Pathophysiology

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Contents

Objectives

- Characterize the pathophysiological processes in abdominal emergencies.
- Categorize these processes into corresponding groups.
- Outline the systemic and local consequences of these processes.
- Link the consequences into the development of symptoms and signs.
- Describe the primary aim of therapy in different pathophysiological conditions.

4.1 Introduction

Acute disease processes in the abdomen, whatever the cause, manifest in the vast majority of cases in a limited number of ways. These manifestations can be grouped according to the principal pathophysiological process and used as a guiding principle toward both diagnosis and therapy. Regardless of the organ or organ system involved, the clinical presentation of a specific pathological process in the abdomen is constant. Knowing the usual presentation of a disease, i.e., appendicitis, ruptured ectopic pregnancy, pelvic inflammatory disease, etc., allows early diagnosis, expeditious formulation of the principal goal of treatment, as well as understanding the natural course of the process if not interrupted by intervention that in most cases is surgical.

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4.2 Hemorrhage

Acute extravasation of blood can:

- Occur freely into the abdominal cavity (e.g., ruptured ectopic pregnancy or liver adenoma)
- Be contained and confined to the retroperitoneal space (ruptured abdominal aortic aneurysm) or specific pathological cavity (bleeding pancreatic pseudocyst)
- Bleed into a hollow organ such as the gastrointestinal (bleeding peptic ulcer), biliary, or urinary tract (renal tumor).

Depending on the amount of blood extravasated and speed of extravasation, the symptoms are dominated either by local irritation or compression caused by the blood and blood clot or by systemic manifestations of *acute hypovolemia* that, if untreated, can result in *exsanguination* of the patient.

If the bleeding stops spontaneously, the extravasated blood or clot can cause delayed problems in form of:

- Compression on adjacent organs
- Obstruction of hollow organs (urinary bladder tamponade)
- Infected hematoma and subsequent abscess formation
- Recurrent bleeding (at high risk if the underlying pathological process is not treated)

The main aim of treatment is to stop the bleeding, utilizing one or more of the following interventions:

- Operation
- Endoscopic procedure
- Interventional radiology (angioembolization)

The urgency of treatment depends on the rate of bleeding. Hypovolemic shock is corrected with intravenous volume expansion avoiding complete normotension in uncontrolled hemorrhage, thus reducing the rate of bleeding and decreasing the risk of recurrent bleeding after spontaneous hemostasis. Extravasated blood is replaced with blood transfusion including clotting factors to maximize the chance of hemostasis that in most cases requires mechanical intervention to seal off the bleeding vessel.

4.3 Contamination

The sources of bacterial contamination in the abdominal cavity include:

- Perforation of a hollow organ containing normal bacteria flora, such as the gastrointestinal tract (most common source of contamination)
- Bacterial translocation through gangrenous intestine (gangrenous appendicitis, ischemic or gangrenous loop of bowel) or other hollow organ wall (gangrenous cholecystitis)
- Previously contained abscess perforating into the free intraperitoneal space

Whether caused by translocation or frank perforation, the bacterial contamination will induce both a *local and systemic inflammatory response*.

Depending on the size and location of the perforation and the ability of the adjacent organs and the greater omentum to seal off the perforation, the condition can progress to:

- Generalized secondary peritonitis
- Walled-off inflammatory process followed either by resolution or formation of a mature abscess

Occasionally, the bacterial contamination is preceded by chemical contamination (e.g., perforated peptic ulcer) causing the initial reaction and symptoms, and the effects of bacterial contamination will manifest within the next few hours.

