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Non-Surgical Causes of Acute Abdominal Pain

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Additional information is available at the end of the chapter

Abstract

Abdominal pain constitutes 5% of the causes of emergency admissions and is an important part in the practice of emergency services in all centers. Patients may suffer from acute surgical abdomen, acute abdomen with nonsurgical diseases or acute problems of chronic diseases. Abdominal pain is sometimes associated with acute trauma. Clinical assessment is a process where diagnosis and treatment must be done quickly and must be well managed. We have tried here to discuss the non-surgical causes of abdominal pain.

Keywords: Emergency Acute, Abdominal, Pain, Non-surgical, Acute abdomen

1. Introduction

Acute abdomen describes the sudden and severe starting of abdominal pain with unexplained etiology [1]. Case management should be done fairly quickly. Nonsurgical diseases as well as surgical pathologies could be the cause of acute abdomen.

Medical history and physical examination findings are very important for assessment. Abdominal pain is the most important sign of acute abdomen but might not be observed in each cases [2]. Especially the elderly and children should be considered for acute abdomen.

Abdominal pain is usually a feature, but a pain-free acute abdomen can occur, particularly in older people, in children, in the immunocompromised, and in the women during their last trimester of pregnancy. Acute abdominal complaints are common [3].

The differential diagnosis of acute abdomen should be done as soon as possible with the medical history, physical examination, laboratory and radiological findings; and the diagnosis should be accelerated for patient management [4].

2. Pathophysiology

2.1. Visceral pain

Visceral pain is a kind of a pain resulting from abdominal, pelvic and thoracic organs whose mechanism is not clearly understood and thus, very difficult to identify [5]. Visceral pain is a common, often superficial pain which cannot be localized. Nausea, vomiting, and emotional changes can accompany this pain [6].

Visceral pain does not result from every organ and is not always associated with tissue damage. Sensitivity of certain organs to pain depends on the properties of the peripheral receptors of organs [7]. The spread of the visceral pain over a large area depends on the distribution of visceral afferent nociceptive pathways in the central nervous system.

Visceral pain usually occurs as a result of the excessive stress of hollow organs such as the digestive tract, the gall bladder, and the ureter or contraction of smooth muscles [8]. Visceral pain may also occur as a result of the stress of the capsule around the organs such as kidney, liver and spleen. The internal organs do not have parenchyma and pain receptors in the brain. Visceral pain receptors are located where there are artery walls, peritoneum, pleura and dura mater and other connective tissues [9].

2.2. Somatic pain

Somatic pain is a type of nociceptive pain. It is the pain growing in all body zones including the skin, muscles and joints except the internal organs. Arising from the somatic nerves, the pain begins suddenly distinct from the visceral pain and is sharp and well localized.

The somatic pain sensation in the portion below the head comes along with the spinal nerve fibers in the spinal ganglia in the posterior radix [10].

Somatic pain can be either *superficial* or *deep*.

2.2.1. Superficial Somatic Pain

Superficial pain arises from nociceptive receptors in the skin and mucous membranes. For example, if you cut your lip, this pain is called superficial somatic pain. Superficial somatic pain is the type of pain that happens with common everyday injuries and is characterized as pricking, sharp, burning or throbbing pain [11].

2.2.2. Deep Somatic Pain

Deep somatic pain originates from structures deeper in your body, such as joints, bones, tendons and muscles. Like visceral pain, deep somatic pain is usually dull and aching. Deep

somatic pain can either be experienced either locally or more generally depending on the degree of trauma. For example, if you bump your knee, then the pain that you experience is local to your knee. However, if you break your kneecap, or patella, you experience pain in your whole leg [12].

2.3. Referred pain

Referred pain is feeling pain in a different region than the area where the abdominal organs affected by the disease are located [13]. Referred pain can be felt in the deeper parts of the skin or tissue and can be well localized. Stress with air or fluid in the intestines can cause this type of pain [14]. It occurs due to the simultaneous arrival of the somatic afferent nerve fibers innervating the dermatome where the referred pain is sensed and the visceral afferent fibers that innervate the affected abdominal organs to the spinal cord. Referred pain sometimes has hyperesthesia along with pain [15].

