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Review

The challenge of intra-abdominal sepsis

Elroy Patrick Weledji*, Marcelin Ngowe Ngowe

Department of Clinical Sciences, Faculty of Health Sciences, University of Buea, Cameroon

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ABSTRACT

An overview of intra-abdominal sepsis is necessary at this time with new experimental studies, scoring systems and audits on management outcomes. The understanding of the pathophysiology of the peritoneum in the manifestation of surgical sepsis and the knowledge of the source of pathogenic organisms which reach the peritoneal cavity are crucial in the prevention of intra-abdominal infection. Inter-individual variation in the pattern of mediator release and of end-organ responsiveness may play a significant role in determining the initial physiological response to major sepsis and this in turn may be a key determinant of outcome. The ability to identify the presence of peritoneal inflammation probably has the greatest influence on the final surgical decision. The prevention of the progression of sepsis is by early goal-directed therapy and source control. Recent advances in interventional techniques for peritonitis have significantly reduced the morbidity and mortality of physiologically severe complicated abdominal infection. In the critically ill patients there is some evidence that the prevention of gut mucosal acidosis improves outcome.

The **aim** of this review is to ascertain why intra-abdominal sepsis remains a major clinical challenge and how a better understanding of the pathophysiology may enable its prevention and better management.

Method: Electronic searches of the medline (PubMed) database, Cochrane library, and science citation index were performed to identify original published studies on intra-abdominal sepsis and the current management. Relevant articles were searched from relevant chapters in specialized texts and all included.

1. Introduction

Intra-abdominal sepsis is one of the most challenging situations in surgery and usually presents as peritonitis.[1–5] Gastrointestinal perforation, with leakage of alimentary contents into the peritoneal cavity, is a common surgical emergency and may have life-threatening sequelae. The mortality of perforated viscus increases with delay in diagnosis and management.[1–3,9–12] The recently reported 12-fold variation in the 30-day mortality rate following emergency abdominal surgery in Britain ranged from 3.6% in the best performing hospital to 41.7% in the worst.[5] This would be alarming in the developing world where an overall mortality rate of less than 17% is reported.[12] This shows that surgical outcome depends on a complex interaction of many factors and the success obtained with the early onset of specific therapeutic procedures. Mortality is lower when operations are conducted by consultant anaesthetists and surgeons rather than trainees and, where patients have ready access to treatment in intensive care following surgery.[9–11] The 'surgeon factor' i.e. decision making on surgical management of the acute abdomen is a critical determinant of outcome.[5] 'Patient factor' is also important as most patients are over 65 with co-morbidity and often seriously ill with internal haemorrhage or a bowel perforation.[5,13,14] Perhaps the variation in surgical management outcome may also be partly explained by the demography and health of the local population.[5,12] A better understanding about susceptibility to infections (patient factor) will explain why a patient with minimal bacterial contamination at surgery may develop a pelvic abscess whereas another patient with massive faecal contamination after stercoral perforation of the colon may not develop infective complications. Inter-individual variation in the pattern of mediator release and of end-organ responsiveness may play a significant role in determining the initial physiological response to major sepsis and this in turn may be a key determinant of outcome.[1–5] Other key determinants of outcome are the initial severity of infection e.g. colonic perforation, the timeliness and adequacy of attempts at treatment and the

* Corresponding author. Department of Clinical Sciences, Faculty of Health Sciences, University of Buea & Department of Surgery, Regional Hospital, Buea, S.W. Region, Cameroon. Tel: +237 99 92 21 44.
E-mail address: elroypat@yahoo.co.uk (E.P. Weledji).

