Hepatitis B

In primary healthcare

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23.05.2026

A GUIDE FOR THE GENERAL PRACTITIONER

DISCLAIMER
FOR EDUCATION, NOT PRESCRIPTION

This presentation has been prepared for the **continuing professional education of general practitioners**. It summarises guidance from the WHO 2024 hepatitis B guidelines, the WHO Global Hepatitis Report 2026 and the peer-reviewed literature cited in the References slide.

The content is intended as a general clinical overview. It is not a substitute for individual clinical judgement, national guidelines, formulary restrictions, or the specific circumstances of any patient.

Doses, thresholds and eligibility criteria evolve. Before acting on this material, consult the current South African national guidelines, your institutional protocols, and the latest WHO recommendations.

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Outline

WHAT WILL BE COVERED TODAY

01 **Epidemiology** – global and regional snapshot

02 **Acute hepatitis B**

03 **Chronic hepatitis B**

04 **Approach to diagnosis**

05 **Approach to treatment**

06 **Co-infections** – HIV . HCV . HDV

07 **Special populations** – Pregnant women . HCWs

08 **Prevention**

09 **Key take-aways for general practice**

Highlights WHO 2024 Guidelines

WHAT CHANGED IN THE 2024 UPDATE

01 SETTING

Decentralised testing and care

Move away from tertiary level (ID/hepatology) to primary healthcare

02 DIAGNOSTICS

Improved HBV diagnostics

Point-of-care HBsAg and HBV DNA testing; reflex approaches to viral load testing

03 TREATMENT

Simplified eligibility

Expanded and simplified treatment criteria for adults and adolescents

04 PREGNANCY SCREENING

Simplified screening in pregnancy

Universal antenatal HBsAg screening integrated into existing maternal services

05 PMTCT

Expanded antiviral prophylaxis

Broader eligibility for antiviral prophylaxis to prevent MTCT

Global Epidemiology

ENDEMICITY = PREVALENCE

Percentage of population testing positive for HBsAg

HIGH
≥ 8%

INTERMEDIATE
2- 8%
South Africa 4.6%

LOW
<2%

Seroprevalence varies by

Country . Rural v Urban . Gender . Ethnicity

Highest burden in Africa

Boys and men in rural areas

HBV BURDEN 2024

240M

people living with HBV infection

Only 27 % diagnosed · < 5 % on treatment · 70 % in WHO African & Western Pacific regions

0.9M

new infections in 2024
↓ 32% since 2015

1.1M

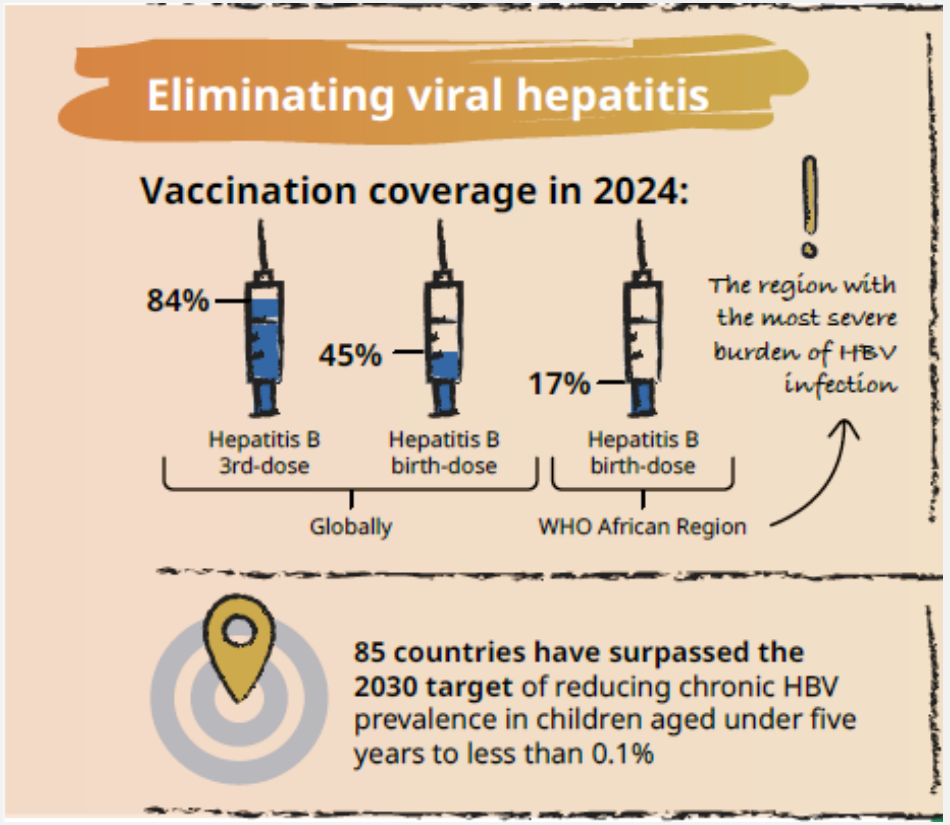
HBV deaths in 2024
↑ 17% since 2015

Jeng et al, 2023 ·WHO Global Hepatitis Report 2026

Priorities for Global and Regional Action

2030 GLOBAL HEPATITIS TARGETS

90%	reduction in new CHB infections (from 2015)
65%	reduction in hepatitis-related mortality (from 2015)
90%	of HBV infections diagnosed
80%	of eligible patients on treatment
90%	coverage for HBV birth-dose vaccine
90%	coverage for 3-dose infant HBV vaccination
100%	of blood donors screened



PRIORITIES WHO AFRICAN & WESTERN PACIFIC REGIONS

- Scale up treatment for CHB
- Improve coverage of HBV birth-dose vaccine
- Improve coverage of antiviral prophylaxis for PMTCT of HBV

HBV Transmission

SOURCE

Infected body fluids

- Blood
- Saliva
- Semen
- Vaginal fluids

ROUTE

How HBV spreads

- Perinatal
- Child-to-child
- Sexual
- Percutaneous
- Blood transfusion

Risk Factors Driving HBV Transmission

Perinatal	Child-to-child	Sexual	Percutaneous	Blood
High maternal viral load HIV co-infection Acute HBV infection during 3rd trimester No PMTCT	No childhood vaccination Incomplete vaccination schedule	High HBV viral load in source person Unvaccinated adult	Unvaccinated HCWs High viral load in source person Absent needle exchange programs	Absent or poor screening of blood and blood products

Main transmission routes in Africa

↓ Risk of progression to CHB ↓

95%

<5y: 30-50%; 5-20y: 6-10%

Acute HBV Infection

Uncomplicated symptomatic acute HBV does not require antiviral therapy.
> 95 % of immunocompetent adults spontaneously clear the infection.

