Review Article

Anesthetic management for patients with perforation peritonitis

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Abstract

Perforation peritonitis is a common surgical emergency. Anesthesia in patients with perforation peritonitis can be challenging. Delayed presentations, old age, hemodynamic instability, presence of sepsis and organ dysfunction are some of the predictors of poor outcome in such patients. Pre-operative optimization can reduce intraoperative and post-operative morbidity and mortality, but surgery should not be unnecessarily delayed. Intensive care in critical care settings may be essential.

Key words: Abdominal sepsis, perforation peritonitis, pre-operative care, post-operative period

Introduction

Perforation peritonitis is a frequently encountered surgical emergency in tropical countries like India, most commonly affecting young men in their prime of life.[1-4] Despite advances in surgical techniques, antimicrobial therapy and intensive care support, management of peritonitis continues to be highly demanding, difficult and complex. A majority of patients present late, with septicemia, thus increasing the incidence of morbidity and mortality thereby complicating the task of anesthesiologist in the perioperative period.

Epidemiology of Perforation Peritonitis

The common age for its occurrence has been reported to be 45-60 years,[3] the median age being 40.5 years in Asian community.[5] Majority of the patient's are male, with a male:female preponderance of 3:1. While, in eastern countries such as India and Pakistan, perforation of the proximal part of the gastrointestinal tract (GIT) is more common,[5] distal gut perforation is more common in the western population. Overall, duodenum is the most common site of perforation.[6]

The overall mortality rate of perforation peritonitis ranges from 6% to 36% depending upon the site and cause of perforation.[3] Mortality rates reported are as follows: Gastric perforation 36%, enteric perforation 17.7% and colorectal perforation 17.5%. Major causes of post-operative morbidity in such patients are respiratory complications such as pneumonia, atelactasis, pleural effusion; wound infection, septicemia and dyselectrolytemia.[3]

Etiology

Perforation of any portion of the gut within the abdominal cavity leads to peritonitis and intra-abdominal sepsis. Besides a perforated peptic ulcer, other important perforations include appendicular perforation, diverticular perforation, perforation of the small bowel in enteric fever and perforation of tuberculous ulcer. [Table 1] Corticosteroids, Non-steroidal anti-inflammatory drugs (NSAIDs) can induce perforation in any portion of GIT.[7] Occasionally, perforation with localized or generalized peritonitis can occur in inflammatory bowel disease. Gangrene of the bowel from strangulation and obstruction or from mesenteric vascular ischemia or occlusion are important causes of peritonitis. Rupture of an empyema of the gall bladder or a gangrenous cholecystitis, also causes generalized peritonitis.

Acute inflammatory disease within the pelvis initially causes pelvic peritonitis, which may progress to generalized peritonitis.

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Table 1: Common causes of perforation peritonitis

Perforated gastric or duodenal ulcer
Perforation of hollow viscus or any portion of the gut
Enteric ulcer
Tubercular ulcer
Inflammatory bowel disease
Diverticulitis
Iatrogenic (corticosteroids and NSAID use)
Penetrating trauma to abdomen

NSAID=Non-steroidal anti-inflammatory drug

In Indian females, septic abortion and puerperal sepsis are important causes of peritonitis. Acalculous cholecystitis and acute pancreatitis can complicate the course of critical illness and are due to poor perfusion in critically ill patients.

Pathophysiology

Many of the systemic as well as abdominal manifestations of peritonitis are mediated by cytokines as tumor necrosis factor (TNF), Interleukin-1, interferon-ϒ and others. Cytokines appear in the systemic circulation of patients and to a much greater extent in peritoneal exudate. The cytokines are produced by macrophages and other host cells in response to bacteria or bacterial products, such as endotoxin.[7-9]

The following changes are observed in peritonitis:

- Local inflammation of peritoneum
- Adynamic ileus
- Hypovolemia
- Changes in other organ systems
- Hormonal and metabolic changes related chiefly to the immediate and acute stress of infection
- The evolution of sepsis syndrome.

