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Group A streptococcal toxic shock syndrome from a traumatic myositis

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Group A streptococcal toxic shock syndrome (StrepTSS) was defined in 1993 by the Working Group on Severe Streptococcal Infections in response to rising cases of severe and life-threatening streptococcal infections worldwide.[1] However, streptococcal myositis presenting as septic shock after blunt injury is rare.[2]

We report StrepTSS in a previously healthy 22-year old Caucasian male who 2 days earlier suffered a blunt injury while playing soccer.

Case report

The patient had seen a doctor on day 1 post-injury and was prescribed diclofenac. He presented to the hospital on day 2 with a systolic blood pressure (SBP) of 60 mmHg, tachycardia of 120 beats/min and tachypnoea of 28 breaths/min. He had right upper quadrant and right flank tenderness with no bruising or external wounds. The initial impression was hypovolaemic shock secondary to retroperitoneal haemorrhage or hepatic injury.

Computed tomography (CT) showed right-sided retroperitoneal oedema and fractures to four right lumbar transverse processes. No solid organ or hollow viscous injury was identified (Figure 1). Table 1 lists the patient's admission bloods.

Figure 1. CT scan images. Blue arrow indicates areas of retroperitoneal oedema

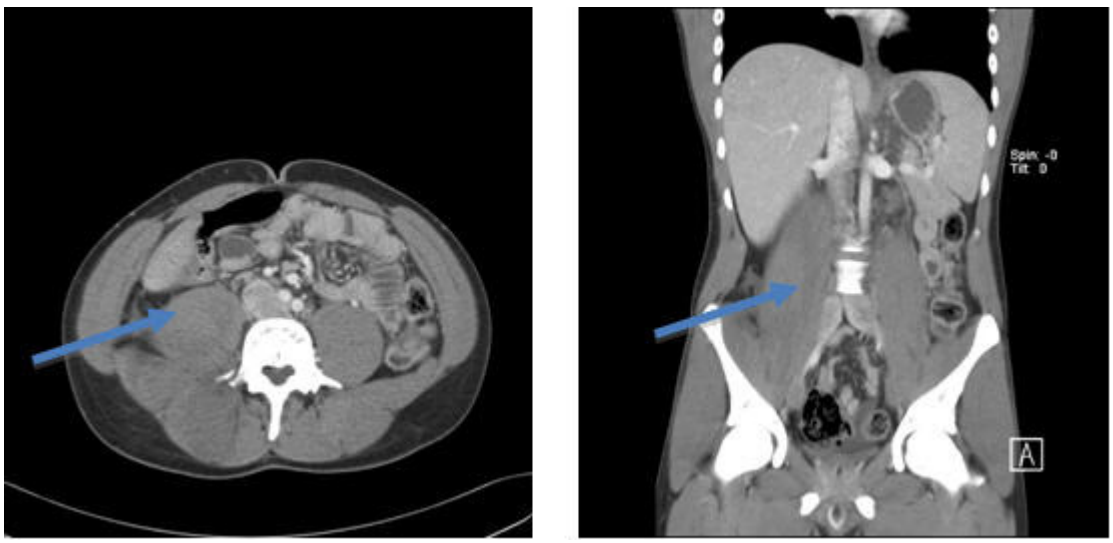

Table 1. Patient's admission blood results

Bloods	
Haemoglobin	130
White cell count	11.8
Myelocytes	0.1
Band neutrophil	3.6
Prothrombin ratio	1.4

Despite fluid resuscitation, SBP remained at 60 mmHg. Seven hours after admission, the patient's temperature rose to 37.8 degrees Celcius. His abdomen became distended but remained non-peritonitic. Due to the history of blunt trauma, abdominal distension and septic shock, the working diagnosis changed to septic shock secondary to small bowel injury.

Laparoscopy revealed fibrinous material in the right upper quadrant, however the subsequent exploratory laparotomy excluded intraperitoneal solid or viscous injury. Straw-coloured fluid from peritoneal cavity was sent for microbiology analyses. Despite antibiotics, he deteriorated with overwhelming sepsis leading to multiorgan failure requiring inotropes, ventilatory support and dialysis.

The following morning, the patient's lower abdomen and scrotum became cellulitic. A repeat CT scan showed no definitive evidence of necrotising fasciitis. However, to exclude a necrotising infection, the right retroperitoneum and scrotum were explored. Extensive oedema was found in the retroperitoneum and scrotum. Muscle and fascia were unremarkable. Retroperitoneal fluid was sent for microbiology analyses.

Eventually, intraperitoneal and retroperitoneal fluid grew *Streptococcus pyogenes*, a type of group A streptococcus. Histologically, debrided tissue revealed acute inflammation of right psoas muscle and scrotal wall.

With the commencement of intravenous penicillin, clindamycin and aztreonam, the patient made a gradual recovery and was discharged from the intensive care unit at day 9. Total hospital length of stay was 19 days.

Discussion

Non-penetrating trauma has been reported as a risk factor for StrepTSS.[3] Vimentin, a 57 kD intermediate filament protein, has been reported to be the primary skeletal-muscle surface protein in injured muscle which binds to group A streptococci.[4] Moderate injury increases surface expression of vimentin.[4]

One to 2 days post-injury, regenerating muscle cells and infiltrating immature muscle-cell precursors maximally express surface vimentin facilitating the adhesion of group A streptococci.[4] Consequently, the organisms proliferate, elaborating potent cytotoxins that cause further cell injury.[4]

Maximum upregulation of surface vimentin (up to 8-fold) is reported to occur at 48 hours after initial insult, and this correlated to our patient's time of presentation.[5] Hamilton et al proved that nonsteroidal anti-inflammatory drugs (NSAIDs) further enhance binding of group A streptococci on injured muscle.[5] Our patient's treatment with NSAIDs for musculoskeletal pain may have contributed to the development of StrepTSS.

Delay in making the correct diagnosis and subsequent treatment initiation can contribute to poor outcome in StrepTSS.[2] StrepTSS has mortality rates of up to 70% and significant morbidity from emergent amputation, extensive surgical debridement and prolonged hospital stay.[2,6]

Our patient's initial presentation with hypotension with abdominal pain clouded the diagnosis. This case should remind clinicians that StrepTSS should be considered in the event of blunt injury with delayed shock (48 hours) in patients with normal haemoglobin and NSAIDs use.

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