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Septic Vasculitis as a Manifestation of Invasive Infection by Community-Acquired Methicillin- Resistant Staphylococcus aureus: A Pediatric Case

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ABSTRACT

Septic vasculitis (SV) is an uncommon complication of bacterial sepsis, characterized by inflammation and thrombosis of small- and medium-sized blood vessels. While it is commonly associated with meningococcemia, a few cases related to Staphylococcus aureus have been reported. Presented is the case of a previously healthy adolescent who developed SV as an initial manifestation of community-acquired Methicillin-resistant Staphylococcus aureus (CA-MRSA) bacteremia. The patient presented with fever, abdominal pain, and purpuric skin lesions, without any known exposure to typical risk factors. Initial clinical suspicion included loxoscelism-a necrotic arachnidism caused by Loxosceles spider bites-based on initial skin lesion morphology. However, further evaluation ruled out loxoscelism, autoimmune etiologies and confirmed MRSA infection through blood cultures and histopathologic analysis of a skin biopsy confirmed septic vasculitis. Imaging revealed secondary infectious foci in the spleen, kidneys, and bone, though no surgical intervention was necessary. The patient was manage with targeted antibiotic therapy, resulting in complete clinical recovery. This case underscores the importance of recognizing septic vasculitis as a rare but serious initial manifestation of invasive bacterial infection in otherwise healthy children. It highlights the diagnostic value of skin findings in febrile illnesses and emphasizes the need for prompt and comprehensive etiological investigation.

Keywords:

invasive bacterial infection, bacteremia, child, community acquired infection, methicillin resistant staphylococcus aureus (mrsa), pediatric, septic vasculitis

Introduction:

Cutaneous vasculitis remains a broad and varied group of conditions mainly involving the skin, characterized histopathologically by inflammation and necrosis of blood vessels. These conditions may evolve as primary idiopathic processes or occur secondary to underlying disease, medication, or infection [1].

A particular type, septic vasculitis (SV), develops in the context of bacteremia or sepsis and presentes clinically as purpuric, blistering, or necrotic skin lesions [2]. A wide range of infectious organisms-viruses, bacteria, and parasites-may also precipitate vasculitic syndromes. Although rare, septic vasculitis may appear as a first sign of a serious systemic infection. At present, drug treatment for primary skin inflammation of vessels relies mainly on

corticosteroids; however, if this inflammation is caused by bacteremia these medications could lead to a worse condition for this patients [3].

Methicillin-resistant Staphylococcus aureus (MRSA), commonly found in hospital settings, is increasingly prevalent in community environments. The infections can range from mild skin infections to bacteremia, endocarditis, osteoarticular and prosthetic device infections. It also triggers activation of the imune system that is fairly responsible for toxic shock syndrome. However, its association with vasculitis is poorly documented, particularly in pediatric populations [4,5].

On the other hand, loxoscelism is caused by the bite of Loxosceles spiders and is a public health concern in countries such as Argentina, Peru, Chile, and Brazil. The venom is dermonecrotic and viscerotoxic, leading to two clinical forms: a mild, localized cutaneous form, and a severe visceral form with systemic symptoms like hemolysis, jaundice, and renal failure [6].

This report describes the case of a previously healthy adolescent who developed septic vasculitis secondary to community-acquired MRSA bacteremia. The clinical, microbiological, histopathological findings, are presented to contribute to the medical literature on this unusual but potentially fatal presentation.

Case Presentation

A previously healthy 12-year-old male adolescent, residing in a rural area of Quilpué, Chile, with na epidemiological history of exposure to Loxosceles laeta spiders. The patient's initial presentation included localized pain and erythema, which subsequently evolved into a violaceous plaque on the hypothenar region of his right hand. Over the next few hours, he developed fever up to 39°C, abdominal pain, vomiting, and diffuse pain in the lower extremities.

He was initially evaluated at a local hospital approximately 12 hours after symptom onset, where vital signs were normal. A cutaneous lesion was localized on his right hand **(Figure 1B)**, with no other clinical findings. Laboratory tests showed moderate leukocytosis of up to 15,200/uL with a neutrophil count of 91%, mild elevation of inflammatory markers (C-reactive protein, CRP - 2.6 mg/dL [N: <1]), and mild proteinuria in the non-nephrotic range, without hematuria (The rest of the laboratory tests are summarized in **Table 1, Control1).** Due to suspected cutaneous-visceral loxoscelism, he was referred to our center. At 20 hours from symptom onset, he arrived at the emergency department in stable general condition, with persistent fever and fatigue. Repeat laboratory tests highlighted increased leukocytosis of up to 22400/uL with neutriphil count of 97% , elevated CRP 6.4mg/dL, and signs of proteinuria accompanied by positive hemoglobinúria (the rest of the laboratory findings are summarized in **Table 1, Control 2),** leading to hospitalization with the presumptive diagnosis of loxoscelism.

