Long-term prognosis of diffuse proliferative glomerulonephritis associated with infection in adults

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Abstract

Background. Infection-associated glomerulonephritis is rare in adults and its long-term prognosis is undefined.

Methods. We retrospectively evaluated the clinical course of 50 adults (30 men, 20 women) with infection-associated glomerulonephritis diagnosed in our department from 1979 to 1999. The mean follow-up was 90±78 months. Patients were subdivided into two groups: group 1 included those without underlying disease and group 2 included those with severe underlying disease.

Results. At presentation, the median age was 54 years, and 33 patients were hypertensive, 31 had nephritic syndrome, eight had nephrotic syndrome and 11 had non-nephrotic proteinuria. Patients in group 2 were significantly older and had a significantly higher proteinuria than patients of group 1. Of the 21 patients in group 2, nine had liver cirrhosis, four cancer, five diabetes, three bronchiectasis, one thalassaemia intermedia, one polymyositis and one had anti-phospholipid antibodies syndrome. At the last follow-up, five patients had died, 21 patients were in complete remission, ten had partial remission, ten had renal insufficiency and three were on chronic dialysis. Multivariate analysis showed that an underlying disease ($P = 0.04$) and interstitial infiltration at biopsy ($P = 0.036$) were predictors of incomplete recovery. A correlation analysis between the year of diagnosis and the clinical/histological characteristics at presentation showed that age ($P = 0.05$), atypical infections ($P = 0.01$), underlying disease ($P = 0.01$) and interstitial infiltration at biopsy ($P = 0.02$) increased over time, while the number of patients with complete remission significantly decreased ($P = 0.001$).

Conclusions. Infection-associated glomerulonephritis may progress to chronic renal failure in a consistent number of adult hospitalized patients, particularly in those with an underlying disease and when associated with interstitial infiltration at biopsy.

Keywords: glomerulonephritis; infections; nephritic syndrome; renal prognosis

Introduction

Infection-associated glomerulonephritis is rare in adults [1] and its incidence is progressively declining in developed countries [2]. The pattern of the disease has changed over recent decades. Not only streptococcal but also other bacterial, fungal, viral and parasitic infections can trigger the disease [3]. An increasing number of cases have been observed in alcoholic [4], diabetic, drug-addicted [5] and/or elderly subjects [6]. The more extensive use of renal biopsies highlighted atypical histological forms of the disease [7]. There is however little information about the long-term prognosis of glomerulonephritis associated with infections in adults. In recent years only a few papers with a small number of patients [8] and relatively short follow-ups [4,5] have been reported.

In this study we retrospectively analysed the cases of glomerulonephritis associated with infections diagnosed in our department over the past 20 years, and followed in our, or in other, renal units in order (i) to evaluate the long-term prognosis in adults, (ii) to identify the clinical and histological predictors of the
renal prognosis and (iii) to verify whether the spectrum of the disease has changed over time.

Subjects and methods

Patient selection

Fifty patients over a period of 14 years entered the study. All patients were submitted to renal biopsies analysed by light microscopy (after haematoxylin-eosin, periodic acid-Shiff and AFOG stains) and by immunofluorescence using antisera against IgG, IgA, IgM, fibrinogen, C3, C4 and Clq.

In 36 cases electron microscopy was performed.

All patients had proteinuria and haematuria at first presentation, and fulfilled at least two clinical/biochemical and two histological criteria. Clinical/biochemical criteria included (i) a recent episode of infection, (ii) antistreptolysin O titre >250 IU/l and (iii) a transient reduction of serum complement fractions. Histological criteria included (i) diffuse proliferative and/or exudative glomerulonephritis, (ii) dominant granular immune-deposits of IgG and/or C3 in the subepithelial position at immunofluorescence (cases with faint deposits of IgA were included according to Silva [7]) and (iii) presence of humps on electron microscopy.

