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E0304 Lack of anti-hepatitis B core antibodies in patients infected with hepatitis B virus

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Background: Diagnosis of hepatitis B virus (HBV) on-going infection is based on the detection of HB surface antigen (HBsAg) and anti-HB core antibodies (anti-HBcAb) in the serum. Anti-HBcAb presence indicates HBV exposure and therefore past or on-going infection. They are known very robust serological markers, with, usually, life-long persistence in case of hepatitis B resolution. Between 2013 and 2017 we diagnosed HBV chronic infection in five patients lacking detectable anti-HBcAb. We analyzed here the epidemiological, clinical and virological features of these infections.

Materials/methods: Routine HBV testing was performed between 06/2013 and 01/2017 (43 months) in Marseille university hospitals, southeastern France. HBV serologies were performed using Architect Abbott assays. HBsAg-positivity was confirmed by seroneutralisation. Serum HBV DNA detection/quantification was performed using the RealTime HBV viral load Abbott assay or the VERIS/MDx System HBV assay. Direct population sequencing of the HBsAg/reverse transcriptase (RT) coding-region and the pre-core/core region of the HBV genome were performed using in-house protocols. HBV sequences were analyzed using the geno2pheno HBV tool, sequence comparisons and phylogenetic analysis.

Results: We observed this lack of anti-HBcAb in five HBV-chronically-infected patients out of 65,910 patients in whom a HBV serology was performed. This serological profile was observed in 0.56% of the 889 patients diagnosed as HBsAg-positive. These five patients had a mean age of 55±15 years, and 4 (80%) were male. Three patients were HIV-infected, with a low CD4 cell count, below 200/mm^3. Two of them were HIV-Hepatitis C virus-co-infected. Mean alanine aminotransferase level was 32±25 IU/L, liver cytolysis being only found in one case. HBV replication was found in three cases, HBV DNA load being above 2 $\log_{10}$IU/mL. HBV genotype was D3 in two cases, A2 and B2 in one case each, and unknown in one case due to the absence of detectable HBV DNA. Core gene sequences (799-nucleotide-long) did not show particular deletion or insertion.

Conclusions: Even if this HBV serological profile is rare, it could appear in few patients, particularly in immunocompromised. This has to be considered because it may impair chronic HBV infection diagnosis in absence of HBsAg testing.