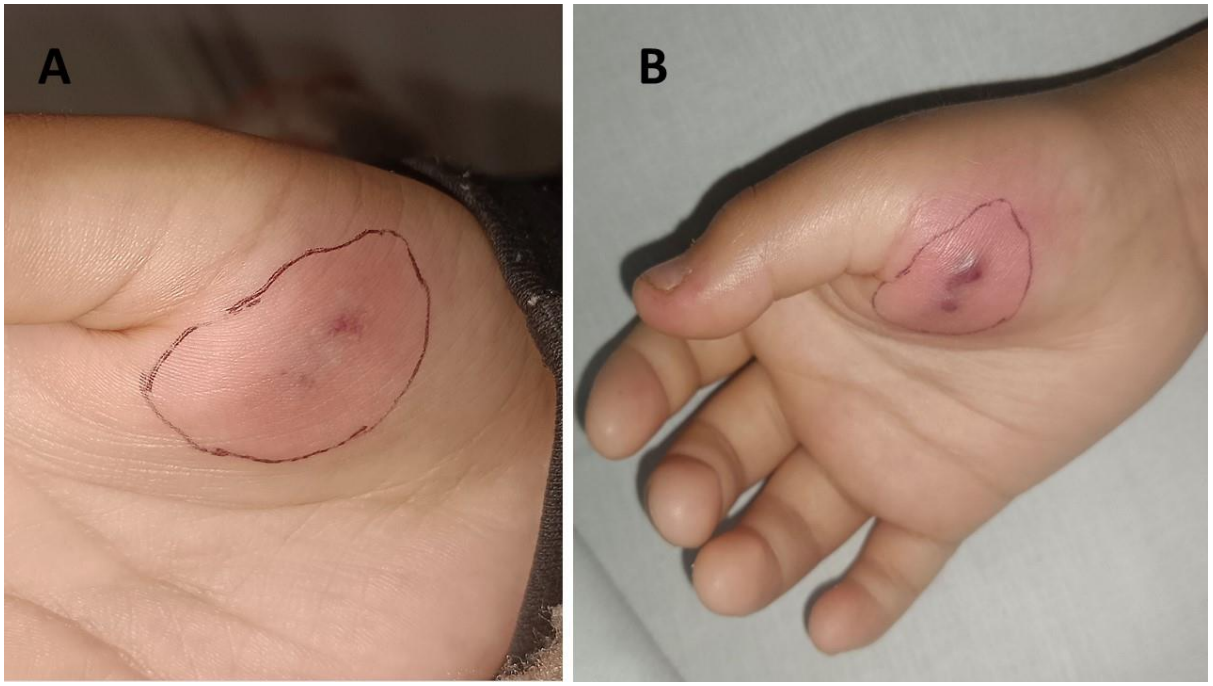

FIGURE 1: Initial presentation (A)Erythema with a slight central violaceous coloration, painful in the hypothenar region of the hand. (B)Evolution after 12 hours to palpable purpura. (photographs taken by the patient's legal guardian prior to hospitalization, who grants image rights and authorizes the publication of the image).

During the first 24 hours of hospitalization, the patient developed purpuric lesions on the fingertips of both hands and feet, as well as the heels, along with an increase in the size of the hand lesion **(Figure 2).** He also experienced persistent abdominal pain, and laboratory findings, summarized in **Table 1 (Control 3)**, revealed elevated inflammatory markers, abnormal liver function tests, and coagulation abnormalities, with normal fibrinogen levels. He was transferred to the Pediatric Intermediate Care Unit (PICU), where empirical antibiotic therapy was initiated.

