



INTRODUCTION

Intra-abdominal sepsis, including peritonitis and abscess formation, present serious and potentially life-threatening events in the management of surgical patients with significant resultant morbidity and mortality. Twenty percent of patients presenting with generalised suppurative peritonitis and over 50% of high risk patients with post-operative intra-abdominal sepsis die despite intensive treatment. Major recent advances have occurred in the conceptual understanding of the pathogenesis, diagnostic methodology, antibiotic therapy and application of non-invasive technology in intra-abdominal sepsis. As a consequence, the management of intra-abdominal infection requires a careful interdisciplinary approach, with close collaboration between surgeon, physician, radiologist, bacteriologist and anaesthetist.

This chapter outlines the relevant anatomy and physiology, pathogenesis, clinical features, diagnostic approach and management of primary and secondary bacterial peritonitis and intra-abdominal abscess.

ANATOMY

The peritoneum is the largest serous membrane in the body and consists of a single layer of mesothelial cells supported on a connective tissue base. The parietal peritoneum lines the anterior, lateral and posterior abdominal walls, the undersurface of the diaphragm and the pelvis and is reinforced by transversalis fascia. The visceral peritoneum is reflected on the intra-abdominal viscera, mesentery and omentum, creating a closed cavity except for the open ends of the fallopian tubes. Although the parietal and visceral surfaces are part of the same membrane, the distinction is relevant with regard to differences in

sensory innervation. The peritoneal cavity forms the potential space

Between the parietal and visceral layers and is divided into the general peritoneal cavity or greater sac, and the lesser sac which communicate via the foramen of Winslow. The anatomic surface area of the peritoneum in adults is about 1.7 sq. meters and approximates the total body surface area.

The parietal peritoneum has a somatic afferent nerve supply and is sensitive to all forms of stimuli. Irritation of the parietal peritoneum produces sharp, well localized, discriminate pain with tenderness, involuntary guarding and rigidity of the abdominal muscles if the stimulus is sufficiently intense. The parietal peritoneum on the undersurface of the diaphragm is supplied centrally by the phrenic nerves and peripherally by lower intercostal nerves. Irritation of the peritoneum over the central portion of the diaphragm results in referred pain in the distribution of the cutaneous branches of the third, fourth and fifth cervical nerves over the shoulder region.

The visceral peritoneum receives afferent innervation only from the autonomic nervous system and is relative insensitive to tactile, thermal and chemical stimuli. Stimuli from the visceral peritoneum characteristically are poorly localized and are perceived as a dull pain. The visceral afferent nerves have no receptors to mediate pain and temperature, but are sensitive to bowel ischaemia and distention or traction on the mesentery.

PHYSIOLOGY

The peritoneum consists of a single surface layer of mesothelial cells

supported on a basement membrane and a deeper well-vascularized connective tissue layer containing collagen and elastic fibres, fat cells, reticulum cells and macrophages. The peritoneal cavity contains less than 50ml of clear fluid consisting of water, electrolytes and solutes derived from interstitial fluid and plasma. Normal peritoneal fluid has a specific gravity of less than 1.016 and less than 3 grams per ml of protein, predominantly albumen. Fibrinogen is not present and the fluid will not clot. Much of the peritoneal membrane acts as a passive, semi-permeable barrier to the bidirectional diffusion of water and most solutes. Normal peritoneal fluid contains less than 3000 cells per ml³ with 50% lymphocytes, 40% macrophages, a few eosinophils, mast cells and occasional desquamated mesothelial cells. Bacteria are absent. Peritoneal fluid has minimal antibacterial activity, mediated both via the complement system and the lymphocyte population in the fluid. The number of granulocytes is significantly increased in the presence of inflammation. Peritoneal aspiration may be of value for culture and chemical analysis to facilitate the diagnosis of inflammatory conditions, tumours or intraperitoneal trauma. The principal route of absorption and clearance of fluids and particulate matter from the peritoneal cavity is by lymphatics. Reverse flow is prevented by one-way valves within the thoracic lymphatic system. Experimentally, particulate matter including red cells and bacteria, are recoverable from the thoracic lymph within 6 minutes and from the blood within 12 minutes after intraperitoneal injection. Absorption of fluid by the diaphragmatic lymphatics produces a cephalad flow of peritoneal fluid which is promoted by increased respiratory movement. The rapid peritoneal clearance of particulate matter and fluid functions as the essential first line of defence following initial peritoneal contamination.

DEFINITION

Peritonitis is the acute inflammatory response of the visceral and parietal peritoneum to bacterial, chemical, radiation or foreign body injury. The two major clinical categories of peritonitis are based on etiology:

- 1) Primary peritonitis is an infection of the peritoneum occurring de novo without obvious intra-abdominal pathology.
- 2) Secondary bacterial peritonitis is a purulent inflammation of the peritoneum due to contamination following a complication of a pre-existing primary intra-abdominal process such as perforated peptic ulcer, ruptured appendix, a disrupted anastomotic suture line or as consequence of bacterial contamination from external sources (eg. penetrating injury).

