SAS Journal of Surgery
Abbreviated Key Title: SAS J Surg
ISSN 2454-5104
Journal homepage: https://www.saspublishers.com

OPEN ACCESS

Surgery

Extensive Bilateral Plantar Necrosis in a Patient with Diabetes Despite Preserved Peripheral Pulses and CT Angiography without Obstructive Lesions: A Diagnostic Challenge (Case Report)

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DOI: https://doi.org/10.36347/sasjs.2026.v12i09.006 | **Received:** 12.07.2026 | **Accepted:** 16.09.2026 | **Published:** 22.09.2026

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Abstract **Case Report**

Background. Extensive bilateral plantar necrosis is an unusual presentation of diabetes-related foot disease, particularly when peripheral pulses are preserved and imaging shows no significant obstructive peripheral artery disease. ***Case presentation.*** A 44-year-old man with poorly controlled type 2 diabetes treated with insulin (HbA1c 10%) was admitted with rapidly progressive bilateral plantar lesions. Initial purpuric and ecchymotic plaques progressed over approximately 15 days to extensive dry necrosis of both soles and several toes. The lower limbs were warm, and the femoral, popliteal, posterior tibial, and dorsalis pedis pulses were palpable and symmetrical. CT angiography of the aortoiliac and lower-limb arteries showed patent aortoiliac, femoropopliteal, tibial, and pedal vessels without abnormalities. On Aug 3, 2026, the patient underwent bilateral plantar necrosectomy with partial toe amputations. After removal of the necrotic tissue, the residual tissue showed macroscopic bleeding. On postoperative day 2, the wounds contained viable red tissue and fibrinous deposits. Intravenous amoxicillin-clavulanate (3 g/day) and ciprofloxacin (500 mg twice daily) were combined with serial debridement every other day. The patient also received 10 sessions of hyperbaric oxygen therapy, five days per week. ***Discussion.*** Patency of the major arteries does not exclude disease of the plantar or digital arteries, impaired tissue perfusion, or microcirculatory dysfunction. Histopathological examination in this case showed microthrombotic vasculopathy, with intraluminal fibrin- and erythrocyte-rich thrombi in small vessels, vessel-wall thickening and hyalinization, narrowing or occlusion of some lumina, and ischemic necrotic lesions, without the overt destructive inflammation that would support vasculitis. These findings strengthen the hypothesis that microvascular disease contributed in the setting of severely uncontrolled diabetes, but they do not establish isolated diabetic microangiopathy as the sole cause of the entire clinical presentation. ***Conclusion.*** Severe plantar necrosis can occur despite palpable pulses and CT angiography showing no significant obstruction. Assessment should combine arterial anatomy, distal perfusion testing, investigation for infection, and evaluation of alternative diagnoses.

Keywords: Diabetes mellitus; diabetic foot; plantar necrosis; gangrene; microcirculation; computed tomography angiography; case report.

INTRODUCTION

Diabetes-related foot complications usually result from interactions among peripheral neuropathy, mechanical stress, infection, peripheral artery disease, and impaired wound healing. The assessment of ulceration or gangrene in a person with diabetes must therefore be multidimensional and cannot be limited to pulse palpation or a single imaging examination [1–3].

Obstructive lower-extremity peripheral artery disease is the vascular mechanism most clearly associated with tissue loss. By contrast, diabetic microangiopathy is generally considered a contributing factor that affects vasoreactivity, oxygenation, the inflammatory response, and tissue repair, rather than an established isolated cause of extensive gangrene [4–7].

We report rapidly progressive bilateral plantar necrosis in a patient with poorly controlled diabetes, preserved peripheral pulses, and CT angiography showing neither significant stenosis nor occlusion of the visualized arteries. We describe the clinical presentation and diagnostic and therapeutic strategy, then discuss the limitations of macrovascular imaging and the possible mechanisms of distal tissue loss.

Case Presentation

Patient information

The patient was afebrile on admission. His footwear was appropriate, with no history of unsuitable shoes. He reported no recent microtrauma, burns, thermal exposure, or prolonged walking before the lesions developed.

The available data indicated a 25-year history of type 2 diabetes, no history of smoking, and peripheral neuropathy demonstrated by loss of protective sensation with a 10-g monofilament. His footwear was appropriate, and no history of microtrauma was identified.