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patient's general health and consequent ability to withstand the process.[9–16] Surgical peritonitis may emanate from perforation, ischaemia (mesenteric or strangulation), pancreatitis and anastomotic leakage.[11] Intra-abdominal abscesses may also occur within an intra-abdominal organ. These include pyogenic abscess in the liver from portal pyaemia when in a septicaemia organisms and neutrophil polymorphs embolize to the liver e.g. following appendicitis or a perforation (now fortunately rare because of the use of antibiotics); pancreas from acute pancreatitis, and in the fallopian tube (pyosalpinx) following adhesions in the fimbrae from an ascending infection.[19] Infections above an obstructing calculi may include an empyema of the gallbladder or in the renal pelvis. A complicated abdominal infection extends beyond the hollow viscus of origin into the peritoneal space and is associated either with peritonitis or abscess formation.

Acute appendicitis is the most common surgical emergency with a life time risk of ~7%, and it is one of a relatively dwindling number of conditions in which a decision to operate may be based solely on clinical findings.[10] There are undoubtedly specific features associated with all acute abdominal conditions which are well established. The aim of both the history and examination is to determine a diagnosis and clinical decision. It remains the ability to identify the presence of peritoneal inflammation which probably has the greatest influence on the final surgical decision.[7,18] Regular re-assessment of patients and making use of the investigative options available will meet the standard of care expected by patients with acute abdominal pain.[7]

2. Pathophysiology of sepsis

Sepsis is an evolving process. It is the systemic inflammatory response to infection frequently associated with hypoperfusion followed by tissue injury and organ failure. Therefore, its sequelae reflect increasing severity of the systemic response to infection and *not* severity of infection.[1–3] Infection is enhanced by the synergy between aerobes e.g. *Escherischia coli* which reduce oxygen content and facilitates growth of obligate anaerobes e.g. *Bacteroides fragilis*, and by the presence of adjuvant substances e.g. faeces, bile or urine.[25]

There is a *balance* between excessive and inadequate responses to infection. Some production of mediators is needed to combat infection but an excessive or prolonged activation of such cellular/humoral mediator pathways is thought to contribute to the development of multiple organ failure (MOF) in patients with major sepsis.[1–3,42] Mortality increases with the degree of the systemic inflammatory response syndrome (SIRS). The mortality following a *bacteraemia* is ~5%, *sepsis* (infection + SIRS) ~15%, *septic shock* (sepsis + hypotension (systolic BP < 90 mmHg) ~50%, *severe SIRS* ~80%, *multiple organ failure* (MOF) ~90%.[1–3] SIRS is a massive systemic response comprising an evolution of a cytokine cascade (TNF, IL-1, IL-6, IL-8), and a sustained activation of the reticuloendothelial system. It finally leads to the elaboration of secondary inflammatory mediators causing cell damage. These mediators include arachidonic metabolites (prostaglandins and leukotrienes), nitric oxide (vasodilator), oxygen free radicals, platelet activating factor causing increase platelet deposition, vasodilatation, increase capillary permeability and activation of coagulation pathways which results in end-organ dysfunction by formation of microthrombi. When three or more systems have failed the ensuing mortality approaches 80–100% and once one organ system has failed, others typically follow (organ failure amplification).[5] Thus it is important to strive to support as far as possible each organ system to avoid each further adverse event (e.g. ventilation, haemofiltration/haemodialysis, inotropic support, use of blood products).

Organs vary in their ability to maintain their own perfusion (through autoregulation) and generally, measurements relate the total body picture, rather than adequacy of perfusion of specific viscera. Renal failure is common in MOF and often established during the early stages. Renal function will return when perfusion and oxygenation are adequate and until this occurs, renal replacement therapy is needed.[1–4,23] Certain organs, notably the gut, are more prone to covert hypovolaemia and the consequent hypoxia may continue to drive the inflammatory process (including multiple organ failure) probably via bacteria translocation even when the initial causal factors are dealt with.[1] To overcome this, one approach has been to try and ensure that the critically ill patient with MOF has a circulation which provides an oxygen delivery greater than normal, thus minimizing the chance of occult hypoxia. A related approach has been to monitor plasma lactate and/or negative base excess as elevated values suggest that tissue hypoxia may be present.[2,3] An alternative strategy is to try and measure specific visceral perfusion in suspect viscera such as gut mucosa pH tonometry particularly in the preoperative preparation of the critically ill surgical patient.[4]

