Minireview

Recurrent Glomerulonephritis After Kidney Transplantation

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Thirty to fifty percent of kidney transplant recipients have glomerular diseases as the underlying causes of end-stage renal failure. While recurrence of glomerulonephritis is an important cause of late renal allograft failure, the risk factors for recurrence are largely unknown or imprecise and prediction remains difficult. Recurrent disease usually presents with similar manifestations as the native disease. With regard to treatment of recurrent glomerular disease in the renal allograft, plasma exchange may be effective in reducing proteinuria in patients with early recurrence of focal and segmental glomerulosclerosis, but immunosuppressive therapy is generally ineffective in the prevention or treatment of recurrent disease. General supportive measures including strict blood pressure control and inhibition or blockade of the rennin-angiotensin pathway are helpful in retarding the rate of deterioration in renal allograft function. Despite the risk of recurrence, kidney transplantation following primary glomerulonephritides enjoys graft and patient survival rates comparable to other causes of end-stage renal failure. With a few exceptions, living related renal transplantation is not contraindicated in view of the favorable outcome and the donor shortage. This review discusses commonly encountered recurrent glomerulonephritides, with special emphasis on the influence of post-transplant prophylactic immunosuppression and emerging treatments.

Key words: Glomerulonephritis, recurrence, renal transplant, treatment

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Introduction

Glomerulonephritis is the underlying cause of end-stage renal failure in 30–50% of kidney transplant recipients (1). These patients are at risk of the recurrence of their original diseases. Recurrent glomerulonephritis was previously considered to be a minor contributor to graft loss. Introduction of newer immunosuppressive agents have reduced graft loss directly by decreasing the incidence of acute rejection and indirectly through the consequent reduction of chronic allograft nephropathy (1,2). With the prolongation of graft survival, the effect of recurrent disease on graft outcome assumes increasing importance. Studies on recurrent disease are difficult since not all patients have undergone native kidney biopsy and most centers perform graft biopsies only when there are abnormal clinical or laboratory features. The reported incidence of recurrent disease is thus influenced by prevailing clinical practice and could over- or underestimate the true occurrence. In this regard, it may be impossible to differentiate between de novo and recurrent disease. Accurate dissection of the contribution by recurrent disease toward graft dysfunction is also difficult in view of the often concomitant histological features of chronic allograft nephropathy or chronic nephrotoxicity due to calcineurin inhibitors. Many a time, full evaluation of biopsy specimen with combination of light microscopy, immunofluorescence and immunohistochemical studies and electron microscopy is needed to delineate different pathologies that coexist in the same patient. Despite these difficulties, there is accumulating evidence that recurrent glomerulonephritis is an important cause of graft loss in the long-term follow-up of renal allograft recipients (1,3,4).

The latest registry study, reported by Briganti et al. on 1505 patients with both native and graft biopsies, showed that graft loss due to recurrent glomerulonephritis was the third most frequent cause for graft loss 10 years after kidney transplantation. The risk of graft loss from recurrence increased with the years of follow-up, from 0.6% at first postoperative year to 8.4% at the 10th year (1). The recurrence rate, clinical course and impact on graft survival vary between different types of glomerulonephritis. This review aims to provide updated knowledge on recurrent renal diseases after kidney transplantation, focusing on recent findings with new post-transplant immunosuppressive regimens and treatment.

Immunoglobulin A Nephropathy

Immunoglobulin A Nephropathy (IgAN) is the most common type of glomerulonephritis worldwide and is the primary cause of renal failure in 20% of kidney transplant recipients. The pathogenetic mechanisms are complex and incompletely understood. It is likely to be related to the aberrant synthesis of abnormally O-glycosylated IgA1 in
patients with IgAN. Mesangial deposition of polymeric IgA1 with abnormal O-glycosylation initiates glomerular inflammation and injury with progressive loss of renal function (5).

