Clinical outcome of immunoglobulin light chain amyloidosis affecting the kidney

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Abstract

Background. The kidney is affected by immunoglobulin light chain amyloidosis (AL) in more than 50% of patients who present with the disease, but long-term predictors for outcomes after renal replacement therapy are not well described.

Methods. Kaplan–Meier and multivariate analyses were performed in a uniformly treated cohort of 145 patients with biopsy-proven AL who were monitored for at least 11 years. Outcome measurements were needed for renal replacement therapy and survival.

Results. Among patients presenting with renal AL, 42% ultimately received renal replacement therapy versus 5% of patients who did not have this presentation. Patients with renal amyloid who received dialysis support had significantly higher serum creatinine and 24-h urine protein levels at presentation. Patients with λ light chain amyloid were significantly more likely to have renal involvement and had significantly greater urinary protein loss than patients with κ light chain amyloid. Serum creatinine level was an independent predictor of overall survival when corrected for cardiac involvement. For 38 patients who received dialysis, median survival from Day 1 of dialysis was 10.4 months, and 26% of patients with AL ultimately received renal replacement therapy versus 42% of patients who presented with renal AL specifically.

Conclusions. Presenting 24-h urine protein loss and creatinine values predict which patients will require dialysis. Median survival for patients starting dialysis is <1 year. The presence of λ light chain amyloid predicts the increased likelihood of renal involvement.

Keywords: amyloidosis; dialysis; immunoglobulin light chains; proteinuric; renal failure

Introduction

Immunoglobulin light chain amyloidosis (AL) is a multisystem disorder in which a clonal population of plasma cells produces a monoclonal light chain or light chain fragment that deposits in tissue as an insoluble, fibrillar protein. Accumulation of the fibril protein or its toxic intermediates in organs ultimately disrupts tissue function and leads to death. The most common clinical presentation of AL is in the kidney, and this presentation occurs in approximately half of patients at diagnosis. Ultimately, many patients with AL have symptomatic renal failure in which dialysis can have a life-saving role. This study was designed to determine the natural history of renal AL and the outcomes after dialysis initiation.

Patients and methods

The study group consisted of 145 patients with biopsy-proven AL, all of whom were seen at Mayo Clinic, Rochester, Minnesota, and were enrolled in three separate studies on amyloid treatment from February 1993 to December 1997. The diagnosis of AL was confirmed with immunohistochemistry for immunoreactivity against immunoglobulin light chains in Congo red stained specimens. Frozen sera were not consistently available for additional studies on these patients after 16 years. Patients returned per protocol for reassessment every 6 months and were monitored by phone or letter when their medical condition made it impossible for them to return to the clinic. All patients gave written informed consent at presentation for participation in the treatment study, and the Mayo Clinic Institutional Review Board approved a retrospective review and reporting of these medical records. All patients were monitored from study entry to death. None were lost to follow-up.

All patients had biopsy-proven AL, defined as green birefringence on a tissue biopsy sample stained with Congo red and viewed under polarized light. If a patient had an overt amyloid syndrome with organ dysfunction, the patient was not excluded from participation on the basis of the percent- age of plasma cells in the bone marrow unless the patient had criteria for symptomatic multiple myeloma. No patient with senile, secondary familial or localized amyloidosis was included. Enrolment began in February 1993, and the last surviving enrollee entered in October 1997.

This cohort of patients was referred for therapy through a haematology clinic and might not be representative of the population that would be seen in a general nephrology population. Patients whose presenting serum creatinine value exceeded 5 mg/dL were excluded on the basis of the data according to which these patients’ disease was too advanced to respond to a trial of therapy. All patients had no underlying process associated with secondary amyloidosis. All patients had a family history negative for amyloidosis and had evidence of an underlying plasma cell dyscrasia either by the presence of a monoclonal protein in the serum or urine or by a clonal population of plasma cells in the bone marrow.

The clinical outcomes from therapy, including response rate and actuarial survival, have previously been reported, but no patient in this
cohort has been described previously with regard to renal function and the natural history of the patient's renal disease. None of these patients received stem cell transplantation at any time during the course of therapy. Patients were prospectively entered into a database that was continuously updated.

