artery (inferior mesenteric artery) to the middle colic artery (SMA); the term central anastomotic artery describes marked enlargement that occurs in the arc of Riolan in patients with high-grade stenosis or occlusion of the SMA and collateralization via a patent inferior mesenteric artery (IMA). This artery lies in the mesentery in close proximity to the inferior mesenteric vein: inadvertent ligation, division or thrombosis of this important collateral artery during aortic exposure or other operative procedures may result in acute ischemia or bowel gangrene. The internal iliac arteries provide a collateral pathway via the hemorrhoidal branches.

The venous network provides the superior mesenteric vein (SMV) formed by the jejunal, ileal, ileocolic, right colic, and middle colic veins, which drain the small intestine cecum, ascending colon, and transverse colon. The right gastroepiploic vein drains the stomach to the SMV, whereas the inferior pancreaticoduodenal vein drains the pancreas and duodenum. The inferior mesenteric vein (IMV) drains the descending colon, the sigmoid colon, and the rectum through the left colic vein, the sigmoid branches, and the superior rectal vein, respectively. The IMV usually joins the splenic vein, which then joins the SMV to form the portal vein.

16.2.3 Pathogenesis

The diagnosis of AMI can be quite challenging: symptoms are initially nonspecific, before evidence of peritonitis and often diagnosis and treatment are delayed when pathology is advanced.

About risk factors: for AMI include atherosclerosis, arrhythmias, hypovolemia, congestive heart failure (CHF), recent myocardial infarction (MI), valvular disease, advanced age, and intra-abdominal malignancy [9, 10]; for CMI the most important cause is atherosclerosis associated with plaques in aorta and renal arteries. A non-atherosclerotic disease can also affect CMI such as vasculitis (giant cell arteritis, Takayasu's disease, and polyarteritis nodosa), systemic lupus, Buerger's disease, spontaneous dissections, fibromuscular dysplasia, neurofibromatosis, radiation arteritis, mesenteric venous stenosis, or occlusion [11].

AMI can be caused by an arterial disease or mechanical obstruction of the intestine (e.g., internal hernia with strangulation, volvulus, or intussusception). In the first case it may be subdivided into non-occlusive mesenteric ischemia (NOMI—20%) and occlusive mesenteric arterial ischemia (OMAI—embolic in 50% and thrombotic in 25%): the latter can be due to arterial embolism (AMAE) and acute mesenteric arterial thrombosis (AMAT) or dissection. It can also be caused by mesenteric venous thrombosis (MVT in less than 10%) [12].

Mesenteric vein thrombosis primarily occurs in the absence of any identifiable factor or secondary as after surgical ligation (splenic vein for splenectomy or portal vein or the superior mesenteric vein (SMV) as part of damage control surgery). The bowel becomes edematous and the intestinal outflow decreases, worsening progressively until it impedes the inflow of arterial blood, leading to bowel ischemia. The colon is usually spared because of better collateral circulation and the ischemia has a patchy distribution.

Once bowel wall infarction has occurred, mortality may be as high as 90%. Even with good treatment, as many as 50–80% of patients die [12].

16.2.4 Clinical Presentation

In normal conditions, approximately 20% of the cardiac output goes through the mesenteric arteries and the flow increases after the ingestion of a meal, approaching 100–150% above normal (2000 mL/min) over the next 3–6 h.

In case of AMI, the patient develops continuous pain, unrelated to food intake, because the mesenteric flow at rest is not guaranteed. This causes tissue hypoxia, leading to initial bowel wall spasm and gut emptying by vomiting or diarrhea.

Symptoms of CMI include abdominal pain, weight loss, and "food fear." The abdominal pain is often postprandial and begins within a few minutes to 30 min after meals, persisting for as long as 5–6 h.

16.2.5 Diagnosis

After clinical evaluation, the first step is a duplex study to evaluate the patency and stenosis of celiac trunk (CT), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA).

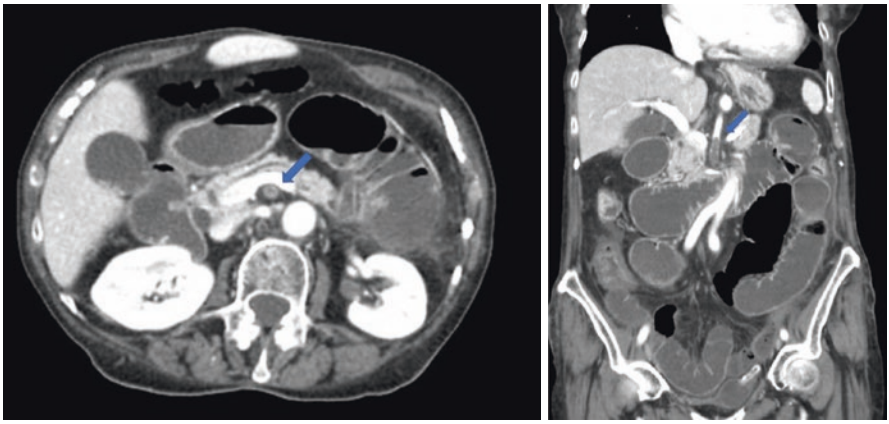

Fig. 16.5 CT that shows AMI due to SMA thrombosis (blue arrow), left axial, right sagittal view

Diagnostic criteria include ≥70% stenosis with peak systolic velocities >275 cm/s for the SMA and >200 cm/s for the celiac axis [13].

As second step CT or MRI should be performed. CT shows the quality of plaque and length of stenosis and possible presence of post stenotic dilatation, conditions that the surgeon should consider in the planning phase.

CT scan is considered the gold standard for diagnosing acute mesenteric ischemia (Fig. 16.5); in case of delayed recognition or non-exhaustive exams, many patients undergo an exploratory laparotomy. With the advent of minimally invasive surgery, diagnostic laparoscopy (DL) has taken a leading role as a less invasive alternative to laparotomy for the early diagnosis of acute mesenteric ischemia, especially if we also consider the possibility to perform it at the bedside in the most critically ill patients. Bedside DL in the Intensive Care Unit (ICU) has been described previously by several authors with a substantial confirmation of the above-mentioned advantages and the possibility to prevent unnecessary laparotomies. Moreover, a negative or non-therapeutic laparotomy can be associated with a morbidity rate as high as 5–22% and in some cases higher. Despite diagnostic laparoscopy is an invaluable tool and can be conducted at the bedside in ICU patients, it has a reduced sensitivity in the early stages of intestinal ischemia unfortunately because the mucosa can be extensively ischemic while the bowel might still appear normal at external inspection. This drawback can be overcome by using fluorescein-assisted laparoscopy with indocyanine green (ICG), with which even early stages of ischemia can be identified.

16.2.6 Treatment

AMI should be immediately treated once recognized; meanwhile CMI can be treated if pharmacological therapy is not sufficient and if the patient develops symptoms such as malabsorption, postprandial pain, vomiting, and diarrhea.

The treatment algorithm should consider if exploratory laparotomy is indicated and if etiology is established (arterial or venous and embolism or thrombosis).

Exploratory laparotomy is not always indicated as the first approach, but in some cases highly suggested. If AMI is due to cardiac embolism, endovascular treatment is not the ideal option because a thrombolysis can cause additional emboli to smaller arteries; so an open vascular approach should be chosen; in case of thrombosis, an endovascular approach can be the right choice for the recanalization.

If MVT is diagnosed, anti-coagulation is suggested unless there is direct evidence of bowel gangrene. If a patient has peritoneal signs or evidence of bowel gangrene, abdominal exploration is indicated regardless of the etiology.

About etiology, atherosclerotic lesions within the SMA and celiac axis represent the group with a higher dilemma in terms of therapeutic options: open, endovascular, or hybrid. If no evidence of bowel gangrene and no definitive indication for laparotomy, an endovascular approach alone, with or without a laparoscopic evaluation could be evaluated.

A vascular approach can be made in open or endovascular surgery and usually SMA is the main target.

Open surgery provides two approaches. One is endarterectomy or embolectomy with angioplasty with patch closure of the artery; the other is performing a bypass with autologous vein if there is frank necrosis or perforation or in the absence of contamination using a polyester or rifampin-soaked graft. If a prosthetic conduit is chosen, the graft should be covered by omentum.

Endovascular surgery has different approaches. Standard antegrade approach through femoral or brachial access or with a retrograde open mesenteric stenting. In the latter, a surgical exposure of SMA is performed by cranial retraction of the transverse mesocolon and exposure of the root of the mesentery to the right side of the abdomen. The mesentery is opened longitudinally on top of the SMA up to the inferior edge of the pancreas, allowing for dissection and control of the SMA and multiple jejunal branches. The segment of stenosis or occlusion is treated by primary stenting using a balloon-expandable stent [14].

After revascularization, the bowel is reassessed and additional bowel resection or anastomosis is performed. Second-look laparotomy is indicated in patients with severe ischemic changes or questionable bowel changes.

16.2.7 Damage Control Surgery

Many patients presenting with clinical signs of intestinal ischemia come to our observation with septic shock and multiorgan failure.

The damage control surgery (DCS) is part of a multidisciplinary strategy known as damage control management (DCM) with the resuscitation phase that will take place in ICU.

The goal of the DMS is the resection of ischemic intestinal tract following open abdomen (OA).

The use of OA after perfusion restoration in a patient with acute mesenteric ischemia as in occlusive proximal or distal superior mesenteric artery emboli and non-occlusive mesenteric ischemia (e.g., post-arrest or resuscitation from shock/arrest) should be considered in case of deranged physiology and bowel edema and necessity to perform a second look or delayed anastomosis. Mesenteric venous thrombosis requiring laparotomy does not routinely mandate OA as often as mesenteric ischemia; however, the risk of abdominal hypertension imposes attention to intra-abdominal pressure.

Negative pressure wound therapy with continuous fascial traction should be suggested as the preferred technique for temporary abdominal closure. Temporary abdominal closure without negative pressure (e.g., Bogota bag) can be applied in low resource settings.

Recent data from the International Register of Open Abdomen (IROA study) showed that different techniques of OA resulted in different results according to the treated disease (trauma and severe peritonitis) and if treated with or without negative pressure in terms of abdominal closure and mortality rate. The results favored the nonnegative pressure systems in trauma and negative pressure temporary closure in severe peritonitis patients.

Open abdomen re-exploration should be conducted no later than 24–48 h (Fig. 16.6). The abdomen should be maintained open if requirements for ongoing resuscitation and/or the source of contamination persists, if a deferred intestinal anastomosis is needed, if there is the necessity for a planned second look for the ischemic intestine and, lastly, if there are concerns about abdominal compartment syndrome development.

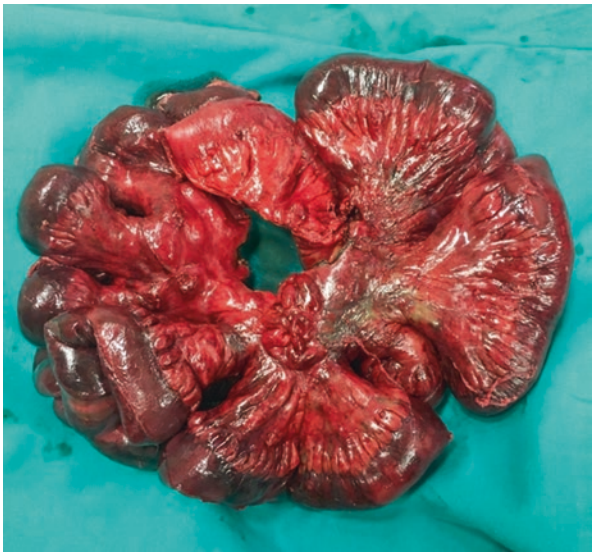

Fig. 16.6 Intestinal resection after damage control surgery

16.2.8 Postoperative Management

Patients are allowed to resume a regular diet within 6–8 h. Anti-platelet therapy is typically started prior to the vascular intervention with acetylsalicylic acid and continued indefinitely. Clopidogrel is started the day of the intervention with a loading dose and continued for 6–8 weeks as a dual anti-platelet agent, after which patients are kept on acetylsalicylic acid alone.

Follow-up includes clinical examination and duplex ultrasound every 6 months during the first year and annually thereafter.

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Part V

Upper and Lower Limbs

Upper and Lower Limbs: Surgical Anatomy and General Consideration in Emergency Settings

17

Matteo Marone and Ilenia D'Alessio

17.1 Introduction

It is extremely important, for every emergency physician, the knowledge of the surgical anatomy of the limbs since vascular injury is one of the most frequent causes of limb loss in trauma and non-trauma situations.

Considering the upper limb, its vascular tree originates in different ways: on the right side the *subclavian* artery originates from the *brachiocephalic* trunk and on the left it originates from the *aortic arch*. The subclavian artery runs in the chest and exits from it at the back of the clavicle, entering in the *prescalenic* area. At this level the *right laryngeal recurrent nerve* is strictly attached to the subclavian artery. To get vascular control at this level the best option is to proceed with a sternotomy and entering the chest.

At the level of the *interscalenic* space, the subclavian artery runs between the anterior and the mid scalene muscles and is surrounded by the brachial plexus. At this level, during the dissection, care must be taken to avoid nerve injuries. The favourite way to access this artery is by a supraclavicular incision.

At the level of *postscalenic* space the subclavian artery becomes *axillary* artery. The latter runs below the pectoral major and it is divided by the pectoral minor in 3 segments. The best way to expose this artery is performing a subclavicular incision and dividing the major and minor pectoralis muscles. The axillary vein usually is encountered first and great care is needed in order to avoid venous and nerve injuries.

