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ORIGINAL RESEARCH

Clinical Characteristics and Disease Course of Livedoid Vasculopathy: A 10-Year Retrospective Analysis

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Objective: Livedoid vasculopathy (LV) is a rare, chronic, recurrent thrombo-occlusive cutaneous disorder, which is also known as atrophie blanche (AB). Its characteristic clinical manifestations include livedo racemosa, painful ulcerations, and white atrophic scars predominantly on the lower extremities. This study aimed to investigate the clinical appearance, histological features and laboratory test results, treatment strategies, and clinical outcomes of LV to provide evidence for clinical diagnosis and management.

Methods: We conducted a retrospective analysis of clinical data from 69 LV patients diagnosed in the First Affiliated Hospital of Sun Yat-sen University between January 2014 and December 2024. Data encompassed demographic characteristics, clinical manifestations, histopathological features, laboratory investigations, treatment regimens, and follow-up results. Treatment efficacy was assessed based on ulcer healing time and patient-reported symptom improvement.

Results: The median age was 31 years (range: 11–85 years), with females constituting 69.6% (48/69). Only 5 patients (7.3%, 5/69) received an initial diagnosis of LV. The predominant clinical presentations were recurrent painful ulcerations (89.9%, 62/69), white atrophic scars (82.6%, 57/69), purpura (84.1%, 58/69), livedo racemosa (75.4%, 52/69) and local pain (85.5%, 59/69) on the lower extremities. Treatment modalities primarily included anticoagulant therapy, antiplatelet therapy, corticosteroids, lipid-lowering therapy, immunosuppressive agents, traditional Chinese medicine and physical therapy modalities.

Conclusion: LV predominantly affects young and middle-aged females. Combination therapy with anticoagulant, antiplatelet and corticosteroids significantly contributes to ulcer healing and rapid pain relief. Long-term follow-up is necessary to monitor for potential progression to systemic diseases. As a single-center retrospective study, these findings warrant further validation through multicenter prospective research.

Keywords: livedoid vasculopathy, anticoagulant therapy, antiplatelet therapy, immunomodulatory agents

Introduction

Livedoid vasculopathy (LV) is a rare, chronic cutaneous disorder characterized pathologically by thrombotic microangiopathy affecting small dermal vessels.[1] With an estimated incidence of approximately 1 per 100,000, LV poses significant diagnostic and therapeutic challenges due to its rarity and clinical complexity.[2] Its histopathological hallmarks include fibrinoid necrosis of superficial dermal vessels, thrombosis, and secondary ischemic damage to the epidermis.[3] Clinically, LV typically manifests as recurrent painful ulcerations, livedo racemosa, and characteristic white atrophic scars, predominantly affecting the lower extremities, which significantly impairs patients' quality of life.[4] Although the precise etiology of LV remains incompletely understood, current evidence suggests its pathogenesis is closely linked to hypercoagulable states, microcirculatory dysfunction, and immunological dysregulation.[5] Some patients have coexisting underlying conditions such as antiphospholipid syndrome or systemic lupus

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erythematosus.[6–8] Beyond these primary manifestations, LV can lead to several serious complications, including chronic neuropathic pain, secondary bacterial infections, and substantial mobility limitations. Furthermore, its association with systemic hypercoagulable states elevates the risk of major thrombotic events, such as deep vein thrombosis and pulmonary embolism, underscoring the necessity for comprehensive evaluation and long-term monitoring.[9,10]

However, due to the non-specific clinical presentation of LV and its symptomatic overlap with conditions like vasculitis and venous insufficiency, the rate of clinical misdiagnosis is high. Previous literature reports an initial diagnostic accuracy rate of less than 20%,[11] which aligns with our finding that only 5.8% (4/69) of patients in this cohort received a correct diagnosis at their first presentation, highlighting the diagnostic complexity of LV. Furthermore, while treatment strategies including anticoagulation and corticosteroids are widely attempted, robust evidence regarding the optimal therapeutic approach and long-term outcomes remains controversial.[12,13] Many existing studies, limited by small sample sizes or short follow-up durations, have been unable to comprehensively evaluate treatment response and recurrence risk.[14,15]

Given this contest, we systematically summarized the clinical data LV patients diagnosed in the First Affiliated Hospital of Sun Yat-sen University over a recent 10-year period. Utilizing this large-sample, longitudinal single-center dataset, our study aims to delineate the disease characteristics and natural history of LV. This will provide a foundation for early recognition, targeted therapeutic interventions, and prognostic assessment, while also establish a basis for future multicenter collaborative research.