2.4. Stimuli leading to abdominal pain

Pain-sensing receptors (nociceptors) are located in the muscle layer of hollow organs such as the bowel and in the capsule of the solid organs such as the liver. Intra-abdominal organs (abdominal viscera) and the mesentery are insensitive to stimuli such as cutting, crashing or tearing that can normally evoke pain in the skin [16]. There are three kinds of stimuli that can alert the nociceptors in the abdominal organs: (1) tension or withdrawal of the visceral walls, (2) inflammation (due to chemical mediators and edema arising in the inflammation area such as bradykinin, serotonin, leukotrienes and prostaglandins) and (3) ischemia (due to the accumulation of metabolites and chemical mediators in the tissues). Visceral peritoneum (serosa), liver parenchyma and greater omentum are insensitive to pain [17]. Inflammation caused by chemicals and bacteria in the parietal peritoneum is an important cause of pain. Inflammation and edema in the tissues lower pain threshold. The other two important reasons for abdominal pain are the stress of the neoplastic formations or fibrotic tissues on the nerve roots [18, 19].

3. Non-surgical disorders causing acute abdominal pain

Abdominal pain is the most constant symptom of acute abdomen whether of surgical or non-surgical origin. Non-surgical causes of acute abdominal pain stimulating an acute abdomen account for up to 30% of patients requiring hospital admission [20].

The history and physical examination remain the critical first step in effective management and must be based on a thorough understanding of the anatomy and physiology of abdominal pain. Laboratory studies are of limited value. Complete blood count (CBC), urinalysis, serum lipase and pregnancy test are most helpful, particularly when abnormal.

Computed tomography (CT) has led to the greatest improvement in the care of patients with acute abdominal pain. Its value for any given patient depends on a given institution's experience in its application and interpretation.

3.1. Metabolic/endocrine causes

3.1.1. Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are among the most serious and accurate metabolic complications of diabetes [21]. These situations can be observed in both type 1 and type 2 diabetes mellitus (DM). While the mortality rate is below 5% in good hands in diabetic ketoacidosis (DKA), it is at a level as high as 15% in hyperosmolar hyperglycemic state (HHS). In both cases, the prognosis worsens with aging in the presence of coma and hypertension. Fifteen percent to 67% of type 1 diabetic patients have DKA in the first diagnosis [22]. Absolute or partial insulin deficiency is the underlying reason in metabolic disorders in DKA. The consequences of insulin deficiency become more pronounced with the strong impact of catecholamines, glucagon, cortisol and counterregulatory hormones (anti-insulin hormones). As a result, glucose production by the liver and kidney increases and finally hyperglycemia and hyperosmolarity occur. Increasing lipolysis and ketone bodies production causes ketosis and acidosis [23]. Hyperglycemia and acidosis result in osmotic diuresis, dehydration and the loss of essential electrolytes.

Polyuria, polydipsia, polyphagia history, weight loss, vomiting, abdominal pain, dehydration, weakness, confusion and as a result coma are the classical clinical observations of DKA.

Abdominal pain can mimic acute abdomen especially in children. The abdominal complaints can be seen in 40%–75% of the patients with DKA [24].

There have been many studies on the mechanism of abdominal pain in DKA; however, a complete mechanism has not been established. It was observed that after hyperglycemia, ketoacidosis and dehydration are improved, especially abdominal pain complaints disappeared. In some DKA cases, it was reported that exploratory laparotomy was applied due to the problem of diagnosis and it was associated with high morbidity and mortality [25].

Diabetic ketoacidosis	High serum glucose
Familial Mediterranean fever	Pleuritis, peritonitis
Porphyria	High porphobilinogen
Addison disease	Low serum cortisol
Hyperthyroidism	Low TSH
Hypokalemia	Low serum potassium
Hypophosphatemia	Low serum phosphate
Hypercalcemia	High serum calcium

Table 1. Metabolic causes.

The possibility of abdomen surgery should always be kept in mind in the cases where abdominal complaints do not disappear despite DKA treatment.

Abdominal pain in DKA might be correlated with the weight of metabolic acidosis and might be confused with an acute abdominal crisis. Other metabolic causes are listed in **Table 1**.

