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Buerger's Disease Revealed by a Leg Ulcer

Hajar Boumehdi[1*], Maryem Aboudourib[1], Layla Bendaoud[1], Ouafa Hocar[1], Said Amal[1]

[1]Dermatology and Venerology, Mohammed VI University Hospital, Bioscience and Health Resarch, Faculty of Medecine and Pharmacy - Cadi Ayyad University, Marrakesh, MAR

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***Corresponding author:** Hajar Boumehdi
Dermatology and Venerology, Mohammed VI University Hospital, Bioscience and Health Resarch, Faculty of Medecine and Pharmacy - Cadi Ayyad University, Marrakesh, MAR

Abstract **Case Report**

A 50-year-old chronic smoker (40 pack-years) presented with a painful leg ulcer evolving for 2 months after scarification. Biopsy suggested pyoderma gangrenosum, and laboratory workup was unremarkable. However, arterial Doppler and angiography revealed total thrombosis of the subrenal abdominal aorta extending to the iliac arteries and bilateral deep vein thrombosis, consistent with Buerger's disease. The patient underwent smoking cessation, double antiplatelet therapy, ACE inhibitors, and aortoiliac stenting. Despite intervention, necrosis progressed, and leg amputation was required. This case highlights the critical role of vascular imaging in evaluating leg ulcers and emphasizes smoking as a key factor in disease progression. Early recognition and intervention are essential for limb salvage and improved outcomes in Buerger's disease.

Keywords: Buerger's disease, foot ulcer, leg ulcer, thromboangiitis obliterans, thrombosis.

INTRODUCTION

Leg ulcers are defined as a loss of tissue involving the epidermis and extending into the dermis. They represent a significant source of morbidity, markedly impairing quality of life through pain, malodor, reduced mobility, social isolation, and sleep disturbance.

Their etiology is heterogeneous and frequently necessitates a multidisciplinary approach. Accurate etiological classification is essential to guide management. Approximately 80% of cases are attributable to chronic venous insufficiency or peripheral arterial occlusive disease; however, a broad spectrum of less common causes-including vasculitis, calciphylaxis, pyoderma gangrenosum, necrobiosis lipoidica, infections, malignancies such as squamous cell carcinoma, and paraneoplastic syndromes-must also be considered [1-2].

Although venous and atherosclerotic arterial diseases predominate, nonatherosclerotic vasculopathies should be considered, particularly in atypical clinical contexts, such as younger patients without conventional cardiovascular risk factors or with predominantly distal ischemic involvement. Among these, Buerger's disease, is a rare, segmental, nonatherosclerotic inflammatory vasculopathy affecting small- and medium-sized vessels of the extremities.

The disease typically affects individuals between 20 and 50 years of age and shows a strong association with tobacco exposure. Initial manifestations commonly include distal extremity claudication, which may be misattributed to musculoskeletal or neurological disorders. Disease progression is characterized by worsening ischemia, leading to rest pain and ischemic ulcerations of the digits.

Management is centered on absolute tobacco cessation, which remains the only intervention capable of altering disease course. Sustained abstinence is associated with symptom remission and a substantial reduction in amputation risk.

CASE PRESENTATION

A 50-year-old male, chronic smoker with a 40 pack-year history, with no known history of diabetes mellitus, hypertension, dyslipidemia, or previous cardiovascular disease, presented with a painful ulcer of the right leg that had been evolving for two months.

The patient reported intermittent claudication of the lower limb, characterized by exertional pain with a limited walking distance, which resolved with rest and

denied any history of Raynaud phenomenon. As a result, the patient underwent non-medical scarification, which was followed by the development of a secondary ulcerative lesion. The pain progressively intensified, becoming severe and significantly limiting ambulation.

On admission, the patient was hemodynamically stable, with a blood pressure of 135/85 mmHg, heart rate of 78 beats/min, respiratory rate of 16 breaths/min and a body temperature of 36.8°C. Peripheral vascular examination revealed weakly palpable distal pulses bilaterally. Both feet were cold to palpation, without signs of acute infection and no evidence of gangrene or digital necrosis.

Dermatological examination revealed a painful ulcer measuring approximately 7 cm on the anterior aspect of the right leg, with irregular undermined borders and a necrotic base. Surrounding erythema was present without purulent discharge. A second, smaller circular ulcerative lesion was noted over the ipsilateral knee (Figure 1).

No regional lymphadenopathy was detected.

FIGURE 1: Lower limb ulcer in a patient with Burher's disease

Given the clinical suspicion of an inflammatory dermatosis, a three-site skin biopsy was performed. Histopathological examination revealed a dense neutrophilic dermal infiltrate without evidence of true vasculitis, findings initially suggestive of pyoderma gangrenosum.

