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Short Review

Utility of MEST and MEST-C Scoring in IgA Nephropathy in Kidney Transplantation: A Mini Review

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Abstract

IgAN is a major cause of end-stage kidney disease (ESKD) leading to kidney transplantation in a significant proportion of patients. However, its recurrence in transplanted kidneys can lead to graft loss. The rate of graft loss attributable to IgAN after transplantation is variably reported in different retrospective cohorts. Previous reports describe recurrence rates of 22-58% with a 1.3% to 16% rate of graft loss. Accurate diagnosis and prediction of graft loss are important for planning effective therapies to improve graft survival in IgAN post transplantation. The Oxford classification using MEST and MEST-C in native kidney disease IgAN has been established for well over a decade. We propose investigating if this classification system can be applied to kidney allografts to standardize the categorization of transplant IgAN. More importantly, successful use of this classification could assist in selecting patients for prospective interventional trials and defining better treatments. In this literature review, we explore the available literature on the Oxford classification and its utility in

describing the disease and predicting graft loss in IgA nephropathy within the context of kidney transplantation.

Keywords
IgA nephropathy; kidney transplant; Oxford score; MEST score; MEST-C score; graft survival

1. Introduction

IgA Nephropathy (IgAN), first described by Berger & Hinglais in 1968 [1], has an incompletely understood pathogenesis. An upstream effect leading to deposition of IgA1-containing immune complexes in the glomerular mesangium is the most accepted theory of IgAN pathogenesis currently. Mesangial IgA deposition drives cellular proliferation, matrix overproduction and synthesis of cytokines and chemokines leading to glomerular injury [2].

Mesangial IgA deposition is accompanied by heterogenous pathological response leading to diverse clinical presentations & clinical course among patients. However, the presence of dominant or co-dominant IgA deposits in the glomerular mesangium on kidney biopsy is the only accepted criteria for diagnosis [3]. IgAN shows a range of clinical manifestations from asymptomatic microscopic hematuria to rapidly progressive glomerulonephritis. However, the two most common clinical presentations are asymptomatic hematuria and progressive kidney disease [4].

Around the world, IgA nephropathy is a commonly reported glomerulonephritis (GN) showing a variable geographical distribution with a higher incidence in Asian population [5]. Overall, the population incidence of IgAN is nearly 2.5 per 100,000 [6]. IgAN is responsible for end-stage kidney disease (ESKD) in 25-30% of patients of all ages within 20 years after diagnosis [7]. As IgAN usually develops in younger patients with less comorbidities, patients with IgAN are generally considered better candidates for renal transplantation compared with other causes of ESKD [8, 9]. Although recurrent GN was previously considered a minor cause of graft loss, with advances in immunosuppressive therapy leading to prolongation of graft survival, recurrent GN is currently considered the third most frequent cause of graft loss [10].

IgAN was reported by some authors as the most common de novo or recurrent nephropathy, especially in living donor transplantation [11-13]. The recurrence of IgAN varies in different reports from 22 to 58%, with a 1.3% to 16% rate of graft loss attributable to IgAN recurrence [14-18]. In a Canadian cohort, de novo IgAN was found to be one of the most common de-novo GN (27%) in transplanted kidney in patients whose ESKD was attributed to non-GN causes [19]. Several studies reported different risk factors for IgAN recurrence including immunologically active native IgA nephropathy (represented by earlier age of onset and greater burden of crescents on native biopsy) [20], younger age at transplant [11, 14, 18, 21], transplant without induction agent [22], higher HLA-mismatch [16], steroid-free sirolimus-based immunosuppression without antilymphocyte globulin induction [23] , living related donor [24], pre-emptive transplantation, preformed DSA, and development of dnDSA after kidney transplantation [25]. Other factors that might be associated with lower IgAN recurrence have also been described; including older age of patients, any triple immunosuppressive therapy [14] & preoperative desensitization [26].

