PERITONITIS

Minsk BSMU 2020

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
1-я КАФЕДРА ХИРУРГИЧЕСКИХ БОЛЕЗНЕЙ
2-я КАФЕДРА ХИРУРГИЧЕСКИХ БОЛЕЗНЕЙ

ПЕРИТОНИТ
PERITONITIS

Учебно-методическое пособие

Минск БГМУ 2020

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Отражены современные аспекты проблемы этиологии, патогенеза, клиники, диагностики перитонита. Освещены современные подходы к выбору методов лечения перитонита.

Предназначено для студентов 4–6-го курсов медицинского факультета иностранных учащихся, обучающихся на английском языке.

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MOTIVATIONAL CHARACTERISTICS OF THE TOPIC

Total in-class hours: 5.

Peritonitis, an inflammation of the peritoneum, is a consequence of the majority of surgical diseases with acute and chronic inflammation and necrosis of abdominal viscera. Even though the onset of peritonitis differs according to the its source, the inflammation, clinical signs and complications become similar in time and bear the overall mortality rate of generalized peritonitis about 40 %.

The purpose is to study the etiology, pathogenesis, main clinical manifestations, diagnosis, and treatment of peritonitis.

Objectives are:

1) to learn main etiological causes of peritonitis;
2) to learn classifications of peritonitis;
3) to learn methods of examinations in peritonitis;
4) to learn common clinical features of peritonitis;
5) to make diagnosis of peritonitis;
6) to be able to assess results of diagnostic studies and laboratory tests;
7) to be able to perform preoperative preparation;
8) to be able to make plan of surgery;
9) to be able to prescribe the postoperative treatment.

Requirements for the initial level of knowledge.

To learn the topic completely the student must know:

– Propaedeutics of internal diseases (methods of clinical evaluation of abdominal organs);

– Human anatomy (localization and structure of internal organs, peritoneum);

– Topographic anatomy and operative surgery (main surgical approaches to abdominal organs);

– Pathologic physiology (response to inflammation);

– General surgery (basic principles of surgical infections and sepsis).

Test questions from related disciplines:

1. Normal and topographic anatomy of abdominal organs.
2. Clinical evaluation of abdominal cavity.
3. Methods of investigations of abdominal organs.
4. Surgical approaches to abdominal organs.
5. General signs of infection and inflammation.

Test questions:

1. Anatomy and functions of peritoneum.
2. Classification of peritonitis.
3. Etiology of peritonitis.
4. Pathogenesis.

5. Complications.
6. Clinical manifestations.
7. Diagnosis.
8. Principles of treatment.
9. Organ specific peritonitis.

ANATOMY AND PHYSIOLOGY

The *peritoneum* is a thin serous membrane that lines the walls of the abdominal and pelvic cavities and clothes the viscera. Its surface consists of a single layer of flat mesothelial cells that reside on a basement membrane (Figure 1), which in turn overlies a bed of connective tissue.

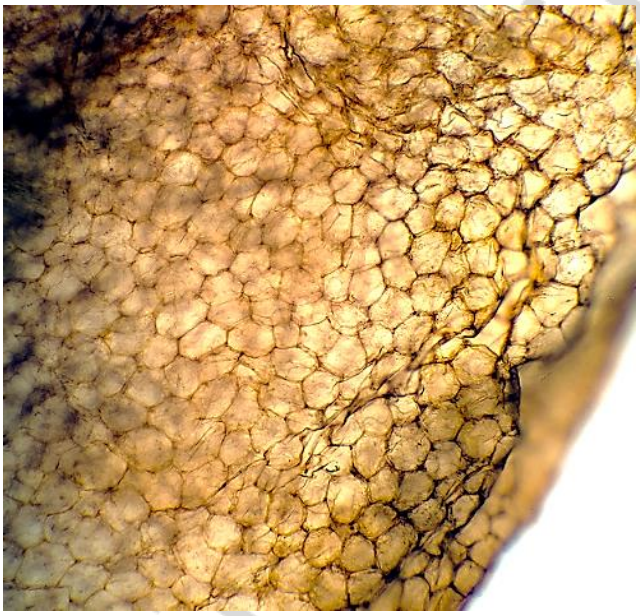


Figure 1. Mesothelial cells

The *peritoneal cavity* is the abdominal space bounded by the diaphragm superiorly, the pelvic floor inferiorly, the retroperitoneum posteriorly, and the anterior abdominal wall anteriorly. In males, this is a closed cavity, but in females, there is communication with the exterior through the uterine tubes, the uterus, and the vagina. The peritoneal cavity is the largest cavity in the body and is divided into two parts: the greater sac and the lesser sac (Figure 2). The greater sac is the main compartment and extends from the diaphragm down into the pelvis.

The lesser sac is smaller and lies behind the stomach. The greater and lesser sacs are in free communication with one another through an oval window called the opening of the lesser sac, or the epiploic foramen (of Winslow, Figure 3).

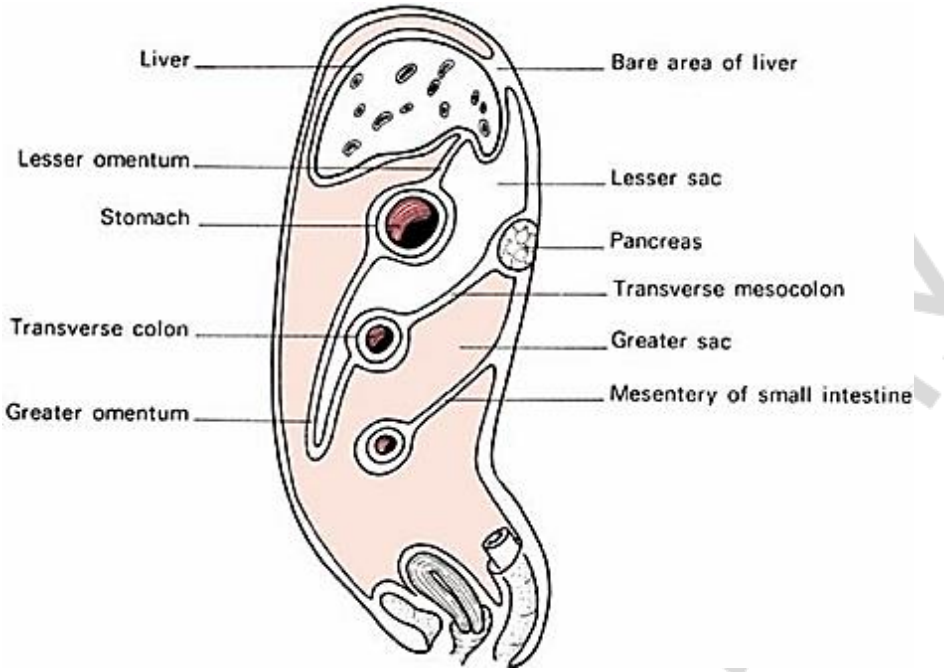


Figure 2. Parts of the peritoneal cavity

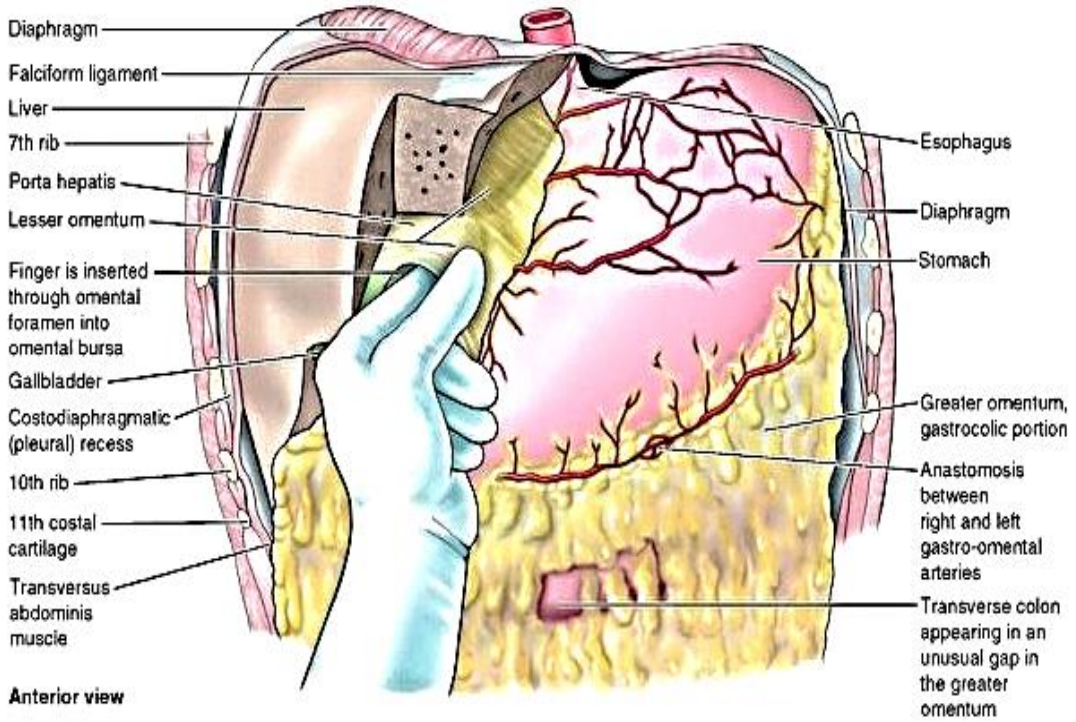


Figure 3. Winslow opening

The peritoneal cavity is normally *sterile*. The peritoneal cavity is lined by the *parietal* peritoneum. The *visceral* peritoneum covers abdominal organs. Abdominal viscera have three variants of peritoneal covering:

– *Intraperitoneal* (total peritoneal covering — small bowel, stomach, transverse colon, sigmoid, spleen);

– *Mesoperitoneal* (partial peritoneal covering — first part of the duodenum, ascending and descending colon);

– *Retroperitoneal* (the second, third, and fourth portion of the duodenum, the distal rectum, the pancreas, the kidneys and ureters, the adrenal glands, and the aorta and inferior vena cava).

The peritoneal surface area is a semipermeable membrane with an area comparable to that of the cutaneous body surface. Nearly 1 m^2 of the total 1.7 m^2 area participates in fluid exchange with the extracellular fluid space at rates of 500 mL or more per hour. Normally, there is less than 50 mL of free peritoneal fluid, a transudate with the following characteristics: specific gravity below 1.016; protein concentration less than 3 g/dL; white blood cell count less than 3000/L; complement-mediated antibacterial activity; and lack of fibrinogen-related clot formation.

The circulation of peritoneal fluid is directed toward lymphatics in the undersurface of the diaphragm (Figure 4). The circulation of fluid from the lower abdomen to the subdiaphragmatic space is due to negative pressure generated in the subdiaphragmatic space with respiration.

