Case Report

Trauma-Associated Purpura Fulminans in a Previously Healthy Child: A Case Report and Review of Literature from a Resource-Limited Setting

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ABSTRACT

Purpura fulminans (PF) is a rare but catastrophic thrombotic disorder characterised by rapidly progressive cutaneous necrosis, disseminated intravascular coagulation (DIC), and multiorgan dysfunction. Although commonly associated with meningococcal sepsis, recent evidences implicate a broader spectrum of bacterial and viral pathogens, including *Staphylococcus aureus* and *β-haemolytic streptococci*, usually following skin barrier disruption or trauma. Data from sub-Saharan Africa are limited. We report a previously healthy 4-year-old boy developed acute infectious Purpura fulminans (PF) following minor trauma to the left knee sustained during play. He presented about a week later, with high-grade fever, vomiting, and rapidly progressive petechiae with evolving ischemia and gangrene of the toes and features of multiorgan dysfunction. Laboratory findings were consistent with consumptive coagulopathy and systemic inflammation. Blood culture was sterile, likely due to antimicrobial exposure prior to sampling. The clinical course was strongly suggestive of a toxin-mediated bacterial septicaemia, most plausibly due to *Staphylococcus aureus*. The patient was managed with broad-spectrum antibiotics including vancomycin, blood product support, intensive supportive care, and close surgical monitoring. Limb perfusion recovered, and amputation was avoided with full recovery. This case highlights the need for high clinical suspicion of trauma-associated infectious PF in the Nigerian child, with the consideration of broader infectious spectrum. It reveals the importance of recognition, early empiric coverage for toxin-producing organisms like *Staphylococcus aureus*, and coordinated multidisciplinary care for optimal outcome. Future research should consider pathogen-specific mechanisms, improved specific diagnosis even after antibiotic exposure, and pragmatic strategies for managing severe pediatric sepsis in low-resource environments.

Keywords:, Disseminated Intravascular Coagulation, Gangrene, Microvascular Thrombosis, Nigeria, Paediatric Septic Shock, Purpura Fulminans

INTRODUCTION

In childhood, minor injuries are generally regarded as benign events, expected to heal spontaneously or with minimal wound care. This usual anticipation is because of both biological resilience and the assumption of timely access to basic health services. In many low-resource settings, however, this ideal course is frequently disrupted. Seemingly trivial trauma may serve as the portal for invasive infection, leading to severe systemic disease with devastating consequences. Purpura fulminans (PF) represents one of the most catastrophic examples of this progression, transforming an initially localized injury into a life-threatening emergency.

Purpura fulminans presents as one of the most severe manifestations of paediatric sepsis. It is a rare thrombotic disorder defined by the triad of disseminated intravascular coagulation (DIC), rapidly progressive cutaneous hemorrhage and necrosis, and multiorgan dysfunction[1-2]. Despite its rarity, PF carries a disproportionate burden of mortality and long-term morbidity, particularly in low- and middle-income countries where delayed presentation and limited access to paediatric intensive care are common[3-4]. Historically, PF has been closely associated with invasive *Neisseria meningitidis* infection in children and sometimes with congenital deficiencies of protein C or protein S in neonates[5]. However, contemporary evidence increasingly recognises PF as a final common pathway of overwhelming infection and endothelial injury rather than a pathogen-specific entity[6-7]. Invasive infections caused by *Staphylococcus aureus* and β-haemolytic streptococci have been reported to produce a clinically indistinguishable syndrome through superantigen release, cytokine amplification (cytokine storm), and profound dysregulation of coagulation pathways[8-9].

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In practice, however, the etiologic spectrum of PF is broader and more complex than often appreciated. Beyond *Neisseria meningitidis*, invasive infections caused by *Staphylococcus aureus* and β-hemolytic streptococci have been increasingly implicated, particularly in settings of skin breach, soft tissue infection, or trauma.[5,6,10] These organisms possess virulence factors, including exotoxins and superantigens, capable of inducing profound endothelial activation, cytokine storm, and activation of the coagulation cascade. This mechanism remains under-reported in African paediatric literature, where PF is often attributed solely to overwhelming sepsis without exploration of antecedent trauma or portal of entry[11,12]. Diagnostic uncertainty, frequent pre-hospital antibiotic exposure, and limited laboratory capacity further complicate microbiological confirmation in these settings[13].

