Preperitoneal Idiopathic Necrotising Fasciitis in Two Patients with Different Outcomes: A Case Report

Priyadarshini Manay, Saneya Pandrowala*, Shilpa A Rao and R R Satoskar

Department of General Surgery, Seth G.S.M.C and K.E.M Hospital, Parel, Mumbai, India

*Corresponding Author: Saneya Pandrowala, Department of General Surgery, Seth G.S.M.C and K.E.M Hospital, Parel, Mumbai, India; E-mail: abdelalims@gmail.com

Introduction

Necrotizing fasciitis is a mixed infection of the skin and subcutaneous tissues with a characteristic clinical and pathological appearance. Early radical surgical excision of all affected tissue is the treatment of choice [1]. Retroperitoneal necrotising fasciitis is a rare, fulminant but potentially fatal soft tissue infection caused by intra-abdominal suppuration [2]. So far cases of necrotising fasciitis of retroperitoneal origin spreading to preperitoneal space have been reported but none were primarily preperitoneal in origin. Retroperitoneal necrotising fasciitis is usually a poly microbial infection [1]. A prompt diagnosis is often difficult but is essential with immediate surgical intervention to help ensure a potentially positive patient outcome. Characteristics that help distinguish patients with idiopathic necrotising fasciitis from secondary necrotising fasciitis include an age greater than 55 years, the presence of co-morbidities such as diabetes mellitus or chronic renal failure, and perineal origin [3]. We present to you two cases of preperitoneal necrotising fasciitis with no evidence of intra-abdominal suppuration.

Case Discussion

Case 1:

The first case is a 36 year old male with one day history of acute onset lower abdomen and right iliac fossa pain with no history of vomiting or constipation or urinary complaints. The patient was a chronic alcoholic with no other significant medical or surgical history. On examination he had lower abdomen guarding and tenderness. Preoperatively patient had counts of 6200 with band forms and creatinine of 1.2, rest blood investigations was normal. An erect chest radiogram did not show free air under diaphragm. Ultrasonogram of abdomen was suggestive of probe tenderness however the appendix could not be visualized. CECT of abdomen and pelvis showed air specs in the right iliac fossa which was mistaken for free intra peritoneal air. The CT also showed a thickened bladder wall. A provisional diagnosis of perforated appendix was made and the patient taken up for emergency exploratory laparotomy.

The intraoperative finding showed necrotising fasciitis of preperitoneal space spreading retro peritoneally (Figure 1). The duskiness spread to anterior wall of the bladder. Intraoperatively methylene blue and saline was infused into bladder through the Foleys catheter but no gross leak found.

Figure 1: Pre peritoneal duskiness extending laterally and in pelvis with bowel appearing grossly normal.
The entire small bowel, large bowel, kidneys and retroperitoneal organs were traced and found to be normal. Intraoperatively the entire necrotic tissue extending behind the right kidney was debrided and a drain placed in the retro peritoneum and one drain placed in pelvis and the patient was shifted to ICU on ventilatory support (Figure 2). Due to extensive involvement patient was started empirically on meropenem, metronidazole and fluconazole for broad coverage. After 4 hours of surgery, patient was unable to wean off ventilator with pulse of 120, SBP 100, creatinine was 5.2 with urine output less than 200ml, total counts were 11900. The drain on the right side was draining 200ml of blackish foul smelling fluid. After 8 hours blood pressure fell to 80mmHg. Ionotropic support was started after adequate fluid resuscitation with a central venous pressure of 10cm of H₂O. After 24 hours of surgery however, patient succumbed to septicemia.