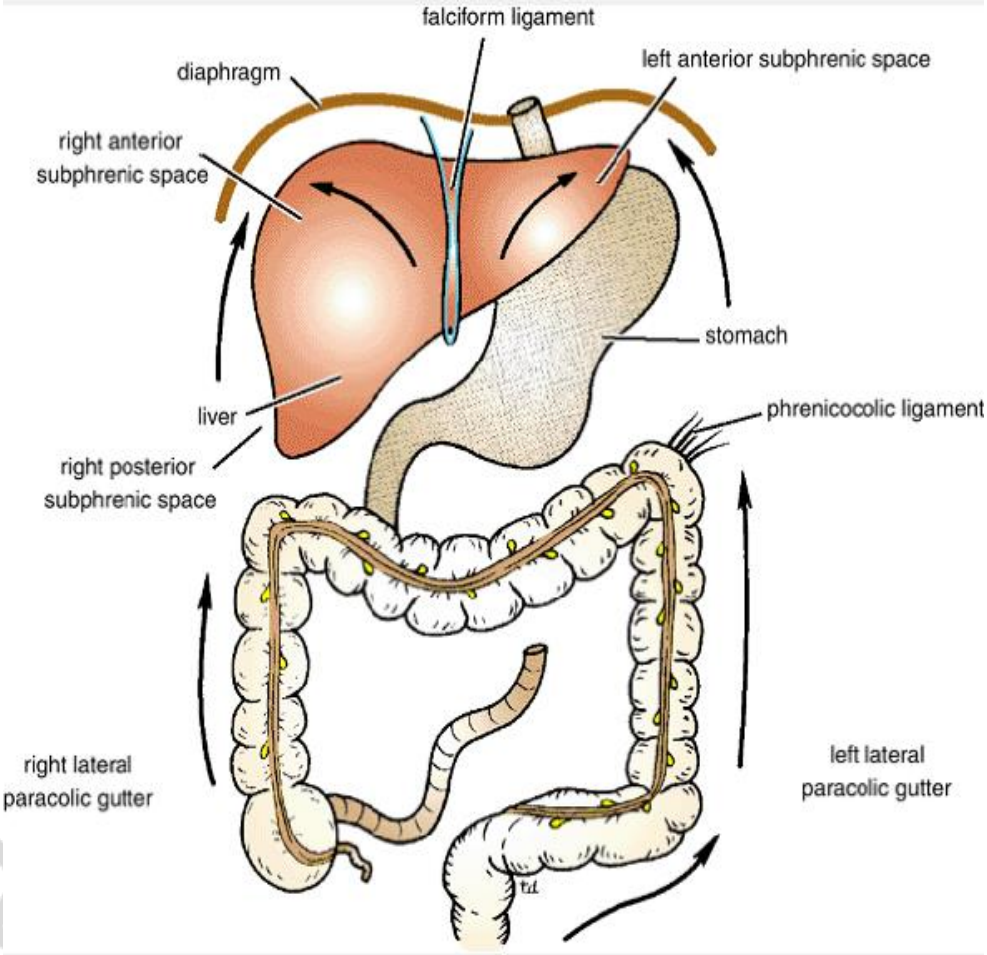


Figure 4. Normal direction of flow of the peritoneal fluid from different parts of the peritoneal cavity to the subphrenic spaces

The parietal peritoneum is supplied by segmental nerves that also supply the abdominal wall directly in contact. The nerve supply of the visceral peritoneum, however, is the nerve supply of the viscus it covers (Figure 5). The entire small intestine, the appendix, ascending and right colon, and the visceral peritoneum covering these structures are supplied by thoracic C-10, which also supplies the skin in the preumbilical region. Hence, pain due to distension, ischemia, or inflammation in these structures is first referred to the periumbilical region. Only when the parietal peritoneum overlying the diseased bowel is involved does the pain localize to the region where the diseased bowel is actually located.

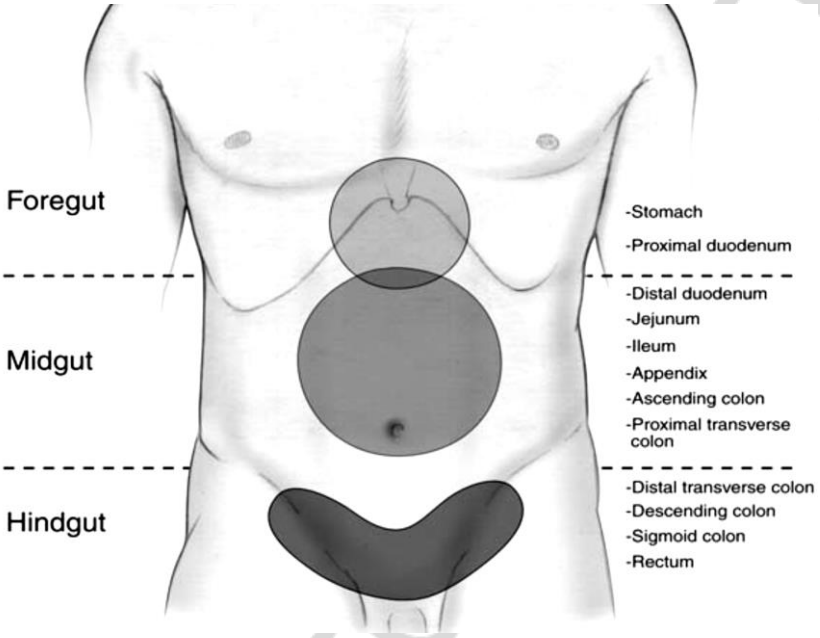
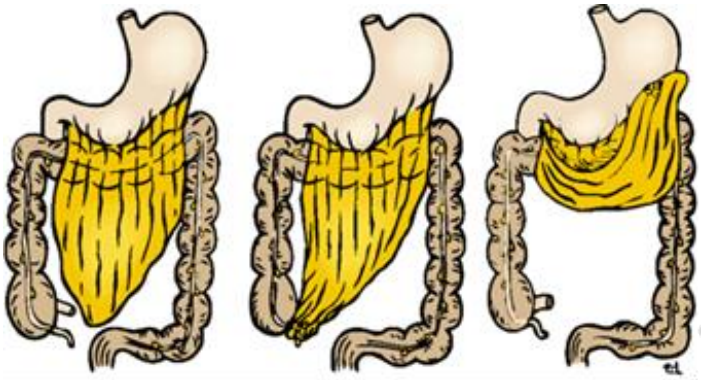


Figure 5. Sites (shaded areas) of referred pain. Sites of pain in the abdominal wall referred from pathology of different parts of the gastrointestinal tract

The ***omentum*** is a well-vascularized pliable, mobile double fold of peritoneum and fat (Figure 6, *a*). The greater omentum is often referred to by the surgeons as the abdominal policeman. The lower and the right and left margins are free, and it moves about the peritoneal cavity in response to the peristaltic movements of the neighboring gut. In the first 2 years of life it is poorly developed and thus is less protective in a young child.

Its composition is well suited to sealing off a leaking viscus (e.g., perforated ulcer) or area of infection (Figure 6, *b*, *c*). In an acutely inflamed appendix, for example, the inflammatory exudate causes the omentum to adhere to the appendix and wrap itself around the infected organ. Its bacteria scavenger functions include absorption of small particles and delivery of phagocytes that destroy unopsonized bacteria. By this means, the infection is often localized to a small area of the peritoneal cavity, thus saving the patient from a serious diffuse peritonitis.



a ***b*** ***c***

Figure 6. The greater omentum:
a — normal; *b* — the greater omentum wrapped around an inflamed appendix;
c — the greater omentum adherent to the base of a gastric ulcer

The peritoneum and omentum play several roles of physiologic significance:

1. Provision of a surface that allows smooth gliding of the small intestine within the peritoneal cavity. This function is aided by the presence of free fluid (50mL of transudate) within the peritoneal cavity.

2. Fluid exchange. Approximately 500mL of fluid or more per hour may be exchanged between the peritoneal cavity and the circulation across the peritoneum. This remarkable property is exploited in the performance of peritoneal dialysis in renal failure. In infants, circulating blood volume may be replenished by the administration of fluid intraperitoneally.

3. Response to tissue damage or infection. The mesothelial and mast cells secrete histamine and other vasodilators in response to injury or infection. This leads to vascular permeability and the exudation of fibrinogenrich plasma, complement, and opsonins. Together with the arrival of neutrophils and macrophages, this process contributes to bacterial destruction.

4. Omental migration. The omentum migrates to areas of inflammation, perforation, or ischemia. This wellvascularized tissue attempts to isolate the pathology and also exerts bacteriophagic function.

5. Elimination of bacteria and toxic products. Bacteria that are not destroyed and other toxic products of infection are circulated to the subdiaphragmatic surfaces, particularly on the right, and absorbed into lymphatic channels and delivered into the right thoracic duct.

DEFINITIONS, CLASSIFICATION AND ETIOLOGY

Peritonitis is an inflammatory or suppurative response of the peritoneum to direct irritation like infection, injury, and leakage into the peritoneal cavity of digestive fluid, bile, pancreatic juice, urine, or blood.

Primary microbial peritonitis occurs when microbes invade the normally sterile confines of the peritoneal cavity via hematogenous dissemination from a distant source of infection or direct inoculation. This process is more common among patients who retain large amounts of peritoneal fluid due to *ascites*, and in those individuals who are being treated for renal failure via *peritoneal dialysis*. These infections invariably are *monomicrobial* and rarely require surgical intervention.

Secondary microbial peritonitis occurs subsequent to contamination of the peritoneal cavity due to *perforation or severe inflammation* and infection of an intra-abdominal organ. The majority of the morbid processes lead to a breach in the integrity of the wall of the gastrointestinal tract, with *transmigration* of intestinal bacteria to the peritoneal cavity or actual perforation with escape of intestinal contents and substantial contamination of the peritoneal space (Table 1). The three most common causes of generalized peritonitis in adults are a *perforated ulcer*, *colonic perforation*, and *perforated appendicitis.*

Table 1

Common causes of secondary peritonitis

Severity	Cause	Mortality Rate
Mild	Appendicitis	< 10 %
	Perforated gastroduodenal ulcers	
	Acute salpingitis	
Moderate	Diverticulitis (localized perforations)	< 20 %
	Nonvascular small bowel perforation	
	Gangrenous cholecystitis	
	Multiple trauma	
Severe	Large bowel perforations	20–80 %
	Ischemic small bowel injuries	
	Acute necrotizing pancreatitis	
	Postoperative complications	

Bacteria may enter the peritoneal cavity via four portals:

1. From the exterior: penetrating wound, infection at laparotomy, peritoneal dialysis.

2. From intra-abdominal viscera:

a) gangrene of a viscus, e.g. acute appendicitis, acute cholecystitis, diverticulitis or infarction of the intestine;

b) perforation of a viscus, e.g. perforated duodenal ulcer, perforated appendicitis, rupture of the intestine from trauma;

c) postoperative leakage of an intestinal suture line.

3. Via the bloodstream: as part of a septicaemia (pneumococcal, streptococcal or staphylococcal). This has been wrongly termed primary peritonitis; in fact, it is secondary to some initial source of infection.

4. Via the female genital tract: acute salpingitis or puerperal infection.

The microbiology of secondary peritonitis is invariably *polymicrobial* and consists of a mixture of gram-negative enteric bacteria and anaerobes:

Facultative gram-negative bacilli:

– E. coli;
– Klebsiella species;
– Proteus species;
– Enterobacter species;
– Morganella morganii;
– Enterococci gram-negative species.

Obligate anaerobes:

– B. fragilis;
– Bacteroides species;
– Fusobacterium species;
– Clostridium species;
– Peptococcus species;
– Peptostreptococcus species;
– Lactobacillus species.

