Hepatitis B and C

Treatment Guidelines for Nigeria

Compiled by

Society for Gastroenterology & Hepatology in Nigeria

Contents

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1. Introduction

Viral hepatitis refers to a group of inflammatory diseases of the liver caused by viruses that have an affinity for the liver. Five viruses have been found to be clinically relevant and they are labeled A, B, C, D and E.

Hepatitis B virus (HBV) and hepatitis C virus (HCV) have been found to be the most important causes of chronic liver disease such as liver cirrhosis and liver cancer ultimately resulting in premature death.

Worldwide serological evidence of past or present infection with HBV is found in about 2 billion people, of this number, 350-400 million are chronically infected and results in about 1 million deaths annually.

Nigeria is an area of high endemicity for HBV with over 70% of the population showing evidence of past infection with the virus. 7.3 - 24% of the populace has serological evidence of current infections (average 13.7%). Going by the 2006 national census, this translates to 19 million Nigerians being currently infected and about 5 million of these will die of the consequences of this infection.

For HCV, the prevalence worldwide is 170 million chronically infected people. The range of prevalence of HCV infection in Nigeria is between 0.5 - 4% in the general population.

The liver disease caused by these 2 viruses is silent and unrecognized. The impact is often underestimated by both the sufferers and the healthcare professionals. Consequently most patients present very late with advanced liver cirrhosis and cancer.

HBV is both preventable (vaccination) and treatable if diagnosed early; currently, there is no vaccine for HCV; however treatment is available if diagnosed early.

1.1 Rationale for management guidelines:

a) Improve awareness and appreciation for HBV/HCV disease and management among healthcare workers.

b) To standardize management and decision making in the approach to HBV/HCV diseases at various levels of healthcare.

c) To adapt the criteria in management of HBV/HCV to the peculiarities in our environment.

d) To facilitate and standardize research into HBV/HCV-related diseases.

2. Hepatitis B

Hepatitis B refers to a viral disease process caused by the hepatitis B virus. Transmission occurs vertically (perinatally from mother to child), horizontally in childhood (during play or intrafamilial non-sexual contact) or horizontally in adolescence/adulthood (sexual contact, contaminated needles/sharp objects, blood transfusion). Providing education about how to avoid risky behavior is essential in improving prevention.

Chronic hepatitis B virus infection is a major cause of end-stage liver disease and hepatocellular carcinoma. The terms 'dynamic, controllable but not curable' are applied to the disease because of the ability of the virus to remain in the hepatocyte nuclei thus posing a lifelong threat of disease reactivation to the patient. This emphasizes the need for regular and life-long monitoring of patients for disease activity.

In Nigeria, most infections occur in childhood (vertical and horizontal transmission). About 70-90% of vertical infections will result in a chronic infection compared with 20-50% of early childhood infections (horizontal) which will progress to the chronic stage. In contrast, when transmission occurs in adolescents or adults, only 1-3% will progress to the chronic infection unless if the individual is immunocompromised.

For a country of high endemicity like Nigeria, studies have shown that universal vaccination at birth is cost effective. However, for children, adolescents and adults not vaccinated at birth, screening would be essential to establish HBV status. Those who are unexposed and are negative for anti-HBc and anti-HBs should be vaccinated while those who are HBsAg positive, should be evaluated for further management.

3. Who to screen?

All Nigerians are at risk and should be counseled and screened for HBV infection at any available opportunity.

Opportunities for screening include:

- Any visit to hospital/clinic
- Preschool entry
- Pre-employment assessment
- Pre-insurance
- Pregnancy
- Blood / organ donation
- Contact tracing through identified cases (siblings, spouse, household contacts, sexual contacts).

healthcare workers are at a particularly high risk of acquiring HBV infection and should be screened routinely.

Screening tools: Screening tools include Hepatitis B surface antigen (HBsAg), antibodies to the surface antigen (anti-HBs) and antibody to the core antigen (anti-HBc).

Recommendation: In Nigeria, HBsAg should be used for general screening (since it is more easily available and more importantly identifies individuals at risk of disease progression / premature death).

All positive subjects require further evaluation for treatment.

All negative subjects should be evaluated for vaccination.