Some of the above-mentioned factors have failed to consistently show an association with IgA nephropathy recurrence in different studies e.g., the benefits of steroid maintenance in preventing the recurrence although was suggested by some studies [23, 27, 28], other studies didn't show any benefits of steroid maintenance [21, 24, 29] or any association between early steroid withdrawal and IgA nephropathy recurrence [16, 25].

As regard the predictors of outcomes in patients with recurrent IgAN, some studies demonstrated an association between clinical presentation at time of recurrence and worse prognosis**.** Uffing et al [25] recently reported unfavorable outcome in patients presenting with proteinuria at the time of recurrence. Kavanagh et al [21] found that serum creatinine and concurrent acute rejection were significant predictors for allograft failure. while Maixnerova et al [30] have reported worse ten-year renal survival in patients presenting with microscopic hematuria.

Specific histological features in kidney biopsy used in Oxford classification of native kidney IgAN help to predict individual patient's risk of progression of kidney disease. It employs four histological features namely - *M*esangial hypercellularity, *E*ndocapillary hypercellularity, *S*egmental glomerulosclerosis, and *T*ubular atrophy/interstitial fibrosis in the cortical area (MEST) [31]. This was subsequently updated to include cellular or fibro-cellular crescent formation (C) leading to MEST-C score [32]. Several studies validated the utility of Oxford classification in predicting renal prognosis in native kidney disease in different populations, including ethnicity, presentations and treatments [33-36].

Predicting graft loss is essential in planning interventions that could improve graft survival in IgAN post transplantation. For this, validated criteria will be of help in determining role of interventions in therapy of Transplant IgAN. It is unclear if there are differences in presentation and histological features in native IgAN and transplant IgAN. Here, we discuss the current evidence for the use of MEST±C score in prediction of graft survival in IgAN in transplant kidneys and discuss their potential for future use in clinical care and their role in interventions.

2. Oxford MEST/MEST-C Score: Prognostic Utility in Transplant IgAN

Both recurrent and de novo IgAN can lead to graft dysfunction in allograft recipients [10, 13, 27]. Occurrence of IgAN recurrence in transplanted kidneys is time-dependent [15, 37]. Nearly 13-25% of patients exhibit some recurrence-related graft dysfunction at 5 years, which may lead to graft loss in nearly 5-10% of cases [10, 38]. The Australia and New Zealand Dialysis and Transplant registry data (ANZDATA) demonstrated recurrence of IgAN in 5.1%, 10.1% and 15% of transplant patients at 5-, 10- and 15-years post-transplant respectively [39]. Recurrence may be determined by both clinical and histological factors, treatment-related factors as well as donor factors. In recent years, some authors suggest that graft loss due to IgAN recurrence has decreased due to changes in immunosuppressive protocols [14, 27]. Early reports of graft loss with IgAN recurrence in transplanted kidneys observed presence of crescents on histological examinations to be associated with graft loss [40-42]. MEST scoring has been widely used in the clinical domain after its first publication in 2009 [31].

Table 1 lists the studies assessing MEST-C scoring in IgAN in allografts, reported till date, with respective baseline characteristics. Table 2 outlines the outcomes and their association with MEST and MEST-C characteristics in each study.

Table 1 Comparison between different studies that assessed the MEST-C score in allograft IgA Nephropathy - Baseline features.

Characteristic	Moroni, 2013 [14]	Lim, 2013 [43]	Agrawal, 2017 [44]	Park, 2019 [45]	Cazorla López, 2020 [46]
Study type	Retrospective (1981-2010)	Retrospective (1992-2006)	Retrospective (2005-2014)	Retrospective (1979-2016) (1990-2016)	Retrospective (1990-2018)
Study setting	Single centre/Italy	Single centre/Korea	Single centre/India	Bi centre/Korea	Single centre/Spain
Study population	190 confirmed native IgA 380 non-diabetic controls	125 renal allograft biopsies (114 patients diagnosed with allograft IgAN regardless of original disease)	22 patients (27 biopsies) diagnosed with allograft IgAN	333 patients *100 with confirmed native IgA *233 patients with unknown cause of ESKD	24 patients
Number of patients	190	114	22	333	24
Males	149	93	19	224 (67.3%)	20
Age	42.5 (33.6-51.4) in rIgAN vs 42.3 (32.9-51.4) in others	30.3 (7.9) in rIgAN vs 35.7 (9.2) in others	31 (27-42)	38 (30-46)	40.3 (8.7)
Living donor kidneys	36/154 in rIgAN vs 72/308 in others	112	22	267	1

