Prevalence of Hepatitis B and C Virus Co–Infection among HIV Positive Patients Accessing Care at Wuse District Hospital, ABUJA, Nigeria

***Abdullahi S. L., Pennap G .R. I., Ibrahim Y.**

Department of Microbiology, Faculty of Natural and Applied Sciences, Nasarawa State University, Keffi P.M.B 1022, Keffi, Nigeria

***Corresponding Author**

DOI: https://doi.org/10.51244/IJRSI.2025.1215000169P

Received: 27 August 2025; Accepted: 03 September 2025; Published: 31 October 2025

ABSTRACT

Co–infection of viruses can be a serious public health problem because most antivirals are designed to control and manage single infection. This study was conducted to determine the prevalence, circulating genotypes and risk factors of hepatitis B, C virus and HIV co–infection among patients accessing care at Wuse District Hospital, Abuja. 5 ml of blood sample was collected from each of the 400 consenting HIV patients accessing care at the medical facility. The 5-Panel hepatitis B and C virus diagnostic Profile kit and Combo Kits were used to screen the blood samples for hepatitis B and C virus infection respectively. All samples positive for HBsAg were genotyped by PCR using type-specific primers. Out of the 400 HIV patients who participated in this study, none was positive for hepatitis C virus, 5(1.3%) were positive for HBsAg, 153(38.3%) were Immune and 235(58.7%) were neither immune nor susceptible to HBV. The 5 positive samples for HBsAg were subjected to genotyping. Hepatitis B virus genotype A was found to be circulating in the study population. History of blood transfusion, sharing of sharp objects and multiple sex partners were found to be significant risk factors for the infection. However, occupation and sharing of clothes did not have any statistically significant association with hepatitis B virus infection in HIV patients. More than half of the study population (58.7%) were found to be unexposed hepatitis B virus while all the study population (100%) were likely unexposed to hepatitis C virus which makes them susceptible to the viruses.

INTRODUCTION

Hepatitis B Virus (HBV) being a double-stranded DNA *hepadnavirus* is an important cause of acute and chronic hepatitis and hepatocellular carcinoma (Kumar *et al.,* 2023). Hepatitis B virus (HBV) infection remains a major health problem despite an extensive vaccination program worldwide. Globally, 260 million people are chronically infected with HBV and 890,000 are dying yearly from complications due to the advancement of the infection (Locarnini *et al.*, 2015). HBV may play a role in the pathogenesis of chronic liver disease, cirrhosis, and hepatocellular carcinoma (HCC) (Kumar *et al.*, 2023). Hepatitis C virus (HCV) is a single-stranded positive-sense virus belongs to the Flaviviridae family (Kim *et al*., 2013). The virus naturally targets the hepatocytes, and it is an important cause of viral hepatitis (Ejiofor *et al.*, 2010).

The coinfection of viruses can be a serious public health problem because most antivirals are designed to control and manage a single infection (Shahriar *et al.*, 2022). Acquired immunodeficiency syndrome (AIDS) caused by the human immunodeficiency virus (HIV) is one of the most important and prevalent disease conditions that has been spread among humans for the last two decades (Kim *et al.*, 2000). More than 75 million people worldwide have been infected with HIV, and approximately 37 million individuals are currently living with this infection (Shahriar *et al.*, 2022). Hepatitis B (HB) and hepatitis C (HC) viral infections are highly prevalent among HIV-infected individuals due to sharing the same transmission routes (Shahriar *et al.*, 2022). Hepatitis caused by both hepatitis B virus (HBV) and hepatitis C virus (HCV) leads to severe liver disorder, and morbidity and mortality are now increasing due to coinfection with HIV (Easterbrook *et al.*, 2017). Coinfection with HIV can modify the natural characteristics of HBV by genome replication status,

higher rates of chronic infection, and liver disease progression (Ndifontiayong*et al.*, 2021). The impact of HIV on HBV is critical, as HIV can provoke chronic HBV infection, which can lead to hepatocellular carcinoma (HCC) (Mapong*aet al.*, 2020). While in HIV-HCV coinfection, HIV increases the HCV viral load and accelerates liver disease progression (Rodrigo, 2020). HIV, HBV, and HCV are transmitted via blood, shared needles, syringes, and other injection equipment, sexually, or even from pregnant mothers to babies (Pfaender*et al.*, 2016). The hepatotropic viruses, both HBV and HCV, attack the liver cell and cause inflammation. However, HIV can attack any targeted cell in the mucosal tissue and spread through the whole lymphoid system (Siebers and Finlay, 1996). As a result of shared transmission routes, HBV, HCV, and HIV can easily cause coinfection, more pervasive than an infection caused by either HBV or HCV (Shahriar *et al.*, 2022).

MATERIALS AND METHODS

Study Area

The study was carried out at Wuse district hospital, which is a modern public medical facility located at Wuse, Abuja Municipal Area Council, Federal Capital Territory.It provides full scale clinical and diagnostic services in various areas such as cardiology, ophthalmology, general surgery, antenatal care, HIV/AIDS services, intensive care services and many others. It has a wide range of participant coverage from all parts of Abuja, Nigeria.

Sample Size Determination

The sample size for this study was determined using the formula by Naing *et al.* (2006), for sample size calculation at 0.05 level of precision;

$$n = \frac{Z^2 pq}{d^2}$$

Where:

n = required sample size

Z = standard normal deviation at the required confidence interval (1.96) which corresponds to 95% confidence interval.

p = Prevalence of HCV/HIV co-infection (6.5% - 0.07, based on previous study in Abuja) (Agboghoroma and Ukaire, 2020)

q = 1 – p = 0.93

d = degree of precision expected (0.05)

$$n = \frac{(1.96)^2 (0.07)(0.93)}{(0.05)^2} = \frac{3.8416 \times 0.07 \times 0.93}{0.0025}$$

$$n = \frac{0.25008816}{0.0025}$$

$n = 100$

This was rounded up to 400 samples.

Inclusion Criteria

The inclusion criteria were consenting HIV positive patients accessing care at Wuse District Hospital, Abuja.

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI | Volume XII Issue XV October 2025 | Special Issue on Public Health

Exclusion Criteria

HIV positive patients who did not give their consent and other patients who were not HIV positive accessing care at Wuse Hospital, Abuja were excluded from the study.

Ethical Consideration

Ethical approval for this study was obtained from the Federal Capital Territory Research and Ethical Commission with the approval number as follows: FHREC/2022/01/85/04 - 05 – 22

Sample Collection and Storage

The serum sample to be used in the study was collected from venous blood aseptically. The harvested serum was stored frozen at 0ºC in the laboratory refrigerator of Wuse District Hospital Abuja until tested for HBV and HCV sero-markers.

Screening for HCV

All blood specimens were screened for HCV using ARCHITECT Anti-HCV (ABBOTT Max-Planck-Ring, Germany). The assay is a chemiluminescent microparticle immunoassay (CMIA) for the qualitative detection of antibody to hepatitis C virus (anti-HCV) in human serum and plasma.

