Revista Portuguesa de Doenças Infecciosas
Relato de Casos Clínicos
Relato de Casos Clínicos / Case Reports 2026 – Volume 20 – Número 1

# Toxic Shock Syndrome due to Group A *Streptococcus* infection in an Intensive Care Unit: Case Report

***Síndrome do choque tóxico por infeção por Streptococcus do grupo A numa Unidade de Cuidados Intensivos: A propósito de um caso clínico.***

Ana Catarina Gonçalves[1], Teresa Valido[2], João Patrício[3], Hugo Inácio[3], André Gordinho[3], Ângela Simas[3], Carlos Pereira[3].

[1] Department of Infectious Diseases, Hospital de Curry Cabral, Unidade Local de Saúde de São José, Lisbon, Portugal.
[2] Department of Internal Medicine, Hospital Professor Doutor Fernando da Fonseca, Lisbon, Portugal.
[3] Department of Intensive Care, Hospital Beatriz Ângelo, Lisbon, Portugal.

**Autor correspondente: Ana Catarina Gonçalves** **Email: acatarina.rg@gmail.com**

**DOI:**10.65332/rpdi.v20.126 **Recebido: 24 Ago 2025** **Aceite: 28 Jan 2026** **Publicado: 03 Mar 2026**

## ABSTRACT

**Introduction:**Toxic shock syndrome (TSS) is a rare but potentially fatal condition caused by exotoxins produced by *Streptococcus pyogenes* or *Staphylococcus aureus*. Streptococcal toxic shock syndrome (STSS) is characterized by rapid clinical deterioration and requires prompt recognition, aggressive supportive care, and multidisciplinary management.

**Case Presentation:** We report the case of a 63-year-old previously healthy Angolan man visiting Portugal who presented to the Emergency Department with a seven-day history of painful swelling of the left thigh and inguinal region, unresponsive to non-steroidal anti-inflammatory drugs (NSAIDs). He rapidly developed septic shock with multiorgan failure, requiring invasive mechanical ventilation, vasopressor support, and renal replacement therapy, and was admitted to the Intensive Care Unit. Blood cultures were negative, but *Streptococcus pyogenes* was isolated from a skin biopsy culture, supporting the diagnosis of STSS. The patient improved with targeted antibiotic therapy and was extubated after five days. Subsequently, he developed severe upper gastrointestinal bleeding from a bleeding antral ulcer, complicated by refusal of blood transfusion, and was managed conservatively.

**Conclusion:** STSS is a fulminant and life-threatening condition. Early diagnosis, aggressive organ support, and multidisciplinary care are essential. NSAIDs use may exacerbate disease severity and contribute to gastrointestinal complications. Refusal of blood transfusion poses major clinical and ethical challenges in the management of critically ill patients.

**Keywords:** Toxic shock syndrome; Intensive Care Units; *Streptococcus pyogenes*.

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## RESUMO

**Introdução:**A síndrome do choque tóxico (TSS) é uma entidade rara, mas potencialmente fatal, causada por exotoxinas produzidas por *Streptococcus pyogenes* ou *Staphylococcus aureus*. A síndrome do choque tóxico estreptocócico (STSS) caracteriza-se por rápida deterioração clínica, exigindo reconhecimento precoce, suporte intensivo agressivo e abordagem multidisciplinar.

**Descrição do Caso:** Apresenta-se o caso de um homem angolano de 63 anos, previamente saudável, que se encontrava em Portugal e recorreu ao serviço de urgência por dor e tumefação da coxa esquerda e região inguinal com sete dias de evolução, sem resposta a anti-inflamatórios não esteróides (AINEs). Evoluiu rapidamente para choque séptico com falência multiorgânica, necessitando de ventilação mecânica invasiva, vasopressores e técnica de substituição renal, sendo internado em Unidade de Cuidados Intensivos. *Streptococcus pyogenes* foi isolado numa biópsia cutânea, confirmando o diagnóstico de STSS. Após antibioterapia dirigida, o doente foi extubado ao quinto dia. Posteriormente, desenvolveu hemorragia digestiva alta por úlcera antral hemorrágica, associada à recusa de transfusão sanguínea, tendo sido tratado de forma conservadora.

