"ANALYSIS OF CORRELATION OF URINARY BIOMARKERS WITH SEROLOGY AND HISTOPATHOLOGICAL PROFILE IN PATIENTS WITH LUPUS NEPHRITIS – A CROSS-SECTIONAL STUDY"

BY

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LIST OF ABBREVIATIONS

- **ACR** – American College of Rheumatology
- **ALN** – Active Lupus Nephritis
- **ANA** – Antinuclear Antibody
- **APRIL** – A Proliferation-Inducing Ligand
- **BAFF/BLyS** – B-cell Activating Factor / B Lymphocyte Stimulator
- **CD163** – Cluster of Differentiation 163
- **CKD** – Chronic Kidney Disease
- **CI** – Chronicity Index
- **CNIs** – Calcineurin Inhibitors
- **CTGF** – Connective Tissue Growth Factor
- **dsDNA** – Double Stranded Deoxyribonucleic Acid
- **EULAR** – European League Against Rheumatism
- **EM** – Electron Microscopy
- **ESKD/ESRD** – End-Stage Kidney Disease / End-Stage Renal Disease
- **GBM** – Glomerular Basement Membrane
- **IF** – Immunofluorescence
- **IFN-α** – Interferon-alpha
- **IL-17** – Interleukin-17
- **IQR** – Interquartile Range
- **iSLE** – Inactive Systemic Lupus Erythematosus
- **ISN/RPS** – International Society of Nephrology / Renal Pathology Society
- **IUGR** – Intrauterine Growth Restriction
- **KIM-1** – Kidney Injury Molecule-1
- **LN** – Lupus Nephritis

- **LUS** – Lupus
- **L-FABP** – Liver-type Fatty Acid Binding Protein
- **miRNA** – Micro Ribonucleic Acid
- **MMF** – Mycophenolate Mofetil
- **MCP-1** – Monocyte Chemoattractant Protein-1
- **NCAM-1** – Neural Cell Adhesion Molecule-1
- **NGAL/LCN2** – Neutrophil Gelatinase-Associated Lipocalin / Lipocalin-2
- **NIH** – National Institutes of Health
- **OPG** – Osteoprotegerin
- **rSLEDAI-2K** – Renal Systemic Lupus Erythematosus Disease Activity Index-2000
- **SLE** – Systemic Lupus Erythematosus
- **SLICC** – Systemic Lupus International Collaborating Clinics
- **suPAR** – Soluble Urokinase Plasminogen Activator Receptor
- **TGF-β** – Transforming Growth Factor-beta
- **TLR** – Toll-Like Receptor
- **TWEAK** – TNF-like Weak Inducer of Apoptosis
- **UPCR** – Urinary Protein-to-Creatinine Ratio
- **VCAM-1** – Vascular Cell Adhesion Molecule-1
- **WHO** – World Health Organization

ABSTRACT

Background: Lupus nephritis (LN) is a serious manifestation of systemic lupus erythematosus (SLE) with diverse histological classes and clinical outcomes. Traditional evaluation relies on renal biopsy; however, non-invasive urinary biomarkers like Monocyte Chemoattractant Protein-1 (MCP-1) and Transforming Growth Factor-β (TGF-β) may provide valuable insights into renal inflammation and chronicity.

Objectives: [redacted]

Methods: [redacted]

Results: [redacted]

[redacted]

Conclusion [redacted]

Keywords: Lupus Nephritis, MCP-1, TGF-β, Urinary Biomarkers, Activity Index, Chronicity Index, rSLEDAI-2K, Renal Biopsy, Proteinuria, Systemic Lupus Erythematosus (SLE), Non-Invasive Markers, Histopathological Class.

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INTRODUCTION

INTRODUCTION

Systemic Lupus Erythematosus (SLE) is a chronic, multisystem autoimmune disease characterized by the production of autoantibodies against a broad range of self-antigens, particularly nuclear and cytoplasmic components. [redacted]

Disease activity often waxes and wanes, with periods of remission and exacerbation. The unpredictable course of the disease, coupled with its potential for severe organ damage, underscores the importance of timely diagnosis and appropriate management.

[redacted]

[redacted]

[redacted]. In Indian cohorts, the prevalence of LN has been reported to be as high as 50–60%, often presenting in advanced stages due to delayed recognition or inadequate access to specialized care. LN is a major contributor to morbidity and mortality in SLE, accounting for significant proportions of hospitalizations, end-stage renal disease (ESRD), and death (11)[redacted]

[redacted]

[redacted]

[redacted]

[redacted]

[redacted]

[redacted]

[redacted]

[redacted]Advances in genomics, proteomics, and systems biology may pave the way for more targeted and individualized treatment approaches in the future (18).

OBJECTIVE OF THE STUDY

OBJECTIVE OF THE STUDY:

Primary objectives:

1. To evaluate the correlation between the serological profile and histopathology among Lupus Nephritis patients

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Global incidence and prevalence of SLE and Lupus Nephritis

Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease with considerable global variability in its incidence and prevalence, influenced by genetic, environmental, and socioeconomic factors. [redacted]

[redacted]

[redacted]While improvements in diagnosis and treatment have led to better outcomes in high-income countries, patients in low- and middle- income countries continue to face challenges due to limited healthcare infrastructure, delayed diagnosis, and inconsistent access to immunosuppressive therapies (22).

Table 1: Class-wise prevalence of Lupus Nephritis (LN) based on the ISN/RPS 2003 classification: (14)

Class	Name	Histological Features	Prevalence (% of LN cases)	Clinical Characteristics
I	[redacted]	[redacted]	[redacted]	[redacted]
II	[redacted]	[redacted]	[redacted]	[redacted]
III	[redacted]	[redacted]	[redacted]	[redacted]
IV	[redacted]	[redacted]	[redacted]	[redacted]
V	[redacted]	[redacted]	[redacted]	[redacted]
VI	[redacted]	[redacted]	[redacted]	[redacted]

[redacted]Indian studies often report Class IV in over 50% of biopsied LN cases.

Incidence and Prevalence of SLE and Lupus Nephritis in India

In India, reliable national epidemiological data on systemic lupus erythematosus (SLE) are limited due to the absence of large-scale population-based studies; however, estimates derived from hospital-based cohorts and regional registries provide useful insights (5). [redacted]Pediatric and adolescent patients in India also demonstrate a high incidence of LN at initial diagnosis, with aggressive proliferative classes being most frequent.

Table 2: SLE and LN in India

Parameter	Estimated Value in India	Notes
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]

Table 3: Class-wise prevalence of Lupus Nephritis (LN) based on studies from India, reflecting biopsy-proven cases across multiple tertiary care centers:

Class	Name	Histological Features	Prevalence in Indian Cohorts (% of LN cases)	Clinical Characteristics
I				
II				
III				
IV				
V				
VI				

Key Observations from Indian Studies

- Class IV (Diffuse Proliferative LN) is consistently the most prevalent, accounting for nearly half or more of all LN cases.

[redacted]

- Children and adolescents with LN in India also show high rates of Class IV disease, often with aggressive presentation.

PREVIOUS RESEARCH STUDIES

[redacted] **(2021).** reviewed immune-related urinary biomarkers that could improve the noninvasive diagnosis of lupus nephritis (LN). [redacted] The paper concluded that immune-related urinary biomarkers have strong translational potential but require standardization and integration into prospective trials before widespread use (25).

(2012) (26)– In a study titled monocyte chemoattractant protein-1 as a urinary biomarker for diagnosis of activity of lupus nephritis done among Brazilian population (n=75) showed positive correlation between uMCP-1 and serum creatinine in active LN (Lupus Nephritis)

2012) (27)– In a similar study titled urinary monocyte chemotactic protein-1 and transforming growth factor-b in systemic lupus erythematosus done among 50 iranian lupus nephritis cases (2012) demonstrated positive correlation with the class of LN

No correlation with the chronicity indexes.

. (2006) (28)**-** In a study titled transforming growth factor beta 1 in children with systemic lupus erythematosus: a possible correlation (2006) done among 32 Egyptian children with active lupus observed that Urinary TGF-β levels are significantly higher in those with active LN ($P < 0.001$).

. (2006) (29)**-** The study measured urinary chemokine and growth factor mRNAs, including TGF-β2, in 26 lupus patients from Thailand and Malaysia.

[redacted]

[redacted], **(2025)** explored the potential roles of soluble urokinase plasminogen activator receptor (suPAR) and vascular endothelial growth factor (VEGF) as biomarkers in lupus nephritis (LN). [redacted]

[redacted]The authors concluded that while suPAR and VEGF are promising, larger multicenter studies are necessary to establish their predictive and prognostic value, potentially integrating them into biomarker panels for noninvasive LN monitoring (30).

[redacted], **(2020).** evaluated urinary biomarkers as noninvasive tools in the diagnosis and monitoring of lupus nephritis. [redacted]

[redacted]

[redacted]Overall, the article reinforced the clinical utility of urinary biomarkers as noninvasive, dynamic tools that can complement histopathology in monitoring lupus nephritis (31).

[redacted] **(2016).** addressed the central role of renal biopsy in lupus nephritis, discussing its timing, frequency, and interpretation. [redacted]

[redacted]The article concluded that biopsy frequency should be individualized, balancing diagnostic benefit with procedural risks, and suggested that biomarkers might reduce reliance on repeat biopsies in the future (32).

[redacted]**. (2004).** This seminal consensus paper by the International Society of Nephrology/Renal Pathology Society (ISN/RPS) redefined the histopathological classification of lupus nephritis. [redacted]

[redacted] Nevertheless, this framework remains essential for research and clinical practice, serving as the backbone for clinical trials, epidemiological studies, and treatment guidelines in lupus nephritis (33).

[redacted] **(2024).** explored the promising role of urinary biomarkers as non-invasive surrogates for renal histopathology in lupus nephritis (LN). [redacted] In closing, they project that these biomarkers could revolutionize LN diagnostics and management—pending further validation in longitudinal and multicenter studies (34).

[redacted]**, (2024).** explored the expanding role of urinary biomarkers in evaluating and managing lupus nephritis (LN), especially by harnessing systems biology and multiomics technologies**.** [redacted]

[redacted] However, it emphasizes that these technologies require extensive validation**,** standardized assays, and translational studies to become clinically actionable (35).

[redacted]**, (2024).** reviewed scrutinizes which urinary protein markers have been repeatedly validated across independent cohorts for differentiating active lupus nephritis (ALN) from inactive SLE (iSLE). An extensive screening of 3,411 patients [redacted] However, they note that clinical integration requires further homegrown assay standardization, prospective validation, and demonstration of utility in guiding treatment decisions (36).

[redacted] **(2024).** reviewed scrutinizes which urinary protein markers have been repeatedly validated across independent cohorts for differentiating active lupus nephritis (ALN) from inactive SLE (iSLE). [redacted]

[redacted]

However, they note that clinical integration requires further homegrown assay standardization, prospective validation, and demonstration of utility in guiding treatment decisions (36).

[redacted] **(2017)** (37) conducted a comprehensive meta-analysis to evaluate the role of urinary monocyte chemoattractant protein-1 (MCP-1) as a biomarker for lupus nephritis (LN). MCP-1, a chemokine involved in monocyte recruitment and renal inflammation, has been consistently implicated in the pathogenesis of LN. [redacted] However, they emphasized the need for larger longitudinal studies to validate its predictive power and establish standardized cut-off values for clinical application.

[redacted] (2023) (38)emphasized that histopathological assessment remains the gold standard in the evaluation of lupus nephritis (LN), providing critical information on both activity and chronicity indices that guide treatment decisions and

prognosis. [redacted] (2023) concluded that systematic histologic evaluation of activity and chronicity provides indispensable information for personalized management in lupus nephritis, serving as both a diagnostic and prognostic biomarker of disease severity and long-term renal outcomes.

PATHOPHYSIOLOGY OF LUPUS NEPHRITIS

Lupus nephritis (LN) arises from a complex interplay of autoimmune mechanisms leading to inflammation, immune complex deposition, complement activation, and structural kidney damage. [redacted]

Loss of Immune Tolerance and Autoantibody Formation

[redacted] These autoantibodies either form circulating

immune complexes or bind to endogenous nucleosomes trapped in the glomerular basement membrane (GBM), leading to in situ immune complex formation (40).

Immune Complex Deposition and Complement Activation

[redacted]

Complement activation produces C3a and C5a (anaphylatoxins) that recruit neutrophils and monocytes to the kidney, propagating inflammation (41).

Role of Innate and Adaptive Immunity

Both innate and adaptive immune responses drive kidney damage in LN:

[redacted]

- **Macrophages** and infiltrating monocytes produce cytokines (TNF-α, IL-6, IL-1β) and chemokines (MCP-1), promoting inflammation and matrix

remodeling.