3.1 Special subjects requiring screening for HBV infection:

- Abnormal liver function tests (LFT) especially elevated ALT of unknown cause.
- Haemoglobinopathies / blood disorders with frequent blood transfusions.
- Patients who develop jaundice or increased aminotransferases after blood transfusion.
- Patients with cirrhosis or suspected hepatocellular carcinoma (HCC).
- Spouses, siblings, parents and children of HBsAg positive patients.
- HIV positive patients.
- Chronic renal failure (CRF) patients, especially if haemodialysis is planned.
- Patients with Chronic Hepatitis C.
- Candidates for chemotherapy or immunosuppressive treatment including organ transplant.

3.2 Further evaluations to be carried out:

Clinical evaluation

a) **History:** Alcohol use, traditional herb use, cigarette smoking, risk factors for co-infection with HIV, family history of liver related death, etc.

b) **Features of advanced chronic liver disease:** Jaundice, weight loss, hepatomegaly or shrunken liver, splenomegaly, asterixis, bleeding tendencies, spider naevi, gynaecomastia, distended anterior abdominal veins, testicular atrophy, breast atrophy, brittle hair, female hair distribution in males, fluid retention, parotid swelling, palmar erythema, erythema on soles, finger clubbing, leuconychia.

Laboratory diagnosis

Phase 1

Initial evaluation for HBsAg positive subjects (mainly asymptomatic patients)

a) **Viral markers:** IgM anti-HBc, IgG anti-HBc, HBeAg, anti-HBe, anti-HBs, anti-HDV*, anti-HCV, anti-HEV*, anti-HAV*, HIV screening.

b) **Assessment of hepatic injury/severity:** Aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), bilirubin, albumin, prothrombin time (PT).

c) **Full blood count** (including platelet count).

d) **Liver imaging:** Ultrasound / CT to determine features of possible advanced liver disease and hepatocellular carcinoma (HCC).

* may not be routinely available.

Phase 2

Patients who are negative for IgM, anti-HBc indicating chronic infection and patients with no features / risk factors of adult aquired hepatitis B.

d) **Molecular biology:** HBV DNA viral load assessment.

e) **Liver biopsy:** If safe and acceptable to the patient, a liver biopsy can be done to grade and stage liver injury in those with high HBV DNA ± high ALT. This may aid treatment decisions in patients not meeting clearcut indications for treatment. Liver biopsy should be processed with special stains including: Masson's trichrome (fibrosis), orcein (elastin fibers, HBsAg), Periodic acid Schiff-diastase (alpha-1 antitrypsin deficiency), reticulin (fibrosis, collapse, HCC), Perls' iron (haemosiderin and iron products) immunohistochemistry for HBsAg, HBcAg, HDAg, AFP, copper (Wilson's disease).

Liver biopsy findings should be categorized into mild, moderate or severe chronic hepatitis or, better still, semi- quantitatively scored by a scoring system like the Knodell Histological Activity Index (HAI). Comments about degree of fibrosis should also be included.