**Conclusão:** A STSS é uma condição fulminante e potencialmente fatal. O diagnóstico precoce, o suporte intensivo e a abordagem multidisciplinar são determinantes para a sobrevivência. Os AINEs podem agravar a gravidade da doença e contribuir para complicações gastrointestinais. A recusa de transfusão sanguínea constitui um desafio clínico e ético significativo.


**Palavras-Chave:** Síndrome do choque tóxico; Cuidados Intensivos; *Streptococcus pyogenes*.

## Introduction

Toxic shock syndrome (TSS) in adults is a rare condition that can lead to acute and progressive multi-organ failure. TSS is caused by exotoxins produced by Group A *Streptococcus* (GAS) or *Staphylococcus aureus* and can be fatal if not recognized and treated promptly[1]. These exotoxins act as superantigens, triggering nonspecific, polyclonal T-cell activation and an uncontrolled immune response, resulting in a cytokine storm. This mechanism underlies the typical clinical manifestations of TSS — high-grade fever, erythroderma, and capillary leak — which can lead to hypotension and subsequent multi-organ failure[2]. Prospective, population-based surveillance studies from Europe and Australia report an incidence of invasive GAS infections of approximately 3 cases per 100,000 inhabitants per year[3]. Among these patients, 13.0 – 15.0% develop streptococcal toxic shock syndrome (STSS). In some series, the reported mortality rate of STSS ranges from 23.0% to 44.0%[4,5]. The portal of entry for *Streptococci* remains unknown in nearly half of the cases. When identified, infection most commonly begins at a site of minor local trauma[6]. The main risk factors for STSS include diabetes mellitus, alcohol dependence, malignancy, human immunodeficiency virus (HIV) infection, heart disease, and narcotic drug use[7]. Clinical criteria for STSS are based on the definitions of the Centers for Disease Control and Prevention (CDC), as summarized in Table I[8].

TSS is rarely associated with positive blood cultures[9]. Results from cultures of needle aspirations from inflamed skin are highly variable, ranging from ≤5.0% to approximately 40.0% in reported series[9]. Skin biopsy cultures yield an organism in 20.0%–30.0% of cases, although bacterial counts are typically low in cellulitis[10]. Admission to the ICU is typically required for patients with STSS[9]. Management includes treatment of septic shock and its associated complications, surgical debridement of the infection when indicated, antimicrobial therapy, and, in some cases, administra-

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tion of intravenous immunoglobulin (IVIG). The use of IVIG may be considered in patients with severe STSS who do not respond to other therapeutic measures, although its efficacy remains controversial[11]. Hemoperfusion in STSS is less well documented, though its use in septic shock is more established[12].

TSS cases frequently require coordinated care from a multidisciplinary team, including specialists in Critical Care, Surgery, and Infectious Diseases[13].

**Tabela I.** Clinical criteria for Streptococcal toxic shock syndrome, based on CDC definitions[8].

| **Streptococcal toxic shock syndrome** | |
|---|---|
| **Mandatory criteria (both required)** | |
| Isolation of GAS | From a normally sterile site. |
| Hypotension | Systolic blood pressure $\leq$ 90 mmHg in adults refractory to adequate volume resuscitation. |
| **Additional criteria ($\geq$2 required)** | |
| Renal dysfunction | Creatinine $\geq$ 2 mg/dL ($\geq$177 $\mu$mol/L) for adults, or >2× the upper limit of normal for age. If preexisting renal disease: >2× increase above baseline. |
| Respiratory distress | Acute onset of diffuse pulmonary infiltrates and hypoxemia in the absence of cardiac failure, or evidence of diffuse capillary leak (generalized edema, pleural/peritoneal effusions with hypoalbuminemia). |
| Hepatic dysfunction | ALT, AST, or total bilirubin $\geq$2× the upper limit of normal for age; if preexisting liver disease: >2× increase over baseline. |
| Coagulopathy | Platelets $\leq$ 100,000/mm$^3$ ($\leq$100 $\times$ 10$^6$/L) and/or disseminated intravascular coagulation. |
| Erythroderma | Erythroderma $\pm$ desquamation. |
| Soft tissue necrosis | Pain, tissue destruction, or skin discoloration. |

