Peritonitis

Introduction

Peritonitis simply means inflammation of the peritoneum. Most surgical cases of peritonitis arise secondary to bacterial contamination of the peritoneal cavity by organisms derived from the gastrointestinal tract. This common form of acute peritonitis is described in detail in this article, while other less common forms are discussed briefly.

Peritonitis can be septic or aseptic, bacterial or viral, primary or secondary, acute or chronic. Most cases of peritonitis are the result of bacterial invasion of the peritoneal cavity from the gastrointestinal tract. Thus when the term 'peritonitis' is used, bacterial peritonitis is implied. Usually there is underlying pathology or injury to the gastrointestinal tract.

Determining the cause of peritonitis presents a diagnostic challenge to the surgeon, physician, GP and obstetrician-gynaecologist. If the underlying cause of peritonitis is amenable to operation, for example perforated peptic ulcer, acute appendicitis or gangrenous cholecystitis, the operation needs to be performed as soon as the patient is fit for anaesthesia.

In peritonitis secondary to pancreatitis or pelvic inflammatory disease, or in cases of primary peritonitis, non-operative treatment is preferred if the diagnosis can be made with certainty. The aetiological complexity of peritonitis requires a careful and methodical approach to arrive at the correct diagnosis. Management should be based upon a thorough knowledge of the natural history of the causative conditions as well as the anatomy and physiology of the peritoneal cavity.

The peritoneum

The peritoneal cavity

The peritoneal membrane consists of a single layer of mesothelial cells supported by a thin layer of connective tissue and is conveniently divided into two parts. The parietal peritoneum lines the abdominal cavity including the anterior abdominal wall, diaphragm and pelvis, while the visceral peritoneum covers the intraperitoneal viscera, creating a cavity that is enclosed except for the open ends of the uterine tubes.

The parietal peritoneum is innervated by both somatic and visceral nerves; when irritated it causes severe pain which is accurately localized to the affected area. The lining of the anterior and lateral abdominal walls is supplied by the lower six thoracic and first lumbar nerves, the central part of the diaphragmatic peritoneum is supplied by the phrenic nerves and the peripheral part by the lower six thoracic nerves. The parietal peritoneum in the pelvis is mainly supplied by the obturator nerve. By contrast, the visceral peritoneum is relatively insensitive and receives afferent innervation from the autonomic nervous system only; irritation of this structure (serosa) causes vague pain that is usually located to the midline. As well as pain perception (parietal), the peritoneum has a number of other functions including visceral lubrication, fluid and particulate absorption, inflammatory and immune responses, and fibrinolytic activity.

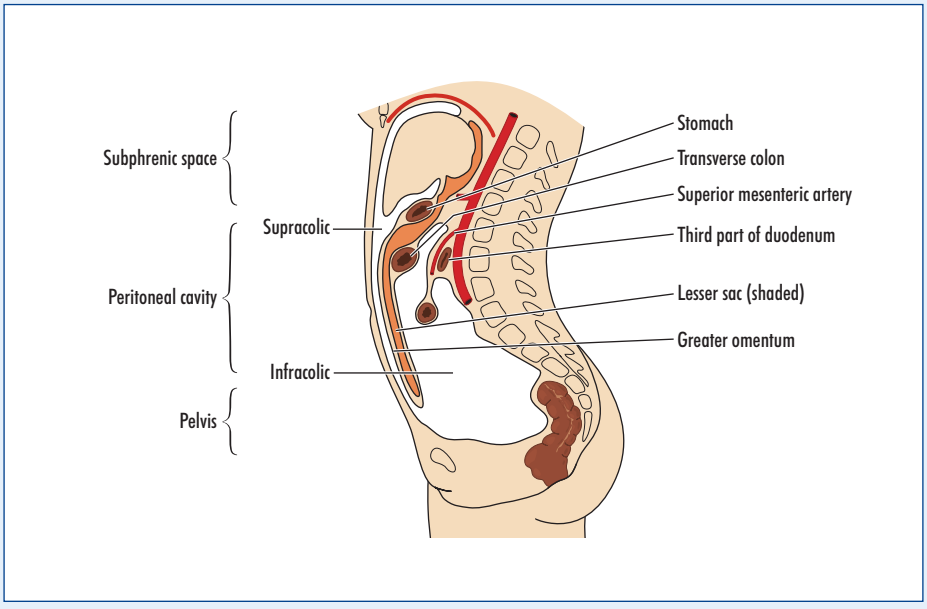
Response to peritoneal injury

The inflammatory response of the peritoneum may involve the entire intra-abdominal cavity or only a portion of either the parietal or visceral peritoneum. The peritoneal response to injury involves the production of an inflammatory exudate plus oedema and vascular congestion of the connective tissue layer immediately adjacent to the single layer of mesothelium. The serous inflammatory exudate is rich in leucocytes and plasma proteins, both of which have an important role in conjunction with fibrin in containing any intraperitoneal infection.