Local peritoneal inflammation

The local response to a peritoneal insult is characterized by hyperemia of the peritoneum with vascular congestion, edema and transudation of fluid from extracellular interstitial compartment in to the abdominal cavity. This transudation is accompanied by the diapedesis of polymorphonuclear leucocytes. It is followed by exudation of a protein rich exudate containing large quantities of fibrin and other plasma proteins. Clotting of this protein rich exudate result in sticking together of the bowel loops to the viscera and the parieties in the area of inflammation.[7,9]

Adynamic ileus

Initially, there is a short period of bowel hypermotility, motility is then depressed and is followed by complete adynamic ileus. The gut distends and is filled with fluid and swallowed air. Fluid secretion in to the gut is markedly enhanced, whilst absorption of the fluid from the gut is markedly impaired. There is therefore, sequestration of a large volume of fluid within the lumen of the gut.[9]

Hypovolemia

The loss and sequestration of fluid in the gut and in the peritoneal cavity may be marked and result in a fall in the volume of the interstitial tissue compartment. Fluid is also trapped as edema beneath the mesothelium of visceral peritoneal linings, adding thereby to the volume of water, electrolytes and proteins translocated into the "third space." The volume of sequestrated fluid may be as large as 4-6 liters in 24 h.[9]

Secondary changes in other organ systems

Cardiac response

Diminished venous return due to fall in circulatory volume leads to fall in cardiac output, hypotension, decrease in oxygen transport and poor oxygenation of tissues. This promotes metabolic acidosis, which in turn further depresses cardiac function.[10-12]

Renal changes

These are secondary to hypovolumia, fall in cardiac output and the effect of increased secretion of the anti diuretic hormone and aldosterone. Renal perfusion suffers and there is fall in glomerular filtration rate. Renal insufficiency develops and enhances metabolic acidosis.[13-15]

Respiratory changes

Abdominal distension due to ileus, together with restriction of diaphragmatic and intercostal movements due to pain results in a fall in tidal volume. This predisposes to atelectasis, which in turn results in ventilation perfusion mismatch and a fall in partial pressure of oxygen (PaO_2) in the blood.[16,17]

Hormonal and metabolic changes

Peritonitis causes an almost immediate response from the adrenal medulla. There is an outpouring of epinephrine and norepinephrine (NE) in to the blood resulting in vasoconstriction, tachycardia and sweating. There is also an increased secretion of adrenocortical hormones for the first 2-3 days following a peritoneal insult. Secretion of antidiuretic hormone and aldosterone is also increased causing a reduction in urine output with conservation of sodium and water. Water retention may exceed sodium retention, leading to hyponatremia.[18,19]

Metabolic rate is generally increased with a corresponding increase in oxygen demand by the tissues. The cardio-pulmonary system is unable to achieve delivery of oxygen to the tissues resulting in tissue hypoxemia and lactic acidosis. Protein catabolism is also increased and progressively becomes more severe. Weight loss of 25-30% of lean body mass is observed

if peritonitis persists. Serum albumin levels progressively falls as albumin accumulates in the peritoneal cavity and is lost to the general circulation.

Anesthetic Management

Pre-operative Assessment

History

Peritonitis presents as an acute abdomen. Abdominal pain is the most important symptom of generalized peritonitis. The pain is maximal over the initial site of inflammation; it then proceeds to involve the whole abdomen as generalized inflammation supervenes. Duration of pain is also important as a long standing pain (>24-48 h) can necessitate detection of features of sepsis and multi-organ dysfunction. Pain may be accompanied by nausea and vomiting.[7]

Examination

The examination should focus on the state of intravascular hydration, the presence of shock or multi-organ dysfunction and the adequacy of hemodynamic resuscitation. Systemic features include fever, tachycardia, tachypnea and leukocytosis. The abdomen may show distension because of ileus. Abdominal movements on respiration are poor; the patient can be tachypneic with shallow intercostal breathing. Tenderness on palpation or rebound tenderness with guarding and rigidity can be present due to peritoneal irritation and inflammation. Bowel sounds will be absent. Rectal examination and pelvic examination may reveal tenderness or a painful inflammatory mass.

Hypovolemic shock

Clinical features of hypovolemic shock such as hypotension, tachycardia and oliguria are often present in the first few days of acute generalized peritonitis. If the circulatory state is uncorrected and if prompt surgery for the peritonitis is delayed, the patient can deteriorate rapidly, which can prove fatal.[12] The Hippocratic facies- "hollow eyes, collapsed temple and brown, black, livid or lead colored face" is the description of the patient affected by generalized peritonitis.

