The peritoneum

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The peritoneum

The peritoneal membrane is conveniently divided into two parts — the visceral surrounding the viscera (Serosa) and the parietal lining the other surfaces of the cavity. The peritoneum has a number of functions

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 which is usually located to the midline. The peritoneal cavity is the largest cavity in the body, the surface area of its lining membrane (2 m\(^2\) in an adult) being nearly equal to that of the skin. The peritoneal membrane is composed of flattened polyhedral cells (mesothelium), one layer thick, resting upon a thin layer of fibroelastic tissue.
During expiration intra-abdominal pressure is reduced and peritoneal fluid, aided by capillary attraction, travels in an upward direction towards the diaphragm. Experimental evidence shows that particulate matter and bacteria are absorbed within a few minutes into the lymphatic network through a number of ‘pores’ within the diaphragmatic peritoneum. This upward movement of peritoneal fluids is responsible for the occurrence of many subphrenic abscesses.
The peritoneum has the capacity to absorb large volumes of fluid: this ability is used during peritoneal dialysis in the treatment of renal failure. But the peritoneum can also produce an inflammatory exudate when injured. When a visceral perforation occurs the free fluid which 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.
Acute peritonitis

Most cases of peritonitis are due to an invasion of the peritoneal cavity by bacteria. 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

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, while high concentrations are found in the colon. 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. The commonest are Escherichia coli, aerobic and anaerobic streptococci, and the bacteroides. Less frequently Clostridium welchii is found; still less frequently staphylococci or Klebsiella.
Gram-negative bacteria contain endotoxins (lipopolysaccharides) in their cell walls which have multiple toxic effects on the host, primarily by causing the release of tumour necrosis factor (TNF).

Other bacteria such as C. welchii produce harmful exotoxins. Bacteroides are commonly found in peritonitis. These Gram-negative, nonsporing organisms, although predominant in the lower intestine. These organisms are resistant to penicillin and streptomycin but sensitive to metronidazole, clindamycin, lincomycin and cephalosporin compounds. Since the widespread use of metronidazole (‘Flagyl’) bacteroides infections have diminished greatly.
Nongastrointestinal causes of peritonitis

Nongastrointestinal causes of peritonitis include chlamydia, gonococcus, beta-haemolytic streptococcus, pneumococcus and Mycobacterium tuberculosis.

In young girls and women, pelvic infection via the Fallopian tubes is responsible for a high proportion of ‘nongastrointestinal’ infections but bacteroides is also found in the female genital tract.
Bacteria in peritonitis
Gastrointestinal source
- *Escherichia coli*
- Streptococci (aerobic and anaerobic)
- *Bacteroides*
- *Clostridium*
- *Klebsiella pneumoniae*
- *Staphylococcus*
Other sources
- *Chlamydia*
- Gonococcus
- b-Haemolytic streptococci
- Pneumococcus
- *Mycobacterium tuberculosis*
Route of infection
Infecting organisms may reach the peritoneal cavity via a number of routes
■ 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
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

The greater sac of the peritoneum is divided into (a) the subphrenic spaces, (b) the pelvis, and (c) the peritoneal cavity proper. The latter is redivided into a supracolic and an infracolic compartment by the transverse colon and transverse mesocolon.
Pathological
1) Adhesions form around the affected organ.
2) Inflamed peritoneum loses its glistening appearance and becomes reddened and velvety.
3) Flakes of fibrin appear and cause loops of intestine to become adherent to one another and to the parieties.
4) There is an outpouring of serous inflammatory exudate rich in leucocytes and plasma proteins that soon becomes turbid;
5) If localisation occurs, the turbid fluid becomes frank pus.
6) Peristalsis is retarded in affected bowel, and this helps in preventing distribution of the infection.
7) The greater omentum, by enveloping and becoming adherent to inflamed structures, often forms a substantial barrier to the spread of infection.
(a) Gas under the diaphragm in a patient with free perforation and peritonitis; (b) representative computed tomography axial image through the pelvis of the same patient showing perforated sigmoid diverticular disease.
Acute pancreatitis seen on computed tomography scanning with swelling of the gland and surrounding inflammatory changes (courtesy of Dr J Healy, Chelsea and Westminster Hospital, London, UK).
Surgical Drains are frequently placed during operation to assist localisation (and exit) of intra-abdominal collections: their value is controversial. They may act as conduits for exogenous infection. Collections detected postoperatively on ultrasound or computerised tomography (CT) scanning may be drained percutaneously.
Diffuse peritonitis