Exclusion criteria were: predominant IgA deposits on immunofluorescence; idiopathic membranoproliferative glomerulonephritis (excluded on the grounds of persistent hypocomplementaemia, presence of extensive subendothelial deposits at light microscopy and extensive doubling of basement membrane by mesangial interposition); cryoglobulinaemic nephritis (excluded in the absence of typical cryostructurel structures of glomerular deposits at electron microscopy); and lupus nephritis (based on the absence of anti-nuclear and/or anti-DNA antibodies, and on the absence of clinical manifestations of systemic lupus erythematosus at diagnosis or during the follow-up).

We subdivided the 50 patients into two groups. Group 1 consisted of patients with post-infective proliferative glomerulonephritis without other underlying disease (29 patients). One of these patients, admitted to our unit for nephritic syndrome 2 weeks after an upper respiratory tract infection, developed septicaemia due to endocarditis a few days later. Group 2 was comprised of patients with severe underlying disease (21 patients). Nine of them had liver cirrhosis (due to alcoholism in six and due to virus C in three), four had malignant neoplasia (two colon cancer, one testis seminoma and one myeloma), five had diabetes mellitus, three had chronic obstructive lung disease with bronchietasis, one had thalassemia intermedia and was submitted to splenectomy, one had polymyositis and one had anti-phospholipid antibodies syndrome. Three patients were affected both by diabetes and by alcoholism. Two patients in this group also developed sepsis.

After discharge, one patient was lost to follow-up. The other 49 patients were followed as outpatients in our department or in other renal units.

Definitions of diagnoses were as follows:

- Arterial hypertension: supine diastolic blood pressure >90 mmHg and/or systolic blood pressure >140 mmHg in three consecutive measurements.
- Renal insufficiency: plasma creatinine ≥1.5 mg/dl.
- Complete remission: plasma creatinine ≤1.2 mg/dl, proteinuria <0.2 g/24 h and urinary red blood cells <5 per high power field.
- Partial remission: plasma creatinine >1.2 and ≤1.5 mg/dl, proteinuria between 0.21 and 3.5 g/24 h and/or >5 red blood cells per high power field.
- Hypocomplementaemia: serum C3 levels <90 mg/dl (normal values, 90–180 mg/dl) and/or serum C4 levels <10 mg/dl (normal values, 10–40 mg/dl).
- Extracapillary proliferation was considered present when it involved >30% of glomeruli.
- Interstitial infiltration was considered present when 2+ or 3+ in a semi-quantitative evaluation from 0–3+.

Treatment

After renal biopsy 24 patients (median plasma creatinine, 3.4 mg/dl; range, 1.4–5.2) were treated with steroid therapy. Among them there were 13 patients in group 1 and 11 patients in group 2. The indication was based on the presence of renal insufficiency and/or extracapillary proliferation at renal biopsy in more than 30% of glomeruli. One patient was given prednisone 1 mg/kg/day for 1 month, while 24 received one i.v. methylprednisolone pulse (0.5–1.0 g each) for three consecutive days. In three patients no further therapy was given. The other 20 patients received oral prednisone (0.5 mg/kg/day for 1 month, then gradually tapered off) for a median period of 6 months (range, 2–6). Four of these patients with normal renal function at presentation and absence of extracapillary proliferation were treated 2–6 months after renal biopsy because of a persistent nephrotic syndrome (two patients) and worsening of renal function (two patients).

The other 26 patients were not treated, of which 13 had normal renal function and absence of extracapillary proliferation at renal biopsy. Ten patients with renal insufficiency and/or extracapillary proliferation at renal biopsy in more than 30% of glomeruli were not treated because of the presence of diabetes (four patients), liver cirrhosis (four patients), and for persistence of infection (two patients). The last three patients with a positive culture for streptococcus and with renal insufficiency at presentation were not treated because renal insufficiency spontaneously reversed within a few days after renal biopsy.

