Abdominal Pain

P. Poitras, A. Archambault, and V. Marchand

Contents

P. Poitras (ed.), *The Digestive System: From Basic Sciences to Clinical Practice*,
https://doi.org/10.1007/978-3-030-98381-9_16

16.1 General Considerations

Pain is defined as "an unpleasant sensory and affective experience associated with tissue damage…." Pain is a subjective sensation, variable according to individuals, time, etc.

The pathophysiology of pain involves (a) a "lesion" of a peripheral tissue, (b) activating a neurological signal afferent from the periphery to the brain, (c) where it generates a sensation of pain. Different steps are therefore involved (as summarized in Fig. 16.1).

1. **Pain transduction**. The transduction of a tissue "lesion" (e.g., distended gut) to a sensory nerve is the first step in the pain pathway where a physical phenomenon (e.g., mechanical, thermal, chemical "trauma," etc.) is converted into a sensory neurological transmission. Various mediators are involved here: the "traumatized" tissue produces prostaglandins, leukotrienes, protons, etc.; circulating anti-inflammatory agents such as platelets or leukocytes secrete bradykinin, histamine, interleukin, etc.; and in nerve endings, substance P is activated. Transduction is a step that can be modulated by certain drugs such as NSAIDs, corticoids, local anesthetics, etc., acting on these mediators.

At the intestinal level, the cutting or burning of the organ is painless (which allows, among other things, the therapeutic gestures of fulguration, electrocoagulation, etc. performed in digestive endoscopy and not perceived by the patient). The distension of the organ, the traction of its mesenteric attachments, the muscle contraction, and the tissue necrosis are sources of pain transduction.

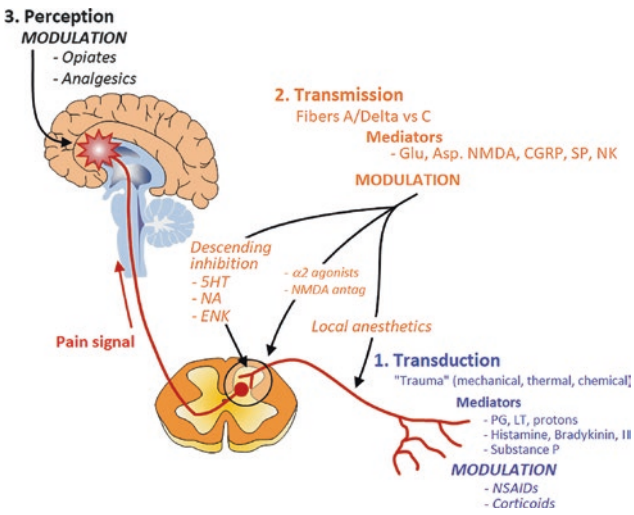

Fig. 16.1 Pain transmission from the peripheral organ to the brain in three steps: (1) transduction, (2) transmission, and (3) perception of the pain signal

2. **Pain transmission.** The transmission of the painful process from the "traumatized" organ to the brain takes place via nerve impulses, mainly from the sympathetic system. The primary nerve (also called first-order or first-class nerve) travels from the organ to the spinal cord through the celiac or mesenteric ganglia in the abdomen; at the level of the spinal cord, in the posterior horn, it meets a second-order neuron which will join the spinothalamic bundle (thermo-algesic sensibility) on its way to the brain.

The neurological fibers transmitting the afferent influx are of two types:

- The A/Delta fibers are myelinated, densely distributed on the organ, and capable of rapid influx transmission. The A/Delta fibers are involved in the somatic pain process (e.g., from the skin, musculoskeletal apparatus, etc.); they allow a precise perception of pain (e.g., a sensation of stinging can be differentiated from a sensation of burning and localized to a very precise site on the body). In the abdomen, A/Delta fibers are rare and limited to the parietal peritoneum and to the capsule of solid abdominal organs.
- The C fibers are non-myelinated, are distributed in limited quantities over their innervated organs, and provide slow impulse transmission. Pain sensation mediated by C fibers is often vague and poorly localized. C fibers are predominant in the abdomen and are responsible for the innervation of the digestive viscera. This explains why visceral pain, as opposed to somatic pain, is often vague and imprecise. For example, in appendicitis (see ▶ Chap. 4), the initial inflammation occurring in the viscera and its immediate vicinity is transmitted by the C fibers to often generate a rather vague, dull discomfort, felt in the periumbilical region before the inflammation ultimately reaches the parietal peritoneum to then activate the A/Delta sensory fibers allowing the precise localization of the pain in the right iliac fossa. The physiology of visceral pain explains the difficulty for patients to report their symptoms and the difficulty for physicians to diagnose them.

