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Sepsis and infections in surgery

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Chapter 124

Diagnosis of intra-abdominal infections

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Introduction

The issue of acute surgical pathology remains a significant concern globally, with persistently high mortality rates and a growing burden on healthcare systems in many countries. The systems of emergency surgical care for patients with intra-abdominal infections (IAI) in many countries of the world differ significantly in their diagnostic and tactical approaches to this category of patients. To facilitate the effective treatment of these patients and to prevent postoperative complications, specialists dealing with these problems must communicate in a common language. In this regard, it is necessary to introduce common concepts at all stages of medical care for this category of patients. Although there is variability in the spectrum of surgical diseases in different countries, the basic concepts of emergency abdominal surgery provide a foundation upon which unified guidelines can be formed. This is particularly evident in the areas of low-income countries, where there are significant deficiencies in the availability of effective surgical care.

The importance of prompt identification of intra-abdominal infections is evident. The primary characteristic of IAI is the rapid progression of endogenous intoxication (EI). In the context of EI, the accumulation of biologically active pathological products – endogenous toxic substances (ETS) – occurs in the tissues and fluids of the body. The sources of ETS are typically as follows: (1) Pathologically functioning organs of the endocrine system; (2) Metabolic products of xenobiotics entering the body from the external environment; (3) Focus of tissue destructive and the areas of ischemia with massive cell death; (4) Primary and secondary infectious-toxic foci; (5) All organs and tissues under hypoic conditions; (6) Endotoxins of microbes when activated in areas of natural vegetation (intestines, etc.); (7) Medications, etc. As a rule, ETS damages all blood cells and tissues of the body, including the endothelium; changes the permeability of cell membranes, and sodium-potassium balance; disrupts vascular tone, and microcirculation of blood, lymph and cerebrospinal fluid; inhibits the function of systems of erythropoiesis, immunity, tissue respiration, synthesis and transport of amino acids; disrupts the function of systems regulating body homeostasis (lipid peroxidation, antioxidant system, etc.). Tension of the abdominal wall muscles indicates the presence of peritonitis. Hypotension and

signs of hypoperfusion, including lactic acidosis, oliguria and acute disturbance of mental status, indicate the onset of sepsis.

The emergency department diagnostic evaluation for patients with intra-abdominal infection consists of the following. The first step in identifying patients with suspected IAI is through a history, physical examination, and laboratory tests. Secondly, patients with inconclusive results in the previous stage of the study, with the presence of central nervous system disorders, and patients with suppressed immunity due to various diseases and treatments with signs of intra-abdominal infection should be considered with emphasis on its confirmation or refutation. Stable patients who have been receiving fluid resuscitation and who cannot have an immediate laparotomy should consider imaging with contrast-enhanced computed tomography as the third option. It is not appropriate to continue with further instrumental diagnosis if there is evidence of diffuse peritonitis requiring emergency laparotomy in patients. Patients with clinical signs of intra-abdominal sepsis should have their severity assessed before surgery based on Sepsis-3 recommendations. Blood cultures do not provide additional clinically relevant information in patients with intra-abdominal infections and are therefore not routinely recommended.

Determination of bacteraemia may be useful in a strategy to determine the duration of antimicrobial therapy when used in conjunction with biomarker studies (procalcitonin, others). Routine Gram staining of infected material in patients with intra-abdominal infection has no proven diagnostic value. In low-resource emergency departments, the diagnosis of intra-abdominal infection is based primarily on clinical findings, supported by basic laboratory tests such as a complete blood count and a differential. Ultrasound, even when available, is not always helpful in diagnosis. Therefore, clinicians must carefully assess each sign and symptom to optimize diagnosis. In recent years, the use of ultrasound has increased and ultrasound scanners have become smaller, more accessible and less expensive. Ultrasound is highly reproducible and easily repeatable but remains highly operator-dependent. In different areas of resource-limited where access to CT is limited, plain radiography and ultrasound are resources that can assist in the diagnosis of emergency abdominal surgical pathology. CT may be useful when the diagnosis is unclear in developed countries, CT has become the gold standard. In instances where abdominal CT and ultrasound do not yield the requisite information, diagnostic peritoneal lavage may be employed as a means of detecting peritonitis. The resulting abdominal contents should be collected for diagnostic studies, with a particular focus on the determination of aerobic and anaerobic bacterial cultures using urgent cytological and bacteriological techniques. Diagnostic laparoscopy is a highly accurate diagnostic test for intra-abdominal pathology and is the recommended diagnostic procedure for patients with abdominal sepsis of unknown origin. It is a widely employed diagnostic procedure for determining the underlying pathology of acute abdominal pain and if surgical pathology is detected, laparoscopic treatment may also be employed. The accuracy of diagnostic laparoscopy is extremely high, with a range of 86-100%. As is well established, all IAIs are categorised as uncomplicated or complicated. It is also known that peritonitis in IAI can be localised or generalised. The occurrence of peritonitis can be attributed to the entry of any microorganism into the abdominal cavity. However, each microorganism exhibits a specific quantitative characteristic and a set of conditions under which peritonitis is likely to occur. This assertion is predicated on the premise that the peritoneum possesses bactericidal and immune properties.

