The ABCs of Acute Peritonitis

Introduction

- Globally, acute peritonitis is a common medical and surgical emergency which is a major contributor to non-trauma deaths despite improvements in diagnosis and surgical and intensive care management.
- Depending on the underlying pathology, the resultant peritonitis may be infectious or sterile (ie, chemical or mechanical). The abdomen is the second most common source of sepsis and secondary peritonitis. Intra-abdominal sepsis is an inflammation of the peritoneum caused by pathogenic microorganisms and their products. The inflammatory process may be localised (abscess) or diffuse in nature.

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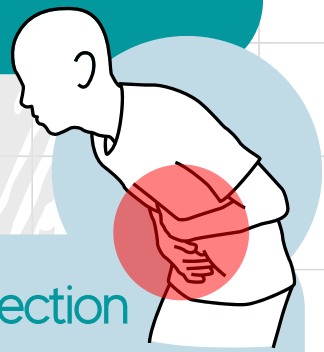
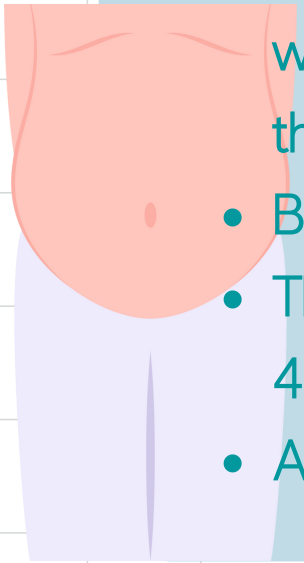
Definition

Peritonitis is defined as an inflammation of the serosal membrane that lines the abdominal cavity and the organs contained therein. The peritoneum, which is an otherwise sterile environment, reacts to various pathologic stimuli with a fairly uniform inflammatory response.



Aetiology

- The two main types of peritonitis are primary spontaneous peritonitis, an infection that develops in the peritoneum; and secondary peritonitis, which usually develops when an injury or infection in the abdominal cavity allows infectious organisms into the peritoneum.
- Both types of peritonitis are life-threatening.
- The death rate from peritonitis depends on many factors, but can be as high as 40% in those who also have cirrhosis.
- As many as 10% may die from secondary peritonitis.



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Types of Peritonitis

1. Primary peritonitis
2. Secondary peritonitis
3. Tertiary peritonitis
4. Chemical peritonitis
5. Peritoneal abscess

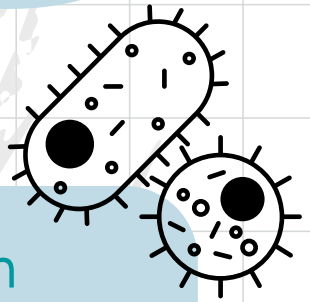
Common microorganisms involved

1. Escherichia coli
2. Streptococci
3. Bacteroides
4. Clostridium
5. Klebsiella pneumoniae

Other Sources

1. Chlamydia trachomatis
2. Neisseria gonorrhoeae
3. Haemolytic streptococci
4. Staphylococcus
5. Streptococcus pneumoniae
6. Mycobacterium tuberculosis and other spp.
7. Fungal infections

Paths to Peritoneal Infection



- Gastrointestinal perforation, e.g. perforated ulcer, appendix, diverticulum
- Transmural translocation (no perforation), e.g. pancreatitis, ischaemic bowel
- Exogenous contamination, e.g. drains, open surgery, trauma
- Female genital tract infection, e.g. pelvic inflammatory disease
- Haematogenous spread (rare), e.g. septicaemia

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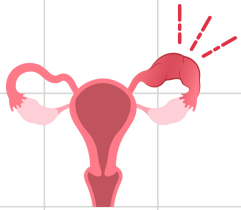
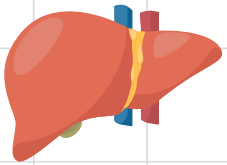
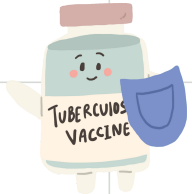
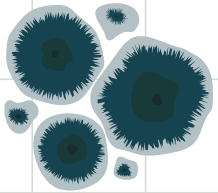




Microbiology





Bacteria from the gastrointestinal tract

- Peritoneal infection is usually caused by two or more bacterial strains.Gram-negative bacteria contain endotoxins (lipopolysaccharides) in their cell walls that have multiple toxic effects on the host, primarily by causing the release of tumour necrosis factor (TNF) from host leukocytes.
- Systemic absorption of endotoxin may produce endotoxic shock with hypotension and impaired tissue perfusion.
- Other bacteria such as Clostridium welchii produce harmful exotoxins. Bacteroides are commonly found in peritonitis. These Gram- negative, non-sporing organisms, often escape detection because they are strictly anaerobic and slow to grow on culture media.