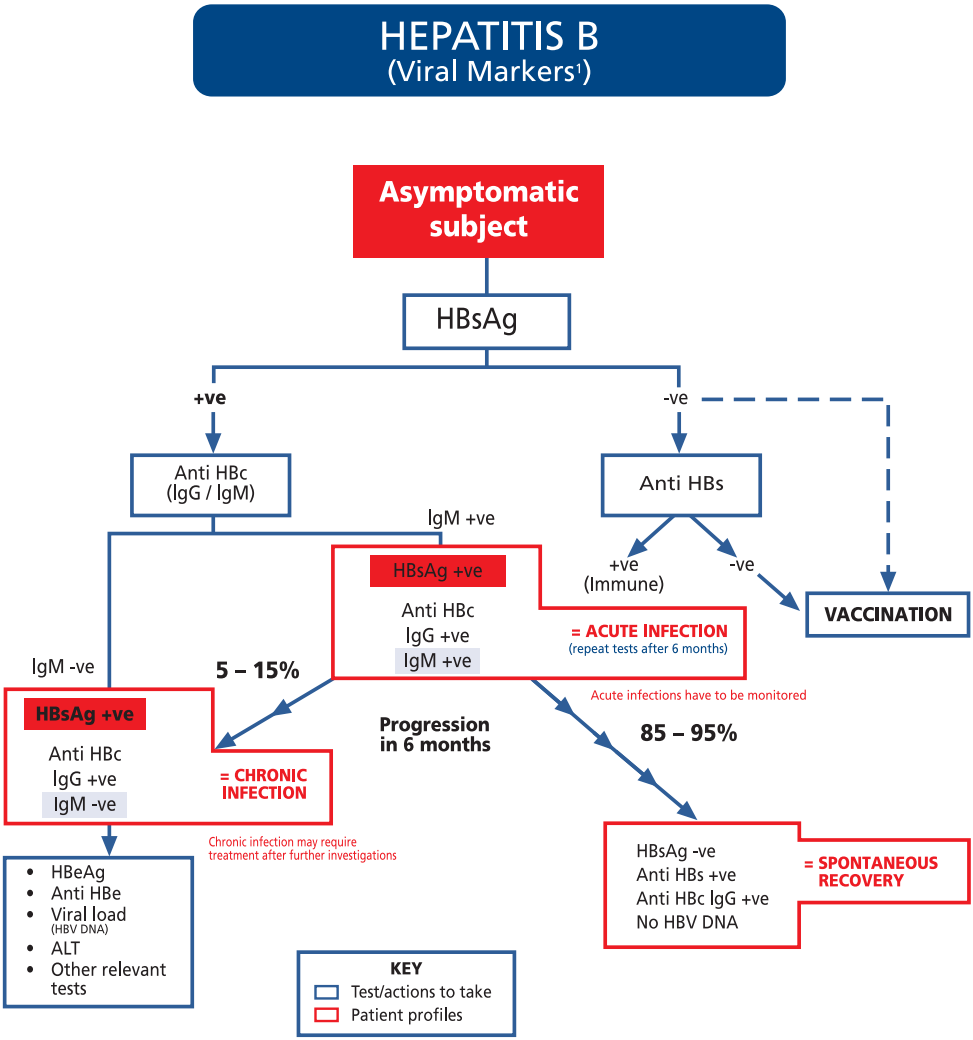
Categorization of subjects after evaluation

Based on the findings of the evaluation above, subjects may be categorized into the following phases:

TABLE 1: Differentiation of Chronic Hepatitis B infection

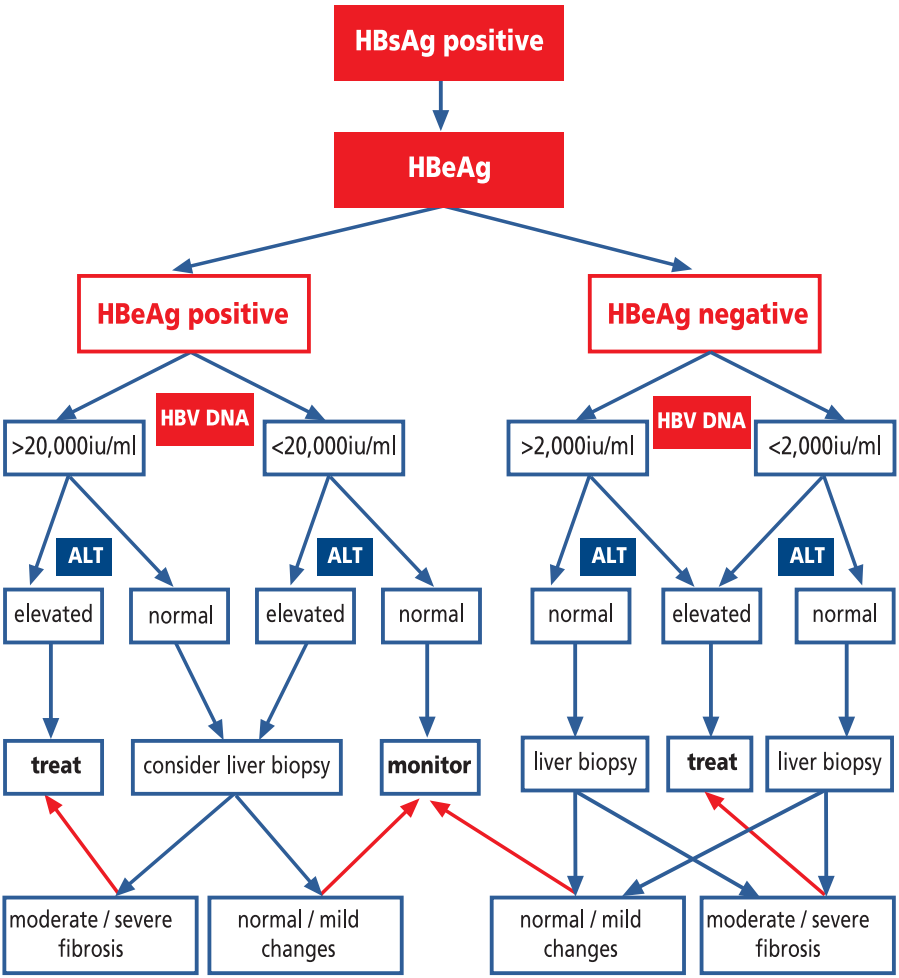
Phase	ALT	Liver histology	HBV-DNA	HBe antigen	Anti HBe	Action
Immune tolerance	Normal or minimally elevated	Normal or Mild activity, no fibrosis	>20,000 IU/ml (>10^5 copies/ml)	positive	negative	monitor
Immune clearance (HBe positive CHB)	Elevated/ fluctuating	Active	>20,000 IU/ml (>10^5 copies/ml)	positive	negative	treat
Inactive HBsAg carrier state	Persistently normal	Inactive, usually with minimal fibrosis	Low, or undetectable <2000IU/ml (<10^4 copies/ml)	negative	positive	monitor
Reactivation (HBe-negative CHB)	Elevated/ often fluctuating	Active with variable amounts of fibrosis	Moderate, often fluctuating, >2000IU/ml (>10^4 copies/ml)	negative	positive	treat

Fig.1: Algorithm of diagnostic workup



Subjects in the immune tolerance and inactive carrier phases are not considered candidates for antiviral therapy. However they will need to be monitored every 3-6 months with serum ALT and HBV DNA.

Fig 2: Algorithm for patient management



4. Management Strategies

Pre-treatment counseling:

It is important that patients are fully informed in simple terms about the following in order to improve compliance:

- The health implications of chronic hepatitis B infection.
- The chronic nature of the disease: monitoring is life-long and treatment may be prolonged / life-long.
- The possibility that spouse(s), children and close relatives may be infected and the need to screen them.
- The need to avoid further health risks such as alcohol and traditional herbs.
- The financial implications of treatment options in relation to the desired goal of treatment.
- Potential side effects of the treatment options should be discussed.
- The objectives and likely outcomes of treatment should be discussed in terms of virological response, normalization of liver functions and prevention or reduction in the risk of further liver damage and liver cancer.

Goals of treatment:

- To achieve a sustained virological response (SVR).
- To prevent liver disease progression to cirrhosis, liver failure and liver cancer.
- To prevent transmission of HBV infection.
- To improve quality of life.

Goals of Treatment Based on HBeAg Status

HBeAg positive	HBeAg negative
Primary Goals - HBeAg seroconversion - HBV DNA viral suppression - ALT normalization	**Primary Goals** - HBV DNA viral suppression - ALT normalization
Secondary Goals - HBsAg seroconversion	**Secondary Goals** - HBsAg seroconversion

The ultimate goal of treatment = HBsAg seroconversion

5. Treatment Options

a. Conventional interferon alpha 2a (IFN)

b. Pegylated interferon alpha 2a (PEGASYS®)

c. Pegylated interferon alpha 2b*

d. Lamivudine

e. Adefovir dipivoxil*

f. Entecavir*

g. Telbivudine*

h. Tenofovir**

*not yet available in Nigeria
**not yet licensed for HBV monoinfection.

