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Case Report

Obstetrics and Gynaecology Section

A Case of Postpartum Methicillin-resistant Staphylococcus Aureus Toxic Shock Syndrome: A Stitch in Time

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ABSTRACT

Toxic Shock Syndrome (TSS) is a sudden, systemic, toxin-induced disease that leads to the failure of multiple organs. It is a potentially fatal illness that might occur in the postoperative period. A 30-year-old woman in her second pregnancy, who had given birth via elective Lower Segment Caesarean Section (LSCS), presented to the Emergency Department on the tenth day after the surgery with septic features. She complained of a high-grade fever persisting for eight days, along with a rash that initially appeared on her upper limbs and gradually spread to her entire body. During the local examination, vulval excoriation with desquamation and peeling of the skin were observed. The imaging revealed a well-defined collection with heterogeneous enhancement in the infraumbilical region of the lower anterior abdominal wall. Multiple intralesional free air foci, primarily involving the bilateral rectus abdominis muscles, were also observed, along with mild fat stranding. These findings suggested an infective origin for the condition. The patient was stabilised and managed in the Surgical Intensive Care Unit (SICU). In order to combat the infection, broad-spectrum antibiotics with coverage against gram-negative bacteria were initiated. Subsequently, the patient developed a wound gap and underwent secondary wound closure. The present case report highlights the importance for healthcare practitioners to recognise the symptoms of postpartum TSS and take proactive measures to prevent its potential complications. Methicillin-resistant Staphylococcus Aureus (MRSA) is an iatrogenic and life-threatening infection; hence, proper treatment with a multidisciplinary approach will prevent maternal morbidity and mortality.

Keywords: Lower segment caesarean section, Postpartum infection, Surgical site infection

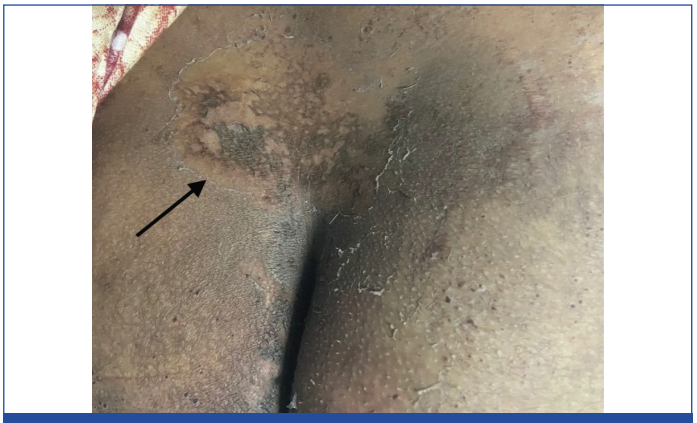
CASE REPORT

A 30-year-old P2L2A1 woman presented to the Emergency Department with septic features on postoperative day 10 of elective LSCS, complaining of a high-grade fever for eight days associated with a rash that appeared on her upper limbs and progressed to involve her entire body. The rash was associated with itching and excoriation. Additionally, she experienced abdominal distension that was insidious in onset and gradually progressive for four days, along with 4-5 episodes of non projectile, non bilious vomiting. The patient had undergone an elective LSCS 10 days ago at a private hospital, and the immediate postoperative period was uneventful. She was a known case of pregnancy-induced hypertension and had been on antihypertensives for the last three months. Her past medical history was not significant, except for a previous caesarean section three years ago.

On examination, she was conscious but uncooperative and disoriented. She had a pulse rate of 108 bpm, blood pressure of 84/50 mmHg, and SpO_2 of 97% on room air. She was icteric. Abdominal examination revealed an abdominal wall induration of 10×11 cm around the LSCS stitch line. The incision was well healed with no evidence of infection. Local examination revealed vulval excoriation [Table/Fig-1] with desquamation and peeling of the skin [Table/Fig-2]. It also involved the medial aspect of the thigh and extended up to the lumbar region on the back side. Vaginal examination revealed no active bleeding. Per urethral catheterisation was present, and there was no abdominal drain.

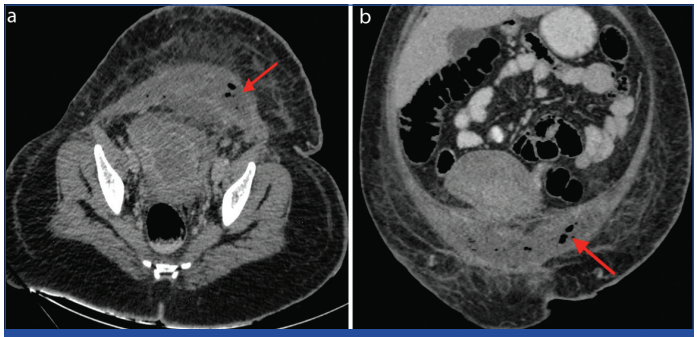
Her blood parameters revealed haemoglobin of 10.8 g/dL, white blood cells at 35,400/µL, platelets at 47,000/µL, D-dimer at 2718, procalcitonin at 1.68, serum urea at 79 mg/dL, alkaline phosphatase at 294 U/L, serum albumin at 2, total protein at 4.5, C-reactive protein at 188 mg/L, conjugated bilirubin at 5.81 mg/dL, and unconjugated bilirubin at 2.39 mg/dL, suggestive of hyperbilirubinaemia. A

[Table/Fig-1]: Vulval excoriation.