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

1) Speed of peritoneal contamination is a prime factor in the spread of peritonitis.
2) Stimulation of peristalsis by the ingestion of food, or even water, hinders localisation.
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.
5) Disruption of localised collections may occur with injudicious and rough handling, e.g. appendix mass or pericolic abscess.
6) Deficient natural resistance (‘immune deficiency’) may result from drugs (e.g. steroids), disease (e.g. AIDS) or old age.
Clinical features

Localised peritonitis

- Peritoneum becomes inflamed; the temperature, and especially the pulse rate, rise.
- Abdominal pain increases, and usually there is associated vomiting.
- Guarding and rigidity of the abdominal wall over the area of the abdomen which 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, deep tenderness of one or both lower quadrants alone being present, but a rectal or vaginal examination reveals marked tenderness of the pelvic peritoneum.
Diffuse (generalised) peritonitis  Early

- Abdominal pain is severe and made worse by moving or breathing.
- Vomiting may occur.
- Tenderness and rigidity on palpation are typically found when the peritonitis affects the anterior abdominal wall. 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,
- 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.
Clinical features of peritonitis

• Abdominal pain, worse on movement, coughing and deep respiration
• Constitutional upset: anorexia, malaise, fever, lassitude
• GI upset: nausea ± vomiting
• Pyrexia (may be absent)
• Raised pulse rate
• Tenderness ± guarding/rigidity/rebound of abdominal wall
• Pain/tenderness on rectal/vaginal examination (pelvic peritonitis)
• Absent or reduced bowel sounds
• ‘Septic shock’ (systemic inflammatory response syndrome (SIRS) and multiorgan dysfunction syndrome (MODS)) in later stages
Diagnostic aids

1) History and repeated examination (very important)
2) A leucocytosis is usually seen in peritonitis but is often delayed for many hours.
3) Peritoneal diagnostic aspiration may be helpful
4) An X-ray film 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 X-ray. If the patient is too ill for an ‘erect’ film to demonstrate free air collecting under the diaphragm, a lateral decubitus film is just as useful showing gas beneath the abdominal wall.
5) Serum amylase estimation may uphold the diagnosis of acute pancreatitis provided it is remembered that moderately raised values are frequently found following other abdominal catastrophes and operations, e.g. perforated duodenal ulcer.
6) Ultrasound and CT scanning.
Management of peritonitis

**General care of patient**
- Correction of fluid and electrolyte imbalance
- Insertion of nasogastric drainage tube and urinary catheter
- Broad-spectrum antibiotic therapy
- Analgesia
- Vital system support

**Operative treatment of cause when appropriate**
- Remove or divert cause
- Peritoneal lavage ± drainage
Specific treatment of the cause

If the cause of peritonitis is amenable to surgery, such as in perforated appendicitis, diverticulitis, peptic ulcer, gangrenous cholecystitis or in rare cases of perforation of the small bowel, operation must be carried out as soon as the patient is fit for anaesthesia. This is usually within a few hours.