Currently, peritonitis is typically caused by a mixed flora. The etiological features of existing forms of peritonitis are largely associated with differences in the sources of bacterial contamination and stages of the course of peritonitis. About the direct causes of peritonitis, their frequency and structure have remained relatively stable for an extended period. Among surgical pathology, the most common cause of peritonitis is perforation of a hollow organ. This group encompasses patients with abdominal trauma, perforated gastro-duodenal ulcers, perforations of the bowel, and failure of gastrointestinal and inter-intestinal anastomoses. A greater proportion of males are affected (60-70%). The age range of patients is broad, spanning from a few

months to over 80 years of age. Approximately 30% of patients are in the elderly and senile age group. In purulent peritonitis, Gram-negative and anaerobic bacteria, including intestinal flora (*Escherichia coli* and *Klebsiella pneumoniae*), enter the abdominal cavity. The endotoxins produced by Gram-negative bacteria lead to the release of cytokines that trigger cellular and humoral cascades, culminating in cellular damage and, in some patients, infectious shock and multi-organ failure syndrome. In particular, abdominal sepsis is initiated by a component of the outer membrane of Gram-negative microorganisms (e.g. lipopolysaccharide, lipid A, endotoxin) or Gram-positive organisms (e.g. lipoteichoic acid, peptidoglycan), as well as toxins from anaerobic bacteria. This results in the release of pro-inflammatory cytokines, including tumour necrosis factor and interleukins 1, 6, and others. These processes contribute to the formation of toxic mediators, which give rise to a complex, multifactorial syndrome that may manifest in a variety of ways and result in functional impairment of one or more vital organs and systems.

Consequently, in the context of this situation, the patient's condition must be evaluated by the Sepsis-3 criteria. At the initial stage of examination, it is also essential to conduct a differential diagnosis and assess the severity of physiological disorders. This is necessary to determine the necessity for correction of water-electrolyte imbalances, replenishment of fluid deficits to stabilise haemodynamic disorders and other procedures. This assessment is of paramount importance for the implementation of diagnostic procedures, the initiation of antimicrobial therapy, the timing and types of surgical intervention, the choice of open or minimally invasive surgery, the use of tact, and other procedures.