Indications for allograft biopsy	Acute episode of renal dysfunction of doubtful origin, persistent proteinuria >0.5 g/day or persistent microscopic haematuria of non-urological origin	Elevated creatinine 37.8% Increased proteinuria 40.5% Persistent microscopic haematuria (21.6%)	No details, but biopsy was done for 'indication'	persistent haematuria, significant proteinuria (>1.0 g/day), or progressive deterioration in graft function Some patients received protocol biopsies	NA
HLA match or mismatch	Mismatch 3 (2-3)	2 haplotype match 4% in recurrent IgAN vs 31	72.7% (16/22) - rise in serum creatinine 91% (20/22) - proteinuria 31.8% (7/22) - haematuria	1-3 mismatch 101 (35.1%) 4-6 mismatch 154 (53.5.5)	0.8 (0.4)
Serum creatinine at biopsy(mg/dL)	NA	2.35 (1.88) in rIgAN vs 1.73 (0.77) in others	1.22 (1.07-1.4)	1.7 (1.4-2.2)	1.8 ± 0.6
Proteinuria at biopsy	NA	1885 (2188) in recurrent IgAN vs 1629 (2156) in others (mg/24 hour)	1.9 (0.95-2.65) * (g/24 hour)	Dipstick albuminuria 1+ 36 (10.9%) ≥2+ 122 (37.1%)	979 (443-1980.5) (mg/24 hour)

* 3 patients did not have urine protein quantification, rIgAN - Recurrent IgA Nephropaghy, NA - not available, where available results represent mean (±SD) or median (IQR) is.

Table 2 Comparison between different studies that assessed the MEST-C score in allograft IgA Nephropathy - Outcomes and Association of MEST/MEST-C with outcomes.

	Moroni, 2013 [14]	**Lim, 2013 [37]**	**Agrawal, 2017 [38]**	**Park, 2019 [39]**		**Cazorla-López, 2020 [46]**

Graft survival	*15-year DCGS*
	• 62.6% in IgAN group
	  ○ 68.3% non-recurrent
	  ○ 51.2% recurrent IgAN
	• 72.4% in controls
		• 10-year graft survival 62.9%.
		• 15-year graft survival 34.3%.
			• 2-year graft survival rates 75%.
			• 5-year graft survival rates 56%.
				10-year DCGF differed significantly according to the presence of the MEST-C score components		5-year DCGS 95.2%

IgAN recurrence	42 allograft IgAN (22.1%)	N/A	N/A	N/A		N/A
Haas classification	N/A	III (28.8%) I (27.2%) V (22.4%) II (15.2%) IV (6.4%)	III (29.1%) IV (20.8%) I (20.8%)	N/A		N/A
	*Excluding 4 patients died with a functioning graft	*Glomeruli showed normal histology in 18.4%		Graft IgAN with confirmed native IgAN	Graft IgAN without confirmed native IgAN	
M	44.7%	12.8%	36.6%	22%	23.6%	57%
E	60.5%	6.4%	22.7%	39%	25.8%	48%
S	55.2%	45.6%	54.5%	38%	47.2%	52%
T	28.94%	20.8%	31.8%	24%	34.3%	48%