Secondary bacterial peritonitis is more common and of greater surgical significance than primary peritonitis; accurate differentiation between the two types is crucial for optimal management.

PRIMARY PERITONITIS

Children

Primary peritonitis is uncommon, accounting for less than 1% of all cases of peritonitis and predominantly affects girls younger than 10 years of age. The bacterial involved are primarily pneumococci and Group A streptococci. Less commonly, gram-negative bacilli and Group B streptococci are found. A useful diagnostic feature in differentiating the usually monobacterial spontaneous peritonitis from the polymicrobial flora of secondary peritonitis is the single species of bacteria cultured. About one half of cases of pneumococcal peritonitis occur in children with nephrosis. The causative bacteria gain access to the peritoneum by a

haematogenous route or uncommonly, via the fallopian tubes.

The clinical manifestations are those of acute diffuse peritonitis. A pre-existing upper respiratory infection, pneumonia or otitis media may mask the initial onset of peritonitis. Severe, generalized abdominal pain is the predominant symptom. High fever, often with chills, irritability, vomiting and diarrhoea are common. On examination, the child appears ill, pyrexial with a temperature of 40°C, and an elevated pulse rate. The abdomen is diffusely tender and ascites with shifting dullness may be demonstrated. Bowel sounds are hypoactive or absent. The white blood count reveals a leucocytosis of 20,000 to 40,000 ml³ with a polymorphonuclear predominance and a shift to the left. Heavy albuminuria is present in cases superimposed on nephrosis. Blood culture is usually positive for streptococci or pneumococci. A peritoneal aspirate under local anaesthesia with an immediate Gram stain is important to differentiate primary peritonitis in which surgical intervention is contra-indicated from peritonitis secondary to a perforated appendix where prompt surgery is necessary. If only cocci are seen on the smear, conservative therapy is appropriate. A mixed bacterial flora on the smear demands an exploratory laparotomy.

Adults

Spontaneous bacterial peritonitis (SBP) is an infective complication caused by enteric organisms and usually occurs in alcoholic cirrhotic patients with ascites. Characteristically, the patient presents with fever, abdominal pain, reduced or absent bowel sounds and encephalopathy in the presence of gross ascites. The presentation may be subclinical and subtle and the only clinical manifestation in a known cirrhotic may be unexplained deterioration in liver function,

progressive encephalopathy or diminishing renal function. A diagnostic ascitic tap is necessary to determine the presence of bacteria, neutrophils, protein, glucose and pH of the ascitic fluid. Gram stains are positive however only in one-third of culture proven cases. Careful aerobic and anaerobic cultures isolate a single organism in 80% of patients with SBP; the most frequent organisms recovered are E.Coli and pneumococci. Anaerobes are virtually never isolated. The ascitic fluid WBC is generally 500/ml³ with more than 75 neutrophils/ml³. A peritoneal acidosis with a pH of less than 7.3 is due to lactic acid production by the infecting organisms. While the bacterial infection may be effectively treated with suitable antibiotics, the prognosis is poor and exceeds 80% due to progressive liver decompensation, renal failure, encephalopathy and haemorrhage.

SECONDARY BACTERIAL PERITONITIS

Secondary bacterial peritonitis is an acute suppurative inflammatory process of the peritoneal cavity arising as a consequence of either:

- Primary disease of the abdominal viscera
- Penetrating or blunt trauma
- Previous intra-abdominal surgery

Aetiology

Secondary peritonitis may complicate almost any abdominal condition including inflammatory, traumatic, obstructive or neoplastic processes (Table 1). Perforation following appendicitis or diverticulitis, is the most common cause of acute suppurative bacterial peritonitis. Delayed treatment of duodenal and gastric perforations are further common causes of bacterial peritonitis. Gangrene of the bowel, either from strangulation or ischaemia, remains an important cause of peritonitis. Pelvic peritonitis often

accompanies pelvic inflammatory disease, progresses to frank generalised peritonitis. Penetrating or blunt abdominal trauma may cause contamination of the peritoneal cavity and subsequent widespread inflammation. Some degree of inflammation follows every surgical procedure within the peritoneal cavity due to inevitable minor contamination from room air, poor surgical technique, glove perforation or spillage during resection or manipulation. Such contamination is nearly always contained by the protective mechanisms within the peritoneal cavity, but gross contamination, virulent organisms or immune compromise may result in clinically significant peritonitis.