Clinical manifestation of intra-abdominal infections

Acute appendicitis in adults

Approximately 20-30% of cases lack the classic clinical features (history, characteristic physical symptoms, laboratory values) that would otherwise be expected. However, in other cases, acute appendicitis (AA) may 'masquerade' as other diseases, which can significantly complicate the diagnosis of AA, especially in elderly patients and in pregnant women. The diseases with which differential diagnosis of AA is mandatory: perforated gastric or duodenal ulcer; acute pancreatitis; complicated Meckel's diverticulum; torsion of the right appendages, disturbed ectopic pregnancy, ovarian apoplexy, pelvic inflammation; right-sided renal colic and/or urinary infections; intestinal infections. As a rule, the patient complains of pain in the right iliac region, which is constant and moderate in intensity, and does not radiate. The disease manifests abruptly, in the absence of any preceding symptoms and abdominal pain is typically persistent. The localisation of pain is dependent on the anatomical features of the appendix location, including ascending, medial, pelvic, retrocecal or retroperitoneal, and left-sided. In the case of an ascending location, the pain is localised in the right subcostal region and may simulate the symptoms of biliary colic or peptic ulcer disease and it is accompanied by vomiting due to duodenal irritation. The location of the appendix in proximity to extrahepatic bile ducts may result in transient jaundice. In the case of a medial location, the process is displaced to the midline and located closer to the root of the mesentery of the small intestine. In this case, the onset of pain may be accompanied by repeated vomiting, which is associated with reflex irritation of the root of the mesentery and the pain is localised in the vicinity of the navel as a rule. In the pelvic position, the inflamed appendix may contact the bladder wall, which is manifested by dysuria and a lower localisation of pain. In the retrocecal or retroperitoneal position, the symptomatology develops more gradually, which frequently results in delayed hospitalisation. In addition, the affected individual may experience pain in the right thigh and even the right hip joint. A left-sided location of the appendix is an extremely rare occurrence, with a prevalence of only 0.1% of observations. All local symptomatology of AA is found in the left iliac region. Furthermore, the

localisation of pain may be influenced by pregnancy, particularly in the second trimester, when the enlarging uterus shifts upwards and laterally, displacing the ileocecal angle. Consequently, the pain may be localised in the right lateral region or the right subcostal region. The patient typically presents with anorexia and nausea or vomiting. The majority of patients experience vomiting on a single or double occasion. The Rovsing's sign can be identified by palpation of the left lower quadrant of the abdomen with the left hand and simultaneous right-hand pressure on the descending colon. The presence of pain in the right iliac region is indicative of a positive symptom. Kocher's sign is characterised by pain in the epigastrium or wandering pain throughout the abdomen that shifts to the right iliac region within two to four hours. The obturator test, which involves passive internal rotation of the flexed right hip when the patient is on their back, is what is causing pain in cases of pelvic AA. The psoas sign is a diagnostic test performed with the patient in the left side position. The doctor then slowly extends the patient's right hip, thus stretching the iliopsoas muscle. This causes pain, which indicates the presence of an irritated and inflamed appendix near the lumbar muscle. Blumberg's symptom is defined as an increase in pain in the right iliac region when the hand is pulled back sharply from the abdominal wall after pressing on it in the right iliac region.

It is recommended that the likelihood of AA be assessed based on the Alvarado, AIR (Appendicitis Inflammatory Response Score), AAS (Adult Appendicitis Score) and RIPASA (Raja Isteri Pengiran Anak Saleha Appendicitis) scores. The sensitivity and specificity of all these scores are inversely proportional: they are sensitive enough to exclude the disease, but not specific enough to confirm AA. The Alvarado score has insufficient specificity to diagnose AA in adults and does not reliably distinguish between complicated and uncomplicated forms of AA in the elderly, so its use in adults is not recommended. However, a borderline score of less than five is sensitive enough to exclude AA (sensitivity 99%). The AIR and AAS scores are recommended for use in suspected AA cases due to their superior predictive value and ability to reduce the incidence of negative appendectomies and the need for imaging studies in low- and intermediate-risk groups. Given the low probability values associated with the individual clinical data mentioned above for determining the likelihood of AA in an adult patient, an individualised approach is currently recommended, taking into account the probability of disease, gender and age of the patient. Furthermore, the AIR and AAS scores are also recommended as clinical predictors of AA.

A complete blood count is recommended as an increase in the number of leukocytes, and in particular, an increase in the number of polymorphonuclear neutrophils (≥ 75%), is of significant diagnostic value in the context of the clinical signs of AA. It is crucial to highlight that biochemical markers (C-reactive protein, procalcitonin, etc.) represent a promising and reliable diagnostic tool for the detection of both negative cases and complicated AA in adults. Nevertheless, there is currently no evidence of sufficient quality to necessity support their use. It is also recommended that a general urinalysis be performed to exclude urinary system pathology. Patients with a medium probability of AA (according to the AAS/AIR score) should be admitted to the hospital for further observation and monitoring. In patients with an intermediate probability of AA (according to AAS/AIR score), ultrasound is recommended as an essential addition to the physical examination. In patients with an intermediate probability of AA (as assessed by AAS/AIR score) and no ultrasound evidence of AA, CT abdominal imaging with intravenous contrast is recommended. In patients under the age of 40 with a high probability of AA, CT is not recommended (as assessed by AAS/AIR). The primary disadvantage of CT is the exposure to ionising radiation, which necessitates a risk-benefit assessment, particularly in younger patients and women of childbearing age. Nevertheless, the routine use of CT has been demonstrated to reduce the number of unnecessary appendectomies and to enhance the detection of abdominal diseases.

