

Hepatitis B Virus Infection

Updated: May 27, 2026

Reviewed: May 27, 2026

Epidemiology

After acquiring hepatitis B virus (HBV) infection, many adults spontaneously recover and develop protective antibodies directed against hepatitis B surface antigen (anti-HBs). However, some progress to chronic hepatitis B, which is a leading cause of chronic liver disease worldwide.¹⁻⁶ Globally and in North America, approximately 8% of people with HIV have evidence of chronic HBV infection, but this varies by region of the world.⁷

Transmission routes vary geographically, with perinatal and early childhood exposures accounting for most HBV transmission in higher-prevalence regions.⁸ In low-prevalence regions—such as Europe and North America—a large proportion of transmission is through sexual contact and injection drug use, but perinatal transmission also occurs.⁹ Although the general modes of transmission are similar to those of HIV, HBV is transmitted more efficiently than HIV.^{4,6} People with HIV are at increased risk for developing chronic HBV infection.¹⁰⁻¹²

Hepatitis D virus (HDV) requires HBV for its propagation because it uses the HBV surface antigen (HBsAg) to form its envelope. Thus, prevalence of HDV mirrors that of chronic HBV infection with geographic variation. In a European study of 1,556 people with HIV and chronic HBV infection, 15.2% had antibodies to HDV, with the highest prevalence observed among people who inject drugs at 50.5%.¹³

Clinical Manifestation

HBV has an average incubation period of 90 days (range 60–150 days) from exposure to onset of symptoms.¹⁴ Acute HBV infection is asymptomatic in approximately 70% of cases, and <1% develop fulminant hepatic failure.^{1,15} When symptoms manifest, they may include right upper quadrant abdominal pain, nausea, vomiting, fever, and arthralgias, with or without jaundice. Most people with chronic HBV infection are asymptomatic or have nonspecific symptoms, such as fatigue. Between 15% and 40% of people with chronic HBV infection will eventually develop cirrhosis, hepatocellular carcinoma (HCC), or liver failure, and up to 25% of people will die prematurely from complications of chronic HBV infection.¹⁶

Diagnosis

Centers for Disease Control and Prevention (CDC) recommends universal hepatitis B screening at least once during a lifetime for all people over the age of 18, including those with HIV.¹⁷ Screening should include three serologic tests (triple panel screening): HBsAg, total hepatitis B core antibody (anti-HBc), and anti-HBs.¹⁷

In acute infection, HBsAg can be detected 4 weeks (range 1–9 weeks) after exposure, and anti-HBc immunoglobulin M (IgM) is usually detectable at the onset of symptoms. See CDC's [Clinical Testing and Diagnosis for Hepatitis B](#) for information on how to interpret laboratory results.

Chronic HBV infection is most commonly defined as persistent HBsAg or hepatitis B e antigen (HBeAg) detected on two occasions at least 6 months apart.¹ Other less common definitions can be found on [CDC’s Case Definitions webpage](#). People with chronic HBV infection should be tested further for HBV e antigen (HBeAg), antibody to HBeAg (anti-HBe), and HBV DNA. “Active” disease, which can be HBeAg negative or HBeAg positive, can be distinguished from “inactive” disease by the presence of both detectable serum HBV DNA and persistent or fluctuating alanine transaminase (ALT) elevations, defined as ≥ 2 times the upper limit of normal (≥ 33 U/L for males and ≥ 25 U/L for females).¹ Because people with chronic HBV infection are at risk for coinfection with HDV, anti-HDV should be checked after chronic HBV infection is diagnosed. If positive, then HDV RNA should be obtained to assess for chronic HDV infection.

People whose past infection has resolved are HBsAg negative with positive anti-HBs and anti-HBc.^{18,19} In a low-prevalence country—such as the United States—an isolated positive anti-HBc may represent a false-positive result; however, its presence can signify infection with HBV in the past, with subsequent loss of anti-HBs. Isolated anti-HBc occurs in 7% to 19% of people with HIV and can be higher in people with underlying hepatitis C virus (HCV) coinfection.^{20–26} Among people with HIV who have isolated anti-HBc, HBV viremia typically occurs in 1% to 10% of cases.^{20,21,27,28} Because most people with HIV who have isolated anti-HBc are HBV DNA-negative,²⁴ routine HBV DNA testing **is not recommended**. However, if a tenofovir-sparing HIV regimen is being considered, some experts recommend that HBV DNA be undetectable prior to the switch (**CIII**) because detectable HBV DNA increases the risk of HBV reactivation (see the [Treating Chronic Hepatitis B Virus Infection section](#) below).²⁹

Diagnosing HBV Disease Progression and the Role of Assessing Liver Fibrosis

Compared with people with HBV mono-infection, those with HIV/HBV coinfection have higher levels of HBV viremia and lower likelihood of resolved infection following acute HBV infection.³⁰ People with HIV/HBV coinfection are also more likely to have detectable HBeAg,^{30,31} lower rates of seroconversion to anti-HBe, and increased risk of HCC and liver-related morbidity and mortality.^{32–34}

Chronic HBV infection is a dynamic disease with a number of phases (see the [American Association for the Study of Liver Diseases/Infectious Diseases Society of America Practice Guideline on the Treatment of Chronic Hepatitis B](#)). In HIV/HBV coinfection, monitoring and treatment are focused on the simultaneous and immediate treatment of both viruses regardless of HBV phase.

People with HIV and chronic HBV should be linked to care and have a complete history and physical examination for signs of cirrhosis or HCC. HBV serologic and quantitative nucleic acid testing (HBeAg/anti-HBe and HBV DNA) and other laboratory testing—complete blood count, ALT, aspartate aminotransferase (AST), albumin, total bilirubin, alkaline phosphatase, international normalized ratio (INR), hepatitis A virus (HAV) immunoglobulin G (IgG) (to determine the need for vaccination), HCV IgG (if positive, then HCV RNA), antibodies to HDV IgG (if positive, then HDV RNA), abdominal ultrasound, and liver fibrosis assessments (transient elastography or serum markers, e.g. FibroSure®)—should be performed at the initial visit.¹ The decision to perform a liver biopsy should be individualized, but the procedure is rarely necessary.¹ People with chronic HBV infection are at increased risk of HCC; therefore, HCC surveillance every 6 months (ultrasound with or without alpha-fetoprotein) is required for people who have cirrhosis and for people in the following groups^{1,35} who are at increased risk of disease progression:

- Males older than age 40 who are from an HBV-endemic country

- Females older than age 50 who are from an HBV-endemic country
- Males older than age 20 who are from sub-Saharan Africa
- People with a first-degree family member with HCC
- People with active HDV coinfection
- People with the 5-year HCC risk score, [PAGE-B](#), score ≥ 10

In one study of 2,963 people with HIV/HBV coinfection (median age 41 years) on tenofovir-containing antiretroviral therapy (ART), the negative predictive value of a PAGE-B score <10 was 99.4% for HCC development over 5 years.³⁶ People with HIV/HBV coinfection are at increased risk of HCC,^{37,38} and some experts recommend ongoing semi-annual HCC surveillance (ultrasound with or without alpha-fetoprotein) for all people aged 40 years and older with HIV/HBV coinfection **(BIII)**.

Preventing Exposure

To reduce exposure to HBV in the general community context, avoid direct contact with blood or bodily fluids, wash hands thoroughly after any potential blood exposure, and avoid sharing personal use household or medical items, such as toothbrushes, razors, nail clippers, or glucometers. Existing recommendations to reduce sexual exposure to other sexually transmitted viral infections can be applied to reduce sexual exposure to HBV. For people who inject drugs, existing harm-reduction principles are recommended to reduce exposure to viruses in this context.

Preventing Hepatitis B and Other Liver Diseases

All family members and sexual contacts of people with chronic HBV infection should be tested, and all susceptible household and sexual contacts should receive hepatitis B (HepB) vaccine, regardless of HIV status **(AII)**.

All people with HIV without chronic HBV infection and without immunity to HBV (negative HBsAg, anti-HBc, and anti-HBs), as well as those who have failed a prior HepB vaccine series, should receive HepB vaccination **(AII)**. Vaccination **should not be deferred** because of low CD4 T lymphocyte (CD4) cell counts in people who are at increased risk of HBV infection.

Hepatitis A (HepA) vaccination is recommended for all people with HIV, including those who are pregnant, who are negative for HAV total antibodies (IgG plus IgM) **(AII)**.

Refer to the [Hepatitis B Vaccine section](#) and the [Hepatitis A Vaccine section](#) in the Immunizations for Preventable Diseases in Adults and Adolescents With HIV chapter for information on available vaccines and detailed HepB and HepA vaccination recommendations.

People with chronic HBV infection should be advised to avoid alcohol consumption since alcohol can accelerate liver disease from chronic HBV **(AII)**.

