Liver fibrosis and steatosis in children after effective treatment of chronic hepatitis C using direct acting antivirals

Maria Pokorska-Śpiewak¹, Ewa Talarek², Anna Dobrzeniecka³, Małgorzata Aniszewska⁴, Magdalena Pluta¹, Magdalena Marczyńska¹

¹Medical University of Warsaw, Regional Hospital of Infectious Diseases in Warsaw, Warsaw, Poland, ²Medical University of Warsaw, Warsaw, Poland, ³Regional Hospital of Infectious Diseases in Warsaw, Warsaw, Poland, ⁴Medical University of Warsaw, Regional Hospital of Infectious Diseases in Warsaw, Warsaw, Poland

Email: maria.pokorska-spiewak@wum.edu.pl

Background and Aims: In this study we aimed to examine the long-term influence of successful treatment with direct acting antivirals (DAAs) on liver fibrosis and steatosis in children with chronic hepatitis C (CHC).

Method: Of 135 children aged 5 to 18 years who had been treated with DAAs due to CHC in our Department between July 2019 and December 2023 as a part of two projects: PANDAA-PED (12-week treatment with sofosbuvir/velpatasvir, SOF/VEL) and POLAC Project (12/24-week treatment with sofosbuvir/ledipasvir, SOF/LDV, and 8-week with glecaprevir/pibrentasvir, GLE/PIB), we selected successfully treated participants, who completed control evaluation at one-year posttreatment. Liver fibrosis and steatosis were assessed using transient elastography at baseline, 12-week posttreatment, and one-year posttreatment. Significant fibrosis was diagnosed when liver stiffness measurement (LSM) was >7 kPa, cirrhosis when LSM was ≥12.5 kPa, and steatosis when controlled attenuation parameter (CAP) was >238 dB/m.

Results: We identified 67 patients (32 boys, 48%) eligible for the study: 49 after SOF/VEL treatment, 9 after GLE/PIB, and 9 after SOF/LDV. At baseline, the mean age was 10.4±2.9 years. There were 62 (93%) patients infected vertically, 58 (87%) were treatment-naïve, 41 (61%) were infected with genotype 1. All children had undetectable HCV RNA at one-year posttreatment. At baseline, 5/67 (7%) participants presented with significant fibrosis, including 2 with cirrhosis. At 12-week posttreatment, only 2 of these patients had LSM >7 kPa, and one-year posttreatment only one cirrhotic patient had abnormal LSM. Significant fibrosis was diagnosed when liver stiffness measurement (LSM) was >7 kPa, cirrhosis when LSM was ≥12.5 kPa, and steatosis when controlled attenuation parameter (CAP) was >238 dB/m. Number of patients with steatosis increased from 4 (6%) at baseline to 7 (10%) at 12-week posttreatment, and to 8 (12%) at one-year posttreatment.

Conclusion: Successful treatment with DAAs leads to regression of significant liver fibrosis in most cases. Increased CAP values after therapy require further analysis on larger groups of patients.