Chronic hepatitis B in British Columbia: Updates from the 2025 Canadian Association for the Study of the Liver/Association of Medical Microbiology and Infectious Disease Canada guidelines

Recent guidelines on treating hepatitis B recommend universal one-time screening for adults, reflex hepatitis D virus testing, expanded treatment indications, and updated hepatocellular carcinoma surveillance.

Kai Zhu, MD, Yvette Ysabel Yao, MD, Richard L. Morrow, PhD, Hin Hin Ko, MD, FRCPC, FAASLD, Nancy Fu, MD, MHSc, FRCPC

ABSTRACT: Chronic hepatitis B is a significant cause of liver disease worldwide and an important public health issue in Canada. In British Columbia, hepatitis B virus (HBV) disproportionately affects immigrants and newcomers from endemic regions, which contributes to ongoing disparities in liver-related morbidity and mortality. Despite effective antiviral therapies, gaps remain across the HBV care cascade, including underdiagnosis, limited engagement in care, and undertreatment. The recently released updated 2025 Canadian Association for the Study of the Liver/Association of Medical Microbiology and Infectious Disease Canada guidelines introduce several important changes, including universal one-time screening for adults, reflex hepatitis D virus testing, expanded treatment indications, and updated hepatocellular carcinoma surveillance recommendations. This review summarizes the epidemiology of chronic HBV in BC and highlights key guideline updates relevant to screening, diagnosis, and treatment of chronic hepatitis B.

Dr Zhu is a postgraduate fellow in the Division of Gastroenterology, University of British Columbia. Dr Yao is a postgraduate resident in the Department of Medicine, UBC. Dr Morrow is a postdoctoral research fellow at the BC Centre for Disease Control and a postdoctoral research fellow in the School of Population and Public Health, UBC. Dr Ko is a clinical professor in the Division of Gastroenterology, UBC. Dr Fu is a clinical assistant professor in the Division of Gastroenterology, UBC.

This article has been peer reviewed.

Hepatitis B virus (HBV) is a DNA virus in the Hepadnaviridae family that can cause a wide spectrum of liver disease, ranging from acute infection to cirrhosis and hepatocellular carcinoma (HCC).[1] Untreated chronic HBV infection is associated with a substantial risk of complications, including liver failure, HCC, extrahepatic manifestations, and premature death.[2,3] Chronic infection is often asymptomatic in its early stages, and many individuals remain undiagnosed until late complications develop.[4]

HBV infection affects an estimated 254 million people worldwide and approximately 262 000 people in Canada.[5,6] Canada has committed to the World Health Organization goal of eliminating HBV as a public health threat by 2030, including targets of a 90% reduction in new cases of chronic HBV infection, diagnosis of 90% of infected individuals, and treatment of 80% of eligible patients.[7,8] Despite these targets, significant gaps remain across the HBV care cascade. It is estimated that in Canada, approximately 29% of individuals with HBV infection remain undiagnosed, about 40% of those diagnosed are not engaged in care, and only 23% of those eligible for treatment receive antiviral therapy.[2,3]

Recently updated Canadian guidelines on the management of chronic hepatitis B were developed jointly by the Canadian Association for the Study of the Liver (CASL) and the Association of Medical Microbiology and Infectious Disease Canada (AMMI).[9] The 2025 guidelines recommend expanded screening, laboratory evaluation, and treatment strategies aimed at improving detection and management of HBV infection. In this article, we highlight key updates in the 2025 guidelines compared with the 2018 Canadian guidelines, with a focus on HBV screening, reflex hepatitis D virus (HDV) testing, laboratory assessment, and antiviral therapy.[9]

Epidemiology

Canada is considered to have low HBV endemicity; however, important regional and population-level variation exists. Immigrants and newcomers account for approximately two-thirds of HBV infections in Canada, and they experience a

disproportionate burden of HBV-related complications, including higher rates of hospitalization and liver cancer mortality.[2,10-13] A population-based study estimated that immigrants with chronic HBV infection lose an average of 4.6 life-years compared with individuals without HBV.[14]