Table 2: Comparative Analysis Of Treatment Options For HBV In Nigeria

Drug	Dose	Advantages	Disadvantages
IFN	4.5-9miu three times wkly s.c	• Finite treatment duration • More durable HBeAg seroconversion • HBsAg loss in HBeAg positive disease • No resistance	• Less favorable response rates compared to peg IFN • Tolerability • Less favorable safety profile • High cost • Sc admin
Pegylated interferon alpha 2a (PEGASYS®)	180mcg wkly s.c	• Very favorable response rates in both HBeAg -ve and +ve disease • Finite treatment duration • More durable HBeAg seroconversion • HBsAg loss in HBeAg positive and negative disease • No resistance	• Tolerability • Less favorable safety profile • High cost • Sc admin
Lamivudine	100mg daily p.o	• Well tolerated • Safe in decompensated disease • Oral admin • More affordable	• Less durable HBeAg seroconversion • Potentially life threatening ALT flares on discontinuation of therapy • Resistance in 70% after 5 years therapy

Drug	Dose	Advantages	Disadvantages
Adefovir	10mg daily p.o	• Well tolerated in advanced liver disease • Effectivet against wild type and Lamivudine resistant HBV • Low rate of HBV resistant in first 2 years	• Higher cost • Resistant HBV mutants in 29% after 5 years • Nephrotoxic at higher doses
Entecavir	0.5mg daily p.o	• Well tolerated • Potent HBV-DNA suppression • Low rate of HBV resistance at 5 years (1.2% at 5 years)	• Higher cost • No long term data in HBV infection • Less effective in some Lamivudine-resistant HBV patients
Tenofovir	300mg daily p.o	• Well tolerated, but limited data in HBV patients • Potent HBV-DNA suppression • Effective against wild type and Lamivudine resistant HBV	• Higher cost • Risk of nephrotoxicity • Lack of long term data in HBV infection

Table 3. Recommendations for therapy

HBeAg Status	HBV DNA (PCR)	ALT	Treatment Strategy
positive	≥ 20,000 IU/ml ≥ 10^5 c/ml	≤ 2 x ULN	• Low efficacy with current treatments • Observe; consider treatment when ALT becomes more elevated • Consider biopsy in persons > 20 years, with ALT persistently high normal to 2 x ULN, or with family history of HCC. • Treatment if HBV DNA ≥ 20,000 IU/ml (≥ 10^5 c/ml) and biopsy shows moderate/severe inflammation or significant fibrosis
positive	≥ 20,000 IU/ml ≥ 10^5 c/ml	> 2 x ULN	• Observe for 3-6 months and treat if spontaneous HBeAg loss fails to occur. Consider liver biopsy prior to treatment if no liver failure present. • Immediate treatment if icteric or if there is clinical decompensation. • PEG-IFN, Tenofovir and Entecavir preferred for initial treatment • Do not use IFN in decompensated disease

HBeAg Status	HBV DNA (PCR)	ALT	Treatment Strategy
positive	≥ 20,000 IU/ml ≥ 10^5 c/ml	> 2 x ULN	• LAM and LdT are not preferred due to the high rate of drug resistance but may be considered where the preferred first line is not available or affordable. • Adefovir not preferred due to low potency. • End point of treatment: seroconversion from HBeAg to anti HBe. **Duration of Therapy:** • IFN-α: 16-24 weeks; if no antiviral response, stop; if HBV DNA becomes undetectable, sufficient. • PEG-IFN-α: 24-48 weeks; if no antiviral response, stop; if HBV DNA becomes undetectable, sufficient. • LAM/ADV/ETV/LdT/TDF: Treat until seroconversion, continue for at least 6 months to 1 year after HBeAg seroconversion; cannot stop unless seroconversion occurs; but if ressistance occurs switch to IFN - α / PEG-IFN.

