RECURRENT GLOMERULONEPHRITIS IN LIVING KIDNEY TRANSPLANTATION

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A b s t r a c t: Glomerulonephritis (GN) is one of the most frequent causes of end-stage renal disease. Recurrent GN can occur very early after transplantation in up to 20% of renal-allograft recipients and should be considered with late graft dysfunction in 2–5%. Importantly, diagnosis of a clinically silent recurrence of the disease will pass undetected unless transplant centers have a policy of protocol biopsies. In addition, the classification of the type of recurrent GN should be done with data on electron microscopy and immunofluorescence, in order to promote prompt treatment and a strategy for long-term graft survival.

The aim of our paper was to present a few typical cases of recurrent GN, showing the actuality of the problem in living related kidney transplant recipients and to ascertain the importance of precise and timely diagnosis by protocol biopsy.

Recurrent focal segmental glomerular sclerosis (FSGS) in childhood is associated with the highest number of graft loss. The treatment of recurrent FSGN is difficult, so prophylactic plasmapheresis prior to transplantation appeared to be more effective in preventing recurrence than plasmapheresis after transplantation, especially in population of children. Mesangio proliferative GN type II is the second most frequent recurrent GN, followed by type I. Here, it is of paramount importance to classify the type of the disease.

The family of the patient at risk for recurrent GN, a candidate for living related kidney transplantation, should be informed for the expected outcome and their voluntary decision whether to proceed with transplantation should be awaited.

Key words: primary renal diagnosis, kidney transplantation, recurrent glomerulonephritis.

Introduction

Glomerulonephritis (GN) is the primary cause of end-stage renal disease in up to 50 percent of those who go on to receive a renal transplant [1]. Recurrence has been reported in 4.0 to 20% of renal-allograft recipients and will lead to graft failure in 2–5% [2, 4]. It is also worth mentioning that the primary renal diagnosis is unknown in many patients, especially if it is because of the restrictive renal biopsy policy in a particular region or country. Furthermore, a clinically silent recurrence of the disease will pass undetected unless transplant centers have a policy of protocol biopsies [5]. Hence, the recurrence rates quoted in the literature may even be underestimated. This variation may be further influenced by the restricted population studied (a small and variable group, e.g. only those with recurrent GN or only with proteinuria) [2, 6], and not adequately classified recurrent GN with an incomplete graft biopsy examination, missing data on electron microscopy and immunofluorescence [7]. Moreover, the definition of recurrent GN is variable (e.g. clinical *vs* histological recurrence), while the differential diagnosis of transplant glomerulopathy, *de novo* GN or GN transmitted from the donor may be very difficult.

On the other hand, chronic allograft dysfunction is the most prevalent cause of renal transplant failure and has a multifactorial origin. The immunological mechanisms include the number of acute rejection episodes, sensitisation with anti-HLA antibodies, HLA mismatching, delayed graft function as a clinical state emerging from the ischemia/reperfusion injury, young recipient age and inadequate immunosuppression, which all lead to chronic rejection. The non-immune mechanisms, such as calcineurin nephrotoxicity, hyperlipidemia, hypertensive damage and smoking, may accelerate deterioration of renal function. Nowadays, with the improved rates of allograft survival mainly because of the prevention of loss due to acute rejection, and early preventive measures on non-immunological factors, the incidence of allograft loss due to the recurrent GN will become more important. Recently, it was demonstrated in an Australian study reporting recurrent GN as a third largest cause of chronic allograft loss in patients with primary GN, exceeded in impact only by chronic allograft nephropathy and death with a functioning graft [2]. However, the findings on the recurrence of GN need to be interpreted with the precaution that other mechanisms may have amplified recurrence-related graft damage in an additive or even synergistic manner. Hence, the expected outcome for patients with end-stage renal failure due to GN and their decision whether to proceed with transplantation may be affected by their risk of allograft loss due to the recurrence of glomerulonephritis. However, current literature data provide variable estimates of the recurrence of the different entities, which are outlined in Table 1.