The unpredictable course of PF poses a major clinical challenge in most settings. In theory, early recognition of sepsis, prompt antimicrobial therapy, and aggressive supportive care should halt progression to fulminant disease. But in practice, particularly in resource-poor regions such as Nigeria, delayed presentation, limited laboratory capacity, and restricted access to pediatric intensive care frequently undermine these ideals. Blood cultures are often negative due to poor yield, poor technics or lack of required equipment and prior empiric antibiotic use. Also, advanced therapies such as recombinant activated protein C are either unavailable or unaffordable.[14] These result to high burden of preventable mortality and morbidity, including limb ischemia, gangrene, and amputation.

With respect to healthcare policy, PF exposes critical gaps in health systems. Much of the existing literature and clinical guidance is derived from high-resource contexts and meningococcal-dominant epidemiology, offering limited practical direction for clinicians in LMICs confronting atypical presentations. This diagnostic uncertainty perpetuates a cycle of late intervention and poor outcomes.

The survivors of PF occasionally face long-term disability, prolonged rehabilitation, and psychosocial distress, adding to the economic and emotional strain on families and already stretched health systems. In sub-Saharan Africa, where paediatric surgical and rehabilitation services are limited, limb-threatening complications carry especially profound implications[11]. These realities underscore the need to contextualize PF not only as a biomedical emergency but also as a public health and health policy concern.

This case report addresses a critical gap in the literature by documenting purpura fulminans in a 4-year-old boy following a minor fall, complicated by plausible *Staphylococcus aureus* septicemia. Unlike the majority of reported African paediatric cases linked to meningococcal disease, this report highlights trauma as a probable initiating factor and expands the recognized infectious spectrum of PF in resource-limited settings. By situating the case within existing theoretical frameworks of coagulation and endothelial dysfunction, and against the practical constraints of care delivery in Nigeria, the report illustrates the diagnostic and management challenges unique to such environments.

The aims of this study are twofold: firstly, to present a detailed clinical account of an unusual case of trauma-associated PF in a Nigerian child; and secondly, to critically review relevant literature to identify areas of consensus, divergence, and novelty regarding causative organisms, pathogenesis, and management. Thereby, the report seeks to inform clinical practice by reinforcing the need for early suspicion and empiric coverage for toxin-producing bacteria, and to inform policy by highlighting the importance of strengthening paediatric sepsis recognition and critical care capacity in low-resource settings.

CASE PRESENTATION

A previously well 4-year-old boy was admitted to the Emergency Paediatric Unit (EPU) with a 3-day history of high-grade fever, repeated vomiting, and rapidly progressive skin eruptions predominantly involving both lower limbs. The illness was preceded by a superficial injury to the left knee after a minor fall one week prior to presentation. Initial care at home was limited to washing the wound with clean water and wiping with methylated spirit. No topical or systemic medications were administered. No previous history of petechiae or purpuric rashes. (See Figure 1A, B, C and D)

Over the subsequent five days, the child developed persistent fever, (up to 40°C), associated with vomiting and pain and tenderness around the injured knee. His clinical condition worsened, with increasing lethargy and generalized bone and joint pains, prompting presentation to hospital. On admission, he appeared acutely ill, lethargic, pale, jaundiced with a necrotic lesion over the left knee (the site of injury), diffuse petechiae and ecchymoses of both lower limbs, and evolving gangrene of multiple toes. No signs of meningeal irritation. Hepatomegaly was present. A provisional diagnosis of overwhelming septicemia complicated by disseminated intravascular coagulation (DIC) was made, with meningococcemia and acute leukemia considered as differentials.