Statistical analysis

The statistical package, S-Plus, was used to analyse sample data. Since histograms of the variable showed non-normal distributions and the sample size was relatively small, medians and inter-quartile ranges were used as descriptive statistics (25th and 75th percentile are reported in parentheses after every median value). The non-parametric Wilcoxon test was used to compare continuous variables in pairs of patient groups. In particular, it was used to evaluate the change in the disease over the years, and the years of diagnosis were compared for pairs of groups with different clinical and histological variables at presentation. Pearson’s product-moment coefficient was used to test the correlation between two continuous variables. A 2 test was used for statistics on cross-tabulated data. To find predictive variables
for the occurrence of complete remission, a survival analysis was performed by using the Cox proportional hazards regression model [9], both uni- and multivariate, considering the complete remission as the event. Harrell z-test on Schoenfeld [10] residuals was used to examine the proportional hazard assumption for each covariate. Model selection was performed by heuristic search through a number of candidate models. Relative risks and their 95% confidence intervals (CI) have been derived as the antilogarithm of the coefficient estimated for each covariate in the Cox model.

Results

Clinical and histological characteristics at presentation

The clinical and histological characteristics of the patients are shown in Table 1. Of 2862 patients submitted to renal biopsy of the native kidney in our department between January 1979 and December 1999, 50 satisfied the criteria for being included in this study. All patients were Caucasians, 30 were men and 20 women. Thirty-eight patients were hospitalized for clinical management of severe disease and 12 for renal biopsy in order to differentiate between the types of glomerular diseases. The median age at biopsy was 54 years (29.5–65.7). Age was significantly higher in patients from group 2. Patients in group 2 had documented normal renal function and urine examination 6–12 months before admission.

Forty-four sources of infection in a median of 4 weeks (2–5 weeks) before admission were recorded; the sites of infection were ‘typical’ in 25 cases (22 pharyngitis/tonsillitis and three skin infections), ‘atypical’ in 20 (eight pneumonia, six urinary tract infection, one testis abscess, one ocular infection, one endocarditis, one gastroenteritis and two septic phlebitis). Multiple infections were observed in six patients, two of which were from group 1 and four from group 2. The infective agents cultured from the sites of infection were: Streptococcus haemolyticus (five patients), Staphylococcus aureus (six patients), Escherichia coli (eight patients), Pseudomonas aeruginosa (two patients), and Haemophilus influenzae (one patient). Twenty-one of the 45 patients evaluated had high antistreptolysin titre.

At presentation the median plasma creatinine was 2.15 mg/dl (1.2–4.1) without significant difference between the two groups. All patients had haematuria, which was macroscopic in 27 cases (54%), and proteinuria (median, 3.7 g/24 h; 1.8–9.0), which was significantly higher in patients from group 2. Thirty-three patients (66%) had arterial hypertension. At presentation, 31 patients (16 from group 1 and 15 from group 2) had nephritic syndrome, which was associated with nephrotic syndrome in 18 patients, nine of group 1 and nine of group 2, (seven had faint deposits of IgA at immunofluorescence). Eight patients (five in group 1 and three in group 2) had nephrotic syndrome (three had faint deposits of IgA at immunofluorescence) and 12 patients (eight of group 1 and three of group 2) had non-nephrotic proteinuria.

The C3 serum complement fraction, determined in 48 patients, was low in 34 of them (71%). Seven of 47 patients tested for serum C4 (15%) had low levels, which were associated with low C3 in six patients. In seven of nine patients with liver involvement, serum C3 was reduced, and in one of them serum C4 was also low. In all these patients serum C3 and C4 returned to normal values within 8 weeks from the start of hospitalization.

All patients were submitted to renal biopsy. The specimens contained a median of 21 glomeruli (15–32). The main histological characteristics are reported in Table 2.

Follow-up

Patients were followed for a mean of 90 ± 78 months (median, 60 months; 20–138) and the results are shown in Table 3. Twenty-one patients (43%) entered complete remission (64% in group 1 and 14% in

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<td>Miscellaneous</td>
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*One patient, admitted to our unit for nephritic syndrome 2 weeks after an upper respiratory tract infection, developed septicaemia due to endocarditis during hospitalization.