Abdominal pain afferents travel mainly with sympathetic fibers (the esophagus and pelvic organs are innervated by vagal or sacral parasympathetic fibers). While the somatic afferents (from the skin, muscles, etc.) give rise to well-established innervation segments that can be recognized according to the dermatomes (Fig. 16.2), the visceral afferents have a much less precise distribution. Afferents from the stomach, liver, and pancreas pass through the celiac ganglion before entering the spinal cord via the paraspinal ganglia located from T5 to

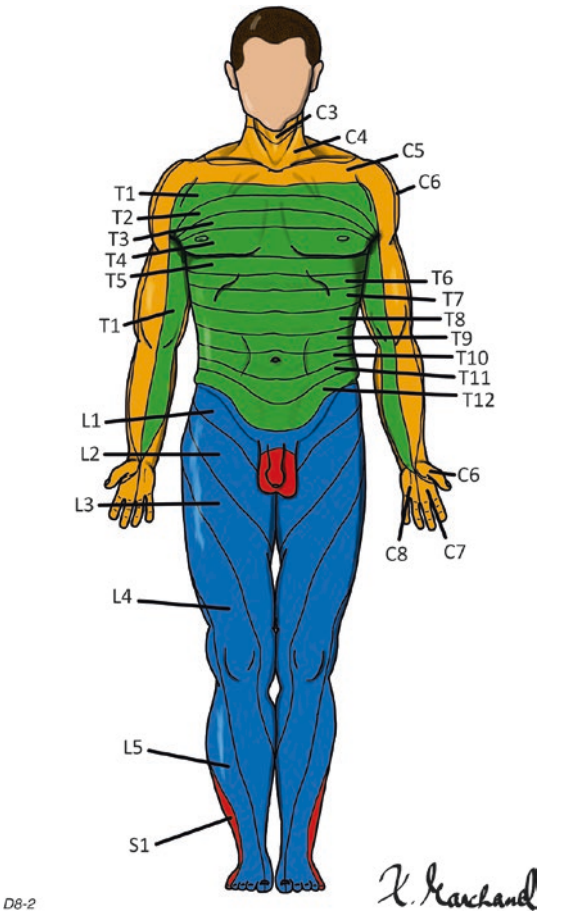

Fig. 16.2 Somatic pain is felt in specific territories corresponding to the dermatomes shown here. Visceral pain is less precise

T9; afferents from the intestine and colon are travelling via the celiac, superior mesenteric, or inferior mesenteric ganglia to the T10 to L2 spinal segments; the distal colon and pelvic organs may use parasympathetic fibers from S2 to S4. The extensive, overlapping segmentation of visceral afferents between different segments explains the relatively unclear parietal localization of a digestive pain. Broadly speaking, the travel of an abdominal pain follows the embryological development of the viscera, and pain originating from the "foregut" organs (stomach, bile ducts, pancreas) is felt in the epigastric region, that from the "midgut" viscera (small intestine) in the periumbilical region, and that from the "hindgut" organs (distal colon) in the hypogastrium.

Visceral pain can be *referred* to the corresponding somatic territory. Thus, the visceral C fibers innervating the diaphragm are found in spinal segments C3, C4, and C5 where the somatic A/Delta fibers innervating the shoulder converge; this explains the shoulder pain experienced during diaphragmatic irritation (e.g., by a suprahepatic abscess, post-laparoscopy air, etc.).

Visceral pain can be *radiating*, i.e., spread to other areas, such as pancreatitis, which causes epigastric pain radiating to the back, or renal colic, which causes pain in the flank or back and radiates to the genitals.

The neurological transmission of a pain impulse involves various neurochemical mediators including glutamate (Glu), aspartate (Asp), NMDA (N-methyl-D aspartate), CGRP (calcitonin gene-related peptide), somatostatin, substance P (SP), neurokinins (NK), etc. The transmission of peripheral nerve impulses can be blocked by local anesthetics (administered to the nerves, plexuses, epidural space, or subarachnoid space). This nerve transmission can also be modulated by pharmacological agents such as alpha-2 agonists (e.g., clonidine) and NMDA antagonists (ketamine) or by descending inhibitory fibers coming from the brain and releasing enkephalins, serotonin, noradrenaline, etc.

