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When Sepsis Kills: A Fatal Cascade of Invasive Group A Streptococcal Sepsis

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Abstract

Presentation
A previously healthy 22-year-old woman presented with right groin pain, unilateral leg swelling, and dyspnoea following recent infection and minor trauma. She was hypotensive and tachycardic with a painful, mottled limb and oxygen saturations of 91%**.**

Diagnosis
Investigations demonstrated lactic acidosis, coagulopathy, pulmonary emboli, visceral infarcts, and necrotising soft-tissue infection. Cultures confirmed *Streptococcus pyogenes*.

Treatment
Despite antibiotics, emergency surgery, and advanced life support, the patient died of multiorgan failure**.**

Discussion
iGAS infection can mimic thromboembolic disease and requires urgent recognition.

Introduction

Invasive Group A Streptococcal (iGAS) infection is a rare but fulminant cause of necrotising fasciitis and multiorgan failure, even in previously healthy adults. Early diagnosis is challenging, as initial features may overlap with deep-vein thrombosis or cellulitis. This case illustrates the rapid progression and diagnostic complexity of iGAS sepsis.

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Case Report

A previously healthy 22-year-old woman presented to the Emergency Department with a two-day history of worsening right groin pain, progressive unilateral leg swelling, and new-onset dyspnoea. It was reported she had recently recovered from a self-limiting upper respiratory tract infection; she also had a minor scratch to the affected leg sustained while playing sport. She was on a combined oral contraceptive pill but had no other medical history.

On arrival, she was triaged as Category 2 and transferred directly to the resuscitation area. Observations showed a Glasgow Coma Scale of 14/15, heart rate 128 beats/min, unrecordable blood pressure, respiratory rate 34 breaths/min, oxygen saturations 91% on 15 L/min oxygen, and temperature 39.0 °C, with prolonged capillary refill (>3 s). The right lower limb was tense, markedly swollen, cool, and mottled, with absent distal pulses.

Initial laboratory investigations demonstrated severe metabolic, inflammatory, and haematological derangement: lactate 25 mmol/L, sodium 129 mmol/L, creatinine 221 µmol/L, phosphate 4.6 mmol/L, C-reactive protein 582 mg/L, white cell count 2.8 × 10^9/L, INR > 10, fibrinogen < 0.6 g/L, alanine aminotransferase > 4200 U/L, bilirubin 34 µmol/L, potassium 6.1 mmol/L, and severe metabolic acidosis. These findings were consistent with septic shock complicated by acute kidney injury, hepatic failure, and disseminated intravascular coagulation (DIC).

Given haemodynamic instability, hypoxia, unilateral limb swelling, and oral contraceptive use, pulmonary embolism was considered a leading differential diagnosis. CT pulmonary angiography, requested from the Emergency Department, demonstrated bilateral segmental pulmonary emboli.

She was admitted to intensive care for refractory shock. CT lower limb angiography demonstrated extensive soft-tissue swelling of the right thigh and calf with marked muscle enlargement and intramuscular gas within the adductor compartment, highly suggestive of necrotising fasciitis (Figure 1). A deep-vein thrombosis was present but deemed insufficient to explain the severity of limb swelling or systemic deterioration. CT of the abdomen and pelvis revealed multiple visceral infarcts involving the liver, spleen, and kidneys, confirming widespread thrombo-embolic and septic insult.

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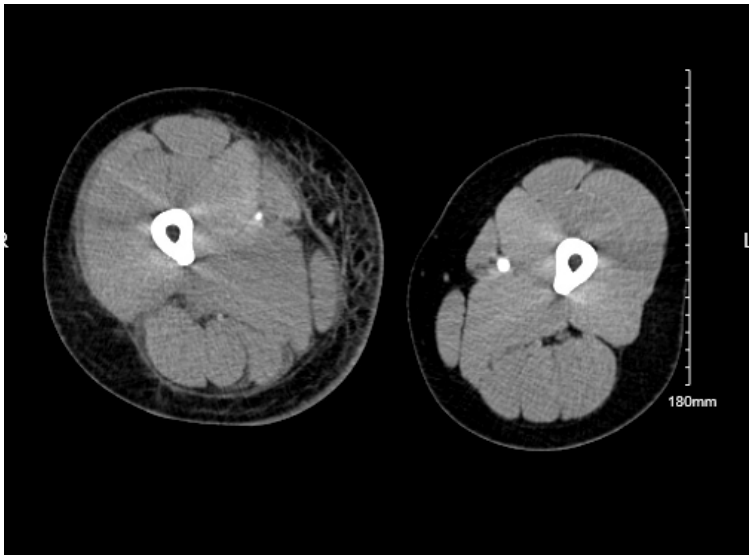

Figure 1: *CT lower limb angiogram showing extensive soft tissue swelling in the right lower limb with muscle enlargement with foci of air in the right adductor musculature suggestive of necrotising fasciitis. DVT was also found but determined unlikely cause of swelling.*

Shortly after ICU admission, the patient suffered two episodes of cardiac arrest (ventricular fibrillation and PEA) in the context of profound metabolic acidosis and circulatory collapse. Return of spontaneous circulation was achieved following advanced life support. She remained dependent on high-dose vasopressors and mechanical ventilation.

