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CASE REPORT

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Necrotising fasciitis of the abdominal wall

Katherine J Williams*

Surgical Registrar, Kingston Hospital NHS Trust, UK

Abstract

A 53 year old gentleman, with a history of intravenous drug use, presented to the Emergency Department in septic shock with an acute abdomen. He had been recently treated with dalteparin for a deep venous thrombosis of his left leg. He was resuscitated with fluids and antibiotics, and cross-sectional imaging suggested a diagnosis of necrotising fasciitis of the abdominal wall. He was admitted to ITU, and a laparotomy, abdominal washout, muscle debridement and laparotomy was performed overnight. No bowel perforation or contamination was identified. Over the next few weeks he underwent multiple debridements, and a negative pressure abdominal dressing was utilised. After a prolonged rehabilitation phase he was discharged home to live independently. He is currently in discussion with the plastics team regarding reconstruction of his abdominal wall. We would like to stress the importance of consideration of necrotising fasciitis as a diagnosis in the context of a critically unwell and septic patient with a history of subcutaneous/intravenous medication usage.

Keywords: *acute abdomen, laparotomy, septic patient*

Background

Necrotising fasciitis can present in an atypical fashion, especially in the context of self-administered subcutaneous medications, or illicit intravenous drug use. The morbidity and mortality associated with necrotising fasciitis is very high, and swift diagnosis and treatment antibiotics, surgical debridement and cardiovascular support is mandatory to maximise chances of survival. If diagnosis is unclear, cross-sectional imaging can be helpful.

Case report

A 53 year old gentleman presented to the Emergency Department of a district general hospital in the early hours of the morning. The presenting symptoms were 6 days of abdominal pain and shortness of breath. He denied any gastrointestinal symptoms, cough, weight loss, or previous similar episodes. He was clinically septic with cold oedematous extremities, and vascular access for resuscitation was achieved through a femoral line (fluids and broad spectrum antibiotics). On examination he was tachypnoea, and tachycardia. He had a distended, peritonitic abdomen with absent bowel sounds. Hernial orifices were normal, and a urinary catheter was placed but drained very little. Digital rectal examination was unremarkable.

Of note, his past medical history included heroin use, both smoking and intravenous. He had been successfully treated for hepatitis C in the past with interferon. He denied any drug allergies, and took daily methadone.

Three months previously, he had attended A+E with a swollen left leg. D-dimer was 2637 ng/ml, and he had been investigated with venous duplex ultrasound. Results were inconclusive for a deep vein thrombosis, and he had been started as an outpatient on empirical low molecular weight heparin.

Investigations

On admission he had a respiratory rate of 45/min, pulse 110/min, blood pressure 110/62 mmHg, saturating at 95% on 4litres oxygen. Bloods results were: Hb 169g/L, white cell count 13.9x10^9 /L, INR 1.4, C-reactive protein 325 mg/L, bilirubin 104 umol/L, ALT19 U/L, ALP 76 U/L, albumin 18 g/L. His urea and electrolytes were within normal limits. Arterial blood gas analysis (15L oxygen) showed a pH 7.43, pO2 14.5, pCO2 3.5, base excess -5, bicarbonate 17, and a lactate of 3.3. Electrocardiogram showed a sinus tachycardia.

Erect chest X-ray showed some basal atelectasis, and abdominal film showed some unusual gas patterning in the lateral abdomen bilaterally.

An urgent IV contrast-enhanced CT of his chest, abdomen, and pelvis was performed (Figure 1). This showed a small amount of intra-peritoneal free fluid, with multiple pockets of gas in the muscles of the abdominal wall, extending from the pelvis to the diaphragm. There was no evidence of bowel ischemia, and liver, pancreas, spleen and kidneys had normal appearances. There was no evidence of pulmonary embolus.

Differential diagnosis

1. Perforated gastro-intestinal viscous
2. Necrotising fasciitis
3. Vascular event, such as infected deep venous thrombosis, endocarditis, or acute pulmonary embolus

Treatment

This gentleman was admitted to intensive care for goal-directed resuscitation, and shortly after stabilisation was admitted to theatre for laparotomy under general anaesthetic. Upon opening

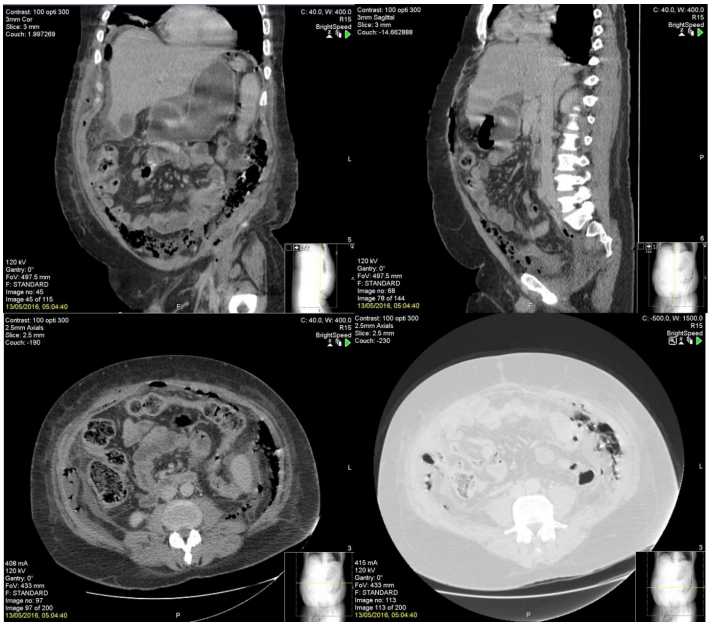

Figure 1. *Admission CT scan of patient's abdomen and pelvis (IV contrast-enhanced), coronal, sagittal and axial views. These demonstrate pockets of free air in the muscles of the abdominal wall.*

