Long-term outcomes of patients with light chain amyloidosis (AL) after renal transplantation with or without stem cell transplantation

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Abstract

Background. Recent advances in the treatment of immunoglobulin light chain amyloidosis (AL) have dramatically improved survival. Kidney transplantation (KTx) has become more common but the long-term outcomes remain unknown and it is the objective of this study.

Methods. Nineteen patients with AL underwent living (n = 18) or deceased (n = 1) KTx at our institution from 1999 to 2008 (median age 57 years, six women). The primary endpoints were patient and kidney allograft survival and recurrence of AL in the allograft. The secondary end point was kidney transplant rejection. Outcome data were stratified according to three treatment modalities: renal transplantation followed by autologous stem cell transplantation (ASCT) (Group 1, n = 8), ASCT followed by renal transplantation (Group 2, n = 6) and renal transplantation after complete remission achieved with nonmyeloablative therapy (Group 3, n = 5).

Results. The median follow-up was 41.4 months. At the time of study, 79% were still alive. Median graft survival did not differ from median overall survival. There was no difference in survival rates between the treatment groups. Five patients had a cellular rejection. Two of the three patients with a rejection in Group 1 died but neither patient with rejection in Groups 2 and 3. Recurrent amyloidosis was diagnosed by biopsy in one patient in Group 2 (preceding ASCT) and in another patient in Group 3.

Conclusions. KTx can be successfully performed in AL patients in complete hematologic response and meet the usual KTx selection criteria. Outcomes appear similar whether hematologic response was achieved with ASCT or by nonmyeloablative therapy.

Keywords: amyloidosis; AL amyloidosis; kidney transplantation; monoclonal; recurrence

Introduction

Immunoglobulin light chain amyloidosis (AL) is a plasma cell dyscrasia characterized by the production of monoclonal light chain fragments [1]. These light chain fragments undergo conformational changes into β-sheets resulting in fibril formation and deposition in multiple organs. The kidney is the most commonly involved organ [2]. Proteinuria is present in 73% of the patients with 28% presenting with nephrotic syndrome. Almost half of the patients have renal insufficiency at the time of presentation. Another study showed that 42% of patients presenting with renal involvement will eventually require renal replacement therapy [3]. The median survival, after initiation of dialysis, was 10.4 months in this study.

Over the past decade, tremendous advances have been made in the treatment of AL. The first major advance was the introduction of high-dose chemotherapy followed by autologous stem cell transplantation (ASCT) [4]. Median survival tripled from 18 months with melphalan and prednisone to 4.6 years [5]. More recently, melphalan and dexamethasone has shown similar hematologic and organ responses as ASCT [6]. In a randomized controlled trial, melphalan and dexamethasone were found to be better tolerated in the high-risk AL patients [7]. Activity against AL has also been reported with novel agents [8–10]. Longer follow-up, however, is needed to fully evaluate their efficacy.

As survival in these patients improves, those that develop renal failure are now faced with a longer time on dialysis. The early experience of kidney transplantation in AL patients was complicated by graft loss due to disease recurrence and early death of the patient [11,12]. However, with modern treatments capable of producing long-term complete hematologic response, kidney transplantation in AL patients has become more feasible [13,14]. We have previously reported our experience with patients receiving kidney transplantation prior to ASCT [14]. This report up-
dates our experience of kidney transplantation after achievement of complete hematologic response via other treatments.

Materials and methods

Study population

All patients with AL who underwent living (n = 18) or deceased (n = 1) kidney transplantation at our institution from 1999 to 2008 were included in this study. Pertinent clinical and laboratory data and information on kidney or bone marrow transplantation, treatment and follow-up were extracted from patient records. All patients had authorized the use of their medical records for research in accordance with Minnesota privacy statutes, and the study was approved by the Institutional Review Board at the Mayo Foundation in accordance with the Declaration of Helsinki and the Health Insurance Portability and Accountability Act guidelines.