Kaplan–Meier analysis was used for evaluation of overall and progression-free survival, and differences between survival curves were tested for statistical significance using a two-tailed log-rank test. Multivariate analysis of factors affecting survival was performed using Cox proportional hazards models. Group comparisons were accomplished using non-parametric statistics of a one-way analysis of variance (Mann–Whitney). When more than two groups were tested, the Kruskal–Wallis statistic was used.

Results

The disposition of the original 145 patients is shown in Figure 1 in accordance with the presence or absence of renal involvement and describing the patients who eventually had renal replacement therapy. Among the 84 patients with renal AL, 72 presented with nephrotic-range proteinuria (>3 g/24 h). Three patients presented with urinary protein loss of 2–3 g/24 h, four presented with urinary protein loss of 1–2 g/24 h and one patient had a renal biopsy for urinary protein loss of 0.8 g/24 h. Four patients had a serum creatinine value of 1.6, 2.0, 3.3 and 3.4 mg/dL, respectively, and all four of them had a renal biopsy for urinary protein loss of 1–2 g/24 h). Three patients presented with urinary protein loss of 2–3 g/24 h and one-third was used in these patients, subsequent dose escalation was permitted if excess myelosuppression was not seen. Forty-four patients were treated with dexamethasone (40 mg) on Days 1–4, prednisone (60 mg/day) by mouth on Days 1–7. In both instances, assessment of midcycle myelosuppression was used to adjust the dose of myelosuppressive agents for subsequent treatment cycles. The goal of therapy was to complete 18 therapy cycles, followed by observation. Only 16 of the patients receiving melphalan had an initial serum creatinine level >2.0 mg/dL. Dose reduction of melphalan by one-third was used in these patients, and subsequent dose escalation was permitted if excess myelosuppression was not seen. Forty-four patients were treated with dexamethasone (40 mg) on Days 1–4, 9–12 and 17–20 every 28 days for 4 months, followed by dosing on Days 1–4 for all subsequent cycles. Treatment was continued indefinitely, but the

dexamethasone daily dose was adjusted to prevent grade 3 or 4 National Cancer Institute common toxicity criteria events. The primary driver for dexamethasone adjustment was neuropsychiatric adverse effects, such as euphoria, insomnia or withdrawal symptoms. Response assessment was performed quarterly with the international criteria [8].

Organ responses were confirmed in 39 of the 49 patients—15 (31%) receiving the five-drug combination, 16 (31%) receiving the two-drug combination and 8 (18%) receiving dexamethasone. Also, 29 of the responses seen in these 39 patients were seen in those with renal AL. Seven patients had cardiac responses and three had hepatic responses.

Of the 145 patients, 16 (11%) are alive, with a minimum follow-up of 11 years. A monoclonal protein was found, by using immunofixation of serum or urine, in 94% of patients. Patients with renal and non-renal AL had results showing no monoclonal protein (8% versus 3%), κ monoclonal protein (7% versus 20%) and λ monoclonal protein (85% versus 77%) (P = 0.02). These patients were seen before the introduction of the immunoglobulin-free light chain assay, and hence information about the free light chain level is not available. There were no differences in age, sex or serum creatinine level at presentation among patients with renal AL and those with non-renal AL (Table 1). Determination of renal AL was made using the published consensus criteria for organ diagnosis and response in amyloidosis [8]. Fifty of the 84 patients with confirmed renal AL also had a renal biopsy. When the biopsy showed only vascular amyloid, renal AL was not considered. A biopsy proof of glomerular or interstitial deposition was required for inclusion. No patient was found to have AL and light chain deposition disease, as previously reported [9]. The other 34 patients had classic AL that fulfilled the published criteria. Cardiac involvement was found with echocardiography in 83 (57%) patients. Of the 145 patients, 92 (63%) were male. The male preponderance of AL has been a persistent finding across decades; it is not believed to reflect referral bias. The median urinary protein loss of patients with renal

