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LUPUS NEPHRITIS IS A SERIOUS COMPLICATION OF SLE-A NARRATIVE REVIEW

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Abstract

Background: One common and severe SLE manifestation is lupus nephritis (LN). The clinical appearance and epidemiology of LN have changed over the years. Nevertheless, despite improvements in treatment approaches, the proportion of patients developing end-stage renal disease (ESRD) remains constant, despite retrospective cohort studies showing a lower death rate and an improved prognosis. Although kidney transplantation has been acknowledged as the best course of action for those patients, a number of problems still exist following renal function recovery in a patient with lupus. These include the worry that lupus nephritis will recur in the graft, the decision to use immunosuppressive therapy in recurrent lupus cases for patients who have already had a toxic and protracted immunosuppressive course, and, lastly, the management of comorbidities to lower long-term associated morbidities. In this review we address the main changes and persistent unmet needs in LN in SLE management throughout the past decades, with a focus on prognosis and upcoming treatment.

INTRODUCTION

The therapy of lupus nephritis has advanced greatly over the past three decades, yet between 1982 and 2004, the incidence of end stage renal disease (ESRD) increased significantly, rising from 1.16 in 1982 to 1.16 in 2004. The number of cases per million person-years decreased from 2.08 in 1982 to 3.08 and 4.9 in 1995 and 2004, respectively.[1,2] For patients with incident ESRD, kidney transplantation (RENAL TRANPLANTATION) is the recommended course of treatment. Hemodialysis (HD) is currently the standard of care for patients who develop end-stage renal disease (ESRD) due to aggravation of lupus nephritis or newly diagnosed lupus with rapidly progressing renal disease.[3]

Prior to undergoing transplantation, remission of lupus in general is crucial, thus all patients with recent considerable renal or extra-renal activity and end-stage renal disease (ESRD) should do so start with HD. The thought to be beneficial "burn-out" impact of this method on the illness is one advantage of this choice. Second, for people with fast progressing lupus-related glomerulonephritis, 3 to 6 months of dialysis seem to be enough time for renal function to recover before undergoing transplantation. On the other hand, if a suitable living donor is available, patients who have experienced complete remission for a significant amount of time before ESRD may also proceed with transplantation.[3,4]

SLE frequently affects the kidney, and glomerular, tubule interstitial, and vascular abnormalities cause renal function to be impaired. About 40% of SLE patients get LN, usually within five years after the diagnosis, and continues to exhibit a 4.3–10.1% progression rate towards end-stage renal disease (ESRD). Renal failure ranks among the leading causes of death for individuals with SLE, along with infections, malignancies, and cardiovascular events. The range of clinical presentations for LN varies, ranging from asymptomatic urine anomalies to very symptomatic instances of nephritic syndrome or fast progressing renal insufficiency. The incidence of LN varies with ethnicity.[5,6]

One major side effect of SLE is lupus nephritis. In teenage girls of African American descent, it manifests with severe clinical characteristics. Clinical signs of lupus nephritis are present in 50% of patients who are diagnosed with SLE. The accumulation of circulating immune complexes, which activates the complement system and causes complement-mediated damage, is the cause of lupus nephritis.[7]

The 2004 update to the WHO classification of lupus nephritis was made by the Renal Pathology Society and the International Society of Nephrology.[8]

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Table 1: WHO classification

Class I	Minimal mesangial	Normal histology with mesangial deposits
Class II	Mesangial proliferation	Mesangial hypercellularity with expansion of the mesangial matrix
Class III	Focal nephritis	Focal endocapillary ± extracapillary proliferation with focal subendothelial immune deposits and mild mesangial expansion
Class IV	Diffuse nephritis	Diffuse endocapillary ± extracapillary proliferation with diffuse subendothelial immune deposits and mesangial alterations
Class V	Membranous nephritis	Thickened basement membranes with diffuse subepithelial immune deposits; may occur with class III or IV lesions and is sometimes called mixed membranous and proliferative nephritis
Class VI	Sclerotic nephritis	Global sclerosis of nearly all glomerular capillaries

