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Chronic high EBV DNA load carriage among paediatric liver transplant patients

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Background: High Epstein-Barr virus (EBV) loads are associated with increased risk for post-transplant lymphoproliferative disorder (PTLD) development. Monitoring EBV loads and pre-emptive reduction of immunosuppression lead to decrease in incidence of PTLD. However serial EBV load monitoring in whole blood identified asymptomatic patients with chronic high viral loads (CHVL). The clinical significance of CHVL carriage is unknown. The aim of the study was to analyse the incidence, characteristics and outcome of CHVL carriage among paediatric liver transplant (LTx) recipients.

Material/methods: Clinical and virological data were collected from 101 children (mean age at LTx = 6.8 years, range: 0.1 - 17.8 years) who underwent LTx at The Children's Memorial Health Institute in Warsaw between January 2013 and December 2015. EBV DNA loads were routinely measured in whole blood as a part of surveillance protocol after LTx. Median follow-up period was 21 months (interquartile range, IQR: 13.8 - 28.9). CHVL carriage was defined as the presence of high EBV loads (>5000 copies/mL) for >50% samples for ≥6 months following either asymptomatic state or resolution of EBV disease. PTLD was defined histologically using WHO definitions.

Results: Seventeen (16.8%) of 101 patients met definition of CHVL carriers. The median time to CHVL onset was 7.8 months after LTx (IQR: 6.3 - 12 months). The CHVL carrier state resolved without progression to PTLD in 6/17 patients, while CHVL continued to persist without evidence of

clinical symptoms in 10 patients. The median time to resolution in those who resolved their CHVL carrier state was 8.8 months (IQR: 7.4 - 12.1 months). The 10 patients who continued CHVL carriage experienced their carrier state for a median time of 10.6 months (IQR: 7.8 - 17.2 months) during the study period. One child developed biopsy-proven PTLD (plasmatic hyperplasia) at 16.4 months after LTx, and 8,6 months after CHVL-onset. Four children experienced more than one episode of CHVL carriage. When compared to patients with undetectable, low or transient high viral load, CHVL carriers were significantly younger at LTx (2.92 vs 7.64 years, $p = 0.004$) and higher proportion of them were EBV-negative prior to LTx (82.3% vs 48.7%; $p = 0.01$). No difference was observed with regard to through tacrolimus levels, concomitant CMV infection or occurrence of acute rejection episodes within the first year after LTx.

Conclusions: CHVL carriage is frequent among paediatric patients after LTx and can lead to PTLD development. Younger and EBV-negative prior LTx children are more likely to be CHVL carriers.