History of the present illness and clinical findings

Approximately 15 days before admission, purpuric and ecchymotic lesions appeared on both soles, mainly over weight-bearing areas. They rapidly progressed to extensive, dry, black plaques involving the forefeet, mid-plantar regions, heels, and several toes (Figures 1 and 2).

On examination, both lower limbs were warm. The femoral, popliteal, posterior tibial, and dorsalis pedis pulses were palpable and symmetrical. The ankle-brachial index (ABI) was 0.92. Neurological examination of the feet demonstrated loss of protective sensation with a 10-g Semmes-Weinstein monofilament, consistent with diabetic peripheral neuropathy. There were no clinical signs of acute proximal arterial occlusion.

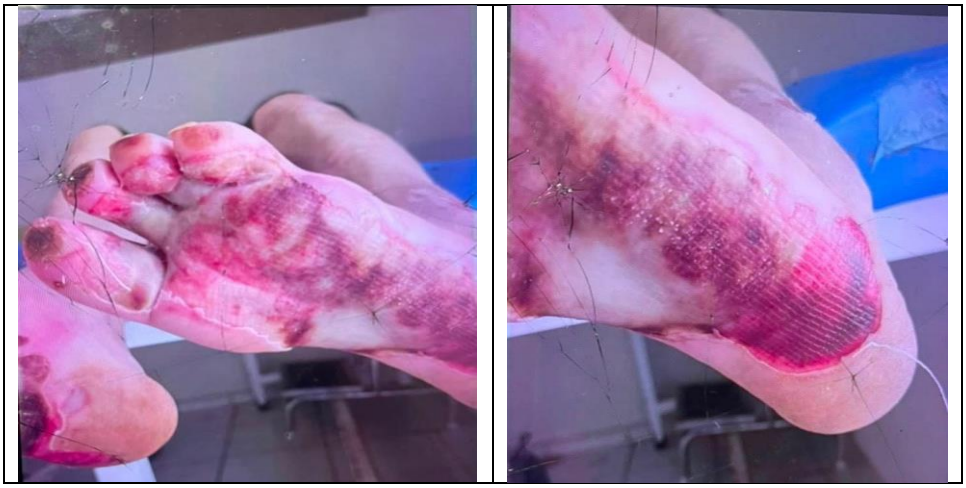

Figure 1: Initial clinical appearance of the bilateral plantar lesions approximately 15 days before necrosectomy. (A) Purpuric and ecchymotic lesions involving the forefoot and toes. (B) Purpuric lesions involving the heel and mid-plantar region. The photographs were cropped without altering the clinical content

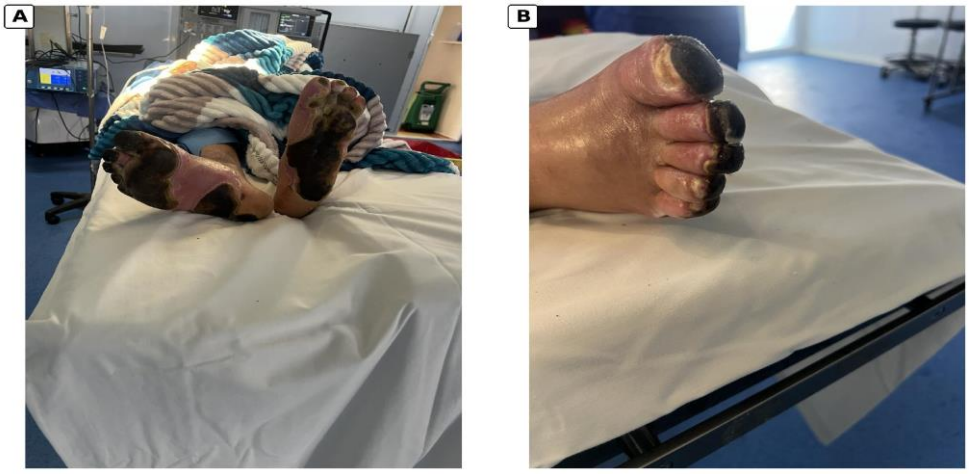

Figure 2: Preoperative appearance. (A) Extensive bilateral dry black necrosis of the soles, predominantly involving the forefeet and heels. (B) Distal necrosis of the toes. The clinical appearance alone cannot distinguish distal ischemia, microthrombotic vasculopathy, or secondary necrotizing infection