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After the axillary artery, the vascular bundle enters in the arm and the artery is named *brachial* artery. The brachial artery lies close to the brachial vein and the median nerve, between the triceps and the biceps on the medial aspect of the arm.

At the level of the elbow the artery divides into the *radial*, *ulnar* and *interosseous* artery. The access to the brachial artery at this level is through a straight incision between triceps and biceps. The artery lies immediately below the fascia and it is important in this case to identify and preserve the median nerve in order to avoid injuries. If the artery needs to be isolated close to its bifurcation, then a lazy S incision needs to be performed and part of the biceps tendon is usually isolated.

Below the elbow radial and ulnar arteries lie deep, below the flexor muscles. The best way to access these arteries is to isolate the bifurcation and follow them into the arm. It is also possible to expose them at the level of the wrist but in this case great care needs to be taken in order to avoid injuries to the median and ulnar nerve [1, 2].

Considering the lower limbs, the *femoral* artery originates from the *external iliac* artery at the level of the inguinal ligament. The common femoral artery is located in the *inguinal triangle*. The border of the inguinal triangle are the inguinal ligament, the Sartorius muscle and the great abductor. At this level the common femoral artery splits in the *superficial femoral artery* that runs in the medial aspect of the thigh and the *profunda femoral artery* that lies deep close to the femur. To expose the common femoral artery and its bifurcation the best option is to perform an arcuate incision at the mid third of the groin. The artery is located lateral to the vein just below the fascia. During the dissection it is important to move the fat pad lateral to medial in order to avoid lymphatic leak.

The superficial femoral artery runs then in the Hunter channel. This channel is delimited medially by the great adductor and superiorly by the Sartorius. At the end of the channel the artery passes from medial to posterior perforating the adductor channel. To expose the first part of the superficial femoral artery a medial thigh incision needs to be performed and the Sartorius muscle needs to be moved from medial to lateral. To expose the distal part of the superficial femoral artery and the proximal popliteal artery a medial thigh incision needs to be performed and the Sartorius muscle needs to be moved from lateral to medial.

The superficial femoral artery continues into the *popliteal* artery. The latter runs posterior to the knee and is divided into three portions: supra-articular, infra-articular and subarticular. The exposure of the popliteal artery below the knee is performed through a medial incision in the upper leg. When the fascia is opened, the gastrocnemius muscle is retracted laterally and the fat pad is exposed. Surgeons usually encounter first the popliteal vein that can be mobilized and, below it, is possible to identify the artery itself.

The popliteal artery then gives the *anterior tibial artery* that runs in the anterior compartment to the leg and the *tibioperoneal trunk* that divides in *peroneal artery* and *posterior tibial artery*. The anterior and posterior tibial artery can be easily exposed at the level of the talocalcaneal joint because they run not too deep and close to the fascia [2, 3].

17.2 Emergency Settings

In emergency settings, there are two types of clinical scenarios that we can face to:

- **Ischaemic limb**
- **Haemorrhagic limb**

In the ***ischaemic*** limb ***scenario*** patients present complaining of intense *pain* of the affected limb, without irradiation, associated, in the early presentation, with *loss of sensation* and, in the delayed presentation, with *complete loss* of sensation *and muscular function* [4].

Causes of ischaemic limb in emergency situations can be numerous and can be divided into trauma related, non-trauma related and iatrogenic injuries

- *Trauma related* causes include blunt and penetrating trauma. In blunt trauma there is no open wound on the skin and the vessels can be injured due to forces that compress the artery causing: rupture with thrombosis of the vessels or dissection (more common). Bones fractures can also kink the vascular bundle of the limb, creating a total arterial and venous occlusion. In penetrating trauma, a foreign body penetrates the skin and can injure vascular structures causing: thrombosis or dissection [5, 6].
- *Non-trauma* related causes of limb ischaemia are acute ischaemia due to thrombosis of pre-existing plaque or distal embolization and dissection.
- Iatrogenic injuries can be caused by access site complications in endovascular procedures.

In the ***haemorrhagic*** limb ***scenario*** patients present with a vascular lesion actively bleeding, usually associated with tachycardia and low blood pressure. The ABG (arterial blood gas) will show high lactate and base excess. Causes of haemorrhagic limb are penetrating traumas, due mainly from stab and gunshot wounds, but also by iatrogenic procedure (e.g. bleeding from percutaneous access site). However, also bone fractures can be associated with haemorrhage if the sharp edge of the bone injuries the vascular bundle of the limb.

17.2.1 Signs of Vascular Lesions in Trauma Patients

Vascular lesions can present with **hard** signs or **soft** signs [7].

Hard signs are:

- ***Expanding haematoma***
- ***Absent pulses***
- ***Bruit or palpable thrill***
- ***Active haemorrhage***

- ***Distal ischaemia****-Ischaemic limb* can be easily identified clinically looking at the patient and looking for the "6P" of Pratt:
  - Pulseless
  - Pain
  - Paresthesia
  - Poikilothermia
  - Paralysis
  - Pallor

  Soft signs are:

- ***Non-expanding haematoma***
- ***Bleeding on the scene no more active***
- ***Nerve injuries***
- ***Proximity of the wound to the vessel***
- ***ABI (Ankle Brachial Index) <0.9***

17.2.2 Signs of Vascular Lesions in Non-trauma Patients

Patients with non-traumatic lesions usually present at E.D. (Emergency Department) with signs of acute ischaemia that are easily recognizable remembering "6P" of Pratt (see above). The most used classification to describe an ischaemic limb is presented in the table below and is known as Rutherford classification, adopted by SVS and ESVS (respectively Society of Vascular Surgery and European Society of Vascular Surgery) [8, 9] (Table 17.1):

It's important to know the existence of other two situations: blue toe syndrome and chronic ischaemia. The former is a digital ischaemia with intact large vessel circulation and is a variation of acute limb ischaemia, except that the size of the embolic material is small enough to travel into the end arteries. The latter is a chronic situation, in contrast to acute limb ischaemia, where repeated emboli to the

Table 17.1 Rutherford classification for acute ischaemia

Category	Sensory impairment	Motor impairment	Arterial Doppler signal	Venous Doppler signal
Class I Viable—no immediate threat	No	No	Audible	Audible
Class IIa Marginally threatened	Minimal in the toes or none	No	Often inaudible	Audible
Class IIb immediately threatened	Involves forefoot ±rest pain	Mild to moderate	Usually inaudible	Audible
Class III Irreversible	Anaesthetic	Paralytic/ rigour	Inaudible	Inaudible

extremities can manifest with a stepwise deterioration of distal arterial flow from individual embolic events that have either no or mild symptoms.

17.3 Diagnosis

It is important to recognize all types of vascular injuries, in order to avoid delay of treatments, which can lead to serious long-term complications and eventually limb loss. Once the clinical picture is defined, it is important to immediately call the vascular physician on call, to proceed with the appropriate diagnostic and therapeutic pathway.

17.3.1 Ischaemic Limb

In most cases, in trauma and non-trauma patients, DUS (Duplex Ultra Sound) is promptly performed due to its non-invasiveness, furthermore it is a diagnostic method radiation free and relatively cheap. DUS can show the presence of clot in the artery. Recent clot is viewed as ipo-anechogenic material in the lumen of the artery, while old clot is viewed as hyperechogenic material. In some centres, DUS is used as the sole diagnostic method before taking the definitive management decision. Other surgeons prefer to perform a CTA (computed tomography angiography) to all the patients presenting with ischaemic limbs. CTA allows a proper visualization of the problem and is important to identify other injuries in case of trauma. Nevertheless, this type of exam is expensive and its risks include radiation exposure and contrast material nephropathy. There is no utility in performing a CT scan without contrast material in the suspect of a vascular injury. DSA (digital subtraction angiography) is the gold standard in the identification of vascular problems and it can be performed in angiographic theatre, especially if below the knee problems are suspected. In traumatized patient, it may be helpful to perform X-ray in order to detect the presence of bone injuries [8, 10].

17.3.2 Haemorrhagic Limb

In this setting the physician usually faces an active bleeding coming from a skin wound or an expanding haematoma in the limb. The diagnostic instruments used in haemorrhagic patients are same used in the ischaemic patients: DUS, CTA, DSA and, if appropriate, X-ray.

17.4 Decision-making and Initial Management

In case of suspected vascular lesions, it is important to inform the trauma surgeon on call immediately, because in limb injuries ***time is life***. After 6 h of total ischaemia the muscular structure of the limbs is completely dead and the only possible intervention is the amputation. If the accepting hospital has no vascular surgery service, it is mandatory to inform the trauma surgeon or general surgeon on call, which task is to stabilize the patient to transport it to a tertiary facility.

17.4.1 Ischaemic Limb

For those who present with acute limb ischaemia, anticoagulation with a heparin and intravenous fluid therapy should be promptly initiated prior to making plans for intervention. These can be started while awaiting evaluation by a specialist trained to treat the embolic event. Options for managing lower extremity embolism include open embolectomy, thrombolysis and transcatheter thrombectomy. Factors such as anatomy, degree of limb threat, runoff, aetiology and patient factors will guide the choice of one over the other. (See Acute and Chronic limb ischaemia, cap 19.) Usually, in case of embolic disease a simple unblocking with a Fogarty catheter can be attempted performing a femoral artery cut down. The majority of the emboli stop at the femoral bifurcation or at the level of the popliteal artery. Sometimes a popliteal cut down needs to be performed to re-vascularize the 3 leg vessels (anterior tibial artery, posterior tibial artery, peroneal artery). In case of thrombotic disease over a previous atherosclerotic plaque an embolectomy procedure can be attempted but usually it fails. The best option is probably performing a DSA on table in order to find an appropriate runoff vessel and proceed with a bypass using synthetic graft or autologous vein. Vein is the favourite conduit if the target vessel is below the knee while, for above the knee vessels, Dracon or PTFE (polytetrafluoroethylene) can be chosen.

17.4.2 Haemorrhagic Limb

First of all, if patients arrive with a bleeding penetrating wound it is important to ask one of the members of the team to stop the bleeding, while the leader of the team performs the ABCDE primary survey (see Chap. 24). Haemostasis can be achieved using direct compression with gauze at the beginning. If the injury is located in a position where it can be compressed, a tourniquet can be applied at the base of the limb that can remain in situ for a maximum of 6 h [11]. If the injury is located in a non-compressible area like the subclavicular space, haemostasis can be achieved using Foley's catheter. Once the vascular control is achieved, the surgeon must evaluate the damage. It is mandatory to perform a debridement of the edge of the artery in order to reduce the risk of contamination and the risk of anastomotic failure. If, after the debridement, the edges of the artery are quite close, a primary repair can be

attempted. This is the best options because it is a quick procedure and it does not use any kind of graft. If the edges of the artery are distant, the situation is more complex. In these cases, a bypass needs to be performed. The preferred conduit is in any case the contralateral great saphenous vein. It is never an option to use the homolateral saphenous vein because it can be injured or, if the deep veins are injured, it is important to preserve it to allow an adequate venous outflow to the limb. Using a prosthetic graft is an option only if no other veins are available due to the high risk of infection of this kind of conduit.

In case of ***hard*** signs, the patient must be referred immediately to the vascular surgeon on call and he needs to go immediately to theatre in order to sort the problem out. Hard signs means that the patient is actively bleeding and it is almost certain to have vascular injuries at the site of the penetrating trauma. For this reason, there is no need for further investigations. It is mandatory to order blood and blood products for these types of patients because the risk of bleeding in theatre is really high [7].

In case of ***soft*** signs, the treating physician needs to rule out the presence of a vascular injury at the site of trauma. In order to do so the preferred techniques are DUS and CTA. However, DSA remains the gold standard in the diagnosis of vascular injuries, especially if foreign bodies can create artefact in the CTA and DUS images. In these kinds of patients there is time to diagnose the presence of an injury and there is no rush to take the patient to theatre (if he/she is stable). Nerve injuries are considered soft signs of vascular injuries because nerves run close to arteries and veins. If there is a nerve fallout probably not only the nerve but also the vascular bundle is damaged. In case of tract close to the anatomical location of the vascular bundle, it is important to check the vascular structure for injuries, because gunshot can create vascular dissection and damage to the surrounding tissue due to cavitation mechanism and, if we speak about stab wound, the physician cannot have the certainty of the direction of the penetration in the body [7].

Key messages

- The knowledge of upper and lower extremities surgical anatomy is mandatory to obtain a prompt and conscious approach to patients with ischaemic and/or haemorrhagic signs.
- In emergency setting there are two possible scenarios: ischaemic limb and haemorrhagic limb.
- To recognize an ischaemic limb remember the 6P of Pratt.
- The clinical diagnosis should precede the instrumental diagnosis that could be made using: Doppler UltraSound, Computed Tomography Angiography or Digital Subtraction Angiography.
- All ischaemic patients should be revascularized in 6 h (remember Time is life) and treated, initially, with heparin and fluids.
- All instable haemorrhagic patients need to be treated as soon as possible.

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Acute and Chronic Limb Ischemia

18

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18.1 Introduction

Limb ischemia is a common clinical condition that causes considerable morbidity and mortality and represents a major drain on healthcare resources. Critical acute (ALI) and chronic limb ischemia (CLI) represent a vascular emergency and are both a life and limb threatening disease.

Best care is required to reduce mortality and morbidity.

Both conditions occur due to the cessation of arterial perfusion to an extremity. If ALI is a vascular surgical emergency, also patients presenting with CLI need for emergent treatment, including medical, surgical, and ischemic lesions management.