Endothelial and Podocyte Injury

Immune complex deposition and cytokine release damage glomerular endothelial cells, causing capillary loop occlusion, endocapillary proliferation, and necrosis. Podocytes, which maintain the filtration barrier

Tubulointerstitial Involvement

Tubular and interstitial damage is a strong predictor of renal prognosis.

Fibrosis and Chronic Progression

These chronic changes are typically irreversible and strongly correlate with progression to end-stage renal disease (ESRD) (44).

Vascular Involvement

Figure 1: A pathophysiology-based approach to the diagnosis and treatment .

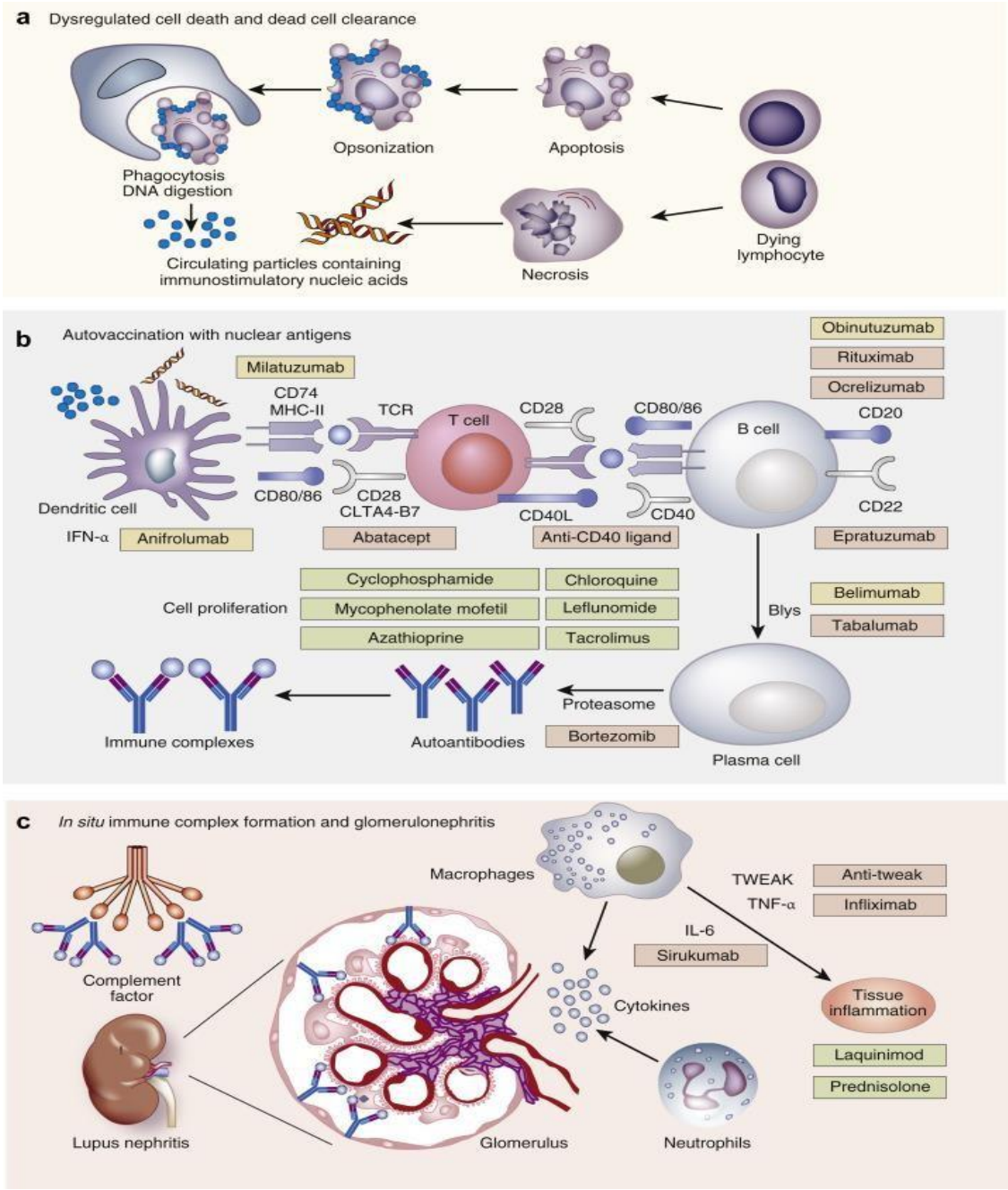
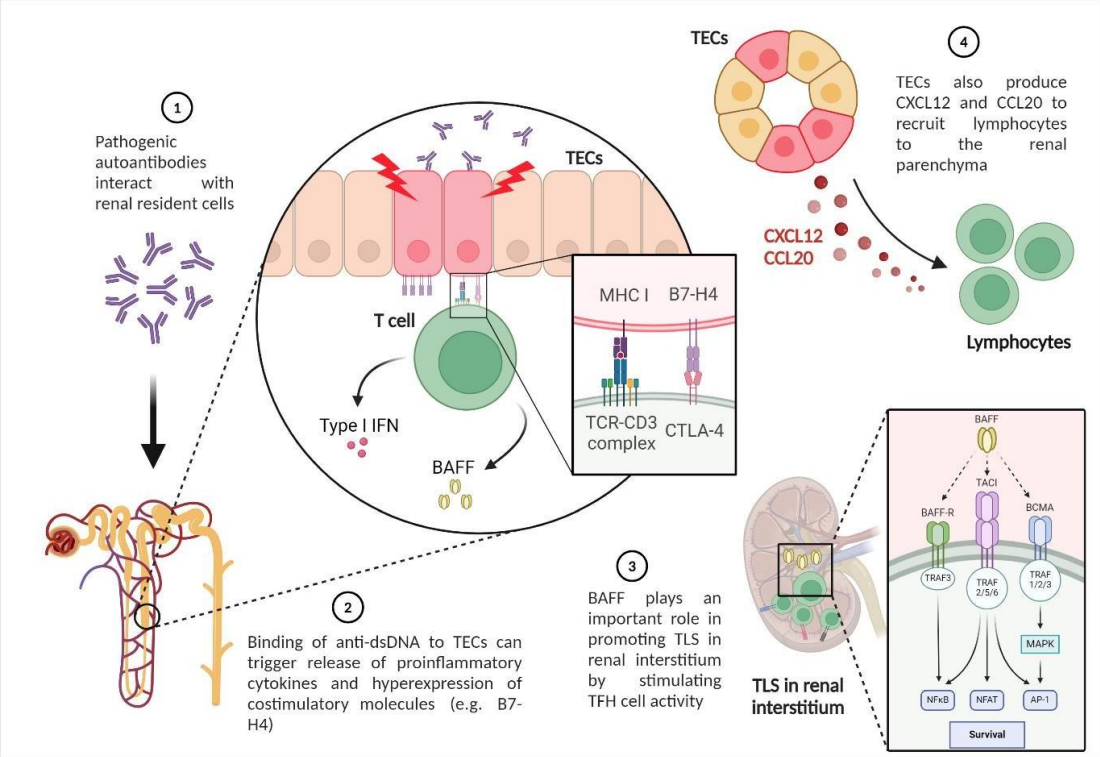


Figure 2: Lupus Nephritis Pathogenesis to New Therapies



Lupus Nephritis Pathogenesis to New Therapies

Urinary and Circulating Biomarkers in Lupus Nephritis

Several urinary and serum biomarkers have emerged as critical tools for assessing disease activity, renal injury, and prognosis in lupus nephritis. [redacted]

[redacted]

[redacted]

[redacted]

[redacted]Finally, anti-dsDNA antibodies, considered hallmark autoantibodies in SLE, directly contribute to immune complex formation and glomerular injury in lupus nephritis (47).

Biomarkers in Lupus Nephritis

Biomarkers play a dual role in both elucidating the pathogenesis of LN and guiding clinical decision-making. [redacted]

[redacted]

[redacted]

[redacted]

In addition, the advent of multi-omics technologies is identifying novel biomarkers for early detection, risk stratification, and treatment response prediction (48).

Table 4: Summary of Biomarkers in Lupus Nephritis

Biomarker	Role in Pathophysiology	Source	Clinical Relevance
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]

CLASSIFICATIONS OF LUPUS NEPHRITIS

Lupus nephritis (LN) is classified based on histopathological features observed on renal biopsy, which is essential for determining prognosis and guiding treatment. [redacted]

[redacted] The classification provides critical information for prognosis and therapeutic decision-making (9,49,50).

Table 5: WHO Classification (1974, revised 1982)

Class	Description
I	[redacted]
II	[redacted]
III	[redacted]
IV	[redacted]
V	[redacted]
VI	[redacted]

[redacted] Despite its usefulness, the WHO system lacked clear definitions for activity and chronicity, leading to poor interobserver reproducibility and challenges in guiding treatment decisions.

Table 6: ISN/RPS 2003 Classification (Current Gold Standard)

Class	Description
I	[redacted]
II	[redacted]
III	[redacted]
IV	[redacted]
V	[redacted]
VI	[redacted]

[redacted]

It also incorporates the use of activity and chronicity indices, enhancing prognostic value and therapeutic planning (14).

2018 ISN/RPS Revision (Proposed)

The 2018 revision, still under review, proposes significant improvements over the 2003 classification. [redacted]

These changes aim to further enhance diagnostic consistency and therapeutic decision-making (51).

Table 7: Comparative Chart: LN Classifications

Feature	WHO (1974/1982)	Modified WHO (1995)	ISN/RPS (2003)	ISN/RPS (2018 Update)
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]

NATURAL HISTORY OF LUPUS NEPHRITIS

The natural progression of LN varies depending on the histological class, response to treatment, and disease control. [redacted] The use of maintenance immunosuppression and novel biologics has shown promise in improving long-term renal survival and reducing flare rates.

CLINICAL FEATURES OF LUPUS NEPHRITIS

Lupus nephritis (LN), a common and potentially severe manifestation of systemic lupus erythematosus (SLE), presents with a broad spectrum of clinical features ranging from asymptomatic urinary abnormalities to full-blown nephritic or nephrotic syndrome and progressive renal failure. [redacted]

Importantly, there may be discordance between clinical presentation and biopsy

findings, making renal biopsy an essential diagnostic tool in all cases of suspected LN, even in those with mild urinary findings.

Table 8: Tabular Summary: Clinical Features of Lupus Nephritis

Feature	Description
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]
[redacted]	[redacted]

Clinical Aspects of Lupus Nephritis

The clinical spectrum of LN is broad and variable. [redacted]

[redacted]

[redacted]Pediatric and adolescent LN tends to be more aggressive, with higher prevalence of Class IV disease and increased risk for rapid progression to end-stage kidney disease (ESKD).

Figure 3: Clinical features of Lupus Nephritis & SLE (male)

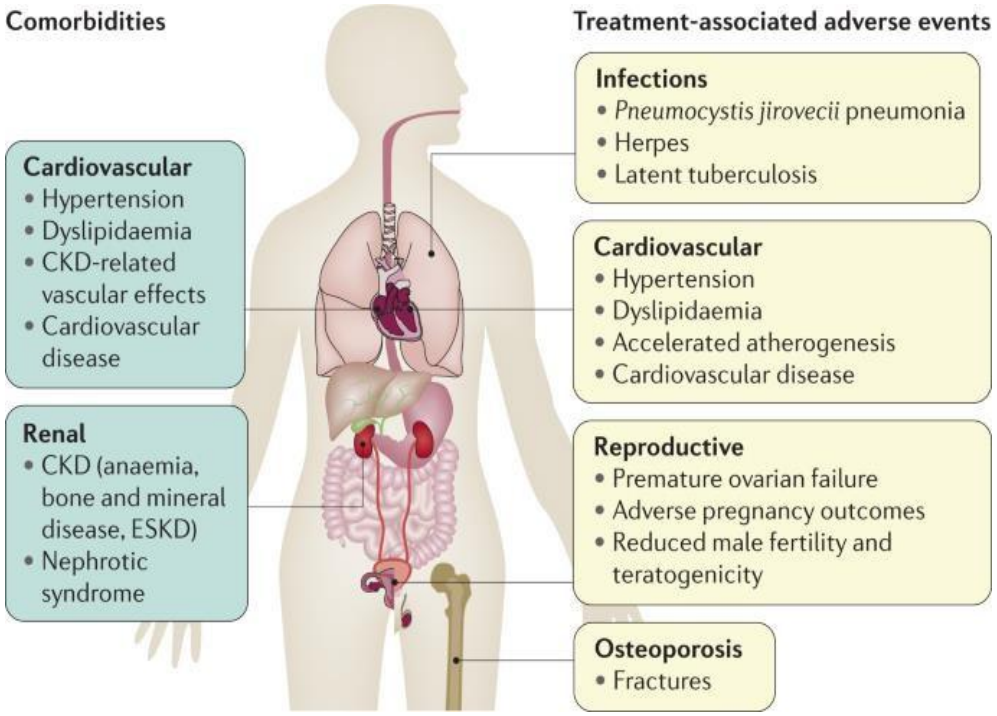
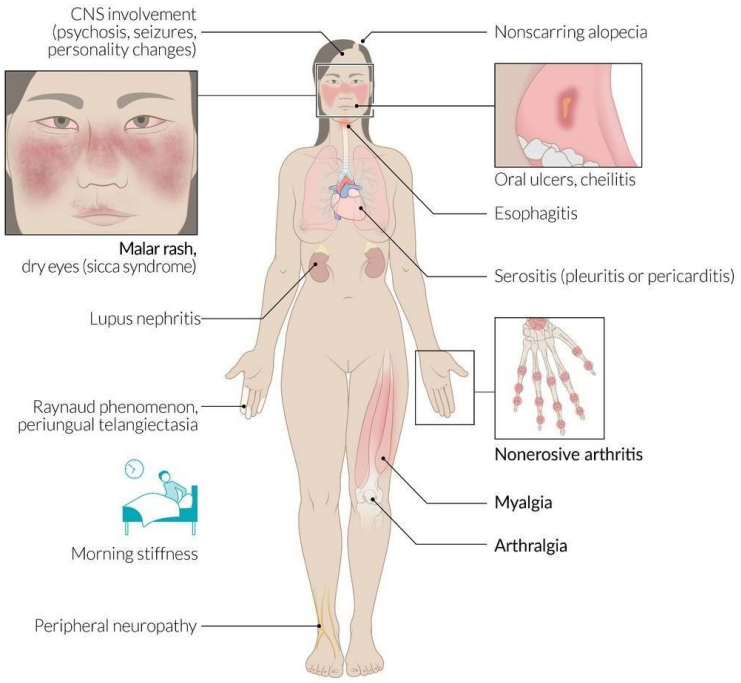