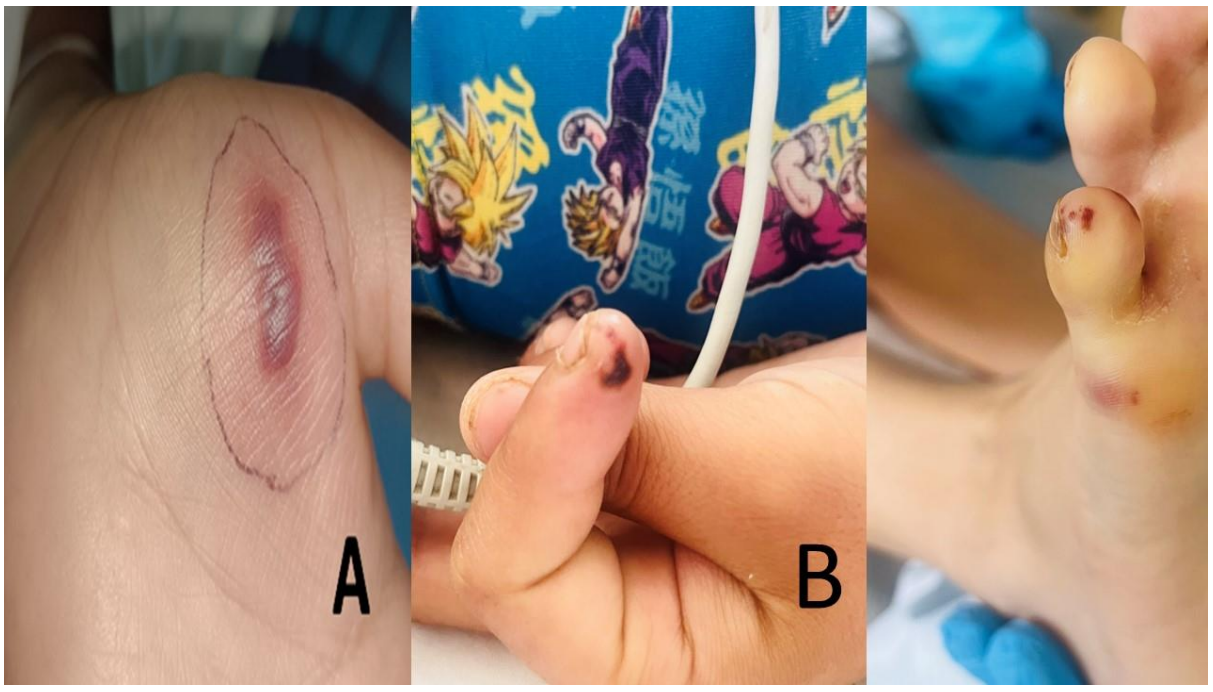

FIGURE 2: (A) Evolution of hand lesion: palpable purura with na increase in the size. (B) New purpuric lesions on the fingertip pulp of the hand. C) Purpuric lesions on the toe of the foot.

In the PICU, the diagnostic evaluation was expanded. Connective tissue disease panels-including rheumatoid factor, antinuclear antibodies, antineutrophil cytoplasmic antibodies, and antiphospholipid antibodies-were all negative; however, hypogammaglobulinemia and hypocomplementemia (low C3 and C4)were documented. Laboratory test results are summarized in **Table 1 (Control 4)**. Microbiological studies revealed blood cultures positive for methicillin-resistant Staphylococcus aureus (MRSA), and a skin biopsy demonstrated superficial and deep perivascular neutrophilic infiltrates, dermis with several small blood vessels exhibiting wall necrosis and luminal thrombosis; epidermal and dermal necrosis, subdermal pustulosis. No microorganisms were detected on Gram stain. Unfortunately, immunofluorescence was not performed.

Test Name	Control 1	Control 2	Control 3	Control 4	Normal Range
Hemoglobin (g/dl)	12.3	12,1	11.7	10	11 - 13.5
Hematocrit %	34	34.7	33	28.2	33 - 39
WBC x 10^9/L	15.2	22.4	22.7	18.8	5.0- 11.0
ANC %	90	97	95	95	25 70
Platelets (µL)	253000	257000	167000	150000	250000- 450000
CRP mg/dl	2.4	6.4	30.5	22.1	0.3 - 1
PT %		59	44	53	78 - 110
aPTT		28	31	33	21 -37
INR		1.3	1.7	1.45	0-97 – 1.2
Fibrinogen mg/dl				321	238-498
Creatinine mg/dl	0.4	0.69	0.59	0.69	0.25 – 1.0
BUN mg/dl		28		23	3 - 25
AST U/L	71	61	208	174	0 - 40
ALT U/L	34	40	174	199	4 - 35
GGT	24	28	96	133	9 - 48
IgA mg/dl				51	58 - 358
IgG mg/dl				338	596 - 1308
IgE UI/ml				11.5	0 - 87
C3 (mg/dl)				32	70 - 150
C4 (mg/dl)				5	13.5 - 45
Hemoglobinuria	(-)	(+)		(-)	(-)
Proteinuria mg/dl	15	Signs	30	30	Negative

TABLE 1: Summary of laboratory results during clinical evolution.