Principle of the Assay

The ARCHITECT Anti-HCV assay is an immunoassay which uses chemiluminescent microparticle immunoassay (CMIA) technology for the qualitative detection of anti-HCV in human serum and plasma. In this step, sample, recombinant HCV antigen coated paramagnetic microparticles and Assay Diluent are combined. Anti-HCV present in the sample binds to the HCV coated microparticles. After washing, anti-human acridinium-labeled conjugate is added in the second step. Following another wash cycle, Pre-Trigger and Trigger Solutions are added to the reaction mixture. The resulting chemiluminescent reaction is measured as relative light units (RLUs). The presence or absence of anti-HCV in the specimen is determined by comparing the chemiluminescent signal in the reaction to the cutoff signal determined from a previous ARCHITECT Anti-HCV calibration. If the chemiluminescent signal in the specimen is greater than or equal to the cutoff signal, the specimen is considered reactive for anti-HCV.

Assay Procedure

The specimen was placed on the well of the cassette using a micropipette after which 3 drops of buffer solution was placed on the well. The kit was left for 5 mins before the result was observed visually.

Interpretation of Results

Negative Result: The presence of a coloured line on only the control (C) line is indicative of a non-reactive test.

Positive Result: The presence of coloured band on both control (C) and test (T) line is indicative of a positive test.

Invalid Result: The absence of a band on the control line is indicative of an invalid result.

Screening for HBV

All blood specimens were screened for HBV serologic markers using HBV-5 rapid panel test kit (CTK Biotech. Inc San Diego, USA) according to the manufacturer's instructions. It is a kit for rapid immunochromatographic assay for the qualitative detection of HBV infection markers such as HBsAg, HBeAg, HBeAb and HBcAb in serum.

Principle of the Assay

HBV-5 Rapid test is a lateral flow chromatographic immunoassay consisting of 5 panel strips assembled in one cassette. Each strip of the panel is composed of a sample pad, colloid gold conjugate pad, nitrocellulose membrane (NC membrane) strip pre-coated with a control line and test line and absorbent pad. The antigen (HBsAg and HBeAg) strips are antibody-based sandwich immunoassays. The conjugate pads contain polyclonal antibodies (HBsAb and HBeAb) conjugated with colloid gold and the NC membrane is precoated with monoclonal antibodies (HBsAb and HBeAb). When an adequate volume of test specimen is applied into the specimen well of the strips, the test specimen migrates by capillary action across the test strips. HBsAg if present in the specimen will bind to the HBsAb gold conjugates and HBsAg will bind to HBeAb if present. The immunocomplex is then captured on the membrane by the pre-coated antibodies (HBsAb and HBeAb), forming a colored T band, indicating a positive test result. The absence of the T band indicates a negative result.

Assay Procedure

The test kit was removed from the pouch, labelled with the specimen identifier and placed on a flat surface on the work bench. The specimens were brought to room temperature using a Pasteur pipette, two drops of a test serum were placed in each of the 5 specimen wells of the strip (HBsAg, HBsAb, HBeAg, HBeAb and HBcAb) and the result read virtually after 15 minutes according to the manufacturer's instructions.

Interpretation of Results

Negative Result: If only the control line (C) developed on the HBsAg, HBeAg strip, or both C and the test (T) lines developed on either the HBeAb or HBcAb strip, the test indicated a negative result on the parameter being tested.

Positive Result: If both C and T lines developed on the HBsAg, HBsAb, or the HBeAg strip, or only C line developed on the HBeAb strip, the test indicated presence of the parameter being tested.

Invalid Result: If no C line developed, the assay on the strip was reported as invalid regardless of color development on the T line. The assay was repeated with a new device.

Molecular Analysis

A genotyping system based on polymerase chain reaction (PCR) using type-specific primers was used in this study for the determination of genotypes A through F of hepatitis B virus according to previously described methods by Abdulqadir *et al.* (2023). Samples that were positive for HBsAg, HBeAg, HBeAb and those that were HBsAg negative but anti-HBc positive were included because the seromarker is an indicator of probable occult hepatitis B virus infection were selected for genotyping.

Preparation of Reagents

Proteinase K was prepared by dissolving 25mg of it in 1.25ml of nuclease-free water and mixed thoroughly. The lysis buffer was prepared by dissolving 25mg of the buffer in 25ml of distilled water which was mixed properly. Washing buffers 1 and 2 were prepared by adding 30ml and 80ml absolute ethanol to the concentrated form of washing buffer 1 and washing buffer 2 provided respectively. The elution buffer was prepared by dissolving 10mg of Tris-chloride in 30ml distilled water.

DNA Extraction Procedure

The accuprep genomic DNA extraction kit (BIONEER Daejeon, North Korea) was used for DNA extraction from serum according to the manufacturer's instructions. Briefly, for each of the samples, 200ul of serum was transferred into a labeled 1.5ml centrifuge tube using an Eppendorf micropipette. Then 20ul and 200ul of already prepared proteinase K and binding buffer were added and mixed immediately using a vortex mixer. The tubes were incubated for 10 minutes at 60^{o}C to activate the enzyme. Then 100ul of isopropanol was added

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI | Volume XII Issue XV October 2025 | Special Issue on Public Health

and mixed again with a vortex mixer. The formed lysate was transferred into the upper reservoir of the binding column and centrifuged at 8,000rpm for 1 minute. The liquid under the tube was decanted and 500ul of the prepared washing buffer 1 was added. The content of the tube was centrifuged at 8,000rpm for 1 minute, the liquid under the tube was again decanted and 500 µl of washing buffer 2, was added. The content of the tube was centrifuged for 1 minute at 8,000rpm to completely remove ethanol.

The binding column tube was transferred into a new 1.5ml centrifuge tube and 200µl of elution buffer was added. The tube was kept at room temperature for 1 minute until the elution buffer was completely absorbed into the glass fiber of the binding column tube and the content was centrifuged at 8,00rpm for 1 minute to finally elute the DNA which settled at the bottom of the tube.

Polymerase Chain Reaction

PCR was carried out in two rounds in a PTC-100 programmable controller (Bio-Rad/MJ Research Inc. USA) using oligonucleotide primers which were adopted from the work of Abdulqadir*et al.* (2023). The first and second round PCR primers were designed based on the nature of nucleotide sequences in regions of the pre-S1 through S gene of the six HBV genotypes (Naito *et al.,* 2011). P1 and S1-2 were universal outer primers. Mix A consisted of sense primer B2 for genotype A, B and C and antisense primers BA1R (type A specific), BB1R (Type B specific) and BC1R (type C specific). B2R was used as the inner primer (antisense) with a combination called mix B for genotypes D, E and F. It consisted of sense primers BD1 (type D specific), BE1 (type E specific) and BF1 (type F specific).These primer combinations for the second round PCR were designed based on the differences in the sizes of the genotype-specific bands. The type-specific primers were designed based on the nature of those sequences within a genotype and based on their poor homology with the sequences derived from other HBV genotypes (Naito *et al.,* 2011). The master mix was prepared by adding 1µl each of the P1 and S1-2 primers into a labelled 1.5ml centrifuge tube and 15µl of deionized water (to make 17µl total volume).