The aims of treatment

- Control the source of contamination
- Correct the disturbed homeostasis caused by the systemic inflammatory reaction

Source control can be achieved by

- Removal of the inflamed organ before or after perforation (acute appendicitis, strangulated bowel loop, acute cholecystitis)

- Surgical closure of the perforation (perforated peptic ulcer)
- Diversion of the intestinal contents with entero- or colostomy, if complete source control in the gastrointestinal tract cannot be reliably achieved or it is not safe to perform primary closure or anastomosis (e.g., the Hartmann' procedure for perforated sigmoid diverticulitis)
- Drainage of the content outside the body with aptly placed drains to create a "controlled fistula," such as in delayed perforation of the duodenum with no chance of reliable primary closure

4.4 Obstruction

A *mechanical obstruction of a hollow organ* leads to a distinct clinical picture dominated by colicky pain when the body tries to overcome the obstruction by enhanced peristaltic contractions. The cause of the obstruction can be intraluminal or caused by external compression, volvulus, or kinking. The progression and complications caused by the obstruction depend on the organ system involved.

Gastrointestinal tract
- Obstruction caused by peritoneal adhesions or bands
- Obstructed hernia
- Twists (volvulus)
- Tumors (especially in the colon)

The obstruction will cause proximal dilatation, ischemic necrosis, and eventual perforation, if the obstruction is not relieved. The risk of perforation increases with the diameter of the dilated bowel thus causing the cecum to be the most likely perforation site in distal colonic obstruction, especially if the ileocecal valve is competent. Temporary relief can be achieved spontaneously (vomiting, incompetent ileocecal valve) or intentionally (nasogastric tube), and sometimes the obstruction may resolve spontaneously, such as in adhesive small bowel obstruction.

Colonic pseudo-obstruction (Ogilvie's syndrome) is a nonmechanical dilatation of the colon that often requires some form of mechanical (endoscopic) or pharmacological (neostigmine) intervention to prevent overdilatation of the colon.

Biliary tract
- Obstruction in the main hepatic or common bile duct (usually caused by stone or tumor) will result in obstructive jaundice.
- If not relieved, a secondary liver injury will follow.
- An obstructed cystic duct will cause dilatation of the gallbladder with ensuing acute cholecystitis, perforation, or empyema.

Urinary tract
- Stone
- Tumor (including prostatic hyperplasia)
- Blood clot

Urinary obstruction will cause proximal dilatation of the urinary tract, renal insufficiency (if bilateral), and eventually loss of a kidney, especially if the obstruction is prolonged or associated with an infection.

Aims of treatment of hollow organ obstruction:

- Relieve the obstruction by correcting or removing the cause.
- Assess the viability of the obstructed organs.
- In urgent or complex situations, temporary relief of the obstruction by proximal diversion followed later by definitive treatment.

Examples of proximal diversion:

- Proximal colostomy in obstructive distal colon or rectal cancer
- Percutaneous transhepatic cholecystostomy or biliary drainage
- Suprapubic cystostomy

Internal drainage utilizing endoscopic placement of stents can also be a definitive procedure, as in patients with advanced cancer or chronic pancreatitis. In most cases, however, *surgical removal of the cause* of obstruction is the definitive treatment with best long-term effect.

4.5 Ischemia

A complete or partial occlusion of a visceral artery leads to end-organ ischemia unless sufficient collateral circulation exists. An acute obstruction is usually caused by thrombosis or an embolus, but occasionally an acute low flow state without obvious localized vascular obstruction can have the same effect.

Depending on the organ involved, the symptoms and localizing signs manifest in different areas of the abdomen. Ischemic pain is usually abrupt, severe, and sometimes poorly localized in the initial stage, and the localizing peritoneal irritation might not be yet present.

Vascular inflow occlusion of the solid abdominal organs (*liver, spleen, kidneys*) will result in ischemic necrosis if revascularization is delayed for more than a few hours.

- Warm ischemia is tolerated poorly by the kidneys, whereas in the liver, either the hepatic artery or the portal vein (if one of them is intact) usually provide sufficient oxygen to prevent cellular necrosis.
- Occlusion of a branch of the main artery can lead to partial infarction of the end organ, such as the spleen or kidney.