Acute cholecystitis

Acute cholecystitis (AC) is defined as an acute inflammation of the gallbladder, accompanied by a local and systemic inflammatory reaction or the threat of its occurrence. In approximately 90% of cases, AC develops in the context of cholelithiasis, while in 10% of cases, it is designated as acute acalculous cholecystitis, as these patients do not have gallstones. The occurrence of intravesical hypertension is the primary factor in reducing the blood supply to the gallbladder mucosa. A reduction in the perfusion of the wall, particularly in elderly and senile patients, contributes to the disruption of the mucous barrier, the penetration and growth of aerobic and anaerobic microorganisms (*Escherichia coli*, *Klebsiella*, *Enterococcus*, *Streptococcus* species, *Enterobacter* species, *Pseudomonas aeruginosa*), which, in turn, increases the inflammatory exudation into the lumen of the organ. The exudation process results in an increase in intravesical pressure, compression of in-wall vessels, disruption of microcirculation and ischemia in the gallbladder wall, and the release of inflammatory exudate into its lumen. Consequently, a pathophysiological vicious circle is formed, whereby the development of AC is perpetuated.

The clinical manifestations of AC are dependent upon the pathomorphological picture of inflammation of the gallbladder, the presence and extent of peritonitis, as well as concomitant changes in the bile ducts. In the majority of cases, the initial symptom of AC is abdominal pain in the right hypochondrium. The pain may also radiate to the right shoulder or scapula, the lumbar region, or the right shoulder girdle, and in some cases, to the heart (it is a cholecystocardiac syndrome). Nausea is a common symptom, as with biliary colic, but vomiting is more common. Severe vomiting may be indicative of the presence of a stone in the common bile duct, acute pancreatitis, or intestinal obstruction. A loss of appetite and malaise are more commonly observed in patients with AC. Purulent complications of AC are manifested by fever above 37–38°C, tachycardia, chills and jaundice. Murphy's sign is characterised by the patient experiencing sharp pain in the right hypochondrium when the examiner presses the gallbladder at the height of inspiration. Mussy's sign is characterised by tenderness at the point of the phrenic nerve, between the heads of the sternocleidomastoid muscle. Kera's sign is characterised by pain during deep palpation in Ker's point. Blumberg's sign is characterised by the patient experiencing an exacerbation of pain in the right upper quadrant when the examiner sharply withdraws their hand from the abdominal wall after pressing it in the right upper region.

The diagnosis of AC can be established clinically, but laboratory confirmation is of great importance. All patients presenting with clinical manifestations of AC are advised to undergo general and biochemical blood tests, as well as additional examinations in unclear cases, to determine the severity of inflammatory changes and to facilitate a timely diagnosis of cholestasis syndrome and differential diagnosis: right-sided lower lobe pneumonia; myocardial infarction; perforated peptic ulcer; acute pancreatitis; shingles; hepatitis; acute intestinal obstruction; acute appendicitis. In diagnostically challenging cases, where all non-invasive research methods have been exhausted, diagnostic video laparoscopy may be employed. When making the final diagnosis, all patients with AC are recommended to use the classification and diagnostic criteria of the Tokyo Agreement (Tokyo guidelines (2013/2018). The diagnostic criteria for AC as set out in the 2013/2018 Tokyo guidelines are as follows:

A. Local signs of inflammation etc.
   (1) Murphy's sign, (2) RUQ mass/pain/tenderness.
B. Systemic signs of inflammation etc.
   (1) Fever, (2) elevated CRP, (3) elevated WBC count
C. Imaging findings
   Imaging findings characteristic of AC.