**Figure 2:** Necrotic tissue above the peritoneum seen extending into the pelvis.

**Case 2:**

The second case was a 20 year old male with history of 1 day lower abdomen pain with 5 episodes of vomiting with history of fever with no significant present or past illness. On examination the patient had generalised abdominal guarding and rigidity. The patient was hypotensive on admission with a SBP of 84 which failed to show increase after fluid challenge and hence patient was started on ionotropic supports with the CVP being 8 to 10cm of H₂O. Ultrasonography of abdomen was suggestive of pelvic collection with a 7mm appendix. Blood investigations showed Hb of 10.9 and counts of 18900 and creatinine of 2 and hence contrast CT was not done. Erect chest radiogram did not show air under diaphragm. Patient was taken for exploratory laparotomy due to a suspicion of sealed off typhoid ileal perforation due to high incidence of typhoid infection in this part of the world. The intraoperative findings were suggestive of similar necrosis above peritoneum which was limited to left lower quadrant going up to the pelvis and was not extending to the retro peritoneum. The small and large bowel was normal; the necrotic tissue was completely debrided. A pre peritoneal drain was placed on the left side along with an intra peritoneal drain in the pelvis and a subcutaneous suction drain. Postoperatively the patient was shifted on ventilatory support to ICU. The patient was started on imipenem and metronidazole for broad coverage. Post operative counts were 3700 with Hb of 10.6 and creatinine of 1.6. The patient was maintaining blood pressure and was slowly weaned off inotropic supports on POD2 and was extubated on POD3. The pelvic drain was removed on POD5 as it was draining less than 20ml however the pre peritoneal drain drained 100 to 200ml of purulent fluid for 7 to 10 days. On POD5 Hb dropped to 4.9 and platelet to 40000 which was probably due to bone marrow suppression due to the infection as no other cause was identified. It was important to note that 3 cultures sent 4 days apart including the intraoperative fluid consistently showed no organisms, however the fourth culture showed presence of E. coli. Fungal culture was also negative as well as TB-MGIT which was sent due to the rampant nature of tuberculosis and its varied presentations in this part of the world. Patient developed burst abdomen on POD 4 for which suction dressing was given. Gradually the purulent drain output ceased and after a prolonged course in the ward patient was discharged with a healthy midline wound and Hb of 9.2 and counts of 9200.

**Discussion**

It is interesting to note that review of literature revealed not a single case of pre peritoneal idiopathic necrotising fasciitis. All cases so far have been those of retroperitoneal origin with or without an identifiable cause spreading anteriorly to the pre peritoneal space. Identifiable sources of infection including chronic pyelonephritis, diverticulitis, peri-anal abscess, colonic cancer, perforation.
Patients with extensive involvement have high mortality despite surgery [1]. Clinically it needs a high index of suspicion to detect retroperitoneal necrotising fasciitis as it manifests as either fever with abdominal pain or signs of peritonism. Skin erythema with crepitus [1, 5-10] is a late sign in the course of the disease. It is also important to note that an earlier diagnosis increases the chances of patient survival. The retroperitoneal and pre peritoneal space infections are dangerous due to a high density of lymphatics and an absence of omentum which prevents localisation of infection. Hence not only can pre peritoneal infections spread to the retro peritoneum and vice versa but also to the chest cavity.

Preperitoneal necrotizing fasciitis is an extremely difficult condition to diagnose preoperatively in the early stages. It requires a high index of suspicion. Factors that can make up suspect it are – a short duration of symptoms, rapid progression to shock with or without intervention, inability of basic investigations like X-Ray and USG to help in diagnosis. Clinically it mimics peritonitis but there is absence of free air under diaphragm on X-rays, ultrasonography is not usually able to pick up the disease or identity the source of infection. Any findings if or when visible on Computed Tomography (CT) happen when infection has already spread extensively. Many patients are too unstable to undergo a CT scan. Even a CT scan cannot locate the source of such an infection in idiopathic cases. Also it has been documented that debridement of extensive retroperitoneal necrotic tissue which is technically irresectable eg. Chest wall muscles, paraspinal muscles etc leads to a fatal outcome1. Antibiotic therapy appears to be of secondary importance to achieving adequate surgical excision [1]. The use of antibiotics at an early stage may help to reduce the amount of tissue destruction [1]. Although in other cases reported to have isolated Bacteroides spp. as a predominant isolate our cases grew E. coli [1].

The placement of a pre peritoneal drain helps to drain necrosum created from the ongoing necrosis post surgery has not been mentioned in literature but we have found it useful in one of our cases.

Conclusion
A high index of suspicion in diagnosis with appropriate antibiotic coverage and prompt radical surgical debridement is the treatment of choice. Despite taking all measures suggested above this condition is associated with high morbidity and mortality.

References