Facultative gram-positive cocci:

– Enterococci;
– Staphylococcus species;
– Streptococcus species;
– Aerobic gram-negative bacilli;
– Pseudomonas aeruginosa.

Peritonitis of bowel origin usually shows a mixed fecal flora (Escherichia coli, Streptococcus faecalis, Pseudomonas, Klebsiella and Proteus, together with the anaerobic Clostridium and Bacteroides). Gynaecological infections may be chlamydial, gonococcal or streptococcal. Blood-borne peritonitis may be streptococcal, pneumococcal, staphylococcal or tuberculous. In young girls, a rare gynecological infection is due to pneumococcus.

Tertiary (persistent) peritonitis is a condition first recognized toward the end of the last century and describes patients who continue to have evidence for poorly localized intra-abdominal infection after initial operative and antimicrobial treatment of secondary infection. At subsequent interventions, these patients tend to have evidence for diffuse inflammation without bowel perforation, and cultures return a variety of bacteria that are not common pathogens in the peritoneum such as *coagulase-negative staphylococci, enterococcus, Pseudomonas species, and even Candida species*, that is resistant to common antibiotics. Immunosuppression is essential in development of the tertiary peritonitis. Death is common outcome.

In ***chemical peritonitis*** the peritoneal inflammation is initially chemical in nature, e.g. early stages of perforated duodenal ulcer, extravasation of uninfected urine (bladder injuries) or bile (after biliary operations). However, if treatment is

delayed, secondary infection supervenes within a few hours. Thus chemical peritonitis represents the initial clinical phase of extravasation of visceral contents into the peritoneal cavity and almost invariably *merges into acute secondary bacterial peritonitis*.

By spreading peritonitis can be divided to **local** that is within one quadrant or region of the abdomen as in early gangrenous or perforated appendicitis (***non-localized***) or ***abscess (localized)***. When peritonitis involve more than two abdominal regions it is called **generalized** (> 2 regions) or **total** (all the peritoneal cavity).

PATHOGENESIS

In response to tissue damage, mesothelial cells of the peritoneum discharge histamine and other vasoactive substances that enhance vascular permeability. The resulting fibrinogen-rich plasma exudate supplies complement and opsonic proteins that promote bacterial destruction. Tissue thromboplastin released by injured mesothelial cells *converts fibrinogen into fibrin*, which may in turn lead to collagen deposition and formation of fibrous adhesions.

In normal condition, this reaction is limited by a plasminogen activator in the cell lining, but the plasminogen activator is inactivated by injury or infection. Bacterial lipopolysaccharide (endotoxin) and cytokines can stimulate production of tumor necrosis factor (TNF). TNF, in turn, mediates the release of plasminogen activator inhibitor produced by inflamed peritoneal mesothelial cells, which can lead to persistence of fibrin. Fibrin clots segregate bacterial deposits, a source of endotoxins that contribute to sepsis, but segregation may also inadvertently shield bacteria from bacteria-clearing mechanisms. This local answer to the damage is called *local inflammatory response syndrome (**LIRS**)*.

Pain fibers within both the visceral and parietal peritoneum are activated. Reflex pathways cause muscular contraction in the abdominal wall to limit movement (*guarding and rigidity*). Similarly, peristaltic movement of the intestine is arrested (*hypoactive or absent bowel sounds*).

*Systemic inflammatory response syndrome (**SIRS**)* is a systemic manifestation of injury caused by infection, multiple trauma, burns, pancreatitis, etc. Serious infection, such as secondary peritonitis, may lead to SIRS through the release of lipopolysaccharide endotoxin from the walls of dying Gram-negative bacilli (mainly Escherichia coli) or other bacteria or fungi. This and other toxins stimulate the release of cytokines from macrophages.

The criteria for Systemic Inflammatory Response Syndrome are:

1. General variables:

– Heart rate > 90 beats/min;

– Core temperature < 36 °C or > 38,3 °C;

– Tachypnea > 20/min or PCO_2 < 32 mm Hg;

– Altered mental status;
– Significant edema or positive fluid balance (> 20 mL/kg over 24 h);
– Hyperglycemia in the absence of diabetes.
2. Inflammatory variables:
– Leukocytosis (white blood cell count > 12,000);
– Leukopenia (white blood cell count < 4000);
– Bandemia (> 10 % band forms);
– Plasma C-reactive protein > 2 above normal value;
– Plasma procalcitonin > 2 above normal value.
3. Hemodynamic variables:
– Arterial hypotension (systolic blood pressure < 90 mmHg, mean arterial pressure < 70, or systolic blood pressure decrease > 40 mmHg);
– Venous oxygen (SVO_2) > 70 %;
– Cardiac index > 3.5 L/min per square meter;
– Organ dysfunction variables;
– Arterial hypoxemia;
– Acute oliguria;
– Creatinine increase.
4. Coagulation abnormalities
– Thrombocytopenia;
– Hyperbilirubinemia;
– Tissue perfusion variables;
– Hyperlactatemia;
– Decreased capillary filling.
In general, the pathological effects of peritonitis are as follows:
1. Widespread absorption of toxins from the large, inflamed surface.
2. The associated paralytic ileus with the following:
a) loss of fluid;
b) loss of electrolytes;
c) loss of protein.
3. Gross abdominal distension with elevation of the diaphragm, which produces a liability to lung collapse and pneumonia.

COMPLICATIONS

The most common complications of peritonitis include:
– *Surgical sepsis* — SIRS in proven bacterial contamination (i.e. *Sepsis = SIRS + Infection*);
– *MODS* — multiple organ dysfunction syndrome;
– *MSOF* — multiple system organ failure;
– *Abdominal compartment syndrome*.

Characteristic features of ***sepsis*** include *fever* (hypothermia occasionally occurs in severe sepsis), *tachycardia*, and *tachypnea*, accompanied by *leukocytosis* (leukopenia is occasionally observed in severe sepsis).

Septic manifestations and *multiple organ dysfunction syndrome (**MODS**)* in SIRS are mediated by the release of proinflammatory cytokines such as interleukin-1 (IL-1) and tumour necrosis factor alpha (TNFα, Figure 7). These cytokines stimulate neutrophil adhesion to endothelial surfaces adjacent to the source of infection and cause them to migrate through the blood vessel wall by chemotaxis. A respiratory burst occurs within such activated neutrophils, releasing lysosomal enzymes, oxidants and free radicals, which are involved in killing the invading bacteria but which may also damage adjacent cells. There are *high circulating levels of cytokines* and *activated neutrophils*, which stimulate fever, tachycardia and tachypnea. The activated neutrophils *adhere to vascular endothelium in key organs* remote from the source of infection and *damage* it, leading to increased vascular permeability, which in turn leads to *cellular damage within the organs*, which become dysfunctional and give rise to the clinical picture of MODS. In its most severe form, MODS may progress into *multiple system organ failure (**MSOF**). Respiratory, cardiac, intestinal, renal and liver failure ensue in combination with circulatory failure and shock.*

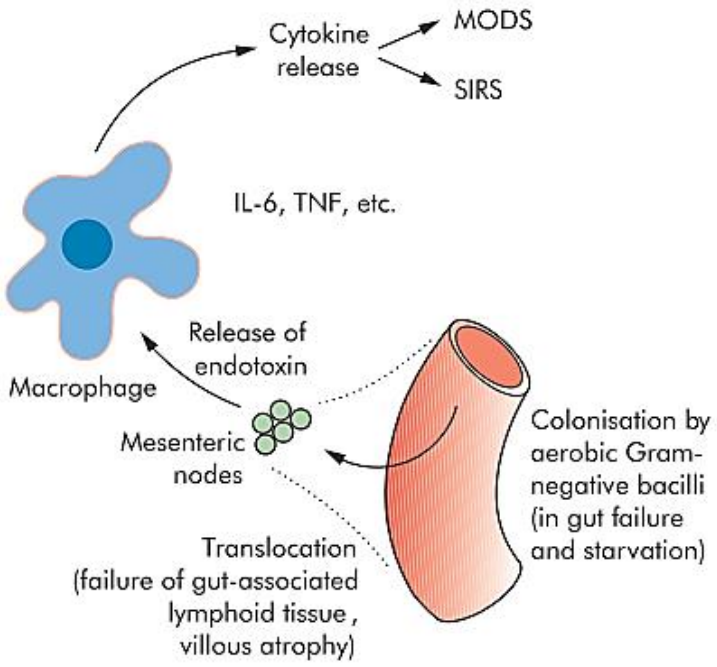


Figure 7. Development of multiple organ dysfunction syndrome (MODS) and systemic inflammatory response syndrome (SIRS). *IL* — interleukin; *TNF* — tumor necrosis factor

Organ dysfunction is quantified by an increase of ≥ 2 points on the Sequential Organ Failure Assessment (SOFA, Table 2).

Table 2

Sequential Organ Failure Assessment score

System	Score				
	0	**1**	**2**	**3**	**4**
Respiratory PaO_2/FiO_2, mmHg (kPa)	≥ 400 (53.3)	< 400 (53.3)	< 300 (40)	< 200 (26.7) with respiratory support	< 100 (13.3) with respiratory support
Coagulation Platelets, $\times 10^3/\mu L$	≥ 150	< 150	< 100	< 50	< 20
Hepatic Bilirubin, mg/dL (μmol/L)	< 1.2 (20)	1.2–1.9 (20–32)	2–5.9 (33–101)	6–11.9 (102–204)	> 12 (204)
Cardiovascular	MAP ≥ 70 mmHg	MAP < 70 mmHg	Dopamine < 5 or dobutamine	Dopamine 5.1–15 or epinephrine ≤ 0.1 or norepinephrine ≤ 0.1	Dopamine > 15 or epinephrine > 0.1 or norepinephrine > 0.1
CNS GCS score	15	13–14	10–12	6–9	< 6
Renal Creatinine, mg/dL (μmol/L)	< 1.2 (110)	1.2–1.9 (110–170)	2–3.4 (171–299)	3.5–4.9 (300–440)	> 5 (440)
Urine output, mL/24 hours				< 500	< 200

MAP = mean arterial pressure; PaO_2 = partial pressure of oxygen; FiO_2 = fraction of inspired oxygen; CNS = central nervous system; GCS = Glasgow Coma Scale. Catecholamine doses in μg/kg/minute.

The SOFA score looks at PaO2/FiO2 ratio, bilirubin, platelet count, mean arterial pressure (MAP), Glasgow Coma Scale (GCS) score, creatinine level, and urine output. An increase in SOFA score of 2 or more is correlated with a 10 % in-hospital mortality risk, which is suggestive of the life-threatening nature of sepsis. An abbreviated version of the scoring system, the quick SOFA (qSOFA) is recommended as a screening and monitoring tool for patients with suspected sepsis. The qSOFA suggests potentially life-threatening sepsis when at least two of the following parameters are met: altered mental status, systolic blood pressure of 100 mmHg or less, and respiratory rate greater than 22 breaths/minute. The qSOFA can readily identify patients at risk of poor outcome from sepsis without reliance upon laboratory or imaging data.

Abdominal compartment syndrome (ACS) is an elevation of intra-abdominal pressure (IAP) and is life-threatening complication of peritonitis. Normal IAP is approximately 5 mmHg, but it can be increased non-pathologically in obese patients and varies with respiration. Abdominal

compartment syndrome is defined as the presence of an IAP of 20 mmHg or greater. Increasing intra-abdominal pressure affects different systems:

– *Renal* — increase in the renal vascular resistance, combined with a moderate resultant decrease in cardiac output.