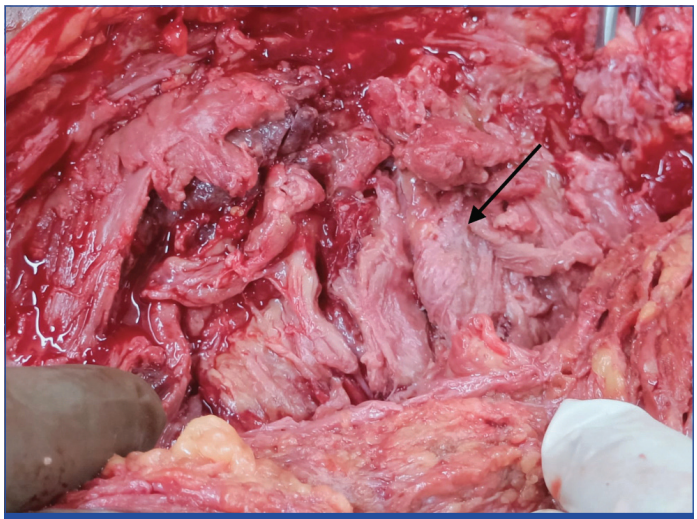
[Table/Fig-2]: Desquamation and peeling of the skin.

Contrast-enhanced Computed Tomography (CECT) scan of the abdomen and pelvis revealed a fairly well-defined, heterogeneously enhancing collection in the infraumbilical region in the midline of the lower anterior abdominal wall with multiple intralesional free air foci predominantly involving bilateral rectus abdominis muscles with adjacent mild fat stranding-findings suggestive of an infective aetiology [Table/Fig-3a,b].

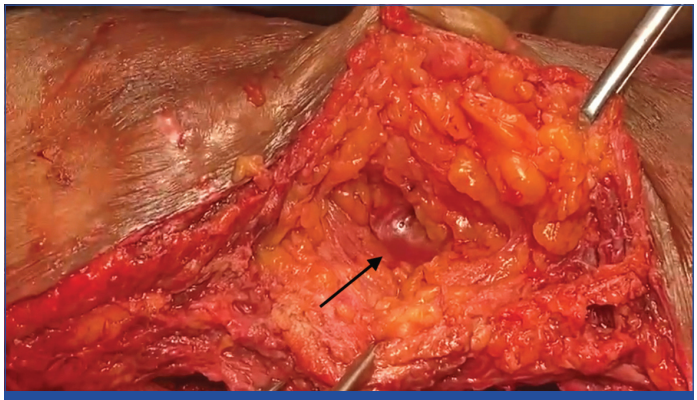


[Table/Fig-3]: a) Axial section and b) Coronal section of CECT of the abdomen and pelvis. The red arrowhead denotes heterogeneously enhancing collection in the rectus abdominis muscle with hypodense air foci and surrounding fat stranding.

The patient was managed and stabilised in the SICU, and broad-spectrum antibiotics with gram-negative coverage were initiated. All relevant investigations were conducted, and after consultations with the intensivist, anaesthetist, and surgeon, an emergency exploratory laparotomy was deemed necessary. The abdomen was opened using a Pfannenstiel incision, and the rectus sheath was separated, revealing offensive-smelling pus beneath the rectus sheath along with necrosed and devitalised rectus abdominis muscle [Table/Fig-4] and the presence of a rectus abdominis muscle abscess [Table/Fig-5].



[Table/Fig-4]: Necrosed and devitalised rectus abdominis muscle.



[Table/Fig-5]: Rectus abdominis muscle abscess.

The abscess was treated by surgically draining the purulent material. Vigorous debridement was performed until vital tissue was encountered, followed by a warm saline wash. Pus swab and necrosed parts of the muscle were sent for culture and sensitivity testing. One unit of packed red blood cells and two random donor plasmas were administered intraoperatively. The patient tolerated the procedure well. The pus culture report indicated MRSA producing Toxic Shock Syndrome Toxin-1 (TSST-1) sensitive to cotrimoxazole, vancomycin, and linezolid. Therefore, she was diagnosed with TSS caused by a rectus abdominis muscle abscess and treated with culture-sensitive antibiotics. The patient's clinical condition improved rapidly postoperatively, and all her blood investigations normalised.