Localized peritonitis

Both anatomical and pathological factors play a role in the localization of peritonitis. Anatomically the greater sac of the peritoneum is divided into the subphrenic spaces, the peritoneal cavity proper – which is further divided into supracolic and infracolic compartments by the transverse mesocolon and colon – and the pelvis (*Figure 1*). Successful localization depends in part on the way in which adhesions form around the affected organ or source of sepsis. The production of a serous inflammatory exudate containing leucocytes, plasma proteins and fibrin causes loops of small bowel and the greater omentum to

Figure 1. Sagittal section of the abdomen, showing the division of the greater sac of the peritoneum into the subphrenic spaces, the peritoneal cavity proper and the pelvis.



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become adherent to one another and to the parietes, forming a substantial barrier to the spread of infection. In addition peristalsis is decreased in the affected bowel, thereby further preventing spread.

Generalized peritonitis

The most important factor in the spread of peritonitis is the speed of peritoneal contamination and the size of the inoculum. When a visceral perforation occurs before localization has taken place, free fluid spills into the peritoneal cavity and can spread over a large area almost instantaneously. Other factors favouring the development of generalized peritonitis include drug-related immunodeficiency (e.g. corticosteroids), disease (e.g. diabetes, acquired immunodeficiency syndrome; AIDS), old age (Watters et al, 1996), disruption of previously localized collections and the virulence of the infecting organism, which can be so great as make localization impossible.

Causes of acute peritonitis

Primary peritonitis

Primary or spontaneous bacterial peritonitis refers to a diffuse bacterial infection without an obvious intra-abdominal source. More than 90% of cases of spontaneous bacterial peritonitis are caused by monomicrobial infection. The condition occurs more commonly in children than adults and in women more than men. The female preponderance may be explained by the entry of organisms into the peritoneal cavity via the uterine tubes. In children there may be a history of an upper respiratory tract or middle ear infection. Children with nephrotic syndrome or, less commonly, systemic lupus erythematosus are particularly susceptible. The causative organisms in these cases are usually streptococcal, pneumococcal or *Haemophilus* bacteria.

In adults spontaneous bacterial peritonitis is observed almost exclusively in patients with ascites secondary to chronic liver disease (Fernández et al. 2006). Contamination of the peritoneal cavity is thought to result from translocation of bacteria across the gut wall or mesenteric lymphatics and, less frequently, via haematogenous spread in patients with bacteraemia. The most common pathogens include *Escherichia coli* (40%), *Streptococcus pneumoniae* (15%) and *Klebsiella pneumoniae* (7%). Because it is often difficult to differentiate between primary and secondary peritonitis, the diagnosis is frequently not made until laparotomy.

Secondary peritonitis

Secondary peritonitis implies bacterial contamination from a known source, usually from within the abdomen and often from a perforation of the gastrointestinal tract (*Table 1*). Small bowel and large bowel perforations are infected from the beginning. Even in patients who initially have non-bacterial peritonitis – i.e. 'chemical' peritonitis secondary to gastric, duodenal or pancreatic juice, bile, blood or urine – the peritoneum usually becomes infected by transmural spread of organisms from the bowel; thus bacterial peritonitis supervenes within a few hours.

Unlike primary peritonitis, bacterial peritonitis is usually polymicrobial with both anaerobic and aerobic organisms being present (*Figure 2*). The proportion of anaerobic to aerobic organisms increases with time. Special mention of peritonitis complicating continuous ambulatory peritoneal dialysis should be made. The two most important routes of infection are the Tenckhoff catheter exit site and the tunnel tract. Hence, cutaneous rather than enteric organisms account for the majority of infections; more than 40% of all positive cultures are coagulase-negative staphylococci. Less common portals of entry are via haematogenous spread in patients with bacteraemia and direct contamination from the gastrointestinal tract.