3. **Pain perception** takes place in the brain. This perception can be modulated by opiate drugs (morphine, etc.), general anesthetics, etc. The spinothalamic network, on its way to the brain, receives messages from the thalamic and limbic systems, and thus pain perception is also influenced by anxiety, emotions, painful memories, etc.

16.2 Clinical Approach to Abdominal Pain

The diagnosis of abdominal pain is difficult due to the visceral neurological system that is less efficient than the somatic system to express pain conditions as discussed above and due to the multiple conditions that need to be considered in the differential diagnosis. A detailed and focused history as well as a careful abdominal examination is essential.

1. ***Location of pain.*** The location of the pain is a primary diagnostic clue to be obtained. Figure 16.3 schematically describes the causes to be considered according to the location of the pain reported by the patient.
2. ***Radiation of pain*** may correspond to classic patterns described in Table 16.1.
3. ***Type of pain***: Burning pain is often caused by an acid peptic lesion (stomach/duodenum ulcer, etc.). Cramps are felt when intestinal or colonic muscle contractions are increased to overcome an obstacle (e.g., intestinal obstruction) or are stimulated by an "irritation" (e.g., viral gastroenteritis). Sudden, explosive pain suggests an abdominal emergency such as a visceral perforation, acute intestinal ischemia, etc. Pain of increasing intensity over 1–2 hours is common in cholecystitis, pancreatitis, or intestinal

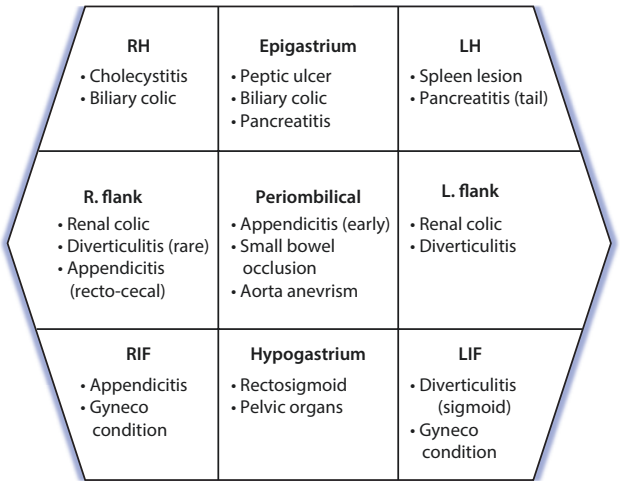

Fig. 16.3 Origin of abdominal pain according to its location in the right hypochondrium (RH), left hypochondrium (LH), right iliac fossa (RIF), etc.

Table 16.1 Radiating/referred pain

Pain	Radiation	Origin to be suspected
Epigastrium	Transfixing to the back	Pancreas
Epigastrium	Right hypochondrium	Gallbladder
Epigastrium	Left hypochondrium	Caudal pancreas
Flank	Groin, genitals	Renal colic
Right hypochondrium	Right shoulder	Subdiaphragm (abscess, air)

obstruction; vague, dull, progressive pain over a few hours is often found in appendicitis, incarcerated hernia, and diverticulitis.

4. ***Associated phenomena*** should be sought. Failure to pass stools and gas is a sign of intestinal ileus (by mechanical obstruction or reflex intestinal paralysis). Relief by bending over in a curled up position is common in pancreatitis. While the patient suffering from peritonitis is usually calm and prostrate, the subject with a renal colic may be rather agitated and inclined to walk.
5. ***Examination*** of the abdomen is a critical step in the assessment of abdominal pain. As described in Table 16.2, examination of the abdomen is based on visual inspection, auscultation, percussion, and palpation of the abdomen.