Table 1 – Табела 1

Published data on recurrent GN and recurrence related graft loss

*Публикувани ӣодаӣоци за рекуренӣни ӣломерулонефриӣи
и ӣубиӣок на ӣрафӣоӣ асоциран со рекуренцијаӣа*

Recurrent diseases in renal transplant recipients		
	Clinical recurrence rate (%)	Graft loss after 5–10 years (%)
FSGS	20–40	10–20
MPGN type I	20–50	10–30
MPGN type II	> 80 (histologically)	10–25
IgA nephropathy	10–25 (> 50 histologically)	2–16
Membranous GN	5–30	5–20

Focal segmental glomerulosclerosis (FSGS)

Primary FSGS recurs in 20–40% of renal transplant recipients, usually within the first 6–12 months after engrafting, although individual cases as long as 5 years after transplantation have been described [8]. The recurrence presents with heavy proteinuria, hypertension and/or lost of graft function within 5 years in 40–50% of the patients [9]. An increased risk of thromboembolic complications has been noted in patients whose proteinuria exceeds 2 g/day [10].

Risk factors for clinically relevant recurrence include young age at the onset of primary FSGS, rapid progression to end-stage renal disease (<3 years), presence of mesangial proliferation in the native kidney biopsy, white race and a very short interval until recurrence of proteinuria after transplantation [4]. The question whether a living related donor (LRD) represents a higher risk for recurrence than living unrelated or cadaveric donors is recently answered in a very large study, based on the US Renal Data System. Graft failure in recipients below 20 years of age was attributed to recurrent FSGS in 24% of living donors (LD) and 11% of cadaveric grafts, although the benefit of graft survival in LD over a cadaveric donor in view of the reduced incidence of rejection has been maintained [11].

The treatment of recurrent FSGN is difficult, since it is resistant to steroids, like the primary disease. Evidence indicates that a circulating humoral mediator (plasma permeability factor) is responsible for the rapid onset of proteinuria [12]. Hence, apart from supportive therapy with ACE inhibitors, angiotensin II receptor blockers, NSAIDs, the therapeutic options are increased

immunosuppression and/or plasma exchange or immunoadsorption as outlined in the latest ERA guidelines [13]. Prophylactic plasmapheresis (PP) prior to transplantation appeared to be more effective in preventing recurrence than plasmapheresis after transplantation, especially in population of children [14]. However, early treatment after diagnosis with a regimen of three daily plasmapheresis treatments followed by six treatments on an alternate-day basis is recommended [15], or maintained chronically once each month [8]. After the first allograft failure with recurrent FSGS, the reported recurrence in the second allograft is increased even to 100% [16].

Mesangioproliferative glomerulonephritis (MPGN)

Mesangioproliferative glomerulonephritis (MPGN) type I recurs up to 50% at 5 years after transplantation, leading frequently to graft failure [17]. The incidence may be overestimated since transplant glomerulopathy may have a similar light microscopic appearance. Recurrence in a second graft, after prior recurrent MPGN, is 80% [17]. The risk factors for recurrence are living related donors and HLA B8DR3, but because of the low number of patients this should be interpreted with caution. No specific therapy for recurrent MPGN type I has been established. Severe recurrent disease has been successfully treated with increased immunosuppression, predominantly cyclophosphamide, and plasmapheresis, while there is anecdotal evidence that acetylsalicylic acid and dipyridamole may stabilaze renal function [9].

Frequently, hepatitis C virus (HCV) might be associated with recurrent or *de novo* MPGN [18]. A short course of high-dose interferon alpha, oral ribavirin, or a combination of both, might be considered as a therapeutical option.

MPGN type II (dense deposit disease) recurrs in 80–100% of the affected allografts [19]. Graft loss within the first 3 years occurs in <20% of transplanted patients.

IgA nephropathy (IgAN)

Glomerular IgA deposition is a common finding in 30–60% of the patients with a primary diagnosis of IgA nephropathy [9]. Several studies have reported that, with increasing duration of post-transplant follow-up, recurrent IgA disease can significantly impair graft function, being a solely predictor of graft dysfunction and loss [20].

No data are available to determine is there a significantly higher recurrence rate in patients receiving LR donors transplant, or that a choice of immunosuppresion might affect the recurrence of the disease. An evidence of long-term follow-up for the benefit of treatment with newer drugs such as mycophenolate mofetil (MMF) or rapamycin should be awaited.

Membranous nephropathy (MN)

The reported risk of recurrence of membranous nephropathy was about 30% after 3 years of engrafting, and the graft was lost in half of them at 10 years [21]. The variability of the reported data is based on the different criteria adopted for graft biopsy in various centers (proteinuria > 0.2 g/day or >1 g/day), suggesting that a significant number of patients may develop a "subclinical" histologic recurrence. There is no evidence of any association of recurrent MN with the immunosuppression used, histoincompatibility, or type of transplant. No particular therapeutic option is proposed.