Treating Chronic Hepatitis B Virus Infection

Recommendations for Treating Chronic Hepatitis B Virus Infection
<p>Indication for Therapy</p> <ul style="list-style-type: none"> All people with HIV/HBV coinfection (HBsAg positive), regardless of CD4 count and HBV DNA level (AII), should be treated with a fully suppressive ART regimen that includes drugs active against both HIV and HBV infections (AII). <p>Note: TDF, TAF, entecavir, 3TC, and FTC should not be used alone in the absence of a fully HIV-suppressive ART regimen because of the potential for development of HIV drug-resistance mutations (AI).</p> <p>Preferred Therapy (CrCl ≥60 mL/min)</p> <ul style="list-style-type: none"> The ART regimen should include two drugs active against HBV: <ul style="list-style-type: none"> TAF (10 or 25 mg)^a plus FTC 200 mg (AII), or TAF 25 mg plus 3TC 300 mg PO once daily (AII), or TDF 300 mg plus (FTC 200 mg or 3TC 300 mg) once daily (AII) <p>Preferred Therapy (CrCl 30–59 mL/min)</p> <ul style="list-style-type: none"> The ART regimen should include two drugs active against HBV: <ul style="list-style-type: none"> TAF (10 or 25 mg)^a plus FTC 200 mg PO once daily (AII), or TAF 25 mg plus 3TC 300 mg^b PO once daily (AII) <p>Preferred Therapy (CrCl <30 mL/min, Not Receiving Hemodialysis)</p> <ul style="list-style-type: none"> A fully suppressive ART regimen with renally dose-adjusted entecavir^c (in place of TDF/[FTC or 3TC] or TAF/[FTC or 3TC]) (AIII), or When recovery of renal function is unlikely, ART with renally dose-adjusted TDF and renally dose-adjusted FTC or 3TC (AIII). If CrCl ≥15 to 29 mL/min, then ART with TAF (10 or 25 mg) once daily plus renally dose-adjusted FTC or 3TC is an option (AIII). <ul style="list-style-type: none"> Some clinicians may choose to continue full-dose FTC to allow for people with CrCl 15–29 mL/min to remain on fixed-dose TAF/FTC products. <p>Preferred Therapy (Receiving Hemodialysis)</p> <ul style="list-style-type: none"> ART with renally dose-adjusted TDF plus [FTC 200 mg or renally dose-adjusted 3TC once daily] (AII), or ART with TAF [10 or 25 mg]^a plus FTC 200 mg PO once daily (given after HD on dialysis days) (AII). TAF and FTC do not require renal dose adjustment in people receiving HD; therefore, fixed-dose TAF/FTC products may be continued. <p>Note: See Table 6 for dosing recommendation for TDF, TAF, FTC, and 3TC for people with renal impairment.</p> <p>Alternative Therapy</p> <ul style="list-style-type: none"> Entecavir 0.5 mg once daily may be used in place of (TAF or TDF) plus (3TC or FTC) (AIII). However, entecavir should not be used in individuals with 3TC-resistant HBV, as prior 3TC resistance reduces susceptibility to entecavir (AI). <p>Duration of Therapy/Monitoring During Therapy</p> <ul style="list-style-type: none"> People on treatment for HBV and HIV should receive therapy indefinitely if HBsAg remains positive (AIII).

- HBV DNA should be monitored at 3 and 6 months after treatment initiation and subsequently at 6-month intervals (AII).
- HBsAg should be monitored yearly (AIII).

Special Considerations When Initiating Tenofovir-Sparing ARV Regimens

- *In people without a history of chronic HBV infection:*
 - Within 3 months prior to initiating or switching to a tenofovir-sparing ARV regimen, HBsAg, anti-HBs, and anti-HBc should be checked to evaluate for unrecognized chronic HBV infection and absence of immunity (AIII), including in individuals with prior receipt of the HepB vaccine (AIII).
- *In people with chronic HBV infection (HBsAg positive):*
 - Anti-HBV therapy (TDF, TAF, or entecavir) must be given if switching to a tenofovir-sparing ARV regimen (e.g. DTG/RPV or long-acting CAB/RPV) (AII).
 - Switching to the one-pill regimen of DTG/3TC without additional anti-HBV therapy (TDF, TAF, or entecavir) should be avoided (AII). Chronic administration of 3TC alone is not recommended because of the high rate of selection of HBV drug-resistance mutations (AI).
 - HBsAg should be checked yearly after ART initiation (AIII). If HBsAg loss occurs and is maintained for at least 6 months and there is a desire to switch to a tenofovir-sparing regimen, this option may be considered.
- *In people with anti-HBc and anti-HBs positivity:*
 - Switching to a tenofovir-sparing ARV regimen without additional anti-HBV therapy is appropriate since there is a low risk of HBV reactivation (AIII). ALT monitoring every 3 months for the first 6 months, then every 6 months thereafter post-switch is recommended (BIII). An ALT increase would warrant HBV DNA testing.
- *In people with isolated anti-HBc positivity (HBsAg negative and anti-HBc positive and anti-HBs negative):*
 - Switching to DTG/3TC or a tenofovir-sparing ARV regimen without additional anti-HBV therapy can be considered, but there is a low risk of HBV reactivation. Therefore, ALT monitoring every 3 months for the first 6 months and then every 6 months thereafter post-switch is recommended (BIII). An ALT increase would warrant HBV DNA testing.
 - HepB vaccination initiated at the time of switch is recommended (see [Vaccination Schedule for People With Isolated Anti-HBc](#)) (AII).
 - Some experts recommend that HBV DNA be undetectable prior to switching to DTG/3TC or tenofovir-sparing ARV regimen (CIII).
- *In people nonreactive for anti-HBc and anti-HBs:*
 - Completing a [HepB vaccination](#) series and checking anti-HBs prior to switching to a tenofovir-sparing ARV regimen is recommended (AI).
- **Note:** If a tenofovir-sparing regimen is initiated, ongoing review of risk factors for reactivation is recommended, e.g., introduction of immunosuppressive therapies (see [Considerations When Using Tenofovir-Sparing Regimens](#)).

Other Considerations

- Because people with HBV/HCV/HIV coinfection appear to have accelerated liver fibrosis progression, higher risk of HCC, and increased mortality, treatment for both HBV and HCV infection should be initiated (AII).
- Because HBV reactivation can occur during treatment for HCV with direct-acting antivirals in the absence of anti-HBV therapy, all people with HIV/HBV coinfection who will be treated for HCV infection should be on HBV-active ART at the time of HCV treatment initiation (AIII).
- When changing ART regimens in people with HIV/HBV coinfection, it is crucial to continue agents with appropriate anti-HBV activity (TDF or TAF) or to initiate entecavir due to risk of HBV reactivation with hepatic flare after stopping anti-HBV treatment (AIII).

- If anti-HBV therapy must be discontinued (i.e., due to medication intolerance, contraindications, or patient preference), serum transaminase and HBV DNA levels should be monitored every 6 weeks for 3 months and then every 3 to 6 months thereafter (AIII).
- If a hepatic flare occurs after drug discontinuation, HBV therapy should be reinstituted because it can be potentially lifesaving (AIII).
- If immunosuppressive therapy is given, HBV reactivation can occur. Thus:
 - o People who are HBsAg positive should be administered treatment for HBV infection regardless of HBV DNA level (AII).
 - o For people who are HBsAg negative/anti-HBc positive and will receive low-risk immunosuppression ([as defined by the AGA](#)), monitoring HBV DNA and ALT at 1 to 3 month intervals is recommended if they are not on a TDF/TAF-based ART regimen (BIII).
 - o For people who are HBsAg negative/anti-HBc positive and will receive moderate- or high-risk immunosuppression ([as defined by the AGA](#)), TDF or TAF plus FTC or 3TC is recommended as part of the ART regimen prior to immunosuppression to prevent reactivation (BII).
 - o For people who are HBsAg negative/anti-HBc positive who cannot be given TDF or TAF and will receive moderate- to high-risk immunosuppression, prophylaxis with entecavir can be administered to prevent reactivation (BII) (see the [Special Considerations During Immunosuppressive Therapy section](#) below).
 - o Treatment to prevent reactivation should be continued for 6 months after immunosuppressive therapy is complete or for 12 months after anti-CD20 therapy is complete (BIII).
- For information regarding drug–drug interactions, such as with tenofovir products and certain antimycobacterial, anti-HCV, or anti-seizure therapies, see the [Liverpool HEP Drug Interaction Checker](#).

Pregnancy Considerations

- During pregnancy, TAF or TDF given in combination with 3TC or FTC is the preferred dual-NRTI backbone in the context of chronic HBV infection (AIII).
- Entecavir has not been well evaluated during pregnancy and is therefore **currently not recommended** in the setting of the HIV/HBV coinfection during pregnancy (AIII).
- Infants born to women who are HBsAg positive should receive HBIG and HepB vaccine within 12 hours of delivery (AI). The second and third doses of vaccine should be administered at 1–2 months and 6 months of age, respectively, for a total of three doses (AI). Infants <2,000 g should receive four doses of HepB vaccine: one dose administered within 12 hours of delivery and the three-dose HepB vaccine series initiated at age 1 month (four doses total: birth, 1 month, 2–3 months, and 6 months) (AII).

^a TAF 10 mg dose is in the fixed-dose combination tablets of elvitegravir/cobicistat/TAF/FTC and darunavir/cobicistat/TAF/FTC; when TAF is used with other antiretrovirals, the dose is 25 mg.