3.2. Hematologic/immunological causes

3.2.1. Sickle cell crisis

Sickle cell anemia (SCA) is a genetic disorder caused by abnormal hemoglobin S production. Hemoglobin S is formed by replacing the glutamic acid in 6th stage of the beta chain with valine. SCA is characterized by recurrent vaso-occlusive crisis [26]. Vaso-occlusive crisis is an emergency that requires frequent hospitalization of the patients with SCA [27]. Although pathophysiology and treatment of various crises are known, mortality rate associated with this disease is high. The mortality rate in this disease is the highest in the first 5 years of life. Almost half of these mortalities occur in the second 6 months of life. Acute infections and serious anemic sequestrations are the most common causes of death [28].

Sickle-shaped erythrocytes reduce the flow of circulation and the blood flow slows down. This leads to congestion especially in the small vessels and creates an anaerobic environment. Some of the sickle cells are recycled and can take their normal form. However, some others cannot go back to their normal form due to the permanent destruction occurring in their cell membranes. These cells lead to hypoxia in the tissues, leading to atherosclerosis and cause painful crisis and organ necrosis as well as tissue damage in the acute and chronic processes.

Vaso-occlusive, in other words, painful crises are usually the first symptom of the disease and are the most common complication after the newborn period [29]. Initiating factors in acute painful episodes may be exposure to cold, dehydration, infection, stress, menstruation or alcohol intake. The initiator cause is infection in 80% of patients [30].

Sickle cell crisis	Sickle cells, effusions
Thrombotic thrombocytopenic purpura	Thrombocytopenia, fever, confusion
Hemolytic uremic syndrome	Schistocytes on smear
Food allergy	Increased Ig E level
Henoch-Schönlein purpura	Leukocytoclastic vasculitis with Ig A deposition
Mast cell activation syndrome	Serologic markers as above
Hereditary angioneurotic edema	Low C1 esterase inhibitor level

Table 2. Hematologic/immunologic causes.

Intravascular sickling causes blockages in small blood vessels and eventually internal organs and soft tissue necrosis [31]. Necrosis shows itself with common bone, joint and muscle pain. Although pain may affect any area of the body, back, chest, abdomen and extremities are most commonly affected. The severity of the pain can be too light that can be ignored or too severe

to be tolerated. Findings such as fever, joint swelling, tenderness, tachypnea, hypertension, nausea and vomiting may accompany painful crisis [32].

Widespread and persistent abdominal pain is one of the most common complaints. It can be difficult to distinguish abdominal pain from abdominal events, such as sickle cell crises, cholecystitis and appendicitis. Patients might be aware of the difference or similarities of the pain with the previous episodes and can distinguish it. There should not be peritonitis symptoms such as rebound tenderness in the abdominal examination of patients with typical vaso-occlusive episodes. This finding is important to distinguish from other diseases that cause acute abdomen.

Other hematologic and immunologic causes are listed in **Table 2**.

3.3. Cardiopulmonary/vascular causes

3.3.1. Myocardial infarction

One of the pathologies that comes to mind foremost in patients presenting acute abdominal pain is acute coronary syndrome. The symptoms of acute myocardial infarction are not always typical. Symptoms especially in inferior myocardial infarction can be confused with gastrointestinal (GI) symptoms. Myocardial infarction pain is very severe and is often described by patients as unbearable. The time is long and often over 30 minutes. It can last for hours. Associated symptoms such as nausea, vomiting and sweating can be seen in approximately half of the cases [33].

Electrocardiogram (ECG) in differentiating the causes of abdominal pain is therefore a very important move. However, although half of the patients experience acute coronary syndrome, it should be noted that the ECG can be negative. In this case, the serum levels of cardiac markers are important for the diagnosis.

Cardiac troponin I (cTnI) is a key protein in cardiac muscle contraction and relaxation. Three to six hours after the start of the acute myocardial infarction (AMI), cTnI release from necrotic myocardium starts and peaks in 24–48 hours. It stays high for 10 days after the initiation of AMI. It is a perfect and specific, long-term, high-residual marker for AMI. In particular, in unstable angina and non-Q wave AMI, detection of elevated serum cTnI level while serum creatine kinase-MB (CK-MB) levels are in the normal range indicates that cTnI is highly sensitive for minimal myocardial damage [34].