Figure 4: Clinical features of Lupus Nephritis & SLE (Female)



LUPUS NEPHRITIS IN PREGNANCY

Pregnancy in women with lupus nephritis (LN) presents a complex interplay between maternal autoimmune disease and the physiological changes of gestation, requiring vigilant planning and monitoring. [redacted]

[redacted]

Despite the high-risk nature, studies have shown that with appropriate preconception counselling and disease control, many women with LN can experience successful pregnancies (54,55).

LUPUS NEPHRITIS IN MALES

Lupus nephritis (LN) in male patients, though less common than in females, tends to follow a more aggressive course and is often associated with poorer outcomes. [redacted]

[redacted]

Whether this reflects intrinsic disease biology, differences in treatment adherence, or pharmacokinetics remains an area of active research (58).

DIAGNOSIS OF LUPUS NEPHRITIS: ROLE AND LIMITATIONS OF RENAL BIOPSY

Renal biopsy remains the gold standard for the diagnosis, classification, and management guidance of lupus nephritis (LN). [redacted]

Through direct visualization of glomerular, tubulointerstitial, and vascular lesions, renal biopsy enables differentiation between proliferative (e.g. Class III and IV) and non-proliferative (e.g. [redacted]

[redacted]

[redacted] Furthermore, histologic activity may not always correlate with clinical or serological markers (e.g., proteinuria, serum creatinine, complement levels), making biopsy both crucial and complementary to laboratory monitoring.

[redacted] However, as of now, renal biopsy remains irreplaceable in providing a definitive diagnosis and guiding evidence-based treatment decisions in lupus nephritis (60).

Figure 5: Renal Biopsy

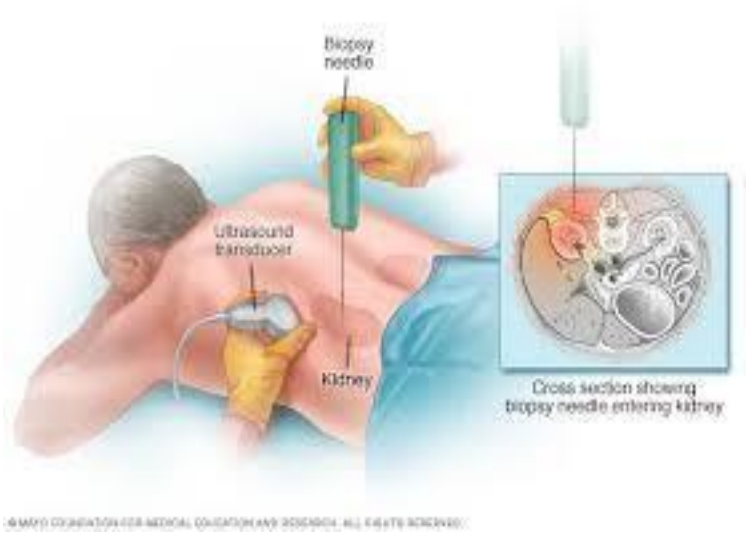
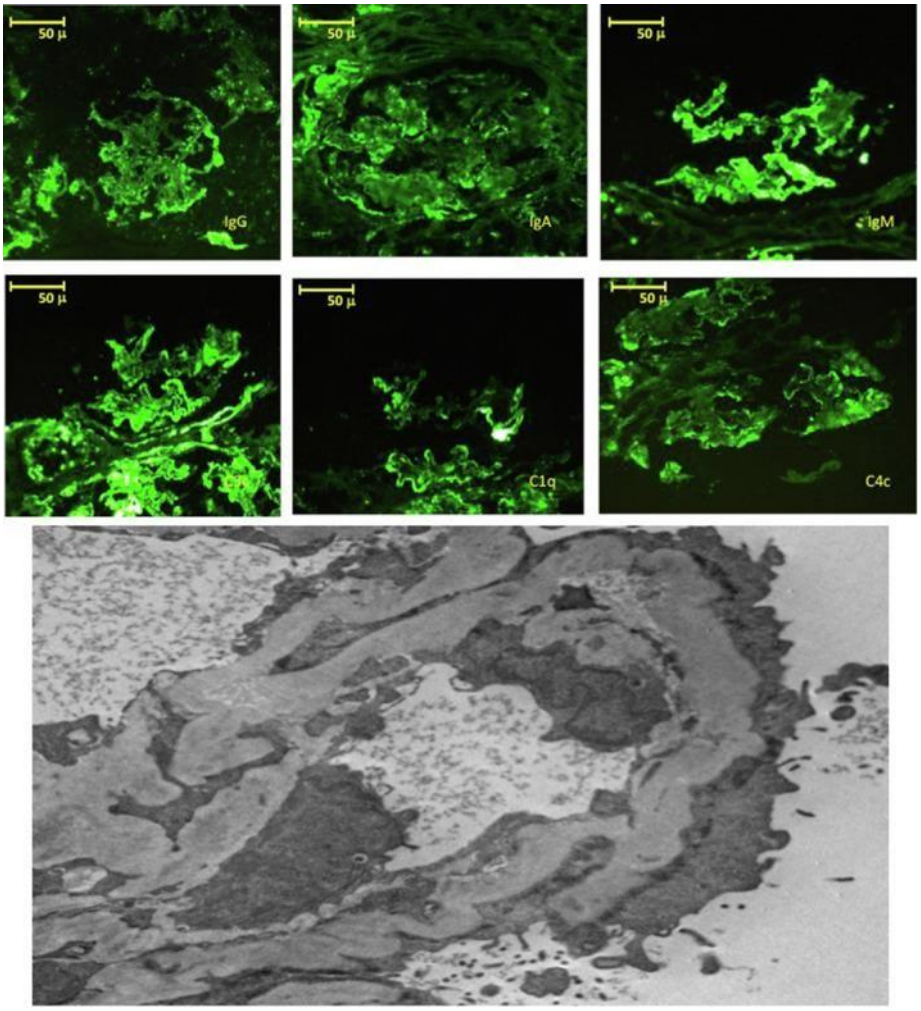


Figure 6: Renal biopsy full house image-



Role of Novel Biomarkers in Lupus Nephritis and Their Influence on the Diagnostic Workup

The search for reliable, non-invasive, and kidney-specific biomarkers in lupus nephritis (LN) has intensified over the past decade, reflecting the inherent limitations of conventional tools like renal biopsy and serological markers. [redacted]

[redacted]

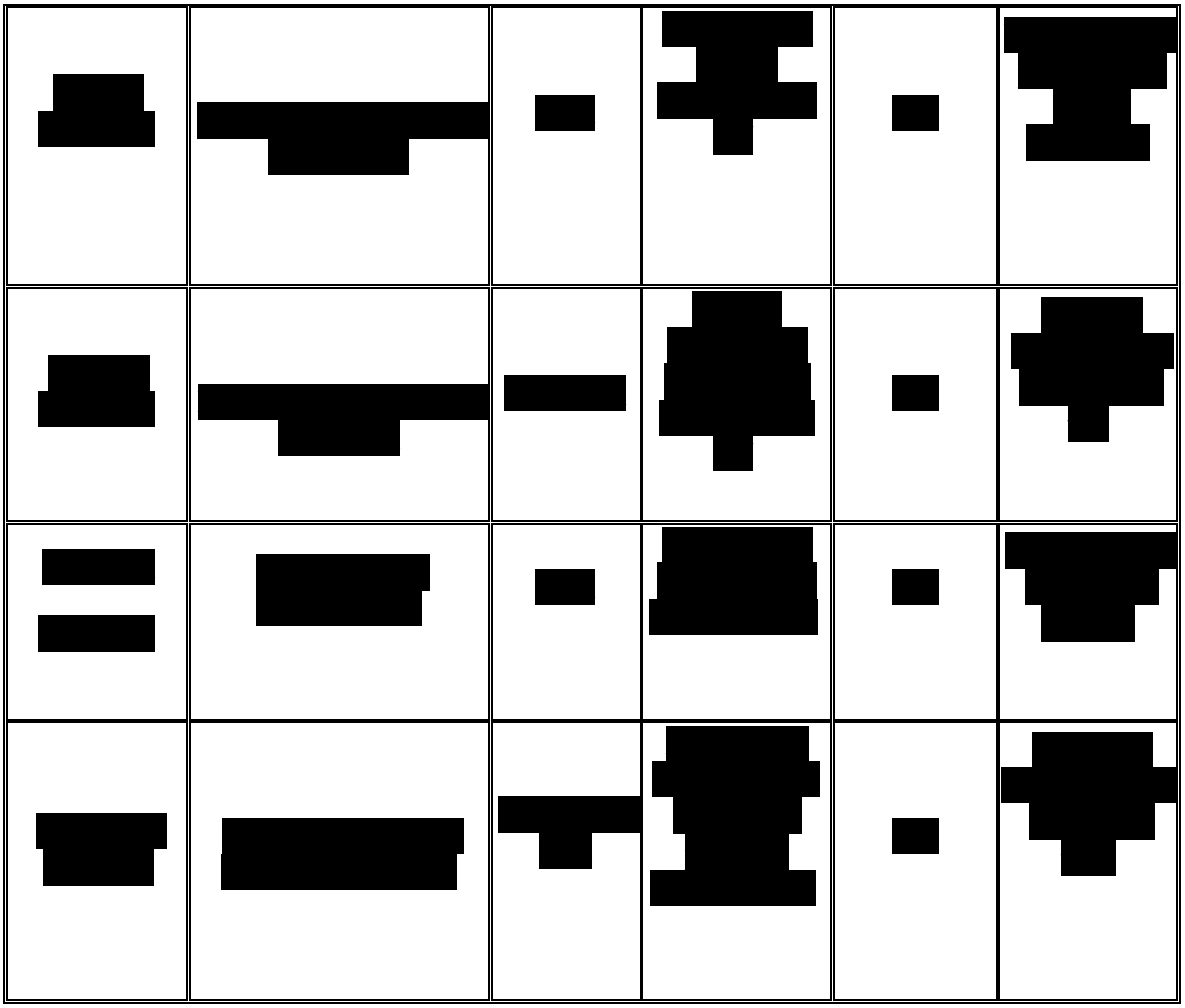
Furthermore, by identifying biomarkers that correlate with early disease activity or treatment resistance, clinicians may be able to intervene sooner and more effectively, potentially preventing chronic kidney damage and progression to end-stage renal disease (ESRD) (63).

[redacted]

[redacted]

Table 9: Comparison of Novel Biomarkers vs Traditional Markers in Proliferative Lupus Nephritis

Parameter	Biological Role	Specificity for Renal Activity	Correlation with Histological Class (Proliferative LN)	Dynamic Monitoring	Limitations
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]



Interpretation and Clinical Focus on Proliferative Lupus Nephritis

In proliferative lupus nephritis (Classes III and IV)—the most severe and aggressive forms of renal involvement in SLE—timely and accurate assessment of disease activity is critical for guiding immunosuppressive therapy. [redacted] They are particularly valuable in distinguishing histologically active disease from chronic damage, thereby preventing overtreatment or undertreatment (64).