In British Columbia, many HBV infections occur among newcomers from countries where HBV is endemic and routine screening and prophylaxis may be limited.[15] Approximately 15% of the population in BC was born in regions with intermediate to high HBV endemicity, defined as hepatitis B surface antigen (HBsAg) prevalence higher than 2%.[2,16-19] Ethnicity has been shown to be associated with HBV infection in the province. South and East Asian individuals are disproportionately affected by chronic HBV infection, and risk is further increased among those with lower socioeconomic status.[11] As of 2023, the highest rate of reported HBV cases in BC occurred in Richmond, where approximately 60% of the population is first-generation immigrants and most recent immigrants are of Chinese origin.[20,21]

Several populations are underserved or disproportionately affected by HBV infection, including individuals who were born in or have lived in HBV-endemic regions, Indigenous people, people living with HIV, individuals with a history of incarceration, people who use unregulated drugs, and individuals with a family history of HBV or HCC.[22] Notably, the disproportionate impact of hepatitis B on Indigenous people reflects historical and ongoing inequities stemming from systemic racism and settler colonialism. HBV risk factors include sexual exposure to an infected partner, sharing personal care items with someone with HBV infection, tattoos or body piercings performed with inadequate infection control practices, occupational exposure such as needlestick injury, and chronic kidney disease requiring hemodialysis.[10,22,23]

Despite the higher burden of disease in some populations, disparities in HBV outcomes are also influenced by important social determinants of health that affect access to screening, longitudinal care, and antiviral treatment **[Table 1]**.[24-30] A survey on awareness and knowledge of HBV in Asian communities in BC reported that 79% of respondents had a general awareness of HBV, although many had misconceptions about the natural history, transmission,

Clinical pearls: What to know about hepatitis B care

- Chronic hepatitis B virus (HBV) infection is often asymptomatic until advanced liver disease develops.
- All adults aged 18 years or older should undergo one-time HBV screening with hepatitis B surface antigen (HBsAg), hepatitis B surface antibody, and hepatitis B core antibody.
- Patients who are HBsAg-positive should undergo reflex hepatitis D virus (HDV) testing.
- Initial evaluation for patients who are HBsAg-positive should include HBV DNA, alanine aminotransferase (ALT) levels, hepatitis B e antigen status, and fibrosis assessment.
- Antiviral treatment should be initiated in patients with HBV cirrhosis, HBV-related hepatocellular carcinoma (HCC), HBV extrahepatic manifestations, planned initiation of potent immunosuppression, elevated ALT above the upper limit of normal with HBV DNA higher than 2000 IU/mL, or significant fibrosis (F2 or higher).
- Antiviral treatment should be considered in those gray-zone patients who have normal ALT, high HBV DNA (higher than 2000 IU/mL), and high risk factors for progression of cirrhosis or HCC. Treatment decisions should involve shared decision making with the patient.
- First-line therapies include entecavir, tenofovir disoproxil fumarate, and tenofovir alafenamide.
- HCC surveillance with abdominal ultrasound and alpha-fetoprotein testing every 6 months is recommended for high-risk groups, including individuals with cirrhosis, men aged 40 years or older, women aged 50 years or older, individuals of African ancestry aged 30 years or older, those with a first-degree family history of HCC, and patients with HIV or HDV co-infection.

TABLE 1. Social determinants of health influencing hepatitis B virus (HBV) screening, access to care, and treatment outcomes.