Recurrent IgAN is common after transplantation. Great variation in the incidence of recurrence has been reported because of difference in duration of follow-up and biopsy policy of different transplant centers (Table 1). Most centers performed renal biopsy only when patients presented with clinical symptoms of proteinuria, hemorrhuria or decline in renal function. This would potentially underestimate the rate of recurrence as patients who were clinically asymptomatic but with histological changes in the graft kidneys would remain undiagnosed. For centers where routine protocol biopsies were being carried out in all transplant recipients, histological recurrence with mesangial IgA deposits and mesangial hypercellularity had been reported in 50–60% of patients (6,7). Recurrence rate reported for patients with renal biopsies for clinical symptoms ranged from 13–50% (9–21) (Table 1). Clinical manifestations are similar to primary IgAN and include microscopic hematuria, proteinuria and slow decline in renal function. Clinical course of recurrent IgAN had been reported to be benign initially (6,8). However, with increasing long-term data, it is apparent that recurrent disease is not as benign as had been reported previously (7,9–18,20,21). Graft loss from recurrence with histological features of diffuse mesangial proliferative expansion and glomerular sclerosis were reported between 1.3% and 16% (1,7,9–21) (Table 1). The estimated 10-year incidence of graft loss due to recurrence was 9.7% (CI 4.7–19.5%) from the latest registry report containing the largest number of IgAN patients (1).

It is interesting to note that renal allograft survival for the first 5 years post-transplant is better in patients with primary IgAN compared to other primary diseases (8,11,19,20). The proposed mechanism included increased occurrence of allo-reactive IgA anti-HLA antibodies which may block the deleterious effect of IgG and IgM antibodies on the graft, and the immunological dysfunction of patients with IgAN (8). Despite the better graft survival of IgAN patients for the early post-transplant period, graft survival becomes comparable and might be worse than patients with other underlying renal diseases when data with follow-up beyond 10 years becomes available (16,18,20), suggesting other factors including recurrent disease contributing to graft loss becomes more apparent with long-term follow-up. No single parameter including age, gender, race, HLA

<table>
<thead>
<tr>
<th>Follow-up duration (mean) (months)</th>
<th>No. of allografts Total (R/NR)</th>
<th>Recurrence rate1</th>
<th>Graft loss due to recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. (%) (R/NR)</td>
<td>No. (%) (R/NR)</td>
</tr>
<tr>
<td>Berger et al. 1984 (6)</td>
<td>&gt;24</td>
<td>32 (13/19)</td>
<td>17(53.1%) (9/8)</td>
</tr>
<tr>
<td>Bachman et al. 1986 (9)</td>
<td>20 ± 13</td>
<td>13 (6/7)</td>
<td>6(46.2%) (5/1)</td>
</tr>
<tr>
<td>Odum et al. 1994 (7)</td>
<td>3–183</td>
<td>51 –</td>
<td>17(33.3%) –</td>
</tr>
<tr>
<td>Hartung et al. 1995 (10)</td>
<td>45.9 ± 10</td>
<td>128 –</td>
<td>47(36.7%) –</td>
</tr>
<tr>
<td>Kesser et al. 19963 (11)</td>
<td>68.1 ± 37.2</td>
<td>84 (3/25)6</td>
<td>13(15.5%) (2/1)5</td>
</tr>
<tr>
<td>Frohnert et al. 1997 (12)</td>
<td>78 (3–156)6</td>
<td>53 (41/12)</td>
<td>10(19%) (6/2)</td>
</tr>
<tr>
<td>Ohmacht et al. 19974 (13)</td>
<td>54 (7–127)6</td>
<td>61 –</td>
<td>20(29.9%) –</td>
</tr>
<tr>
<td>Bumgardner et al. 1998 (14)</td>
<td>61 ± 37</td>
<td>61 (18/43)</td>
<td>18(29.5%) (6/12)</td>
</tr>
<tr>
<td>Freese et al. 1999 (15)</td>
<td>67 (11–159)6</td>
<td>104 (47/57)</td>
<td>13(12.5%) (11/2)</td>
</tr>
<tr>
<td>Kim et al. 2001 (16)</td>
<td>2–164</td>
<td>90 (60/30)</td>
<td>19(21.1%) (13/6)</td>
</tr>
<tr>
<td>Wang et al. 2001 (17)</td>
<td>52 (18–155)6</td>
<td>48 (17/31)</td>
<td>14(29.2%) (6/8)</td>
</tr>
<tr>
<td>Ponticelli et al. 2001 (18)</td>
<td>70.4 ± 50.5</td>
<td>106 (21/85)</td>
<td>37(35%) (9/25)</td>
</tr>
<tr>
<td>Andreosdottir et al. 2001 (19)</td>
<td>67.2 ± 54</td>
<td>79 –</td>
<td>17(21.5%) –</td>
</tr>
<tr>
<td>Briganti et al. 2002 (1)</td>
<td>12–120</td>
<td>532 –</td>
<td>–</td>
</tr>
<tr>
<td>Choy et al. 2003 (20)</td>
<td>100.0 ± 5.8</td>
<td>75 (32/43)</td>
<td>14(18.7%) (9/5)</td>
</tr>
<tr>
<td>Moriyama 2005 (21)</td>
<td>67.8 ± 19.9</td>
<td>49 (44/5)</td>
<td>13(26.5%) (12/1)</td>
</tr>
</tbody>
</table>

