RESEARCH ARTICLE

Symmetrical Peripheral Gangrene Without Shock: A Rare Manifestation of Legionella Pneumonia

Asif Dabeer Jafri[1*], Ishank Jaiswal[2], Muktesh Singh[3], Ratender Kumar Singh[4]

1. Department of Emergency Medicine and Tele-ICU Service, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India
2. Department of General Medicine, Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh, India
3.Department of Anaesthesia and Critical Care, Moti Lal Nehru Medical College, Prayagraj, Uttar Pradesh, India
4. Department of Emergency Medicine, Telemedicine and Digital Health, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India

Abstract

Symmetrical peripheral gangrene (SPG) is a rare and life-threatening condition characterized by symmetrical distal ischemia progressing to gangrene in the absence of large-vessel obstruction or vasculitis. It is most frequently associated with septic shock, disseminated intravascular coagulation, and the use of vasopressors. We describe an unusual case of SPG occurring in a hemodynamically stable patient with Legionella pneumonia. A 43-year-old previously healthy man presented with a four-day history of high-grade fever and productive cough, followed by rapidly progressive blackish discoloration of the fingers and toes. On admission, he was febrile but hemodynamically stable, with preserved peripheral pulses and no history of shock or exposure to vasopressors. Laboratory evaluation revealed neutrophilic leukocytosis and marked thrombocytopenia. Autoimmune, vasculitis, and thrombophilia screening were negative, and Doppler ultrasonography excluded large-vessel occlusion. Blood and pleural fluid cultures were sterile; however, urinary antigen testing confirmed Legionella pneumophila. The patient was treated with intravenous meropenem, clarithromycin, and clindamycin along with supportive care. Following initiation of therapy, his fever resolved, respiratory symptoms improved, and the ischemic changes stabilized without further progression. He was discharged in stable condition without the need for amputation. This case underscores the importance of early recognition and prompt targeted antimicrobial therapy to prevent limb loss in atypical presentations of SPG.

Keywords: Legionella pneumonia, symmetrical peripheral gangrene, sepsis.

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Introduction

Symmetrical peripheral gangrene (SPG) is a rare, life-threatening condition marked by distal ischemic injury in two or more limbs, progressing to gangrene despite the absence of large-vessel obstruction or vasculitis [1]. The condition predominantly affects acral areas and typically presents bilaterally, involving at least two sites such as the toes, fingers, earlobes, or scrotum, all occurring without any major occlusive vascular disease. It was first reported by Hutchinson in 1891 as gangrene affecting the distal limbs in a symmetrical pattern. It can occur in individuals of any age or sex. The condition carries a high mortality rate, ranging from 40% to 90%, and the risk of amputation can reach up to 50% [2]. The cause is multifactorial, but it is frequently associated with the use of vasopressors during the management of septic shock [3]. The occurrence of this condition in a hemodynamically stable patient, in the absence of vasculitis, shock, vasopressor exposure, or autoimmune disease, is exceptionally rare; to our knowledge, no published case has described its occurrence under such circumstances. Identifying the exact etiology is often difficult, particularly in resource-limited settings. Prompt recognition of this atypical presentation and early initiation of broad-spectrum antibiotics can prevent limb amputation and lead to optimal clinical outcomes.

Case Presentation

A 43-year-old healthy male presented with a 4-day history of high-grade fever associated with cough and expectoration. Over the preceding 3 days, he developed progressive blackish discoloration of both upper and lower limbs, consistent with rapidly evolving peripheral gangrene, as shown in Figure 1. He had no past medical history of any chronic diseases. There was no history of trauma, prolonged travel, immobilization, or intermittent claudication. He also denied exposure to aerosolized contaminated water sources, including cooling towers, air-conditioning systems, showers, or hot tubs, as well as recent hotel or cruise ship stays, healthcare-associated exposures, and residence in long-term care facilities.

On examination, the patient was alert but febrile, with a temperature of 38.4°C. Admission vitals were: blood pressure (BP) 130/80 mmHg, pulse rate (PR) 116/min, respiratory rate (RR) 20/min, and oxygen saturation (SpO2) 97% on room air. His fingers and toes appeared dusky with clear demarcation, though sensation was preserved and all peripheral pulses were palpable. Respiratory examination revealed crepitations predominantly over the left lung field, consistent with left-sided pneumonia. Imaging confirmed

