Renal biopsy in liver transplant recipients

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Abstract

Background. Renal impairment post-liver transplant (LT) is often attributed to calcineurin inhibitors (CNIs). A renal biopsy can be a useful tool but may be complicated in LT recipients. We aimed to determine the clinical scenarios that prompted a decision to perform a renal biopsy in this patient population, to assess histological findings and evaluate patient management and survival and renal outcome.

Methods. Information on clinical variables and renal histology was extracted from single-centre prospectively compiled databases from 1996 onwards.

Results. Over 2100 adults received an LT in the time period studied, and 54 of these (35 males and 19 females) were referred for renal review. Of these, 43% underwent a renal biopsy. They had a higher creatinine ($P < 0.01$), a greater deterioration in creatinine over the year prior to review and were more likely to be nephrotic (both $P < 0.01$). Histological findings included hypertensive changes (44%), CNI nephrotoxicity (48%), IgA nephropathy (9%), membranoproliferative glomerulonephritis (17%), acute tubular necrosis (4%), crescentic glomerulonephritis (4%) and diabetic nephropathy (9%). Major bleeding complications occurred in 17%. Treatment changed in the majority but, it
was not significantly different in the two groups. Although initial renal function was worse in the biopsied group, final patient and renal survival did not differ between the two groups.

**Conclusion.** A renal biopsy is a valuable tool in those with renal insufficiency and/or proteinuria and haematuria but the benefits must be weighed against the relatively high complication rate in LT recipients.

**Keywords:** biopsy; histology; liver; renal; transplantation

**Introduction**

Renal dysfunction in liver transplant (LT) recipients is not uncommon, and development of chronic kidney disease (CKD) has been widely reported [1–3]. Calcineurin inhibitors (CNIs) form the backbone of immunosuppression regimens post-LT and have revolutionized graft and survival outcomes [4,5]. Unfortunately, they can cause both reversible and irreversible nephrotoxicity. The classical histological hallmarks of CNI nephrotoxicity include isometric cytoplasmic proximal tubular vacuolation, afferent arteriolar hyalinization, thrombotic microangiopathy, adventitial hyaline arteriolaripathy, striped interstitial fibrosis, tubular atrophy and focal segmental glomerulosclerosis [6,7]. They can also cause post-LT diabetes and hypertension, which could further exacerbate renal impairment [8,9].

Other potential aetiologies of CKD have been demonstrated in this patient population [10,11]. There may be a common pathophysiology underlying both the liver and renal diseases, as seen with hepatitis C-related cryoglobulinaemia and immune complex-mediated glomerulonephritis [12,13]. LTs have also been performed to correct the metabolic defects of hereditary amyloidosis and some of these patients may already have established renal damage by the time they come to transplantation [14,15]. A number of studies have reported renal biopsy findings from patients with normal renal function taken at the time of LT. They revealed that almost all had evidence of glomerular lesions including IgA nephropathy, mesangiocapillary glomerulonephritis and hepatic glomerulosclerosis [16–18]. These individuals did not have a worse renal outcome post-LT, but the propensity for those with hepatic disease to develop glomerular pathology should be borne in mind if renal impairment develops post-LT.

Recipients of solid organ transplants are increasingly being referred to renal services to establish the cause of a decrement in renal function and provide advice on future management and, in this setting, a kidney biopsy is useful and often necessary [19]. In LT recipients, a renal biopsy may not always be indicated or possible as the precipitant for the decline in renal function may be obvious or the biopsy may be considered high risk because of bleeding tendency [20]. In addition, these patients are already immunosuppressed, so it is important to question whether a biopsy will change patient management.

This study was performed to evaluate the spectrum of renal histological diagnoses seen post-LT, the clinical characteristics that prompted a decision to perform a biopsy, the complication rate, patient management post-referal/biopsy and patient and renal survival.

**Subjects and methods**

**Patient population**

The renal and liver departments’ prospectively compiled databases (1996 onwards) were interrogated retrospectively to identify LT recipients referred for renal review. There were no defined criteria for referral, which was left to the discretion of individual hepatologists. Those with a pre-existing renal diagnosis, such as those who had undergone a kidney biopsy prior to LT, were excluded.

