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A Case of Livedoid Vasculopathy with Rapid Response to Rivaroxaban: Case Report and Current Treatment Overview

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Abstract

Introduction: Livedoid vasculopathy (LV) is a rare chronic microvascular disorder characterized by persistent skin ulcers and atrophie blanche. It is often misdiagnosed as other conditions such as chronic venous insufficiency or necrotizing vasculitis.

Case presentation: A 42-year-old woman presented with painful erythematous patches and superficial ulcers affecting both her lower limbs. She was initially treated for chronic venous insufficiency and later for nonspecific necrotizing vasculitis, but without improvement. Skin biopsy confirmed the diagnosis of livedoid vasculopathy. The patient did not respond to corticosteroids but showed rapid improvement after switching to oral rivaroxaban, with dry ulcers, reduced pain, and fading erythema observed after 7 days.

Conclusion: This case highlights the essential role of skin biopsy in accurately diagnosing LV and the effectiveness of targeted anticoagulant therapy with rivaroxaban in achieving rapid symptom relief.

Keywords: Livedoid vasculopathy; cutaneous microvascular thrombosis; chronic leg ulcer; atrophie blanche; rivaroxaban.

1. INTRODUCTION

Livedoid vasculopathy (LV) is a rare superficial microvascular disease characterized by thrombosis in small dermal vessels, leading to tissue ischemia, chronic skin ulceration, and the development of distinctive porcelain-white atrophic scars (atrophie blanche). Its estimated annual incidence is approximately 1 in 100,000 people annually and it predominantly affects young to middle-aged women, with a female-to-male ratio of about 3:1 and a mean age of onset between 35 and 53 years [1].

The pathogenesis of LV involves a complex interplay of prothrombotic factors, reduced fibrinolytic activity, and endothelial injury, resulting in microvascular thrombosis [2]. Approximately 50–77% of LV patients have at least one coagulation abnormality, though the absence of such findings does not exclude the diagnosis [3].

Clinically, LV presents insidiously with painful purpuric macules or erythematous patches, which progress to persistent superficial ulcers and heal with white atrophic scarring. The morphology of these lesions resembles that of necrotizing vasculitis, leading to frequent misdiagnosis, especially in the early stages. In this context, skin biopsy serves as a pivotal test to confirm the thrombotic — rather than inflammatory — nature of the lesions, thereby distinguishing LV from true vasculitic entities and guiding appropriate treatment strategies [2].

We report a typical case of LV initially misdiagnosed as nonspecific necrotizing vasculitis, which showed significant improvement after transitioning to oral anticoagulation with rivaroxaban. This report highlights the essential role of histopathology in diagnosing LV and the therapeutic potential of targeted anticoagulants in clinical practice.

2. CASE PRESENTATION

A 42-year-old female patient with no known underlying medical conditions was admitted due to painful ulcers and erythema on the lower limbs. One month prior to admission, she began to notice erythematous patches appearing on both shins and dorsum of the feet, without any accompanying systemic symptoms. She initially visited a local healthcare facility, where she was diagnosed with chronic venous insufficiency of the lower limbs and treated with Daflon 500 mg, two tablets daily. However, despite treatment, the lesions did not improve and progressively expanded, accompanied by the development of painful ulcers.

Seven days prior to admission, due to worsening symptoms, the patient sought evaluation at a dermatology specialty hospital for definitive diagnosis and management. At that time, she was provisionally diagnosed with nonspecific necrotizing vasculitis. Treatment was initiated with antibiotics (amoxicillin–clavulanate), alphachymotrypsin, and a skin biopsy was performed. The patient was then discharged for outpatient follow-up.

As her condition continued to deteriorate, with increasing pain and swelling in both feet, she returned for a follow-up and was advised to be hospitalized for inpatient treatment.

