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British Association of Sexual Health and HIV guidelines on the management of viral hepatitis 2026

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Abstract

The 2026 British Association for Sexual Health and HIV (BASHH) guidelines for the management of viral hepatitis are in line with current evidence and practice within the United Kingdom. We provide evidence-based recommendations for the prevention, diagnosis and initial management of viral hepatitis A, B, C and D. Key updates are detailed at the start of the article.

Keywords

hepatitis virus, antiviral, vaccine

What is new in the guideline?

- Offer screening for chronic hepatitis B virus (HBV) infection to all sexual health clinic attendees at least once.
- Expanded recommendation for opportunistic HBV vaccination as prevention in those with ongoing risk of sexually transmitted HBV.
- Updated recommendations to include novel HBV vaccines in sexual health clinics.
- Guidance on use of tenofovir-containing human immunodeficiency virus (HIV) post-exposure prophylaxis (PEP) and pre-exposure prophylaxis (PrEP) in people with chronic HBV infection.
- Updated recommendations for hepatitis C virus (HCV) testing in sexual health clinics.
- Reduced detail on use of antiviral therapies in hepatitis B and C management. Clinicians should refer to European Association for the Study of the Liver (EASL) Guidelines.[1,2]

Introduction and methodology

Objectives

The objective of this guideline is to answer health questions about how to prevent, diagnose and manage viral hepatitis care within a sexual health service and to support the development of appropriate local care pathways. The guideline is primarily for use by clinicians and policymakers in sexual health services within the United Kingdom (UK). It is recommended that management of people diagnosed with viral hepatitis is via local pathways agreed with Hepatologists.

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The guideline offers recommendations for the prevention, diagnosis and initial management of viral hepatitis A, B, C and D. This is an update to the 2017 British Association for Sexual Health and HIV (BASHH) National Guidelines for the Management for Viral Hepatitides.[3]

The guideline is aimed primarily at patients aged 16 years or older presenting to healthcare professionals working in departments offering specialist level 3 care in sexually transmitted infection (STI) management within the UK. However, the principles of the recommendations are applicable across levels of STI care providers, and non-specialist services may need to develop, where appropriate, local referral pathways.

Search strategy

This guideline was produced according to specifications set out in the Clinical Effectiveness Group's (CEG) document 'Framework for guideline development and assessment' (2015, updated 2019) accessed at https://www.bashh.org/_userfiles/pages/files/resources/2020_guidelines_framework.pdf.

The Grading of Recommendations Assessment, Development and Evaluation (GRADE, refer to Appendix 1 system was used to assess the evidence and make recommendations as detailed in the guidance.

Methods

This work reviews and updates the 2017 BASHH guidelines[3] and incorporates the findings of a comprehensive literature review on hepatitis A, B, C and D infections. In addition, sections on hepatitis in relevant national and international guidelines were reviewed including the European International Union Against STI (IUSTI) Guideline for the Management of Hepatitis B and C infections in Sexual Health Settings 2017[4]; the EASL Guidelines for the Management of Hepatitis B[1] and Hepatitis C[2]; the British HIV Association (BHIVA) guidelines for the Management of Hepatitis Viruses (2013),[5] the American Association for the Study of Liver Disease (AASLD) Guidelines for Hepatitis B[6] and Hepatitis C[7] and the BHIVA guidelines on Use of Vaccines in people living with HIV.[8] Vaccine updates were cross-referenced with the Green Book.[9] Forward and backward searching from key references was also conducted. All writing group members underwent GRADE training. The strength of the recommendation is graded as 1 (strong) or 2 (weaker/conditional) and the quality of the evidence is graded from A (high-quality) to D (low-quality). Good practice points (GPP) are recommendations based on the clinical judgement of the working group. The recommendations are the result of a series of meetings of the writing committee and incorporate input from the public consultation process (comments available on request).

Equality impact assessment

An assessment of the guideline and its recommendations was undertaken to ensure the principles of equality and diversity were adhered to and is available in Appendix 2.

BASHH has adopted an anatomical approach without assuming gender in the majority of guidelines and uses gender terminology in line with BASHH 'sexual health standards for trans, including non-binary, people'.

Stakeholder involvement, piloting and feedback

The guideline working group included: patient representatives; clinicians from Sexual Health/HIV, Infectious Diseases, Hepatology, Virology; representatives from BASHH, BHIVA, Society of Sexual Health Advisers (SSHA), HIV Pharmacy Association (HIVPA), The Hepatitis C Trust and UK Health Security Agency (UKHSA).

The first draft was produced by the writing group and then circulated to the BASHH CEG for review using the Appraisal of Guidelines, Research and Evaluation (AGREE, refer to Appendix 3) tool. The second draft of the guideline was posted on the BASHH website for wider consultation (2 months) and any comments received during the consultation period were reviewed by the authors and acted on appropriately. The final draft was presented to the CEG for review and piloting.

Once the guideline is published, the CEG will keep it under review should critical new evidence become available that affects the current recommendations. The guideline will be formally reviewed and updated, if necessary, every 5 years.

Summary of recommendations

Acute viral hepatitis in sexual health clinics

- If acute viral hepatitis is suspected, we recommend the following tests to establish hepatitis aetiology and assess severity: liver function tests (LFT), clotting, hepatitis A virus-specific immunoglobulin type M (HAV-IgM), hepatitis B surface antigen (HBsAg), antibody against hepatitis B core antigen (anti-HBc) IgM, antibody against HCV (anti-HCV), HCV ribonucleic acid (RNA)/core antigen, antibody against hepatitis E virus (anti-HEV) and HEV RNA (**1B**).
- Following clinical assessment, a management plan for acute viral hepatitis should be devised in conjunction with Hepatology (**1A**).
- Public health authorities should be notified (**1C**).
- Perform contact tracing in conjunction with public health colleagues where appropriate. (**1C**).

Assessment of HAV immunity

- We recommend HAV immunity and vaccine assessment for the following people: gay, bisexual or other men who have sex with men (GBMSM), trans women who have sex with men, people who inject drugs (PWID), people with HBV, HCV or HIV (**1B**).
- Screening for pre-existing HAV immunity before vaccination may be performed in a non-outbreak situation if the person is likely to re-attend (**2B**).
- If HAV antibody test is performed, the first dose of vaccine can be given at the same time (**1B**). The antibody result will aid decision-making regarding further doses.

HAV vaccination

- HAV monovalent vaccine schedule: 2 doses (at 0, 6–12 months) provide 95% protection for at least 10 years (**1A**). If using Twinrix® a 3-dose schedule should be used.
- There is increasing evidence that HAV vaccine-induced immunity may be >25 years and possibly lifelong, so no further booster doses are needed after the primary course in immunocompetent individuals (**1B**).

HBV screening

- We recommend that all sexual health clinic attendees should be offered testing for chronic HBV infection at least once (**1D**).
- We recommend HBsAg test is used to screen for the presence of chronic HBV infection (**1B**).
- In those eligible for vaccination, further assessment of HBV immunity (hepatitis B surface antigen antibody (anti-HBs) titre and anti-HBc tests) is not routinely required in vaccine naïve immunocompetent individuals.

HBV vaccination

- We recommend prophylactic HBV vaccination (**1B**) for:
  - GBMSM, trans people who have sex with men.
  - Sex workers.
  - People requesting HIV PEP/PrEP.
  - People from, or with sexual partners from, countries of HBV prevalence >2% (see Figure 1).
  - People who have injected drugs or who are smoking heroin/crack cocaine.
  - People with HIV.
  - Close contacts of people with HBV or at increased risk of HBV.
  - Prisoners.
  - Immigration detainees.
  - People with chronic liver disease.
- We recommend clinicians consider opportunistic prophylactic HBV vaccination for people who may be at increased risk of sexual HBV acquisition including (**GPP**):
  - People with more than one sexual partner during the previous 3 months.

Figure 1. Map showing seroprevalence of HBsAg (all ages).[10] HBsAg=Hepatitis B surface antigen. Created with mapchart.net.

- People seeking treatment for a STI.

- We recommend HBV vaccination is initiated within 7 days following potential HBV exposure (**GPP**):
  - After sexual assault.
  - After occupational/community risk exposures.

HBV vaccine schedules

- We recommend people commence an HBV vaccine course which provides optimal uptake, completion and immunity in accordance with schedules published in the Green Book (see Section 217.4).[11] (**GPP**)
- HBV-monovalent or combined HAV/HBV vaccines have been widely used in sexual health services for HBV prevention.
- The recommended schedules for conventional HBV vaccines include:
  - HBVaxPRO® and Engerix B® preferred accelerated course (doses at 0, 1, 2, 12 months), alternative course (0, 1, 6 months).
  - If need to provide rapid immunity, the super accelerated or very rapid schedule can be used: Engerix B® or Twinrix Adult® (0, 1, 3 weeks and 12 months).
  - For people with HIV or renal insufficiency, HBVaxPRO® 40 mcg, Engerix B® 40 mcg dose or Fendrix adjuvanted 20 mcg can be used (schedule 0, 1, 2, 6 months).
  - Heplisav B®, an HBV vaccine with a novel adjuvant and faster 2-dose schedule has demonstrated superior immunogenicity when compared with Engerix B®. If available, Heplisav B® (0, 1 month) may be preferred in those who are likely to have a poorer response to vaccine, have not responded to other monovalent vaccines, where a rapid response is required or if compliance may be an issue (**GPP**).

People with HBV (HBsAg positive): Local pathways and responsibilities to be determined with Hepatology

- Partner notification and contact tracing (**1B**).
- Provide information on HBV, alcohol avoidance and transmission (**GPP**).
- Test for HIV and HCV infections (**1D**).
- Test for other sexual infections (**1D**).
- Vaccinate for HAV if non-immune (**1D**).
- Refer to Hepatology specialist for ongoing management, hepatitis delta virus (HDV) testing and hepatocellular carcinoma (HCC) surveillance (**1B**).

Use of tenofovir-containing HIV PrEP in people with HBV infection

- Tenofovir-containing HIV PrEP may be initiated while HBV serology results are pending (**GPP**).
- Any individual found to be HBsAg positive should be referred to a Hepatology specialist service whilst remaining on tenofovir-containing PrEP (**GPP**).
- People with chronic HBV taking tenofovir-containing HIV PrEP are recommended to use daily dosing, NOT event-based dosing schedules (**1D**).
- There is a risk of HBV rebound viraemia and liver dysfunction upon stopping tenofovir in people with HBV infection. The management of stopping tenofovir-containing PrEP should be discussed in advance with a Hepatology specialist (**GPP**).

HCV screening

- Testing individuals from the following groups for HCV infection is recommended in (**1B**, see Table 4):
  - People with a past/current history of injecting drug use.
  - People with HIV.
  - GBMSM with risk factors for acquiring HCV (see further details section 11.3.2).
  - People with a recent history of HBV
  - GBMSM recently diagnosed with syphilis or lymphogranuloma venereum (LGV).
  - People born in a country of intermediate or high HCV prevalence.
- Consider testing individuals with other risk factors for HCV acquisition (**2D**):
  - People with a history of intranasal/inhaled recreational drug use; have exchanged sex for money, goods or favours; have experienced homelessness; have been in prison or other secure/detained settings; with parenteral exposures in unregulated settings (e.g., tattoos, piercings); with a possible healthcare-related exposure in a low- or middle-income country (e.g., non-sterile needle use).

People with positive anti-HCV

- Confirm active HCV infection using HCV RNA or core antigen (**1B**).

People with positive HCV RNA or core antigen - local pathways and responsibilities to be determined with Hepatology

- Partner notification and contact tracing.
- Provide information on HCV, alcohol avoidance and transmission.
- Check for HAV and HBV immunity and vaccinate if non-immune (**1D**).
- Test for HIV infection (**1D**).
- Test for other sexual infections. (**1D**).

- Refer to Hepatology specialist for ongoing management and antiviral therapy.

HCV screening for individuals with past HCV infection

- Use HCV RNA or core antigen testing for individuals with ongoing risk for HCV reinfection (**1D**).

Prevention of hepatitis A/B in sexual health

HAV and HBV: Vaccination in sexual health

Table 1 presents a summary of HAV and HBV vaccination in sexual health.

HAV: Vaccination

- We recommend that all GBMSM, trans women who have sex with men, people who have injected drugs, people with HBV, HCV and/or HIV attending a sexual health clinic should be opportunistically offered HAV vaccine, where available, unless they have documented evidence of two doses of HAV vaccine or of previous HAV illness.[13]
- Screening for pre-existing HAV exposure before vaccination has been found to be cost-effective in one study and therefore may be performed, depending on other factors such as funding, local epidemiology and clinic access.[14] (**2B**) Data from the ANRS IPERGAY trial document that out of 427 men who have sex with men (median age 34.8 years), 50.1% were not immune to HAV at baseline, and lack of immunity was associated with younger age.[15]
- If HAV antibody test is performed, or if it is likely that the individual will not return, the first dose of vaccine should be given at the same time. (**1B**) The antibody result will aid decision making regarding further doses.[16]
- To prevent sustained outbreaks, it is estimated that at least 70% of GBMSM should have HAV immunity.[17]
- Vaccination should be obtained via alternative sources e.g., primary care or travel clinic for people with travel or occupational risk.
- HAV vaccine schedule: doses at 0 and 6–12 months confers 95% protection for at least 10 years. (**1A**) There is increasing evidence that vaccine induced immunity may be >25 years and possibly lifelong, so no further booster doses are needed after the primary course in immunocompetent patients.[13,18] (**1B**)
- People with HIV respond to HAV vaccine (as demonstrated by antibody production in 46%–88%) but titres are lower than in HIV-negative individuals, and correlate with CD4 count.[19,20] All adults living with HIV should be screened for HAV IgG and seronegative individuals should be offered HAV vaccination. A three-dose schedule (0, 1 and 6 months) is preferred for individuals with CD4 counts <300 cells/μL or HIV viraemia (1B). For further details see BHIVA Immunisation Guidelines.[8]
- Side effects of hepatitis A vaccines: these are usually mild and likely only for the first few days following immunisation. Very common side effects can include mild injection site pain and redness. Common side effects include fever, myalgia, headache, nausea, vomiting, decreased appetite, diarrhoea and abdominal pain.

HAV: Management of contact with acute hepatitis A

- Non-immune contacts (no history of vaccination or lab-confirmed immunity) maybe given HAV vaccine up to 14 days following exposure, providing exposure took place within the source's period of infectiousness (i.e., during the prodromal illness or first week of jaundice).[13] (**1A**)
- For exposures presenting at beyond 14 days, HAV vaccine should be offered to those with chronic liver disease and to members of the household if there is >1 contact to prevent tertiary cases.[13] Vaccine should also be offered to those at ongoing sexual risk.[21–23]
- Human normal immunoglobulin (HNIG) 1000 mg intramuscularly should be considered in addition to HAV vaccine for contacts who are less able to respond to the vaccine or are at higher risk of complications (e.g., concurrent chronic HBV or HCV, chronic liver disease, patients with advanced HIV (CD4 + cell count <200 cells/μL) or other forms of immunosuppression, or age >60 years).[13] (**1A**)
- HNIG works best if given in the first few days after first contact with an efficacy of 90% and is unlikely to give any protection if given more than 2 weeks after first exposure, but may reduce disease severity if given up to 28 days after exposure.[13] Where HNIG is being considered, prompt discussion with the local UKHSA team +/− local Microbiology/Virology/Public Health authorities is recommended.[24]

HBV: Vaccination

We recommend prophylactic HBV vaccination for the following people, if non-immune, in sexual health settings.[11,25–35] (1A)

- GBMSM, trans people who have sex with men.
- Sex workers.
- People requesting HIV PEP/PrEP.

Table 1. Vaccinations in sexual health.