All patients underwent baseline evaluation of their amyloidosis including bone marrow biopsy, serum and urine electrophoresis and immunofixation and light chain serum concentration analysis. Amyloidosis was identified in biopsy specimens by both characteristic electron microscopy patterns and positive Congo red staining. The subtyping of amyloidosis into AL and AA (serum amyloid A) amyloidosis was done by immunofluorescence staining of κ and λ light chains and immunoperoxidase staining for serum amyloid A. Renal function, i.e. glomerular filtration rate, was estimated by the Modification of Diet in Renal Disease formula [15].

Kidney transplantation KTx was performed in accordance to standard listing criteria from United Network for Organ Sharing (UNOS). Four patients with chronic kidney disease Stage V underwent preemptive KTx before initiation of hemodialysis. All patients had a good Eastern Cooperative Oncology Group (ECOG) performance status (0–1) and limited cardiac involvement. The initial strategy was to perform KTx before ASCT (Group 1, n = 8). The order was then changed to ASCT first with the goal to achieve complete hematologic response prior to KTx (Group 2, n = 6). The remaining patients underwent KTx after complete remission with nonmyeloablative therapy alone (Group 3, n = 5). ASCT was not pursued in the latter group because of sustained remission, patient preference or precluding infections.

Stem cell mobilization was accomplished with saeragramostim (granulocyte-macrophage colony-stimulating factor) or filgrastim (granulocyte colony-stimulating factor) (5 μg/kg/day). Apheresis was performed until a minimum of 2 × 10⁹/kg of CD34⁺ cell was collected. All patients prior to the ASCT underwent conditioning with high-dose melphalan (140–200 mg/m²). Engraftment was defined as the amount of time to achieve 500 × 10⁹ neutrophils for three consecutive days and 50000 × 10⁹ platelets without platelet transfusion for 7 days.

Hematological and renal response to therapy was assessed by criteria from the consensus opinion of the 10th International Symposium on Amyloid and Amyloidosis [16]. Complete hematologic response requires absence of monoclonal protein in the serum and urine and normal free light chain ratio. Renal allografts were biopsied as per the usual kidney transplant protocol or as indicated by a decline in renal function or an increase in proteinuria. These biopsy samples were processed for light microscopy and immunofluorescence. Recurrences of amyloidosis were confirmed by electron microscopic examination of the allograft biopsy. Time to end-stage kidney disease (ESRD) was calculated from time of diagnosis to initiation of dialysis. Overall survival was calculated from the day of kidney transplantation and diagnosis of AL.

Statistical analysis

Variables were presented as median (range). Long-term survival of the patients and their renal allograft were graphically displayed using the Kaplan–Meier method and group comparisons were made using the log rank test. Other group comparisons were made using analysis of variance or rank summed test for normally or not normally distributed continuous variables, respectively. All statistical calculations were performed using SPSS 10.1 statistical software (SPSS Science, Chicago, IL) and Microsoft Excel (Microsoft Corporation, Redmond, WA). Significance was considered for P < 0.05.

Results

Patient population

A total of 19 patients with AL amyloidosis who underwent kidney transplantation were included in this study. The median age of the study population was 57 years (range 35–67 years). Thirteen patients (68%) were men. The hematological and renal characteristics of the study population are outlined in Tables 1 and 2, respectively.

Ten patients (53%) had a monoclonal protein present in the serum (IgG-κ, n = 1; IgD-κ, n = 1; IgG-κ, n = 1; IgD-κ, n = 1; monoclonal λ, n = 4; monoclonal κ, n = 2). Urine protein electrophoresis with immunofixation showed monoclonal λ in 10 patients, monoclonal κ was present in 5 patients and biclonal κ and λ in 1 patient. Extra-renal involvement was identified in 12 patients. Involvement of the heart, gastrointestinal tract and the liver was found in nine, four and two patients, respectively. Before renal transplantation, 13 patients were on hemodialysis and 1 patient was on peritoneal dialysis. Of those who were not on renal replacement therapy, the median creatinine serum concentration was 4.4 μg/dL (range 3.4–6.3 μg/dL).