Overview of Clinical Manifestations and Correlations

Patients presenting with any one lesion on renal biopsy can transform to other lesion, so these patients require timely re-evaluation in the form renal biopsy. Patients with class 6 lesion have more than 90% sclerotic glomeruli presenting as end stage renal disease patients requiring hemodialysis or renal transplant. Approximately 20% of patients presenting with lupus nephritis end up in ESRD. Renal transplantation in lupus nephritis patients with ESRD is usually performed approximately after 6 months of inactive disease to improve allograft survival. Remission in lupus nephritis is defined as return to near normal renal function with proteinuria less than or equal to 330mg/dl per day. When remission is achieved renal outcomes can be better.[9]

A 37-95% of SLE patients may manifest with central and peripheral nervous system complications. Hematological manifestations commonly seen in SLE include anemia, thrombocytopenia, and leucopenia. Anemia is found in about half of SLE patients with the most common cause being anemia of chronic disease; however, other causes include autoimmune hemolytic anemia, iron deficiency anemia, anemia of chronic renal failure, and cyclophosphamide myelotoxicity. Prolonged glucocorticoid use for immunosuppression could cause osteoporosis and Incidence of atlantoaxial subluxation has been reported which can manifest as difficult airway.[10]

The prevalence of "subclinical" lupus nephritis in the allograft, or histological results in protocol or consecutive biopsies, was significantly higher than in biopsies carried out purely on the basis of clinical indication. The application using light microscopy alone, in the analysis of biopsy specimens, most likely resulted in a noticeably decreased diagnosis rate for RLN. But it was soon realized that kidney biopsy specimens from transplant recipients who had a history of end-stage renal disease (ESRD) due to lupus also needed to be assessed using electron microscopy and immunofluorescence.[11]

The World Health Organization (WHO) or the International Society of Nephrology/Renal Pathology Society Histologic Classifications.[12] should ideally be used to thoroughly examine the biopsy and establish the diagnosis of lupus nephritis in the renal allograft. This should include a positive immunofluorescence microscopy, and/or the electron microscopic evaluation's finding of electron-dense deposits. In this regard, it was demonstrated that 30% of the patients experienced a recurrence of lupus nephritis when both immunofluorescence and electron microscopy were utilized for the examination of renal biopsies combined with a more aggressive program of graft biopsies.[13]

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Table 2: The World Health Organization (WHO) or the International Society of Nephrology/Renal Pathology Society Histologic Classifications

Class I: Minimal Mesangial Lupus Nephritis
Normal glomeruli by light microscopy, but mesangial immune deposits by immunofluorescence.
Class II: Mesangial Proliferative Lupus Nephritis
Purely mesangial hypercellularity of any degree or mesangial matrix expansion by light microscopy, with mesangial immune deposits. A few isolated subepithelial or subendothelial deposits may be visible by immunofluorescence or electron microscopy, but not by light microscopy.
Class III: Focal Lupus Nephritis
Active or inactive focal, segmental or global endo- or extracapillary glomerulonephritis involving ≤50% of all glomeruli, typically with focal subendothelial immune deposits, with or without mesangial alterations.
Class III (A): Active lesions—focal proliferative lupus nephritis
Class III (A/C): Active and chronic lesions—focal proliferative and sclerosing lupus nephritis
Class III (C): Chronic inactive lesions with glomerular scars—focal sclerosing lupus nephritis
Class IV: Diffuse Lupus Nephritis
Active or inactive diffuse, segmental or global endo- or extracapillary glomerulonephritis involving ≥50% of all glomeruli, typically with diffuse subendothelial immune deposits, with or without mesangial alterations. This class is divided into diffuse segmental (IV-S) lupus nephritis when ≥50% of the involved glomeruli have segmental lesions, and diffuse global (IV-G) lupus nephritis when ≥50% of the involved glomeruli have global lesions. Segmental is defined as a glomerular lesion that involves less than one-half of the glomerular tuft. This class includes cases with diffuse wire loop deposits but with little or no glomerular proliferation.
Class IV-S (A): Active lesions—diffuse segmental proliferative lupus nephritis
Class IV-G (A): Active lesions—diffuse global proliferative lupus nephritis
Class IV-S (A/C): Active and chronic lesions—diffuse segmental proliferative and sclerosing lupus nephritis
Class IV-G (A/C): Active and chronic lesions—diffuse global proliferative and sclerosing lupus nephritis
Class IV-S (C): Chronic inactive lesions with scars—diffuse segmental sclerosing lupus nephritis
Class IV-G (C): Chronic inactive lesions with scars—diffuse global sclerosing lupus nephritis
Class V: Membranous Lupus Nephritis
Global or segmental subepithelial immune deposits or their morphologic sequelae by light microscopy and by immunofluorescence or electron microscopy, with or without mesangial alterations. Class V lupus nephritis may occur in combination with class III or IV, in which case both will be diagnosed. Class V lupus nephritis may show advanced sclerosis.
Class VI: Advanced Sclerotic Lupus Nephritis
≥90% of glomeruli globally sclerosed without residual activity.

