The peritoneum

BY: DR. AHMED MODHER KHALAF

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 m2 in an adult) being nearly equal to that of the .skin

The peritoneal membrane is composed of flattened polyhedral cells

one layer thick, resting upon a thin layer of flbroelastic ,(mesothelium) .tissue

Beneath the peritoneum, supported by a small amount of areolar tissue, lies a

network of lymphatic vessels and rich plexuses of capillary blood vessels from

which all absorption and exudation must occur. In health, only a few millilitres

,of peritoneal fluid is found in the peritoneal cavity The fluid is pale yellow

somewhat viscid and contains lymphocytes and other leucocytes; it lubricates

.the viscera allowing easy movement and peristalsis

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

‚in which a pure infection with streptococcal (spontaneous' peritonitis')

.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. 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. 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) from host leucocytes. Systemic

absorption of endotoxin may produce endotoxic shock with hypotension and

impaired tissue perfusion. 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, 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. In many laboratories, the culture is discarded if there is no growth in 48 hours. These organisms are resistant to penicillin and streptomycin but sensitive to metronidazole, clindamycin, lincomycin and cephalosporin compounds. Since the widespread use of metronidazole .bacteroides infections have diminished greatly (’Flagyl‘)

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 ■

,Even in patients with nonbacterial 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 itis 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. The proportion of .anaerobic to aerobic organisms increases with the passage of time

: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. مهم

Anatomical-

,The greater sac of the peritoneum is divided into **(a)** the subphrenic spaces the pelvis, and **(c)** the peritoneal cavity proper. The latter is redivided into **(b)** a supracolic and an infracolic compartment by the transverse colon and transverse mesocolon, which deter 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 thence to the pelvis. Posture can assist in directing collections into the .pelvis, as in the 'Sherren' regime for perforated appendicitis

Pathological-

.Adhesions form around the affected organ (1
Inflamed peritoneum loses its glistening appearance and becomes (2
.reddened and velvety
Flakes of fibrin appear and cause loops of intestine to become (3
.adherent to one another and to the parieties
There is an outpouring of serous inflammatory exudate rich in (4
;leucocytes and plasma proteins that soon becomes turbid
.If localisation occurs, the turbid fluid becomes frank pus (5
Peristalsis is retarded in affected bowel, and this helps in preventing (6
.distribution of the infection
The greater omenturn, by enveloping and becoming adherent to (7
inflamed structures, often forms a substantial barrier to the spread of
.infection

Surgical

Drains are frequently placed during operation to assist localisation (and exit)

of intra-abdominal collections: their value is contraversial. 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

Speed of peritoneal contamination is a prime factor in the spread of **(1** peritonitis. 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

,**Stimulation of peristalsis** by the ingestion of food, or even water **(2** 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 **(3** .render the localisation of infection difficult or impossible

.**Young children** have a small omentum **(4**

Disruption of localised collections may occur with injudicious and **(5**
.rough handling, e.g. appendix mass or pericolic abscess
Deficient natural resistance (‘immune deficiency’) may result from **(6**
.drugs (e.g. steroids), disease (e.g. 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 which is involved, with a positive ‘release’ sign (rebound
tenderness), if inflammation arises under the diaphragm shoulder tip .pain may be felt (’phrenic‘)

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. With appropriate treatment localised peritonitis .usually resolves

In about 20 per cent 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 typically found 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

. 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

History and repeated examination must not be forgotten (very (1 (important

A leucocytosis is usually seen in peritonitis but is often delayed for (2 .many hours

Peritoneal diagnostic aspiration may be helpful but is usually (3 unnecessary. After infiltrating the skin of the abdomen with local anaesthetic, the peritoneum is entered in one or more quadrants with a sterile needle and or intravenous cannula attached to a syringe into which is sucked any free fluid. Bile-stained fluid indicates perforated peptic ulcer or gall bladder, the presence of pus indicates bacterial peri-tonitis; blood is aspirated in a high proportion of patients with intraperitoneal bleeding. When aspiration fails, the introduction of a small quantity of sterile physiological saline, followed after a few .minutes by peritoneal aspiration may produce fluid of diagnostic value

Microscopy of the fluid may show neutrophils (indicative of acute .inflammation) and bacteria (confirming infection)

An X-ray film of the abdomen may confirm the presence of dilated (4 ,gasfilled 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

Serum amylase estimation may uphold the diagnosis of acute (5

pancreatitis provided it is remembered that moderately raised values

are frequently found following other abdominal catastrophes and

.operations, e.g. perforated duodenal ulcer

Ultrasound and CT scanning, when available, may also be helpful in (6 some patients by identifying a cause of peritonitis e.g. perforated appendicitis, acute pancreatitis. Such knowledge may influence .operative approach or contraindicate operation

Treatment

:Treatment consists of

;General care of the patient

;Specific treatment for the cause

.Peritoneal lavage when appropriate

General care of the patient

Correction of circulating volume and electrolyte imbalance (1
.(Patients are frequently hypovolaemic with electrolyte disturbances)
Central venous catheterisation and pressure monitoring may be (2
helpful in correcting fluid and electrolyte balance particularly in patients
.with concurrent disease as heart failure
Plasma protein depletion may also need correction as the inflamed (3
.peritoneum leaks large amounts of protein
,If the patient's recovery is delayed for more than 7—10 days (4
intravenous feeding **('hyperalimentation' or 'total parenteral**
.nutrition') is required

Gastrointestinal decompression. A nasogastnic tube is passed into (5 the stomach and aspirated. Intermittent aspiration is maintained until .the paralytic ileus resulting from peritonitis has recovered Measured volumes of water are allowed by mouth when only small ,amounts are being aspirated. If the abdomen is soft and not tender and bowel sounds return, oral feeding may be progressively introduced. It is important not to prolong the ileus by missing this .stage

Antibiotic therapy. Administration of antibiotics prevents the (6
multiplication of bacteria and the release of endotoxins. As the infection
is usually a mixed one, initially parenteral broad-spectrum antibiotics
.active against aerobic and anaerobic bacteria should be given
A fluid balance chart must be started so that daily output by gastric (7
,aspiration and urine is known. Additional losses from the lungs, skin
and in faeces are estimated, so that the intake requirements can be
.calculated and seen to have been administered

Throughout recovery, **the haematocrit and serum electrolytes and** (8

.urea must be checked regularly

Analgesia. The patient should be nursed in the sitting-up position (9 and

must be relieved of pain before and after operation. Once the diagnosis

.has been made morphine may be given, and continued as necessary

If appropriate expertise is available epidural infusion may provide

excellent analgesia. Freedom from pain allows early mobilisation and

adequate physiotherapy in the postoperative period which helps to

prevent basal pulmonary collapse, deep-vein thrombosis and

.pulmonary embolism

Vital system support. Especially if septic shock is present, special(10

.measures may be needed for cardiac, pulmonary and renal support

Administration of oxygen postoperatively can help to prevent and settle

down the effects of septic shock, especially adult respiratory distress

syndrome (ARDS) which may require a period of mechanical

ventilation. If oligunia persists despite adequate fluid replacement, both

.**diuretics** and **inotropic** agents such as dopamine may be needed

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 penitoneal 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

Systemic complications of peritonitis

Bacteraemic/endotoxic shock ■

Bronchopneumonia/respiratory failure ■

Renal failure ■

Bone marrow suppression ■

Multisystem failure ■