Post-kidney transplant courses

In total, 18 patients underwent living donor renal transplantation (13 from living-related donors and 5 from living-unrelated donors). One patient received a deceased donor renal transplant. The initial immunosuppressive regimen included tacrolimus (n = 17), cyclosporine A (n = 2) or mycophenolate (n = 12) in addition to prednisone (Table 2). In the majority of patients, allograft biopsies were performed at 4 months, 1 and 2 years post-KTx. The median follow-up time was 41.4 months. At the time of study, 79% were still alive. The median graft survival did not differ from median overall survival. Neither median overall survival assessed from day of kidney transplantation (Figure 1) nor day of diagnosis of AL (Figure 2) had been reached. At the last follow-up, the median estimated glomerular filtration rate was 51.5 (10–75) mL/min/1.73m² (Table 2). Five patients had a documented cellular rejection of the kidney transplant. Two of three patients (#7 and #8) with cellular rejection in Group 1 died but neither patient with a rejection in the Groups 2 and 3 have died. Renal allograft loss was not encountered other than in those patients with fatal outcomes (see below).

Recurrent disease in the renal allograft

Amyloidosis was diagnosed by per-protocol renal transplant biopsies in one patient in Group 1 (preceding ASCT) and in another patient in Group 3. The patient (#1) in Group 1 had a positive amyloid-biopsy status prior to ASCT, but no progression of the disease was demonstrated on the subsequent surveillance biopsies after ASCT. One patient (#16) in Group 3 had recurrence of the disease 52 months after the kidney transplantation and received melphalan and dexamethasone. The latest renal allograft biopsy was performed with 21 ± 17 months for the entire cohort. Stratifying according to the treatment group was
and uniformly fatal. Even as recent as 1999, the survival
termed
the disease process that resulted from these deposits
similar to cellulose when exposed to iodine [2]. Historically,

Discussion

Even though descriptions date back to the early 1600s, it
was Rudolph Virchow in 1854 who adopted the term
‘amyloid’ to refer to tissue deposits that stained in a manner
similar to cellulose when exposed to iodine [2]. Historically,
the disease process that resulted from these deposits
termed ‘amyloidosis’ has been considered untreatable and
uniformly fatal. Even as recent as 1999, the survival
rates of AL were 51%, 16% and 5% at 1, 5 and 10 years,
respectively [17]. However, the development of new treat-
ment approaches, in particular ASCT, melphalan and dexam-
ethasone, has improved the outlook for many individuals
with these disorders [4,6]. In one of the largest series on
ASCT, a 5-year survival of 82% was estimated for AL pa-
tients with a complete hematologic response [4].

Renal involvement is common in AL amyloidosis and
frequently leads to renal failure [2,3]. Patients with ad-
vanced renal dysfunction not yet requiring dialysis are at
increased risk for treatment-related mortality following
ASCT [18]. On the other hand, the risk is reduced when
the patient undergoes ASCT after dialysis has been initiated
[13]. As a result, two separate strategies have been used in
order to insure adequate treatment of the AL in these pa-
tients. In one strategy, renal function was restored prior to
ASCT [18]. On the other hand, the risk is reduced when

Fatal outcomes

All renal allograft losses were the result of patient death.
Two patients in Group 1 died. One patient (#7) presented
to the hospital with candida esophagitis and died of acute
pulmonary embolism ~9 months after the transplant. The
second patient (#8) died 8 months after renal transplantation
due to cytomegalovirus (CMV) infection and post-transplant
lymphoproliferative disorder. In Group 2, one patient (#10),
who had undergone cardiac transplantation prior to ASCT
and kidney transplant, died 41 months after the kidney
transplantation from a stroke. Cardiac transplant and gastro-
intestinal biopsies in this patient demonstrated amyloid
(retinal biopsy not performed) and she opted not to pursue
further therapy. In Group 3, one patient (#15) developed
failure to thrive soon after the kidney transplantation along
with progressive anorexia, nausea, vomiting and diarrhea.
He opted to return to his home country where he died, 1.5
months after kidney transplant.