Chronicity Index in Lupus Nephritis (NIH Scoring System)

Chronicity scores in Lupus Nephritis (LN) are essential components of the renal biopsy histopathological evaluation, especially according to the NIH Activity and Chronicity Index. [redacted]

[redacted]

[redacted]

Component	Scoring	Description
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]

Interpretation of Chronicity Scores

- 0–3 (Mild chronicity): Minimal irreversible damage; good prognosis if treated.

[redacted]

MONOCYTE CHEMOATTRACTANT PROTEIN-1 (MCP-1) AND TRANSFORMING GROWTH FACTOR-BETA (TGF-B) IN LUPUS

1. Role in Pathophysiology

Urinary MCP-1 (Monocyte Chemoattractant Protein-1)

[redacted]

It is secreted by glomerular endothelial cells, mesangial cells, tubular epithelial cells, and infiltrating leukocytes in response to

cytokine stimulation (e.g., IL-1, TNF-α).

TGF-β levels are particularly elevated in chronic and later stages of LN, marking the transition from active inflammation to irreversible renal damage (65).

2. Correlation with Disease Activity

MCP-1 levels in urine have been shown to closely correlate with renal disease activity, particularly in proliferative LN (Class III and IV).

It is less sensitive for acute activity but predictive of long-term prognosis (e.g.

Together, they complement each other: MCP-1 reflects active inflammation, while TGF-β reflects fibrosis and progression.

4. Comparison with Renal Biopsy

Feature	Renal Biopsy	Urinary MCP-1	Urinary TGF-β
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	[redacted]	[redacted]	[redacted]

[redacted]

[redacted]As assays become standardized and incorporated into clinical trials, these biomarkers are expected to play a central role in precision nephrology and non-invasive disease monitoring in SLE.

MANAGEMENT OF LUPUS NEPHRITIS

The management of lupus nephritis (LN) has evolved significantly over the past decade, and the KDIGO 2021 and 2024 guidelines represent a paradigm shift toward a more targeted, patient-centered, and less toxic approach. [redacted]

[redacted] The first option is mycophenolate mofetil (MMF) combined with corticosteroids, which is especially effective in non-White populations and in patients with moderate disease. MMF is favored due to its [redacted]

[redacted]

[redacted] However, its use requires careful monitoring due to nephrotoxicity risk and drug interactions. These biologic and targeted therapies reflect a shift toward personalized medicine, addressing both disease biology and patient variability (66).

[redacted] The treat-to- target strategy endorsed by KDIGO calls for predefined targets such as reduction of proteinuria to <0.5–0.7 g/day and stabilization of kidney function, with regular monitoring of urinalysis, serum creatinine, complement levels, and anti-dsDNA titers.

[redacted]

[redacted]

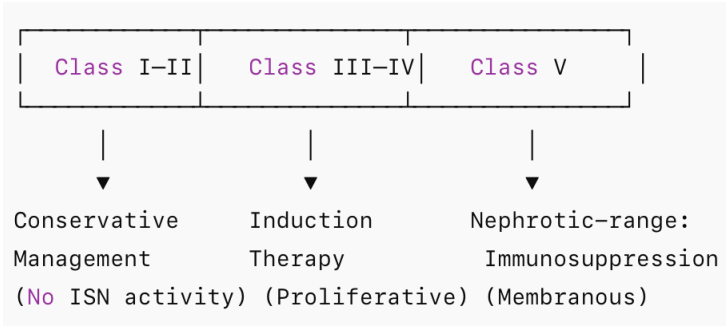
KDIGO 2021 and 2024 guidelines emphasize a risk-adapted, histology-guided, and dynamically reassessed approach to lupus nephritis. [redacted] The overarching goal remains achieving durable remission, preventing relapses, and avoiding long-term kidney damage while minimizing treatment-related toxicity (66,67).

Lupus Nephritis: Evaluation & Management Algorithm

[redacted]

Perform Kidney Biopsy (Gold Standard)

→ ISN/RPS Classification (Class I–VI)



Initial Therapy

Class I–II:

- Hydroxychloroquine (HCQ)

Class III–IV:

- **Induction:**
- Add RAAS inhibitors if proteinuric

Class V:

- If nephrotic-range proteinuria:

Re-evaluate at 6 Months

- Assess Clinical Response:
  - [redacted]

Next Steps

Good Response:

- Switch to maintenance:
  - [redacted]

Poor/No Response:

- Switch or intensify therapy:
  - [redacted]

Maintenance & Monitoring

- Maintain HCQ in all patients unless contraindicated

[redacted]

- **Monitor:**
  - [redacted]

TRIALS ON LUPUS NEPHRITIS

Lupus nephritis (LN), a severe manifestation of systemic lupus erythematosus (SLE), has been the subject of extensive clinical research aimed at improving patient outcomes and understanding the disease's pathophysiology. [redacted]

Figure 7: Landmark Trials in Lupus Nephritis (Image 1)

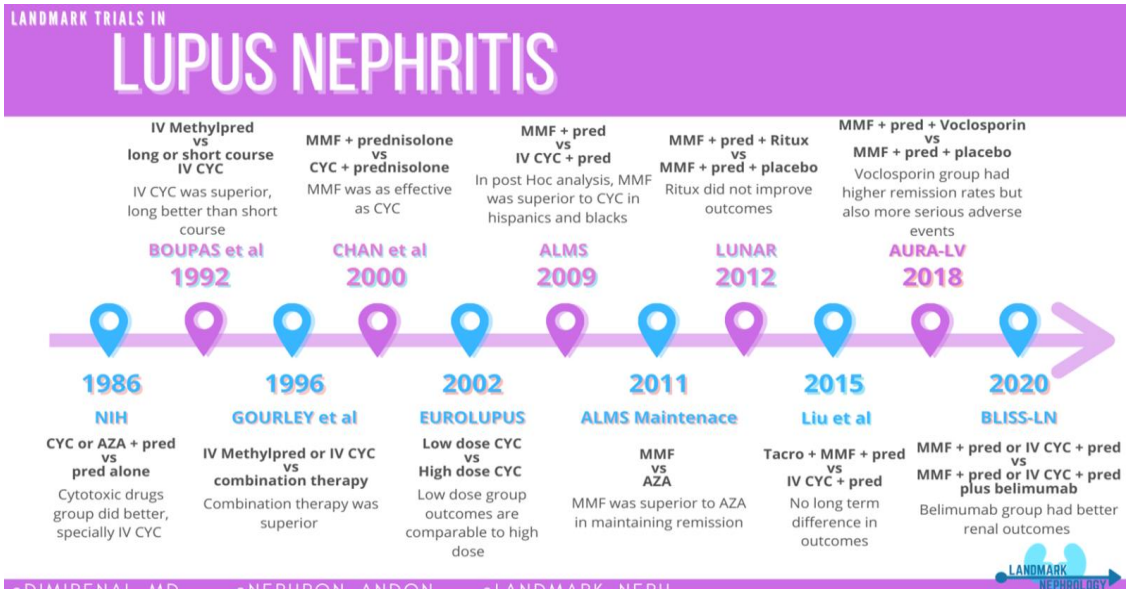


Figure 8: Landmark Trials in Lupus Nephritis (Image 2)

Studies	Study details	Inclusion / Exclusion	Outcome	Comment
HONG-KONG STUDY	MMF (2 grams/day * 6 months f/b 1 gram/day * 6 months) + Prednisolone Vs Prednisolone + oral CYC * 6 months followed by Prednisolone + Azathioprine * 6 months	Patients with proliferative lupus nephritis; and those with a serum creatinine > 3.4 mg/dl were excluded.	One year Outcome Complete remission (81% Vs 76%) Partial remission (14% Vs 14%)	Introduced MMF as induction agent; The critics were small sample size, shorter follow up and serum creatinine >3.4 mg/dl were excluded. Study in Asian population
ELNT (Euro-lupus nephritis trial)	Trial of high dose Cyclophosphamide Vs Low dose Cyclophosphamide IV MPS (750mg) * 3 doses followed by Wysolone (0.5 mg/kg/day) * 4 weeks ; steroid taper 2.5 mg every 2 weeks till 5mg to 7.5 mg maintained for two and half years. High dose IVCP– 6 monthly IVCP f/b Two Quarterly pulses; Initial dose is 0.5grams/m2, escalating the dose upto 1grams/m2 Low dose IVCP – 500 mg every 15 days * 3 months (6 doses) Maintenence – Azathioprine 2mg/kg (after last dose of CYC)	Predominantly Caucasians Mean serum creatinine was 1.15 mg/dl (range 0.5 to 4.8mg/dl) Predominantly class IV lupus nephritis	Follow up for a median period of 41 months Primary end point was treatment failure (20 % in high dose Vs 16% in low dose) The renal remission was (54% Vs 71%) not significant statistically. Renal flare (29% Vs 27%) By 10 years, the rate of ESRD was similar	Infection rate was less with low dose IVCP Best predictor of good long term outcome in ELNT is proteinuria of less than 800mg/day at 12 months The drawbacks of this study was Only mild to moderate renal impairment patients were included Predominantly Caucasians

Figure 9: Landmark Trials in Lupus Nephritis (Image 3)

Studies	Study details	Inclusion / Exclusion	Outcome	Comment
ALMS trial (Asperva lupus nephritis management study)	MMF Vs CYC for induction Multinational study MMF (2 – 3 grams/day) * 6 months Vs C[illegible] (0.5 – 1g/m2 IV monthly *6 months	Class III – Class IV Primary endpoint at 6 months Renal response defined as (a)Stabilization of serum creatinine or improvement in serum creatinine (b)If nephrotic range proteinuria – UPCR <3gm/gm (c) If initial UPCR <3gm/gm – 50% reduction from baseline.	Renal response at 6 months 56% Vs 53 % (MMF Vs CYC) at 6 months Complete remission is 8% in both the groups	Strength Larger number of patients (n =370)

Table 6. Landmark maintenance trials in lupus nephritis.

Studies	Study details	Inclusion / Exclusion	Outcome	Comment
MAINTAIN	MMF Vs AZA maintenence	Patients with proliferative lupus ; Induction – ELNT regime ; irrespective of remission status, patients were given MMF Vs AZA for a maintenece period of 3 years	20% renal flare with MMF 25% renal flare with Azathioprine	Supports AZA and MMF are equivalent for preventing relapses in moderately severe lupus nephritis following induction with IV CYC.

(Grand Rounds in Nephrology. Kasi Vishweswaran. [redacted] ISBN: 9789383989768.)

1. Trials of Calcineurin Inhibitors (CNIs)

a. Tacrolimus vs. Cyclophosphamide (CYC):

- [redacted] **(2012):** This randomized, non-inferiority trial compared the efficacy and safety of tacrolimus to intravenous cyclophosphamide (IV CYC) in patients with active LN. [redacted]
- **Li et al. (2013):** In this randomized trial, tacrolimus was compared to IV CYC in Chinese patients with active LN. [redacted]

Figure 10: Voclosporin in Lupus Nephritis (AURORA 1 Trial):

Table 9. Voclosporin in lupus nephritis.

Trial	Inclusion/ Exclusion	Intervention	Endpoint	Outcome
AURORA 1 Phase 3 RCT	Patients with Class III,IV Lupus nephritis	Voclosporin 23.7 mg twice a day or placebo in combination with MMF 1000 mg twice a day and rapidly tapered prednisone for 52 weeks Patients with an eGFR of <45 mL/min/1.73 m2 BSA were excluded from the study	The primary end point of the study was a complete kidney response at 1 year.	The primary end point of complete renal response at week 52 was achieved in the voclosporin than in the placebo group

Based on the results of AURORA 1, voclosporin is FDA-approved for the treatment of lupus nephritis in combination with MMF and glucocorticoids.

b. Cyclosporine vs. Cyclophosphamide:

[redacted] **(1990s):** This randomized trial evaluated the efficacy of cyclosporine compared to IV CYC in patients with proliferative LN. [redacted]

2. Trials of Biologic Agents

a. Belimumab (Anti-BAFF Antibody):

[redacted] **(2020):** The BLISS-LN trial was a phase 3, multinational, randomized, double-blind, placebo-controlled study conducted over 104 weeks at 107 sites in 21 countries. [redacted]

[redacted]

[redacted]

[redacted] The safety profile of belimumab was consistent with previous studies, indicating no new safety concerns (71).

Figure 11: Belimumab in Lupus Nephritis (BLISS-LN Trial):

Table 8. Belimumab in lupus nephritis.