Social determinant or barrier	Potential impact on HBV care
Language barriers[24]	Reduced understanding of HBV transmission, screening, and treatment
Limited health literacy[25]	Delayed diagnosis and reduced engagement in longitudinal care
Lack of access to primary care[26]	Reduced screening and delayed specialist referral
Immigration and newcomer status[27]	Difficulty navigating the health care system and accessing culturally appropriate care
Stigma related to HBV infection[28]	Reduced disclosure, testing, and treatment uptake
Low socioeconomic status[27]	Difficulty affording transportation, child care, or time away from work for appointments
Lack of extended medication coverage[29]	Reduced access to antiviral therapy
Cultural barriers and mistrust of health systems[25]	Reduced engagement in preventive care and follow-up
Rural or remote residence[30]	Limited access to laboratory testing and specialist care

and health consequences of HBV.[31] Barriers to HBV screening and care include language barriers, lack of familiarity with the health care system, difficulty finding a family physician, and stigma associated with HBV diagnosis.[32-35] Among individuals who are aware of their HBV diagnosis, additional socioeconomic barriers may include lack of extended health care benefits for medication coverage, travel and child care costs, and time taken off work to attend medical appointments or laboratory testing.[36] Given persistent gaps in the HBV care cascade and ongoing inequities in HBV prevalence and outcomes, chronic HBV infection remains an important public health challenge in BC.

HBV screening

Early identification of chronic hepatitis B infection is essential for preventing long-term complications, including cirrhosis and HCC. Screening also allows timely vaccination of individuals who lack immunity.

The 2025 CASL/AMMI guidelines represent a major shift from the earlier 2018 recommendations regarding HBV screening and monitoring. The 2018 guidelines emphasized a risk-based approach that targeted screening toward individuals in higher-risk groups.[37] In contrast, the 2025 update recommends universal one-time screening for all adults aged 18 years or older using a triple serologic panel consisting of hepatitis B surface antigen (HBsAg), antibody to HBsAg, and antibody to hepatitis B core antigen.[9] Reflex testing for HDV infection is also recommended for individuals who test positive for HBsAg. Individuals who remain nonimmune or who have ongoing risk factors should undergo periodic repeat testing. This universal screening approach aims to improve case detection among asymptomatic individuals and eliminates the need for complex individual risk assessments. The updates in the CASL/AMMI guidelines from 2018 to 2025 are summarized in **Table 2**.

Screening recommendations during pregnancy remain particularly important, because most chronic HBV infections worldwide are acquired through perinatal transmission.[38] All pregnant people should undergo HBsAg testing in the first trimester of every pregnancy.[9] Despite these recommendations, screening remains imperfect. A population-based study from Ontario reported prenatal HBV screening rates of approximately 92.7%, with suboptimal follow-up laboratory assessment among patients who tested positive for HBsAg.[39]

HBV diagnosis and initial evaluation

Diagnosis of chronic HBV infection is established by the presence of HBsAg for longer than 6 months. Once infection is identified, further laboratory assessment is required to determine the phase of disease, level of viral replication, and degree of liver injury. Initial evaluation typically includes hepatitis B e antigen (HBeAg), antibody to HBeAg (anti-HBeAg), quantitative HBV DNA, and alanine aminotransferase (ALT) levels. Assessment of liver fibrosis using noninvasive tests such as transient elastography may also help guide treatment decisions.

Testing for HDV infection has also evolved in recent guidelines. HDV requires HBV for replication and causes the most severe form of viral hepatitis. Because HDV shares similar transmission routes with HBV, co-infection may occur through simultaneous exposure.[40] Earlier guidelines recommended HDV testing only in higher-risk groups, including individuals from endemic regions, people who inject drugs, those with unexplained ALT elevation despite antiviral therapy, and patients with advanced liver disease. The 2025 guidelines now recommend universal reflex anti-HDV testing for all individuals who are HBsAg-positive, with repeat testing if ongoing risk factors or suggestive clinical features persist.[9]

Recent updates have also addressed the management of patients in the so-called gray zone, whose serum HBV DNA and ALT levels cannot be readily categorized into a defined phase of chronic HBV infection.[41] The natural history and optimal management of these patients remain areas of ongoing study. Under the 2018 guidelines, treatment was generally recommended when HBV DNA levels exceeded 2000 IU/mL and ALT levels were persistently above the upper limit of normal for approximately 6 months, or when significant fibrosis was

TABLE 2. Summary of updates in the Canadian Association for the Study of the Liver/Association of Medical Microbiology and Infectious Disease Canada hepatitis B virus (HBV) guidelines from 2018 to 2025.[9,37]