However, on postoperative day 10 of the exploratory laparotomy, the patient developed a wound gap and underwent secondary wound closure. The pus culture showed no growth. She was discharged on postoperative day 10 after the secondary wound closure, following suture removal, with a healthy stitch line.

DISCUSSION

The TSS is characterised by a sudden onset of symptoms, including fever, rash, low blood pressure, multiple organ system dysfunction, and skin peeling. The prevalence of TSS is roughly estimated to range from 0.8 to 3.4 cases per 100,000 individuals [1]. This uncommon but serious condition is typically triggered by different bacterial strains, with group A *Streptococcus or Staphylococcus aureus* being the most frequently implicated pathogens [2].

Phillips C and Walsh E opined that early recognition of signs and symptoms and aggressive intervention are critical to decrease the risk of mortality. However, unlike our case, theirs was caused by Group-A streptococcus in the postpartum period [3]. Postpartum TSS typically manifests soon after childbirth, although instances have been documented as late as two months following delivery [4].

Multiple strains of *S. aureus* have been distinguished; yet, in contrast to other bacteria, a significant portion of *S. aureus's* TSS stems from an exotoxin, dubbed TSST-1 [5].

The clinical manifestations of TSS are caused by bacterial toxins that induce a massive release of cytokines, resulting in a systemic inflammatory response, leading to severe organ damage and death.

Severe TSS can occur as a result of MRSA infections during the puerperium. It is well-documented that TSS can manifest during the initial postoperative phase following different surgical procedures [6].

The TSS has a rapid, dramatic, and fulminant onset. There exist six clinical criteria that enable the diagnosis of TSS without the need for evaluating streptococcal infections. These criteria include fever exceeding 38.9°C, the presence of a rash, desquamation of the skin, hypotension, and the involvement of at least three organ systems. These organ systems encompass the gastrointestinal, muscular, mucous membranes, renal, hepatic, haematologic, and central nervous systems [7]. Furthermore, it is crucial to consider the absence of any evidence indicating an alternative cause for the illness. The confirmation of TSS can be achieved through the application of specific laboratory criteria, involving obtaining negative results for Rocky Mountain spotted fever, measles, or leptospirosis in blood, throat, and cerebrospinal cultures. It is important to note that blood cultures may exhibit positive results for *S. aureus* [8].

TSS caused by a Staphylococcus infection commonly presents in individuals without underlying health conditions through symptoms such as elevated body temperature, hypotension, fatigue, and cognitive impairment, which may swiftly advance to organ dysfunction and mortality. Characterised by a sunburn-like rash that can affect various body areas, the condition typically emerges promptly and leads to skin peeling within a few days [9].

In this instance, the patient satisfied the clinical criteria for TSS, as evidenced by the presence of a fever, rash, hypotension, and the involvement of a minimum of three organ systems (vomiting, elevated liver enzymes, acute renal disturbance, and thrombocytopenia), thereby confirming the diagnosis of TSS.

Thorough evaluation is essential, as delayed identification of TSS is linked to higher rates of illness and death [10]. Quick and timely recognition of the syndrome is important for enabling appropriate and prompt treatment. Its diagnosis depends on a high degree of clinical suspicion. It is crucial to promptly identify TSS to ensure the administration of suitable treatment. This treatment should encompass immediate drainage of the affected area to regulate the continuous production of toxins, the utilisation of antimicrobial therapy that specifically targets *S. aureus* (such as clindamycin, which inhibits protein synthesis and further toxin production, and vancomycin if MRSA is the responsible organism), and supportive therapy involving fluid resuscitation and pressor support, if necessary [7]. Thus, early diagnosis should be made, and doctors should be familiar with the manifestations of TSS.

In the initial hours, it is important to consider surgical intervention if the patient's condition worsens despite antibiotic treatment, as the possibility of clostridial infection cannot be excluded. Hysterectomy has been associated with the successful resolution of infection in many instances [11].

CONCLUSION(S)

All women should be administered a prophylactic dose of antibiotics during the perioperative period to mitigate the potential risks associated with caesarean section. In cases where patients with puerperal pyrexia fail to show improvement despite receiving suitable treatment, it is crucial to consider the possibility of other complications. The possibility of TSS should be ruled out in a case of sepsis following an emergency procedure, and a timely call for an exploratory laparotomy should be made if conservative management is not successful. Timely surgical intervention is needed. If not attended to aggressively, the infection can spread rapidly and be life-threatening. The present case report emphasises the need for early recognition of symptoms of sepsis in the postoperative period and aggressive intervention to prevent its detrimental effects and reduce the morbidity and mortality associated with it.

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