



ABDOMINAL PAIN

ESSENTIAL DIAGNOSIS AND

MANAGEMENT IN ACUTE MEDICINE

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Abdominal Pain: Essential Diagnosis and Management in Acute Medicine

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PREFACE

Almost all aspects of medicine are challenging for both healthcare providers and sufferers. Although subthemes covered by medicine can be classified as discrete systems or organs of the body, caregivers in primary and acute care prefer to conceive these with complaints, symptoms and semiological approaches. Therefore, no patient presents to the acute care area with an ‘acute appendicitis’ or ‘acute cholangitis’, instead, there comes ‘*a girl in her 18 with vomiting and abdominal pain for a couple of days*’, or, ‘*an elderly gentleman who is not looking very good recently*’ in the boxes. The patient’s diagnosis seems very elusive, for it can turn out to be pelvic inflammatory disease, urinary tract infection, ruptured ovarian cyst or abscess, intestinal obstruction, diverticulitis, mesenteric ischemia, etc, while the patient is screaming for emergency relief for both pain and vomiting in the acute care area.

Abdominal pain is one of the most common chief complaints in both emergency settings and primary care, which constitutes around 10% in most large studies in the world. Unlike many entities presenting to outpatient clinics and those admitted to the wards, the origin of abdominal pain is harder to diagnose provided with the complexity and closeness of the structures in the abdomen and also extra abdominal causes that can trigger the symptomatology. Nonetheless, recent decades witnessed giant leaps and advances in the recognition and treatment of patients with acute abdominal pain. Apart from advanced studies such as computed tomography, and magnetic resonance imaging, bedside point-of-care procedures like ultrasonography have eased diagnosis and facilitated the management of patients with abdominal pain. We should note that regardless of technological advances, the most important contributor to the diagnosis and management process consists of the evaluation of the patient with an elaborate history and physical examination. Using a tailored approach for evaluation, an experienced physician can not only narrow the list of differential diagnosis, but also expedite the complex pathway to definitive treatment, preventing unnecessary delays with cumbersome investigations.

The optimal management of the patients with abdominal pain warrants a multifaceted approach undertaken in harmony. COVID-19 pandemic era has brought *de novo* challenges for the delivery of ‘usual’ medical care into the scene for most patients. This book titled “**Abdominal Pain: Essential Diagnosis and Management in Acute Medicine**”, therefore, is intended to highlight the contemporary approaches with respect to diagnostic and therapeutic modalities for diseases of digestive tract and other entities precipitating abdominal pain. Abundant figures, tables, and radiological images have been used to render understanding easier and to illustrate key findings. We hope this project can be used as a reference and an everlasting source for caregivers facing sufferers of abdominal pain, albeit a small step in the history of medical progress lasting for thousands of years.

CONSENT FOR PUBLICATION

Not applicable.

CONFLICT OF INTEREST

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CHAPTER 1

The “Stomachache” of Medicine: Concepts and Mechanisms of Abdominal Pain

Abstract: Abdominal pain (AP) is by far among the most common complaints in healthcare institutions. Approximately every tenth patient in the acute setting is estimated to present with AP. Although cultural, geographical and sociodemographic variations exist, it is an outstanding complaint in all patient groups, independent of age and gender. Although it can be a manifestation of an intraabdominal pathology itself, a serious systemic or extraabdominal condition can be revealed following a thorough investigation of AP. Therefore, it is vital to evaluate the patient systemically, a focused but elaborate history, and extensive physical examination not confined to the abdomen in order to establish important diagnoses. Inspection, auscultation, percussion, superficial and deep palpation are important elements of the examination methods for the abdomen. Each positive or negative finding on examination should be interpreted cautiously for the individual patient. After history and evaluation narrow the list of differential diagnoses (DD), ancillary investigations including laboratory tests and radiological modalities can be ordered.

Keywords: Abdominal pain, Physical examination, Laboratory tests, Work up, Imaging, Differential diagnoses.

GENERAL CONSIDERATIONS

Abdominal pain (AP) is one of the commonest reasons for admission to emergency departments (ED) and other healthcare institutions. Considering the singular ‘chief’ main complaints, it is established that 8% to 10% of patients both in Turkey and in the world are admitted to the ED with AP. Since registry databases are operated inadequately in developing worlds, most data are derived from Europe and America, therefore substantial variations may occur especially in regard to entities affected by geographical properties. While the rate of hospitalization among adult patients who present to ED with AP varies between 18% and 42%, the same can be found higher, up to 65% in elderly patients.

Pearl: The main goal of the physician in the management of AP is to decide which patients can be safely discharged and which patients should be kept in the hospital.

Distinction Between Acute And Chronic AP

In many sources, acute AP is noted as pain within the first 7 days. The term “chronic” mostly refers to the pain of the same character over months and years. In this case, it should be borne in mind that the patient presenting with pain for 6 or 7 days can still be diagnosed with “acute” cholecystitis or pancreatitis, although somehow difficult to be comprehended. For example, it is possible to extend this period even longer, since the pain associated with choledocholithiasis may last (and neglected) for a few days and then turn into acute cholecystitis. In brief, real-life scenarios may not fit into the encyclopedic information or what we can expect to see.

Why does My Belly Hurt?

The structure related to the digestive system in embryonic life differentiates into 3 separate parts.

- **Foregut:** It includes stomach, duodenum, liver, gall bladder, and pancreas. Upper abdominal pain is remarkable in their malfunctions or inflammations.
- **Midgut** (small intestine, proximal colon, appendix) causes periumbilical pain.
- **Hindgut** structures (distal colon and genitourinary system) cause pain in the left, midline or right lower abdomen.

Anatomophysiologic mechanisms include the splanchnic and the cerebrospinal neural pathways activated for transmission of AP. Pacinian corpuscles and free nerve endings in the visceral walls are the splanchnic afferent receptors sensitive to *stretch* and *spasm*. On the other hand, cerebrospinal receptors are sensitive to pressure, friction, cutting, burning that are discerned by the skin. In the dorsal root ganglia (DRG) the splanchnic and cerebrospinal cell bodies are in close proximity. The proximal fibers terminate around the spinal cord. The close relationship of these anatomic pathways may contribute to the fact that severe visceral pain, such as rapid distention of a viscus, may “spill over” into somatic segments (viscerosensory and visceromotor reflexes) in the absence of somatic nerve irritation (Fig. 1).

The embryonic gut and its appendages emerge as midline organs, therefore, their splanchnic innervation is bilateral. This is why the perception of visceral pain locates in the midline. In fact, there are not any nerve fibers in the visceral peritoneum. Cerebrospinal nerves to the parietal peritoneum (T6 through T12) have the same segmental organization as the lower thoracic dermatomes.

Dichotomous Classification of Pain: Visceral or Somatic?

Visceral pain is a type of pain originating from the internal organs. This type of pain is ill-defined, not well localized, is felt in a wide area, and its changes are also felt much slower than somatic pain. With these features, it can cause pain to be felt away from the diseased or inflamed organ.

Somatic pain, on the other hand, is the stimulation of **peripheral nociceptive nerves**. Abdominal pain is essentially triggered by irritation of the parietal peritoneum. It is also provoked by infection, chemical irritation (like spilled bile, faeces or urine into peritoneal cavity), and/or trauma. When the inflammatory process expands and eventually irritates the peritoneum, the **somatic component** is activated and the pain is much better localized by the conscious patient. It is often described by patients in the form of severe ‘sharp’ pain. Typically, this course is observed in pathologies such as acute appendicitis (AAp) and acute cholecystitis (AC), which are very close to the peritoneum.

It has been documented in rats that there is an organ-to-organ cross-sensitization of pelvic viscera, including colon, bladder, and female reproductive organs, which may contribute to the overlap of lower abdominal pain. At the peripheral and spinal level, there are two potential pathways involved in such cross-sensitization: (1) axonal dichotomy of visceral sensory afferents (Fig. 1A) and (2) convergence of two visceral afferents from two organs on spinal neurons (Fig. 1B).

Pitfalls

The term “stomachache ” seems to be justified as the difficulty of diagnosis in patients presenting with AP is a real challenge for physicians. Delay in diagnosis and misdiagnosis is a common problem even for the most experienced emergency physician or general surgeon. Disruptions that may be related to ancillary services such as radiology and biochemistry also increase the difficulty.

Age and Sex

Two important factors for the definitive diagnosis of AP are age and gender. Peptic ulcer, gastritis, urinary stone disease and AAp are more common causes of AP encountered in men, while nonspecific abdominal pain (NSAP), biliary tract diseases, functional bowel disorders including IBS, urinary tract infections (UTI) and pelvic inflammatory disease (PID) are outstanding in women.

As with every symptom, the cause of AP should be explained, diagnosed and resolved. Many diseases known in medicine can cause AP directly or indirectly.

Laboratory and Radiological Investigations and Cost-effectiveness

Abstract: Abdominal pain (AP) is a very common complaint that renders it elusive to diagnose in most instances. After history and evaluation narrow the list of differential diagnoses (DD), ancillary investigations including laboratory tests and radiological modalities are ordered. Of note, these adjuncts will only help the clinician who bears a presumptive diagnosis on the mind. Non-invasive, repeatable and cost-efficient options such as ultrasound are preferred initially, although, in most instances, more specific and definitive information warrants advanced imaging techniques including computed tomography and contrast studies. Laboratory work-up needs to be tailored to the individual based on findings on evaluation. ECG, complete blood count and blood chemistry can provide inappreciable clues for specific diagnoses while none will be sufficient *per se*. Urinalysis and specific cultures including stool studies will expedite recognition of urinary tract infection, amebiasis and other infections when indicated. B-hCG level can prevent unwanted exposure to radiation and drug effects on an unrecognized pregnant woman. Thus, all these adjunctive investigations should be included in the management plan individualized to the patient, based on the history and evaluation findings.