Upon admission, physical examination revealed superficial ulcers with well-demarcated borders and dry bases, surrounded by purpuric patches and mild edema on the dorsum of both feet (Figure 1A). Initial laboratory tests—including hematology, biochemistry, electrolytes, and coagulation profile (PT: 10.4 seconds; TT: 16.6 seconds; aPTT: 24.2 seconds; bleeding time: 3 minutes; clotting time: 8 minutes) — were all within normal limits. Histopathological examination of the skin biopsy confirmed the diagnosis of livedoid vasculopathy (Figure 3A-B).

Regarding treatment, the patient was prescribed methylprednisolone at a dose of 32 mg/day for two days. However, during this period, the clinical condition showed signs of deterioration: pain and edema did not improve, the erythematous base became more pronounced, and the ulcers continued to exude. The treatment regimen was then switched to rivaroxaban at 10 mg/day. After seven days of rivaroxaban therapy, the lesions demonstrated marked improvement: the ulcers became dry with reduced exudation and no further deepening; erythematous areas gradually faded; and both pain and edema significantly decreased.

The patient was discharged with instructions to continue rivaroxaban at a maintenance dose of 10 mg/day. At the two-month follow-up, the ulcers had completely healed with scar formation, no new lesions had developed, and both pain and edema had fully resolved. The patient's general condition was stable, with no signs of cachexia or impaired mobility.

Notably, the patient experienced no serious adverse effects related to the treatment, including bleeding complications — a common concern with oral anticoagulant use.

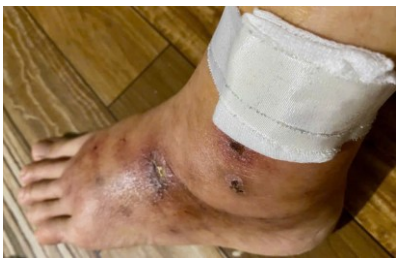

Figure 1: Cutaneous lesions before hospital admission: Superficial ulcers with well-defined borders and dry bases, surrounded by purpuric macules and mild edema on the dorsum of both feet.

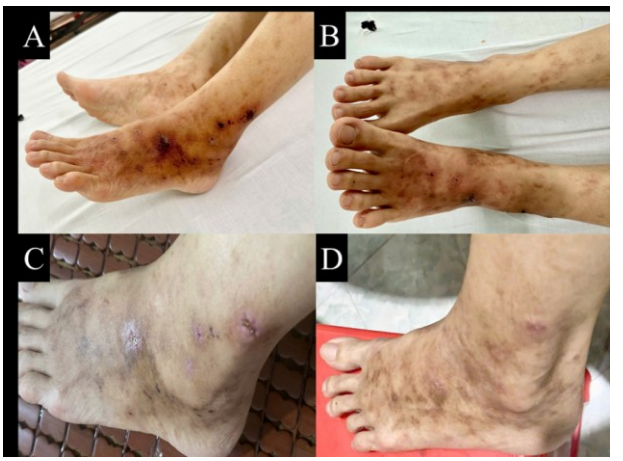

Figure 2: A-B. Cutaneous lesions on day 4 of hospitalization: Several dry ulcers with dark brown crusts, measuring 0.2–0.5 cm, on a background of dusky erythema over the dorsum of both feet. Ill-defined, non-blanching erythematous patches and areas are also present on the shins and dorsum of both feet.

C. Cutaneous lesions on day 7 of hospitalization: The dorsum of the feet shows multiple small, dried ulcers with brown crusts. Interspersed are hyperpigmented patches and ivory-white, stellate-shaped atrophic scars with surrounding hyperpigmented borders.

D. Cutaneous lesions after two months: The ulcers have completely healed, leaving only a few flat, ivory-white atrophic scars accompanied by diffuse post-inflammatory hyperpigmentation. No signs of acute inflammation are present; the skin appears dry and stable.