	HAV	HBV
Offer vaccine to following people in sexual health clinics	• GBMSM • Trans people who have sex with men • PWID • People with HBV, HCV or HIV	• GBMSM • Trans people who have sex with men • Sex workers • People requesting PEP/PrEP • People from/with sexual partners from countries of HBV prevalence >2% • PWID or who are smoking heroin/crack cocaine • People with HIV • Close contacts of people with HBV or at increased risk of HBV • Prisoners • Immigration detainees • People with chronic liver disease • Consider offering HBV vaccine to people with more than one sexual partner in the previous 3 months and people seeking treatment for a sexually transmitted infection
Type of vaccines for adults >16 years	**Monovalent HAV vaccines e.g.:** • Havrix Monodose® 1 mL (>16 years) • Avaxim® 0.5 mL (>16 years) • Vaqta® 1 mL (>18 years) **Combined HAV/HBV vaccines:** • Twinrix adult® 1 mL (>16 years)	**Monovalent HBV vaccines e.g.:** • Engerix B® 20 mcg/1 mL (>16 years) • Engerix B® 2 × 20 mcg/1 mL (>16 years and dialysis/pre-dialysis) • HBvaxPRO® 10 mcg/1 mL (>16 years) • HBvaxPRO® 40 mcg/1 mL (in people living with HIV and adults with renal insufficiency [dialysis/pre-dialysis]) **Adjuvanted:** • Fendrix® 20 mcg/0.5 mL (>15 years and dialysis/pre-dialysis) • Heplisav B® 20 mcg/0.5 mL (>18 years) **Combined HAV/HBV vaccines:** • Twinrix adult® 1 mL (>16 years)
Under 16 years	• Havrix Junior Monodose® 0.5 mL (1–15 years) • Avaxim Junior® 0.5 mL (1–15 years) • Vaqta Paediatric® 0.5 mL (1–17 years)	• Engerix B® 10 mcg/0.5 mL (0–15 years) • HBvaxPRO Paediatric® 5 mcg/0.5 mL (0–15 years)
Route of administration	**Monovalent HAV vaccines e.g.,**: • Intramuscularly into the upper arm or anterolateral thigh. Vaccine should not be administered in the gluteal region **Twinrix® adult:** • Intramuscular in the deltoid region	**Engerix B®/HBvaxPRO®:** • Intramuscularly in the upper arm or anterolateral thigh. The buttock must not be used because vaccine efficacy may be reduced **Heplisav B/Twinrix adult®:** • Intramuscular in the deltoid region

(continued)

Table 1. (continued)

	HAV	HBV
Schedule (adults)	**Monovalent HAV vaccine:** • Two doses at 0 and 6–12 months provides protection for up to 25 years • Delays in second dose (even up to several years) results in successful boosting so restarting schedule not required **Twinrix adult®:** • 0,1 and 6 months	**Engerix B®/HBvaxPRO®:** • Accelerated schedule: 0, 1, 2 and 12 months (preferred) • Standard schedule: 0,1 and 6 months • If need to provide rapid immunity, the super accelerated or very rapid schedule can be used: Engerix B® or Twinrix adult® (0, 1, 3 weeks and 12 months). Although unlicensed, can be used in 16-18years if rapid immunity required to maximise compliance **Fendrix® 20 mcg/Engerix B® (double dose = 2 x 20 mcg):** • 0, 1, 2 and 6 months – adults with renal insufficiency **Heplisav B[a]:** • 2 doses (0, 1 month) • Adults with severe renal impairment (eGFR <30 ml/min including patients undergoing haemodialysis): 0, 1, 2 and 4 months
People with HIV. Further details in BHIVA immunisation guidelines[12]	**Monovalent HAV vaccine eg Havrix Monodose®:** • Two doses (0 and 6 months) for most adults; • a three-dose schedule (0, 1 and 6 months) is preferred for individuals with CD4 counts <300 cells/mm^3 or viraemia.	**HBvaxPRO® 40 mcg, Engerix B® 40 mcg (2x20 mcg doses), Fendrix® 20 mcg:** • Standard schedule: 0, 1, 2, and 6 months **Heplisav B®:** • Two doses (0, 1 month). The need for a third dose at 6 months is less clear at present, but it is recommended in individuals with age ≥40 years, CD4 counts <500 cells/mm^3, viraemia, chronic renal disease or diabetes

BHIVA, British HIV Association; eGFR, Estimated glomerular filtration rate; HAV, Hepatitis A virus; HBV, Hepatitis B virus; HCV, Hepatitis C virus; HIV, Human immunodeficiency virus; PEP, Post-exposure prophylaxis; PrEP, Pre-exposure prophylaxis; PWID, People who inject drugs.
[a]Additional eligibility criteria in Green Book Chapter 18.

- People from, or with sexual partners from, countries of HBV prevalence >2%.
- People who have injected drugs or who are smoking heroin/crack cocaine.
- People with HIV.
- Close contacts of people with HBV or at increased risk of HBV.
- Prisoners.
- Immigration detainees.
- People with chronic liver disease.

We recommend sexual health clinicians consider opportunistic HBV vaccination for people attending their services who may be at increased risk of sexual HBV acquisition and have not benefitted from universal HBV vaccination programmes (GPP) including:[11,35,36]

- People with more than one sexual partner during the previous 3 months.
- People seeking treatment for a STI.
- It is acknowledged that this recommendation may have logistical and/or financial implications for sexual health services.

HBV vaccination should be initiated as soon as possible (ideally within 24–48 h) following potential HBV exposure:

- After sexual assault.
- After occupational/community risk exposures.

There is likely to be little benefit of post-exposure prophylaxis after 7 days, but vaccination causes negligible harm and can provide offer protection for future HBV exposures.

In addition, the Green Book Chapter 18 also recommends HBV vaccination for the following individuals[11]:

- Foster carers and families adopting children from intermediate/high HBV prevalence countries.
- People receiving regular blood products and their carers.

- Recipients of solid organ transplants.
- People with chronic kidney failure (stage 4 or 5) or chronic liver disease.
- People resident in prison or detention centres.
- People in residential care for people with learning disabilities.
- Individuals with travel or occupational risk.

Side effects of hepatitis B vaccines: Very common side effects can include irritability, pain and redness at injection site and fatigue. Common side effects include loss of appetite, drowsiness, headache, nausea, vomiting, diarrhoea, abdominal pain and fever.

HBV vaccination as primary prevention: Choice of vaccine and schedule

The recommended schedules for conventional HBV vaccines include:

- HBVaxPRO® and Engerix B® preferred accelerated course (doses at 0, 1, 2, 12 months); alternative course (0, 1, 6 months).
- If need to provide rapid immunity, the super accelerated or very rapid schedule can be used: Engerix B® or Twinrix Adult® (0, 1, 3 weeks and 12 months).
- For people with HIV or renal insufficiency, HBVaxPRO® 40 mcg, Engerix B® 40 mcg or Fendrix® adjuvanted 20 mcg can be used (schedule 0, 1, 2, 6 months).
- Fendrix® 20 mcg can also be used for others who have not responded to conventional vaccine (**1A**).[37–40]
- Heplisav B®, a HBV vaccine with a novel adjuvant and faster 2-dose schedule has demonstrated superior immunogenicity when compared with Engerix B®. Heplisav B® showed 95% protective immunity after two doses compared to 81.3% for Engerix B® 3-dose schedule in people aged 18–55 years. Observational efficacy data has also been published showing 93.4% seroprotection after two doses of Hepislav B® in people with HIV on antiretroviral therapy (ART) when compared with 53.6% of people vaccinated using Engerix B®.[41–43]
- If available, Heplisav B® (0, 1 month) may be preferred in those who are likely to have a poorer response to vaccine, have not responded to other monovalent vaccines, where a rapid response is required or if compliance may be an issue (**GPP**).

Assessing HBV vaccine-induced immunity

Post HBV vaccination immunity testing is NOT routinely recommended in the Green Book[11] for most adults as conventional vaccines are highly effective. Testing for HBV immunity should only be performed where there are clinical concerns that an individual will not mount a robust immune response, for example:

- People with HIV, renal insufficiency and immunosuppression. For people living with HIV, please refer to the BHIVA Immunisation Guidelines.[8]
- People presenting following a significant exposure.

If an immunity check is required, the recommended time to check anti-HBs titre is 4–8 weeks following the last dose of either the standard (0, 1, 6 months) or accelerated (0, 1, 2, 12 months) vaccine course. In the accelerated course, protection is expected after the first three doses, with the 12-months dose required to reach immunogenic comparison with the standard course.[11]

Antibody responses vary widely between individuals (Table 2). It is preferable to achieve anti-HBs levels >100 mIU/ml, although levels of 10 mIU/ml or more are generally accepted as enough to protect against infection.

Following a full primary course:

Table 2. HBV vaccine-induced immunity.

Anti-HBs titre following full course of vaccine	
>100 mIU/mL	No further doses or titre check required in absence of immunosuppressive medication or morbidity
>10–100 mIU/mL	One immediate additional dose vaccine
<10 mIU/mL	Test for markers of current infection (HBsAg and anti-HBc) If no evidence of infection: Repeat a full course of vaccine Re-test Anti-HBs titre 4-8 weeks after completion of course If still <10 mIU/mL non-responder. Consider use of adjuvanted vaccine (e.g. Fendrix® Heplisav B®) if at ongoing risk.

Anti-HBc, Antibody against hepatitis B core antigen; Anti-HBs, Antibody against hepatitis B surface antigen; HBsAg, Hepatitis B surface antigen.

Responders with anti-HBs levels greater than or equal to 100 mIU/ml do not require any further doses. In immunocompetent individuals, once a satisfactory response has been established, further assessment of antibody levels is not indicated.

Responders with anti-HBs levels of 10 to 100mIU/ml should receive one additional dose of vaccine at that time (or whenever identified, even if years later). In immunocompetent individuals, further assessment of antibody levels or any booster doses are not required.

An antibody level below 10mIU/ml (taken at the correct interval, 4–8 weeks after a primary course), is classified as a non-response to vaccine. In non-responders, a repeat course of vaccine is recommended, followed by retesting 4–8 weeks after the second course.

A low antibody level (<10 mIU/ml) in someone tested at the wrong interval, may not indicate non-response, as they may still have immune memory. If a booster is given, testing at the correct interval should be undertaken to inform future management. Interval titre checking and reinforcing doses should be considered for people in the following categories:

- People with HIV, renal insufficiency and immunosuppression (for people with HIV refer to the BHIVA Immunisation Guidelines.[8]
- People presenting following a significant exposure.
- People with occupational risk.

Incomplete HBV vaccinations

Evidence suggests that if vaccine courses are not completed in immunocompetent patients, the outstanding doses can be given years later without the need to restart a course. (**1B**) One or two doses of vaccine may provide immunity in 40% and over 90% of immunocompetent patients, respectively.[44,45]

HBV: Vaccination in people with HIV

- People living with HIV show a reduced response rate to HBV vaccine and become anti-HBs negative more quickly, although higher dose vaccine increases the response by 13%.[3,46–48] (**1B**) Response correlates with CD4 count if not on ART but also with viral load and ART use. Vaccine response improves if the CD4 count rises, and the viral load is undetectable on ART.
- BHIVA recommends high dose vaccination (40 µg EngerixB® or HBvaxPro40®) or Fendrix® 20 mcg at 0, 1, 2, 6 months.[12] Only to use a single dose 0, 1, 3 weeks ultra-rapid course if CD4 count >500 and a rapid course is essential. Anti-HBs levels should be measured 4–8 weeks after completion of the primary course and regularly monitored at routine follow-up.

HBV: Post-exposure management of contacts

We recommend consulting the Green Book Chapter 18[11] for further information on HBV PEP. Recommendations depend upon the HBsAg status of the source, the vaccination status of the contact and timing of exposure. Where the presenting contact is non-immune or has unknown immunity after a single sexual or parenteral exposure to HBsAg positive index, baseline HBV serology should be undertaken and vaccination initiated whilst awaiting the results.

Vaccination as HBV PEP

- Following an exposure, the accelerated course of recombinant vaccine should be offered to all sexual and household contacts as soon as possible. Vaccination should also be offered following possible HBV exposure in occupational or community needlestick exposures; responsibility for where this care is provided should be determined locally.
- For HBV PEP, an accelerated schedule of monovalent HBV vaccine (or a combined vaccine of equivalent strength) should be used, with vaccine given at 0, 1, 2 months. A dose at 12 months should be given if they remain at higher risk.[11]
- If previously partially vaccinated, one dose should be given immediately and the course completed.
- If previously fully vaccinated, a booster dose should be given if last dose was >1 year ago.

Specific HBIG

- Contact details for hepatitis B immunoglobulin (HBIG) use are in the Green Book Chapter 18 supplies section.[11]
- HBIG provides passive immunity and can give immediate but temporary protection after exposure. HBIG does not affect the development of active immunity when given with HBV vaccine. If infection has already occurred at the time of immunisation, severe illness and development of chronic HBV may be prevented.
- HBIG is used after exposure to give rapid protection until HBV vaccine, which should be given at the same time, becomes effective. As vaccine alone is highly effective, the use of HBIG in addition to vaccine is only recommended in high-risk situations or in a known non-responder to vaccine. Whenever immediate protection is required, immunisation with the vaccine should be given.
- If indicated, HBIG works best within 12 h and ideally within 48 h. It is not indicated after 7 days.[11,49,50] (**1A**)
- Any sexual partner of people with acute HBV should be offered vaccine, and if seen within 1 week of last contact, should also be offered HBIG.

- HBIG dose for individuals >10 years old = 500 IU IM.[11,49]
- HBIG is in limited supply in the UK.

General advice for contacts of HBV

- Contacts should avoid sexual contact, especially unprotected penetrative sex, until it is shown that infection has not been acquired and it has been shown that vaccination has been successful (anti-HBs titres >10 IU/L).[11,32,51–56] (**1D**)
- Discuss condom use and how to reduce the risk of catching HBV through avoiding needle sharing. (**1C**)

Hepatitis A virus infection

New in hepatitis A section

- Updated HAV epidemiology.
- Hepatitis A vaccine updates including booster doses and duration of immunity.

Background

HAV belongs to the *Picornaviridae* family and is an RNA virus. It is particularly common in areas of the world with poor access to clean water and adequate sanitation. It is transmitted via faecal-oral route (via ingestion of contaminated food or water, or via close personal contact).[57–63]

Risk factors and epidemiology

- Non-immune travellers to areas with a high or intermediate prevalence,[64] GBMSM linked to oro-anal or digital-rectal contact, individuals with multiple sexual partners, anonymous partners, sex in public places and group sex[65–72]; people at occupational risk (e.g., sewage workers, laboratory personnel). More information for travel health can be found via the National Travel Health Network and Centre website (https://nathnac.net).
- Higher HAV viral load and more protracted HAV viraemia may occur with HIV, which may prolong faecal shedding and increase the risk of HAV transmission to others.[73–75]
- People who experience homelessness; migrants; refugees; incarcerated persons.[76]
- People with chronic liver disease and older patients are at higher risk of adverse outcomes.[77,78]
- Outbreaks have been reported amongst PWID,[79] in institutions for people with learning difficulties,[80] and following ingestion of contaminated food products e.g., shellfish, fruit and frozen berries.[81]
- The European Centre for Disease Prevention and Control (ECDC) reported that between June 2016 and June 2017, 1500 confirmed HAV cases and 2660 probable or suspected cases were reported in the European Union (EU), predominantly among adult MSM. The viruses identified belonged to three separate clusters based on genetic sequencing of HAV: Event 1-Cluster VRD_521_2016, Spain; Event 2-Cluster RIVM-HAV16-090 ('EuroPride strain') initially linked to men who have sex with men who participated in the EuroPride festival in Amsterdam (23 July to 7 August 2016); Event 3 -Cluster V16-25801 ('Berlin Strain'), initially reported in Berlin in November and December 2016.[82]
- A total of 797 confirmed or probable cases were identified in England and Wales associated with the 2016 to 2018 outbreak in men who have sex with men. This incident was declared over in June 2018. A number of incidents of the outbreak spilling over into the community were also identified.[57]
- In 2019, 503 laboratory confirmed cases of HAV infections were reported in England and Wales, with the greatest proportion (31.8%) from the London region.[57]
- In 2021, ECDC reported 3864 cases of HAV, with a notification rate of 0.9 cases per 100,000 population, which is the lowest number of reported case and notification rate since the beginning of EU surveillance in 2007. This was most likely due to the impact of the COVID19 pandemic, with reduced international travel, restaurant closures, and limited gatherings and social interactions.