The management of ALI and CLI remain particularly challenging because amputation rates are variable and despite therapy and mortality rates range from 10 to 40% of all patients due to increase in cardiovascular risk for this cohort of patients.

Moreover, distinction between ALI and CLI patients should be difficult, and patients admitted for ALI suffered chronic arterial disease. At last but not at least, oftentimes, patients presenting with ALI and CLI are the sickest patient cohort that vascular specialists treat.

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Surgical management has improved according to technological innovation and even if open surgical revascularization has been the mainstay of therapy, over the last several years, many percutaneous technologies have emerged that have provided alternative modalities of restoring perfusion. In addition, pharmacological development has provided new drugs, quickly become mandatory in emergent and long-life treatment of these patients.

18.2 Multimodal Approach to ALI and CLI

Patients suffering ALI and CLI need a multidisciplinary approach.

Emergent management of these patients aims to prevent major cardiovascular events, to treat ischemic pain and at last to guarantee distal perfusion.

Even if vascular surgeon could play a role of team leader, several specialists have to be involved in ALI and CLI emergent management and moreover during follow-up. Particularly cardiological evaluation is mandatory, all ALI and CLI patients should be considered at high risk for further cardiovascular complication and pharmacological control of major CV risk factors as hypertension, diabetes, and LDL level has to be started as soon as possible.

Anesthesiologists should be involved in management of ALI and CLI patients, although surgical procedures require only local anesthesia.

Emergent treatment of ALI and CLI
• Drugs: ALI/CLI anticoagulation/antiaggregation
• Pain control: anesthesiological evaluation
• Cardiac status: cardiac evaluation and specific therapy (heart rate control, statins, hypertension)
• Lesions control (CLI): foot drainage

18.3 Diagnostic Imaging

Imaging in ALI and CLI aim to identify anatomic localization of arterial occlusion.

Decision-making is crucial to reduce time to reperfusion and to minimize iodine contrast acute kidney injuries.

Bed-side ultrasound evaluation should follow clinical evaluation. Patients suspected for ALI and CLI have to be referred in centers that could guarantee complete diagnostic and therapeutic management.

In terms of diagnostic accuracy, digital subtraction angiography (DSA) is still considered the standard investigation for ALI and CLI. In patients with severe renal insufficiency, carbon dioxide angiography may be considered. Isolated DSA should be avoided, and it represents the first therapeutic line and should be performed in the operating theater where we intend to revascularize the patient.

Duplex-Ultrasound evaluation (DUS) is able to obtain the necessary information in 90% of cases where revascularization is considered, and is an accurate modality with which to detect complete or incomplete obstruction in the common femoral, superficial femoral, and popliteal arteries, and in bypass grafts. The diagnostic

accuracy is lower for detection of stenosis or occlusions in the aorto-iliac and tibial arteries. Computed tomography angiography (CTA) is considered more useful than DSA because it can combine evaluation of the possible primary cause of ALI, with high resolution evaluation of the outflow tract and provide a roadmap to guide treatment in both conditions. However, as mentioned for DSA, there is an association between the use of iodinated contrast and acute kidney injury and some papers evidenced as patients treated based on DUS as the sole preoperative modality, presented similar outcomes to those who had preoperative computed tomography angiography.

Imaging modality		
	CTA	Ultrasound
Aortic	+++	+
Iliac	+++	+
Femoral	+++	+++
Popliteal	+++	++
Tibial	++	+++ (hemodynamic evaluation)
Foot arteries	– (completion DSA required)	–

18.4 Surgical Approach to ALI and CLI

As mentioned current guidelines consider percutaneous approach as first-line treatment for majority of patients suffering ALI and CLI.

However, patients suffering ALI and CLI have to be treated in centers that could offer both techniques.

Operating theaters should be equipped for endovascular and open surgical treatment at the same time.

Although the number of patients treated purely with the endovascular procedure has grown, there is a significant number of patients that requires a combined (hybrid) approach using both techniques, preferably in one session in both acute patients and on chronic conditions. Moreover in an emergency setting or in some special conditions (patient's compliance, renal failure…) a comprehensive preoperative evaluation could be disregarded. This results in uncertain operating strategy in most of the patients.

The majority of patients presents significant comorbidities and requires complex multilevel arterial repair.

We could identify three different settings to perform both techniques:

- the interventional radiology suite, which is fully equipped for endovascular procedures, including excellent imaging tools and a broad portfolio of materials. However, it is not ideal for open surgery.
- The operating room (OR) is ideal for open revascularization but is usually equipped with only a mobile X-ray C-arm and a basic CAT laboratory. These shortcomings can be reduced due to recent technological developments. The modern C-arm already possesses the capability for digital subtract angiography with high resolution, which allows for road mapping, data backup, etc. The ste-

rility, illumination, and handling of the operating table are the advantages of an OR. It may be equipped with an X-ray-transparent operating table and an automatic injector for contrast agents. Although the image quality will never be the same as it is in the intervention suite, it is still preferable to perform ALI cases and CLI patients with undefined strategy.

- The third and ideal solution is a special hybrid operating theater that combines the advantages of the two previous options, but at a greater cost.

In all settings an ultrasound machine and the possibility to employ DUS scan in a sterile field became more and more mandatory. DUS allow access evaluation and a noninvasive control of distal perfusion in all patients.

Even the next sentence might seem obvious, it is important to stress that physician who intend to treat ALI and CLI patients should have skillfulness, experience, and competence in all diagnostic imaging evaluation and both operating techniques.

This goal could be achieved in a multidisciplinary team that could provide an emergent and simultaneous procedure for all patients.

18.5 Operating Theater Requirements

- **SPACE:** One of the most necessary aspects that is often the least considered is the need for adequate and proximate storage space for a stock of implantable materials and disposable items that is easily and quickly retrievable. These will include an inventory system to track and replace many readily used items, such as catheters, sheaths, wires, percutaneous transluminal angioplasty balloons, and stents. In addition to space for storage, there is a need for increased working space within the operating room. Meeting this increasing need for space may not necessarily be obtained by expanding the total square footage of the room but by eliminating unnecessary elements that obstruct the working space. Although a conventional operating room requires some basics, such as an instrument table, operating table, and anesthesia area, there is increased need in an endovascular suite to accommodate a movable table, the imaging equipment, and additional technologic supplements, such as intravascular ultrasound, mechanical thrombolytic machinery, and accessory tables to hold the percutaneous equipment. Furthermore, added space is necessary to allow for flexibility when approaching patients with percutaneous access. It may be necessary to access a patient via either the brachial or femoral artery, and the room must be convertible enough to accommodate this capability. Steric constraints can be modeled using computer-aided design applications to help the entire surgical team understand and optimize procedural flow.
- **IMAGING ACQUISITION AND STORAGE**: Pros and Cons using a portable C-arm rather than fixed equipment could be resumed in: increasing imaging quality reduce room flexibility. Data acquisition upgrades are not limited to the addition of a C-arm. In addition, surgeons should consider the construction of a control room. The control room houses the computer equipment necessary for postprocessing of the images and provides for an ample workspace to accomplish this. This is not easy to obtain in the majority of old hospital.

- **DATA VISUALIZATION:** capability to visualize preoperative data, and definition of intraoperative DSA improve results in ALI and CLI treatment.
- **RADIATION PROTECTION:** This point represents an issue for both patients and physicians. The increased endovascular workload leads to considerably extended exposure times for vascular surgeons comparable to those otherwise only encountered in interventional radiology and cardiology. Therefore, it is important that the vascular specialist is not only aware of all measures of radiation protection for the patient and also for the operating room personnel but is also fully informed on the topic of radiation protection at the administrative and legal levels.
- **FULL SURGICAL EQUIPMENT:** Open surgical procedures could be less invasive and more effective in CLI and ALI patients.

Surgical Vascular Access in Acute and Chronic Limb Ischemia Treatment

- **Axillary artery:** It could be dissected in the first and third part (minor pectoralis muscle division)—axillo-femoral bypass, necklace bypass, good access for endovascular procedure for aorto-iliac pathologies.
- **Brachial artery:** The whole artery could be isolated, distal part is compressible so could be eligible as puncture site for percutaneous procedures—Fogarty embolectomy of upper limbs, omeral access in percutaneous or hybrid procedure-.
- **Forearm arteries:** Surgical exposure should be useful in posttraumatic ischemic injuries; moreover, distal radial artery including snuff box segment represents a common percutaneous access.
- **Descending thoracic aorta:** It is uncommon cutdown in ischemic patients; however, there are some reports in complete infrarenal aortic occlusion treatment—thoracic aorta-femoral bypass.
- **Infrarenal Aorta:** Access to infrarenal aorta is necessary during aortic endarterectomy or aorto-bifemoral bypass.
- **Common and External Iliac arteries:** iliac endarterectomy, cross over bypass, iliac-femoral bypass.
- **Common Femoral artery:** This is the most common access in ischemic patient treatment for surgical or endovascular procedures (fit for large percutaneous access >8 Fr) (Fig. 18.1).
- **Superficial Femoral artery:** It represents an easy access during surgical procedures in the medial and distal part; however, it is not eligible for non-US-guided percutaneous access (Fig. 18.2).
- **Popliteal artery:** It represents the most common outflow vessel of infrainguinal bypass (Fig. 18.3).
- **Below the knee and below the ankle vessels:** They represent distal anastomosis site of ultradistal bypass and they have an increasing role (as all infrainguinal vessels) as puncture site for endovascular retrograde recanalization.

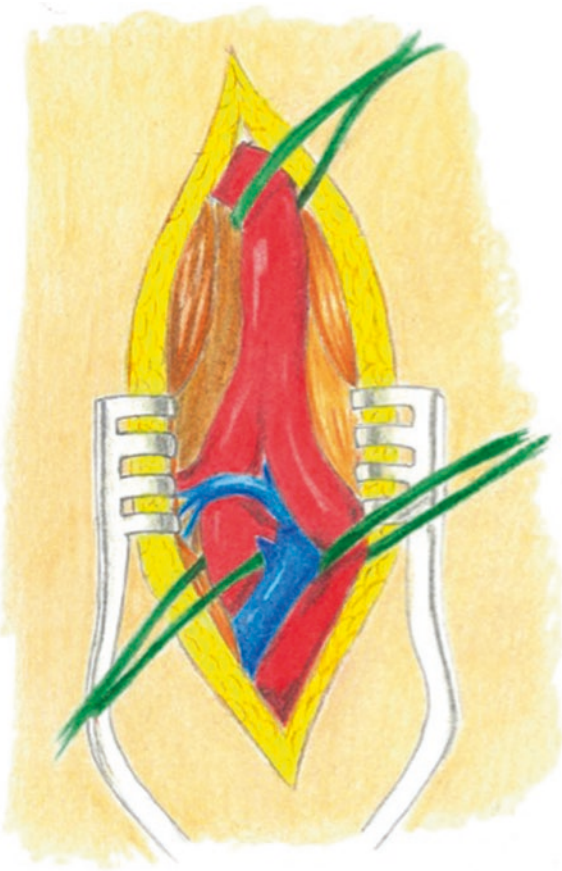

Fig. 18.1 Common femoral artery surgical access

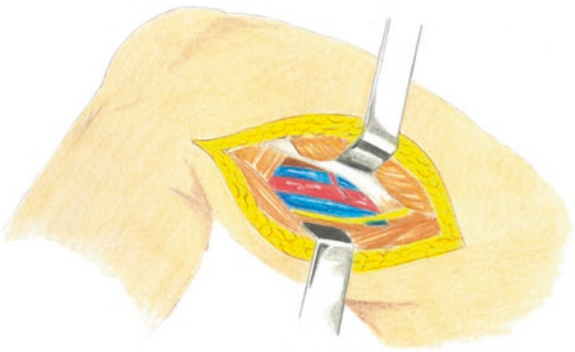

Fig. 18.2 Distal superficial femoral artery surgical access

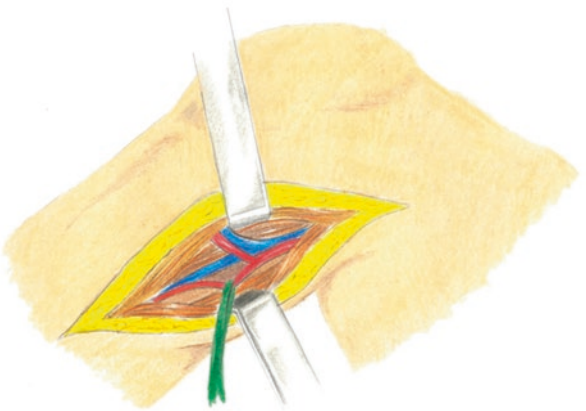

Fig. 18.3 Distal popliteal artery surgical access

18.6 Glossary of Most Common Procedure Using in Acute and Chronic Limb Treatment

18.6.1 Thrombus Management

- *"Fogarty Embolectomy"* since 1963 represents the mainstay in thrombus removal procedure for acute limb ischemia, more recently the original catheter was improved with over the wire technology.
- *Catheter directed thrombolysis (CDT):* In catheter directed thrombolysis, thrombolytic medication (tissue plasminogen activator, t-PA) is infused over time that spans hours to days, in the region of thrombus using a multi side-hole catheter.
- *Pharmacomechanical thrombectomy and percutaneous aspiration thrombectomy:*
- Pharmacomechanical thrombectomy (PMT) devices are utilized stand alone or more commonly in adjunct with CDT with the goal of endovascular thrombus maceration and removal. PMT devices in general can be categorized as rheolytic, rotational, or ultrasound enhanced. Along with the introduction of aspiration, PMT devices enhance the surgeon's ability to remove thrombus quickly, resulting in lower doses of thrombolytic drugs and reducing the time to reperfusion. Vacuum-assisted percutaneous aspiration thrombectomy (PAT) with the Penumbra Indigo Mechanical Thrombectomy System (Penumbra, Alameda, CA, USA) is emerging as an effective tool to remove thrombi and emboli from arteries and veins.