	2018 guidelines	2025 guidelines
Screening population	Risk-based screening for high-risk groups	Universal one-time screening for adults ≥ 18 years of age, using a triple serologic panel (HBsAg, anti-HBs, anti-HBc)
HDV testing	Testing in high-risk HBV patients	Universal reflex anti-HDV testing for all HBsAg-positive individuals
HCC surveillance	Ultrasound every 6 months in defined high-risk groups	Ultrasound plus AFP every 6 months in high-risk groups
Treatment eligibility	HBV DNA > 2000 IU/mL with ALT > ULN or significant fibrosis	Expanded indications, including select patients with normal ALT
Treatment endpoint	Treatment cessation mainly after HBsAg loss or HBeAg seroconversion with consolidation therapy	Additional criteria for stopping therapy in non-cirrhotic HBeAg-negative patients using quantitative HBsAg thresholds
Role of quantitative HBsAg	Limited clinical role	Used to guide treatment cessation and risk stratification in selected patients

AFP = alpha fetoprotein; ALT = alanine aminotransferase; anti-HBc = hepatitis B core antibody; anti-HBs = hepatitis B surface antibody; HBeAg = hepatitis B e antigen; HBsAg = hepatitis B surface; HCC = hepatocellular carcinoma; HDV = hepatitis D virus; ULN = upper limit of normal.

present.[37] The 2025 guidelines adopt a broader treatment approach that includes selected populations with normal ALT levels. In particular, treatment is recommended for individuals older than 40 years of age with HBV DNA levels higher than 2000 IU/mL, regardless of HBeAg status, as a strategy to reduce the risk of HCC.[9]

Together, these changes reflect a broader and more proactive approach to HBV detection and management that emphasizes earlier diagnosis, more comprehensive risk stratification, and expanded treatment strategies aimed at reducing long-term complications.

Chronic hepatitis B treatment

The main treatment goals for chronic hepatitis B are to reduce disease-related morbidity and mortality.[42] Functional cure is defined as sustained loss of HBsAg with undetectable HBV DNA after completion of treatment. This outcome demonstrates good durability after treatment cessation and is associated with improved clinical outcomes. However, functional cure is achieved in only 1% to 12% of patients who receive nucleos(t)ide analogue therapy.[23] For most patients, a more practical treatment goal is sustained virologic suppression with undetectable HBV DNA and normalization of ALT levels.

Chronic hepatitis B treatment indications

The 2025 CASL/AMMI guidelines emphasize increasing awareness and treatment of patients in the gray zone. These patients do not fit within the traditional phases of HBV infection and account for approximately 30% to 50% of individuals with chronic hepatitis B.[42] The most common gray-zone subgroup includes patients who are HBeAg-negative and have normal ALT levels and HBV DNA higher than 2000 IU/mL **[Table 3]**. Increasing evidence demonstrates that gray-zone patients are at increased risk of progressive liver disease and that antiviral treatment is associated with reduced rates of progression to cirrhosis, hepatic decompensation, and HCC.[43-45] As a result, the updated 2025 CASL/AMMI guidelines have expanded treatment criteria and support earlier treatment initiation in selected patients with normal ALT levels.[9] These indications are summarized in the **Figure**.

In BC, PharmaCare Special Authority provides indefinite coverage for lamivudine, tenofovir disoproxil fumarate (TDF), tenofovir alafenamide (TAF), and entecavir in patients with HBV DNA higher than 2000 IU/mL and ALT levels above the upper limit of normal. Coverage is also available for patients with evidence of fibrosis stage F2 or higher, as assessed by liver biopsy, transient elastography (FibroScan), or the aspartate-aminotransferase-to-platelet ratio index. PharmaCare Special Authority criteria have not yet been updated to reflect the new guideline recommendations. During this interim period, clinicians may discuss self-funded antiviral therapy with patients or closely monitor those who do not meet coverage criteria and initiate treatment once eligibility is reached. In selected circumstances where antiviral therapy is clinically indicated but falls outside the listed criteria, such as hepatitis B prophylaxis in the setting of potent immunosuppression, additional clinical information may be submitted with the Special Authority application for review by the BC Hepatitis Drug Benefit Adjudication Advisory Committee.