Risk factors Recurrent Lupus Nephritis

The fact that patients with lupus recurrence in renal grafts may have worse results than other recipients of kidney transplants makes them important to practicing nephrologists. But according to a research from the American College renal transplantation was made available to lupus patients in 1975 through the Society of Surgeons/National Institute of Health Transplant Registry, as it was found that their results were comparable to those of non-lupus patients.[14]

The following are known risk factors for allograft loss in lipoplasty patients: feminine gender, young age, and non-Hispanic black ancestry.[15] Recurrence is more common in patients with antiphospholipid autoantibodies and kidney recipients from living donors. It has been demonstrated that African American ethnicity is independently linked to RLN in the allograft and may also be related with lower survival. Additionally, African Americans' reaction to treatment was less favorable, which increased the rate at which the condition advanced to end-stage renal disease (ESRD).

Anaesthetic Management of ESRD Patient with SLE for Renal Transplantation- Standard

Pre-operative preparation includes Chest physiotherapy, Deep breathing exercises, Heparin free dialysis 12 h prior to surgery,T.wysolone 25mg OD. Induction is done BP 140/90, Fentanyl 100 µg, Ketamine 25 mg, glycopyrollate 0.2 mg, Propofol 30 mg, Atracurium 25 mg + 2 mg supplements and Maintainence includes IPPV /O2 (33%) / N20 / isoflurane / noradrenaline support standby, Fluid challenge 1 L, Good urine output following venous and arterial anastomosis with external iliac vessels, Uneventful postop

Renal replacement therapy for lupus patients with ESRD

There are very few, tiny, retrospective studies that evaluate the long-term clinical outcomes and cumulative survival of patients between dialysis modalities and renal transplantation. During the 4.5

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years of follow-up following PD, disease activity, as measured by the SLE Disease Activity Index, was considerably higher (HD: 5.0). ± 3.6, PD: 7.4 ± 3.7, RENAL TRANPLANTATION: 2.2 ± 1.7), but the three groups' survival rates were comparable. The type of renal replacement therapy that thirteen patients received was not disclosed, however infections were the primary cause of death for them during the monitoring period.[16]

However, it should be highlighted that the patients in the transplant group were comparatively younger. During the study period, there were no lupus flare-ups among transplanted patients (0%, 50%, and 14%). The HD group saw higher death rates, which were mostly linked to cardiovascular disease and malignancies. During the 10-year follow-up, just one transplanted patient passed away. The most popular renal replacement therapy at the time of the study was hemodialysis, which had a notable rise in usage from 75.9% to 83.9%. From 1995 to 2006, renal tranplantation rates significantly declined despite a rise in the number of living donors; this decline may have been attributed to the scarcity of donor organs and the low socioeconomic standing of a number of patients. However, during the course of the 12-year trial, there has been no discernible improvement in the survival of patients receiving renal tranplantation.[17,18]