Abbreviations: GAS, Group A *Streptococcus*; ALT, alanine aminotransferase; AST, aspartate aminotransferase

## Case Presentation

We present the case of a 63-year-old Angolan man with a medical history notable only for an appendectomy performed 10 years earlier. He had been in Portugal for ten days to attend a religious meeting. The patient was a Jehovah's Witness and refused any form of blood transfusion on religious grounds.

He presented to the Emergency Department with a primary complaint of pain and swelling in his left thigh and inguinal region, which had started seven days prior and had not improved with non-steroidal anti-inflammatory drugs (NSAIDs). He reported sustaining an injury to the fourth and fifth toes of his left foot on a metal gate before leaving Angola. The patient denied fever, chills, malaise, or any respiratory, urinary, or gastrointestinal symptoms, and reported no history of risky sexual behavior.

On physical examination, he was tachycardic (136 beats per minute), hypotensive (95/81 mmHg), and tachypneic (30 breaths per minute). Examination revealed a painful, swollen area with erythema and warmth over the left lower abdomen, involving the inguinal region and extending down the anterior and medial aspect of the left thigh (figure 1), as well as evidence of prior trauma to the fourth and fifth toes of the left foot (figure 2).

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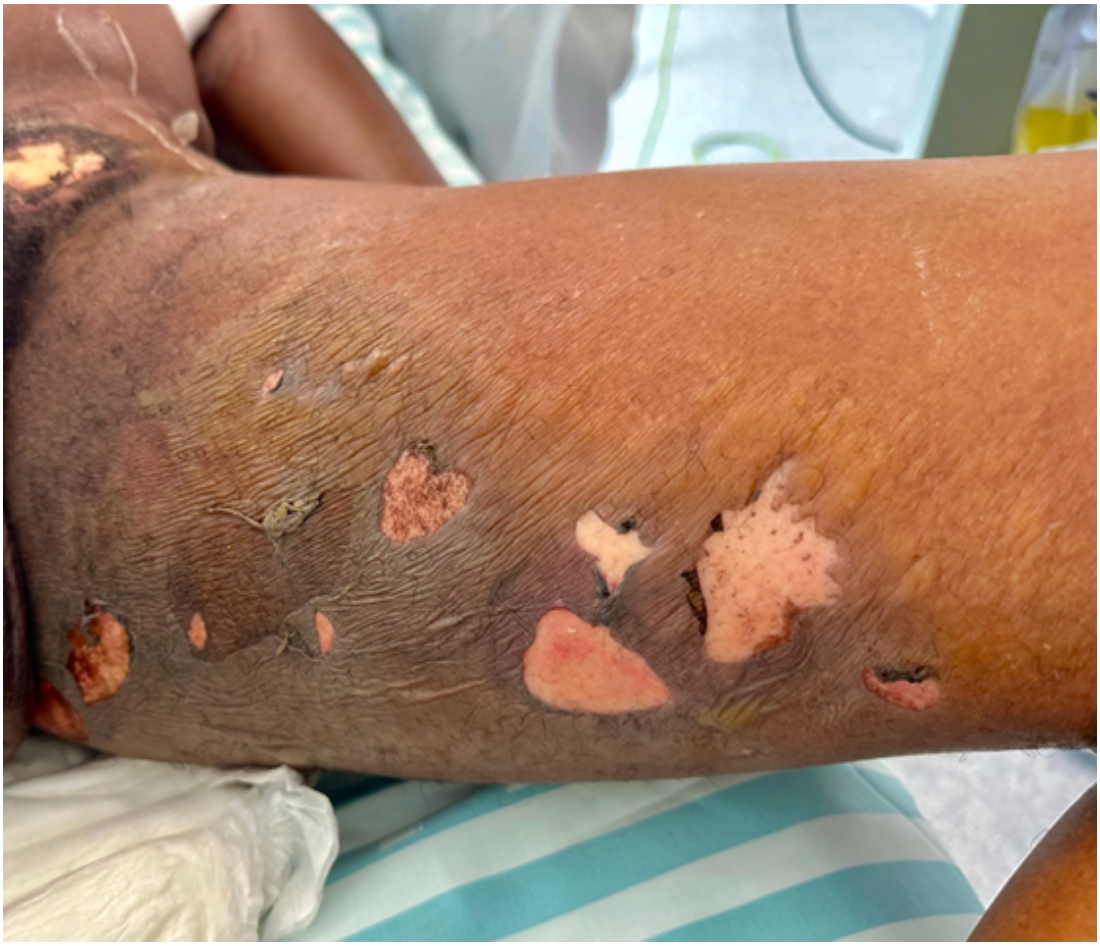