RD = related donor, NRD = nonrelated donor.

Recurrence rate for RD = 29.8%; NRD = 22.7%. Breslow–Day test of Homogeneity of odds ratio: chi-square = 10.29, df = 10, p = 0.416. Mantel–Haenszel estimate of Common odds ratio: 2.14 (95% CI = 1.42, 3.23; p < 0.001).

Percentage of graft loss from RD = 34.8%; NRD = 24.1%. Breslow–Day test of Homogeneity of odds ratio: chi-square = 7.37, df = 5, p = 0.194. Mantel–Haenszel estimate of Common odds ratio: 1.95 (95% CI = 0.64, 5.97; p = 0.243). 

(%) = Percentage was calculated from number of graft loss due to recurrent IgAN/total number of patients with primary IgAN.

1Recurrence rate in patients with clinical symptoms of proteinuria/hematuria/renal impairment.

2Recurrence rate in patients with histological changes but clinically asymptomatic.

3Included 13 patients who suffered from underlying Henoch-Schonlein purpura.

4Included 4 patients who suffered from underlying Henoch-Schonlein purpura.

5Only 28 allografts had information with respect to the donor type.

6Median.
typing, pre-transplant course or biochemical characteristic of serum IgA can predict recurrence.

The relationship between the risk of recurrence and the donor type remains controversial. Some studies had reported a higher risk of disease recurrence in related donors (6,9,11,15,17), while others reported no added risk (12,14,16). Pooling all available data from literature that contained information on graft recurrence (6,9,11,12,14–18,20,21) and graft loss (9,12,14,17,20,21) in relation to donor type and estimate the risk by Mantel–Haenszel estimate of common odds ratio showed a higher risk of disease recurrence among transplant recipients with related donors (common odds ratio 2.14, \( p < 0.001 \)), but the risk of graft loss was not increased (common odds ratio 1.95, \( p = 0.24 \)) (Table 1). Whether this apparent paradox could be due to insufficient follow-up remains to be investigated. Given the fact that the graft survival of patients with primary IgAN is excellent for the first decade post-transplant, it is inappropriate to refrain from living related donor transplantation even though there may be a slight risk of recurrence. In contrast, familial IgAN should be rigorously excluded in potential living related donors since familial IgAN is associated with high risk of development of renal failure in affected members (22). Moriyama et al. reported higher risks of recurrence and graft loss in patients with latent IgA deposition from donor kidneys (majority were living related donors) (21). Whether such latent IgA deposition or the load of immune deposits might be detrimental to graft survival remain speculative.

