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RENAL TRANSPLANTATION
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Outcome of glomerulonephritis in live-donor renal transplant recipients: A single-centre experience

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KEYWORDS

Post-transplantation glomerulonephritis (GN);
Renal transplantation;
Long-term survival;
Recurrent GN;
De novo GN

ABBREVIATIONS

ESRD, end-stage renal disease;
FSGS,

Abstract ***Objectives:*** To investigate the frequency and risk factors affecting the incidence of post-transplantation glomerulonephritis (GN) and the impact of GN on the survival of the graft and the patient.

Patients and methods: Patients were classified based on histological findings into three groups. Graft survival was ascertained using the Kaplan–Meier method and significance calculated using log-rank tests. For multivariate analysis the Cox model was used.

Results: Transplant glomerulopathy was the most prevalent glomerular disease in our series followed by recurrent GN and lastly *de novo* GN. In all, 50% of the *de novo* GN group had diabetes. The worst graft outcomes were in the recurrent GN group ($P = 0.044$). Multivariate analysis revealed ageing of the graft and mammalian target of rapamycin (mTOR) immunosuppression as risk factors for development of GN. While, the age of the recipient and donor, anti-lymphocyte globulin induction therapy, and acute rejection were risk factors for poor graft outcomes.

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focal segmental glomerulosclerosis;
GN, glomerulonephritis;
HCV, hepatitis C virus;
HR, hazard ratio;
MPGN, membrano-proliferative GN;
PTGN, post-transplantation GN

Conclusions: GN is an important issue after transplantation. Tracking the incidence and progression of histological findings in the graft may help to guide proper management and improve graft outcome.

Introduction

Transplantation has proven to be the best therapy for end-stage renal disease (ESRD), being superior to maintenance dialysis therapy with better quality of life and lower mortality risk [1]. The impact of glomerulonephritis (GN) on graft outcome is not fully understood [2].

GN has been reported to recur in renal grafts at different rates depending on histological type [3]. Impairment of graft function and even loss has mostly been reported with recurrent GN [4]. Exact diagnosis of GN before transplantation is not easy, as most of the patients present with ESRD without an available histological diagnosis for varying reasons. Thus, most reported diagnoses of GN are based on clinical judgement rather than histological evidence, leading to an incorrect estimation of the true incidence of GN [5]. Another problem is the difficultly in differentiating between GN histological findings and calcineurin-inhibitor nephrotoxicity and chronic allograft nephropathy [6].

Recurrent GN is clinically relevant, as it can result in long-term graft loss; it was reported to be the third most common cause of graft loss during the 10-year period after transplantation. The negative impact of recurrent GN increased from 0.6% during the first year after transplantation to 8.4% after 10 years [3]. In the present study, we analysed the incidence of different types of GN reported after transplantation, potential precipitating factors, and their potential risk on graft survival.

Patients and methods

This study comprised 2000 transplant recipients who received their grafts between March 1976 and February 2010 at Mansoura Urology and Nephrology Center. In all, 1648 patients received their grafts from related donors, while the other 352 received their grafts from unrelated donors. Among the unrelated group, 122 were spouses. The procedures were approved by the ethics committee of human experimentation in our centre and in accordance with the Helsinki declaration of 1975.

Exclusion criteria included: couples with historical positive lymphocytotoxic cross match, recent malignancy, addiction, psychiatric disorders, type I diabetes mellitus, significant extra-renal organ failure (pulmonary, hepatic, or cardiac), other exclusion criteria for donors included: unwilling donors, diabetes mellitus, hypertension, positive hepatitis B surface antigen (HBsAg), anti-hepatitis C virus (HCV) antibodies, anti-HIV and anti-cytomegalovirus (CMV) IgM antibodies. All clinical records of all kidney transplant recipients were entered prospectively in our computer network transplant database.

Study design

This is a retrospective study in which the patients were divided according to graft biopsy results into two groups: Group I (No GN) included patients who did not have post-transplantation GN (PTGN). Group II (PTGN) included patients who developed PTGN. This group was further divided according to the nature of GN into:

- *de novo* GN, which included patients who did not have biopsy confirmed GN before transplantation or had a different type of GN than the one discovered after transplantation;
- recurrent GN, which included patients with PTGN of the same histopathological type as that before transplantation;
- transplant glomerulopathy, which included patients with glomerular injury with unique pathological and pathogenic entity distinct from other forms of chronic allograft injury.

GN management was according to the international protocols valid at the time of graft biopsy. The protocol table is provided in the Appendix.

Statistical analysis

Qualitative data are presented in cross tabulation and quantitative data are presented as the mean (standard deviation, SD). Univariate analyses were used for initial evaluation of differences using the chi-square and