Group 1, ocular infection in one patient and febrile diarrhea in one patient; group 2, testis abscess in one patient. P values refer to the differences between group 1 and group 2; the numbers reported in parentheses after every median value are 25th and 75th percentile.
group 2) after a median period of 12 months (10–12). At presentation, 12 of these patients had nephritic syndrome (median plasma creatinine, 3.2 mg/dl; 2.1–6.6) and 11 had a nephrotic syndrome. During the subsequent follow-up (median, 138 months) none of them had a recurrence of renal disease.

Ten other patients (20%) had a partial remission (18% in group 1 and 24% in group 2) which persisted after a median observation of 38 months. At presentation, three patients had had nephritic syndrome (plasma creatinine 1.5, 2.5 and 3.5 mg/dl, respectively), which partially remitted within 9 months; non-nephrotic proteinuria and microscopic haematuria persisted. Three patients had presented with a nephrotic syndrome; proteinuria slowly reduced (from 5 to 12 months) to non-nephrotic range. In the other four patients, non-nephrotic proteinuria slightly improved during the first year of observation but persisted with microscopic haematuria until the end of the follow-up.

The other 13 patients (three in group 1 and ten in group 2) showed chronic renal insufficiency and proteinuria, which was associated with microscopic haematuria in 12 patients. They had been followed for a median of 46 months. Eleven patients had had nephritic syndrome at presentation (median plasma creatinine, 4.5 mg/dl; 2.5–5.7), which was associated with nephrotic syndrome in seven patients. In eight of them, renal insufficiency partially improved during the first year (median plasma creatinine decreased from 4.4 to 2 mg/dl). In the other three nephritic patients plasma creatinine did not improve and slowly progressed with time (from 1.5, 1.5 and 2.3 mg/dl to 1.7, 1.8 and 7 mg/dl, respectively). The last two patients had normal plasma creatinine at presentation (1.2 and

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<th>Table 3. Renal status of 49 patients at the last observation</th>
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<td>Dialysis* (number of patients)</td>
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<td>Follow-up (months)</td>
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Mean follow-up, 90 ± 78 months. *Five patients died: three with renal insufficiency and two on dialysis (one in group 1 and four in group 2). The numbers reported in parentheses after every median value are 25th and 75th percentiles.
1.4 mg/dl) and non-nephrotic proteinuria but renal insufficiency developed during the follow-up. At the last observation their plasma creatinine levels were 1.9 and 1.7 mg/dl, respectively. Three of these patients died 2–9 months after the diagnosis of acute glomerulonephritis; one of sepsis, one of mieloma and one of stroke.

Five further patients (two in group 1 and three in group 2) had to be submitted to chronic dialysis within 1–6 months. All had nephritic syndrome at presentation (plasma creatinine, from 3 to 6.9 mg/dl) and four had nephrotic syndrome. Four of them had faint IgA deposits at biopsy. Two of them died 2 and 3 months, respectively, after dialysis was started because of sepsis.

Fifty-four per cent of patients treated with steroids achieved complete (six patients) or partial (seven patients) remission compared with 72% of untreated patients (16 complete remission, two partial remission) \((P = \text{n.s.})\). Of note, patients treated with steroids had higher plasma creatinine levels than untreated patients \((P = 0.05)\), and a higher prevalence of interstitial infiltration \((P = 0.04)\).

Prognostic indicators of complete remission

Univariate analysis showed that among the clinical parameters at presentation only the absence of an underlying disease \((P = 0.01)\) was predictive of complete remission (Table 4). Age \((P = 0.08; \text{threshold 62 years, } P = 0.04)\) and proteinuria \((P = 0.06; \text{threshold 1.5 g/24 h, } P = 0.02)\) were more weakly associated with complete remission. Among the histological features, the absence of interstitial infiltration \((P = 0.017)\), of extracapillary proliferation \((P = 0.03)\) and of subendothelial deposits on electron microscopy \((P = 0.05)\) were forerunners of complete remission.

At multivariate analysis only the absence of an underlying disease, \((P = 0.04)\) and the absence of interstitial infiltration \((P = 0.036)\) were predictive of complete remission. The relative risk of incomplete recovery were 3.5 (95% CI, 1.03–12.2) and 8.7 (95% CI, 1.15–65.5).