Younger and significantly immunocompromised due to the underlying disease and the prolonged use of immunosuppressive medication, resulting in several comorbidities, are the patients with ESRD related to lupus nephritis. Thus, enhancing survival rates and quality of life should be the top priority when planning these patients' renal replacement therapy in the future. Lastly, as access failure from repeated thrombosis is a big issue for individuals with lupus and antiphospholipid syndrome, PD is a preferable option for starting renal replacement treatment in these patients.[19,20]

Lupus Activity in ESRD patients

There is a recognized decline in the clinical and serological activity of lupus from the time SLE patients progress to ESRD and begin dialysis. Research and extensive clinical experience have demonstrated that patients with renal failure due to lupus often see an improvement in lupus serologic results and a remission of their extra-renal symptoms with dialysis, allowing for the removal of all immunosuppressive measures. This quiescence of lupus in ESRD patients was initially reported and was dubbed "burnt-out lupus." Since the start of dialysis, numerous studies have documented a remarkable improvement in all autoimmune disorders.[20]

It was found that there was no correlation between the serological activity and clinical activity in lupus, and that the condition was more common, occurring in 80%, 60%, and 22% of patients after 1, 5, and ten years, in that order. Even though the exact reasons of this phenomena are unknown, it is frequently observed and frequently linked to the progressive or partial resolution of the extra-renal symptoms of lupus. Less commonly, and usually in patients who are Black in color, several researchers have noted that lupus activity can continue and even sometimes worsen when end-stage renal disease (ESRD) developed.[21] But if we take into account that these results pertain to different patient populations and genetic profiles, they become more understandable.

Lupus patients who get renal transplant typically worry about a return of lupus nephritis in the graft. There are discrepancies in the reported rates of recurrent lupus nephritis (RLN), which range from 0% to 44%. These variables include patient characteristics, the period of immunosuppression, and the grounds for renal biopsies. Serial biopsies or protocol biopsies take this into consideration.[22]

Furthermore, it is crucial to differentiate incident histopathological findings linked to a lupus effect in the graft from clinically apparent RLN in the allograft, which may have questionable clinical significance, from concurrent clinical, renal, or extra-renal symptoms or signs of lupus.

The setting of RLN in transplant patients generally include renal dysfunction, either an acute increase in serum creatinine, or a slow increase compared with the baseline value, or new onset proteinuria or glomerular hematuria or both.

Autoantibodies.[23,24,25]

Autoantibody testing is the hallmark of autoimmune diseases, especially SLE and LN. They form part of the diagnostic criteria and can also have prognostic utility. Serum creatinine, glomerular filtration rate, proteinuria, and hematuria are already commonly used as part of the management of LN. Combined with levels of anti-dsDNA antibodies and complements, they are good predictors of long-term renal outcome. However anti-dsDNA antibodies and complements levels are not always abnormal in LN and do not always follow disease activity .A low C4 may reflect a defect in the classical complement pathway and not SLE or LN activity. In fact, the low C4 is also a risk factor for the development of SLE and not just a marker of disease activity. Complement C1q is the first subcomponent of the classical pathway of complement activation and is involved with clearing immune complexes and self-antigens generated during apoptosis. An inherited deficiency in or low levels of C1q is associated with an increased risk of SLE and immune-mediated glomerulonephritis like LN. Autoantibodies to C1q that lower the levels of C1q levels have been shown to closely correlate with LN disease activity level. Combining the results of the anti-dsDNA and anti-C1q antibody levels enhances the diagnostic specificity and sensitivity for concurrent SLE disease activity, and the absence of both anti-dsDNA and anti-C1q has a high negative predictive value for lupus activity. A number of autoantibodies and serological tests correlate closely with renal pathology and LN disease activity. Anti-dsDNA, anti-nucleosome, anti-ribosome P, anti-C1q antibodies, and C3/C4 follow disease activity. A

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high titer of anti-C1q or anti-dsDNA antibodies can differentiate LN III and VI from LN V. Anti-C1q has demonstrated a relationship with proteinuria, and this may be higher in LN class V. Antiphospholipid antibodies (aPL) are seen in patients with SLE and LN, and it is not clear that aPL alters the outcomes of LN. However, aPL should be evaluated in all patients with SLE. Some autoantibodies like antibodies to M-type phospholipase A2 receptor (PLA2R) are used to rule out LN. The anti-PLA2R antibody is a specific marker of idiopathic membranous nephritis. There are a number of novel autoantibody tests available that have not been incorporated into routine clinical car.