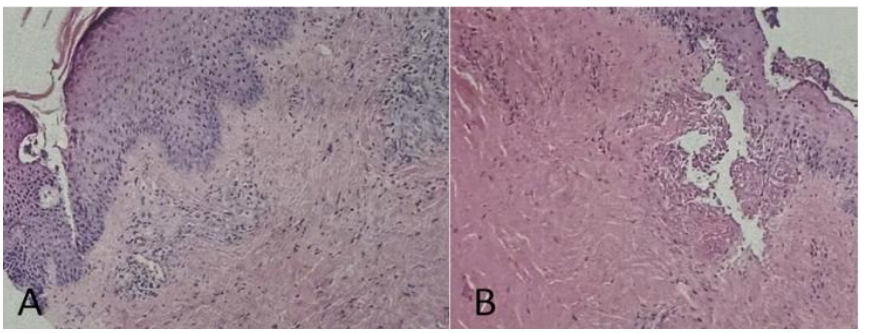

Figure 3: A-B. Histopathological findings: Mild epidermal hyperplasia. In the dermis, numerous blood vessels with thickened, hyalinized walls are observed, along with intraluminal fibrin deposition due to thrombosis, accompanied by hemorrhage and mild perivascular lymphocytic infiltration.

3. DISCUSSION

Livedoid vasculopathy (LV) is a rare but clinically significant condition due to its chronic course, often associated with persistent pain and non-healing skin ulcers, which markedly impair quality of life. Unlike necrotizing vasculitis—which is characterized by immune-mediated inflammatory mechanisms—LV is now considered the result of localized microvascular thrombosis .

Histopathology plays a central role in differentiating these two entities: while necrotizing vasculitis typically shows vascular wall necrosis, neutrophilic infiltration, and granular complement deposition, LV is characterized by fibrin-rich thrombi occluding small dermal vessels, along with endothelial proliferation and fibrin deposition, but without clear evidence of inflammation or vascular wall necrosis [4]. This fundamental pathophysiological distinction has important therapeutic implications, as it indicates that anticoagulant therapies targeting the underlying mechanism of disease play a more central role than anti-inflammatory agents alone.

In this case, the skin biopsy revealed dilated superficial vessels filled with erythrocytes and fibrin thrombi, without evidence of vessel wall necrosis or perivascular inflammatory infiltrates—findings that are characteristic of LV and inconsistent with necrotizing vasculitis. The absence of vasculitic features carries diagnostic significance comparable to the presence of thrombotic changes, as it reinforces the non-inflammatory nature of the disease. These histopathological findings, together with the clinically progressive ischemic presentation, provided a clear scientific rationale for adjusting the therapeutic strategy.

In clinical practice, both the indication for skin biopsy and the biopsy technique are crucial for establishing a definitive diagnosis. Biopsy should be performed at the edge of a newly formed ulcer, with a depth extending at least into the deep dermis to maximize the likelihood of detecting characteristic thrombotic changes [4]. In contrast, biopsies taken from stellate scars [atrophie blanche] or from the base of chronic ulcers often reveal only fibrotic tissue, increasing the risk of false-negative results.

In a French cohort study, Gardette et al. (2018) confirmed the diagnostic value of histopathology in LV, reporting that 100% of patients had thrombi in tissue samples, and 73% showed perivascular lymphocytic infiltration without features of necrotizing inflammation [5].

In addition to routine histopathology, direct immunofluorescence staining may occasionally reveal deposition of fibrin, C3, and immunoglobulins—predominantly IgM—but such deposition is typically non-specific, diffuse, and lacks a granular pattern. This helps distinguish LV from immune complex-mediated vasculitis, which characteristically presents with granular deposition and is often associated with decreased serum complement levels. Therefore, properly performed skin biopsy remains the most valuable tool for definitive diagnosis and differentiation of LV from similar vasculitic conditions [4].