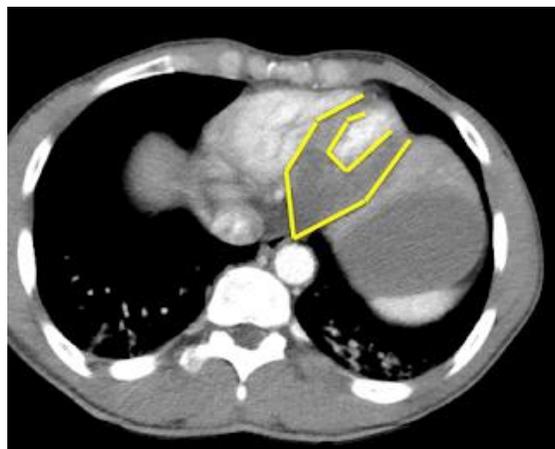
Keywords: Abdominal pain, Computed tomography, Contrast media, Diagnosis, Imaging, Laboratory tests, Physical examination, Radiological.

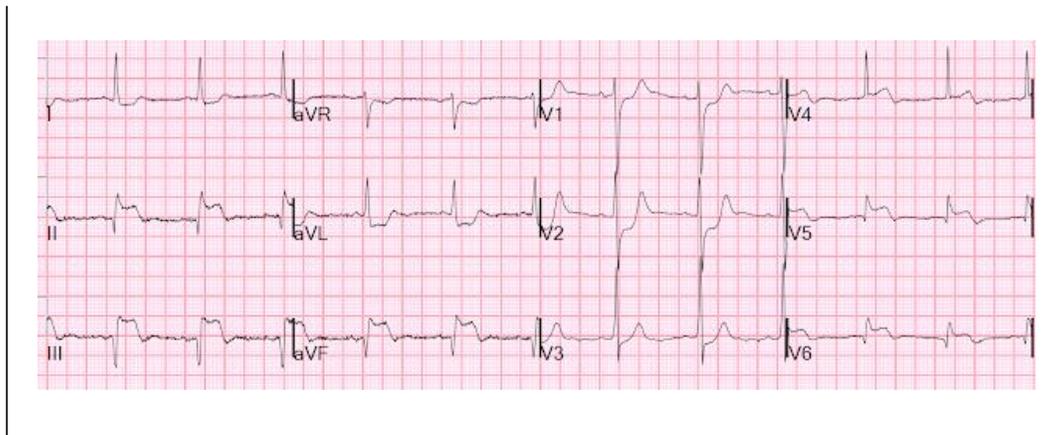
There are myriad types of investigations in the medical area and they are getting more varied over time. The clinician assumes the difficult task of tailoring these tests for each patient. A focused history and thorough examination are the key features to judge the test strategy, which will prevent unnecessary testing and expedite diagnosis and management.

ECG: It should be taken and interpreted urgently in all patients over 40 years of age, especially in patients presenting with atypical presentations of AP or additional systemic signs and symptoms. Since the sensitivity of the first ECG in detecting acute coronary syndrome does not exceed 50% to 60%, serial ECG monitoring is the rule in suspected cases. It should be noted that myocardial infarctions involving the “inferior” wall of the heart, neighbouring diaphragm,

often present with AP (or thoracoabdominal pain). There may be upper abdominal or epigastric pain here, not the lower abdomen. In short, AMI and ACS are in the DD of epigastric pain.

Case example: A 30-year-old male with no previous cardiac history was admitted to the ED with epigastric pain and vomiting. There was no chest pain, shortness of breath, or shoulder or jaw pain. Chest and abdomen CT scans of the patient were taken and interpreted as unremarkable for acute abdominal conditions (Figs. A and B). Although acute inferolateral AMI is recognized in the ECG (C) taken later, it is seen that the inferior wall myocardial tissue is darker than normal myocardium in the retrospective scrutiny on CT sections. This appearance was thought to be related to the lack of contrast enhancement of the infarcted tissue.





CBC: For adults, a leukocyte count of $10,000/\text{mm}^3$ and a neutrophil count above 75% are considered supranormal or pathological in most laboratories. If leukocytosis is compatible with the history and physical examination findings, it may be significant and guide physicians in the decision making for the presumptive diagnosis. However, its sensitivity and specificity are lower than an “ideal test” in most intraabdominal conditions. For example, in 10% to 60% of AAP cases, there is no leukocytosis on presentation. If the leukocyte count is over $30,000/\text{mm}^3$, different diagnoses such as leukemia/leukomoid reaction, intraabdominal sepsis should be thought of, not the “classical” acute abdominal entities such as AAP and cholecystitis. While leukocytosis is expected in sepsis, values below $4000/\text{mm}^3$ (leukopenia) may also be encountered as an ominous sign of mortality. For these reasons, it should be tried not to rule in or out diagnoses based solely on leukocyte counts.

Amylase and Lipase: Serum amylase increases with many intra- and extra-abdominal conditions such as acute pancreatitis, peptic ulcer, ileus, ectopic pregnancy, diabetic ketoacidosis (DKA), liver diseases, small bowel ischemia/perforation, intestinal wall injury, parotid gland diseases. Serum lipase is more sensitive and specific in the diagnosis of AP and other pancreatic disorders.

Pill information: If the blood levels of amylase and lipase exceed 3 times the upper limit of normal, it indicates pancreatitis in most patients with AP or vomiting. In other situations, it can be viewed as a nonspecific test that should be verified *via* other supportive tests such as lipase and trypsinogen activation.

Liver Enzymes: AST, ALT, and bilirubin are commonly ordered tests in 'routine' biochemistry, but this is a wrong practice. Apart from acute viral hepatitis,

Pain: Methods for the Assessment

Abstract: International Association for the Study of Pain defines pain as an unpleasant subjective sensation that includes the past experiences of the person with or without tissue damage.

Acute pain, generally lasting for hours to days, is the primary complaint at a rate of up to 70-80% at first admission. Headache, myalgia, arthralgia, back pain, local pain induced by minor trauma (such as sprains), thoracoabdominal pain, ear, facial pain, etc. are the most common types of presentations in the acute setting related to pain. Analgesia, on the other hand, is the relief of the perception of pain without causing sedation or any change in vital signs.

It is one of the few areas a physician can make a difference to implement more efficient patient care. The subjective and multidimensional nature of the pain experience make pain assessment really challenging. Patients' evaluation of pain should be the main reference for decision-making to provide analgesics or not. Implementation of dimensional recording of pain in clinical practice include the addition of pain as the "fifth" vital sign to be noted during initial assessment; the use of pain intensity ratings; and posting of a statement on pain management in all patient care area.

Our motto should be "pain cannot be treated if it cannot be assessed". The most important principle is that clinicians should somehow assess their patients' pain levels, independent of the specific method or scale to achieve this. Although all pain-rating scales are valid, reliable and appropriate for use, the VAS has somehow appeared more difficult than the others. Pain reassessment should be guided by pain severity reported by the patients themselves.

Keywords: Assessment, Evaluation, Multidimensional, Pain, Pain rating, Physical examination, Unidimensional.

Pain is the most common reason for presenting to EDs, regardless of age, gender, occupation or education level (Cordell, 2002, Wheeler 2010). Researchers cited that pain was the main complaint of more than 80% of patients admitted to the ED. In the last three decades, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO), which has issued the accreditation principles of health institutions in many items, has announced that appropriate assessment and management of pain is mandatory and therefore, standard care rules have

commenced to be established in ED setting all over the world.

Pain is not simply a response to physical injury, instead, it is a behavioral pattern that is affected by anxiety, depression, expectations and other psychological changes, together with current and past experiences. Thus, the International Association for the Study of Pain (IASP) has described pain as a “sensory and emotional experience originating from any part of the body, accompanied by tissue damage or potential tissue damage, or defined in the process of such damage” (Raja, 2020).

Furthermore, JCAHO stated that **pain should be measured and recorded as a 5th vital sign** in the ED evaluation of the patients in the acute setting.

THE TERMINOLOGY OF PAIN

The IASP published the terminology for pain in 1979. In routine clinical practice, many different terms are used to describe different pain syndromes and patterns. These terms and their explanations are given below:

- **Analgesia:** Absence and sensation of pain caused by painful stimulation.
- **Anesthesia Dolorosa:** Pain in an anesthetic site or area.
- **Causalgia:** Burning pain that persists after a traumatic nerve lesion.
- **Central pain:** Pain associated with a central nervous system lesion.
- **Dysesthesia:** An unpleasant (undesirable) abnormal sensation (feeling).
- **Hyperalgesia:** Increased sensitivity and responsiveness to painful stimuli.
- **Hyperesthesia:** Increased sensitivity to stimulation.
- **Hyperpathy:** Painful syndrome characterized by hyperalgesia and overreaction.
- **Hypoalgesia:** Decreased sensitivity and responsiveness to painful stimuli.
- **Hypoesthesia:** decreased sensitivity to stimulation.
- **Neuralgia:** Pain that radiates to a nerve.
- **Neuritis:** An inflammation of the nerve itself.
- **Neuropathy:** Pathological change or dysfunction in a nerve.
- **Nociceptor:** Specific receptor sensitive to painful or potentially painful stimulus.
- **Paresthesia:** Abnormal sensation (in the absence of a stimulus)
- **Stimulus:** A material effect that causes a change or damage in the tissue.
- **Pain threshold:** The intensity of the smallest stimulus that causes pain in the person.
- **Pain tolerance level:** The largest stimulus triggering pain that the person can tolerate.