Despite maximal supportive therapy, she developed rapidly progressive multiorgan failure, including anuric acute kidney injury requiring continuous renal replacement therapy, worsening hepatic dysfunction with coagulopathy, and marked haematological deterioration. Platelet count fell from 277 × 10^9/L to 34 × 10^9/L, haemoglobin decreased from 15 g/dL to 7.3 g/dL, and white cell count rose to 51 × 10^9/L on the final day of life.

Urgent fasciotomy revealed extensive muscle necrosis without active bleeding, consistent with necrotising fasciitis, and both lower limbs were deemed non-viable. A laparotomy and cholecystectomy were performed due to gallbladder necrosis. Blood and wound cultures grew Streptococcus pyogenes, confirming invasive Group A Streptococcal infection. Empirical antimicrobial therapy with meropenem and linezolid was administered. Neurological status failed to improve, and CT brain demonstrated bilateral large-volume territorial infarcts with uncal and cerebellar tonsillar herniation (Figure 2). In the context of refractory shock, irreversible neurological injury, and multiorgan failure, care was redirected following multidisciplinary discussion. The patient died from multiorgan failure.

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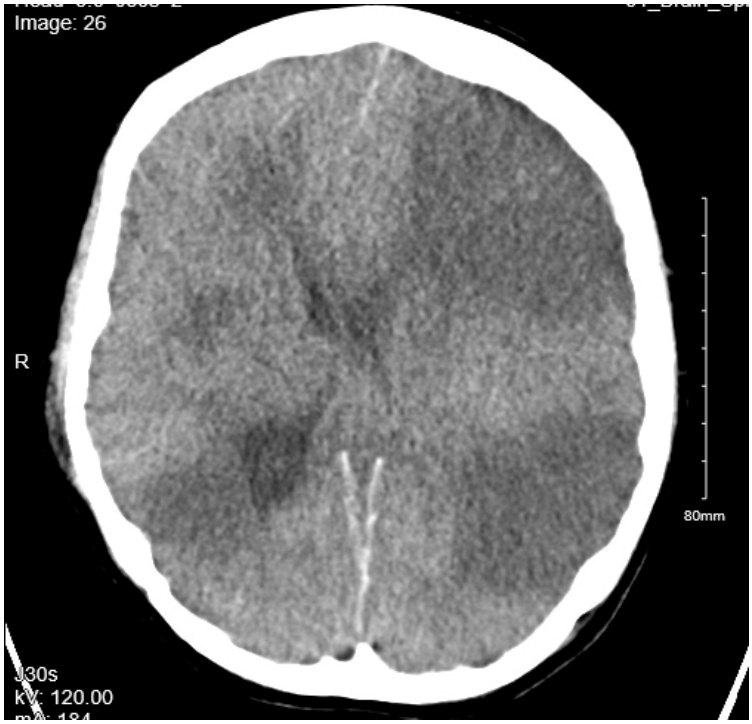

Figure 2: *CT Brain image showing bilateral large volume territorial infarcts and evidence of both uncal and cerebellar tonsillar herniation. Subcortical haemorrhages left vertex.*

Discussion

This case highlights the fulminant nature of iGAS infection in a previously healthy young adult following minor trauma. A small leg abrasion combined with preceding pharyngitis likely facilitated autoinoculation of Streptococcus pyogenes, a recognised mechanism in community outbreaks[1]. The initial impression of deep-vein thrombosis reflects a well-recognised diagnostic pitfall, as early necrotising soft-tissue infection can closely mimic thromboembolic pathology. Disproportionate pain, early haemodynamic compromise, and rapid systemic deterioration should prompt urgent imaging and early surgical review[2].

The coexistence of pulmonary emboli, visceral infarction, and DIC illustrates the intense thrombo-inflammatory response characteristic of severe sepsis[3]. Early leucopaenia followed by extreme leukocytosis and precipitous thrombocytopenia reflected rapid immune dysregulation and consumptive coagulopathy. Once cerebral oedema and herniation developed, the prognosis was non-survivable[4]. Early recognition, prompt radical debridement, and toxin-suppressive antimicrobial therapy remain the cornerstone of management.

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Declarations of Conflicts of Interest:
None declared.

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