Clinical features

The signs, symptoms and physical findings of patients with peritonitis depend on the aetiology and duration of the disease and whether the process is localized or generalized. A thorough history will often suggest the source of the problem, which can then be confirmed on clinical examination. When evaluating a patient with suspected peritonitis it is essential that a complete physical examination is performed. Thoracic pathology with diaphragmatic irritation (e.g. empyema), extraperitoneal pathology (e.g. pyelone-

Table 1. Causes of acute peritonitis

Gastrointestinal tract	Perforation	Gastric or duodenal ulcer
		Sigmoid diverticula
		Caecum secondary to large bowel obstruction
		Gallbladder
		Appendix
		Meckel's diverticulum
		Iatrogenic (oesophagogastroduodenoscopy, endoscopic retrograde cholangiopancreatography, colonoscopy)
		Postoperative anastomotic dehiscence
	Transmural bacterial contamination	Ischaemic bowel
		Inflammatory bowel disease
		Appendicitis
Chemical peritonitis	Acute pancreatitis	
Vascular	Ruptured aneurysm	Aortoiliac
		Splenic
Trauma	Traumatic laceration of liver, spleen, pancreas, mesentery, bowel	
	Ruptured bladder	
Female genital tract	Pelvic inflammatory disease	
	Ruptured ovarian cyst	
	Ruptured ectopic pregnancy	
Haematogenous spread	Septicaemia	

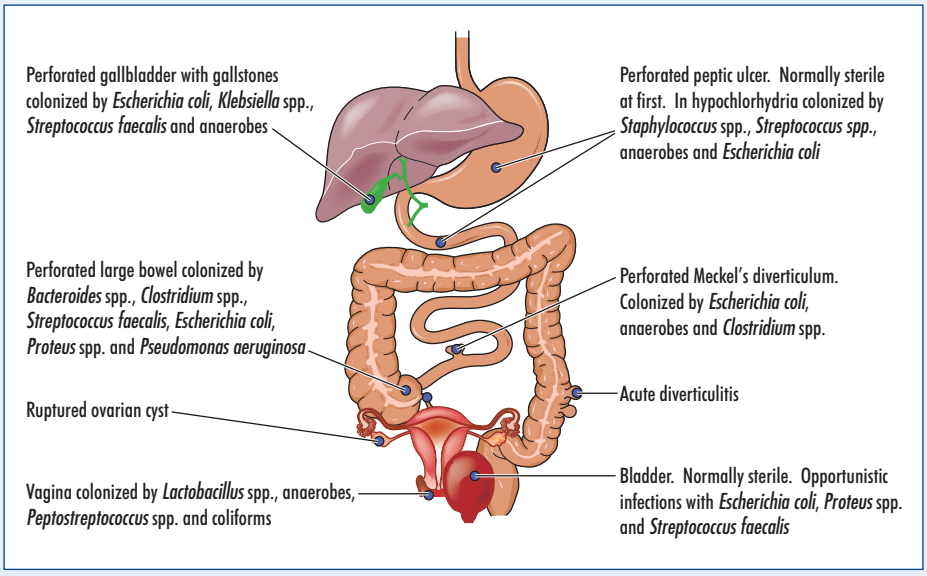


Figure 2. Common causes of secondary peritonitis illustrating the polymicrobial aspect of bacterial contamination from a known source.

phritis, urinary tract infection) and abdominal wall pathology (e.g. rectus sheath haematoma) can all mimic the signs and symptoms of peritonitis.

Localized peritonitis

Localized peritonitis is intimately associated with the causative condition. For example, pain is typically experienced in the right upper quadrant with acute cholecystitis, in the epigastrium with peptic ulcer disease and acute pancreatitis, the right iliac fossa with appendicitis (often an initial umbilical location), in the left iliac fossa with diverticulitis and suprapubically with pelvic inflammatory disease.

The most important sign is guarding and rigidity of the abdominal wall over the affected organ, which is typically associated with rebound (percussion) tenderness. If inflammation originates close to the diaphragm, shoulder tip (phrenic) pain may be felt. In patients with pelvic peritonitis secondary to pelvic inflammatory disease or an inflamed pelvic appendix, abdominal signs may be minimal while rectal or vaginal examination elicit marked tenderness. Localized peritonitis is usually associated with pyrexia, tachycardia, nausea and vomiting.