Laboratory investigations on admission revealed marked systemic inflammation and hematologic derangement, with marked leukocytosis of 34,000/μL, and severe thrombocytopenia of 30,000/μL, highly suggestive of consumptive coagulopathy. Prothrombin time and activated partial thromboplastin time were within normal limits at presentation. Blood cultures, taken on the third day of admission, subsequently yielded no growth, likely reflecting prior antimicrobial exposure. Malaria rapid diagnostic testing was positive, most likely a potential co-morbid stressor rather than the primary driver of illness[14]. Empirical broad-spectrum antimicrobial therapy with intravenous cefotaxime and gentamicin was commenced on admission. Vancomycin was added shortly thereafter in view of the rapidly progressive soft tissue involvement and the concern for toxin-mediated bacterial sepsis. (See Table 1).

Diagnosis and Management

Based on the constellation of rapidly progressive cutaneous necrosis, severe thrombocytopenia, and other

laboratory features of severe sepsis, a diagnosis of acute infectious purpura fulminans was made. Given the antecedent skin trauma and aggressive soft tissue involvement, toxin-mediated *Staphylococcus aureus* infection was considered the most plausible aetiology[15,16].

The child was managed in isolation with intensive monitoring and multidisciplinary input. Intravenous vancomycin (to ensure coverage for methicillin-resistant *S. aureus*) and cefotaxime were continued, with plans for de-escalation guided by microbiological data, although this was ultimately limited by negative culture result.

Supportive care included cautious fluid resuscitation, supplemental oxygen, and fresh whole blood transfusions in lieu of unavailable platelet concentrates and fresh frozen plasma. The child, in the course of the illness developed transient multiorgan dysfunction, including acute kidney injury, respiratory distress with severe hypoxemia and sepsis-associated hepatocellular injury, but improved with sustained supportive care. Surgical teams performed serial assessments and limited debridement, confirming gradual restoration of tissue perfusion. The clinical hematologist reviewed the patient and supported the diagnosis and management. He was discharged after a two-week hospital stay in stable condition, with no requirement for surgical amputation. (See Figure 2, and Table 2)

This case illustrates the severe and unpredictable course of purpura fulminans following minor trauma in a child, and highlights both the diagnostic challenges and adaptive management strategies required in resource-constrained settings.

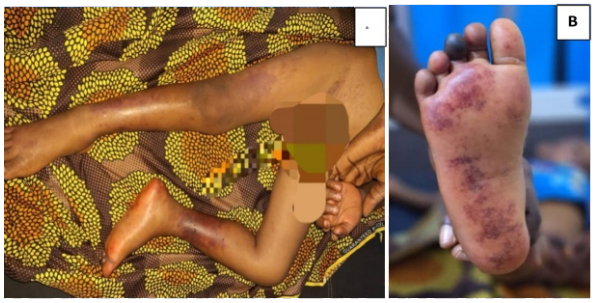

Figure 1. Petechial hemorrhages and ecchymosis(A, B, C and D)

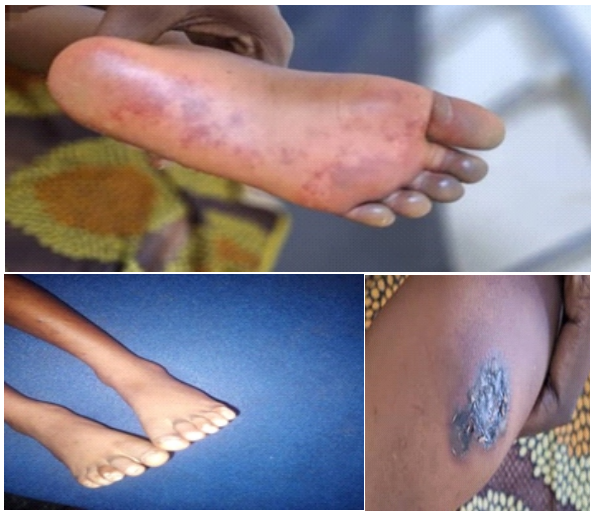

Figure 2. Both feet after treatment with no gangrene.

