Case Report

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Tertiary peritonitis in patients with cirrhotic ascites; Case report

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Abstract

Peritonitis is the inflammation of peritoneum of clinical emergency importance either in operation room or intensive care unit. Peritonitis is divided into primary, secondary, and tertiary. Primary peritonitis or spontaneous peritonitis is arises in the absence of an identifiable anatomical causes and has a low incidence on surgical intensive care units. Secondary peritonitis (SP) is the commonest peritonitis which is defined as an infection of the peritoneal cavity resulting from perforation, anastomotic disruption, ischemic necrosis, or other injuries of the gastrointestinal tract. Tertiary peritonitis can be defined as the persistence or recurrence of intra-abdominal infection with multiple organ failure and a systemic inflammatory response in an immune compromised host that develops after what was thought to be a effective attempt at primary source control. The management of tertiary peritonitis should include the provision of appropriate physiologic support, the administration of antimicrobial therapy, and operation or intervention to control the source of contamination and to decrease the bacterial load. Moreover, two crucial components must be present, which include the time period, which is 48 hours, and there must be successful surgical source control. Moreover, tertiary peritonitis remains a vital cause of hospital death mainly among patients with associated risk factors.

Introduction

Peritonitisis a most important consequence of hollow visceral perforation, anastomotic disruption, ischemic necrosis, or other injuries of the gastrointestinal tract, often initiatives acute care in the emergency department, operating room, and the ICU [1-3]. Resuscitation joined with rapid source control of the infectious focus and appropriate empiric antibiotic therapy to reduce bacterial load are effective treatment [4-7]. Such an integrated approach is incorporated by a variety of medical professional organization guidelines as well as the Surviving Sepsis Campaign [8]. Several pathogens are associated with the onset of the TP. The inflammation depends on the peritoneum capacity to allow the activation, differentiation, and proliferation of immune cells, which leads to complex immune responses against invading microorganisms[9].

Because TP is a life-threatening condition, an accurate and rapid diagnosis is crucial for the appropriate management of the disease. Such diagnosis is made on the basis of clinical manifestations related to the peritoneal and systemic inflammation and can be supported by laboratory findings, imaging, and some score systems[10]. In the cases when secondary peritonitis is diagnosed during the initial operation, a successful attempt of surgical source control (e.g., exteriorization of perforated viscus) is often adequate to save prolong morbidity and mortality in the majority of patients. However, a reoperation may be indicated in a subset of patients who show clinical signs of recurrent or persistent intra-abdominal infection in spite of apparently successful source control, which is referred to as TP [11].

The management of affected individuals is made through measures aimed at infection control, which range from antimicrobial therapy to percutaneous drainage or open surgical intervention[12].

Case Report

Cirrhotic ascitic patient 65 years old man presented to the hospital because of one week history of abdominal pain and distension, he received antispasmodic many time to relief abdominal pain. The patient had past history of splenoctomy since 30 years.

Esophagogastroduodenoscopy was done several time as diagnostic and therapeutic intervention.

On examination, the patient appeared ill, with prominent abdominal distension, pain with some guarding and rebound. However he was haemodynamically stable and afebrile. No masses or organomegaly were palpated. On percussion, a shifting dullness was observed, which suggested ascites. Normal bowel sounds were present blood pressure was 110/70, heart rate was 90/min, temperature was 37 ^{c}, and O_2 saturation was 99%. The patient was admitted to Damietta fever and gastroenterology hospital.

Initial investigations showed raised C reactive protein 96 mg/dl, WBCs was 21,700/cmm, neutrophil was 19,000/ cmm, hemoglobin count within the normal range, platelet count was 145000/cmm, INR was 1.3, serum creatinine was 2.5 mg/dl, serum albumin was 2.8 g/dl, bilirubin was 1.5 mg/dl, serum amylase levels within the normal range, the urine analysis did not show significant proteinuria or hematuria. Abdominal CT was avoided because of initial high serum creatinine however, abdominal ultrasound scan