Non-gastrointestinal causes of peritonitis

- Pelvic infection via the Fallopian tubes is responsible for a high proportion of 'non-gastrointestinal' infections. The most common offending organisms are chlamydia and gonococcus. These organisms lead to a thinning of cervical mucus and allow bacteria from the vagina into the uterus and oviducts, causing infection and inflammation.
- Another variant of transperitoneal spread of such organisms is perihepatitis which can cause scar tissue to form on Glisson's capsule, a thin layer of connective tissue surrounding the liver (Fitz-Hugh–Curtis syndrome).
- Other bacterial variants that are discussed separately include tuberculosis and other mycobacterial strains and those causing primary peritonitis (pneumococcus, staphylococcus and streptococcus spp).
- Fungal peritonitis is uncommon but may complicate severely ill patients

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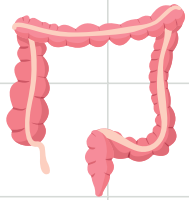
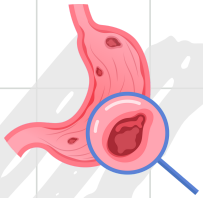
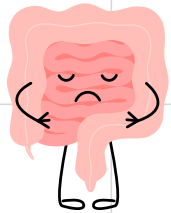




Local Peritonitis



Note: Anatomical and pathological factors may favour the localisation of peritonitis.

1

Anatomical

The greater sac of the peritoneum is divided into (1) the sub- phrenic 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.

2

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 leukocytes and plasma proteins that soon becomes turbid; if localisation occurs, the turbid fluid becomes frank pus. Peristalsis is retarded in the 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 (see below).

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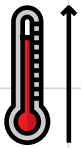
Clinical features



The initial symptoms and signs of localised peritonitis are those of the underlying condition usually visceral inflammation causes are;

- abdominal pain
- specific GI symptoms + malaise
- anorexia
- nausea

When the peritoneum becomes inflamed, abdominal pain will worsen and, in general, temperature and pulse rate will rise.

The pathognomonic signs are;

- localised guarding
- rebound tenderness
- Rigidity

Note: If inflammation arises under the diaphragm, shoulder tip ('phrenic') pain may be felt as the pain is referred to the C5 dermatome.

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Diffused (Generalised) Peritonitis

A number of factors may favour the development of diffuse peritonitis:
Speed of peritoneal contamination is a prime factor. If an inflamed appendix or other hollow viscus perforation before localisation has taken place, there will be an efflux of contents into the peritoneal cavity
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.
The virulence of the infecting organism may be so great as to render the localisation of infection difficult or impossible.
Deficient natural resistance may result from use of drugs (e.g. steroids), disease (e.g. acquired immune deficiency syndrome (AIDS)) or old age.

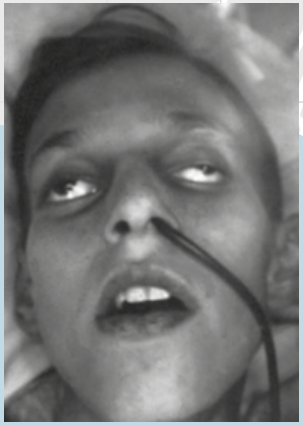
Clinical Features

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.**
- **Tenderness and generalised guarding**
- **Infrequent bowel sounds may be heard**
- **Bowel sounds cease with the onset of paralytic ileus**
- **Pulse and temperature rise**

Late

- **generalised rigidity of the abdomen**
- **Distension**
- **bowel sounds are absent.**
- **Cold and clammy extremities**
- **sunken eyes**
- **dry tongue**
- **thready (irregular) pulse**
- **drawn and anxious face (Hippocratic facies)**



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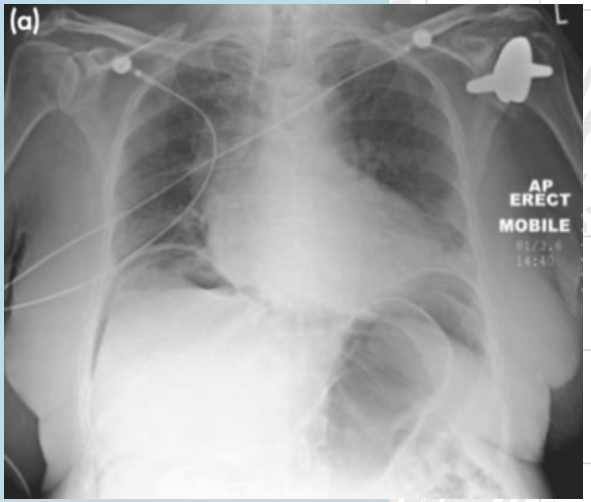
Investigations

Bedside

- Urine dipstick for urinary tract infection
- ECG if diagnostic doubt (as to cause of abdominal pain) or cardiac history.