Generalized peritonitis

Abdominal pain is severe and is usually experienced suddenly at the site of the original lesion, rapidly spreading outwards from this point. Since pain is exacerbated by movement or breathing, the patient usually lies still. Tenderness and rigidity of the whole abdomen is noted on palpation, whilst absent or hypoactive bowel sounds are the result of paralytic ileus. Vomiting may occur. The pulse rate rises gradually, but temperature changes are variable, the patient occasionally being hypothermic. If treatment is not initiated, the abdomen increasingly distends and septic shock becomes manifested by hypotension, tachycardia, tachypnoea, pallor and restlessness. The Hippocratic facies denotes a patient in the terminal phases of peritonitis.

Investigations

A number of investigations can help confirm a doubtful diagnosis, but even if these tests are negative, treatment should be based on a combination of a careful history and clinical signs.

Blood tests

There is usually leucocytosis or a slower rise in C-reactive protein. A raised serum amylase may confirm the diagnosis of acute pancreatitis, but it should be remembered that lesser increases can be associated with other causes of peritonitis such as a perforated duodenal ulcer, ischaemic small bowel or a ruptured aortic aneurysm.

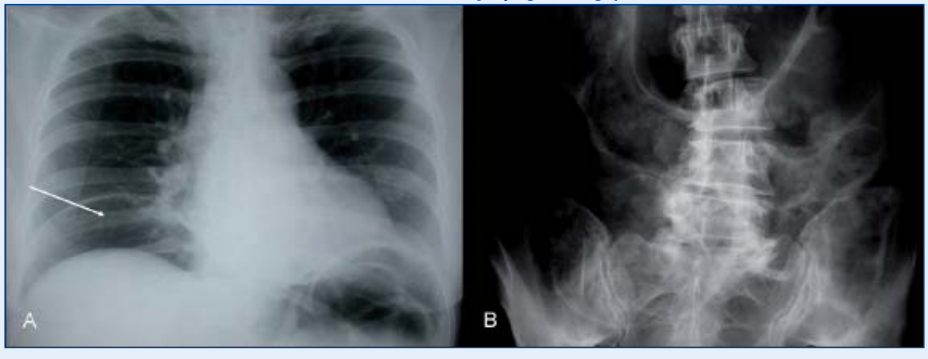
Radiology

An erect chest X-ray may demonstrate free peritoneal gas secondary to a perforated viscus, as can a plain abdominal X-ray; Rigler's sign denotes air is present both within the lumen and outside the wall of the intestine (*Figure 3*). If the patient is too unwell for an erect chest X-ray, a lateral decubitus film can show gas beneath the abdominal wall. Ultrasound and computed tomography (CT) scans can also be helpful in identifying the cause of peritonitis, e.g. ischaemic bowel or acute pancreatitis (*Figure 4*) (Bartlett, 1995). In a doubtful case of acute pancreatitis, clear-cut swelling of the pancreas helps to avoid an unnecessary laparotomy, while the presence of a right-sided ovarian cyst may help avoid an appendicectomy in a suspected case of appendicitis.

Treatment

If there is any doubt as to the diagnosis and the patient is unwell, early operation is indicated (after appropriate resuscitation). This principle is particularly important in patients who have previously been healthy or in those with postoperative peritonitis. More patients die from delay in intervention than from a negative laparotomy.

Figure 3. Plain radiograph findings of a perforated duodenal ulcer. a. Erect chest X-ray demonstrating free peritoneal gas under the right hemidiaphragm (arrow). b. Plain abdominal film in which pneumoperitoneum shows clear demarcation of the walls of small bowel loops (Rigler's sign).