Acute HAV

- People with acute HAV may present to sexual health services. The incubation period can range from 15 to 45 days (average 28 days).
- People with acute HAV are most infectious for approximately 2 weeks before jaundice onset and 1 week after the period of jaundice.
- Up to half of adults are asymptomatic or have mild non-specific symptoms with little or no jaundice. The prodromal illness is characterised by flu-like symptoms (i.e., malaise, myalgia, fatigue), often with right upper abdominal pain. This phase lasts for 3–10 days. The icteric illness follows, which is characterised by jaundice, anorexia, nausea and fatigue. The icteric phase usually lasts for 1–3 weeks. It can persist for 12 or more weeks in a minority who have cholestatic symptoms (pruritus and jaundice).[83]
- Over 99% of people with HAV have full resolution of infection and immunity is presumed to be lifelong. Individuals may develop severe hepatitis during acute infection requiring hospitalisation but acute liver failure (ALF) is extremely rare and the overall mortality rate is <0.1%.[83,84]

- HAV infection in pregnancy does not have any teratogenic effects but there is an increased rate of miscarriage and premature labour, proportional to the severity of the illness.[85,86]
- Laboratory tests for suspected acute HAV should include serum HAV-IgM which usually remains positive for 45–60 days.[87,88] HAV-IgG does not distinguish between current or past infection and may remain positive for life.[87] Liver function tests and clotting studies should be performed and results discussed with Hepatology colleagues. All individuals with suspected acute hepatitis should also be tested for other pathogens including (but not limited to) acute HBV, HCV and HEV.
- Most people with acute HAV can be managed as an outpatient, emphasising rest and oral hydration.[84] Severe disease with vomiting, dehydration or signs of hepatic decompensation (change in conscious level or personality) requires hospital admission with specialist Hepatology input.[84,89] (**1A**)
- Information should be provided with advice on minimising transmission to contacts.
- People are most infectious for 2 weeks before the onset of jaundice or other symptoms consistent with hepatitis if no jaundice (i.e., before the illness is recognised).[24]
- Sexual intercourse and food-handling should be avoided while infectious (from 2 weeks before to 1 week after the onset of jaundice). (**1B**) Employment should be considered.
- Acute HAV (and all forms of acute hepatitis) should be notified formally to the appropriate health protection team based on patient's postcode.
- Screening for other STIs and assessing for other sexual health need (e.g., HIV PrEP) is recommended.[71]
- Pregnancy and Breast Feeding: pregnant women should be advised of the increased risk of miscarriage/premature labour and the need to seek medical advice if this happens.[64,85,86](**1B**) The risk from breast feeding is uncertain although there are no reported cases of HAV transmission from breast milk. Even if the infant acquires HAV, the disease is normally mild or asymptomatic. Therefore, the balance of risks between infection and stopping breast feeding should be considered on an individual basis. (**2C**)
- Partner notification should be performed for at-risk sexual contacts (i.e., oro/anal, digital/rectal and penetrative anal sex) within the period 2 weeks before, until 1 week after, the onset of jaundice. (**1D**) This should be recorded and the outcome documented at subsequent follow-up visits. Other at-risk individuals (e.g., household contacts, those at risk from shared food/water contamination) should be contacted via public health authorities.[90] (**1D**)
- For management of contact with acute HAV see section 7.3

Hepatitis B virus infection

New in hepatitis B section

- Updated HBV epidemiology.
- New recommendation to offer screening for chronic HBV infection to all adults attending sexual health services at least once.
- Expansion of targeted HBV vaccination to prevent morbidity and mortality from future HBV acquisition.
- Inclusion of novel HBV vaccines.
- Guidance on use of tenofovir-containing HIV PrEP in people with chronic HBV.
- Shortened section on HBV staging and management.

Background

- HBV is a hepadna deoxyribonucleic acid [DNA] virus. It is endemic worldwide with variable prevalence (see Figure 1). In high prevalence regions, such as Southeast Asia, China and Sub-Saharan Africa, up to 8% of the population have chronic HBV.[10,11,91]
- HBV continues to be an important global cause of mortality, with 820,000 deaths attributed to HBV in 2019, predominantly from HCC and cirrhosis.[92] In 2019, an estimated 296 million people (3.8%) were living with chronic HBV globally, with an incidence of 1.5million new infections annually.[92] In the EU/EEA there were approximately 3.6 million people living with chronic HBV infection in 2024.[93]
- In May 2016, the UK signed up to the World Health Organisation (WHO) Global Health Sector Strategy on Viral Hepatitis committing to meet targets that include a 90% reduction in incidence of hepatitis B infection and a 65% reduction in mortality from hepatitis B by 2030 from a 2015 baseline.[91]
- In England there are an estimated 270,000 people living with chronic HBV, which equates to approximately 0.6% of the population, increasing to 1.5% in London. UKHSA figures demonstrate that over 95% of cases are amongst migrant populations, with infections acquired abroad. They may not present to services until they have advanced liver disease or attend for antenatal care.[94]
- Other groups at higher risk include GBMSM, sex workers, PWID and people detained within prisons.[94]
- Of those with newly diagnosed HBV infection in England (1999–2021), 57% were in men. The proportion aged 34 years and over increased from 41.2% in 1999 to 63.7% in 2021.[94]
- The cascade of care for HBV in the UK (from testing, diagnosis to treatment) remains poorly understood and work is underway to estimate the proportion of

people with chronic HBV that are unaware of the infection.[94]

- Vaccination is the cornerstone of control of HBV in adult risk group populations and improved vaccine implementation, and uptake is required in prisons, sexual health services for GBMSM, and drug services. The Green Book: Immunisation against Infectious Disease recommends HBV vaccination in '*people who change sexual partners frequently, GBMSM and sex workers*'.[91]
- At sexual health clinics, rates of HBV vaccination coverage in GBMSM fell from 95% in 2008 to below 20% (varying between clinics) of non-immune first-time attendees receiving first dose HBV vaccination in 2019 based on Genitourinary Medicine Clinic Activity Dataset (GUMCAD) data. These rates fell further during COVID-19 pandemic.[94]
- Between 2015 and 2021 an average of 350 acute HBV infections were reported annually to UKHSA (range 175 to 457). Since 2015, cases of acute hepatitis B in England have continued to decrease.[94] In 2022, 213 cases of acute hepatitis B were reported across England (confirmed, probable and possible). Where information was provided, the most likely route of transmission was heterosexual then GBMSM.[95]
- In 2022 a programme funded by National Health Service (NHS) England began opt-out testing for blood borne viruses (HIV, HBV, HCV) in selected Emergency Departments in areas of very high HIV diagnosed prevalence across England. In the first 2 years, the highest number and proportion of new diagnoses was for HBV (1957 new HBV diagnoses). Similar to diagnosis in other settings, new HBV diagnoses were higher in men than women. A greater proportion of new HBV diagnoses through the programme were among people living in the most deprived quintiles compared to people diagnosed in other settings.[96]
- 2023 Centers for Disease Control and Prevention (CDC) recommendations include testing all adults over 18 years for chronic HBV at least once in their lifetime.[97]

Transmission

HBV is transmitted by parenteral or mucosal exposure to HBV infected blood or body fluids.[91]

Transmission mostly occurs:

- Through perinatal transmission or more rarely in utero.[92] 95% of chronic HBV infections in the UK occur in migrant populations and were acquired perinatally or during childhood in endemic countries.[94] Infection during early childhood (perinatal or <5 years old) leads to chronic infection more frequently than at older ages.[92,98]
- Through vaginal or anal sex.[25,26,28,30,94,99]
- As a result of blood-to-blood contact through percutaneous exposure (e.g., sharing needles, syringes and other paraphernalia; occupational needlestick injuries; transfusion of unscreened blood products; and non-sterile acupuncture, piercings and tattoo needles.[11,94,95]

Other transmissions:

- Sporadic horizontal transmission can occur between non-sexual contacts in people without apparent risk factors, e.g., within institutions for people with learning difficulties; children in countries of high background prevalence; or in household contacts. In these cases, the mode of transmission is poorly understood but may involve transmission via small cuts and open wounds, sharing of dental hygiene products or bites.[11,94,100,101]

Incubation period and window period

- Incubation period (time to appearance of symptoms) 40–160 days.[11]
- Window period (time to positive HBsAg test): Usually 30–60 days.[11,93,94]
- The virus can survive outside of the body for at least 7 days.[92]

Acute HBV

- Acute infection is asymptomatic in up to 70% of adults. Virtually all infants and children have asymptomatic acute infection.[83,102–104]
- During the prodromal phase symptoms are often subclinical or non-specific and include a flu-like illness, anorexia and nausea. Approximately 2 weeks later the icteric phase (jaundice with pale stools and dark urine) occurs in 30%–50% of adults, and in less than 10% of children. Liver enlargement and tenderness are common. This phase may last up to 3 months.
- ALF occurs in less than 1% of symptomatic cases of acute HBV and is associated with a poorer prognosis than if caused by HAV.[83] It may present with worsening coagulopathy, encephalopathy, and cerebral oedema. There is an increased rate of miscarriage or premature labour during acute HBV. In acute HBV during pregnancy, the risk of transmission ranges from 10 to 60% depending on pregnancy trimester, with greatest risk of transmission with acute HBV infection around the time of delivery.[86]

- Laboratory tests for suspected acute HBV should include HBV serology including HBsAg. Liver function tests and clotting studies should be performed and results discussed with Hepatology.
- Patients with mild/moderate acute HBV can often be managed as an outpatient with rest and oral hydration. Where patients have severe hepatitis or evidence of ALF, liaise with Hepatology specialist for ongoing management.
- Patients with acute HBV should be seen at one or two weekly intervals until transaminases are normal (usually 4–12 weeks). Arrangements should be decided locally in discussion with Hepatology. Serology should be repeated after 6 months even if the LFT are normal to assess for chronic HBV. Immunity after recovery from infection (HBsAg negative, anti-HBc positive/negative, anti-HBs positive) is life-long in over 90%.[103,104]
- Acute HBV infection is a clinically notifiable disease (seropositivity for HBV is reported upon laboratory testing, regardless of acute or chronic as HBV is a notifiable organism too).[11]
- Acute HBV partner notification should be performed and documented, and the outcome documented at subsequent follow-up. Contact tracing should include any sexual contact (penetrative vaginal or anal sex or oro/anal sex) or needle sharing partners during the period in which the index case is thought to have been infectious.[105,106] (**1D**) The infectious period is from 2 weeks before the onset of jaundice until the patient becomes surface antigen negative. In index cases without an episode of jaundice, assess the risk to estimate when infection might have occurred, and use that period to guide partner notification.[105]

Chronic HBV

- The persistence of HBsAg in serum/plasma for 6 months or more after acute infection.[11] This occurs in 5%–10% of HBV infections acquired in adulthood.[103]
- There is a higher risk of chronic infection seen in immunocompromised patients (e.g., people with HIV, renal insufficiency, taking immunosuppressive medicines).[11,107,108]
- Over 90% of infants born to hepatitis B 'e' antigen [HBeAg] positive women will develop chronic HBV infection unless immunisation is commenced immediately at birth and infant immunisation schedule completed.[11,86] The likelihood of developing chronic HBV infection decreases with increasing age in childhood.[91]
- Around 20%–25% of individuals with chronic HBV infection worldwide have progressive liver disease, leading to cirrhosis in some people.

Chronic HBV: Phases of Infection

Chronic HBV infection represents the balance between viral replication and host response, with active liver inflammation (hepatitis) not occurring in all states. The natural history of chronic infection is divided into five phases, taking into account both viral and host factors, such as the presence of antigens, antibodies and transaminase levels. Progression through the phases is non-linear and can be non-sequential. More detailed information is available in the EASL guidelines.[1]

- In HBeAg positive infection (phases 1 and 2), there are high levels of HBV DNA detectable, and the presence or absence of raised transaminases gives an indication of associated inflammation, or disease. These phases are extended in perinatally acquired and childhood infections.
- Spontaneous seroconversion to HBeAg negative infection (phases 3 and 4) occurs at a rate of around 10%–15%/year. During these phases, there is less HBV DNA detected, and transaminase levels are lower, but the level of active inflammation differentiates HBeAg negative infection (phase 3) from HBeAg negative disease (phase 4). The latter is associated with progression to cirrhosis, and monitoring of alanine transaminase (ALT) and DNA levels are essential.
- Finally, in some people who have cleared HBsAg and remain anti-HBc positive, there is HBV DNA present in the liver and serum (phase 5). Known as occult HBV infection, there is a risk of reactivation particularly in those with severe immunosuppression from other conditions, or during administration of chemotherapy, which may lead to ALF. Another group has detectable serum/plasma HBV DNA, usually at low level (<200 IU/ml) and detection is often intermittent. Anti-HBc is usually positive. In a subset with S gene or other mutations, altered HBsAg is produced which is not recognised by some assays but HBV DNA levels are high.[109,110]

Progression risk to cirrhosis and of HCC

- Progression to cirrhosis may occur during the HBV phases associated with inflammation with an annual incidence rate of 8%–20%.
- HCC may complicate chronic HBV infection at any stage, but most commonly after development of cirrhosis. It is estimated there is an annual incidence rate of 2%–5% in HBV patients with cirrhosis.

- Host and viral factors associated with a higher risk of developing HCC include older age, male sex, African origin, alcohol excess, diabetes, smoking, HBV genotypic mutations and coinfection with hepatitis C, hepatitis D, or HIV.[1,107,108]

Co-infections with chronic HBV

Acute HAV (see chapter 8)

- Acute HAV can be severe in patients with chronic HBV.[111]

HCV infection (see chapter 11)

- Concurrent HCV and HBV infections can be associated with increased hepatitis severity and greater risk of cirrhosis and liver cancer.[112–114]

HDV infection (see chapter 10)

- HDV co-infection or superinfection is associated with hepatitis of increased severity, and may be associated with more rapidly progressive fibrosis, cirrhosis and end-stage liver disease.
- HDV is likely under diagnosed, but may be identified in 5% of adults with chronic HBV globally.[92]
- Between 10 and 50% of chronic HBV and HDV carriers will develop cirrhosis
- 10% or more of cirrhotic patients will progress to liver cancer.
- Premature death occurs in approximately 50%.[114–117]

HIV

- Concurrent HIV increases the risk of HBV fibrosis progression and of both liver-related and all-cause mortality.[108,118]

HBV: Screening in sexual health settings

Table 3 presents the available serological and biochemical tests for HBV.

Who to screen for HBV in sexual health settings?

- Previous BASHH guidelines advised a risk-factor based approach to screening for chronic HBV infection in sexual health clinic attendees.[3]
- National Institute for Health and Care Excellence (NICE) guidelines (2013) recommend sexual health and genitourinary medicine clinics should offer and promote HBV and HCV testing to all service users at increased risk of infection, including people younger than 18.[103]
- NHS England have begun opt-out testing for blood borne viruses (HIV, HBV, HCV) in selected Emergency Departments in areas of very high HIV prevalence across England. Within 2 years, the highest number of new diagnoses identified was for HBV. New HBV diagnoses were higher in men, people aged 35–64 years and in people of black African ethnicity.[95]
- A recent retrospective study of general practice medical records in England found associations of HBsAg positivity with lower area deprivation index, being from high HBV prevalence country, GBMSM sexual risk group, close HBV contacts, people with a history of injection drug use or a recorded diagnosis of HIV, HCV, or syphilis.[119]
- Modelling studies based on a United States (US) model suggest HBV testing is cost-effective in adults seeking care for STIs.[120]
- The CDC now recommend all adults>18 years are tested for chronic HBV at least once in their lifetime.[97]

Table 3. Serological and biochemical tests for HBV.

