IgA nephropathy recurs early in the graft when assessed by protocol biopsy

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Abstract

Background. The recurrence of IgA nephropathy (IgAN) in the allograft is common. Factors related to IgA recurrence are unclear. The aims of this study were to determine the incidence of IgAN recurrence as assessed by protocol biopsies and to identify predictive factors for recurrence.

Methods. We identified 65 protocol biopsies taken before the second year post-transplantation in patients with IgAN as primary renal disease. Diagnosis of recurrence of IgA was based on the detection of at least 1+ mesangial deposits of IgA. Pathological findings and clinical characteristics were retrospectively compared between recurrent and non-recurrent cases.

Results. IgAN recurrence rate was 32%. Mesangial C3 was detected in 83% of recurrent cases versus 17% in non-recurrent patients (P < 0.001). Normal urinalysis was observed in 52%. Non-recurrent patients had arteriolar hyalinosis in 31% of the cases versus none in IgAN recurrence (P = 0.006). Seventy-nine per cent of cyclosporine users were free of recurrence, whereas 45% of the patients without cyclosporine experienced recurrence (P = 0.03). The odds ratio (OR) for IgAN recurrence in patients using cyclosporine was 0.3 (confidence interval 0.1–0.9). Zero HLA-DR mismatch was associated with non-recurrence (P < 0.01). The OR for IgA recurrence was 6.7 if any degree of DR mismatch was present. IgAN recurrent patients had better glomerular filtration rate, but after censoring delayed graft function, the differences disappeared. Graft loss due to IgA recurrence was only 3%.

Conclusions. IgAN recurrence rate was 32%. The histological diagnosis was not accompanied by abnormalities in the urinalysis in one-half of the patients. Full DR match and cyclosporine were associated with non-recurrence.
Keywords: glomerulonephritis recurrence; IgA glomerulonephritis; kidney transplantation; protocol biopsy

Introduction

IgA nephropathy (IgAN) is the most common glomerular disease worldwide. Prevalence appears highest in Asia, Australia, Finland and southern Europe, ranging from 20 to 40% among patients with primary glomerulonephritis, being the first cause of end-stage renal failure in this population [1]. In the USA, the prevalence rate is only 6.6% [2]. Despite the recommended therapeutic approaches for the treatment of IgA, patients with known risk factors for progression evolve into uraemia in 20–30% of the cases [3, 4].

Kidney transplantation is a successful treatment for patients with end-stage renal disease due to IgAN. However, Berger noticed recurrence of the disease as early as in 1975 [5]. The incidence of IgAN recurrence has been described to range between 12 and 53% [6]. This great variation can be explained by disparities between studies concerning the length of the follow-up period, biopsy policies, different ethnicities and the proportion of living related donors. The improvements in kidney transplantation in the past decades have significantly prolonged graft survival. As a consequence, the effect of recurrent glomerular disease has developed an increasing importance since the longer the time after transplantation, the higher the probability of recurrence. In these lines, the longest follow-up of IgAN-transplanted patients reported in the literature was 10 years (mean) and the recurrence rate was 18.7%, based on biopsies obtained by cause [7]. However, in 32 serial protocol biopsies taken at 6 months, 2, 4 and 10 years, the recurrence rate was 53% [8]. Another study including 11 protocol biopsies reported a 27% of IgAN recurrence [9].

The contribution of recurrence of IgAN to graft loss has been considered to be of minor importance in the past, though it is far to be addressed. One of the largest studies on the risk of graft loss due to recurrent glomerulonephritis revealed that it comes third, after chronic rejection and patient’s death. The incidence of allograft loss due to recurrence of IgAN at 10 years was 2.8% [4]. Also, a recent report from the United States Renal Data System described an incidence of 2.6% of 10-year graft loss due to recurrent glomerulonephritis [2]. While it is well established that IgAN can recur after transplantation, time, frequency, risk factors, effect of immunosuppression and the long-term consequences of recurrence are still uncertain. The aims of this study were to determine the incidence of IgAN recurrence assessed by protocol biopsies and to identify predictive factors including pathological and clinical characteristics in a long-term retrospective analysis.

Materials and methods

Altogether, 155 patients with IgAN as the aetiology for end-stage renal disease received a kidney transplant in Bellvitge University Hospital or Helsinki University Hospital between January 2001 and April 2010. For the analysis, we excluded recipients of multiple organs.