Myocardial infarction	Abnormal ECG, high troponin
Aortic dissection	Diagnostic CT angiogram
Pulmonary embolus	High D-dimer diagnostic CT angiography
Pneumonia	Chest radiograph

Table 3. Cardiopulmonary/vascular causes.

No classic findings in the beginning of AMI and frequent complaints of abdominal pain may present various clinical cases that may delay early and accurate diagnosis [35].

Other cardiopulmonary or vascular causes are listed in **Table 3**.

3.4. Infectious causes

3.4.1. Malaria

Malaria is a parasite infection formed by protozoa from the plasmodium family and transmitted to humans by anopheles breed of mosquitoes, which progress with bouts of fever, anemia and splenomegaly, and tends to be initially acute and chronic when left untreated [36]. Malaria agents are *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, *Plasmodium malaria* and, the newly defined in 2008, *Plasmodium knowlesii*. According to the data obtained from 106 endemic countries where the majority is African countries by the World Health Organization (WHO), about 3.3 billion people faced the risk of malaria in 2010. In 2010, 216 million malaria episodes occurred worldwide, 81% of these were observed in African countries. Because of the infection, 655,000 people died, and the majority of deaths (91%) are still in the African continent. Unfortunately, 86% of the deaths in the world are of children under 5 years of age [37].

Abdominal pain can be traced in malaria due to many reasons. More abdominal symptoms are seen in falciparum malaria when compared to vivax malaria. The abdominal pain is believed to be secondary developing microvascular occlusions to the developing excessive red blood cell sequestration [38].

The pain in malaria cases is usually transient and mild, but in some cases it may be very severe and take longer time to reside. It should be kept in mind that complications such as acalculous cholecystitis, GI bleeding, splenic rupture and splenic infarction may develop in malaria cases [39].

Other infection causes are listed in **Table 4**.

Malaria	Fever, hemolytic anemia, myalgia, multiorgan disease
Staphylotoxin	Fever, rush
Tuberculosis mesenteritis	Fever, diarrhea, ascites
Yersinia enterocolitica	Diarrhea, fever, positive stool culture
Dengue fever	Fever, hemolytic anemia, low platelets

Table 4. Infectious causes.

3.5. Drug/toxin causes

Drugs and toxins can manifest themselves as acute abdominal pain sources through several mechanisms. For example, corrosives can cause acute abdomen by causing serious side effects

on the GI tract. Anticholinergic drugs and narcotics may be the cause of partial obstruction of ileus. Again, vasoconstrictor drugs can create acute abdominal pain due to ischemic colitis. Many drugs can cause serious toxic effects especially on the properties of the liver and pancreas, and patients may present with severe acute abdominal symptoms.

Some drugs and toxins are listed in **Table 5**.

Salicylate	Tinnitus, confusion, metabolic acidosis
Tricyclic antidepressants	Anticholinergic symptoms, ECG changes, delirium
Anticholinergics	Tachycardia, confusion, ileus
Heavy metals	Renal, neurologic toxicity
Cocaine	Hypertension, systemic and organ ischemia

Table 5. Drug/toxin causes.

3.6. Neuropsychiatric causes

3.6.1. Herpes zoster

It is the reactivation of zoster latent varicella zoster virus (VZV) infection. VZV that can reside in the sensory nerve ganglia after chickenpox can cause shingles with lesions anatomically similar to those in chicken pox along the dorsal ganglia usually years later [40]. These lesions are controlled with host immune responses (especially, anti-VZV cytotoxic T cells) and recover within a few weeks [41]. Rarely in immune-sufficient people, complications can develop such as very painful, ongoing post-herpetic neuralgia, meningitis, encephalitis, eye involvement, perivasculitis and atypical necrotizing retinopathy [42]. Very severe and life-threatening shingles can be seen in immunocompromised people (suffering from HIV infection and some cancers) [43].