* Correspondence to: Asif Dabeer Jafri
E-mail: asif_jafri2003@yahoo.co.in

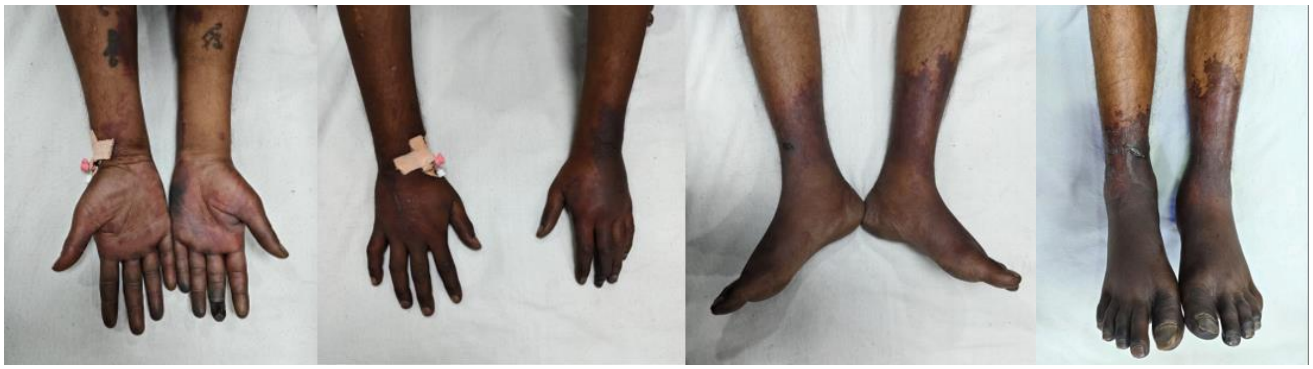

Figure 1. Ischemic and gangrenous changes of the extremities.

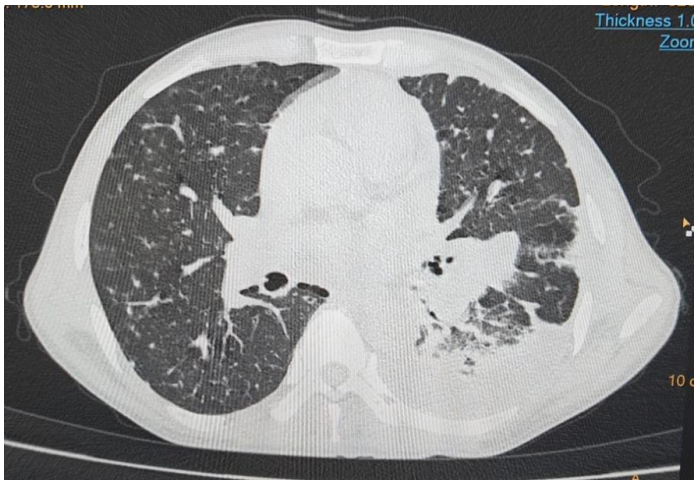

Figure 2. High-Resolution Computed Tomography (HRCT) of the chest shows left-sided consolidation consistent with pneumonia, accompanied by a left-sided pleural effusion.

left-sided pneumonia with an associated ipsilateral pleural effusion (Figure 2). Cardiovascular, neurological, and abdominal examinations were unremarkable. There were no signs of infective endocarditis or lymphadenopathy, and all cranial nerves were intact.

The laboratory parameters are summarized in Table 1. Viral markers, including hepatitis B surface antigen (HBsAg), hepatitis C virus (HCV), and human immunodeficiency virus (HIV), were negative. The tropical infection panel for scrub typhus, leptospirosis, dengue, and malaria also yielded negative results. Peripheral blood evaluation showed normocytic normochromic anemia with neutrophilic leukocytosis and marked thrombocytopenia. Autoimmune and vasculitis panels were negative, with normal antistreptolysin O (ASLO), anti-deoxyribonuclease B (anti-DNase B), and complement levels. Thrombophilia screening was normal. Doppler ultrasonography of both upper and lower limbs showed no abnormalities. Pleural fluid analysis was consistent with an exudative effusion, suggestive of an uncomplicated parapneumonic effusion. Blood, urine, sputum, and pleural fluid cultures were sterile. Urinalysis was within normal limits; however, the urinary Legionella pneumophila antigen test was positive.

He was treated with intravenous antibiotics (meropenem, clarithromycin, and clindamycin), along with mucolytics, bronchodilators, proton pump inhibitors, and IV fluids. Following initiation of therapy, his condition steadily improved, with no progression of limb ischemia,

Table I. – Laboratory parameters of the patient.

Parameter	Patient Value	Reference Range
Hb	12	13–17 g/dL (male)
TLC	2800	4,000–11,000 /µL
DLC	88/10	Neutrophils: 40–75%; Lymphocytes: 20–45%
PLT	50,000	150,000–450,000 /µL
Sr. Creatinine	1.43	0.6–1.3 mg/dL
Sr. Urea	93	15–40 mg/dL
Sr. Uric Acid	6.8	3.5–7.2 mg/dL
S. Na	124.4	135–145 mEq/L
S. K	3.6	3.5–5.0 mEq/L
Ca (T/I)	1.75/0.9	2.15–2.55 mmol/L / 1.12–1.32 mmol/L
Total. Bilirubin	1.07	0.2–1.2 mg/dL
Protein	4.3	6.0–8.3 g/dL
Albumin	2.6	3.5–5.0 g/dL
AST	81	10–40 IU/L
ALT	80	7–56 IU/L
ALP	105	44–147 IU/L
PT	20	11–13.5 seconds
INR	1.6	0.8–1.2
B12	2000	200–900 pg/mL
Folate	15.9	2–20 ng/mL
HbA1C	5.1	4.0–5.6%
Procalcitonin	14.01	<0.05 ng/mL
D-dimer	7.84	<0.5 µg/mL (FEU)
ESR	16	Male: <15 mm/h Female: <20 mm/h
CRP	121.2	<5 mg/L
S. TSH	1.6242	0.4–4.0 µIU/mL
Lactate	2.1	0.5–2.0 mmol/L