A kidney allograft biopsy was taken by protocol, after patient’s written consent, in 80 patients. The reasons for not taking a protocol biopsy were patient’s refusal, anticoagulation therapy or other medical contraindication. Whereas a protocol allograft biopsy was obtained in 80 cases, immunofluorescence techniques were available in 65. Therefore, this cohort constituted the material for this retrospective study. Protocol biopsies were taken at a median time of 6.9 months after transplantation. The policy regarding the time point for obtaining a protocol biopsy varied in both institutions, with a time frame of 2.2–25 months. However, 75% of the biopsies were obtained between 6.2 and 12 months after transplantation. Two core samples were obtained under ultrasound guidance with an automatic biopsy gun. There were no major complications related to the procedure. Histological material was examined under light and immunofluorescence microscopy. Biopsies were classified according to Banff’97 by two different pathologists (M.C. and A.R.-S.). Immunoglobulins and C3 deposits were studied in all biopsies. C4d was routinely analysed with immunofluorescence in 80% of them. Diagnosis of recurrence of IgA was based on the detection of at least 1+ mesangial deposits of IgA.

Clinical data collected from Bellvitge University Hospital Transplant Registry and Finnish National Transplant Registry included donor and recipient demographics, human leucocyte antigen (HLA) antibodies, HLA mismatches, type and number of transplant. The data collected from medical records were diagnosis of hypertension before and after transplantation, body mass index (BMI) pre- and post-transplantation; presence of diabetes mellitus previous to transplantation and new-onset diabetes mellitus, occurrence of delayed graft function (DGF), acute rejection (AR) episodes, length of follow-up and time from transplantation to biopsy. Also evaluated was the immunosuppressive therapy, the use of statins, angiotensin-converting enzyme inhibitors (ACE-i) or angiotensin 2 receptor blockers (ARB). Serum creatinine and proteinuria were collected at 6 months and thereafter yearly over a 10-year follow-up period. Haematuria at the time of protocol biopsy was also observed. Kidney function was assessed with the estimated glomerular filtration rate (eGFR) calculated with the modified Modification of Diet in Renal Disease formula [10].

Statistical analysis was performed using SPSS Software (version 17.0; Chicago, IL). Data are presented as median and standard deviation for continuous variables and in proportion for categorical variables. Categorical data were compared using chi-square test and the odds ratio (OR) for recurrence of IgAN, with a confidence interval (CI) of 95%. Continuous data were analysed with the Mann–Whitney U-test. Logistic regression was used to evaluate the effect of the use of immunosuppression and HLA match on the recurrence. P-values <0.05 were considered significant.

Results

Clinical data

Demographic and clinical data are depicted in Table 1. IgAN recurrence was diagnosed in 32.3% of the protocol biopsies. HLA-A2 was more common in patients without recurrence (44 versus 31%, respectively), which was not statistically significant. No other HLA types in the recipient were specifically associated with recurrence. Full DR match was observed in 89.5% of the patients without recurrence, whereas in cases with recurrence, it accounted for 10.5% (P < 0.01). The OR for IgAN recurrence was 6.7 (CI 1.4–32.8) if there was any degree of DR mismatch. As shown in Table 1, patients without recurrence were older and received kidneys from older donors (47 versus 40 years, P = 0.02). Accordingly, DGF was more common in non-recurrent patients, but the differences did not reach statistical significance. There were similar rates of AR between the recurrence and non-recurrence groups.

Clinical data at the time of the protocol biopsy are detailed in Table 2. The histological recurrence was not accompanied by significant haematuria or proteinuria. In fact, in 52% of the patients, IgA recurrence was lanthanic. Four of the 21 patients with IgA recurrence (19%) developed proteinuria of over 0.5 g/24 h during the follow-up, while
in the non-recurrent group, it was detected in 9 of 44 patients (20%). Serum creatinine and eGFR were better in IgAN recurrent patients early after transplantation. The differences were statistically significant between the first and third year but vanished after the fourth year (Figure 1). After censoring the patients who experienced DGF, we were not able to find any statistical difference in renal function between the IgAN recurrent and non-recurrent patients (Figure 2). During a median follow-up of 56 months, two patients died with a functioning graft and four returned to dialysis (two due to IgAN recurrence).