The aim of our paper was to put an accent on how actual the problem with the recurrent GN in kidney transplant patients is and to raise the question of protocol biopsy as a matter of precise and timely diagnosis, in particular when the examination is followed by immunohistology and electron microscopy as well.

Material and methods (case presentations)

Three typical cases of recurrent glomerulonephritis from a cohort of 159 living related kidney transplant patients with their first allograft from our country, were selected for case presentation. The patients received induction with methylprednisolone (500 mg) and Antilymphocyte globulin – ALG (Lymphoglobuline; 7–10 mg/kg BW for the first 7–10 days post transplantation) or Daclizumab (Zenapax; 1 mg/kg BW at implantation and thereafter every 2 weeks x five doses). The post-transplant immunosuppression consisted of cyclosporine A – CyA (Neoral; 6 to 8 mg/kg/day) to reach C2 target levels (blood concentration at 2 hours after administration of the drug), steroids (1 mg/kg/day tapered to 0.1 mg/kg/day after 4 weeks) and azathioprine (Imuran; 1.5–2.5 mg/kg BW) or mycophenolate mofetil (Cellcept 2 x 1 g/day)

The indication for graft biopsy was an increase in serum creatinine >20% and/or a decrease in urine output for 2 consecutive days (suspected acute rejection); proteinuria > 1 g/day and/or hematuria (suspected recurrent glomerulonephritis); slow and gradual deterioration of graft function (uncertain etiology of chronic allograft nephropathy). At the beginning of 2002, a clinical practice of routine protocol biopsy at 1 and 6 months was established. All biopsies were performed using an ultrasound-guided automated biopsy "gun". The formalin-fixed biopsy samples were embedded in paraffin, serially sectioned at 3 to 5 μm thickness and stained with hematoxylin-eosin (HE), periodic acid-Schiff (PAS), Masson's trichrome and methenamine silver. The graft biopsies were considered adequate when they contained ≥ 7 glomeruli and at least one artery. Renal lesions were blindly reviewed for evidence of acute and chronic changes by the same pathologist using descriptive morphologic criteria according to the Banff

97 scoring scheme [22], and classical pathological knowledge for differentiation of the type of glomerulonephritis.

The clinical and biochemical data were recorded according to the routine clinical practice.

Results

Case 1. *(Recurrence of FSGS): A 13-year-old male patient was admitted to the Pediatric Department in December 1993 for proteinuria (17 g/day), edema and macrohematuria. The paternal grandmother was supposed to have a nephrotic syndrome because of the edema. In January 1994 FSGS was diagnosed as a primary cause of renal disease, and a fast progression to end-stage renal failure and the need for dialysis treatment was observed in September 1996. In October 1997 he underwent living related kidney transplantation from his 41-year-old mother. As induction therapy prednisolone and an 8 days course of ALG was administered. Maintenance therapy consisted of steroids, Azathioprine and Cyclosporine A. After initial polyuria on the first post operative day, there was a subsequent decrease in urine output with a need for HD treatment on day 5 post-transplantation. Two days later a three-day methylprednisolone pulse (MPP) of 500mg each was administered. Renal biopsy (RB) was performed on day 13, since an insufficient response under steroid treatment was obtained. The histology showed an interstitial injury but no vascular or changes for acute rejection. The next few days the graft function slowly improved, although a progressive rise in proteinuria up to 3.9 g/day was observed. A one-month biopsy revealed recurrence of FSGS and three days MPP of 1 gr/day was administered, followed by mycophenolate mofetil. Serum creatinine decreased to 114 µmol/l, but proteinuria was increased to 10 g/day. In May 1998 a rise in serum creatinine around 300 µmol/l was observed and 3 plasmapheresis (PP) sessions were started, with no effect on proteinuria. The renal function remained stable until August 1998, when serum creatinine increased to 430 µmol/l and another three MPP of 500 mg each were administered. In January 1999 proteinuria increased to 16 g/day and graft function deteriorated, raising serum creatinine to 530 µmol/l. The patient received again 3 MPP of 500 mg each and 3 PP sessions and proteinuria decreased to 11.7 g/day. However, in the following months serum creatinine steadily increased to 1150 µmol/l, and dialysis treatment was started.*