**Immunosuppression**

Prior to 1995, the standard immunosuppression regimen was cyclosporine, prednisolone and azathioprine. Thereafter, tacrolimus replaced cyclosporine, which was administered with prednisolone. Maintenance serum 12-h trough cyclosporine and tacrolimus levels were 100 µg/L and 4–8 µg/L, respectively. Prednisolone was tapered from 20 mg to 5 mg over 3 months and withdrawn, where possible. However, if the underlying cause of end-stage liver disease was autoimmune, patients were maintained on 5 mg. Prednisolone, mycophenolate mofetil and sirolimus were used to facilitate CNI minimization.

**Clinical characteristics**

Clinical information on the following parameters was obtained at referral: age, gender, cause of end-stage liver disease, hepatitis C virus status, date of initial renal assessment and of LT and any history of diabetes or hypertension. Blood pressure and dipstick urinalysis findings were noted. The following laboratory parameters were identified: urea, creatinine, the Modification of Diet in Renal Disease glomerular filtration rate, bilirubin, aspartate aminotransferase, gamma-glutamyl transpeptidase, international normalized ratio, platelet count, albumin, cryoglobulins and autoimmune screen [21]. Any abnormal renal findings on radiological imaging were noted such as multiple renal cysts, cortical thinning, increased echogenicity, a discrepancy or reduction in renal size or collecting system dilatation. The change in creatinine, and glomerular filtration rate (GFR) over the year prior to referral, was determined. Renal biopsy histological findings were noted along with any serious complications and patient management before and after the renal review or biopsy. Individuals were followed up to determine patient survival from the time of their first LT. Renal survival was defined as a final GFR over 15 ml/min. Those who received a renal transplant or required chronic dialysis during the follow-up were deemed to have a GFR under 15 ml/min. Where patients developed acute kidney injury at the time of a final illness, the last stable creatinine was used to determine the final GFR.

**Renal biopsy**

The decision to perform a renal biopsy was made by individual nephrologists according to published guidelines [22]. The renal biopsies were percutaneous in all but one case, where a nephrectomy rather than a biopsy was performed, at the time of a combined liver and kidney transplant. Five of the biopsies were done in other institutions, so information was not available on the biopsy technique. In the remainder, a lower pole renal biopsy was obtained using real-time ultrasound guidance and a 16-gauge automated biopsy needle. Prior to the biopsy coagulopathy, thrombocytopenia or uncontrolled hypertension were excluded. Post-biopsy, patients were monitored for 24 h for complications by frequent evaluation of vital signs and urine [23]. If the biopsies were done at a different institution, the formal reports or slides were forwarded for review. Biopsies were embedded and stained with haematoxylin-eosin, silver methenamine, elastica van Gieson’s and periodic acid-Schiff stains. All were examined by light microscopy and 29% by electron microscopy. Immunofluorescence was performed in 43% and immunoperoxidase studies were performed in the remainder looking for deposition of IgG, IgA, IgM, C3, C1q, fibrinogen, kappa and lambda. The final histological diagnosis was determined in each case. The number of focally or completely sclerosed glomeruli was scored as a percentage of the total number of glomeruli seen on each biopsy. Interstitial fibrosis and tubular atrophy were also scored as a percentage of...
Table 1. Aetiology of end-stage liver disease in all patients referred for a renal review

<table>
<thead>
<tr>
<th>Causes of liver disease</th>
<th>Percentage of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcoholic liver disease</td>
<td>24%</td>
</tr>
<tr>
<td>Viral hepatitis</td>
<td>22%</td>
</tr>
<tr>
<td>Primary biliary cirrhosis</td>
<td>11%</td>
</tr>
<tr>
<td>Viral hepatitis and alcoholic liver disease</td>
<td>7%</td>
</tr>
<tr>
<td>Fulminant hepatic failure</td>
<td>7%</td>
</tr>
<tr>
<td>Chronic active hepatitis</td>
<td>6%</td>
</tr>
<tr>
<td>Cryptogenic cirrhosis</td>
<td>6%</td>
</tr>
<tr>
<td>Primary sclerosing cholangitis</td>
<td>4%</td>
</tr>
<tr>
<td>Biliary atresia</td>
<td>4%</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>2%</td>
</tr>
<tr>
<td>Carcinoïd syndrome</td>
<td>2%</td>
</tr>
<tr>
<td>Nodular regenerative hyperplasia</td>
<td>2%</td>
</tr>
<tr>
<td>Vanishing bile duct syndrome</td>
<td>2%</td>
</tr>
<tr>
<td>Budd–Chiari syndrome</td>
<td>2%</td>
</tr>
</tbody>
</table>