Laboratory investigations

Parameter	Aug 2, 2026	Aug 4, 2026
Hemoglobin (g/dL)	10.4	8.1
Leukocytes (G/L)	9.3	13
Platelets (G/L)	378	289
Prothrombin time (%)	92	—
aPTT (ratio)	0.9	—
Sodium (mmol/L)	131	130
Potassium (mmol/L)	3.67	3.89
Bicarbonate (mmol/L)	22	18
Urea (g/L)	0.52	0.40
Creatinine (mg/L)	15	15
Total protein (g/L)	78	—
CRP (mg/L)	45.7	138.2
HbA1c (%)	10	—

Initial laboratory tests showed moderate anemia and an inflammatory response, with C-reactive protein (CRP) at 45.7 mg/L and a leukocyte count of 9,300/mm³. Renal function was preserved, with serum creatinine at 15 mg/L and a preserved glomerular filtration rate. On postoperative day 2, inflammatory markers increased transiently (CRP 138.2 mg/L; leukocytes 13,000/mm³) and then progressively decreased. At discharge, CRP was 89 mg/L and the leukocyte count was 11,600/mm³, in parallel with clinical improvement.

Vascular assessment

CT angiography of the aorta and lower limbs showed a patent abdominal aorta of normal caliber to the iliac bifurcation, with no plaque or caliber discrepancy. The iliac, common femoral, profunda femoris, superficial femoral, popliteal, tibioperoneal, anterior tibial, posterior tibial, fibular, and dorsalis pedis arteries were patent bilaterally to their distal segments. The radiological conclusion reported no arterial abnormality (Figure 5).

Etiologic workup. Given the bilateral, symmetrical, and initially purpuric nature of the lesions, an immunological workup was performed to investigate systemic vasculitis. Antinuclear antibodies (ANA) and antineutrophil cytoplasmic antibodies (ANCA) were negative, providing no immunological evidence of systemic vasculitis.

An ABI of 0.92, symmetrical distal pulses, and patent arteries on CT angiography did not suggest severe macrovascular ischemia that could independently explain the extent of the necrosis. Toe pressure, transcutaneous oxygen pressure ($TcPO_2$), and skin perfusion pressure were not available, limiting direct assessment of tissue perfusion.

Therapeutic intervention and early outcome

On Aug 3, 2026, the patient underwent bilateral plantar necrosectomy with partial toe amputations. Empirical antibiotic therapy with amoxicillin-clavulanate 3 g/day and ciprofloxacin 500 mg twice daily was administered intravenously for one week, followed by the same doses orally for two weeks, for a total antibiotic duration of three weeks.

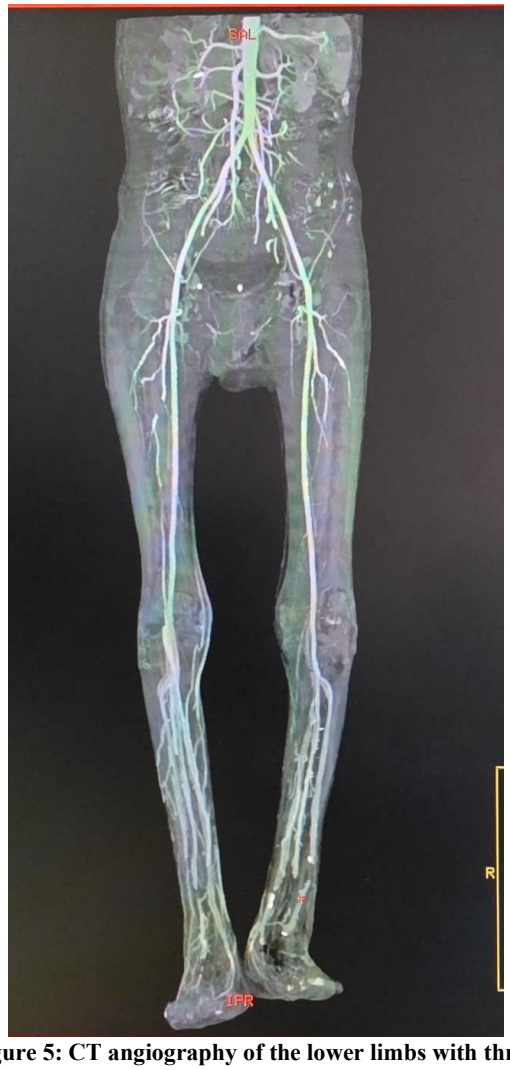

Figure 5: CT angiography of the lower limbs with three-dimensional angiographic reconstruction. The major aortoiliac, femoropopliteal, and infrapopliteal arteries appear patent bilaterally, without abnormalities. This macroarterial patency contrasts with the extent of the bilateral plantar necrosis. Interpretation should remain correlated with the radiology report

