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The management of Hepatitis B infection in kidney transplant recipients

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Chronic hepatitis B virus (HBV) infection confers adverse clinical outcomes in kidney transplant recipients (KTR) due to increased hepatic complications. This presents a clinical challenge especially in areas endemic for HBV infection such as the Asia-Pacific region, where the prevalence rates can be up to 10-15% in chronic dialysis patients. With advancement in pre- and post-transplant care including the availability of effective oral nucleoside/tide analogues (NA), there is a changing paradigm for the management of HBV-infected KTR and patient outcomes has also improved substantially. Pre-transplant management includes meticulous infection control measures in dialysis units, reduced blood transfusion with the use of erythropoietin stimulating agents and also a universal HBV immunization program. Careful donor-recipient serological matching and the use of HBV hyperimmunoglobulins can also help reduce HBV transmission peri-operatively. The pre-emptive or prophylactic use of NA in post-renal transplant setting has brought major impact to this area. Lamivudine, the first oral NA available, can effectively suppress viral replication and improve short- and long-term patient survival in HBV-infected KTR, but is also associated with high rates of resistance. Development of drug resistance remains an important issue as most KTR require prolonged anti-viral treatments. In this context, recent data suggested that entecavir is efficacious for HBV DNA suppression and transaminase improvements in both treatment-naïve and lamivudine-resistant KTR, and was associated with very low resistance rates in the former group. More importantly, entecavir-treated patients all showed stable renal allograft functions after 3 years of follow-up. Other rescue options include adefovir and tenofovir which are nephrotoxic, and hence appropriate dosage adjustment and careful allograft function monitoring should be exercised when used in KTR.

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New advances in approaching of the alteration of Intrahepatic microcirculation in cirrhotic livers

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From a hemodynamic point of view, the hepatic vascular resistance and portal inflow determine the level of portal pressure. Factors that determine the hepatic vascular resistance include both structural and dynamic components. Among the structural components are histological characteristics such as steatosis, fibrosis and regeneration nodules and neoangiogenesis. Dynamic structures include cells with contractile properties such as hepatocytes, hepatic stellate cells, sinusoidal endothelial cells and Kupffer cells. The contributions of the interactions between four cells in cirrhotic livers resulted in hepatic endothelial dysfunction, hepatic microcirculatory dysfunction, hepatic venous dys-regulation, hepatic fibrogenesis and subsequently increased intrahepatic resistance and portal hypertension in cirrhosis. The pathogenic mechanisms triggering the associated abnormalities in hepatic microcirculations including persistent endotoxemia, increased hepatic oxidative stress, activated endocannabinoids substances, pathogenic sinusoidal remodeling and hypo-perfusion in cirrhotic livers. Cumulative data suggested various therapeutic strategies targeting on the hepatic microcirculation effectively improvement of the systemic abnormalities of cirrhosis. Accordingly, the mechanistic and therapeutic approaches focusing on the disarrangement of hepatic microcirculation will introduce in this article.

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