Trial	Inclusion/ Exclusion	Intervention	Endpoint	Outcome
BLISS LN	Biopsy proven lupus nephritis (Class III,IV,V), UPCR > 1g; Exclusion: eGFR < 30 ml/ min/1.73m2	Belimumab (at a dose of 10 mg/kg) or placebo, on a background of standard induction therapy (Cyclophosphamide Vs Mycophenolate mofetil). Intravenous belimumab or placebo on days 1 (baseline), 15, and 29 and every 28 days thereafter to week 100.	Primary efficacy renal response at week 104, defined as: (a) UPCR of 0.7 or less, (b) eGFR that was no worse than 20% below the pre-flare value or at least 60 ml/ min/ 1.73 m^2, and (c) No use of rescue therapy for treatment failure. Secondary endpoint was complete renal response at week 104.	At week 104, primary efficacy renal response was significantly higher in patients in the belimumab group than in the placebo. Significantly more patients had a complete renal response at week 104 in the belimumab group than placebo group

b. Obinutuzumab (Anti-CD20 Monoclonal Antibody):

[redacted] **(2023):** The REGENCY trial was a phase 3, randomized, controlled trial that assessed the efficacy and safety of obinutuzumab, a humanized type II anti-CD20 monoclonal antibody, in adults with biopsy-proven active LN. [redacted]

[redacted]

MATERIALS AND METHODS

MATERIALS AND METHODS

[redacted]

Inclusion Criteria :

[redacted]

Exclusion criteria:

[redacted]

All patients will be subjected to renal biopsy

Lab investigations done included the following :

[redacted]

Other routine investigations such as CBC, Urine routine, Mini Renal (Creatinine estimation by Enzymatic Method), LFT and demographic details were obtained.

Novel Biomarkers

- **Urinary MCP-1**

Sample Collection and Processing

[redacted]

Assay Procedure

[redacted]

[redacted]

Cutoff

[redacted]

- **Urinary TGF-beta**

Sample Collection and Processing

[redacted]

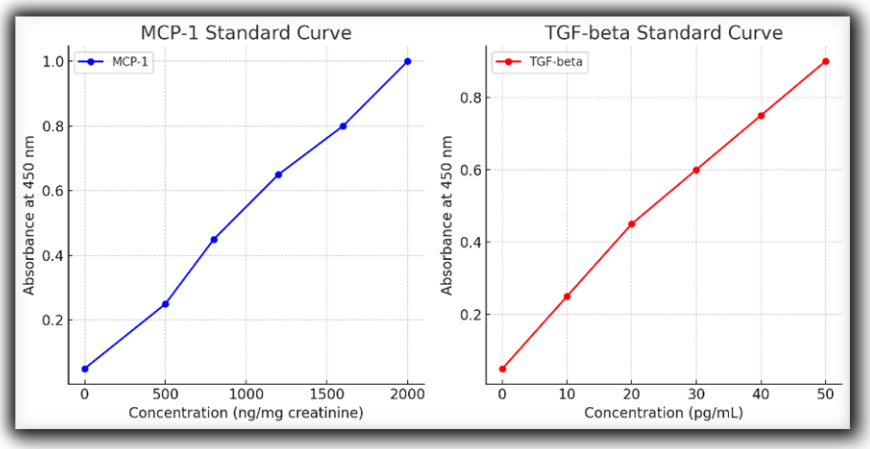
Assay Procedure

[redacted]

[redacted]To confirm the assay's accuracy, each batch of testing included a standard curve that was used to compare the absorbance of the unknown samples to known concentrations of TGF-beta, ensuring correct calibration and consistency.

TGF-beta Cutoff

[redacted]



[redacted]Study Design: [redacted]

[redacted]Study Period: [redacted]

c. **Sample Size:**

$$\text{Sample Size} = \frac{Z\alpha^2 \quad P\,(1\text{-}P)}{d^2}$$

[redacted]

Sample size	[redacted]

[redacted]

Minimum of 35 cases of lupus nephritis were selected.

[redacted] **Sampling method** : [redacted]

- **Statistical Analysis :**

[redacted] **Study protocol:** [redacted]

[redacted] **Data collection procedure:** [redacted]

[redacted] **Data processing and analysis/statistical analysis** [redacted]

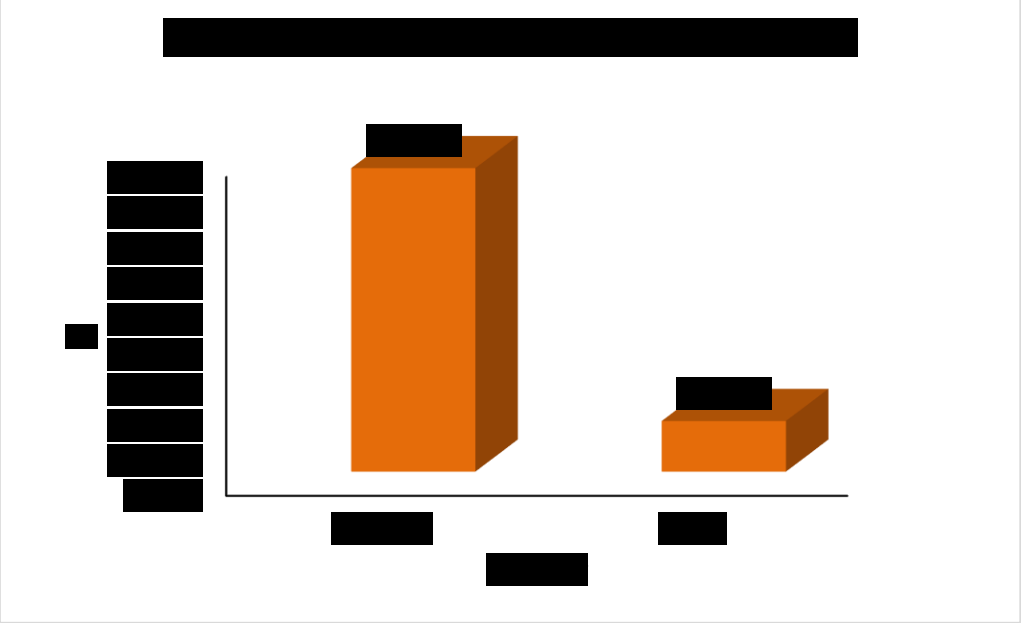
RESULTS AND OBSERVATION

RESULTS AND OBSERVATION

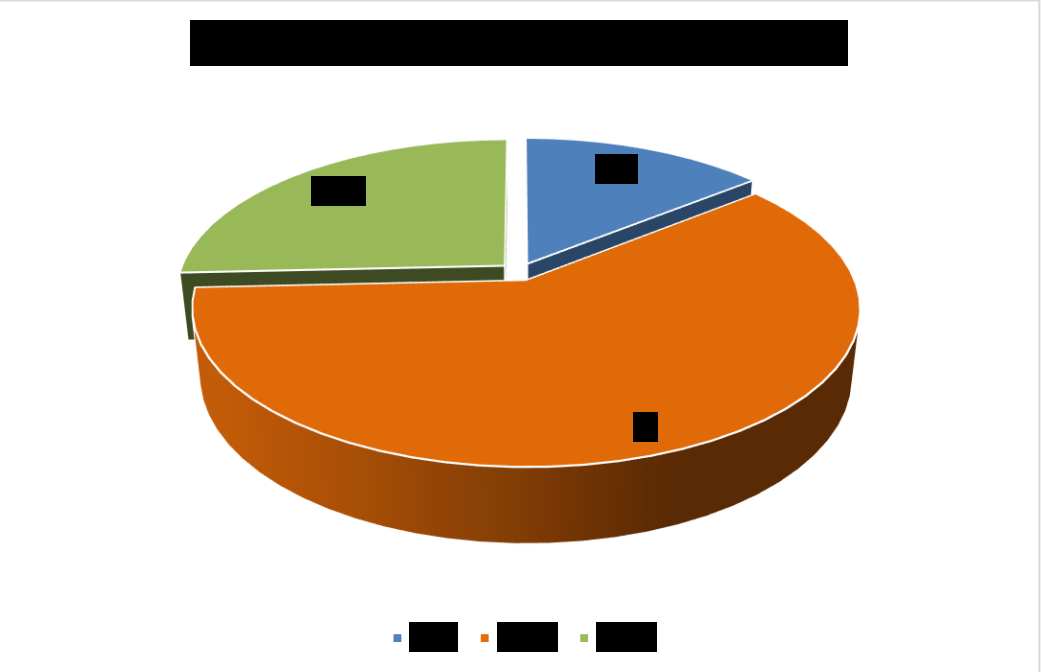
Table 10: Distribution according to demographic details

Parameter	Value / n (Median, IQR)	Percentage
Total patients		
Age (years)		
1-20		
21-40		
41-60		
Gender: Female		
Gender: Male		
Biopsy Class distribution		
Class III		
Class IV		
Class IV/V		
Class V		
Class II		
Class III/V		
Class VI		
Anti-dsDNA Positive		
Anti-dsDNA Negative		
C3 (mg/dl)		
C4 (mg/dl)		
uMCP-1 (pg/mgCr)		
uTGF-β (pg/mL)		
Activity Index		
Chronicity Index		
Serum Creatinine (mg/dL)		
UPCR (g/g)		
rSLEDAI-2K		

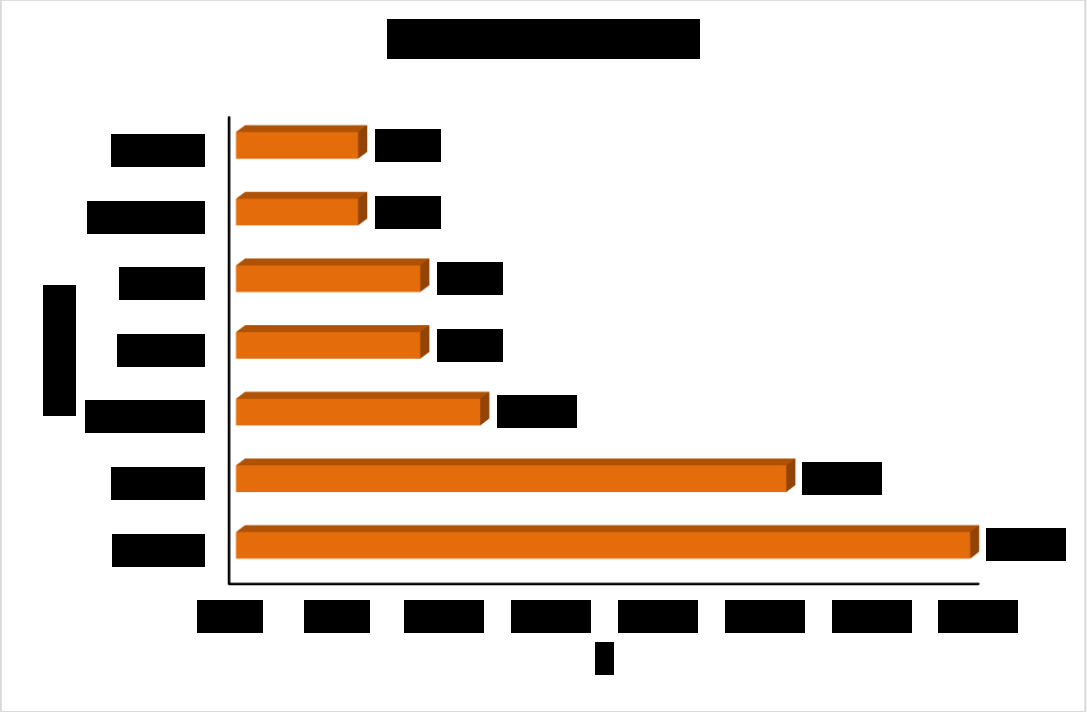
Graph 1: Gender distribution of Lupus Nephritis Patients



Graph 2: Age distribution of Lupus Nephritis Patients

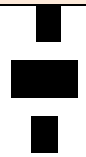
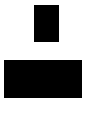
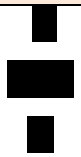
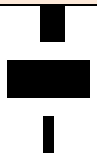
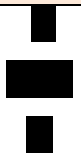
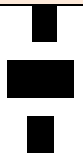
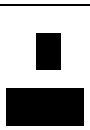
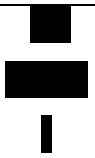
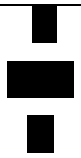
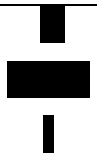
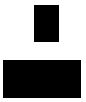
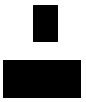
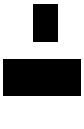
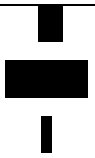
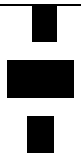
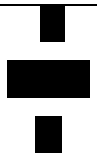
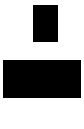
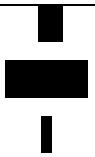
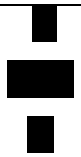
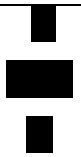
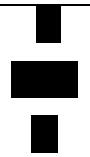
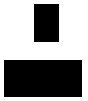
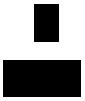
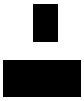
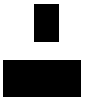
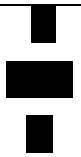
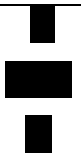
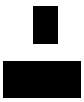
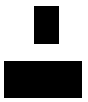
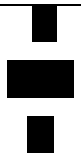