After removal of the necrotic tissue, the residual tissue showed macroscopic bleeding. Serial surgical debridement was performed every other day, with regular reassessment of tissue viability. Local wound care used a lipidocolloid contact layer dressing (UrgoTul®). The patient also received 10 hyperbaric oxygen therapy sessions at 2 ATA, each lasting 60 minutes, administered once daily on five days per week, with no sessions on weekends. The clinical and laboratory course was favorable, with no further clinical extension of necrosis.

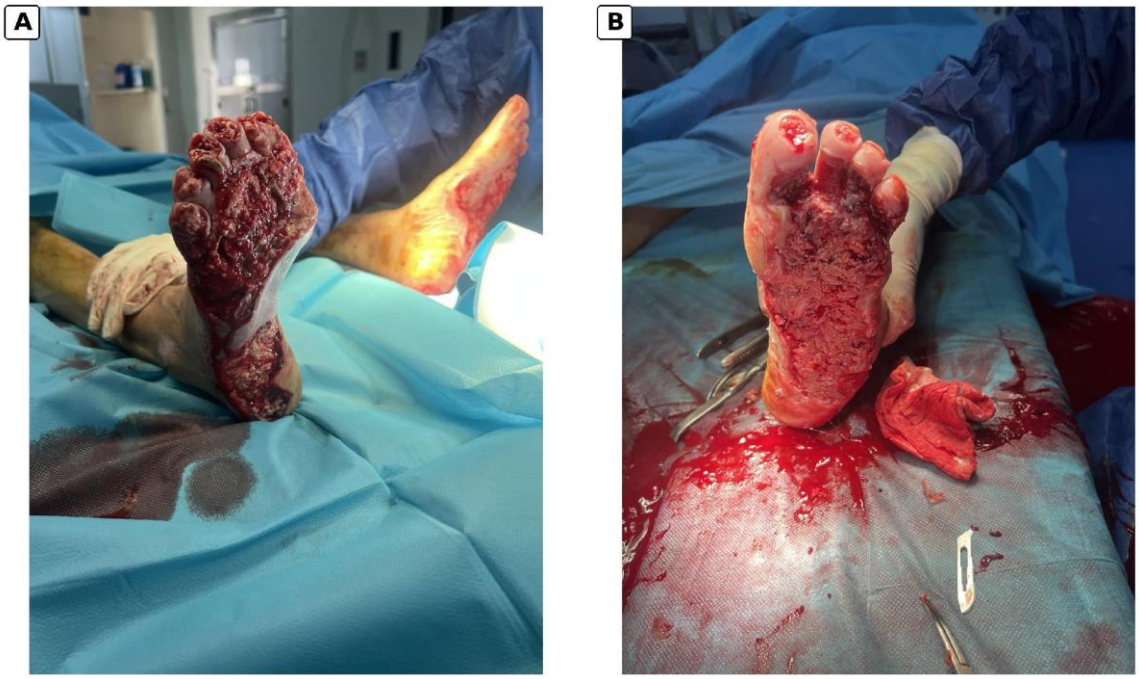

Figure 3: Intraoperative appearance after bilateral plantar necrosectomy. (A–B) Removal of necrotic tissue with exposure of deep tissue planes and macroscopic bleeding from residual tissue. This bleeding indicates residual local perfusion but does not exclude microcirculatory impairment or deep infection