In peritonitis due to pancreatitis or salpingitis, or in cases of primary peritonitis of streptococcal or pneumococcal origin, nonoperative treatment is preferred (if the diagnosis can be made with certainty).
**Peritoneal lavage**

In operations for general peritonitis it is essential that after the cause has been dealt with the whole peritoneal cavity should be explored with the sucker and mopped dry, if necessary until all seropurulent exudate is removed. The use of a large volume of saline (1—2 litres) containing dissolved antibiotic (e.g. tetracycline) has been shown to be very effective (Matheson).
Prognosis
With modern treatment diffuse peritonitis carries a mortality of about 10 per cent. The systemic complications and lethal factors are listed below.
Complications of peritonitis

Systemic complications
Bacteraemic/endotoxic shock
Systemic inflammatory response syndrome
Multiorgan dysfunction syndrome
Death

Abdominal complications
Paralytic ileus
Residual or recurrent abscess/inflammatory mass
Portal pyaemia/liver abscess
Adhesional small bowel obstruction
Common situations for residual abscesses: (1) subphrenic; (2) paracolic; (3) right iliac fossa; (4) pelvic.
Pelvic abscess

• The pelvis is the commonest site of an intraperitoneal abscess because the vermiform appendix is often pelvic in position and also the Fallopian tubes are frequent sites of infection.
• A pelvic abscess can also occur as a sequel to any case of diffuse peritonitis and is a common sequel of anastomotic leakage following large bowel and rectal surgery.
• The most characteristic symptoms of a pelvic abscess are diarrhoea and the passage of mucus in the stools.
• The passage of mucus, occurring for the first time in a patient who has, or is recovering from, peritonitis, is pathognomonic of pelvic abscess.
• Rectal examination reveals a bulging of the anterior rectal wall.
Opening a pelvic abscess into the rectum.
Subphrenic abscess
Anatomy
The complicated arrangement of the peritoneum results in the formation of four intraperitoneal and three extraperitoneal spaces in which pus may collect. Three of these spaces are on either side of the body, and one is approximately in the midline.
**Figure 58.8** Intraperitoneal abscesses on transverse section. (1) The left subphrenic space; (2) left subhepatic space/lesser sac; (3) right subphrenic space; (4) right subhepatic space.

**Figure 58.9** Intraperitoneal abscesses on sagittal section. (1) Left subphrenic; (2) left subhepatic/lesser sac; (3) right subphrenic; (4) right subhepatic.
Intraperitoneal abscesses

Left superior (anterior) intraperitoneal (‘left subphrenic’) The common cause of an abscess here is an operation on the stomach, the tail of the pancreas, the spleen or the splenic flexure of the colon.

Left inferior (posterior) intraperitoneal (‘left subhepatic’) is another name for the ‘lesser’ sac. The commonest cause of infection here is complicated acute pancreatitis. In practice a perforated gastric ulcer rarely causes a collection here because the potential space is obliterated by adhesions.
Right superior (anterior) intraperitoneal (‘right subphrenic’) Common causes here are perforating cholecystitis, a perforated duodenal ulcer, a duodenal cap ‘blow out’ following gastrectomy and appendicitis

Right inferior (posterior) intraperitoneal (‘right subhepatic’) lies transversely beneath the right lobe of the liver in Rutherford Morison’s pouch. It is the deepest space of the four and the commonest site of a subphrenic abscess which usually arises from appendicitis, cholecystitis, a perforated duodenal ulcer or following upper abdominal surgery.
Extraperitoneal abscesses

Right and left extra peritoneal which are terms given to perinephric abscesses; Midline extraperitoneal which is another name for the ‘bare’ area of the liver which may develop an abscess in amoebic hepatitis (the commonest cause) or a pyogenic liver abscess.
Clinical features

The symptoms and signs of subphrenic infection are frequently nonspecific, and it is well to remember the aphorism, ‘pus somewhere, pus nowhere else, pus under diaphragm’.

