

INTRODUCTION

Kidney transplantation is the treatment of choice for end-stage renal disease. It offers better quality of life and minimizes the mortality risk for patients when compared with maintenance dialysis therapy (**Worawon Chailimpamontree, et al., 2009**).

Little data are available concerning the impact of the post-transplantation glomerulonephritis on graft outcome (**Gaston R, 2006**). The post-transplant glomerulonephritis (GN) may be De novo GN or recurrence of the original kidney disease. De novo GN appear to have poorer prognosis than the recurrent type.

Different types of glomerulonephritis were reported to recur in the graft with different recurrence rates (**Briganti EM, et al., 2002**). The recurrent glomerulonephritis was reported to be important cause of impaired graft function and consequent graft loss (**Choy B. et al., 2006**).

Studies on recurrent disease are difficult since not all patients have undergone native kidney biopsy or it was non-representative. The reported incidence of recurrent GN is thus judged by clinical suspension and could be over- or under-estimates of the true incidence (**Hariharan S, et al., 1999**). Precise diagnosis of recurrent disease in view of concomitant histological features of chronic allograft nephropathy or chronic drug nephrotoxicity by calcineurine inhibitors is often difficult to be determined (**Requião-Moura LR, et al., 2007**).

There is accumulating evidence that recurrent GN is an important and clinically relevant cause of graft loss in the long-term follow-up of

renal allograft recipients. It was reported that recurrent GN is considered to be the third most common cause for graft loss 10 years after kidney transplantation. The risk of graft loss from recurrence was found to be increased from 0.6% during the first year post-transplant to 8.4% after 10 year of follow up (**Briganti EM, et al., 2002**).

The introduction of newer immunosuppressive agents and induction protocols improved the graft survival. The improvement of graft survival was through the direct reduction of the incidence of acute rejection. The incidence of post-transplant glomerulonephritis whether recurrence or De novo was not influenced (**Hariharan S, et al., 2000**).