WHO TO TREAT

Immunocompromised

HIV, patients on chemotherapy, biologics, transplant recipients

Pregnant women

If in liver failure, or for PMTCT when acute HBV acquired in 2nd / 3rd trimester or HBV DNA > 200 000 IU/mL in 3rd trimester.

Fulminant/severe

Fulminant hepatic failure or severe acute hepatitis with coagulopathy or encephalopathy.

Protracted course

Severe symptoms persisting beyond 4 weeks - ↑ risk of progression.

TREATMENT DURATION IF STARTED

Anti-HBs seroconversion route

Continue antivirals for ≥ 3 months after seroconversion to anti-HBs

Anti-HBe seroconversion route

Continue for ≥ 12 months after anti-HBe seroconversion without HBsAg loss.

The Problem with CHB

A **silent** and **incurable** disease

NATURAL HISTORY

- Chronic disease established **during childhood** in Africa and Asia.
- Causes liver damage over **decades**, then presents in adulthood.

Leading cause of chronic liver disease, cirrhosis and HCC worldwide.

BURDEN OF CHB

254M globally living with CHB

2.5M South Africans affected

Moonsamy et al, 2023

BUT…

SAFE & EFFECTIVE VACCINE SINCE 1982

98 - 100 % protection conferred

Introduced into South African EPI in **1995**.

COMPOUNDED BY CO-MORBIDITIES

- HIV co-infection · **1.9 M**
- HDV co-infection · **1.6 M**
- Rising alcohol consumption
- Rising metabolic risk factors (MASLD)

THE WHO SIMPLIFIED PATHWAY TO CARE

01 Screen

02 Stage

03 Treat

A stepwise algorithm to manage HBV in primary care

01 Screen

01 TESTING METHODS

- Lab immunoassays
- Rapid diagnostic tests (RDTs)
- Validated point-of-care tests

https://sl1nk.com/qyot1qb

02 PREFERRED SCREENING TEST - HBsAg

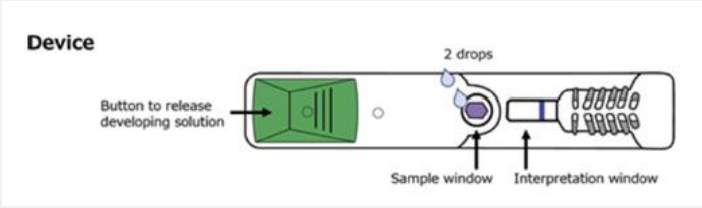
Positive test = current HBV infection (acute or chronic)

Persistence ≥ 6 months defines **chronic** HBV infection

03 CONFIRM WITH HBV DNA

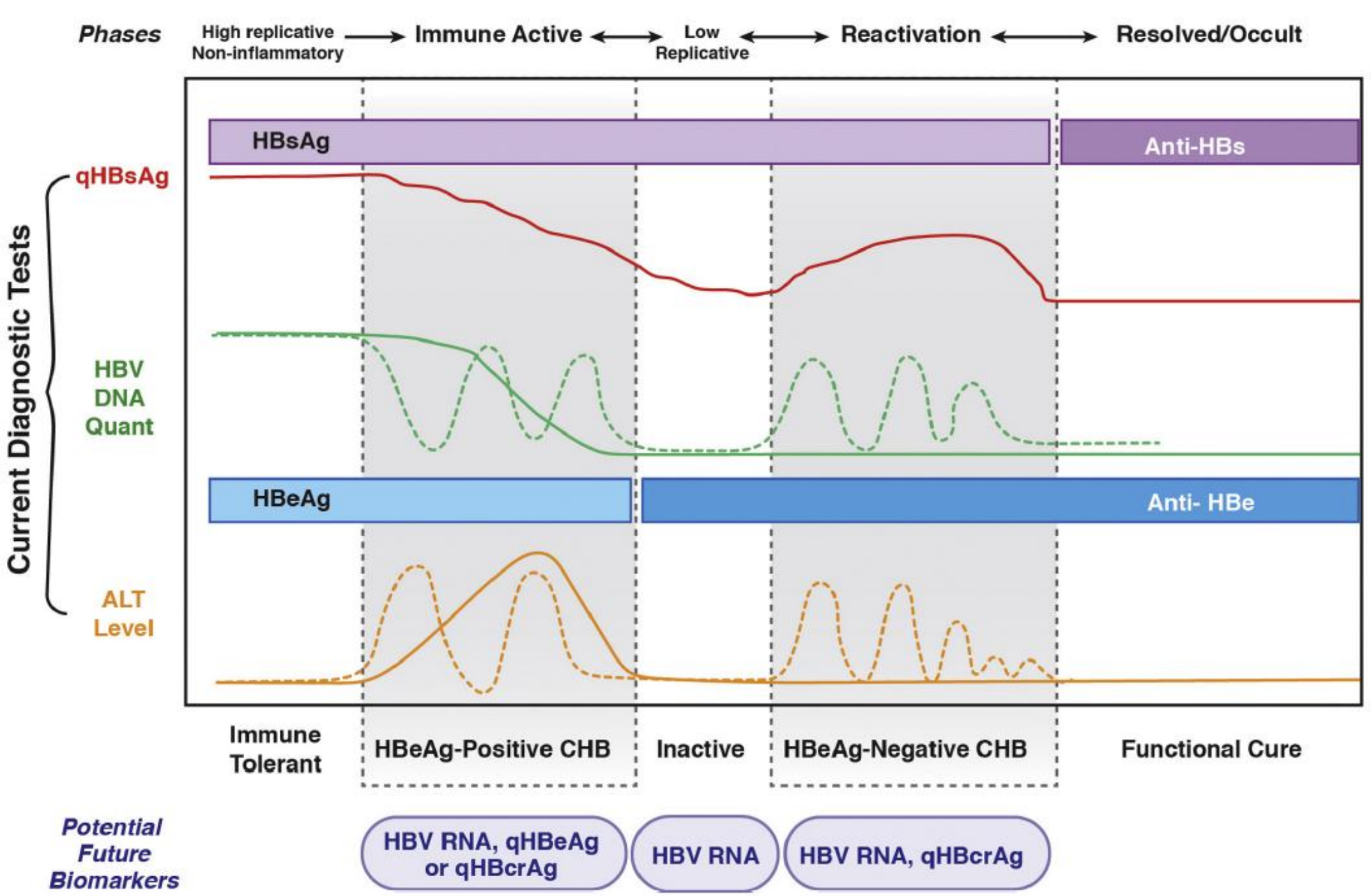
Once HBsAg positive → simplified access to HBV DNA testing

- **Preferred:** HBV DNA (costly) — ideally reflex testing, POC testing
- Surrogates if HBV DNA unavailable:
  - HBeAg (where no DNA access)
  - Hep B core-related antigen (HBcrAg) POC test — reflects high viral load



Shimakawa et al 2022

HBcrAg POC: 91 % sensitivity & 86 % specificity for VL > 200 000 IU/mL


Phases
High replicative
Non-inflammatory
Immune Active
Low
Replicative
Reactivation
Resolved/Occult
Current Diagnostic Tests
qHBsAg
HBV
DNA
Quant
ALT
Level
HBsAg
Anti-HBs
HBeAg
Anti- HBe
Immune
Tolerant
HBeAg-Positive CHB
Inactive
HBeAg-Negative CHB
Functional Cure
Potential
Future
Biomarkers
HBV RNA, qHBeAg
or qHBcrAg
HBV RNA
HBV RNA, qHBcrAg

WHO Recommendations for HBsAg Testing

01

Universal adult screening

Where HBsAg prevalence is ≥ **2 %,** i.e. intermediate and high prevalence settings.