Case 2. *(Recurrence of FSGS): A 25-year-old male patient with diagnosed FSGS as a primary cause of renal disease in 1987, slowly progressed to end-stage renal failure and a need for dialysis treatment in May 2002. In October 2002 he underwent living related kidney transplantation from his 66-year-old mother. Immediate postoperative renal function was normalized after*

two weeks, but proteinuria (< 2 g/day) persisted, and temporary steroid diabetes appeared with a need for 2 weeks insulin treatment. Protocol biopsies at 1and 6 months after transplantation revealed recurrence of FSGS and no acute or chronic changes according to the Banff classification. Hence, there were no changes in standard therapeutical regimen other than maintaining cyclosporinemia at the upper level of the proposed range for C_2 or C_0. In the meanwhile, determination of hemostasis showed a hyperthrombotic state and an adjuvant therapy with dypiridamol and acetyl salicylic acid was administered. At the same period of time, a gradual increase in the patient's body mass index and signs of hip osteonecrosis were observed and adequately treated thereafter. All these conditions were considered as contributing factors for the observed gradual increase in serum creatinine, maintaining a level between 250 and 280 μmol/l. In November 2003 proteinuria decreased to 0.4 g/day and remained stable thereafter. At the last check-up, in October 2005 the graft function was at the same level.

Case 3. (Recurrence of MPGN): A 22-year-old male patient with primary cause of renal disease MPGN diagnosed in 1994, progressed to end-stage renal failure and a need for dialysis treatment in 1999. In September 2002 he received a kidney from his mother (50 years of age). There was an immediate posttransplant proteinuria (< 2 g/day), but renal function remained stable. He underwent the first episode of pneumonia 2 weeks after transplantation. At the end of the first month proteinuria increased to 4.2 g/day while the histology of the1-month protocol biopsy revealed recurrence of MPGN and a Banff classification of acute rejection type IIb. The patient received three consecutive MPP of 1 gr each, graft function was stable, and there was no need for PP treatment. Additionally, an angiotensin receptor blocker was administered and after 4 months proteinuria decreased to 0.6 g/day with the graft function remaining stable. In February 2003 there was another episode of broncho-pneumonia. At the end of the sixth month an increase in serum creatinine to 162 μmol/l was observed. Confirmation of the recurrent MPGN and borderline acute and moderate chronic changes according to the Banff classification was reported at the sixth-month protocol biopsy. In May 2003, there was again an episode of broncho-pneumonia. Renal function worsened in May 2004 with an increase in serum creatinine to 216 μmol/l and proteinuria of 5 g/day. At the beginning of 2005 CyA was replaced by Tacrolimus with further progression of the graft dysfunction. In the following months the patient underwent a few episodes of broncho-pneumonia and become severely anemic, with no efficient response to EPO therapy because of persistent inflammatory state and a functional iron deficiency. In September 2005 proteinuria diminished but the graft function failed and serum creatinine increased to 680 μmol/l. The patient started dialysis and died from sepsis in October 2005.

Discussion

We have reported two patients with recurrent FSGS. Although the same histological type, the outcome of these two cases was completely opposite. Studies dealing with the risk factors for clinically relevant recurrence of FSGS include young age at the onset of primary FSGS, rapid progression to end-stage renal disease (< 3 years), and a very short interval until recurrence of proteinuria after transplantation [4]. The first patient matched all these risk factors. However, we reported a marked proteinuria of 10 g/day at the 1-month biopsy and very short graft survival of less than 2 years. Despite the induction therapy with ALG, increased immunosuppression by cyclosporine and MMF thereafter, as well as plasmapheresis treatments, the disease took a rapidly progressive course and was highly resistant to MPP. A recent study published by Ohta T *et al.* in 2001, reported a beneficial effect of prophylactic plasmapheresis prior to transplantation, especially population of in children [14]. Unfortunately, the prophylactic PP treatment was not applied in this case of severe primary and recurrent FSGS thereafter.

Our experience with protocol biopsies performed in stable allografts has uncovered a high prevalence of borderline and subclinical rejection and features of CAN in stable allografts [5]. In addition, a few cases with recurrent primary renal disease were found. Hence, this timely diagnosis might give an advantage in the adjustment of the therapeutical approach to patients with recurrent glomerulonephritis. The early diagnosis of recurrent FSGS presented in the second case, allowed maintaining an increased immunosuppression with CyA and additional adjuvant therapy with dypiridamol and acetyl salicylic acid, which might be of some benefit for the graft function and long-term graft survival. However, it should be mentioned that beside young age at the onset of primary FSGS, this patient presented with slow progression to end-stage renal disease (15 years), and early proteinuria after transplantation which did not exceed the value of 2 g/day.