Changes over time

The incidence of new case/year of acute glomerulonephritis did not significantly change between 1979 and 1999 (Figure 1A).

A correlation analysis between the year of diagnosis and clinical and histological parameters at presentation showed that age \((P = 0.05)\), atypical sites of infection \((P = 0.01, \text{Figure 1B})\) and interstitial infiltration \((P = 0.02, \text{Figure 1C})\) significantly increased over time. The other clinical, biochemical and histological characteristics at presentation did not change over the past 20 years.

The number of patients who achieved complete remission significantly decreased over time \((P = 0.001, \text{Figure 1D})\).

The percentage of patients in group 1 significantly decreased over time, while that of patients of group 2 increased \((P = 0.01, \text{Figure 2})\).

Discussion

The main aim of this study was to retrospectively evaluate the long-term prognosis of adults who were hospitalized because of an infection-associated proliferative glomerulonephritis over the past 20 years. After a mean follow-up of 7.5 years, only 43% of our patients were in complete remission, 20% entered partial remission, 27% had chronic renal insufficiency, and 10% had to be submitted to chronic dialysis. Three patients with renal insufficiency and two on regular dialysis died. The probability of complete remission in this series significantly declined from the early 1980s to the late 1990s. The impression of a worsening prognosis in recent years is supported by the few studies with adequate follow-up periods. In the 1970s, complete remission was reported in 70–80% of adults with acute glomerulonephritis [11,12]. More recently Vogl et al. [13] reported a complete remission in 69% of adults with post-streptococcal glomerulonephritis after a mean follow-up of 4.8 years, while Chugh et al. [14] found recovery in only 59% of adults followed for more than 2 years. In the most recent study, Montseny et al. [5] found complete remission in only 26% of adults, half of them having an underlying disease. However, the short follow-up of that study, with a mean 9 months, may have precluded the observation of late recoveries in other patients, since complete remission occurred after a median of 12 months in our series as well as in other older studies.

Table 4. Clinical and histological predictors of complete remission: univariate analysis

| Variables at presentation                | Remission (21 patients) | No remission (28 patients) | *P*
|-----------------------------------------|-------------------------|---------------------------|---
| Age (years, median)                     | 47 (17–58)              | 63 (42–68)                | 0.08
| Underlying diseases (%)                 | 14.3                    | 64.3                      | 0.01
| Proteinuria (g/24 h, median)            | 3.7 (0.7–6)             | 4.7 (2.3–10.3)            | 0.06
| Extracapillary proliferation (%)        | 14                      | 61                        | 0.03
| Interstitial infiltration (%)           | 5                       | 57                        | 0.017
| Subendothelial deposits on electron microscopy (%) | 19                      | 68                        | 0.05

The numbers reported in parentheses after every median value are 25th and 75th percentiles. *P values refer to the statistical significance of the variables in the Cox proportional hazard regression. The following parameters at presentation were not significantly correlated with the outcome: typical sites of infection; plasma creatinine; low serum C3 and C4; white blood cell count; haemoglobin; platelet count; total serum proteins; macroscopic haematuria; nephritic syndrome; nephrotic syndrome; arterial hypertension; and Ig positivity at immunofluorescence.
Together with the reduced probability of complete recovery, in recent years there was an increasing incidence of chronic renal insufficiency, from 3.3% in the 1970s [11], to 16–28% in the 1980s [13,14,16] to 37% in our series. All these data strongly suggest that the long-term prognosis of post-infectious glomerulonephritis is worsening in adults. This is probably due to the fact that the typology of infection-associated proliferative glomerulonephritis requiring hospitalization has changed over the years in developed countries, with the typical post-streptococcal-glomerulonephritis becoming rarer, while the number of cases with severe underlying diseases is progressively increasing.