Suspected diagnosis: one item in A + one item in B

Definite diagnosis: one item in A + one item in B + C

The severity of AC is determined by the following criteria:

- Grade II (moderate) AC: "Grade II" AC is associated with any one of the following conditions: An elevated white blood cell (WBC) count (greater than 18,000 per microliter) is indicative of the condition. Additionally, a palpable tender mass in the right upper abdominal quadrant, duration of complaints exceeding 72 hours, and marked local inflammation (gangrenous cholecystitis, pericholecystic abscess, hepatic abscess, biliary peritonitis, emphysematous cholecystitis) are also indicative of the condition.
- Grade III (severe) AC: "Grade III" AC is associated with dysfunction of any one of the following organs/systems:
  1. Cardiovascular dysfunction: hypotension requiring treatment with dopamine ≥ 5 μg/kg per min, or any dose of norepinephrine.
  2. Neurological dysfunction: decreased level of consciousness.
  3. Respiratory dysfunction: A PaO_2/FiO_2 ratio of less than 300 indicates.
  4. Renal dysfunction characterized by oliguria and a creatinine level exceeding 2.0 mg/dl.
  5. A PT-INR of greater than 1.5 indicates hepatic dysfunction.
  6. A platelet count of less than 100,000/mm^3 indicates haematological dysfunction.

The Tokyo Guidelines 2013/2018 propose that imaging studies, such as ultrasound, CT and HIDA scans, should be employed to diagnose AC, in conjunction with a comprehensive history, a thorough clinical examination and laboratory tests. Furthermore, it highlighted the value of ultrasound as a diagnostic tool due to its non-ionising, inexpensive, and easy-to-use characteristics. The positive results of bile culture are found in 80-100% of patients with acute cholangitis. In the majority of cases, the causative agents of the biliary infections are microorganisms of the intestinal microflora. These microorganisms include representatives of the *Enterobacteriaceae* family, among which *Escherichia coli* plays a dominant role (50-60%), *Klebsiella* spp. (8-20%), *Serratia* spp., and *Acinetobacter* spp. Gram-positive microorganisms (*Streptococcus* and *Enterococcus*) are identified in 2-30% of cases, while non-spore-forming anaerobes (*Bacteroides*, *Fusobacteria* and *Peptococci*) are found in up to 20% of cases. *Pseudomonas* spp. are present in 2-4% of cases, as reported by Salvador *et al*.

Perforation of hollow organs

Perforation may occur at any level of the gastrointestinal tract, with the contents of the stomach or intestine entering the peritoneal cavity. The causes of this condition are diverse. The clinical symptoms manifest suddenly, with the onset of intense pain and a rapid deterioration in the patient's condition. Diagnosis is typically based on the identification of free gas within the abdominal cavity through the use of radiography.

Esophagus

Esophageal rupture may be iatrogenic during endoscopic procedures or other manipulations or may occur spontaneously (Burhave's syndrome). The patient's condition is severe, with evidence of mediastinitis. The most common site of oesophageal rupture is the distal left oesophagus. Acid and gastric contents cause fulminant mediastinitis and shock. The symptoms of oesophageal rupture include chest and abdominal pain, fever, vomiting, haematemesis, and shock. Approximately 30% of patients present with subcutaneous emphysema. Crepitation, also known as Hammen's symptom, may be heard in the mediastinal region. This is a distinctive crunching or clicking noise that is synchronised with heartbeats. The presence of air in the mediastinum, fluid in the pleural cavity, and widening of the mediastinum, as observed on radiological examination of the chest and abdomen, are indicative of the diagnosis and it is confirmed by esophagography with

water-soluble contrast. A CT scan of the chest reveals the presence of air and fluid in the mediastinum, yet does not provide information regarding the precise location of the rupture.