25 ± 24 months for Group 1, 19 ± 9 months for Group 2
and 14 ± 8 months for Group 3.

Table 1. Hematological characteristics of the study population with AL amyloidosis undergoing renal transplantation

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Patient demographics</th>
<th>Extra-renal involvement</th>
<th>Initial treatment</th>
<th>Hematologic response</th>
<th>Relapse</th>
<th>Relapse therapy</th>
<th>Stem cell tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>52 years male</td>
<td>Cardiac</td>
<td>L-PAM</td>
<td>CR</td>
<td>Positive amyloid on previous renal bx but no progression after ASCT</td>
<td>None</td>
<td>After renal tx</td>
</tr>
<tr>
<td>2.</td>
<td>64 years male</td>
<td>None</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>After renal tx</td>
</tr>
<tr>
<td>3.</td>
<td>60 years female</td>
<td>GI</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>After renal tx</td>
</tr>
<tr>
<td>4.</td>
<td>64 years female</td>
<td>None</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>After renal tx</td>
</tr>
<tr>
<td>5.</td>
<td>50 years male</td>
<td>Cardiac</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>After renal tx</td>
</tr>
<tr>
<td>6.</td>
<td>61 years female</td>
<td>None</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>After renal tx</td>
</tr>
<tr>
<td>7.</td>
<td>58 years male</td>
<td>None</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>After renal tx</td>
</tr>
<tr>
<td>8.</td>
<td>56 years male</td>
<td>Cardiac</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>After renal tx</td>
</tr>
<tr>
<td>9.</td>
<td>62 years female</td>
<td>Cardiac and Hepatic</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>Before renal tx</td>
</tr>
<tr>
<td>10.</td>
<td>55 years female</td>
<td>Cardiac and GI</td>
<td>L-PAM</td>
<td>CR</td>
<td>Positive amyloid on cardiac allograft bx</td>
<td>None</td>
<td>Before renal tx</td>
</tr>
<tr>
<td>11.</td>
<td>57 years male</td>
<td>GI</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>Before renal tx</td>
</tr>
<tr>
<td>12.</td>
<td>37 years male</td>
<td>Hepatic</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>Before renal tx</td>
</tr>
<tr>
<td>13.</td>
<td>35 years female</td>
<td>Cardiac</td>
<td>L-PAM</td>
<td>PR</td>
<td>Positive λ light chain Revlimid</td>
<td>None</td>
<td>Before renal tx</td>
</tr>
<tr>
<td>14.</td>
<td>53 years male</td>
<td>None</td>
<td>L-PAM</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>Before renal tx</td>
</tr>
<tr>
<td>15.</td>
<td>57 years male</td>
<td>None</td>
<td>Thal + DEX</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>16.</td>
<td>51 years male</td>
<td>None</td>
<td>VBMCP</td>
<td>CR</td>
<td>Positive Amyloid on Kbx L-PAM and DEX</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>17.</td>
<td>60 years male</td>
<td>Cardiac and GI</td>
<td>L-PAM + DEX</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>18.</td>
<td>47 years male</td>
<td>Cardiac</td>
<td>L-PAM + DEX</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>19.</td>
<td>67 years female</td>
<td>Cardiac</td>
<td>L-PAM + DEX</td>
<td>CR</td>
<td>Negative</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

*GI, gastrointestinal tract; L-PAM, melphalan; Thal, thalidomide; DEX, dexamethasone; VBMCP, vincristine, bcnu, melphalan, cyclophosphamide, prednisone; CR, complete remission; PR, partial remission; Kbx, kidney biopsy; tx, transplantation.