Table 1. Clinical Presentation and Laboratory Indices of the Patient

Category	Parameter	Values/Findings	Comments
Clinical Presentation	Injury site	Left knee injury from fall	Initial mild and bruising.
	Time of onset of symptoms	5 days after injury	High grade fever, vomiting, lethargy, worsening pains
	Skin manifestations	Petechiae, ecchymosis, gangrenes of the toes.	Rapid progression purpura fulminans
	Organ dysfunctions	Multi-organ failures	Liver, renal, pulmonary requiring renal support and Oxygen therapy
Laboratory Findings	Renal function test	Elevated urea and creatinine(mild)	Suggestive of Acute kidney injury
	Urinalysis	Proteinuria+,	
	White Blood Cell Count	34,000/µL	Markedly elevated; suggestive of bacterial infection
	Packed Cell Volume	25.8%	Moderate Anemia
	Platelet Count	30,000/µL	Severe thrombocytopenia associated with DIC
	Total Bilirubin	178.2mmol/L	Deranged liver functions.
	Cong Bilirubin	43.5mmol/L	
	Total Protein	61g/L	
	Albumin	31g/L	
	Aspartate Transaminase	115 IU/L	
	Alanine Transaminase	80 IU/L	
	Prothrombin Time	Test 18sec, Control 18sec	Normal clotting time
	Activated Partial Thromboplastin Time	Test 38sec, Control 41sec.	
	Malaria parasite (RDT)	Positive	
	HIV, HBsAg, Anti HCV.	All negative	
	Throat swab M/C/S	Gram stain - No bacterial seen. Culture - No pathogen isolated	

Table 2. Serial Haematological Parameters in the course of admission.

Day on admission	WBC (x 10^9/L)	RBC (x 10^9/L)	HGB(g/dL)	Platelets (x 10^9/L)	HCT (%)
1	35.7	3.53	9.58	9	25.8
4	30.1	4.07	11.0	27	29.9
7	20.5	4.51	13.6	40	37.6
14	12.5	4.51	12.3	106	36.0

LITERATURE REVIEW

Purpura fulminans (PF) represents one of the most severe thrombotic microangiopathies encountered in paediatric infectious disease practice. It is defined by the abrupt onset of haemorrhagic skin necrosis, disseminated intravascular coagulation (DIC), and rapidly progressive multiorgan dysfunction. PF carries a disproportionate burden of mortality and long-term disability, particularly in low- and middle-income countries where delayed recognition and limited access to advanced supportive care remain pervasive.[17,18] While meningococcal sepsis has historically dominated the literature, emerging evidence supports a broader and more complex aetiological landscape, including trauma-associated bacterial sepsis, most notably due to *Staphylococcus aureus*.[5,6,10].

Trauma as a Precipitating Factor in Purpura Fulminans

Purpura fulminans is classified into three clinical entities: acute infectious PF, post-infectious or idiopathic PF, and PF associated with congenital or acquired deficiencies of natural anticoagulants such as protein C and protein S.[2,19,20] Infectious form of PF predominates in children, with *Neisseria meningitidis* accounting for the majority of reported cases globally. However, trauma-associated PF has been increasingly recognised as a distinct and underappreciated clinical trigger.

High-income settings studies commonly describe PF developing after seemingly trivial injuries, superficial lacerations, or localized soft tissue infections.[5,7,10] These cases support a pathophysiological model which suggests endothelial disruption from trauma facilitating bacterial invasion and local inflammatory activation, subsequently leading to systemic coagulopathy. Other studies have proposed that trauma may unmask latent vulnerabilities in the coagulation pathway, including subclinical deficiencies of protein C or transient acquired anticoagulant depletion during sepsis.[6,10]

In contrast, African literatures on trauma-associated PF are rare. Some studies from Nigeria, Kenya, and South Africa largely attribute PF to overwhelming sepsis without detailed exploration of antecedent trauma.[4,11,12] This disparity likely reflects under recognition rather than true epidemiological absence. Early PF lesions are most probably misdiagnosed as cellulitis, necrotising fasciitis, vasculitis, or uncomplicated post-traumatic bruising, resulting in delayed escalation of care or the delay of the needed wholistic management. Where trauma-associated PF is recognised early in high-resource settings, prompt intervention has been associated with limb salvage and improved survival.[5,10] In low-resource settings, however, presentation often occurs after the onset of gangrene and multiorgan failure, with reported mortality rates approaching 40–60%.[4,12]