Table 1. Characteristics of all 145 patients with immunoglobulin light chain amyloidosis

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Renal (n = 84)</th>
<th>Non-renal (n = 61)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, median (IQR) (years)</td>
<td>61 (52–67)</td>
<td>62 (55–68)</td>
<td>0.47</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>52/32</td>
<td>40/21</td>
<td>0.72</td>
</tr>
<tr>
<td>24-h urine protein loss, median (IQR) (g/day)</td>
<td>7.0 (4.4–11.7)</td>
<td>0.3 (0.1–2.6)</td>
<td>NAb</td>
</tr>
<tr>
<td>Creatinine, median (IQR) (mg/dL)</td>
<td>1.1 (0.9–2.2)</td>
<td>1.1 (0.9–1.3)</td>
<td>0.22</td>
</tr>
<tr>
<td>Cardiac involvement (yes/no)</td>
<td>35/49</td>
<td>45/16</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Albumin, median (IQR) (g/dL)</td>
<td>2.17 (0.93–3.93)</td>
<td>3.36 (1.77–4.37)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

aRenal biopsy was done in a sole patient and showed no amyloid deposits.
b24-h urine protein loss is a part of the definition of renal amyloidosis.
IQR, interquartile range; NA, not applicable.

Fig. 1. Outcomes of the original cohort of 145 patients with amyloidosis. AL indicates immunoglobulin light chain amyloidosis.
renal AL. The ratio of had renal AL are compared with patients who had non-gammopathy of undetermined significance. Moreover, the ratio of 2:1 seen in multiple myeloma and monoclonal cell clones was 18:118, or more than 1:6, unlike the typical ratio of 1:12 seen in 24-h urine protein loss, 7.4 (5.0–14.8) 5.9 (3.7–9.1) 0.03

Table 3. Characteristics of 84 patients presenting with renal amyloidosis

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Subsequently required (n = 35)</th>
<th>Not required (n = 49)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, median (IQR) (years)</td>
<td>60 (52–67)</td>
<td>62 (52–67)</td>
<td>0.90</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>21/14</td>
<td>31/18</td>
<td>0.76</td>
</tr>
<tr>
<td>24-h urine protein loss, median (IQR) (g/d)</td>
<td>7.4 (5.0–14.8)</td>
<td>5.9 (3.7–9.1)</td>
<td>0.03</td>
</tr>
<tr>
<td>Creatinine, median (IQR) (mg/dL)</td>
<td>1.4 (0.9–2.8)</td>
<td>1.1 (0.9–1.5)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

IQR, interquartile range.

Table 2. Distribution of urinary protein and creatinine based on monoclonal protein

<table>
<thead>
<tr>
<th>Test</th>
<th>κ (n = 18)</th>
<th>λ (n = 118)</th>
<th>None (n = 9)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients (N = 145)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-h urine protein loss, median (IQR) (g/day)</td>
<td>0.73 (0.2–2.4)</td>
<td>3.61 (0.4–7.8)</td>
<td>5.68 (0.3–12.2)</td>
<td>0.001</td>
</tr>
<tr>
<td>Creatinine, median (IQR) (mg/dL)</td>
<td>1.1 (0.9–1.3)</td>
<td>1.1 (0.9–1.3)</td>
<td>1.5 (1.2–1.6)</td>
<td>7.50</td>
</tr>
<tr>
<td>Patients with renal amyloid at presentation (n = 84)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-h urine protein loss, median (IQR) (g/day)</td>
<td>2.93 (1.1–5.0)</td>
<td>7.2 (4.8–11.8)</td>
<td>7.8 (3.8–12.6)</td>
<td>0.04</td>
</tr>
<tr>
<td>Creatinine, median (IQR) (mg/dL)</td>
<td>1.0 (0.8–2.6)</td>
<td>1.1 (0.9–2.2)</td>
<td>1.5 (1.1–1.6)</td>
<td>&gt;0.30</td>
</tr>
</tbody>
</table>

IQR, interquartile range.

AL was 7.0 g for 24 h (Table 1). Only 56 (39%) of the 145 patients had <1 g of urinary protein loss over 24 h.