HBeAg Status	HBV DNA (PCR)	ALT	Treatment Strategy
negative	≥ 20,000 IU/ml ≥ 10^5 c/ml	> 2 x ULN	• End point of treatment not defined. • Liver biopsy preferred before initiation of therapy to evaluate severity of fibrosis. • PEG-IFN. Tenofovir and Entecavir are preferred as first choice. **Duration Of Therapy*** • IFN - α / PEG-IFN- α: 1 year or more. • LAM/ADV/ETV/LdT/TDF: Until loss of HBsAg.
negative	≥ 2,000 IU/ml ≥ 10^4 c/ml	> 2 x ULN	• Consider liver biopsy and treat if biopsy shows moderate / severe necroinflammation.
negative	≥ 2,000 IU/ml ≥ 10^4 c/ml	> 2 x ULN	• Liver biopsy for > 20 years; treat if there is moderate /severe necroinflammation; observe for < 20 years; treat if HBV DNA or ALT becomes higher. **Compensated Cirrhosis:** Treat using ADV/ETV/TDF. Long term therapy required. PEG-IFN - α may be used in a well compensated cirrhosis
negative	undetectable		**Compensated cirrhosis:** observe **Decompensated cirrhosis:** refer for liver transplant.

ADV- Adefovir; ETV- Entecavir; IFN-α- Interferon alfa; PEG-IFN-α- Pegylated interferon alfa , LdT - Telbivudine, LAM-Lamivudine; TDF-Tenofovir; ULN-Upper limit of normal.

6. Monitoring and evaluation

IFN and PEG-IFN

1) 4 weekly Full Blood Count, LFT,
2) 12 weekly Thyroid Function, HBV DNA,
3) 24 weekly HBsAg, HBeAg / anti-HBe

Oral NUCs

1) 12 weekly Full Blood Count, Electrolytes, Urea, Creatinine, LFT, HBV DNA (till undetectable for Tenofovir and Entecavir but continuously for other NUCs).
2) 24 weekly HBeAg / anti-HBe, HBV DNA (when undetectable with Tenofovir and Entecavir).
3) Annual HBsAg/anti-HBs.

7. Treatment response

- **Primary response:** Decreased viral load by more than 10 fold after 12 weeks of therapy.
- **Complete response:** Undetectable viral load after 24 weeks of therapy.
- **Partial response:** Reduction of viral load below 10,000 copies/ml but still detectable after 24 wks.
- **Inadequate response:** There is a decrease in viral load but remains above 10,000c/ml after 24 wks.
- **Sustained virological response**: Undetectable viral load 6 months after cessation of therapy.

- **Treatment failure:** Patients with primary non-response, partial virological response and virological breakthrough (resistance).
- **Primary non-response:** Less than 10 fold drop in HBV-DNA level at 12 weeks in a treatment compliant patient.
- **Virological breakthrough:** Increase in serum HBV-DNA by more than 10 fold after achieving a virological response during continued therapy.

8. Special groups

1. HBV/HCV co-infection

Co-infection with HCV is associated with a more severe disease and rapid progression to both liver failure and HCC.

Treatment: Treatment must be for the dominant infection while monitoring the latent one. The dominant infection is the one with the greater viral load. Latent infection may flare up after the dominant infection has been successfully treated.

2. HBV/HIV co-infection

HIV infection worsens the liver disease in HBV infected persons and is also associated with rapid progression to end stage liver disease.

Treatment: Simultaneous treatment of both diseases usually required. The preferred drug combinations include Tenofovir + Emtricitabine / Lamivudine in combination with a non-nucleoside RTI or protease inhibitor.

3. HBV/HDV co-infection

HDV co-infection is also associated with rapid progression to end stage liver disease. A high index of suspicion is required if the serum HBV-DNA is low and the ALT is high. Diagnosis is based on the presence of anti-HDV, HDV-RNA, or immunohistochemical evidence of delta antigen in the liver.

Treatment: Use IFN (conventional or pegylated) for 12 - 24 months.

4. Pregnancy

Standard care is to immunize the infant within 12 hours of delivery with Hepatitis B immunoglobulin and Hepatitis B vaccine. However, there are reports that indicate that treatment of pregnant women with high HBV-DNA levels in the last trimester with nucleoside analogues reduces the risk of intra uterine and perinatal transmission of the virus.

5. Chemotherapy (cancer patients) and immunosuppressive therapy (including transplant patients)

Reactivation of HBV infection may occur in this situation and may be accompanied by fatalities. Chronic HBV carriers should thus be commenced on anti-HBV therapy at least one week before the commencement of the above therapy and continued for 6 months after cessation. Note that patients should be on life long anti HBV suppression treatment post transplant.