In South Africa, this applies broadly across the adult population.

02

Targeted testing in all settings

HIGH-YIELD GROUPS

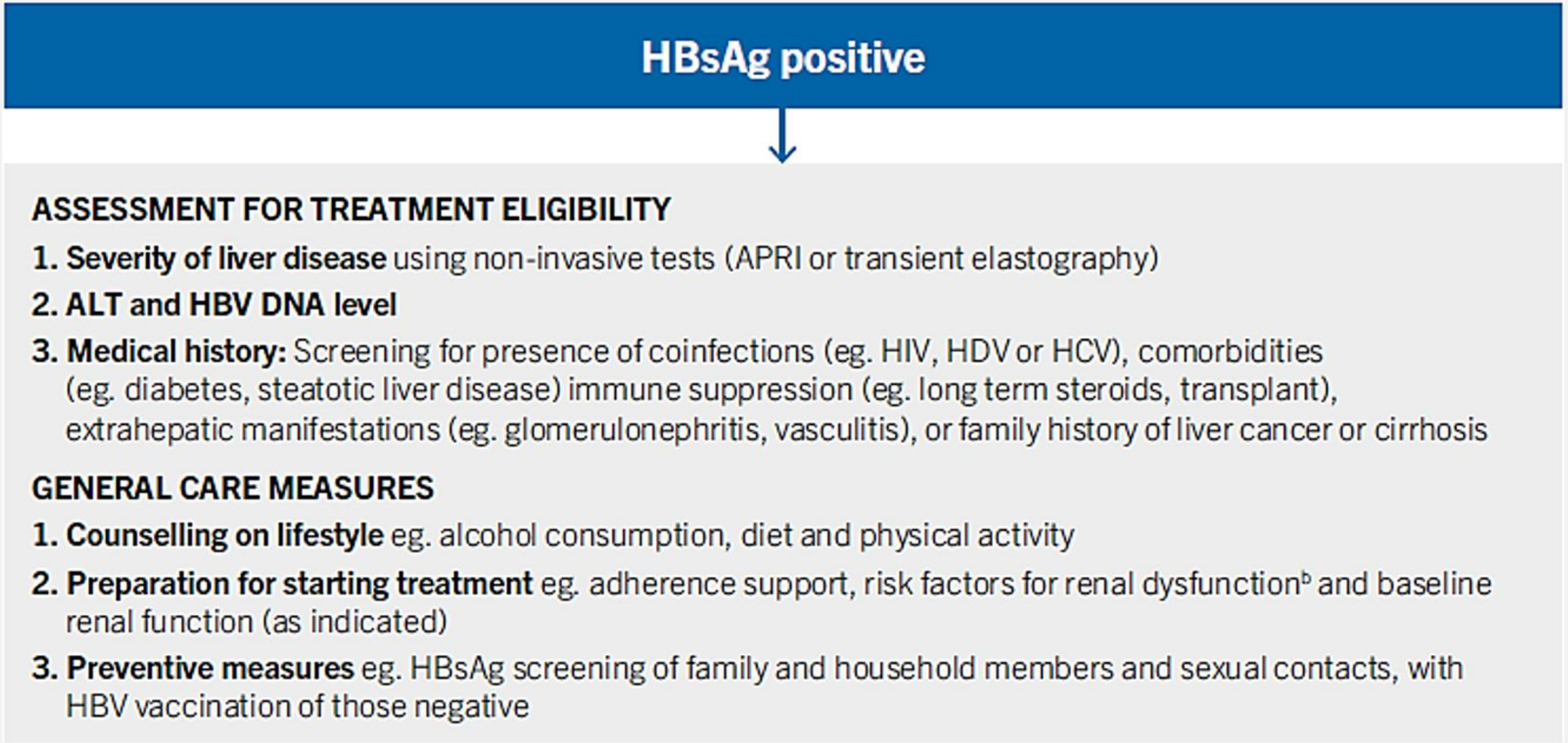
- Pregnant women
- Clinical suspicion of liver disease*
- High-risk adolescents
- Blood donors
- Migrants from high-endemicity areas
- Close contacts* (household & sexual)

KEY POPULATIONS

- Multiple sexual partners including CSW
- MSM
- Transgender persons
- Prisoners & closed-setting populations
- PWID
- PLWH
- HCWs