Crescents	20.9%	12%	0%	16%	14.6%	3/24
Comments	The presence of crescents at graft biopsy in recurrent IgAN patients is associated with a worse graft outcome.	• E, S, T predicted graft survival. • E and T predicted serum creatinine doubling. • The presence of at least one crescent correlated significantly with proteinuria. • There were significant correlations between Oxford-MEST score and GIS or Haas subclass	• High serum creatinine, low estimated glomerular filtration rate, E1 and T lesions, and degree of interstitial inflammation predicted graft survival. • The Oxford MEST scheme is useful in predicting graft survival in post-transplant IgAN. • The degree of interstitial inflammation is also an important feature for determining graft outcomes in post-transplant IgAN.	• M, E, S, and C were significantly associated with the prognosis of recurred IgAN, and T was the only independent prognostic parameter for allograft IgAN without confirmed native IgAN. • Presence of multiple MEST-C components was associated with worse outcomes		The presence of crescents is an indicator of poor prognosis. Patients with post- RT IgAN had received less induction immunosuppressive.

Limitations	Retrospective nature. Non-uniform immunosuppressive regimen due to the long duration of the observation & lack of control biopsy.	Acute rejection and acute tubular injury are still the major concerns influencing graft dysfunction, which cannot be assessed by the Oxford classification.	Limited study size	Selection bias (A number of patients were excluded owing to slide availability) Distinguishing between recurrent IgAN, de novo IgAN, and donor driven IgAN was not conclusive.	Limited study size

DCGF: death-censored graft failure, M = *M*esangial hypercellularity, E = *E*ndocapillary hypercellularity, S = *S*egmental glomerulosclerosis, and T = *T*ubular atrophy/interstitial fibrosis in the cortical area.

In one of the early studies, Moroni et al [14] reported in 2013, a retrospective evaluation of 190 IgAN patients and compared the renal transplant outcomes with 380 non-diabetic controls. During a 15-year follow up, death-censored graft survival (DCGS) was 62.6% in IgAN group and 72.4% in controls (p = 0.038). IgAN recurred in 22.1% grafts. The 15-year DCGS was 68.3% and 51.2% in non-recurrent and recurrent IgAN respectively (p = 0.069). Graft survival of non-recurrent IgAN patients was similar to that of controls (p = 0.406).

In a similar study, Lim et al [43] assessed 125 allograft biopsies from 114 patients diagnosed with IgAN irrespective of the native disease. Graft survival at 10- and 15-years was observed in 62.9% and 34.3%, respectively. Endocapillary hypercellularity, segmental sclerosis, and tubulointerstitial fibrosis were significant predictors of graft survival. S1 and T1-2 were correlated with elevated serum creatinine level, proteinuria, and decreased estimated glomerular filtration rate, and E1 was correlated with decreased estimated glomerular filtration rate at the time of biopsy. Also, the correlation between Oxford-MEST scores and GIS or Haas subclass was significant. The prognostic value of Haas classification [47] was found to be comparable to Oxford classification in native kidney disease by Park et al [48] and lower than that of Oxford classification in native kidney disease by Duan et al [49].

In a study from India, Agrawal et al [44] observed 27 biopsies from 22 patients with post-transplant IgAN. The 2- to 5-year graft survival rates were 75% and 56%, respectively. Recurrent disease occurred mostly between 4 and 8 years after transplant. The mean duration of follow-up was 75.3 +/- 64 months (range, 4-116; median, 25.7). Eight patients had graft failure, with a mean follow-up duration of 20 +/- 18 months (range 2-48 months). These patients had high urinary protein levels at follow-up. Two patients with associated chronic active antibody-mediated rejection at biopsy were dialysis-dependent within 2 months of biopsy. Predictors of graft survival included elevated serum creatinine levels, E and T lesions, and degree of interstitial inflammation. Also, the authors observed that the percentage (>25%) of segmentally sclerosed glomeruli and not S correlated with graft outcome. There was no significant correlation of Oxford MEST score M1 and S1 with raised serum creatinine, low estimated glomerular filtration rate (eGFR), and nephrotic proteinuria. E1 and T score correlated with high serum creatinine levels and low eGFR at presentation. S1 correlated with raised mean arterial pressure.