18.6.2 Atherosclerotic Lesions Management

- *Endarterectomy:* It consists in surgical removal of the plaque. Moreover, using a hybrid technique (Moll ring catheter), endarterectomy could be extended to arterial segment far from surgical cutdown. Procedure could be completed with patch angioplasty to improve long-term patency.

- *Bypass:* It is probably the gold standard treatment in chronic ischemia treatment, especially employed autologous conduit. However, improvement in endovascular techniques is still reducing number of these procedures in the majority of vascular centers.
- *Balloon angioplasty:* It is the most used endovascular procedure and after "Dotterization" the former one. Drug coated surfaces is one of the most common improvement of this technique as scoring device or intravascular lithotripsy.
- *Stenting:* A *peripheral artery stent* is a metal mesh tube that expands inside an artery. Use of stent as both primary treatment of bail-out is controversial. Innovation development, including drug eluting technology, is still reviving stenting in peripheral artery disease.
- *Endovascular bypass:* This technique involves cover stents deployment after intraluminal recanalization.
- *Endovascular Atherectomy:* It represents a sort of endovascular endarterectomy. It is an evolving technology with more and more devices available with promising results.

Further Reading

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Popliteal Artery Aneurysm and Non-atherosclerotic Limb Disease

19

Alberto M. Settembrini and Pasqualino Sirignano

19.1 Thoracic Outlet Syndrome (TOS)

Thoracic outlet syndrome (TOS) describes one or more patterns of symptoms as a result of compression of the neurovascular bundle at the thoracic outlet.

Neurogenic TOS (NTOS) is present when the brachial plexus is compressed at the scalene triangle or by the pectoralis minor muscle as it inserts on the coracoid process. Venous TOS (VTOS) is present when the vein is compressed by the anterior junction of the clavicle and first rib, or, less commonly, by the pectoralis minor muscle. It can manifest as acute thrombosis or by intermittent positional obstruction despite an open vein. Arterial TOS (ATOS) is thought to be present when true arterial damage occurs (thrombosis, distal embolization, and/or aneurysm formation), but may also be described as present when occlusion of the artery causes true ischemic symptoms.

The incidence of TOS is from 3 to 80 per 1000 people, and between 2500 and 3000 first rib resections (FRR) are performed yearly in the United States.

To understand the pathophysiology, it is essential to know the anatomy of the scalene triangle because its compression causes TOS. The triangle is formed by the anterior scalene muscle, the middle scalene muscle, and, inferiorly, the upper border of the first rib. This area has space just for its contents as subclavian artery, and anything that narrows the triangle (posttraumatic fibrosis and scarring of the anterior scalene muscle, a cervical rib or band) can cause its compression (Fig. 19.1).

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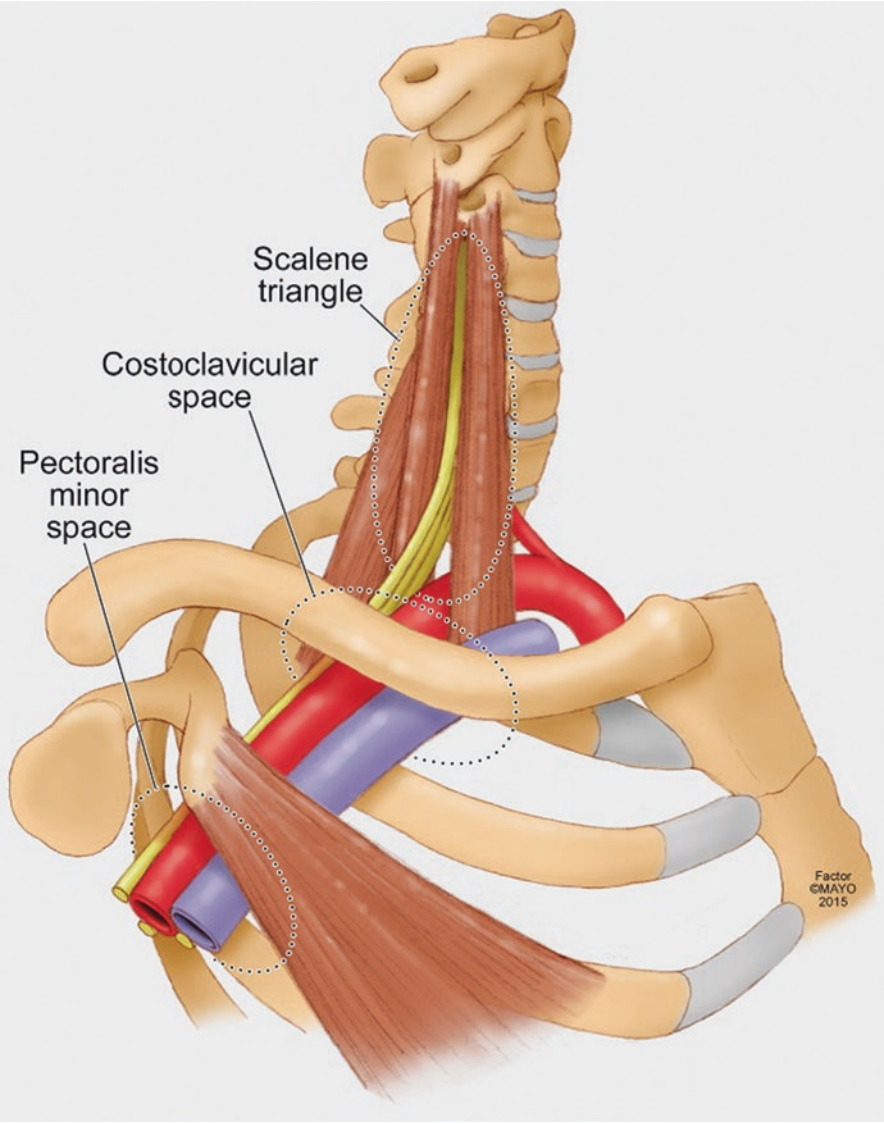

Fig. 19.1 Right thoracic outlet. Nerve (yellow) compression can occur at the scalene triangle (upper dotted line) or pectoralis minor space (lower dotted line), whereas venous compression most often occurs at the costoclavicular space (medium dotted line). Arterial injury is most often due to bone trauma at the scalene triangle. (from Reporting standards of the Society for Vascular Surgery for thoracic outlet syndrome. Illig KA, Donahue D, Duncan A, Freischlag J, Gelabert H, Johansen K et al. J Vasc Surg. 2016 Sep;64(3):e23–35)

In NTOS, local pain and tenderness are the symptoms of this compression and they can be worsened by all the movements that narrow the triangle (arms overhead) or stretch the brachial plexus.

In VTOS, the compression of the vein anteriorly at the junction of the first rib and clavicle is the cause. Such compression can narrow the vein (intermittent positional obstruction) or, much more commonly, cause no symptoms until actual vein injury and thrombosis are present (effort thrombosis). Subclavius muscle, tendon, and costoclavicular ligament are essential, but ultimately, the anterior portion of the rib (or clavicle) must be addressed to treat this entity [1].

True ATOS (defined as actual damage to the artery with or without distal sequelae) is almost always caused by a bony abnormality (cervical rib, abnormal first rib, long C7 transverse process). It should be noted that cervical ribs are relatively common (2.8% in women and 1.4% in men) [2].

19.1.1 Clinical Evaluation and Decision-Making

Patients with NTOS have symptoms caused by compression of the brachial plexus at the scalene triangle. Patients usually have two different symptomatology. Firstly pain and/or inflammation at the area of compression at anterior chest wall, axillary, shoulder, trapezius, and neck pain, and typically have headaches as well. Secondly, distal symptoms consistent with nerve compression: forearm and hand numbness, pain, paresthesia, and, in advanced cases, weakness and/or atrophy. Maneuvers that narrow the thoracic outlet (arms overhead or extended; elevated arm stress test) or stretch the brachial plexus (dangling, driving, vacuuming, running, and upper limb tension test).

VTOS is easier to diagnose. Patients always present acute thrombotic occlusion of the subclavian vein, causing suddenly swollen arms, often with cyanosis and dilated upper arm or chest wall collateral veins. In the history vigorous activity, often with the arms overhead, will be present (hence the term "effort thrombosis"). The acute thrombus is located very focally at the costoclavicular junction and later after clot propagation duplex becomes positive. Diagnosis by a skilled vascular ultrasonographer should be straightforward, although if suspicion is high and duplex is negative, diagnostic venography should be performed.

ATOS can be diagnosed because it presents with distal ischemia and/or a palpable supra- or infraclavicular mass suggesting an aneurysm. Things that suggest ATOS include young age, a bony abnormality, or absence of systemic atherosclerosis or inflammatory arteriopathies.

Decision-making in patients with a TOS depends on recognizing that the problem is due to the thoracic outlet. First, the original bony problem must be addressed, usually by excision. Secondly, any arterial damage should be repaired, and, thirdly, any distal issues should be addressed. Diagnosis is usually made by arteriography and ultrasound, and a plain chest X-ray is mandatory.

19.1.2 Treatment

As mentioned above, the treatment depends on the cause of the TOS. The different reasons provide different surgical approaches. There is the transaxillary approach,

especially in the case of the first rib removal in case of NTOS or ATOS, for VTOS the supra- or para-clavicular access to remove the muscular conflict.

19.2 Adventitial Cystic Lesion of Popliteal Artery

Cystic adventitial disease (CAD) is an uncommon condition in which mucoid cysts form within the adventitial layer of the arterial or venous wall. It is a rare cause of intermittent claudication that was first described in 1947 by Atkins and Key.

The cystic lesion can affect arteries and veins, but venous involvement is sporadic. The popliteal artery is affected in about 85% of cases. The cysts may be unilocular or multilocular. Gelatinous material characteristically builds up within the adventitial wall of the affected vessel resulting in narrowing of the lumen, compromising distal blood flow.

The cause is still uncertain. One possibility can be repetitive trauma especially in childhood; another reason could be a disorder of connective tissue or developmental inclusion of mucin-secreting cells within the adventitia of the artery could allow a cyst to develop within its adventitia or ganglion theory [3].

19.2.1 Clinical Evaluation and Decision-Making

Clinical evidence of this disease is usually in men (ratio to female 4:1) in the third or fourth decade because there is an onset of claudication or symptoms referred to as alteration of popliteal artery. Some degree of stenosis may be associated with the cyst for an extended period with preservation of luminal patency in the popliteal artery until the intracystic pressure exceeds that of the artery, causing compression and occlusion or stenosis causing significant associated symptoms.

Patients can be evaluated with standard vascular evaluation in an outpatient clinic, especially those without risk factors for peripheral vascular disease. After the initial sudden onset of cramping pain in the calf, the patient may experience some relief followed by typical intermittent claudication as good collateral circulation develops as shown in peripheral atherosclerotic disease. The Ishikawa sign is the loss of pedal pulses upon passive flexion of the knee due to compression on the popliteal pulses by cysts and can be helpful in the presence of large cysts.

At duplex the affected limb will show a flattening of the pulse wave below the cystic lesion. Because of the excellent collateral circulation that usually develops in young men, the total absence of pulse waves over the distal tibial arteries is rare. Magnetic resonance and CTA can show the “scimitar sign” a smooth occluding contour of the popliteal artery.

19.2.2 Treatment

Treatment of adventitial cystic disease of the popliteal artery can be conservative through the aspiration, under duplex localization and percutaneous approach, of the cyst eradicating it and resultant arterial stenosis or occlusion in some patients. Because the cystic content is quite viscous and gelatinous, aspiration must be done with a relatively large-bore needle.

A surgical intervention provides effective treatment. These can be the evacuation of the cyst or enucleation in case of no occlusion of the artery approaching the popliteal artery as the same for popliteal aneurysm through the posterior way with S-shaped incision or medial incision.

If occlusion of the popliteal artery has occurred, a total resection and reconstruction with prosthesis or autogenous saphenous vein is the choice with better results with a vein graft.

The recurrence rate of popliteal artery CAD after revascularization surgery is very low (0% to 10%) compared with the rate after cyst excision alone (10–34%) [4].

19.3 Popliteal Entrapment

Popliteal entrapment syndrome is a rare, potentially limb-threatening, vascular disorder causing intermittent claudication in young adults without a previous history of cardiovascular disorders. Similar to cystic lesions, this disease can be revealed in young patients without any cardiovascular risk factor.

It was first described in 1879 by a medical student in Edinburgh, Scotland. T. P. Anderson Stuart was dissecting the amputated leg of a 64-year-old man, and he described the anatomic abnormality associated with the abnormal course of the popliteal artery. Love and Whelan first used the term popliteal artery entrapment syndrome in reporting two cases in 1965.

Over the last decades, the incidence has increased to 0.17–3.5% [5]. Furthermore, 85% of patients affected with this condition are males, with almost 60% of cases occurring in young athletes during the third decade of life [6] (Fig. 19.2). Moreover, in about 30% of the cases, the disease has a bilaterally symptomatic presentation.

19.3.1 Clinical Evaluation and Decision-Making

The pathophysiology of this disease is understood considering the embryologic development of structures of popliteal fossa: the in utero development of the gastrocnemius muscle is a dynamic process with the potential for the occurrence of several anatomic abnormalities. The medial head of gastrocnemius muscle (MHGM) is one of the most important causes of entrapment syndrome classified into six different types (Fig. 19.3).

