THE PERITONEUM, OMENTUM, MESENTERY AND RETROPERITONEAL SPACE

THE PERITONEUM

Functions of the peritoneum

- Pain perception (parietal peritoneum)
- Visceral lubrication
- Fluid and particulate absorption
- Inflammatory and immune responses
- Fibrinolytic activity

The parietal portion is richly supplied with nerves and, when irritated, causes severe pain accurately localised to the affected area. The visceral peritoneum, in contrast, is poorly supplied with nerves and its irritation causes vague pain that is usually located to the midline.

During expiration, intra-abdominal pressure is reduced and peritoneal fluid, aided by capillary attraction, travels in an upward direction towards the diaphragm. This upward movement of peritoneal fluids is responsible for the occurrence of many subphrenic abscesses.

When a visceral perforation occurs, the free fluid that spills into the peritoneal cavity runs downwards, largely directed by the normal peritoneal attachments. For example, spillage from a perforated duodenal ulcer may run down the right paracolic gutter.

Causes of a peritoneal inflammatory exudate

- Bacterial infection, e.g. appendicitis, tuberculosis
- Chemical injury, e.g. bile peritonitis
- Ischaemic injury, e.g. strangulated bowel
- Direct trauma, e.g. operation
- Allergic reaction, e.g. starch peritonitis

ACUTE PERITONITIS

Most cases of peritonitis are caused by an invasion of the peritoneal cavity by bacteria, so that when the term ‘peritonitis’ is used without qualification, bacterial peritonitis is implied. Bacterial peritonitis is usually polymicrobial, both aerobic and anaerobic organisms being present. The exception is primary peritonitis (‘spontaneous’ peritonitis), in which a pure infection with streptococcal, pneumococcal or Haemophilus bacteria occurs.

Bacteriology
<table>
<thead>
<tr>
<th>Bacteria in peritonitis source</th>
<th>Gastrointestinal Other sources</th>
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<tbody>
<tr>
<td>■ <em>Escherichia coli</em></td>
<td>■ <em>Chlamydia</em></td>
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<tr>
<td>■ Streptococci (aerobic and anaerobic)</td>
<td>■ Gonococcus</td>
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<tr>
<td>■ <em>Bacteroides</em> streptococci</td>
<td>■ b-Haemolytic</td>
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<td>■ <em>Clostridium</em></td>
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<td>■ <em>Klebsiella pneumonia</em></td>
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<td>Pneumococcus</td>
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<tr>
<td>■ <em>Staphylococcus</em></td>
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<tr>
<td><em>Mycobacterium tuberculosis</em></td>
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*Bacteria from the gastrointestinal tract*

The number of bacteria within the lumen of the gastrointestinal tract is normally low until the distal small bowel is reached, whereas high concentrations are found in the colon. However, disease (e.g. obstruction, achlorhydria, diverticula) may increase proximal colonisation. The biliary and pancreatic tracts are normally free from bacteria, although they may be infected in disease, e.g. gallstones. Peritoneal infection is usually caused by two or more bacterial strains. *Bacteroides* are commonly found in peritonitis. These Gram negative, non-sporing organisms, although predominant in the lower intestine, often escape detection because they are strictly anaerobic and slow to grow on culture media unless there is an adequate carbon dioxide tension in the anaerobic apparatus. These organisms are resistant to penicillin and streptomycin but sensitive to metronidazole, clindamycin, lincomycin and cephalosporin compounds.

*Non-gastrointestinal causes of peritonitis*

Pelvic infection via the fallopian tubes is responsible for a high proportion of ‘non-gastrointestinal’ infections. Immunodeficient patients, for example those with human immunodeficiency virus (HIV) infection or those on immunosuppressive treatment, may present with opportunistic peritoneal infection, e.g. *Mycobacterium avium-intracellulare* (MAI)
**Route of infection**
Infecting organisms may reach the peritoneal cavity via a number of routes (Even in patients with non-bacterial peritonitis (e.g. acute pancreatitis, intraperitoneal rupture of the bladder or haemoperitoneum), the peritoneum often becomes infected by transmural spread of organisms from the bowel, and it is not long (often a matter of hours) before a bacterial peritonitis develops. Most duodenal perforations are initially sterile for up to several hours, and many gastric perforations are also sterile at first; intestinal perforations are usually infected from the beginning. Mortality reflects:
- the degree and duration of peritoneal contamination;
- the age of the patient;
- the general health of the patient;
- the nature of the underlying cause.

**Localised peritonitis**
Anatomical, pathological and surgical factors may favour the localisation of peritonitis.

**Paths to peritoneal infection**
- Gastrointestinal perforation, e.g. perforated ulcer, diverticular perforation
- Exogenous contamination, e.g. drains, open surgery, trauma
- Transmural bacterial translocation (no perforation), e.g. inflammatory bowel disease, appendicitis, ischaemic bowel
- Female genital tract infection, e.g. pelvic inflammatory disease
- Haematogenous spread (rare), e.g. septicaemia

**Anatomical**
The greater sac of the peritoneum is divided into (1) the subphrenic spaces, (2) the pelvis and (3) the peritoneal cavity proper. The last is divided into a supracolic and an infracolic compartment by the transverse colon and transverse mesocolon, which deters the spread of infection from one to the other. When the supracolic compartment overflows, as is often the case when a peptic ulcer perforates, it does so over the colon into the infracolic compartment or by way of the right paracolic gutter to the right iliac fossa and hence to the pelvis.

**Pathological**
The clinical course is determined in part by the manner in which adhesions form around the affected organ. Inflamed peritoneum loses its glistening appearance and becomes reddened and velvety. Flakes of fibrin appear and cause loops of intestine to become adherent to one another and to the parietes. There is an outpouring of serous inflammatory exudate rich in leucocytes and plasma proteins that soon becomes turbid; if localisation occurs, the turbid fluid becomes frank pus. Peristalsis is retarded in affected bowel and this helps to prevent distribution of the infection.