Acute Pain and its Perception

Acute pain is provoked by harmful stimuli produced by abnormal muscle or organ function, deep somatic structures, organs, skin disease, or trauma. The corresponding mechanisms in the clinical practice are muscle contraction, tissue ischemia, and traumatic tissue destruction.

Individual perception of pain differs according to continents, countries, or smaller geographical units, whilst age and gender also affect perception and management.

In brief, perception of and response to pain vary from person to person. The clinician should not expect a standard pattern of pain behavior. Another important point is that perceived pain intensity does not appear to be directly related to the degree of tissue damage that occurred.

One of the most commonly used pain classifications is given in Fig. (1).

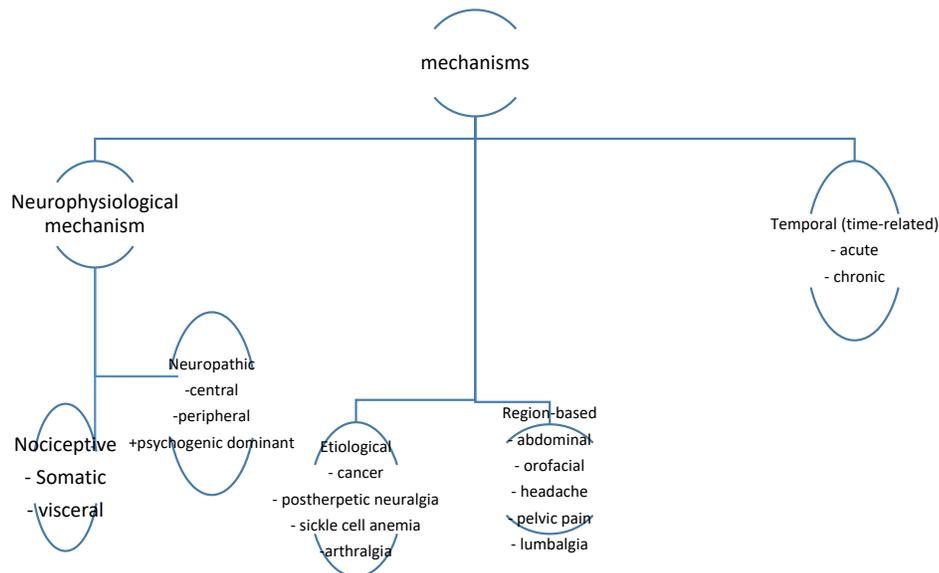


Fig. (1). Types and subtypes of pain.

Pain in the Clinical Practice

It is known that pain is one of the most common problems in daily practice, since

CHAPTER 4**Specific Diagnoses and Management Principles of the Upper Digestive Canal**

Abstract: Acute abdominal conditions which frequently necessitate emergency interventions and/or surgery include visceral perforations *i.e.*, gastric and duodenal ulcer, bleeding and rarely, ingested foreign bodies causing tissue damage, *e.g.*, button batteries. However, the differential diagnosis (DD) of patients presenting with acute abdominal pain is much broader than this, including many benign conditions as well. Acute gastroenteritis, acute gastritis and peptic ulcer disease are benign and mostly temporary diseases which may be relieved with simple treatments and follow-up. Gastrointestinal bleeding (with or without esophageal varices) may cause hemorrhagic shock unless expedient management is pursued. Ingested foreign bodies can constitute emergency conditions with tissue damage, especially when lodged in a specific site. The most important thing about button batteries is the prevention of their ingestion. Complications increase in direct proportion to time wasted.

Keywords: Acute abdominal pain, Esophageal varices, Gastrointestinal bleeding, Peptic ulcer, Surgical abdomen, Visceral perforation.

ACUTE GASTROENTERITIS (AGE)

Diarrhea or acute gastroenteritis (AGE) is defined as the passage of loose or watery stools typically at least three times in 24 hours. Every individual experiences this condition in one way or another throughout his life. Diarrhea represents one of the top five causes of death worldwide and is the second leading cause of death in children under the age of five (following only acute respiratory infections). Although seasonal variability is remarkable, diarrhea constitutes a substantial rate of 3-4% in the ED patient population (Yilmaz S, 2018). In a number of studies, the average age was around 35 and more than half (up to 2/3) of the cases were women in Turkey (Yilmaz S, 2018, Bozdemir MN, 2014, Tiryaki O, 2005). Its incidence is higher in rural areas than in cities.

Diarrhea is usually caused by a viral or bacterial infection that is relieved within a few days. Most children and adults with AGE are associated with contaminated

food and water supplies (WHO). Paying attention to some basic principles in treatment will provide satisfactory results for patients and physicians.

Why do we have diarrhea? Apart from bacteria, viruses and fungi, emotional factors such as stress and anxiety can precipitate diarrhea. In some susceptible individuals, consumption of alcohol or caffeine, and overt ingestion of sweet or heavy foods have also been reported to be among factors for diarrhea which is mostly short-lived and self-limiting. Diarrhea occurs when babies cannot digest lactose-containing foods and beverages, especially feeding with cow's milk. Diarrhea can spread rapidly due to unhygienic pools used in the summer period. In general, toilet hygiene and infrastructural factors are prominent variables causing a surge in diarrhea incidences worldwide, especially in developing countries and regions.

Causative Agents and Spread: *Campylobacter*, *Salmonella*, *Shigella* and *E. coli*. are among the most common bacterial agents detected in developing countries related to diarrhea. Differences in rates vary depending on age, eating habits, socioeconomic level and geographical distribution. For example, it spreads rapidly among young children who have not fully learned hygienic toilet principles at home and in schools.

The most common etiologies of diarrhea are given in Table 1.

Table 1. The most common causes of diarrhea.

Microbial diseases (virus, bacteria, fungus, protozoa) ingested with water and food
Consumption of milk and dairy products other than breastfeeding in infants.
Parasites
Use of agents such as laxatives and purgatives
Other drug-related untoward effects, <i>e.g.</i> , chemotherapy
Radiotherapy
Diseases that impair intestinal structure or absorptive functions, such as inflammatory bowel disease (Crohn's disease, ulcerative colitis).

Inadequate Storage of Foods: Most often, milk, cream, mayonnaise, and meat-containing foods that were left out for a long time cause diarrhea. Minced meat is much more likely than bulk meat to get infected. Quick-melting and corruptible foods such as ice cream and chocolate can also cause diarrhea if not stored properly.

Chronic Diarrhea lasting more than two weeks may be inflicted by a more serious condition and requires further investigation. Conditions such as

immunodeficiency, AIDS, radiotherapy and some specific GI diseases can cause this. Chronic diarrhea can also be caused by diseases such as untreated dysentery, irritable bowel syndrome (IBS), and pancreatitis.

Large Bowel Infection (Infectious Colitis, IC)

IC can be precipitated by bacterial or viral agents, or parasites and represents a form of inflammatory diarrhea. Most cases with IC manifest with acute purulent, bloody, and mucoid types of diarrhea. Fever, tenesmus, and AP are also encountered in a majority of patients with IC.

Causative Agents for IC: Table 2 summarizes the agents isolated in patients with IC.

Table 2. Causative agents isolated from stool samples of patients with infectious colitis.

Bacteria	Viral	Parasitic Infestation	Sexually Transmitted Diseases in Rectum
<i>Campylobacter jejuni</i> (most common) <i>Shigella</i> <i>Salmonella</i> , <i>Escherichia coli</i> , <i>Clostridium difficile</i> , <i>Yersinia enterocolitica</i> <i>Mycobacterium tuberculosis</i>	<i>Norovirus</i> , <i>Rotavirus</i> , <i>Adenovirus</i> , <i>Cytomegalovirus (CMV)</i>	<i>Entamoeba histolytica</i>	<i>Chlamydia trachomatis</i> <i>Neisseria gonorrhoeae</i> , <i>Treponema pallidum</i> Herpes simplex,

Short Notes on Specific Agents

Campylobacter Jejuni is the leading causative agent among bacterial agents inciting diarrhea throughout the world with a prevalence of around 30 / 100000 people (Azer, 2020).

Salmonellae are another commonly isolated agent which is responsible of 1.2 million patients with non-typhoidal salmonellosis diagnosed in the US every year.

Shigellosis is also important with severe clinical course and systemic complications. Although the global incidence is still high (around 170 million patients), attributed death rates are estimated to have decreased nowadays. Huge developments in laboratory techniques in recognition and management are thought to have resulted in this achievement.

Shiga Toxin-producing *E. coli* O157: H7 represents a prominent food and waterborne agent that causes diarrhea, hemorrhagic colitis, and hemolytic-uremic

Specific Diagnoses and Management Principles of the Intestines and Lower Digestive Canal

Abstract: Acute appendicitis, visceral perforations, diverticulitis (including bleeding and abscesses) acute calculous cholecystitis, acute ischemic bowel, mesenteric artery ischemia and infarction can cause acute abdominal conditions which prompt emergency interventions. Inflammatory bowel diseases (ulcerative colitis and Crohn's disease) may be followed up in some time without remarkable complications, although at some point with abscesses, hemorrhagic diarrhea and acute abdominal syndromes. However, the differential diagnosis (DD) of patients presenting with acute abdominal pain is much broader than this, including many benign conditions as well. Some etiologies of abdominal pain such as cholangitis strangulated hernias, colonic diverticulitis, perianal/ perirectal abscesses and fistulas may progress and turn into life-threatening conditions like abdominal sepsis without proper management.

