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# Tofacitinib Combined with Hirudoid and Jinyin Peptide for the Treatment of Livedoid Vasculopathy: A Case Report

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## Abstract

A 25-year-old woman presented with a 10-year history of recurrent eruptions on both lower extremities, with exacerbation during the preceding month. Routine blood and urine tests, blood biochemistry, lipid profile, humoral immune indices, and rheumatologic screening showed no obvious abnormalities; antinuclear antibody and the ENA-17 panel were negative, and color Doppler ultrasonography of the bilateral lower-extremity arteries and deep veins was unremarkable. Histopathological examination of the skin revealed fibrinoid necrosis in the superficial dermal vessel walls and intravascular thrombus formation. Livedoid vasculopathy was diagnosed. After treatment with tofacitinib combined with topical Hirudoid and Jinyin Peptide, the lesions healed.

## Keywords

Tofacitinib, Livedoid Vasculopathy, Hirudoid, Jinyin Peptide

## 1. Introduction

Livedoid vasculopathy (LV) is a rare, recurrent, and refractory thrombotic cutaneous disease characterized by painful, persistent punched-out ulcers around the ankles, often accompanied by livedo reticularis and healing with porcelain-white atrophic scars (atrophie blanche). The disease typically affects bilateral lower extremities and can severely impair quality of life. Its pathogenesis is complex and incompletely understood, involving hypercoagulable states, inflammatory and immune factors, and metabolic abnormalities. Current treatments are largely empirical, including anticoagulants, antiplatelet agents, glucocorticoids, and various combination therapies, but response is variable and many patients remain refractory.

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Janus kinase (JAK) inhibitors, such as baricitinib and tofacitinib, have shown potential in inflammatory and thrombotic skin disorders by modulating the JAK-STAT signaling pathway and downstream cytokines. Here we report a case of recurrent LV successfully treated with tofacitinib combined with topical Hirudoid and Jinyin Peptide, aiming to provide a reference for the management of refractory LV.

## 2. Clinical Data

A 25-year-old woman presented with recurrent eruptions on both lower extremities for 10 years, with aggravation for 1 month. Ten years earlier, she had developed scattered petechiae, ecchymoses, vesicles, and hemorrhagic bullae on both lower extremities without an obvious precipitating factor. The lesions were symmetrically distributed and accompanied by joint swelling and mild pain or discomfort. She had received multiple treatments at outside hospitals; the details were unknown, but the response was acceptable and the disease remained controlled without recurrence for 5 years.

On June 9, 2021, the lesions worsened again without an obvious trigger, and the patient attended our department. A biopsy of the left ankle lesion showed fibrinoid necrosis in the superficial dermal vessel walls and intravascular thrombus formation, consistent with the pathological features of livedoid vasculopathy (**Figure 1**). A diagnosis of livedoid vasculopathy was made. She received circulation-improving and anti-inflammatory therapy, including ginkgo leaf extract and dipyridamole injection and compound glycyrrhizin, and was discharged after the disease stabilized. On May 18, 2022, the lesions recurred without an obvious trigger. She returned to our department and received circulation-improving, anti-inflammatory, and anticoagulant therapy, including ginkgo leaf extract and dipyridamole injection, compound glycyrrhizin, pentoxifylline, and rivaroxaban. The disease was controlled, and she was discharged.

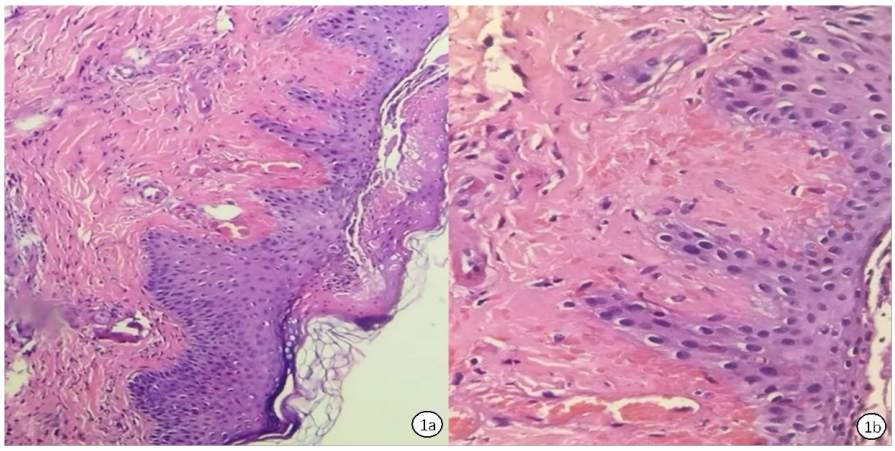


**Figure 1.** Histopathological examination of the left ankle lesion. Fibrinoid necrosis was observed in superficial dermal vessel walls, with intravascular thrombus formation.

