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Livedoid Vasculopathy: A Comprehensive Review of Pathogenesis, Histopathology, Therapeutic Algorithms, Emerging Therapies, and Rehabilitation Strategies

Abstract

Livedoid vasculopathy (LV) is a rare, chronic, and debilitating thrombo-occlusive disorder of the dermal microvasculature. Characterised by painful ulcers and atrophic blanche, LV presents diagnostic and therapeutic challenges due to its overlap with inflammatory vasculitides and its relapsing nature. This review synthesises current literature on LV's pathogenesis, histopathological features, treatment algorithms, emerging therapies, and rehabilitation interventions. Histopathology confirms a non-inflammatory vascular occlusion, guiding a tiered therapeutic approach that includes anticoagulants, immunomodulators, and biologics. Emerging therapies such as JAK inhibitors and hyperbaric oxygen therapy offer promise in refractory cases. Rehabilitation, though underexplored, is essential for pain management, mobility restoration, and wound care. Future research should focus on biomarker-driven stratification, standardised rehabilitation protocols, and long-term outcome studies.

Keywords: Livedoid vasculopathy; Thrombo-occlusive microangiopathy; Histopathology; Anticoagulant Therapy; Treatment Algorithms; Emerging Therapies; Rehabilitation Strategies.

Literature Review

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Introduction

Livedoid Vasculopathy (LV), previously termed livedoid vasculitis, is increasingly recognised as a thrombo-occlusive microangiopathy rather than a primary inflammatory vasculitis [1]. It predominantly affects middle-aged women and presents with painful ulcers, purpura, and white stellate scars known as atrophic blanche [2]. The pathogenesis involves hypercoagulability, endothelial dysfunction, and fibrin deposition, often triggered by autoimmune or prothrombotic states. Despite its rarity, LV poses significant diagnostic and therapeutic challenges due to its chronicity, pain burden, and risk of misclassification. This review aims to consolidate recent advances in understanding LV's pathogenesis, histopathology, treatment algorithms, emerging therapies, and rehabilitation strategies.

Methods

A structured literature search was conducted using PubMed, Scopus, and Google Scholar databases from January 2018 to October 2025. Keywords included "livedoid vasculopathy," "treatment algorithm," "histopathology," "JAK inhibitors," "biologics," "rehabilitation," and "emerging therapies." Inclusion criteria were peer-reviewed articles, systematic reviews, and case reports focusing on LV's clinical management and pathophysiology. Reference lists of key articles were manually screened for additional sources. Articles were evaluated for relevance, methodological rigour, and clinical applicability.

Results

Pathogenesis and Histopathology

LV is now understood as a thrombo-occlusive disorder of the dermal microvasculature, with minimal inflammation. Histopathological examination reveals fibrin thrombi within small dermal vessels, endothelial swelling, and extravasated red blood cells [1]. Unlike vasculitis, there is no leukocytoclastic infiltration. Direct immunofluorescence may reveal fibrin, IgM, and complement components, supporting a thrombotic pathogenesis. These findings underscore the importance of distinguishing LV from inflammatory vasculitis, as treatment strategies differ significantly.

Treatment Algorithms

Therapeutic approaches to LV are tiered and individualised based on disease severity, comorbidities, and response to prior treatments. A 2018 systematic review by Kellner et al. [2] proposed the following algorithm:

- First-line therapy involves anticoagulants such as warfarin, rivaroxaban, or low-molecular-weight heparin. These agents target the underlying thrombotic mechanism and have shown consistent ulcer healing and pain reduction.
- Second-line agents include antiplatelet drugs (aspirin, clopidogrel) and vasodilators like pentoxifylline, which improve microcirculatory flow.
- Third-line options encompass immunomodulatory therapies such as corticosteroids, intravenous immunoglobulin (IVIG), and hydroxychloroquine, particularly in patients with autoimmune overlap.
- Refractory cases may benefit from biologics (e.g., rituximab, TNF-α inhibitors) or targeted therapies like JAK inhibitors [3].

The “CHAP” regimen—colchicine, hydroxychloroquine, aspirin, and pentoxifylline—has been proposed for mild-to-moderate disease, offering a low-cost, synergistic approach [4].

Emerging Therapies

Recent advances have introduced novel agents for refractory LV:

- Upadacitinib, a selective JAK1 inhibitor, demonstrated rapid ulcer healing and pain relief in a 2025 case report [3]. Its mechanism involves modulation of cytokine signalling and vascular inflammation.
- Biologics, including rituximab and TNF-α inhibitors, have shown efficacy in autoimmune-associated LV, particularly in patients with systemic lupus erythematosus or antiphospholipid syndrome [5].
- Hyperbaric oxygen therapy and plasmapheresis are under investigation for severe, treatment-resistant cases, aiming to enhance tissue oxygenation and remove circulating immune complexes.

These therapies represent a paradigm shift toward precision medicine in LV, though randomized controlled trials are needed to validate their efficacy.

Rehabilitation Interventions

Rehabilitation is a critical yet underreported aspect of LV management. Chronic pain, ulceration, and scarring significantly impair mobility and quality of life. A 2023 Springer chapter emphasised the need for multidisciplinary care integrating dermatology, vascular medicine, and rehabilitation [6].

Key rehabilitation strategies include:

- **Pain management:** Topical lidocaine, systemic analgesics, and cognitive-behavioural therapy address both nociceptive and neuropathic pain.
- **Mobility support:** Compression therapy improves venous return, while physiotherapy aids in gait training and offloading pressure from ulcerated areas.
- **Wound care:** Regular debridement, use of advanced dressings (e.g., hydrocolloid, silver-impregnated), and infection control are essential.
- **Psychosocial support:** Chronic dermatologic pain is associated with depression and anxiety; psychological counselling and peer support groups can improve coping and adherence.

Despite its importance, rehabilitation lacks standardised protocols in LV, highlighting a gap in comprehensive care.

Conclusion

Livedoid vasculopathy is a complex, chronic thrombo-occlusive disorder requiring a multidisciplinary approach. Histopathology confirms its non-inflammatory nature, guiding anticoagulant-based therapy. Treatment algorithms must be tailored to individual patient profiles, with emerging therapies offering hope for refractory cases. Rehabilitation plays a vital role in improving functional outcomes and quality of life. Integration of dermatologic, vascular, and rehabilitative care is essential for holistic management.

Future Directions

- Development of biomarker-driven treatment stratification to personalise therapy.
- Randomised controlled trials evaluating JAK inhibitors, biologics, and combination regimens.
- Establishment of standardised rehabilitation protocols for chronic dermatologic pain and mobility impairment.
- Longitudinal studies assessing psychosocial outcomes, recurrence rates, and cost-effectiveness of therapies.

Limitations

- Most evidence is derived from case reports and small series, limiting generalizability.
- Lack of randomised controlled trials for emerging therapies.
- Sparse data on rehabilitation outcomes, with no standardised protocols.
- Potential publication bias favouring successful treatment outcomes.

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