Peptic ulcer of the stomach and duodenum

The clinical signs of perforative peptic ulcer are as follows: 1. The disease typically manifests acutely, presenting with sharp abdominal pain, akin to a "dagger blow." 2. The abdominal muscles are tensed. 3. Palpation of the abdomen elicits pain. The patient is often positioned with bent knees, and the pain is exacerbated by movement, accompanied by a distressed facial expression, pale skin, cyanotic lips, and dry mucous membranes of the mouth and lips. The patient's blood pressure is often reduced by 5-10%, with a slow pulse and frequent, shallow respiration. Palpation of the anterior abdominal wall reveals a tense appearance. In the initial hours of the disease, sharp pain is experienced in the epigastrium and mesogastrium. Upon percussion of the abdomen, a sharp pain is elicited. The disappearance of hepatic dullness and the appearance of high tympanitis over the liver can be established. In the presence of fluid in the lower and lateral abdomen, a dulling of the percussion sound is determined. The physical examination findings may be contradictory, as it has been observed that symptoms of peritonitis are present in 35% to 50% of patients with perforated peptic ulcers. This is more commonly observed in cases of occluded perforation or patients with atypical localisations. Abdominal pain is less pronounced in perforated ulcers of the posterior wall of the duodenum and the cardia of the stomach when the contents enter the retroperitoneal fascia or omental sac. Localized or generalized peritonitis is a typical manifestation of perforated peptic ulcer, although it may be present in only two-thirds of patients.

Patients with perforated ulcers are advised to undergo a standard set of laboratory tests, including blood, urine, blood chemistry, blood glucose, bilirubin, creatinine, blood amylase, and so forth. However, it should be noted that changes in laboratory parameters in these patients are non-specific, laboratory tests are conducted for differential diagnosis and to ascertain the extent of damage to various organs. All patients are recommended to undergo chest and abdominal radiography to detect free gas under the diaphragm (the diagnostic accuracy of X-ray images in the vertical position and on the left side is the same). If other diagnostic methods are not informative and if technically possible, abdominal CT is recommended. In patients with suspected perforation of a peptic ulcer in the absence of signs of free gas in the abdominal cavity, according to the data of radiological methods of imaging, it is recommended that an oral water-soluble contrast agent be administered with repeated radiological examination. Ultrasound can also reveal free intraperitoneal exploration or free fluid but the role of it in the diagnostic workup of suspected peptic ulcer perforation remains to be determined. The 2020 WSES recommendation suggests the adoption of scoring systems including the BOEY, PULP and ASA scores to stratify patients by risk and predict outcomes. However, these recommendations are weak and based on low-quality evidence. The scoring systems in question are relatively straightforward to use, clinically meaningful, and can be employed to predict postoperative morbidity and mortality. In cases of perforated peptic ulcer with sepsis/septic shock, it is crucial to determine the parameters for assessing the severity of the disease. This entails identifying several symptoms, such as altered mental status and dyspnea, as well as signs, including tachycardia, tachypnea, decreased pulse pressure, and decreased urine output. Additionally, laboratory parameters, such as hyperlactatemia, arterial hypoxemia, increased creatinine, and blood coagulation disorders, must be considered. The systems of organ failure assessment (SOFA) or quick SOFA (qSOFA) are acceptable for assessing disease severity.

Gastric tumour

The occurrence of gastric adenocarcinoma perforation is a rare phenomenon. Patients with perforation of gastric malignancy typically present with a history and symptoms that are similar to those of patients with

benign gastroduodenal or other cavitary perforations. In the context of examining a patient with suspected perforation, it is recommended to pay attention to the sudden and violent onset of symptoms, including diffuse acute abdominal pain, weakness, marked pain on palpation of the anterior abdominal wall, and abdominal muscle tension. It is important to note that changes in laboratory values in perforation are nonspecific. However, physicians must be aware of the presence of leucocytosis and increased amylase levels, which may be observed in gastric perforation. Instrumental methods of examination can reveal characteristic signs of perforation, including free gas in the abdominal cavity, free fluid in the abdominal cavity, and the presence and localisation of the perforation. The diagnostic accuracy of review radiography varies considerably, ranging from 30% to 85%. CT of the abdomen is recommended for all patients with suspected perforation, and if other diagnostic methods are not informative (it has a high diagnostic accuracy of 95-98%). In patients with suspected perforation in the absence of signs of free gas in the abdominal cavity according to the data of radiological methods of investigation, oral administration of water-soluble radiopaque iodine-containing contrast agent with repeated radiological examination is recommended. In all patients with suspected perforation, in the event of a negative radiological examination or the inability to perform a CT scan, it is recommended to perform an ultrasound of the abdominal cavity organs to detect free gas and free fluid (diagnostic accuracy in perforation up to 92%).
Esophagogastroduodenoscopy is indicated for patients with suspected tumour perforation in cases where the clinical and instrumental picture is ambiguous, as well as for the diagnosis of other complications (bleeding, pylori duodenal stenosis), and for biopsy in cases of an unresectable tumour process (allows the presence of a tumour with perforation to be established in 90% of cases). In the absence of free gas in the abdominal cavity, diagnostic laparoscopy is recommended for patients with suspected perforation.