Keywords: Acute abdominal pain, Anal abscess, Anal fistula, Acute appendicitis, Colonic diverticulitis, Surgical abdomen, Visceral perforation.

ACUTE APPENDICITIS (AAP)

(Information is also Provided in the Introductory Chapters)

It is one of the most common causes of acute AP and undoubtedly the most common etiology of surgical acute abdominal syndromes. Diagnosis becomes difficult at the extremes of age and during pregnancy. There are certain diagnostic misdiagnosis rates at all ages. It can be confused with many entities, causing missed or delayed diagnoses. PID in women and ureteral stones in men are the most common misdiagnoses.

History: AP with severe, colicky pain in the middle of the abdomen often accompanied by nausea and vomiting is the classic history of onset. Pain that migrates to the right iliac fossa, a.k.a. McBurney point after a certain period is typical, although not the rule.

A substantial proportion of atypical presentations are recorded. Some patients present with diarrhea or dysuria/pollakiuria. The location of appendix affects this situation (retrocecal 74%; pelvic 21%; paracecal 2%; others 3%).

EXAMINATION

While there may be few or subtle findings in the early stage in AAP, a patient with a poor general condition, septic shock or generalized peritonitis may be encountered in the advanced stage. In patients between these two extremes, fever, tachycardia, decreased/loss of appetite, guarding in the right lower quadrant, Rovsing sign, tenderness in the rectal exam in the right and proximal (close to the appendix) part can be noted. It should be known that the findings will not be apparent and definite in cases with plastron appendicitis. Especially in elderly patients, ambiguous findings, subfebrile temperature, moderate pain, and palpable mass may be noted (Table 1).

Table 1. The predictive values (pooled likelihood ratios) of the findings in the history and examination in diagnosing appendicitis (Andersson, 2004).

Signs/symptoms	Positive likelihood ratio (LR+)	95% confidence interval	Negative likelihood ratio (LR-)	95% confidence interval
Age \geq 20	1.25	(1.10–1.42)	0.74	(0.62–0.89)
Male sex	1.62	(1.49–1.76)	0.62	(0.57–0.68)
Vomiting	1.63	(1.45–1.84)	0.75	(0.69–0.80)
Migratory pain	2.06	(1.63–2.60)	0.52	(0.40–0.69)
Indirect tenderness (Rovsing)	2.47	(1.38–4.43)	0.71	(0.65–0.77)
Rectal tenderness	1.03	(0.83–1.27)	0.96	(0.85–1.08)
Psoas sign	2.31	(1.36–3.91)	0.85	(0.76–0.95)
Rebound tenderness	1.99	(1.61–2.45)	0.39	(0.32–0.48)
Percussion tenderness	2.86	(1.95–4.21)	0.49	(0.37–0.63)
Guarding	2.48	(1.60–3.84)	0.57	(0.48–0.68)

The probability of diagnosis of AA can be predicted significantly using the Alvarado score (Table 2).

Table 2. Alvarado score is a tool recommended to be used in the preliminary diagnosis of appendicitis.

Migrating pain	1 point
Loss of appetite	1 point

(Table 2) cont....

Nausea or vomiting	1 point
Tenderness in the right iliac fossa	2 point
Indirect tenderness	1 poin
Fever (>37.3C)	1 point
Leukocyte count>10,000/mm ³	2 point
Left shift > 75% PNL	1 point
TOTAL	10 point

Descriptions and notes: Eight points and above obtained from Alvarado score indicates AAP in 96%. A score of 0 to 4 excludes AAP. Of note, scoring changes during pregnancy (see the relevant section).

Elderly: 1/3 of operated AAP in the elderly patient is already perforated at the time of diagnosis (Yeo, 2006). Mortality is higher compared to young people, mainly due to delays in treatment resulting from time losses until diagnosis. Pain localization to the right lower quadrant occurs as a late finding in the elderly. Distention mimicking small bowel obstruction is common. Elderly patients have also a higher likelihood to deteriorate due to missed or delayed diagnosis and succumb to sepsis and/or septic shock more commonly.

WORK UP

The presumptive diagnosis of AAP is made by clinical picture and follow-up, and the definitive diagnosis is made by pathological examination. Urine test and pregnancy test must be taken, but USG and CT may be ordered in accord with the patient's condition. Although an increase in leukocytes is usually expected, a diagnosis cannot be made based solely on this, since it will also ensue in other diagnoses such as intra-abdominal abscess or PID. Since leukocyturia may also occur in patients with AAP, a patient with suspected AAP who has not been diagnosed with urinary tract infection (UTI) and whose pain has not resolved should not be discharged.

AAP and CT: MDCT is viewed as the diagnostic modality of choice to diagnose most acute abdominal diseases including AAP. Satisfactory figures of sensitivity (91% to 94%) and specificity (90% to 95%) as a diagnostic study for AAP were achieved, respectively.

How can I Diagnose AAP? Established Criteria are as Follows

An enlarged appendix (greater than 6 mm in diameter)

Wall thickening over 2 mm,

CHAPTER 6**Specific Diagnoses and Management Principles of the Hepatobiliary and Pancreatic Diseases**

Abstract: Hepatobiliary and pancreatic diseases are among common illnesses which cause major morbidity and mortality in the middle-aged and elderly patients and some specific subpopulations. Some geographic predispositions also exist for some diseases. For example, pain, fever, jaundice, and hepatomegaly can be noted in hydatid cyst disease which may cause allergic reaction and portal hypertension in the Southeast Europe and the Middle East. Of note, hepatobiliary and pancreatic diseases are commonly confused with each other, which may complicate diagnostic and therapeutic processes. A patient with biliary stones may be asymptomatic or suffer from acute or chronic cholecystitis, biliary colic, obstructive jaundice, cholangitis, mucocele, empyema, acute pancreatitis, gallstone ileus, and carcinoma. Cholecystitis and cholangitis are among diseases with high morbidity especially in the elderly and thus need to be ruled out in any patient with abdominal pain evaluated in acute and primary care setting. Some diagnostic clues are extremely helpful, such as Charcot triad which suggest severe cholecystitis (right upper quadrant AP, jaundice and fever) or cholangitis when complicated by altered mental status and hemodynamic instability. Acute pancreatitis refers to acute response to injury of the pancreas is referred to. Chronic pancreatitis, on the contrary, results from permanent damage to the endocrine and exocrine functions of the gland. Ultrasound, computed tomography and magnetic resonance imaging are among invaluable tools in diagnosing these diseases, together with specific laboratory adjuncts such as serum lipase for pancreatitis and bilirubin for obstructive jaundice. Definitive treatment encompasses surgical procedures, mostly in patients with acute abdomen due to gallstones or pancreatic necrosis.

Keywords: Acute pancreatitis, Acute cholecystitis, Biliary tract diseases, Cholangitis, Hepatobiliary diseases, Hydatid cyst disease.

LIVER DISEASES**Hydatid Cyst**

It consists of the larvae of the Echinococcus parasite (*E. granulosus* and *E. Multilocularis*). Sheep carry it, and it is transmitted to humans by dogs eating the uncontrolled meat (esp. liver) of these sheep. Infection is usually acquired in childhood, and it rarely presents clinically before the fourth decade of life.

Larvae → portal vein → liver (in 75% of cases)

- **In E. Granulosus**, which is more common in most parts of the World including Southeast Europe and the Middle East, hydatid cysts grow up to 1 cm in the first 6 months and grow 2 to 3 cm every year. The outcome is better than the other subspecies.
- **In E. Multilocularis**, the larvae proliferate and penetrate the surrounding tissue. They cause a diffuse and infiltrative granulomatous reaction and may mimic **malignancy**. It progresses as **necrosis** → **cavitation** → **calcification**.

Microscopic Features of Hydatid Cysts Include

- Cyst fluid: Antigenic, light yellow, neutral pH
- Endocyst: Forms daughter vesicles/capsule may detach, revealing sediments or daughter cysts.
- Ectocyst: Acellular substance secreted by the parasite
- Pericyst: granulation/fibrosis tissue layer formed by host response

Clinical Presentation

Cysts are initially asymptomatic. Symptoms appear as the size increases or infection or rupture develops.

Pain, fever, jaundice, hepatomegaly can be noted (therefore may be confused with biliary tract disease, cholecystitis/cholangitis).

May cause allergic reaction and portal hypertension.

Typical clinical profile: Middle-aged (farming) patient presents with sudden-onset right upper quadrant pain, jaundice, and palpable mass.

Diagnostic Checklist

Hydatid cysts can masquerade (or are masqueraded by) biliary cystadenoma, pyogenic liver abscess, cystic metastases, hemorrhagic/ infected cysts. Both the imaging and clinical courses of E. multilocularis mimic a solid malignant neoplasm.

Lab: - eosinophilia, boosted serological titers are detected in 80% of patients.

- Increased levels of alkaline phosphatase (ALP) and GGT.

Presumptive diagnosis is confirmed by advanced **imaging**. First of all, USG should be performed (Fig. 1).