Summary of clinical features of an abdominal/pelvic abscess

- Malaise
- Sweats with or without rigors
- Abdominal/pelvic (with or without shoulder tip) pain
- Anorexia and weight loss
- Symptoms from local irritation, e.g. hiccoughs (subphrenic), diarrhoea and mucus (pelvic)
- Swinging pyrexia
- Localised abdominal tenderness/mass
A common history is that when some infective focus in the abdominal cavity has been dealt with, the condition of the patient improves temporarily, but after an interval of a few days or weeks, symptoms of toxaemia reappear. The condition of the patient steadily, and often rapidly, deteriorates. Sweating, wasting and anorexia are present. There is sometimes epigastric fullness and pain, or pain in the shoulder on the affected side, owing to irritation of sensory fibres in the phrenic nerve, referred along the descending branches of the cervical plexus. Persistent hiccups may be a presenting symptom.
A swinging pyrexia is usually present, unless antibiotics or drugs (steroids) have interfered. If the abscess is anterior, abdominal examination will reveal some tenderness, rigidity or even a palpable swelling. Sometimes the liver is displaced downwards, but more often it is fixed by adhesions. Examination of the chest is important, and in the majority of cases collapse of the lung or evidence of basal effusion or empyema is to be found.
**Investigations**

Anumber of these may be helpful as follows.

1) **Blood count usually shows a leucocytosis.**
2) **A plain radiograph sometimes demonstrates the presence of gas or a pleural effusion.** On screening, the diaphragm is often seen to be elevated (so-called ‘tented’ diaphragm) and its movements impaired.
3) **Ultrasound or CT scanning is the investigation of choice and permits early detection of subphrenic collections.**
4) **Radiolabelled white cell scanning may occasionally prove helpful when other imaging techniques have failed.**
Differential diagnosis. Pyelonephritis, amoebic abscess, pulmonary collapse and pleural empyema give rise to most of the diagnostic difficulties.
Treatment of abdominal abscess

- The clinical course of suspected cases is watched, and blood and imaging investigations are made at suitable intervals.
- If suppuration seems probable, intervention is indicated. If skilled help is available it is possible to insert a percutaneous drainage tube under ultrasound or CT control.
- The same tube can be used to instil antibiotic solutions or irrigate the abscess cavity.
- If an operative approach is necessary and a swelling can be detected in the subcostal region or in the loin, an incision is made over the site of maximum tenderness, or over any area where oedema or redness is discovered.
• If no swelling is apparent, the subphrenic spaces should be explored either by an anterior subcostal approach or from behind after removal of the outer part of the 12th rib according to the position of abscess on imaging.

• When the cavity is reached, all of the fibrinous loculi must be broken down with the finger and one or two drains or drainage tubes must be fully inserted.

• The appropriate antibiotics are also given
Acute nonspecific ileocaecal mesenteric adenitis

Aetiology

Nonspecific mesenteric adenitis was so named to distinguish it from specific (tuberculous) mesenteric adenitis. Aetiology often remains unknown although some cases are associated with Yersinia infection of the ileum. In other cases an unidentified virus is blamed. In about 25 per cent of cases a respiratory infection precedes an attack of nonspecific mesenteric adenitis. This self-limiting disease is never fatal but may be recurrent.
There is a small increase in the amount of peritoneal fluid. The ileocaecal mesenteric lymph nodes are enlarged and can be seen and felt between the leaves of the mesentery. There is often a leucocytosis of 10000—12000/mm³ (10—12 x 10⁹/litre) or more on the first day of the attack, but this falls on the second day.
Clinical features

During childhood, acute nonspecific mesenteric adenitis is a common condition. It is unusual after puberty but is sometimes seen in teenage girls. The typical history is one of short attacks of central abdominal pain lasting from 10 to 30 minutes, and associated with circumoral pallor. Vomiting is common but there is no alteration of bowel habit. If vomiting is absent, it is more likely to be a case of mesenteric adenitis than appendicitis.
On examination

• There are spasms of general abdominal colic, usually referred to the umbilicus, with intervals of complete freedom, which never occurs in obstructive appendicitis.
• The patient seldom looks ill.
• In more than half of the cases the temperature is elevated; in severe examples it exceeds 38.30°C.
• Abdominal tenderness is greatest along the line of the mesentery..
• The pelvic peritoneum is tender to rectal palpation in 30 percent of cases.
• The neck, axillae and groins should be palpated for enlarged lymph nodes—if these nodes are enlarged, brucellosis should come to mind.
Treatment

When the diagnosis can be made with assurance, bed rest for a few days is the only treatment necessary. If in doubt, it is safer to perform either appendicectomy or diagnostic laparoscopy.
Thank you