Pathogenesis

The initial local responses of the peritoneum to contamination or injury are vascular dilatation, hyperaemia and increased capillary permeability, followed by transudation of fluid from the vascular and interstitial spaces. Fluid containing opsonins, polymorphonuclear leucocytes and macrophages accumulates in the involved tissues and the free peritoneal space. Phagocytosis of bacteria and foreign material is the major local peritoneal defense mechanism and the peritoneal exudate contains antibodies and complement which enhance aggregation, adherence and engulfment of micro-organisms, in addition to promoting chemotaxis and diapedesis of white cells. The peritoneum loses its normal sheen and acquires a dull ground-glass appearance due to deposition of fibrinogen. The release of tissue thromboplastin converts fibrinogen to fibrin. Normal peritoneum however, has a fibrinolytic capacity due to plasminogen activator present in mesothelial cells. Injury to the peritoneum produces an inhibition in fibrinolytic activity which permits the development of fibrinous adhesions that wall-off and surround the inflamed

area of injury. This abundant fibroblastic exudate aids specific humoral and cellular immune defense mechanisms in localizing the inflammatory process. Further restriction of the inflammatory process is due to peritoneal and omental tissue adaptation with adhesions between loops of bowel and the parietal wall with migration of the greater omentum in to the area of inflammation to further compartmentalise the infective process. The regional lymphatic circulation provides an important peritoneal defense mechanism by absorption of bacteria through the lymphatic system present beneath the diaphragmatic mesothelium. Bacteria removed via the lymphatics are filtered by the thoracic lymph nodes or, once in the systematic circulation, are eliminated by the reticulo-endothelial cells in the liver and spleen.

If the peritoneal defenses are able to contain the inflammatory process, the disease may end by resolution. A second possible outcome is localization of the process with contained suppuration and abscess formation. If the peritoneal and systemic defence mechanisms are unable to localise the inflammation, generalized suppurative peritonitis occurs. The factors favoring spread of the inflammatory process are:

- overwhelming or continued bacterial contamination.
- virulence and synergistic bacterial action
- presence of foreign bodies or adjuvant factors deleterious to resolution of the inflammation..

Continuing contamination of the peritoneal cavity occurring with free anterior perforation of a duodenal ulcer or perforation secondary to an obstruction colonic carcinoma results in rapid dissemination of bacteria throughout the peritoneal cavity. The volume and rate of spill are critical factors. Intra-abdominal spread of

contamination is promoted by the peristaltic movement of the viscera and by diaphragmatic motion producing a cyclic pressure differential between the subphrenic space and the peritoneal cavity. The subphrenic area is contaminated by movement of bacteria into the potential space by the negative suction of the diaphragm during respiration. In addition, the effect of gravity in the recumbent patient allows fluid to drain into the most dependent spaces.

A mixed bacterial flora is usually found in acute suppurative peritonitis after secondary contamination from the intestinal tract. The most common organisms are aerobic coliforms, in particular *E. Coli*, *Proteus*, *Klebsiella*, and anaerobes including *Bacteroides*, anaerobic cocci and *Clostridia*. (Table II) All these bacteria are potentially lethal pathogens in the human. The mixed aerobic-anaerobic faecal flora commonly found in peritonitis frequently acts synergistically. Particular combinations such as *E. Coli* and *B. fragilis* produce a mortality rate significantly greater than the mortality rate of each organism alone. The mortality rate resulting from peritonitis is in part a dose-dependent phenomenon correlating directly with the number of pathogenic bacteria introduced into the peritoneal cavity. In duodenal perforations, few bacteria are cultured initially, but bacterial proliferation increases significantly after six hours. Perforation of obstructed distal small bowel or colon however, results in immediate gross contamination. The site of the gastrointestinal perforation and by inference, the number of bacteria escaping from the lumen are therefore critical determinants in the risk of subsequent infection. Not all organisms cultured from the intraperitoneal fluid however have equivalent virulence; *E. Coli* and *B. fragilis* are regarded as the major pathogens in intra-abdominal sepsis since they are found most often in positive blood cultures.

Certain adjuvant substances increase the lethality of peritonitis, eg. the presence of necrotic tissue in the peritoneal cavity significantly enhances the risk of continuing infection. The virulence of contaminating micro-organisms is further enhanced by mucus, bile, barium sulphate and haemoglobin. This characteristic is attributed to the detrimental effect of the adjuvant on defence mechanisms, especially phagocytosis. The co-existence of haemoglobin and bacterial inoculum within the peritoneal cavity produces an increased susceptibility to the micro-organisms, since clearance of bacteria from the peritoneal cavity is inhibited by haemoglobin. Haemoglobin specifically inhibits intraperitoneal chemotaxis of neutrophils, phagocytosis and intracellular killing of bacteria. Haemoglobin also interferes with lymphatic absorption of bacteria and exerts an additional local toxic effect after degradation.

Pathophysiology

Peritonitis produces profound physiological alterations both locally within the abdominal cavity and also systemically affecting the cardiovascular, respiratory, renal and neuro-endocrine systems.

- **Fluid Shifts**

The peritoneum reacts to inflammation by vascular dilatation, hyperaemia and exudation of fluid from the vascular space into the free peritoneal cavity, the loose connective tissues beneath the mesothelium of viscera and mesenteries as oedema fluid and into the lumen of the atonic dilated gastro intestinal tract. This translocation of water, electrolytes and protein into the sequestered third space effectively removes fluid from the body economy. The rate of fluid loss is proportional to the surface area of peritoneum involved in the inflammatory

process and with extensive peritonitis, may reach 4 to 6 litres in 24 hours.

- Ileus

Generalised peritonitis produces an inhibition of intestinal motility with resultant adynamic ileus. Distention of the bowel with unabsorbed fluid and gas, aggravated by relative mural ischaemia if intraluminal pressure exceeds capillary perfusion pressure, permits bacteria to penetrate the mucosal barrier and enter the vascular compartment.