The situation is quite different for patients with prior graft loss due to recurrent IgAN because the risk of recurrence in the second transplant (20–100%) is much increased (13–15,18). Omhacht et al. reported a graft loss rate of 60% in their patients with a follow-up duration of 21–51 months (13) while two other series reported good graft function despite of recurrence in their patients up to 92 months of follow-up (14,18). In this regard, living donor transplant should be discouraged if recurrence and graft failure occur within few years after first transplant. However, such a transplantation is not a problem if their first graft functions beyond 10 years post-transplantation.

There is no effective therapy for the prevention or treatment of recurrent IgAN. Calcineurin inhibitors, in the presence or absence of induction therapy, do not influence the recurrent risk. Despite initial enthusiasm, newer immunosuppressive drugs are ineffective in preventing recurrence. Anecdotal reports that mycophenolate mofetil might have averted progression to allograft failure in recurrent IgAN are not substantiated by recent studies (18,23). Data on sirolimus are limited. Development of IgAN with nephrotic range of proteinuria had been reported in two transplant recipients after conversion from a calcineurin inhibitor-based immunosuppression to sirolimus (24). Steroid free or rapid steroid withdrawal regimen does not seem to affect the recurrent risk (25). The effect of fish oil in recurrent IgAN has not been systematically examined. Angiotensin converting enzyme inhibitor and angiotensin receptor blocker are commonly used for reduction of proteinuria and preservation of renal function in patients with recurrence as in IgAN of native kidneys (26,27).

Henoch-Schönlein purpura (HSP) has been regarded by many as the systemic variant of IgAN. Renal manifestation of HSP is indistinguishable from IgAN. Currently available data suggest that the recurrence rate after transplantation in patients with HSP is similar to that of IgAN (13,19,28).

### Focal and Segmental Glomerulosclerosis

Focal and segmental glomerulosclerosis (FSGS) is a histological diagnosis that encompasses not only the idiopathic form (primary FSGS) but also a variety of secondary causes including glomerular hyperfiltration, toxic injury or viral infection leading to similar sclerotic lesions, recurrence risk of which depends on the underlying disorder. Primary FSGS has a recurrence rate of 20–50% after kidney transplantation leading to graft failure in 13–20% of patient in 10 years after kidney transplantation (1,4). Clinical manifestations of recurrent FSGS include early onset of massive proteinuria, usually within first year post-transplant, hypertension and graft dysfunction.

The pathogenesis of recurrent FSGS is unclear. A circulating permeability factor which increases the glomerular permeability to albumin and is removable by plasmapheresis or immunoabsorption therapy has long been suspected to play an important role. Savin et al. developed an *in vitro* bioassay for the permeability factor (29), and had shown that patients with high permeability factor activity in pretransplant sera were more likely to develop recurrence (29,30). However, recent data suggest that the absence or loss of an inhibitor of a normally present factor in plasma rather than the addition of a circulating factor could be the underlying cause for the glomerular permeability alteration (31,32). Further complicating the picture is the recognition of the pivotal role of the podocyte in the pathogenesis of proteinuria in various glomerulopathies. Acquired or inherited defect in the slit—diaphragm proteins (podocin [NPHS2], nephrin [NPHS1], α-actinin 4 and CD2AP) on the glomerular basement membrane have been reported in 15% of patients with primary FSGS (33,34). Recurrence which would not be expected in the genotypically normal donor kidneys have been reported in recipients with mutations of podocin, more so for the heterozygous than the homozygous mutations (34). This suggested that etiology of recurrent FSGS is likely multifactorial involving interaction between genetic and extra-renal mechanisms (putative permeability factor).

Risk factors for recurrence include younger age, rapid progression of original disease with development of end-stage renal failure within 3 years, mesangial hypercellularity of
Data from small series have implicated increased recurrent disease, although long-term data are still awaited (25,46). An increase in recurrent FSGS or graft loss from recurrent underlying FSGS. Early steroid withdrawal did not lead to complete or partial remission of their proteinuria after 6 months of therapy although efficacy and long-term safety need further evaluation with prospective trial.