** applies to children, adolescents and adults*

02 Stage



Initial assessment algorithm ·WHO 2024

03 Treat

SIMPLIFIED CRITERIA

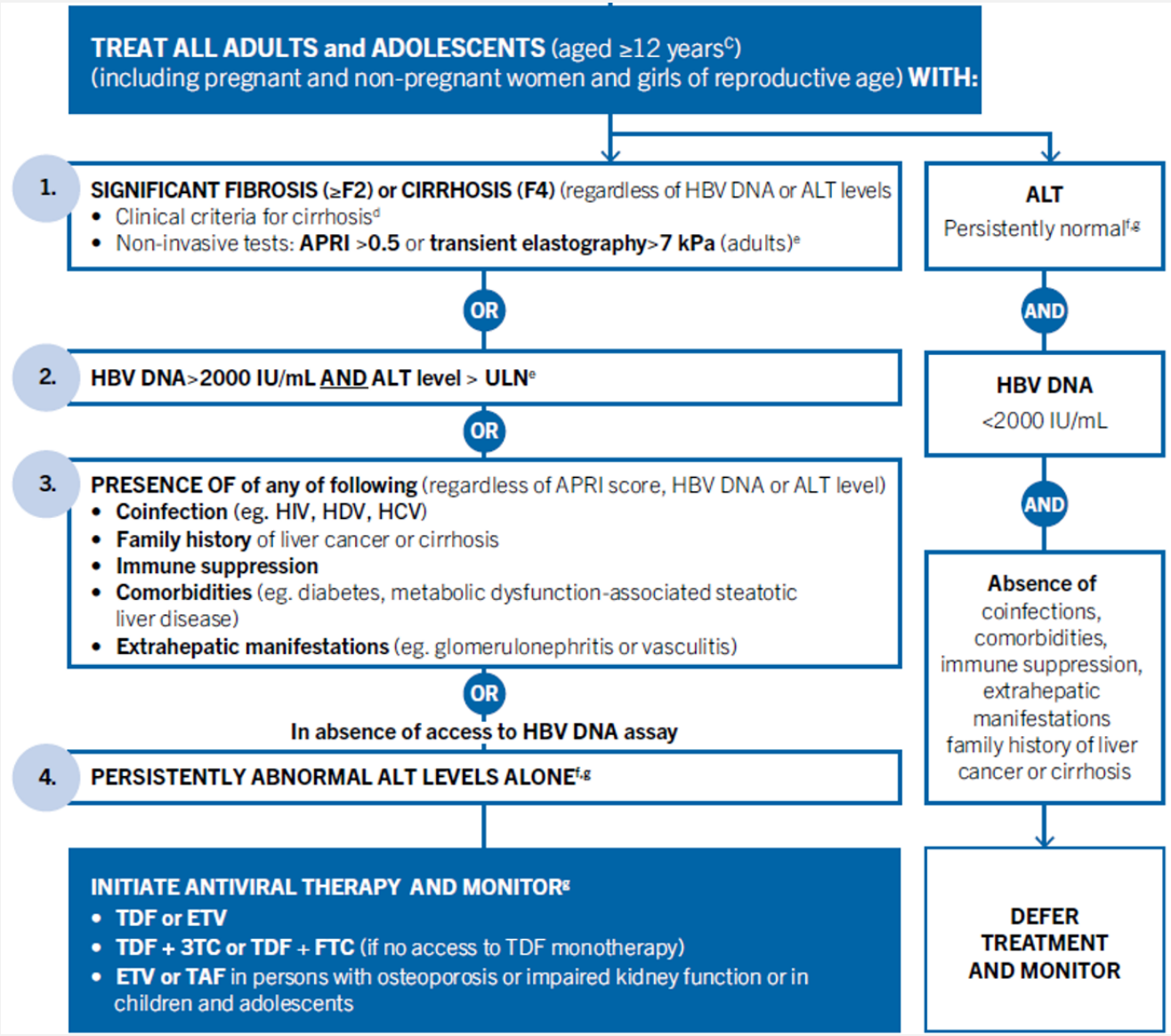
01 Cirrhosis APRI >0.5 or Fibroscan >7 kPa

02 HBV DNA >2000 and ALT > ULN

03 Viral co-infections

04 Other

- Family history HCC
- On immunosuppressants
- Diabetes
- MASLD
- EHM



Treatment eligibility algorithm WHO 2024

Evaluating Liver Fibrosis

INVASIVE

Liver biopsy

Only if considering pegylated interferon for treatment or exploring differential diagnoses

NON-INVASIVE

Transient elastography (Fibroscan®)

Best tool but expensive; treatment threshold >7 kPa

Fibrosis scores

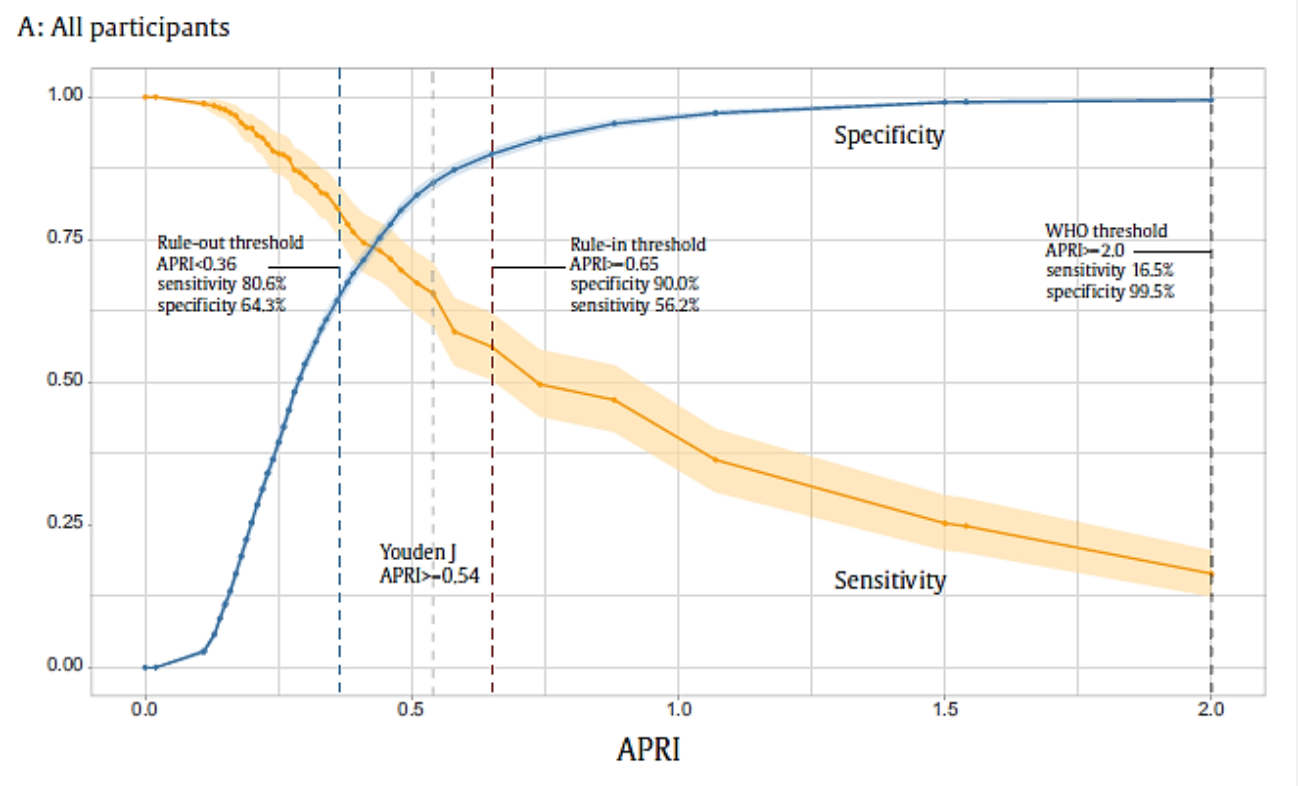
- APRI (WHO recommended)