Primer Sequences Used in the Study

Primers	**Sequences**		**Specificity**	**Ampliconsbp**
P1	5'–TACACCATATTCTTGGGAACAAGA–3'	First round PCR	Universal sense	1,063bp
S1 – 2	5' – CGAACCACTGAACAAATGGC – 3'		Universal antisense	
µB2	5' – GGCTCAAGTTCAGGAACAGT – 3'	Mix A (second round PCR)	Type A to C specific, sense	
BAIR	5' - CTCGCGGAGATTGAGATGT – 3'		Type A specific, antisense	68bp
BB1R	5' – CAGGTTGGTGAGTGACTGGAGA – 3'		Type B specific, antisense	281bp
BC1R	5' – GGTCCTAGGAATCCTGATGTTG – 3'		Type C specific, antisense	122bp
B2R	5' – GGAGGCGGATCTGCTGGCAA – 3'	Mix B (second round PCR)	Types D to F specific antisense	
BD1	5' – GCCAACAAGGTAGGAGCT – 3'		Type D specific, sense	119bp
B E 1	5' – CACCAGAATCCAGATTGGGACCA – 3'		Type E specific, sense	167bp
B F 1	5' – GCTACGGTCCAGGGTTACCA – 3'		Type F specific, sense	97bp

PCR Procedure

The PCR mix tube contained Taq DNA Polymerase, dNTPs, MgCl2 and 1 × PCR buffer and 17µl of master mix and 3µl of the extracted DNA were added to make 20µl total volume. Positive and negative control tubes were prepared by adding 17µl of the master mix into each of the 2 tubes and 3µl of a known DNA for the positive control and 3µl of deionized water for the negative control to make 20µl each. The content of the tube

was then centrifuged for 30 seconds using a microcentrifuge and the tubes were loaded into the PCR machine. The first round PCR was programmed to first incubate the samples for 5 minutes at 95ºC, followed by 40 cycles consisting of 94º C for 1 minute, 55ºC for 1 minute and 72ºC for 2 minutes.

For the second round PCR, two reactions were performed for each sample with the common universal sense primer (B) and mix A for types A to C and the common universal antisense primer (B2R) and mix B for types D to F. Fresh PCR mix tubes were labeled in duplicates for each sample (1 and 2) for mix A and B where 2µl each of the primer mix A and B was added to the corresponding tubes followed by the addition of 1µl each of the aliquot of the first PCR product to all the samples.

Finally, 17µl of deionized water was added to each tube to make 20µl total volume. The content of the tube was centrifuged, loaded into the PCR machine, and allowed to run using the following parameters: one amplification for 40 cycles consisting of preheating at 95ºC for 5 min, 30 cycles of amplification at 94ºC for 2 minutes, 58ºC for 1 minute and 75ºC for 5 minutes.

Agarose Gel Electrophoresis

The products from the PCR second round were used to run the Agarose gel-electrophoresis which separates the genotype-specific DNA bands according to their sizes.

The agarose gel (2%) was prepared by adding 2g of the agarose powder into 100ml of 1 × TAE (Tris-acetic Ethylene Diamine Tetra acetic Acid, EDTA) in a conical flask. The mixture was heated in a microwave until the agarose powder was completely dissolved and allowed to cool in a water bath set at 50ºC. This was followed by the addition of 8µl of ethidium bromide and poured into a gel cast with combs to make well. The gel was allowed to solidify in the cast and the combs were carefully removed from the cast resulting in the formation of wells.

Agarose Gel Electrophoresis Procedure

The solidified gel was placed in the electrophoresis chamber then covered with TAE buffer. In the first well, 8µl of DNA molecular marker (BIONEER Deajeon, North Korea) was introduced and 8µl each of the samples (mix A, B, positive and negative controls) were added into the corresponding wells in a definite order. The electrophoresis was run for 40 minutes at 100 volts and the resultant DNA bands were visualized using the gel imaging and documentation system.

Interpretation of Gel Electrophoresis Results

The sizes of PCR products were estimated in relation to the migration pattern of a 100bp plus DNA molecular marker (BIONEER Daejeon, North Korea). The DNA molecular marker is a set of DNA molecules of known length which consisted double stranded DNA fragments ranging in size from 100bp to 1000bp increments. By comparing the sizes of the bands on the DNA molecular marker with those of the samples, the results were interpreted with respect to the specific size of each genotype.

Statistical Analysis

Data obtained from the questionnaires and results of the laboratory tests were analyzed using SPSS 25 (Statistical Package for Social Sciences version 25). Descriptive Statistics were presented in tables, figures, graphs and charts. The seroprevalence of HBV infection were determined from total population under consideration and expressed as a percentage. A comparison of the frequency was analyzed using the Chi-square test and a p value of ≤ 0.05 was considered statistically significant.

RESULTS

Data Presentation

A total of 400 people living with HIV participated in this study. The blood samples were collected from Wuse District Hospital., Abuja. Out of the 400 participants enrolled, 322 (80.5%) were male and 78(19.5%) were

female. With respect to marital status, 281(70.3%) were unmarried and 119 (29.7%) were married. The sociodemographic characteristics of the participants is as shown in Table 1.

Among the 400 participants, 5(1.3%) were positive for HBsAg, 153(38.3%) were positive for HBsAb, 4(1.0%) were positive for HBcAb, 2(0.5%) for HBeAg, 1(0.3%) was positive for HBeAb and 235(58.7%) were unexposed to HBV. (Figure 1and Table 2). The pattern of prevalence is as shown in figure 3.2. None of the 400 participants were positive for hepatitis C.

The married participants were 281 with the prevalence of 0.8% for HBsAg while the unmarried participants were 119 with a prevalence of 1.4% for HBsAg. There was no significant association between marital status and prevalence of HBsAg ($p>0.05$). The participants were between the ages of 15 to 45 years. Age 25 to 34 years participants were 284 with the prevalence of 1.0% forHBsAg. Age 35-44 years was only1 with the highest prevalence 1.7% for HBsAg. The age of the participants had no significant association ($p>0.05$) with prevalence of HBV infection. (Table 3).

Of the 150 participants who are civil servants, 3 were positive for HBsAg with the prevalence of 2.0%. Artisans were 178 with the prevalence of 1.1%. There was no significant association between occupation and HBV infection ($p>0.05$).