Chronic hepatitis B antivirals

The first-line antiviral agents recommended for the treatment of chronic hepatitis B are entecavir, TDF, and TAF. These nucleos(t)ide analogues are preferred because of their high antiviral potency, favorable safety profiles, and very low rates of antiviral resistance.[46] TDF or TAF is preferred over entecavir in patients with prior exposure to lamivudine because lamivudine resistance compromises the efficacy of entecavir. In patients with renal impairment or risk factors for

TABLE 3. Clinical phases in chronic hepatitis B.[9,37]

Clinical phase	HBeAg-positive chronic infection	HBeAg-positive chronic hepatitis	HBeAg-negative chronic infection	HBeAg-negative chronic hepatitis	Gray zone	HBsAg-negative
Synonymous terminology	Immune tolerant, high replication	Immune active, high replication	Inactive carrier, low replication	Immune active, high replication	Indeterminate phase	Functional cure, resolved/occult
HBsAg	+					–
HBeAg	+		–		+/–	–
Anti-HBc	+					
Anti-HBe	–		+		+/–	+
Anti-HBs	–					+/–
ALT	Normal	Elevated or fluctuating	Normal	Elevated or fluctuating	Normal or mildly elevated	Normal
HBV DNA (IU/mL)	$> 10^7$	10^4–10^7	Often < 2000	10^3–10^7	Often > 2000	Undetectable

ALT = alanine aminotransferase; anti-HBc = hepatitis B core antibody; anti-HBe = hepatitis B e-antibody; anti-HBs = hepatitis B surface antibody; HBeAg = hepatitis B e antigen; HBsAg = hepatitis B surface antigen; HBV = hepatitis B virus.