^b The prescribing information for 3TC (Epivir) recommends dosage adjustment from 300 mg once daily to 150 mg once daily for people with CrCl 30–49 mL/min. However, the prescribing information for several FDC products that contain 3TC (including ABC plus 3TC, DTG plus 3TC, and 3TC plus ABC) recommends no dose adjustment for CrCl 30–49 mL/min. The recommendation in this table incorporates the dosing guidance from the FDC products.

^c Entecavir **should not be used** in individuals with 3TC-resistant HBV because prior 3TC resistance reduces susceptibility to entecavir (AI).

Key: 3TC = lamivudine; ABC = abacavir; AGA = American Gastroenterological Society; anti-HBc = HBV core antibody; anti-HBs = HBV surface antibody; anti-HBV = hepatitis B virus antibody; ART = antiretroviral therapy; ARV = antiretroviral; CD4 = CD4 T lymphocyte; CrCl = creatinine clearance; CAB = cabotegravir; DTG = dolutegravir; FDC = fixed-dose combination; FTC = emtricitabine; HBsAg = HBV surface antigen; HBV = hepatitis B virus; HBIG = hepatitis B immune globulin G; HCC = hepatocellular carcinoma; HCV = hepatitis C virus; HD = hemodialysis; HepB = hepatitis B [vaccine]; NRTI = nucleoside reverse transcriptase inhibitor; PO = orally; RPV = rilpivirine; TAF = tenofovir alafenamide; TDF = tenofovir disoproxil fumarate

Preferred Regimens

The ultimate treatment goals in HIV/HBV coinfection are the same as for HBV mono-infection: to prevent disease progression and to reduce HBV-related morbidity and mortality. All people with HIV/HBV coinfection (HBsAg positive), regardless of CD4 count and HBV DNA level (**AII**), should be treated with an ART regimen that includes drugs active against both HIV and HBV infections (**AII**).

The [Adult and Adolescent Antiretroviral Guidelines](#) recommend the fixed-dose coformulations of tenofovir disoproxil fumarate (TDF)/emtricitabine or lamivudine (FTC or 3TC), tenofovir alafenamide (TAF)/FTC, abacavir/3TC, or 3TC alone (with dolutegravir) as nucleoside reverse transcriptase inhibitor (NRTI) regimen backbones for ART-naïve people regardless of CD4 count.³⁹ Because both components of the tenofovir combinations (tenofovir and either FTC or 3TC) have anti-HBV activity, they are also the treatment of choice for people with HIV/HBV coinfection (**AII**), regardless of CD4 count (**AI**) and HBV DNA level (**AII**) (see [Hepatitis B Virus/HIV Coinfection](#) in the Adult and Adolescent Antiretroviral Guidelines). TDF and TAF are both active against wild-type and 3TC-resistant HBV strains. Studies among people with HIV/HBV coinfection (most of them carrying 3TC-resistant HBV) receiving tenofovir have shown, on average, 4 log₁₀ declines in HBV DNA levels.⁴⁰⁻⁴⁵ TDF and TAF have a high genetic barrier for development of resistance mutations.^{1,46}

There are limited data suggesting that TAF may be superior to TDF in people with HIV/HBV coinfection. In a randomized, placebo-controlled study of people with HIV/HBV coinfection (predominantly Asian participants), TAF/FTC/bictegravir (BIC) was superior to TDF/FTC/dolutegravir (DTG) in achieving HBV DNA <29 IU/mL at 48 weeks (63% vs. 43%, respectively; $P = 0.002$), but efficacy was similar at 96 weeks (75% vs. 70%, respectively; $P = 0.64$).⁴⁷ At 96 weeks, participants receiving TAF/FTC/BIC had higher rates of HBeAg seroconversion (32% vs. 15%; $P = 0.008$), while rates of HBsAg loss did not differ significantly between groups (23% vs. 14%; $P = 0.07$). Although the data are intriguing, it remains undetermined whether these responses are related to ethnicity, HBV genotype, or other HIV-related factors.

The decision to use TAF/FTC versus TDF/FTC should be based upon creatinine clearance (CrCl) and an assessment of risk for nephrotoxicity and accelerated bone loss.

- Among people with CrCl ≥ 60 mL/min, TAF/FTC or TAF plus 3TC or TDF/(FTC or 3TC) can be considered (**AII**).
- Among people with a CrCl 30 to 59 mL/min, either TAF/FTC or TAF plus 3TC is preferred (**AII**).
- In people with CrCl <30 mL/min who are not on hemodialysis, renally dosed entecavir with a fully suppressive ART regimen is recommended because entecavir without suppressive ART can lead to emergence of the HIV M184V mutation (**AIII**).⁴⁸ Renally dosed TDF and FTC or 3TC also can be used if recovery of renal function is unlikely (**AIII**). If renally dosed TDF is used, then the CrCl needs to be monitored carefully. In people with CrCl ≥ 15 to 29 mL/min, ART with TAF (as a single agent) and renally dosed FTC or 3TC may be used (**AIII**). Of note, some clinicians may choose to continue full-dose FTC to allow for people with CrCl 15 to 29 mL/min to remain on fixed-dose TAF/FTC products.
- In people receiving hemodialysis, ART with either renally dosed TDF plus (FTC or renally dosed 3TC) (**AII**) or TAF/FTC (**AII**) may be used. TAF and FTC do not require dose adjustment in

patients receiving hemodialysis; coformulated full-dose products may be continued and given after dialysis on the day of hemodialysis. Refer to [What to Start: Nucleoside Reverse Transcriptase Inhibitor Options as Part of Initial Therapy](#) and [Appendix B, Table 12. Antiretroviral Dosing Recommendations in Adults With Renal or Hepatic Insufficiency](#) in the Adult and Adolescent Antiretroviral Guidelines for more information.

Chronic administration of 3TC or FTC as the only active drug against HBV **is not recommended** because of the high rate of selection of HBV drug-resistance mutations (**AI**).

People with HIV/HBV coinfection who are receiving ART should continue HBV therapy indefinitely if they remain HBsAg positive (**AIII**) because relapses after response can occur, particularly in those with lower CD4 counts.¹ Additionally, discontinuation of nucleos(t)ide analog therapy is associated with an HBV reactivation in approximately 30% of cases,^{49,50} as well as possible decompensation of liver disease and even death.⁵¹⁻⁵⁴ If anti-HBV therapy and ART must be discontinued for people with chronic HBV, serum transaminase levels and HBV DNA should be monitored every 6 weeks for 3 months and every 3 months thereafter while off anti-HBV agents (**AIII**). If a hepatic flare occurs, anti-HBV therapy and ART should be reinstituted and can be potentially lifesaving (**AIII**).

Some people with HIV/HBV coinfection also have chronic HCV infection. Scant information is available on the treatment of HBV/HCV/HIV coinfection. Because people with HBV/HCV/HIV coinfection appear to have accelerated progression of liver fibrosis, higher risk of HCC, and increased mortality,⁵⁵⁻⁵⁷ treatment for both HBV and HCV should be initiated (**AII**). Because HBV reactivation can occur during treatment for HCV infection with direct-acting antivirals in the absence of anti-HBV therapy, all people with HIV/HBV coinfection who will be treated for HCV infection should be on HBV-active ART at the time of HCV treatment initiation (**AIII**).⁵⁸⁻⁶¹ See the [Hepatitis C Virus](#) chapter for information on HCV treatment.

For information regarding drug–drug interactions, such as with tenofovir products and certain antimycobacterial, anti-HCV, or anti-seizure therapies, see the [Liverpool HEP Drug Interaction Checker](#).

Considerations When Using Tenofovir-Sparing Regimens

With increasing use of tenofovir-sparing regimens, HBV status must be considered before such a switch. The Panel on Guidelines for the Prevention and Treatment of Opportunistic Infections in Adults and Adolescents With HIV (the Panel) recommends checking HBsAg, anti-HBc, and anti-HBs within 3 months of changing to a tenofovir-sparing regimen to avoid reactivation of an unrecognized chronic HBV infection (**AIII**). In people who are not immune (anti-HBc and anti-HBs negative), completing a vaccination series and checking anti-HBs prior to starting a tenofovir-sparing regimen is recommended (**AI**). In people with prior receipt of the HepB vaccine, anti-HBs should be rechecked prior to switching to a tenofovir-sparing regimen because incident infections have been reported in such situations (**AIII**).⁶²

In people with chronic HBV infection, switching to the one-pill regimen of DTG/3TC should be avoided because 3TC is then the only active drug against HBV (**AII**). Further, switching to a tenofovir-sparing regimen such as long-acting cabotegravir/rilpivirine (RPV) or DTG/RPV without addition of an anti-HBV drug (TAF, TDF, or entecavir) should be avoided (**AIII**).

In people with recovery from a past HBV infection (anti-HBc positive, anti-HBs positive), DTG/3TC or a tenofovir-sparing regimen without additional anti-HBV agents is appropriate because the risk of HBV reactivation is low **(AIII)**.⁶³ ALT monitoring every 3 months for the first 6 months and then every 6 months thereafter post-switch is recommended **(BIII)**. If ALT elevations occur, then checking HBV DNA is recommended.