Another important pathogenic factor frequently observed in LV is an underlying prothrombotic state. Studies have shown that approximately 50–77% of LV patients have at least one coagulation abnormality. The most commonly reported include factor V Leiden mutation, protein C or protein S deficiency, antiphospholipid syndrome (APS), or elevated lipoprotein(a) levels [4]. This high prevalence suggests that LV is not merely a localized cutaneous

disorder but may represent a dermatologic manifestation of a systemic thrombotic syndrome such as APS. This underscores the importance of thrombophilia screening in the evaluation of patients, particularly in cases characterized by recurrence, early onset, or poor response to treatment.

In our case, the patient underwent only basic coagulation tests (PT, aPTT, TT, bleeding time, and clotting time), all of which were within normal limits. Advanced investigations for thrombophilia screening (e.g., Factor V Leiden mutation, antiphospholipid antibodies, protein C/S deficiency) were not performed; therefore, an underlying hypercoagulable state cannot be definitively excluded. Although several prothrombotic factors have been identified, it is important to emphasize that a substantial proportion (nearly half) of patients with LV have no identifiable precipitating factor [3] . These cases are classified as idiopathic LV, similar to our patient. Accordingly, the absence of detectable coagulation abnormalities does not exclude the diagnosis of LV [6].

Notably, despite the absence of comprehensive screening, the patient exhibited a marked clinical response to rivaroxaban therapy: the ulcers healed with scarring, pain and edema improved, and no new lesions developed during short-term follow-up. This suggests that even in the absence of confirmed congenital or acquired coagulopathy, oral anticoagulation may still provide significant clinical benefit. However, to ensure long-term therapeutic success—particularly regarding the optimal duration of anticoagulation and the prevention of thrombotic recurrence—it is important to consider implementing a thrombophilia workup during the follow-up phase. Conversely, in cases where patients fail to respond to anticoagulant therapy, the diagnosis should be reassessed, with particular attention to the possibility of systemic vasculitis or connective tissue disease.

Current treatment strategies for livedoid vasculopathy (LV) focus on symptom control—particularly relief of ischemic pain—promotion of ulcer healing, and prevention of recurrence [5]. Given the primarily thrombotic pathogenesis, anticoagulation is considered the cornerstone and mainstay of therapy. Indeed, recent studies have shown that approximately 98% of LV patients receive anticoagulant therapy [5], with rivaroxaban—a direct oral factor Xa inhibitor—being the most commonly used agent, prescribed in about 54% of cases. The preference for rivaroxaban is largely due to its high efficacy, convenient oral formulation, fixed dosing, and lack of need for INR monitoring [5].

Regarding dosing, studies have used rivaroxaban for LV at doses ranging from 10 to 20 mg per day. During the active phase of the disease, some authors recommend initiating therapy at a higher dose (15–20 mg/day), followed by a reduction to a maintenance dose of 10 mg/day once a stable clinical response is achieved [7]. Lower doses (10 mg/day) have been reported to be better tolerated while still providing therapeutic benefit in many patients, although more severe cases may require prolonged treatment at higher doses. In the RILIVA trial, Weishaupt et al. (2016) demonstrated that rivaroxaban led to improvement in pain and lesion healing after 12 weeks of treatment. The protocol used included an initial loading dose of 10 mg twice daily, followed by a maintenance dose of 10 mg once daily [5]. A systematic review of 73 patients reported a clinical response rate of 82.2%, with a low incidence of serious adverse effects [5]. More recently, retrospective data published

in *JAAD International* (2023–2024) further supported the role of rivaroxaban in long-term maintenance therapy and its effectiveness when reintroduced during disease flares [8].

In our case, the decision to initiate rivaroxaban at a dose of 10 mg/day was based on the specific clinical characteristics: lesions were predominantly superficial and confined to the skin, there was no evidence of systemic thrombosis, and basic coagulation tests were within normal limits. In addition, the potential bleeding risk associated with anticoagulation therapy was carefully considered. Although the phase II trial mentioned an initial dose of 20 mg, its primary objective was rapid pain control. However, in our patient, the baseline pain was not severe. Therefore, to optimize safety and minimize the risk of dose-related adverse effects, we opted for a more cautious starting dose of 10 mg/day. This decision is also consistent with previous clinical reports. For example, Lee et al. (2015) demonstrated that their patients showed clinical improvement with rivaroxaban 10 mg within 7 days, similar to the response observed in our case [9].