Table 1: Socio-demographic Characteristics of HIV Patients Attending Wuse District Hospital, Abuja

Parameters	**No. Screened (%) n = 400**
Gender	322(80.5)
Male	78(19.5)
Female	
Age (years)	
15-24	33(8.3)
25-34	286(71.5)
35-44	60(15.0)
≥ 45	21(5.2)
Marital Status	
Unmarried	281(70.3)
Married	119(29.7)
Occupation	
Students	59(14.7)
Farmers	2(0.5)
Unemployed	11(2.8)
Entrepreneurs	178(44.5)
Civil Servants	150(37.5)
History of Blood Transfusion	
Yes	46(11.5)
No	354(88.5)
Multiple sex Partners	
Yes	11(2.3)
No	389(97.2)
Body Scarification	
Yes	156(39.0)
No	244(61.0)
Alcohol Consumer	
Yes	32(8.0)
No	368(92.0)
Sharing of Sharp Objects	
Yes	16(4.0)
No	384(96.0)

ISSN No. 2321-2705 | DOI: 10.51244/IJRSI | Volume XII Issue XV October 2025 | Special Issue on Public Health

Sharing of Clothes	
Yes	141(35.3)
No	259(64.7)
History of HBV infection in the Family	
Yes	56(14.0)
No	344(86.0)

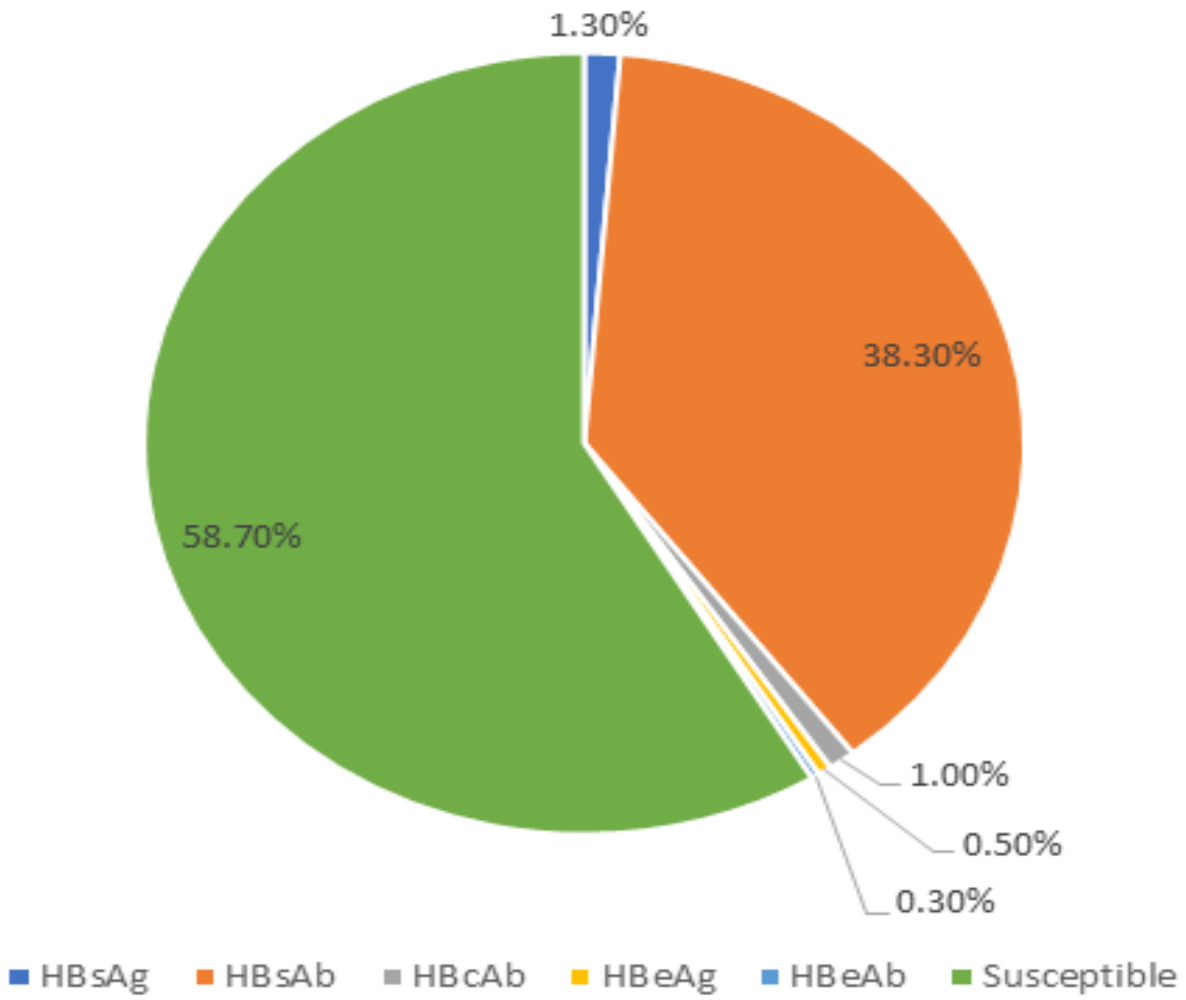

Figure 1: Prevalence of HBV infection serologic markers among HIV patients accessing care at Wuse District Hospital, Abuja

Table 2: Patterns of HBV Infection Serologic Markers among HIV Positive Patients Accessing care at Wuse District Hospital, Abuja

Serologic Markers	Interpretation	No. of Participants	Percentage (%)
$HBsAg^{+}$, $HBsAb^{-}$, $HBcAb^{+}$, $HBeAg^{+}$, $HBeAb^{-}$	Chronic infection with high viral replication	2	0.5
$HBsAg^{+}$, $HBsAb^{-}$, $HBcAb^{+}$, $HBeAg^{-}$, $HBeAb^{+}$			0.3
$HBsAg^{-}$, $HBsAb^{-}$, $HBcAb^{+}$, $HBeAg^{-}$, $HBeAb^{-}$	Carrier with low viral replication	1	1.0
$HBsAg^{+}$, $HBsAb^{-}$, $HBcAb^{-}$, $HBeAg^{-}$, $HBeAb^{-}$	Window period of infection	4	1.3
$HBsAg^{-}$, $HBsAb^{+}$, $HBcAb^{+}$, $HBeAg^{-}$, $HBeAb^{-}$	Recently vaccinated	5	38.3
$HBsAg^{-}$, $HBsAb^{-}$, $HBcAb^{-}$, $HBeAg^{-}$, $HBeAb^{-}$	Immune due to natural previous exposure	153	58.7
	Unexposed (Susceptible)	235	
Total		**400**	**100**

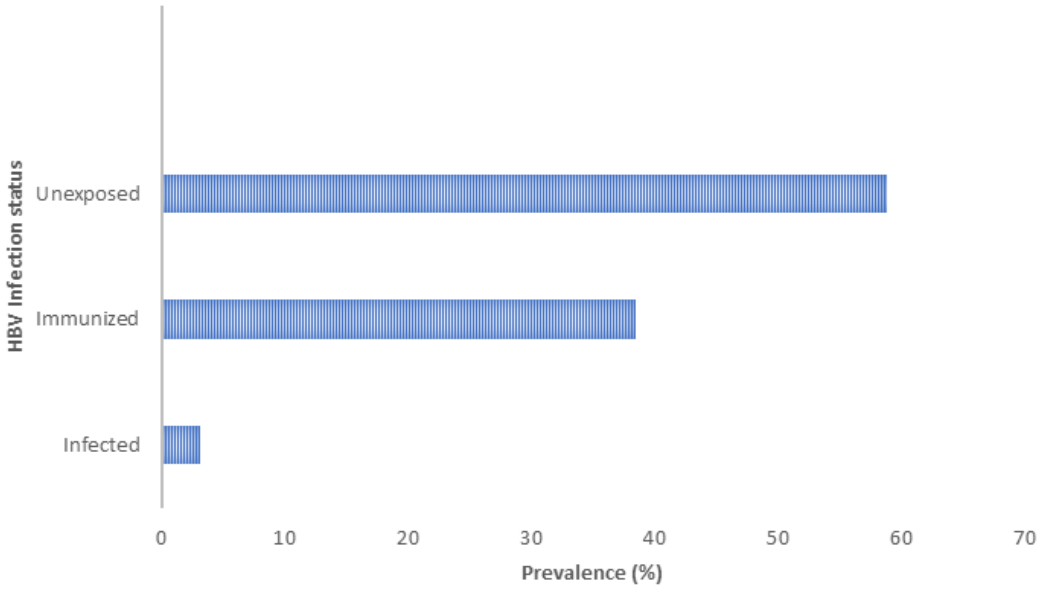

Figure 2: Prevalence pattern of HBV infection among HIV positive patients accessing care at Wuse District Hospital, Abuja.