Although the risk of HBV reactivation is low in people with isolated anti-HBc (HBsAg negative, anti-HBc positive, anti-HBs negative), the risk is higher than in those with past HBV infection who are anti-HBs positive. Thus, in people with isolated core positivity, switching to DTG/3TC or a tenofovir-sparing regimen can be considered with ALT monitoring every 3 months for the first 6 months, then every 6 months thereafter post-switch **(BIII)**.⁶⁴ If ALT increases, then checking HBV DNA is recommended. Some experts recommend undetectable HBV DNA prior to such a switch **(CIII)**. Vaccination initiated at the time of such a switch is recommended **(AII)** (see [Vaccination Schedule for People With Isolated Anti-HBc](#)). In a study using data from the Veterans Aging Cohort Study, HBV reactivation occurred in 1.6% of individuals who were anti-HBc positive and HBsAg negative after switching to a nucleos(t)ide-sparing regimen.⁶⁴

Alternative Treatment of HBV Infection Among People With HIV Who Are Not Receiving HBV-Active Antiretroviral Therapy

All people with HIV should receive ART. Among people with HIV/HBV coinfection, cotreatment is essential and recommended.³⁹ Pegylated interferon (IFN)- α -2a (or 2b) monotherapy is approved for HBV treatment, but it should only be used in rare cases with consultation of an expert.

Regimens That Are Not Recommended

TDF, TAF, entecavir, 3TC, and FTC **should not be used alone** in the absence of a fully HIV-suppressive ART regimen because of the potential for development of HIV drug-resistance mutations **(AI)**.^{65,66}

Monitoring of Response to Therapy and Adverse Events

To evaluate response, HBV DNA should be monitored at 3 and 6 months after treatment initiation and then at 6-month intervals **(AII)**. Treatment responses are slower than responses to HIV therapy and are defined as the following for nucleos(t)ide analog therapy:

- Virologic response: undetectable HBV DNA (<10 IU/mL) by real-time polymerase chain reaction⁶⁷
- Partial virologic response: HBV DNA ≥ 1 log₁₀ IU/mL decline, but still detectable HBV DNA at 12 months⁶⁷
- Primary nonresponse: HBV DNA <1 log₁₀ IU/mL decline after 3 months⁶⁸

See the [Managing Treatment Failure section](#) below for information on managing partial virologic response.

For people who are HBeAg positive, loss of HBeAg is also a measure of virologic response. Other markers that indicate treatment success include improvement in liver histology based on biopsy, transient elastography, or other noninvasive tests; normalization of serum aminotransferases; and, in

those with loss of HBeAg, the development of anti-HBe. Sustained loss of HBsAg is considered by some to be a functional cure; however, this desirable serologic response is uncommon (<1% of HBsAg-positive people without HIV per year).¹ Interestingly, some studies indicate that after ART initiation, people with HIV/HBV coinfection have up to 20% chance of HBsAg loss, most commonly within the first 1 to 2 years of treatment.^{69,70} For this reason, HBsAg should be checked yearly after ART initiation in these individuals **(AIII)**. If HBsAg loss occurs and is maintained for at least 6 months and there is a desire to switch to a tenofovir-sparing regimen, this option may be considered as discussed above.

Adverse Events

Renal toxicity with TDF, including increased serum creatinine or renal tubular dysfunction, has been observed; both increased serum creatinine and renal tubular dysfunction are more frequent among people with HIV who have underlying renal insufficiency, are older, or have been treated with TDF for prolonged periods.⁷¹ These biochemical changes are usually reversible when TDF is discontinued or changed to TAF.⁷²

Electrolytes and serum creatinine levels should be evaluated at baseline and every 6 months. Because renal toxicity may be reversible, alternative anti-HBV therapy should be used if renal toxicity occurs **(AI)**. If TDF is used among people with baseline renal insufficiency, either a dose adjustment as noted in the package insert is required or a change to TAF if appropriate based on degree of renal insufficiency.⁷² See [Table 6. Dosing Recommendations in People With Renal Insufficiency](#) for more information.

TDF has been associated with a decrease in bone mineral density (BMD). TAF is associated with less of a decrease in BMD than TDF in studies of hepatitis B treatment.⁷³ TAF also has been associated with early weight gain among people with HIV, although the long-term consequences of this are unclear.⁷⁴

See [Considerations for Antiretroviral Use in Patients With Coinfection: Hepatitis B Virus/HIV Coinfection](#) in the Adult and Adolescent Antiretroviral Guidelines for more information on adverse events related to TAF and TDF.

Entecavir-associated lactic acidosis is uncommon but has been reported among people with HBV mono-infection with advanced cirrhosis.⁷⁵⁻⁷⁷

Immune Reconstitution Inflammatory Syndrome

Return of immune competence after ART (or after steroid withdrawal or chemotherapy) can lead to reactivation of HBV-associated liver disease. Any immune reconstitution can lead to a rise in serum aminotransferases, so-called “hepatitis flare,”⁷⁸ which constitutes immune reconstitution inflammatory syndrome (IRIS) among people with HIV/HBV coinfection. IRIS can manifest when serum aminotransferase levels dramatically increase as CD4 counts rise within the first 6 to 12 weeks after ART is started, with signs and symptoms characteristic of acute hepatitis and without another cause for the flare.^{79,80} Flares are more consequential among people with more severe liver disease, especially those with cirrhosis,⁸¹ and it can be difficult to distinguish between IRIS and other causes of hepatitis flare, such as drug-induced liver injury, HBV drug resistance, HBeAg seroconversion, or acute hepatitis caused by infection with HAV, HCV, HDV, hepatitis E virus, Epstein-Barr virus, herpes simplex virus, or cytomegalovirus.

After introduction of ART, serum ALT levels should be monitored closely; some experts recommend ALT testing at 6 and 12 weeks and then every 3 to 6 months thereafter. Any association between abnormal aminotransferases and clinical jaundice or synthetic dysfunction (elevated INR and low serum albumin) should prompt consultation with a hepatologist **(CI)**.⁷²

Most (90%) people with HIV/HBV coinfection do not experience ART-associated hepatotoxicity,⁸² and clinically significant hepatotoxicity (elevated direct bilirubin and INR) is rare. ART-associated hepatotoxicity may be dose dependent or idiosyncratic. Among people with HIV, the risk of ART-associated hepatotoxicity has been associated consistently with elevated pre-ART aminotransferases (ALT, AST) and the presence of HBV or HCV coinfection. In HIV/HBV coinfection, baseline elevated HBV DNA levels are predictive of hepatotoxicity.⁸³⁻⁸⁶ Nonetheless, aminotransferase levels return to baseline in most cases, even if the ART is continued.^{87,88} Therefore, discontinuing ART usually is not necessary in the presence of hepatotoxicity unless the following symptoms are observed: hypersensitivity (e.g., fever, lymphadenopathy, rash), symptomatic hepatitis (i.e., nausea, vomiting, abdominal pain, or jaundice), or elevations in serum aminotransferase levels >10 times the upper limit of normal **(AIII)**.⁸⁹ Liver histology also may help to differentiate drug toxicity (e.g., increased eosinophils) from viral hepatitis (e.g., portal inflammation). If aminotransferases increase >2 times the baseline level, the Panel recommends monitoring aminotransferases weekly and also obtaining bilirubin and INR until the aminotransferases begin declining **(AIII)**.

Managing Treatment Failure

HBV treatment failure on nucleos(t)ide analogs is defined as primary nonresponse (HBV DNA <1 log₁₀ decline) after 3 months of therapy among people who consistently adhere to HBV therapy or an increase in HBV DNA levels >1 log₁₀ above nadir. In either situation, treatment failure generally is due to either drug-resistant HBV if the person is on 3TC/FTC monotherapy or to nonadherence to therapy.¹ If drug-resistant HBV is present, a change in treatment is needed **(AII)**. Many experts will obtain HBV-resistance testing because it has value in distinguishing between nonadherence and drug resistance, evaluating people with unclear prior drug history, and predicting the level of resistance to entecavir.⁹⁰ However, TDF/TAF is associated infrequently with clinical resistance, although slow response has been noted, as discussed above. Addition of entecavir has led to suppression of HBV DNA among people whose response to TDF/TAF is slow and adherence is confirmed.⁹¹ (see [Monitoring of Response to Therapy and Adverse Events](#))

With 3TC monotherapy for HBV, the rate of developing 3TC-resistance is approximately 20% per year among people with HIV/HBV coinfection, hence it **should not be used** as monotherapy for HBV.⁹² If 3TC resistance is suspected or documented, TDF or TAF should be added to the ART regimen **(AIII)**.⁹³⁻⁹⁵ Because people with 3TC-resistant HBV will have cross-resistance to the other L-nucleosides (FTC), and partial resistance to entecavir, those agents **should not be used** among people found to have 3TC-resistant HBV **(AI)**.⁹⁶ If consideration is given to entecavir use in this situation, consultation with an expert in the management of drug-resistant HBV is recommended. All nucleoside analogs must be dose-adjusted for renal insufficiency per package insert guidelines and [Table 6. Dosing Recommendations in People With Renal Insufficiency](#).