Newer immunosuppressive agents such as sirolimus have increasingly been used to replace calcineurin inhibitors to avoid calcineurin inhibitors associated nephrotoxicity and to avoid chronic allograft nephropathy. However, a number of case reports have reported the development of de novo or recurrent FSGS when cyclosporine in replaced with sirolimus, with subsequent improvement after switching back to cyclosporine (24,42,43). The beneficial effect in this regard seemed specific to cyclosporine (43). Paradoxically, sirolimus had been reported in a recent study that 12 out of 21 patients with steroid resistant FSGS achieved complete or partial remission of their proteinuria after 6 months of therapy (44). In view of the accentuation of glomerular damage due to the proinflammatory effects of sirolimus and its derivatives in animal models (45), caution still need to be exercised when sirolimus is used in patients with underlying FSGS. Early steroid withdrawal did not lead to an increase in recurrent FSGS or graft loss from recurrent disease, although long-term data are still awaited (25,46). Data from small series have implicated increased recurrent FSGS with antilymphocytic antibodies (47) and anti-IL2 receptor antibodies (48).

Membranoproliferative (Mesangiocapillary) Glomerulonephritis

Secondary causes of membranoproliferative (mesangiocapillary) glomerulonephritis (MPGN) (type I) include infections such as viral hepatitis B or C and systemic diseases. Treatment of these underlying causes may thus reduce the risk of recurrence. Recurrent disease should also be differentiated from de novo MPGN which occurs as part of the histological changes in patients with chronic transplant nephropathy.

Both type I (with mesangial and subendothelial deposits) and type II (dense deposit disease) primary MPGN have high rates of recurrence after transplantation. Type I MPGN recurs in 20–50% of patients. Clinical manifestations include proteinuria and deterioration of renal function. Risk factors for recurrence include HLA-B8DR3, living related donors and previous graft loss from recurrence (49). The overall incidence of allograft loss at 10 years due to recurrence is around 15% (1). The risk of graft loss from recurrence in a second graft in patients who have experienced a recurrence in the first graft is as high as 80% (49).

Recurrence disease is much more frequent in type II disease, and up to 80–100% of patients are affected. These patients usually present with nonnephrotic range proteinuria within the first year posttransplant and slowly declining renal function. There is no correlation between complement level and recurrence risk. Graft loss due to recurrence occur in 15–30% of patients after 5 years (50). Type III MPGN (with both subepithelial and subendothelial deposits) has been considered as a variant of type I disease, and there are few data regarding its recurrence after kidney transplantation.

A recent report has suggested that the severity of histological abnormalities in the native kidney (interstitial fibrosis, crescent formation and mesangial proliferation) rather than the type of MPGN is related to recurrence risk. Nevertheless, type II MPGN usually has more aggressive glomerular changes and thus a higher risk of recurrence, and poorer prognosis (51). No effective therapy is available for prevention or treatment of recurrent MPGN.

Membranous Nephropathy

Secondary causes of membranous nephropathy (MN) including viral infections and malignancy should be screened. Treatment of these underlying causes may reduce the risk of recurrence in secondary MN. Idiopathic MN recurs in 10–30% of patients after kidney transplantation. Recurrent disease should also be differentiated from de novo MN.
which is the most common de novo glomerulopathy in renal allografts. The clinical presentation of recurrent disease is characterized by nephrotic range proteinuria. The mean onset time is approximately 10 months post-transplant as compared with the more insidious and later onset of symptoms in de novo MN, an entity thought to be related to chronic rejection (52,53). Recent demonstration of antibodies against ‘neutral endopeptidase’, a protein expressed on the human podocyte cell membrane, causing severe membranous glomerulonephritis in a fetus, suggested that ‘neutral endopeptidase’ probably plays a significant role in the pathogenesis of the membranous glomerulonephropathy (54). No risk factor for recurrence has been identified. The initial concerns with regard to the risk of recurrence with living related donors, presence of HLA-DR3 in the recipient, and the aggressiveness of native disease have not been substantiated (53). Graft failure from recurrence occurs in 10–15% of patients after 10 years (1). Cyclosporine and mycophenolate mofetil which have been used in treatment of primary MN do not prevent or change the course of recurrent disease (53). There is also no report to suggest therapeutic advantage of tacrolimus or cyclophosphamide over cyclosporine.