2.1. The Mannheim peritonitis index score

Many scoring systems have been created for assessing patient risks of death during an event of peritonitis. The *Mannheim peritonitis Index* (MPI) is a reliable predictor of the peritonitis outcome as the increase of MPI scores is proportional to that of morbidity and mortality. MPI adverse factors include presence of organ failure, time elapsed >24 h, presence of malignancy, origin of sepsis, the presence of faecal peritonitis and generalized peritonitis.[12,13]

3. Early goal-directed therapy

It is not possible to practice fully the ideal management of early diagnosis and surgery for the acute abdomen, thus reducing morbidity and mortality to zero, because patients and the disease are variable. However, because *infection*, *inadequate tissue perfusion* and *a persistent inflammatory state* are the most important risk factors for development of multiple organ failure it seems logical that initial therapeutic efforts should be directed at their early treatment or prevention (early goal-directed therapy).[20,21]

Early initiation of broad spectrum antibiotics has been shown to be critical during the SIRS phase for prevention of sepsis and septic shock. It has been shown clearly that patient mortality is significantly lower when appropriate antibiotics are prescribed early in the course of the patient's illness.[24] It is also important to appreciate that fungi and atypical organisms can contribute to the sepsis syndrome, and to take cultures and prescribe appropriately.[17] Prolonged 'prophylaxis' is detrimental as superinfection by fungi, antibiotic-resistant *Pseudomonas, Enterococci* and *Staphylococci* is encouraged.[17,41] These infections carry a high mortality and are difficult to treat. Enteric streptococci account for 10–20% of severe infections related to the abdomen and are not sensitive to all common prophylactic antibiotics.[15–17]

Early goal-directed resuscitation during the first 6 h after recognition of shock has moved towards the use of whole blood as it appears to eliminate the problems of expansion of extravascular volume and marked fluid retention seen with crystalloid on a background of leaky capillaries, and also appears to provide a lower incidence of organ failure.[20]

Adequate replacement of fluid loss via oesophageal doppler monitoring of left atrial filling, stroke volume and aortic flow (goal-directed fluid management) avoids the complications of fluid overloading or under-filling and thus favour faster recovery.[42–44] Critically ill patients who are either physiologically unstable or at high risk of failed source control especially following septic shock

where resuscitation with crystalloid will likely lead to an abdominal compartment syndrome may benefit from a laparostomy.[34,35]

"Source control" defined as any procedure, or series of procedures that eliminates infectious foci, controls factors that promote on-going infection and corrects or controls anatomic derangements to restore normal physiologic function.[21,22] Source control failure is more likely in patients with delayed (>24 h) procedural intervention, higher severity of illness (Acute physiology and chronic health evaluation score or APACHE > 15), advanced age (>70 years), co-morbidity, poor nutritional status, and a higher degree of peritoneal involvement (i.e. a high MPI score), and is heralded by persistent or recurrent intra-abdominal infection, anastomotic failure, or fistula formation.[12,13,16,21,22] Untreated, colonic perforation with faecal peritonitis is rapidly fatal – death results from septicaemia and multiple organ failure caused by the absorption of pathogenic bacteria load and their toxins from the peritoneal cavity.[3,5]