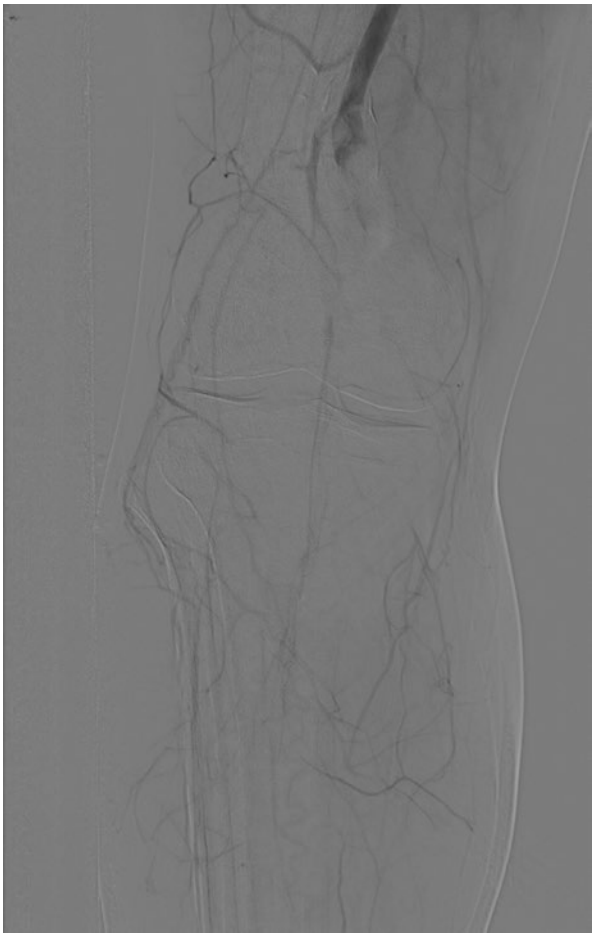

Fig. 19.2 Lower limb angiography that shows thrombosis of popliteal artery at the knee due to entrapment of MHGM

Type I: MHGM is the typical position, but the popliteal artery has an aberrant medial course under the muscle; **Type II**: MHGM insertion has a more lateral way on the posterior face of the femur causing a medial and inferior course of the popliteal artery.

Type III: Abnormal fibrous band or accessory muscle arising from the medial or lateral condyle forcing the popliteal artery.

Type IV: Popliteal artery lying in its primitive deep or axial position within the fossa, becoming compromised by the popliteus muscle or fibrous bands.

Type V: The entrapment of both the popliteal artery and vein due to any of the causes mentioned above.

Type VI: The muscular hypertrophy, resulting in a functional compression of both the popliteal artery and vein.

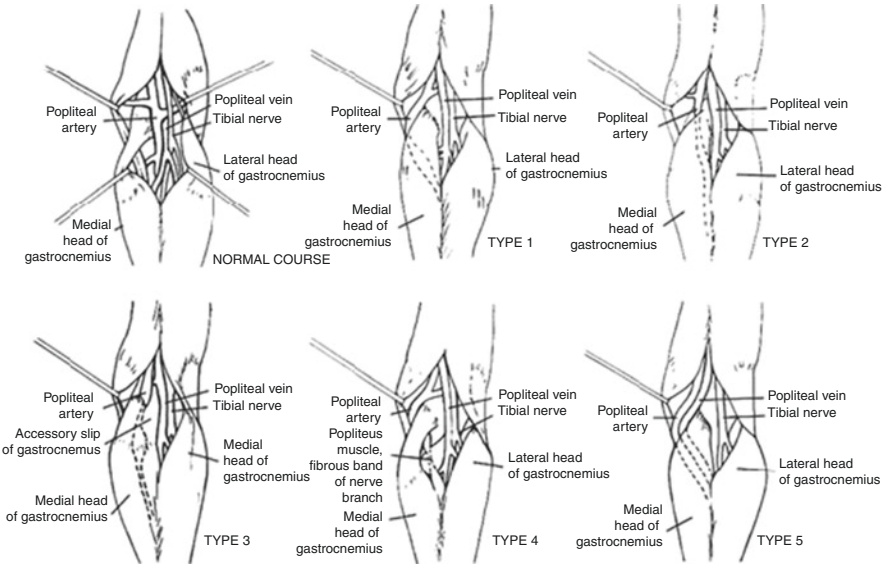

Fig. 19.3 Different types of popliteal entrapment. Two are the crucial points: medial head of the gastrocnemius and the popliteal artery. (A. L. Rochier, B. E. Sumpio. variant popliteal entrapment syndrome involving the lateral head of the gastrocnemius muscle: a case report, Ann Vasc Surg 2009;23:535.e5–535.e9)

Clinical evaluation should start with the history collecting. Usually the patients are young and report symptoms similar to peripheral arterial disease with leg and foot claudication, associated with lower extremity numbness, paresthesia, discoloration, pallor, and coolness. Physical examination may reveal hypertrophy of the calf muscles. A regular foot pulse disappearing during active plantar flexion and passive dorsiflexion is a characteristic phenomenon. Usually the differential diagnosis is with orthopedic syndromes [7].

The diagnosis can be confirmed with duplex ultrasound using provocative maneuvers (leg/foot positioned first in a neutral position and then in resisted plantar flexion) that provides a quick, inexpensive, and non-invasive initial screening test.

CTA or angio-MRI is mandatory to obtain a precise and definitive diagnosis.

The management is tailored to the patient based on the presence and absence of the symptoms. Incidental findings can be conservatively treated, but if muscle insertion abnormalities are the cause even in an asymptomatic patient, surgical correction is the preferred method of treatment.

In cases of extensive popliteal artery wall damage, occlusion, or aneurysm development, interposition of a bypass graft using an autogenous vein has been proposed.

19.4 Popliteal Artery Aneurysm

Popliteal artery aneurysms (PA) are the most frequent peripheral aneurysm with 7/100,000 in men and 1/100,000 in women incidence.

The popliteal artery in normal anatomy has a mean diameter of 7 mm in men and 6 mm in women so the threshold for defining a popliteal artery aneurysm is more or less an artery greater than 1 cm. Dilatation often is bilateral (Fig. 19.4**,** up to 60%) and associated to other aneurysms mainly in abdominal district: 5% of people presenting with an AAA have a popliteal aneurysm, while about 20% of people diagnosed with a popliteal aneurysm have a concomitant AAA [8, 9].

19.4.1 Clinical Evaluation and Decision-Making

Clinical presentation is a palpable mass in the popliteal fossa behind the knee, and the chance to detect it is directly correlated with the dimension of the sac. Most popliteal aneurysms are asymptomatic and can be discovered during routine physical examination or diagnostic imaging.

Most popliteal aneurysms are asymptomatic, like other aneurysms, but symptoms can be with claudication to critical limb ischemia in case of popliteal aneurysm thrombosis. Popliteal aneurysms have a laminar thrombus inside the sac and mechanical stress produced by the movements of the knee on the artery squeezes out particles of the thrombus which embolizes the runoff vessels causing claudication or acute peripheral ischemia or blue toe syndrome.

Usually duplex studies can reveal in popliteal fossa the dilatation of the popliteal artery; as second-level exams, a CT angio or MRI can be performed to plan for the intervention, considering that CT angio is more helpful in case of endovascular treatment.

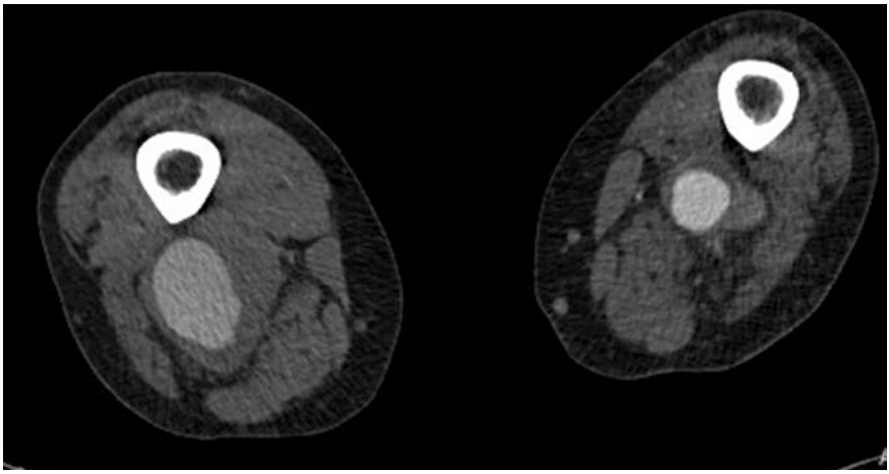

Fig. 19.4 Axial images of bilateral popliteal aneurysm

19.4.2 Treatment

Considering that the popliteal aneurysm thrombosis can be a catastrophic condition with high-grade acute limb ischemia due to the poor collateral network, it is crucial to know when it is indicated the treatment of a popliteal aneurysm.

Some studies suggested a possible growth rate for asymptomatic popliteal aneurysms: when <2 cm the annual expansion rate is about 1.5 mm/year, it increases at 3 mm/year when the diameter is between 2 cm and 3 cm and jumps at 3.7 mm/year if it exceeds 3 cm [10].

In our experience, aneurysms smaller than 2 cm can cause acute limb ischemia, which requires prompt intervention to reduce the risk of leg amputation.

In emergency in case of PA thrombosis thrombolysis is considered in most cases the choice through the use of Urokinase and rt-PA. The advantage of the latter is the shortened procedure, as its infusion usually takes considerably less time [11].

In any case, physicians should judge if the limb can sustain the obvious delay associated with this technique: a motor deficit or sensory loss indicates the need for immediate treatment.

19.4.3 BOX Indication for PA Treatment

Symptomatic aneurysms

- TREAT REGARDLESS OF SIZE (UNLESS UNFIT)

Asymptomatic aneurysm

- <2 cm: monitor
- 2–2.5 cm: consider treatment, but keep in mind
  - life expectancy (age and comorbidities)
  - operative risk
  - factors associated with possible evolution (thrombus, distortion)
- ≥2.5 cm: treat (unless unfit)

The approach to PA can be in open or endovascular surgery. The open treatment provides two different surgical approaches: medial or posterior that have no significant difference in terms of nerve lesions, but the posterior has shorter in-hospital stay [12]. With the onset of stent-graft endovascular treatment of popliteal aneurysm is preferred in many centers because it reduces the invasivity through the femoral access and length of stay, but there is the risk of endoleak, the sac is not removed, and the graft can slide down. So it is not indicated in giant aneurysms because the involvement of the fossa risks remain important maintaining compressive symptoms.

Through a medial approach, the operator can easily reach any arterial segment proximal to the dilatation. Through an opening of the origin of the soleus muscle, access to the lower popliteal artery, to the origin of the anterior tibial and the tibioperoneal trunk, is obtained as well.

The advantages are the possibility to go up to superficial femoral artery or common femoral artery in case of highly calcified arteries and the saphenous vein harvesting; the disadvantages are complete removal of the aneurysmatic sac or complete exclusion through ligation of every single collateral vessel is problematic.

The posterior approach can be used if the extension of the dilatation is limited to the popliteal fossa (usually aneurysms behind the knee joint), this route has the enviable advantage of allowing for a complete and easier aneurysmectomy and a very short interposition graft and shorter hospital stay and quicker recovery [13].

The exposure, in prone patients, proceeds through veins (popliteal, small saphenous, gastrocnemius veins) and nerves (sciatic nerve, tibial nerve, and common fibular nerve). On the contrary, the harvesting of great saphenous vein is more complex considering that a small saphenous vein is often too small to be the suitable conduit.

19.4.4 BOX indications for type of PA treatment

19.4.4.1 Endovascular IF

- Adequate proximal and distal neck
- Possibly, diameter at proximal neck not very different from diameter at distal neck
- At least 1 (better if 2) runoff vessels
- Older patients
- Increased surgical risk

19.4.4.2 Open Surgery

Posterior approach

- Only for aneurysm limited to the popliteal fossa.
- Only if minimum probability of tibial arteries being involved in surgery.
- Plan vein harvesting in advance (proximal thigh vs. knee level vs. short saphenous).
- Very short aneurysms can be substituted with prosthetic grafts (PTFE).

Medial approach

- The most versatile approach: allows for treating all kinds of aneurysms in all situations: use whenever in doubt for any reason.

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Part VI

Specific Clinical Pictures: Surgeon Perspectives

The Infected and Septic Patient

20

Emanuele Botteri, Nicoletta Lazzeri, Silvia Mazzoleni, and Frank A. Rasulo

20.1 Infection and Sepsis

20.1.1 Definitions

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Infection is defined as the invasion of normally sterile tissue by organisms resulting in infectious pathology.

Bacteremia is the presence of viable bacteria in the blood.

Infections and/or bacteremia can lead, if not diagnosed or treated properly, to sepsis or even to septic shock; therefore, care must be taken in identifying the subjects at risk [1].

20.2 Epidemiology

20.2.1 Incidence

Since 1970s rates of sepsis have increased. A retrospective analysis of an international database reported a global incidence of 437 per 100,000 person-years for sepsis between the years 1995 and 2015, although this rate was not reflective of contributions from low- and middle-income countries [2]. The Global Burden of Disease Study reported that in 2017, an estimated 48.9 million incident cases of

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sepsis were reported [3]. Approximately 11 million deaths were reported, representing 19.7% of all global deaths. While incidence and mortality varied across regions, the overall mortality decreased by almost 53% between 1990 and 2017. Moreover, this study highlights the need for greater prevention and treatment of sepsis, particularly in areas of the world with the lowest socio-demographic index.

