

## **Introduction**

Chronic hepatitis C virus (HCV) infection exists in a large proportion of patients undergoing renal transplantation. Nowadays it is not considered to be an absolute contraindication to transplantation; however, it is associated with an increased risk for the patient and accounts for a shorter half-life of the renal allograft (*Caeiro et al., 2011*).

The natural history of hepatitis C virus (HCV) infection after renal transplantation is incompletely understood. HCV is still highly prevalent among patients with end-stage renal disease (ESRD) and transplantation both in developed and less developed countries (*Fabrizi et al., 2002*).

Chronic liver disease and its complications are major problems in renal transplant recipients. Hepatitis C virus (HCV) infections are the most important causes of chronic liver disease in this population (*Ghafari. and Sanadgol. 2008*).

Chronic liver disease is a frequent complication after transplantation, and HCV is the leading cause of chronic liver

disease among renal transplant recipients. Since aminotransferase activity is lower in patients with chronic renal failure than nonuremic populations, some HCV-positive recipients may be overlooked, so predicting the natural history of HCV among recipients becomes difficult (*Savas et al., 2007*).

Chronic Infection with hepatitis C virus (HCV) is a serious public health problem affecting an estimated 2% of the world's population. The main routes of HCV transmission in the dialysis setting include transfusions, multiple procedures for dialysis access, transplanted organs, and contamination within the dialysis unit (*Fissell et al., 2004*).

HCV reactivation occurred in nearly half of the renal transplant recipients, mostly in the second year. Patient survival and graft survival were not affected by HCV reactivation. Anti-HCV positivity should not preclude chronic renal failure patients from renal transplantation (*Savas et al., 2007*).

The immunosuppressive regimens used for the prevention of allograft rejection result in increased HCV replication leading to pathological deterioration in 75% of patients and 25%

incidence of cirrhosis within 5 years after transplantation (*Zylberberg et al., 2002*).

Patients who have undergone kidney transplantation and suffer from hepatitis C (HCV) cannot be treated with standard therapy (pegylated interferon combined with ribavirin) due to the risk of acute rejection. Furthermore, immunosuppressive therapy facilitates the progression of infection and chronic hepatopathies. Monocytes and macrophages are known to produce extrahepatic breeding sites that spread disease (*Novelli et al., 2009*).

Amantadine monotherapy is safe and is well tolerated in HCV (+) renal transplant patients. However, it lacks of efficacy. It is able to improve liver enzymes but it has no impact neither upon HCV viremia nor upon liver histology (*Kamar et al., 2004*).