  AST-to-**P**latelet **R**atio **I**ndex

  [AST/AST(ULN)/Platelet count/mm^3] x100

  Score >0.5 predicts significant fibrosis

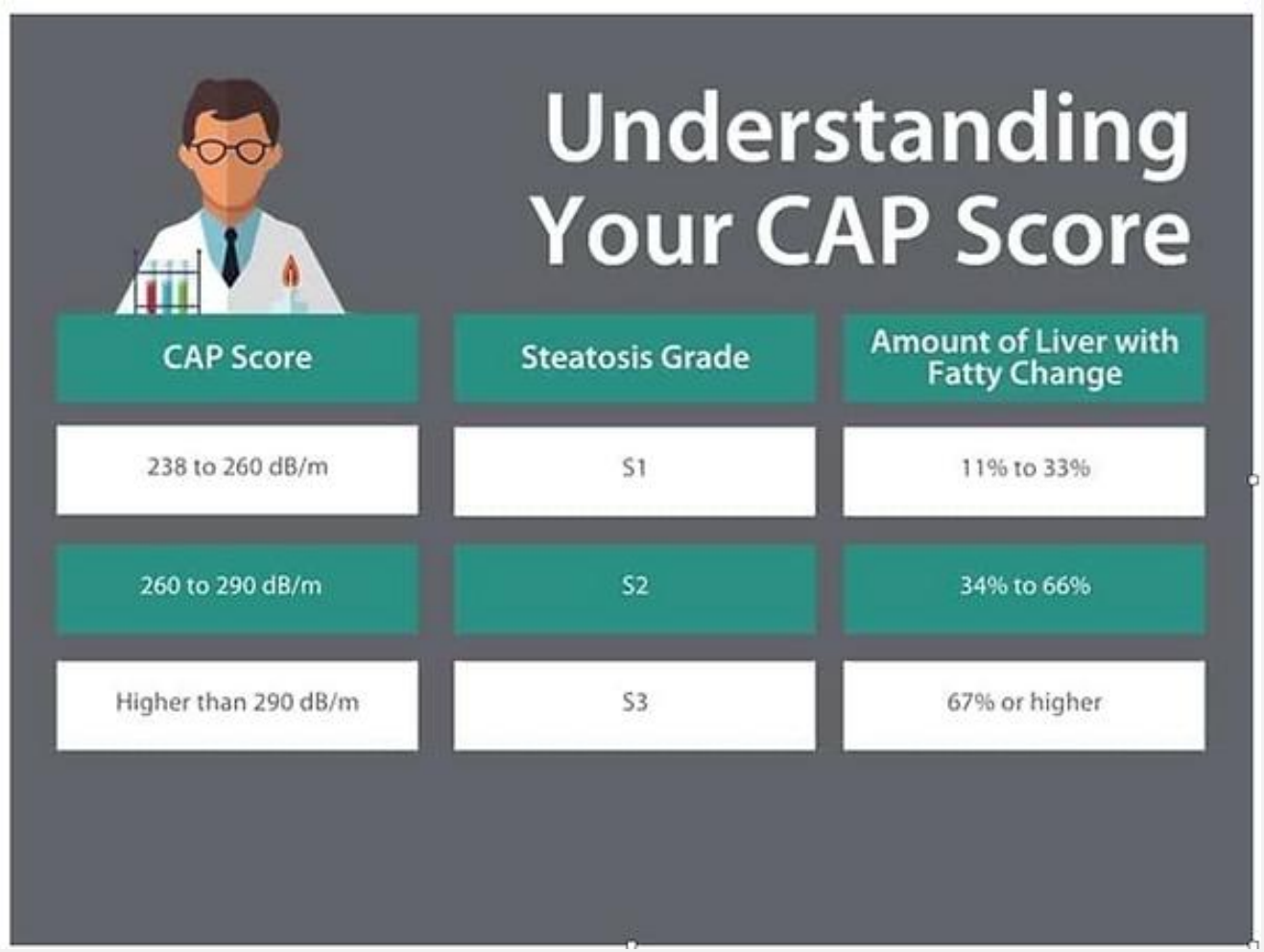
- FIB-4 Alternative composite score



Johannessen et al 2023, N = 3548 Meta-analysis in African populations

Challenged previous WHO APRI cut-off of ≥ 2

Fibroscan® How it Works

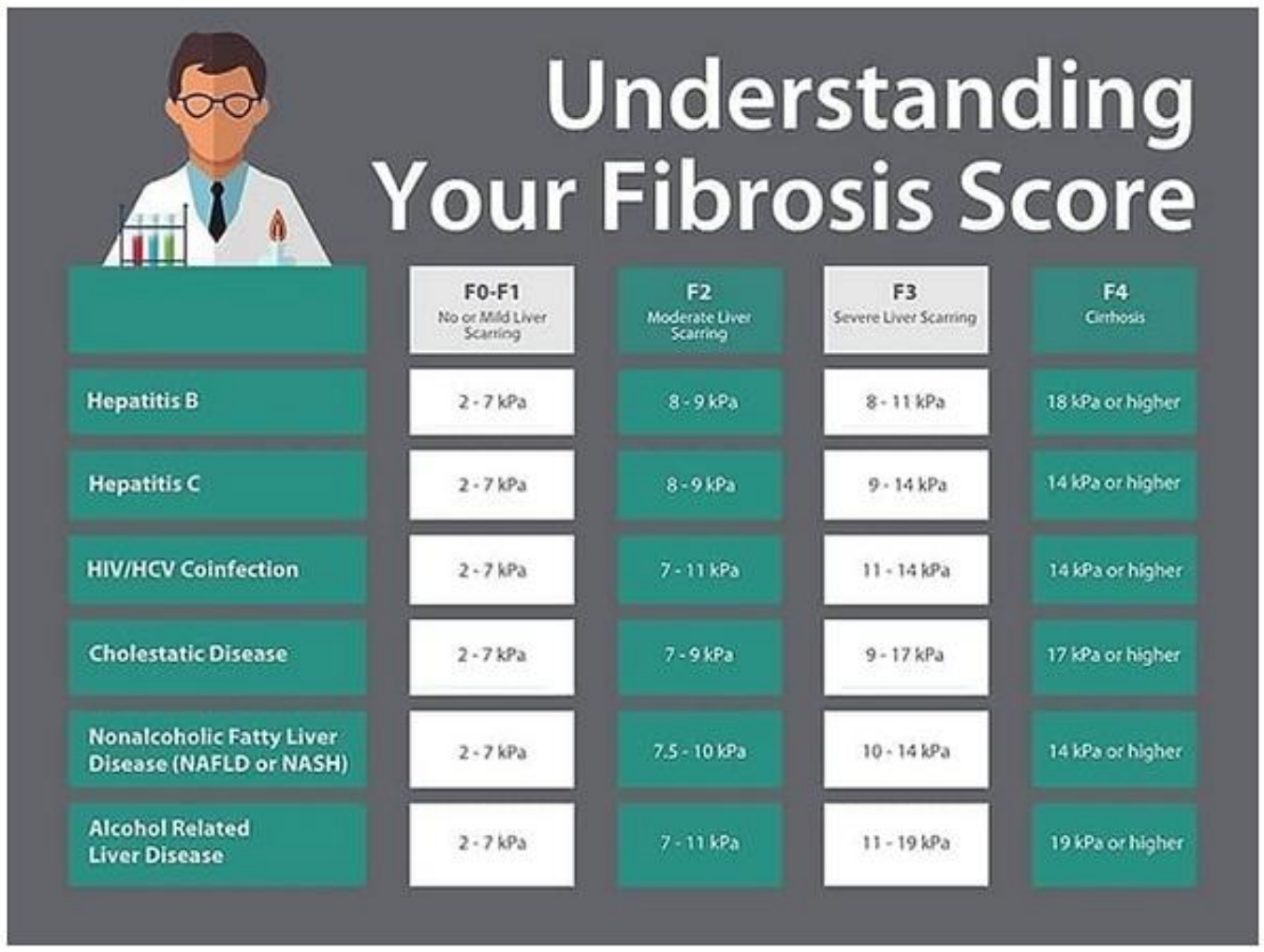


The probe sends a shear wave through the liver — its velocity is proportional to stiffness