If treatment failure occurs on entecavir, then TDF or TAF (with or without FTC) is recommended because of the cross-resistance that occurs with L-nucleosides (3TC, FTC) **(AI)**.

People whose HBV infection initially failed to respond to pegylated IFN- α can be given nucleos(t)ide analog therapy following the recommendations previously described **(CIII)**.

Declines in HBV DNA levels can be slow, especially when pretherapy HBV DNA levels are very high. HBV DNA levels usually drop quickly among people who are receiving an HBV drug with high potency and a high genetic barrier to resistance—such as tenofovir—but HBV DNA levels may still be detectable for some years.¹ Thus, in a person who is adherent to therapy with a partial virologic response to tenofovir, the drug should be continued with monitoring of HBV DNA levels **(BII)**. Improved virologic response has been reported with the addition of entecavir to TDF; however, whether such “intensification therapy” is required is unclear.

Special Considerations for Treating Cirrhosis and End-Stage Liver Disease

People with HIV/HBV coinfection who have cirrhosis or end-stage liver disease should be managed the same as people with HBV mono-infection with cirrhosis or end-stage liver disease, including referral to a hepatologist **(AIII)** (see the [American Association for the Study of Liver Diseases \(AASLD\) 2018 Hepatitis B Guidance](#)). Among people with HIV/HBV coinfection with end-stage liver disease, IFN- α is **contraindicated (AI)**, but nucleos(t)ide analogs are safe and efficacious **(AI)**.^{92,97,98} In addition, treatment cessation is **contraindicated**.

People with HIV/HBV coinfection with decompensated liver disease and/or early HCC are candidates for liver transplantation. HIV infection is not a contraindication to organ transplantation among people on suppressive ART.⁹⁹ Because transplantation does not cure HBV infection, post-transplant hepatitis B immune globulin (HBIG) and HBV treatment are required **(AII)**. People with HIV who are potential candidates for liver transplantation should be referred to a liver specialist for further evaluation.

Special Considerations During Immunosuppressive Therapy

With immunosuppressive therapy, both in the context of malignancy and rheumatologic/autoimmune diseases, reactivation of HBV infection can occur. HBV reactivation in people without HIV with HBsAg-positive/anti-HBc-positive disease receiving immunomodulatory therapy is well described, especially with anti-CD20 antibodies.¹⁰⁰⁻¹⁰² Even among people with HBsAg-negative/anti-HBc-positive disease, HBV reactivation occurs in up to 18% of people receiving anti-cancer drugs¹⁰³ and 1.7% of people receiving rheumatologic disease drugs.¹⁰⁴

People with HIV undergoing immunosuppressive therapy should have the triple panel screening performed (HBsAg, anti-HBc, and anti-HBs) before initiating therapy unless they are known to have chronic HBV. People who are HBsAg positive regardless of HBV DNA level should receive treatment with TDF or TAF plus 3TC or FTC-based ART regimen (see [Preferred Regimens](#) above) **(AII)**. For people with HBsAg-negative/anti-HBc-positive disease, the risk of HBV reactivation during immunosuppressive therapy is divided into low (<1%), moderate (1% to 10%), and high (>10%) risk according to the American Gastroenterological Society (AGA).¹⁰⁵ With moderate or high-risk immunosuppression, TDF or TAF plus FTC or 3TC are recommended before initiating immunosuppressive therapy **(BII)**. If TDF or TAF plus FTC or 3TC cannot be used as part of their HIV regimen, these people could receive entecavir for anti-HBV prophylaxis **(BII)**. For people whose ART regimen does not include TDF or TAF plus FTC or 3TC and who will receive low-risk immunosuppression, monitoring with HBV DNA and ALT every 1 to 3 months is recommended **(BIII)**. No studies have been performed on the appropriate length of therapy, but the Panel agrees with the [AGA guidance recommendation](#) to continue treatment for a minimum of 6 months after cessation of immunosuppressive therapy or of 12 months in the setting of anti-CD20 antibodies **(BIII)**.

Special Considerations During Pregnancy

Screening for HBV with the triple panel is recommended during pregnancy **(AIII)**, followed by linkage to care or vaccination as needed.¹⁰⁶ HBsAg should be screened during each pregnancy, preferably in the first trimester, even if vaccinated or tested previously.^{17,106} If a history of appropriately timed triple panel screening is elicited without subsequent risk for exposure to HBV (i.e., no new HBV exposures since triple panel screening), then only HBsAg screening is needed. Such testing enables documentation of the HBsAg test result during pregnancies with maternal chronic infection or maternal immunity to ensure timely prophylaxis for exposed infants (see below).

Those who are both HBsAg negative and anti-HBs negative should be offered vaccination against HBV **(AII)**. In the context of chronic HBV infection during pregnancy, if the HepA vaccine series has not been previously administered, then screen for immunity to HAV infection. Those who screen negative for total anti-HAV should receive the HepA vaccine series **(AIII)**.¹⁰⁷ Refer to the [Hepatitis B Vaccine section](#) and the [Hepatitis A Vaccine section](#) in the Immunizations for Preventable Diseases in Adults and Adolescents With HIV chapter for information on available vaccines.

Treatment of symptomatic acute HBV infection during pregnancy is supportive, with special attention given to maintaining blood glucose levels and normal clotting status. High maternal HBV DNA levels correlate strongly with perinatal HBV transmission, including failures of HBV passive-active immunoprophylaxis.¹⁰⁸⁻¹¹¹ See [Hepatitis B Virus/HIV Coinfection](#) in the Perinatal Guidelines for additional information.

As for all people with HIV/HBV coinfection, ART that includes drugs active against both HIV and HBV should be used during pregnancy **(AII)**. TAF or TDF given in combination with 3TC or FTC is the preferred dual-NRTI backbone **(AIII)**.¹⁰⁷ Entecavir has not been well evaluated in pregnancy, with too few exposures to assess overall risk; thus, it is **currently not recommended** in the setting of HIV/HBV coinfection during pregnancy **(AIII)**.¹⁰⁷

Cases of adverse events during pregnancy related to any of the antiretroviral or anti-HBV drugs listed should be reported to the [Antiretroviral Pregnancy Registry](#) (1-800-258-4263 or SM_APR@APRegistry.com). TDF, TAF, FTC, and 3TC use during pregnancy has been well tolerated and are recommended for use in pregnancy **(AII)**.¹¹²

Infants born to women who are HBsAg positive should receive HBIG and the first dose of HepB vaccine within 12 hours of delivery **(AI)**. The second and third doses of vaccine should be administered at 1 to 2 months and 6 months of age, respectively **(AI)**. Infants who weigh <2,000 g at birth should receive HBIG and four doses of HepB vaccine; administer one dose of HepB vaccine within 12 hours of delivery, and initiate the three-dose HepB vaccine series beginning at age 1 month (four doses total: birth, 1 month, 2–3 months, and 6 months) **(AII)**.

References

1. Terrault NA, Lok ASF, McMahon BJ, et al. Update on prevention, diagnosis, and treatment of chronic hepatitis B: AASLD 2018 hepatitis B guidance. *Hepatology*. 2018;67(4):1560-1599. Available at: <https://pubmed.ncbi.nlm.nih.gov/29405329>.
2. Schweitzer A, Horn J, Mikolajczyk RT, Krause G, Ott JJ. Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013. *Lancet*. 2015;386(10003):1546-1555. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/26231459>.
3. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet*. 2020;396(10258):1204-1222. Available at: <https://www.sciencedirect.com/science/article/pii/S0140673620309259?via%3Dihub>.
4. Jeng WJ, Papatheodoridis G, Lok ASF. Hepatitis B. *Lancet*. 2023;401(10381):1039-1052. Available at: <https://www.sciencedirect.com/science/article/pii/S0140673622014684?via%3Dihub>.
5. Huang DQ, Terrault NA, Tacke F, et al. Global epidemiology of cirrhosis - aetiology, trends and predictions. *Nat Rev Gastroenterol Hepatol*. 2023;20(6):388-398. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/36977794>.
6. Devarbhavi H, Asrani SK, Arab JP, Nartey YA, Pose E, Kamath PS. Global burden of liver disease: 2023 update. *J Hepatol*. 2023;79(2):516-537. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/36990226>.
7. Leumi S, Bigna JJ, Amougou MA, Ngouo A, Nyaga UF, Noubiap JJ. Global burden of hepatitis B infection in people living with human immunodeficiency virus: a systematic review and meta-analysis. *Clin Infect Dis*. 2020;71(11):2799-2806. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/31813969>.
8. Gomes C, Wong RJ, Gish RG. Global perspective on hepatitis B virus infections in the era of effective vaccines. *Clin Liver Dis*. 2019;23(3):383-399. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/31266615>.
9. Ghany MG, Perrillo R, Li R, et al. Characteristics of adults in the hepatitis B research network in North America reflect their country of origin and hepatitis B virus genotype. *Clin Gastroenterol Hepatol*. 2015;13(1):183-192. Available at: <https://pubmed.ncbi.nlm.nih.gov/25010003>.
10. Hyams KC. Risks of chronicity following acute hepatitis B virus infection: a review. *Clin Infect Dis*. 1995;20(4):992-1000. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/7795104>.
11. Liu CJ, Kao JH. Global perspective on the natural history of chronic hepatitis B: role of hepatitis B virus genotypes A to J. *Semin Liver Dis*. 2013;33(2):97-102. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/23749665>.

