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Intra-abdominal sepsis—Epidemiology, aetiology and management

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ABSTRACT

Peritonitis is a progressive disease leading inexorably from local peritoneal irritation to overwhelming sepsis and death unless this trajectory is interrupted by timely and effective therapy. In children peritonitis is usually secondary to intraperitoneal disease, the nature of which varies around the world. In rich countries, appendicitis is the principal cause whilst in poor countries diseases such as typhoid must be considered in the differential diagnosis. Where resources are limited, the clinical diagnosis of peritonitis mandates laparotomy for diagnosis and source control. In regions with unlimited resources, radiological investigation, ultrasound, CT scan or MRI may be used to select patients for non-operative management. For patients with appendicitis, laparoscopic surgery has achieved results comparable to open operation; however, in many centres open operation remains the standard. In complicated peritonitis "damage control surgery" may be appropriate wherein source control is undertaken as an emergency with definitive repair or reconstruction awaiting improvement in the patient's general condition. Awareness of abdominal compartment syndrome is essential. Primary peritonitis in rich countries is seen in high-risk groups, such as steroid-dependent nephrotic syndrome patients, whilst in poor countries the at-risk population is less well defined and the diagnosis is often made at surgery.

Introduction

Peritoneal inflammation is the final common pathway of a myriad primary intra-abdominal insults. Both the primary disease and the peritoneal inflammation have sequelae outside the peritoneal cavity and result in systemic illness. Whilst peritonitis may be generalised or localised, acute or chronic, primary or secondary and bacterial or non-bacterial, the most common is acute bacterial peritonitis secondary to some primary intra-abdominal disease.

In small children, the lack of omental fat inhibits the omentum's function as a barrier to the spread of an infected effusion, and generalised peritonitis is more commonly seen in children than in adults. Omental fat deposits increase with age having important metabolic sequelae, which may presage adult obesity.[1]

Whilst local inflammation may result in a leucocyte-rich effusion into the peritoneal cavity[2] with few constitutional symptoms other than pain, fever, and anorexia, the absorption of bacteria and toxins via sub-epithelial lymphatics[3] promotes the systemic manifestations of the disease ranging from SIRS through MODS, to septic shock and ultimately death. Mortality rates are clearly influenced by the nature of the primary insult and the severity of systemic disease at presentation. Measuring the severity of the local and systemic disease is not easy but is essential if comparison of outcomes is to be rational and one approach is to use the disordered physiology that the disease process has caused as a surrogate for disease severity. As no custom designed scoring system exists, modification of the Apache II score to suit children is a reasonable compromise.[4]

Peritonitis

The peritoneum has traditionally been thought of as little more than a lubricated semi-permeable serosal membrane that prevents abdominal organs from snagging during movement and provides a large surface area for peritoneal dialysis. It is now realized that the peritoneum has important mechanical, immunological and synthetic functions that are essential to homoeostasis[5] and that the omentum is an important immunological and endocrine organ.[6]

The cellular response to any intraperitoneal insult requires the co-ordinated efforts of both peritoneal macrophages and the mesothelial cell,[2] and the clinical picture to a large extent depends upon the success of localisation, primarily an omental function[6]; the nature and magnitude of the insult and general patient factors, such as nutritional status, age and co-morbidities.

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With the exception of chronic peritonitis which is seen in patients undergoing continuous ambulatory peritoneal dialysis, and in some patients with tuberculosis, the clinical picture is of an "acute abdomen" with attendant abdominal pain, guarding and rebound tenderness over the affected region, varying degrees of toxaemia and signs of intestinal paralysis.

However, in acute peritonitis the peritoneal effusion rich in macrophages, complements, immunoglobulins and cytokines[7] may significantly deplete intravascular volume. Additionally, fluid will accumulate within static loops of bowel exacerbating the fluid deficit. Either the primary insult or translocation of organisms that have thrived in static bowel loops, across an impaired intestinal mucosa, result in endotoxaemia and septicaemia with attendant multi-organ dysfunction.

Epidemiology

The primary diseases that lead to peritonitis differ from region to region around the world and also differ according to the age of the patient. Amongst older children in rich countries, appendicitis is by far the most common cause, reaching 82% of patients with acute abdominal symptoms in America[8] and 92% in Israel,[9] whilst necrotising enterocolitis (NEC) predominates in neonates.[10] In Africa, NEC is still a common neonatal problem,[11] but in half of older children with peritonitis the primary cause is perforation of the ileum due to typhoid.[12] Appendicitis is frequent, and perhaps increasing in frequency in Africa,[13] but remains outweighed by typhoid, ascariasis, trauma, intussusception and primary peritonitis. In India whilst appendicitis is the most common single pathology, diseases such as typhoid, tuberculosis and ascariasis are prominent.[14]

In some parts of the world, unusual pathologies predominate, for example, pigbel in the Highlands of Papua New Guinea[15] and this informs local investigational, preventative and therapeutic algorithms.