Table 3: Prevalence and Distribution of HBV infection Serologic Markers with Respect to Socio-demographic Factors among HIV Patients at Wuse District Hospital, Abuja

Parameters	**No. of Samples Examined**	**No. Positive (%)**				
		HbsAg	**HBsAb**	**HBcAb**	**HBeAg**	**HBeAb**
Age (Years)						
15-24	33	1(3.0)	13(3.0)	1(3.0)	0(0.0)	0(0.0)
25-34	286	3(1.0)	68(23.7)	2(0.7)	2(0.7)	0(0.0)
35-44	60	1(1.7)	51(85.0)	1(1.7)	0(0.0)	1(1.7)
≥ 45	21	0(0.0)	21(100.0)	0(0.0)	0(0.0)	0(0.0)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.6220**	**0.0010***	**0.5610**	**0.7710**	**0.9990**
Gender						
Male	322	4(1.3)	136(42.2)	4(1.2)	2(0.6)	1(0.3)
Female	78	1(1.2)	17(21.7)	0(0.0)	0(0.0)	0(0.0)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.8710**	**0.0011***	**0.5100**	**0.3120**	**0.1100**
Marital Status						
Unmarried	281	4(1.4)	91(32.3)	1(0.3)	1(0.7)	1(0.3)
Married	119	1(0.8)	62(52.1)	3(2.5)	1(0.0)	0(0.0)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.3320**	**0.1100**	**0.1010**	**0.8900**	**0.5510**
Occupation						
Student	59	0(0.0)	32(54.2)	0(0.0)	0(0.0)	0(0.0)
Farmer	2	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Unemployed	11	0(0.0)	8(72.7)	0(0.0)	0(0.0)	1(9.0)
Artisan	178	2(1.1)	71(39.9)	3(1.6)	2(1.1)	0(0.0)
Civil servant	150	3(2.0)	42(28.0)	1(0.7)	0(0.0)	0(0.0)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.3110**	**0.0001***	**0.5320**	**0.9990**	**0.7120**

*= Significant ($p<0.05$)

With respect to history of blood transfusion, participants who had been transfused were 46 among which 2(4.3%) were positive for HBsAg while participants who had never been transfused were 354 with the lowest prevalence of 0.8% for HBsAg. The history of blood transfusion among the participants had significant association with the prevalence of HBV HBsAg ($p<0.05$). Similarly, participants with multiple sexual partners were 11 with the highest prevalence of 9.0% for HBsAg while participants with no multiple sexual partners were 389 with the lowest prevalence of 1.0% for HBsAg. Similarly, participants who consume alcohol were 19 with the highest prevalence of 10.0% for HBsAg while participants who do not consume alcohol were 381 with the lowest prevalence of 0.8% for HBsAgas seen in Table 3.

With respect to sharing of clothes, participants who share clothes were 141 with the prevalence of 0.7% for HBsAg while participants who do not share clothes were 259 with the prevalence of 1.5% for HBsAg. Similarly, participants with history of HBV in the family were 56 with the prevalence of 3.5% for HBsAg while participants with no history of blood transfusion in the family were 344 with the prevalence of 0.8%. The history of blood transfusion was not statistically significant for HBsAg. ($p > 0.05$) as seen in Table 4.

Table 4: Prevalence and Distribution of HBV infection Serologic Markers with Respect to Possible Risk Factors among HIV Patients at Wuse District Hospital, Abuja

Parameters	**No. of Samples Examined**	**No. Positive (%)**				
		HBsAg	**HBsAb**	**HBcAb**	**HBeAg**	**HBeAb**
History of Blood Transfusion						
Yes	46	2(4.3)	41(89.1)	1(2.1)	1(2.1)	1(2.1)
No	354	3(0.8)	112(31.6)	3(0.8)	1(0.3)	0(0.0)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.0001***	**0.0020***	**0.1020**	**0.0990**	**0.1010**
Multiple Sex Partners						
Yes	11	1(9.0)	5(45.4)	1(9.0)	0(0.0)	0(0.0)
No	389	4(1.0)	148(38.0)	3(0.8)	2(0.5)	1(0.3)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.0010***	**0.3220**	**0.9120**	**0.7010**	**0.9900**
Body Scarification						
Yes	156	2(1.3)	57(36.5)	1(0.6)	0(0.0)	1(0.6)
No	244	3(1.2)	96(39.3)	3(1.2)	2(0.8)	0(0.0)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.9100**	**0.7170**	**0.8100**	**0.9820**	**0.5610**
Alcohol Consumption						
Yes	19	2(10.5)	12(63.1)	1(5.2)	0(0.0)	1(5.2)
No	381	3(0.7)	141(37.0)	3(0.8)	2(0.5)	0(0.0)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.6700**	**0.0001***	**0.9231**	**0.5660**	**0.7230**

*= Significant ($p<0.05$)

Table 5:Prevalence and Distribution of HBV infection Serologic Markers with Respect to Possible RiskFactors among HIV Patients at Wuse District Hospital, Abuja.

Parameters	**No. of Samples Examined**	**No. Positive (%)**				
		HBsAg	**HBsAb**	**HBcAb**	**HBeAg**	**HBeAb**
Sharing of Sharp Objects						
Yes	16	0(0.0)	7(43.7)	0(0.0)	1(6.2)	1(6.2)
No	384	5(1.3)	146(38.0)	4(1.0)	1(0.3)	0(0.0)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.0030***	**0.0710**	**0.9100**	**0.8110**	**0.9999**
Sharing of Clothes						

Yes	141	1(0.7)	26(18.4)	1(0.7)	1(0.7)	0(0.0)
No	259	4(1.5)	127(49.0)	3(1.1)	1(0.3)	1(0.3)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.1010**	**0.8123**	**0.9810**	**0.2300**	**0.9900**
History of HBV in the Family						
Yes	56	2(3.5)	12(21.4)	2(3.5)	0(0.0)	0(0.0)
No	344	3(0.8)	141(40.9)	2(0.5)	2(0.5)	1(0.3)
Total	**400**	**5(1.3)**	**153(38.3)**	**4(1.0)**	**2(0.5)**	**1(0.3)**
p-value		**0.9000**	**0.0001***	**0.2300**	**0.0670**	**01010**

*= Significant (p<0.05)

Prevalence of HBV Genotypes

A total of five samples were positive for HBsAg by the immunoassay which were then marked for genotyping by PCR. The samples were genotyped by PCR using type-specific primers and they were determined according to the amplified sizes of the PCR product. Two of the samples were found to be HBV genotype B while one of the samples was identified to be genotype A as shown in Plate A. On Plate B, the two samples were identified to be genotype D and E as shown in Plate B.