**Renal histology**

From the 21 protocol biopsies with IgA mesangial deposits, 14 showed normal histomorphology and in two cases, these deposits were associated with increased mesangial matrix. Concomitant sub-clinical AR was observed in one case. Recurrence of IgAN was accompanied with chronic nephropathy in four cases. Among the 44 protocol biopsies without recurrence, 23 had normal histology. Sub-clinical AR was observed in four protocol biopsies and one showed borderline AR. Twelve cases had chronic nephropathy. There was one case of cyclosporine A-related toxicity and one polyoma virus nephropathy. C4d in peritubular capillaries was seldom seen; however, of the four cases that were seen, all cases were in non-recurrent patients. The mesangial deposition of C3 was concomitant with IgA and was seen in 83% of recurrent cases compared to only 17% of C3 deposition in patients without recurrence (P < 0.001).

Individual scoring components of the Banff classification were equally seen in both groups, with the exception of arteriolar hyalinosis. This feature was seen in 31% of the patients without IgAN recurrence, while none of the cases with IgAN recurrence showed hyalinosis (P = 0.006). The presence of arteriolar hyalinosis was associated with donor age (P = 0.04) but not with the use of cyclosporine (P = 0.18), diabetes, BMI or arterial hypertension.

**Immunosuppression and concomitant medications**

The use of immunosuppressive drugs, ACE-i/ARB and statins are summarized in Table 3. Induction therapy with thymoglobulin was administered to 10 (15.4%) of the patients. These patients were, however, at low immunological risk. The recurrence rate in the patients who received induction with thymoglobulin was 60%. On the contrary, only 27% of the patients without early recurrence of IgAN received induction with thymoglobulin (P < 0.04). Induction with interleukin-2 receptor monoclonal antibody (IL-2 R-mAb) was equally used in both recurrent and non-recurrent patients. The vast majority of the patients (91%) were on triple therapy with a calcineurin inhibitor, myco-phenolate mofetil and prednisone. Interestingly, among the 34 patients receiving cyclosporine, 79% were free of recurrence, whereas 45% of the patients without cyclosporine experienced recurrence of IgAN (P = 0.03). The OR for

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**Table 1. Demographic and clinical data**

<table>
<thead>
<tr>
<th></th>
<th>All patients (N = 65)</th>
<th>Without recurrence (N = 44)</th>
<th>With recurrence (N = 21)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recipient sex (male/female)</td>
<td>55/10</td>
<td>37/7</td>
<td>18/3</td>
<td>0.86</td>
</tr>
<tr>
<td>Recipient age (median, SD)</td>
<td>43 (±11.5)</td>
<td>47 (±12.1)</td>
<td>43 (±9.5)</td>
<td>0.11</td>
</tr>
<tr>
<td>Donor sex (male/female)</td>
<td>38/27</td>
<td>24/20</td>
<td>14/7</td>
<td>0.35</td>
</tr>
<tr>
<td>Donor age (median, SD)</td>
<td>48.5 (±14.1)</td>
<td>52 (±14.3)</td>
<td>38 (±11.9)</td>
<td>0.02</td>
</tr>
<tr>
<td>Re-transplantation (%)</td>
<td>9.2</td>
<td>9.1</td>
<td>9.5</td>
<td>0.95</td>
</tr>
<tr>
<td>DD/LRD/LURD (%)</td>
<td>91/9/0</td>
<td>93/7/0</td>
<td>86/14/0</td>
<td>0.33</td>
</tr>
<tr>
<td>Panel reacting antibodies (median, range)</td>
<td>1 (0–7)</td>
<td>1 (0–7)</td>
<td>0</td>
<td>0.06</td>
</tr>
<tr>
<td>Acute rejection (%)</td>
<td>25</td>
<td>23</td>
<td>29</td>
<td>0.61</td>
</tr>
<tr>
<td>DGF (%)</td>
<td>28</td>
<td>34</td>
<td>14</td>
<td>0.09</td>
</tr>
<tr>
<td>Hypertension pre-Tx (%)</td>
<td>64.6</td>
<td>63.6</td>
<td>66.7</td>
<td>0.81</td>
</tr>
<tr>
<td>Hypertension after Tx (%)</td>
<td>76.9</td>
<td>81.8</td>
<td>66.7</td>
<td>0.17</td>
</tr>
<tr>
<td>BMI pre-Tx (median and SD)</td>
<td>24.9 (±3.7)</td>
<td>29 (±3.9)</td>
<td>24.7 (±3.2)</td>
<td>0.72</td>
</tr>
<tr>
<td>BMI after Tx (median and SD)</td>
<td>26.1 (±3.6)</td>
<td>26.2 (±3.5)</td>
<td>25.8 (±3.7)</td>
<td>0.76</td>
</tr>
<tr>
<td>DM pre-Tx (%)</td>
<td>6.2</td>
<td>4.5</td>
<td>9.5</td>
<td>0.43</td>
</tr>
<tr>
<td>NODAT (%)</td>
<td>13.8</td>
<td>13.6</td>
<td>14.3</td>
<td>0.94</td>
</tr>
</tbody>
</table>

*DD, deceased donor; LRD, living-related donor; NODAT, new-onset diabetes mellitus; Tx, transplantation.