Reasons for a possible increased rate of sepsis include advancing age, immunosuppression, and multidrug-resistant infection [4–6].

The incidence is also greatest during the winter, probably due to the increased prevalence of respiratory infections [7].

20.2.2 Pathogens

The contribution of various infectious organisms to the burden of sepsis has changed over time. Gram positive bacteria are the pathogens that are most commonly isolated from patients with sepsis. The incidence of fungal sepsis has increased over the past decade, but remains lower than bacterial sepsis [5]. In approximately half of cases of sepsis, an organism is not identified (culture negative sepsis) [8].

20.3 Pathophysiology

When a pathogen enters a sterile site in the body, it is detected by the immune system, which starts a host response. Innate immune cells, specifically macrophages, can recognize and bind non-self components on the surface of the pathogen, leading to phagocytosis of both invading bacteria and of debris from injured tissue. Moreover, macrophages start releasing proinflammatory cytokines, with consequent recruitment of additional inflammatory cells, namely leukocytes. If the bacterial load is limited, a local host response is sufficient in clearing the pathogen [9].

Sepsis occurs when activation of immune system exceeds the burden of the local environment, leading to a generalized and sometimes dysregulated host response involving multiple tissues, with consequent cellular injury and organ dysfunction.

Some of the mechanisms proposed to explain this process of injury include: tissue ischemia (insufficient oxygen relative to oxygen need), cytopathic injury (direct cell injury by proinflammatory mediators and/or other products of inflammation), and an altered rate of apoptosis (programmed cell death). No organ system is protected from the consequences of sepsis. Those that are most commonly involved include the circulation, lung, gastrointestinal tract, kidney, and nervous system [10].

20.4 Disease Severity

Sepsis exists on a continuum of severity ranging from infection and bacteremia to sepsis and septic shock, which can lead to multiple organ dysfunction syndrome (MODS) and death.

According to the Society of Critical Care Medicine (SCCM) and the European Society of Intensive Care Medicine (ESICM) we can define:

Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection.

Early sepsis—no formal definition of early sepsis has been validated until now. Nevertheless, it is important to monitor patients at risk of developing more serious infections or those that can progress to septic shock and MODS. The qSOFA (quick Sequential Organ Failure Assessment score) is a simple and validated score that can be used by non-intensivists to identify the subjects at risk of increased morbidity and mortality. The three components of the scale are readily identifiable at the bedside and are allocated one point each:

- Respiratory rate ≥22/min
- Altered mentation
- Systolic blood pressure ≤100 mmHg

A score >2 indicates an increased risk of progression to septic shock and MODS.

The qSOFA score was originally validated in 2016 as most useful in patients suspected as having sepsis outside of the intensive care unit (ICU) [11].

Septic shock—a state of severe hypotension that doesn't respond to adequate fluid resuscitation, that requires vasopressors to maintain a mean arterial pressure (MAP) ≥65 mmHg and have lactate >2 mmol/L (>18 mg/dL).

Septic shock is a type of vasodilatory or distributive shock.

Multiple organ dysfunction syndrome—progressive organ failure requiring immediate life support. MODS represents a final common stage of both infectious and non-infectious insults.

Common parameters to be monitored in septic patients to diagnose MODS are [12, 13]:

- Respiratory—Partial pressure of arterial oxygen (PaO_2)/fraction of inspired oxygen (FiO_2) ratio
- Hematology—Platelet count
- Liver—Serum bilirubin
- Renal—Serum creatinine (or urine output)
- Brain—Glasgow coma score
- Cardiovascular—Hypotension and vasopressor requirement

In general, the greater the number of failing organs the higher the mortality, with the greatest risk being associated with respiratory failure requiring mechanical ventilation.

The most common manifestations of severe organ dysfunction are acute respiratory distress syndrome, acute renal failure, and disseminated intravascular coagulation [14].

20.5 Risk Factors

Risk factors for sepsis include the following [15]:

- Intensive care unit admission.
- Bacteremia.
- Advanced age (≥65 years).
- Immunosuppression.
- Type 2 diabetes and obesity.
- Cancer.
- Community acquired pneumonia.
- Previous hospitalization.

20.6 Clinical Presentation and Diagnosis

Importantly, the presentation is nonspecific such that many other conditions (e.g., pancreatitis, acute respiratory distress syndrome) may present similarly.

20.6.1 Symptoms and Signs

- Specific to an infectious source (e.g., pain and purulent exudate in a surgical wound may suggest an underlying abscess).
- Arterial hypotension (e.g., systolic blood pressure <90 mmHg, mean arterial pressure <70 mmHg).
- Temperature >38.3 or <36 °C.
- Heart rate >90 beats/min.
- Tachypnea, respiratory rate >20 breaths/min.
- Altered mental status, obtundation, or restlessness.
- Oliguria urine (output <0.5 mL/kg/h for at least 2 h despite adequate fluid resuscitation) or anuria.

These findings may be modified by preexisting disease or medications. As examples, older patients, diabetic patients, and patients who take beta-blockers may not exhibit an appropriate tachycardia as blood pressure falls.

20.6.2 Laboratory Signs

- Leukocytosis (WBC > 12,000/μL) or leukopenia (WBC count < 4000/μL).
- Hyperglycemia (plasma glucose > 140 mg/dL) in the absence of diabetes.
- PCR.
- Arterial hypoxemia.
- Creatinine increase.
- Coagulation abnormalities (INR > 1.5 or aPTT > 60 s).
- Thrombocytopenia (platelet count < 100,000/μL).
- Hyperbilirubinemia (plasma total bilirubin > 4 mg/dL).
- Hyponatremia, hyperkalemia.
- Hyperlactatemia: an elevated serum lactate (e.g., >2 mmol/L) can be a manifestation of organ hypoperfusion [16].
- PCT elevated.

20.6.3 Imaging

To identify the focus of infection, multiple radiologic exams can be performed (RX, TC, echography).

20.6.4 Microbiology

The identification of an organism in culture is highly supportive of the diagnosis of sepsis. A key point in the appropriate management of septic shock is to obtain cultures, including blood cultures, as soon as possible, always before starting any antimicrobial treatment.

20.6.5 Diagnosis

A constellation of clinical, laboratory, radiologic, physiologic, and microbiologic data is typically required for the diagnosis of sepsis and septic shock. The diagnosis is often made empirically at the bedside upon presentation, or retrospectively when follow-up data returns (e.g., positive blood cultures in a patient with endocarditis) or a response to antibiotics is evident.

20.6.6 Initial Evaluation of Common Sources of Sepsis: Symptoms/Signs

respiratory tract: pharyngeal inflammation, exudate, swelling and lymphadenopathy, productive cough, pleuritic chest pain, consolidative auscultatory findings

central nervous system: altered mental status, signs of meningeal irritation, focal signs, irritation at insertion site of peridural catheter

urinary tract: urgency, dysuria, loin, or back pain

vascular catheters: redness or drainage at insertion site

pleural catheter: redness or drainage at insertion site

gastrointestinal: abdominal pain, distension, diarrhea, and vomiting

20.6.7 Initial Microbiologic Evaluation

respiratory tract: throat swab for aerobic culture, sputum of good quality, rapid influenza testing, urinary antigen testing (e.g., pneumococcus, legionella), quantitative culture of protected brush or bronchoalveolar lavage

central nervous system: CSF analysis and culture

urinary tract: urine culture

vascular catheters: culture of blood (from the catheter and a peripheral site), culture catheter tip (if removed)

pleural catheter: culture of pleural fluid (through catheter)

gastrointestinal: stool culture for Salmonella, Shigella, or Campylobacter; detection of Clostridium difficile toxin

intra-abdominal: aerobic and anaerobic culture of percutaneously or surgically drained abdominal fluid collections [17]

20.7 Prognosis

Sepsis has a high mortality rate, estimated in a range from 10 to 52% [4, 5, 18–20].

Mortality rates increase linearly according to the disease severity [21].

Most deaths occur within the first 6 months, but the risk remains elevated at 2 years. Patients who survive sepsis are more likely to be admitted to long-term care facilities in the first year after the initial hospitalization, and also appear to have a persistent decrement in their quality of life [20, 22–24].

Clinical characteristics that impact the severity of sepsis and, therefore, the outcome include the host's response to infection, the site and type of infection, and the timing and type of antimicrobial therapy.

20.8 Management of Sepsis and Septic Shock

The key point in managing sepsis and septic shock is to act fast, within the first hours of onset of symptoms, to avoid increased morbidity and mortality. When MODS ensues, the patient must be transferred to an ICU ward. The

recommendations cited in this chapter are taken from the "International Guidelines for Management of Sepsis and Septic Shock: 2016" [25].

20.8.1 Initial Resuscitation

- Treatment and resuscitation should begin immediately at least 30 ml/kg of IV crystalloid should be given within the first 3 h.
- Crystalloids are the fluids of choice for both initial resuscitation and subsequent volume replacement. Both balanced crystalloid solutions and saline solutions can be used; ringer lactate should be preferred as initial fluid.
- Albumin can be used for volume replacement in selected case; it is always advised to contact the intensivist to implement fluid therapy as needed if the target MAP of 65 mmHg is not reached with crystalloids.
- Following initial resuscitation, additional fluids should be given according to the hemodynamic status, which must be reassessed frequently. Reassessment should include a thorough clinical examination and evaluation of available physiologic variables (heart rate, blood pressure, arterial oxygen saturation, respiratory rate, temperature, urine output, and others, as available) as well as other noninvasive or invasive monitoring, such as echocardiography. An initial reasonable target of MAP is > 65 mmHg. Lactate levels can be used as markers of tissue hypoperfusion.

20.8.2 Specimen Analysis

Appropriate culture specimen (including blood) should be obtained before starting antimicrobial treatment in patients with suspicion of sepsis or septic shock. The collection of these materials must not delay the onset of antimicrobial therapy.

20.8.3 Antimicrobial Therapy

- Antimicrobials must be started as soon as possible, ideally within 1 h from the diagnosis of sepsis/septic shock.
- Starting with broad-spectrum antimicrobial therapy is a good strategy to cover all likely pathogens (including fungi) while waiting for the results of culture specimen. Consultation with an infectious disease specialist may be useful to choose the appropriate treatment regimen.
- Antibiotic de-escalation can be started once pathogen identification, together with its level of resistance, has occurred. It is advised to discuss every change in antimicrobial therapy with an infectious disease specialist.

- A proper antimicrobial therapy should last around 7–10 days; longer courses may be appropriate in patients with a slow clinical response, with undrainable foci of infection, with bacteremia from S. aureus and from some fungal/viral infections, and in immunocompromised patients. As usual, discussion with an infectious disease specialist is essential to evaluate eventual continuation of therapy.
- Procalcitonin can be measured throughout the septic state to evaluate the progression of the patient and can be viewed as a marker, together with other inflammation markers, for eventual discontinuation of antimicrobial therapy.

20.8.4 Vasoactive Medications and Corticosteroids

- If MAP cannot be maintained >65 mmHg after appropriate fluid replacement, it is recommended to contact the intensivist to evaluate the admission to ICU.
- Once admitted to the ICU, vasopressors can be started to.
- Norepinephrine must be the first vasopressor to be used in septic shock.
- If adequate pressure values cannot be maintained with noradrenaline alone, either adrenaline, dobutamine, or vasopressin can be added.
- Hydrocortisone can be used in septic shock, at a dose of 200 mg/day.

20.8.5 Venous Thromboembolism Prophylaxis and Stress Ulcer Prophylaxis

- LMWH can be used, in the absence of contraindications, in patients with septic shock, to prevent TVP/PE.
- Gastroprotection is to be started as soon as possible once a day; if hydrocortisone is added to therapy, the dose must be doubled.

The key points in the diagnosis and management of septic shock are summarized in the flowchart below (Fig. 20.1).

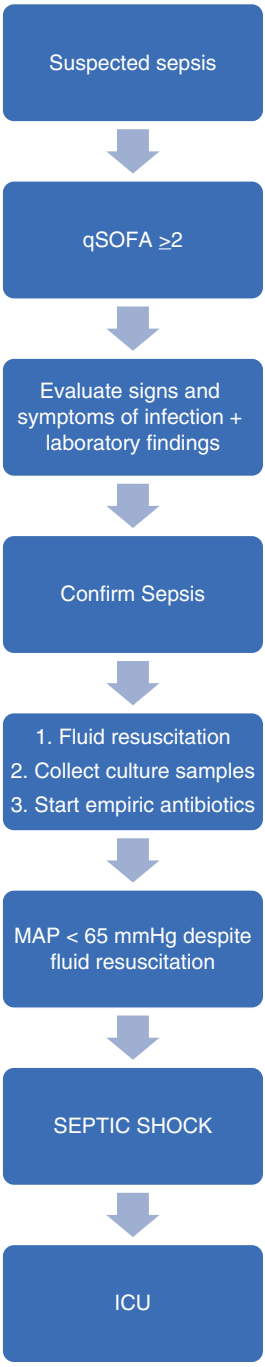

Fig. 20.1 Flowchart—identification and management of sepsis

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Hemorrhagic Patient

21

Samuele Colombo and Daniele Bissacco

21.1 Diagnosis and Management

It is of vital importance to early recognize and treat a hemorrhagic shock, in fact, death can occur within few hours of the onset of symptoms. The treatment consists in identifying the source of the bleeding as quickly as possible, stopping it with simple and effective maneuvers like direct manual compression or by means rapid surgical maneuvers (Damage Control Surgery, DCS) and restoring a correct volume of circulating blood. Hemorrhagic shock, especially when caused by occult sources of bleeding, is difficult to diagnose based on signs and symptoms. In fact, in most patients, hypotension is a late symptom that appears when there has been a loss of about 30% of the circulating volume. This occurs due to compensatory mechanisms such as peripheral vasoconstriction present mainly in young patients or pregnant women. Other signs and symptoms such as tachycardia, tachypnea, paleness, and altered consciousness (decrease in Glasgow GCS score) are also signs of late and impending shock. It is important to remember that tachycardia may not be present, even in the presence of significant blood loss, in patients taking beta blocker therapy. Table 21.1 shows the four shock classes based on vital parameters, very useful for quickly classifying the patient based on the severity of his condition.