Plate A: Agarose gel electrophoresis of PCR Mix A. Lane M represents the molecular maker, lane 1 and 4 indicates genotype B (281bp). Lane 3 indicates genotype A (68bp).

Plate B: Agarose gel electrophoresis of PCR Mix B. Lane M represents the molecular maker, lane 6 indicates genotype D (119bp). Lane 3 indicates genotype E (167bp).

DISCUSSION

The prevalence of HBV co-infection among people living with HIV/AIDS in this study was 1.3 % using HBsAg as a surrogate for HBV infection (5/400). This confirms earlier reports stating that HBV infection exists among people living with HIV/AIDS. Findings from this study shows that a total of 235 (58.7%) of the people living with HIV were neither positive for any of the markers of HBV nor anti-HCV which makes them susceptible to HBV and/or HCV infection.

The prevalence of HBV/HIV coinfection from this study is lower than the 12.5% reported in Kano, North Central Nigeria (Hamza *et al.,* 2019),7.8% in south-east Nigeria (Nnakenyi *et al.,* 2020), 11.5% in Abuja (Adewole *et al.,* 2019) and 11.8% in Jos (Lar *et al.,* 2013). The variation in prevalence of this study may be due to the fact that infections vary from one locality to another and from one country to another depending on the level of associated risk factors (WHO,2013; Yu *et al.,* 2020).

HCV/HIV infection was not recorded in this study as none of the 400 samples tested positive for HCV. This may be due to the lower number of participants in this study compared to 0.7% in 440 participants by Diwe*et al.* (2013) and 4.7% in 4663 participants by Nnakenyi *et al* (2020). This may also suggest that although the three viruses have similar routes of transmission, they are not transmitted at the same rate.

HBV/HIV coinfection is a growing concern because of increasing toxicity to antiretroviral medications in HBV coinfected individuals as well as higher levels of HBV replication, lower rates of spontaneous resolution of the HBV infection, and higher risk of reactivation of previous infections. This implies that there is an increased risk of developing cirrhosis of the liver among HBV/HIV coinfected persons (Gilson *et al.,* 1997; Feld *et al.,* 2015).

The first antibody to appear in HBV infection is HBcAb and its presence in an individual symbolizes earlier contact with the virus (Liu *et al.,* 2010). This seromarker was found in 1.0% of the study population. These were those that have had contact with the virus at one time or the other in their lives. A HBsAg negative status does not rule out the possibility of HBV infection. An Individual might be in the window period, and detection of HBcAb serves as a determinant serologic marker during the window period of the infection (Ogunfemi *et al.,* 2017). HBcAb can also be an indication of HBV occult infection when it is present in the absence of HBsAg and other seromarkers. The common reason for the absence of HBsAg is the change in the steric configuration in HBsAg molecule, determined by mutations within the "a" determinant region. These modified HBsAg molecules, either cannot be detected by commercially available assays or are weakly exposed in the surface of hepatocytes due to a poor recognition by the immune system (Cento *et al.,* 2013).

The prevalence of 0.5% HBeAg in this study is associated with active HBV replication and transmission of infection. However, it means that this individual has acute or chronic HBV infection and have 70-90% chances of transmitting the virus to others and high chances of developing persistent liver disease leading to cirrhosis and even primary liver cancer if not treated (Abah and Aminu, 2016). The prevalence reported in this study (0.5%) is however lower than 1.9% reported earlier by Agbesor *et al.* (2013) in Abuja, 1.5% in Ilorin (Ogunfemi *et al.,* 2017) among blood donors and 4.7% among pregnant Nigerian women (Abahand Aminu, 2016). The reason for these differences may not be unrelated to the fact that the studies were conducted in different populations and as such population differences should understandably impact the outcome.

HBeAb is the antibody produced by the body against HBeAg and its presence indicates lowered infectivity and transmission of the virus. Like the HBsAb, it may also imply recovery from HBV infection (WHO, 2017). The prevalence of HBeAb in this study was 0.3%. Higher rates have been reported, and these include 8.0% reported by Odimayo*et al.* (2016) among HBsAg seropositive individuals, 13.0% by Mbaawuaga*et al.* (2014) and 51.6% by Abah and Aminu (2016) among a population of pregnant women in Nigeria. The differences in study populations may account for the observed differences in the different studies.

The data from this study showed that HBV/HIV coinfection rates was highest among HIV infected individuals who are between the ages of 25 to 34 years, although the difference was not statistically significant ($p > 0.05$). This finding is similar to reports of Okechukwu *et al.* (2014) showing that those between ages 26 to 40 years

have higher frequencies of HBV /HIV coinfection infection. Higher prevalence of coinfection in younger people could be attributed to the many routes of transmission which operate among the younger persons compared to the older ones. This is because HBV is more infectious than HIV and can be transmitted via dried blood, open cuts, and shared toothbrushes, razors, clippers and having unprotected sex with one or multiple partners who could be infected. The younger people are generally more vulnerable to these risky behaviors and factors than the adults.

Findings from this study also showed that more men (1.5%) were infected with HBV than women (0.0%) although the difference was not statistically significant ($p > 0.05$). The findings are at variance with those reported by Okechukwu *et al.* (2014) who reported higher prevalence in females than males. Another study by Adewole *et al.* (2019) in Abuja Nigeria showed higher female preponderance of HIV/HBV co-infection. The difference between males and female coinfection could mean that men in the study area are more engaged in risky behaviors that favor transmission compared to the women. Similarly, the study showed that HBV/HIV coinfection rate was higher in unmarried individuals than their married counterparts although the difference was not statistically significant ($p > 0.05$). This might imply that the exposure to risk factors among the different groups of persons is fairly the same although it appears higher among the unmarried. The higher prevalence recorded among the unmarried has been attributed to absence of family cover which could shield or prevent them from having multiple sexual partners (Sule *et al.,* 2011).

Occupation was not found to be significantly associated with HBV infection ($p > 0.05$). The highest prevalence of infection reported among the civil servants might be because of the nature of their occupation which involves frequent contact with people. This may sometimes enhance their chance of exposure to HBV. This, however, contrasts with the findings of Okechukwu *et al.* (2014) in Nigeria, who reported highest prevalence among farmers.

In this study, high coinfection rate (1.3%) was recorded among the participants who have had blood transfusion. This may be due to transfusion of improperly screened blood since not all Nigerian hospitals have the facilities for effective screening of HBV. Therefore, there is risk of transfusing infected blood. However, the finding of this study is similar to that of Isa *et al.* (2015) who identified blood transfusion as one of the major risk factors for HBV infection but contradicted that of Buseri *et al.* (2010) who reported a higher seropositivity among people who have never been transfused.

A higher seroprevalence of HBsAg was reported among people with multiple sexual partners (9.0%) than those with single sexual partners (1.0%) and this difference was found to be statistically significant. This finding is similar to that of Pennap *et al.* (2011) who reported higher prevalence of HBsAg among eligible blood donors with multiple sexual partners. However, this finding is contrary to that of Isa *et al.* (2015) who reported higher HBsAg prevalence in those with single sexual partners than those with multiple sexual partners. The reason for this is not very obvious.