An extensive laboratory evaluation was undertaken. Results demonstrated a mild elevation of inflammatory markers, while fasting blood glucose levels remained within normal limits. The autoimmune panel, including antinuclear antibodies (ANA), anti-neutrophil cytoplasmic antibodies (ANCA), antiphospholipid antibodies, and rheumatoid factor, was negative.

Biological screening for occult malignancy, including lactate dehydrogenase (LDH) measurement and serum protein electrophoresis, yielded unremarkable results. Thrombophilia screening was negative, and viral serologies were nonreactive (Table 1).

TABLE 1: Laboratory Tests and Results

Laboratory Test	Result	Reference range
Basophils	29	0-150 /㎣
Eosinophils	156	0-500 /㎣
Hematocrit	47.7	35-47%
Hemoglobin	12.3	13-15 g/dL
Lymphocytes	3456	2500-4500/㎣
Monocytes	29.7	25-30 pg
Neutrophils	604	200-1000/㎣
Red Blood Cells (RBC)	87.7	80-90 µm³
White Blood Cells (WBC)	4355	4000-6000/㎣
Platelets	215000	150000-40000 /㎣
Mean Platelet Volume (MPV)	5.43	4.5-5.5 M/㎣
Mean Corpuscular Hemoglobin (MCH)	9740	6000-11000 /㎣
Erythrocyte Sedimentation Rate (ESR)	22	0-10 MM
C-Reactive Protein (CRP)	72	0-5 mg/L
Activated Partial Thromboplastin Time (aPTT)	24,2	30-35 sec
aPTT Ratio (aPTT-R)	0.97	-
International Normalized Ratio (INR)	1.08	0.8-1.2
D-Dimer	380	< 500 ng/mL
Antithrombin III	90	80-100%
Fibrinogen	2.5	2-4 g/L
Protein C	99	65-140 %
Protein S	101	70-140 %
Factor V Leiden Mutation	Negative	-
Prothrombin G20210A Mutation	Negative	-
Lupus Anticoagulant	Negative	-
Anticardiolipin Antibodies	Negative	IgG/IgM < 20 U/mL
β2-Glycoprotein I Antibodies	Negative	IgG/IgM < 20 U/mL
Alanine Aminotransferase (ALT)	20	10-41 U/L
Aspartate Aminotransferase (AST)	33	10-50 U/L
Creatinine	10.40	7-12 mg/L
Glucose	1.18	0,70-1,09 g/L
Hemoglobin A1c (HbA1c)	5.7	4.8O-5.9 %
Triglycerides	0.87	0.35-1.50 g/L
Low-Density Lipoprotein (LDL)	0.87	0-1.60 g/L
Cholesterol	1.86	1.54-2.01 g/L
Uric Acid	58	35 – 72 mg/L
Lactate Dehydrogenase (LDH)	211	0-250 U/L
Prostate-Specific Antigen (PSA)	0.7	0-4 ng/ml
Alpha-Fetoprotein (AFP)	1.4	0-5.80 UI/ml
Carcinoembryonic Antigen (CEA)	1.5	0-6.50 ng/mL
Cancer Antigen 125 (CA-125)	12	0-35 UI/mL
Cancer Antigen 19-9 (CA 19-9)	6.4	0-27 UI/mL
Antinuclear Antibodies (ANA)	Negative	< 1/80
Anti-DNA Antibodies	Negative	<16UI/ML
Antineutrophil Cytoplasmic Antibodies (ANCA)	Negative	<5
Rheumatoid Factor (RF)	Negative	<10 UI/mL
Anti-Cyclic Citrullinated Peptide Antibodies (Anti-CCP)	Negative	< 20 U/mL
Complement Component 3 (C3)	1.66	0.90-1.80 g/L
Complement Component 4 (C4)	0.23	0,10 à 0,40 g/L
Cryoglobulins	Negative	-
VIH serology	Negative	-
Hepatitis B surface antigen	Negative	-
Anti-HBs antibodies 45 IU/L >10 IU/L	45 IU/L	>10 IU/L
Anti-HBc antibodies	Negative	-

Summary of Laboratory Test Results on Hospital Admission, Including Hematologic, Coagulation, Biochemical, Metabolic, Viral Serology, Tumor Marker, and Autoimmune Parameters.

A comprehensive malignancy evaluation was performed. Contrast-enhanced computed tomography of the chest, abdomen, and pelvis revealed no evidence of solid malignancy or lymphoproliferative disease. Given the persistence of severe pain, delayed wound healing, and weak peripheral pulses, vascular imaging was pursued. Arterial Doppler ultrasound demonstrated markedly reduced flow in the distal abdominal aorta and iliac arteries, with monophasic distal waveforms. Computed tomography angiography (Figure 2, 3,) revealed a complete thrombosis of the infrarenal abdominal aorta, extending to the aortoiliac bifurcation.