Causative Organisms, Antimicrobial Resistance, and Treatment Delays

Timely initiation of appropriate antimicrobial therapy remains one of the most critical determinants of outcome in acute infectious PF. Multiple studies demonstrate a strong association between early antibiotic administration, ideally within the first hour of sepsis recognition, and improved survival.[3,21] However, this standard is often difficult to achieve when early symptoms follow minor trauma and are perceived as benign or self-limiting.

While *Staphylococcus aureus* is a common cause of paediatric skin and soft tissue infection, it is an infrequent but particularly aggressive trigger of PF. When implicated, the disease is often severe and progresses rapidly, driven by exotoxin-mediated endothelial injury, cytokine storm, and profound activation of the coagulation cascade.[15,22] Several case series have highlighted the role of virulent strains, including Panton–Valentine leukocidin-producing isolates, in precipitating PF.[9,22] Warkentin et al. described fulminant PF in children with *S. aureus* sepsis, many of whom deteriorated within hours despite early antimicrobial therapy, underscoring the organism's pathogenic potency.[9]

Some literatures document culture-negative purpura fulminans in which *Staphylococcus aureus* remains a likely pathogen, particularly following prior antibiotic exposure that may suppress bacterial growth[23,24]. Given its well-established virulence and superantigen-mediated inflammatory effects, the clinical pattern in some studies support a presumptive *S. aureus*–associated purpura fulminans despite negative cultures.[25]

The global rise of methicillin-resistant *S. aureus* (MRSA) has been associated with delays in effective therapy and worse outcomes in severe paediatric infections.[16,26] In sub-Saharan Africa, surveillance studies have indicated significant increase in resistance to first-line antistaphylococcal agents, alongside widespread empiric antibiotic use prior to hospital presentation.[1 3] Consequently, blood cultures could be negative, limiting pathogen confirmation and targeted therapy. African PF case reports infrequently include antimicrobial susceptibility data, representing a major gap in understanding microbial drivers of disease severity.[4,11,12]

Management of Purpura Fulminans in Resource-Limited Settings

The management of PF is essentially centred on rapid treatment of sepsis, correction of coagulopathy, haemodynamic stabilisation, and early surgical involvement. Recombinant activated protein C was explored as adjunctive therapy following early adult sepsis trials, but failed to demonstrate benefit and raised safety concerns in paediatric studies, leading to its withdrawal.[27] Current best practice relies on blood products, including fresh frozen plasma and platelets, alongside broad-spectrum antimicrobials and organ support.

In many African settings, these interventions are inconsistently available. Limited paediatric intensive care capacity, delayed access to blood products, and inadequate laboratory monitoring constrain optimal care delivery[4,11]. Also basic coagulation studies and other laboratory parameters monitoring unavailability, complicate disease monitoring. Nkambule et al. demonstrated improved survival with early multidisciplinary care and intensive monitoring, yet such models remain inaccessible to many children.[11]

Prevention strategies commonly receive little attention in PF literatures, despite their relevance. Vaccine-preventable infections, particularly meningococcal and pneumococcal disease, remain important PF triggers in Nigeria probably due to suboptimal immunisation coverage in some regions.[28] Also, community wound-care practices, delayed presentation, and unregulated antibiotic use contribute to progression from minor injury to severe sepsis, yet are rarely addressed in paediatric emergency frameworks.

Synthesis and Contribution of the Present Study

Collectively, most existing literatures reveal several consistent themes. PF secondary to *Staphylococcus aureus* is uncommon but associated with rapid progression and high morbidity when diagnosis is delayed. Trauma is an under-reported precipitating factor, particularly in African children. Antimicrobial resistance, delays in effective treatment and poor implementation of proper management principles significantly worsen outcomes ultimately especially in resource-limited settings.

