Acute peritonitis

The term peritonitis refers to a constellation of signs and symptoms, which includes abdominal pain and tenderness on palpation, abdominal wall muscle rigidity, and systemic signs of inflammation.

Classifications

The first classification of peritonitis was suggested by Miculich (1886):

1. septic;
2. purulent;
3. progressive fibrino-purulent;
4. local.

In 1912 Grekov defined the phases of peritonitis:

1. early (1–2 days);
2. late (3–5 days);
3. final (6–21 days).

Modern classification of acute peritonitis
(Shalimov A. A., 1981)

According to origin

1. Primary. It occurs in the absence of an apparent intra-abdominal source of infection and is observed almost exclusively in patients with ascites formation.
2. Secondary.

According to aetiology

1. Microflora of the digestive tract (E. coli, Enterococcus, Pseudomonas, Proteus, Streptococcus, Staphylococcus, Anaerobic infection).
2. Microflora non-connected with the digestive tract (tuberculous infection, gonococcal infection).
3. Aseptic (pancreatogenic, bilious).
4. Carcinomatous.
5. Parasitogenic.

According to cause

1. Traumatic.
2. Postoperative.
3. Inflammatory.
4. Perforated.

According to character of exudate

1. Serous.
2. Sero-fibrinous.
3. Fibrinous.
4. Fibrino-purulent.
5. Purulent.

**According to spreading of infection**
1. Local (inflammatory process is localized only in 1 anatomy region).
2. Diffuse (inflammatory process is localized in 2–5 anatomy regions).
3. Total (inflammatory process is localized in 6–9 anatomy regions).

**Phases of peritonitis**
1. Reactive (< 24 hours).
2. Toxic (24–72 hours).
3. Terminal (>72 hours).

**Pathophysiology**

The leading parts are:

a) pathogenic microflora;

b) intoxication;

c) hypovolemia;

d) deep metabolic disturbances.

**The first phase – reactive phase**

1. From the moment the aggressive factor has gotten into abdominal cavity, the signs of inflammation (oedema, hyperemia, and exudation) start.
   
a) 10 minutes – appearance of exudate into abdominal cavity.
   
b) 2 hours – formation of adhesions between peritoneum, bowels and large omentum starts.
   
c) 18 hours – formation of friable mass has been completed.
2. Activation of function of hypothalamo-pituitary, -adrenal axis.
3. Activation of biologically active substances: kinins, biogenic amines.

They control blood circulation and intestinal motor function.

4. Activation of immune response.
5. Onset of microcirculation disturbances.

**The second phase – toxic phase**

1. The leading factor at this stage is intoxication. Exactly intoxication usually determines the result of treatment. The constituents of intoxication are:
   – peritoneal exudate which is absorbed;
   – microflora (exo- and endotoxins);
   – proteolytic enzymes, which are released after destruction of leucocytes (cathepsins);
   – toxic substances, which are produced and absorbed into paretic bowels (phenols, skatoles).

2. Adynamic ileus. Distended bowel wall loses barrier function and translocation of microorganisms starts. Free liquid is collected into intestinal lumen – “sequestration of liquid into the third space” (transudate, digestive juice).
3. The organism can’t limit the infection and inflammatory process spreads into all abdominal cavity.

4. Immune response is depressed.

**The third phase – terminal phase**

This is a stage of septic shock and multiple organ failure. The changes are irreversible. Intoxication, disturbances of metabolism and haemodynamic are fatal.

Clinical manifestations of acute peritonitis depend on:

- disease which causes the peritonitis;
- stage of peritonitis;
- spreading of peritonitis;
- characteristics of microflora;
- activity of immune response

All patients present with some degree of abdominal pain. Anorexia and nausea are frequently present. Vomiting may occur because of the underlying visceral organ pathology or secondary to the peritoneal irritation.

**Treatment** of peritonitis and intra-abdominal sepsis always begins with volume resuscitation, correction of potential electrolyte and coagulation abnormalities, and empiric broad-spectrum parenteral antibiotic.

**Surgical treatment**

Surgery remains an important therapeutic modality for all cases of peritoneal infection. Any operation should address the **first 2 principles** of the treatment of intra-peritoneal infections:

1. early and definitive source control
2. elimination of bacteria and toxins from the abdominal cavity.

**Gastro-intestinal decompression.** It is indicated in patients with severe peritonitis and ileus. It is achieved by transnasal, transstomal or transanal small or/and large bowel tube. The aims of procedure are:

- removing of toxic bowel content;
- stabilization of bowel motor function;
– enteral nutritional supporting;
– prevention of postoperative adhesion bowel obstruction.