ABSTRACT

Does the orthotopic liver transplantation have an impact on hypersplenism in children with chronic liver disease and portal hypertension?

Introduction

Hypersplenism is a condition associated with hyperactivity of the enlarged spleen, leading to destruction of blood cells and causing anemia, leucopenia and thrombocytopenia. Portal hypertension is one of possible causes of splenomegaly. Hypersplenism occurs in 15 to 70% of patients with chronic liver disease and increases risk of bleeding from gastrointestinal tract.

Restoration of normal portal blood flow after liver transplantation (LTx) should in theory reduce the signs of hypersplenism, such as thrombocytopenia and splenomegaly.

The aim of the study

The aim of the study is to evaluate the impact of the orthotopic liver transplantation on regression of hypersplenism in children with chronic liver disease and portal hypertension and to identify risk factors of persistent thrombocytopenia after LTx.

Material and methods

Between 2000-2017 in Department of Pediatric Surgery and Organ Transplantation of Children's Memorial Health Institute in Warsaw 678 pediatric liver transplantations were performed. In this group 174 children with thrombocytopenia at transplantation were identified. Inclusion criteria were: thrombocytopenia below $75 \cdot 10^3/mm^3$ with or without leucopenia, splenomegaly and chronic liver disease. Assumed follow-up time was 5 years after transplantation. Exclusion criteria were: LTx for acute liver failure or oncological disease, retransplantation or death in 5-year observation time. 69 children matched those criteria. Demographical data, the course of underlying liver disease, hypersplenism signs before and after LTx, perioperative period and follow-up data were investigated.

Results

Statistical analysis showed increase in platelet count from 2 weeks after LTx. A rise in platelet count above $75 \cdot 10^3/mm^3$ in early postoperative period was observed in 84% of children, in 94,2% after a year, 93,1% and 95,2% respectively 3 and 5 years after

transplantation. After one, 3 and 5 years of follow-up normal thrombocytes level was reported in 46,4%, 55,9% and 69,6% of patients.

A significant reduction in spleen size and SSHR was detected one year after transplantation. This decreasing tendency continued throughout the whole observation time.

The platelet count below $50 \cdot 10^3/mm^3$ before transplantation, hemodynamic disorders of portal flow and poor allograft function were factors contributing to slower improvement of hypersplenism signs. No impact of immunosuppressive treatment on hypersplenism regression was detected. The platelet count at 4 weeks after LTx was prognostic factor for persistent thrombocytopenia at 1 year post-transplant.

Conclusion

The orthotopic liver transplantation, by normalizing portal pressure, significantly reduces the severity of hypersplenism present before transplantation and increases platelet count. In most patients, thrombocytopenia is reduced by at least one grade or platelet count normalizes with concomitant reduction in spleen size.

A significant rise in platelet count is observed as early as two weeks after LTx, stabilization of this parameter occurs approximately 12 months after liver transplant. Spleen dimensions systematically decrease throughout the post-transplantation observation period.

Factors negatively affecting the rate and extent of resolution of hypersplenism after liver transplantation include severe hypersplenism before LTx, abnormal function of the transplanted organ and portal flow disturbances.

Isolated hypersplenism without additional risk factors (significant spleen size, GRWR<1%, thrombocytopenia below 50 $10^3/mm^3$) is not an absolute indication for simultaneous splenectomy during LTx in children.

Special immunosuppressive protocol after liver transplantation in children with hypersplenism does not seem to be justified. However the coexistence of persistent hypersplenism, perioperative complications and adverse effects of drugs used in the early period after transplantation may lead to severe thrombocytopenia and the need to modify immunosuppression.