Alcohol consumption was not statistically significant in this study ($p > 0.05$). High prevalence of the viral infection (10.5%) was found among participants who consume alcohol compared to those who do not consume alcohol (0.7%). This is in conformity with the result of Ndako*et al.* (2012). The reason for this could be that alcohol consumers are likely to have multiple sexual partners as alcohol increases libido in both men and women.

There was a statistically significant association between sharing sharp objects and the viral infection in this study. This implies that the virus can be transmitted from one person to another through unsterilized sharp objects. The prevalence of HBsAg in the study was higher among individuals who share clothes (1.5%) in this study. However, there was no significant association between this potential risk factor and the infection. This finding agrees with that of Isa *et al.* (2015) who reported higher prevalence of HBsAg among individuals who shared clothes. This could be because HBV can be transmitted through the sweat of infected persons as reported by Kazuhide *et al.* (2020).

HBV Genotyping is important in determining HBV disease progression and treatment (Zaman *et al.,* 2018). This study was able to detect and genotype infecting HBV in 5 of the collected samples that were HBsAg-

positive and that were HBV-DNA positive. HBV genotypes A, B, D, E were found to be circulating in the study population. This was consistent with the findings in earlier report in Nigeria (Ahmad *et al*., 2019 and Nnakenyi *et al.,* 2020) and in other African countries (Kramvis and Kew, 2007).

CONCLUSION

The prevalence of HBV among people living with HIV accessing care at Wuse District Hospital, Abuja was found to be 1.3% using HBsAg as surrogate for infection. History of blood transfusion, sharing of sharp objects and multiple sex partners were found to be significant risk factors for infection ($p \leq 0.05$). Genotype A, B,D and E were found to be circulating in the study population. More than half of the study population (58.7%) were found to be unexposed to HBV while all the study population (100%) were found to be unexposed to HCV which makes them susceptible to the viruses.

REFERENCES

1. Abdala, N., Krasnoselskikh, T. V., Durante, A. J., Timofeeva, M. Y., Verevochkin, S. V., and Kozlov, A.P. (2008). Sexually transmitted infections, sexual risk behaviours and the risk of heterosexual spread of HIV among and beyond IDUs in St. Petersburg, Russia. European Addiction Research, 14(1): 19-25
2. Abdelwahab, K.S., and Ahmad, Said, Z.N. (2016). Status of hepatitis C virus vaccination a recent update. World Journal of Gastroenterology, 22(2), 862-873.
3. Abdulqadir, M.O., Rashid, P.M.A., Hussain, A.H., Rahman, H.S., and Ezzaddin, S.A. (2023). Genetic characterization of hepatitis B virus genotypes among patients with chronic infection in Sulaimaniyah city, Iraq. Peer Journal,11(1): 44-54.
4. Adewole, O.O., Anteyi, E., Ajuwon, Z., Wada, I., Elegba, F., and Ahmad, P. (2019). Hepatitis B and C virus coinfection in Nigerian patients with HIV infection. Journal of Infectious diseases in developing countries, 3(5): 369-375.
5. Agbesor, I., Smart, A., and Zacchaeus, J. (2013). Hepatitis B profile among blood donors in the capital territory Abuja, Nigeria. International Journal of Science and Research, 5(7).
6. Agboghoroma, C.O., and Ukaire, B.C. (2020). Prevalence and risk factors of human immunodeficiency virus and hepatitis C infection among pregnant women attending antenatal care at a tertiary hospital in Abuja, Nigeria. Nigerian Medical Journal, 61: 245-251.
7. Ahmad, A.E., Adamu, G.B., Bolanle, O,P,M., Shettima, K., Bello, Y.J., Idris, N.A., Mohammed, T.I., Abdulqadri, O.O., Sumayya, H.M., Ahmed, B.S., Afolaranmi, T., Claudia, H., Atiene, S.S, Ayuba, Z. and Adebola, T.O. (2019). Pattern of prevalent hepatitis B virus genotypes in Zaria, Nigeria. Niger Postgraduate Medical Journal, 26(2): 80-86.
8. Al-Mohri, H., Cooper, C., Murphy, T. and Klein, M.B. (2005). Validation of a simple model for predicting liver fibrosis in HIV/hepatitis C virus-coinfected patients, HIV Medicine, 6(6): 375-378.
9. Althoff, K.N., Stewart, C.N., Humes, E., Zhang, J., Gerace, L., Boyd, C. M., Wong, C., Justice, A. C., Gebo, K. A., Thorne, J. E., Rubtsova, A. A., Horberg, M. A., Silverberg, M. J., Leng, S. X., Rebeiro, P. F., Moore, R. D., Buchacz, K., andKasaie, P. (2022). The shifting age distribution of people with HIV using antiretroviral therapy in the United States. AIDS, 36(3), 459-471.
10. Altice, F.L., Zelenev, A., Mazhnaya, A., and Basu, S. (2018). Hepatitis C virus treatment as prevention in an extended network of people who inject drugs in the USA: a modelling study. Infectious Diseases, 18(2): 215-224.
11. Aminu, M., and Aba H. (2016). Seroprevalence of hepatitis B virus serological markers among pregnant Nigrian women. Annals of African Medicine,15(**1**).
12. Amini, A., Varsaneux, O., Kelly, H., Tang, W., Chen, W., Boeras, D.I., Falconer, J., Tucker, J.D., Chou, R., Ishizaki, A., Easterbrook, P., and Peeling, R.W. (2017). Diagnostic accuracy of tests to detect hepatitis B surface antigen: A systematic review of the literature and meta-analysis. BMC Infectious Diseases, 17(1), 698.
13. Anugwom, C.M., Allaire, M., Akbar, S.M.F., Sultan, A., Bollipo, S., Mattos, A. Z., andDebes, J. D. (2021). Hepatitis B-related hepatocellular carcinoma: surveillance strategy directed by immuneepidemiology. Hepatoma Research, 7, 23.