- Endocrine response

Peritonitis results in a major systemic stress producing a vigorous response from the pituitary-adrenal axis. Adrenal medullary secretion of catecholamines is in large part responsible for the vasoconstriction, tachycardia and sweating accompanying the initial response to peritonitis. Aldosterone secretion increases as a response to hypovolaemia and further aggravates potassium loss and sodium retention. Release of antidiuretic hormone results in renal conservation of water which may exceed sodium retention with consequent dilutional hypotonicity of plasma sodium.

- Cardiovascular System

Loss of extracellular fluid volume depletes central venous return, lowering cardiac output and increasing heart rate. Compensatory vasoconstriction results in an increased total peripheral resistance to maintain blood pressure and cardiac and cerebral perfusion pressures. Decreased perfusion and oxygenation to the splanchnic bed, kidneys and inactive muscles results in anaerobic glycolysis with progressive accumulation in lactic acid.

Metabolic acidosis is aggravated by decreased renal clearance, secondary to reduced renal perfusion. If acidosis progresses, depression of cardiac contractility further decreases cardiac output.

- Respiratory System

Demands on the respiratory system increase significantly in peritonitis, with a decrease in total respiratory capacity. Abdominal distention secondary to ileus causes an elevation and restriction of diaphragmatic movement. Respiratory restriction, fatigue and inefficient respiratory effort diminish ventilatory volume with ensuing atelectasis which may progress to ventilation-perfusion imbalance, intrapulmonary arteriovenous shunting and peripheral hypoxaemia.

- Kidneys

Renal changes induced by peritonitis are primarily a reflection of hypovolaemia, reduced cardiac output and increased secretion of aldosterone and antidiuretic hormone. Renal blood flow, glomerular filtration and urine volume are reduced. Aldosterone promotes sodium retention and antidiuretic hormone results in increased reabsorption of water from the distal tubules with a further decrease in urine output.

Protein catabolism progresses during the duration of peritonitis and serum albumen concentrations decrease with further loss into the peritoneal cavity. Hepatic glycogen stores are depleted and increased insulin secretion mobilises fat as an energy source. The net effect of these metabolic changes in the body results in a significant energy deficit.

Clinical features

The predominant symptom in peritonitis is abdominal pain

aggravated by any movement, including respiration. Perforation of a peptic ulcer with massive contamination of gastric contents produces sudden onset epigastric pain with rapid spread to involve the entire abdomen corresponding to spread of acid-pepsin throughout the peritoneal cavity.

The spread of pain from lesions such as a perforated appendix or a diverticular abscess is more gradual corresponding to progression of bacterial inflammation. Anorexia, nausea and vomiting commonly accompany peritonitis. Patients may complain of feeling feverish, sometimes with rigors, and of thirst and decreased urine output. Other symptoms of a dynamic ileus produced by the peritonitis are the inability to pass faeces or flatus and abdominal distention.

Physical findings

Patients characteristically lie quietly in bed with the knees flexed and frequent shallow respiration. With advancing infection, respiration becomes more rapid as the demand for tissue oxygen utilization increases and acidosis worsens. Respiration is primarily intercostal since diaphragmatic movement worsens pain. Body temperature elevation in peritonitis is usually 39° to 40°C, but may reach 42°C in overwhelming sepsis. Subnormal temperatures are seen in the early stages of chemical peritonitis and late in septic shock, where progressive peripheral hypothermia is a grave sign. Immunosuppressed patients do not show a marked febrile response; fever is usually higher and more spiking in character in younger, healthier patients than in the elderly. Increased tachycardia with weak peripheral pulses reflects the circulatory effects of both hypovolaemia and toxemia. With progression of peritonitis, blood pressure may be significantly reduced due to septic shock.

Abdominal examination elicits generalized tenderness with reflex guarding of the abdominal muscles. Extensive abdominal hyperresonance due to gaseous bowel distention may be demonstrated by percussion. Pneumoperitoneum from a ruptured hollow viscus may produce decreased liver dullness. Bowel sounds are audible on auscultation in the initial stages of peritonitis, but with spread of the inflammation the silent abdomen of adynamic ileus supervenes. Rectal and vaginal examination are an essential part of the diagnostic procedure and must not be omitted in evaluating the patient with signs of peritonitis. Anterior tenderness on pelvic examination, or the presence of a mass in the pouch of Douglas, may provide an important clue to pelvic sepsis. Palpation of the cervix with excitation tenderness suggests the presence of inflammation in the uterus and adnexae, while examination of material on the glove may give a clue to the etiology of the process.

Diagnostic Studies

A leucocytosis between 15,000 and 20,000 per ml³ is usual in peritonitis with the differential count showing a polymorphonuclear predominance and a shift to the left. Blood chemistry shows variable degrees of haemoconcentration and dehydration. Acidosis, both metabolic and respiratory, is present in severe and late cases. Urinalysis serves to exclude infection of the urinary tract as a source of abdominal pain. An increased serum amylase may indicate pancreatitis or a surgically correctable cause of hyperamylasaemia including perforated duodenal ulcer or strangulated small bowel.