**Antineutrophil Cytoplasmic Antibody-Associated Glomerulonephritis (Pauci-Immune Crescentic Glomerulonephritis)**

Despite better recognition and improved treatment of antineutrophil cytoplasmic antibody (ANCA)-associated glomerulonephritis, a proportion of patients still progress to end-stage renal failure. Recurrence in patients with Wegener’s granulomatosis (WG), microscopic polyangiitis (MPAN) and idiopathic necrotizing crescentic glomerulonephritis (CGN) have been reported. Nachman et al. pooled data from 127 patients and reported that 17% of patients had recurrence of vasculitis after 4–89 months of follow-up. Three-fifths of them had renal manifestation and two patients lost their grafts due to recurrence (55). A more recent study by Briganti reported a 10-year incidence of allograft loss of 7.7% in patients with pauci-immune crescentic glomerulonephritis (1).

Pre-transplantation disease course, cANCA or pANCA specificity, disease subtype (WG, MPAN or CGN), ANCA titer (in the absence of clinically active disease) at the time of transplantation, duration of follow-up or donor type do not predict recurrence (55). It is advisable to defer kidney transplantation until the disease is inactive (55). Patients with renal relapses generally showed good response to cyclophosphamide (55–57). For patients with cellular crescents on renal biopsies and high ANCA titer, favorable outcome with combination therapy comprising cyclophosphamide, plasmapheresis with or without intravenous immunoglobulin had been reported (57,58).

**Systemic Lupus Erythematosis**

Although the prognosis of lupus nephritis has improved over the past few decades, lupus nephritis remains an important cause of end-stage renal failure. Histological recurrence has been reported in up to 30% (59) of transplant recipients. Clinically significant recurrent disease occurs in 2–9% (2,60). With the higher morbidity and poorer general condition of patients during active disease, most centers would postpone renal transplantation until the disease becomes quiescent for at least 6–9 months (60,61). The duration of dialysis before transplantation and serological status in the absence of clinically active disease do not predict recurrence (59–61). There are anecdotal reports on the efficacy of mycophenolate mofetil in recurrent lupus nephritis (62,63). Graft loss due to recurrent lupus nephritis is uncommon, occurring in 2–4% (59–61). Long-term patient and graft survival are similar to kidney allograft recipients with other underlying diseases (59–61).

**Antiglomerular Basement Membrane Disease**

Histological recurrence had been reported in up to 50% of patients when kidney transplantation was performed while circulating antiglomerular basement membrane disease (anti-GBM) antibodies were still present (64). With the current practice of deferring transplantation until the disease becomes quiescent and circulating anti-GBM antibody levels become undetectable for at least 12 months, clinical recurrence is rare and consisted of isolated case reports only (1,3). Good treatment response had been reported in one patient who developed recurrence with positive anti-GBM antibody and crescentic glomerulonephritis treated with pulse steroid, plasmapheresis and cyclophosphamide (65) while another patient responded to treatment with immunoadsorption and cyclophosphamide (57).