Materials and Methods

Study Design and Patient Selection

This retrospective cohort study was conducted in the First Affiliated Hospital of Sun Yat-sen University. We reviewed the medical records of 69 consecutive patients diagnosed with LV between January 2014 and December 2024. The study protocol was approved by the Ethics Committee of the First Affiliated Hospital of Sun Yat-sen University, and the research was conducted in accordance with the ethical standards outlined in the Declaration of Helsinki. Given the retrospective nature of the study and the use of de-identified patient data, the requirement for informed consent was waived by the Institutional Review Board.

Diagnostic Criteria

The diagnosis of LV was based on characteristic clinical features, including typical painful ulcerations, livedo racemosa, purpura, and white atrophic scars on the lower extremities and histopathological examination of skin biopsies characterized by thrombosis within small dermal vessels, fibrin deposition within vessel walls, possible extravasation of erythrocytes, absent or minimal perivascular inflammation, and no significant perivascular neutrophilic infiltration and characteristic of leukocytoclastic vasculitis is observed in the affected vessels.

Data Collection

The following information was systematically extracted from electronic medical records: demographic characteristics, clinical manifestations, past medical history, laboratory investigations, imaging studies, and treatment regimens.

Inclusion and Exclusion Criteria

The diagnosis of LV was established based on the combination of characteristic clinical manifestations and confirmatory characteristic histopathological findings (as mentioned in the diagnostic criteria).

Inclusion Criteria: All consecutive patients hospitalized at our institution between January 2014 and December 2024 who fulfilled the above diagnostic criteria for LV and had complete medical records were considered for inclusion.

Exclusion Criteria: Patients were excluded if they had incomplete clinical data, missing clinical photographs, or photographs of suboptimal quality that precluded reliable assessment.

Statistical Analysis

Descriptive statistics are presented as mean combined with standard deviation and frequencies (percentages) for categorical variables. Categorical variables were evaluated using the chi-square test or Fisher's exact test, as appropriate. Statistical significance was defined as $p < 0.05$ (SPSS version 21.0).

Results

Baseline Clinical Characteristics

We identified 69 patients initially presenting to our hospital between January 2014 and December 2024 who ultimately received a confirmed diagnosis of LV. Within this cohort, the median age was 31 years, and ages ranged from 11 to 85 years. Forty-eight patients (48/69, 69.6%) were female. The mean duration from symptom onset to definitive diagnosis was 62.0 ± 89.0 months (corresponding to a mean disease duration of 5.4 ± 7.2 years). The mean hospital stay duration was 11.1 ± 4.3 days. Skin lesions were predominantly localized to the bilateral lower limbs in the majority of patients (56/68, 82.4%). The onset of symptoms occurred during the summer months in 44.9% (31/69) of patients (Table 1). Prior to the final LV diagnosis, 64 patients (64/69, 92.8%) had received alternative diagnoses, most commonly vasculitis, purpura, and infection. The definitive diagnosis of LV was established through subsequent skin biopsies performed at our institution or pathology consultation of prior pathological specimens.

Concomitant Symptoms

The predominant clinical manifestations in these patients were ulcerations (62/69, 89.9%), purpura (58/69, 84.1%), and white atrophic scars (57/69, 82.6%) (Figure 1A). Livedo racemosa was observed in 75.4% (52/69) of patients (Figure 1B), and local pain accompanied skin lesions in 85.5% (59/69) of cases. Concomitant systemic conditions included hypertension in 9 patients (13.0%) and diabetes mellitus in 4 patients (5.8%). Lower limb edema occurred in 29.0% (20/69), while foot numbness was reported by 3 patients (4.3%). Metabolic comorbidities included hepatic steatosis (20.3%, 14/69), hyperlipidemia (23.2%, 16/69), and lower limb atherosclerosis (20.3%, 14/69). Vascular ultrasound detected thromboses in the lower limbs in 2 patients (2.9%) (Table 2).