3.1. Implications on surgical management

Localised peritonitis occurs because peritoneal resistance to infection relies upon localization rather than dispersal of a contaminant.[28,29] The inhibition of peritoneal fibrinolysis permits stabilization of fibrinous exudates and limits the spread of infection. The omentum 'abdominal policeman' and the intraperitoneal viscera also have a remarkable ability to confine infection as seen for example in acute appendicitis, perforated duodenal ulcer/diverticular disease.[29–31] Thus, localised peritonitis implies either contained or early perforation of a viscus or inflammation of an organ in contact with anterior parietal peritoneum. For instance, a palpable mass in the right iliac fossa represents either an inflamed mass of adherent omentum, appendix and adjacent viscera, or an abscess. Conservative treatment with later drainage of any abscess had been the standard and diffuse peritonitis was usually fatal.[30,31] Surgery for appendicitis evolved when the mortality associated with perforated appendicitis was high. Although only a few patients progressed to the potentially lethal complications, early surgery for all patients with suspected appendicitis became the definitive method of preventing severe peritoneal sepsis.[10] The prognosis after appendicectomy is excellent.[30] An appendix mass is often detected only after the patient has been anaesthesized and paralysed. Thus, the differentiation of a phlegmonous mass from an abscess is not a practical problem because surgery is the correct management for both. Such a policy renders any debate on interval appendicectomy redundant. Operation during the first admission is expeditious and safe, provided steps are taken to minimize postoperative sepsis. The consequences of missing a carcinoma in the elderly patient or other pathology including ileo–caecal TB, lymphoma etc are also abolished.[10]

Generalised peritonitis will occur when there is failure of localization. Failure of localization may arise for the following reasons: a) a rapid contamination that does not permit localization as in a perforated colon/anastomotic leak, b) persistent or repeated contamination that overwhelms an attempt to overcome it, c) a localized abscess that continues to expand and ruptures into the peritoneal cavity (e.g. appendix, diverticular abscess).[26,30,31] The peritoneal cavity becomes acutely inflamed with production of an inflammatory exudate which spreads through the peritoneum leading to intestinal dilatation and paralytic ileus. Early definitive primary or re-operative surgery leading to the removal of necrotic tissue, the drainage of abscesses, and the control of peritoneal soilage (source control) may be effective in the intra-abdominally septic patient.[12,21,49] Ongoing intestinal ischaemia with doubt about intestinal viability is best managed by exteriorizing the bowel ends after resection of the ischaemic bowel and a second-look laparotomy undertaken 24-48 h later.[9] Diffuse peritonitis from perforated appendicitis, which has been diagnosed preoperatively, should be dealt with by formal laparotomy, rather than by making a gridiron incision, to allow thorough peritoneal toilet and lavage.[30]

3.1.1. Acute colonic perforations

It may be difficult to reach the correct pre-operative diagnosis in many patients with colonic perforations. As patients often present with generalized peritonitis, the diagnosis is being established only at laparotomy.[7,9] There may be clues in the history e.g. known diverticular disease, ulcerative colitis, Crohn's disease, collagen disorder; a recently altered bowel habit or rectal bleeding suggesting carcinoma, or there may be a short history of severe diarrhoea in patients with infective colitis.[11] Patients with faecal peritonitis are more severely ill with signs of septic shock than those with a purulent peritonitis from a small perforation.[2,3] As the most common operative finding in patients with a colonic perforation is a perforated inflammatory mass in the sigmoid colon, differentiation between diverticular disease and carcinoma may be difficult.[9] Current opinion favours resection of the inflammatory phlegmon and its perforation, with or without primary anastomosis.[11] The former policy of peritoneal drainage, construction of a proximal defunctioning stoma and subsequent colonic resection at a second operation carries a postoperative mortality of 30%.[9,13] The stoma does not protect against continued faecal contamination from the perforated segment. Thus, the importance of 'source control' of sepsis.[19] Many surgeons favour a Hartmann's procedure with excision of the diseased bowel, construction of an end colostomy and closure of the rectum, or alternatively exteriorization as a mucous fistula.[26] Reversal of Hartmann's is a difficult procedure however with increased complications including anastomotic leakage. Thus 30–50% of Hartmann's procedures are never actually reversed.[26] The recent systematic review comparing outcomes following primary resection and anastomosis (PRA) and Hartmann's procedure in emergency surgery for acute diverticulitis confirms the above observations. The mortality after PRA was 7.4% and Hartmann's 15.6% and these results have not improved over the intervening 25 yrs.[27] Patients with a toxic megacolon as a complication of inflammatory bowel disease or infective colitis require a subtotal colectomy with preservation of the rectal stump and formation of a terminal ileostomy. This allows the option of construction of an ileo-anal pouch once the sepsis has resolved.[9,11] However, light clothing, hot climate, high residue diet (vegetables) and poor availability of appliances all make the management of an ileostomy more difficult in the tropics and so ileorectal anastomosis is preferred unless the rectum is extensively diseased with stricture formation.[8] Sepsis also induces high ileostomy output.[36,37] Careful resuscitation in order to avoid congestive heart failure from the toxic myocarditis of typhoid and prompt surgical intervention has reduced the mortality rate from typhoid perforation from 50% to 20%. The perforated ulcer is excised by wedge excision, a single area of diseased bowel may be resected or, in the very ill patient, exteriorization of the small bowel may be the best procedure.[8]