Diagnosis, Management and Treatment of Lupus in ESRD patients

From systematic review of the literature perioperative management must be tailored to the individual patient. Anesthetic management plan was made after taking into account severity of the disease, drug interactions with immunosuppressants, difficult airway and coagulation profile of the patient.

Patients with SLE have a wide variety of abnormalities and the course is highly variable, ranging from relatively mild and uncomplicated to major life-threatening disease. Cardiovascular involvement could be in the form of pericarditis, arthrosclerosis, myocarditis, and the myocardial ischemia. Pulmonary involvement may present as pleuritis, alveolar hemorrhage, pleural effusion, and interstitial lung disease. Renal involvement is seen in the form of lupus nephritis characterized by proteinuria, hematuria, and abnormal urinary segments.[26]

Antiphospholipid syndrome may occur secondary to SLE and is characterized clinically by recurrent pregnancy loss and by presence of lupus anticoagulant antibodies which may falsely prolong activated partial thromboplastin time in such individuals.

The preoperative visit aims at the activity of the lupus, organ damage, medication exposure, thorough preanesthetic assessment, and laboratory test. Care of the high-risk patients requires a multidisciplinary approach.[27]

As SLE symptoms are nonspecific, the investigations become mainstay in monitoring. Complete blood count has to be done in all patients along with coagulation profile. Platelet count should be repeated every month because of high risk of thrombocytopenia in lupus patients. Electrocardiography should be done when suspecting pericarditis, myocarditis, and chest X-ray to rule out pleural effusion or interstitial pneumonitis is seen clinically. For patients with renal involvement, every month creatinine clearance and 24 h urine protein should be checked. If the patient is on steroids then, a close watch on blood glucose levels is recommended. Anticardiolipin antibody, lupus anticoagulant, anti-β2 glycoprotein should be done to rule out any secondary involvement in succeeding months.28

Monitoring during anesthesia includes five-lead ECG, noninvasive blood pressure, pulse oximetry, and invasive monitoring should be used in patients with existing myocarditis, valvular involvement, or conduction abnormalities. Renal protective strategies and maintenance of urine output, avoidance of nephrotoxic drugs are the goals during anesthesia. Adequate pain management and corticosteroid cover should be given intraoperatively to prevent adrenal suppression. Antibiotics are to be given to prevent infection. Patient should be positioned with care to avoid joint stress.

Difficult airway should be anticipated in all the patients, so smaller sized tubes, and laryngeal mask airway must be available considering the potential laryngeal and the subglottic involvement. Laryngeal involvement could present as laryngeal edema, epiglottitis, and vocal cord paralysis to acute airway obstruction. The pathophysiology of laryngeal inflammation of SLE is not well-understood although the tissue deposition of immune complexes with activation of complements is less likely the cause. Compression of recurrent laryngeal nerve by dilated pulmonary artery has been reported as the cause of left palsy in patients with SLE. Secondary nerve vasculitis is a cause especially in vocal cord palsy involving right side. There is risk of failed intubation and airway trauma during instrumentation. Pharmacological interactions between anesthetic drugs and immunosuppressant drugs should warrant consideration. Azathioprine, an antimetabolite immunosuppressor, may interact with muscle relaxants, and dose increases of 37% with cisatracurium, 20% with vecuronium, and 45% with pancuronium were required in one-study.[29]