Source · fibroscantt.com

Fibroscan® Interpreting Fibrosis Results

Fibroscan readout · correspondence with METAVIR fibrosis stages



	F0-F1 No or Mild Liver Scarring	F2 Moderate Liver Scarring	F3 Severe Liver Scarring	F4 Cirrhosis
Hepatitis B	2 - 7 kPa	8 - 9 kPa	8 - 11 kPa	18 kPa or higher
Hepatitis C	2 - 7 kPa	8 - 9 kPa	9 - 14 kPa	14 kPa or higher
HIV/HCV Coinfection	2 - 7 kPa	7 - 11 kPa	11 - 14 kPa	14 kPa or higher
Cholestatic Disease	2 - 7 kPa	7 - 9 kPa	9 - 17 kPa	17 kPa or higher
Nonalcoholic Fatty Liver Disease (NAFLD or NASH)	2 - 7 kPa	7.5 - 10 kPa	10 - 14 kPa	14 kPa or higher
Alcohol Related Liver Disease	2 - 7 kPa	7 - 11 kPa	11 - 19 kPa	19 kPa or higher

Metavir Scoring System for Fibrosis staging	
F0	No Fibrosis can be detected
F1	Fibrosis exists with expansions of portal zones
F2	Fibrosis exists with expansions of most portal zones and occasional bridging
F3	Fibrosis exists with expansion of most portal zones, marked bridging, and occasional nodules
F4	Presence of cirrhosis

Source: fibroscantt.com

Treat

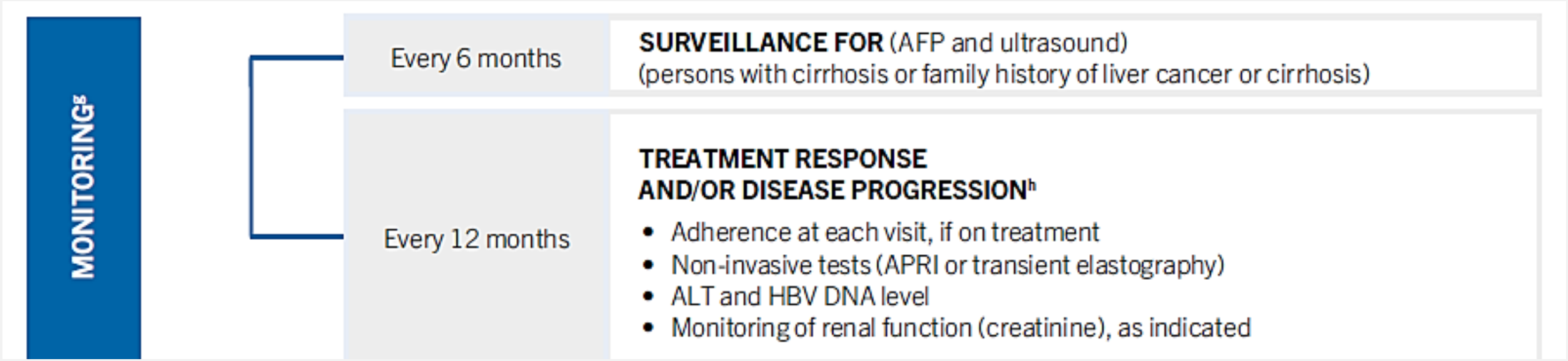
Antiviral drug	Potency against HBV	Resistance barrier	Activity against HIV	Cost
Interferons	Moderate	Not applicable	Moderate	High
Tenofovir disoproxil fumarate	High	High	High	Low (high in Hong Kong SAR, China and elsewhere in Asia)
Entecavir (ETV)	High	High	Weak	Low
Tenofovir alafenamide fumarate (TAF)	High	High	High	High

- **THE GOAL – FUNCTIONAL CURE**
  - Sustained loss of HBsAg **and** undetectable HBV DNA after stopping treatment (usually ≥ 6 months off therapy)
  - Rare in practice – achieved in ≤ 1 % of most cohorts
    - cccDNA and integrated HBV DNA persist in hepatocytes as viral reservoirs

WHO 2024

Follow-up

MONITORING THE HBV-POSITIVE PATIENT



IF ON TREATMENT
Every 6 months

IF NOT YET ELIGIBLE
Annual reassessment

WHO 2024

PREGNANT WOMEN

Two parallel goals

01
Prevent mother-to-child transmission

02
Reduce maternal HBV disease progression

WHO 2024 Priorities for Antenatal HBV Care

01 **Universal ANC HBV screening by HBsAg**

- First test ideally during 1st trimester of ***each*** pregnancy
- If HBsAg negative → vaccination (safe in pregnancy)