Graph 3. Biopsy class distribution

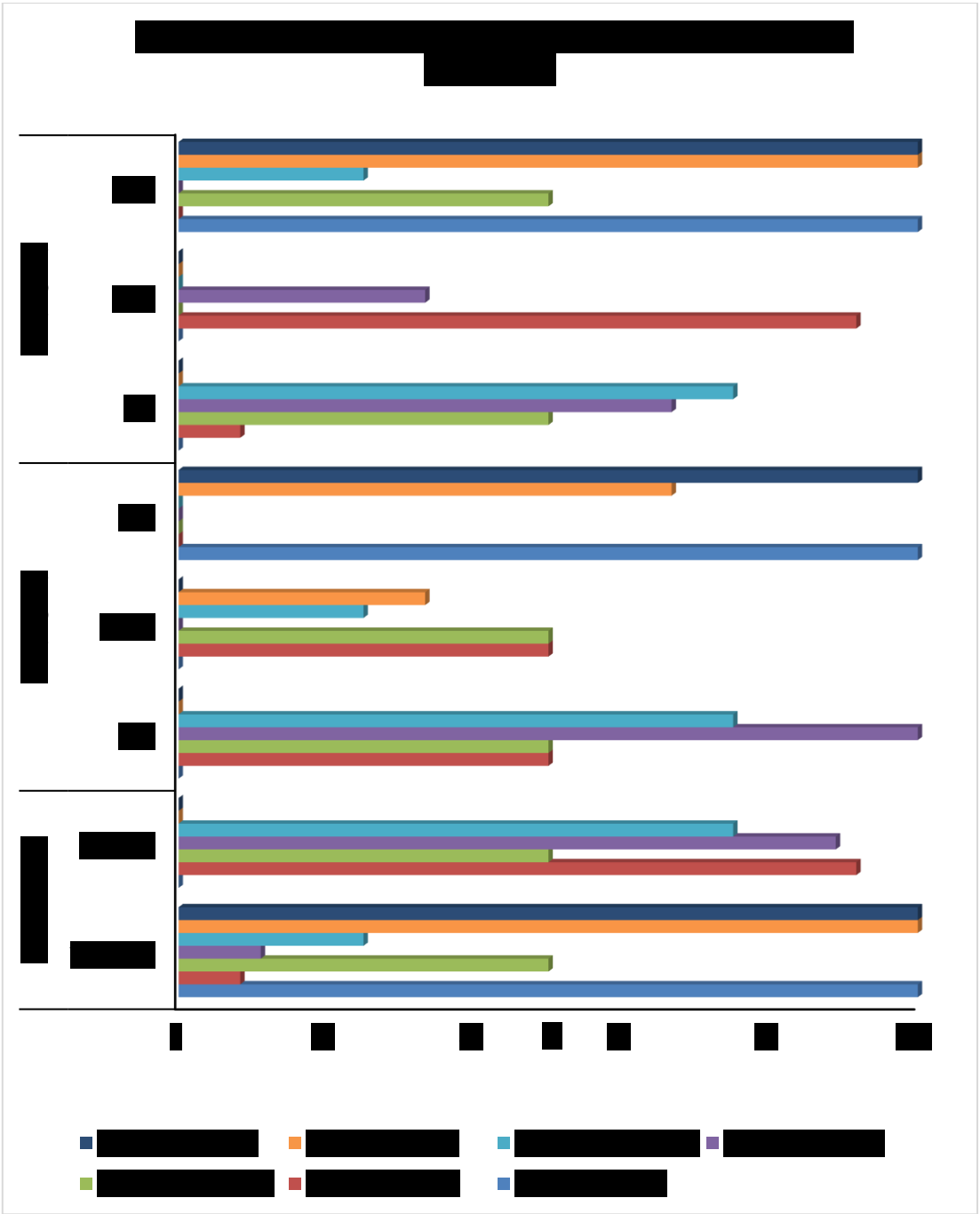


[redacted]

Table 11: Biopsy Class and Its Association with Serological markers such as C3, C4, Anti-dsDNA

		Biopsy Class							Chi-Square (Sig.)
		Class II	Class III	Class III/V	Class IV	Class IV/V	Class V	Class VI	
Anti-DsDNA	Negative								
	Positive								
C3 (Mg/Dl)	< 60								
	60-90								
	>90								
C4 (Mg/Dl)	< 5								
	5-10								
	> 10								

Graph 4: Biopsy Class and Its Association with Serological markers such as C3, C4, Anti-dsDNA



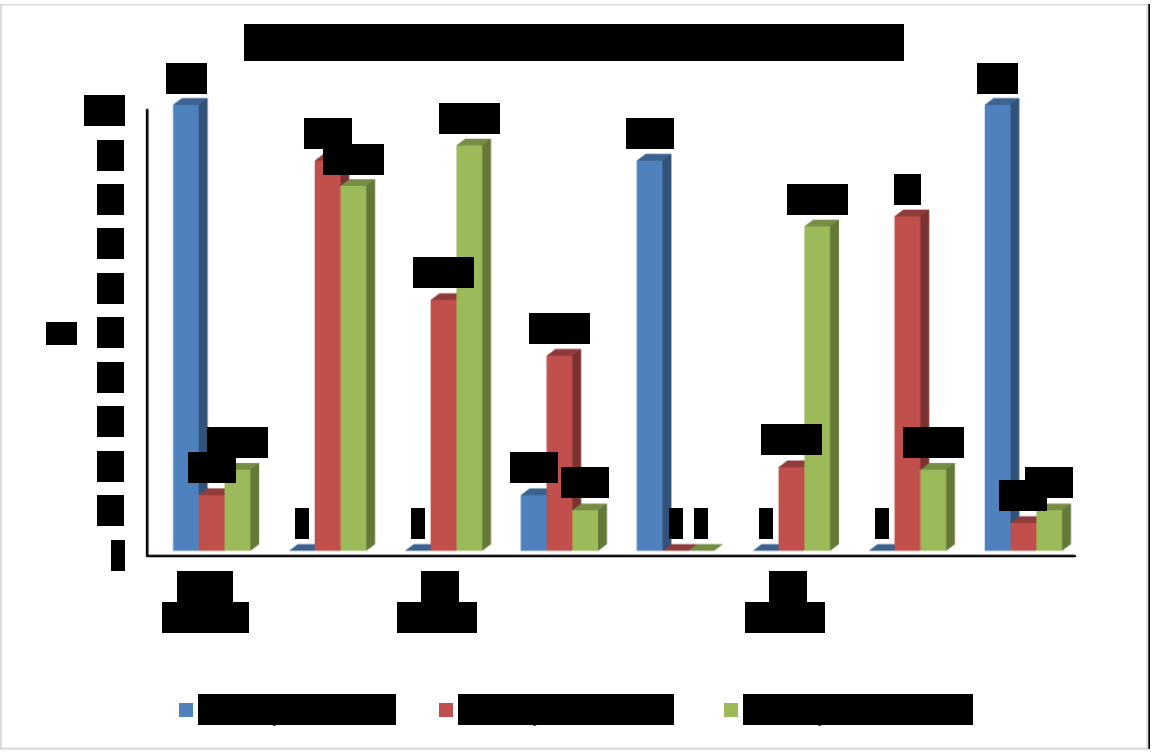
[redacted]

[redacted]

Table 12: Activity Index and Its Association with Serology - C3, C4, Anti-dsDNA

	Activity Index			Chi-Square (Sig.)
	0-5	6-12	13-24	
Anti-DsDNA	[redacted]	[redacted]	[redacted]	[redacted]
C3 (Mg/dL)	[redacted]	[redacted]	[redacted]	[redacted]
C4 (Mg/dL)	[redacted]	[redacted]	[redacted]	[redacted]

Graph 5: Activity Index and Its Association with Biomarkers

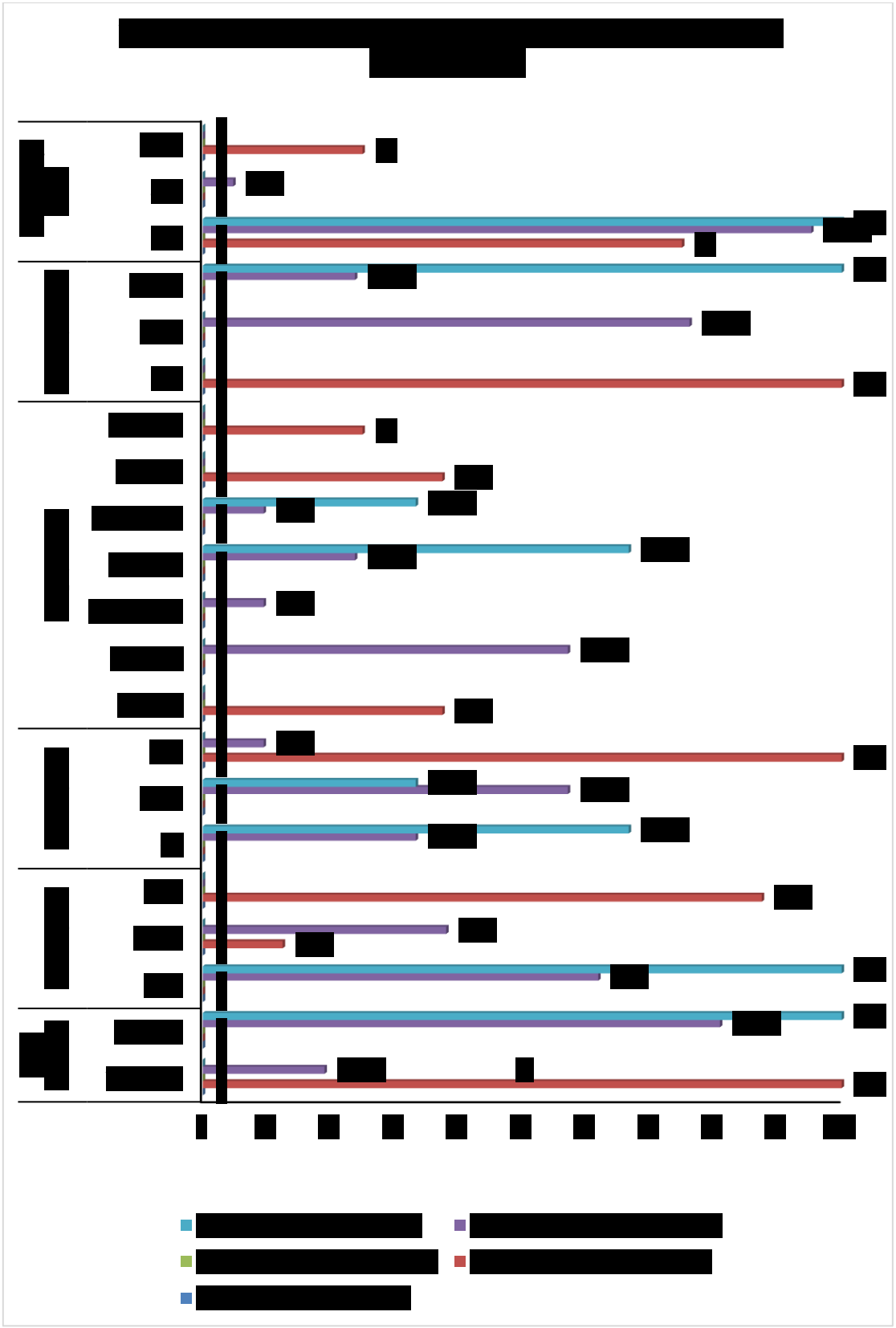


[redacted]

Table 13: Urinary MCP-1 Levels and Its Association with Serological and Biopsy Classes

		uMCP-1 (pg/mgCr)					Chi-Square (Sig.)
		<100	100-300	300-500	500-1000	>1000	
Anti-DsDNA	Negative						
	Positive						
C3 (mg/dL)	< 60						
	60-90						
	> 90						
C4 (mg/dL)	< 5						
	5-10						
	>10						
Biopsy Class	Class II						
	Class III						
	Class III/V						
	Class IV						
	Class IV/V						
	Class V						
	Class VI						
Activity Index	0-5						
	6-12						
	13-24						
Chronicity Index	0-3						
	4-6						
	7-12						

Graph 6: Urinary MCP-1 Levels and its Association with Serological parameters and Biopsy Classes, Activity index and Chronicity index

