mean the patient would become upset, or decide to refuse treatment.

Obtaining informed consent cannot be an isolated event. It involves a continuing dialogue keeping patients abreast of changes in their condition and the treatment or investigation proposed. Whenever possible, treatment options should be discussed at a time when the patient is best able to understand and retain the information.

Emergency situations:

In an emergency, where consent cannot be obtained, medical treatment may be provided to anyone who needs it, provided the treatment is limited to what is immediately necessary to save life or avoid significant deterioration in the patient's health.

INTRA - ABDOMINAL SEPSIS – EPIDEMIOLOGY AND CLINICAL PRESENTATION

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Severe Sepsis (acute organ dysfunction secondary to infection) and septic shock (severe sepsis plus hypotension not reversed with fluid resuscitation) are major healthcare problems, affecting millions of individuals around the world each year, killing one in four (and often more), and increasing in incidence [1].

- Incidence increased from 82 to 240/100,000 population
- Men > women 1.28 CI
- Reduced mortality rate 27 to 18%
- Highest mortality black men
- Decreased length of hospital stay
- Increased discharge to chronic care
- Predominance gram positive organism after 1987
- Fungal organism increase 207 %

Similar to major trauma, acute myocardial infarction or stroke, the speed and appropriateness of therapy administered in the initial hours after severe sepsis develops are likely to influence outcome. In 2004, an international group of experts in the diagnosis and management of infection and sepsis, representing 11 organizations, published the first internationally accepted guidelines that the bedside clinician could use to improve outcomes in severe sepsis and septic shock [2]. These guidelines represented phase II of the Surviving Sepsis Campaign (SSC) an international effort to increase awareness and improve outcomes in severe sepsis. Joined by additional organizations the group met again in 2006 and 2007 to update the guidelines document using a new evidence-based methodology system for assessing quality of evidence and strength of recommendations [3].

Intra-abdominal sepsis

These infections include secondary peritonitis, abdominal abscesses and cholangitis. The infection generally occurs because enteric microorganisms enter the peritoneal cavity through a defect in the wall of the intestine or other viscus as a result of obstruction, infarction, or direct trauma. Mixed aerobic and anaerobic flora can be recovered. The predominant aerobic isolates are Escherichia coli, and enterococci and the main anaerobic bacteria are Bacteroides fragillis, Peptostreptococcus. and clostridium species.

The treatment of abdominal infection includes surgical correction and drainage of pus and administration of antimicrobials effective against both the aerobic and anaerobic pathogens.

Pathophysiology

Intraperitoneal infections are caused by members of the gastrointestinal flora, mainly Escherichia coli, enterococcil, Klebsiella, Enterobacter, Proteus, Bacteroides, anaerobic coccil, Glostridia and Fusobacteria. The gram-negative aerobic bacteria exert their pathogenic potential mainly by endotoxin which acts by way of mediators, causing systemic septic response and, initially, the local response of the peritoneal cavity.

The anatomic aspect of peritonitis describes the division of the abdominal cavity into supracolic, infracolic and paracolic spaces, the lesser sac and the cul-d-sac of the pelvis. Endotoxin which is elaborated by bacteria activates the classical as well as the alternative complement pathway. It activates also the arachidonic acid metabolism, leading to the release of prostaglandins (PG) and leukotriens (LTC). The local host defense against a bacterial invasion includes the activation of cellular and humeral immunologic defense mechanism, in which the final product of the complement pathway (C5b-9), as well as chemoattractants C3a, C5a and C567 play a key role.

Peritoneal infections are truly synergistic infections. The most important synergistic mechanisms are protection against host defense and creation of a suitable environment by one member of the flora for another. Certain adjuvant substances, ie. bile, gastric juice, blood and necrotic tissue, play a role in the pathogenesis of peritonitis. The peritoneum deals either an infection in 3 ways:

➢ Direct absorption of bacteria into the lymphatics via the stoma of the diaphragmatic peritoneum

➢ Local destruction of bacteria through phagocytosis by either resident macrophages or polymorphonuclear granulocytes attracted to the perioteneal cavity

➢ Localization of the infection in the form of an abscess.

