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Toxic shock syndrome following inguinal hernia repair: a rare condition

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ABSTRACT

A 25-year-old man developed fulminant multisystem failure 28 hours after elective repair of an inguinal hernia. Toxic shock syndrome (TSS) was diagnosed. The patient recovered fully with supportive care in ICU, antibiotics, and IV human immunoglobin . To the best of our knowledge, only one case of TSS following inguinal hernia repair have ever been previously published.

INTRODUCTION

Toxic shock syndrome is a severe systemic illness characterized by shock, pyrexia, an erythematous rash, gastrointestinal disturbance and central nervous system signs including lethargy or irritability. It is mediated by toxins produced by some strains of bacteria, most commonly *Staphylococcus aureus* or *Group A Streptococcus*. To the best of our literature search, only one case of TSS following inguinal hernia repair have been published.[1] So, this case is being reported for its rarity.

CASE REPORT

A 25 year aged young man; with American Society of Anesthesiologist (ASA) physical status 1 underwent hernioplasty under spinal anesthesia for left sided indirect incomplete inguinal hernia.

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On first post operative day, patient tolerated oral liquid diet during daytime. But in the evening, patient complained of 4-5 episodes of vomiting & loose stool, generalised bodyache and decreased urine output. On examination patient though conscious and oriented, had tachycardia(pulse rate was 110/min) and low blood pressure (80 / 60 mmHg) and conjunctival chemosis. He developed erythematous macular rash mainly over abdominal wall. Wound site was dry and appeared healthy.

Patient was resuscitated with IV fluids. Failure to maintain blood pressure necessitated use of inotropes (dopamine).

On second post operative day patient developed full blown picture of staphylococcal toxic shock syndrome

- GI manifestations – vomiting diarrhoea
- Hepatic Manifestation –'! Bilirubin (T-6mg, D- 3.8mg)

- Renal Manifestation – Creatinine (2 mg/ dl)
- CNS Manifestation – drowsiness +
- Cpk – Nac '!
- Thrombocytopenia – platelets count (48000/mm3)
- Sun burn rashes, Septic Shock
- Chemosis
- Fever – 102 oF

and anti-Staphylococcal antibiotics(Injection Vancomycin and injection Clindamycin) were started. On the third postoperative day platelet count further decreased to 19000/cu mm. Subconjunctival hemorrhage (fig.1) purpuric rashes and bruises especially localized to the soles (fig. 2)were evident. Persistent fever more than 100^{o} F, tachypnoea (respiratory rate more than 40/min), failure to maintain oxygen saturation inspite of Fio2 and bilateral infiltrate on chest x-ray necessitated intubation and mechanical ventilation under sedation under neuromuscular blockade. Pooled human immunoglobin was administered at a dose of 5 gm stat followed by 0.01ml/kg infusion over a period of half an hour and 0.06ml/kg infusion over 8 hours. Blood and stool culture did not grow any organism. Surgical wound was healthy and dry. The antibiotics were readjusted to Clindamycin, Ciprofloxacin and Cefepime. Since there was no clinical or radiological improvement of the ongoing sepsis, on an empirical basis glycopeptide antiobitic Teicoplanin, Ceftazidime and Tobramycin were started. The patient responded and he showed a gradual but remarkable recovery within the next few days. On the 11th day of ICU stay, after 9 days of mechanical ventilatory support, patient could be gradually weaned off and he was successfully extubated. On the 16th postoperative day patient was discharged from the hospital. On follow up after 15 days patient had near total recovery.

Legends to Figures

Figure 1: subconjunctival hemorrhage

Figure 2: Purpuric rashes and bruises on the soles

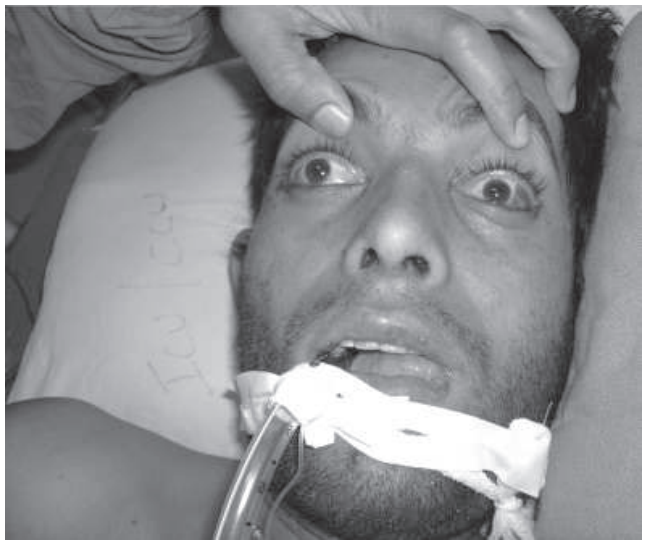

Fig 1

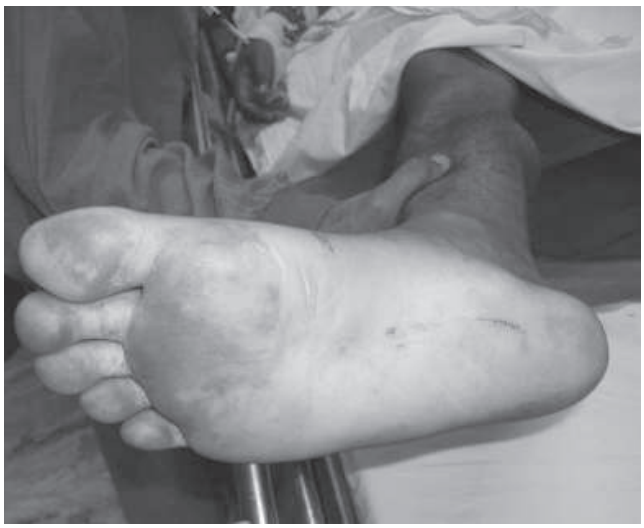

Fig 2

DISCUSSION

Todd et al.[2] first described toxic shock syndrome (TSS) in 1978 among seven children with an infection caused by *Staphylococcus aureus*. The present case meets all the criteria proposed by Centers for Disease Control and Prevention for diagnosis of TSS.[3]

Both Streptococcus and Staphylococcus species cause shock through the elaboration of toxins. Differing strains of *Staphylococcus aureus* produce TSS toxin 1 and staphylococcal enterotoxin B and C, all of which act as superantigens. These indiscriminately bind and activate T-cells, which lead to the amplification of cytokines TNF-[alpha] and IL-1; together, these cytokines cause fever, hypotension, and tissue injury.[4]

The treatment of post operative TSS involves early wound exploration and excisional debridement, coupled with aggressive fluid resuscitation often in the intensive care unit. Antistaphylococcal antibiotic agents should be given intravenously. However, antibiotic therapy alone may not alter the clinical course because most symptoms of post operative TSS are caused by the toxin, not by the bacteria; therefore, the intravenous administration of gamma globulin or corticosteroids has also been advocated.[5]

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