Currently, there are no official guidelines regarding the optimal duration of rivaroxaban therapy in the treatment of LV. Studies and case reports suggest that treatment duration typically ranges from several months to approximately one year. Many clinical reports have documented an average treatment period of 3–8 months: some patients achieved symptom resolution within 3–4 months and continued therapy thereafter [6], while others attained complete remission after 8–12 months.

In clinical practice, most authors recommend a minimum treatment duration of 3–6 months, followed by individualized adjustments based on clinical response and the risk of recurrence [10].

The decision to discontinue rivaroxaban is primarily based on clinical criteria and a careful balance between benefits and risks. Discontinuation may be considered when the patient has achieved complete clinical remission—characterized by healed skin ulcers (leaving atrophic white scars), significant pain relief, and the absence of new lesions [11].

However, recurrence after cessation is not uncommon, with a reported average relapse interval of approximately 24–25 weeks. Therefore, close monitoring is essential, and resumption of rivaroxaban should be considered in the event of disease flare-ups [4,10,12].

Furthermore, decisions regarding discontinuation or dose reduction must also take into account the risk of bleeding complications, particularly heavy menstrual bleeding in women and gastrointestinal hemorrhage [4,10]. For this reason, monitoring for signs of bleeding and assessing renal function during treatment is essential. Dose reduction should be considered in high-risk populations, such as elderly patients or those with renal impairment.

Low-molecular-weight heparin (LMWH) is also a potent treatment option, particularly in the acute phase, due to its rapid onset of action and effective pain control. Although long-term use is limited by the need for subcutaneous injections, LMWH is still recommended as a first-line therapy by many international guidelines, alongside oral anticoagulants.

According to the German S1 guidelines (2021), treatment typically begins with therapeutic-dose LMWH, followed by a step-down to intermediate dosing once symptoms stabilize. Oral rivaroxaban may then be introduced for maintenance therapy to improve long-term adherence [8].

Warfarin and other vitamin K antagonists

(VKAs) are no longer preferred treatment options due to several limitations, including the need for regular INR monitoring, high potential for drug interactions, and delayed onset of action. However, VKAs still play a role in patients with confirmed hypercoagulable states, such as high-risk antiphospholipid syndrome (APS), where rivaroxaban may be less effective or contraindicated [8].

Finally, intravenous immunoglobulin (IVIG) may serve as a salvage therapy in refractory cases. Although its use is limited by high cost and the requirement for intravenous administration, IVIG has shown a high response rate in several cohort reports. In a French study, 6 out of 8 patients who were unresponsive to anticoagulation therapy experienced clinical improvement after receiving IVIG, suggesting it may be an effective adjunctive option when standard therapies have been optimized [4,8].

4. CONCLUSION

Livedoid vasculopathy is a chronic thrombotic microvascular disorder with persistent clinical manifestations that are often misdiagnosed as forms of necrotizing vasculitis. Properly performed skin biopsy, combined with appropriate laboratory investigations—particularly thrombophilia screening—plays a crucial role in establishing an accurate diagnosis and distinguishing LV from other systemic diseases. Anticoagulant therapy, especially with rivaroxaban and low-molecular-weight heparin, is currently considered the cornerstone of LV treatment due to its high efficacy in relieving pain, promoting ulcer healing, and preventing recurrence. Drug selection should be based on disease phase, individual risk factors, treatment adherence potential, and underlying comorbidities. In refractory cases or those unresponsive to anticoagulation alone, adjunctive therapies such as IVIG or immunomodulatory agents may be considered. Early recognition and a rational, individualized treatment approach are key to improving prognosis and quality of life in patients with livedoid vasculopathy.

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