The overall ratio of patients having κ versus λ plasma cell clones was 18:118, or more than 1:6, unlike the typical ratio of 2:1 seen in multiple myeloma and monoclonal gammopathy of undetermined significance. Moreover, the distribution continues to be significant when patients who had renal AL are compared with patients who had non-renal AL. The ratio of λ-to-κ patients in the cohort with non-renal amyloid was 4:1; for patients with renal amyloid, nearly 12:1 (P = 0.02). Therefore, the presence of a λ light chain appears to have some degree of tropism for binding to the kidney and leading to glomerular proteinuria.

When patients with renal amyloid were likewise divided into patients who ultimately received dialysis and those who did not, there was no significant difference between the κ and λ distribution in the two groups. Among patients with dialysis, 6% had no monoclonal protein, 6% had κ and 89% had λ. Among those with no dialysis, 10% had no monoclonal protein, 8% had κ and 82% had λ. However, the presence of a λ light chain predicted increased urinary protein loss (P = 0.001) (Table 2). When the records of all patients were analysed, the patients who had a λ light chain had greater urinary protein loss (3.61 g/day) than patients with a κ light chain (0.73 g/day). Moreover, when only the 84 patients with renal AL were analysed, the median urinary protein loss was nearly 3-fold greater in those who had a λ light chain (7.20 g/day) than those who had a κ light chain (2.93 g/day) (Table 3), suggesting that not only does λ result in a higher prevalence of renal involvement, but the degree of glomerular damage in patients with renal involvement is greater with λ than with κ (P < 0.02).

Among patients with renal amyloid, those who required renal replacement therapy presented with a higher serum creatinine level than those who did not require renal replacement therapy (median, 1.4 mg/dL versus 1.1 mg/dL) (Table 3). A similar analysis was performed with the immunoglobulin light chain and the serum creatinine level in all patients and in only those with renal involvement, and no significant creatinine differences were found (Table 2).

### Survival

Of the 145 patients, 130 (90%) have died at the time of this report. As Figure 1 shows, 38 patients ultimately received renal replacement therapy. Only five patients had peritoneal dialysis, and these patients are not reported separately. Of the 84 patients who presented with renal AL, 35 eventually had end-stage renal disease (Table 3). However, the actuarial risk reached 50% at 12 years (Figure 2). Of the 61 patients who did not present with renal AL, 3 ultimately had dialysis-dependent renal failure. Among the 38 patients who received dialysis, only 2 are alive. The median time from diagnosis to dialysis for the 38 patients was 29.5 months (interquartile range, 9.3–72.4 months). The overall median survival of the entire cohort of 145 patients was 26.8 months (Figure 3). Patients with renal AL had a significantly superior survival outcome compared with patients without renal AL (38 months versus 19 months; P = 0.05) (Figure 4). The surprising survival seen in patients with renal AL is related to the fact that patients without renal
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AL had a higher prevalence of cardiac AL (Table 1). When the analysis of survival between patients with renal AL and patients without it was limited to those without cardiac AL, no survival difference was seen ($P = 0.14$).

The median survival from biopsy-proven diagnosis was 59 months for the 38 patients who received dialysis. The median survival of patients from the start of dialysis was 10.4 months, with 20% of patients dying within the first month and 12% of patients having a 5-year survival while receiving dialysis (Figure 5). The cause of death for the 36 patients who have died to date was cardiac amyloidosis for 11 patients; voluntary cessation of dialysis, 6; progressive amyloidosis that occurred predominantly in the gastrointestinal tract, 5; sepsis, 3; refractory hypotension (probably related to cardiac amyloid), 2; gastrointestinal tract haemorrhage, 2 (one with ischaemia and one with haemorrhage); second malignancy, 2; peritonitis (on peritoneal dialysis), 1; and unknown cause, 4. A sole patient received a renal transplant; this patient, who had a diagnosis of AL at age 26 years, received dialysis for 5 years and 9 months after the diagnosis. He received a renal transplant 1 month after starting dialysis and died of a posttransplant lymphoproliferative disorder 7 years and 1 month after receiving his renal transplant. The patient’s overall survival was 12 years and 8 months.