12. Kafeero HM, Ndagire D, Ocamo P, et al. Mapping hepatitis B virus genotypes on the African continent from 1997 to 2021: a systematic review with meta-analysis. *Sci Rep*. 2023;13(1):5723. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/37029173>.
13. Béguelin C, Atkinson A, Boyd A, et al. Hepatitis delta infection among persons living with HIV in Europe. *Liver Int*. 2023;43(4):819-828. Available at: <https://pubmed.ncbi.nlm.nih.gov/36625770>.
14. Centers for Disease Control and Prevention. Clinical overview of hepatitis B. 2024. Available at: <https://www.cdc.gov/hepatitis-b/hcp/clinical-overview/index.html>.
15. Wright TL, Mamish D, Combs C, et al. Hepatitis B virus and apparent fulminant non-A, non-B hepatitis. *Lancet*. 1992;339(8799):952-955. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/1348798>.
16. McMahon BJ. The natural history of chronic hepatitis B virus infection. *Hepatology*. 2009;49(5 Suppl):S45-55. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/19399792>.
17. Connors EE, Panagiotakopoulos L, Hofmeister MG, et al. Screening and testing for hepatitis B virus infection: CDC recommendations - United States, 2023. *MMWR Recomm Rep*. 2023;72(1):1-25. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/36893044>.
18. Terrault NA, Lok ASF, McMahon BJ, et al. Update on prevention, diagnosis, and treatment of chronic hepatitis B: AASLD 2018 hepatitis B guidance. *Hepatology*. 2018;67(4):1560-1599. Available at: <https://pubmed.ncbi.nlm.nih.gov/29405329>.
19. Block TM, Locarnini S, McMahon BJ, Rehermann B, Peters MG. Use of current and new endpoints in the evaluation of experimental hepatitis B therapeutics. *Clin Infect Dis*. 2017;64(9):1283-1288. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/28200098>.
20. Palacios R, Mata R, Hidalgo A, et al. Very low prevalence and no clinical significance of occult hepatitis B in a cohort of HIV-infected patients with isolated anti-HBc seropositivity: the BHOI study. *HIV Clin Trials*. 2008;9(5):337-340. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/18977722>.
21. Shire NJ, Rouster SD, Rajcic N, Sherman KE. Occult hepatitis B in HIV-infected patients. *J Acquir Immune Defic Syndr*. 2004;36(3):869-875. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/15213572>.
22. Witt MD, Seaberg EC, Darilay A, et al. Incident hepatitis C virus infection in men who have sex with men: a prospective cohort analysis, 1984-2011. *Clin Infect Dis*. 2013;57(1):77-84. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/23532480>.
23. Bhattacharya D, Tseng CH, Tate JP, et al. Isolated hepatitis B core antibody is associated with advanced hepatic fibrosis in HIV/HCV infection but not in HIV infection alone. *J Acquir Immune Defic Syndr*. 2016;72(1):e14-17. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/26829660>.
24. Gandhi RT, Wurcel A, McGovern B, et al. Low prevalence of ongoing hepatitis B viremia in HIV-positive individuals with isolated antibody to hepatitis B core antigen. *J Acquir Immune Defic Syndr*. 2003;34(4):439-441. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/14615664>.

25. Gandhi RT, Wurcel A, Lee H, et al. Response to hepatitis B vaccine in HIV-1-positive subjects who test positive for isolated antibody to hepatitis B core antigen: implications for hepatitis B vaccine strategies. *J Infect Dis.* 2005;191(9):1435-1441. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/15809901>.
26. Spradling PR, Xing J, Harris AM, Ly KN. Estimated prevalence and number of persons with isolated antibody to hepatitis B core antigen and associated occult hepatitis B, United States, 2001-2018. *J Infect Dis.* 2022;225(3):465-469. Available at: <https://pubmed.ncbi.nlm.nih.gov/34252183>.
27. French AL, Operskalski E, Peters M, et al. Isolated hepatitis B core antibody is associated with HIV and ongoing but not resolved hepatitis C virus infection in a cohort of US women. *J Infect Dis.* 2007;195(10):1437-1442. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/17436223>.
28. Neau D, Winnock M, Jouvencel AC, et al. Occult hepatitis B virus infection in HIV-infected patients with isolated antibodies to hepatitis B core antigen: Aquitaine cohort, 2002-2003. *Clin Infect Dis.* 2005;40(5):750-753. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/15714424>.
29. Dieterich DT, Brunet L, Hsu RK, et al. Monitoring and risk of hepatitis B reactivation and hepatitis flare during tenofovir interruption among people with HIV and hepatitis B. *AIDS.* 2026;40(1):43-51. Available at: <https://pubmed.ncbi.nlm.nih.gov/40971446>.
30. Colin JF, Cazals-Hatem D, Lioriot MA, et al. Influence of human immunodeficiency virus infection on chronic hepatitis B in homosexual men. *Hepatology.* 1999;29(4):1306-1310. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/10094979>.
31. Gilson RJ, Hawkins AE, Beecham MR, et al. Interactions between HIV and hepatitis B virus in homosexual men: effects on the natural history of infection. *AIDS.* 1997;11(5):597-606. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/9108941>.
32. Thio CL, Seaberg EC, Skolasky R, Jr., et al. HIV-1, hepatitis B virus, and risk of liver-related mortality in the Multicenter Cohort Study (MACS). *Lancet.* 2002;360(9349):1921-1926. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/12493258>.
33. Brau N, Fox RK, Xiao P, et al. Presentation and outcome of hepatocellular carcinoma in HIV-infected patients: a U.S.-Canadian multicenter study. *J Hepatol.* 2007;47(4):527-537. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/17692986>.
34. Rajbhandari R, Jun T, Khalili H, Chung RT, Ananthakrishnan AN. HBV/HIV coinfection is associated with poorer outcomes in hospitalized patients with HBV or HIV. *J Viral Hepat.* 2016;23(10):820-829. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/27291562>.
35. Singal AG, Llovet JM, Yarrow M, et al. AASLD Practice Guidance on prevention, diagnosis, and treatment of hepatocellular carcinoma. *Hepatology.* 2023;78(6):1922-1965. Available at: <https://pubmed.ncbi.nlm.nih.gov/37199193>.
36. Surial B, Ramírez Mena A, Roumet M, et al. External validation of the PAGE-B score for HCC risk prediction in people living with HIV/HBV coinfection. *J Hepatol.* 2023;78(5):947-957. Available at: <https://doi.org/10.1016/j.jhep.2022.12.029>.

37. Wandeler G, Maunon E, Atkinson A, et al. Incidence of hepatocellular carcinoma in HIV/HBV-coinfected patients on tenofovir therapy: relevance for screening strategies. *J Hepatol*. 2019;71(2):274-280. Available at: <https://www.sciencedirect.com/science/article/pii/S0168827819302260?via%3Dihub>.
38. Kim HN, Newcomb CW, Carbonari DM, et al. Risk of HCC with hepatitis B viremia among HIV/HBV-coinfected persons in North America. *Hepatology*. 2021;74(3):1190-1202. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/33780007>.
39. Panel on Antiretroviral Guidelines for Adults and Adolescents. Guidelines for the use of antiretroviral agents in adults and adolescents with HIV. 2025. Available at: <https://clinicalinfo.hiv.gov/en/guidelines/adult-and-adolescent-arv/whats-new-guidelines>.
40. van Bommel F, Wunsche T, Schurmann D, Berg T. Tenofovir treatment in patients with lamivudine-resistant hepatitis B mutants strongly affects viral replication. *Hepatology*. 2002;36(2):507-508. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/12143063>.
41. Nunez M, Perez-Olmeda M, Diaz B, Rios P, Gonzalez-Lahoz J, Soriano V. Activity of tenofovir on hepatitis B virus replication in HIV-co-infected patients failing or partially responding to lamivudine. *AIDS*. 2002;16(17):2352-2354. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/12441815>.
42. Ristig MB, Crippin J, Aberg JA, et al. Tenofovir disoproxil fumarate therapy for chronic hepatitis B in human immunodeficiency virus/hepatitis B virus-coinfected individuals for whom interferon-alpha and lamivudine therapy have failed. *J Infect Dis*. 2002;186(12):1844-1847. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/12447773>.
43. Nelson M, Portsmouth S, Stebbing J, et al. An open-label study of tenofovir in HIV-1 and hepatitis B virus co-infected individuals. *AIDS*. 2003;17(1):F7-10. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/12478090>.
44. Benhamou Y, Tubiana R, Thibault V. Tenofovir disoproxil fumarate in patients with HIV and lamivudine-resistant hepatitis B virus. *N Engl J Med*. 2003;348(2):177-178. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/12519935>.
45. Peters MG, Andersen J, Lynch P, et al. Randomized controlled study of tenofovir and adefovir in chronic hepatitis B virus and HIV infection: ACTG A5127. *Hepatology*. 2006;44(5):1110-1116. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/17058225>.
46. Huhn GD, Tebas P, Gallant J, et al. A randomized, open-label trial to evaluate switching to elvitegravir/cobicistat/emtricitabine/tenofovir alafenamide plus darunavir in treatment-experienced HIV-1-infected adults. *J Acquir Immune Defic Syndr*. 2017;74(2):193-200. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/27753684>.
47. Avihingsanon A, Lu HZ, Leong CL, et al. Bictegravir, emtricitabine, and tenofovir alafenamide versus dolutegravir, emtricitabine, and tenofovir disoproxil fumarate for initial treatment of HIV-1 and hepatitis B coinfection (ALLIANCE): a double-blind, multicentre, randomised controlled, phase 3 non-inferiority trial. *Lancet HIV*. 2023;10(10):E640-E652. Available at: <https://pubmed.ncbi.nlm.nih.gov/37494942>.