Statistical analysis

Univariate logistic regression analysis was used to determine the clinical variables that were significantly different in individuals who underwent a biopsy compared with those who did not, and significant *P*-values (<0.05) are displayed. Continuous variables are displayed as mean ± standard deviation or as a percentage. Kaplan–Meier analysis was performed to evaluate patient and renal survival from the time of first LT in those who underwent a biopsy compared with those who did not, using the log-rank test. Patients were censored at the time of death or a GFR under 15 mL/min, respectively, or at the end of the follow-up. Statistical analysis was performed using SPSS 14.0.

Results

Patient population, immunosuppression and clinical characteristics

Over 2100 adults received a LT in the time period studied, and 54 of these (35 males and 19 females) were referred for renal review. Twenty-three (43%) of these underwent a renal biopsy. Causes of end-stage liver disease are illustrated in Table 1. There was no difference in aetiology between those who were biopsied and those who were not. The mean time post-LT to renal referral was 5.3 ± 4.6 years. Details on the patterns of CNI immunosuppression at the time of referral are illustrated in Tables 2 and 3. There were several significant differences in the clinical characteristics of both groups (Table 2). Of note, none of the patients with viral hepatitis had evidence of cryoglobulinaemia. Those who were biopsied had significantly worse renal function, had a greater deterioration in renal function over the year prior to review and were more likely to have nephrotic range proteinuria (Table 2).

Renal histology

The average number of glomeruli seen was 25. Histology findings, with some biopsies having more than one pathology, were as follows: hypertensive vascular changes (44%), CNI nephrotoxicity (48%), IgA nephropathy (9%), membranoproliferative glomerulonephritis (17%), acute tubular necrosis (4%), focal and proliferative glomerulonephritis with crescents (4%) and diabetic nephropathy (9%). The patients with IgA nephropathy did not have alcohol-related liver disease. Electron microscopy confirmed that membranoproliferative glomerulonephritis was type 1 in two out of the four cases, and there were no immune deposits seen on a third. The fourth patient did not have electron microscopy but had hepatitis C, so most likely had type 1. Acute tubular necrosis was demonstrated on one biopsy performed because of failure to recover renal function.

Table 2. Clinical characteristics at the time of initial renal review in patients who underwent a renal biopsy versus those who did not