Investigations

Investigations which are recommended in patients are complete blood count including platelet count, serum electrolytes, liver and kidney function tests, blood sugar and electrocardiogram. Systemic features of sepsis should warrant investigation like coagulation profile and blood gas analysis. Samples for blood, urine and peritoneal fluid culture should be sent before starting empiric antibiotic therapy. Imaging studies like X-ray Chest or abdomen in the upright position will reveal gas under the diaphragm. Paralytic ileus is characterized by marked distension of small gut. If the patient is too sick for an X-ray in the erect posture, then a left lateral decubitus X-ray of the abdomen is of help. It may show the presence of free air between the liver margin and the abdominal wall. Presence of free fluid and gas in the peritoneal cavity after perforation of gut is visible as a fluid collection with a clear horizontal air fluid level.[7]

Pre-operative predictors of mortality in adult patients with perforation peritonitis

Elective surgery in peritonitis patients have a favorable outcome as compared to emergency surgery.[20,21] [Table 2] Cohen[22] and Møller *et al*.[23] reported a high risk of mortality in persons over 60 years due to multiple pathological processes preceding concomitantly. Jhobta *et al*. and Afridi *et al*., have stressed that delayed presentation to hospital accounts for significant mortality.[3,5] Similarly Kocer *et al*. found that patients who were admitted after 24 h had a 3.4 times higher morbidity risk than patients admitted before 24 h.[24] The mannheim peritonitis index (MPI)[25] [Table 3] also consider the duration of peritonitis >24 has one of the factor contributing to mortality.[26] Svanes *et al*. have reported

Table 2: Pre-operative predictors of mortality

Age
Site of perforation
Duration of symptoms
Pre-operative shock
Hypoglycemia
Renal dysfunction
Serum lactate levels
Acidosis
Base excess
Delay in surgical treatment
Increased tumor necrosis factor
Increased procalcitonin level
Increased intramuscular gastric pH

Table 3: Manheim peritonitis index

Risk factors	Score
Age>50 years	5
Female sex	5
Organ failure*	7
Malignancy	4
Pre-operative duration of peritonitis>24 h	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudate	
Clear	0
Cloudy, purulent	6
Fecal	12

**Kidney failure: Creatinine level>177 mmol/l or Urea level>167mmol/L or Oliguria<20ml/h. Pulmonary insufficiency: PO_2<50 mm Hg or PCO_2>50 mm Hg. Intestinal obstruction/paralysis>24h or complete mechanical ileus, Shock hypodynamic or hyerdynamic*

that a delay of more than 24 h increases mortality by seven to eight fold, complication rate by three fold and length of hospital stay by two fold, compared with a delay of 6 h or less.[27] Hypoglycemia during hospitalization occurs in patients with and without diabetes and has been associated with increased in-hospital mortality.[28,29] Sepsis, starvation, malignancy and low serum albumin levels were risk factors for developing hypoglycemia. Depleted glycogen stores, impaired gluconeogenesis and increased peripheral utilization may all be contributing factors. Renal dysfunction is an important marker of mortality.[30] MPI score include serum creatinine levels as one of the marker. Møller *et al*. have reported renal insufficiency on admission as an independent risk factor related to mortality, in patients operated for peptic ulcer perforation.[29,30] Elevated blood lactate levels have been used to define the prognostic value of occult hypoperfusion in critically ill patients without signs of clinical shock. Vorwerk *et al*. reported a specificity of 74.3% for mortality with lactate levels of >4 mmol/L in patients with sepsis.[31] Mikkelsen *et al*. concluded that intermediate and high lactate levels are independently associated with mortality in severe sepsis, independent of organ failure and shock.[32] Low values of bicarbonate levels at admission are also associated with higher mortality in ICU.[33] Lee *et al*. .and Møller *et al*. ascertained metabolic acidosis as an independent predictor of mortality.[34-36] Increased Tumor necrosis factor (TNF)[37], procalcitonin levels[38], and intramucosal gastric pH[39] have also been used as markers of hypoperfusion resulting from sepsis.

Several scoring indices have been compiled to predict the prognosis of patients with sepsis [Table 4].[40] The Boey score encompasses three factors – major medical illness, preoperative shock and longstanding perforation >24 h. The mortality rate increases progressively with an increasing number of risk factors: 0, 10, 45 and 100% in patients with none, one, two and all the three risk factors respectively.[41]

The MPI has eight parameters and a score ranging from 0 to 57. Accuracy of MPI is comparable or slightly superior to that of other sepsis classification system.[42-44] Patients with a score of <21 have a reportedly low mortality (0-23%) as compared to those with a score >29 (100%). The MOF score considers various organ systems and is tedious to apply. Despite advances in intensive care and aggressive surgical techniques, recent studies suggest an operative mortality rate ranging from 10- 30%.[45]

Table 4: Scoring indices

Acute physiology and chronic health evaluation
Simplified acute physiology score
Boey score
Multi-organ failure score
Manheim peritonitis index

Management

Management consists of the following important features:

- Quick restoration of the circulatory hemodynamics followed promptly by surgery
- Use of appropriate antibiotics
- Critical Care and support of different organ systems
- Maintenance of nutrition.