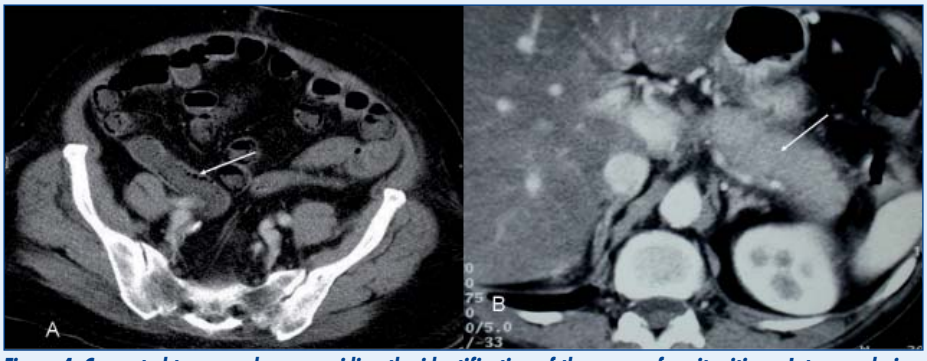


Figure 4. Computed tomography scans aiding the identification of the cause of peritonitis. a. Intramural air (arrow) in a patient with small bowel ischaemia. b. Oedema of the tail of the pancreas (arrow) confirms the diagnosis of acute pancreatitis.

Supportive care

Analgesia

Once the diagnosis has been made analgesia should be given and continued as required. Pain relief is particularly important postoperatively, when early mobilization and adequate physiotherapy helps avoid basal pulmonary atelectasis, deep vein thrombosis and pulmonary embolus.

Nasogastric intubation

Gastrointestinal decompression should be achieved by a combination of free drainage and regular aspiration of the stomach until the associated paralytic ileus has resolved.

Fluid balance

Hypovolaemia and plasma electrolyte deficiencies need to be corrected. A detailed fluid balance chart should document daily losses by gastric aspiration and urine output. In combination with central venous pressure monitoring, these records act as a guide to adequate intravenous administration. If there is a delay in the patient's recovery of more than 7 days total parenteral nutrition should be considered.

Antibiotics

Intravenous administration of broad-spectrum antibiotics active against anaerobic and aerobic organisms (e.g. metronidazole and cefuroxime) prevents bacterial proliferation and release of endotoxins (Farber and Abrams, 1997).

Specific treatment of the cause of peritonitis

Localized peritonitis

In patients with subacute bowel perforations (e.g. diverticulitis, Crohn's disease, appendicitis and postoperative anastomotic dehiscence), radiologically-guided percutaneous drainage of localized intra-abdominal abscesses should be considered as an alternative to operation (Hemming et al, 1991; Bufalari et al, 1996). Laparoscopy has gained wider acceptance in the diagnosis and treatment of localized peritonitis. It is particularly useful in differentiating appendicitis from pelvic inflammatory disease in young female patients with right iliac fossa pain (Slim and Chipponi, 2006).

Generalized peritonitis

If the cause of peritonitis is amenable to surgical intervention (such as perforated bowel, peptic ulcer, gangrenous cholecystitis and appendicitis), operation should be performed as soon as the patient has been adequately resuscitated. The operation required for each cause is beyond the scope of this article. Irrespective of the cause, however, cultures of peritoneal fluid should be taken to guide postoperative antibiotic administration then copious peritoneal lavage should be carried out before closing the abdomen, this being particularly important with faecal contamination. Consideration should also be given to the placement of abdominal drains.

Prognosis and complications

With modern treatment, generalized peritonitis carries a mortality of up to 10% (Lee et al, 2001). This figure is obviously influenced by the associated co-morbidity of each individual patient. In addition to endotoxic shock, systemic complications include organ failure (respiratory, renal, multiple) and bone marrow suppression. Local complications include prolonged paralytic ileus, adhesional small bowel obstruction, liver abscesses and other residual abscesses (subphrenic, inter-loop, pelvic).

Conclusions

Determining the cause of peritonitis is a difficult but intriguing challenge – incorrect treatment can be fatal. Current treatment of both localized and generalized peritonitis consists of a multimodality approach aimed at correction of the underlying cause. It usually entails operation, administration of systemic antibiotics and supportive therapy to prevent or limit secondary complications caused by multiple organ failure. BJHM

Conflict of interest: none.

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KEY POINTS

- Most cases of peritonitis are secondary to bacterial invasion of the peritoneal cavity from the gastrointestinal tract.
- Biochemical and radiological investigations can help confirm a doubtful diagnosis, but treatment should be based on the clinical features.
- If there is any doubt as to the diagnosis and the patient is unwell, early operation is preferable.
- If the cause of peritonitis is amenable to operation, the operation needs to be performed as soon as the patient has been adequately resuscitated.
- Administration of systemic antibiotics and other supportive measures are required to prevent or limit secondary complications.