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SYSTEMIC LUPUS ERYTHEMATOUS VASCULITIS: A SYSTEMIC REVIEW OF NOVEL BIOMARKERS, AND THERAPEUTIC BREAKTHROUGHS (2020 – 2026)

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Systemic Erythematous lupus (SLE) is a chronic autoimmune condition in which vasculitis – inflammation of blood vessels, is a major cause of morbidity and mortality. It affects vessels of all diameters in the kidney, skin, liver, heart, brain, lung, and gastrointestinal tract, among other organ systems. The published literature from January 2020 to March 2026, which includes the time of transition after the EULAR/ACR 2019 categorisation criteria and the Relevance. of new targeted medicines, is compiled in this systematic review.

Organ Specific pathological findings demonstrate unique morphological signatures. Fibrinoid necrosis, wire-loop lesions, and the typical full-house immunofluorescence pattern (IgG, IgM, IgA, C3, C1q) are specific signs of lupus nephritis in the kidney. Leukocytoclastic vasculitis along with neutrophil infiltration, fibrinoid necrosis, and the lupus band test showing granular immunoglobulin deposits specific to the dermoepidermal junction is how cutaneous vasculitis manifests. Portal lymphoplasmacytic infiltrates with interface hepatitis are revealed by hepatic vasculitis. In heart Coronary arteritis and Libman-Sacks endocarditis, which are non-bacterial verrucous vegetations made of fibrin and immune complexes, are examples of cardiac involvement. Perivascular lymphocytic cuffing and small vessel vasculitis are linked to neuropsychiatric lupus. Mesenteric vasculitis causes intestinal bowel ischaemia and necrosis, whereas pulmonary vasculitis manifests as capillaritis with alveolar haemorrhage.

Novel mechanistic insights have significantly transformed the world's understanding. Immunohistochemical detection of citrullinated histone H3 (citH3) is an important diagnostic sign of active vasculitis, and NETosis - the release of neutrophil extracellular traps, (NETs) - is acknowledged as a key pathogenic mechanism. Both innate and adaptive immunological dysregulation are caused by the type I interferon signature, which also forecasts how well interferon-targeted treatments will work. The increased cardiovascular risk in SLE patients can be explained by endotheliopathy - characterised by degradation of the endothelium glycocalyx with higher syndecan-1. This process accelerates atherosclerosis and causes arterial leakage.

Therapeutic breakthroughs have changed the way they are treated. Based on phase III trials showing notable decreases in disease activity and vasculitis flare-ups, the FDA approved anifrolumab, a monoclonal antibody that targets the type I interferon receptor, in 2021. Due to its improved renal response rates, the calcineurin inhibitor voclosporin was licensed for the treatment of lupus nephritis. The most remarkable development is CAR-T cell therapy, which targets CD19+ B cells. Case studies show that individuals with refractory SLE, including those with severe vasculitis, can achieve complete clinical and serological remission.

This systematic review, which integrates organ-specific morphological discoveries with new mechanistic insights and treatment advancements, offers the first thorough synthesis of SLE vasculitis pathology in the post-EULAR/ACR 2019 age. CitH3 immunohistochemistry for NETosis, full-house immunofluorescence for immune complex deposition, and syndecan-1 as a crucial biomarker of endotheliopathy are important diagnostic tools for pathologists and rheumatologists.