Hemodynamic Resuscitation

The objective of pre-operative resuscitation is to rapidly restore adequate oxygen delivery to peripheral tissues. Most patients are hypovolemic from the massive sequestration of fluid into the peritoneum and into the lumen of gut. In high surgical or trauma patients with sepsis, early hemodynamic optimization before development of organ failure reduced mortality by 23% in comparison with those who were optimized after development of organ failure.[46]

In hemodynamically unstable, other than vital parameters, invasive arterial pressure monitoring and ICU or high dependency unit admission must be considered. Placement of a central venous catheter will allow measurement of central venous pressure (CVP), mixed venous oxygen saturation (SVO2), administration of intravenous fluids (IVF) and vasopressors.[47]

Intravenous infusion of normal saline, dextrose saline and ringer lactate should be given to raise the CVP, the filling pressure of the ventricle and the cardiac output. Too rapid infusions in patient with overt cardiac function can cause pulmonary edema. Over resuscitation of shock can cause hypervolemia and should be avoided as this can precipitate the subsequent development of ARDS. Vasopressor support with norepinephrine may be considered even before optimal IVF loading has been achieved. Low dose Vasopressin (0.03 unit/min) may be subsequently added to reduce the requirement of high dose of norepinephrine alone.[48] Inotropes are added to volume resuscitation and vasopressors, if there is evidence of continued low cardiac output despite adequate cardiac filling and fluid resuscitation. The surviving sepsis campaign [Table 5] recommends that dobutamine is the first line inotrope therapy to be added to vasopressors in septic patients in the presence of myocardial dysfunction and on-going signs of hypoperfusion.[48] Resuscitation efforts should be continued as long as hemodynamic improvement accompanies.

Abnormalities in electrolyte balance and acid base balance should be corrected. Hemoglobin should be raised by packed cell infusion and kept close to 11 g/dl. Deranged coagulation profile should be corrected by infusion of fresh frozen plasma. Input/output charts should be carefully maintained.

Table 5: Surviving sepsis campaign international guidelines 2012

Fluid resuscitation recommendations
Use crystalloid for initial fluid resuscitation in severe sepsis and septic shock
Do not use hetastarch/hydroxyethyl starch greater than 200 KDa molecular weight
Albumin can be added, if the patient require substantial amount of crystalloid
Initial fluid challenge in patients with sepsis-induced tissue hypoperfusion with suspicion of hypovolemia to achieve a minimum of 30 mL/kg of crystalloids (a portion of this may be albumin equivalent). More rapid administration and greater amounts of fluid may be needed in some patients
Fluid challenge technique be applied wherein fluid administration is continued as long as there is hemodynamic improvement either based on dynamic (eg, change in pulse pressure, stroke volume variation) or static (eg, arterial pressure, heart rate) variables
Recommendations for vasopressors, inotropes
NE is the first choice for vasopressor therapy
Vasopressin 0.03 units/min may be added to norepinephrine with intent Of either raising MAP or decreasing NE dosage
When a second agent is needed epinephrine is weakly recommended
Dopamine is recommended only in highly selected patients whose risk of arrhythmias is very low and who had a low heart rate and or cardiac output
Low-dose dopamine should not be used for renal protection
A trial of dobutamine infusion up to 20 micrograms/kg/min be administered or added to vasopressor (if in use) in the presence of (a) myocardial dysfunction as suggested by elevated cardiac filling pressures and low cardiac output, or (b) ongoing signs of hypoperfusion, despite achieving adequate intravascular volume and adequate MAP

NE=Norepinephrine, MAP=Mean arterial pressure

Supplemental oxygen therapy is valuable in severely septic patients even if they don't have signs of respiratory distress.