Table 1 Baseline Clinical Characteristics of Patients with Livedoid Vasculopathy (N=69)

Characteristics	N (%)
Gender	
Male	21 (30.4%)
Female	48 (69.6%)
Age (years)	38.0±17.7
Length of Stay (days)	11.1±4.3
Duration of skin lesions (years)	5.4±7.2
Season of onset	
Spring	6 (8.6%)
Summer	31 (44.9%)
Autumn	18 (26.0%)
Winter	14 (20.2%)
Initial diagnosis from external hospital	
Livedoid vasculopathy	4 (5.7%)
Other dermatoses	64 (92.8%)
Time from initial external diagnosis to current admission (months)	62.0±89.0

Note: Data are presented as n (%) or mean ± standard deviation.

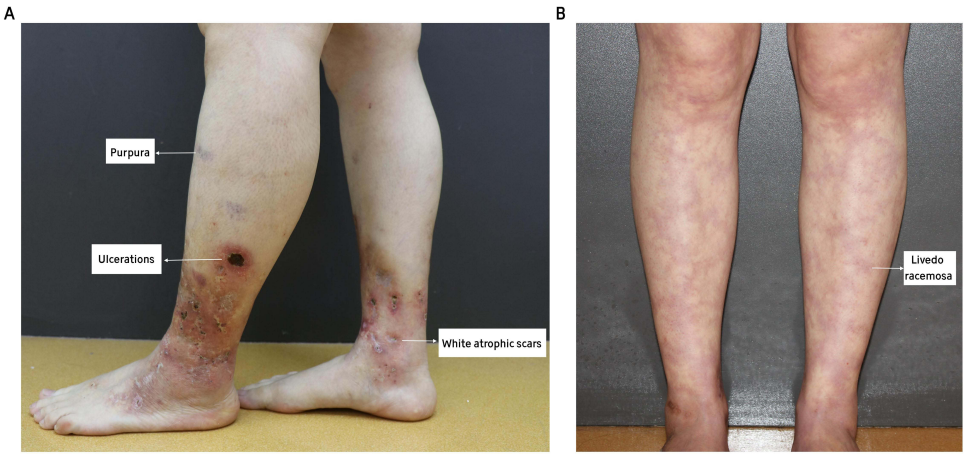

Figure 1 Clinical manifestations of livedoid vasculopathy. (**A**) Ulcerations, purpura and white atrophic scars. (**B**) Livedo racemosa.

Laboratory Findings

Laboratory investigations of 69 patients with livedoid vasculopathy revealed multiple abnormalities across various parameters. Platelet counts were abnormal in 20.6% of patients. Coagulation studies showed that 38.1% of patients showed elevated fibrinogen and 20.8% showed elevated D-dimer. It also revealed abnormal activated partial thromboplastin in 32.8% of subjects, abnormal coagulation factor VIII in 25.9%, and abnormal antithrombin III in 7.7% of patients. Normal fibrin degradation products was shown in 96.2% of patients. Lipid metabolism abnormalities was found in some patients, including hypercholesterolemia in 25.0%, elevated high-density lipoprotein in 25.0%, elevated low-density lipoprotein in 48.3% and hypertriglyceridemia in 21.7% of patients. Apolipoprotein disturbances were observed with normal A-I levels in all patients, elevated A-II in 27.3%, and elevated C-III in 59.1% of patients. Laboratory parameters demonstrated elevated serum amyloid A in 56.3% of patients, abnormal complement C3 in 42.1%, elevated rheumatoid factor in 2.1%, elevated anti-nuclear antibody in 5.3% of patients and normal anti-cyclic citrullinated peptide antibodies in all patients. Laboratory teats revealed that 44.8% of patients exhibited elevated C-reactive protein and 2.1% had elevated antistreptolysin O (Table 3).

Table 2 Concomitant Symptoms of 69 Patients with Livedoid Vasculopathy

Characteristics	N (%)
Cutaneous Symptoms and Signs	
Ulcerations	62 (89.9%)
Purpura	58 (84.1%)
Livedo racemosa	52 (75.4%)
White atrophic scar	57 (82.6%)
Pain	59 (85.5%)
Lower limb edema	20 (29.0%)
Foot numbness	3 (4.3%)
Comorbidities and Associated Conditions	
Hyperlipidemia	16 (23.2%)
Hepatic steatosis	14 (20.3%)
Lower limb arteriosclerosis	14 (20.3%)
Peripheral neuropathy symptoms	9 (13.0%)
Hypertension	9 (13.0%)
Diabetes mellitus	4 (5.8%)
Lower extremity ultrasound thrombus	2 (2.9%)

Note: Data are presented as n (%).

