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Summary

HBV INTEGRATION PATTERNS IN HBSAG-POSITIVE CHB AND HCC PATIENTS UNDER ENTECAVIR TREATMENT

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Hepatobiliary

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Long-term entecavir therapy reduces HBV integration intensity in chronic hepatitis B liver tissue but not in adjacent non-tumorous tissue of hepatocellular carcinoma patients, where integration remains five-fold higher than in chronic hepatitis B.

KEY POINTS

- In chronic hepatitis B, entecavir significantly reduced the intrahepatic HBV/HG19 coverage ratio compared with treatment-naïve patients (9.65 vs. 14.98, $p < 0.0001$), but in adjacent non-tumorous tissues of hepatocellular carcinoma, the ratio remained markedly elevated and unchanged between entecavir-treated and treatment-naïve groups (47.42 vs. 47.10, $p > 0.9999$).
- Hepatocellular carcinoma patients showed a three- to five-fold higher HBV integration intensity in adjacent non-tumorous tissues compared with chronic hepatitis B patients ($p < 0.05$).
- All HBV DNA integration events were characterized by a high proportion (>80%) of chimeric alignment fragments; entecavir did not substantially reduce this proportion in either chronic hepatitis B or hepatocellular carcinoma adjacent tissues.
- HBV integrations were randomly distributed across chromosomes in both chronic hepatitis B and hepatocellular carcinoma cohorts, with preferential enrichment at enhancer regions in the HBV genome, regardless of entecavir treatment.
- The presenter concluded that long-term entecavir neither reverses genomic damage nor reduces integration intensity in hepatocellular carcinoma, indicating limited cancer prevention, and suggested that high-intensity HBV DNA integration may serve as a potential molecular early warning for hepatocellular carcinoma.

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