			Serological and biochemical tests									
			HBsAg	HBeAg	IgM Anti-HBc	IgG Anti-HBc	HBV DNA (PCR)	Anti-HBe	Anti-HBs	ALT	Liver disease/ necroinflammation	Relative infectivity
Stage of infection	Acute	Early	+	+	+	+	+	-	-	+++	Moderate to severe. Fulminant in ALF	+++
		Resolving	+	-	+	+	-	+/–	-	++	Moderate/resolving	++
	Chronic	Phase 1	+	+	-	+	$>10^7$	-	-	N	None/minimal	+++
		Phase 2	+	+	-	+	10^4–10^7	-	-	+	Moderate/severe	+++
		Phase 3	+	-	-	+	<2000	+	-	N	None	+
		Phase 4	+	-	-	+	>2000	+/–	-	+	Moderate/severe	++
Immune	Phase 5 - past infection		-	-	-	+	<10	+/–	+/–	N	None	-
	Vaccinated		-	-	-	-	-	-	+	N	N/A	Immune

ALF, Acute liver failure; ALT, Alanine transaminase; Anti-HBc, Antibody against hepatitis B core antigen; Anti-HBe, Antibody against hepatitis B 'e' antigen; Anti-HBs, Antibody against hepatitis B surface antigen; DNA, Deoxyribonucleic Acid; HBeAg, Hepatitis B 'e' antigen; HBsAg, Hepatitis B surface antigen; HBV, Hepatitis B virus; IgG, Immunoglobulin type G; IgM, Immunoglobulin type M; N/A, Not applicable.

- Due to the opportunity to identify HBV infections and link people to care preventing progression to chronic liver disease, we now recommend that all sexual health clinic attendees should be tested at least once for chronic HBV infection.
- It is acknowledged that this recommendation may have financial implications for sexual health services.

Which tests to perform for chronic HBV screening and immunity status?

- HBsAg
- Anti-HBc and anti-HBs titre to assess immunity only if required. (**1B**) Routine assessment in vaccine-naive immunocompetent adults not required.[121–123]

How to manage the results?

- Offer vaccination if at ongoing risk. (**1A**)
- If HBsAg screening test is reactive then further serology is required for staging HBV infection including:
  - Anti-HBc – IgG and IgM.
  - HBeAg.
  - Antibody against hepatitis B 'e' antigen (anti-HBe).
  - HBsAb.
  - HBV DNA polymerase chain reaction (PCR).
- HBV testing algorithms for confirmatory testing of isolated anti-HBc (HBsAg negative, anti-HBc positive; anti-HBs<10mIU/ml) will vary in different laboratories and therefore discussion with the local virology laboratory colleagues about the testing algorithm for isolated anti-HBc is recommended. (**GPP**)[124] Where there is concern that the anti-HBc may be a false positive, anti-HBe can be helpful. A single HBV vaccine dose will induce anti-HBs if there has been past natural HBV exposure (amnestic response, measured 4 weeks after a single dose of HBV vaccine). If anti-HBs is still negative after a single booster, regard as non-immune and give a full course of vaccine. (**1C**)

HBV: Management

General advice

- People who are HBsAg positive (and/or HBV DNA positive) should be referred to a Hepatology service for ongoing management.
- They should be given detailed information about their condition, the implications on the health of themselves, and sexual partners, as well as about the routes of transmission.[92,125,126]
- Advice should be given to avoid unprotected sexual intercourse (including oro-anal and oro-genital contact), unless their partners have been successfully vaccinated (see below). (**1D**) They should not donate organs/semen/blood.[11,92,125,126] (**1B**) They should avoid drinking alcohol.
- Further information is available at https://www.nhs.uk/conditions/Hepatitis-B and via the British Liver Trust.[122,125]
- Partner notification for chronic HBV should attempt to trace contacts as far back as any episode of jaundice or to the time when the infection is thought to have been acquired although this may be impractical for periods of longer than 3 years.[106] (**1D**)
- Arrange screening for HBV of children who have been born to HBsAg positive women if the child was not vaccinated at birth[1,11,106] (**1C**) via primary care/Paediatrics depending on local pathways.
- For screening of other nonsexual partners who may be at risk, discuss with the public health authorities.[1,11] (**1C**)

Chronic HBV: Initial management in sexual health settings)- local pathways and responsibilities to be determined with Hepatology

- Partner notification and contact tracing (**1B**).
- Provide information on HBV, alcohol avoidance and transmission (**GPP**).
- Test for HIV and HCV infections (**1D**).
- Test for other sexual infections (**1D**).
- Vaccinate for HAV if non-immune (**1D**).
- Refer to Hepatology specialist for ongoing management, hepatitis delta virus (HDV) testing and hepatocellular carcinoma (HCC) surveillance (**1B**).[103,116]

Chronic HBV: Further management (usually via Hepatology specialist)

Goals of therapy

- To improve survival and quality of life by prevent disease progression to cirrhosis and reduce risk of HCC.
- To prevent vertical transmission, HBV reactivation, and prevent and treat HBV-associated extrahepatic manifestations.

Treatment

- The decision to treat depends on factors including serum ALT, HBV DNA level, and severity of associated liver disease. Treatment decisions should be made in conjunction with Hepatology specialists. See EASL and WHO guidelines for further information.[1,127]

HIV/HBV coinfection

- HIV treatment (ART) should be started as soon as possible, including tenofovir disoproxil fumarate (TDF) or tenofovir alafenamide (TAF) as part of ART, in order to suppress HBV replication and prevent HBV associated liver damage during treatment,[3,33] irrespective of CD4 count.
- There is a risk of hepatitis flare and decompensation if ART containing TDF/TAF are stopped.

Pregnancy and breastfeeding

- The management of pregnant women with HBV should be in conjunction with Hepatology.
- In the absence of intervention, vertical transmission occurs in 90% of pregnancies where the mother is HBeAg positive and in about 10% of HBsAg positive and HBeAg negative mothers.
- Most (>90%) infants acquiring HBV perinatally develop chronic HBV infection.[128]
- Infants born to women with HBV infection are vaccinated from birth. HBIG is also given in certain situations where the risk of transmission is deemed to be high such as a viral load during that pregnancy of ≥1,000,000 IU/ml.[11] (**1A**) This intervention reduces vertical transmission by 90%. See Green Book chapter 18 for criteria for HBIG in neonates.
- TDF monotherapy may be recommended for pregnant women at week 24 if HBV DNA >200,000 IU/ml or quantitative HBsAg>4 log10 IU/ml to reduce the risk of transmission of HBV to the baby (**1A**), and should continue up to 12 weeks following delivery.[129] HBV replication may increase immediately following pregnancy but is seldom associated with clinical consequences.
- Breastfeeding is not contraindicated in HBsAg positive women off-treatment, or on TDF-based treatment.

Chronic HBV: Monitoring and surveillance

- HCC surveillance is offered for some individuals with chronic HBV and should be discussed in a Hepatology specialist clinic. Risk factors for HCC in individuals without cirrhosis include: ethnicity, gender, age, family history of HCC and stage of infection.[130,131]
- PAGE-B score may be used in specialist clinics to assess the risk of progression to HCC.[131]

Tenofovir-containing PrEP and PEP for HIV infection in individuals with HBV[128,132]

- Tenofovir disoproxil/Emtricitabine or Tenofovir alafenamide/Emtricitabine may be used as HIV PrEP or as part of PEP when clinically indicated.
- Tenofovir-containing PrEP/PEP may be initiated while HBV serology results are pending.
- People found to be HBsAg positive should be referred to a Hepatology specialist service whilst remaining on tenofovir.
- People with HBV on tenofovir-containing PrEP are recommended to take daily NOT event-based or 'on-demand' dosing schedules.
- There is a risk of HBV rebound upon stopping tenofovir, and the management of stopping therapy should be discussed in advance with a Hepatology specialist.
- Further details can be found in BASHH/BHIVA PrEP and PEP guidance.

Hepatitis delta infection

HDV is a small incomplete RNA virus that can only be acquired in the presence of HBsAg carriage and is associated with a higher rate of fulminant hepatitis, progression to cirrhosis, and HCC.[133,134]

HDV: Epidemiology and transmission

- It is estimated that 5% of people with chronic HBV also have HDV infection globally.
- Transmission routes are similar to HBV and occurs through broken skin and contact with infected blood and blood products.
- HDV has an incubation period of 3–7 weeks, and acute illness may present with a non-specific flu like illness with associated transaminitis.[135] Chronic HDV is defined as the presence of HDV infection for greater than 6 months.[133]

HDV: Risk factors

HDV may occur in any person with HBV, but is observed at higher frequency in:

- People who have acquired HBV infection abroad.
- PWID and their sexual partners.
- Sex workers.
- People with HCV or HIV infection.

HDV should be suspected if:

- The acute hepatitis is severe.
- If there is a further acute hepatitis episode in patients with chronic HBV.
- If liver disease is rapidly progressing in chronic HBV and HBV is well-controlled.

HDV: Investigations

- All people with HBV should be screened for HDV using antibody against HDV (anti-HDV) test. Active

infection is confirmed via HDV RNA testing. Local pathways should agree whether this test should be performed in Sexual Health or Hepatology.

HDV: Management

- All people with HDV should be referred to Hepatology for ongoing management.
- Until recently, pegylated interferon alpha was the only treatment strategy available with a 20% virological response rate. Novel therapies are being developed following the research into the mechanism of HDV persistence. Bulevirtide (Hepcludex) was NICE approved for use in the UK in 2023, for selected patients who have not responded/cannot have pegylated interferon with compensated liver disease and significant fibrosis (METAVIR stage F2 or above or Ishak stage 3 or above. Bulevirtide is an entry inhibitor of the HBsAg and HDV into hepatocytes.[136] Compared to standard care, bulevirtide resulted in significantly improved virological response at 48 weeks with continuous treatment, with ongoing benefit seen at 96 weeks.[135]

Hepatitis C infection

New in hepatitis C section

- Updated HCV epidemiology.
- Updated information on management of recently acquired HCV.

Hepatitis C: Background and epidemiology

- HCV is a single stranded RNA virus in the family *Flaviviridae.*
- HCV is endemic worldwide and, in 2020, an estimated 57 million individuals were living with active HCV, with a global prevalence of 0.7% 95% UI 0.7–0.9. Prevalence varies by region, with the highest prevalence in Eastern Europe (2.9% [95% UI 2.3–3.2]).[137]
- In 2022 in England, approximately 62,600 (95% credible interval (CrI) 48,900–77,800) were estimated to be living with chronic HCV infection in 2022, a 51.6% decrease from 2015. This is equivalent to a prevalence of 0.14% (95% CrI 0.11–0.17%) The reduction was predominantly driven by the roll out of highly effective, oral direct acting antiviral (DAA) therapies which provide cure rates of ~95%.
- This has led to significant declines in the incidence of HCV-related end stage liver disease and HCC.[138] In 2022, HCV-related deaths in England were 0.44 per 100,000 population.
- In 2016, The World Health Assembly proposed the elimination of HCV as a public health threat by 2030. Updated 2030 global targets aim to reduce the number of new HCV cases to 350,000 (5 per 100,000) and the number of people dying from HCV to 40,000 deaths (2 per 100,000).[139,140]
- A proxy measure set is the reduction in HCV viraemia prevalence by 80% from 2015 baseline (in general population and PWID). In 2022 in England this figure was 51.6% in general population and 60.8% in PWID.
- Achievement and maintenance of HCV elimination is likely to require early diagnosis and access to treatment for all first and re-infections, as well as additional preventive measures, both in the UK and internationally.
- Around 4% of people who have been treated for HCV have a re-infection.
- There are eight currently recognised HCV genotypes, which are further divided into subtypes, and in some cases may show differences in susceptibility to different DAA regimens.[141] Most (~90%) UK infections are caused by subtypes 1a or 3a.[138]

HCV: Transmission

Parenteral transmission

- Parenteral spread accounts for most HCV cases and the majority of people with HCV in the UK report a history of injection drug use. Transmission usually occurs through sharing needles or injecting equipment.[138]
- Other parenteral routes include:
  - Sharing personal hygiene items which have come into contact with blood such as razors.
  - Tattoos or body piercings in an unregulated setting.
  - Blood transfusions and organ transplants in the UK before 1992.
  - Healthcare related transmissions such as re-use of needles or haemodialysis.
  - Occupational exposure such as needlestick injury.[138]
- In many low and middle income countries, iatrogenic transmission remains a major HCV transmission route.[142]

Sexual transmission

- Amongst monogamous heterosexual couples without HIV, sexual transmission is extremely rare (<0.1%/year)[143–147] but this risk increases in the context of HIV coinfection.[148]
- Amongst GBMSM with HIV and without a history of injection drug use, the prevalence of anti-HCV is greater (4.1%) compared to the general population[108] and an increased risk of HCV sexual transmission has been described.[148,149]

- A global epidemic of recently acquired HCV was described amongst GBMSM with HIV from 2000 onwards, driven by mucosal and parenteral transmissions. Specific risk factors were identified, both behavioural (receptive condomless anal sex, traumatic sex, sex in a group environment, sharing sex toys, use of recreational drugs [chems] during sex, and seroadaptive behaviours) and biological (concurrent STIs, especially ulcerative conditions such as LGV and syphilis).[150–155]
- Chems may be injected (slamming) or non-injected, and may cause loss of attention to safer sex, mucosal exposure such as from nasal administration and/or parenteral exposures.[155] Chems include methamphetamine (crystal), mephedrone, gamma-hydroxybutyric acid/gamma-butyrolactone.
- The epidemic has also been characterised by a high incidence of HCV reinfection (3.8/100 person-years (95% CI 2.8–5.1).[156]
- However, data from the UK and internationally suggest a declining incidence of recently-acquired HCV in GBMSM with HIV since 2016, likely driven by expanded DAA access.[157–159]
- Sexual transmission of HCV may also be more frequent amongst HIV-negative GBMSM groups who share risk factors with HIV-positive GBMSM and are involved in the same HCV transmission networks, including those eligible for or using HIV PrEP.[149,160,161]
- Amongst HIV negative GBMSM overall, HCV prevalence is only slightly higher than in the general population. HCV diagnosis rates in English Sexual Health clinics were 113/100,000, 24/100,000 and 12/100,000 in HIV-positive GBMSM/HIV-negative GBMSM/all attendees, respectively.[138,149]
- There are currently insufficient data on HCV incidence in transgender persons.