02 Expanded access to HBV DNA testing

03 Simplified pathway cascades and linkage to treatment

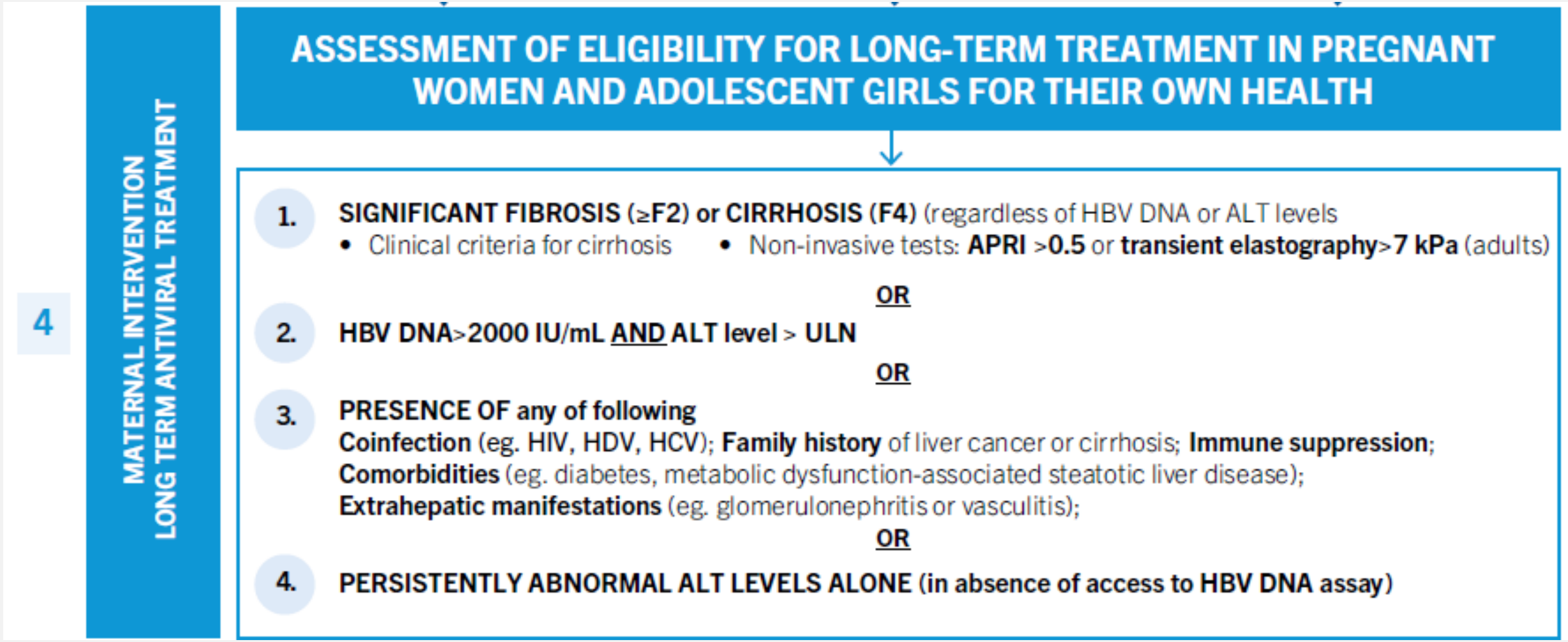
04 Simplified treatment eligibility algorithms

05 Integration into maternal HIV services

- Triple screen HIV/HBV/Syphilis

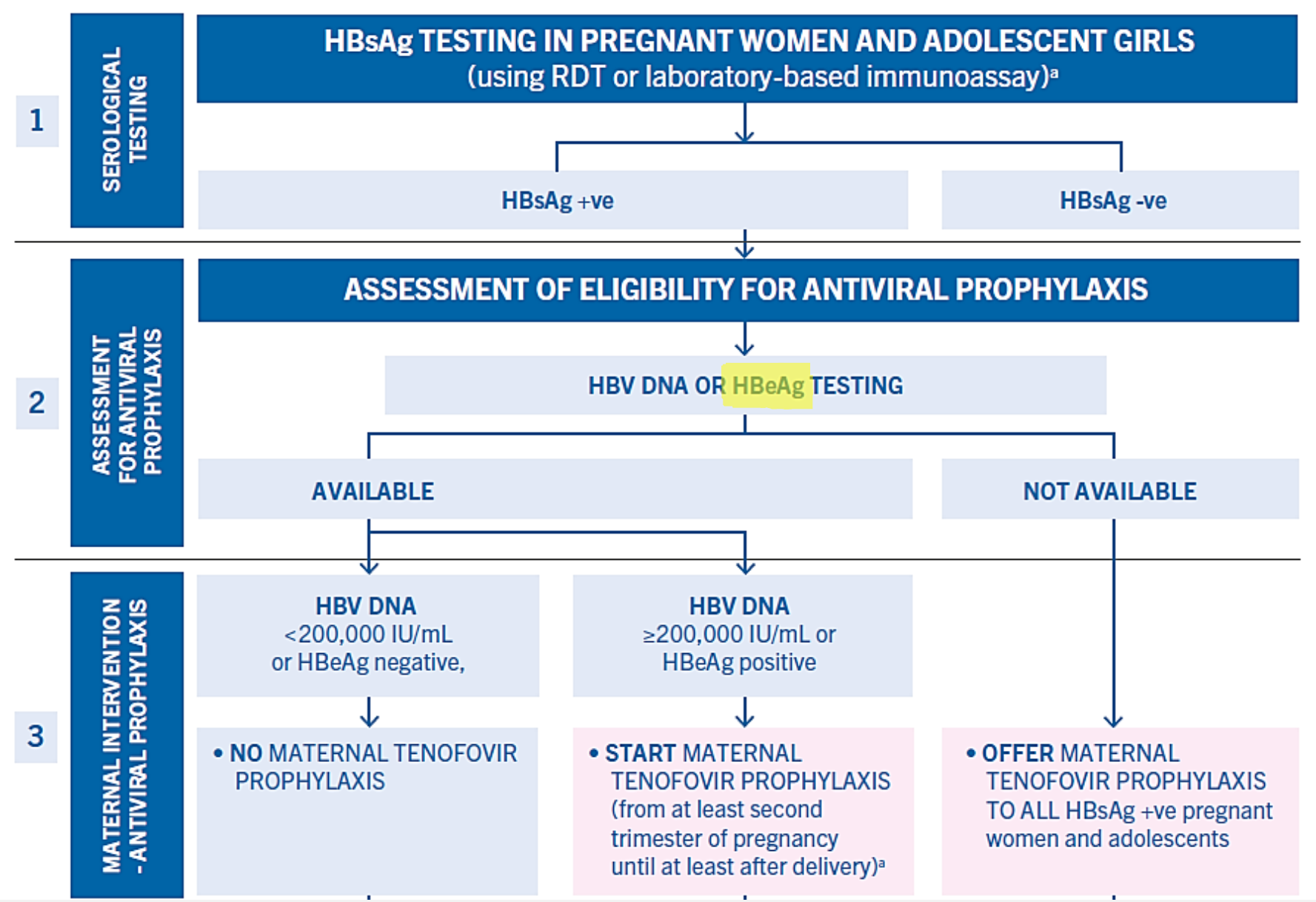
Source: angelp/iStockphoto/Getty Images

Treatment Eligibility



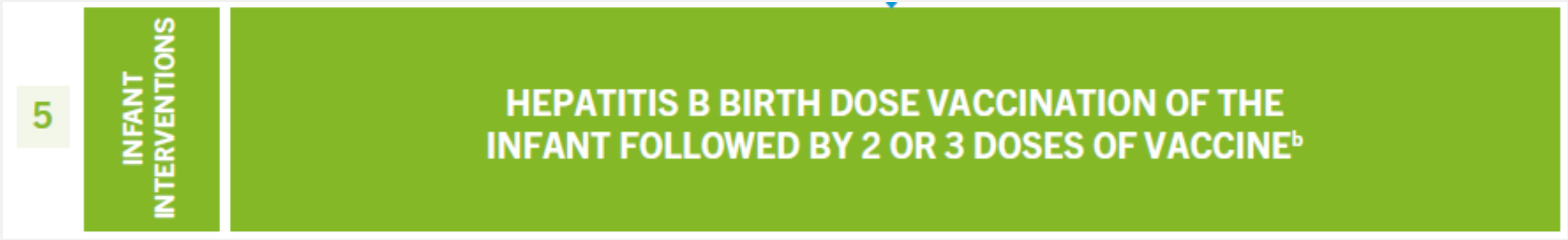
WHO 2024

Antiviral Prophylaxis



WHO 2024

Infant Interventions



- **Timely HB birth-dose vaccine** within **24 hours** of delivery
  - Given as the **first dose of the HB infant schedule** — regardless of the mother's HBsAg status
- **HBIG** (often out-of-stock in our setting) recommended for infants born to HBsAg-positive mothers
  - Especially those with high HBV DNA
- **Breastfeeding is safe** — no transmission risk
  - Beware of cracked, bleeding nipples in mothers with high HBV DNA
- **Caesarean section** for usual obstetric indications only — *not* solely for HBV PMTCT

