Summary and conclusion

Liver disease is one of the leading causes of death in long-term survivors after kidney transplantation. Chronic liver disease is a frequent complication affecting about 15 % of renal allograft recipients. Infection by hepatitis B and C viruses is the predominant cause of hepatic dysfunction in these patients. Hepatic cirrhosis and clinically active hepatitis due to HBV and HCV infection clearly contraindicate kidney transplantation. More controversial is the attitude to be adopted towards candidates with clinically quiescent chronic HBV or HCV. It was proved by many authors that schistosomiasis does not affect short as well as long-term outcome of kidney transplantation.

In an attempt to study the impact of pretransplant liver dysfunction and etiology of liver disease on the short-term outcome of renal transplantation, we prospectively followed up 75-end stage renal disease patients on regular hemodialysis and arranged for living donor kidney transplantation at Mansoura Urology and Nephrology Center from the pre- to post-transplant period.

The patients were classified into two main groups:

Group (1): twenty-five patients with healthy liver considered a control group.

Group (2): fifty patients with abnormal liver function of different etiologies. So, they were subclassified into:

Subgroup (a): patients with HCV antibodies and / or HCV RNA positive (28 patients); **(i) Treated by interferon (before transplantation)**: 16 patients and **(ii) Non-treated by interferon**: 12 patients

Subgroup (b): patients with mixed HCV and HBV infection (5 patients).

Subgroup (c): patients with mixed HCV and schistosomal infection (8 patients).

Subgroup (d): patients with Schistosomiasis (9 patients)

We noticed high sustained response to IFN in HCV viremic haemodialysis patients (68.2 %) which is much better than non-uremic HCV positive patients and only one patient turned HCV RNA positive after renal transplantation. Also,

we found that HCV patients treated by IFN as well as pure schistosomal renal allograft recipients had excellent graft function during the period of follow up and significantly better than other groups. Moreover, number of rejection episodes was nearly equal to the control group.

Also, we found that HCV (not treated by IFN) showed non-significant elevation of serum creatinine especially during the first 8 months after kidney transplantation. However, after that, serum creatinine dropped and was non-significantly different from the control group.

The mixed HCV and HBV infection group showed significant pre-transplant impaired liver function in comparison to control group. After kidney transplantation, serum creatinine was non-significantly higher than control group especially during the first year after transplantation. Moreover, the number of rejection episodes was non-significantly higher than control group.

Unfortunately, mixed HCV and schistosomiasis group had bad results. Only 25 % did not experience rejection, 25 % experienced 2 episodes and 50 % experienced more than 3 rejection episodes during their follow up period. Their serum creatinine ranged from 1-12 mg / dl (mean 2.5 mg / dl) versus 0.6-3.4 mg / dl (mean 1.24 mg /dl) in control group. Also, their serum creatinine reached its peak in the 7^{th} month (4.94 versus 1.5 mg /dl). Moreover, number of rejection episodes was nearly double that of control group (2.38 versus 1.24) but it was non-significant due to high standard deviation.

None of our patients developed fulminant hepatitis or chronic liver disease.

In summary, our presented data confirmed that IFN is highly effective in management of pretransplant HCV infection which may contribute to better post-transplant graft function. Neighther HCV nor schistosomiasis affect the short term out come either of the patients or of the graft survival after kidney transplantation. Mixed HCV and HBV may cause more susceptibility to early

post-transplant graft impairment and more rejection episodes. Also, mixed HCV and schistosomiasis was associated with post-transplant graft impairment. So, it carries a high risk to patients as well as graft survival after kidney transplantation.

We conclude that problems occurring in renal transplant recipients must be diagnosed as early as possible to avoid either irreversible rejection or a rapidly fatal outcome: the management of these patients appears to be one of the most important factors for the long term success of an allograft. Such management must be performed by a well-trained clinical and laboratory consultants. One must keep in mind that an otherwise benign disease can be rapidly lethal in a transplant recipient.

Again, we conclude that pre-transplant treatment of HCV in chronic renal failure patients by interferon is highly effective in management of pretransplant HCV infection which may contribute to better post-transplant graft function. Also, patients with mixed HCV and Schistosomal infection are liable to bad post-transplant results either in the patient or the graft survival.

We recommend IFN therapy for HCV RNA positive haemodialysis patients and caution must be taken in renal transplantation to patients with mixed HCV and Schistosomiasis. Further studies are needed to complete this work, to detect the possible pathogenesis of impaired graft survival in mixed HCV and schistosomiasis patients and postulation of new regimens of immunosuppression therapy suitable to these patients as well as close monitoring of graft function after kidney transplantation.