Table 16.2 Abdominal examination: important signs

Inspection	↑ abdominal volume: fluid = ascites; air = bowel obstruction
	Hernia (wall/inguinal areas) = potential incarceration site
	Surgical scar = potential intestinal occlusion by adhesions
Auscultation	Bowel sounds ↑ = bowel obstruction
	Bowel sounds ↓ = paralytic ileus (peritonitis, etc.)
Percussion	Tympanic = air (bowel obstruction) vs dull = fluid (ascites)
	Liver dullness absent = pneumoperitoneum due to visceral perforation
Palpation	Abdominal guarding (abdominal wall contraction during palpation)
	Voluntary (protection against pain) vs involuntary rigidity (peritonitis)
	Localized (local inflammation) vs diffuse (generalized peritonitis)
	Rebound pain (pain evoked by sudden release of abdominal palpation) = peritonitis
	Localized (Blumberg's sign) or at distance (Rovsing's sign)
	Specific pain sites
	McBurney's point (right lower quadrant = appendicitis)
	Murphy's sign (right upper quadrant: pain on inspiration) = cholecystitis
	Psoas sign (pain at thigh elevation) = psoas irritation (abscess, etc.)
	Obturator sign (pain during hip internal rotation) = obturator muscle irritation
	Rectal exam: pelvic pain = pelvic inflammation/abscess

16.3 Diagnostic Strategies in Abdominal Pain

The management of acute abdominal pain is primarily aimed at detecting severe conditions that require "urgent" surgical treatment.

1. **Laboratory tests**. In addition to the clinical examination, various laboratory tests (serum tests, X-rays, etc.) are obtained to guide the diagnosis. The blood count is used to check for a low hemoglobin (which could be due to internal bleeding) or elevated

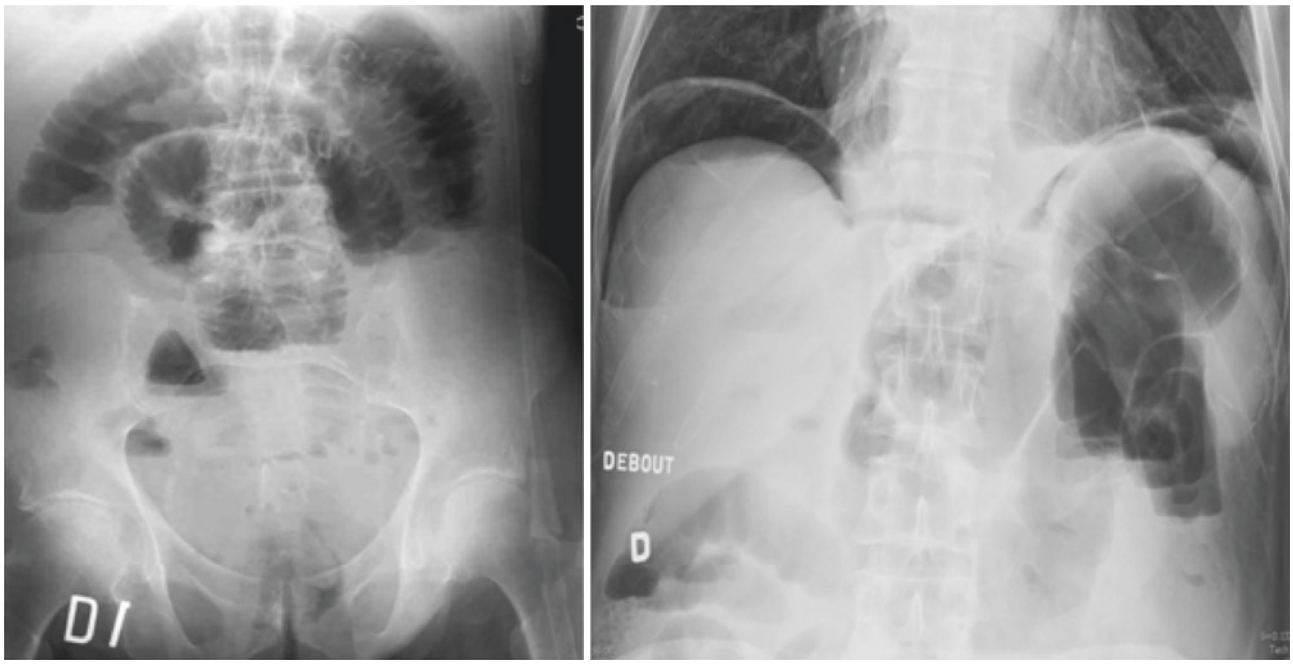

Fig. 16.4 Plain X-rays (flat plates) of the abdomen: Left: distension of small intestinal loops occluded by a scar adhesion. Right: free air under the diaphragm due to a perforated duodenal ulcer