Park et al [45]**,** in a more recent retrospective cohort study, determined the MEST-C scores of the 333 recipients diagnosed with allograft IgAN (100 with known IgAN in native kidneys +233 with other or unknown primary causes of ESKD). The 10-year death-censored graft failure (DCGF) outcome differed significantly according to the presence of the MEST-C score components. MEST-C score predicted graft failure. The presence of multiple MEST-C components was associated with worse outcomes. M, E, S, and C were significantly associated with the prognosis of recurrent IgAN, and T was the only independent prognostic parameter for allograft IgAN without confirmed native IgAN. In another retrospective study, Park et al [48] assessed 10-year DCGF since the establishment of allograft IgAN diagnosis. In patients with allograft IgAN, 88 (15.9%) had glomerular crescents, including 40 patients (7.2%) with >10% crescent formation in the total biopsied glomeruli. All MEST-C components had a significant association with the graft outcomes. The presence of glomerular crescents in IgAN was associated with a worse graft prognosis, and the association was valid with the C scores of Oxford classification.

In a recent study, Cazorla-López et al [46] retrospectively assessed 24 patients who developed IgAN in the renal graft. Time from transplant to development of IgAN was 7 ± 5.3 years. In total,

seven patients lost the graft. In comparison to patients who had functioning graft at the end of 11 ± 6.4 years of follow-up, three patients who lost the graft had crescents in transplant biopsy. No differences were observed for any other histological characteristics of MEST-C except crescents. These pieces of evidence indicate MEST and MEST-C scores have potential utility in predicting graft survival in post-transplant IgAN.

On the other hand, the study by Kavanagh et al [21] was unable to establish this negative impact of MEST-C scores on allograft survival of 282 transplanted patients with failure secondary to IgAN including 80 with recurrent IgAN and 202 without recurrence. However, the authors used combined MEST-C score in the multivariate analysis and the sample size was smaller compared to other studies.

3. Primary CKD Etiology, lgAN Recurrence and Graft Loss: Is Oxford Classification Score Still Useful?

As etiologies of CKD are varied and may be unknown in several transplant recipients, development of IgAN in transplanted graft (in those with absence of confirmed IgAN as primary disease) may pose some difficulty in extrapolating the Oxford classification for prediction of graft survival. Among the studies discussed above, three studies – Moroni et al [14], Park et al [48] and Park et al [45], included patients who had biopsy proven IgAN as primary cause of ESKD. However, other studies included patients with varied etiologies of CKD. Lim et al [43] included patients with IgAN in transplanted graft irrespective of the original CKD etiology. Agrawal et al [44] also observed varied etiologies of CKD with chronic glomerulonephritis (CGN) being frequent (16/22 patients) and primary IgAN being confirmed in only three cases. Cazorla-López et al [46] observed CKD etiology in native kidneys being unknown (41.6%) in majority of patients followed by IgAN, RPGN and FSGS.

These observations are of great interest, but studies are limited by variable patient cohorts, some with small numbers from single or bi-centres and equally importantly the retrospective nature. Graft survival after diagnosis of IgAN in transplant kidneys irrespective of baseline CKD etiology is an area of further research in establishing the utility of Oxford classification. Lastly, acute rejection and acute tubular injury which are other major concerns influencing graft dysfunction cannot be assessed with MEST score. Validation of these findings in further studies with larger and prospective cohorts are warranted to firmly establish the role of the Oxford classification for predicting graft survival in post-transplant IgAN.

4. Conclusions

IgA nephropathy after kidney transplantation is a significant risk factor for graft loss. Evidence is indicative of significant association of MEST/MEST-C criteria, especially crescents with subsequent graft loss. Inclusion of these features in biopsies of posttransplant IgAN cases appears desirable. However, larger, multi-centre and prospective studies of IgAN in allograft recipients are required to establish the utility of Oxford MEST/MEST-C score in kidney transplantation. This is equally important in cohorts with confirmed IgAN as primary kidney disease as well as those with other primary diseases. Such studies will be necessary for better disease stratification for embedding use of MEST-C scoring in clinical practice and will be indispensable in paving the way for therapeutic interventions that will ultimately help in improving outcomes in Transplant IgAN.