**Figura 1.** Lesion of the left inguinal region and left lower limb on the second day of hospitalization. Photograph showing erythema, swelling, and signs of inflammation extending from the left inguinal region to the proximal thigh and lower limb. The lesion is associated with pain and tenderness on palpation.

Laboratory tests revealed elevated C-reactive protein (416 mg/L), serum creatinine (3.2 mg/dL), and creatine kinase (6379 U/L). Arterial blood gas analysis showed metabolic acidosis. A contrast-enhanced computed tomography scan of the abdomen, pelvis, and left thigh demonstrated edema adjacent to the left femoral and iliac vessels and an ill-defined left inguinal collection measuring 2.5 cm. Cardiac ultrasound revealed a reduced ejection fraction (35.0%) without vegetations.

The patient was evaluated by the General Surgery team and did not meet criteria for drainage. He was started on fluid resuscitation and empiric antibiotic therapy with piperacillin/tazobactam. The patient rapidly deteriorated in the ED, developing respiratory, renal, and hemodynamic dysfunction, which required invasive mechanical ventilation and vasopressor support with norepinephrine (maximum 60 $\mu$g/min) and adrenaline (maximum 0.08 $\mu$g/kg/min).

He was admitted to the ICU on the same day, with an Acute Physiology and Chronic Health Evaluation II (APACHE II) score of 13. Upon ICU admission, antibiotic therapy was switched to amoxicillin/clavulanate 2.2 g and clindamycin 800 mg every eight hours. Corticosteroid therapy (hydrocortisone 50 mg every six hours) was also initiated. Due to prolonged anuria and severe metabolic acidemia, continuous renal replacement therapy was required. The patient gradually improved, with normalization of fluid balance and renal function after 48 hours.

Over the subsequent days, the patient's skin lesions progressed, with the development of diffuse erythema and desquamative changes, characterized by peeling of the superficial skin layers, particularly in the inguinal region and along the left lower limb (figure 3).