6. Children

This category of patients is mostly in the immune tolerant phase of the disease and thus will require a longer period of observation.

Indications for treatment are similar as for adults namely elevated ALT, and or increased HBV-DNA.

Lamivudine and interferon have been successfully used in children.

9. Prevention of Hepatitis B

1) Health education for the public and health care providers.

2) Universal immunization (infants, children, adolescents) and implementation of national Programme on Immunization (NPI) scheme for HBV vaccination.

3) Contact tracing and immunization of non-immune persons.

4) Screening and vaccination of all special risk groups especially surgeons, laboratory workers, dentists, emergency workers and law enforcement agents.

5) Screening of pregnant women at ante-natal clinics and immunization of the non-immune. Immunization of all babies born to HBV-positive pregnant women immediately after birth.

6) Screening of all blood/organ donors and blood products before transfusion/transplantation.

7) Proper disposal of all sharp instruments (needles, lancets, blades, etc.)

8) Non recycling of disposable instruments used in medical procedures (needles, lancets).

9) Sterilization of all instruments used by traditional medical practitioners for invasive procedures e.g circumcision, tattooing, ear piercing, etc.

10) Easy availability of HB immunoglobulins for post-exposure prophylaxis.

11) Practice of ABC (Abstinence, Be faithful and Condom) as in the prevention of HIV infection should be encouraged.

10. Hepatitis C infection

The hepatitis C virus (HCV) is different from the hepatitis B virus (HBV) in many respects. HCV is a single strand RNA virus unlike HBV which is a double strand DNA virus. However, just like HBV, infection by HCV is usually insidious and most patients are asymptomatic or suffer only a mild attack. Routes of transmission are largely similar to those of HBV namely; transfusion of unscreened blood, or blood products, organ transplant, needle stick injuries, injection drug use, unsterilized medical instruments, body piercing, tattoos/scarification, sex, mother to child transmission (not as common as in HBV) and shared personal items. Mother to child transmission is not as effective as in HBV infection. However the risk for this increases in mothers who are HIV positive or who have very high HCV RNA titres in late pregnancy.

About 80% of those who are infected go on to become chronic carriers for many years; 10 - 20% develop chronic liver disease including cirrhosis and a further 10% develop hepatocellular carcinoma (HCC) after 10 - 20 years.

Unlike HBV, there is no immunity after an infection; this is thought to be due to the diversity and numerous strains of the virus. Furthermore, there are no vaccines against HCV.

In Nigeria, most studies on hepatitis C are not community based with the majority of reported studies among blood donors. Therefore the available data significantly under-represents the actual burden of the condition. The best estimate possible suggests the prevalence at between 0.5 - 4%.

Management of chronic hepatitis C (CHC) is somewhat complex and requires the immediate attention of doctors with specialized training in the condition.

11. Who to screen?

- Persons who have injected illicit drugs.
- Children born to HCV infected mothers.
- Healthcare, emergency, medical and public health workers.
- Persons at special risk: persons with hepatomegaly and/or unexplained serum ALT elevate levels, persons on haemodialysis, recipients of blood/blood products and organ transplants; HIV positive persons; HBsAg positive persons.
- Persons with STIs and high risk sexual behavior with multiple sexual partners; male homosexuals.
- Commercial sex workers.
- Pregnant women.

11.1 Screening tools

1) Antibody to HCV (3rd gen ELISA recommended)
2) HCV-RNA used for patients with HIV infection and end stage renal disease.

11.2 Investigations for HCV positive persons

- Confirmation of a positive anti HCV test using the most sensitive PCR available.
- Quantitative HCV-RNA assay.
- HCV genotyping.

11.3 Further Evaluations

1) LFT.
2) Full blood count including platelet count.
3) Prothrombin time.

4) Liver imaging.

5) Liver biopsy to determine the grade, stage and prognosis of liver disease.

6) Serological test for HBsAg and HIV.

11.4 Pre-treatment evaluation

1) Markers for auto immune disease.

2) Thyroid function tests.

3) Assess for alcohol and drug abuse.

4) Evaluate for depressive or psychiatric illness.

5) BMI.