– *Cardiovascular* — increased IAP decreases cardiac output as well as increasing central venous pressure, systemic vascular resistance, pulmonary artery pressure, and pulmonary artery wedge pressure.

– *Respiration* – in conjunction with increased IAP, there is diaphragmatic stenting, exerting a restrictive effect on the lungs with a resultant decrease in ventilation and lung compliance, an increase in airway pressures, and a reduction in tidal volumes.

– *Visceral Perfusion* — increasing IAP may result in visceral hypoperfusion and subsequently in secondary bacterial translocation

The most accurate method for direct, invasive IAP measurement is direct needle puncture and transduction of the pressure within the abdominal cavity (e.g., during peritoneal dialysis or laparoscopy), but the gold standard for intermittent, indirect, non-invasive IAP measurement is *transduction of the pressure within the urine bladder*. The reference method for continuous indirect IAP measurement is a balloon-tipped catheter in the stomach or a continuous bladder irrigation method.

CLINICAL PRESENTATION

Irrespectively of the exact etiology, florid peritonitis is usually accompanied by well-recognized systemic and local symptoms and signs.

Systemic manifestations. These emanate from the presence of a serious infection: the patient looks ill and is toxic with a high metabolic rate, pyrexia, tachycardia and leukocytosis. If bacteria have invaded the bloodstream (bacteraemia, septicaemia), attacks of rigors (shivering) are encountered; the patient feels cold even though his or her temperature is elevated above 38 °C. The combination of fluid and electrolyte losses (vomit, fluid inside the oedematous intestinal loops and peritoneal exudate that is sequestrated) and the enhanced insensible loss caused by the pyrexia lead to dehydration with dry mouth, loss of skin turgor and collapse of the peripheral veins.

Local symptoms. The clinical picture of generalized peritonitis consists of diffuse, severe abdominal pain in a patient who looks sick and toxic. The patient typically lies motionless, and has an extremely tender abdomen with peritoneal signs consisting of board-like rigidity, rebound-tenderness, and involuntary defense-guarding. The pain of acute peritonitis is due to irritation of the somatic nerves supplying the parietal peritoneum. Its extent and exact location depend on whether the peritonitis is generalized or localized to a particular quadrant of the intra-abdominal cavity. It is always severe, constant and aggravated by

movement (passive or active) and thus the patient lies still in the supine position and may at times draw up the knees to relax the abdominal musculature.

Besides pain, one can encounter signs of intestinal paresis (absence of peristalsis, distension, vomiting and constipation).

Clinical signs are not constant; they are changing along with progression of intra-abdominal inflammation.

Clinical manifestations according to phase of peritonitis.

First phase (2–12 hours) — pain:

– Local signs are prevailing, MODS is absent;

– Severe acute pain made worse by moving or breathing., pain shock. It is firstly experienced at the site of the original lesion and spreads outwards from this point. The patient usually lies still;

– Tenderness and defense-guarding, "peritoneal signs";

– Moderate tachycardia, hypotension, increasing respirations rate, vomiting;

– Infrequent bowel sounds may still be heard for a few hours but they cease with the onset of paralytic ileus;

– Inflammatory syndrome — elevated temperature, moderate leukocytosis with left shift.

Second phase (2–3 days) — toxemia:

– Increasing signs of systemic intoxication;

– Local signs expression is reducing;

– The whole abdomen becomes rigid (generalized rigidity);

– A good way to elicit peritoneal irritation is by asking the patient to cough, shaking (gently) his bed, or by very gentle percussion of the abdomen;

– Geriatric patient may have weak abdominal musculature or may not exhibit the classical peritoneal signs;

– Distension is common and bowel sounds are absent;

– A patient is weak and thirsty, anorexic and nauseated;

– He vomits, and may have diarrhoea or be constipated;

– Thready (irregular) pulse, hypotension, breathing is shallow;

– Inflammatory syndrome — high temperature (> 38) or low (< 36) in advanced stage, leukocytosis with left shift or leucopenia.

Third phase (> 3 days) — multiple organ failure:

– MODS develops — renal, cardiovascular, pulmonary systems failure;

– Hippocratic face — sunken eyes, dry tongue, grey and drawn and anxious face;

– The patient lapses into unconsciousness;

– If not treated, finally, patient dies in peripheral circulatory failure;

– Mortality rate > 60 %.

DIAGNOSIS

Clinical examination. The diagnosis of peritonitis is mostly made by clinical examination. The local or abdominal signs are elicited by a methodical sequence of inspection, palpation, percussion and auscultation of the abdomen and digital rectal examination.

Inspection. On inspection of the normal abdomen, the abdominal wall is seen to move with respiration; it bulges with inspiration as the diaphragm descends. This normal excursion is often absent in patients with peritonitis as the abdominal muscles over the area of peritoneal inflammation undergo reflex spasm.

Palpation. The same phenomenon accounts for the tight feel of the abdominal musculature noted during light palpation and often referred to as guarding. When marked, the abdominal muscles actually feel rigid (rigidity), although descriptions of board-like rigidity are exaggerated and have conveyed the wrong impression that deep palpation is necessary to elicit this sign. In fact, deep palpation is absolutely contraindicated in all patients with acute abdomen as it serves no purpose other than to inflict severe pain on the patient and thereby lose his or her confidence. Tenderness on light palpation elicited over the affected region is a most useful and reliable sign. Rebound tenderness is experienced by the patient when the pressure of the palpating hand is released. Considerable store has been laid on this physical sign in the past. More recent studies have cast some doubt on its value in clinical practice. Certainly it must be elicited with great gentleness.

Percussion. A more humane way to evoke rebound tenderness is to tap the affected area gently or better still ask the patient to cough, which, by moving the inflamed viscera against the inflamed parietal peritoneum, reproduces the localized pain.

Auscultation. Auscultation of the abdomen in patients with peritonitis reveals a silent abdomen (no identifiable borborygmus) due to absence of the normal peristaltic activity. At times, tinkling bowel sounds may be heard. These are due to passive movement of fluid within dilated loops of inflamed gut and signify the presence of a paralytic ileus.

Digital rectal examination. No examination of a patient with an acute abdomen is complete without a rectal examination. Although this is best carried out in the left lateral position, if the patient is in severe pain it may be conducted in the supine posture with flexion and abduction of the hip joints. The specific findings on digital rectal examination may include pelvic tenderness, boggy swelling in the rectovesical pouch and tenderness caused by movement of the cervix in the female.

Additional Diagnostic Studies. X-Ray (free gas in abdominal cavity (Figure 8), atypical gas patterns (gas in the biliary tree (pneumobilia), portal

vein gas); leakage of contrast media into abdominal cavity, signs of intestinal obstruction and paresis — abnormal distension/dilatation of small bowel loops or colon with or without fluid levels).

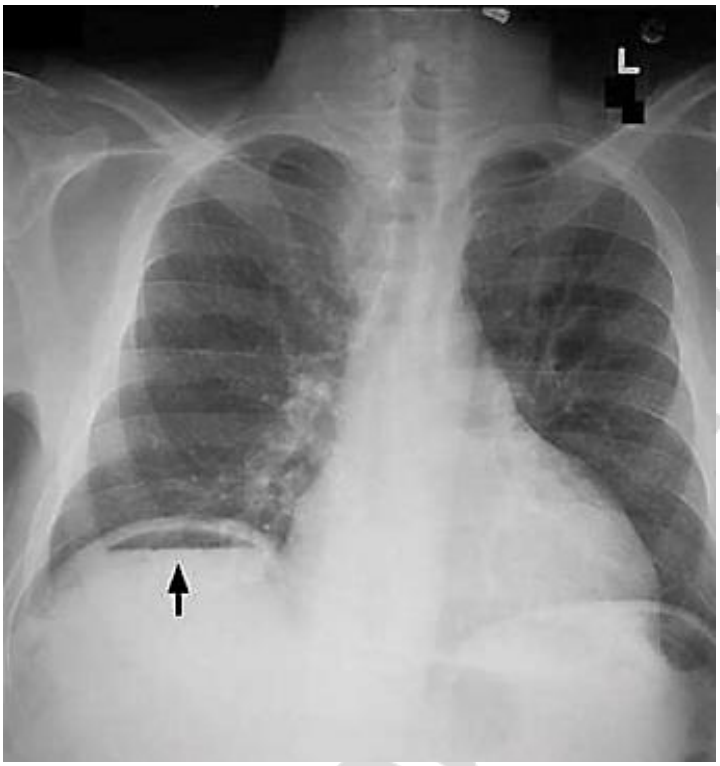


Figure 8. Abdominal X-ray in the upright position demonstrating a pneumoperitoneum with air under both diaphragms (arrow)

Ultrasound is a readily available diagnostic modality in most places. US is very accurate in the diagnosis of acute cholecystitis, acute pelvic pathology in female patients and urological pathologies. It can be diagnostic of acute appendicitis, useful in demonstrating intra-abdominal fluid — ascites, pus, or blood, localized or diffuse. An US-guided aspiration of unexplained intraperitoneal fluid can clarify the diagnosis.

CT (Figure 9) and MRI shows details that no other diagnostic method does: free gas, fluid, masses, tissue planes, inflammatory changes, opacities, blood vessels and organ perfusion.

Endoscopy is useful for diagnosing perforations of hollow organs, finding biliary or colonic occlusion etc.

In majority of cases of diffuse peritonitis there is no need in multiple and prolonged investigations because the diagnosis of “Peritonitis” was already made clinically and emergency operation is indicated anyway.

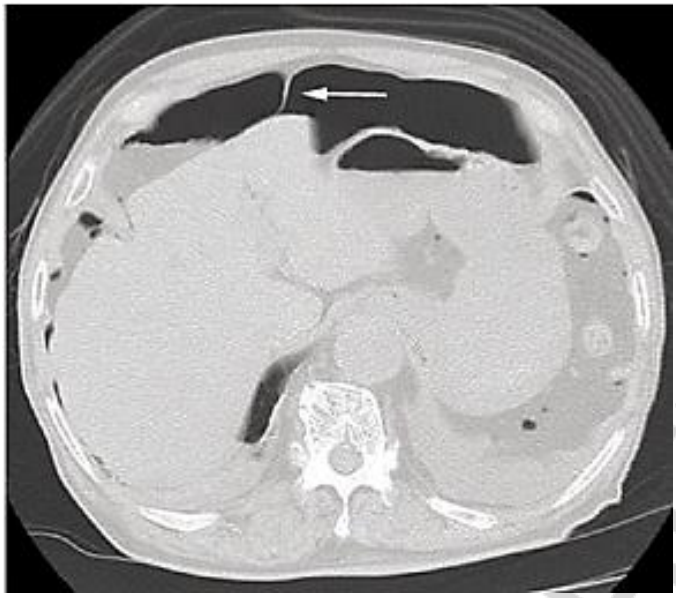


Figure 9. CT demonstrating two pockets of extraluminal gas in the epigastric region outlying the falciform ligament (arrow)

TREATMENT

The main treatment method of peritonitis is surgery.

Aggressive surgical infection cannot be cured only by the administration of antibiotics, and never was cured in the face of an ongoing source of contamination.