Table 3 Laboratory Findings of Patients with Livedoid Vasculopathy

Characteristics	Mean ± SD (Proportion of Abnormal Individuals, %)
Platelet (10^9/L)	260.0 ± 75.1 (20.6%)
Activated Partial Thromboplastin Time (S)	29.2 ± 8.6 (32.8%)
Coagulation Factor VIII (%)	139.3 ± 63.8 (25.9%)
Fibrinogen (g/L)	3.0 ± 1.3 (38.1%)
Fibrin Degradation Products (μg/mL)	2.1 ± 1.4 (3.8%)
Antithrombin III (%)	94.0 ± 20.6 (7.7%)
D-dimer (mg/L)	0.5 ± 0.5 (20.8%)
Total Cholesterol (mmol/L)	4.8 ± 1.3 (25.0%)
High-Density Lipoprotein (mmol/L)	1.3 ± 0.2 (25.0%)
Low-Density Lipoprotein (mmol/L)	3.0 ± 0.8 (48.3%)
Triglycerides (mmol/L)	1.3 ± 0.8 (21.7%)
Apolipoprotein A-I (g/L)	1.3 ± 0.3 (0.0%)
Apolipoprotein A-II (mg/L)	317.8 ± 57.4 (27.3%)
Apolipoprotein C-III (mg/L)	62.7 ± 33.1 (59.1%)
Rheumatoid Factor (IU/mL)	10.4 ± 2.7 (2.1%)
Anti-cyclic Citrullinated Peptide Antibody (U/mL)	0.6 ± 0.2 (0.0%)
Serum Amyloid A (mg/L)	21.2 ± 32.7 (56.3%)
Anti-Nuclear Antibody (U/mL)	15.4 ± 69.7 (5.3%)
Complement C3 (g/L)	1.0 ± 0.2 (42.1%)
Antistreptolysin O (IU/mL)	43.0 ± 52.2 (2.1%)
C-reactive protein (mg/L)	6.6 ± 9.5 (44.8%)

Note: Data are presented as mean ± standard deviation (SD) for all continuous laboratory variables.

Pathological Results

Skin pathological were performed for 34 patients at our hospital. The other patients were excluded from the statistical analysis, because they had previously sought medical attention elsewhere and had undergone pathological examinations prior to presenting here, and they declined repeat testing. Histopathological findings indicated that the changes were predominantly localized in the superficial to mid-dermis. Acanthosis and elongation of rete ridges were observed in 38.2% and 35.3% of patients, respectively. Increased capillary density and dilated and congested capillaries were present in 79.4% and 73.5% of patients, respectively (Figure 2A). Luminal thrombosis and occlusion was found in 55.9% of patients (Figure 2B), and fibrinoid necrosis and thickening of the vascular wall was noted in 64.7% (Figure 2C and Table 4). While no overt vasculitis was detected in any patient, sparse perivascular inflammatory cell infiltration was observed in some cases. Direct immunofluorescence (DIF) examination was performed on 29 patients, revealing varying

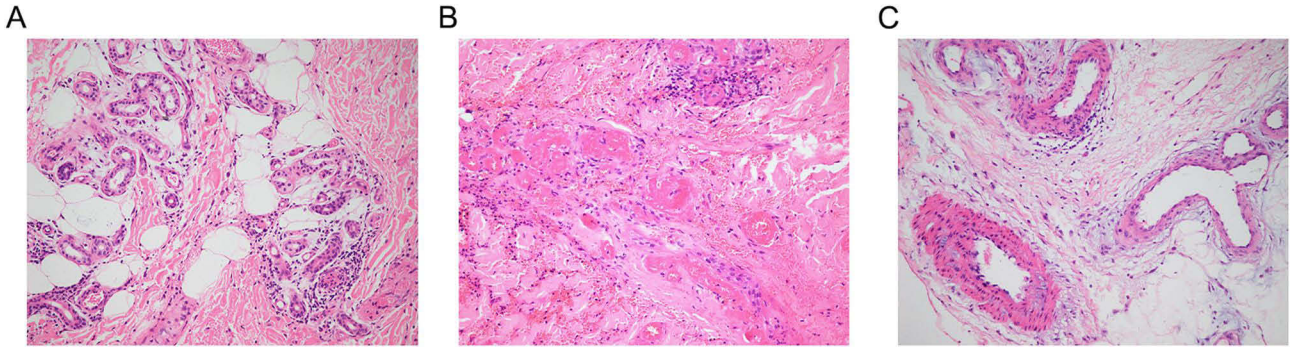

Figure 2 Skin pathological of livedoid vasculopathy. (**A**) Increased, dilated, and congested small superficial dermal blood vessels. (**B**) Fibrinoid vascular wall deposition and intraluminal thrombosis in superficial dermal vessels with extravasated erythrocytes. (**C**) Thickening of the walls of small dermal blood vessels with segmental fibrinoid degeneration.