Vertical transmission

- HCV vertical transmission is estimated at 5%–7%, increasing to 10%–12% for women with HIV and HCV coinfection.[162,163]
- Transmission risk has reportedly been correlated with maternal HCV RNA level in blood.[164–167]
- It is safe for a mother with hepatitis C to breastfeed her infant. There is no documented evidence that breastfeeding spreads HCV, but caution required if cracked/bleeding nipples. See CDC Guidance for further information.[168]

Other groups with increased HCV transmission. Compared to the general population, an increased prevalence of HCV infection has been reported in:

- People who have been in prison.[138,169]
- People who have exchanged sex for money, goods or services.[29,170]
- People who have experienced homelessness.[171]
- People with links to countries where HCV is endemic.[138]
- People who snort/inhale recreational drugs.[172,173]
- People with high levels of alcohol consumption.[174]

However, amongst UK blood donors, 25% of individuals with HCV did not report any risk factors.[175]

HCV: Incubation period

- The HCV incubation period is 4–20 weeks. HCV antibody conversion may take 12 weeks or more. However, HCV RNA will usually be detectable within 2 weeks.[137,138,141–145]
- HCV antibody seroconversion may be delayed in people living with HIV, particularly in those with a low CD4 count (<200 cells/μL). HCV antibody production may rarely be absent in these individuals, or in other immunocompromised people.[142,143]
- Spontaneous viral clearance is estimated to occur in occurs in up to 37% of individuals (24.4% of PWID and 15.4% of GBMSM living with HIV). Other factors affecting clearance rates include gender, age, ethnicity, presence of symptoms, HBV coinfection and HCV genotype. The variation in estimates may relate to both the impact of HIV on the natural history of HCV and the relative rates of HCV reinfection.[176]

HCV: Symptoms

The majority (>60%) have asymptomatic infection or non-specific symptoms.[177]

HCV: Who to test for HCV in sexual health settings?

Table 4 presents a summary of who to test for HCV in sexual health settings.

HCV screening and diagnostics: Which tests to perform?

- HCV antibody testing should be performed if the possible HCV risk exposure occurred more than 3 months ago. If HCV antibody is negative, a repeat should be considered at 6 months. It may take 3 months or more for the anti-HCV test to become positive after exposure, increasing to 6 months or more in people with HIV.[180]
- Where HCV antibody is positive, further testing for hepatitis C ribonucleic acid (HCV RNA) or hepatitis

Table 4. Who to test for HCV in sexual health settings.

Group	Notes
People with a past/current history of injection drug use. This includes people who injected only once. Injected drugs may be recreational, anabolic steroids, other image/performance enhancing drugs	Test every 3–6 months if ongoing IDU.
People with HIV	Test at least annually unless confirmation of no risk. Test every 3-6 months if high risk for acquiring HCV[a]
GBMSM with risk factors for HCV[a]	Test every 3–6 months if ongoing risk[a] Routine HCV screening in HIV-negative GBMSM is not recommended in the absence of HCV risk factors[a] (**1B**)
People newly diagnosed with HBV	
GBMSM newly diagnosed with syphilis or LGV	
People born in a country of intermediate or high HCV prevalence	Africa, asia, the Caribbean, Central and South America, Eastern and Southern Europe, the Middle East, and the Pacific islands[178]
People with a history of intranasal/inhaled recreational drug use	
People who have exchanged sex for money, goods or favours	
People who have shared personal items which may have come into contact with blood e.g. razors	
People who have experienced homelessness	
People who have been in prison or other secure/detained settings	
People with parenteral exposures in unregulated settings e.g. tattoos, piercings	
People with a possible healthcare-related exposure in a low- or middle-income country e.g. non-sterile needle use	
People with a history of haemodialysis	
Children born to women with viraemic HCV	
People who received a blood product or organ transplant in the UK before 1992 and are previously unscreened	
People reporting an occupational exposure e.g. needlestick injury from a source with viraemic HCV or of unknown HCV status	UKHSA guidance[179]
People with abnormal liver function tests	
People with symptoms or signs of acute hepatitis, such as jaundice	
Close contacts of people with viraemic HCV including sexual partners, also family members, close friends or household contacts[178]	

GBMSM, Gay, bisexual or other men who have sex with men; HBV, Hepatitis B virus; HCV, Hepatitis C virus; HIV, Human immunodeficiency virus; IDU, Injection drug use; LGV, Lymphogranuloma venereum; PrEP, Pre-exposure prophylaxis; UKHSA, UK Health Security Agency.
[a]Risk factors in GBMSM: condomless anal sex, chemsex, injection drug use, sex in a group environment, traumatic sex, sharing sex toys, individuals eligible for or using PrEP.

C core antigen (HCV Ag) should be performed to determine whether there is active infection. (**1A**)

- HCV Ag is a surrogate marker of HCV replication and can be used instead of HCV RNA to diagnose acute or chronic HCV infection. HCV Ag assays are less sensitive than HCV RNA assays (lower limit of detection equivalent to approximately 500 to 3000 HCV RNA IU/ml, depending on HCV genotype.[138,141,181,182]
- HCV Ag or HCV RNA testing can be performed in cases where possible HCV risk exposure occurred less than 6 months ago to allow earlier detection. (**1A**)

- Where there is a high suspicion of recently acquired HCV and anti-HCV is negative, consider HCV RNA testing, with HCV Ag as an alternative.
- HCV Ag or HCV RNA testing should be performed to detect reinfection in people who have previously resolved HCV infection (spontaneously or through treatment). (**1A**)
- HCV Ag or HCV RNA testing should be considered for people with HIV and CD4 count <200 cells/μL and other immunocompromised people at risk for HCV, particularly where clinical suspicion remains despite negative antibody testing. (**2C**)
- Rapid diagnostic tests for HCV antibody using serum, plasma, fingerprick blood or saliva are available and may be useful in outreach settings and in individuals who decline venepuncture or are difficult to bleed.
- Dried blood spot (DBS) testing on fingerprick blood is an alternative to venepuncture and can be used to test for the presence of HCV antibody or RNA. Cartridge-based RNA point-of-care testing (POCT) is available and can be performed on fingerprick blood, serum or plasma. Self-sampling antibody testing can be undertaken as part of online sexual health screening using fingerprick blood.

HCV: Further investigations in sexual health services

HCV shares transmission routes with other blood borne viruses and therefore people with HCV are at increased risk for HBV and HIV exposure. People with HCV who acquire HBV or HAV are at greater risk of severe liver disease.

- HAV IgG testing should be performed in individuals with no history of HAV vaccination or previous infection, and HAV vaccination is recommended whilst awaiting results.
- All people with HCV should therefore be tested for HBsAg, anti-HBc and anti-HBs, and offered HBV vaccination if susceptible.
- HIV testing should be performed.
- An STI screen is recommended in people who may have acquired HCV sexually, including treponemal serology and chlamydia and gonorrhoea testing.

HCV: Management

- Individuals with active HCV infection should be referred to a Hepatology specialist service for further assessment and ongoing management.
- Bloods tests including liver function tests, renal function, full blood count and clotting studies should be performed.
- Assessment of liver fibrosis is recommended in all people with HCV as (1) this may inform antiviral treatment decisions and (2) individuals with advanced (>/=F3) fibrosis may require monitoring for complications of end stage liver disease and HCC.
- Liver ultrasound should be performed in people with active HCV and is particularly important in individuals with advanced (>/=F3) fibrosis to exclude hepatocellular carcinoma.
- In some settings, it may not be possible to assess liver disease stage, and this should not be a barrier to starting HCV treatment.

HCV: Treatment

- The primary goal of HCV treatment is cure, in order to prevent liver fibrosis, cirrhosis, decompensation of cirrhosis, HCC, extrahepatic manifestations and death.
- Secondary goals of HCV treatment are to improve quality of life and reduce stigma and to prevent onward transmission.
- HCV cure is defined by undetectable HCV RNA in serum or plasma 12 weeks after the end of treatment (SVR12, sustained virological response). In some settings, depending on resources, HCV Ag may be used as an alternative to HCV Ag RNA.
- All people with detectable HCV RNA should be considered for treatment with DAAs without delay, according to local pathways.
- Determination of genotype is currently recommended if available but does not limit access to treatment. Treatment with pan-genotypic regimens can be initiated without knowledge of the genotype and subtype with a high probability of success.
- A treatment history including prior HCV treatment, current prescribed medication, over-the-counter remedies and herbal supplements should be obtained in order to check for a small number of drug-drug interactions that may influence choice of regimen. The University of Liverpool website should be consulted for potential interactions: Liverpool HEP Interactions (hep-druginteractions.org).
- The same all-oral DAA regimens should be used in people living with HIV and HCV.

Recognising and managing recently acquired HCV

- Historically, HCV infection has been classified as acute (the first 6 months of infection) or chronic (absence of spontaneous clearance of HCV within the first 6 months of infection).
- Acute HCV is usually asymptomatic and sub-clinical. Precise timing of infection is therefore difficult to establish. The term ‘recently acquired’ hepatitis C is now

preferred and is defined by the presence of anti-HCV antibodies, HCV RNA and/or HCV Ag that were not detectable in previous samples up to 12 months prior. In practice, historical results are often unavailable and DAA treatment in the early phase of infection is thought to be both cost-effective and useful to achieve elimination. Most care pathways now adopt a 'test and treat' strategy with immediate treatment offered to all individuals with a positive HCV RNA or HCV Ag by point of care test or venepuncture.
- All patients with detectable HCV RNA or core antigen should be referred directly to their local specialist team without delay.

HCV: Partner notification and contact tracing

- Individuals diagnosed with HCV should preferably be referred to a sexual health advisor.
- Partner notification should be performed and documented. The outcome should be documented at subsequent follow-up.
- Contract tracing should include any contacts during the period in which the index case is thought to have had HCV viraemia. This should include sexual contacts reporting condomless sex and needle sharing partners.
- Sexual contacts with HIV should be advised of the increased risk of HCV transmission, with regular testing and condom use encouraged.[108,148]
- If the HCV infection was not known to be recently acquired, trace back to the likely time of infection (e.g., blood transfusion, first needle sharing) although this may be impractical for periods longer than two or 3 years.
- Screen contacts for evidence of past or current HCV infection. For contacts who were exposed within the window period (6 months), consider use of HCV RNA or core antigen testing.
- Test children born to women with HCV viraemia.[162,163] This can be arranged in conjunction with primary care or Paediatrics.
- For other non-sexual contacts thought to be at risk, consider on a case-by-case basis.
- Spontaneous resolution of HCV infection and previous successful treatment do not provide protection against re-infection if further HCV exposure occurs.[162,163]
- There is currently no vaccine or immunoglobulin which will prevent HCV transmission.
- Use of HCV DAA as PrEP or PEP is not currently recommended.

HCV: Advice for people with active HCV

- All people with detectable HCV RNA or core antigen should be given information on how to reduce the risk of onward transmission including avoiding sharing injecting equipment and personal hygiene items which may contain traces of blood such as toothbrushes and razors.
- People with HCV viraemia should be advised to avoid condomless vaginal or anal sex. Consistent condom use is likely to prevent sexual transmission. However, HIV-negative heterosexual individuals in monogamous relationships should be advised of very low rates of sexual HCV transmission.[143–148]
- GBMSM with HCV should be recommended to use condoms for anal sex, single person only sex toys/condoms on sex toys and changed between partners and recommended not to share lube and avoid group sex situations, given the risk of mucosal transmission from HCV RNA in semen, rectal fluid and blood.
- All people with HCV should be referred to specialist services for early treatment with DAA therapies.
- People with HCV should be reassured that HCV is curable.
- People with HCV should be advised not to donate blood, semen or organs and provided advice on other routes of transmission.
- People with HCV should be given a detailed explanation of their condition with particular emphasis on the long-term implications for the health of themselves and their partner(s). This should be reinforced by providing clear and accurate written information.
- As an acute infectious hepatitis, recently acquired HCV is a notifiable disease and should be reported to the local Health Protection Team.

HCV: Primary prevention

- GBMSM with HCV-acquisition risk factors should be given advice on safer practices including not sharing injecting equipment, safe disposal of syringes/needles, not sharing straws for nasally administered drugs, condom use for anal sex, not sharing anal douches or sex toys.
- All people reporting injection drug use should be offered referral to needle and syringe exchange schemes.
- Where harmful use of psychoactive substances is identified in people with or at risk of HCV, psychological and medical support should be offered, including opioid substitution therapy for opiate dependence and general harm reduction advice/support. Referral to specialist drug services may be indicated.

HCV: Reinfection

- Individuals with ongoing risk behaviours should be offered testing with HCV Ag or HCV RNA at least annually. (**1A**)
- People who are found to have HCV reinfection should be re-referred for treatment. (**1A**)
- Discuss risk reduction for HCV reinfection in all individuals with ongoing risk for acquisition. (**1D**)

- Use of HCV DAA PrEP or PEP is not currently recommended.

Auditable outcome measures

- Proportion of new attendees at sexual health clinic with risk factors for HAV-acquisition offered HAV vaccine/immunity check (target 90%).
- Proportion of new attendees at sexual health clinic with risk factors for HBV-acquisition offered HBV vaccine/immunity check (target 90%).
- In those offered HAV or HBV vaccination, completed vaccine course if non-immune (target 70%).
- Proportion of new attendees at sexual health clinic offered testing for HBV (target 90%).
- Proportion of individuals diagnosed with chronic HBV referred to Hepatology (target 90%) and evidence of attendance for HBV care (target 70%).
- Proportion of sexual health clinic attendees at risk of HCV infection being screened for chronic HCV (target 90%).
- Proportion of people testing anti-HCV positive undergoing HCV RNA or core antigen test for active infection (target 90%).
- Proportion of individuals with active HCV referred to Hepatology services for DAA therapy (target 90%) and evidence of attendance for HCV care (target 70%).

Resource implications of these guidelines

Recommendations for testing and prophylactic vaccination in this guideline may incur additional costs for Sexual Health clinics including laboratory tests, vaccination costs and additional clinic attendances.

Qualifying statement

The recommendations in this guideline may not be appropriate for use in all clinical situations. Decisions to follow these recommendations must be based on professional clinical judgement, consideration of individual patient circumstances and available resources.

All possible care has been undertaken to ensure specification of the correct dosage of medication and route of administration. However, it remains the responsibility of the prescribing clinician to ensure the accuracy and appropriateness of the medication they prescribe.

Review arrangements

An author group will be invited by the BASHH CEG to review and revise the guideline in 2030 using the BASHH framework for guideline development. However, addenda may be issued sooner than 2030, particularly if relevant new data are available relating to testing or treatment options.

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Editorial independence

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Membership of the clinical effectiveness group

Current membership of the BASHH Clinical Effectiveness Group is available at https://www.bashh.org/bashh-groups/clinical-effectiveness-group/.

Supplemental material

Supplemental material for this article is available online.

References

1. European Association for the Study of the Liver. EASL 2017 Clinical Practice Guidelines on the management of hepatitis B virus infection. *J Hepatol* 2017; 67: 370–398. https://doi.org/10.1016/j.jhep.2017.03.021
2. European Association for the Study of the Liver, Clinical Practice Guidelines Panel Chair and EASL Governing Board representative, et al. EASL recommendations on treatment of hepatitis C: final update of the series. *J Hepatol* 2020; 73: 1170–1218. https://doi.org/10.1016/j.jhep.2020.08.018
3. British Association for Sexual Health and HIV (BASHH). 2017 interim update of the 2015 BASHH National Guidelines for the Management of the Viral Hepatitides. https://www.bashh.org/_userfiles/pages/files/resources/viral_hepatitides_2017_update_181217.pdf (2017, accessed 28 January 2025).
4. Brook G, Brockmeyer N, van de Laar T, et al. 2017 European guideline for the screening, prevention and initial management of hepatitis B and C infections in sexual health settings.