The patient subsequently presented to our department again because of recur-

rent and aggravated skin lesions. She had been generally healthy previously and reported no family history of hereditary disease. General physical examination showed no obvious abnormality. Dermatological examination revealed a 3 cm × 2 cm × 1 cm punched-out ulcer on the left ankle. On the right lower limb and ankle, one soybean-sized and three broad bean-sized moth-eaten ulcers were observed. Erythema and swelling were present around the ulcers on both lower extremities (Figure 2).

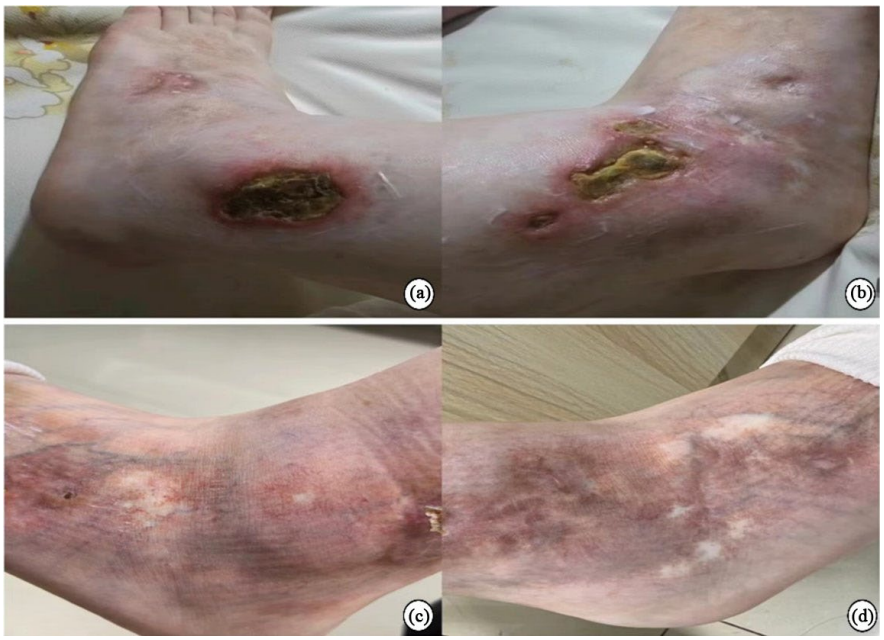


**Figure 2.** Clinical presentation and outcome. (a), (b) A 3 cm × 2 cm × 1 cm punched-out ulcer was observed on the left ankle, and one soybean-sized and three broad bean-sized moth-eaten ulcers were present on the right lower limb and ankle, with erythema and swelling around bilateral lower-extremity ulcers. (c), (d) Residual hyperpigmentation and linear superficial white atrophic scars were observed at the previous ulcer sites after treatment.

Laboratory tests, including routine blood and urine tests, blood biochemistry, lipid profile, humoral immunity, and rheumatologic screening (C-reactive protein, Erythrocyte Sedimentation Rate, Rheumatoid Factor, Anti-Streptolysin O), showed no obvious abnormalities. Antinuclear antibody and the ENA-17 panel were negative. However, comprehensive thrombophilia screening (e.g., antiphospholipid antibodies, protein C/S, antithrombin, homocysteine, cryoglobulins) was not performed, which is a limitation of this case. Color Doppler ultrasonography of the bilateral lower-extremity arteries and deep veins showed no abnormality. Based on the clinical and histopathological findings, livedoid vasculopathy was diagnosed.

Given the recurrent and refractory nature of the ulcers despite prior treatments with circulation-improving, anti-inflammatory, and anticoagulant agents, a JAK inhibitor was considered. Tofacitinib was chosen for its dual anti-inflammatory and immunomodulatory effects, as well as its cost advantage compared with other

JAK inhibitors. Before initiating tofacitinib, previous systemic therapies were discontinued.