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Table 4 Pathological Results of 34 Patients with Livedoid Vasculopathy

Pathological Features	N (%)
Epidermis	
Acanthosis	13 (38.2%)
Elongation of rete ridges	12 (35.3%)
Dermis	
Increased capillary density	27 (79.4%)
Dilated and congested capillaries	25 (73.5%)
Endothelial cell swelling and hypertrophy	16 (47.1%)
Luminal thrombosis and occlusion	19 (55.9%)
Fibrinoid necrosis and thickening of the vascular wall	22 (64.7%)
Extravasation of erythrocytes	22 (64.7%)
Perivascular focal lymphocytic infiltration	20 (58.8%)
Scattered perivascular neutrophilic infiltration	18 (52.9%)
Mild perivascular histiocytic infiltration	16 (47.1%)
Subcutis	
Congestion and erythrocyte extravasation in subcutaneous fat	13 (38.2%)

Note: Data are presented as n (%).

degrees of deposition of immunoglobulins, complement, and fibrin within the vascular walls. The most common DIF pattern involved deposition of immunoreactants in both vessels and the dermo-epidermal junction. C3 (12/29, 41.4%) was the most frequently deposited immunoreactant, followed by IgM (6/29, 20.7%), IgA (5/29, 17.2%), and IgG (1/29, 3.5%).

Treatment

During hospitalization, all 69 patients received combination therapy regimens consisting of two or more of the following interventions: anticoagulant therapy (100%, 69/69), antiplatelet therapy (82.6%, 57/69), corticosteroids (56.5%, 39/69), lipid-lowering therapy (21.7%, 15/69), immunosuppressive agents (43.5%, 30/69), vitamin supplementation (65.2%, 45/69), traditional Chinese herbal medications (66.7%, 46/69), androgen preparations (4.4%, 3/69), and hyperbaric oxygen therapy (10.1%, 7/69) (Table 5). The management of ulcers involves the application of topical therapeutic agents to promote a moist wound milieu and actively prevent superinfection. They also received general management, includeing bed rest, elevation of the affected limb, smoking cessation, and lower extremity compression therapy, such as the use of elastic stockings or bandages. Patient pain management and ulcer care are also critical components of the treatment regimen. Regarding treatment outcomes, the healing time of the ulcer in the patient's affected area is 11.5 ± 4.6 days, all

Table 5 Treatment Regimens of 69 Patients with Livedoid Vasculopathy

Characteristics	N (%)
Anticoagulant therapy	69 (100%)
Antiplatelet therapy	57 (82.6%)
Corticosteroids	39 (56.5%)
Lipid-lowering therapy	15 (21.7%)
Immunosuppressant	30 (43.5%)
Vitamin (B, C and E)	45 (65.2%)
Traditional Chinese Medicine	46 (66.7%)
Androgen	3 (4.4%)
Hyperbaric Oxygen Therapy	7 (10.1%)
Discharge Outcome (improvement)	69 (100%)

Note: Data are presented as n (%).