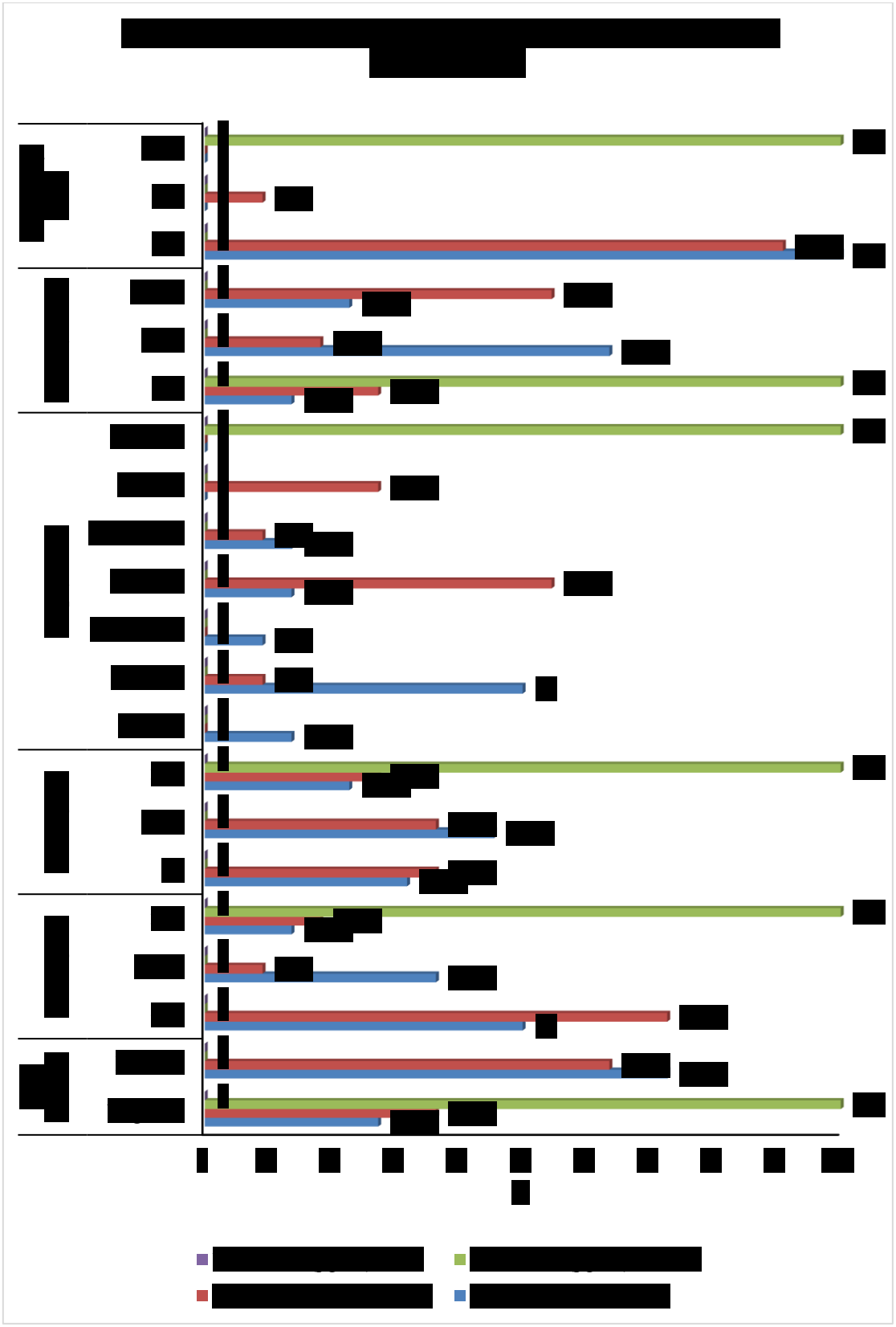


[redacted]

Table 14: Urinary TGF-β Levels and Its Association with Serological and Biopsy Classes, Activity index and Chronicity index

		uTGFbeta (pg/ml)				Chi-square (Sig.)
		<10	10-40	40-100	>100	
Anti-DsDNA	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
C3 (Mg/Dl)	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
C4 (Mg/Dl)	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
Biopsy Class	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
Activity Index	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
Chronicity Index	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	

Graph 7: Urinary TGF-β Levels and Its Association with Serological and Biopsy Classes, Activity Index and Chronicity Index



[redacted]

Table 15: Correlations Between Urinary TGF-β and Serum Creatinine and uPCR

	uTGFbeta		
	N	R	Sig.
S. Creatinine (mg/dL)	[redacted]	[redacted]	[redacted]
uPCR (g/g)	[redacted]	[redacted]	[redacted]

[redacted]

[redacted]

Table 16: Urinary MCP-1 Levels and Their Correlation with Clinical Parameters

	uMCP-1 (Pg/mgCr)		
	N	R	Sig.
S. Creatinine (mg/dL)	[redacted]	[redacted]	[redacted]
uPCR (g/g)	[redacted]	[redacted]	[redacted]

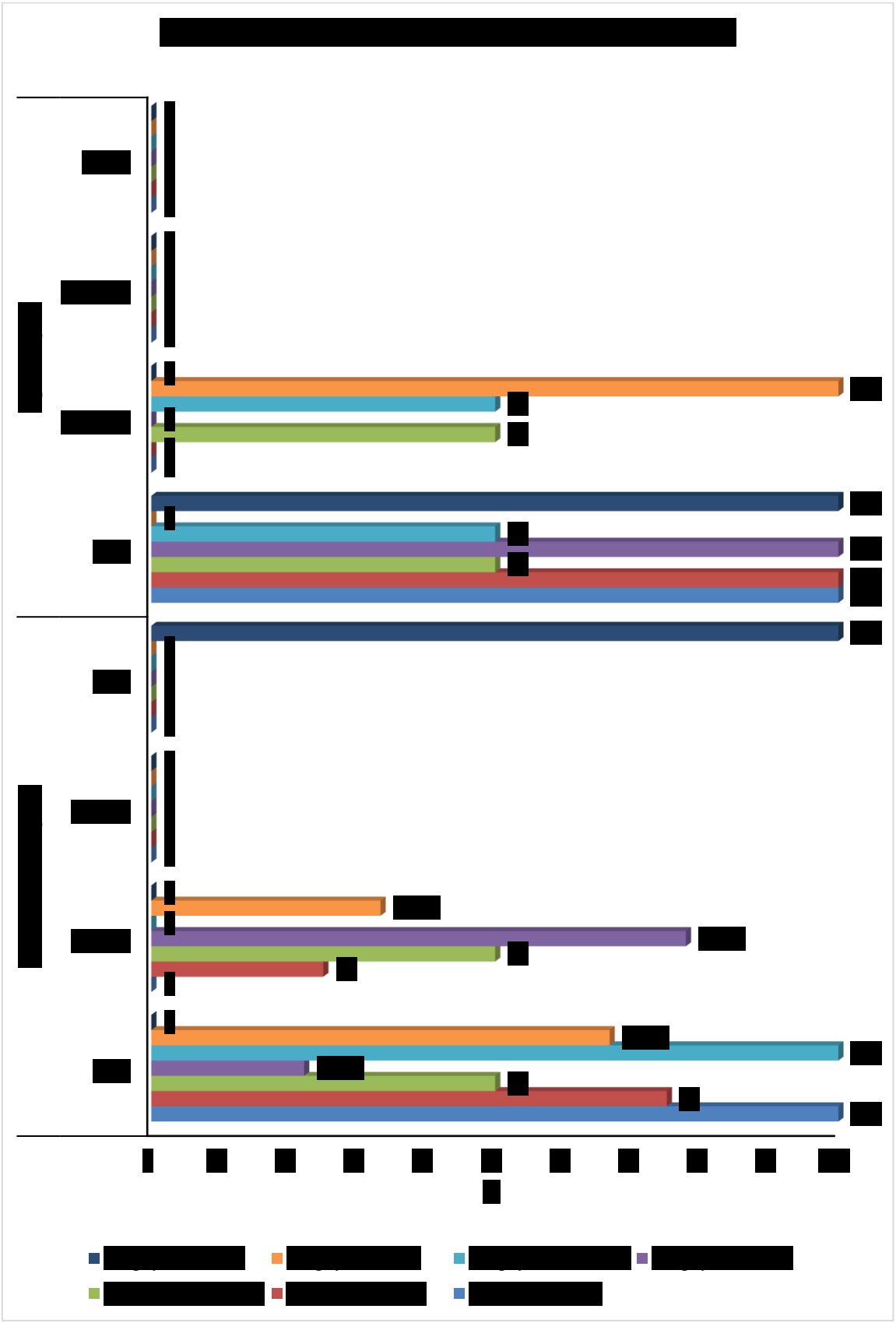
[redacted]

[redacted]

Table 17: Biopsy Class and Its Correlation with Serum Creatinine and uPCR

		Biopsy Class							Chi-Square (Sig.)
		Class II	Classii i	Classi ii/V	Class IV	Class IV/V	Class V	Class VI	
S. Creatinine (Mg/Dl)	<1.0								
	1.0 - 1.5								
	1.5 - 3.0								
	>3.0								
UPCR (G/g)	<1.5								
	1.50- 5.0								
	5.0 - 10.0								
	>10.0								

Graph 8: Biopsy Class and Its Correlation with Serum Creatinine and uPCR

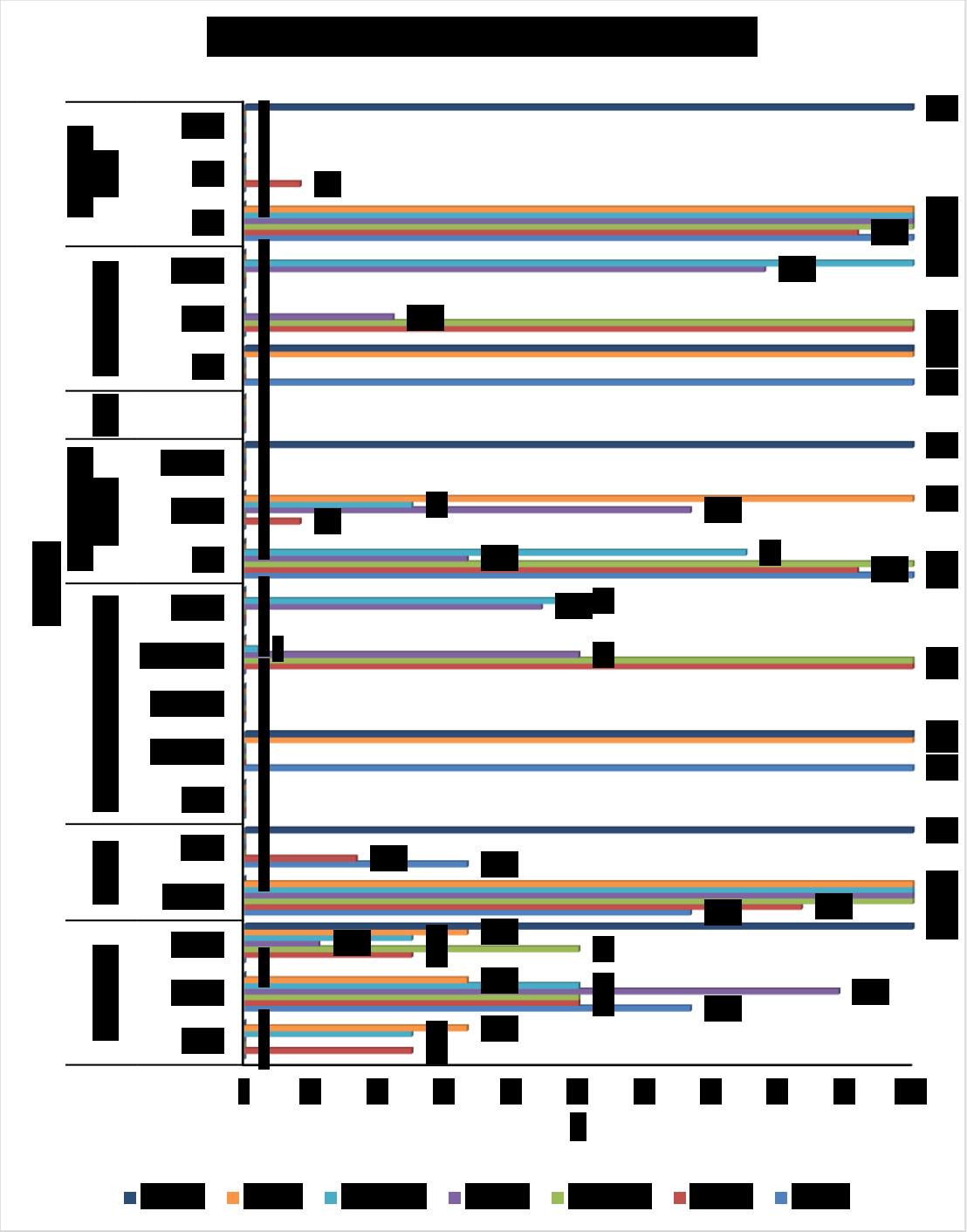


[redacted]Some proliferative LN patients (Classes III and IV) who had low proteinuria (<1.5 g/g) despite biopsy-proven active disease with NIH activity scores ≥6, indicating that reliance on proteinuria alone may fail to detect active inflammation.