In the initial evaluation of a patient who has a possible source of bleeding, as mentioned above, it is very important to identify the source of hemorrhage as quickly as possible. The most common sources of bleeding are the gastrointestinal

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Table 21.1 The American College of Surgeons Advanced Trauma Life Support (ATLS) hemorrhagic shock classification

Blood loss	Class I	Class II	Class III	Class IV
HR beats/min	<100	100–120	120–140	>140
Blood press (mmHg)	Normal	Normal	Decrease (<90)	Decrease
Pulse press	Normal	Narrowed	Narrowed	Narrowed
Resp rate	14–20	20–30	30–40	>35
GCS	Normal	Normal	Decrease	Decrease
Base deficit	0 to −2	−2 to −6	−6 to −10	<−10
Blood loss ml (%)	<750 (15)	750–1500 (15–30)	1500–2000 (30–40)	>2000 (>40)

tract manifesting as hematemesis, melaena, proctorrhagia, or vaginal bleeding. In these cases, the hemorrhage can clinically manifest itself with external bleeding after a few hours from the onset, consequently causing a significant loss of circulating volume before reaching the identification of the hemorrhagic source. Other areas of the body where the signs and symptoms of bleeding may be overlooked in the initial stages of clinical evaluation are cavities such as chest, abdomen, or pelvis and even more subtle is the retroperitoneum. In fact, large quantities of blood can be collected in these spaces, causing severe hemorrhagic shocks. In other cases, however, such as bleeding from the upper airways (epistaxis or hemoptoe) or the lesion of superficial vessels such as in the case of trauma, the source of bleeding is quickly detectable, favoring a more timely intervention.

21.2 Imaging

As previously mentioned, the chest, abdomen, and pelvis are common sources of bleeding in trauma patients. For this reason, these districts must be rapidly evaluated with first level examinations such as chest and pelvic radiograph and a targeted evaluation with ultrasound (Extended Focused Assessment with Sonography for Trauma, EFAST) [1]. Furthermore, ultrasound is very useful in identifying occult sources of bleeding even in non-traumatic patients. A diagnostic tool of extreme usefulness is the CT scan with contrast enhancement which, however, should be performed only as a second level examination in case the source of the bleeding remains unrecognized at the first evaluation and if the patient has normal hemodynamics or responsive to the fluid resuscitation. In case of massive hemorrhage, before performing second level diagnostic examinations, it's mandatory to obtain the stabilization of the hemodynamic parameters by resuscitations maneuvers, such as massive transfusion and surgical procedures (DCS) [2].

21.3 Lab Tests

The fastest laboratory test to evaluate the biochemical changes caused by bleeding is the blood gas analysis. The base deficit and lactate values are, in fact, indirect signs of peripheral oxygenation. Other laboratory tests useful for predicting the

need for a massive transfusion are hemoglobin (even if its decrease occurs later) and INR. Platelets counts and fibrinogen levels should also be evaluated and normalized. It is essential to detect coagulopathy and measure the rate of clot formation using tests such as the thromboelastogram to guide ongoing resuscitation of blood products by transfusion of coagulation factors. Electrolytes must also be measured both in the initial stages and during resuscitation with blood products so that they can be corrected early avoiding the onset of malignant arrhythmias [3, 4].

21.4 Treatment

Restoration of intravascular volume and rapid control of hemorrhage are the priorities for the management of bleeding. Protocols of intravascular volume restoration have evolved during the past several decades, finally getting back to a resuscitation approach that supports the use of plasma, platelets, and red blood cells [5]. Massive-transfusion protocols is required to treat patients presenting with shock class III or IV and any delay in their activation is associated with an increase in mortality. Recent evidences indicate that a ratio of plasma to platelets to red cells close to 1-1-1 is safe and reduces short-term mortality from hemorrhage due to trauma. For patients with hemorrhage from different causes than trauma, a ratio of platelets to red cells of more than 1:2 reduced mortality in the first 48 h [6]. The anticoagulant citrate is a usual component of all these blood products; it is rapidly metabolized by the liver in healthy people, while in patients with hemorrhagic shock receiving a large volume of blood product it can become toxic and cause life-threatening hypocalcemia and progressive coagulopathy [4]. For this reason it's very important an empirical calcium dosing and its correction during large-volume transfusions (e.g., 1 G of intravenous calcium chloride after transfusion of the first 4 units of any blood product). Despite a frequent use of isotonic crystalloid resuscitation in the early management of bleeding, these solutions have no effective therapeutic benefit other than a transient intravascular volume expansion. When isotonic crystalloid is overdosed, the risk of complications such as respiratory failure, compartment syndromes, and coagulopathy increases. So it is safer to limit crystalloid infusions to 3 L in the first 6 h after arrival in Emergency Department (ED) [7]. As in prehospital resuscitation, colloids or hypertonic saline have not proved of any benefit for the management of severe bleeding [8]. Procoagulant hemostatic products, such as activated recombinant factor VII, prothrombin complex concentrate, tranexamic acid, and fibrinogen, can be added to promote clot formation in patients with severe bleeding [9], especially in patients taking oral anticoagulants affected by hemophilia. For these classes of patients, the use of these agents is accepted despite it is considered off-label in healthy patients. However, the use of these drugs presents some non-negligible risks such as thrombotic complications, paradoxical hemorrhage, and multiorgan failure. For this reason it is important to refer to the protocols and guidelines in force in your department. Norepinephrine and other vasoactive drugs can be used to counteract hypotension in the early stages of shock.

Patients with acute hemorrhage caused by a traumatic event should be held less than 15–20 min in the ED for initial diagnosis and resuscitation so as to decrease the risk of death. Patients with hemorrhage in an extremity should be carried

immediately to the OR for vascular exploration using a tourniquet to limit blood loss in case of wounds far from joint areas such as armpits and groins. Avoid blind exploration of the wound in the emergency room, especially with surgical instruments like forceps or clamps due to reduction of the risk of iatrogenic vascular injuries. In patients with torso injuries, EFAST is of vital importance to identify the site of major bleeding so as to concentrate firstly DCS maneuvers (thoracostomy or Extraperitoneal Pelvic Packing) in that district. Patients with exclusive abdominal or pelvic bleeding from any source may benefit from a new technique called REBOA (Resuscitative Endovascular Balloon Occlusion of the Aorta) [10]. REBOA is used as a temporary bridge-system for performing second level diagnostic investigations and for transporting the patient to the operating or angiography room for definitive care by a temporary reduction of bleeding and normalization of vital parameters.

21.5 Conclusions

Patients with severe bleeding need adequate and on time hemostasis in order to increase their survival chances. A prolonged time to hemostasis has been linked to increased blood-transfusion requirements and increased mortality. The DCS maneuvers, described above, can be performed by trained personnel with experience in trauma, but those who are not specialist in surgery can also be fundamental in saving patient's life by implementing a few but precise precautions:

1. Recognize the signs and symptoms of bleeding early.
2. Stop bleeding immediately by manual pressure, direct soft tissue suturing, and tourniquet placement.
3. Start infusion of liquids early, preferring the use of blood components.
4. In case of noncompressible torso hemorrhage, call early a specialist capable of performing DCS maneuvers.
5. Once hemodynamic stability is achieved, transfer the patient as soon as possible to a first level trauma center if it is not possible to guarantee all the necessary care in your department.
6. Participate in training courses on bleeding management (such as the Stop the Bleeding campaign) or in more intensive courses such as Advanced Trauma Life Support (ATLS).

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The Polytrauma Patient

22

Matteo Marone and Ilenia D'Alessio

22.1 Introduction

A polytrauma patient is a person who experiences injuries to multiple body parts and organ systems as a result of an external physical force applied to that person [1].

There are two main types of trauma:

1. *Blunt*
2. *Penetrating*

In blunt trauma, also known as non-penetrating trauma, there is no perforation of the skin by foreign body. Classical examples of non-penetrating trauma are motor vehicle accidents. In contrast, in penetrating trauma there is a perforation of the skin and foreign objects can penetrate the patient's body. These objects can either enter and exit the body or they can be retained by the body itself. Classical examples of penetrating trauma are stab-wounds and gunshot-wounds [2, 3].

Polytrauma patients often experience a mixture of these two types of trauma, it is common to see patients after a car accident (blunt trauma) with penetrating injuries due to glasses or pieces of metal. Other types of injury include:

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- Thermal injuries
- Chemical injuries
- Blast injuries

Polytrauma patients are the patients whose management is the most difficult and time consuming for emergency physicians. It can and it may involve a multidisciplinary trauma team with many figures as: emergency medical doctors, paramedics, specialist doctors (surgeons, anaesthetists), nurses and residents in training. Usually are needed many instruments (e.g. CT scan, Doppler Ultrasound, Chest XR).

Every member of the team needs to be specifically trained to accomplish particular tasks like: intubation, IV line insertion, ICD (Intercostal drain) positioning and so on. It is mandatory that every member of the team communicate and refer his or her action to the figure that guides the team: the Trauma Leader. The trauma leader is a fundamental element of the team because it guides the resuscitation and takes decisions about the diagnostic and therapeutic steps to perform. He or she gives tasks to the other members of the team and his duties involve teaching and helping other members of the team if they are unable to carry out the task assigned to them.

Patients treatment is not limited to one centre but it can involve many structures and doctors and it is divided into three different phases:

1. Pre-hospital care
2. Acute care
3. Hospital care

When treating polytrauma patients it is important for every professional involved to remember that trauma patients have a tri-modal distribution of death, that means that mortality from trauma occurs mainly in the three peaks of Fig. 22.1 [4].

During the first peak death occurs within seconds or minutes from trauma and it is usually from non-treatable injuries like: aortic complete transection, cardiac rupture, high spine injuries, TBI (Traumatic Brain Injuries). There is no way to reduce these peak but primary preventive measures.

Death within the second peak occurs between minutes to hours from trauma and it can be prevented by the application of standard Advanced Trauma Life Support (ATLS®) [5] principles in order to diagnose and treat causes like: injuries of spleen and liver, haemopneumothorax, fractures, active bleeding and so on.

Death within the third peak occurs days to months after trauma and they can be prevented by the appropriate management of the patient in every stage of the pre-hospital and hospital stay. Examples of problems that lead to death during this peak are sepsis and organ failure.

As can be clearly seen, doctors cannot do anything to reduce the high of the first peak but they have a fundamental role in reducing the mortality in the second and third. This is possible having a good knowledge and applying ATLS principle to all patients that arrive in the emergency department with a polytrauma.

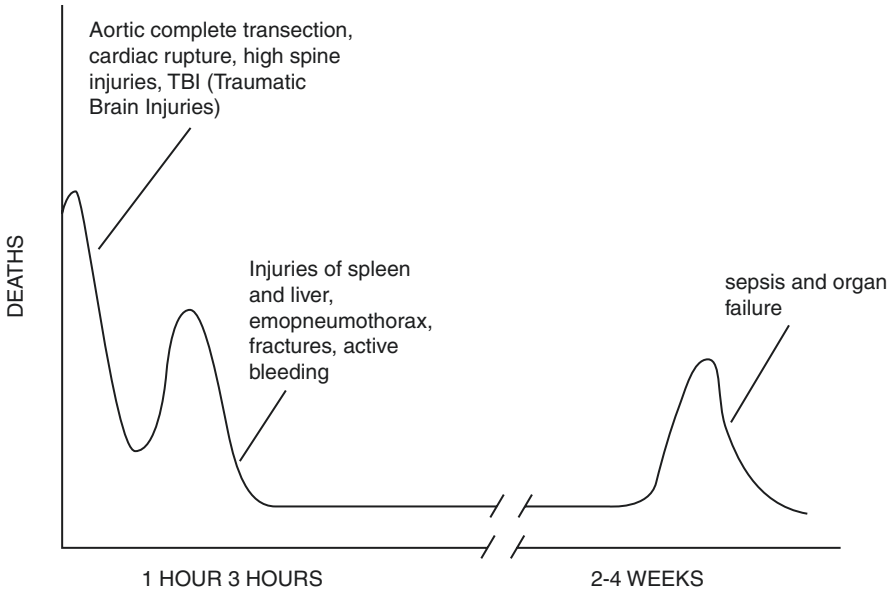

Fig. 22.1 Tri-modal distribution of causes of death in polytrauma patients

22.2 Signs and Symptoms

The main sign in polytrauma patients is *shock*, that is universally defined as the inadequate delivery of oxygen and nutrients and the inadequate removal of toxic metabolites to and from tissues that leads to the loss of cells' function and causes cell damage [6, 7].

During the initial phase of shock, cellular damages are reversible, however with the persistence of the shock status these damages can become irreversible.