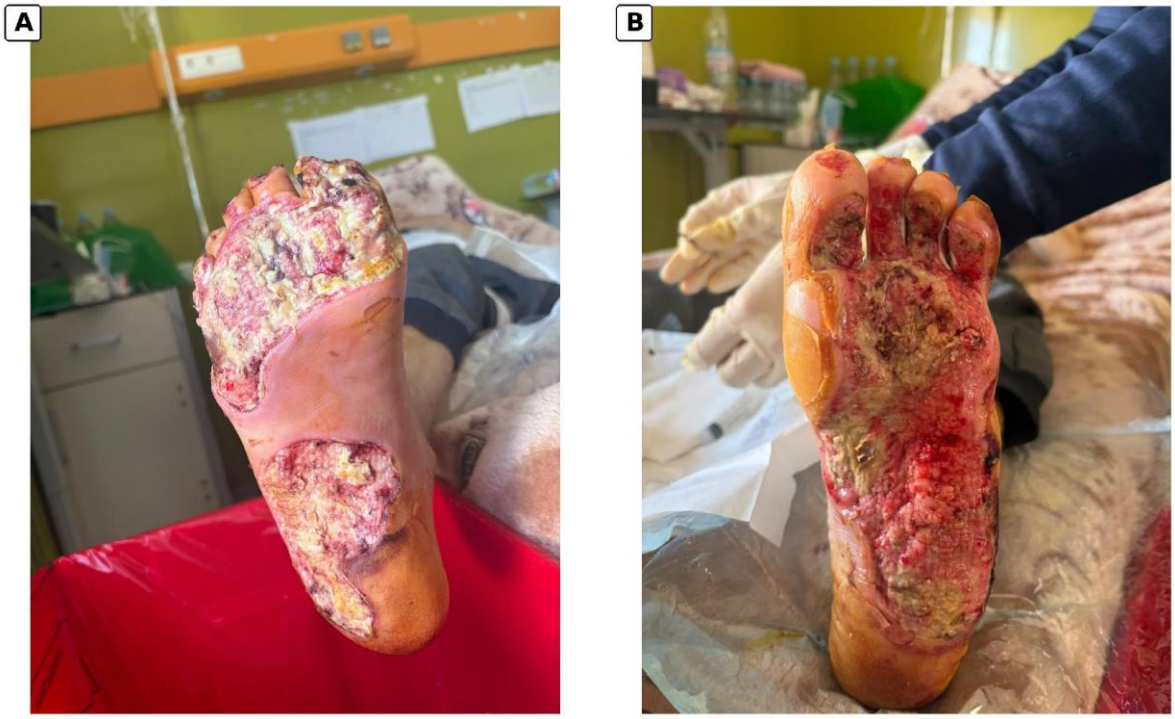

Figure 4: Wound appearance during dressing changes on postoperative day 2. (A–B) Extensively debrided wound beds with viable red areas and fibrinous deposits. Assessment of healing potential requires prolonged follow-up, distal perfusion measurements, infection control, and management of mechanical stress

Timeline

Time	Event
Approximately day −15	Onset of bilateral purpuric and ecchymotic plantar lesions.
Aug 2, 2026	Initial laboratory tests: Hb 10.4 g/dL, leukocytes 9.3 G/L, CRP 45.7 mg/L, and HbA1c 10%.
Admission	Extensive bilateral plantar necrosis; warm feet; all peripheral pulses palpable and symmetrical.
Before surgery	CT angiography: patent aortoiliac, femoropopliteal, tibial, and pedal arteries without abnormalities.
Aug 3, 2026	Bilateral plantar necrosectomy and partial toe amputations; initiation of intravenous amoxicillin-clavulanate 3 g/day and ciprofloxacin 500 mg twice daily.
Aug 4, 2026, POD 1–2	Hb 8.1 g/dL, leukocytes 13 G/L, and CRP 138.2 mg/L; postoperative dressings showed debrided wound beds with viable and fibrinous areas.
From Aug 11, 2026	Hyperbaric oxygen therapy: 10 sessions, one session daily from Monday through Friday; surgical debridement continued every other day.

DISCUSSION

An unusual and probably multifactorial presentation

This case is distinguished by rapidly progressive, extensive bilateral plantar necrosis in a relatively young patient, contrasting with symmetrical peripheral pulses and CT angiography showing no significant obstructive peripheral artery disease. Diabetes-related foot complications rarely have a single cause. Neuropathy, mechanical stress, infection, arterial insufficiency, microvascular dysfunction, metabolic disorders, and impaired immune responses may act simultaneously [1–3].

The bilateral and relatively symmetrical distribution, with initial lesions over weight-bearing areas, is less consistent with a single arterial embolus or segmental occlusion of a major vessel. However, progression from plantar purpura to extensive necrosis within approximately 15 days indicates severe tissue injury. The sequential images are an important strength of this case because they document the transition from a potentially reversible or diagnostically informative stage to established tissue loss.

Palpable pulses do not exclude inadequate tissue perfusion

An ABI of 0.92 did not support severe obstructive peripheral artery disease. In patients with diabetes, however, this measurement must be interpreted cautiously because arterial stiffness may affect the result. Neuropathy was demonstrated by loss of protective sensation with a 10-g monofilament, supporting a multifactorial pathophysiology involving neuropathy, plantar mechanical stress, and probable impairment of microcirculatory perfusion.