48. McMahon MA, Jilek BL, Brennan TP, et al. The HBV drug entecavir — effects on HIV-1 replication and resistance. *New England Journal of Medicine*. 2007;356(25):2614-2621. Available at: <https://www.nejm.org/doi/full/10.1056/NEJMoa067710>.
49. Dore GJ, Soriano V, Rockstroh J, et al. Frequent hepatitis B virus rebound among HIV-hepatitis B virus-coinfected patients following antiretroviral therapy interruption. *AIDS*. 2010;24(6):857-865. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/20216301>.
50. Bellini C, Keiser O, Chave JP, et al. Liver enzyme elevation after lamivudine withdrawal in HIV-hepatitis B virus co-infected patients: the Swiss HIV Cohort Study. *HIV Med*. 2009;10(1):12-18. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/18795964>.
51. Rey D, Krantz V, Partisani M, et al. Increasing the number of hepatitis B vaccine injections augments anti-HBs response rate in HIV-infected patients. Effects on HIV-1 viral load. *Vaccine*. 2000;18(13):1161-1165. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/10649616>.
52. Bessesen M, Ives D, Condreay L, Lawrence S, Sherman KE. Chronic active hepatitis B exacerbations in human immunodeficiency virus-infected patients following development of resistance to or withdrawal of lamivudine. *Clin Infect Dis*. 1999;28(5):1032-1035. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/10452630>.
53. Proia LA, Ngui SL, Kaur S, Kessler HA, Trenholme GM. Reactivation of hepatitis B in patients with human immunodeficiency virus infection treated with combination antiretroviral therapy. *Am J Med*. 2000;108(3):249-251. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/10723980>.
54. Neau D, Schvoerer E, Robert D, et al. Hepatitis B exacerbation with a precore mutant virus following withdrawal of lamivudine in a human immunodeficiency virus-infected patient. *J Infect*. 2000;41(2):192-194. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/11023772>.
55. Puoti M, Spinetti A, Ghezzi A, et al. Mortality for liver disease in patients with HIV infection: a cohort study. *J Acquir Immune Defic Syndr*. 2000;24(3):211-217. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/10969344>.
56. Bonacini M, Louie S, Bzowej N, Wohl AR. Survival in patients with HIV infection and viral hepatitis B or C: a cohort study. *AIDS*. 2004;18(15):2039-2045. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/15577625>.
57. Donato F, Boffetta P, Puoti M. A meta-analysis of epidemiological studies on the combined effect of hepatitis B and C virus infections in causing hepatocellular carcinoma. *Int J Cancer*. 1998;75(3):347-354. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/9455792>.
58. Collins JM, Raphael KL, Terry C, et al. Hepatitis B virus reactivation during successful treatment of hepatitis C virus with sofosbuvir and simeprevir. *Clin Infect Dis*. 2015;61(8):1304-1306. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/26082511>.
59. Takayama H, Sato T, Ikeda F, Fujiki S. Reactivation of hepatitis B virus during interferon-free therapy with daclatasvir and asunaprevir in patient with hepatitis B virus/hepatitis C virus co-infection. *Hepatol Res*. 2015. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/26297529>.

60. De Monte A, Courjon J, Anty R, et al. Direct-acting antiviral treatment in adults infected with hepatitis C virus: reactivation of hepatitis B virus coinfection as a further challenge. *J Clin Virol*. 2016;78:27-30. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/26967675>.
61. Ende AR, Kim NH, Yeh MM, Harper J, Landis CS. Fulminant hepatitis B reactivation leading to liver transplantation in a patient with chronic hepatitis C treated with simeprevir and sofosbuvir: a case report. *J Med Case Rep*. 2015;9:164. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/26215390>.
62. Vasishta S, Dieterich D, Mullen M, Aberg J. Brief report: hepatitis B infection or reactivation after switch to 2-drug antiretroviral therapy: a case series, literature review, and management discussion. *J Acquir Immune Defic Syndr*. 2023;94(2):160-164. Available at: <https://pubmed.ncbi.nlm.nih.gov/37345994>.
63. Denyer RV, Tate JP, Benator DA, Lim JK, Weintrob A. Hepatitis B reactivation in a US cohort of people with human immunodeficiency virus and hepatitis B core antibody after switch to antiretroviral therapy without hepatitis B activity. *Clin Infect Dis*. 2026. Available at: <https://pubmed.ncbi.nlm.nih.gov/41848012>.
64. Denyer RV, Tate JP, Benator DA, Kim JK, Weintrob AC. 1026. Hepatitis B reactivation in persons with HIV with positive hepatitis B core antibody after switching to antiretroviral therapy without hepatitis B activity. *Open Forum Infect Dis*. 2023;10:1-2. Available at: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10679264>.
65. McMahon MA, Jilek BL, Brennan TP, et al. The HBV drug entecavir — effects on HIV-1 replication and resistance. *N Engl J Med*. 2007;356(25):2614-2621. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/17582071>.
66. Koziel MJ, Peters MG. Viral hepatitis in HIV infection. *N Engl J Med*. 2007;356(14):1445-1454. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/17409326>.
67. European Association For The Study Of The Liver. EASL clinical practice guidelines: management of chronic hepatitis B virus infection. *J Hepatol*. 2012. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/22436845>.
68. Zoulim F, Locarnini S. Management of treatment failure in chronic hepatitis B. *J Hepatol*. 2012;56 Suppl 1:S112-122. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/22300461>.
69. Matthews GV, Ali RJ, Avihingsanon A, et al. Quantitative HBsAg and HBeAg predict hepatitis B seroconversion after initiation of HAART in HIV-HBV coinfecting individuals. *PLoS One*. 2013;8(4):e61297. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/23593455>.
70. Chihota BV, Wandeler G, Chilengi R, et al. High rates of hepatitis B virus (HBV) functional cure among human immunodeficiency virus-HBV coinfecting patients on antiretroviral therapy in Zambia. *J Infect Dis*. 2020;221(2):218-222. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/31613956>.
71. Nishijima T, Kawasaki Y, Tanaka N, et al. Long-term exposure to tenofovir continuously decrease renal function in HIV-1-infected patients with low body weight: results from 10 years of follow-up. *AIDS*. 2014;28(13):1903-1910. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/25259702>.

72. Pozniak A, Arribas JR, Gathe J, et al. Switching to tenofovir alafenamide, coformulated with elvitegravir, cobicistat, and emtricitabine, in HIV-infected patients with renal impairment: 48-week results from a single-arm, multicenter, open-label phase 3 study. *J Acquir Immune Defic Syndr*. 2016;71(5):530-537. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/26627107>.
73. Agarwal K, Brunetto M, Seto WK, et al. 96 weeks treatment of tenofovir alafenamide vs. tenofovir disoproxil fumarate for hepatitis B virus infection. *J Hepatol*. 2018;68(4):672-681. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/29756595>.
74. Mallon PW, Brunet L, Hsu RK, et al. Weight gain before and after switch from TDF to TAF in a U.S. cohort study. *J Int AIDS Soc*. 2021;24(4):e25702. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/33838004>.
75. Lange CM, Bojunga J, Hofmann WP, et al. Severe lactic acidosis during treatment of chronic hepatitis B with entecavir in patients with impaired liver function. *Hepatology*. 2009;50(6):2001-2006. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/19937695>.
76. Marzano A, Marengo A, Marietti M, Rizzetto M. Lactic acidosis during entecavir treatment in decompensated hepatitis B virus-related cirrhosis. *Dig Liver Dis*. 2011;43(12):1027-1028. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/21782535>.
77. Mao H, Kang T. Lactic acidosis during entecavir antiviral treatment in a patient with hepatitis B virus-related decompensated cirrhosis. *West Indian Med J*. 2015;64(2):165-166. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/26360694>.
78. Lau GK. Does treatment with interferon-based therapy improve the natural history of chronic hepatitis B infection? *J Hepatol*. 2007;46(1):6-8. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/17112628>.
79. Drake A, Mijch A, Sasadeusz J. Immune reconstitution hepatitis in HIV and hepatitis B coinfection, despite lamivudine therapy as part of HAART. *Clin Infect Dis*. 2004;39(1):129-132. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/15206064>.
80. Shelburne SA, 3rd, Hamill RJ, Rodriguez-Barradas MC, et al. Immune reconstitution inflammatory syndrome: emergence of a unique syndrome during highly active antiretroviral therapy. *Medicine (Baltimore)*. 2002;81(3):213-227. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/11997718>.
81. Crane M, Oliver B, Matthews G, et al. Immunopathogenesis of hepatic flare in HIV/hepatitis B virus (HBV)-coinfected individuals after the initiation of HBV-active antiretroviral therapy. *J Infect Dis*. 2009;199(7):974-981. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/19231993>.
82. Sulkowski MS, Mehta SH, Chaisson RE, Thomas DL, Moore RD. Hepatotoxicity associated with protease inhibitor-based antiretroviral regimens with or without concurrent ritonavir. *AIDS*. 2004;18(17):2277-2284. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/15577540>.
83. Pineda JA, Santos J, Rivero A, et al. Liver toxicity of antiretroviral combinations including atazanavir/ritonavir in patients co-infected with HIV and hepatitis viruses: impact of pre-existing liver fibrosis. *J Antimicrob Chemother*. 2008;61(4):925-932. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/18276600>.