Antibiotic Therapy

Empirical Therapy should be started promptly after relevant cultures have been sent. One should never await culture results as this would waste precious time.[49] The recommended antimicrobial regimen for patients with intra-abdominal infections have been outlined by the Surgical infection society based on prospective randomized clinical trials [Table 6].[50] All the recommended regimens are effective against gram negative enteric aerobic and anaerobic microorganisms.[51] A recent review of prospective randomized studies of antibiotic regimen for secondary peritonitis of gastrointestinal origin in adults from the Cochrane colorectal cancer group concluded that 16 antibiotic regimens had similar rates of clinical success.[52] There was no difference in mortality between any of these regimens. The use of appropriate empiric antimicrobial treatment has been associated with improved survival in a variety of clinical settings. Study by Baré *et al*. has demonstrated that selection of an appropriate treatment regimen as recommended by Surgical Infection Society was associated with a significant and marked improvement in successful treatment.[53]

Table 6: Recommended antimicrobial regimen for Patient with intraabdominal infection

Single agents
Ampicillin/sulbactam
Cefotatan
Cefoxitin
Ertapenem
Imipenem/cilastatin
Meropenem
Moxifloxacin
Piperacillin/tazobactam
Ticarcillin/clavulanic acid
Combination regimens
Aminoglycoside plus an anti anaerobicagent (clindamycin or metronidazole)
Aztreonam plus clindamycin
Cefuroxime plus metronidazole
Ciprofloxacin plus metronidazole
Third or fourth generation cephalosporins (cefepime, ceftazidime, ceftizonin or ceftrioxone) plus an antianaerobe

Definitive Surgical Treatment

Surgical therapy alone may be sufficient to cure otherwise healthy young patients who have no signs of severe sepsis, hence should never be delayed unnecessarily.[54] Though the risk of surgery is high, however, the passage of time only increases the risk. The surgical procedure depends on the nature, extent and duration of the problem.

Intraoperative Management

The primary goal of anesthesiologist during the intraoperative period is to provide safe and optimal care. General anesthesia with endotracheal intubation and controlled ventilation is the technique of choice. Almost all the laparotomies are done on an emergency basis. A quick and thorough airway assessment must be done to identify any potential difficulty. One should make sure that appropriate help is available in the operation theatre. In addition to the standard intraoperative monitoring, invasive hemodynamic monitoring should be considered in hemodynamically unstable patients. Special care should be taken to maintain normothermia and fluid, electrolytes and acid base balance. Advanced age, comorbid illnesses, delayed presentation, presence of features of sepsis or organ dysfunction are some of the predictors that the patient will require ICU care after surgery.[23-30]

Induction of Anesthesia

De-nitrogenation of the lungs, breathing 100% oxygen through a face mask should be considered before induction of anesthesia. A rapid sequence induction and intubation using succinylcholine to facilitate tracheal intubation may be required. If the patient is having hyperkalemia or any other contraindication to succinylcholine, rocuronium can be employed for facilitating neuromuscular relaxation.[55]

Use of opioids to prevent pressor response to airway manipulation & gentle mask ventilation (inspiratory pressure <20 cm H2O) before tracheal intubation in obese, pediatric, pregnant and critically ill patients are some of the new emerging consensus.[56]

Options for induction drugs include ketamine, etomidate, slow administration of propofol, or titrated doses of thiopentone sodium. Most intravenous or inhalational anesthetic agent causes vasodilation or impaired ventricular contractility, so induction of anesthesia should be a step wise process, using small incremental doses titrated to clinical response. Ketamine or midazolam may be used in hemodynamically compromised or critically ill patients. Short acting opioids such as fentanyl, alfentanil or remifentanil will enable a reduction in the dose of anesthetic induction agent. Continued volume resuscitation and vasopressor infusions are helpful to counteract the hypotensive effect of anesthetic agent and positive pressure ventilation.[54]

Maintenance of Anesthesia

Anesthesiologist should choose the technique which they believe fits with their assessment of the individual patient risk factors and co-morbidities and their own experience and expertise. Either inhalational agents or intravenous agents may be used with opioids. Minimum alveolar concentration (MAC) of inhalational anesthetic agents is reduced in severe sepsis.[57] During surgery the hemodynamic state may be further complicated by blood loss or systemic release of bacteria and endotoxins. Intravascular volume resuscitation should be continued throughout the surgical procedure. Intraoperative CVP values may be increased by raised intra-thoracic and intra-abdominal pressure. Throughout the surgical procedure, cardiovascular parameters (heart rate, cardiac filling pressure, inotropic state, systemic arterial pressure) can be adjusted to optimize tissue oxygen delivery. If the patient has intraoperative hypoxemia, it can be managed by increasing the inspired oxygen concentration. The inspired oxygen concentration can be increased until oxygen saturation (SaO_2) is at least 90% and the use of PEEP may be considered. Intraoperative hypothermia should be avoided as it has been found to be associated with impaired platelet and coagulation factor dysfunction.[58]

Opioids, NSAIDs, tramadol etc., can be used for analgesia in titrated doses as permissible by the renal and hepatic functions of the patient. Local anesthetic functions may get confounded by the presence of sepsis, local infection or acidosis.