Small bowel (not traumatic)
The clinical picture of intestinal perforation is dependent on a multitude of factors, including the aetiology, age of the patient, localization of the defect, type of perforation, medication, and others. It is important to note that the clinical picture of intestinal perforation develops in the context of any underlying disease, and it is accompanied by the rapid onset of complications such as peritonitis and sepsis. Consequently, it is only possible to speak of specific signs of perforation in the context of a specific etiologically significant pathology. A diagnosis is made based on clinical findings and is then confirmed by instrumental diagnostics, which have different diagnostic values. The most prevalent and distinctive symptom is considered to be abdominal discomfort of varying locations. An examination may reveal abdominal tension and bloating, as well as decreased peristalsis and symptoms of peritoneal irritation. In addition, tachycardia, moderate arterial hypotension, and fever (in the event of peritonitis) may be observed. Moderate dehydration may be evidenced by an increased thirst, a dry tongue, and oliguria. A review of radiography of the abdominal cavity in various positions may reveal the accumulation of free gas in the abdominal cavity. It is recommended that all patients undergo abdominal ultrasound on admission for diagnostic purposes. CT and MRI are considered the "gold standard" for the diagnosis of intestinal perforation, as well as for identifying the causes of perforation. In cases where the diagnosis remains uncertain, laparoscopy is recommended. Laboratory diagnostics lacks highly specific features; therefore, blood, urine, C-reactive protein, and blood biochemical analysis are recommended.

Colon (not traumatic)
Diverticulitis of the colon is defined as inflammation of the diverticulum, which may or may not be accompanied by infection. This may result in phlegmon of the bowel wall, peritonitis, perforation, fistula formation or abscess. The most severe complication of diverticular disease is perforation of the diverticulum, which occurs

in 7-10% of patients with diverticular disease, according to the literature. The outcome of the disease is the formation of an abdominal cavity infiltrate or peritonitis. In the acute form of diverticulitis, the most common symptom is abdominal pain. In the majority of cases, the condition is localized to the lower abdomen on the left side. In some cases, patients may experience symptoms in other locations, including the upper abdomen, the left side, and, on rare occasions, the lower abdomen on the right side. The pain may be continuous or intermittent. In most cases, the pain is not particularly intense and is not accompanied by an increase in body temperature. Other symptoms that are not observed in all patients with intestinal diverticulitis include flatulence, an increased frequency of stools or constipation, nausea, vomiting, and frequent urination. The general condition of patients before the onset of complications remains unaltered. Palpation may reveal a compacted but mobile area of the affected intestine. The symptoms of diverticulitis are more pronounced in cases with a complicated course.

Acute para-intestinal infiltrate

The subsequent phase of acute diverticulitis. The exudate (inflammatory fluid) that forms within the diverticulum soaks the intestinal wall and surrounding tissues, causing them to become inflamed. This subsequently results in an exacerbation of the symptoms. The pain becomes more pronounced. A tumour-like mass with indistinct boundaries may be palpated in the abdomen. Palpation of the abdomen results in the development of pain. The motility of the inflamed intestinal wall is diminished. The symptoms of intoxication also manifest, including weakness and a body temperature above 38 degrees Celsius. In two out of three patients, there is an increase in symptoms associated with impaired intestinal function, including flatulence, abdominal bloating, and changes in the frequency and consistency of stool. In some cases, faecal discharge may be challenging due to compression of the intestine by the infiltrate, yet intestinal obstruction does not develop. The intestinal function is rapidly restored in the context of antibacterial treatment.