Other treatment modalities such as antimicrobial agents are of secondary importance to effective surgery with regard to treatment of surgical infections and overall outcome.

Also, it has been repeatedly demonstrated that delay in operative intervention, whether due to misdiagnosis or the need for additional diagnostic studies, is associated with increased morbidity and occasional mortality.

The treatment of peritonitis always involves the combination of preoperative preparation, **source control, antimicrobial agents and postoperative care**.

Preoperative preparation. Diffuse peritonitis involves inflammation and fluid production throughout the peritoneal surface, an area comparable to total body surface area. This is a major hemodynamic challenge and inflammatory stress to the host organism and must be addressed by attention to circulating blood volume, respiratory status, and other systemic parameters just as in any other critical systemic illness.

The fluid requirements of diffuse peritonitis approximate those of a 50 % body surface area burn. These fluid shifts accompanied by the cytokine response to peritonitis can easily lead to respiratory and renal failure initially followed by MODS if not treated early and aggressively. Attention to airway, breathing, and circulation must occur simultaneously with antimicrobial management.

The systemic disturbance associated with intra-abdominal infection is most readily measured by critical care scores such as Acute Physiology and Chronic Health Evaluation (APACHE) II score, SOFA scores, etc., which has been demonstrated to correlate with mortality and inversely with successful initial treatment of intra-abdominal infection.

Indications for preoperative resuscitation:

– Toxic and terminal phase of a generalized peritonitis;

– Severe comorbidities;

– Children, senior people;

– Signs of dehydration.

The standard principles of resuscitation are followed, after an initial assessment of the patient's general condition:

1. Oxygen therapy, if the patient is hypoxic, or haemoglobin oxygen saturations are less than 95 %.

2. Intravenous fluid and electrolyte replacement; plasma expanders or blood may be required in the presence of shock.

The massive transfer of fluid into the peritoneal cavity must be replaced by an appropriate amount of intravenous fluid. If systemic toxicity is evident or if the patient is old or in fragile health, a central venous pressure (or pulmonary artery wedge pressure) line and bladder catheter should be inserted; a fluid balance chart should be kept; and serial body weight measurements should be taken to monitor fluid requirements. Sufficient balanced or lactated Ringer's solution must be infused rapidly enough to correct intravascular hypovolemia promptly and to restore blood pressure and urine output to satisfactory levels. Potassium supplements are withheld until tissue and renal perfusion are adequate and urine is produced. Blood is reserved for anemic patients or those with concomitant bleeding.

3. Antibiotic therapy, with specificity to treat the broad spectrum of bowel organisms, e.g. penicillin and gentamicin or a cephalosporin together with metronidazole; therapy is guided, where possible, by checking the sensitivity of the responsible organisms isolated on a peritoneal swab or from blood cultures.

Loading doses of intravenous antibiotics directed against the anticipated bacterial pathogens should be given after fluid samples have been obtained for culture. Initial antibiotics employed include third-generation cephalosporins, ampicillin-sulbactam, ticarcillin-clavulanic acid, aztreonam or imipenem-cilastatin for gram-negative coliforms, and metronidazole or clindamycin for anaerobic organisms. The choice of single-, double- or triple-drug therapy is of less importance than adequate coverage of both anticipated aerobic and anaerobic organisms. Inadequate initial drug dosing and scheduling contribute to treatment failures. Aminoglycosides should be used judiciously because renal impairment is often a feature of peritonitis and because lowered intraperitoneal pH may impair their in vivo activity.

Empirically chosen antibiotics should be modified postoperatively by culture and sensitivity results if there is persistent or subsequent infection (seen in 15–20 % of patients). Antibiotics are continued until the patient has remained afebrile with a normal white count and a differential count of less than 3 % bands.

4. Relief of pain with NSAD or opiates, e.g. intravenous morphine.

5. Gastric aspiration by means of a nasogastric tube reduces the risk of aspiration of vomit under anaesthesia and prevents further abdominal distension by removing swallowed air.

Operative Treatment. The objectives of surgery for peritonitis are to remove all infected material (exudate, pus, necrotic tissues) with microbiologic culturing and correct the cause of peritonitis (source control), and prevent late complications (tertiary peritonitis, abscesses, wound infection etc.).

Source Control. The primary precept of surgical infectious disease therapy consists of drainage of all purulent material, débridement of all infected, devitalized tissue, and debris, and/or removal of foreign bodies at the site of infection, plus remediation of the underlying cause of infection. A discrete, walled-off purulent fluid collection (i.e., an abscess) requires drainage via percutaneous drain insertion or an operative approach in which incision and drainage take place. An ongoing source of contamination (e.g., bowel perforation) requires operative intervention, both to remove infected tissue and fluids and to remove the initial cause of infection (e.g., bowel resection).

Except in early, localized peritonitis, a midline incision offers the best surgical exposure in case of diffuse peritonitis. Materials for aerobic and anaerobic cultures of fluid and infected tissue are obtained immediately after the peritoneal cavity is entered. Occult pockets of infection are located by thorough exploration, and contaminated or necrotic material is removed. Routine radical debridement of all peritoneal and serosal surfaces does not increase survival rates. The primary disease is then treated. This may require resection (e.g., ruptured appendix or gallbladder), repair (e.g., perforated ulcer), or drainage (e.g., acute pancreatitis).

Attempts to reanastomose resected bowel in the presence of extensive sepsis or intestinal ischemia often lead to leakage. Temporary stomas are safer, and these can be taken down several weeks later after the patient has recovered from the acute illness. Surgical wounds should seldom be closed primarily. They should be left open in grossly soiled cases or delayed primary closure employed in those with less contamination.

Intraoperative Peritoneal Lavage. In diffuse peritonitis, lavage with copious amounts (> 3 L) of warm isotonic crystalloid solution removes gross particulate matter as well as blood and fibrin clots and dilutes residual bacteria. The addition of antiseptics or antibiotics to the irrigating solution is generally useless or even harmful because of induced adhesions (e.g., tetracycline, povidone-iodine). Antibiotics given parenterally will reach bactericidal levels in

peritoneal fluid and may afford no additional benefit when given by lavage. Furthermore, lavage with aminoglycosides can produce respiratory depression and complicate anesthesia because of the neuromuscular blocking action of this group of drugs. After lavage is completed, all fluid in the peritoneal cavity must be aspirated because it may hamper local defense mechanisms by diluting opsonins and removing surfaces upon which phagocytes destroy bacteria.

Peritoneal Drainage. Drainage of the free peritoneal cavity is ineffective and often undesirable. Not only are drains quickly isolated from the rest of the peritoneal cavity, but they also still act as a channel for exogenous contamination. Prophylactic drainage in diffuse peritonitis does not prevent abscess formation and may even predispose to abscesses or fistulas. Drainage is useful for residual focal infection or when continued contamination is present or likely to occur (e.g., fistula). It is indicated for localized inflammatory masses that cannot be resected or for cavities that cannot be obliterated. Soft sump drains with continuous suction through multiple side perforations are effective for large volumes of fluid. Smaller volumes of fluid are best handled with closed drainage systems (e.g., Jackson–Pratt drains). Large cavities with thick walls may be drained by several large Penrose drains placed in a dependent position.

To achieve more effective peritoneal drainage in severe peritonitis, some surgeons have previously left the entire abdominal wound open to widely expose the peritoneal cavity — laparostomy (open abdomen, Figure 10). Besides requiring intensive nursing and medical support to cope with massive protein and fluid losses (averaging 9 L the first day), there are serious complications such as spontaneous fistulization, wound sepsis, segmental colonic necrosis, and large incisional hernias. Consequently, this method is seldom employed now.

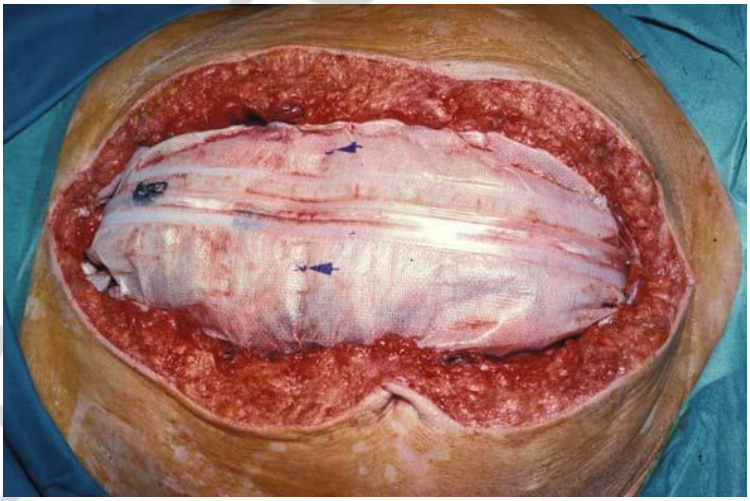


Figure 10. Laparostoma (open abdomen)

An alternative method is to re-explore the abdomen every 1–3 days until all loculations have been adequately drained (second look). The wound may be closed temporarily with a sheet of polypropylene (Marlex) mesh that contains

a nylon zipper or Velcro to avoid a tight abdominal closure and to facilitate repeated opening and closing. Other options include the use of a plastic sheet (Bogota bag) or a wound vacuum device bridging over the open fascia. Exploration may even be performed in the intensive care unit with heavy sedation. Available data suggest that this method should be restricted to selected patients with long-standing (more than 48 hours) extensive intraperitoneal sepsis associated with multiple organ failure (high sepsis scores).

Management of Abdominal Distention. Abdominal distention caused by ileus frequently accompanies peritonitis, and intraoperative decompression of the intestine is often needed.

– Pushing the small bowel content to the large bowel or stomach;

– Gastrostomy;

– Jejunostomy (transnasal, percutaneous) — plus prolonged nutritional support;

– Colostomy (drainage or loop colostomy).

An alternative approach is to close the abdomen temporarily with a sheet of plastic (Bogota bag) to avoid further distention, increased intra-abdominal pressure, and respiratory or renal problems (abdominal compartment syndrome).

Postoperative care. Intensive care monitoring, often with ventilatory support, is mandatory in unstable and frail patients. Achieving hemodynamic stability to perfuse major organs is the immediate objective, and this may entail the use of cardiac inotropic agents besides fluid and blood product supportive measures. Antibiotics are given for 10–14 days, depending on the severity of peritonitis. A favorable clinical response is evidenced by well-sustained perfusion with good urine output, reduction in fever and leukocytosis, resolution of ileus, and a returning sense of well-being. The rate of recovery varies with the duration and degree of peritonitis.

The early removal of all nonessential catheters (arterial, central venous, urinary, and nasogastric) reduces the risk of secondary infected foci. Drains should be removed or advanced once drainage diminishes and becomes more serous in nature. Excessive or prolonged suction may produce fistulas or bleeding even within a few days.

Growing awareness of the association between proximal gut colonization with Candida, Streptococcus faecalis, pseudomonas, and coagulase-negative staphylococci and secondary nosocomial infections and subsequent multiple organ failure has encouraged early gut feeding and discontinuation of unnecessary antibiotics whenever feasible.