Role of Neuraxial Blockade

Sepsis is considered to be a relative contraindication to regional neuraxial blockade. It should be undertaken with caution; since the hemodynamic effects of these techniques in the setting of sepsis can induce cardiovascular compromise which may be difficult to reverse.[59] Recent blood tests confirming normal coagulation are required.

Embu *et al*. have reported use of spinal anesthesia for surgery for typhoid perforation in rural African hospital.[60] The anesthesiologist involved had limited training and resources. A sensory level of T6 was found to be adequate for exploratory laparotomy, though sometimes sedative doses of ketamine were required to make the procedure more tolerable to the patient. Epidural blockade has also been used in these patients, but is better to be used as adjunct rather than sole anesthetic. It is more technically demanding and may not be suitable for rural hospitals, especially where a catheter needs to be inserted. Recent evidence suggests thoracic epidural blockade to be beneficial during sepsis by improving gut perfusion.[61] Spackman *et al*. have also shown that epidural analgesia resulted in improvement in gastric mucosal perfusion and the ultrasound appearance of the small bowel.[62]

While epidurals have very low risk of permanent neurological sequelae, severely septic patients may be at increased risk of this and other complications.[63]

Post-operative Management

In all critically ill patients, analgesia, sedation and mechanical ventilation are maintained at the conclusion of surgery. Safe transportation of the patient to the ICU and elaborate handover should also be ensured.

Critical Care

Pre-resuscitation measurement should be used to calculate the intensive care admission APACHE score. Attention to fluid, electrolyte & acid base balance and to nutrition is

vital. Cardiorespiratory support and in particular ventilator support are necessary in critically ill patients. In patients with severe sepsis sufficient fractional inspired oxygen concentration (FIO2) should be used to maintain adequate oxygenation (PaO2 > 12 Kpa) i.e., 90 mmHg.[64] Low tidal volume (up to 6 ml/Kg of the predicted body weight), limitation of inspiratory plateau pressure and permissive hypercapnia may be considered to prevent lung volume and baro-trauma, provided that the arterial pH does not decrease by 7.20.[65] Ongoing infusion of vasopressor medication can be adjusted to match the present intravascular volume and the new mechanical ventilator setting. Application of at least a minimal amount of positive end-expiratory pressure (PEEP) in ARDS; head-of-bed elevation in mechanically ventilated patients unless contraindicated; a conservative fluid strategy for patients with established ARDS who do not have evidence of tissue hypoperfusion; protocols for weaning and sedation; minimizing use of sedation; avoidance of neuromuscular blockers if possible in the septic patient without ARDS; a short course of neuromuscular blocker (no longer than 48 hrs) for patients with early ARDS are some of the key recommendations of surviving sepsis campaign.[48]

Antimicrobial therapy which was started before operation should be continued in ICU. Antibiotic regimens can be reassessed in the light of microbiological results, and adjusted. Continuation of adequate glycemic control is important in the control of septic process. In severely septic patients blood glucose should be maintained in the range of 6-10 mmol/L.[66]

Nutrition is one of the cornerstones of management in these patients. Though enteral route should be started as soon as feasible, parenteral nutrition should be considered if there is surgical contraindication for enteral administration[67]

Intravenous hydrocortisone may be considered when hypotension responds poorly to fluid resuscitation and vasopressors. Hydrocortisone in a dose of 200 mg/day in four divided doses or as a continuous infusion in a dose of 240 mg/day (10 mg/hr) for 7 days is recommended for septic shock in ICU setting.[68,69]

Acute renal failure occurs in 23% of patients with severe sepsis. Renal replacement therapy may be initiated to correct acidosis, hyperkalemia or fluid overload. Continuous renal replacement therapy (CRRT) and sustained low efficiency dialysis (SLED) can be considered in hemodynamically unstable patients.[70]

Stress ulcer and deep vein thrombosis prophylaxis are also recommended.

Conclusion

Perforation peritonitis is the most common surgical emergency with high mortality. The anesthesiologist has a crucial role in coordinating and in delivery of resuscitation and therapeutic strategies to optimize patient survival outcome.

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