6) Assess for insulin resistance using fasting blood sugar and serum insulin.

12. Management Strategies

Goals of treatment

- Achieve sustained virological response.
- Prevent complications of HCV infection such as liver cirrhosis and liver cancer.
- Reduce risk of infecting others.
- Improve quality of life.

Who should be treated?

1) Persons of at least 18 years of age.

2) Abnormal ALT values.

3) Compensated liver disease.

4) Acceptable hematological and biochemical indices.

5) Liver biopsy showing chronic hepatitis with significant fibrosis.

6) Persons willing to be treated and to conform to treatment requirements.

7) Acute Hepatitis C after 12 weeks of observation with no fall in HCV RNA titres.

Contra-indications to treatment

- Pregnancy.
- Continuing alcohol abuse.
- Hepatic decompensation.
- Severe cardiac disease.
- Uncontrolled psychiatric illness.
- Uncontrolled auto-immune disease.
- Untreated thyroid disorder.
- Under three (3) years of age.
- Uncontrolled seizure disorder.
- Severe co-morbidity e.g. severe hypertension, heart failure, poorly controlled diabetes mellitus, obstructive pulmonary disease.

Treatment

The current standard of care is a combination of peg IFN and Ribavarin. Duration and dosage of therapy depend on the genotype and the weight of the patient:

Genotype 1, 4, 5 and 6

PEG-IFN alpha 2a (PEGASYS®) 180µg (or PEG- IFN alpha 2b 1.5µg/kg) weekly s.c. for 48 weeks + weight based - Ribavirin p.o. (1000mg/day for those less than 75kg; 1200mg/day for those above 75kg) for 48 weeks.

Genotype 2 and 3

PEG-IFN alpha 2a (PEGASYS®) 180µg (or PEG- IFN alpha 2b 1.5µg/kg) weekly s.c. for 24 weeks + weight based - Ribavirin p.o. (1000mg/day for those less than 75kg; 1200mg/day for those above 75kg) for 24 weeks.

Response to treatment

- Treatment of genotype 1 achieves 40 - 50% SVR.
- Treatment of genotype 2 and 3 achieves 80 - 90% SVR.
- Combined SVR for all genotypes is 50 - 60% .

Monitoring response

1) HCV-RNA assessed at initiation of therapy and at 12 weeks for genotypes 1, 4, 5 and 6. Treatment may be discontinued if patient does not achieve early virological response (i.e. more than 2 log drop or 100 fold reduction in viral load). Patients, who achieve early virological response (EVR), should have viral loads assessed at 24 and 48 weeks. For patients who have an end of treatment response, the sustained virological response (SVR) should be assessed at 24 weeks after cessation of therapy.

   For genotype 2 and 3, HCV-RNA should be assessed at initiation of therapy and at 4 & 12 weeks for rapid and early virological response. End of treatment response should be assessed at the end of 24 weeks and SVR assessed 24 weeks after cessation of treatment.

2) Monthly hematological and biochemical profile.

3) Three monthly thyroid function test.

4) Monthly evaluation for depression.

Adverse effects of drug therapy

1) Flu - like syndrome (fever, chills, headaches, myalgia) which is self-limiting and can be controlled by anti-pyretics/analgesics e.g paracetamol.
2) Fatigue, weight loss, anorexia, diarrhea, alopecia.
3) Hematological derangements: anemia, thrombocytopenia, neutropenia.
4) Psychiatric disorder: mood changes, depression, insomnia, suicidal ideation, psychosis.
5) Metabolic abnormalities: glucose intolerance, thyroid disorders.
6) Lung complications: pneumnitis, pneumonia.
7) Dermatological problems: various skin rashes, pruritis.
8) Eye complications: retinal haemorrhages, retinal artery and venous occlusion.

13. Prevention of Hepatitis C

Currently there is no vaccine available against HCV infections. The following preventive measures are however advisable:

1) Screening of all potential blood and organ donors.
2) Sterilization of equipments and tools for surgical/invasive procedures.
3) Use of disposable syringe and needles.
4) Public health enlightenment campaign.
5) Observance of universal precautions in the care of high risk patients e.g dialysis patients.
6) Practice of ABC.

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