The greater omentum, by enveloping and becoming adherent to inflamed structures, often forms a substantial barrier to the spread of infection.

**Surgical**

Drains are frequently placed during operation to assist localization (and exit) of intra-abdominal collections: their value is disputed. They may act as conduits for exogenous infection.

**Diffuse peritonitis**

A number of factors may favour the development of diffuse peritonitis:

1. **Speed of peritoneal contamination** is a prime factor. If an inflamed appendix or other hollow viscus perforates before localisation has taken place, there is a gush of contents into the peritoneal cavity, which may spread over a large area almost instantaneously. Perforation proximal to an obstruction or from sudden anastomotic separation is associated with severe generalised peritonitis and a high mortality rate.

2. **Stimulation of peristalsis** by the ingestion of food or even water hinders localisation. Violent peristalsis occasioned by the administration of a purgative or an enema may cause the widespread distribution of an infection that would otherwise have remained localised.

3. The **virulence of the infecting organism** may be so great as to render the localisation of infection difficult or impossible.

4. **Young children** have a small omentum, which is less effective in localising infection.

5. **Disruption of localised collections** may occur with injudicious handling, e.g. appendix mass or pericolic abscess.

6. **Deficient natural resistance** (‘immune deficiency’) may result from use of drugs (e.g. steroids), disease [e.g. acquired immune deficiency syndrome (AIDS)] or old age.

**Clinical features**
Localised peritonitis
Localised peritonitis is bound up intimately with the causative condition, and the initial symptoms and signs are those of that condition. When the peritoneum becomes inflamed, the temperature, and especially the pulse rate, rise. Abdominal pain increases and usually there is associated vomiting. The most important sign is guarding and rigidity of the abdominal wall over the area of the abdomen that is involved, with a positive ‘release’ sign (rebound tenderness). If inflammation arises under the diaphragm, shoulder tip (‘phrenic’) pain may be felt. In cases of pelvic peritonitis arising from an inflamed appendix in the pelvic position or from salpingitis, the abdominal signs are often slight; there may be deep tenderness of one or both lower quadrants alone, but a rectal or vaginal examination reveals marked tenderness of the pelvic peritoneum. With appropriate treatment, localised peritonitis usually resolves; in about 20% of cases, an abscess follows. Infrequently, localised peritonitis becomes diffuse. Conversely, in favourable circumstances, diffuse peritonitis can become localised, most frequently in the pelvis or at multiple sites within the abdominal cavity.

Diffuse (generalised) peritonitis
Diffuse (generalised) peritonitis may present in differing ways dependent on the duration of infection.

Early
Abdominal pain is severe and made worse by moving or breathing. It is first experienced at the site of the original lesion and spreads outwards from this point. Vomiting may occur. The patient usually lies still. Tenderness and rigidity on palpation are found typically when the peritonitis affects the anterior abdominal wall. Abdominal tenderness and rigidity are diminished or absent if the anterior wall is unaffected, as in pelvic peritonitis or, rarely, peritonitis in the lesser sac. Patients with pelvic peritonitis may complain of urinary symptoms; they are tender on rectal or vaginal examination. Infrequent bowel sounds may still be heard for a few hours but they cease with the onset of paralytic ileus. The pulse rises progressively but, if the peritoneum is deluged with irritant fluid, there is a sudden rise. The temperature changes are variable and can be subnormal.

Late
If resolution or localisation of generalised peritonitis does not occur, the abdomen remains silent and increasingly distends. Circulatory failure ensues, with cold, clammy extremities, sunken eyes, dry tongue, thready (irregular) pulse and drawn and anxious face.
(Hippocratic facies; The patient finally lapses into unconsciousness.

**Diagnostic aids**
Investigations may elucidate a doubtful diagnosis, but the importance of a careful history and repeated examination must not be forgotten.

- A *radiograph of the abdomen* may confirm the presence of dilated gas-filled loops of bowel (consistent with a paralytic ileus) or show free gas, although the latter is best shown on an erect chest radiograph. If the patient is too ill for an ‘erect’ film to demonstrate free air under the diaphragm, a lateral decubitus film is just as useful, showing gas beneath the abdominal wall.

- *Serum amylase estimation* may establish the diagnosis of acute pancreatitis provided that it is remembered that moderately raised values are frequently found following other abdominal catastrophes and operations, e.g. perforated duodenal ulcer.

- *Ultrasound* and *computerised tomography (CT) scanning* are increasingly used to identify the cause of peritonitis. Such knowledge may influence management decisions.

- *Peritoneal diagnostic aspiration* may be helpful but is usually unnecessary. Bile-stained fluid indicates a perforated peptic ulcer or gall bladder; the presence of pus indicates bacterial peritonitis. Blood is aspirated in a high proportion of patients with intraperitoneal bleeding.

**Clinical features in peritonitis**
- Abdominal pain, worse on movement
- Guarding/rigidity of abdominal wall
- Pain/tenderness on rectal/vaginal examination (pelvic peritonitis)
- Pyrexia (may be absent)
- Raised pulse rate
- Absent or reduced bowel sounds
- ‘Septic shock’ [systemic inflammatory response syndrome (SIRS)] in later stages

**Treatment**
In case of doubt, early surgical intervention is to be preferred to a ‘wait and see’ policy. This rule is particularly true for previously healthy patients and those with postoperative peritonitis. Caution is required in patients at high operative risk because of comorbidity or advanced age.
Treatment consists of:  
• general care of the patient;  
• specific treatment of the cause;  
• peritoneal lavage when appropriate.