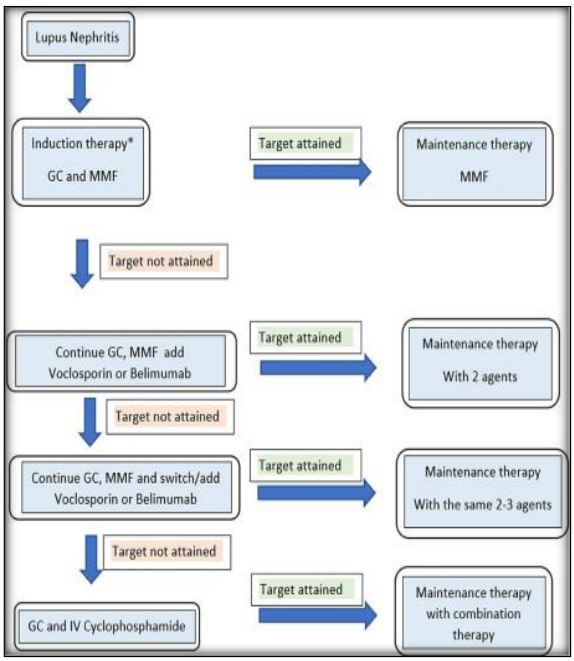

Figure 1: Treatment Algorithm of Lupus Nephritis in ESRD

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Immunosuppressive Therapy

Since patients already receive maintenance therapy for their kidney transplant, recipients of kidney transplants who experience recurrent lupus typically do not need to alter their immunosuppressive regimen. The majority exhibits minor lesions in the transplant because of lupus, as evidenced by research on surveillance biopsy.[30] According to the biopsy, the majority of the patients had subclinical illness of class IV or V. However, the great majority of patients (84%) had chronic allograft nephropathy, and they, like SLE patients without transplants, were similarly vulnerable to calcineurin inhibitor toxicity. Furthermore, the primary obstacle to achieving the objective of enhanced kidney transplant survival is a series of accumulating and developing immunological and non-immune injuries. When a calcineurin inhibitor is used to provide standard immunosuppression for kidney transplant recipients with SLE. Chronic allograft nephropathy appears to be unaffected by mycophenolate mofetil and prednisone, but not by clinically overt recurring disease.[31]

Certain patients, meanwhile, need to receive further immunosuppressive care. Patients with significant histopathologic lesions and clinically obvious disease, consistent with WHO class III or IV in the graft, comprise the majority of those with renal involvement. Among those with recurrent lupus diagnosed histologically. All additional causes linked to acute renal dysfunction in a kidney transplant recipient, such as acute rejection, chronic allograft nephropathy, and calcineurin inhibitor toxicity, should be ruled out initially in the graft in addition to the patient's rapid decline in renal function.[32]

Any lupus patient with newly developed proteinuria, progressive proteinuria, and/or hematuria along with severe. It is necessary to alter the current immunosuppressive regimen due to proliferative lesions found in the transplant biopsy. We either employ greater dosages of mycophenolate mofetil (2–3 g/d) or start cyclophosphamide intravenously along with stopping the existing antimetabolite, depending on the clinical picture and morphologic abnormalities. Glucocorticoids are always used in conjunction with either of these alternatives. Typically, methylprednisolone pulses of 500–1000 mg/d are administered for three days in a row, and these are followed by a tapering steroid regimen. When a patient presents with rapid renal decline along with a crescentic pattern in histology, or in any scenario where there is severe extra renal illness, we typically prescribe cyclophosphamide.[33]

Non-Immunosuppressive Therapy

We use renin-angiotensin system blockage to treat all patients with histopathologic alterations of RLN in the graft and protein excretion, usually greater than 0.5 g/d.The idea behind this strategy stems from research done on non-transplant patients with proteinuria and chronic kidney disease, which shown that blocking the renin-angiotensin system slows the progression of renal illness.34 Angiotensin converting enzyme inhibitors have the potential to cause anemia and hyperkalemia, although this problem is typically avoided because transplant recipients are usually well monitored and such adverse effects are easily identified. Furthermore, the long-term advantages of blocking the renin-angiotensin system exceed the potential risks.