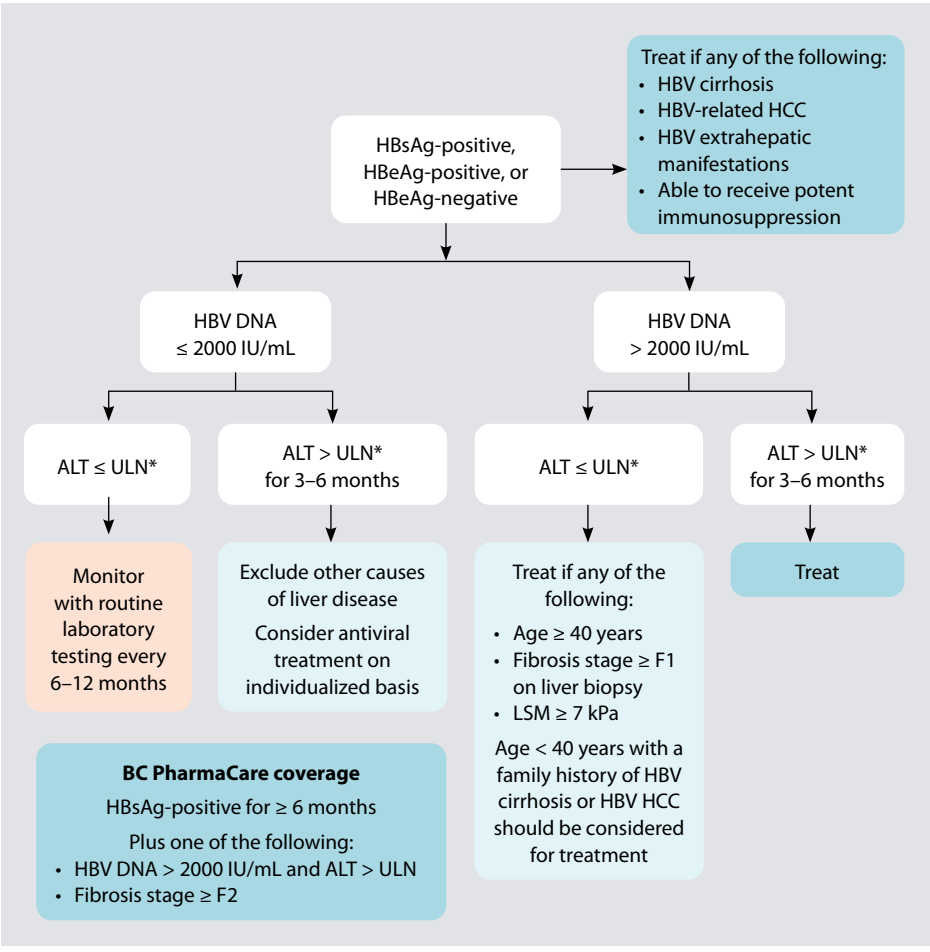

FIGURE. Canadian Association for the Study of the Liver/Association of Medical Microbiology and Infectious Disease Canada treatment recommendations for chronic hepatitis B.[9]

ALT = alanine aminotransferase; HBeAg = hepatitis B e antigen; HBsAg = hepatitis B surface antigen; HBV = hepatitis B virus; HCC = hepatocellular carcinoma; LSM = liver stiffness measurement; ULN = upper limit of normal.
* ULN = 25 U/L for females and 35 U/L for males.

osteoporosis, entecavir or TAF is preferred over TDF because long-term use of TDF has been associated with reductions in bone mineral density and renal tubular injury.

Pegylated interferon alfa offers a finite treatment course of 48 weeks and may be appealing for selected patients—for example, young adults who prefer a finite course of treatment. However, treatment requires subcutaneous injections and is associated with significant adverse effects, including flu-like symptoms, neutropenia, thrombocytopenia, depression, and exacerbation of autoimmune disease. Pegylated interferon alfa is also contraindicated in pregnancy, decompensated cirrhosis, autoimmune disease, uncontrolled psychiatric disorders, and cytopenias.[42] Older nucleos(t)ide analogues, including lamivudine, adefovir, and telbivudine, are no longer recommended because of lower antiviral potency and high rates of resistance; lamivudine resistance is reported in up to 70% of patients at 5 years of age.[46]

Treatment endpoints

The ideal endpoint of HBV therapy is functional cure, defined as sustained HBsAg loss with undetectable HBV DNA. This outcome is associated with improved clinical outcomes and durable viral suppression after treatment discontinuation.[47] Treatment endpoints vary according to HBeAg status because it reflects different phases of infection with distinct treatment responses.

In noncirrhotic HBeAg-negative patients, treatment cessation may be considered after loss of HBsAg or when quantitative HBsAg levels are lower than 100 IU/mL in Asian patients or lower than 1000 IU/mL in White patients, followed by an additional 12 months of consolidation therapy. The ethnicity-specific thresholds were derived from cohorts that were overwhelmingly Asian or White, with minimal representation of other ethnic groups. Therefore, there are currently no recommendations in the guidelines for other groups. Although not included in current guideline recommendations, an anti-HBs level higher than 100 mIU/mL at the time of HBsAg loss has been associated with improved clinical outcomes and a lower risk of reverse HBV seroconversion.[48] In contrast, HBeAg-positive patients without cirrhosis may be considered for treatment cessation after HBeAg seroconversion, followed by an additional 12 months of consolidation therapy. Patients should be monitored closely after treatment discontinuation for evidence of virologic relapse. Patients with HBV-related cirrhosis are generally recommended to continue antiviral therapy indefinitely until loss of HBsAg, regardless of HBeAg status. Decisions regarding treatment discontinuation should be made in consultation with a gastroenterologist or hepatologist, an infectious diseases specialist, or another clinician experienced in the management of chronic hepatitis B.

Local centres of excellence include the Digestive Health Centre of BC/Pacific Gastroenterology Associates and the Vancouver General Hospital BC Hepatitis Program. There are also other clinicians and clinics throughout BC with experience in the management of hepatitis B.