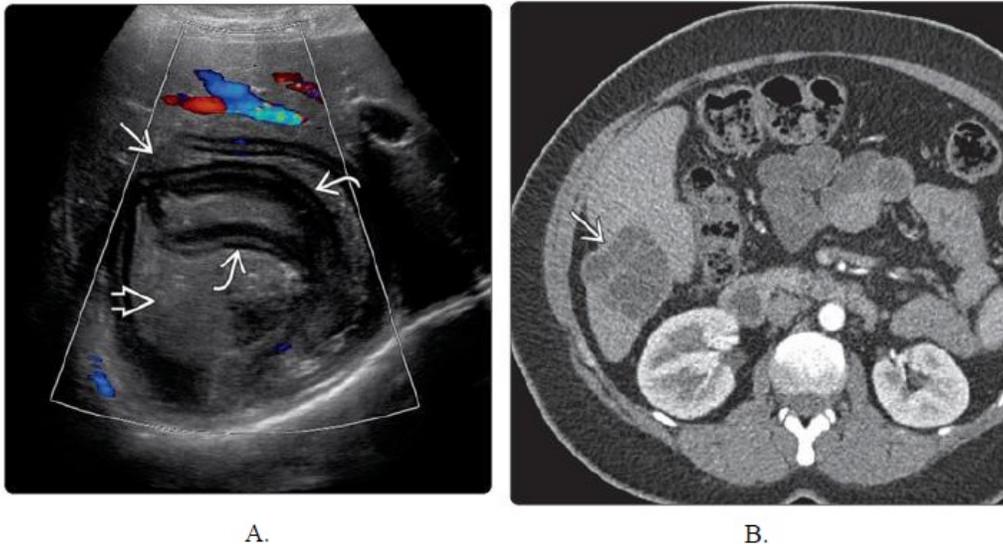


Fig. (1). **A.** Oblique abdominal color Doppler USG shows hepatic echinococcal cyst (arrow) and fine echogenic debris (hydatid sand) (hollow arrow). Separation of the endocyst membrane causes the membranes to float within the pericyst, what we call the “water lily” sign (curved arrow). **B** Axial abdominal contrast-enhanced CT shows a multiloculated cystic mass (arrow) in the right lobe of the liver.

Percutaneous cyst aspiration should be performed carefully for there is a risk of peritoneal spillage and anaphylaxis.

Tips for Radiology

Daughter cysts float inside the main cysts, and their situation changes when the patient changes his/her position.

Treatment

E. granulosus

- **Medical:** Albendazole/mebendazole
- **Direct injection** of scolicid agent
- **“PAIR”** procedure: Puncture, Aspiration, Injection, Respiration
- **Surgery:** Segmental or lobar hepatectomy
- **E. multilocularis**

Specific Diagnoses and Management Principles of the Urinary and Genital Tract Diseases

Abstract: Urinary tract infections (UTIs) and genital tract diseases (GTD) are among the most common infectious diseases with female predominance. On the other hand, acute epididymitis and orchitis are the most common GTDs which cause scrotal pain in adult males. Testicular torsion is a true medical emergency with vascular compromise and mandates immediate intervention to beware of serious complications. Although a majority are self-limiting diseases which can be treated easily, rapid diagnosis and management of certain UTIs and GTDs are a must to prevent grave outcomes. The infections may inflict the lower and/or the upper parts of urinary tract which also determines the severity of the disease. The urinary stone disease generally presents with ureteral colicky pain, blunt flank pain, nausea/vomiting, and hematuria with a male predominance. Most patients are managed easily in the acute setting but some are prone to deterioration with protracted urinary obstruction and resultant renal damage. The utilization of reliable, easy-to-use diagnostic tools with high accuracy is the key to expedient detection, identification and treatment. Ultrasound provides invaluable information in point-of-care diagnosis of most urinary tract diseases in both sexes. Management should be individualized in accord with the patients' signs and symptoms, general status and outcome estimations.

Keywords: Acute epididymitis, Genital tract disease, Gynecological pain, Orchitis, Ovarian torsion, Pelvic inflammatory disease, Pyelonephritis, Testicular torsion, Ureteral colic, Urinary stone disease.

ACUTE EPIDIDYMITIS AND ORCHITIS (AEO)

AEO is the most common cause of scrotal pain in adult males. Men in their third decade in life constitute nearly half of all cases. It occurs with sexual transmission under 35 years of age, or with chlamydial / gonococcal infection. If the patient is over 35 years old, it is mostly accompanied by a urinary tract infection (UTI). *Escherichia coli*, and *Pseudomonas* are mostly involved in this group. In one series, epididymitis occurred with orchitis in 58% of patients (Kaver, 1990). Rarely, it can also be triggered by trauma or autoimmune diseases.

Mumps (epidemic parotitis) infection can lead to orchitis (14% to 35% of the involved patients) in postpubertal males (Azmat, 2021). When associated with mumps infection, orchitis ensues four to seven days after the emergence of parotitis (Trojian, 2009). Unaccompanied by AEO, it is almost always indicative of mumps. 1/5 to 2/3 of these patients suffer from bilateral orchitis. The dreadful complication of this appears to be **testicular atrophy**.

Orchitis is usually viral in young patients, while mumps and rubella are the most common causative agents (Kanda, 2014). In addition, coxsackievirus, varicella, echovirus, and cytomegalovirus can lead to orchitis. Bacterial orchitis can be caused by *Escherichia coli*, *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Staphylococcus* and *Streptococcus* spp. (Azmat, 2021). In sexually active males *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, and *Treponema pallidum* can also be isolated. There are also reports of orchitis caused by autoimmunity, which are classified as primary and secondary.

Acute epididymitis encompasses pain and scrotal swelling for less than 45 days, while chronic cases are symptomatic for longer than 12 weeks and are typically recognized with pain without scrotal swelling (Trojian, 2009).

Etiology varies with the patient’s age and the likely pathogens. AEO is manifested as the gradual onset of posterior scrotal pain that may be accompanied by urinary symptoms such as dysuria and urinary frequency (McConaghy, 2016).

How do we Evaluate Acute Scrotal Pain (ASP) in the Emergency Setting?

DD for the patient with ASP is summarized in Table 1. Empiric treatment with antibiotics for acute epididymitis is given in Table 2.

Table 1. Selected differential diagnosis of acute scrotal pain (Trojian 2009, Crawford, 2014).

Diagnosis	Most Common Presentation	Signs	USG/Color Doppler (CD) Findings
Epididymitis	Slow-onset ASP on the posterior side and swelling in a few days	Scrotal swelling or inflammation and tenderness of the epididymis; Prehn sign (+) (pain relieved by scrotal elevation); cremasteric reflex spared	Hyperemia, swelling, and increased blood flow of the epididymis on CD
Testicular cancer	Some cases can present with pain	Firm and tender nodule in the involved testis	Distinct mass involving the testis on CD
Testicular torsion	Sudden onset of severe unilateral ASP	High-riding testis, absent cremasteric reflex, increased pain with scrotal elevation	Absent blood flow on CD

(Table 1) cont....

Diagnosis	Most Common Presentation	Signs	USG/Color Doppler (CD) Findings
Torsion of testicular appendix	Sudden onset of ASP	Blue-dot sign (bluish discoloration of the scrotum, over the torted appendage), indicating infarction or necrosis	Appendage larger than 5 mm, spherical form, or enhanced periappendiceal blood flow on CD

Table 2. Empiric antibiotic therapy for acute epididymitis (Adapted from CDC, 2015).

Population	Most likely Pathogen(s)	Antibiotic
Children younger than 24 mo	Various	Antibiotic treatment for likely underlying enteric organism and referral to a urologist
Children 2 to 14 years of age	Various, +/- anatomic abnormalities	Treatment based on urinalysis and/or urine cultures
Sexually active men younger than 35 years	Gonorrhea or chlamydia	IM ceftriaxone (single 250-mg dose) <i>and</i> Doxycycline (PO, 100 mg twice daily for 10 days)
Adults who frequently commit insertive anal intercourse	Gonorrhea or chlamydia AND an enteric organism	IM ceftriaxone (single 250-mg dose) <i>and</i> Levofloxacin PO (Levaquin; 500 mg once daily for 10 days) or ofloxacin PO (300 mg BID for 10 days)
Adults older than 35 years or who have had recent urinary tract surgery or instrumentation	Enteric organism	Levofloxacin (PO, 500 mg once daily for 10 days) <i>or</i> ofloxacin (PO 300 mg BID for 10 days)

Clinical course: Pain in the testicle(s), increases steadily over time, which is also associated with swelling in the epididymis and testicular tissues. In advanced AEO cases, there is the triad of pain+swelling+tenderness on palpation.

History of fever, dysuria and urethral discharge may be obtained. Typically, the epididymis is tender, with the testicle located well below the scrotum. Abscess formation may be seen in delayed and untreated cases. Erythema and reactive hydrocele may be noted in the scrotal wall.

A positive Prehn sign (where the passive elevation of the scrotum reduces pain) is strongly supportive of epididymitis, contrary to testicular torsion. Cremaster reflex is intact.

Elderly men (>50 years) should be evaluated for urinary tract obstruction secondary to prostatic enlargement. All patients treated for an STD should be recommended to undergo treatment with their partners (CDC, 2015).