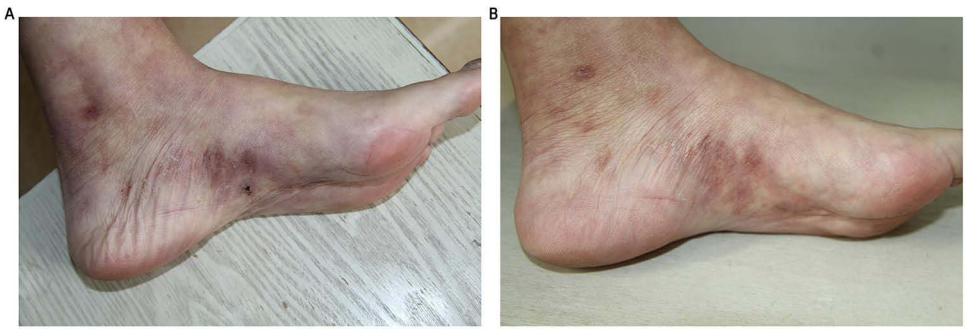

Figure 3 (**A**) Before treatment, irregular, branching livedo racemosa and dusky purpuric patches were observed on the lower legs and feet, which did not blanch on diascopy. Punctuate superficial ulcers with crusts and stellate white atrophic scars were present on the ankles and soles. (**B**) After 2 months of treatment with rivaroxaban 15 mg daily, prednisone 15 mg daily, and aspirin 50 mg daily, the livedo racemosa and dusky purpuric patches showed significant fading and reduction in size, and the ulcers had completely healed with detachment of crusts.

patients (100%, 69/69) demonstrated clinical improvement at discharge (The livedo racemosa and dusky purpuric patches on the lower legs and feet decreased in size and faded in color. The ulcers healed, and the crusts detached). (Figure 3).

Discussion

This single-center retrospective study provides an in-depth analysis of the clinicopathological features, treatment strategies, and outcomes in 69 LV patients, offering valuable data and clinical insights for managing this rare disease, thereby holding significant clinical and academic relevance.

The wide age range and striking female predominance observed in our cohort align with previous reports, confirming LV's predilection for young and middle-aged women.[16] This demographic profile is consistent with larger cohort studies, which reported a similar female-to-male ratio and age distribution 3. This may be attributed to multifactorial influences, including hormonal fluctuations across various physiological stages and sex-specific differences in immune status. Hormones may play a contributory role in the pathogenesis, potentially impacting vascular physiology and pathological processes, rendering women more susceptible.[16] For instance, estrogen has been shown to modulate endothelial function and coagulation pathways, potentially explaining the gender disparity observed in our study and others.[17] However, the precise mechanisms underlying this association warrant further investigation to elucidate the quantitative relationships and specific pathways involved.

The clinical manifestations observed – ulcerations, purpura, white atrophic scars, and livedo racemosa – constitute the classic tetrad of LV. Painful ulcers, in particular, cause substantial morbidity, severely impacting patients' quality of life and functional capacity.[18] Our finding that ulcers were present in 89.9% of patients is consistent with prior studies reporting prevalence rates ranging from 70% to 95% 3,4. These hallmark presentations should alert clinicians evaluating patients with lower extremity skin lesions to consider LV promptly and initiate appropriate diagnostic workup. Notably, only 7.2% of patients received an initial diagnosis of LV in our study, reflecting a high rate of misdiagnosis and delayed recognition in clinical practice. This low initial diagnostic accuracy mirrors the experience of Goerge et al, who highlighted the frequent confusion of LV with venous ulcers or vasculitis.[19] This diagnostic challenge likely stems from the variable clinical presentation and its overlap with other dermatoses, leading to difficulties in accurate identification, especially by non-specialists or less experienced clinicians.[20] Therefore, enhancing physician education on LV's clinical spectrum is crucial. A meticulous approach involving detailed history-taking, thorough physical examination, and judicious use of ancillary investigations is essential to improve initial diagnostic accuracy and prevent treatment delays.

Laboratory investigations revealed a complex interplay of abnormalities. While overt hypercoagulable states were not universally present, perturbations were noted: elevated Factor VIII, hyperfibrinogenemia, and elevated D-dimer suggest localized or intermittent activation of coagulation pathways, a central tenet in LV pathophysiology. These findings corroborate those of Kaya et al, who similarly reported a high frequency of fibrinogen and D-dimer elevation in their LV cohort.[21] The high prevalence of elevated inflammatory markers, particularly CRP and SAA, indicates

significant underlying inflammation, despite the relative absence of classical systemic autoimmunity (low rates of RF, anti-CCP, and ANA positivity). This reinforces the concept of LV as an inflammatory thrombo-occlusive disorder rather than a primary vasculitis. The lipid abnormalities, while present in subsets, lacked a consistent pattern, suggesting they may be epiphenomena rather than primary drivers of pathogenesis, a observation also made by other groups.[22]

Histopathological analysis proved pivotal. The key findings centered on the dermal microvasculature: marked capillary proliferation and congestion, frequent luminal thrombosis, vessel wall fibrinoid degeneration, and extravasation of erythrocytes. These vascular changes are highly characteristic of LV and have been detailed in prior histopathological series.[23,24] Critically, no overt leukocytoclastic vasculitis was identified in any case, definitively distinguishing LV from conditions like cutaneous small vessel vasculitis. The frequent perivascular lymphocytic and neutrophilic infiltrates, alongside DIF findings demonstrating vascular deposits (particularly C3, IgM, and IgA), confirm an inflammatory component with immune complex deposition likely contributing to endothelial damage and subsequent thrombosis. This combination of microvascular thrombosis, characteristic vessel wall changes, inflammation without vasculitis, and immune deposits is the diagnostic hallmark of LV.