Postoperative Complications. Postoperative complications are frequent and may be divided into local and systemic problems. Deep wound infections, residual abscesses and intraperitoneal sepsis, anastomotic breakdown, and fecal fistula formation usually become manifest toward the end of the first postoperative week. Persistent high or swinging fever, inability to wean off

cardiac inotropes, generalized edema with unexplained continued high fluid requirements, increased abdominal distention, prolonged mental apathy and weakness, or general failure to improve despite intensive treatment may be the sole indicators of residual intra-abdominal infection. This should prompt a thorough examination of the patient for infected catheters and an abdominal CT scan. Percutaneous catheter drainage of localized abscesses or open reexploration is undertaken as needed.

The role of planned relaparotomy, as opposed to laparotomy on demand (in relation to deterioration in oxygenation, organ function, and inotrope requirements), in such cases remains controversial, but case-control studies have shown that planned relaparotomy does not appear to reduce mortality significantly and may increase secondary complications, including the incidence of intestinal fistula. In severe cases of tertiary peritonitis, where sepsis frequently coexists with abdominal hypertension, management may necessitate leaving the abdomen open (laparostomy) to facilitate drainage of the septic focus and planned, staged reconstructive surgery of the gastrointestinal tract and abdominal wall after recovery.

Indications to reoperation:

1. Persisting (tertiary) peritonitis.
2. Early intestinal obstruction by adhesions.
3. Hemorrhage.
4. Formation of intraperitoneal abscesses.
5. Anastomosis leakage.

Regardless of the approach to source control, review of multiple series suggests that in approximately 25 % of cases a second effort at source control will be necessary to resolve the infection. This may be another open operation or another percutaneous drain or conversion from one approach to the other.

A subsequent intervention is indicated when the patient fails to improve or worsens following the primary intervention. Ideally, the initial effort at source control should be successful.

Prognosis. If properly treated, typical cases of surgical peritonitis (e.g., perforated peptic ulcer, appendicitis and diverticulitis) have an overall mortality rate less than 10 %. The mortality rate rises to about 40% in the elderly, and/or in those with significant underlying illness, as well as in cases that present late (after 48 hours).

The mortality rate of generalized peritonitis is about 40 %. Factors contributing to a high mortality rate include the type of primary disease and its duration, presence of multiple organ failure before treatment, and the age and general health of the patient. Mortality rates are consistently below 10 % in patients with perforated ulcers or appendicitis; in young patients; in those having less extensive bacterial contamination; and in those diagnosed and operated upon early. Patients with distal small bowel or colonic perforations or

postoperative sepsis tend to be older, to have concurrent medical illnesses and greater bacterial contamination, and to have a greater propensity to renal and respiratory failure; their mortality rates are about 50 %. Markedly poor physiologic indices (e.g., APACHE II or Mannheim Peritonitis Index), reduced cardiac status, and low preoperative albumin levels identify high-risk patients who require intensive treatment to reduce a daunting mortality rate.

Non-operative Treatment of Peritonitis. Non-operative treatment of peritonitis is possible in following situations:

1. Acute pancreatitis.
2. Some cases of typhoid peritonitis.
3. Pelvic peritonitis. Pus can be drained vaginally, or rectally in a male (uncommon).
4. Pus which is mainly under the diaphragm.
5. Peritonitis which has been confirmed by aspiration, but the patient is too ill to withstand laparotomy. In this case operation should be delayed until patient will improve.

Management includes antibiotics, nasogastric aspiration, infusion therapy, correction of electrolytes, and drainage of abdominal fluid collections.

Principles of Antimicrobial Therapy. The initial antimicrobial treatment of intra-abdominal infection is always empiric. The diagnosis is made clinically, and antimicrobial therapy is started when the diagnosis is made and while the patient is being prepared for the source control procedure.

Cultures should be taken in the operating room or radiology suite, but bacterial identification will not be available for at least a day for aerobic and facultative species and for at least 2 days or more for anaerobes. Susceptibility data will not be available for an equivalent amount of time after that. Accordingly, the first 2 to 5 days of treatment are empiric and will be completed without specific susceptibility data.

The potential bacterial flora of peritonitis is complex. At least 400 different bacterial species can be identified in the human colon. Clinical microbiology laboratories usually recover only 3 to 5 different pathogens from intra-abdominal infections. The clinical findings represent a major simplification of the flora that is the usual contaminating source.

Effective source control and antibiotic therapy is associated with low failure rates and a mortality rate of approximately 5 to 6 %; inability to control the source of infection leads to mortality greater than 40 %.

Empiric therapy. Empiric antimicrobial regimen for the treatment of peritonitis should have broad activity against aerobic and facultative gram-negative bacilli and against enteric anaerobes. The regimen should not be completely inactive against staphylococcus and streptococcus species. It should be limited to a short course of drug (3 to 5 days), and should be curtailed as soon

as possible based on microbiologic data (i.e., absence of positive cultures) coupled with improvements in the clinical course of the patient.

Antimicrobial agents for intra-abdominal infection:

Single Agents:

– Cefoxitin;

– Cefotetan;

– Ampicillin/sulbactam;

– Ticarcillin/clavulanic acid;

– Piperacillin/tazobactam;

– Imipenem/cilastatin;

– Meropenem.

Combination Regimen:

– Cefuroxime + metronidazole;

– Third-/fourth-generation cephalosporin (cefotaxime, ceftriaxone, ceftizoxime, ceftazidime, cefepime) + an antianaerobic;

– Aztreonam + clindamycin;

– Ciprofloxacin + metronidazole;

– Aminoglycoside (gentamicin, tobramycin, netilmicin, amikacin) + an antianaerobic (clindamycin or metronidazole).

Among surgical patients, the manner in which therapy is used, particularly in relation to the use of microbiologic data (culture and antibiotic sensitivity patterns), differs depending on whether the infection is monomicrobial or polymicrobial.

It is important, however, to ensure that the antimicrobial coverage choice is adequate, because delay in appropriate antibiotic treatment has been shown to be associated with increased mortality. Within 24 to 72 hours, culture and sensitivity reports will allow refinement of the antibiotic regimen to select the most efficacious agent.

The antibiotic regimen should not be modified solely on the basis of culture information, as it is less important than the clinical course of the patient. For example, patients who undergo appendectomy for gangrenous, perforated appendicitis, or bowel resection for intestinal perforation, should receive an antimicrobial agent or agents directed against aerobes and anaerobes for 3 to 5 days, occasionally longer.

Duration. Cogent data exist to support the opinion that satisfactory outcomes are achieved with 12 to 24 hours of therapy for penetrating GI trauma in the absence of extensive contamination, 3 to 5 days of therapy for perforated or gangrenous appendicitis, 5 to 7 days of therapy for treatment of peritonitis due to a perforated viscus with moderate degrees of contamination, and 7 to 14 days of therapy to adjunctively treat extensive peritoneal inflammation (e.g., feculent peritonitis) or that occurring in the immunosuppressed host. It needs repeating that the eventual outcome is more closely linked to the ability of

the surgeon to achieve effective source control than to the duration of antibiotic administration.

In the later phases of postoperative antibiotic treatment of serious intra-abdominal infection, the absence of an elevated WBC count and lack of fever [< 38.6 °C (100.5 °F)] provide close to complete assurance that infection has been eradicated. Under these circumstances, antibiotics can be discontinued with impunity. However, the presence of one or more of these indicators does not mandate continuing antibiotics or changing the antibiotic(s) administered. Rather, a search for an extra-abdominal source of infection or a residual or ongoing source of intra-abdominal infection (e.g., abscess or leaking anastomosis) should be sought, the latter mandating maneuvers to effect source control.

SPECIFFIC TYPES OF PERITONITIS

Primary Peritonitis (PP)

PP is the uncommon condition seen in:

– Cirrhosis with ascites;
– Nephrotic syndrome;
– Following splenectomy;
– Systemic lupus erythematosus.

Impairment of the hepatic reticuloendothelial system and compromised peripheral destruction of bacteria by neutrophils promotes bacteremia, which readily infects ascitic fluid that has reduced bacterium-killing capacity. Primary peritonitis is most closely associated with cirrhosis and advanced liver disease with a low ascitic fluid protein concentration. It is also seen in patients with the nephrotic syndrome or systemic lupus erythematosus, or after splenectomy during childhood. Recurrence is common in cirrhosis and often proves fatal.

Clinical Findings. The clinical presentation simulates secondary bacterial peritonitis, with abrupt onset of fever, abdominal pain, distention, and rebound tenderness. However, one-fourth of patients have minimal or no peritoneal symptoms. Most have clinical and biochemical manifestations of advanced cirrhosis or nephrosis. Leukocytosis, hypoalbuminemia, and a prolonged prothrombin time are characteristic findings. The diagnosis hinges upon examination of the ascitic fluid, which reveals a white blood cell count greater than 500/L and more than 25 % polymorphonuclear leukocytes. A blood-ascitic fluid albumin gradient greater than 1.1 g/dL, a raised serum lactic acid level (> 33 mg/dL), or a reduced ascitic fluid pH (< 7.31) supports the diagnosis. Bacteria are seen on Gram-stained smears in only 25 % of cases. Culture of ascitic fluid inoculated immediately into blood culture media at the bedside usually reveals a single enteric organism, most commonly E. coli, Klebsiella, or Streptococci, but Listeria monocytogenes has been reported in immunocompromised hosts.

The diagnosis is established based on a patient who has ascites for medical reasons, physical examination that reveals diffuse tenderness and guarding without localized findings, absence of pneumoperitoneum on abdominal flat plate and upright roentgenograms, the presence of more than 100 WBCs/mL, and microbes with a single morphology on Gram's stain performed on fluid obtained via paracentesis. Subsequent cultures will typically demonstrate the presence of gram-positive organisms in patients receiving peritoneal dialysis. In patients without this risk factor, organisms can include E. coli, K. pneumoniae, pneumococci, and others, although many different pathogens can be causative.

Treatment. Antibiotic prophylaxis is of no proven value. Systemic antibiotics with third-generation cephalosporins (e.g., cefotaxime) or a beta-lactam-clavulanic acid combination along with supportive treatment are begun once the diagnosis has been established. The 50 % average mortality rate is due to peritonitis in only about a third of cases. Multiple organ failure as indicated by gastrointestinal bleeding, hepatic encephalopathy, and renal failure are ominous signs.

Treatment consists of administration of an antibiotic to which the organism is sensitive; often 14 to 21 days of therapy are required. Removal of indwelling devices (e.g., peritoneal dialysis catheter or peritoneovenous shunt) may be required for effective therapy of recurrent infections.

Tuberculous Peritonitis

Pathophysiology. Tuberculosis peritonitis is encountered in 0.5 % of new cases of tuberculosis. It presents as a primary infection without active pulmonary, intestinal, renal, or uterine tube involvement. Its cause is reactivation of a dormant peritoneal focus derived from hematogenous dissemination from a distant nidus or breakdown of mesenteric lymph nodes (Figure 11).