Author Contributions

AL was responsible for performing the literature review, reviewing and finalizing the manuscript. JJ assisted in designing the review, performing the literature review, and drafting the manuscript. DAK conceptualized the review, defined the scope, designed the review, and finalized the manuscript.

Competing Interests

The authors have declared that no competing interests exist.

References

1. Berger J. Intercapillary deposits of IgA-IgG, French. J Urol Nephrol. 1968; 74: 694-695.
2. Suzuki H, Kiryluk K, Novak J, Moldoveanu Z, Herr AB, Renfrow MB, et al. The pathophysiology of IgA nephropathy. J Am Soc Nephrol. 2011; 22: 1795-1803.
3. Yeo SC, Cheung CK, Barratt J. New insights into the pathogenesis of IgA nephropathy. Pediatr Nephrol. 2018; 33: 763-777.
4. Rodrigues JC, Haas M, Reich HN. IgA nephropathy. Clin J Am Soc Nephrol. 2017; 12: 677-686.
5. Utsunomiya Y, Koda T, Kado T, Okada S, Hayashi A, Kanzaki S, et al. Incidence of pediatric IgA nephropathy. Pediatr Nephrol. 2003; 18: 511-515.
6. McGrogan A, Franssen CF, de Vries CS. The incidence of primary glomerulonephritis worldwide: A systematic review of the literature. Nephrol Dial Transplant. 2011; 26: 414-430.
7. Barratt J, Feehally J. Treatment of IgA nephropathy. Kidney Int. 2006; 69: 1934-1938.
8. O'Shaughnessy MM, Montez Rath ME, Lafayette RA, Winkelmayer WC. Patient characteristics and outcomes by GN subtype in ESRD. Clin J Am Soc Nephrol. 2015; 10: 1170-1178.
9. Saran R, Robinson B, Abbott KC, Bragg Gresham J, Chen X, Gipson D, et al. US renal data system 2019 annual data report: Epidemiology of kidney disease in the United States. Am J Kidney Dis. 2020; 75: a6-a7.
10. Briganti EM, Russ GR, McNeil JJ, Atkins RC, Chadban SJ. Risk of renal allograft loss from recurrent glomerulonephritis. N Engl J Med. 2002; 347: 103-109.
11. Ponticelli C, Glassock RJ. Posttransplant recurrence of primary glomerulonephritis. Clin J Am Soc Nephrol. 2010; 5: 2363-2372.
12. McDonald SP, Russ GR. Recurrence of IgA nephropathy among renal allograft recipients from living donors is greater among those with zero HLA mismatches. Transplantation. 2006; 82: 759-762.
13. Zhang J, Chen GD, Qiu J, Liu GC, Chen LZ, Fu K, et al. Graft failure of IgA nephropathy in renal allografts following living donor transplantation: Predictive factor analysis of 102 biopsies. BMC Nephrol. 2019; 20: 446.
14. Moroni G, Longhi S, Quaglini S, Gallelli B, Banfi G, Montagnino G, et al. The long-term outcome of renal transplantation of IgA nephropathy and the impact of recurrence on graft survival. Nephrol Dial Transplant. 2013; 28: 1305-1314.
15. Odum J, Peh CA, Clarkson AR, Bannister KM, Seymour AE, Gillis D, et al. Recurrent mesangial IgA nephritis following renal transplantation. Nephrol Dial Transplant. 1994; 9: 309-312.