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Biopsy</th>
<th>No biopsy</th>
<th><em>P</em>-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>50.0 ± 14.5</td>
<td>56.5 ± 10.5</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>65%</td>
<td>65%</td>
<td>NS</td>
</tr>
<tr>
<td>Female</td>
<td>35%</td>
<td>35%</td>
<td>NS</td>
</tr>
<tr>
<td>Calcium inhibitor use</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cyclosporin</td>
<td>70%</td>
<td>58%</td>
<td>NS</td>
</tr>
<tr>
<td>Tacrolimus</td>
<td>26%</td>
<td>35%</td>
<td>NS</td>
</tr>
<tr>
<td>Botha</td>
<td>4%</td>
<td>6%</td>
<td>NS</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>120.5 ± 26.5</td>
<td>118.3 ± 18.5</td>
<td>0.01</td>
</tr>
<tr>
<td>Proteinuria (g/24 h)</td>
<td>3.6 ± 2.9</td>
<td>1.3 ± 1.8</td>
<td>0.01</td>
</tr>
<tr>
<td>Haematuria (dipstick urinalysis)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td>69%</td>
<td>87%</td>
<td>NS</td>
</tr>
<tr>
<td>+1</td>
<td>25%</td>
<td>9%</td>
<td>NS</td>
</tr>
<tr>
<td>+2</td>
<td>0%</td>
<td>4%</td>
<td>NS</td>
</tr>
<tr>
<td>+3</td>
<td>6%</td>
<td>0%</td>
<td>NS</td>
</tr>
<tr>
<td>Urea (mmol/L)</td>
<td>17.1 ± 8.3</td>
<td>12.2 ± 4.8</td>
<td>0.02</td>
</tr>
<tr>
<td>Creatinine (µmol/L)</td>
<td>277.5 ± 143.5</td>
<td>196.0 ± 66.3</td>
<td>0.02</td>
</tr>
<tr>
<td>Glomerular filtration rate (mL/min)</td>
<td>22.9 ± 15.2</td>
<td>26.5 ± 10.6</td>
<td>NS</td>
</tr>
<tr>
<td>Bilirubin (µmol/L)</td>
<td>9.8 ± 4.9</td>
<td>11.0 ± 8.8</td>
<td>NS</td>
</tr>
<tr>
<td>Aspartate aminotransferase (IU/L)</td>
<td></td>
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<tr>
<td>Gamma-glutamyl transpeptidase (IU/L)</td>
<td>289.4 ± 408.6</td>
<td>161.6 ± 343.2</td>
<td>NS</td>
</tr>
<tr>
<td>Alkaline phosphatase (IU/L)</td>
<td>225.7 ± 274.1</td>
<td>182.9 ± 169.0</td>
<td>NS</td>
</tr>
<tr>
<td>International normalized ratio</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Albumin (g/L)</td>
<td>33.1 ± 6.1</td>
<td>38.4 ± 5.4</td>
<td>0.01</td>
</tr>
<tr>
<td>Platelet (10⁹/L)</td>
<td>196.5 ± 157.1</td>
<td>181.7 ± 68.4</td>
<td>NS</td>
</tr>
<tr>
<td>Renal ultrasound findings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>53%</td>
<td>74%</td>
<td>NS</td>
</tr>
<tr>
<td>Abnormal</td>
<td>47%</td>
<td>26%</td>
<td>NS</td>
</tr>
<tr>
<td>Time from first transplant to renal referral/biopsy (years)</td>
<td>5.1 ± 4.3</td>
<td>5.5 ± 5.1</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are displayed as mean ± standard deviation or as a percentage. NS, not significant.

*aBoth patients were switched from cyclosporin to tacrolimus or visa versa during the follow-up.
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4 months after drug toxicity from paracetamol. A selection of these histological findings is illustrated in Figure 1. On average, there was 39% glomerular sclerosis, 45% interstitial fibrosis, 44% chronic tubular atrophy and 65% of biopsies showed moderate to severe vascular damage.

Complications post-renal biopsy

Of the 23 patients, information was available regarding post-biopsy complications in 18 (78%). There were no complications seen in 15 (83%) of these but bleeding requiring either transfusion or arteriography with embolization of renal vessels was required in the remaining 3 (17%). One of these three patients had a platelet count of $60 \times 10^9/L$, at the time of the biopsy, but otherwise clotting parameters were normal. Intravenous desmopressin was routinely given to all individuals with impaired renal function prior to the biopsy and patients had controlled hypertension.

Patient management pre- and post-renal review or biopsy

CNI was minimized before the renal review or biopsy in 17 (31%) and after in 7 (13%) of cases. The corresponding values for CNI cessation were 10 (19%) and 13 (24%). In order to facilitate this, 37 (69%) of individuals were started on mycophenolate mofetil and 4 (7%) on sirolimus. All patients treated with sirolimus had $<1$ g/L proteinuria prior to initiation of this agent, but the proteinuria rose from a mean of 0.6 to 1.8 g/L with treatment. Prednisolone and azathioprine were started or continued in 39 (72%) and 8 (15%) patients, respectively. The pre- and post-renal review or biopsy, ACE or angiotensin receptor blockers (ARB) were used in 6 (11%) and 32 (59%) patients, respectively. The specific immunosuppression regimens are detailed in Table 3. None of these treatment interventions differed significantly between the two groups and none had any influence on the change in GFR from the time of initial presentation to the final stable GFR. In the one individual with crescentic focal and proliferative glomerulonephritis, cyclophosphamide and steroids were used for a period of 1 month but treatment was limited because of recurrent
to receive a renal transplant during the follow-up (including two combined liver and kidney transplants). Of these, four had stage 3 CKD and one had developed acute renal failure at the end of the follow-up.