As previously reported, response to therapy had a dramatic effect on outcomes [7]. Among 36 patients evaluable for response who went on to receive dialysis, the 24 non-responders had a median of 6.3 months until dialysis. Of 12 responders who ultimately received dialysis, the median period until dialysis was 92 months ($P < 0.001$). Of the renal AL patients, 78 were evaluable for response; the median overall survival was 14 months for the 49 non-responders to therapy and 108 months for the 29 responders. Of note, patients have to survive in order to respond, so patients with early death are by definition non-responders; therefore, caution must be exercised in imputing the effect of successful therapy on overall outcomes.

The Cox proportional hazards model was used to determine predictors of overall survival. Cardiac involvement was a significant predictor ($P = 0.005$). Serum creatinine level, urine total protein level, the serum albumin level and the light chain did not contribute in the multivariate model as predictors of survival.

### Discussion

In this study of 145 patients with AL, renal involvement defined by consensus criteria was seen in 58% of patients. This study confirms the importance of cardiac involvement in predicting survival for patients with AL. One reason why the patients with renal AL had an improved survival (Figure 4) was their underrepresentation of cardiac involvement compared with the patients who presented without renal AL, who had a prevalence of cardiac involvement of 75%. A surprising number of patients ultimately had renal replacement therapy, representing 26% of all patients and 42% of the patients who presented with renal AL. The causes of death differed, but most deaths were related to progressive amyloid in either the heart or the gastrointestinal tract, with a substantial number of patients withdrawing from dialysis. It is notable that renal failure occurred in only 5% of the patients who did not have AL in the kidney at presentation. Systemic amyloidosis accounted for 0.3% of all patients newly admitted to US dialysis centres from 1995 to 2004 [10]. These patients had a first-year mortality rate of 43.5% compared with our patients’ mortality rates of 20% at 1 month and 45% at 1 year (Figure 5). Because patients with a serum creatinine level exceeding 5 mg/dL were excluded, the results may be better than they would be in an entirely unselected cohort of patients with renal AL.

In one of the earliest reports of amyloidosis, 37 patients were reported, 30 of whom received renal replacement therapy [11]. The 2-year survival from diagnosis was 57%; the median duration of survival after renal replacement therapy

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**Fig. 3.** Kaplan–Meier survival of all 145 patients with amyloidosis from the point of diagnosis.

**Fig. 4.** Kaplan–Meier survival of patients with amyloidosis at diagnosis, separating patients with renal involvement from patients without renal involvement.

**Fig. 5.** Kaplan–Meier survival of 38 patients from the start of dialysis therapy.
was 7.5 months. Of seven patients who underwent renal transplant, three patients died within 6 months of transplant, their death being related to undiagnosed cardiac involvement. Failure to provide posttransplant chemotherapy to suppress light chain production likely has a role as well. In a large Italian centre, 61 patients with renal amyloid were treated, but only 20 had AL and 4 died within 1 month of therapy initiation [12]. In the group, the most important extrarenal complication of AL was related to cardiovascular involvement, which rarely occurs in secondary systemic amyloidosis (AA). Due to cardiac involvement, intradialytic hypotension is a major problem in patients with AL and results in a decreased quality of life for these patients compared with the general dialysis population [12]. A report of 35 AL patients reported results similar to those reported here [13]. The mean time to dialysis was 15 months and the mean survival was 24 months, with most deaths due to AL.

Among the largest centres reporting on dialysis outcomes in patients with amyloidosis are centres with a high prevalence of familial Mediterranean fever. When the heart is not a major end-organ target of amyloidosis, the major cause of death is infection [14]. A superior outcome for patients with AA has been repeatedly reported [15]. In a recent report from a Spanish dialysis centre, 28% of the patients with amyloidosis had AL and only 5% achieved 24-month survival [16]. European reports of amyloidosis are primarily of patients with AA, which rarely involves the heart and nervous system [17]. Therefore, results of dialysis in these patient groups cannot be directly compared with results of a cohort of patients with AL because of the markedly different clinical course.