3.1.2. Is there a role for abdominal drains?

It is generally futile to attempt to drain an anastomosis or the general peritoneal cavity as an enterocutaneous fistula may ensue.[46] The evidence is that drains may cause more problems than they solve if they are placed 'just in case' of a leak. The adhesions that occur in the healing process of the anastomosis or general peritoneal cavity will attract the peritoneal drain (foreign body) which may physically damage the anastomosis or small bowel. Secondly, the anastomosis needs to gain some extra blood supply, which it does by forming adhesions to adjacent vascular structures.

If a piece of corrugated plastic is placed beside an anastomosis it will be unable to do this and a leak will be encouraged. The only exceptions to this are where the anastomosis is not watertight, such as with bile and urine, and a collection will interfere with healing. Most surgeons are wary of the potential danger suction may do to an anastomosis. Redivac drains are deliberately not placed in the vicinity of anastomoses and are removed after 48 h.[47] Drains can indeed mislead the surgeon as they easily get blocked. It is preferable for an anastomotic leak to reveal itself so that it can be managed accordingly. If there is no drain you can tell if an anastomosis has leaked by clinical signs backed by a water-soluble contrast study – the definitive investigation to determine if there is a leak.[32,33] Vigilance in the post-operative period is the key, and to remember that anastomotic failure can occur. Large bore drains are useful in sepsis following inadequate peritoneal lavage or residual sepsis. These should be placed in the appropriate dependent areas of the abdominal cavity such as the paracolic gutters, pelvis and subphrenic spaces away from the intestine.

4. Post operative sepsis

Postoperative sepsis is usually caused by a failure to eradicate infection at the original laparotomy or anastomotic breakdown and can be extremely difficult to diagnose.

4.1. Anastomotic leak

Interestingly, postoperative mortality from post operative sepsis due to anastomotic leak is higher than any natural condition.[32] The mortality rate of individuals who developed an anastomotic disruption was 39.3%, and anastomotic leak was found to be an independent predictor of mortality.[15] This may be due to the fact that sepsis is the leading cause of death following an anastomotic leak and corroborated by the fact that delayed diagnosis worsens the prognosis. The acute onset of abdominal pain and generalized peritonitis is a serious manifestation of an anastomotic leak and, these patients may quickly progress to septic shock, requiring intensive care monitoring and resuscitation with fluids and inotropic agents.[1,2,40] Patients with diffuse peritonitis from an anastomotic leak or perforated viscus cannot be fully resuscitated until ongoing soiling has been controlled.[12,13,19] In such patients resuscitation should be continued intraoperatively (resuscitation surgery) with exteriorization of bowel ends as stomas.[11,26,34] Laparostomy as opposed to primary closure of abdominal fascia may be indicated if there is a risk of developing an abdominal compartment syndrome from severe sepsis and septic shock.[36] For haemodynamically stable patients without generalised peritonitis e.g. abscess, a delay of up to 24 h may be appropriate to allow further clinical assessment and image-guided minimally invasive interventional therapy.[22,23,39,40]