WHO 2024

HEALTHCARE WORKERS

Two key actions

01 Screen
Confirm HBsAg and Immune Status

02 Vaccinate
Full 3-dose schedule for non-immune HCWs

The HBsAg + HCW

01 TREATMENT

Are they eligible for treatment?

Follow the same WHO recommendations as any other adult

02 OCCUPATIONAL RISK

Risk of occupational transmission?

For exposure-prone procedures (EPPs):

- HBV DNA > 200 IU/mL → **treat** with antivirals
- Return to EPPs once HBV DNA below the limit of detection (or * < 200 IU/mL)

03 PRACTICE RIGHTS

- HBV infection **does not** exclude a HCW from the practice of medicine
- Maintain standard IPC and occupational safety measures

** Western guidelines threshold · WHO uses < 2 000 IU/mL*

CO-INFECTIONS

Viruses in concert

01 HIV

02 HCV

03 HDV

HIV/HBV Co-infection – Overview

THE REGIONAL BURDEN

2.6 M

HBV / HIV co-infected adults in sub-Saharan Africa
Often reflects childhood-acquired HBV and adult-acquired HIV

LIVER-RELATED MORTALITY

2 X HIGHER

Versus HBV mono-infection

HIV INCREASES HBV MORBIDITY

↑ Risk of **perinatal HBV transmission**

↑ Likelihood of progression to **chronic HBV**

↑ Progression to **fibrosis & cirrhosis**

↑ **Persistent HBeAg** positivity

↑ Risk of **acute liver failure**

↑ Risk of **occult HBV** (HBsAg-negative, DNA-positive)

↑ **HCC younger age of onset** & more aggressive course

HIV/HBV Co-infection – Treatment

TREATMENT IMPROVES OUTCOMES

What you can expect on therapy

- High rates of HBV DNA suppression, HBeAg and HBsAg loss – comparable to HIV-negative cohorts
- No evidence of HBV resistance among co-infected populations
- Reduced progression to cirrhosis
- Lower but persistent risk of HCC

Preferred NUC

Tenofovir POTENT ANTI-HIV ACTIVITY

- Avoid 3TC / FTC monotherapy – low genetic barrier to resistance
- Entecavir does **not** have reliable anti-HIV activity

WATCH FOR HBV-IRIS

Potentially higher risk of HBV-IRIS leading to **hepatic decompensation**, especially in ART regimens *without* tenofovir and where adherence is poor

De Vries-Sluijs et al, 2010 · Plaza et al, 2013

HIV/HBV Co-infection – Viral Load Discordance

THREE DISCORDANCE PATTERNS

PATTERN 01 · MOST COMMON
Suppressed HIV VL + **high** HBV DNA

PATTERN 02
Suppressed HIV VL + **partially suppressed** HBV DNA

PATTERN 03
High HIV VL + **low** HBV DNA

WHAT THIS MEANS IN PRACTICE

- Expect **slower HBV suppression** – > 1 log drop over 3 months is reasonable
- HBV DNA may still be detectable after 1 year if baseline VL was very high — *do not* reflexively switch therapy

WHY THIS HAPPENS

- **Different viral biology** – HIV reservoir is fast-decaying CD4 cells; HBV reservoir is slow-decaying hepatocytes
- Different **immune control mechanisms** for each virus
- Different **timing of acquisition** – childhood HBV vs adult HIV
- HBV **resistance patterns** – 3TC monotherapy and *YMDD* mutations.
- ART **regimen effects**, e.g. poor adherence in elite controllers

HBV/HDV Co-infection

HBV / HCV and HBV / HDV co-infections: cause **more severe hepatitis,** accelerated progression to cirrhosis and HCC

HBV / HCV

CLINICAL PATTERN

- HBV DNA levels typically low and HCV is the primary driver of liver inflammation
- Risk of HBV reactivation during and after HCV clearance by DAAs

THEREFORE ·IF HCV CO-INFECTED

① Treat HBV first

② Once HBV DNA suppressed, initiate DAA for HCV

③ Continue HBV therapy for **12w** after DAA completion (unless eligible for long-term HBV treatment)

HBV / HDV

THE BASICS

- HDV is a small defective RNA virus - requires HBV to replicate
- In Africa, **low seroprevalence** south of the Equator, much higher to the north

TWO CLINICAL SCENARIOS

Simultaneous	**Super-infection**
Severe acute hepatitis & ALF	In established CHB
Recovery often complete Chronic infection rare	Usually leads to chronic hepatitis D (CHD)

Prevention

Prevent transmission – at every contact point

01 ANTENATAL

Screen & prophylax

- **Antenatal HBsAg screening** for every pregnancy
- **Antiviral prophylaxis** in eligible pregnant women

02 VACCINATION

Cover every life-stage

- **BDV within 24 h** to prevent perinatal transmission
- Complete the **3-dose infant schedule** to prevent horizontal transmission in < 5y
- Vaccinate **non-immunes:** close contacts, pregnant women and HCWs; PEP for percutaneous injuries
- Vaccinate **targeted groups:** immunocompromised, chronic liver disease, haemodialysis, key populations, travellers to high-endemicity areas

WHO Global Hepatitis Report 2026

Key take-away: HBV management belongs in PHC

01 **Screen widely**
In SA's intermediate-prevalence setting, standard is **universal adult HBsAg screening** – not just risk-based

02 **Lower treatment eligibility thresholds**
Use non-invasive methods **(APRI > 0.5)** to predict liver fibrosis

03 **Tenofovir is first-line**
Especially for HIV co-infection. Avoid 3TC/FTC monotherapy. Treatment is lifelong unless clear stopping criteria

04 **Antenatal care = HBV care**
Each pregnancy warrants an HBsAg test. Every newborn should receive BDV **within 24 h of delivery**

05 **Screen for HIV and HCV co-infections in CHB**
Co-infection doubles liver-related mortality. Choose a tenofovir-containing regimen for HIV/HBV and treat HBV before HCV

06 **Prevent transmission** through vaccination and antenatal antiviral prophylaxis

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