CHAPTER 8

“Chronic” Abdominal Pain in the Acute Setting: Functional Bowel Diseases, Irritable Bowel Syndrome (IBS) and Cancer-related Pain

Abstract: Chronic abdominal pain is a very common condition all over the world. Although not expected to present emergently, acute exacerbations of chronic pain or the slightest change that worsens the patient's condition (*e.g.* acute-onset diarrhea, vomiting, or loss of appetite) will trigger admissions to ED. Functional bowel diseases include irritable bowel syndrome (IBS, a.k.a. spastic colon), functional bloating, functional constipation, functional diarrhea, and unspecified functional bowel disorders. Epidemiologic, pathophysiologic and therapeutic studies of functional bowel diseases, employed the Rome Criteria with universal validity. Patients with malignancy can experience different types of cancer-related pain at any time during the disease process, perceived by the organs or systems involved.

Keywords: Abdominal pain, Cancer-related pain, Chronic abdominal pain, Functional bowel diseases, Irritable bowel syndrome, Spastic colon, Rome Criteria.

Evaluation and history should be focused towards differentiating benign functional disease from organic disease with serious outcomes. For example, significant recent weight loss, hypovolemia or cachectic appearance, signs of blood loss, and *de novo* anemia warrant further investigation to exclude organic etiology (Table 1). In contrast, the laboratory values are expected to be normal in the case of “functional” abdominal pain (AP). **Irritable bowel syndrome (IBS)** may be considered in a patient with bowel habit changes accompanying AP.

Iron deficiency, if diagnosed, should suggest celiac disease, IBD (including ulcerative colitis/ Crohn's disease) or malignancy. As an accurate biomarker, detection of **fecal calprotectin** supports the presumptive diagnosis of IBD and helps to direct patients to colonoscopy. **Chronic pancreatitis** may be the cause of chronic AP, especially in patients with a history of previous pancreatitis attacks, alcoholism or gallstones. The flip side is that some patients without a previously

diagnosed risk factors mentioned above can also present with recurrent AP which will some day be diagnosed with ‘chronic AP’ in the ED, as a challenge for the physicians.

Table 1. The following tests may be requested in the patient with chronic AP.

• CBC+ leukocyte formula
• Electrolyte, BUN, creatinine, glucose
• Calcium
• ALT/AST, alkaline phosphatase, bilirubin
• Lipase/amylase
• Serum iron, iron binding capacity, ferritin
• Transglutaminase

DD of IBS and other functional disorders should include a broad range of disorders causing AP at some point of the disease course (Table 2).

Table 2. Uncommon causes of abdominal pain include the following entities.

<ul style="list-style-type: none"> • AAA- Abdominal aortic aneurysm • Abdominal compartment syndrome • Abdominal migraine • Acute intermittent porphyria • Angioedema (hereditary or due to ACE inhibitor) • Celiac artery compression syndrome • Chronic abdominal wall pain • Colonic pseudoobstruction • Eosinophilic gastroenteritis • Epiploic appendagitis • Familial Mediterranean Fever (FMF) • Parasite-Helminth infections • Herpes zoster • Hypercalcemia • Hypothyroidism • Chronic lead poisoning • Meckel's diverticulum • Paroxysmal nocturnal hemoglobinuria (PNH) • Pseudoappendicitis • Causes of “pulmonary abdominal pain” (lower lobe pneumonia) • Rectus sheath hematoma • Renal infarction • Lower rib-related pain • Sclerosing mesenteritis • Somatization
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Irritable Bowel Syndrome (IBS); a.k.a. Spastic Colon

IBS is a functional disorder that causes occasional disturbances in the GIS and chronic AP associated with remarkable changes in bowel habits.

It is more common in the urban young population and women. Incidence figures of 5% to 20% have been reported in different geographic regions. The continent with the highest frequency is South America with 21%. It is rare over 50 years of age. The prevalence of IBS in Turkey has been found 5% in males, 7.4% in females and 6.3% overall, and there is a female predominance (around 2/3) (Celebi, 2004).

The mechanism that is most emphasized can be summarized as **“the nerves that carry messages from the gut to the brain are working too hard”** (Table 3).

Table 3. The mechanisms focused on the etiology of IBS are as follows.

● Changes in intestinal motility,
● Visceral hypersensitivity,
● Intestinal barrier dysfunction,
● Intestinal bacterial overgrowth and
● Changes in intestinal flora,
● Intestinal mucosal inflammation,
● Genetic and epigenetic changes,
● Brain-intestinal system interaction
● Psychosocial problems.

Findings in the History and Examination

There is colic/cramp-like AP with exacerbations in the history. Symptoms are mostly intermittent and there may be asymptomatic periods for weeks in between. The most disturbing ones following AP are the feeling of urgency to defecate, bloating and increased number of defecations.

The severity of attacks changes over time. The location and definition of AP also varies from patient to patient. There may be patients who are relieved by defecation, as well as those with tenesmus and increased pain with defecation. **Bloating** is a prominent complaint.

Medication history should be taken carefully, as the side effects of some drugs can cause bloating and mimic IBS. Oral antidiabetic use should be questioned, especially in diabetic patients as diabetic gastroparesis should also be in the DD.

Special Groups and Abdominal Pain

Abstract: Specific patient groups have inherent characteristics when they suffer from diseases, including those of the digestive system and other causes of abdominal pain. Both diagnostic features and treatment measures differ regarding the patient's age, sex, previous medical / surgical history, and comorbid diseases. Pregnancy has its unique features in both anatomy and physiology of the woman which result in substantial variation in physical examination finding, radiological and laboratory adjuncts (*e.g.*, the location of the appendix is shifted away from its usual site and computed tomography is hardly ever used to diagnose etiologies of abdominal pain in pregnant women). Likewise, children have many differences in presentation, examination findings, work up and treatment principles, complicating the management process.

In addition, the pandemic disease has caused a paradigm shift in the evaluation of almost all diseases, including those with abdominal pain. Many data suggest a close relationship between COVID-19 and the digestive system. Patients with COVID-19 carry a high risk of digestive symptomatology including abdominal pain, nausea and vomiting, diarrhea and others. HIV (+) patients exhibit various GI symptoms such as diarrhea, abdominal pain and proctitis.

Healthcare providers should have robust knowledge of various forms of presentations and characteristics of special subgroups with abdominal pain in this regard, to prevent misdiagnoses and treatment errors in those patients.

Keywords: Acute abdominal pain, Acute appendicitis, Acute cholecystitis, COVID-19, Diverticulitis, Ectopic pregnancy, Elderly, Human Immunodeficiency Virus (HIV), Invagination, Mesenteric ischemia, Mesenteric lymphadenitis, Intussusception, Pregnancy.

PREGNANCY-RELATED ACUTE AP

Although the etiologies of AP in pregnancy are essentially similar to other patients, it is necessary to pay attention to some key issues.

Ectopic pregnancy (EP) is the development of the gestational sac implanted outside the uterus. It is estimated that 5 to 10 out of 100 pregnancies can be

located ectopically. 96% of EPs are found in the ampulla or isthmus of the Fallopian tubes, 2% in the interstitial tissue of the uterus, and the remainder in the other intraabdominal sites.

EP is diagnosed most commonly in women who have had their first pregnancies or in multiparous women who have become pregnant for the first time after a long time.

EP is the most common cause of pregnancy deaths in the first trimester, resulting mostly from delayed diagnosis. Therefore, in every woman of childbearing age presenting with AP and/or vaginal bleeding, the diagnosis of EP should be presumed until it is ruled out. If the triad of pain+bleeding+syncope can be identified in a woman of child bearing age, the diagnosis is far more likely.

Among the risk factors are pathologies that slow down or stop the migration of the fertilized ovum to the uterus: PID, pelvic adhesions, endometriosis, *in vitro* fertilization, intrauterine device, use of progesterone-containing birth control pills, congenital anatomical variants, malignancies or other tumoral masses.

PATHOLOGY

EP Implanted In The Fallopian Tube can have 3 Potential Consequences

- Rupture towards the peritoneal cavity (tubal abortion)
- Spontaneous abortion (with varying degrees of pain and bleeding).
- Rupture into the tube (with varying degrees of pain and bleeding).

EP implanted in one of the uterine horns can be viable till the 10th to 14th weeks. Very rarely, there are intraperitoneal pregnancies reaching the term.

Clinical Presentation

The most common form of presentation is sudden onset lower abdominal pain and bleeding, associated with syncope or presyncope. Although not a rule, delayed menstruation can be noted. Nausea and vomiting are common. There may also be a history of irregular bleeding in different colors from time to time. Admissions can also be seen after one to four weeks of waxing and waning pain episodes.

EP risk factors, delayed menstruation, and pregnancy symptoms should be questioned, but none of them is a *sine qua non* for the diagnosis of EP. Findings compatible with hemorrhagic shock may be encountered. Fluid resuscitation is to be initiated even before the diagnosis is confirmed.

Abdominal findings can range from very mild pain to peritoneal irritation. Tenderness and sometimes a mass may be identified in bimanual vaginal examination if performed in the emergency setting.

The patients should be consulted with OB/GYN without waiting for verification of the diagnosis of EP.

Emergency Treatments and Consultations Should not be Delayed for the Results of the Laboratory Work Up

B-HCG levels are typically much lower than the expected gestational week, but the pregnancy test is positive.