One of the concerns in the literatures lies in the role of trauma. While Western reports increasingly recognise trauma-associated PF as a distinct clinical pathway, African publications largely attribute PF to classical septic causes, likely reflecting diagnostic and reporting gaps rather than true differences in disease biology.

The present study contributes to the literature by documenting a rare case of trauma-associated PF in a Nigerian child, highlighting early clinical signs that may facilitate timely recognition; drawing attention to the

probable role of *Staphylococcus aureus*; and contextualising management within the constraints of a low-resource health system. By integrating clinical, microbiological, and health-system perspectives, this report offers insights relevant to paediatric clinicians, researchers, and public health policymakers seeking to reduce preventable morbidity and mortality from this devastating illness.

DISCUSSION

The present case of a previously healthy 4-year-old boy who developed PF following minor knee trauma and presumed *Staphylococcus aureus* septicemia provides an instructive framework for examining disease mechanisms, contextualising clinical patterns, and drawing implications for practice and policy.

Pathophysiological Considerations

Several complementary theories support the pathogenesis of PF, which endothelial injury is central to its severity. The coagulation cascade model proposes that bacterial toxins trigger excessive activation of coagulation pathways, leading to consumption of platelets, and clotting factors, ultimately leading to DIC and tissue infarction[15,19]. Similar activation of inflammatory pathways, including complement activation and massive cytokine production, further amplifies endothelial damage and promotes microvascular thrombosis.[7] Likewise in congenital or acquired deficiencies of protein C or protein S, the loss of endogenous anticoagulant control predisposes to unchecked thrombin generation and fulminant thrombosis.[6,19]

This case aligns most closely with infection-driven coagulation dysregulation as the clinical course strongly supports an acquired mechanism precipitated by severe sepsis, culminating into a rapid onset of thrombocytopenia, evolving cutaneous necrosis, and multiorgan dysfunction. This following a localized injury, is consistent with toxin-mediated endothelial injury, a well-described feature of severe *S. aureus* infection.[10,20] Although congenital anticoagulant deficiency could not be definitively excluded due to limited laboratory capacity, there was no history of neonatal PF nor past history suggestive.

Consistency with Existing Literature

The clinical trajectory observed mirrors usual descriptions of PF: fever followed by petechiae, ecchymoses, and progression to gangrene [5,19]. PF evolves once systemic infection is established as elaborated by previous studies, with speed making it been referred to as a time-critical emergency in which delays of hours may determine survival or limb loss.[5] Also, Warkentin et al. characterised PF as a "final common pathway" of overwhelming sepsis, irrespective of the initiating pathogens.[9] This case study reinforces these clinical presentation, demonstrating that once the threshold of endothelial and coagulation collapse is crossed, clinical manifestations follow a remarkably consistent pattern.

Novelty and Points of Divergence

Where this case meaningfully differs from other scenarios is in its aetiological pathway. Trauma-associated PF remains rare, with few isolated paediatric cases reported globally.[6,10] The combination of minor trauma, presumed *S. aureus* septicemia, and PF in a child without known coagulation disorders challenges the prevailing perception that PF is almost meningococcal or genetically mediated. Also, the marked leukocytosis and severe thrombocytopenia at presentation followed a steady normalization with effective antimicrobial therapy, which is in keeping with the clinical resolution seen in acute infection–associated purpura fulminans with disseminated intravascular coagulation. In contrast, acute leukaemia would typically show persistent marrow dysfunction without cytotoxic treatment, and inherited protein C or S deficiencies would usually not demonstrate such rapid recovery solely with infection control.

While *Neisseria meningitidis* continues to dominate PF literatures, *S. aureus* and other pathogens are increasingly recognised as a cause of severe toxin-mediated syndromes, including PF, although infrequently.[29-32] When *S. aureus* is involved, as highly suggested in the case, disease progression is often rapid and severe.[27]. This case therefore broadens the microbial spectrum of PF and underscores the need for empiric antimicrobial strategies that extend beyond classical meningococcal coverage.