leukocytes (suggestive of severe inflammation). Pancreatic enzymes (amylase, lipase) are elevated in pancreatitis (but also in other conditions). Hepatic enzymes (ALT, AST, alkaline phosphatases) are elevated in liver or biliary diseases. Lactic acid increases (unfortunately late) in the case of intestinal ischemia. A simple plain X-ray of the abdomen can reveal signs of visceral perforation (free air under the diaphragm) or intestinal obstruction (distended intestinal loops, gas-fluid levels, etc.; see Fig. 16.4).

2. **Surveillance**. Classically, the diagnostic strategy for acute unexplained abdominal pain was based on repeated clinical (and biological) evaluation (q 1–2 h) to monitor the evolution of the pain and the development of obvious or very significant signs for a precise diagnosis that would allow a specific therapeutic (often surgical) management. For example, appendicitis in its initial stage is most often manifested by a vague periumbilical discomfort (visceral pain mediated by C fibers) before the irritation of the parietal peritoneum (and activation of A/Delta fibers) allows the typical perception of pain in the right iliac fossa. Repeated physical examination allows early detection of signs of localized peritonitis and thus to decide on surgical intervention before more serious complications such as generalized fecal peritonitis may occur.
3. **Imaging.** In the last 20 years, however, abdominal imaging techniques have revolutionized the management of patients with acute abdominal pain. Ultrasound is crucial in gallbladder diseases and, depending on local expertise, may be very useful in other conditions such as appendicitis, diverticulitis, etc. CT scan is the most commonly used technique to elucidate abdominal pain investigated in the emergency room. It allows early identification of lesions that previously were, unfortunately, diagnosed only at an advanced (and complicated) stage of their evolution (e.g., ruptured appendix, intestinal ischemia with necrosis and perforation, etc.). It can also clarify the disease condition (e.g., extent of pancreatic damage, cause of intestinal obstruction, etc.) to guide the treatment procedures.

16.4 Therapeutic Strategies for Abdominal Pain

Specific treatment Treatment of the underlying cause of the abdominal pain (e.g., appendectomy if appendicitis, antibiotics if diverticulitis, etc.) will of course be undertaken as soon as possible.

General treatment will include:

- *Support treatment* to correct and maintain the vital parameters (O_2 saturation, hemodynamic stability, hydroelectrolytic equilibrium, etc.). Comorbidities (e.g., diabetes or coronary disease decompensated by stress, infection, blood loss, etc.) may need to be addressed.
- *Pain relief*: A nasogastric decompression tube can be installed in patients with vomiting and/or intestinal obstruction.

Analgesics such as morphine (5–15 mg sc, iv), hydromorphone (1–4 mg sc), or meperidine (50–75 mg im) are commonly administered. The use of analgesics in patients with acute abdominal pain was previously discouraged to avoid masking clinical signs (e.g., increasing pain and abdominal guarding in response to progressive peritoneal irritation) that could have justified an urgent surgical treatment; but this attitude now seems to be less important since, among other things, the diagnosis is now most often obtained earlier with radiological imaging. However, opiates can cause nausea, vomiting, and paralytic ileus, which can become confounding elements in the evaluation of an abdominal pain.

The most common urgent abdominal conditions are listed in Table 16.3.