16. Ortiz F, Gelpi R, Koskinen P, Manonelles A, Räisänen Sokolowski A, Carrera M, et al. IgA nephropathy recurs early in the graft when assessed by protocol biopsy. Nephrol Dial Transplant. 2012; 27: 2553-2558.
17. Cosio FG, Cattran DC. Recent advances in our understanding of recurrent primary glomerulonephritis after kidney transplantation. Kidney Int. 2017; 91: 304-314.
18. Jäger C, Stampf S, Molyneux K, Barratt J, Golshayan D, Hadaya K, et al. Recurrence of IgA nephropathy after kidney transplantation: Experience from the Swiss transplant cohort study. BMC Nephrol. 2022; 23: 178.
19. Chailimpamontree W, Dmitrienko S, Li G, Balshaw R, Magil A, Shapiro RJ, et al. Probability, predictors, and prognosis of posttransplantation glomerulonephritis. J Am Soc Nephrol. 2009; 20: 843-851.
20. Avasare RS, Rosenstiel PE, Zaky ZS, Tsapepas DS, Appel GB, Markowitz GS, et al. Predicting post-transplant recurrence of IgA nephropathy: The importance of crescents. Am J Nephrol. 2017; 45: 99-106.
21. Kavanagh CR, Zanoni F, Leal R, Jain NG, Stack MN, Vasilescu ER, et al. Clinical predictors and prognosis of recurrent IgA nephropathy in the kidney allograft. Glomerular Dis. 2022; 2: 42-53.
22. Berthoux F, El Deeb S, Mariat C, Diconne E, Laurent B, Thibaudin L. Antithymocyte globulin (ATG) induction therapy and disease recurrence in renal transplant recipients with primary IgA nephropathy. Transplantation. 2008; 85: 1505-1507.
23. Von Visger JR, Gunay Y, Andreoni KA, Bhatt UY, Nori US, Pesavento TE, et al. The risk of recurrent IgA nephropathy in a steroid-free protocol and other modifying immunosuppression. Clin Dis. 2014; 28: 845-854.
24. Nijim S, Vujjini V, Alasfar S, Luo X, Orandi B, Delp C, et al. Recurrent IgA nephropathy after kidney transplantation. Transplant Proc. 2016; 48: 2689-2694.
25. Uffing A, Pérez Saéz MJ, Jouve T, Bugnazet M, Malvezzi P, Muhsin SA, et al. Recurrence of IgA nephropathy after kidney transplantation in adults. Clin J Am Soc Nephrol. 2021; 16: 1247-1255.
26. Sato Y, Ishida H, Shimizu T, Tanabe K. Evaluation of tonsillectomy before kidney transplantation in patients with IgA nephropathy. Transplant Immunol. 2014; 30: 12-17.
27. Clayton P, McDonald S, Chadban S. Steroids and recurrent IgA nephropathy after kidney transplantation. Am J Transplant. 2011; 11:1645-1649.
28. Di Vico MC, Messina M, Fop F, Barreca A, Segoloni GP, Biancone L. Recurrent IgA nephropathy after renal transplantation and steroid withdrawal. Clin Transplant. 2018; 32: e13207.
29. Ahn S, Min SI, Min SK, Ha IS, Kang HG, Kim YS, et al. Different recurrence rates between pediatric and adult renal transplant for immunoglobulin a nephropathy: Predictors of posttransplant recurrence. Exp Clin Transplant. 2015; 13: 227-232.
30. Maixnerova D, Hruba P, Neprasova M, Bednarova K, Slatinska J, Suchanek M, et al. Outcome of 313 Czech patients with IgA nephropathy after renal transplantation. Front Immunol. 2021; 12: 726215.
31. Cattran DC, Coppo R, Cook HT, Feehally J, Roberts IS, Troyanov S, et al. The Oxford classification of IgA nephropathy: Rationale, clinicopathological correlations, and classification. Kidney Int. 2009; 76: 534-545.
32. Trimarchi H, Barratt J, Cattran DC, Cook HT, Coppo R, Haas M, et al. Oxford classification of IgA nephropathy 2016: An update from the IgA nephropathy classification working group. Kidney Int. 2017; 91: 1014-1021.

