Mild hepatic disorder occurs early after Fontan procedure and worsens at time progresses

Hamamichi Y., Ishii Y., Ishii R., Terashima Y., Narita J., Kawazu Y., Inamura N., Kayatani F., Kawata H., Kishimoto H.
Osaka Medical and Research Institute for Maternal and Child Health, Osaka, Japan

Background: Hepatic impairment may occur early after Fontan procedure. But hepatic damage is indistinct because of its mild change. In Fontan patients elevation of central venous pressure causes hepatic fibrosis for a long time. In patients with hepatitis thrombocytopenia is index of liver fibrosis. We attempted to detect the process of hepatic lesion from early-stage to middle-stage in Fontan patients.

Methods: The medical records of 122 patients after Fontan surgery were reviewed. Blood tests ranged 1 month to 15 years after procedure. We divided the term after Fontan four groups: from 1 month to 2 years; from 2 to 5 years; from 5 to 10 year; from 10 to 15 years. Labolatory data of healthy person were used as control who were matched sex and age. First, indexes of liver abnormality in Fontan patients were compared with those of controls. Second, we defined thrombocytopenia as platlet count (Plt) 19.0x104/ml under, which was the lower (2.5th percentile) reference limit of Plt in control. We compared cardiac functions between patients with thrombocytopenia and patients with non-thrombocytopenia. Results: Hepatopacific markers, such as alanine aminotransferase, aspartate aminotransferase, lactate dehydrogenase, and gamma-glutmyl transpeptidase were significantly higher than those of matched controls in all periods after Fontan procedure. The levels of total bilirubin became significantly higher after 2 years postoperatively and its disparity continued. Similarly Plt decreased after 5 years and total protein after 10 years. Plt lessened more highly in later stage. The number of patients with thrombopenia was 46 in Fontan group. The levels of brain natriuretic peptide and ejection fraction of major ventricle were not different in patients with thrombopenia and in patients with non-thrombopenia. Conclusion: Our study showed liver abnormality arose early after Fontan procedure. Each variation of marker appeared in different period. Thrombocytopenia, which reflected hepatic fibrosis, occurred in Fontan patients certainly and developed with time. But hepatic impairment in Fontan patients was not introduced by viral hepatitis. We don’t know whether thrombocytopenia is proper to assess hepatic fibrosis in Fontan patients. We should establish assessment method of liver damage by routine blood tests in patients after Fontan procedure.