Intra-peritoneal abscess should be suspected in any patient who develops signs of sepsis 7–10 days after a laparotomy.[7] CT or Ultrasound – guided percutaneous drainage of abdominal abscesses via a pigtail catheter has emerged as the procedure of choice in many circumstances as morbidity and mortality is lower than following operative drainage.[38–40,49] Laparoscopy if available or laparotomy should be undertaken if doubt remains despite negative radiology. Operative drainage is necessary for those abdominal abscesses which are multiple, isolated but cannot safely be approached percutaneously, and/or are associated with systemic sepsis unresponsive to percutaneous drainage.[22,39] Occasionally, a pelvic abscess bulging into the rectum may be drained through it. The mortality from post operative intra-abdominal abscess is greater than 50% and the mortality increases with each operation to treat recurrent or persistent sepsis.[15,22] This is due to the deteriorating septic state of the patient superimposed on the stress of surgery and, the increased dissection required by re-operative surgery with increased risk of injury and ischaemia to tissues.[33,40] Therefore, the best opportunity to eradicate infection is the first operation.

4.2. How important is the intensive care unit (ICU)?

Recent studies show that some 30–40% of patients admitted to ICU received suboptimal care on the ward at some stage. Too many deaths or unplanned admissions to ICU occur because appropriate, thoughtful and early action was not taken.[10,11,16] Consideration should be given to early ICU admission for patients with significant co-morbidity, since ICU can then play a prophylactic role. Nevertheless, despite the poor prognosis of MOF with a 10% survival rate in ICU, for there to be any real prospect of recovery, the underlying cause or source of sepsis must be dealt with.[16,20–22,48] Determining whether or not an unstable patient on ICU has an abdominal process which requires intervention can be difficult as the usual signs may be absent due to sedation and paralysis. Distension, appearance of wounds, stomas, drain effluent and any systemic deterioration may give clues. Joint management (intensivist, surgeon and the radiologist) offers the best chance of success.[48,50] Further surgical intervention for an unstable patient with a recurrent problem e.g. further sepsis in patient with recent faecal peritonitis or a new complication e.g. acalculous cholecystitis in patient with a head injury can be hazardous yet vital.[50] Even in the former case, there will usually be alternative sources of sepsis (chest, urine, lines, etc.) which need quite different treatments with no less urgency. However, missed significant abdominal sepsis in a patient with organ failure is almost always fatal.[4,21–23] The aim should be to operate in a timely manner on as well prepared a patient as possible although it may not be possible to make the patient stable until the underlying cause is dealt with.[48,50]

4.2.1. Energy requirements

As sepsis potentiates the catabolic response in the critically ill patient and contributes very significantly to the clinical picture, nutritional support is frequently needed.[1,23,36,37,42] Although some of the exaggerated muscle protein breakdown might be due to the catabolic hormonal environment and insulin resistance, the release of cytokines e.g. IL-1, 6 and TNF may also play a part.[1,16] Energy requirements are highly variable and difficult to predict, especially in critically-ill ventilated patients. Historically, critically ill patients have been given large amounts of energy, often far in excess of requirements. This resulted in complications, particularly related to excessive lipid deposition within the liver. In critically ill patients, intolerance to glucose, may result in inability to meet these energy requirements if glucose is used as the principal calorie source. Glucose administration also leads to increased respiratory workload for critically ill patients due to production of more carbon dioxide and should be avoided as far as possible. These difficulties can be avoided by using lipid emulsions in total parenteral nutrition (TPN) and lipid-rich enteral feeding solutions. At least 50% of the energy requirements should be administered as lipid which further reduce the volume of feed required significantly.[37] Some glucose, however, is required in all feeding regimens to prevent ketosis. Overall, the effect of feeding the critically ill is to halve net protein catabolism. However, positive nitrogen balance (indicating repletion of visceral and muscle protein) is unlikely to be restored to septic patients unless the underlying cause of sepsis is dealt with.[23]