WBC: white blood cell; ANC: absolut neutrophil count; CRP: C-reactive protein; PT: prothrombin time; aPTT: activated partial thromboplastin time; INR: international normalized ratio; BUN: blood urea nitrogen; AST: Aspartate aminotransferase; ALT: alanine aminotransferase; SGPT: Gamma-glutamyl transpeptidase; IgA: Immunoglobulin A: IgG: Immunoglobulin G; IgE: Immunoglobulin E; C3: C3 complement; C4 complement.

Dissemination studies confirmed the presence of small splenic and left renal abscesses, as well as early signs of osteomyelitis in the distal left femur and distal right tibia. Other metastatic infections, such as endocarditis, were ruled out. The patient received targeted antibiotic therapy for 6 weeks, without the need for surgery and responded well clinically: fever resolved within 72 hours, subsequent blood cultures became negative, laboratory parameters normalized, skin lesions disappeared, and he was discharged without functional sequelae.

Discussion

Cutaneous vasculitis associated with severe bacterial infection accounts for approximately 1.5% of biopsyconfirmed vasculitis cases [1]. A study aimed to analyze cutaneous lesions in patients with bacterial septic vasculitis. In a cohort of 32 patients with bacterial sepsis, skin lesions, and biopsy-confirmed septic vasculopathy, cutaneous manifestations were the initial event in 90.6% of cases-a finding that may be critical for early diagnosis [7].

Neisseria meningitidis is the most common causative agent, followed by N. gonorrhoeae. However, cases have also been reported with Staphylococcus aureus, Streptococcus pyogenes, Streptococcus pneumoniae, Pseudomonas spp., and Rickettsia spp. [2].

The pathophysiology of this entity involves the following principal mechanisms: (a) disseminated intravascular coagulation, (b) direct invasion of the microorganism into the vascular wall, (c) immunemediated mechanisms, (d) septic embolism, and (e) action of bacterial toxins. More than one of these mechanisms may coexist in a single patient [8,9]. Clinically, septic vasculitis presents with palpable purpura, vesicular lesions, and bullae in acral areas. Histologically, neutrophilic vasculitis and occlusive thrombi composed of neutrophils, erythrocytes, platelets, and fibrin, can be observed with or without detectable bacteria within vessels [7].

In the presented case, acral purpuric lesions were early signs, followed by fever and systemic involvement. The skin biopsy showed intravascular fibrin thrombi and inflammatory infiltrate composed of neutrophils. Together with hypogammaglobulinemia and hypocomplementemia, these findings suggest a vascular phenomenon secondary to immune mechanisms and systemic inflammatory response. Techniques such as immunohistochemistry and polymerase chain reaction can facilitate pathogen identification, aiding diagnosis in complex or nonspecific cases [9].

This case highlights an unusual but severe manifestation of Invasive Infection by MRSA in pediatrics. Unlike entities such as Henoch-Schönlein purpura, systemic lupus erythematosus, or isolated leukocytoclastic vasculitis, the patient’s immunologic and complement profile, along with microbiological and histological evidence, confirmed septic vasculitis secondary to MRSA infection.

Conclusions

Septic vasculitis in pediatrics is an infrequent yet critical manifestation of bacterial sepsis. It should be considered in the differential diagnosis of purpuric skin lesions in patients presenting with fever and systemic symptoms, even in previously healthy children. Cutaneous lesions may represent the first clinical sign of occult bacteremia and should prompt a comprehensive etiological evaluation, including blood cultures and skin biopsy. A multidisciplinary approach and a high clinical suspicion are essential for the timely diagnosis and management of this potentially life-threatening condition.

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Ethics Approval and Informed Consent

This clinical case report was exempted from ethics committee approval as it involves the description of a single clinical case. However, informed consent was obtained from the patient’s legal guardian for the use of clinical data and photographs for the purpose of case presentation in a medical journal. All data submitted have been anonymized to ensure the patient’s confidentiality.

Conflict of Interest Statement

The author declares that there are no conflicts of interest.

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