**Discussion**

While CNI nephrotoxicity is a common cause of renal dysfunction post-LT, it is not the only potential aetiology. The examination of renal histology is the only way to determine the exact cause, but it is not always indicated or possible. In this study, we have determined the clinical characteristics that prompted a decision to perform a renal biopsy post-LT, the variety of histological diagnoses seen along with changes to patient management and patient and renal survival post-renal review or biopsy.

Overall, <3% of LT recipients were referred for a renal opinion. This is low when considering that 18% of patients have been reported to develop a GFR <30 mL/min by 5 years post-LT [3]. One reason for the low referral rate is that ours is a tertiary referral centre with many LT recipients having their renal care locally. Clinical guidelines suggest that a renal specialist see all patients with a GFR <30 mL/min, nephrotic syndrome or malignant hypertension. A renal review should also be sought in those with a GFR <60 mL/min where there is a progressive deterioration in the GFR or when the dipstick urinalysis reveals associated haematuria or proteinuria. In addition, renal advice should be sought where there is evidence of complications of CKD such as anaemia, hyperkalaemia, renal bone disease or uncontrolled hypertension [24]. Failure to refer patients in a timely fashion delays introduction of strategies to potentially slow the progression of CKD. These include minimization of CNI exposure, aggressive control of hypertension particularly with ACE and ARBs and investigation and treatment of the complications of CKD [25]. Early renal referral is also required, as CKD is associated with increased morbidity and mortality [3]. In this study, the 10-year survival rate was lower than that described in another study of 4000 consecutive LTs where the equivalent rate was 57% [26]. This is perhaps due to the impact of renal impairment on mortality.

The clinical variables that prompted a decision to perform a kidney biopsy were a more rapid progression in renal dysfunction over the previous year, a greater degree of proteinuria and hypoalbuminaemia and the severity of renal impairment. The characteristic clinical features of CNI nephrotoxicity are mild proteinuria, bland urinary sediment, slowly progressive renal dysfunction and hypertension [27,28]. Patients were therefore more likely to undergo a renal biopsy if they departed from this clinical phenotype.

We demonstrated that the aetiology of CKD is multifactorial with many individuals having more than one lesion on biopsy. In the majority, CKD could be attributed to hypertension, CNI toxicity or both but in 48% of cases, other pathologies were seen. Studies evaluating renal biopsy findings post-LT are rare. The largest study to date comprised 26 patients, and similar clinical characteristics were seen to those described in this study. Patients were biopsied at ~5 years post-LT with a mean creatinine of

![](image)

**Fig. 2.** Kaplan–Meier survival curves are displayed showing patient and renal survival from the time of their first liver transplant in those who underwent a renal biopsy compared with those who did not. Renal survival is defined as a glomerular filtration rate over 15 mL/min based on each patient last stable creatinine at death or at the end of the follow-up. The log-rank test was used to compare the strata.

**Patient and renal survival**

Mean patient survival from the time of each patient’s first LT was not significantly different in the biopsied versus non-biopsied patients at 7.9 ± 5.2 versus 8.9 ± 4.9 years (Figure 2). One-, 5- and 10-year post-LT survival rates were 100%, 73.6% and 37.7%, respectively. Twenty-four (44%) patients died, and the cause of death was unknown in 10. The remainder died of multi-organ failure due to sepsis or haemorrhage. The mean length of the renal follow-up from the time of referral was 3.2 ± 2.5 years. There was no overall difference in renal survival from the time of first LT between the two groups (Figure 2). Mean serum creatinine (213.4 ± 135.8 versus 257.6 ± 194.5 µmol/L) and GFR (26.7 ± 18.4 versus 25.4 ± 12.7 mL/min) were not significantly different between the two groups at death or the end of the follow-up. Ten (19%) patients developed acute kidney injury at the end of the follow-up and did so in the setting of a final fatal illness. Five (9%) patients went on

neutropaenia and this patient did not recover renal function. Two of the patients were noted to have membranoproliferative glomerulonephritis and were hepatitis C RNA positive; however, neither were treated with anti-virals because of contraindications to interferon therapy.