The x-ray findings of peritonitis may be non-specific with features of a paralytic ileus with distention of both small intestine and colon. Inflammatory exudate and oedema of the bowel wall

produce widening of the space between adjacent bowel loops and peritoneal fat lines and psoas shadows are obliterated. Free air may be visible beneath the right hemidiaphragm and occasionally beneath the left hemidiaphragm. An intra-abdominal abscess may be detected by extraluminal air fluid levels or by the mottled soap-bubble appearance of an anaerobic abscess. In advanced cases with bowel necrosis, intramural gas may be seen in the intestinal wall. Portal vein gas is a late sign of bowel infarction in adults and may occur in infants with necrotizing enterocolitis.

Management of bacterial peritonitis

Every patient with acute peritonitis is potentially critically ill and requires the rapid institution of physiological monitoring and aggressive treatment. The essential elements of management in sequence are fluid resuscitation, antibiotic administration, decompression of the gastro-intestinal tract and, when appropriate, exploratory laparotomy. The patient with established peritonitis requires repeated systematic clinical evaluation with frequent determination of blood pressure, pulse, central venous pressure, and urine output with specific gravity. In all older patients and in younger patients with cardiovascular compromise, a Swan-Ganz pulmonary artery balloon catheter should be inserted to determine pulmonary artery and wedged pulmonary venous pressures. A Foley catheter is essential to determine hourly urine output and a nasogastric tube should be inserted for decompression of the stomach to avoid vomiting and further progression of intestinal distention. Laboratory tests should be obtained for haematocrit, blood count, electrolytes, urea and creatinine, amylase and liver function. Respiratory function is assessed with repeated arterial blood gas determination, while in critically ill patients, a radial arterial line provides

access for arterial samples for blood gas analysis and a constant mean arterial pressure record.

Hypoxaemia is a frequent complication in diffuse peritonitis due to increased permeability of the pulmonary vasculature with transudation of fluid into alveoli and decreased oxygen exchange and is treated by increasing inspired oxygen using a 40% ventimask and assessing response with regular blood gas analysis. Endotracheal intubation and positive pressure respiration with a volume controlled respirator are indicated if the patient remains hypoxic.

A critical caveat in the treatment of peritonitis is the frequent underestimation of prior fluid loss. Replacement with crystalloid should take into account:

- maintenance fluid requirements.
- ongoing loss via the nasogastric tube.
- prior fluid loss including dehydration from prolonged vomiting.
- third space loss into the peritoneal cavity and bowel lumen.

Elevation of the serum haematocrit and blood urea are useful indicators of the degree of dehydration and third space loss. The primary goals of fluid management are the maintenance of a normal blood pressure and the establishment of an adequate urinary output approaching 30ml per hour. Rapid initial fluid replacement should use crystalloid supplemented by colloid solutions and is monitored continuously against central venous and pulmonary wedge pressures. If the haematocrit is low in the septic patient, blood transfusion is an appropriate addition to crystalloids, both for replacement and correction of anaemia.

Parenteral antibiotic administration should commence before surgery and

should anticipate both aerobic and anaerobic flora with provision for the full bacteriological spectrum. The parenteral route is important to ensure adequate antibiotic tissue levels during surgery. The aim of antibiotic therapy is to reduce both the systemic and local infectious complications of peritonitis. The best current results are achieved by initial empiric triple antibiotic therapy designed to eliminate the three major groups of commonly isolated bacteria including coliforms, enterococci and anaerobes. Aminoglycosides are bactericidal to most facultative gram-negative enteric organisms and are indicated in peritonitis caused by these bacteria. Plasmid-mediated resistance to aminoglycosides is common among gram-negative organisms and the therapeutic agent may require changing in persistent or recurrent infections. Aminoglycosides are excreted in the urine and patients with impaired renal function require reduced dosages to prevent renal and ototoxicity. The volume of distribution and rate of excretion is variable in individual patients and dosage requirements should be based on trough and peak levels to minimise complications after intravenous administration. In patients with significant renal impairment, third generation cephalosporins provide an effective substitute for aminoglycosides. Most anaerobes, including *Bacteriodes* species, are resistant to aminoglycosides. Metronidazole is the anti-anaerobic agent of choice. *Enterococcus* is a frequent isolate in peritonitis and is an important synergistic partner with anaerobes in experimental peritonitis. *Enterococcus* is usually sensitive to ampicillin and is advisable in patients with secondary bacterial peritonitis. No precise criteria exist for the length of antibiotic therapy in intraperitoneal sepsis and a period of 7-10 days is usually adequate while clinical improvement continues. Breakthrough bacteraemia during administration of appropriate antibiotics is presumptive

evidence of residual or recurrent infection, provided nosocomial infection has been excluded.

