ISSN 2472-5056

Vol.9 No.5:279

# Lupus Nephritis Explained: Renal Risks and Management in Systemic Lupus Erythematosus

### John Lucas\*

Department of Internal Medicine, University of Liverpool, Liverpool, UK

Corresponding author: John Lucas, Department of Internal Medicine, University of Liverpool, Liverpool, UK, E-mail: lucasjohn@liv.uk

**Received date:** September 25, 2024, Manuscript No. IPJCEN-24-19844; **Editor assigned date:** September 30, 2024, PreQC No. IPJCEN-24-19844 (PQ); **Reviewed date:** October 14, 2024, QC No. IPJCEN-24-19844; **Revised date:** October 21, 2024, Manuscript No. IPJCEN-24-19844 (R); **Published date:** October 28, 2024, DOI: 10.36648/2472-5056.9.5.279

Citation: Lucas J (2024) Lupus Nephritis Explained: Renal Risks and Management in Systemic Lupus Erythematosus. J Clin Exp Nephrol Vol.9 No.5: 279.

## Description

Lupus Nephritis (LN) is a severe and potentially lifethreatening manifestation of Systemic Lupus Erythematosus (SLE), an autoimmune disease that can affect multiple organs, including the kidneys. Characterized by inflammation and damage to the kidney's glomeruli, lupus nephritis can significantly impair kidney function and lead to Chronic Kidney Disease (CKD) or End-Stage Renal Disease (ESRD) if not managed properly. This article aims to provide an overview of lupus nephritis, its pathogenesis, clinical presentation, diagnostic approach and current treatment strategies. The exact mechanisms underlying lupus nephritis are complex and involve a variety of immune system components. In lupus nephritis, the immune system mistakenly attacks the body's own tissues, causing inflammation in the kidneys. A hallmark of SLE and lupus nephritis is the production of autoantibodies, particularly antidouble-stranded DNA (anti-dsDNA) antibodies. These antibodies form immune complexes that deposit in the glomeruli of the kidneys, triggering an inflammatory response that damages the delicate filtration system of the kidneys. This inflammation can lead to proteinuria (excess protein in the urine), hematuria (blood in the urine) and eventually decreased kidney function. The development of lupus nephritis is influenced by both genetic and environmental factors. Certain genetic markers, such as variations in the Human Leukocyte Antigen (HLA) system, are associated with a higher risk of developing lupus nephritis. Additionally, environmental factors like infections, stress and hormonal changes can act as triggers for the onset of the disease.

#### **Clinical presentation**

Lupus nephritis can present in a wide range of severity, from asymptomatic proteinuria to rapidly progressive kidney failure. Patients may not show any overt signs of kidney disease early on, which makes regular monitoring essential for individuals diagnosed with SLE. Common clinical features of lupus nephritis include: One of the most common signs of lupus nephritis, proteinuria is indicative of glomerular damage. A 24 h urine collection may reveal significant amounts of protein. Microscopic or gross hematuria can occur, signifying inflammation or injury within the kidneys. Swelling, especially in the lower extremities, can result from the kidneys' inability to

regulate fluid balance, often due to protein loss. Elevated blood pressure is frequently seen in patients with lupus nephritis and can further exacerbate kidney damage. As the disease progresses, the kidneys' filtering capacity declines, leading to reduced kidney function and in severe cases, renal failure. Lupus nephritis is classified into six histologic classes based on kidney biopsy findings, according to the International Society of Nephrology/Renal Pathology Society (ISN/RPS) classification system. A kidney biopsy is essential for diagnosing lupus nephritis and determining the specific class, which in turn guides treatment decisions. Blood tests, such as anti-dsDNA antibodies and complement levels and urine tests for proteinuria and hematuria are also important diagnostic tools.

#### Lupus nephritis

The treatment of lupus nephritis is aimed at reducing inflammation, preventing kidney damage and preserving longterm kidney function. Therapy is typically divided into two phases: Induction therapy and maintenance therapy. The initial phase of treatment is aggressive and aimed at achieving remission. Corticosteroids, such as prednisone, are commonly used to suppress inflammation. In combination with steroids, immunosuppressive agents like cyclophosphamide or Mycophenolate Mofetil (MMF) are often administered. Cyclophosphamide has been the standard therapy for severe cases (Class III, IV and V), though MMF has become more commonly used due to its better safety profile and similar efficacy. Once remission is achieved, the goal is to maintain it with less aggressive therapy. Maintenance therapy often includes lower doses of corticosteroids and immunosuppressive agents, such as azathioprine or MMF. Regular monitoring of kidney function, proteinuria and blood pressure is critical during this phase to detect any signs of relapse early. Recent advances have introduced biologic therapies targeting specific immune pathways involved in lupus nephritis. Belimumab, a monoclonal antibody that inhibits B-cell activation, has been approved for treating lupus nephritis, offering a more targeted approach than traditional immunosuppressive drugs. The prognosis of lupus nephritis varies depending on the severity of the disease and the response to treatment. With early diagnosis and appropriate therapy, many patients can achieve remission and preserve kidney function. However, relapses are common and approximately 10%-30% of patients with severe lupus nephritis

Vol.9 No.5:279

may progress to ESRD, requiring dialysis or transplantation. Factors associated with poor outcomes include shown to have a higher risk of developing severe lupus nephritis delayed diagnosis, poor response to treatment and the presence of other comorbidities, such as hypertension and cardiovascular

kidney disease. African American and Hispanic patients have been and worse outcomes, likely due to genetic and socio-economic factors.