Abbreviations; Hb: hemoglobin; TLC: total leukocyte count; DLC: differential leukocyte count; PLT: platelet count; S. Na: serum sodium; S. K: serum potassium; Ca: calcium (total and ionized); TB: total bilirubin; S. Protein: serum protein; S. Albumin: serum albumin; AST: aspartate aminotransferase; ALT: alanine aminotransferase; ALP: alkaline phosphatase; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; HbA1c: glycated hemoglobin; INR: international normalized ratio; S.TSH: serum thyroid-stimulating hormone.

and he was discharged in a hemodynamically stable state. After completing a 10-day course of inpatient intravenous antibiotics, he was discharged in a hemodynamically stable condition. At discharge, all laboratory parameters were within normal limits. He was prescribed oral faropenem and azithromycin for an additional 7 days.

Discussion

Symmetrical peripheral gangrene, also referred to as purpura fulminans, is an uncommon condition marked by rapid ischemic injury to the extremities, occurring in the absence of vessel obstruction or vasculitis.

SPG primarily arises from altered microcirculation and vasospastic states, causing poor peripheral perfusion [4]. Disseminated intravascular coagulation (DIC) may act as a final common pathway of microvascular injury, and SPG has been proposed as a distinct cutaneous manifestation of DIC. In DIC patients with hypovolemia, vasopressors such as epinephrine or norepinephrine can further reduce tissue perfusion, aggravate ischemia, and precipitate tissue necrosis and gangrene [5]. SPG develops in approximately 2% to 6% of patients with sepsis, and the coagulopathy triggered during sepsis can progress to DIC, a life-threatening state that is recognized as a key pathological driver of SPG [6].

SPG can arise from a broad range of both infectious and non-infectious causes. Infection leading to sepsis is a major contributor, with bacterial pathogens such as Pneumococcus, Neisseria meningitidis, Staphylococcus aureus, and Streptococcus pyogenes commonly implicated [7]. Rarely, severe infections like Plasmodium falciparum malaria have also been associated with SPG [8]. Non-infectious causes include malignancies, hypovolemic shock, myeloproliferative disorders, vasospastic conditions, and connective tissue diseases such as systemic lupus erythematosus and antiphospholipid antibody syndrome. Certain medications, including noradrenaline, adrenaline, and dopamine, have also been reported as potential triggers in some cases. Immunosuppression, such as asplenia, diabetes mellitus, or renal failure serves as an important aggravating factor [9].

Conditions that closely resemble this presentation include thromboangiitis obliterans, advanced atherosclerosis, thromboembolic gangrene, secondary Raynaud's phenomenon, diabetes-related vascular disease, severe neuropathy, exposure to toxic or chemical agents, calciphylaxis, and various forms of vasculitic gangrene.

Management focuses on prompt fluid resuscitation for shock and early broad-spectrum antibiotic therapy for suspected infection. Patients with bleeding require replacement of depleted clotting factors. Interventions such as prostacyclin, tissue plasminogen activator, plasmapheresis, leukapheresis, and sympathetic blockade may offer limited benefit in selected cases but are not effective in SPG. The use of anticoagulants, including heparin and aspirin, remains controversial. Johansen and Hansen reported that heparin, aspirin, and streptokinase did not prevent the progression of gangrene in SPG. Oral corticosteroids have also shown no therapeutic advantage.

This case describes the occurrence of SPG in a hemodynamically stable patient in the absence of shock, vasopressor exposure, or underlying vasculitis, with preserved peripheral pulses. Such a presentation, particularly its association with Legionella pneumonia, has rarely been described. These observations suggest that SPG may develop even in the early phases of sepsis associated with DIC, likely mediated by sepsis-related endothelial dysfunction, microvascular thrombosis, and coagulation abnormalities.

Conclusion

Symmetrical peripheral gangrene can occur in hemodynamically stable patients even in the absence of shock, vasopressor therapy, or underlying autoimmune disease. Its presence may serve as an early clinical marker of evolving sepsis.

Authors' contributions

The following statements should be used:
Conceptualization: ADJ, IS, MS, RKS; Methodology: ADJ, IS, RKS; Data curation: ADJ, IS, RKS; Validation: ADJ, IS, MS, RKS; Writing - Original Draft: ADJ; Writing - Review & Editing: ADJ, IS, MS, RKS; Validation: ADJ, IS, MS, RKS; Supervision: RKS.

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Conflict of interest

We declare that we do not have any conflict of interest.

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