Table 16.3 Most common abdominal emergencies

Disorders	Symptoms	Diagnostic tests	Treatment
Appendicitis	Pain periumbilical →RLQ	CT scan (ultrasound)	Cx: appendectomy urgent
Biliary colic	Pain epigastrium/RUQ (<4–6 h)	Ultrasound	Analgesia Cx: cholecystectomy elective
Cholecystitis	Pain RUQ Murphy +	Ultrasound	Analgesia/antibiotics? Cx: cholecystectomy < 24–48 h
Cholangitis	Pain RUQ Fever Jaundice	Ultrasound Blood cultures Serum liver tests	Antibiotics ERCP sphincterotomy 1–7 days
Pancreatitis	Pain epigastrium→back	Serum tests CT scan (non-urgent)	NPO, analgesia, hydration ICU (severe pancreatitis)
Visceral perforation	Pain periumbilical or diffuse Generalized peritonitis	X-ray (flat plate) (CT scan)	Cx: urgent Antibiotics
Intestinal occlusion	Cramp/colicky pain Abdomen distension	X-ray (flat plate) (CT scan)	Hydration, NG tube, analgesia? Cx: maximum wait 72 h
Intestinal ischemia	Pain periumbilical or diffuse	Serum lactate CT scan	Heparin? Vascular Cx/RX: urgent
Aortic aneurysm	Pain periumbilical	Ultrasound (CT scan)	Cx urgent
Ischemic colitis	"Colon" (flank, LLQ) pain Bloody stools	CT scan (Colonoscopy)	Hydration, analgesia, antibiotics?
Diverticulitis	Pain LLQ (most often) (Fever)	CT scan Blood WBC	Antibiotics vs NSAID+ analgesia Abscess drainage (RX/if >3 cm) (Surgery)
Ectopic pregnancy	Pain LLQ or RLQ	Ultrasound	Cx: urgent
Renal colic	Pain flank	CT scan	Hydration, Analgesia, tamsulosin [Stone extraction (ureteroscopy)]
"Solid" lesion (liver, spleen, intestine/tumor, abscess)	Pain at lesion site	CT scan	Rx of underlying condition

Abbreviations: *Cx* surgery; *ICU* intensive care unit; *LLQ* left lower quadrant (left iliac fossa); *LUQ* left upper quadrant (left hypochondrium); *NPO* nil per os (fasting); *NG tube* nasogastric tube; *RLQ* right lower quadrant; *RUQ* right upper quadrant; *Rx* treatment; *RX* radiology

16.5 Miscellaneous

Abdominal pain may be due to non-visceral, nonsurgical disorders as listed in ◘ Table 16.4.

Table 16.4 Non-visceral/nonsurgical abdominal pain

A. **Musculoskeletal disorders**
- Radiculopathy (from T6 to L2: disk herniation, diabetic neuritis, shingles, etc.)
- Parietal scar neuroma
- Psoas, rectus abdominis tendinopathy
- Xyphoiditis

B. **Radiating pain**
- Inferior wall myocardial infarction (epigastric pain)
- Inferior lobe pneumonia/pulmonary embolism (hypochondrium pain)

C. **Systemic or metabolic disorders**
- Porphyria
- Mediterranean fever
- Hemolytic crises (sickle cell anemia, malaria)
- Diabetic ketoacidosis
- Lead and arsenic poisoning
- Spider (black widow)/reptile bite
- Epilepsy/abdominal migraine
- Narcotic withdrawal
- Visceral hypersensitivity
- Somatization/anxiety

16.6 Abdominal Pain in Children

In children, some unique features are worth mentioning.

(a) ***Acute abdominal pain***

- In children, appendicitis is, by far, the most common cause of acute abdominal pain.
- In a child suffering from altered general condition, abdominal bloating, and bilious vomiting, an intestinal obstruction must be suspected; particularly, a volvulus secondary to an intestinal malrotation is to be feared since it can have catastrophic consequences if it is not recognized rapidly (short bowel syndrome, death).
- Intestinal intussusception occurs most often in children under 2 years of age. Typically, the child has a sudden abdominal pain, is crying loudly when lying down in a stooping position with the knees pulled to the chest, and presents red jelly-like ("currant jelly") stools. Most often located at the ileocolic junction, "telescoped" intestinal loops suffer from strangulation with venous stasis, parietal edema, and vascular compression; intestinal ischemia may lead to necrosis if the intussusception is not reduced rapidly. The diagnosis is made on ultrasound when the typical "donut" appearance is visualized. In the absence of contraindications (suspicion of perforation, hemodynamic instability, prolonged symptoms), a hydrostatic (or pneumatic) enema is attempted as a first-line treatment. In some cases, surgical reduction (or even intestinal resection) may be necessary. In children over 2 years of age, the possibility of a luminal process precipitating the intussusception, such as a Meckel's diverticulum, a polyp, or a small bowel tumor, should be considered.

(b) ***Chronic abdominal pain***

In 80% of cases, chronic abdominal pain in children is of functional origin. Other common causes are constipation and lactose intolerance.

Signs suggesting organicity include anorexia, weight loss, vomiting, diarrhea, nocturnal pain, and precise localization away from the periumbilical region.