Surgical Treatment

Operative management of bacterial peritonitis is directed at the control of the source of contamination by closure or resection of the area of perforation, removal of the bacterial inoculum by suction or lavage, debridement of grossly necrotic tissue and prevention of recurrence of sepsis. For generalized peritonitis, a midline incision provides optimal access to all quadrants of the abdomen. A specimen of peritoneal fluid should be obtained for aerobic and anaerobic culture. All purulent material and blood should be evacuated from the peritoneal cavity once the source of contamination has been controlled. Careful separation of loops of matted bowel may uncover unsuspected intermesenteric abscesses. In generalized peritonitis, the sub-diaphragmatic, subhepatic and lateral peritoneal gutters and pelvis should be irrigated with warm saline. All gross foreign material including necrotic tissue, faecal residue, blood or bile should be sucked out after irrigation since the virulence of peritoneal infections is enhanced by the presence of adjuvant foreign substances. In local abscesses such as a contained appendix abscess, generalised peritoneal irrigation is unnecessary and undesirable after complete evacuation and drainage of the abscess cavity.

An alternative approach which has been proposed is radical peritoneal debridement as a primary method of treatment in severe generalised peritonitis during which the entire peritoneal cavity is meticulously debrided of fibrin strands, blood clot and purulent membrane, followed by thorough irrigation with saline until the effluent is clear. While good initial results have been presented in

patients treated with radical peritoneal debridement, randomised clinical data have found no advantage using this technique over standard surgical treatment. Local intra-operative peritoneal irrigation with saline or antibiotic instillation have been used in several forms. Although peritoneal irrigation may spread bacteria throughout the peritoneal cavity, lavage does reduce the number of bacteria present, as well as diminishing the concentration of adjuvant substances. However, the mortality and infective complications of experimental peritonitis are not significantly reduced by saline irrigation alone. Residual saline left in the peritoneal cavity following irrigation dilutes bacterial opsonins and suspends bacteria in a fluid medium, decreasing phagocytosis and permitting bacterial proliferation. All residual fluid should therefore be aspirated upon completion, prior to abdominal closure. The use of intra-operative irrigation with antibiotic solutions have, in a randomised study, shown reduction only in infective complications without a reduction in mortality. Other studies have, however, been unable to show any advantage of intra-operative antibiotic irrigation over saline irrigation in patients with peritonitis who in addition received systemic antibiotics which achieve intraperitoneal levels comparable to serum levels. Patients with massive peritoneal contamination or with depressed host defenses may benefit from further post-operative peritoneal lavage.

Drainage of the free peritoneal cavity in peritonitis is seldom effective since standard drains are rapidly isolated and sealed by omentum, exudate and loops of intestine. Drains may also act as a foreign body aggravating intraperitoneal infection and allowing external bacteria to enter the peritoneal cavity. Drains are effective only when their purpose is to evacuate an abscess cavity, establish controlled fistulae or offer a preferential drainage

route after extensive surgery. Triple lumen sump drains which have inlets for both air and irrigation fluid, and a shielded, fenestrated outlet to which suction can be applied, provide more efficient drainage than corrugated or vacuum drains. Sump drains are therefore preferred when large, dependent cavities require drainage. Controlled open drainage leaving the peritoneum open, without closing the abdominal wall, has been advocated as a technique in selected patients with gross contamination and abdominal wall defects. In this situation, polypropylene mesh (Marlex) is used to protect the bowel and prevent evisceration. Closure of the abdominal wound after surgery for peritonitis is best accomplished by approximating only fascia as a single layer mass closure using either monofilament synthetic suture such as Nylon or Prolene, or an absorbable polyglycolic acid (Dexon) suture. With gross contamination, the skin and subcutaneous tissues should be left open and packed with a fine mesh saline-soaked gauze. The gauze dressings are changed daily and, when a clean wound with granulation tissues is evident, usually within 4 to 5 days, the wound is closed with sterile adhesive strips. In selected cases, the use of retention sutures or placement of a plastic zipper may be appropriate to avoid wound dehiscence and evisceration. The zipper has the advantage of allowing repeat laparotomies to be performed without suturing the sheath.

The ultimate outcome of secondary peritonitis depends on the patient's age, previous nutritional status, concomitant disease states including immunosuppression, and the source of sepsis. Important management factors determining prognosis include:

- a) avoidance of unnecessary delay before operative therapy.
- b) the correct choice and administration of antibiotics.

c) nutritional and haemodynamic support.

Continued peritoneal contamination, septicaemia, fluid and electrolyte abnormalities and respiratory failure are the principal causes of death during the first week. Later causes are renal and hepatic failure. Stress ulceration, upper GIT bleeding and intestinal obstruction are further late causes of morbidity, in addition to being a mortality factor.

INTRAPERITONEAL ABSCESSSES

Intraperitoneal abscesses may be either solitary or multiple and develop when a localized purulent fluid collection becomes walled off by the host tissue reaction in one of the potential spaces of the peritoneal cavity or between adjacent loops of bowel.