The patient was treated with tofacitinib 5 mg per tablet, two tablets daily, for 12 weeks, combined with topical Hirudoid and Jinyin Peptide. Safety monitoring: Before treatment, baseline screening including complete blood count, liver and renal function tests, lipid profile, and screening for latent tuberculosis and viral hepatitis was performed; no abnormalities were detected. During the 12-week treatment, laboratory monitoring was performed every 4 weeks, and no significant adverse events (including infection, cytopenia, or liver enzyme elevation) were observed. The patient tolerated the regimen well.

Clinical course after treatment: The patient's ulcer-associated pain began to subside within the first two weeks of treatment. Partial re-epithelialization was observed at week 6, and complete healing of all ulcers was achieved by week 10. At the 12-week follow-up, the eruptions had healed, and residual hyperpigmentation and linear superficial white atrophic scars remained at the previous ulcer sites (**Figure 2**). The patient was followed for an additional 6 months after the 12-week treatment course, during which no recurrence was observed.

## 3. Discussion

Livedoid vasculopathy is a rare, recurrent, and refractory cutaneous disease that manifests as painful, persistent, punched-out ulcers around the ankle. In some patients, obvious livedo reticularis can be observed around the ulcers. The ulcers may heal with porcelain-white atrophic scars, termed atrophie blanche [1]. The disease usually affects the bilateral lower extremities, ankles, and dorsum of the feet, and it can severely impair quality of life. A retrospective study reported that baricitinib showed favorable efficacy and safety in refractory livedoid vasculopathy [2]. In our clinical practice, the JAK inhibitor tofacitinib combined with Hirudoid and Jinyin Peptide achieved a satisfactory therapeutic effect.

Livedoid vasculopathy is rare, and reported sample sizes are generally small. Its pathogenesis is complex and remains incompletely understood. Current evidence suggests that the mechanism may involve hypercoagulable and prothrombotic factors, including abnormalities in platelets, coagulation and anticoagulation systems, and fibrinolysis; metabolic factors, including homocysteine and lipoprotein(a); and inflammatory and immune factors [3]. The main therapeutic objectives are to improve skin lesions, relieve pain, and reduce recurrence. At present, treatment is mostly empirical and includes anticoagulants, antiplatelet agents, glucocorticoids, thrombolytic therapy, hyperbaric oxygen therapy, intravenous immunoglobulin, vitamin supplementation, ultraviolet therapy, and one or more of these approaches in combination with other therapies; overall efficacy is variable but can be acceptable [4].

The JAK family mainly consists of JAK1, JAK2, JAK3, and tyrosine kinase 2. It has been reported that the JAK3-STAT signaling pathway can drive JAK3 inflammation-related microvascular formation and intimal hyperplasia in inflamed ves-

sel walls. The immunosuppressive effect of JAK3 inhibitors is particularly evident for effector cytokines and can significantly reduce IL-17 levels [5]. Tofacitinib is a JAK inhibitor that primarily inhibits JAK3 and also inhibits JAK1 and JAK2 [6]. It can regulate the JAK-STAT signaling pathway and suppress the expression of downstream inflammatory cytokines, including IL-1β, IL-6, and TNF-α [7].

A previous report showed that adalimumab achieved good efficacy in refractory livedoid vasculopathy [8]. In addition, IL-17 and TNF-α have been reported to act synergistically on vascular endothelial cells and vessel walls, promoting coagulation, increasing platelet aggregation, and leading to thrombus formation [9]. Intravascular thrombosis is one of the important mechanisms underlying livedoid vasculopathy. Compared with other JAK inhibitors, tofacitinib has a cost advantage, which may reduce patients' economic burden and improve treatment adherence. Because this disease is characterized by punched-out ulcers, Hirudoid may help control local inflammation, improve blood circulation in the affected area, and promote exudate absorption, while Jinyin Peptide may facilitate wound repair.

In this single case, the combination of tofacitinib, topical Hirudoid, and Jinyin Peptide was associated with ulcer healing. However, as this is an observational finding from a combined regimen, the specific contribution of tofacitinib alone remains unclear. The proposed mechanistic rationale is inferential based on known JAK-STAT pathways and cytokine effects, rather than disease-specific proof. Further studies with larger sample sizes are needed to validate the efficacy of this approach.

## Ethics Statement

This study is a single case report. According to the policy of our Institutional Review Board (IRB), ethical approval was not required. The patient has provided written informed consent for the publication of this case report and any accompanying images.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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