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212 μmol/L. The principal aetiologies were CNI nephrotoxicity, diabetic nephropathy, thrombotic microangiopathy and tubular changes attributed to hydroxyethyl starch use [29]. Smaller case series have found similar lesions in addition to focal segmental glomerulosclerosis, membranous and IgA nephropathy [30–33]. Renal biopsies have also been performed to assess progression of hereditary amyloidosis both pre- and post-LT [34].

In some individuals, particularly those with poorly functioning LTs, there may be evidence of coagulopathy and/or ascites, making a biopsy risky and technically difficult. Collateral circulation in the region of the kidney has been reported which could potentially result in haemorrhage post-procedure [20]. In this study, there was a high incidence of bleeding complications (17%) requiring either transfusion or arteriography. Normally, a renal biopsy is a safe procedure, and the risk of such complications in non-LT patients has been reported to be in the region of 0.4–2.4% [35–37].

To minimize bleeding complications, patients should not be biopsied with uncontrolled coagulopathy or hypertension [23]. In addition, Doppler ultrasound may be useful in excluding collateral circulation in the region of the kidney. Depending on local expertise, performing trans-jugular renal biopsies could reduce the major complication rate, in this patient population [38,39]. In one study with 800 patients, the risk of severe complications was similar in those who underwent either a trans-jugular biopsy or a percutaneous renal biopsy. This was despite the fact that those who had the trans-jugular biopsies were selected because of greater bleeding tendency [40]. This suggests that there may be a reduced risk of bleeding complications with this approach.

A renal biopsy should only be undertaken if the findings will influence treatment or help determine renal prognosis. In one study, where trans-jugular biopsies were performed in patients with cirrhosis, biopsy findings influenced management in 38% of cases [38]. We found that, while treatment changed after biopsy or renal review in the majority of cases, frequently changes had already been commenced prior to this. Overall, there was no significant difference in therapy between those who were biopsied and those who were not in terms of ACE/ARB use and CNI minimization. Of note, there was no significant difference in overall patient survival or renal outcome between the two groups. However, it should be noted that those who underwent a renal biopsy had significantly poorer renal function compared with the other group to begin with. So, although there was no significant difference in renal function at the end of the follow-up, it is possible that the biopsy findings influenced patient management sufficiently to stabilize renal function in that group.

Despite the relatively high complication rate, a renal biopsy is still an important tool in certain LT recipients. In addition to those with renal impairment and/or haematuria and proteinuria, hepatitis CRNA-positive patients with proteinuria disproportionately higher than would be expected with CNI nephrotoxicity should be biopsied to exclude underlying glomerulonephritis. These individuals may benefit from anti-viral therapy [41–43]. Also, as demonstrated in one patient in this study with crescentic glomerulonephritis, those with rapidly progressive renal failure in the presence of an active urinary sediment require a renal biopsy. Another scenario would be a patient with a nephrotic range proteinuria. If an underlying glomerulonephritis were found on biopsy, then increasing immunosuppression would be justified, whereas with CNI toxicity the opposite treatment would be required. Randomized trials have shown that CNI-sparing regimens with mycophenolate mofetil or sirolimus can potentially improve renal function [44–47]. A presumptive diagnosis of CNI toxicity can often be made but some clinicians may be uncomfortable changing immunosuppression in patients without renal biopsy confirmation, given the potential risk of LT rejection with mycophenolate monotherapy [47,48].

There are some limitations to this study. The numbers of patients are small, and it is a retrospective review. We cannot give a true representation of the percentage of LT recipients referred for renal review as not all the patients were followed up at our institution. In addition, there was inadequate database documentation of minor complications post-biopsy, such as microscopic or frank haematuria.

CNI nephrotoxicity is a common cause of renal impairment in LT recipients, but it is not the only cause. We have outlined the clinical features that prompted a decision to perform a renal biopsy and have also illustrated the spectrum of potential aetiologies. Despite having a renal biopsy, patient management and survival did not differ significantly from those patients who did not undergo a biopsy. There was no difference in renal survival although function was significantly worse in the biopsy group at initial referral. Serious bleeding complication rates were high, so this must be borne in mind when deciding to perform a renal biopsy in LT recipients with renal impairment and/or proteinuria and haematuria.

Conflict of interest statement. None declared.

References


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