Some recent studies investigated new markers such as serum protein calponin 2 (CNN2) as a biomarker for tubal EP (Zhang, 2021). Serum CNN2 concentration was found to be increased in those with EP than in women with viable intrauterine pregnancy (vIUP) and miscarriage. The serum CNN2 predicted EP from vIUP and miscarriage with areas under the curve (AUCs) of 0.931 (95% confidence interval: 0.889-0.975). Their data highlight the usability of serum CNN2 as a single marker for the diagnosis of EP.

Although transvaginal USG gives better results, it can also be started with transabdominal USG and findings such as significant Intraabdominal free fluid (pelvic hematocele) accelerate the diagnosis and management (Table 1). On the contrary, visualization of an intrauterine pregnancy help ruling out EP and mandate a search for other DD which can explain the patient's findings such as a missed abortion, placenta previa, *etc.* (Fig. 1).

Table 1. Findings that increase the likelihood of a diagnosis of EP with a compatible clinical scenario.

1 Extruterine gestational sac, containing yolk sac or embryo
2 Adnexal ring sign
3 Adnexal mass
4 Significant free fluid in the abdomen, including Morison's pouch (hepatorenal space)
5 Complex free fluid (suggesting hemoperitoneum)
6 Pseudogestational sac
7 Weak decidual reaction
8 Blood in the endometrium

Extraabdominal Causes of Abdominal Pain

Abstract: Diabetes mellitus (DM), chronic renal failure (CRF), amyloidosis, sickle cell anemia (SCA) and acute intermittent porphyria are among diseases that can be associated with abdominal pain (AP) at some point in the course of the pathological process. Diabetic ketoacidosis (DKA) is a severe life-threatening syndrome characterized by fluid loss, electrolyte changes, hyperosmolarity and acidosis. These pathophysiologic factors can explain AP in patients with DKA. Vomiting and AP can also be initial manifestations of DKA even in euglycemic patients.

SCA is one of the most common autosomal recessive diseases classified in hemoglobinopathies. The disease is first recognized by history, then by peripheral smear and hemoglobin electrophoresis, and advanced studies. Splenic sequestration crisis is a severe complication of SCA that prompts emergent treatment, Opiate analgesia and hydration is the main treatment.

Patients with chronic renal failure (CRF) and end-stage renal disease are also prone to severe AP due to peritonitis which is triggered by continuous ambulatory peritoneal dialysis in vulnerable patients. Amyloidosis is mostly recognized with typical attacks *i.e.*, febrile episodes, exanthema, AP, myalgias and arthralgias.

Acute intermittent porphyria is an autosomal dominant disorder characterized by severe neurovisceral attacks of AP, nausea, vomiting, tachycardia, and hypertension in the absence of signs compatible with peritonitis. Management of mild attacks comprises symptomatic treatment, optimized calorie intake, and fluid replacement to beware dehydration.

Keywords: Abdominal pain, Acute intermittent porphyria, Amyloidosis, Chronic renal failure, Diabetes mellitus, Peritonitis, Porphyria, Sickle cell anemia, Splenic sequestration crisis.

DIABETIC KETOACIDOSIS (DKA)

DKA is a syndrome characterized by fluid loss, electrolyte changes, increased osmolarity due to high levels of plasma glucose, and a shift in blood pH to acidosis. All these reasons may trigger AP along with dehydration in the peritoneum. As a result, the acute AP emerges and can be seen as an entity that can masquerade all other etiologies of AP.

DKA occurs with either a sudden increase in the need for insulin (such as pneumonia, other infections, stress, overwork) or a decrease in insulin production and secretion (such as pancreatitis, pancreatic insufficiency, sudden cessation/decrease in previously used insulin therapy).

Although DKA is mostly seen in childhood diabetes, which we call type I diabetes, it is also seen in seniors with type II. DKA may be the first sign of diabetes in one of 4 cases.

The severity of the clinical findings in DKA is not proportional to the level of hyperglycemia.

The main findings of DKA are dehydration, Kussmaul breathing, and unconsciousness. Ketones in the urine and high blood sugar associated with low pH also support this. Of note, children with Type-I Diabetes and severe DKA were found to be more likely to report vomiting, fatigue, and abdominal pain, but less likely to report polyuria, polydipsia, and polyphagia than those with mild/moderate DKA (Peng, 2021).

AP in the course of DKA is usually generalized and radiates throughout the abdomen, but sometimes it can be confined to a specific region.

Especially in children and those with communication problems, signs of dehydration should be sought by the physician (Table 1). Interestingly, vomiting and AP can be initial manifestations of DKA even in euglycemic patients with long-term diabetes (Mumtaz, 2020)

Table 1. Evidence of significant dehydration (at least 5%).

● Extended capillary refill time (> 2 sec)
● Abnormal skin turgor
● Hyperpnea.
Adjunctive findings:
● Sunken eyes
● Dryness of mucous membranes
● Absence of tears
● Scummy extremities

Fluid replacement, correction of acidosis, and elimination of electrolyte imbalance which are the cornerstones of the treatment of DKA, will also treat pain. Likewise, altered mental status attributed to severe DKA also improves with treatment.

Alternative and additional diagnoses should also be excluded. ACS/AMI, CVA, acute heart failure, pyelonephritis, pneumonia, adrenal insufficiency, and thyroid problems that may trigger or accompany DKA should be investigated.

Wide-bore vascular access is opened bilaterally, preferably in the upper half of the body. There is no additional advantage of central vascular access. On the contrary, peripheral vascular access such as antecubital or external jugular veins should be preferred for rapid fluid infusion. 1 L of crystalloid fluid is given in the first hour, and 500 mL to 1 L of crystalloid fluid in the next several hours. Thus, 3.5-5 L is given in the first 5 hours. For the treatment of severe dehydration attributed to DKA, 6-12 L of fluid should be given in a total of 24 hours. Fluid therapy and insulin need to be administered simultaneously.

IV insulin infusion is given at a rate of 0.1 to 0.14 IU/kg/hour. When blood glucose readings fall below 250 mg/dL, insulin is stopped and 5% dextrose infusion is commenced. The HCO₃ level should be kept above 18. The additional infusion of HCO₃ is not helpful at pH values above 7.0 or 7.1, thus not indicated at all. On the contrary, it can deepen hypokalemia and cerebral complications.

- If serum K < 3.3 mEq/L, K replacement can take priority, insulin can be stopped and potassium solutions can be infused at 20-30 mEq/hour, with due caution.
- If serum K is between 3.3 and 5.3 mEq/L: 20 – 30 mEq K is put into each liter of IV crystalloids and administered to maintain blood K level between 4 and 5 mEq/L.
- If serum K > 5.3 mEq/L, K is not replaced, and the levels are frequently checked.

The Most Common Complications of DKA Treatment are Hypoglycemia and Hypokalemia

The serum phosphate level is also important. Levels below 1.5 mg/dl [0.48 mmol/L]), warrant oral replacement if the patient's consciousness is satisfactory to ingest the tablets.

If there is significant cerebral edema, 0.3 g/kg mannitol can be infused and titrated upwards. The patient must be admitted to the intensive care unit.

The reduction of ketone bodies in the urine is also one of the indicators that the treatment is successful, but it can also be misleading.

Non-diabetic ketoacidosis, on the other hand, is also a source of ketone bodies and can present with AP as well (Bashir, 2020). Starvation ketoacidosis and alcoholic ketoacidosis are subtypes of non-diabetic ketoacidosis.

Abdominal Trauma and Pain

Abstract: Trauma is the most common cause of death in the young population, predominantly males. Abdominal trauma is a leading source of occult bleeding which is the second cause of early-phase deaths following major head injury. Uncontrollable bleeding constitutes the most common cause of preventable deaths especially if the management of shock is delayed. Penetrating trauma leads to significant morbidity and mortality, nonetheless, diagnosed more easily with its remarkable presentation.

The main goal in the evaluation of the abdomen in the acute setting is to uncover (*i.e.*, not to overlook) the injuries requiring surgery without delay, rather than to diagnose specific injuries in detail. Signs and symptoms of progressing shock states vary from patient to patient, and sometimes very subtle changes can herald impending doom.

The clinician should be proactive in detecting the injuries, using both evaluation findings and bedside ultrasound together with other advanced imaging techniques when necessary, keeping in mind that occult injuries can evolve in time insidiously. Ongoing intraabdominal bleeding is an ominous finding which precedes advanced hemorrhagic shock and needs to be sought for carefully in patients with trauma.

Keywords: Abdominal pain, Abdominal trauma, Bleeding, Computed tomography, Fluid management, Hemorrhagic shock, Injury, REBOA, Resuscitation, Rupture, Shock, Trauma, Ultrasound.

Abdominal trauma (AbT) is a type of injury that requires attention due to the difficulties in the evaluation as well as its course with a significant morbidity and mortality rate. Although the main title of AbT is a very general scope, there are countless different combinations of injury possibilities and presentations within this context. Likewise, inspection and examination have their own difficulties. Our goal in the evaluation of the abdomen is to identify (or not to miss) the injuries requiring surgery without delay, rather than to diagnose specific injuries. The most serious error in AbT is not missing a specific diagnosis, but the failure to pursue or delay the surgical intervention mandatory for stabilization. The most common preventable deaths occur as a result of uncontrollable bleeding due to delayed management. Many lesions are related to intra-abdominal injuries within the concept of the 'golden hour'.