The second aim of this study was to identify the predictors of renal prognosis. Only a few studies have investigated the prognostic factors of infection-associated glomerulonephritis and, to the best of our knowledge, a multivariate analysis was not performed in any of them. Some papers have reported that nephrotic syndrome [5,13,14] and renal insufficiency [14,17] at onset were reliable predictors of a poor prognosis. Instead, in another study [18], as well as in our population, neither the levels of plasma creatinine nor the amount of urinary protein excretion at presentation were correlated with the long-term prognosis, although we observed a non-significant trend...
towards a better outcome for patients with proteinuria lower than 1.5 g/day. The importance of age has also been pointed out. Some investigators [6,19] have found that patients older than 60 years had a higher likelihood of mortality, cardiovascular complications and incomplete recovery of renal function. In our series, patients older than 62 years tended to have a worse prognosis than younger adults in a univariate analysis, but the difference disappeared in the multivariate analysis. Instead, the only parameter significantly associated with recovery both in the univariate and the multivariate analyses was the absence of an underlying disease at presentation, confirming the results of two recent studies. Keller et al. [4] reported that, of 30 patients with infection-associated glomerulonephritis, after a mean follow-up of 12.5 months, none of the 12 non-alcoholic patients developed chronic renal failure, in comparison with 50% of the 18 alcoholics. Montseny et al. in 1995 [5] reported that 41 out of 76 adults (54%) with post-infectious glomerulonephritis had an immunocompromized background due to alcoholism, diabetes, drug-addiction or obstructive lung disease. Unfortunately, the correlation between the presence of an immunocompromized background and prognosis was not evaluated in that study. In our series, about half of the patients had an underlying disease which probably caused an immunocompromized background; namely liver cirrhosis mainly due to alcoholism, diabetes, obstructive lung diseases or malignancy. Each of these pathological conditions significantly worsened the outcome. This subgroup of patients was significantly older and had more severe proteinuria than patients without underlying disease, confirming the change in demographic and clinical characteristics of post-infectious glomerulonephritis in recent years. Histological findings may also be helpful in assessing the prognosis. There is a general agreement that crescents are a forerunner of a bad prognosis [6,11,14]. Atypical humps [11] and interstitial fibrosis [5] were also found to be associated with incomplete recovery in previous studies. Our results confirmed the unfavorable influence of crescents in the long-term. The presence of subendothelial deposits on electron microscopy was also predictive of a bad prognosis. However, in the multivariate analysis, only the absence of interstitial infiltration at initial renal biopsy together with the absence of an underlying disease were independent predictors of complete recovery.

The third aim of this study was to investigate if the spectrum of infection-associated glomerulonephritis is changing over the years. In developed countries, the incidence of the typical post-streptococcal glomerulonephritis is progressively decreasing [2] probably due to the improved standard of living, better public health services and the early treatment of pharyngeal infections. In the meantime, however, the number of cases of glomerulonephritis either caused by atypical infective agents [3], or developed in patients with an underlying disease [4,5], and showing atypical clinical and/or histological features at presentation [7,20] is increasing. To verify whether the spectrum of the disease has actually changed over the past 20 years, we performed a correlation analysis between the year of diagnosis and several clinical and histological parameters at presentation. The atypical sites of infection, the number of older patients, and the number of renal biopsies with interstitial infiltration did significantly increase. Of much importance, the number of patients with an immunocompromized background also significantly increased. This probably accounted for the decreasing number of patients who achieved a complete recovery.

In conclusion, this study shows that infection-associated glomerulonephritis should be considered a serious disease in adults, particularly when there is a previous disease and/or when it is associated with severe interstitial infiltration at renal biopsy. Even in favorable cases recovery may require several months. Patients with incomplete recovery should be regularly monitored as many of them may progress to chronic renal insufficiency. The treatment with steroids did not improve the outcome; rather it was associated with a worse prognosis. However, these results were probably influenced by the fact that only patients with severe disease received steroid therapy. A careful management of underlying diseases, abstinence from alcohol and strict control of arterial hypertension may be of use in slowing the renal progression in these patients.

Acknowledgment. This study was supported by the grant: ‘Project Glomerulonephritis’ in memory of Pippo Neglia.

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Post-infectious glomerulonephritis in adults


Received for publication: 21.9.01
Accepted in revised form: 15.2.02