Discussion

Even though descriptions date back to the early 1600s, it
was Rudolph Virchow in 1854 who adopted the term
‘amyloid’ to refer to tissue deposits that stained in a manner
similar to cellulose when exposed to iodine [2]. Historically,
the disease process that resulted from these deposits
termed ‘amyloidosis’ has been considered untreatable and
uniformly fatal. Even as recent as 1999, the survival
rates of AL were 51%, 16% and 5% at 1, 5 and 10 years,
respectively [17]. However, the development of new treat-
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ethasone, has improved the outlook for many individuals
with these disorders [4,6]. In one of the largest series on
ASCT, a 5-year survival of 82% was estimated for AL pa-
tients with a complete hematologic response [4].

Renal involvement is common in AL amyloidosis and
frequently leads to renal failure [2,3]. Patients with ad-
vanced renal dysfunction not yet requiring dialysis are at
increased risk for treatment-related mortality following
ASCT [18]. On the other hand, the risk is reduced when
the patient undergoes ASCT after dialysis has been initiated
[13]. As a result, two separate strategies have been used in
order to insure adequate treatment of the AL in these pa-
tients. In one strategy, renal function was restored prior to
ASCT [18]. On the other hand, the risk is reduced when

ASCT. Similarly, Sanada et al. [20] described a case of regression of amyloid deposits in the native kidney biopsy 2 years after ASCT. Another patient in our series had recurrence of disease 52 months after the kidney transplantation and received melphalan and dexamethasone successfully. A third patient with recurrent disease in the cardiac transplant could have had recurrent disease in the renal allograft as well but he declined renal biopsy. Overall, our study appears to show that recurrent disease in renal allograft is related to the hematological response: patients with a complete hematologic response have a lower probability of relapse after kidney transplantation.

Overall, there was no statistically significant difference in the rate of rejection between the groups. However, there was a tendency in the patients who had undergone kidney transplantation first to present with more episodes of acute cellular rejection. We cannot make final conclusions, but this could be related to the need to withhold mycophenolate for a short period of time to allow faster engraftment after ASCT. This hypothesis would need further assessment in future studies.

We would like to point out the retrospective nature and the small sample size as limitations of the study. The study is insufficiently powered to allow comparisons of the treatment strategies or any conclusion whether life expectancy was improved by kidney transplantation. This hypothesis would need further assessment in future studies.

Table 2. Renal characteristics of the study population with AL amyloidosis undergoing renal transplantation