Hepatitis B in pregnancy

Perinatal transmission accounts for up to 50% of HBV infections worldwide, but there is substantial variability across geographic regions.[49] Vertical transmission is clinically significant because approximately 90% of neonates infected perinatally develop chronic infection, compared with only about 5% of immunocompetent adults. As a result, all pregnant people should be

screened for HBsAg during the first trimester. If positive, further evaluation should include testing for HBeAg, anti-HBe, HBV DNA, and ALT. Pregnant people who meet standard indications for antiviral therapy should begin treatment promptly. In addition, pregnant people with HBV DNA levels higher than 200 000 IU/mL should initiate antiviral therapy between 28 and 32 weeks of gestation to reduce parent-to-child transmission.[9] TDF is the preferred antiviral during pregnancy based on well-established safety and efficacy data. However, TAF is increasingly recognized as a safe and effective alternative.[50] For patients who are receiving treatment solely to reduce the risk of parent-to-child transmission, antiviral therapy can be continued until 3 months postpartum to reduce the risk of postpartum flares. Breastfeeding is not contraindicated in HBV infection or while receiving TDF or TAF.

All infants born to parents who are positive for HBsAg should receive hepatitis B vaccination and hepatitis B immunoglobulin as soon as possible after birth, followed by completion of the second and third vaccine doses by 6 months of age. The combination of passive and active immunization is approximately 95% effective in preventing parent-to-child transmission.[48] However, the risk of transmission increases in infants born to parents with high HBV viral loads, which is why pregnant people with HBV DNA levels higher than 200 000 IU/mL are recommended to receive antiviral therapy during pregnancy.

Hepatocellular carcinoma surveillance

HCC surveillance is recommended in patients with chronic hepatitis B, because HCC can develop even in the absence of cirrhosis. HBV DNA can integrate into the host genome and disrupt tumor suppressor genes or activate oncogenes, leading to carcinogenesis independent of cirrhosis. The estimated annual incidence of HCC in noncirrhotic patients with chronic hepatitis B ranges from 0.3% to 0.5%.[51] Additional host, viral, liver-related, and environmental factors further increase the risk of HCC and influence the recommended age at which surveillance should begin. Accordingly, the CASL/AMMI guidelines recommend surveillance in high-risk patients with chronic hepatitis B, including those with cirrhosis, men aged 40 years or older, women aged 50 years or older, individuals of African ancestry aged 30 years or older, those with a first-degree family history of HCC, and patients with HIV or HDV co-infection. The recommended surveillance strategy is abdominal ultrasound with alpha-fetoprotein measurement every 6 months, which is cost-effective, noninvasive, free of radiation exposure, and more sensitive than imaging alone.[52]

Future directions in HBV management

Future directions in HBV management focus on achieving functional cure, defined as sustained loss of HBsAg with undetectable HBV DNA after a finite course of therapy. Several novel agents, including siRNA therapies and capsid assembly modulators, are currently in advanced clinical development. Emerging biomarkers, such as quantitative HBsAg, HBV RNA, and hepatitis B core-related antigen, hold promise for more precise disease monitoring and personalized treatment stratification, although only quantitative HBsAg is available in clinical practice. The recent CASL/AMMI guidelines advocate for broader HBV screening and expanded treatment indications, which should enable earlier diagnosis and treatment initiation. However, successful implementation will require public health studies to assess cost-effectiveness, alongside updates to provincial health coverage policies. Functional cure is more likely to be achieved with these potential new drugs, as early phase trials of siRNA-based regimens and related agents have demonstrated substantially higher rates of sustained HBsAg loss compared with current standard therapies. In the long term, the ultimate goal remains complete cure through eradication of covalently closed circular DNA and integrated viral DNA, although this remains unattainable with existing or foreseeable therapies. ■

Competing interests

Dr Ko has received honoraria and consulting fees from Gilead and GSK (formerly GlaxoSmithKline). The other authors declared no competing interests.

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