Case Report

An unwonted case of Meleney’s like abdominal wall necrotizing soft tissue infection

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ABSTRACT

Poly-microbial infection leading to necrotizing soft tissue infection is known to cause fulminant sepsis resulting in significant mortality and morbidity despite aggressive medical and surgical management. We present a case of Meleney’s like synergistic gangrene of abdominal wall culminating in full blown sepsis and death in the background of penetrating injury to the abdomen. Our patient was a 36 year old female, who presented with a penetrating injury to the abdomen due to a road traffic accident resulting in a laceration over the anterior abdominal wall with evisceration of bowel and omentum. After initial resuscitation, patient was immediately taken up for exploration and multiple small bowel perforations were primarily repaired. Postoperatively, she developed severe polymicrobial infection at the laceration site which extended all over the abdomen. She was treated with broad spectrum antibiotics based on culture and sensitivity and serial debridement of necrosis was done. She developed ventral hernia during the course, which was complicated by necrosis of bowel which was managed by resection and anastomosis. Postoperatively, she developed multi-organ dysfunction due to sepsis and succumbed to infection. We imply on the necessity of innovative techniques and multidisciplinary approach for controlling sepsis and compensating the significant soft tissue loss due to widespread necrotizing infection.

Keywords: Penetrating abdominal injury, Polymicrobial infection, Meleney’s gangrene, Broad-spectrum antibiotics

INTRODUCTION

Penetrating injuries to the abdominal wall often leads to poly-microbial infections which lead to spreading inflammation and necrosis of skin and other soft tissues. Various risk factors were implicated in the development of such fulminant infections such as diabetes, tumors, immunodeficiency etc. Few special poly-microbial infections that were noted for their fulminant course were - Fourniers gangrene, that was described in 1883 and Meleney’s synergistic gangrene, that was first described in 1926. Initial bacteriological insult, superadded with reduced host defense mechanisms, causes a rapid spread of infection along fascial planes causing massive tissue necrosis. In our case, a young lady who sustained a penetrating abdominal injury due to a road traffic accident developed a fulminant Meleney’s like necrotizing soft tissue infection of the abdominal wall. Despite the early aggressive management, the patient could not be salvaged.

CASE REPORT

A 36 year old female presented with an alleged history of a road traffic accident resulting in penetrating trauma by an iron rod to the antero-lateral abdominal wall and upper abdomen with evisceration of bowel contents 6 hrs after the inciting event. The patient was disoriented with GCS 13 and severely pale with pulse rate of 110 per minute
and a blood pressure of 60/40 mmHg. There was a 40×10 cm laceration over the umbilical and left lumbar region with bowel and omental evisceration and a 10×3 cm laceration over the epigastric region (Figure 1).

Initial fluid resuscitation with blood transfusion was carried out. Pelvic X-ray had revealed left iliac blade fracture. Chest X-ray and NCCT brain was normal. She was immediately taken up for exploration under anaesthesia. A midline laparotomy incision was given and other abdominal viscera and solid organs were explored. Multiple small bowel transections and serosal injuries were identified for which primary closure was done. Edges of the laceration site were debrided and stay sutures were taken after a thorough lavage. Patient was started on broad spectrum antibiotics and later culture specific antibiotics were given despite which the necrosis continued to spread to involve the entire abdominal wall (Figure 2).

Serial debridement and twice daily dressings were done. She developed a superadded fungal infection for which anti-fungal intra-venous amphotericin was given. Patient developed multiple episodes of paralytic ileus, owing to the high doses of analgesics she received, which were treated using regular enemas. On post-operative day 12, patient developed herniation of a bowel loop through the abdominal wall defect which progressed to bowel gangrene (Figure 3).

Owing to severe sepsis and bowel gangrene, patient was again taken up for re-exploration. Intra-operatively patchy gangrene was observed extending from distal ileum to the mid transverse colon. The gangrenous bowel was resected and an ileo-transverse anastomosis was done as there was no abdominal wall available for stoma construction. Post operatively patient had developed severe septic shock and multi-organ dysfunction with complete renal shut down and had succumbed to death.

**DISCUSSION**

Necrotizing soft tissue infection, characterized by extensive destruction of tissues and toxemia culminating in fulminant sepsis, is a known entity caused by anaerobes, gram- negative organisms and coliforms. The presence of various co-morbidities increases the chances of rapid deterioration. Presence of excess subcutaneous fat due to obesity acts as an independent risk factor of wound complication in view of poor vascular supply. Our case, although had no co-morbidities, had excess subcutaneous fat leading to a potential space for the bacteria to thrive. These infections usually cause a small vessel thrombosis on account of the localized infection and inflammation which culminated in necrosis. In the
present scenario, the penetrating injury to the abdomen severed the inferior epigastric artery leading to the devascularisation of the part of the tissue between the laceration on the lower antero-lateral abdomen and the laceration over the epigastrium. Bacterial infection, ischemia and reduced defense mechanisms are the key factors in the spread of infection in necrotizing fasciitis. Control of local sepsis by surgical debridement and the appropriate antimicrobial coverage are the two mainstay in the treatment of the condition. Multiple linear incisions should be made to assess the area and extent by passing probe or finger. Debridement should continue until the subcutaneous tissue no longer separates from the underlying tissue. Various techniques such as negative pressure wound therapy would help in improving the local perfusion and nutritional supply and optimizes the granulation tissue. But presence of a severe necrotic infection with the visceral herniation through the defect limited its usage in the current case. Hyperbaric oxygen therapy was found useful only to tackle the anaerobic infection but cannot be substituted for radical surgical debridement. Presence of polymicrobial infection precluded the use of this therapy. Technique of preserving the unaffected skin during debridement limits the need for excess graft harvest at the later stage. These techniques are difficult to employ and practically challenging in cases with generalized septicemia and rapidly spreading infection like necrotising soft tissue infections.

CONCLUSION

This case had demonstrated the essential need for development of novel techniques in complicated abdominal wall defects in the presence of an active infection and need for a multidisciplinary team involvement in managing complicated abdominal wall necrotizing soft tissue infections.

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REFERENCES