5. The dilemma

Despite modern surgical techniques and significant improvements in the peri-operative care of the surgical patient, the

colorectal anastomosis still has an anastomotic leak rate reported to range from 3% to 22%.[32,33] This wide variation indicates that the pathogenesis for leakage is probably multifactorial. Impaired microcirculation from life-style related factors such as smoking could be another reason.[45] However, the prevention of sepsis from adequate attention to technique, and correct surgical decision making from the findings at operation, (surgeon-related factor) remains the single most important factor that can influence the morbidity and mortality in bowel surgery.[11,15,26,32] Also, despite improvements in resuscitation techniques, antibiotic therapy and anaesthesia, the mortality associated with a perforated peptic ulcer has not changed over the last two decades. It remains around 25% almost certainly due to the fact that the age-mix of the disease has changed during this time with more elderly (female) patients with the increasing use of non-steroidal anti-inflammatory drugs (NSAIDS) presenting with perforated peptic ulcers and many of whom have serious concomitant medical illnesses (poor American society of anaesthesiologists score – ASA).[6] Similarly, the mortality of appendicitis is associated with the age of the patient and delayed diagnosis.[14,30] The overall mortality for appendicitis is less than 1%, but it rises to over 5% when perforation is present and most deaths occur in the elderly.[10,30] The most important prognostic factors in emergency colorectal surgery are again the preoperative status – age and faecal peritonitis. Together the mortality is greater than 60%.[26] Thus, peritoneal sepsis is seldom the sole cause of death, but compounds coincidental cardiovascular, respiratory or renal pathology. When severe SIRS is in progress, prognosis is poor and surgical intervention may be late as the cascade is fully in progress. Gut mucosal hypoperfusion as an early consequence of hypovolaemia may continue to drive the inflammatory process even when the initial causal factors are dealt with. Supportive treatment may be all that is required as there is as yet no known drug to abort this cascade.[3,5] Several multicentre randomized trials using monoclonal antibodies or antagonists to endotoxin, TNF and IL-1 as adjuvant to the established basic principles of management have not reduced mortality and it is now recognized that the redundancy in the inflammatory response is such that if one component is removed, another mediator will continue the response. Moreover, if the pool of endogenous antagonists (e.g. IL-1 receptor antagonist or soluble TNF receptors) is replete, addition of exogenous antagonists is unlikely to be efficacious.[4]

6. Conclusions

The understanding of the pathophysiology of the peritoneum in the manifestation of surgical sepsis, and knowledge of the source of pathogenic organisms which reach the peritoneal cavity are crucial in the prevention of intra-abdominal surgical infection. Inter-individual variation in the pattern of mediator release and of end-organ responsiveness may be a key determinant of outcome. The ability to clinically identify the presence of peritoneal inflammation probably has the greatest influence on the final surgical decision. The Mannheim peritonitis index score may assist surgeons in identifying and aggressively managing high risk patients so as to improve outcome. Early recognition, prompt resuscitation, early judicious and appropriate antibiotic treatment and, prompt diagnosis and eradication of the source of sepsis (i.e. early goal-directed therapy and source control) should abort the evolving process of sepsis. The prevention of gut mucosal acidosis in critically ill patients may also improve outcome. Established septic shock or multiple organ failure is thus really only treatable by prevention through attention to detail. Sepsis represents a major clinical challenge and their management continues to demand a multidisciplinary approach.

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Author contribution

Dr Elroy Patrick Weledji (FRCS) – Senior Lecturer, Anatomy & Surgery in the Department of Clinical Sciences is the main Author and Researcher.

Prof Marcelin Ngowe Ngowe – Professor of Surgery and Dean of the Faculty provided facilities and contributed ideas to the manuscript.

Conflict of interest

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