These studies, in large part, are compromised because of the likelihood that the patients were referred and had renal failure, rather than being a long-term cohort of patients who had a diagnosis of amyloidosis and were observed indefinitely to determine an accurate prevalence of renal failure development over time. As Figure 2 shows, the actuarial risk of renal failure at 11 years was nearly 50%, which was far higher than what we had previously reported for a different cohort of patients [18]. In that cohort, only 18%—compared with our current report of 26%—received dialysis, and only one-third of patients who had renal amyloid had dialysis, compared with our current report of 42%. The increased willingness may be a reflection of the increasing comfort that dialysis centres have in providing renal replacement therapy for patients with systemic disorders.

A second advantage of the current report is the remarkably longer follow-up of survivors—11 years or longer—with no patients lost to follow-up. Recent reports on outcomes of patients presenting with systemic amyloidosis have median follow-ups as short as 2–3 years; as Figure 2 shows, many patients start dialysis after the 5- and 10-year mark. A recent report from a French centre identified 19 patients with AL who underwent dialysis [3]. Once again, there is no way to determine the number of patients in the original cohort who ultimately had end-stage renal disease. The report does show, however, that patients with AL have a shorter time from diagnosis to dialysis than patients with AA, reflecting the more aggressive nature of AL. It also reconfirms the shorter survival for patients with AL, primarily related to the development of cardiac amyloid.

Of note, the current study did not include patients receiving high-dose therapy and stem cell transplant, because acute renal insufficiency after high-dose therapy is a common event. We also did not include patients in whom the need for dialysis was a treatment-related phenomenon [19]. Our program for stem cell transplant began only 10 years ago, and thus a long follow-up is not available. In addition, only 25% of patients with AL who are seen at Mayo Clinic in Rochester are transplant eligible, whereas no such restrictions applied to the reported patients. The patients in the current cohort received either alkylating agent-based chemotherapy or corticosteroid therapy, neither of which is nephrotoxic, so the renal failure that was seen was not related to the underlying therapy.

The current options available to patients with renal AL all use chemotherapy to reduce light chain synthesis, the presumed precursor to the amyloid deposit. Treatment decisions include offering high-dose therapy, which has a high response rate [20] but a significant risk of renal-related complications posttransplantation [19]. Alternatively, oral chemotherapy that is melphalan based has been used; although it is much safer initially, outcomes may not be optimal [21]. Recently, bortezomib has been reported to have higher response rates in AL, and its ultimate role in therapy has yet to be defined [22].

One of the largest centres to report survival in patients with amyloidosis is an Italian group [23]. The group provided clinical and laboratory information of 290 patients. The median time of the follow-up, however, was 24 months. Its report noted that the patients with AL had a significantly shorter survival than those with AA. Multivariate analysis showed that cardiac involvement significantly influenced survival, whereas age and creatinine level did not impact the outcome in AL.

In summary, of 145 patients with biopsy-proven AL, 35 of the 84 patients who presented with renal amyloid ultimately received dialysis, and 3 of 61 patients without renal amyloid ultimately had renal amyloid and underwent dialysis. Long-term outcome was poor. The only predictors of the ultimate need for dialysis in patients with renal amyloid were their presenting creatinine and 24-h urine protein levels.

Of note, patients with renal amyloid are far more likely to have λ light chain than κ light chain, considering all patients with amyloid and the patients who have renal amyloid only. Recently, investigators showed that clones derived from the 6a (λ VI) germ line gene were more likely to present with dominant renal involvement, which may explain the serum and urine findings [24]. The underlying mechanism for why patients with λ light chain amyloid tend to have increased urinary protein loss continues to be unclear. In a multivariate analysis of survival, the presence of cardiac involvement independently predicted survival.

Conflict of interest statement. None declared.

(See related article by P. Ronco and P. Aucouturier. Renal involvement in AL amyloidosis: the facts, the promise and the hope. Nephrol Dial Transplant 2009; 24: 2967–2969.)
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