Int J STD AIDS 2018; 29: 949–967. https://doi.org/10.1177/0956462418767576

5. Wilkins E, Nelson M, Agarwal K, et al. British HIV Association guidelines for the management of hepatitis viruses in adults infected with HIV 2013. *HIV Med* 2013; 14(Suppl 4): 1–71. https://doi.org/10.1111/hiv.12106
6. 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: 1560–1599. https://doi.org/10.1002/hep.29800
7. Bhattacharya D, Aronsohn A, Price J, et al. Hepatitis C guidance 2023 update: AASLD-IDSA recommendations for testing, managing, and treating hepatitis C virus infection. *Clin Infect Dis* 2023, Online ahead of print.
8. Geretti AM, Brook G, Cameron C, et al. British HIV Association Guidelines on the use of vaccines in HIV-positive adults 2015. *HIV Med* 2016; 17 Suppl 3: s2–s81. https://doi.org/10.1111/hiv.12424
9. UK Health Security Agency. Immunisation against infectious disease: the green book. https://www.gov.uk/government/collections/immunisation-against-infectious-disease-the-green-book (2013, updated 15 October 2024, accessed 27 January 2025).
10. GBD 2019 Hepatitis B Collaborators. Global, regional, and national burden of hepatitis B, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet Gastroenterol Hepatol* 2022; 7: 796–829. https://doi.org/10.1016/S2468-1253(22)00124-8
11. UK Health Security Agency. Hepatitis B: the green book, chapter 18. Hepatitis B immunisation information for public health professionals. https://www.gov.uk/government/publications/hepatitis-b-the-green-book-chapter-18 (2013, updated 14 August 2024, accessed 27 January 2025).
12. British HIV Association (BHIVA). British HIV Association guidelines on the use of vaccines in HIV-positive adults 2015, 2015. https://bhiva.org/wp-content/uploads/2024/10/2015-Vaccination-Guidelines.pdf
13. UK Health Security Agency. Hepatitis A: the green book, chapter 17. Hepatitis A immunisation information for public health professionals. https://www.gov.uk/government/publications/hepatitis-a-the-green-book-chapter-17 (2013, updated 15 January 2024, accessed 27 January 2025).
14. O'Riordan M, Goh L and Lamba H. Increasing hepatitis A IgG prevalence rate in men who have sex with men attending a sexual health clinic in London: implications for immunization policy. *Int J STD AIDS* 2007; 18: 707–710. https://doi.org/10.1258/095646207782193902
15. Le Turnier P, Charreau I, Gabassi A, et al. Hepatitis A and B vaccine uptake and immunisation among men who have sex with men seeking PrEP: a substudy of the ANRS IPERGAY trial. *Sex Transm Infect* 2023; 99: 140–142. https://doi.org/10.1136/sextrans-2022-055634
16. Chen GJ, Lin KY, Sun HY, et al. Incidence of acute hepatitis A among HIV-positive patients during an outbreak among MSM in Taiwan: impact of HAV vaccination. *Liver Int* 2018; 38: 594–601. https://doi.org/10.1111/liv.13468
17. Regan DG, Wood JG, Benevent C, et al. Estimating the critical immunity threshold for preventing hepatitis A outbreaks in men who have sex with men. *Epidemiol Infect* 2016; 144: 1528–1537. https://doi.org/10.1017/S0950268815002605
18. Theeten H, Van Herck K, Van Der Meeren O, et al. Long-term antibody persistence after vaccination with a 2-dose Havrix (inactivated hepatitis A vaccine): 20 years of observed data, and long-term model-based predictions. *Vaccine* 2015; 33: 5723–5727. https://doi.org/10.1016/j.vaccine.2015.07.008
19. Kourkounti S, Papaizos V, Leuow K, et al. Hepatitis A vaccination and immunological parameters in HIV-infected patients. *Viral Immunol* 2013; 26: 357–363. https://doi.org/10.1089/vim.2012.0100
20. Mena G, Garcia-Basteiro AL, Llupia A, et al. Factors associated with the immune response to hepatitis A vaccination in HIV-infected patients in the era of highly active antiretroviral therapy. *Vaccine* 2013; 31: 3668–3674. https://doi.org/10.1016/j.vaccine.2013.06.012
21. Van Damme P and Van Herck K. A review of the long-term protection after hepatitis A and B vaccination. *Travel Med Infect Dis* 2007; 5: 79–84. https://doi.org/10.1016/j.tmaid.2006.04.004
22. Nelson NP, Weng MK, Hofmeister MG, et al. Prevention of Hepatitis A virus infection in the United States: recommendations of the Advisory Committee on immunization practices, 2020. *MMWR Recomm Rep* 2020; 69: 1–38. https://doi.org/10.15585/mmwr.rr6905a1
23. Rendi-Wagner P, Korinek M, Winkler B, et al. Persistence of seroprotection 10 years after primary hepatitis A vaccination in an unselected study population. *Vaccine* 2007; 25: 927–931. https://doi.org/10.1016/j.vaccine.2006.08.044
24. UK Health Security Agency. Public health control and management of hepatitis A - 2024 updated guidance. https://assets.publishing.service.gov.uk/media/65ccdbbd1d93950012946677/Hepatitis-A-guidance-13-february-2024.pdf (2024, accessed 27 January 2025).
25. Gilson RJC, de Ruiter A, Waite J, et al. Hepatitis B virus infection in patients attending a genitourinary medicine clinic: risk factors and vaccine coverage. *Sex Transm Infect* 1998; 74: 110–115. https://doi.org/10.1136/sti.74.2.110
26. Struve J, Giesecke J, Lindh G, et al. Heterosexual contact as a major route for transmission of acute hepatitis B among adults. *J Infect* 1990; 20: 111–121. https://doi.org/10.1016/0163-4453(90)93288-4
27. Hyams KC, Phillips IA, Tejada A, et al. Hepatitis B in a highly active prostitute population: evidence for a low risk of chronic antigenemia. *J Infect Dis* 1990; 162: 295–298. https://doi.org/10.1093/infdis/162.2.295

28. Koff RS, Slavin MM, Connelly JD, et al. Contagiousness of acute hepatitis B. Secondary attack rates in household contacts. *Gastroenterology* 1977; 72: 297–300.
29. Ward H, Day S and Weber J. Risky business: health and safety in the sex industry over a 9 year period. *Sex Transm Infect* 1999; 75: 340–343. https://doi.org/10.1136/sti.75.5.340
30. Wu JC, Chen CM, Sheen IJ, et al. Evidence of transmission of hepatitis D virus to spouses from sequence analysis of the viral genome. *Hepatology* 1995; 22: 1656–1660.
31. Huo TI, Wu JC, Huang YH, et al. Evidence of transmission of hepatitis B virus to spouses from sequence analysis of the viral genome. *J Gastroenterol Hepatol* 1998; 13: 1138–1142. https://doi.org/10.1111/j.1440-1746.1998.tb00590.x
32. Palmović D and Crnjaković-Palmović J. Prevention of hepatitis B virus (HBV) infection in health-care workers after accidental exposure: a comparison of two prophylactic schedules. *Infection* 1993; 21: 42–45. https://doi.org/10.1007/BF01739311
33. Vogt TM, Perz JF, Van Houten CK Jr., et al. An outbreak of hepatitis B virus infection among methamphetamine injectors: the role of sharing injection drug equipment. *Addiction* 2006; 101: 726–730. https://doi.org/10.1111/j.1360-0443.2006.01407.x
34. Walsh B, Maguire H and Carrington D. Outbreak of hepatitis B in an acupuncture clinic. *Commun Dis Public Health* 1999; 2: 137–140.
35. Centers for Disease Control and Prevention (CDC). Haber P and Schillie S. Epidemiology and prevention of vaccine-preventable diseases. Chapter 10: Hepatitis B. https://www.cdc.gov/pinkbook/hcp/table-of-contents/chapter-10-hepatitis-b.html?CDC_AAref_Val=https://www.cdc.gov/vaccines/pubs/pinkbook/hepb.html, (09 May 2024, accessed 27 January 2025).
36. National Institute for Health and Care Excellence (NICE). Hepatitis B and C testing: people at risk of infection. Public health guideline [PH43]. https://www.nice.org.uk/guidance/ph43/chapter/Recommendations#recommendation-2-awareness-raising-for-people-at-increased-risk-of-hepatitis-b-or-c-infection (2012, updated 01 March 2013, accessed 28 January 2025).
37. de Silva TI, Green ST, Cole J, et al. Successful use of Fendrix in HIV-infected non-responders to standard hepatitis B vaccines. *J Infect* 2014; 68: 397–399. https://doi.org/10.1016/j.jinf.2013.12.009
38. Hoebe CJ, Vermeiren AP and Dukers-Muijrers NH. Re-vaccination with Fendrix® or HBVaxPro® results in better response rates than does revaccination with three doses of Engerix-B® in previous non-responders. *Vaccine* 2012; 30: 6734–6737. https://doi.org/10.1016/j.vaccine.2012.08.074
39. Kundi M. New hepatitis B vaccine formulated with an improved adjuvant system. *Expert Rev Vaccines* 2007; 6: 133–140. https://doi.org/10.1586/14760584.6.2.133
40. Pichichero ME. Improving vaccine delivery using novel adjuvant systems. *Hum Vaccin* 2008; 4: 262–270. https://doi.org/10.4161/hv.4.4.5742
41. Hyer RN and Janssen R. 2287. Recently approved HEPLISAV-B(R) [Hepatitis B vaccine (recombinant), adjuvanted] Shows a higher proportion of subjects achieving seroprotection with a more consistent immune response compared with Engerix-B(R) [Hepatitis B vaccine (Recombinant)] in three comparative trials. *Open Forum Infect Dis* 2018; 5: S677–S678. https://doi.org/10.1093/ofid/ofy210.1940
42. Marks KM, Kang M, Umbleja T, et al. Immunogenicity and safety of Hepatitis B Virus (HBV) vaccine with a toll-like receptor 9 agonist adjuvant in HBV vaccine-naive people with human immunodeficiency virus. *Clin Infect Dis* 2023; 77: 414–418. https://doi.org/10.1093/cid/ciad201
43. Schnittman S, Zepf R, Cocohoba J, et al. 21. Current and Nadir CD4+ counts are associated with Heplisav-B seroprotection rates in people with HIV. *Open Forum Infect Dis* 2020; 7: S33–S35. https://doi.org/10.1093/ofid/ofaa439.066
44. van der Sande MA, Mendy M, Waight P, et al. Similar long-term vaccine efficacy of two versus three doses of HBV vaccine in early life. *Vaccine* 2007; 25: 1509–1512. https://doi.org/10.1016/j.vaccine.2006.10.023
45. Wistrom J, Ahlm C, Lundberg S, et al. Booster vaccination with recombinant hepatitis B vaccine four years after priming with one single dose. *Vaccine* 1999; 17: 2162–2165. https://doi.org/10.1016/s0264-410x(99)00012-2
46. Fonseca MO, Pang LW, de Paula Cavalheiro N, et al. Randomized trial of recombinant hepatitis B vaccine in HIV-infected adult patients comparing a standard dose to a double dose. *Vaccine* 2005; 23: 2902–2908. https://doi.org/10.1016/j.vaccine.2004.11.057
47. Overton ET, Sungkanuparph S, Powderly WG, et al. Undetectable plasma HIV RNA load predicts success after hepatitis B vaccination in HIV-infected persons. *Clin Infect Dis* 2005; 41: 1045–1048. https://doi.org/10.1086/433180
48. Paitoonpong L and Suankratay C. Immunological response to hepatitis B vaccination in patients with AIDS and virological response to highly active antiretroviral therapy. *Scand J Infect Dis* 2008; 40: 54–58. https://doi.org/10.1080/00365540701522975
49. UK Health Security Agency. Human hepatitis B specific immunoglobulin for hepatitis B post-exposure prophylaxis. https://www.gov.uk/government/publications/immunoglobulin-when-to-use/hepatitis-b-immunoglobulin-issued-march-2021 (updated 7 November 2024, accessed 07 January 2025).
50. Zuckerman JN. Review: hepatitis B immune globulin for prevention of hepatitis B infection. *J Med Virol* 2007; 79: 919–921. https://doi.org/10.1002/jmv.20816

51. Ahmed SM, Volpellier M and Forster G. The use of the super accelerated hepatitis B vaccination regimen in a north London sexual assault referral centre (SARC). *J Forensic Leg Med* 2007; 14: 72–74. https://doi.org/10.1016/j.jcfm.2006.02.018
52. Baral S, Sherman SG, Millson P, et al. Vaccine immunogenicity in injecting drug users: a systematic review. *Lancet Infect Dis* 2007; 7: 667–674. https://doi.org/10.1016/S1473-3099(07)70237-2
53. Connor BA, Blatter MM, Beran J, et al. Rapid and sustained immune response against hepatitis A and B achieved with combined vaccine using an accelerated administration schedule. *J Travel Med* 2007; 14: 9–15. https://doi.org/10.1111/j.1708-8305.2006.00106.x
54. Kallinowski B, Jilg W, Buchholz L, et al. Immunogenicity of an accelerated vaccination regime with a combined hepatitis a/b vaccine in patients with chronic hepatitis C. *Z Gastroenterol* 2003; 41: 983–990. https://doi.org/10.1055/s-2003-42929
55. Keystone JS and Hershey JH. The underestimated risk of hepatitis A and hepatitis B: benefits of an accelerated vaccination schedule. *Int J Infect Dis* 2008; 12: 3–11. https://doi.org/10.1016/j.ijid.2007.04.012
56. Mast EE, Weinbaum CM, Fiore AE, et al. A comprehensive immunization strategy to eliminate transmission of hepatitis B virus infection in the United States: recommendations of the Advisory Committee on Immunization Practices (ACIP) Part II: immunization of adults. *MMWR Recomm Rep* 2006; 55: 1–33; quiz CE31-34.
57. Public Health England. Laboratory reports of hepatitis A infections in England and Wales, 2019. https://assets.publishing.service.gov.uk/media/608c22e38fa8f51b9988cc42/hpr0715_HAV19_v2.pdf (2021, accessed 27 January 2025).
58. Roberts RJ and Palmer SR. Exposure to school children as a risk factor in a community outbreak of hepatitis A in young adults: a case control study. *Epidemiol Infect* 2006; 134: 803–807. https://doi.org/10.1017/S0950268805005625
59. Botelho-Nevers E and Gautret P. Outbreaks associated to large open air festivals, including music festivals, 1980 to 2012. *Euro Surveill* 2013; 18: 20426. https://doi.org/10.2807/ese.18.11.20426-en
60. Martin A and Lemon SM. Hepatitis A virus: from discovery to vaccines. *Hepatology* 2006; 43: S164–S172. https://doi.org/10.1002/hep.21052
61. Rowe SL, Tanner K and Gregory JE. Hepatitis A outbreak epidemiologically linked to a food handler in Melbourne, Victoria. *Commun Dis Intell Q Rep* 2009; 33: 46–48.
62. Schmid D, Fretz R, Buchner G, et al. Foodborne outbreak of hepatitis A, November 2007-January 2008, Austria. *Eur J Clin Microbiol Infect Dis* 2009; 28: 385–391. https://doi.org/10.1007/s10096-008-0633-0
63. Shapiro CN and Margolis HS. Worldwide epidemiology of hepatitis A virus infection. *J Hepatol* 1993; 18(Suppl 2): S11–S14. https://doi.org/10.1016/s0168-8278(05)80371-x
64. National Institute for Health and Care Excellence (NICE). Clinical Knowledge summary: hepatitis A - risk factors. https://cks.nice.org.uk/topics/hepatitis-a/background-information/risk-factors/ (2025, accessed 27 January 2025).
65. Bell A, Ncube F, Hansell A, et al. An outbreak of hepatitis A among young men associated with having sex in public venues. *Commun Dis Public Health* 2001; 4: 163–170.
66. Cotter SM, Sansom S, Long T, et al. Outbreak of hepatitis A among men who have sex with men: implications for hepatitis A vaccination strategies. *J Infect Dis* 2003; 187: 1235–1240. https://doi.org/10.1086/374057
67. Dodge B, Reece M, Herbenick D, et al. Relations between sexually transmitted infection diagnosis and sexual compulsivity in a community-based sample of men who have sex with men. *Sex Transm Infect* 2008; 84: 324–327. https://doi.org/10.1136/sti.2007.028696
68. Reintjes R, Bosman A, de Zwart O, et al. Outbreak of hepatitis A in Rotterdam associated with visits to 'darkrooms' in gay bars. *Commun Dis Public Health* 1999; 2: 43–46.
69. Stene-Johansen K, Tjon G, Schreier E, et al. Molecular epidemiological studies show that hepatitis A virus is endemic among active homosexual men in Europe. *J Med Virol* 2007; 79: 356–365. https://doi.org/10.1002/jmv.20781
70. Urbanus AT, van Houdt R, van de Laar TJ, et al. Viral hepatitis among men who have sex with men, epidemiology and public health consequences. *Euro Surveill* 2009; 14: 19421. https://doi.org/10.2807/ese.14.47.19421-en
71. Van Rijckevorsel GG, Sonder GJ, Bovee LP, et al. Trends in hepatitis A, B, and shigellosis compared with gonorrhea and syphilis in men who have sex with men in Amsterdam, 1992-2006. *Sex Transm Dis* 2008; 35: 930–934. https://doi.org/10.1097/OLQ.0b013e3181812cdf
72. Ross JD, Ghanem M, Tariq A, et al. Seroprevalence of hepatitis A immunity in male genitourinary medicine clinic attenders: a case control study of heterosexual and homosexual men. *Sex Transm Infect* 2002; 78: 174–179. https://doi.org/10.1136/sti.78.3.174
73. Gallego M, Robles M, Palacios R, et al. Impact of acute hepatitis A virus (HAV) infection on HIV viral load in HIV-Infected patients and influence of HIV infection on acute HAV infection. *J Int Assoc Physicians AIDS Care (Chic)* 2011; 10: 40–42. https://doi.org/10.1177/1545109710385692
74. Ida S, Tachikawa N, Nakajima A, et al. Influence of human immunodeficiency virus type 1 infection on acute hepatitis A virus infection. *Clin Infect Dis* 2002; 34: 379–385. https://doi.org/10.1086/338152
75. Lee YL, Chen GJ, Chen NY, et al. Less severe but prolonged course of acute hepatitis A in human immunodeficiency virus (HIV)-infected patients compared with HIV-uninfected patients during an outbreak: a multicenter observational study. *Clin Infect Dis* 2018; 67: 1595–1602. https://doi.org/10.1093/cid/ciy328
76. Ngui SL, Granerod J, Jewes LA, et al. Outbreaks of hepatitis A in England and Wales associated with two co-circulating