Gastrointestinal tract

- Acute occlusion of the *superior mesenteric artery* leads to massive necrosis of most of the small bowel and the right hemicolon.
- Thrombosis is usually more proximal than an embolus where the first jejunal branches might ensure the viability of a part of the proximal jejunum.
- Occlusion of the *celiac axis or the inferior mesenteric artery* seldom has dramatic effects.
- Decreased flow to the left hemicolon can lead to *ischemic colitis.*
- Thrombosis of the *superior mesenteric vein* leads to venous congestion and bowel edema with less clearly demarcated areas than arterial thrombosis. In most cases, it can be managed nonoperatively (anticoagulation) since the risk of necrosis is low.
- *Bowel strangulation* is a special form of ischemia where vascular occlusion is preceded by mechanical closed loop obstruction of the bowel caused by adhesions or incarcerated external hernia. If not corrected in time, the strangulated bowel loop will become necrotic and perforates.

4.6 Toxic Injury

Ingested drugs, toxins, alcohol, or corrosive agents cause a wide range of acute emergency surgical problems including:

- Alcohol-induced acute pancreatitis
- Toxic hepatitis
- Corrosive injury to the esophagus, stomach, and less commonly, duodenum
- Inflammation of a solid organ can lead to necrosis (liver, pancreas) and subsequent infection (infected peripancreatic necrosis) or decrease or loss of function (endo- and exocrine pancreatic insufficiency)
- In hollow organs, full-thickness necrosis usually leads to perforation and generalized infection (mediastinitis, peritonitis)

The primary aim of surgical treatment is to *manage the complications* caused by the toxic injuries (pancreatic necrosectomy, resection of necrotic esophagus) and subsequent *restoration of function* that can include complex reconstructive procedures or organ transplantation.

4.7 Abdominal Compartment Syndrome (ACS)

Increased intra-abdominal pressure (IAP) can be the result of

- Space-occupying process in the abdomen (primary ACS)
- Extensive fluid resuscitation (sepsis, burns) leading to visceral edema (secondary ACS)

Abdominal compartment syndrome is defined as sustained intra-abdominal hypertension (>20 mmHg) combined with evidence of new end-organ dysfunction.

Increased IAP will cause dysfunction of most organ systems within and outside the abdomen (most commonly in the renal, gastrointestinal, and respiratory systems) and can, if untreated, lead to multiple organ dysfunction syndrome and death.

While clinical recognition can be difficult, the IAP is easily measured through a urinary bladder catheter.

The main aim of treatment is to lower the IAP.

- Initially with *conservative* methods by decreasing intra-abdominal contents (nasogastric and rectal tubes, percutaneous drainage of ascites) and increasing abdominal wall compliance (short-term muscle relaxants, optimization of hemodynamics, removing constrictive bandages).
- If intra-abdominal hypertension persists, prompt *surgical decompression is necessary, leaving the abdomen open.*

The management of the ensuing open abdomen aims for delayed fascial closure when the principal cause of the ACS has been treated. If fascial closure is not possible, a planned hernia approach (usually with split-thickness skin grafting) is instituted and followed by delayed abdominal wall reconstruction procedure performed 6–12 months later. If the abdominal fascia cannot be closed, coverage with native tissue is always preferred, i.e., skin only closure.

Pitfalls

- Failure to recognize early systemic signs of hemorrhage
- Inability to achieve reliable source control of contamination
- Incomplete or delayed relief of hollow organ obstruction
- Delayed recognition and treatment of intestinal ischemia
- Ignoring the possibility of abdominal compartment syndrome

4.8 Summary

Acute abdominal emergencies present in a few distinct forms that have specific local and systemic manifestations, but with consequences that have many similarities regardless of the site or organ of the lesion. Massive hemorrhage leads to hypovolemic shock and exsanguination if the bleeding is not stopped. Bacterial contamination of the peritoneal cavity can lead to generalized peritonitis and septic shock unless limited by the body or source control achieved by surgical means. Hollow organ obstruction leads to proximal dilatation that often requires temporary or definitive measures to prevent permanent organ damage or perforation. Acute ischemia can cause irreversible damage to the organ involved unless revascularization can be performed rapidly. Finally, the complications caused by ingestion of toxic or corrosive agents often require surgical intervention.

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