**Conclusions**

With improving long-term renal allograft survival, recurrent disease has increased prominence as a significant contributor to late graft loss. Knowledge on the risk factors for recurrence, onset time and impact on graft function is prerequisite to informed decisions (Table 2). There are minimal data on the risk of recurrent disease with new immunosuppressive agents, although anecdotal observations caution cyclosporine and/or corticosteroid withdrawal in patients with a history of FSGS, and animal data suggest that it is pertinent to examine the impact of sirolimus on recurrent glomerular diseases. Apart from plasmapheresis for patients with recurrent FSGS, there is no consensus on strategies to prevent or treat recurrent glomerular disease in the kidney allograft. It is important to emphasize that the majority of patients with primary glomerulonephritis as the underlying cause of renal failure enjoy excellent graft and patient survival.
### Table 2: Risk of recurrence and graft loss and treatment strategies for different types of glomerulonephritis

<table>
<thead>
<tr>
<th>Type</th>
<th>Risk of recurrence</th>
<th>Risk of graft loss due to recurrence</th>
<th>Prevention/treatment strategies</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IgAN</strong></td>
<td>13–46%</td>
<td>2–16%</td>
<td>ACEI and/or ARB for patients with proteinuria ± renal impairment due to recurrent IgAN (26,27)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Avoid living donors for patients with history of rapid graft loss from recurrence (38)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Preemptive perioperative plasmapheresis (PP) for 2 weeks for patients with high risk of recurrence (39,40)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Chronic PP with or without cyclophosphamide or cyclosporine for patients with relapse after initial course of PP (29,30,35)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>? Avoid omission of calcineurin inhibitors in sirolimus based immunosuppressive regimen (24,42,43)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>? Avoid induction therapy (47,48)</td>
</tr>
<tr>
<td><strong>FSGS</strong></td>
<td>20–50%</td>
<td>13–20%</td>
<td>No effective preventive or treatment measures</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Exclude secondary causes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Defer transplant till disease inactive (55)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Cyclophosphamide for recurrence (55,56)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Combine therapy with PP, cyclophosphamide ± intravenous immunoglobulin for recurrence with high titer of ANCA and cellular crescents in renal biopsies (57,58)</td>
</tr>
<tr>
<td><strong>MPGN</strong></td>
<td></td>
<td></td>
<td>No effective preventive or treatment measures</td>
</tr>
<tr>
<td>Type I</td>
<td>20–25%</td>
<td>~15%</td>
<td>Exclude secondary causes</td>
</tr>
<tr>
<td>Type II</td>
<td>80–100%</td>
<td>15–30%</td>
<td>Exclude secondary causes</td>
</tr>
<tr>
<td>Membranous nephropathy</td>
<td>10–30%</td>
<td>10–15%</td>
<td>Defer transplant till disease inactive (60,61)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Consider mycophenolate for recurrence (62,63)</td>
</tr>
<tr>
<td><strong>ANCA-associated glomerulonephritis</strong></td>
<td>~17%</td>
<td>6–8%</td>
<td>Defer transplant till disease inactive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Combine therapy with PP, cyclophosphamide ± intravenous immunoglobulin for recurrence with high anti-GBM titer and cellular crescents in renal biopsies (57,65)</td>
</tr>
<tr>
<td><strong>SLE</strong></td>
<td>2–9%</td>
<td>2–4%</td>
<td>Defer transplant till disease inactive</td>
</tr>
<tr>
<td><strong>Anti-GBM</strong></td>
<td>Rare</td>
<td>Rare</td>
<td>Defer transplant till disease inactive</td>
</tr>
</tbody>
</table>

1Clinical relevant refer to patients with clinical symptoms of proteinuria/hematuria/renal impairment.  
2% of transplanted patients.

...survival. Also, in spite of the controversy over the risk of recurrence with certain types of glomerulonephritis when the source of allografts is from living donors, the graft survival is largely comparable to patients with other causes of end-stage renal failure. Thus, living related kidney donation can still be encouraged in carefully selected patients and donors. Caution should be exercised in patients with previous rapid graft loss due to recurrent disease in view of the markedly increased risk with subsequent transplants. Research toward identification of biological or immunological markers for individual glomerulonephritis should provide tools to better identify and prevent recurrence.

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