hepatitis A virus strains. *J Med Virol* 2008; 80: 1181–1188. https://doi.org/10.1002/jmv.21207
77. Van Damme P, Pinto RM, Feng Z, et al. Hepatitis A virus infection. *Nat Rev Dis Primers* 2023; 9: 51. https://doi.org/10.1038/s41572-023-00461-2
78. Lemon SM, Ott JJ, Van Damme P, et al. Type A viral hepatitis: a summary and update on the molecular virology, epidemiology, pathogenesis and prevention. *J Hepatol* 2017, Online ahead of print. https://doi.org/10.1016/j.jhep.2017.08.034
79. O'Donovan D, Cooke RP, Joce R, et al. An outbreak of hepatitis A amongst injecting drug users. *Epidemiol Infect* 2001; 127: 469–473. https://doi.org/10.1017/s0950268801006185
80. Bohm SR, Berger KW, Hackert PB, et al. Hepatitis A outbreak among adults with developmental disabilities in group homes -Michigan, 2013. *MMWR Morb Mortal Wkly Rep* 2015; 64: 148–152.
81. Ruscher C, Faber M, Werber D, et al. Resurgence of an international hepatitis A outbreak linked to imported frozen strawberries, Germany, 2018 to 2020. *Euro Surveill* 2020; 25: 1900670. https://doi.org/10.2807/1560-7917.ES.2020.25.37.1900670
82. European Centre for Disease Prevention and Control (ECDC). Rapid risk assessment: hepatitis A outbreak in the EU/EEA mostly affecting men who have sex with men. https://www.ecdc.europa.eu/en/news-events/epidemiological-update-hepatitis-outbreak-eueea-mostly-affecting-men-who-have-sex-men (updated 29 September 2017, accessed 29 January 2025).
83. Zuckerman JN and Zuckerman AJ. Viral hepatitis. In: Cook GC and Zumla AI (eds). *Manson's Tropical Diseases*. Eds Saunders. Saunders Elsevier, 2009, pp. 697–714.
84. Jeong SH and Lee HS. Hepatitis A: clinical manifestations and management. *Intervirology* 2010; 53: 15–19. https://doi.org/10.1159/000252779
85. Sookoian S. Liver disease during pregnancy: acute viral hepatitis. *Ann Hepatol* 2006; 5: 231–236.
86. Elinav E, Ben-Dov IZ, Shapira Y, et al. Acute hepatitis A infection in pregnancy is associated with high rates of gestational complications and preterm labor. *Gastroenterology* 2006; 130: 1129–1134. https://doi.org/10.1053/j.gastro.2006.01.007
87. Charuworn P and Cheung R. Diagnostic markers for hepatitis virus infection. *Expert Opin Med Diagn* 2008; 2: 303–314. https://doi.org/10.1517/17530059.2.3.303
88. Lee HK, Kim KA, Lee JS, et al. Window period of anti-hepatitis A virus immunoglobulin M antibodies in diagnosing acute hepatitis A. *Eur J Gastroenterol Hepatol* 2013; 25: 665–668. https://doi.org/10.1097/MEG.0b013e32835df262
89. European Association for the Study of the Liver, Clinical Practice Guidelines Panel. Wendon J, et al. EASL clinical practical guidelines on the management of acute (fulminant) liver failure. *J Hepatol* 2017; 66: 1047–1081. https://doi.org/10.1016/j.jhep.2016.12.003
90. UK Health Security Agency. Hepatitis A immunoglobulin (issued 2024). https://www.gov.uk/government/publications/immunoglobulin-when-to-use/hepatitis-a-immunoglobulin-issued-2024 (updated 7 November 2024, accessed 27 January 2025).
91. World Health Organisation. Global hepatitis report 2024: action for access in low- and middle-income countries. https://www.who.int/publications/i/item/9789240091672 (2024, accessed 28 January 2025).
92. World Health Organisation. Hepatitis B fact sheet. https://www.who.int/news-room/fact-sheets/detail/hepatitis-b (2024, accessed 28 January 2025).
93. European Centre for Disease Prevention and Control (ECDC). ECDC evidence brief: prevention of hepatitis B and C in the EU/EEA. https://www.ecdc.europa.eu/en/publications-data/prevention-hepatitis-b-and-c-eueea-2024 (2024, accessed 27 January 2025).
94. UK Health Security Agency. Hepatitis B in England - 2024. https://www.gov.uk/government/publications/hepatitis-b-in-england/hepatitis-b-in-england-2024 (2025, updated 06 January 2025, accessed 27 January 2025).
95. UK Health Security Agency. Acute hepatitis B: national enhanced surveillance report January to December 2022. https://www.gov.uk/government/publications/acute-hepatitis-b-england-enhanced-surveillance-reports/acute-hepatitis-b-national-enhanced-surveillance-report-january-to-december-2022 (updated 24 January 2024, accessed 08 January 2025).
96. UK Health Security Agency. Public health evaluation of BBV opt-out testing in EDs in England: 24-month interim report. https://www.gov.uk/government/publications/bloodborne-viruses-opt-out-testing-in-emergency-departments/public-health-evaluation-of-bbv-opt-out-testing-in-eds-in-england-24-month-interim-report (updated 28 November 2024, accessed 27 January 2025).
97. Centers for Disease Control and Prevention (CDC). Clinical testing and diagnosis for Hepatitis B. https://www.cdc.gov/hepatitis-b/hcp/diagnosis-testing/?CDC_AAref_Val=https://www.cdc.gov/hepatitis/hbv/testingchronic.htm (06 March 2024, accessed 27 January 2025).
98. Yeung LT and Roberts EA. Hepatitis B in childhood: an update for the paediatrician. *Paediatr Child Health* 2001; 6: 655–659. https://doi.org/10.1093/pch/6.9.655
99. Gerlich WH. Medical virology of hepatitis B: how it began and where we are now. *Virol J* 2013; 10: 239. https://doi.org/10.1186/1743-422X-10-239
100. Cramp ME, Grundy HC, Perinpanayagam RM, et al. Seroprevalence of hepatitis B and C virus in two institutions caring for mentally handicapped adults. *J R Soc Med* 1996; 89: 401–402.
101. Wallace RA. Clinical audit of gastrointestinal conditions occurring among adults with Down syndrome attending a specialist clinic. *J Intellect Dev Disabil* 2007; 32: 45–50. https://doi.org/10.1080/13668250601146761
102. Hyams KC. Risks of chronicity following acute hepatitis B virus infection: a review. *Clin Infect Dis* 1995; 20: 992–1000. https://doi.org/10.1093/clinids/20.4.992
103. National Institute for Health and Care Excellence (NICE). Hepatitis B (chronic): diagnosis and

management. Clinical guideline [CG165]. https://www.nice.org.uk/guidance/cg165 (2013, updated 20 October 2017, accessed 28 January 2025).
104. Hann HW, Han SH, Block TM, et al. Symptomatology and health attitudes of chronic hepatitis B patients in the USA. *J Viral Hepat* 2008; 15: 42–51. https://doi.org/10.1111/j.1365-2893.2007.00895.x
105. Oxman AD, Scott EA, Sellors JW, et al. Partner notification for sexually transmitted diseases: an overview of the evidence. *Can J Public Health* 1994; 85(Suppl 1): S41–S47.
106. McClean H, Radcliffe K, Sullivan A, et al. 2012 BASHH statement on partner notification for sexually transmissible infections. *Int J STD AIDS* 2013; 24: 253–261. https://doi.org/10.1177/0956462412472804
107. Landrum ML, Fieberg AM, Chun HM, et al. The effect of human immunodeficiency virus on hepatitis B virus serologic status in co-infected adults. *PLoS One* 2010; 5: e8687. https://doi.org/10.1371/journal.pone.0008687
108. Thornton AC, Jose S, Bhagani S, et al. Hepatitis B, hepatitis C, and mortality among HIV-positive individuals. *AIDS* 2017; 31: 2525–2532. https://doi.org/10.1097/QAD.0000000000001646
109. Im YR, Jagdish R, Leith D, et al. Prevalence of occult hepatitis B virus infection in adults: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol* 2022; 7: 932–942. https://doi.org/10.1016/S2468-1253(22)00201-1
110. Raimondo G, Locarnini S, Pollicino T, et al. Update of the statements on biology and clinical impact of occult hepatitis B virus infection. *J Hepatol* 2019; 71: 397–408. https://doi.org/10.1016/j.jhep.2019.03.034
111. Sagnelli E, Coppola N, Pisaturo M, et al. Clinical and virological improvement of hepatitis B virus-related or hepatitis C virus-related chronic hepatitis with concomitant hepatitis A virus infection. *Clin Infect Dis* 2006; 42: 1536–1543. https://doi.org/10.1086/503840
112. Fattovich G, Bortolotti F and Donato F. Natural history of chronic hepatitis B: special emphasis on disease progression and prognostic factors. *J Hepatol* 2008; 48: 335–352. https://doi.org/10.1016/j.jhep.2007.11.011
113. Lin L, Verslype C, van Pelt JF, et al. Viral interaction and clinical implications of coinfection of hepatitis C virus with other hepatitis viruses. *Eur J Gastroenterol Hepatol* 2006; 18: 1311–1319. https://doi.org/10.1097/01.meg.0000243881.09820.09
114. Chiaramonte M, Stroffolini T, Vian A, et al. Rate of incidence of hepatocellular carcinoma in patients with compensated viral cirrhosis. *Cancer* 1999; 85: 2132–2137.
115. Papatheodoridis GV, Manolakopoulos S, Dusheiko G, et al. Therapeutic strategies in the management of patients with chronic hepatitis B virus infection. *Lancet Infect Dis* 2008; 8: 167–178. https://doi.org/10.1016/S1473-3099(07)70264-5
116. Hoofnagle JH. Chronic hepatitis B. *N Engl J Med* 1990; 323: 337–339. https://doi.org/10.1056/NEJM199008023230510
117. Thomas HC. Best practice in the treatment of chronic hepatitis B: a summary of the European Viral Hepatitis Educational Initiative (EVHEI). *J Hepatol* 2007; 47: 588–597. https://doi.org/10.1016/j.jhep.2007.07.017
118. 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: 1921–1926. https://doi.org/10.1016/s0140-6736(02)11913-1
119. Geretti AM, Austin H, Villa G, et al. Hepatitis B virus infection in general practice across England: an analysis of the Royal College of General Practitioners Research and Surveillance Centre real-world database. *J Infect* 2023; 86: 476–485. https://doi.org/10.1016/j.jinf.2023.03.001
120. Hutton DW, Toy M, Salomon JA, et al. Cost-effectiveness of hepatitis B testing and vaccination of adults seeking care for sexually transmitted infections. *Sex Transm Dis* 2022; 49: 517–525. https://doi.org/10.1097/OLQ.0000000000001632
121. Conners 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: 1–25. https://doi.org/10.15585/mmwr.rr7201a1
122. National Institute for Health and Care Excellence (NICE). Clinical knowledge summary: hepatitis B - how to test for hepatitis B. https://cks.nice.org.uk/topics/hepatitis-b/diagnosis/how-to-test-for-hepatitis-b/#:~:text=Initialtestingshouldideallyinclude,requireddependingonthefindings (2022, accessed 27 January 2025).
123. The Royal College of Pathologists. UK standards for microbiology investigations. https://www.rcpath.org/profession/publications/standards-for-microbiology-investigations.html (accessed 29 January 2025).
124. ABC. https://www.rcpath.org/static/7875b72a-521a-4755-88ae270a2e230bf7/cbe174cd-5788-49bb-8499d47428cbb441/uk-smi-v-4i6-1-investigation-of-hepatitis-b-infection-july-2021-pdf.pdf
125. Public Health England. Hepatitis B factsheet. https://www.england.nhs.uk/wp-content/uploads/2014/11/hepatitis-b-fctsht.pdf (2014, accessed 29 January 2025).
126. British Liver Trust. Hepatitis B: breaking the silence. https://britishlivertrust.org.uk/hepatitis-b-breaking-the-silence/?gad_source=1&gclid=EAIaIQobChMIh4X0wv_WiQMVw6NQBh1s7iVPEAAYASAAEgK6BPD_BwE (2014, accessed 29 January 2025).
127. World Health Organisation. Guidelines for the prevention, diagnosis, care and treatment for people with chronic hepatitis B infection. https://www.who.int/publications/i/item/9789240090903 (2024, accessed 03 February 2025).
128. Brady M, Rodger A, Asboe D, et al. BHIVA/BASHH guidelines on the use of HIV pre-exposure prophylaxis (PrEP) 2018. *HIV Med* 2019; 20 Suppl 2: s2–s80. https://doi.org/10.1111/hiv.12718
129. British Viral Hepatitis Group (BVHG) and Maternal and Paediatric Subgroup. Guideline for the management of hepatitis B in pregnancy and the exposed infant. https://www.basl.org.uk/uploads/BVHGPerinatalHBV3.3.21.pdf (2021, accessed 29 January 2025).