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**Table 2. Clinical data at the time of the protocol biopsy**

<table>
<thead>
<tr>
<th></th>
<th>All patients (N = 65)</th>
<th>Without recurrence (N = 44)</th>
<th>With recurrence (N = 21)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Follow-up (median and range, in months)</td>
<td>56.4 (6–117)</td>
<td>54 (2–117)</td>
<td>60 (9.6–109)</td>
<td>0.81</td>
</tr>
<tr>
<td>Time to protocol biopsy (median, range in months)</td>
<td>6.9 (2.2–25)</td>
<td>7.2 (2.6–24)</td>
<td>6.8 (2.2–25)</td>
<td>0.93</td>
</tr>
<tr>
<td>Haematuria (%)</td>
<td>24</td>
<td>19</td>
<td>36</td>
<td>0.13</td>
</tr>
<tr>
<td>Proteinuria in g/day (median, SD)</td>
<td>0.10 (±0.51)</td>
<td>0.10 (±0.57)</td>
<td>0.10 (±0.34)</td>
<td>1.00</td>
</tr>
<tr>
<td>Serum creatinine (µmol/L, median, SD)</td>
<td>126 (±37)</td>
<td>128 (±40)</td>
<td>119 (±29)</td>
<td>0.75</td>
</tr>
<tr>
<td>Cyclosporin trough level (median, SD)</td>
<td>110 (±25.6)</td>
<td>117 (±26)</td>
<td>93 (±19)</td>
<td>0.39</td>
</tr>
<tr>
<td>Tacrolimus trough level (median, SD)</td>
<td>7.5 (±2.6)</td>
<td>7.3 (±2.1)</td>
<td>10.8 (±4.4)</td>
<td>0.37</td>
</tr>
</tbody>
</table>
IgA recurrence in patients using cyclosporine was 0.3 (CI 0.1–0.9). We compared the cyclosporine trough levels of both groups and the differences were not statistically different. To further evaluate the association between cyclosporine and the risk of recurrence, we focused our analysis on the biopsies without hyalinosis. Even in the absence of hyalinosis, the use of cyclosporine was associated with non-recurrence in 76% of the cases. The use of tacrolimus and its trough concentrations were similar both in the recurrent and in non-recurrent patients.

The prescription of statins or ACE-i/ARB was similar among the recurrent and non-recurrent patients. Of note, logistic regression analysis considering recurrence of IgA as a dependent variable and as covariates showed full DR mismatch and use of cyclosporine reaffirmed the statistical significance of both parameters (risk of recurrence = −1.46 + 1.89 × DR mismatch − 1.33 × cyclosporine; P = 0.023 and P = 0.026, respectively).

Discussion

In this study, we evaluated the incidence of IgAN recurrence as assessed by protocol biopsies in 65 patients and aimed to identify clinical and pathological factors that could predict recurrence. We found that the histological recurrence of IgAN can be recognized in almost one-third of the patients after 2 years from transplantation. To our knowledge, only two studies reviewed IgAN recurrence in protocol biopsies: one of them included 32 biopsies reporting a recurrence rate of 53% [8] and another included 11 biopsies with a recurrence rate of 27% [9]. Regarding the incidence of IgAN recurrence, our results are similar to those in previous reports, despite the fact that our study included patients using modern immunosuppression. In most centres, the suspicion of IgAN recurrence is based on the presence of haematuria, proteinuria or a decline in renal function, being the recurrence rate between 12.5 and 50% [6]. The use of these criteria neglect cases with lanthanic IgAN. In fact, we found that 52% of the IgAN recurrences diagnosed by protocol biopsies were not accompanied by proteinuria or haematuria. Thus, protocol biopsies and immunofluorescence analysis constitute an essential tool for the diagnosis of recurrence, especially when it is clinically silent.