Definitions

Sepsis is defined as infection plus systemic manifestations of infection. Severe sepsis is defined as sepsis plus sepsis-induced organ dysfunction or tissue hypoperfusion. The threshold for this dysfunction has varied somewhat from one severe sepsis research study to another.

Sepsis-induced hypotension is defined as a

➢ Systolic blood pressure (SBP) <90 mmHg

➢ Mean arterial pressure (MAP) <70 mmHg

➢ SBP decrease >40 mmHg or <2 SD below normal for age in the absence of other causes of hypotension.

Septic shock is defined as sepsis-induced hypotension persisting despite adequate fluid resuscitation. Sepsis induced tissue hypoperfusion is defined as either septic shock, an elevated lactate or oliguria.

Management of Severe Sepsis

A. Initial Resuscitation

Protocol resuscitation of a patient with sepsis induced shock, defined as tissue hypoperfusion (hypotension persisting after initial fluid challenge or blood lactate concentration ≥4 mmol/L) have been developed. This protocol should be initiated as soon as hypoperfusion is recognized and should not be delayed pending ICU admission. During the first 6 hours of resuscitation, the goals of initial resuscitation of sepsis-induced hypoperfusion should include all of the following as one part of a treatment protocol:

➢ Central venous pressure 8-12 mmHg

➢ Mean arterial pressure (MAP) ≥ 65 mmHg

➢ Urine output ≥0.5 mL-kg^{-1}.hr^{-1}

➢ Central venous (superior venal cava) or mixed venous oxygen saturation ≥70% or ≥65% respectively

B. Diagnosis

Appropriate cultures should be obtained before antimicrobial therapy is initiated if such cultures do not cause significant delay in antibiotic administration. To optimize identification of causative organisms, at least two blood cultures should be obtained before antibiotics with at least one drawn percutaneously and one drawn through each vascular access device.

C. Antibiotic Therapy

Intravenous antibiotic therapy be started as early as possible and within the first hour of recognition of septic shock and severe sepsis without septic shock . Appropriate cultures should be obtained before initiating antibiotic therapy but should not prevent prompt administration of antimicrobial therapy.

The antibiotics should include one or more drugs that have activity against all likely pathogens - aerobic isolates Escherichia coli, and enterococci and the main anaerobic bacteria are Bacteroides fragillis, Peptostreptococcus. and clostridium species and that penetrate in adequate concentrations into the presumed source of sepsis.

When choosing empirical therapy, clinicians should be cognizant of the virulence and growing prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) in some communities and healthcare settings. Clinicians should also consider whether candidemia is a likely pathogen when choosing initial therapy. When deemed warranted, the selection of empirical antifungal therapy (e.g. fluconazole, amphotericin B, or echinocandid) should be tailored to the local pattern of the most prevalent *Candida* species and any prior administration of azoles. Because patients with severe sepsis or septic shock have little margin for error in the choice of therapy, the initial selection of antimicrobial therapy should be broad enough to cover all likely pathogens.

D. Source Control

➢ Specific anatomical diagnosis of inection requires consideration for emergent source control (eg necrotizing fasciitis, diffuse peritonitis, cholangitis, intestinal infarction) be sought and diagnosed or excluded as rapidly as possible.

➢ All patients presenting with severe sepsis should be evaluated for the presence of a focus on infection amenable to source control meausres, specifically the drainage of an abscess or local focus on infection, the debridement of infected necrotic tissue, the removal of a potentially infected device, or the definitive control of a source of on-going microbial contamination.

➢ When source control is required, the effective intervention associated with the least physiologic insult be employed (eg. percutaneous rather than surgical drainage of an abscess.

➢ Intravascular access devices are a possible source of severe sepsis or septic shock, they be promptly removed after other vascular access has been established.

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