130. Sherman M. Hepatocellular carcinoma: epidemiology, risk factors, and screening. *Semin Liver Dis* 2005; 25: 143–154. https://doi.org/10.1055/s-2005-871194
131. Papatheodoridis G, Dalekos G, Sypsa V, et al. PAGE-B predicts the risk of developing hepatocellular carcinoma in Caucasians with chronic hepatitis B on 5-year antiviral therapy. *J Hepatol* 2016; 64: 800–806. https://doi.org/10.1016/j.jhep.2015.11.035
132. British HIV Association (BHIVA). UK guideline for the use of HIV Post-exposure prophylaxis following sexual exposure (PEPSE) 2015, 2015. https://www.bhiva.org/file/6074031a87755/PEPSE-guidelines.pdf
133. Da BL, Heller T and Koh C. Hepatitis D infection: from initial discovery to current investigational therapies. *Gastroenterol Rep (Oxf)* 2019; 7: 231–245. https://doi.org/10.1093/gastro/goz023
134. Urban S, Neumann-Haefelin C and Lampertico P. Hepatitis D virus in 2021: virology, immunology and new treatment approaches for a difficult-to-treat disease. *Gut* 2021; 70: 1782–1794. https://doi.org/10.1136/gutjnl-2020-323888
135. Farci P and Niro GA. Clinical features of hepatitis D. *Semin Liver Dis* 2012; 32: 228–236. https://doi.org/10.1055/s-0032-1323628
136. National Institute for Health and Care Excellence (NICE). Bulevirtide for treating chronic hepatitis D. Technology appraisal guidance [TA896]. https://www.nice.org.uk/guidance/ta896/evidence (2023, accessed 28 January 2025).
137. Collaborators Polaris Observatory HCV. Global change in hepatitis C virus prevalence and cascade of care between 2015 and 2020: a modelling study. *Lancet Gastroenterol Hepatol* 2022; 7: 396–415. https://doi.org/10.1016/S2468-1253(21)00472-6
138. UK Health Security Agency. Hepatitis C in UK 2023: working to eliminate hepatitis C as a public health threat. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1133731/hepatitis-c-in-the-UK-2023.pdf (2023, accessed 08 January 2025).
139. World Health Organisation. Global health sector strategy on viral hepatitis, 2016–2021: towards ending viral hepatitis, 2016. https://www.who.int/publications/i/item/WHO-HIV-2016.06
140. World Health Organisation. Global health sector strategies on, respectively, HIV, viral hepatitis and sexually transmitted infections for the period 2022-2030, 2022. https://iris.who.int/bitstream/handle/10665/360348/9789240053779-eng.pdf?sequence=1
141. Hedskog C, Parhy B, Chang S, et al. Identification of 19 novel hepatitis C virus subtypes-further expanding HCV classification. *Open Forum Infect Dis* 2019; 6: ofz076. https://doi.org/10.1093/ofid/ofz076
142. Sonderup MW, Afihene M, Ally R, et al. Hepatitis C in sub-Saharan Africa: the current status and recommendations for achieving elimination by 2030. *Lancet Gastroenterol Hepatol* 2017; 2: 910–919. https://doi.org/10.1016/S2468-1253(17)30249-2
143. Vandelli C, Renzo F, Romano L, et al. Lack of evidence of sexual transmission of hepatitis C among monogamous couples: results of a 10-year prospective follow-up study. *Am J Gastroenterol* 2004; 99: 855–859. https://doi.org/10.1111/j.1572-0241.2004.04150.x
144. Terrault NA, Dodge JL, Murphy EL, et al. Sexual transmission of hepatitis C virus among monogamous heterosexual couples: the HCV partners study. *Hepatology* 2013; 57: 881–889. https://doi.org/10.1002/hep.26164
145. Marincovich B, Castilla J, del Romero J, et al. Absence of hepatitis C virus transmission in a prospective cohort of heterosexual serodiscordant couples. *Sex Transm Infect* 2003; 79: 160–162. https://doi.org/10.1136/sti.79.2.160
146. McMahon JM, Pouget ER and Tortu S. Individual and couple-level risk factors for hepatitis C infection among heterosexual drug users: a multilevel dyadic analysis. *J Infect Dis* 2007; 195: 1572–1581. https://doi.org/10.1086/516785
147. Kao JH, Liu CJ, Chen PJ, et al. Low incidence of hepatitis C virus transmission between spouses: a prospective study. *J Gastroenterol Hepatol* 2000; 15: 391–395. https://doi.org/10.1046/j.1440-1746.2000.02165.x
148. Tohme RA and Holmberg SD. Is sexual contact a major mode of hepatitis C virus transmission? *Hepatology* 2010; 52: 1497–1505. https://doi.org/10.1002/hep.23808
149. Jin F, Dore GJ, Matthews G, et al. Prevalence and incidence of hepatitis C virus infection in men who have sex with men: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol* 2021; 6: 39–56. https://doi.org/10.1016/S2468-1253(20)30303-4
150. van de Laar TJ, van der Bij AK, Prins M, et al. Increase in HCV incidence among men who have sex with men in Amsterdam most likely caused by sexual transmission. *J Infect Dis* 2007; 196: 230–238. https://doi.org/10.1086/518796
151. Browne R, Asboe D, Gilleece Y, et al. Increased numbers of acute hepatitis C infections in HIV positive homosexual men; is sexual transmission feeding the increase? *Sex Transm Infect* 2004; 80: 326–327. https://doi.org/10.1136/sti.2003.008532
152. Danta M, Brown D, Bhagani S, et al. Recent epidemic of acute hepatitis C virus in HIV-positive men who have sex with men linked to high-risk sexual behaviours. *AIDS* 2007; 21: 983–991. https://doi.org/10.1097/QAD.0b013e3281053a0c
153. Marcellin F, Lorente N, Demoulin B, et al. Comparison of risk factors in HIV-infected men who have sex with men, coinfected or not with hepatitis C virus (ANRS VESPA2 French cross-sectional national survey). *Sex Transm Infect* 2015; 91: 21–23. https://doi.org/10.1136/sextrans-2014-051542
154. Ward H, Martin I, Macdonald N, et al. Lymphogranuloma venereum in the United Kingdom. *Clin Infect Dis* 2007; 44: 26–32. https://doi.org/10.1086/509922

155. Recently acquired and early chronic hepatitis C in MSM: recommendations from the European treatment network for HIV, hepatitis and global infectious diseases consensus panel. *AIDS* 2020; 34: 1699–1711.
156. Hosseini-Hooshyar S, Hajarizadeh B, Bajis S, et al. Risk of hepatitis C reinfection following successful therapy among people living with HIV: a global systematic review, meta-analysis, and meta-regression. *Lancet HIV* 2022; 9: e414–e427. https://doi.org/10.1016/S2352-3018(22)00077-7
157. Garvey LJ, Cooke GS, Smith C, et al. Decline in Hepatitis C Virus (HCV) incidence in men who have sex with men living with human immunodeficiency virus: progress to HCV microelimination in the United Kingdom? *Clin Infect Dis* 2021; 72: 233–238. https://doi.org/10.1093/cid/ciaa021
158. Boerekamps A, van den Berk GE, Lauw FN, et al. Declining Hepatitis C Virus (HCV) incidence in Dutch human immunodeficiency virus-positive men who have sex with men after unrestricted access to HCV therapy. *Clin Infect Dis* 2018; 66: 1360–1365. https://doi.org/10.1093/cid/cix1007
159. Kusejko K, Salazar-Vizcaya L, Shah C, et al. Sustained effect on Hepatitis C elimination among men who have sex with men in the Swiss HIV cohort study: a systematic Re-Screening for hepatitis C RNA two years following a nation-wide elimination program. *Clin Infect Dis* 2022; 75: 1723–1731. https://doi.org/10.1093/cid/ciac273
160. Desai M, White E, Vora N, et al. High incidence of Hepatitis C virus infection observed in the PROUD study of HIV pre-exposure prophylaxis. *J Viral Hepat* 2020; 27: 852–857. https://doi.org/10.1111/jvh.13297
161. Bradshaw D, Vasylyeva TI, Davis C, et al. Transmission of hepatitis C virus in HIV-positive and PrEP-using MSM in England. *J Viral Hepat* 2020; 27: 721–730. https://doi.org/10.1111/jvh.13286
162. Ades AE, Gordon F, Scott K, et al. Overall vertical transmission of hepatitis C virus, transmission net of clearance, and timing of transmission. *Clin Infect Dis* 2023; 76: 905–912. https://doi.org/10.1093/cid/ciac270
163. Benova L, Mohamoud YA, Calvert C, et al. Vertical transmission of hepatitis C virus: systematic review and meta-analysis. *Clin Infect Dis* 2014; 59: 765–773. https://doi.org/10.1093/cid/ciu447
164. Ohto H, Terazawa S, Sasaki N, et al. Transmission of hepatitis C virus from mothers to infants. The vertical transmission of Hepatitis C virus collaborative study group. *N Engl J Med* 1994; 330: 744–750. https://doi.org/10.1056/NEJM199403173301103
165. Mast EE, Hwang LY, Seto DS, et al. Risk factors for perinatal transmission of hepatitis C virus (HCV) and the natural history of HCV infection acquired in infancy. *J Infect Dis* 2005; 192: 1880–1889. https://doi.org/10.1086/497701
166. Ceci O, Margiotta M, Marello F, et al. Vertical transmission of hepatitis C virus in a cohort of 2,447 HIV-seronegative pregnant women: a 24-month prospective study. *J Pediatr Gastroenterol Nutr* 2001; 33: 570–575. https://doi.org/10.1097/00005176-200111000-00011
167. Azzari C, Moriondo M, Indolfi G, et al. Higher risk of hepatitis C virus perinatal transmission from drug user mothers is mediated by peripheral blood mononuclear cell infection. *J Med Virol* 2008; 80: 65–71. https://doi.org/10.1002/jmv.21023
168. *Hepatitis B or C infections and breastfeeding | breastfeeding special circumstances* | CDC (accessed 2 March 2026).
169. Perrett SE, Plimmer A, Shankar AG, et al. Prevalence of HCV in prisons in Wales, UK and the impact of moving to opt-out HCV testing. *J Public Health* 2020; 42: 423–428. https://doi.org/10.1093/pubmed/fdaa022
170. Terrault NA. Sexual activity as a risk factor for hepatitis C. *Hepatology* 2002; 36: S99–S105. https://doi.org/10.1053/jhep.2002.36797
171. Public Health England. Evaluation of hepatitis C test and treat interventions targeted at homeless populations (outside London) in England during the COVID-19 pandemic. 2020 Report, 2021. https://assets.publishing.service.gov.uk/media/5ffc9a85e90e07639fd8d4b9/Evaluation_of_initiatives_to_test_and_treat_the_housed_homeless_2020_report.pdf
172. Macias J, Palacios RB, Claro E, et al. High prevalence of hepatitis C virus infection among noninjecting drug users: association with sharing the inhalation implements of crack. *Liver Int* 2008; 28: 781–786. https://doi.org/10.1111/j.1478-3231.2008.01688.x
173. Fernandez N, Towers CV, Wolfe L, et al. Sharing of snorting straws and hepatitis C virus infection in pregnant women. *Obstet Gynecol* 2016; 128: 234–237. https://doi.org/10.1097/AOG.0000000000001507
174. Haley RW and Fischer RP. Commercial tattooing as a potentially important source of hepatitis C infection. Clinical epidemiology of 626 consecutive patients unaware of their hepatitis C serologic status. *Medicine (Baltim)* 2001; 80: 134–151. https://doi.org/10.1097/00005792-200103000-00006
175. Neal KR, Jones DA, Killey D, et al. Risk factors for hepatitis C virus infection. A case-control study of blood donors in the Trent Region (UK). *Epidemiol Infect* 1994; 112: 595–601. https://doi.org/10.1017/s0950268800051293
176. Smith DJ, Jordan AE, Frank M, et al. Spontaneous viral clearance of hepatitis C virus (HCV) infection among people who inject drugs (PWID) and HIV-positive men who have sex with men (HIV+ MSM): a systematic review and meta-analysis. *BMC Infect Dis* 2016; 16: 471. https://doi.org/10.1186/s12879-016-1807-5
177. Hajarizadeh B, Grebely J and Dore GJ. Case definitions for acute hepatitis C virus infection: a systematic review. *J Hepatol* 2012; 57: 1349–1360. https://doi.org/10.1016/j.jhep.2012.07.007
178. National Institute for Health and Care Excellence (NICE). Clinical knowledge summary: who should I test for hepatitis

C? https://cks.nice.org.uk/topics/hepatitis-c/diagnosis/who-to-screen-test/ (2022, accessed 27 January 2025).
179. Public Health England. Hepatitis C: guidance on the investigation and management of occupational exposure. https://webarchive.nationalarchives.gov.uk/ukgwa/+/. https://www.hpa.org.uk/cdph/issues/CDPHVol2/no4/guides_hepC.pdf (1999, accessed 03 February 2025).
180. Thomson EC, Nastouli E, Main J, et al. Delayed anti-HCV antibody response in HIV-positive men acutely infected with HCV. *AIDS* 2009; 23: 89–93. https://doi.org/10.1097/QAD.0b013e32831940a3
181. Khan H, Hill A, Main J, et al. Can hepatitis C virus antigen testing replace ribonucleic acid polymearse chain reaction analysis for detecting hepatitis C virus? A systematic review. *Open Forum Infect Dis* 2017; 4: ofw252. https://doi.org/10.1093/ofid/ofw252
182. Freiman JM, Tran TM, Schumacher SG, et al. Hepatitis C core antigen testing for diagnosis of hepatitis C virus infection: a systematic review and meta-analysis. *Ann Intern Med* 2016; 165: 345–355. https://doi.org/10.7326/M16-0065

Appendix

Abbreviations

AASLD	American Association for the Study of Liver Disease
AGREE II	Appraisal of Guidelines, Research and Evaluation
ALF	Acute Liver Failure
ALT	Alanine Transaminase
Anti-HBc	Antibody Against Hepatitis B core antigen
Anti-HBe	Antibody Against Hepatitis B ‘e’ antigen
Anti-HBs	Antibody Against Hepatitis B surface antigen
Anti-HCV	Antibody Against HCV
Anti-HDV	Antibody Against HDV
Anti-HEV	Antibody Against HEV
ART	Antiretroviral Therapy
BASHH	British Association for Sexual Health and HIV
BHIVA	British HIV Association
CDC	Centers for Disease Control and Prevention
CEG	Clinical Effectiveness Group
CrI	Credible Interval
DAA	Direct acting antiviral
DBS	Dried Blood Spot
DNA	Deoxyribonucleic Acid
EASL	European Association for the Study of the Liver
ECDC	European Centre for Disease Prevention and Control
EU	European Union
GBL	Gamma-butyrolactone
GBMSM	Gay, bisexual or other men who have sex with men
GHB	Gamma-hydroxybutyric Acid
GPP	Good practice point
GRADE	Grading of Recommendations, Assessment, Development, and Evaluation
GUMCAD	Genitourinary Medicine Clinic Activity Dataset
HAV	Hepatitis A Virus
HAV-IgM/G	HAV–specific Immunoglobulin Type M/G
HBeAg	Hepatitis B ‘e’ Antigen
HBIG	Hepatitis B Immunoglobulin
HBsAg	Hepatitis B Surface Antigen
HBV	Hepatitis B Virus
HBV-DNA	Hepatitis B Virus Deoxyribonucleic Acid
HCC	Hepatocellular Carcinoma
HCV	Hepatitis C Virus
HCV Ag	Hepatitis C Core Antigen
HCV-RNA	Hepatitis C Virus Ribose Nucleic Acid
HDV	Hepatitis Delta Virus
HEV	Hepatitis E Virus
HIV	Human Immunodeficiency Virus
HIVPA	HIV Pharmacy Association
HNIG	Human Normal Immunoglobulin
IgG	Immunoglobulin Type G
IgM	Immunoglobulin Type M
IUSTI	International Union Against Sexually Transmitted Infection
LFT	Liver Function Test
LGV	Lymphogranuloma Venereum
NHS	National Health Service
NICE	National Institute for Health and Care Excellence
PCR	Polymerase Chain Reaction
PEP	HIV Post-exposure Prophylaxis
POCT	Point-of-Care Testing
PrEP	HIV Pre-exposure Prophylaxis
RNA	Ribonucleic Acid
PWID	People Who Inject Drugs
SSHA	Society of Sexual Health Advisers
STI	Sexually Transmitted Infection
TAF	Tenofovir Alafenamide
TDF	Tenofovir Disoproxil Fumarate
UKHSA	UK Health Security Agency
US	United States
WHO	World Health Organisation