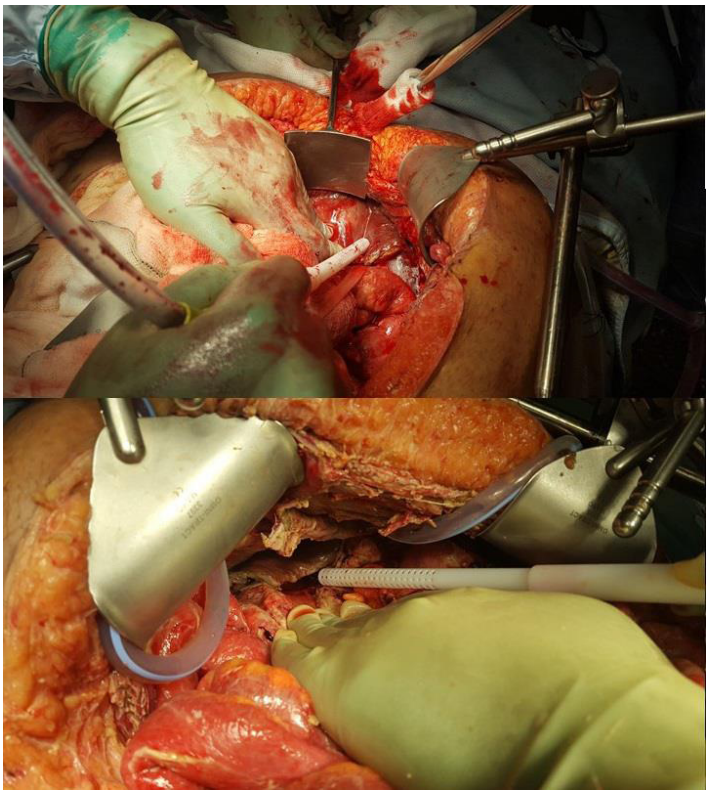

Figure 2. *Intra-operative photographs showing necrosis of abdominal wall muscles.*

the abdomen an extremely offensive odour was released. He was found to have circumferential, necrotic, gas-filled abdominal wall muscles (Figure 2). Intra-abdominal organs were examined and found to be intact. There was some free fluid but no faecal contamination. No evidence of gastrointestinal tract perforation was identified. The necrotic muscle was extensively excised, and a laparotomy was formed with a Bogota bag.

Over the next few weeks he underwent multiple abdominal wall debridements and peritoneal washouts under general anaesthetic. A negative pressure dressing was applied to his open abdomen. The necrosis spread to involve the perinephric fat, posterior abdominal wall, and upper abdomen, threatening the diaphragm. Tissue, blood, and urine cultures were all negative. He was transferred to a tertiary centre for HPB and cardiothoracic input to his care. He required two weeks supportive care only, not requiring further debridement.

Outcome and follow-up

He did extremely well, responded to treatment, and was repatriated to the district general hospital, eating and drinking, mobilising, with negative pressure dressing laparotomy. His laparotomy epithelialized over time, and the pressure dressing was removed. He was discharged home on oral antibiotics for a pseudomonas wound infection. He will be evaluated as an outpatient by the plastics team regarding abdominal wall reconstruction options. At four months follow up this gentleman is doing well, and living independently.

Discussion and conclusion

Necrotising fasciitis is globally recognised as a dangerous and virulent pathology, carrying a mortality rate greater than 20% [1]. It is defined as an infection of the deep soft tissues, progressing rapidly to tissue necrosis and systemic sepsis. Presentation is usually with pain, swelling/erythema, and fever, and surgical crepitus in the absence of trauma is pathognomonic. Pain out of proportion to clinical signs should prompt closer inspection and investigation, whilst the presence of shock is a bad prognostic indicator. Three-dimensional imaging and/or tissue biopsy (including fascial layers) can also be helpful where diagnosis is unclear. High risk groups include diabetics, cirrhotics, and the immunocompromised.

The clinical course of necrotising fasciitis progresses rapidly despite the use of antibiotics, and requires urgent resuscitation and surgical debridement. Attention to repeated re-examination of the wound (with further debridement if necessary), and aggressive nutritional support can improve prognosis [2].

The patient described in this case report presented to the emergency department in septic shock, with few clinical signs and an inability to give a medical history. A systematic approach to resuscitation and management, with the early involvement of intensive care physicians and surgeons, saved his life. It is unknown if his use of subcutaneous or intravenous medications played a part in the aetiology of his disease, but it is suspected that an infected deep venous thrombosis may have played its part.

Learning points

1. Necrotising fasciitis can present in an atypical fashion, and is a life threatening surgical emergency.
2. The presence of surgical crepitus on examination is not always present in atypical cases, and urgent cross-sectional imaging can be helpful when the diagnosis is in doubt. The presence of air in subfascial muscle planes is diagnostic.

3. The treatment of necrotising fasciitis must always include resuscitation with fluid and antibiotics, and early involvement of intensivist physicians is important. Definitive treatment requires emergent surgical debridement.

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Correspondence: Katherine J Williams, Surgical Registrar, Kingston Hospital NHS Trust, UK, Tel: +44 20 8546 7711; E-mail: k.williams@ic.ac.uk

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