Collateral circulation was noted, with recanalization of the external iliac arteries via the epigastric arteries. Additionally, there was complete occlusion of both internal iliac arteries, with distal reconstitution through parietal collateral vessels, consistent with a chronic arterial occlusion. There was also evidence of bilateral deep venous thrombosis, indicating associated venous thrombotic involvement. Diffuse atherosclerotic changes with medial arterial calcification were noted.

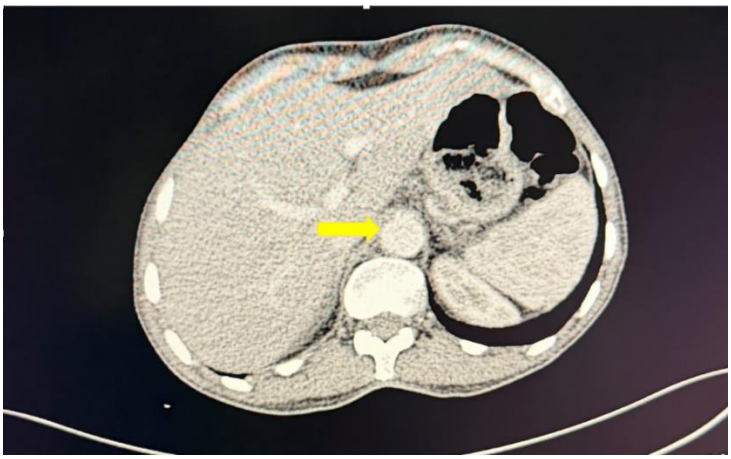

Figure 2: Contrast-Enhanced CT Angiography Demonstrating Distal Aortic Thrombosis (arrow)

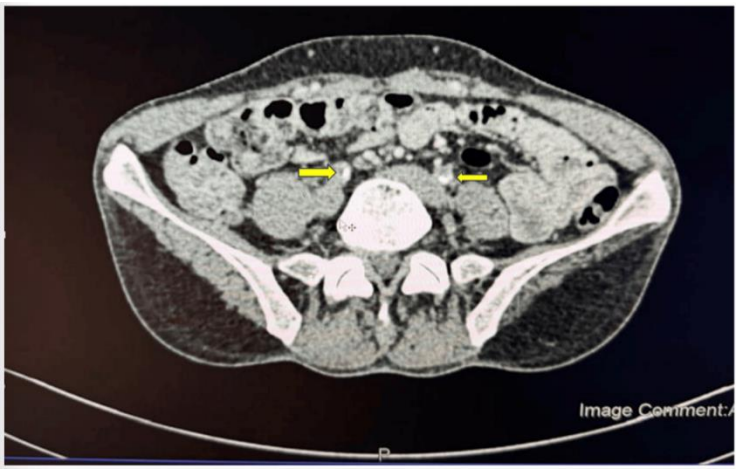

Figure 3: Axial computed tomography angiography image demonstrating thrombotic occlusion of the primitive iliac arteries (arrows indicate areas of thrombosis)

In the absence of major atherosclerotic risk factors other than heavy tobacco use, and after exclusion of autoimmune, thrombophilia, and neoplastic causes, the findings were considered highly suggestive of Buerger's disease presenting as chronic large-vessel arterial occlusion associated with concomitant venous thrombosis.

Strict smoking cessation was immediately recommended. The patient was managed with dual antiplatelet therapy (aspirin 75 mg/day and clopidogrel 75 mg/day), an ACE inhibitor (ramipril 2.5 mg/day), and therapeutic anticoagulation with low-molecular-weight heparin (LMWH) for the associated deep vein thrombosis.

An endovascular procedure with aortoiliac stent placement was performed in an attempt to restore perfusion. The procedure consisted of catheter-directed thrombectomy followed by balloon angioplasty and stent placement in the aortoiliac segments. Partial recanalization was achieved; however, distal arterial flow remained severely compromised. Post procedural Doppler ultrasound demonstrated markedly reduced arterial flow in the distal vessels, with monophasic waveforms and low velocities, consistent with persistent severe

DISTAL ISCHEMIA

Clinically, despite revascularization and optimized medical therapy including antiplatelet treatment, anticoagulation, and strict smoking cessation, the patient showed no clinical improvement and instead exhibited worsening ischemic symptoms, with the development of new ulcerations, increased claudication and pain, and the development of livedo (figure 4).