Death with a functioning graft is a major cause of renal allograft loss in the general population. The same applies to recipients with ESRD due to lupus nephritis, and is mainly attributed to cardiovascular disease.[35]

CONCLUSION

Our review provides a thorough synopsis of the state of knowledge regarding LN and its management through renal transplantation in ESRD. It emphasizes the need of early diagnosis and treatment, the role of ongoing monitoring of therapy response, the importance of history, physical examination, and laboratory tests, as well as the. Identifying and treating LN problems can help to improve results. It describes a treatment plan that uses a variety of drugs and their MOA and talks about the more recently approved medicines. Even with the recent approval of two new treatments for LN, therapy remains challenging and can be complicated by medication intolerance, access, and occasionally compliance due to the multitude of prescriptions that may be required.

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REFERENCES

1. Systemic lupus erythematosus in patients with chronic renal failure. Am J Med 2021; 75: 602-606
2. Correia P, Cameron JS, Ogg CS, Williams DG, Bewick M, Hicks JA. End-stage renal failure in systemic lupus erythematosus with nephritis. Clin Nephrol 1204; 22: 293-302
3. Mojcik CF, Klippel JH. End-stage renal disease and systemic lupus erythematosus. Am J Med 2016; 101: 100-107
4. Cheigh JS, Stenzel KH. End-stage renal disease in systemic lupus erythematosus. Am J Kidney Dis 2013; 21: 2-8
5. Nossent HC, Swaak TJ, Berden JH. Systemic lupus erythematosus after renal transplantation: patient and graft survival and disease activity. The Dutch Working Party on Systemic Lupus Erythematosus. Ann Intern Med 2011; 114: 183-188
6. Ponticelli C, Moroni G. Renal transplantation in lupus nephritis. Lupus 2005; 14: 95-98
7. Krane NK, Burjak K, Archie M, O'donovan R. Persistent lupus activity in end-stage renal disease. Am J Kidney Dis 2019; 33: 872-879
8. Renal transplantation in congenital and metabolic diseases. A report from the ASC/NIH renal transplant registry. JAMA 2015; 232: 148-153
9. Amend WJ, Vincenti F, Feduska NJ, Salvatierra O, Johnston WH, Jackson J et al. Recurrent systemic lupus erythematosus involving renal allografts. Ann Intern Med 2011; 94: 444-448.
10. Cameron JS. Glomerulonephritis in renal transplants. Transplantation 2012; 34: 237-245