Aetiology

Intra-abdominal abscess formation is due to either:

1. effective localization of the primary pathology such as an appendiceal or diverticular abscess
2. a sequel or complication following generalized peritonitis or
3. intraperitoneal contamination secondary to external trauma or complicating previous surgery

The four major intraperitoneal anatomic spaces in which abscesses commonly localise are: (Fig 1)

- the right and left subphrenic spaces and the lesser sac
- the subhepatic space or Morrison's pouch
- the intermesenteric area, including the paracolic gutters and interloop areas
- the pelvis

The increased tendency for peritonitis to resolve with the formation of a pelvic or subphrenic abscess reflects the anatomy of the peritoneal cavity with preferential drainage either into

the pelvis by gravity or the subphrenic spaces due to diaphragmatic movement.

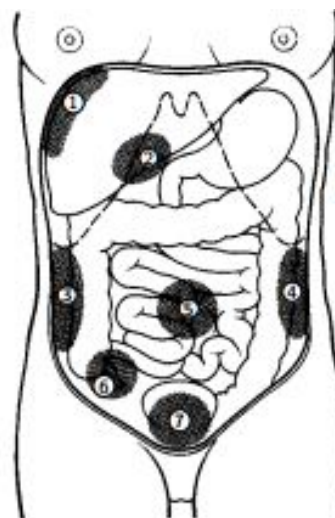


Fig 1.

1. Subphrenic space
2. Subhepatic space
- 3/4. Paracolic gutters
5. Interloop
6. Appendiceal
7. Pelvic

Clinical: Symptoms and Signs

Symptoms and signs may be variable due to the anatomy of the intraperitoneal spaces. Fever is the most common symptom and is intermittent at first becoming progressively higher and careful with maturation of the abscess. Although localized sepsis is generally associated with a swinging temperature, the presence of fever is more significant than the pattern of the pyrexia. Fever may be absent in very young, elderly, malnourished, uraemic or immunocompromised patients. Fever may also be absent in patients with localized infections on antibiotics which suppresses, but do not eradicate infection. When antibiotics are discontinued, fever may recur. Overwhelming septicaemia may present without pyrexia or with hypothermia. Rigors and temperature peaks above 39°C are due to transient bacteraemia. Abdominal tenderness, pain and a palpable mass may be

present only when the parietal peritoneum forms a portion of the abscess wall. Peritoneal signs with intermesenteric, subphrenic and pelvic abscesses frequently are absent. Although abdominal pain may occur, it is seldom well localized. Paralytic ileus is a frequent accompaniment and may be localized or diffuse. Symptoms of nausea, vomiting and diarrhoea may be present. Pelvic abscesses may be heralded by tenesmus or urinary frequency and a tender mass may be palpable on rectal or vaginal examination. In the postoperative patient, persistent ileus, fever, leucocytosis, a falling platelet count and deteriorating liver function with low-grade hyperbilirubinaemia or pulmonary and renal insufficiency should initiate a persistent search for a hidden abscess. In patients receiving hyperalimentation, the development of persistent hyperglycaemia or acidosis may be due to an unsuspected abscess. The only indirect clinical findings with a subphrenic abscess may be pulmonary changes including a pleural effusion, elevation of the diaphragm and diminished basal breath sounds.

Diagnosis & Special Investigations

A leucocytosis greater than 15 000 per ml³, accompanied by a left shift with an elevated ESR is invariably present. Blood cultures may detect bacteraemia and identify enteric gram-negative bacilli, anaerobes or enterococci which are the organisms most commonly involved. Plain abdominal x-rays may show a soft tissue mass, localized gas bubbles or an extraluminal air-fluid level. The chest x-ray provides indirect evidence of a subphrenic abscess with unilateral elevation and splinting of the diaphragm, pleural effusion and persistent basal atelectasis. Barium studies of the gastro-intestinal tract may show organ displacement due to fluid collections or a spiculated mucosal pattern characteristic of surrounding inflammation and

occasionally a sinus tract or extravasation of contrast from the bowel.

Radionuclide scintigraphy using gallium or indium are two isotopes potentially useful for imaging nonvisceral inflammatory processes. Gallium binds to lactoferrin, a protein present in neutrophils, after an intravenous injection, and accumulates at an inflammatory focus. Indium is lipophilic and binds to leucocyte cell protein. Indium-labeled cells migrate to an infective site where the isotope can be detected by a gamma camera. While both isotope studies have applicability in selected patients, their nonspecific and limited anatomic information has been superseded by more specific and anatomically accurate radiological evaluation.

Ultrasonography and computerized tomography are currently the most useful investigations for the identification and localization of intraperitoneal abscesses. Ultrasonography offers a rapid, non-invasive method of detecting intra-abdominal abscesses without the risk of radiation exposure. Considerable operator skill is required for effective use of the technique in identifying small abscesses. The major technical limitations of ultrasound examination are poor penetration through bone and distended gas-filled bowel making evaluation of deeper structures inadequate. Potential practical scanning difficulties which may limit the skin area required for adequate transducer contact include large open wounds, recent incisions, bulky dressings, stomas and external appliances. The majority of abscesses appear as well-defined echo-free cavities that readily transmit ultrasound waves, although some abscesses have internal echoes which represent debris within the cavity. Although real-time ultrasound scanning allows visualisation of peristalsis and vascular pulsation within the abdomen, fluid-filled masses

including abscess cavities, haematomas and loculated ascites may be difficult to differentiate. A logical extension now in general use is percutaneous ultrasound-guided aspiration of intraperitoneal collections with a fine 22-gauge needle allowing fluid to be retrieved for diagnosis and culture.