Distal ischemia further progressed, with necrosis initially involving the foot and subsequently extending proximally to the leg.

Given the progression to extensive irreversible tissue damage and non-salvageable limb ischemia, a below-knee amputation was performed. Postoperative recovery was uneventful. The patient was referred for rehabilitation and structured smoking

CESSATION FOLLOW-UP

DISCUSSION

Buerger's disease, also known as thromboangiitis obliterans, is a nonatherosclerotic, inflammatory, segmental, and occlusive vascular disorder affecting small- and medium-sized arteries and veins of the extremities. It predominantly occurs in young male smokers, typically younger than 50 years. This epidemiologic pattern is well established in the literature and reflects a strong and consistent association with tobacco exposure, which remains the primary environmental risk factor.

In the present case, the patient's demographic characteristics and smoking history are consistent with previously reported series, which emphasize that thromboangiitis obliterans predominantly affects heavy smokers, often in a dose-dependent manner. Although historically considered a disease of men, recent reports suggest that women may also be affected, with an apparent increase in reported cases, possibly reflecting changes in global smoking patterns [3-4].

Although its precise etiology remains unclear, tobacco exposure is considered the central pathogenic factor. Proposed mechanisms include immune-mediated vascular injury, endothelial dysfunction, and a prothrombotic inflammatory state.

Clinically, Buerger's disease classically presents with distal extremity ischemia, including rest pain, digital ulcers, and Raynaud phenomenon. Involvement of the upper or lower limbs typically affects distal segments of small- and medium-sized vessels and is often bilateral. In contrast, our patient presented with intermittent claudication and a leg ulcer, with severe and atypical ischemic manifestations. The presence of

complete thrombosis of the subrenal abdominal aorta extending to the iliac bifurcation represents an unusual and advanced presentation. Although rare, proximal large-vessel involvement has been reported in the literature, particularly in patients with delayed diagnosis or persistent heavy smoking, suggesting a more aggressive disease phenotype in such cases.

From a diagnostic perspective, thromboangiitis obliterans is a diagnosis of exclusion, based on clinical presentation, imaging findings, and the absence of alternative causes of vascular occlusion, such as atherosclerosis, diabetes mellitus, autoimmune disease, or thrombophilia. No single diagnostic test is pathognomonic. In this case, extensive laboratory and imaging evaluations excluded autoimmune, hematologic, and neoplastic conditions, thereby supporting the diagnosis. The initial histopathological findings suggestive of pyoderma gangrenosum highlight the diagnostic complexity and underscore the importance of correlating clinical, histological, and vascular findings. Similar diagnostic challenges have been described in prior reports in which inflammatory cutaneous lesions masked an underlying ischemic vascular etiology.

Management of thromboangiitis obliterans is centered on complete and sustained smoking cessation, which remains the cornerstone of therapy and the only intervention consistently associated with disease stabilization and improved outcomes. Pharmacologic treatments, including vasodilators, antiplatelet agents, and anticoagulants, have demonstrated limited and inconsistent benefits. Revascularization procedures may be considered in selected cases; however, their effectiveness is often restricted due to the distal, segmental, and diffuse nature of the vascular lesions. Published data indicate that advanced disease is associated with significant morbidity, with a substantial proportion of patients progressing to critical limb ischemia and requiring major amputation in severe or late-stage presentations [5-6].

In our patient, despite aggressive medical management and endovascular stenting, the clinical course was unfavorable, ultimately resulting in limb amputation. This outcome is consistent with prior reports emphasizing that delayed diagnosis and ongoing tobacco exposure are strongly associated with poor prognosis and increased risk of limb loss.

It also underscores that advanced ischemic damage is often irreversible, even with optimal intervention.

Overall, this case demonstrates both concordance with and divergence from established literature. While the epidemiologic characteristics and underlying pathophysiology are consistent with prior descriptions, the extensive proximal aortic involvement and severe disease progression represent an atypical and aggressive presentation. These findings underscore the importance of early recognition, comprehensive vascular evaluation in patients with atypical ulcers, and strict smoking cessation as the cornerstone of management.

CONCLUSIONS

Although the exact etiology of thromboangiitis obliterans remains incompletely understood, robust evidence consistently identifies tobacco use as the central factor driving both the onset and progression of the disease.

While thromboangiitis obliterans classically affects distal small- and medium-sized vessels, this case illustrates that occlusion may, in rare instances, involve large vessels, highlighting an atypical and more extensive vascular presentation.

Our observation reinforces the crucial role of comprehensive vascular imaging in the assessment of any leg ulcer, as early and accurate diagnosis is essential to guide appropriate management, prevent complications, and improve patient outcomes.

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