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Initial treatment</th>
<th>Renal tx type</th>
<th>Renal tx survival</th>
<th>Renal tx rejection</th>
<th>Follow-up, eGFR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Cellcept; Prograf; PDN</td>
<td>LRD</td>
<td>103.9 months</td>
<td>None</td>
<td>47</td>
</tr>
<tr>
<td>2</td>
<td>Thymo; CyA; Cellcept; PDN</td>
<td>LURD</td>
<td>95.1 months</td>
<td>None</td>
<td>63</td>
</tr>
<tr>
<td>3</td>
<td>Thymo; Prograf; PDN</td>
<td>LRD</td>
<td>83.7 months</td>
<td>Borderline</td>
<td>47</td>
</tr>
<tr>
<td>4</td>
<td>Thymo; Prograf; PDN</td>
<td>LRD</td>
<td>78.3 months</td>
<td>None</td>
<td>48</td>
</tr>
<tr>
<td>5</td>
<td>Thymo; Prograf; PDN</td>
<td>LRD</td>
<td>72.7 months</td>
<td>Cellular</td>
<td>48</td>
</tr>
<tr>
<td>6</td>
<td>Thymo; Prograf; PDN</td>
<td>LRD</td>
<td>61.7 months</td>
<td>None</td>
<td>66</td>
</tr>
<tr>
<td>7</td>
<td>Thymo; Prograf; PDN</td>
<td>LURD</td>
<td>8.8 months</td>
<td>Cellular</td>
<td>26</td>
</tr>
<tr>
<td>8</td>
<td>Thymo; Prograf; PDN</td>
<td>LURD</td>
<td>7.8 months</td>
<td>Cellular</td>
<td>&lt;15</td>
</tr>
<tr>
<td>9</td>
<td>Thymo; Prograf; CellCept; PDN</td>
<td>LRD</td>
<td>42.4 months</td>
<td>None</td>
<td>53</td>
</tr>
<tr>
<td>10</td>
<td>Basiliximab; Prograf; CellCept; PDN</td>
<td>LRD</td>
<td>41.4 months</td>
<td>None</td>
<td>41</td>
</tr>
<tr>
<td>11</td>
<td>Daclizumab; Prograf; CellCept; PDN</td>
<td>CD</td>
<td>33.9 months</td>
<td>Cellular</td>
<td>39</td>
</tr>
<tr>
<td>12</td>
<td>Basiliximab; Prograf; CellCept; PDN</td>
<td>LRD</td>
<td>60.5 months</td>
<td>Borderline</td>
<td>39</td>
</tr>
<tr>
<td>13</td>
<td>Thymo; Prograf; CellCept; PDN</td>
<td>LRD</td>
<td>23.1 months</td>
<td>None</td>
<td>75</td>
</tr>
<tr>
<td>14</td>
<td>Basiliximab; Cyclosporin; CellCept; PDN</td>
<td>LURD</td>
<td>10.4 months</td>
<td>None</td>
<td>64</td>
</tr>
<tr>
<td>15</td>
<td>Campath; Prograf; CellCept; PDN</td>
<td>LURD</td>
<td>52.4 months</td>
<td>Cellular</td>
<td>39</td>
</tr>
<tr>
<td>16</td>
<td>Campath; Prograf; PDN</td>
<td>LRD</td>
<td>16.7 months</td>
<td>None</td>
<td>60</td>
</tr>
<tr>
<td>17</td>
<td>Thymo; Prograf; CellCept; PDN</td>
<td>LRD</td>
<td>29.8 months</td>
<td>None</td>
<td>68</td>
</tr>
<tr>
<td>18</td>
<td>Basiliximab; Prograf; CellCept; PDN</td>
<td>LRD</td>
<td>10.3 months</td>
<td>None</td>
<td>54</td>
</tr>
</tbody>
</table>

aTx, transplantation; eGFR, estimated glomerular filtration rate; Thymo, thymoglobulin; PDN, prednisone; LRD, living-related donor; LURD, living-unrelated donor; CD, cadaveric donor. The eGFR in mL/min/1.73m².

Fig. 1. Kaplan–Meier survival curves for assessed from the day of kidney transplantation. Overall survival was not significantly different among the three groups, P = 0.94.

Fig. 2. Kaplan–Meier survival curves for assessed from the day of diagnosis of AL. Overall survival was not significantly different among the three groups, P = 0.89.
a kidney transplantation should be based on estimated survival of the patient which is most correlated with cardiac involvement. Our selection criteria stipulate that patients must have a good performance status (0 or 1) and limited cardiac involvement (echocardiographic features, cardiac biomarkers and stress testing). In addition, those who meet the criteria for multiple myeloma may not be good candidates given the higher risk for recurrence. Acute rejection during ASCT is possible as maintenance of immunosuppression can be challenging and graft loss can occur as a result of treatment-related mortality [21]. To reduce the risk to the renal allograft, we changed our strategy to requiring hematologic complete response prior to KTx. This would also avoid performing a kidney transplant in patients who are resistant to therapy. As patients with AL are surviving longer, kidney transplantation should be an option for those who developed ESRD. More studies are needed to improve the selection criteria for these patients.

Conflict of interest statement. None declared.

(See related article by Bridoux et al. Renal transplantation in light chain amyloidosis: coming out of the cupboard. Nephrol Dial Transplant 2011; 26: 1766–1768)

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