The third case of recurrent mesangioproliferative glomerulonephritis (MPGN) presented with heavy proteinuria and rapid impairment of the graft function for 3 years after transplantation. This graft survival was much shorter even than the reported recurrence rate of 50% at 5 years after transplantation [17]. An increased immunosuppression with CyA and tacrolimus thereafter, as well as the PP treatments, was not successful in slowing down the progression of the disease. The patient was HCV negative, HLA matched with 4 antigens and experienced only one episode of acute rejection at the 1-month protocol biopsy. This episode of vascular rejection appeared to be sensitive to MPP therapy and there was no need for PP treatment. Importantly, we were not able to differentiate the type of MPGN (immunofluorescence) and missed the opportunity for an aggressive treatment with cyclophosphamide and PP.

According to the clinical manifestation, this MPGN had similarities with type II MPGN, whose reported rate of graft loss within first 3 years was < 20% of transplanted patients [19]. In addition, the clinical condition of the patient was complicated with frequent and severe episodes of broncho-pneumonia, based on exacerbated chronic bronchitis. The patient was a heavy smoker since his 15th year of age, although he stopped this habit after transplantation. The substantial amount of antibiotic received in the treatment of this vital clinical condition might have further contributed to the rapid deterioration of the graft function.

Conclusion

Recurrent disease can occur very early after transplantation and should be considered together with late graft dysfunction. The classification of the type of recurrent GN with data on electron microscopy and immunofluorescence is inevitable for appropriate treatment and a strategy for long-term graft survival. Protocol biopsies are useful in timely diagnosis of histological, clinically silent recurrent GN, giving an opportunity for prompt treatment in order to salvage the graft. The family of the patient at risk for recurrent GN, the candidate for living related kidney transplantation, should be informed of the expected outcome and their voluntary decision whether to proceed with transplantation should be awaited.

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Р е з и м е

РЕКУРЕНТНИ ГЛОМЕРУЛОНЕФРИТИ КАЈ БУБРЕЖНА ТРАНСПЛАНТАЦИЈА ОД ЖИВ ДАРИТЕЛ

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А п с т р а к т: Гломерулонефритот (ГН) е една од најчестите причини за терминално бубрежно заболување. Рекурентниот ГН може да се создаде многу рано по трансплантацијата кај 20% од примателите на бубрежен алографт и би требало да се смета како причина за подоцнежна дисфункција на графтот кај 2–5% од нив. Важно е да се напомене дека дијагнозата на клинички мирната рекуренција ќе помине незабележана, освен во случаите кога центрите за трансплантација изведуваат рутински протокол биопсии на графтот. Понатаму, класификацијата на типот на рекурентен ГН би требало да се направи со добивање на податоци од електронската и имунофлуоресцентна микроскопија, со цел да се овозможи брз третман и планирање на стратегија за долгорочното преживување на графтот.

Цел на нашиот труд беше да се презентираат неколку типични случаи на рекурентен ГН, за да се покаже актуелноста на проблемот кај бубрежната трансплантација од жив дарител и да се утврди важноста на прецизната и навремена дијагноза преку протокол биопсијата на графтот.

Рекурентната фокал сегментна гломеруларна склероза (ФСГС) во детството е асоцирана со најголемиот број на изгубени графтови. Третманот на рекурентната ФСГС е тежок, па затоа профилаксата со примена на плазмафереза пред трансплантацијата се претпоставува дека е поефи-

касна за превенција на рекуренцијата отколку плазмаферезата по тран-
сплантација, особено кај детската популација. Мезангио пролиферативниот
ГН тип 2 е втор најчест рекурентен ГН, а потоа следи типот 1. Кај овие
случаи, од неизмерна важност е да се класифицира типот на заболувањето.

Фамилијата на пациентот кој е кандидат за бубрежна трансплан-
тација од жив дарител а е со ризик за рекурентен ГН, би требало да биде
информирана за очекуваниот исход и треба да се почека на нивната свое-
волна одлука дали да продолжат со трансплантацијата.

Клучни зборови: примарна ренална дијагноза, бубрежна трансплантација,
рекурентен гломерулонефрит.

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