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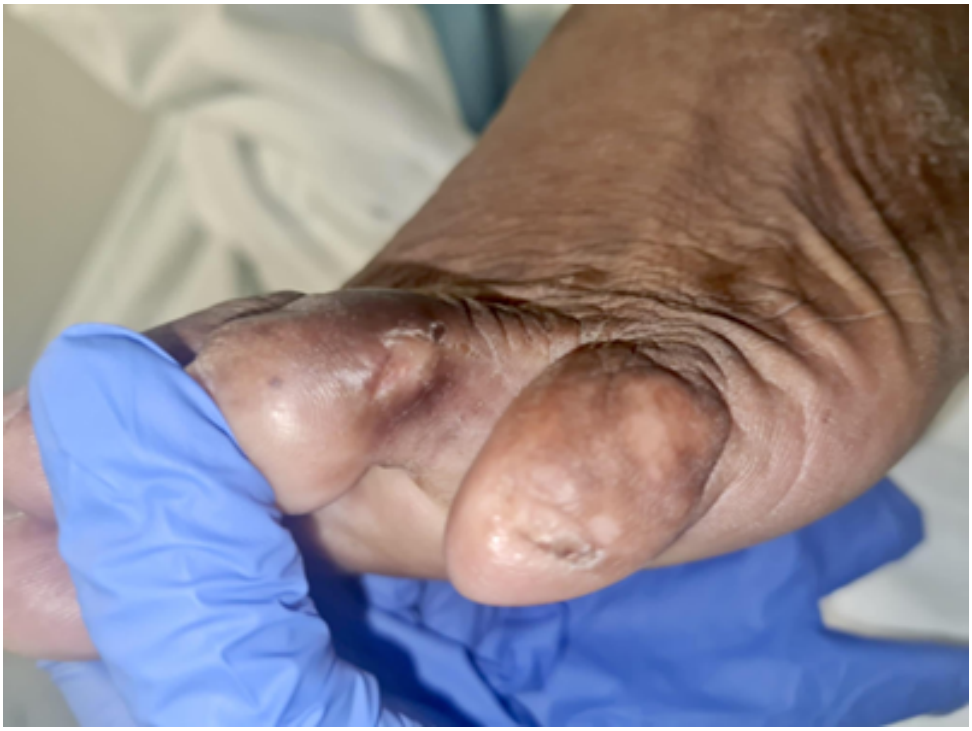

**Figura 2.** Local trauma on the fourth and fifth toes of the left foot on the day of admission. Photograph showing superficial abrasions and minor lacerations on the fourth and fifth toes of the left foot, corresponding to the reported injury from a metal gate before hospitalization.

**Figura 3.** Progression of skin lesions in the left inguinal region and lower limb on the third day of hospitalization. Photograph showing marked erythema, swelling, and extension of inflammatory changes in the left inguinal region and proximal lower limb compared to admission.

Blood cultures and all other microbiological tests were negative, prompting a punch biopsy of the skin on the third day of hospitalization. Histology showed leukocytoclastic vasculitis, intravascular thrombosis, and focal necrosis of epidermal keratinocytes. *Streptococcus pyogenes* was isolated from

the skin biopsy culture, confirming a diagnosis of STSS.

Another contrast-enhanced computed tomography scan of the abdomen, pelvis, and left thigh was performed on the third day of hospitalization and showed no worsening findings. The patient gradually improved with antibiotic therapy. Over the course of hospitalization, the erythema and swelling in the left inguinal region and lower limb slowly subsided, with no formation of abscesses or necrotic tissue, and he continued to have no indications for surgical debridement after repeated evaluations by the General Surgery and Plastic Surgery teams.

He was successfully extubated after five days. On the day of extubation, in addition to receiving a proton pump inhibitor (PPI) for stress ulcer prophylaxis, an upper gastrointestinal endoscopy was performed due to an episode of melena without hemodynamic instability (Blatchford score 13). The procedure revealed an acute antral ulcer with a bleeding vessel, which was treated with three hemoclips. The patient received PPI infusion for 72 hours, followed by high-dose oral PPI (pantoprazole 40 mg twice daily). A progressive decline in hemoglobin was observed, reaching a nadir of 5 g/dL. As the patient was a Jehovah's Witness, he refused blood transfusions; his anemia was therefore managed with intravenous iron and folate. Despite this profound anemia, the patient remained hemodynamically stable and never developed hemorrhagic shock.

After ten days in the ICU, he was transferred to a level 1 care unit for continuation of care, no longer requiring constant monitoring and with resolution of multiorgan dysfunction.