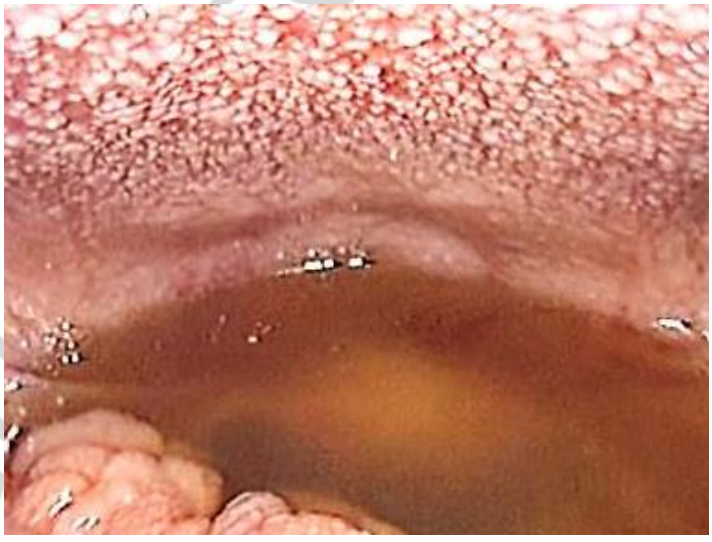


Figure 11. Laparoscopic view of Tuberculosis peritonitis

Some cases occur as a systemic manifestation of extra-abdominal infection. Multiple small, hard, raised, whitish tubercles studding the peritoneum,

omentum, and mesentery are the distinctive finding. A cecal tuberculoma, matted lymph nodes, or omental involvement may form a palpable mass.

The disease affects young persons, particularly women, and is more prevalent in countries where tuberculosis is still endemic. AIDS patients are especially susceptible to development of extrapulmonary tuberculosis.

Clinical Findings. Chronic symptoms (lasting more than a week) include abdominal pain and distention, fever, night sweats, weight loss, and altered bowel habits. Ascites is present in about half of cases, especially if the disease is of long standing, and may be the primary manifestation. A mass may be felt in a third of cases. The differential diagnosis includes Crohn's disease, carcinoma, hepatic cirrhosis, and intestinal lymphoma. One-fourth of patients have acute symptoms suggestive of acute bowel obstruction or peritonitis that mimics appendicitis, cholecystitis, or a perforated ulcer.

Detection of an extra-abdominal site of tuberculosis, evident in half of cases, is the single most useful diagnostic clue. Pleural effusion is present in up to 50 % of patients. Paracentesis, laparoscopy, or peritoneal biopsy is applicable only in patients with ascites. The peritoneal fluid is characterized by a protein concentration above 3 g/dL with less than 1.1 g/dL serum-ascitic fluid albumin difference and lymphocyte predominance among white blood cells. Definitive diagnosis is possible in 80 % of cases by culture (often taking several weeks) and direct smear. A PPD skin test is useful only when positive (about 80 % of cases). Hematologic and biochemical studies are seldom helpful, and leukocytosis is uncommon. The sedimentation rate is elevated in many cases. The presence of high-density ascites or soft tissue masses on ultrasonography or CT scan supports the diagnosis. Young patients from endemic areas who present with classic symptoms or who have suggestive imaging findings should undergo diagnostic laparoscopy, which may obviate laparotomy.

Treatment. In chronic cases, nonoperative therapy is preferable if the diagnosis can be established. Most patients presenting with acute symptoms are diagnosed only by laparotomy. In the absence of intestinal obstruction or perforation, only a biopsy of a peritoneal or omental nodule should be taken. Obstruction due to constriction by a tuberculous lesion usually develops in the distal ileum and cecum, although multiple skip areas along the small bowel may exist. Localized short segments of diseased bowel are best treated by resection with primary anastomosis. Multiple strictured areas may be managed either by side-to-side bypass or a stricturoplasty of partially narrowed segments.

Combination antituberculosis chemotherapy should be started once the diagnosis is confirmed or considered likely. A favorable response is the rule, but isoniazid and rifampin must be continued for 18 months postoperatively.

Granulomatous Peritonitis

Pathophysiology. Talc (magnesium silicate), cornstarch glove lubricants, gauze fluffs, and cellulose fibers from disposable surgical fabrics may elicit

a vigorous granulomatous (probably a delayed hypersensitivity) response in some patients 2–6 weeks after laparotomy. The condition is uncommon now that surgeons wipe clean their gloves before handling abdominal viscera. Less rarely, granulomatous peritonitis may develop as a hypersensitivity reaction to other foreign material (e.g., intestinal ascariasis or food particles from a perforated ulcer). This process should be distinguished from congenital peritoneal encapsulation or abdominal cocoon.

Clinical Findings. Besides abdominal pain, which is often out of proportion to the low-grade fever, there may be nausea and vomiting, ileus, and other systemic complaints. Abdominal tenderness is usually diffuse but mild. Free abdominal fluid, if detectable, should be tapped and inspected for the diagnostic Maltese cross pattern of starch particles.

Treatment. Reoperation achieves little and should be avoided if the diagnosis can be made. Most patients undergo reexploration because they present an erroneous impression of postoperative bowel obstruction or peritoneal sepsis. The diffuse hard, white granulomatous masses studding the peritoneum and omentum are easily mistaken for cancer or tuberculosis unless a biopsy specimen is taken to demonstrate foreign body granulomas.

If granulomatous peritonitis is suspected, the response to treatment with corticosteroids or other anti-inflammatory agents is often so dramatic as to be diagnostic in itself. After clinical improvement, intravenous methylprednisolone can be replaced by oral prednisone for 2–3 weeks. The disease is self-limited and does not predispose to late intestinal obstruction.

Postoperative peritonitis

Postoperative peritonitis is an organ-space surgical-site infection. 15–30 % of all intra-abdominal infections occur following an operation. The diagnosis is usually delayed. The most common cause is a technical error compromising the vascular supply to an anastomosis, resulting in necrosis and leakage of intestinal contents into the peritoneal cavity. Iatrogenic perforation of a hollow viscus is another cause. An intra-abdominal hematoma may become secondarily infected, resulting in an abscess.

Treatment. Management is operative, as in other forms of secondary peritonitis, although nonoperative drainage with ultrasound or CT guidance is a valid option for an abscess not associated with an anastomosis.

Antimicrobial therapy is more difficult, due to the possible selection of resistant bacteria by preoperative antibiotic therapy. Any antibiotics administered should not only cover the specific bacteria isolated but should also be effective against possible pathogens from the facultative and obligate anaerobic bowel flora; usually a third-generation cephalosporin or a quinolone + metronidazole are sufficient. Other options are imipenem or one of the extended-spectrum penicillin/β-lactamase-inhibitor combinations. If a resistant Enterobacter or Serratia present problems for treatment, the synergism seen between

aminoglycosides and β-lactam antibiotics should be exploited. Enterococci rarely cause a problem and don't need to be treated specifically when otherwise adequate aerobic and anaerobic antibiotic coverage is provided.

Localized peritonitis (intraperitoneal abscesses)

An intra-abdominal abscess is a collection of infected fluid within the abdominal cavity. Gastrointestinal perforations, postoperative complications, penetrating trauma, and genitourinary infections are the most common causes.

An abscess forms by one of two modes: it may develop (1) adjacent to a diseased viscus (e.g., with perforated appendix, Crohn's enterocolitis, or diverticulitis) or (2) as a result of external contamination (e.g., postoperative subphrenic abscesses) (Table 3).

Table 3

Common Sites and Causes of Intraperitoneal Abscesses

Site	Cause
Right lower quadrant	Appendicitis, perforated ulcer, regional enteritis
Left lower quadrant	Colorectal perforation (diverticulitis, carcinoma, inflammatory bowel diseases)
Pelvis	Appendicitis, colorectal perforation, gynecologic sepsis, postoperative complications
Subphrenic region	Postoperative complications following gastric or hepatobiliary surgery or splenectomy, perforated ulcer, acute cholecystitis, appendicitis, pancreatitis (lesser sac)
Interloop	Postoperative bowel perforation

In 30 % of cases, the abscess occurs as a sequela of generalized peritonitis. Interloop and pelvic abscesses form if extravasated fluid gravitating into a dependent or localized area becomes secondarily infected.

A number of potential spaces in the peritoneal cavity can be the site of intraabdominal abscesses. Figure 12 shows a simplified classification of the peritoneal spaces.

The colon divides the abdomen into the supra- and infracolic spaces and into the right and left paracolic gutters. The supracolic space contains the left and right subphrenic spaces and the subhepatic space, which is continuous with the hepatorenal space. When a patient is lying supine, the hepatorenal space is the most dependent part of the peritoneal cavity. The supracolic space also contains the lesser sac behind the lesser omentum. The infracolic space is divided by the mesentery of the small intestine, whose oblique attachment to the retroperitoneum extends from the right side of L-4 to the left side of L-1, into a right and left infracolic space. The pelvic cavity begins at the promontory of the sacrum. The rectum divides the pelvic cavity into a prerectal space anteriorly — rectovesical in men and rectovaginal (pouch of Douglas) in women — and posteriorly into a retrorectal or presacral space.

All of these spaces are potential sites of intraabdominal abscesses. Abdominal abscesses may also develop between loops of small intestine, where they are referred to as interloop abscesses.

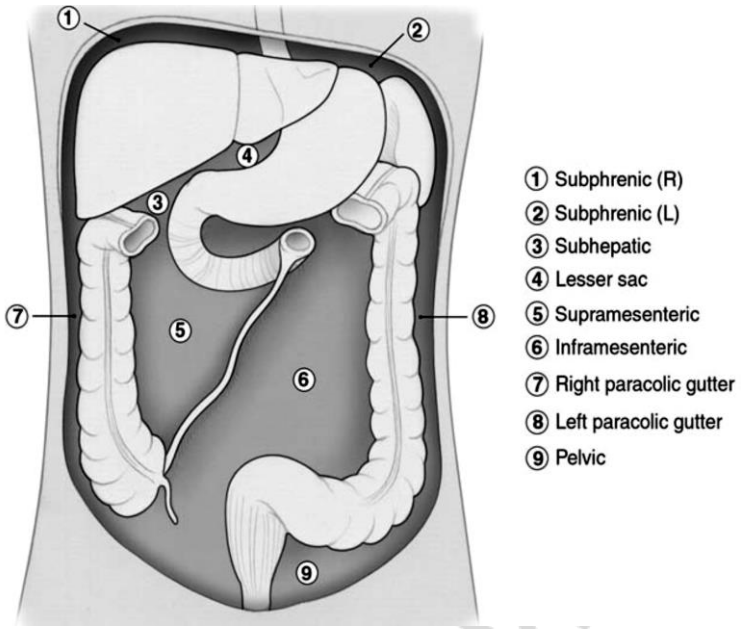


Figure 12. Peritoneal spaces

Symptoms and Signs. An intraperitoneal abscess should be suspected in any patient with a predisposing condition. Fever, tachycardia, and pain may be mild or absent, especially in patients receiving antibiotics. A deep-seated or posteriorly situated abscess may exist in seemingly well individuals whose only symptom is persistent fever. Not infrequently, prolonged ileus or a sluggish recovery in a patient who has had recent abdominal surgery or peritoneal sepsis, rising leukocytosis, or nonspecific radiologic abnormality provides the initial clue. A mass is seldom felt except late in patients with lower quadrant or pelvic lesions. Irritation of contiguous structures may produce lower chest pain, dyspnea, referred shoulder pain or hiccup, or basilar atelectasis or effusion in subphrenic abscesses; or diarrhea or urinary frequency in pelvic abscesses. The diagnosis is more difficult in postoperative, chronically ill, confused, or diabetic patients and in those receiving immunosuppressive drugs, a group particularly susceptible to septic complications.