Whilst the aetiology of acute bacterial peritonitis varies around the world so do morbidity and mortality. In rich countries, the overall mortality from appendicitis in children hovers around 0.04% and in the age group 9–19 years is around 0.006%.[16] In Nigeria the reported mortality is 0.9%,[17] and in a South African series which contained both adults and children, the mortality was 2%.[18] These high mortality rates relate to delayed presentation and systemic sepsis syndromes related to difficulties in accessing health care structures as well as a lack of resources including, inter alia, access to intensive care.[19] Even within rich countries, there are socio-economic and ethnic differences in the complications and mortality of appendicitis, again reflecting difficulties in accessing surgical care.[20]

The mortality rate from peritonitis due to typhoid ileal perforation similarly varies but is high everywhere, ranging from 23% in Tanzania[21] to 13% in Nigeria.[22] Many centres report an improvement in mortality rate over time but identify late presentation as the principal cause of treatment failure.[23,24] Whilst the recognition of the importance of pre-operative resuscitation and the evolution of surgical techniques may have contributed greatly to the improved outlook for typhoid patients, mortality is likely to remain high until this element is effectively addressed.

Chronic peritonitis is seen frequently in children undergoing continuing ambulatory peritoneal dialysis (CAPD)[25] and is commonly seen in children with abdominal tuberculosis. The latter group has increased in regions severely affected by the HIV pandemic.[26] Tuberculous peritonitis may be one component of widespread extrapulmonary tuberculosis or may arise de novo.[27] Clinical clues to the diagnosis of tuberculous peritonitis may be found by inspection of the umbilicus,[28] but tuberculosis should be suspected in any patient with ascites, abdominal discomfort and weight loss.

Primary peritonitis in rich countries is seen in children with cirrhosis or nephrotic syndrome who are being treated with steroids,[29] and patients in this clinical situation can confidently be initially treated non-operatively. In poor countries, the condition is usually seen in otherwise well children, making it more difficult to suspect and diagnose.

The migration of people from developing countries to Europe and America suggests that it would be unwise for surgeons in the developed world to ignore patterns of disease seen in poor countries.

Principles of management of peritonitis in children

Acute peritonitis is a potentially lethal condition and should always be treated as an emergency. In children, the *suspicion* of peritonitis mandates close observation and an early decision on intervention.

The diagnosis of peritonitis is a clinical responsibility based on the findings of abdominal pain and tenderness, toxaemia with resultant tachycardia and pyrexia, frequently an associated ileus and evidence of a fluid deficit. Laboratory tests such as C-reactive protein, pro-calcitonin, serum lactate[30] and radiological studies may be indicative of inflammatory or ischaemic pathology, and may direct resuscitation efforts, but this remains a clinical responsibility. Peritonitis is an evolving pathology that worsens with time,[31] and awareness of the inevitable progression forms the rationale for a policy of frequent repeated observations by the same observer when critical decisions, such as the need for surgery, must be made.

Recognition that the removal of a normal appendix in adults in whom a clinical diagnosis of appendicitis had been entertained is associated with a higher mortality than ruptured appendicitis[16] has driven the expansion of radiological investigation of patients presenting with abdominal pain. It must be remembered that in many patients investigation is neither necessary nor desirable as clinical signs and symptoms are diagnostic. Scoring systems are of little practical help[32] but may be used to select patients for further investigation, particularly those who present with non-diagnostic scores on either the Alvarado or Samuel's scoring system.[33] Ultrasound is the most frequently used aid with a reported accuracy of 71–97%[34] but is limited by being operator dependent and not freely available. Computerised tomography is even less widely available but has a reported diagnostic accuracy of 93–98%,[34] although it is potentially hazardous in children. Each CT scan exposes the patient to approximately the equivalent of 250 chest radiographs[35] and whilst modern scan protocols might reduce this exposure,[36] formal clinical practice guidelines can make up to 80% of the investigations unnecessary.[37] Magnetic resonance imaging has been proposed for children and pregnant women but is no more able to differentiate simple from complicated appendicitis than ultrasound.[38] Of course in most parts of the world, none of these modalities are available and reliance must be placed on clinical findings and serial review of patients in whom there is diagnostic doubt. Clinical diagnosis in most centres has an accuracy of between 85% and 95%.[39]

The neonate can be very difficult to assess particularly in an ICU situation where mechanical ventilation, paralysis or deep sedation may impact the precision of clinical diagnosis. Trends in levels of inflammatory markers such as pro-calcitonin or C-reactive protein may assist in reaching a decision,[40] but under such circumstances it is often expedient to open the abdomen to confirm or refute the diagnosis. Awaiting objective signs such as abdominal wall