The patient was discharged after a 21-day hospital stay, clinically stable, with no organ dysfunction. He was referred for outpatient follow-up in Internal Medicine, Plastic Surgery, and Gastroenterology.

## Discussion

STSS is a rare but highly aggressive condition associated with significant morbidity and mortality, even in previously healthy individuals[9]. In nearly half of reported cases, the portal of entry remains unidentified, with infection often originating from minor or non-penetrating trauma[6].

The present case illustrates this diagnostic challenge, reinforcing the need for a high index of suspicion in patients presenting with rapidly progressive soft-tissue symptoms and systemic deterioration. Compared with previously reported cases, this patient exhibited a particularly fulminant course, with early progression to septic shock, multiorgan dysfunction, and acute renal failure requiring continuous renal replacement therapy. Despite negative blood cultures — a frequent finding in STSS — *Streptococcus pyogenes* was successfully isolated from a skin punch biopsy, underscoring the diagnostic value of tissue sampling when clinical suspicion is high. Histological evidence of leukocytoclastic vasculitis further supports infection-mediated small-vessel injury, a feature consistent with toxin-driven disease described in the literature[6].

Early initiation of combination antibiotic therapy with a $\beta$-lactam and clindamycin was pivotal to the favourable outcome. Clindamycin's ability to suppress exotoxin production and modulate host immune response is well documented and remains a cornerstone of STSS management[14]. This case reinforces current recommendations advocating early adjunctive clindamycin, even in the absence of microbiological confirmation. The patient's prior use of NSAIDs may have contributed both to delayed presentation and to increased disease severity, as suggested in previous studies linking NSAIDs use with more severe invasive streptococcal infections and gastrointestinal complications. In addition, although surgical intervention is often required in STSS, careful multidisciplinary assessment in this case supported a conservative approach, highlighting the importance of individu-

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alized decision-making based on infection extent, organ dysfunction, and bleeding risk.

This case also raises important ethical and therapeutic considerations. Refusal of blood transfusion in the context of life-threatening anemia posed a significant management challenge, necessitating alternative strategies. Similarly, intravenous immunoglobulin was not administered due to both clinical improvement and patient refusal, illustrating real-world limitations in applying guideline-supported therapies.

The added value of this case lies in its demonstration of successful STSS management despite negative blood cultures, absence of surgical intervention, and refusal of key supportive therapies. It highlights the diagnostic utility of skin biopsy, reinforces critical learning points regarding early antimicrobial strategies, and emphasizes the need for individualized, multidisciplinary, and ethically informed care in complex STSS presentations.

## Conclusion

This report illustrates a severe and life-threatening presentation of STSS, complicated by septic shock and multiorgan dysfunction, requiring invasive mechanical ventilation, vasopressor support, and continuous renal replacement therapy.

Although surgical debridement was not required, early clinical suspicion was essential for timely initiation and appropriate adjustment of antibiotic therapy. Clindamycin played a key role as an antitoxin agent in combination with $\beta$-lactam therapy, helping to suppress exotoxin production.

This case also highlights the limitations of negative blood cultures in ruling out STSS and the value of skin biopsy in establishing a definitive diagnosis.

Finally, the management of a patient refusing blood transfusions due to religious beliefs underscores the importance of ethical decision-making and individualized care in critical, life-threatening situations.

## Author Contributions

*Ana Catarina Gonçalves*: Conceptualisation; Methodology; Investigation; Formal Analysis; Data Curation; Writing – Original Draft; Writing – Review & Editing; Visualization.

*Teresa Valido and João Patrício*: Conceptualisation; Methodology; Investigation; Writing – Review & Editing.

*Hugo Inácio, André Gordinho, Ângela Simas, Carlos Pereira*: Supervision; Critical Review of the Manuscript.

## Funding

No funding was received for this study.

## Ethics Statement

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Written informed consent was obtained from the patient for publication of clinical details and images.

## Conflicts of Interest

The authors declare no conflicts of interest.

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