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showed mild to moderate ascites, coarse cirrhotic liver and splenoctomy. Ascetic fluid aspiration was done which revealed purulent peritoneal fluid figure (1) turbid color and WBC in ascetic was 92,000/cmm mainly neutrophils, ascitic protein was 3.2 gm/dl**,** SAAG was<1.1**.** Abdominal erect X ray to exclude perforated viscous was free, the bowel habit was normal. We asked surgical consultants to exclude secondary peritonitis and he was advised medical treatment and follow up the condition. We aspirate ascitic fluid and wait the culture and sensitivity test. The patient received empirical treatment in the form of third generation cephalosporin's and metronidazole intravenously until the result of culture. Three days following initiation of antibiotic therapy, WBCs was 18, 200 and neutrophil 90% and serum creatinine increased to 3.3 mg/dl, albumin decreased to 2.4gm/dl, bilirubin was 1.4 mg/dl and the amount of ascites increased by abdominal ultrasound. Again we asked surgical consultation, they advised continues of medical treatment and follow up. The patient was admitted ICU and received treatment then surgical exploration was done and peritoneal toilet and search for the cause but the surgeon don't find the cause and diagnosed the case as tertiary peritonitis, then closed the abdominal incision and fixed drained catheter and continue the medical treatment. The patient was discharged after 2 week from hospitalization but still had mild ascites, continued antibiotic as levofloxacin and metronidazole. 4weeks after discharge the laboratory investigation were WBCs 8700, serum creatinine 1.2, INR 1.3, serum albumin 2.7 and serum bilirubin 1.3. Ascetic fluid aspiration was done and WBC in ascetic was 85 mainly neutrophils.

Table 1. Demographic, anthropometric and biochemical data between both studied groups.

	1 day	3 day	5 day	8day	15 day	4weeks post-discharge
WBCs	21.700	18200	33000	25400	17000	**8700**
Creatinine	2.5	3.3	1.1		1.2	1.2
INR	1.2	1.3	1.3	1.4	1. 5	1.3
S Albumin	2.8	2.4	2.0	1.8	1.5	2.7
S bilirubin	1.5	1.4	1.3	1.3	1.3	1.3

Discussion

Age of the patient, underlying etiology of peritonitis, malnutrition, and presence of multidrug resistant microorganisms are significant risk factors which may predispose towards TP. The differentiation between secondary and TP becomes really difficult as there is usually a continuum between the two and the exact time point when secondary peritonitis turns into TP is often missed. In our case when secondary peritonitis is not diagnosed during the initial operation, an unsuccessful attempt of surgical source control but peritoneal toilet was done often enough to save prolong morbidity and mortality in the majority of patients. However, a reoperation may be indicated in a subset of patients who show clinical signs of recurrent or persistent intra-abdominal infection, which is referred to as TP. It is important to differentiate the entity with an ongoing secondary peritonitis developing because of failure of the surgical attempt of the source control (e.g., failure of the surgical procedure or missed pathology), whereas in TP there is no obvious anatomical defect or perforation of the hollow viscus. A planned or on-demand re laparotomy after an initial operation is probably most frequent way to diagnose TP, but is a late event to occur [13,14] Hence, it is desirable to have timely and no operative diagnosis of TP after the initial operation and subsequent initiation of an appropriate therapy to reduce the complications and to improve the outcome. It is also essential to identify patients at risk for developing TP as early as possible after initial operation for secondary peritonitis.

There has been no consensus till date concerning value of clinical and laboratory parameters and scoring systems for sufficient diagnosis and monitoring of TP [15]. The ICU consensus conference guideline provides a precise definition of TP as intra-abdominal infection that persists or recur ≥48 h following successful and adequate surgical source control. This definition contains two important conditions, which have to be encountered; the time period (≥48 h) and successful surgical source control **[16].** Mannheim Peritonitis Index, Simplified Acute Physiology Score II (SAPS II), and C-reactive protein are three initial and simply accessible parameters which may be help for recognition of patients who might further develop TP **[15].** However, there are conflicting data concerning the value applying such parameters for the detection of TP [17,18]. There is still a lack of studies addressing the identification of risk factors for patients prone to develop TP. Hence it still remains to have diagnostic markers that could predict the likelihood of a patient to develop TP even at the onset of peritonitis, during the initial operation, or during the first postoperative days.

Conclusion

Although advances have been achieved in our knowledge of TP, much has to be done aiming for a better understanding of this condition. Successful diagnosis is essential for an appropriate and rapid treatment, which should include both procedural approaches aiming for the definitive control of the infectious focus and the use of

antimicrobial drugs, to decreasing the complications and adverse outcomes

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