A focused history coupled with a complete physical examination and evaluation of the patient is essential. As a bedside test, point-of-care ultrasonography (USG/POCUS) is widely used by clinicians and is a regular part of physical examination. While diagnostic peritoneal lavage (DPL) was used in the past to evaluate abdominal trauma in the diagnosis of the unstable patient, it has now been almost abandoned with the development of both USG/POCUS and whole-body computed tomography (CT) scanning and MDCT. However, retroperitoneal injuries still represent a challenge in recognition in the emergency setting. CT imaging has replaced DPL in specific clinical situations and is invaluable in diagnosis. No test can replace the need for careful clinical evaluation and monitoring for the development of signs of serious injury. In recent years, diagnostic thoracoscopy and laparoscopy have been increasingly used in the evaluation and treatment of AbT (Table 1).

Table 1. Indications for laparotomy in abdominal trauma.

Absolute (Prepare the OR immediately)
Ongoing severe hemodynamic instability, +/- positive FAST result
Generalized peritonitis (guarding, rigidity)
Evisceration
Relative indications (Consult trauma surgeon)
Free air image on X-ray or CT (can stem from thorax or other sources apart from abdominal trauma)
Signs of GI bleeding on NG or rectal exam
Penetrating abdominal trauma: firearm injuries (mostly operative) and stabbing wounds (can be operative)
Multisystem trauma + Ongoing hemodynamic instability, source of injury not defined yet

One of the most important points is to consider the victim as having multiple trauma, even in the case of presentation with significant AbT. The completeness of both the primary survey summarized with ABCDE mnemonic and the secondary survey formulated as “head-to-toe” is essential. Otherwise, non-abdominal vital injuries may be missed.

PATHOPHYSIOLOGY

Abdominal injuries mainly develop *via* two mechanisms: Blunt and penetrating trauma. It is also important to remember that these two types of injury can coexist. For example, a person with a stab wound on the chest may also have been bluntly beaten to lacerate his spleen. Similarly, there may be significant blunt trauma as well as penetration by sharp objects in a motor vehicle injury. Thus, the clinician should not set limit his/her vision with the easily seen.

Blunt Trauma

There are three mechanisms of injury by blunt trauma:

1. direct impact
2. crush injury
3. acceleration-deceleration injury. These types of injuries can also be found in a double or triple combination.

The abdomen consists of the peritoneal cavity and the extraperitoneal space. The extraperitoneal space includes the retroperitoneum and the extraperitoneal pelvis, which are hard to examine and evaluate, since their injuries usually cause minimal findings on physical examination. Peritoneal lavage is unreliable for injuries in this area and negative lavage may give a false sense of comfort. Ancillary secondary tests are more useful in the evaluation of the retroperitoneum and pelvis. Intravenous pyelography (IVP), upper GI contrast studies, cystography, angiography, and CT imaging will all be useful in selected cases. Oral and IV contrast-enhanced CT imaging is the single most useful test in the evaluation of retroperitoneal organs such as the kidney and pancreas (Fig. 1). Although USG can also be used in the retroperitoneum, care should be taken in interpretation.

Rectal digital exam and stool occult blood test can be interpreted in selected cases to evaluate distal bowel injuries. Fresh blood coming directly to the finger on the digital exam may mean pelvic fracture and perforation of the rectum, which prompts further investigation.

Serum amylase level is a useful predictor of potential injury, but normal amylase level can be seen even in the presence of significant pancreatic injury.

In case of injury in the peritoneal space, there are usually obvious findings on physical examination of the abdomen, as the peritoneum responds to irritation very rapidly. Findings such as tenderness, guarding, or palpable mass raises suspicion of injury.

CHAPTER 12**Treatment and Resuscitation of the Patient with Acute Abdominal Pain**

Abstract: Provision of airway patency, effective breathing and gas exchange and circulatory functions producing adequate perfusion (ABC) are vital elements in all emergent and critical patients. Initial resuscitation should begin with control in a primary survey both in the field and in the hospital in the management of the patients with abdominal pain. In a patient whose respiratory patency is under threat, evaluation and management of the inflammatory process in the abdomen should not be considered before this is resolved. Differential diagnosis and proper management of abdominal pain follow the primary survey, resuscitation and resolution of vital threats. IV fluid therapy and pain management are commenced as prompted by the general condition. Prehospital providers should operate in communication with the command control center in this context. “Tubes or fingers for all orifices” can be accepted as a general approach for the moribund patient to monitor the clinical course. In the hospital, the emergency physician should relieve the pain expediently after evaluating and recording the initial vital signs and findings on systemic examination. Antiemetic therapy and other symptomatic measures should be individualized for the given patient.

Keywords: Abdominal pain, Airway, Breathing, Circulation, Management, Resuscitation.

As in all emergency and critical patients, initial resuscitation should begin with airway, breathing and circulation (ABC) control in a primary survey both in the field and in the hospital (Table 1). In a patient whose respiratory patency is under threat, evaluation and management of the inflammatory process in the abdomen should not be considered before this is resolved.

MANAGEMENT IN THE HOSPITAL

When performing the first-line resuscitative interventions for diseases that will require operative treatment, a quick contact should be made with the general surgery consultant (+cardiovascular surgery for vascular pathologies, +OB/GYN

for gynecological or obstetric problems). About 1/3 of patients with AP presenting to the ED are consulted, and eventually around 1/10 are hospitalized. For this reason, the education of front-line physicians about AP is critical.

Table 1. Treatment steps and mainstays in the prehospital area. EMS command control center (CCC) instructions are followed throughout all procedures.

IV fluid therapy is started as prompted by the general condition. Two wide-bore (16-14G) vascular accesses should be opened from the antecubital or equivalent area in the upper half of the body. Femoral or calf veins are not preferred.
Cardiac rhythm and pulse oximetry are monitored continuously.
Give supplemental O ₂ . Additional O ₂ is given by 100% face mask or nasal cannula based on findings if SpO ₂ is below 94%, or in those with symptoms such as fever, tachycardia, agitation, ischemic chest pain, or unconsciousness.
IV analgesic agents, preferably opiates (morphine/fentanyl) are titrated to effect.
Since abdominal pain may be a sign of ACS, especially over the age of 40, an ECG should be obtained and interpreted.
Anti-emetics (preferably ondansetron and/or H ₁ antagonists or metoclopramide 10 mg <i>via</i> slow infusion) may be given.
Antibiotherapy should be started as early as possible according to local protocols in patients who are prepared to undergo operation and in those with suspected sepsis. Empirical combination therapy takes priority in emergency situations.
Insert NG tube.
Monitor urine output with a Foley catheter.
Admit the patient to intensive care / general surgery beds.

Hypovolemic or septic shock should be rapidly recognized and resuscitated. In addition, perhaps more important is the recognition of the case that will succumb to shock (eg ruptured spleen or ruptured aneurysm) even though clinical findings of shock are not obvious.

How much of which Liquid Should we Give in Which Way?

Since there may be external (bleeding, vomiting, diarrhea) or internal (sequestration to the third cavities in the abdomen) fluid loss, the volume status should be evaluated urgently and fluid replacement should be initiated as soon as possible. Crystalloid fluids (normal saline and lactated Ringer's solution) take priority for initial resuscitation. Colloid fluids have not been found to have any advantage over crystalloids, on the contrary, they can cause coagulation problems (*e.g.*, Dextran solutions).

For this, at least two antecubital wide-bore (16 or 14 G) vascular accesses must be opened. Peripheral wide vascular access is superior to central catheters in almost every aspect except for indications of CVP monitoring.

20 mL/kg formula is a concrete strategy that can be employed initially in every case including weight extremes in hypovolemic patients with the poor general conditions. In an elderly patient with suspected heart failure, the behavior of not giving fluid paves the way to hypovolemia and hypotension. In these cases, starting with 100 to 150 ml/hour, the response to fluid resuscitation can be titrated within a few hours by monitoring the inferior vena cava with bedside USG. In other hypovolemic/hypotensive cases, a bolus of 20 mL/kg of fluid can be infused. For patients with acute blood loss, the 10 mL/kg formula for the initial dose of blood transfusion is more suitable.

A nasogastric (NG) tube is indicated for evacuation and decompression of gastric contents since aspiration may be common in acute abdominal syndromes with persistent vomiting such as mechanical bowel obstruction and pancreatitis. Caution should be exercised in the insertion of NG catheter in EVB, for it may rarely trigger bleeding. Nonetheless, it is not contraindicated. It should be inserted after endotracheal intubation in patients with impaired consciousness. In patients with injuries to the head and/or maxillofacial area involving the midface, it should not be inserted without ruling out a basilar fracture of the skull.

In critically ill patients, who may be prone to hypovolemia, monitoring urinary output *via* catheterization will be useful. The same is true in cases such as postrenal obstruction, and/or prostatism. In cases where there is difficulty in defecation due to anal/rectal mass, and/or fecaloma(s), a rectal tube can be placed to ease excretion.

ACUTE PAIN MANAGEMENT

In cases presenting with acute AP, the emergency physician should relieve the pain expediently after evaluating and recording the initial vital signs and findings on systemic examination. There are no contraindications for cessation of pain in a patient with pain. A point to be noted is that acute AP of inflammatory origin can be disguised or 'masked' by anti-inflammatory agents and cause misdiagnosis. Therefore, some authors emphasized that NSAID group drugs such as ketorolac and tenoxicam should not be used in undiagnosed patients with AP. A meta-analytic study disclosed that postoperative IV ketorolac infusion may provide substantial pain relief for most patients, but further research may impact this estimate (McNicol, 2021). Adverse events are recorded at a slightly higher rate when compared to placebo and other NSAIDs.

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