Palpable dorsalis pedis and posterior tibial pulses reduce the likelihood of major proximal occlusion but do not exclude distal arterial disease, involvement of the plantar or digital arteries, or impaired cutaneous oxygenation. The intersocietal IWGDF, ESVS, and SVS guidelines state that clinical examination alone cannot exclude peripheral artery disease in a person with diabetes and a foot ulcer or gangrene [2].

Diabetes-related medial arterial calcification may also produce a falsely elevated or noncompressible ABI. Toe pressures, $TcPO_2$, skin perfusion pressure, and Doppler waveform morphology provide complementary functional information. Their absence in the present case limits interpretation of actual perfusion, particularly in the plantar region.

Value and limitations of CT angiography

CT angiography reasonably excludes occlusion of the visualized aortoiliac, femoropopliteal, and tibial arteries. The reported patency of the dorsalis pedis arteries to their distal segments strengthens this anatomical conclusion. However, CT angiography does not measure flow distribution in arterioles, capillaries, or arteriovenous shunts and does not directly assess tissue oxygenation. The plantar arteries and digital arches may also be less well visualized.

The discrepancy between satisfactory arterial anatomy and severe necrosis therefore requires a distinction among vascular patency, vasodilatory reserve, and nutritive perfusion. A patent artery may deliver insufficient blood flow to injured tissue, while abnormalities in microvascular flow distribution may coexist with preserved macroscopic arterial flow.

Potential role of diabetic microcirculatory dysfunction

Chronic diabetes is associated with endothelial dysfunction, reduced nitric oxide bioavailability, oxidative stress, inflammation, protein glycation, impaired erythrocyte deformability, and thickening of capillary basement membranes. In the foot, these abnormalities may impair autoregulation, reactive hyperemia, and adaptation of blood flow to mechanical or infectious stress [4–8].

An HbA1c level of 10% makes advanced microvascular dysfunction and an impaired healing response plausible but does not prove causation. Historical and contemporary studies do not support the concept that isolated structural microangiopathy is usually sufficient to cause gangrene. It is more accurate to consider it a contributing mechanism that reduces

tissue reserve and exacerbates the effects of infection, pressure, and inflammation [4–7].

Demonstrating functional impairment would have required $TcPO_2$, toe pressure, skin perfusion pressure, capillaroscopy, laser Doppler flowmetry, or another microcirculatory technique. Their absence precludes a definitive diagnosis of "microangiopathic gangrene" and should be explicitly acknowledged.

In this case, histopathological examination provided objective evidence of small-vessel disease. It showed intraluminal fibrin- and erythrocyte-rich thrombi, thickening and hyalinization of vessel walls with narrowing or occlusion of some lumina, and associated ischemic injury and necrosis of the epidermis and dermis with fibrin deposits. The absence of overt destructive inflammation of the vessel walls did not support histological vasculitis. The concordance among these microvascular lesions, negative ANA and ANCA results, patent macroscopic arteries, and poor glycemic control strengthens the hypothesis of microthrombotic vasculopathy in the setting of diabetes. Nevertheless, histology alone cannot establish diabetic microangiopathy as the sole mechanism of necrosis; the findings must be interpreted alongside potential neuropathic, metabolic, and infectious factors.

Poor glycemic control, anemia, and tissue vulnerability

Chronic hyperglycemia impairs chemotaxis and phagocytic function, promotes glycation and oxidative stress, and compromises matrix synthesis and angiogenesis. In this patient, an HbA1c level of 10% was therefore a major aggravating factor. The initial anemia, which worsened after surgery, may have reduced oxygen delivery to tissues already under high metabolic demand. These factors do not independently explain the bilateral distribution but reduce the capacity for recovery.

Neuropathy and plantar mechanical stress

The forefoot and heel locations correspond to the main weight-bearing areas. Sensory neuropathy may abolish protective pain, while motor neuropathy and deformities alter plantar pressure. The previous amputation of the right fifth toe and orthopedic sequelae of the leg may have increased mechanical stress on the right side but cannot explain the bilateral involvement.