Herpes zoster	Unilateral, painful vesicular rash in dermatomal distribution
Temporal lobe seizures	Aura, abnormal EEG
Radiculopathy	Mechanical pain in dermatomal distribution
Functional abdominal pain syndrome	In woman, fatigue
Abdominal migraine	Adolescent, cyclic occurrence
Irritable bowel syndrome	Diarrhea, constipation

Table 6. Neuropsychiatric causes.

In zona zoster infection, acute abdominal pain is one of the symptoms and may be strong enough to cause laparotomic evaluation by mixing with surgical acute abdominal pain. The reason for 3 of the 121 laparotomies made in a series was found to be zona zoster [44].

A much more acute clinical picture is the abdominal zoster clinic that may occur in the immune-suppressed patients. These patients present dermis findings that progress very commonly

within hours and very serious abdominal pain. Especially for these patients, mortality rate is very high despite treatment.

Other neurophsychiatric causes are listed in **Table 6**.

3.7. Renal causes

3.7.1. Nephrolithiasis

It gives similar symptoms to renal and ureteral stones. There are two main symptoms: lumbar pain and hematuria. Lumbar pain may be blunt or renal colic. Pain is felt in the costovertebral angle; it can stay in this area especially during renal colic and it can may spread downwards in the abdomen, groin, genital or femoral region. In addition, complaints such as cold sweats, nausea and vomiting can also be added to the case with increased sympathetic tone. If the infection occurs in the upper urinary tract, symptoms such as fever and pyuria occur in addition to this scene. Fever varies according to the degree of obstruction made by the stone from the remittent fever case to the continuous high fever case.

Other renal causes are listed in **Table 7**.

Nephrolithiasis	Hematuria
Papillary necrosis	Hematuria, diabetes, sickle cell disease

Table 7. Renal causes.

3.8. Vasculitis/connective tissue causes

3.8.1. Systemic lupus erythematosus

Systemic lupus erythematosus (SLE) is a chronic, of unknown cause, connective tissue disease with immunologic disorders and is of autoimmune nature and affects many organs and systems [45]. The disease varies from clinical fever, swelling in the joints, erythematous skin rash to impact on organs and systems, such as kidney, central nervous system and lungs. The majority of patients might have specific organs and system symptoms along with nonspecific systemic symptoms such as weakness and fatigue, fever, muscle aches and weight loss. The disease may sometimes mimic infection starting with fever or may progress in an insidious way in months and years with fever, fatigue and weakness symptoms. Clinical course may range from mild to severe; remission and flare-ups occur typically at varying periods.

Nonspecific gastrointestinal symptoms are seen in lupus patients [46]. Loss of appetite, nausea, vomiting and abdominal pain may develop due to inflammation of the peritoneum (aseptic peritonitis), intestinal vascular disease (mesenteric vasculitis) or drug therapy (NSAIDs and corticosteroids). It may progress to mesentery vasculitis intestinal ischemia, infarction or perforation. The participation of esophagus in the disease is as esophagitis,

esophageal ulceration or esophageal motility disorder. Hepatitis and chronic pancreatitis are rare.

Gastrointestinal (GI) manifestations occur in approximately 25–40% of patients with SLE [47]. Many of these symptoms are nonspecific and often reflect either lupus of the GI tract or the effects of medication. Lupus mesenteric vasculitis (LMV) is a predominant cause of acute abdominal pain in SLE patients [48], and it occurs at a high frequency in association with active disease. LMV varies from 29 to 65% among SLE patients with acute abdominal pain. CT is the imaging method of choice to confirm the diagnosis because it permits the visualization of the bowel wall and abdominal vasculature. CT findings include characteristic dilated bowel loops, focal or diffused bowel-wall thickening, abnormal bowel-wall enhancement (target sign), mesenteric edema, stenosis or engorgement of the mesenteric vessels, causing the comb sign and ascites [49, 50]. The CT findings in the present case were consistent with that of acute abdomen because of SLE.

Other connective tissue causes are listed in **Table 8**.

Systemic lupus erythematosus	SLE criteria
Systemic vasculitis	Multiorgan disease with positive p-ANCA
Scleroderma	Skin changes, visceral disease, Scl-70

Table 8. Vasculitis/connective tissue causes.

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