Table 18: Biopsy Class and Age/Gender Distribution

Parameter	Category	Class II	Class III	Class III/V	Class IV	Class IV/V	Class V	Class VI	χ^2 (p)
Age (years)	1–20	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	21–40	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	41–60	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
Gender	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	Male	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
Urinary MCP-1 (pg/mgCr)	<100	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	100–300	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	300–500	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	500–1000	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	>1000	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
Urinary TGF-β (pg/mL)	<10	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	10–40	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	40–100	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
>100		[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
Activity Index	0–5	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	6–12	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	13–24	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
Chronicity Index	0–3	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
	4–6	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	
	7–12	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	

Graph 9: Biopsy Class and Age/Gender Distribution



[redacted]

Table 19: Biopsy Class and rSLEDAI-2K Correlation

		rSLEDAI-2K		
		Median	**IQR**	**P-Value**
Biopsy Class	**Class II**	[redacted]	[redacted]	[redacted]
	Class III	[redacted]	[redacted]	
	Class III/V	[redacted]	[redacted]	
	Class IV	[redacted]	[redacted]	
	Class IV/V	[redacted]	[redacted]	
	Class V	[redacted]	[redacted]	
	Class VI	[redacted]	[redacted]	

[redacted]

[redacted]

[redacted]

[redacted]However, some Class III patients with moderate scores had relatively low proteinuria (<1.5 g/g) despite active histology, indicating that rSLEDAI-2K can detect activity not evident from proteinuria alone.

DISCUSSION

DISCUSSION

Lupus nephritis (LN) is a major organ manifestation of systemic lupus erythematosus (SLE) that substantially influences morbidity and mortality in affected individuals. Despite advances in therapy, up to [redacted] of LN patients progress to end-stage renal disease, underscoring the clinical imperative for early diagnosis, accurate activity assessment, continual monitoring, and effective therapeutic guidance ([redacted]

[redacted] who first highlighted urinary MCP-1's robust association with active LN and proliferative histology in Indian cohorts using sensitive ELISAs. They also demonstrated MCP-1's predictive capacity for renal flares, suggesting utility in early diagnosis and monitoring.

[redacted]

[redacted]

In striking contrast to MCP-1, urinary TGF-β levels in our cohort showed a strong positive correlation with serum creatinine and chronic histologic indices but weak or no association with histological activity scores, anti-dsDNA status, or acute immunological markers. High urinary TGF-β concentrations were most pronounced in chronic sclerotic Class VI and advanced Class IV/V LN, supporting its established role as a marker of renal fibrogenesis rather than acute inflammation.

[redacted]

[redacted]

Morell et al. (2021) (25) echo this perspective, focusing on immune-related urine biomarkers as adjunct tools for early diagnosis and tracking response to therapy, reflecting the dynamic inflammatory and remodelling processes intrinsic to LN.

[redacted]

[redacted]

Emerging research on cellular profiling in urine sediment, microRNA biomarkers, and complement activation product measurement also shows promise for enhanced biomarker performance (Aragón et al., 2020) (31).

[redacted]

[redacted]

[redacted]Future prospective, multicenter, and longitudinal studies are needed to validate and standardise these biomarkers, and to develop composite activity and chronicity indices that can guide precision nephrology in SLE.

STRENGTHS AND LIMITATIONS

STRENGTHS OF THE STUDY

1. **Comprehensive Clinicopathological Correlation:**

[redacted]

2. **Use of Novel Urinary Biomarkers:**

[redacted]

3. **Well-Defined Biopsy Subclassification:**

[redacted]

4. **Statistical Rigor:**

[redacted]

5. **First-of-its-Kind Regional Data:**

[redacted]

LIMITATIONS OF THE STUDY

1. **Small Sample Size per Subgroup:**

2. **Cross-Sectional Design:**

3. **Lack of Longitudinal Biomarker Follow-Up:**

4. **Absence of Treatment Data Correlation:**

5. **Single-Centre Study:**

CONCLUSION

CONCLUSION

This study establishes a significant clinicopathological correlation in patients with lupus nephritis, emphasizing the diagnostic and prognostic value of combining renal biopsy classes with clinical parameters and urinary biomarkers. [redacted]

[redacted]

[redacted]

[redacted]

[redacted]Future research with larger cohorts and longitudinal follow-up is warranted to validate urinary biomarkers as predictive tools for disease progression and therapeutic response.

SUMMARY

SUMMARY

This prospective observational study aimed to evaluate the clinicopathological correlation in patients with biopsy-proven lupus nephritis and assess the utility of urinary biomarkers—MCP-1 and TGF-β—as indicators of disease activity and chronicity. [redacted]

BIBLIOGRAPHY

BIBLIOGRAPHY

1. García-Carrasco M, Pinto CM, Poblano JCS, Morales IE, Cervera R, Anaya JM. Systemic lupus erythematosus. 2013 Jul 18. Stear Pearls
2. Vaillant AAJ, Goyal A, Varacallo MA. Systemic Lupus Erythematosus. Dermatology: Volume 1–2, Fifth Edition. 2023 Aug 4;1:670–88.

[redacted]

[redacted]

[redacted]

[redacted]

[redacted]

72. Furie RA, Rovin BH, Garg JP, Santiago MB, Aroca-Martínez G, Zuta Santillán AE, et al. Efficacy and Safety of Obinutuzumab in Active Lupus Nephritis. N Engl J Med. 2025 Apr;392(15):1471–83. doi:10.1056/NEJMoa2410965

ANNEXURE

ANNEXURE I – INFORMED CONSENT

“[redacted]

Name of Principal Investigator: [redacted]

[redacted]

Withdrawal from participation in the study: [redacted]

Possible benefits from participating in the study: [redacted]

Possible risks from participating in the study: [redacted]

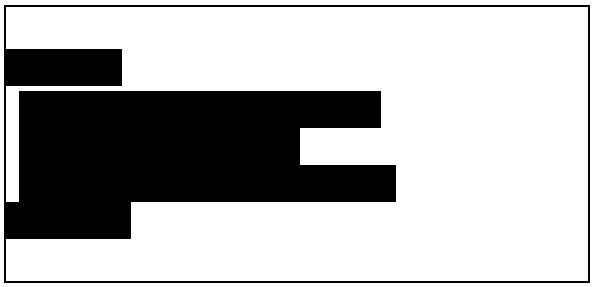
Privacy and confidentiality: [redacted]

Financial incentives: [redacted]

The cost of investigations [redacted]

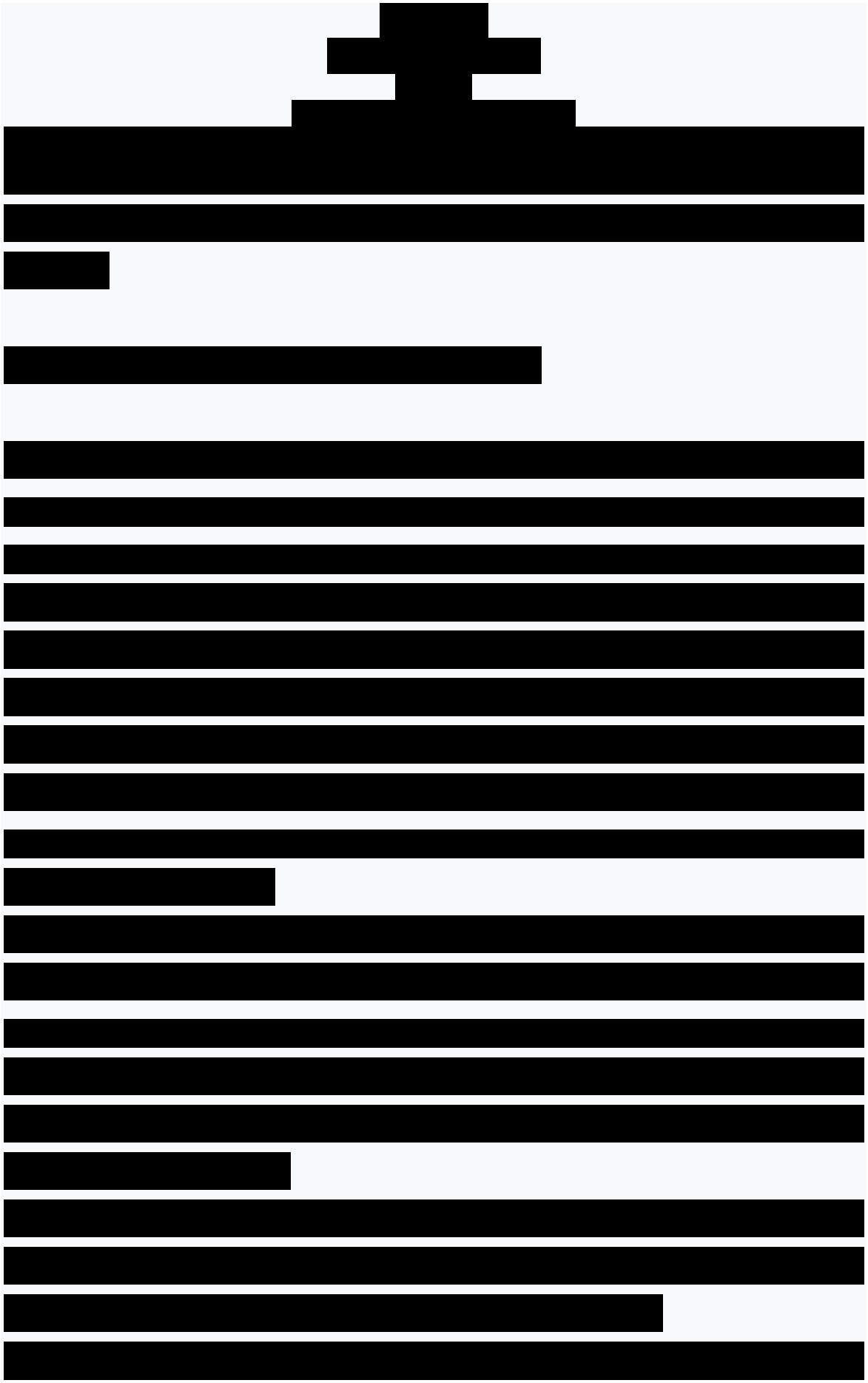
Authorization for publication of aggregated data [redacted]

Questions: [redacted]



[redacted]

Legal rights: [redacted]



[redacted]

[redacted]

[redacted]

[redacted]

[redacted]

ANNEXURE II – ETHICAL CLEARANCE

ANNEXURE III – CLINICAL PROFORMA

Name: **Age :** **Sex :**

Address: **Phone No:**

History: **IP Number :**

[redacted]	[redacted]	[redacted]	[redacted]	[redacted]

[redacted]	[redacted]	[redacted]

[redacted]	[redacted]	[redacted]	[redacted]

[redacted]	[redacted]	[redacted]

General examination

[redacted]

Systemic examination

[redacted]

[redacted]

[redacted]

[redacted]	[redacted]	[redacted]

[redacted]

[redacted]

[redacted]

[redacted]	[redacted]	[redacted]	[redacted]	[redacted]

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[redacted]	[redacted]	[redacted]

[redacted]

[redacted]	[redacted]	[redacted]	[redacted]

[redacted]

[redacted]

[redacted]

[redacted]

[redacted]	[redacted]	[redacted]

[redacted]

[redacted]

[redacted]

[redacted]

ANNEXURE – IV MASTER CHART

Sr. No.	Age	Gender	ANA	BIOPSY CLASS	ANTI-DSDNA	C3 (mg/dl)	C4 (mg/dl)	uMCP-1 (pg/mgCr)	uTGFbeta (pg/ml)	ACTIVITY INDEX	CHRONICITY INDEX	S. Creatinine (mg/dl)	UPCR (g/g)	rSLEDAI-2K
[redacted]	28 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	41 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	19 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	44 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	68 years	Male	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	23 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	15 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	42 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	18 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	34 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	28 years	Male	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	24 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	43 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	45 years	Male	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	42 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	23 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	30 years	Male	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	22 years	Male	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	22 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	36 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	16 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	24 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	27 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	29 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	42 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	26 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	16 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	21 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	31 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	46 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	34 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	35 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	39 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	34 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]
[redacted]	33 years	Female	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]	[redacted]