33. Lv J, Shi S, Xu D, Zhang H, Troyanov S, Cattran DC, et al. Evaluation of the Oxford Classification of IgA nephropathy: A systematic review and meta-analysis. Am J Kidney Dis. 2013; 62: 891-899.
34. Herzenberg AM, Fogo AB, Reich HN, Troyanov S, Bavbek N, Massat AE, et al. Validation of the Oxford classification of IgA nephropathy. Kidney Int. 2011; 80: 310-317.
35. Coppo R, Troyanov S, Bellur S, Cattran D, Cook HT, Feehally J, et al. Validation of the Oxford classification of IgA nephropathy in cohorts with different presentations and treatments. Kidney Int. 2014; 86: 828-836.
36. Roberts IS. Oxford classification of immunoglobulin a nephropathy: An update. Curr Opin Nephrol Hypertens. 2013; 22: 281-286.
37. Park S, Baek CH, Cho H, Yu MY, Kim YC, Go H, et al. Glomerular crescents are associated with worse graft outcome in allograft IgA nephropathy. Am J Transplant. 2019; 19: 145-155.
38. Floege J. Recurrent IgA nephropathy after renal transplantation. Semin Nephrol. 2004; 24: 287-291.
39. Allen PJ, Chadban SJ, Craig JC, Lim WH, Allen RD, Clayton PA, et al. Recurrent glomerulonephritis after kidney transplantation: Risk factors and allograft outcomes. Kidney Int. 2017; 92: 461-469.
40. Benabdallah L, Rerolle JP, Peraldi MN, Noël LH, Bruneel MF, Carron PL, et al. An unusual recurrence of crescentic nephritis after renal transplantation for IgA nephropathy. Am J Kidney Dis. 2002; 40: e20.1-e20.4.
41. Jeong HJ, Kim YS, Kwon KH, Kim SI, Kim MS, Choi KH, et al. Glomerular crescents are responsible for chronic graft dysfunction in post-transplant IgA nephropathy. Pathol Int. 2004; 54: 837-842.
42. Díaz Tejeiro R, Maduell F, Diez J, Esparza N, Errasti P, Purroy A, et al. Loss of renal graft due to recurrent IgA nephropathy with rapidly progressive course: An unusual clinical evolution. Nephron. 1990; 54: 341-343.
43. Lim BJ, Joo DJ, Kim MS, Kim YS, Kim SI, Kim Y, et al. Usefulness of Oxford classification in assessing immunoglobulin A nephropathy after transplantation. Transplantation. 2013; 95: 1491-1497.
44. Agrawal V, Singh A, Kaul A, Verma R, Jain M, Pandey R. Utility of Oxford classification in post-transplant immunoglobulin a nephropathy. Transplant Proc. 2017; 49: 2274-2279.
45. Park S, Go H, Baek CH, Kim YH, Kim YC, Yang SH, et al. Clinical importance of the updated Oxford classification in allograft IgA nephropathy. Am J Transplant. 2019; 19: 2855-2864.
46. Cazorla López JM, Wu J, Villanego Fernández F, Naranjo Muñoz J, Vigara Sánchez LA, García García Doncel A, et al. IgA nephropathy after renal transplant: Recurrences and de novo cases. Transplant Proc. 2020; 52: 515-518.
47. Haas M. Histologic subclassification of IgA nephropathy: A clinicopathologic study of 244 cases. Am J Kidney Dis. 1997; 29: 829-842.
48. Park KS, Han SH, Kie JH, Nam KH, Lee MJ, Lim BJ, et al. Comparison of the Haas and the Oxford classifications for prediction of renal outcome in patients with IgA nephropathy. Hum Pathol. 2014; 45: 236-243.
49. Duan SW, Mei Y, Liu J, Chen P, Li P, Chen YZ, et al. Predictive capabilities of three widely used pathology classification systems and a simplified classification (Beijing classification) in primary IgA nephropathy. Kidney Blood Press Res. 2019; 44: 928-941.