Diverticular abscess

An abscess is a localized accumulation of pus. The pain is constant and localised to the area of the purulent cavity. In the majority of cases, the pain is located in the lower abdomen on the left side. The patient's temperature is within the normal range. An increase in body temperature to more than 37.5 degrees Celsius, severe pain, vomiting, and an elevated heart rate typically indicate the breakthrough of the purulent cavity into the abdominal cavity with the development of peritonitis.

Diverticular perforation

Perforative diverticulitis is characterized by the sudden onset of severe abdominal pain. Initially, the pain is localised to a specific area of the abdomen, but subsequently becomes diffuse, lacking a clear localisation. Following the perforation of the colon, the symptoms rapidly intensify, with the onset of high fever and severe intoxication.

Colorectal cancer perforation

Colorectal perforation in colorectal cancer is a less common occurrence. The integrity of the intestinal wall may be violated in cancer as a result of tumour decay or obstruction of the intestinal lumen, pressure of faeces on the intestinal wall, disruption of its blood supply and subsequent necrosis. Furthermore, colon perforation may be observed in the context of non-oncological bowel obstruction, Crohn's disease, ulcerative colitis and toxic megacolon. In certain instances, medical procedures, such as colonoscopy, have been identified as a potential cause of perforation. According to research, the incidence of such complications is approximately 0.2%. The use of immunosuppressants and certain hormonal drugs is associated with an

increased risk of non-traumatic perforation of the colon, regardless of the underlying cause. Colon perforation is defined by the presence of acute abdominal symptoms. Patients typically present with severe abdominal pain, which is often exacerbated by movement and relieved by resting. They often adopt a flexed position on the side or back with bent legs, which may be a result of the pain. The body temperature of patients with intestinal perforation initially exhibits a sub-febrile pattern, followed by a rise to febrile figures. Additionally, patients may exhibit signs of weakness, pallor of the skin, nausea, vomiting, decreased urine output, tachycardia, and respiratory disorders. The abdomen is distended, and the anterior abdominal wall is tense. Palpation reveals an increase in pain, with the greatest discomfort occurring in the area of the perforation. Following the perforation of the colon, the intensity of the pain syndrome gradually decreases as the receptors of the peritoneum adapt to the pathological changes occurring within the abdominal cavity. As peritonitis develops, pain and abdominal wall tension increase in intensity and become more diffuse, affecting all regions of the abdomen. The percussion of the liver in patients with perforation of the colon results in the disappearance of hepatic dullness, indicating the presence of gas in the abdominal cavity. In the low-lying areas of the abdomen, the presence of fluid may result in a dulling of the sound. Auscultation reveals a weakening or disappearance of intestinal noises due to peristaltic disorders. The clinical manifestations of colonic perforation are contingent upon the extent of the perforation, the dimensions of the perforation hole, and the presence or absence of obstruction of the large intestine. In general, the proximal lesion of the colon is more severe due to the rapid dissemination of liquid intestinal contents in the abdominal cavity. The exception to this rule is when the cause of colonic perforation is intestinal obstruction. In such cases, periods of apparent improvement, due to a reduction in the pressure of the contents on the intestinal wall, are more pronounced than usual. On occasion, colonic perforation may be practically asymptomatic due to the small size of the perforation or the proximity of the omentum, which prevents the contents from escaping into the abdominal cavity.

Conclusion

This chapter addresses the clinical question of which diagnostic procedures are appropriate for the initial evaluation of patients with suspected IAI. Intra-abdominal infection is a prevalent clinical issue, and the classification (uncomplicated and complicated IAI) encompasses a spectrum of processes affecting diverse organs. An uncomplicated infection causes intramural inflammation of the digestive tract and has a significant chance of becoming a complicated infection if not treated properly. Complicated intra-abdominal infection is defined as the spread of infection beyond the jejunum into the peritoneal space, which is associated with the formation of an abscess or peritonitis. The necessity for prompt diagnosis and intervention, coupled with the ongoing debate surrounding the optimal procedure to be employed, further complicates the management of these patients. The management of these infections has been significantly advanced by the development of supportive intensive care, diagnostic imaging, minimally invasive interventions, and antimicrobial therapy.

Competing interests

The authors have no financial and non-financial competing interests to declare.

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