There are different types of shock:

- *Haemorrhagic*
- *Non-haemorrhagic*

Haemorrhagic shock is the most frequent cause of shock in polytrauma patients. Until it is not proven differently doctors have to consider any polytrauma patients in haemorrhagic shock. Patients can bleed from different sites and an easy way to not miss any site is remembering the following phrase: *"blood on the floor and four more"*. This simple expression identifies the potential bleeding site of a polytrauma patient: "*Blood on the floor*" refers to the blood lost on the scene and during transport from penetrating and open wounds. "*Four more*" represents the body spaces where blood can collect in a closed trauma: chest, abdomen, pelvis and long bones fractures [6].

Non-haemorrhagic shock can be classified into:

- Cardiogenic
- Neurogenic—spinal
- Septic

It is extremely rare to have a septic shock in a recently traumatized patient, however, cardiogenic and spinal shock are not so rare. These types of shock have to be ruled out in particular conditions. If patients have signs of high velocity chest impact ECG and troponin level need to be checked. If patients have neurological problems with high suspicion of spinal cord injuries, a CT scan to check for medulla injuries needs to be performed [6].

Not every patient with shock is similar to one other. Depending on the gravity of shock, patients can experience different signs and symptoms.

It is important, however, to remember that the *heart rate* is the first parameter to change in response to blood loss and it's the first parameter to change when doctors infuse fluids in shock patients. Other parameters that can change are the urine output and the mental status. The last parameter that varies is the blood pressure.

As shown in the table below (Table 22.1), there are four different classes of shock depending on the percentage of blood loss.

It is important to recognize the appropriate class of shock in order to provide the optimal fluid therapy for the patient. In fact, in class I or II *crystalloids* (e.g. lactated Ringer's/Hartman's solution, acetate buffered solution, acetate and lactate buffered solution, acetate and gluconate buffered solution, 0.45% NaCl (hypotonic solution), 3% NaCl (hypertonic solution), 5% Dextrose in water) are a good option; however, in class III and IV blood and blood products are probably the best choice. In any case it is important in high volume trauma centres to use rotational thromboelastometry (ROTEM) in order to guide the infusion of blood and blood products [8].

Polytrauma patients always experience pain. Pain leads to tachycardia and discomfort. It is important to provide every patient with appropriate analgesia. Morphine IM (intramuscular) is often a good option because it is easily administered and absorbed.

Table 22.1 Classes of shock

	Class I	Class II	Class III	Class IV
% of blood loss	0–15	15–30	30–40	>40
Heart rate bpm	<100	100–120	120–140	>140
Blood pressure	Normal	Normal	Reduced	Reduced
Urine output (ml)	>30	20–30	5–15	None
Mental function	Anxious	Moderately anxious	Confused	Confused and lethargic

22.3 Diagnosis

The diagnosis of polytrauma patients is represented by the primary and secondary survey.

During the primary survey it is mandatory to remove the clothes from the patients in order to not miss any injuries. Every emergency physician should know ABCDE ATLS principles [5, 9].

22.3.1 Briefly

A—Airways: In this phase it is important to check for signs of airways obstruction like stridor, change in the voice level and so on. Immediate intubation is mandatory in case of airways obstruction and neck haematoma, as well as if signs of fume inhalation are present (burn nostril, carbon particle in the mouth). Succinylcholine and ketamine are the drugs of choice for intubation. If the arterial blood gases show high potassium, use Rocuronium and not Succinilcolina to avoid cardiac toxicity. During this phase it is useful to check for neck injuries and jugular distention that can be early signs of tension pneumothorax if monolateral or cardiac tamponade if bilateral.

B—Breathing: During this phase it is fundamental to check for chest movement and air entry on both sides of the thorax. If there is an open wound in the chest, it is mandatory to suture it or close it with a three-sided adhesive dressing and insert an ICD. In any case of reduced air entry in an hemithorax, it is mandatory to insert an ICD and this needs to be done before the X-ray confirmation of the problem. If the patient is in shock with reduced air entry on one side and with distended jugular veins, a tension pneumothorax is suspected and a chest decompression with a 18G needle in the second intercostal space needs to be performed immediately.

C—Circulation: During this phase, the physician has to monitor the patient with 12 lead ECG, a pressure cuff and an oximeter. The second step is to insert two large IV lines (16–18G) and start infusing warm fluid. Before infusing, blood samples need to be drawn for haematologic tests and blood chemistries (complete blood count [CBC] and differential, platelet count, electrolytes, serum creatinine, liver function tests, uric acid), in addition to toxicology and X-Match. If the patient is unstable, it is advisable to activate immediately the local massive transfusion protocol. Check for possible open wounds and apply compression or tourniquet by Foley catheter. During this phase the referring physician can apply pelvic binder and do Focused Assessment with Sonography for Trauma (eco-FAST) to check for fluid in the abdomen.

D—Disability: During this phase it is important to evaluate the Glasgow Coma Scale and the gross body movements in order to detect possible nerve injuries. The physician needs to evaluate the pupils and their light reactivity and reflexes. Careful attention needs to be put in looking for signs of paraparesis or quadriplegia.

E—Exposure: During this phase it is important to log-roll the patient protecting the spine in order to check for other possible injuries and expose all the body.

The secondary survey follows the primary survey and it is performed on a stabilized patient. It represents a head to toe examination comprehensive of full neurological examination, chest and abdominal examination and limb examination. The only two XR needed during the primary survey are a chest XR and a pelvic XR. The pelvic imaging is performed because it is really difficult to rule out pelvic fracture clinically and the chest imaging is performed to check the appropriate position of all the lines and tubes that the physician inserted during the resuscitation.

22.4 Therapy

Polytrauma patients, as we said, are shocked. In order to improve this status, it is mandatory that every trauma patient is treated with 100% 02 facemask. This measure will increase the peripheral tissue oxygenation.

It is important and mandatory to keep the patient warm in order to avoid the development of the lethal triad: hypothermia, hypercoagulability, acidosis. This can be achieved using thermal blanket, heater devices and devices that allow the infusion of warm fluid into the body.

In all the polytrauma patient it is mandatory to consider the C spine injured, if not proven otherwise. In order to do so, the best way to clear it is using a CT scan, that is more sensitive and specific than a plane XR. Until the CT scan is done, it is important to maintain the neck immobilized with a C collar in situ.

In polytrauma patients with signs of shock, it is important to stop the bleeding to stabilize the patient.

As previously stated, blood can come from five different sources:

- Open wound
- Bone fractures
- Pelvic fractures
- Chest
- Abdomen

In case of bone fracture with misalignment of the limb it is important to put the limb on tractions and to reduce the fracture. Reducing the fracture will recreate a sort of continuity in the bone reducing in this way the space where the blood can collect. Traction is also important because it avoids the fracture to lose its alignment.

In case of pelvic fracture, it is important to close the pelvic ring in order to reduce the space where the patient can bleed. There are different and effective ways to do so. Special devices are called T-pods or pelvic binders. These devices are placed around the hip of the trauma patient and allow the closure of the pelvic ring. The same principle can be applied in rural setting using a towel to close the pelvis. There

are two major currents of thought about pelvic fractures: some surgeons prefer to apply pelvic binder to all trauma patients and then clear the pelvis with a pelvic XR; others prefer to perform a manual examination of the pelvis and apply the binder only in case of pelvic instability [10, 11].

If the pelvic bleeding persists, despite binder, probably the cause is arterial, in this case interventional radiology can be involved to perform an embolization. The use of Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) in zone III (below renal arteries) can be a good option in these patients.

Considering the open wound their problem is that blood can come out from them, especially if there is a vascular structure injuries underneath. Manual compression is the most efficient way to stop the bleeding. However, this prevents one member of the team from performing other tasks. If the open wound is on a limb, the bleeding can be stopped using a tourniquet. This device can be used to exclude the specific limb from the systemic circulation stopping arterial and venous flow. Tourniquets need to be put in areas of the limb with only one bone underneath like arm and tight in order to achieve a complete vascular occlusion. It is mandatory, after the tourniquet is placed, to write on the patient the time of application. In fact, one of the main tourniquet drawbacks is that they create a complete ischaemia of the limb whom they are applied to. This means that the tourniquet needs to be removed within 6 h from his application. If we consider wounds in non-compressible areas of the body like the neck or the supraclavicular area, haemostasis can be achieved by inflating Foley catheter in the wound. The balloon of the catheter will compress the artery and the vein and will allow a temporary stop of the haemorrhage. More than one Foley catheter can be placed in the same spot to stop the bleeding, if necessary [12].

Considering the abdomen, its management is the priority of the trauma surgeon on call. Emergency room laparotomies are not advisable for the lack of proper instrumentation and illumination. If surgical control of the bleeding needs to be achieved immediately the best way to do so is performing a resuscitative thoracotomy and cross clamp the aorta. Nowadays, with endovascular technique, an aortic balloon occlusion can be performed using a technique called REBOA. In this situation a balloon is inflated in the aorta (usually zone I, above visceral arteries) in order to exclude the abdomen from the aortic arterial blood flow to stabilize the patient [13].

Considering the chest, haemothorax and suspected haemothorax need to be treated using ICD. The ICD output guides the physician in the following step. An output of more than 1200 ml of blood is associated with haemodynamic instability and indicates the necessity of performing a resuscitative thoracotomy. With this procedure the chest is open passing through the fourth intercostal space in the emergency department. This procedure allows the surgeon to control the bleeding directly and to cross clamp the aorta in case of necessity. In responder patient a blood loss of more than 200 ml every hours or more than 1200 ml indicates the necessity of a thoracotomy in OR (operating room) [14].

22.5 Diagnostic Algorithm (Fig. 22.2)

Dealing with a trauma patient after the initial resuscitation can be challenging.

Initial resuscitation helps identify the main problems that can lead a patient to death. However, once the initial assessment is over the doctor in charge has to decide what to do. The decision depends on the haemodynamic status of the patients. After administering the first litre of fluid (crystalloid) to the patient, the trauma leader has to assess the patient's status.

There are three possible scenarios that he can face (see Table 22.2):

- Responder patient
- Transient responder patient
- Non-responder patient

To identify in which class the patient is, the physician has to evaluate the clinical parameter, especially the heart rate (HR), the patient general condition (from pale to pink) and most importantly the change in arterial blood gases (ABG) parameters of lactates and base excess (BE).

If after the initial resuscitation the patient HR returns to normal values, the lactates and the BE on ABG improve, probably this patient is a responder. In this case eventually the bleeding stopped by itself. In this scenario the patient needs to be referred to the trauma surgeon in order to be admitted in a trauma ward and continue with clinical observation and further management.

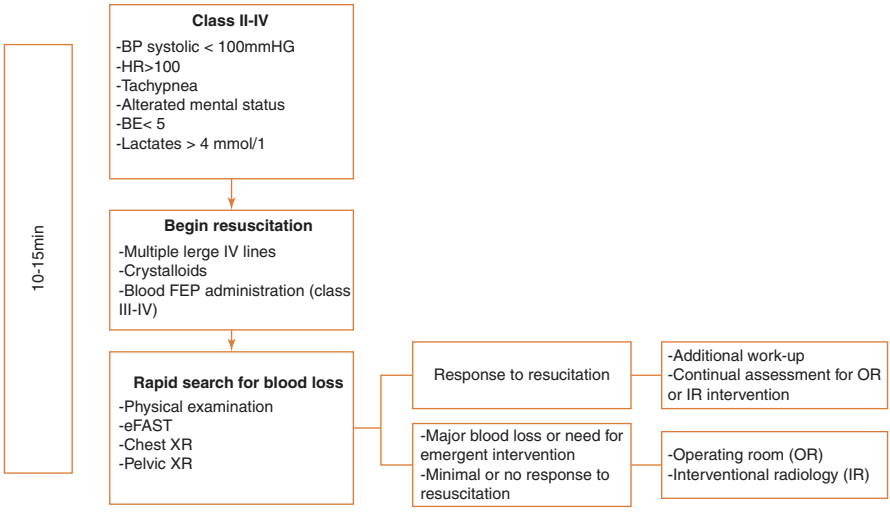

Fig. 22.2 Diagnostic algorithm

Table 22.2 Responses to initial fluid resuscitation

	Responder	Transient responder	Non-responder
Vital signs	Return normal	Improve and then deteriorate after infusion	Do not improve
Blood loss	Minimal	Moderate	Severe
Type of fluid for resus	Crystalloid	Blood X matched	Blood massive transfusion
Need for intervention	Not likely	Interventional radiology	Urgent OR

If after the initial resuscitation the patient HR, lactates and the BE on ABG improve only for a short time during the fluid administration and after stopping the infusion they deteriorate, probably this patient is a transient responder. In this case the bleeding is not massive but it is still ongoing. In this scenario the best option is to refer the patient to the trauma surgeon for further management and it is also useful to involve the interventional radiologist on call for possible embolization or angiosuit procedures.

If after the initial resuscitation the patient HR, lactates and BE do not improve, this patient is a non-responder. In this case the bleeding is massive and still ongoing. Urgent referral to trauma surgeon is mandatory in order to make a definitive plan to stop the bleeding.

Key Messages

- Follow ABCDE and ATLS principle every time.
- Consider physiology, anatomy and mechanism of injuries.
- Check for signs of shock and consider every trauma patient in shock if not proved otherwise.
- Assess the patient continuously, prevent the complications and sort the problems out as they present to you.
- Trauma patients need fluids and blood, do not be afraid of infusing the patient.
- Blood on the floor and 4 more.
- Stabilize the patients, intubate, ICD, pelvic binding, limb traction.
- Follow diagnostic algorithm, in trauma there is no space for inventions or new techniques it is a matter of life or death.

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