Thrombosis leads to vascular stenosis and occlusion, resulting in cutaneous ischemia, necrosis, ulceration, and scarring. Hypercoagulability may arise from hereditary or acquired coagulation disorders, such as prothrombotic gene mutations or secondary conditions like antiphospholipid syndrome.[25] In our cohort, the high prevalence of elevated Factor VIII and fibrinogen underscores the role of acquired hypercoagulability in disease pathogenesis. Immune abnormalities involve mechanisms like autoantibody production and immune complex deposition, activating the complement cascade and triggering vascular inflammation, which exacerbates endothelial damage and thrombogenesis.[26] Our DIF findings, showing vascular complement and immunoglobulin deposition, provide direct support for this mechanism. Identifying these underlying etiologies is paramount for targeted therapy. Routine screening for relevant coagulation parameters and immunological markers is recommended to detect associated conditions and guide management, as advocated by international consensus guidelines.[27] Anticoagulant or antiplatelet therapy addresses hypercoagulability, while immunomodulatory agents, including corticosteroids, immunosuppressants, are indicated for immune-mediated pathology, aiming to control disease activity and improve prognosis.

Our study encompassed various therapeutic modalities, including anticoagulant therapy, antiplatelet therapy, corticosteroids, lipid-lowering therapy, immunosuppressive agents, traditional Chinese medicine and physical therapy. Anticoagulants and antiplatelet agents prevent thrombus propagation and improve microcirculation, while immunomodulatory mitigate aberrant immune responses and vascular inflammation, yielding synergistic benefits.[28,29] This multimodal approach is supported by a growing body of evidence suggesting that targeting both thrombotic and inflammatory pathways improves outcomes in refractory cases.[30] Close monitoring for potential adverse effects, such as bleeding risk with anticoagulants or infection with immunosuppressants, is essential to ensure treatment safety and efficacy. Physical therapy modalities may provide adjunctive benefits by promoting ulcer healing and enhancing local circulation, though more robust data is needed to confirm its efficacy.

Despite the efficacy of combination therapy, long-term follow-up is imperative to monitor for potential progression to systemic diseases or complications. Given the chronic, relapsing nature of LV, patients may experience disease flares or develop new complications, including deep vein thrombosis, pulmonary embolism, or involvement of other organ systems.[31] Regular follow-up facilitates early detection of such risks, enabling timely intervention to improve long-term outcomes and quality of life. Establishing structured follow-up protocols, with scheduled reassessments and appropriate investigations, is recommended for ongoing disease monitoring.

As a single-center retrospective analysis, this study provides valuable insights but has inherent limitations. The relatively small sample size may limit the generalizability of findings and statistical power. Retrospective designs are susceptible to information bias and selection bias; incomplete follow-up data in some cases may have impacted the accuracy of prognostic analyses. Future multicenter, large-scale, prospective studies are essential to validate our findings, further elucidate LV pathogenesis, optimize treatment strategies, and generate higher-level evidence to advance precision medicine in LV management.

Conclusion

This study comprehensively analyzed the clinical characteristics, laboratory findings, and treatment outcomes of 69 patients with LV diagnosed between January 2014 and December 2024. The majority of patients were female, and the mean duration from symptom onset to definitive diagnosis was approximately 5.41 years, indicating a potential delay in diagnosis. Skin lesions predominantly localized to bilateral lower extremities and often presented as ulcerations, livedo racemosa, purpura, and white atrophic scars. Laboratory abnormalities included various coagulation and inflammatory markers and dyslipidemia. Treatment with combination therapy regimens led to clinical improvement in all patients at discharge. However, this study was limited by its relatively small sample size and retrospective design. Future studies with larger sample sizes and longer follow - up periods are needed to further confirm these findings and explore the long - term outcomes of LV patients.

Data Sharing Statement

The data supporting the findings of this study are available from the corresponding author (Hui Zhou) upon reasonable request.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no conflict of interest.

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