Bloods

- Baseline U&E for treatment
- Full blood count for white cell count (WCC)
- Serum amylase to rule out acute pancreatitis



Imaging

- Erect chest radiograph to demonstrate free subdiaphragmatic gas.
- A supine radiograph to see dilated gas-filled loops of bowel (consistent with a paralytic ileus)
- Multiplanar computed tomography (CT)
- Ultrasound scanning

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Management

General care of patients

- 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

Complications

Abdominal complications

- Paralytic ileus
- Residual or recurrent abscess/inflammatory mass
- Portal pyaemia/liver abscess
- Adhesional small bowel obstruction

Systemic complications

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

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SIGMUM CASE BASED DISCUSSION

Answers Sheet & Explanations

Acute Peritonitis

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Question 1 Answer

C. Cloudy fluid

Upon initial presentation and management, Peritoneal dialysis patients presenting with cloudy PD fluid should be presumed to have peritonitis until proven otherwise.

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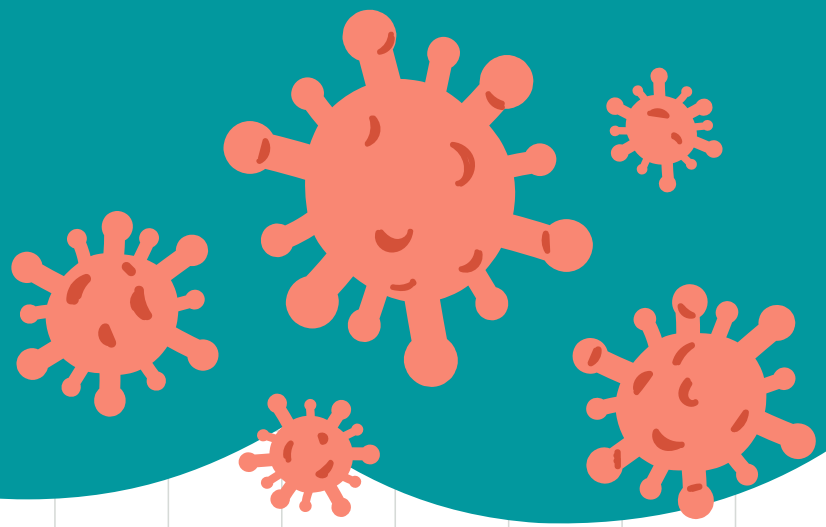
Question 2 Answer

B. Patient may be asymptomatic of pain

Patients with spontaneous bacterial peritonitis may present in various ways and not necessarily exhibit the classical symptoms like abdominal pain or fevers. They may even have atypical or no overt symptoms at all.



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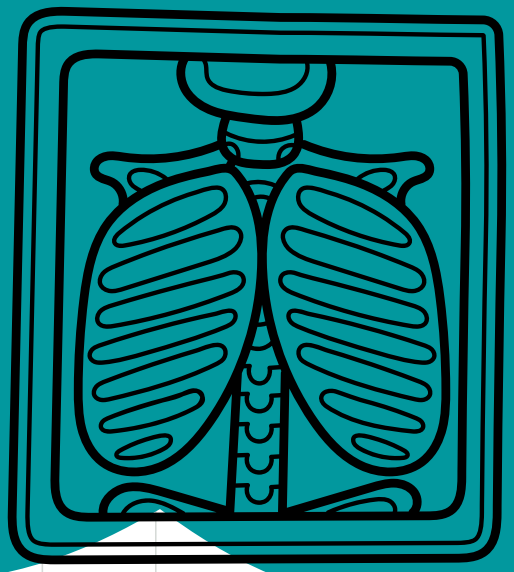
Question 3 Answer

C. Perforated duodenal ulcer

Long term use of NSAIDs can cause peptic ulcers in the duodenum and an untreated duodenal ulcer may perforate, with signs of large amounts of free intraperitoneal gas appreciated on an abdominal X-ray.



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