The choice of immunosuppressive drugs for the treatment of primary IgAN in patients at risk for progression remains uncertain [11]. The largest study on the risk of graft loss due to recurrent glomerulonephritis could not find any difference in the rate of graft loss due to recurrence when azathioprine and mycophenolate mofetil (MMF), cyclosporine and tacrolimus, sirolimus and prednisone were compared [2]. This study has limitations related to the registry data, thus the diagnosis of disease recurrence may not be accurate. We found the use of cyclosporine to be associated with a lower risk of recurrence, independently of the trough levels. Moreover, patients on cyclosporine may have lower MMF exposure because of a pharmacokinetic interaction [12]. Nevertheless, it has been recently reported that cyclosporine therapy may be effective in reducing proteinuria and regressing renal pathology in a subset of children with IgAN [13].

Recommended treatment for primary IgAN includes ACE-i or ARB [14]. In our study, only one-third of the patient population was using ACE-i or ARB, and this was seen equally in the recurrent as in the non-recurrent patients, which may explain at least in part the low level proteinuria detected in this cohort.

The use of induction therapy to prevent recurrence remains controversial. A retrospective study of 57 kidney allograft biopsies from 116 renal transplant patients suggested a protective effect from IgAN recurrence when thymoglobulin was used for induction and a deleterious effect with IL-2 R-mAb compared to no induction [15]. We found that 60% of the patients who received induction with thymoglobulin experienced recurrence compared with 27% of the patients without recurrence. Of note, the induction with thymoglobulin followed by a tacrolimus-based immunosuppression in this cohort was not due to a high immunological risk but was chosen by protocol. This issue remains speculative due to the small number of patients receiving induction therapy included in our study and the retrospective nature of the analysis.

Several reports linked specific types of HLA to IgAN, such as HLA-DR4, HLA-DQw4 and HLA-Bw35 and HLA-B8, DR3 [16–18]. This suggests that various HLA-associated genes might predispose to IgAN or be related to the disease progression [19]. In our study, we were not able to detect any HLA types particularly associated with recurrence. On the other hand, full DR match was significantly more frequent in patients without recurrence. However, recurrence has been reported to be 17% among zero-HLA mismatch in living donation [20]. Also, our study mainly
follow-up was 5 years [6, 29], but others reported that the in accordance with previous reports where the median this study was better in the early years after transplantation, recurrence rather than merely IgA deposition. moreover, our findings suggest that there was already IgAN to persistence of donor IgA deposits in the graft. Further-
ed we consider it unlikely that our recurrent cases were related
tocol biopsy and the concomitant complement deposition,
tory response. Taking into account both, the timing of pro-
osition in most cases, suggesting that there was already
investigation, recurrent IgAN was associated with C3 dep-
deposition of C3 was detected in only 19% [17]. In our
donors, the study revealed a latent mesangial deposition
IgAN was found in 4–8% [24–26]. A gradual resolution of
were already present in the donor in our study population.
arteriolar hyalinosis was not detected in biopsies with IgAN recurrence, and arteriolar hyalinosis [23]. We found that arteriolar hya-
in the recurrent patients, same as tubulointerstitial injury
AR, but others found that chronic rejection was less common
at assessed in the recurrent disease when assessed
glomerulonephritis.
References
2. Mulay AV, van Walraven C, Knoll GA. Impact of immunosuppres-
sive medication on the risk of renal allograft failure due to recurrent

including deceased donors and this may account for these discrepancies.

We observed that in over 65% of the cases with IgAN recurrence the light microscopic histology was normal, which is in accordance with the absence of clinical findings in 52% of the cases. It has been previously reported that biopsies from patients with IgAN recurrence showed milder glomerular changes compared to IgAN in native kidneys, although there may be more glomerular obsoles-
cence or interstitial fibrosis/tubular atrophy [21, 22]. The histological changes reported in the literature are highly de-
dependent on the time elapsed from transplantation. IgAN recur-
rence was not associated with acute or chronic rejection in our
study. A similar observation was reported previously regarding AR, but others found that chronic rejection was less common in the recurrent patients, same as tubulointerstitial injury and arteriolar hyalinosis [23]. We found that arteriolar hya-
inosis was not detected in biopsies with IgAN recurrence, while it was present in 31% of those without recurrence. We related it to donor age while we were not able to find any association between arteriolar hyalinosis and hypertension, diabetes, BMI or the use of cyclosporine in this cohort.

