Peritonitis

Peritonitis is an **inflammation of the peritoneum**.

Types

1. **chemical** – through the action of chemical substances (bile, urine, pancreatic juice, gastric content, blood). If these fluids are sterile, it can also be referred to as *aseptic peritonitis* ;
2. **microbial** – through the action of microorganisms (bacteria), both aerobic (*Escherichia coli*, genus *Proteus*, *Klebsiella*, streptococci) and anaerobic (*Bacteroides fragilis*, anaerobic cocci, Clostridia);

1. **primary** – infection without disruption of the abdominal wall and intra-abdominal organs ((i.e. peritonitis from displacement of organs and hematogenous spread);
2. **secondary** – the infection is due to perforation of an organ or because of a penetrating injury to the abdominal wall;
3. **terciary** – infection due to surgery;

1. **diffuse** (peritonitis diffusa) – the inflammation is spread throughout the peritoneum;
2. **localised** (peritonitis circumscripta) – the inflammation is limited to a part of the peritoneum by fibrous adhesions.

Causes

1. Primary peritonitis:

a. *peritonitis due to organ displacement* (from a pathologically modified organ without perforation of its wall):

- ulcers penetrating deep (but not completely) into the wall;
- phlegmon of the intestine or stomach;
- hemorrhagic intestinal infarction;
- in purulent pleuritis and Pericarditis through the diaphragm (so-called **induced peritonitis**);

b. *. metastatically by blood*:

- in generalised infectious diseases (influenza, scarlet fever, measles);
- tonsilitis.

2. Secondary peritonitis:

a. *Injuries penetrating the abdominal wall*:

- stabbing, cutting, gunshot wound;
- postoperative infections (peritonitis post laparotomiam);

b. *perforation of the wall of an intra-abdominal organ* (inflamed, ulcerative or traumatic):

- appendicitis;
- gastric ulcer or intestinal ulceration;
- salpingitis;
- diverticulitis;
- perforation of the small or large intestine;
- cholecystitis;
- pancreatitis.

3. Tertiary peritonitis:

a. peritonitis that has occured after adequate treatement of a primary or secondary peritonitis during a persistent or reccurent intra-abdominal infection;
b. peritonitis that has developed in connection with surgery.

According to the pathological-anatomical picture, we divide peritonitis into **exudative**, **productive** and **tuberculous**.

Acute exudative peritonitis

- It is a superficial exudative inflammation during which exudation occurs to the surface of the peritoneum and into the peritoneal cavity, where it accumulates mainly in recesses. The peritoneum itself is red (hyperemia), often with small ecchymoses.
- The exsudate can be:

1. **serous** (p. serosa) – a small amount of exsudate can occur during the developement of acute Acute appendicitis. If mixed with blood, serous-hemorrhagic peritonitis can occur, usually with hemorrhagic intestinal infarction, peritoneal tuberculosis or concomitant carcinomatosis of the peritoneum;
2. **fibrinous** (p. fibrinosa) – the surface of the peritoneum loses its luster, the intestinal loops are glued together, the fibrin rapidly liquefies through the action of leukocytes and the inflammation becomes purulent in nature - peritonitis fibrinoso-purulenta;
3. **purulent** (p. purulenta) – the most common form of exudative peritonitis, either due to fibrinous inflammation or can be purulent from the beginning;
4. **putrefactive** (p. putrida) – during simultaneous anaerobic infection of the intestine (during putrefactive processes in the intestinal wall).

- **Sterkoral peritonitis** – during **intestinal perfration**, the exudate mixes with the intestinal contents and gases.
- **Biliary peritonitis** – when the **gallblader** or bile duct is perforated, e.g. by a concretion, bile is mixed into the exudate. This can also happen during organ displacement. It can present itself as:
    - aseptic serous-fibrinous peritonitis - in the case of sterile bile;
    - purulent peritonitis - in the case of infected bile.
- **Urinous peritonitis** due to **perforation** or rupture of the **bladder**, more rarely due to rupture of the pelvis or ureter (urine is absorbed from the peritoneal cavity into the blood and **uremia** occurs).
- **Meconium peritonitis** – during perforation of the intestine in **newborns** (e.g. in cystic fibrosis, where the thickened intestinal content causes their obstruction leading to ileus and subsequent perforation).

- When **pancreatic enzymes** are released, a characteristic whitish necrosis of adipose tissue occurs – Balser necrosis.
- An **autopsy finding** of peritonitis is characterized by the presence of a large amount of watery pus in the peritoneal cavity - it covers the intestinal loops and fills the peritoneal recesses. If it is caused by **intestinal perforation**, the abdomen is inflated with gas content.

Productive peritonitis

- Generally occurs during the **reparative phase of exudative inflammation** due to the organisation of the exudate – the fibrous organization of fibrin creates numerous adhesions (flat or striped) in the peritoneal cavity – between the intestinal loops or with the omentum and parietal peritoneum (common in hernias - hernia accreta). Peristaltic movements can change these adhesions into vascularized fibrous bands that can cause strangulation. In the case of circumcriptive peritonitis, adhesions can encapsulate the abscess that can then become a reservoir of infection and can later collapse and lead to diffuse peritonitis.
- **Primary productive peritonitis** is characterized by an increase in collagenous, often hyalinized connective tissue, macroscopically manifested by a thickening of the peritoneum, which has lost its transparency and whitish color. The surface is smooth and shiny. Primary productive peritonitis is common in larger hernias or polyserositis (e.g. systemic lupus erythematosus, where these changes affect the pericardium, pleura and peritoneum – most often the liver and spleen).

Tuberculous peritonitis

- It occurs either during **hematogenous spread** of infection (miliary tuberculosis) or spreading through infected organs (tuberculosis of the intestine, fallopian tube, nodes).
- Macroscopically, it corresponds to a miliary spread of nodules that is accompanied by serous-hemorrhagic exudate or has the character of a productive inflammation with the formation of ligaments and adhesions.

Pathogenesis

- The defense mechanisms of the peritoneum are mainly the presence of complement in the fluid and fluid circulation (it flows into the lymphatic nodes under the diaphragm). In the presence of a large number of bacteria, these mechanisms are not sufficient – peritonitis develops: mesothelial cells damaged by inflammation release histamin and other vasoactive substances that increase the permeability of capillaries – this leads to the creation of exudative inflammation. The exudate contains a lot of fibrinogen (peritonitis begins as a fibrinous inflammation, later associated with neutrophil infiltration and the inflammation then turns into purulent inflammation) which coagulates into fibrin and delimits the inflammatory deposit. Other components of the exudate are complement and other opsonins facilitating phagocytosis. The omentum is also an important defense mechanism, since it can seal the perforation of the organ or limit the infection and improve the circulation of ischemized organs with its collaterals.
- Bacteria present in the peritoneal cavity release toxins that are resorbed by the peritonal surface (because inflammation increases capillary permeability). This can lead to general intoxication and shock from vascular paralysis – **the cause of death is not sepsis** (bacteremia usually does not occur), **but circulatory failure**. A regular complication of diffuse bacterial peritonitis is **paralytic ileus** (due to the action of toxins directly on the intestinal muscle and on the autonomic nerve plexuses).

Links

Related articles

- Acute appendicitis
- Diffuse peritonitis
- Pankreatitis
- Spontaneous bacterial peritonitis

External links

- Peritonitida (česká wikipedie)
- Peritonitis (anglická wikipedie)

Sources

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