Category: Research Spotlight

Preventing the preventable: Why adolescent liver cancer is a policy failure, not a clinical inevitability

Inae Kim

MSc in International Health and Tropical Medicine, 2025

Hepatocellular carcinoma (HCC), also known as liver cancer, is often explained as the price of time: decades of cumulative liver injury eventually tipping into malignancy. That fits many high-income settings, where HCC commonly follows long-standing cirrhosis and is increasingly linked to metabolic disease.[1-2] Yet in parts of sub-Saharan Africa (SSA), HCC is observed at younger ages and remains tightly coupled to chronic hepatitis B virus (HBV) infection.[3]

This study presents a simple argument: *adolescent HCC should signal gaps in HBV prevention and early-life health system delivery*, rather than as an unfortunate clinical rarity.

Why adolescence reframes the problem

Adolescence is not a life stage in which clinicians or families anticipate end-stage liver disease. A cancer diagnosis at 13 or 18 years old interrupts schooling, destabilises households, and enforces long-term economic consequences. What makes adolescent HCC especially confronting is that it is not only catastrophic; it is also preventable. In HBV-endemic settings, it points back to early acquisition of infection, and ultimately to delayed, inconsistent or incomplete prevention measures.

A preventable early infection with lifelong consequences

The timing of HBV infection is decisive. When HBV is acquired at birth or in early childhood, it is more likely to persist as chronic infection, silently shaping risk across the life course.[6] Chronic HBV increases risk through sustained inflammation and direct viral effects, including integration of HBV DNA into the host genome. These mechanisms can accelerate disease progression even without the classic decades-long trajectory.[5] In SSA, mother-to-child transmission and early childhood transmission remain key routes sustaining HBV endemicity, with downstream implications for chronic liver disease and cancer.[6]

Using an “extreme outcome” to interrogate the system

The Groote Schuur Hospital (GSH) study was a single-centre descriptive case series of adolescents aged 10-19 years with HCC treated between 1 January 2012 and 31 December 2024.[4] Clinical records and imaging were reviewed for demographics, symptoms, laboratory markers, tumour stage, treatments received, and survival.[4] The design interrogates a critical question: what does adolescent HCC look like at presentation, and what does that imply about missed upstream opportunities for HBV control?

Five adolescents, late presentation, limited options, premature death

Among 726 patients managed with HCC at GSH, five were adolescents (0.5%). Rarity should not reassure, as all five adolescents had chronic HBV infection.[4] Presentations were late and clinically overt, most commonly abdominal pain and/or an abdominal mass. At diagnosis, every patient had advanced disease: four had Barcelona Clinic Liver Cancer (BCLC) stage C and one stage D. Prognostically adverse features were frequent, including

extrahepatic metastases (40%) and portal vein tumour thrombosis (60%), findings that typically exclude curative treatment options.[4]

Only one adolescent underwent liver resection; two received systemic therapy (sorafenib or lenvatinib), and two received best supportive care. Outcomes were stark as the median survival was 137 days, and only one patient remained alive at analysis. In plain terms, these were teenagers dying within months from a cancer that, in this series, traced back to a preventable infection.[4]

HBV prevention as a public health imperative

Adolescent HCC should be treated as an alarm for prevention. The aggressive and advanced disease mirrors findings from other HBV-endemic settings where early acquisition drives earlier-onset HCC.[3,7]

South Africa introduced national HBV immunisation in 1995, yet targeted HBV birth-dose vaccination for neonates born to HBsAg-positive mothers was introduced only in 2023.[4,10-11] Meanwhile, ongoing maternal and paediatric infection persists: HBsAg prevalence among pregnant women has been reported at 11.2%, and among children under five years at 4.8%.[4,8-9] These figures represent continuing opportunities for infection when risk is highest, and protection most efficient.

Targeted prevention, including antenatal screening, maternal antivirals when indicated, and prophylaxis for exposed infants, is crucial. It depends on early antenatal attendance, consistent testing, reliable linkage to care, and uniform implementation across facilities.[4,10-11] Universal hepatitis B birth-dose vaccination within 24 hours, as recommended by the World Health Organisation (WHO), is designed to remove these points of failure by providing early protection regardless of maternal HBV, yet not implemented in South Africa. Regional studies suggest universal birth-dose delivery is feasible in SSA but requires deliberate investment: cold-chain resilience, staffing at delivery sites, monitoring systems, and community engagement.[6,10-11] In practice, the birth dose is not merely an injection; it is a test of whether an equitable, time-critical intervention can be delivered to every newborn.

Conclusion

The adolescents in this series arrived beyond the reach of curative treatment options.[4] If chronic HBV acquired early in life is the proximate driver, then adolescent HCC is a preventable endpoint, and its occurrence is best read as a policy failure rather than a clinical inevitability. Preventing even one case is not only saving a life; it is preserving education, family stability, and future livelihoods, while demonstrating that elimination targets can be made real where they matter most: at birth.

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Inae is a South Korean-South African junior doctor, whose work sits at the intersection of clinical research, particularly in viral hepatitis B and hepatocellular carcinoma (liver cancer), and global health. She holds both MBChB and BMedSci (Hons) in Infectious Diseases and Immunology from the University of Cape Town. She is currently pursuing an MSc in International Health and Tropical Medicine at Nuffield Department of Medicine to deepen her expertise in global health and aims to integrate this with her clinical research interests during her DPhil in Clinical Medicine, which she will begin at Oxford in 2026/27. Learn more about Inae here.