Computerized tomography (CT) currently provides the highest resolution with accurate anatomic localization and is the most useful modality for demonstrating multiple collections within the abdominal cavity. Oral contrast material, given prior to the procedure may facilitate identification of the fluid collections. Intravenous contrast may be added to produce rim enhancement of a mass which is a characteristic feature of an abscess. A technical limitation of CT is the inability to fully differentiate haematoma from an abscess since the densities may be similar. In addition, a reduction in quality of the CT image may be produced by metallic surgical clips in the postoperative patient. Despite the use of a battery of diagnostic tests, the occasional patient with evidence of sepsis, persistent abdominal pain, leucocytosis and fever may not reveal the site, and in such patients, exploratory laparotomy still serves an important role.

Treatment

The initial resuscitation of patients with intraperitoneal abscess is similar to patients with diffuse peritonitis. Diagnosis and treatment may be delayed because of subtle clinical manifestations. Overt septicaemia, nutritional deficiency, fistulae and extra-abdominal complications including respiratory or renal failure may be present and require specific attention.

Antibiotic therapy should cover the same spectrum as generalised peritonitis and should include both aerobic and anaerobic organisms.

The operative management of intraperitoneal abscesses requires:

- effective drainage of the abscess.
- correction of the underlying cause when appropriate
- careful exploration to exclude other abscesses within the peritoneal cavity

In patients with a single accessible well-localized abscess, simple drainage through an extraperitoneal approach may be adequate. Localized appendiceal abscesses may be drained through a flank incision without contaminating the peritoneal cavity. Well-localised pelvic abscesses can be drained through the vagina and rectum. A transperitoneal approach should be used for patients who have multiple abscesses and require effective evacuation of all septic material, debridement of necrotic tissue and closure with drainage at the site of a gastrointestinal leak. The operation is best performed through a midline incision similar to diffuse peritonitis. The choice between the small extraperitoneal operation and the larger transperitoneal procedure requires individualisation and careful clinical judgement and no specific rules can be made.

The advent of percutaneous CT-aspiration and drainage has modified the various operative considerations: this technique, however, requires careful selection of suitable patients and definite localisation by imaging of the abscesses. A drainage tract that will not transverse adjacent bowel is important. The precautions in selecting patients for percutaneous drainage are:

- the presence of single non-loculated abscess cavity
- a drainage route that does not traverse bowel, uncontaminated viscera, sterile peritoneum or pleura
- no perforated viscus is present and

- highly viscous pus or thick necrotic debris is not present

Percutaneous abscess drainage can be employed in critically ill patients as a temporising measure permitting definitive surgical treatment at a later stage. Despite failures and complications which include fistulae, bacteraemia, bleeding and peritonitis, percutaneous drainage of abscesses has become an important addition to the therapeutic armamentarium of intraperitoneal abscesses in selected patients.

Despite recent advances in surgery and intensive care, mortality in patients with intra-abdominal sepsis

remains distressingly high. Assessment of the variety of published treatment regimens is hampered by the uncontrolled nature of many of the studies. Objective validated scoring systems such as the sepsis score, the APACHE II Score and the surgical infection stratification system have been devised to aid audit and comparison between groups of patients.

The ultimate prognosis in patients with intraperitoneal abscesses is influenced by risk factors which include the presence of multiple, recurrent or lesser sac abscesses, positive blood cultures, multiple organ failure and age over 60 years

Table 1: Aetiology of secondary bacterial peritonitis

1. **INFLAMMATORY**
 - a. Intestine
 - i) Diverticulitis with perforation
 - ii) Appendicitis
 - iii) Meckel's diverticulitis
 - iv) Necrotizing enterocolitis
 - v) Ulcerative colitis
 - vi) Amoebic colitis
 - b. Visceral
 - i) Salpingitis
 - ii) Empyema of gallbladder
 - iii) Necrotizing pancreatitis
 - iv) Liver abscess
2. **STRANGULATION OBSTRUCTION**
 - a. Small bowel
 - i) Closed loop adhesive obstruction
 - ii) Hernia: internal, external
 - iii) Volvulus with strangulation
 - iv) Intussusception
 - b. Colon
 - i) Volvulus: sigmoid, caecal
 - ii) Closed loop obstruction (neoplasm, diverticulitis)
3. **TRAUMA**
 - a. Blunt rupture of viscus: stomach, small bowel, colon
 - b. Penetrating
 - c. Iatrogenic
 - i) endoscopy/biopsy
 - ii) anastomotic disruption
4. **PERFORATION**
 - a. Neoplasms: gastric, colon carcinoma
 - b. Foreign body
 - c. Duodenal, gastric ulcer
5. **VASCULAR**
 - a. Mesenteric embolus
 - b. Ischaemic colitis