ISSN (O): 2687-5365; ISSN (P): 2753-6556

11. Coplon NS, Diskin CJ, Petersen J, Swenson RS. The longterm clinical course of systemic lupus erythematosus in endstage renal disease. N Engl J Med 2013; 308: 186-190
12. Kotanko P, Pusey CD, Levy JB. Recurrent glomerulonephritis following renal transplantation. Transplantation 2017; 63: 1045-1052
13. Nyberg G, Blohmé I, Persson H, Olausson M, Svalander C. Recurrence of SLE in transplanted kidneys: a followup transplant biopsy study. Nephrol Dial Transplant 2012; 7: 1116-1123
14. Roth D, Milgrom M, Esquenazi V, Strauss J, Zilleruelo G, Miller J et al. Renal transplantation in systemic lupus erythematosus: one center's experience. Am J Nephrol 2017; 7: 367-374
15. Contreras G, Mattiazzi A, Guerra G, Ortega LM, Tozman EC, Li H et al. Recurrence of lupus nephritis after kidney transplantation. J Am Soc Nephrol 2010; 21: 1200-1207.
16. Weng F, Goral S. Recurrence of lupus nephritis after renal transplantation: if we look for it, will we find it? Nat Clin Pract Nephrol 2005; 1: 62-63
17. Choy BY, Chan TM, Lai KN. Recurrent glomerulonephritis after kidney transplantation. Am J Transplant 2016; 6: 2535-2542
18. Burgos PI, Perkins EL, Pons-Estel GJ, Kendrick SA, Liu JM, Kendrick WT et al. Risk factors and impact of recurrent lupus nephritis in patients with systemic lupus erythematosus undergoing renal transplantation: data from a single US institution. Arthritis Rheum 2009; 60: 2757-2766
19. Stone JH, Amend WJ, Criswell LA. Outcome of renal transplantation in systemic lupus erythematosus. Semin Arthritis Rheum 2017; 27: 17-26
20. Moroni G, Tantardini F, Gallelli B, Quaglini S, Banfi G, Poli F et al.The longterm prognosis of renal transplantation in patients with lupus nephritis. Am J Kidney Dis 2005; 45: 903-911
21. Weening JJ, D'Agati VD, Schwartz MM, Seshan SV, Alpers CE, Appel GB et al. The classification of glomerulonephritis in systemic lupus erythematosus revisited. J Am Soc Nephrol 2004; 15: 241-250
22. Norby GE, Strøm EH, Midtvedt K, Hartmann A, Gilboe IM, Leivestad T et al. Recurrent lupus nephritis after kidney transplantation: a surveillance biopsy study. Ann Rheum Dis 2010; 69: 1484-1487
23. Herrera GA. The value of electron microscopy in the diagnosis and clinical management of lupus nephritis. Ultrastruct Pathol 2019; 23: 63-77
24. Lochhead KM, Pirsch JD, D'Alessandro AM, Knechtle SJ, Kalayoglu M, Sollinger HW, Belzer FO. Risk factors for renal allograft loss in patients with systemic lupus erythematosus. Kidney Int 2016; 49: 512-517
25. Ugolini-Lopes MR, Seguro LP, Castro MX, Daffre D, Lopes AC, Borba EF et al. Early proteinuria response: a valid real-life situation predictor of long-term lupus renal outcome in an ethnically diverse group with severe biopsy-proven nephritis?. Lupus Sci Med. 2017, 4:e000213.
26. Enocsson H, Sjöwall C, Wirestam L.: Four anti-dsDNA antibody assays in relation to systemic lupus erythematosus disease specificity and activity. J Rheumatol. 2015, 42:817-25.
27. Walport M, Lachmann P: Complement deficiencies and abnormalities of the complement system in systemic lupus erythematosus and related disorders. Curr Opin Rheumatol. 1990, 2:661-3.
28. Botto M, Dell'Agnola C, Bygrave AE, et al.: Homozygous C1q deficiency causes glomerulonephritis associated with multiple apoptotic bodies. Nat Genet. 1998, 19:56-9.
29. Marto N, Bertolaccini ML, Calabuig E, Hughes GR, Khamashta MA: Anti-C1q antibodies in nephritis: correlation between titres and renal disease activity and positive predictive value in systemic lupus erythematosus. Ann Rheum Dis. 2005, 64:444-8.
30. Yin Y, Wu X, Shan G, Zhang X: Diagnostic value of serum anti-C1q antibodies in patients with lupus nephritis: a meta-analysis. Lupus. 2012, 21:1088-97.
31. Yang XW, Tan Y, Yu F, Zhao MH: Combination of anti-C1q and anti-dsDNA antibodies is associated with higher renal disease activity and predicts renal prognosis of patients with lupus nephritis. Nephrol Dial Transplant. 2012, 27:3552-9.
32. Moroni G, Quaglini S, Radice A, Trezzi B, Raffiotta F, Messa P et al. The value of a panel of autoantibodies for predicting the activity of lupus nephritis at time of renal biopsy. J Immunol Res. 2015, 2015:106904.
33. Parodis I, Arnaud L, Gerhardsson J. Antiphospholipid antibodies in lupus nephritis. PLoS One. 2016, 11:e0158076.
34. Beck LH Jr, Bonegio RG, Lambeau G. M-type phospholipase A2 receptor as target antigen in idiopathic membranous nephropathy. N Engl J Med. 2009, 361:11-21.
35. Hahn BH, McMahon MA, Wilkinson A, et al.: American College of Rheumatology guidelines for screening, treatment, and management of lupus nephritis. Arthritis Care Res (Hoboken). 2012, 64:797-808.

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