Sequential multiple organ failure — principally respiratory, renal, or hepatic failure — or stress gastrointestinal bleeding with disseminated intravascular coagulopathy is highly suggestive of intra-abdominal infection.

Laboratory Findings. A raised leukocyte count, abnormal liver or renal function test results, hyperglycemia, and abnormal arterial blood gases are nonspecific signs of infection. Serial postoperative measurement of serum lysozyme (derived from phagocytic cells) is a promising but not widely available test that appears to be highly specific for intra-abdominal pus.

Persistently positive blood cultures point strongly to an intra-abdominal focus. A cervical smear demonstrating gonococcal infection is of specific value in diagnosing tubo-ovarian abscess.

Imaging Studies. Plain *x-rays* may suggest an abscess in up to one-half of cases. In subphrenic abscesses, the chest x-ray may show pleural effusion, a raised hemidiaphragm, basilar infiltrates, or atelectasis. Abnormalities on plain abdominal films include an ileus pattern, soft tissue mass, air-fluid levels, free or mottled gas pockets, effacement of properitoneal or psoas outlines, and displacement of viscera. Many of these findings are vague or nonspecific, but they may suggest the need for a CT scan. Barium contrast studies interfere with and have been largely superseded by other imaging techniques. A water-soluble upper gastrointestinal series may reveal an unsuspected perforated viscus or outline perigastric and lesser sac abscesses.

Real-time *ultrasonography* is sensitive (about 80 % of cases) in diagnosing intra-abdominal abscesses. The findings consist of a sonolucent area with well-defined walls containing fluid or debris of variable density. Bowel gas, intervening viscera, skin incisions, and stomas interfere with ultrasound examinations, limiting their efficacy in postoperative patients. Nevertheless, the procedure is readily available, portable, and inexpensive, and the findings are specific when correlated with the clinical picture. Ultrasonography is most useful when an abscess is clinically suspected, especially for lesions in the right upper quadrant and the paracolic and pelvic areas.

CT scan of the abdomen, the best diagnostic study, is highly sensitive (over 95 % of cases) and specific. Neither gas shadows nor exposed wounds interfere with CT scanning in postoperative patients, and the procedure is reliable even in areas poorly seen on ultrasonography. Abscesses appear as cystic collections with density measurements of between 0 and 15 attenuation units. Resolution is increased by contrast media (e.g., sodium diatrizoate) injected intravenously or instilled into hollow viscera adjacent to the abscess. One drawback of CT scan is that diagnosis may be difficult in areas with multiple thick-walled bowel loops or if a pleural effusion overlies a subphrenic abscess, so that occasionally a very large abscess is missed. CT- or ultrasonography-guided needle aspiration can distinguish between sterile and infected collections in uncertain cases.

Gallium-67 citrate and indium 111-labeled autologous leukocyte scans are rarely indicated because, compared to other modalities, they do not provide a timely answer, have high false-positive and false-negative rates, and provide less anatomic localization.

The scanning time, patient inaccessibility during scan acquisition, and upper respiratory motion have limited the usefulness of MRI in the investigation of upper abdominal abscesses. CT scan is generally preferable.

Principles of treatment. Treatment consists of prompt and complete drainage of the abscess, control of the primary cause, and adjunctive use of

effective antibiotics. Depending upon the abscess site and the condition of the patient, drainage may be achieved by operative or nonoperative methods. Percutaneous drainage is the preferred method for single, well-localized, superficial bacterial abscesses that do not have fistulous communications or contain solid debris. Following CT scan or ultrasonographic delineation, a needle is guided into the abscess cavity; infected material is aspirated for culture; and a suitably large drainage catheter is inserted.

Postoperative irrigation is vital to remove debris and ensure catheter patency. This technique is not appropriate for multiple or deep (especially pancreatic) abscesses or for patients with ongoing contamination, fungal infections, or thick purulent or necrotic material. Percutaneous drainage can be performed in about 75 % of cases. The success rate exceeds 80 % in simple abscesses but is often less than 50 % in more complex ones. It is heavily influenced by the availability of appropriate equipment and the experience of the radiologist performing the drainage. Complications include septicemia, fistula formation, bleeding, and peritoneal contamination.

Open drainage is reserved for abscesses for which percutaneous drainage is inappropriate or unsuccessful. These include many cases where there is a persistent focus of infection (e.g., diverticulitis or anastomotic dehiscence) that needs to be controlled. In cases without evidence of continued soiling, the direct extraserous route has the advantage of establishing dependent drainage without contaminating the rest of the peritoneal cavity. Only light general anesthesia or even local anesthesia is necessary, and surgical trauma is minimized. Right anterior subphrenic abscesses can be drained by a subcostal incision (Figure 13). Posterior subdiaphragmatic and subhepatic lesions can be decompressed posteriorly through the bed of the resected twelfth rib (Nather-Ochsner incision, Figure 13) or by a lateral extraserous method (DeCosse incision). Most lower quadrant and flank abscesses can be drained through a lateral extraperitoneal approach. Pelvic abscesses can often be detected on pelvic or rectal examination as a fluctuant mass distorting the contour of the vagina or rectum. If needle aspiration directly through the vaginal or rectal wall returns pus, the abscess is best drained by making an incision in that area. In all cases, digital or direct exploration must ensure that all loculations are broken down. Penrose and sump drains are used to allow continued drainage postoperatively until the infection has resolved. Serial sonograms or imaging studies help document obliteration of the abscess cavity.

Transperitoneal exploration is indicated if the abscess cannot be localized preoperatively, if there are several or deep-lying lesions, if an enterocutaneous fistula or bowel obstruction exists, or if previous drainage attempts have been unsuccessful. This is especially likely in postoperative patients with multiple abscesses and persistent peritoneal soiling. The need to achieve complete drainage fully justifies the greater stress of laparotomy and the small possibility

that infection might be spread to other uninvolved areas. Laparoscopy alone is often inadequate, especially in critically toxic patients without a localized focus.

Satisfactory drainage is usually evidenced by improving clinical findings within 3 days after starting treatment. Failure to improve indicates inadequate drainage, another source of (or ongoing) sepsis, or organ dysfunction. Additional localizing studies and repeated percutaneous or operative drainage should be undertaken urgently (i.e., within 24–48 hours, depending on the severity of the case). Failure to acknowledge adequate progress delays essential studies and incurs higher mortality.

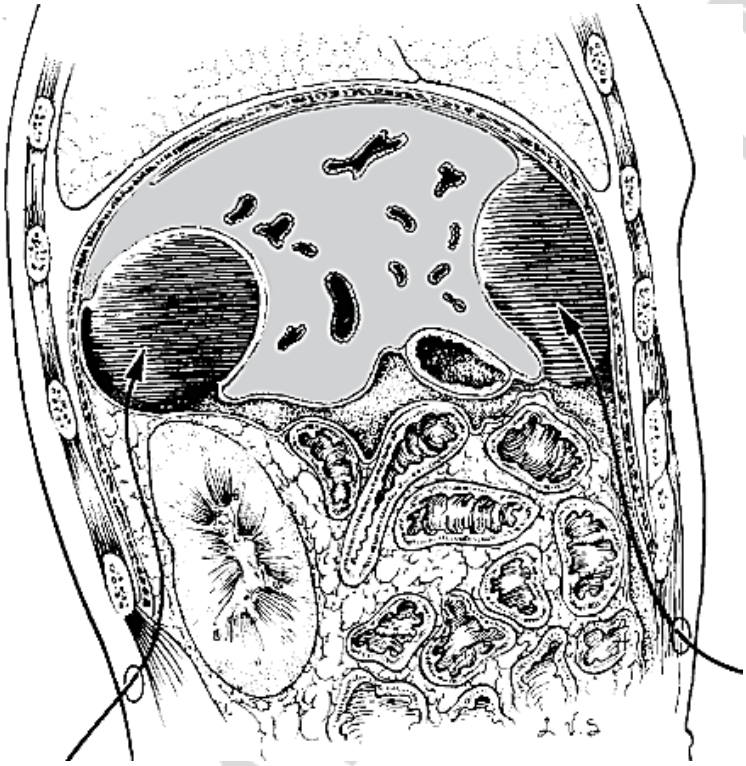


Figure 13. Extraperitoneal approaches to the right subphrenic spaces. An abscess in the anterior subhepatic space usually requires transperitoneal drainage. Posterior abscesses may also be drained laterally

Prognosis. The mortality rate of serious intra-abdominal abscesses is about 30 %. Deaths are related to the severity of the underlying cause, delay in diagnosis, multiple organ failure, and incomplete drainage. Right lower quadrant and pelvic abscesses are usually caused by perforated ulcers and appendicitis in younger individuals. They are readily diagnosed and treated, and the mortality rate is less than 5 %. Diagnosis is often delayed in older patients; this increases the likelihood of multiple organ failure. Decompensation of two major organ systems is associated with a mortality rate of over 50 %. Shock is an especially ominous sign. Subphrenic, deep, and multiple abscesses frequently require operative drainage and are associated with a mortality rate of over 40 %. An untreated residual abscess is nearly always fatal.

TESTS

1. The pathological effects of peritonitis are:
a) Formation of lung emphysema;
b) Loss of fluid;
c) Paralytic ileus;
d) Absorption of toxins from the large, inflamed surface of peritoneum;
e) Abdominal distension that lead to lung collapse and pneumonia;
f) Loss of electrolytes;
g) Hemorrhage.

2. The common species that are found in tertiary peritonitis are:
a) E. coli;
b) Bacteroides species;
c) Candida;
d) Enterococci;
e) Streptococcus fecalis;
f) Coagulase-negative staphylococci.

3. Classification of peritonitis by spreading:
a) Total;
b) Local;
c) Minimal;
d) Large;
e) Small;
f) Generalized.

4. Manifestations of the third (terminal) phase of peritonitis are:
a. Hippocratic face;
b) Prevail of the local signs;
c) Bowel sounds are absent;
d) Renal, cardiovascular, pulmonary systems failure;
e) Hypotension;
f) Distention of the abdomen.

5. The main treatment method of peritonitis is:
a) surgery;
b) conservative treatment.

6. Best surgical approach to the subphrenic abscess:
a) drainage under ultrasound/CT guidance;
b) median laparotomy;
c) laparoscopy;
d) systemic antibiotics only.

7. According to peritoneal covering, the 1st part of duodenum has:

a) Retroperitoneal covering;
b) Mesoperitoneal covering;
c) Intraperitoneal covering.

8. The functions of the peritoneum are:

a) Smooth gliding of the small intestine within the peritoneal cavity;
b) Digestive processes;
c) Fluid exchange;
d) Control the body temperature;
e) Elimination of bacteria and toxic products.

9. According to peritoneal covering, the stomach is:

a) Mesoperitoneal organ;
b) Retroperitoneal organ;
c) Intraperitoneal organ.

10. Goals of preoperative preparation in peritonitis are:

a) to find the patient's relatives;
b) to improve respiratory status;
c) to prepare the surgeon (food, coffee etc.);
d) to prepare the operation room for surgery;
e) to fill in medical records;
f) to correct a major hemodynamic challenge and inflammatory stress.

Answers: 1 – b, c, d, e, f; **2** – c, f; **3** – a, b, f; **4** – a, c, d, e, f; **5** – a; **6** – a; **7** – b; **8** – a, c, e; **9** – c; **10** – b, f.

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