The absence of a reported injury or burn does not exclude an unperceived exposure. Retrospective investigation of thermal contact, prolonged walking, unsuitable footwear, caustic products, or prolonged compression would be relevant. The 10-g monofilament test, vibration perception, Achilles reflexes, examination for deformities, and subsequent plantar pressure analysis should be documented.

Differential diagnoses to exclude

The purpuric onset, bilateral distribution, and preserved pulses required consideration of small-vessel vasculopathies. The immunological workup for systemic vasculitis was negative, including negative ANA and ANCA results. Most importantly, histopathological analysis of the resected tissue showed fibrin- and erythrocyte-rich thrombi in small vessels and hyaline vessel-wall changes, without overt destructive inflammation of the vessel walls. These findings favor microthrombotic vasculopathy over inflammatory vasculitis but must be interpreted in the overall clinical context.

Cholesterol microembolization was less likely in the absence of a recent vascular procedure, significant aortoiliac atheroma, systemic livedo reticularis, or acute kidney injury. Calciphylaxis also appeared less likely with preserved renal function, although a nonuremic form cannot be excluded without the appropriate clinical and histological context. Symmetrical peripheral gangrene associated with shock, disseminated intravascular coagulation, or vasopressors was not supported by the available data.

Interpretation of intraoperative bleeding

Macroscopic tissue bleeding after necrosectomy is consistent with residual local perfusion and patency of the major arteries. However, it does not prove that microvascular reserve is sufficient for healing. Perfusion may be heterogeneous, and a bleeding operative bed may subsequently become necrotic in the presence of persistent infection, excessive pressure, edema, or hypoxia.

The appearance on postoperative day 2 was too early to draw conclusions. Essential data for the final report will include progression of granulation tissue, additional debridement, possible use of negative-pressure wound therapy or tissue coverage, preservation of weight-bearing capacity, development of osteomyelitis, and whether a more proximal amputation becomes necessary.

Multidisciplinary management

Local control consisted of initial necrosectomy followed by serial surgical debridement every other day. Empirical intravenous antibiotic therapy combining amoxicillin-clavulanate 3 g/day and ciprofloxacin 500 mg twice daily was administered concurrently. This approach should be combined with deep-tissue sampling, glycemic control, correction of laboratory abnormalities, plantar offloading, and close reassessment [1,3,9].

Hyperbaric oxygen therapy was used as an adjunctive treatment. The patient received 10 sessions, each lasting 60 minutes, once daily on five days per week, with no sessions on weekends. This strategy did not replace surgical source control, antibiotic therapy, or metabolic optimization.

Lessons from the case

The most robust conclusion is that severe bilateral plantar necrosis can occur despite palpable pulses and the absence of obstruction in the visualized arteries. Anatomical patency of the major vessels does not predict the quality of nutritive tissue perfusion. The most likely explanation is multifactorial, involving poor glycemic control, probable microcirculatory dysfunction, potential neuropathic plantar stress, and possible necrotizing infection with secondary microvascular thrombosis.

CONCLUSION

This case illustrates an unusual presentation of rapidly progressive bilateral plantar necrosis in a patient with poorly controlled diabetes, preserved peripheral pulses, and normal CT angiography. It demonstrates the limitations of an assessment focused exclusively on the macrocirculation and supports functional assessment of distal perfusion, microbiological and histological investigations, and exclusion of inflammatory or thrombotic vasculopathies. Diabetic microangiopathy may be considered a contributing factor but should not be presented as the sole cause on the basis of the current data.

Patient perspective

The patient reported being deeply affected by the rapid progression of the lesions and the need for surgical procedures. He understands the goal of maximizing limb preservation and hopes to achieve wound healing that will allow a gradual return to weight bearing and walking.

Informed consent

Written informed consent was obtained from the patient for publication of the clinical data and anonymized medical photographs. The images and manuscript contain no direct identifiers.

Declarations

Conflicts of interest: The authors declare that they have no conflicts of interest.

Funding: No specific funding was received for this work.

Author contributions (CRediT): El Mehdi El Khadir: conceptualization, investigation, data collection, and writing of the original draft; Nizar Taoussi, Mehdi Benmoussa, Karima El Houassli, Noureddine Lahlou, and Mohamed Zoulati: investigation, clinical care, and manuscript review; Tarik Bakkali and Hassan Toufik Chtata: supervision, validation, and critical review of the manuscript. The exact allocation of roles must be confirmed by all authors.

Data availability: The clinical data are available from the corresponding author, subject to confidentiality requirements and patient consent.

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