We cannot fully rule out the possibility that IgA deposits were already present in the donor in our study population. In autopsy studies from cases without known renal disease, IgAN was found in 4–8% [24–26]. A gradual resolution of IgA deposits was reported to occur within 45 days following transplantation [27, 28]. In a Japanese study of 0-h renal biopsies, where 87% of transplants were from living donors, the study revealed a latent mesangial deposition of IgA in 16% of its cases. Interestingly, in this study co-
deposition of C3 was detected in only 19% [17]. In our investigation, recurrent IgAN was associated with C3 dep-
osition in most cases, suggesting that there was already complement activation and therefore potential inflammatory response. Taking into account both, the timing of proto-
col biopsy and the concomitant complement deposition, we consider it unlikely that our recurrent cases were related to persistence of donor IgA deposits in the graft. Furthermore, our findings suggest that there was already IgAN recurrence rather than merely IgA deposition.

Kidney function in IgAN recurrent patients included in this study was better in the early years after transplantation, in accordance with previous reports where the median follow-up was 5 years [6, 29], but others reported that the differences in kidney function vanished when the follow-
up was prolonged [30]. Our finding could be explained, at
least in part, by older donor age and the higher incidence of DGF in non-recurrent patients. In fact, kidney function in patients without DGF revealed similar GFR in both recurrent and non-recurrent patients. Regarding allograft survival, recurrent IgAN was the cause of graft loss in 3% of the cases in our study. This is in accordance with previous reports based on registry data where the follow-up was 10 years [2, 4]. Of note, using a more accurate tool for the recognition of IgAN recurrence provided by protocol biopsies and immunofluorescence microscopy, we ob-
served a similar rate of graft loss already at 5 years. We can speculate that with a longer follow-up period, the im-
pact of graft loss in our population due to recurrent IgAN might be higher.

In conclusion, we observed that 32.3% of the cases with IgAN had recurrence of the primary disease when assessed by protocol biopsies during the first 2 years after transplanta-
tion. The histological diagnosis was not accompanied by abnormalities in the urinalysis in one-half of the patients. Full DR match and the use of cyclosporine were associated with non-recurrence of IgAN. This observation should be confirmed in a study including a larger number of cases focussing on the donor selection and the use of new immunosuppressive drugs. Although graft function is well preserved early after transplantation, the consequences of IgAN recurrence should be evaluated in a long-term follow-up.

Acknowledgements. This research was financially supported by grants from the ESOT, the Helsinki University Central Hospital research funds (EVO TYH2010206) and Red de Investigacio´n Renal (REDinREN, ISCIII 06/0016).

Conflict of interest statement. None declared.

Table 3. Use of immunosuppressive agents, ACE-i/ARB and statins

<table>
<thead>
<tr>
<th></th>
<th>All patients (N = 65)</th>
<th>Without recurrence (N = 44)</th>
<th>With recurrence (N = 21)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Induction with thymoglobin</td>
<td>10 (15.4%)</td>
<td>4 (9.1%)</td>
<td>6 (28.6%)</td>
<td>0.04</td>
</tr>
<tr>
<td>Induction with IL-2-R-mAb</td>
<td>8 (12.3%)</td>
<td>4 (9.1%)</td>
<td>4 (19%)</td>
<td>0.25</td>
</tr>
<tr>
<td>Cyclosporin</td>
<td>34 (52.3%)</td>
<td>27 (61.4%)</td>
<td>7 (33.3%)</td>
<td>0.03</td>
</tr>
<tr>
<td>Tacrolimus</td>
<td>28 (43.1%)</td>
<td>16 (36.4%)</td>
<td>12 (57.1%)</td>
<td>0.11</td>
</tr>
<tr>
<td>Rapamycin</td>
<td>5 (7.7%)</td>
<td>3 (6.8%)</td>
<td>2 (9.5%)</td>
<td>0.70</td>
</tr>
<tr>
<td>Mycophenolate mofetil</td>
<td>50 (82.9%)</td>
<td>40 (90.9%)</td>
<td>18 (85.7%)</td>
<td>0.52</td>
</tr>
<tr>
<td>Steroids</td>
<td>59 (90.8%)</td>
<td>41 (93.2%)</td>
<td>18 (85.7%)</td>
<td>0.33</td>
</tr>
<tr>
<td>ACE-i/ARB</td>
<td>18 (27.7%)</td>
<td>13 (29.5%)</td>
<td>5 (23.8%)</td>
<td>0.65</td>
</tr>
<tr>
<td>Statins</td>
<td>22 (33.8%)</td>
<td>15 (34.1%)</td>
<td>7 (33.1%)</td>
<td>0.95</td>
</tr>
</tbody>
</table>


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