Short Communication The Neurologist: Clinical & Therapeutics Journal 2021
Vol.5 No.1

Relationship between contributor/beneficiary MTRR quality polymorphisms and the danger of new-beginning neurological intricacies after liver transplantation

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Liver transplantation (LT) is one of the best options for the treatment of end-stage liver disease. At present, because of the high survival rate, many complications associated with LT patients are increasing. NCs are one of them. It is reported that up to 30% of the patients in western countries developed neurological complications after LT, but only 4% after heart transplantation and 0.5% after renal transplantation (Agildere et al., 2006, Amodio et al., 2007, Bronster et al., 2000, M et al., 2006). Frequent complications include acute confusion, epilepsy, cerebrovascular disease (Otan et al., 2015a, Otan et al., 2015b), and central system infection (Weiss and Thabut, 2019). These symptoms lead to a decline in the quality of life of patients, and even increase the mortality rate (Ghaus et al., 2001). Early and accurate identification of NCs high-risk patients is helpful to prevent disease and improve the prognosis of recipients.Since there is no accepted model or indicator for the prediction of NCs before, our research has practical guiding significance.

Methionine synthase reductase (MTRR), maintains sufficient levels of methylcob (III)alamin, the activated cofactor for methionine synthase, which catalyzes the remethylation of homocysteine to methionine. Hyperhomocysteinemia is caused by nutrition, such as folic acid, vitamin B6 and B12, and genetic factors, including the functional polymorphism of key enzymes related to homocysteine metabolism (Szvetko et al., 2007). Epidemiological evidence accumulated over the past decade has shown that elevated plasma homocysteine levels (hyperhomocysteinemia) were associated with an increased risk of neural tube defects (NTDs) (Gaughan et al., 2001). Many studies have shown that MTRR is associated with neurological symptoms (Fang et al. Kumar et al., 2018, Wang et al., 2015). MTRR and Methylenetetrahydrofolate reductase (MTHFR) are involved in the metabolism of amino acids in the body, so scholars often study the two together. In this study, we aimed to determine whether donor or recipient gene (MTRR) mutations contribute to the development of NCs after LT, and to identify potential risk factors for NCs.

A total of 166 patients who underwent primary LT treatment at the Affiliated Hospital of Qingdao University from January 2015 to July 2017 were included. We excluded children under the age of 18 who underwent liver transplantation; patients with missing follow-up data; patients with combined liver and kidney transplantation; patients had acute rejection. The average age of the recipients at the time of transplantation was 50.5 ± 9.8 years (range: 18–75 years), with 138 males and 28 females. Table 1 summarizes the main clinical characteristics of the study population. None of the patients had a family history of neuropsychiatric illness. Causes of liver disease include viral hepatitis B (n = 119), alcohol (n = 7), hepatitis C virus (n = 5), Other etiology tumor (n = 9), primary biliary cirrhosis (n = 6), sclerosing cholangitis (n = 6), autoimmune hepatitis (n = 6), cholangiocarcinoma (n = 4), and hepatic cyst (n = 4). Application of lamivudine combined with low-dose hepatitis B immunoglobulin in the treatment of hepatitis B virus related liver disease (Lu et al., 2008). Immunosuppressive regimens are triple therapies, including tacrolimus, steroids and mycophenolate mofetil. The amount of steroids during the operation is 6-8 mg/kg, and the dosage will be reduced gradually according to the pathogenesis.