84. Neukam K, Mira JA, Collado A, et al. Liver toxicity of current antiretroviral regimens in HIV-infected patients with chronic viral hepatitis in a real-life setting: The HEPAVIR SEG-HEP Cohort. *PLoS One*. 2016;11(2):e0148104. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/26848975>.
85. Hoffmann CJ, Charalambous S, Martin DJ, et al. Hepatitis B virus infection and response to antiretroviral therapy (ART) in a South African ART program. *Clin Infect Dis*. 2008;47(11):1479-1485. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/18937580>.
86. Sulkowski MS, Thomas DL, Chaisson RE, Moore RD. Hepatotoxicity associated with antiretroviral therapy in adults infected with human immunodeficiency virus and the role of hepatitis C or B virus infection. *JAMA*. 2000;283(1):74-80. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/10632283>.
87. Stern JO, Robinson PA, Love J, Lanes S, Imperiale MS, Mayers DL. A comprehensive hepatic safety analysis of nevirapine in different populations of HIV infected patients. *J Acquir Immune Defic Syndr*. 2003;34 Suppl 1:S21-33. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/14562855>.
88. Sherman KE, Shire NJ, Cernohous P, et al. Liver injury and changes in hepatitis C Virus (HCV) RNA load associated with protease inhibitor-based antiretroviral therapy for treatment-naïve HCV-HIV-coinfected patients: lopinavir-ritonavir versus nelfinavir. *Clin Infect Dis*. 2005;41(8):1186-1195. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/16163639>.
89. Reuben A. Hy's law. *Hepatology*. 2004;39(2):574-578. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/14768020>.
90. Lok AS, Zoulim F, Locarnini S, et al. Antiviral drug-resistant HBV: standardization of nomenclature and assays and recommendations for management. *Hepatology*. 2007;46(1):254-265. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/17596850>.
91. Park ES, Lee AR, Kim DH, et al. Identification of a quadruple mutation that confers tenofovir resistance in chronic hepatitis B patients. *J Hepatol*. 2019;70(6):1093-1102. Available at: <https://pubmed.ncbi.nlm.nih.gov/30794889>.
92. Benhamou Y, Bochet M, Thibault V, et al. Long-term incidence of hepatitis B virus resistance to lamivudine in human immunodeficiency virus-infected patients. *Hepatology*. 1999;30(5):1302-1306. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/10534354>.
93. Matthews GV, Seaberg E, Dore GJ, et al. Combination HBV therapy is linked to greater HBV DNA suppression in a cohort of lamivudine-experienced HIV/HBV coinfecting individuals. *AIDS*. 2009;23(13):1707-1715. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/19584701>.
94. Vassiliadis TG, Giouleme O, Koumerkeridis G, et al. Adefovir plus lamivudine are more effective than adefovir alone in lamivudine-resistant HBeAg- chronic hepatitis B patients: a 4-year study. *J Gastroenterol Hepatol*. 2010;25(1):54-60. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/19780875>.

95. Lampertico P, Vigano M, Manenti E, Iavarone M, Sablon E, Colombo M. Low resistance to adefovir combined with lamivudine: a 3-year study of 145 lamivudine-resistant hepatitis B patients. *Gastroenterology*. 2007;133(5):1445-1451. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/17983801>.
96. Ze E, Baek EK, Lee JJ, et al. Long-term outcomes of two rescue therapies in lamivudine-refractory patients with chronic hepatitis B: combined lamivudine and adefovir, and 1-mg entecavir. *Clin Mol Hepatol*. 2014;20(3):267-273. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/25320730>.
97. Lai CL, Chien RN, Leung NW, et al. A one-year trial of lamivudine for chronic hepatitis B. Asia Hepatitis Lamivudine Study Group. *N Engl J Med*. 1998;339(2):61-68. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/9654535>.
98. Dienstag JL, Schiff ER, Wright TL, et al. Lamivudine as initial treatment for chronic hepatitis B in the United States. *N Engl J Med*. 1999;341(17):1256-1263. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/10528035>.
99. Miro JM, Laguno M, Moreno A, Rimola A, Group. HCOiHW. Management of end stage liver disease (ESLD): what is the current role of orthotopic liver transplantation (OLT)? *J Hepatol*. 2006;44(1 Suppl):S140-145. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/16352366>.
100. Lau GK, Yiu HH, Fong DY, et al. Early is superior to deferred preemptive lamivudine therapy for hepatitis B patients undergoing chemotherapy. *Gastroenterology*. 2003;125(6):1742-1749. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/14724827>.
101. Lee YH, Bae SC, Song GG. Hepatitis B virus reactivation in HBsAg-positive patients with rheumatic diseases undergoing anti-tumor necrosis factor therapy or DMARDs. *Int J Rheum Dis*. 2013;16(5):527-531. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/24164839>.
102. Reddy KR, Beavers KL, Hammond SP, Lim JK, Falck-Ytter YT, Institute. AGA. American Gastroenterological Association Institute guideline on the prevention and treatment of hepatitis B virus reactivation during immunosuppressive drug therapy. *Gastroenterology*. 2015;148(1):215-219; quiz e216-217. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/25447850>.
103. Huang YH, Hsiao LT, Hong YC, et al. Randomized controlled trial of entecavir prophylaxis for rituximab-associated hepatitis B virus reactivation in patients with lymphoma and resolved hepatitis B. *J Clin Oncol*. 2013;31(22):2765-2772. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/23775967>.
104. Mori S, Fujiyama S. Hepatitis B virus reactivation associated with antirheumatic therapy: risk and prophylaxis recommendations. *World J Gastroenterol*. 2015;21(36):10274-10289. Available at: <https://www.ncbi.nlm.nih.gov/pubmed/26420955>.
105. Ali FS, Nguyen MH, Hernaez R, et al. AGA Clinical Practice Guideline on the Prevention and Treatment of Hepatitis B Virus Reactivation in At-Risk Individuals. *Gastroenterology*. 2025;168(2):267-284. Available at: <https://doi.org/10.1053/j.gastro.2024.11.008>.

106. Schillie S, Vellozzi C, Reingold A, et al. Prevention of hepatitis B virus infection in the United States: recommendations of the Advisory Committee on Immunization Practices. *MMWR Recomm Rep*. 2018;67(1):1-31. Available at: <https://www.cdc.gov/mmwr/volumes/67/rr/rr6701a1.htm>.
107. Panel on Treatment of HIV During Pregnancy and Prevention of Perinatal Transmission. Hepatitis B virus/HIV coinfection. 2026. Available at: <https://clinicalinfo.hiv.gov/en/guidelines/perinatal/hepatitis-b-virus-hiv-coinfection?view=full>.
108. del Canho R, Grosheide PM, Schalm SW, de Vries RR, Heijtkink RA. Failure of neonatal hepatitis B vaccination: the role of HBV-DNA levels in hepatitis B carrier mothers and HLA antigens in neonates. *J Hepatol*. 1994;20(4):483-486. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/8051386>.
109. Ngui SL, Andrews NJ, Underhill GS, Heptonstall J, Teo CG. Failed postnatal immunoprophylaxis for hepatitis B: characteristics of maternal hepatitis B virus as risk factors. *Clin Infect Dis*. 1998;27(1):100-106. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/9675462>.
110. Wiseman E, Fraser MA, Holden S, et al. Perinatal transmission of hepatitis B virus: an Australian experience. *Med J Aust*. 2009;190(9):489-492. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/19413519>.
111. Kubo A, Shlager L, Marks AR, et al. Prevention of vertical transmission of hepatitis B: an observational study. *Ann Intern Med*. 2014;160(12):828-835. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/24862434>.
112. Panel on Treatment of HIV During Pregnancy and Prevention of Perinatal Transmission. Recommendations for use of antiretroviral drugs during pregnancy and interventions to reduce perinatal HIV transmission in the United States. 2026. Available at: <https://clinicalinfo.hiv.gov/en/guidelines/perinatal/whats-new-guidelines>.