Contextual Factors in Resource-Limited Settings

Several contextual factors likely influenced the evolution of this case of PF. The delayed presentation following an inadequate home wound care, limited early diagnostic capacity, and empiric antibiotic exposure prior to blood culture sampling are common in low-resource environments as seen here, and complicate both recognition and microbiological confirmation. Rising prevalence of methicillin-resistant *S. aureus* in sub-Saharan Africa increases the risk of initial therapeutic mismatch[13,16], which informed our choice of antimicrobial coverage to mitigate the progression of septicaemia and coagulopathy.

Implications for Theory, Practice, and Policy

From a theoretical point, this case supports a syndromic rather than pathogen-restricted conceptualisation of PF. Purpura fulminans should be viewed as the end result of a combined pathogenesis (endothelial injury, inflammatory amplification, and consumptive coagulopathy) rather than a condition confined to specific organisms or inherited defects.

Clinically, this case report reinforces the need for high index of suspicion of PF in any child with septicaemic illness associated with evolving haemorrhagic skin lesions, regardless of antecedent trauma or presumed pathogen. Minor injuries accompanied by systemic symptoms should be carefully evaluated. To salvage limb and life in this situation, early commencement of treatment with appropriate broad-spectrum antibiotics with antistaphylococcal coverage, necessary supportive care, and multidisciplinary collaboration, including early surgical assessment are critical.

From a policy perspective, this case of PF exposes structural gaps in paediatric emergency and critical care services in low-income settings. Limited access to

coagulation assays, blood products, and intensive monitoring constrains optimal management as documented in other regions[11,12]. Strengthening laboratory capacity, ensuring availability of essential blood components, and improving early sepsis recognition protocols are achievable priorities even in resource limited settings. Preventive strategies, particularly improved vaccine coverage against invasive bacterial infections, remain central to reducing the burden of PF.

LIMITATIONS

This single-case report design limits generalisability, and negative blood cultures restrict microbiological certainty of this study. Also, the interpretation of this case without protein C and S assays precludes definitive exclusion of underlying anticoagulant deficiency (whether congenital or acquired), even though highly unlikely due to lack of supportive history. These limitations only reflect systemic constraints rather than methodological oversight and are themselves illustrative of challenges faced in sub-Saharan African settings like ours.

FUTURE DIRECTIONS

Future researches should prioritise prospective registries of PF in African children to better define epidemiology, microbial patterns, and outcomes. Traumatic injury studies exploring trauma-induced endothelial activation as a cofactor in PF will be reasonable. Comparative analyses of PF associated with different pathogens, particularly *S. aureus*, may clarify organism-specific trajectories. Finally, policy-oriented research evaluating the impact of improved sepsis pathways, antimicrobial stewardship, and immunisation coverage on PF incidence is essential.

CONCLUSION

This case report documents a rare and severe presentation of purpura fulminans in a previously healthy 4-year-old boy following minor trauma, complicated by probable *Staphylococcus aureus* septicemia. It demonstrates how PF can rapidly progress from localized injury to systemic coagulopathy, multi-organ dysfunction, and extensive necrosis. Beyond its clinical significance, the case expands the conceptual understanding of PF to include non-meningococcal bacterial pathogens and trauma as potential triggers. It underscores the critical need for early recognition, multidisciplinary management, and improved pediatric critical care capacity, including limited microbiological diagnostics, delayed access or unavailability of coagulation assays, and lack of advanced therapies particularly in resource-limited settings. Future research should focus on multicenter registries, mechanistic studies of trauma-associated endothelial dysfunction, and strategies to enhance outcomes in children with PF.

RECOMMENDATIONS

In general, clinicians should maintain a low threshold for suspecting *Staphylococcus aureus*–associated purpura fulminans after minor trauma in children, initiating immediate broad-spectrum antibiotics and sepsis care when likely. Healthcare systems in resource-limited settings must establish pediatric early warning tools, make available essential diagnostics, and create multidisciplinary referral pathways. And lastly, public education should emphasize rapid recognition of spreading purpura with fever as a medical emergency, thereby promoting urgent healthcare-seeking behaviour.

Conflict of Interest

The authors declare that there are no financial, personal, or professional conflicts of interest that could have influenced the content, interpretation, or conclusions of this manuscript.

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