14. Arankalle, V.A., Gandhi, S., Lole, K.S., Chadha, M.S., Gupte, G.M., andLokhande, M.U. (2011). An outbreak of hepatitis B with high mortality in India: Association with precore, basal core promoter mutants and improperly sterilized syringes. Journal of Viral Hepatitis, 18(4), 20–28.
15. Archampong, T. N., Boyce, C. L., Lartey, M., Sagoe, K. W., Obo-Akwa, A., Kenu, E., Blackard, J. T., andKwara, A. (2017). HBV genotypes and drug resistance mutations in antiretroviral treatment-naive and treatment-experienced HBV-HIV-coinfected patients. Antiviral therapy, 22(1), 13–20.
16. Audsley, J., Avihingsanon, A., Littlejohn, M., Bowden, S., Matthews, G. V., Fairley, C. K., Lewin, S.R., andSasadeusz, J. (2020). Long-term TDF-inclusive ART and progressive rates of HBsAg loss in HIV-HBV coinfection-lessons for functional HBV cure? Journal of Acquired Immune Deficiency Syndromes, 84(5), 527–533.
17. Bacon, B.R., andKanwal, F. (2011). Does treatment alter the natural history of chronic HCV? Chronic Hepatitis C Virus Advances in Treatment, Promise for the Future, 4(6), 103-104.
18. Bedimo, R., Westfall, A. O., Mugavero, M., Drechsler, H., Khanna, N., and Saag, M. (2010). Hepatitis C virus coinfection and the risk of cardiovascular disease among HIV-infected patients. HIV Medicine, 11(7), 462–468.
19. Berretta, M., Garlassi, E., Cacopardo, B., Cappellani, A., Guaraldi, G., Cocchi, S., De Paoli, P., Lleshi, A., Izzi, I., Torresin, A., Di Gangi, P., Pietrangelo, A., Ferrari, M., Bearz, A., Berretta, S., Nasti, G., Di Benedetto, F., Balestreri, L., Tirelli, U., and Ventura, P. (2011). Hepatocellular carcinoma in HIV-infected patients: check early, treat hard. Oncologist, 16(9), 1258-1269.
20. Bhattarai, M., Baniya, J. B., Aryal, N., Shrestha, B., Rauniyar, R., Adhikari, A., Koirala, P., Oli, P. K., Pandit, R.D., Stein, D. A., and Gupta, B.P. (2018). Epidemiological profile and risk factors for acquiring HBV and/or HCV in HIV-infected population groups in Nepal. Biomed Research International, 9241679.
21. Bhuyan, G.S., Aftab, U.N., Rosy, S., Farjana, A.N., Nusrat, S., Suprovath, K.S., and Muhammad, A.S. (2021). Frequency of heparitisB,C and HIV among transfusion dependent beta thalassemia patients in Dhaka. Journals of Infectious Diseases, 13(1): 89-95.
22. Boyd, A., Bottero, J., Miailhes, P., Lascoux-Combe, C., Rougier, H., Girard, P. M., Serfaty, L., and Lacombe, K. (2017). Liver fibrosis regression and progression during controlled hepatitis B virus infection among HIV-HBV patients treated with tenofovir disoproxil fumarate in France: a prospective cohort study. Journal of International AIDS Society, 20(1), 21426.
23. Bruss, V. (2007). Hepatitis B virus morphogenesis. World Journal of Gastroenterology, 13(1), 65-73.
24. Buchanan, R., Khakoo, S. I., Coad, J., Grellier, L. and Parkes, J. (2017). Hepatitis C bio-behavioural surveys in people who inject drugs-a systematic review of sensitivity to the theoretical assumptions of respondent driven sampling. Harm Reduction Journal, 14, 44.
25. Buseri, F.I., Seiyaboh, E. and Jeremiah, Z.A. (2010). Surveying infections among pregnant women in the Niger Delta, Nigeria. Journal of Global Infectious Diseases, 2:203-211.
26. Buxton, J., Jafari, S., Copes, R., Baharlou, S., andEtminan, M. (2010). Tattooing and the risk of transmission of hepatitis C: a systematic review and meta- analysis. International Journalof Infectious Diseases, 14(11): 928-940.
27. Carias, A.M., and Hope, T.J. (2019). Barriers of mucosal entry of HIV/SIV. Current Immunology Reviews, 15(1): 4–13.
28. Centers for Disease Control and Prevention (CDC). (2012). Hepatitis C FAQs for Health Professionals. Archive.
29. Centers for Disease Control and Prevention (CDC). (2013). Monitoring selected national HIV prevention and care objectives by using HIV surveillance data-United States and 6 U.S. dependent areas. Surveillance Supplemental Report, 18(5), 1-35.
30. Cento, V., Carmen, M., Salvatore, D., Romina S., and Yue, H. (2013). Overlapping structure of hepatitis B virus genome and immune selection pressure are critical forces modulating HBV evolution. Journal of General Virology, 94, 143-149.
31. Chang, M.H., You, SL., Chen, C.J., Liu, C.J., Lee, C.M., Lin, S.M., Chu, H.C., Wu, T.C., Yang, S.S., Kuo, H.S., Chen, D.S. and Taiwan Hepatoma Study Group. (2009). Decreased incidence of hepatocellular carcinoma in hepatitis B vaccinees: A 20-year follow-up study. Journal of National Cancer Institute, 101(19), 1348–1355.

32. Collin, F., Duval, X., Le Moing, V., Piroth, L., Al Kaied, F., Massip, P., Villes, V., Chêne, G., and Raffi F. (2009). Ten-year incidence and risk factors of bone fractures in a cohort of treated HIV1-infected adults. AIDS, 23(8), 1021–1024.
33. Cunha, L., Carrilho, C., Bhatt, N., Loforte, M., Maueia, C., Fernandes, F., Guisseve, A., Mbofana, F., Maibaze, F., Mondlane, L., Ismail, M., Dimande, L., Machatine, S., Lunet, N., Liu, Y. T., Gudo, E. S. andPineau, P. (2019). Hepatocellular carcinoma: Clinical-pathological features and HIV infection in Mozambican patients. Cancer Treatment and Research Communications, 19, 100129.
34. Dammacco, F., Tucci, F.A., and Lauletta, G. (2010). Pegylated interferon-alpha, ribavirin, and rituximab combined therapy of hepatitis C virus-related mixed cryoglobulinemia: Along-term study. Blood, 116: 343-353.
35. Debes, J.D., Bohjanen, P.R., andBoonstra, A. (2016). Mechanisms of accelerated liver fibrosis progression during HIV infection. Journal of Clinical and Translational Hepatology, 4(4), 328-335.
36. Deeks, S.G., Overbaugh, J., Phillips, A., and Buchbinder, S. (2015). HIV infection. Nature Reviews. Disease Primers, 1(1), 15-35.
37. Deressa, T., Damtie, D., and Fonseca, K. (2017). The burden of hepatitis B virus (HBV) infection, genotypes and drug resistance mutations in human immunodeficiency virus-positive patients in Northwest Ethiopia. PLoS One, 12(1), 90-149.
38. Diwe, C.K., Okwara, E.C., Enwere, O.O., Azike, J.E., and Nwaimo, N.C. (2013). Sero-prevalence of hepatitis B virus and hepatitis C virus among HIV patients in a suburban university teaching hospital in soth-east Nigeria. Pan African Medical Journal 16: 7.
39. Easterbrook, P.J., Roberts, T., Sands, A. and Peeling, R. (2017). Diagnosis of viral hepatitis. Current Opinion in HIV and AIDS, 12(3), 302–314.
40. Egypt Today staff. (2021). Hepatitis C prevalence in Egypt drops from 7% to 2% thanks to Sisi's initiative. Egypt today (Cairo).
41. Ejiofor, O.S., Emechebe, G.O., Igwe, W.C., Ifeadike, C.O., &Ubajaka, C.F. (2010). Hepatitis C virus infection in Nigerians. Nigerian Medical Journal,51(4), 173.
42. Epeldegui, M., Vendrame, E., & Martínez-Maza, O. (2010). HIV-associated immune dysfunction and viral infection: role in the pathogenesis of AIDS-related lymphoma. Immunologic Research, 48(1-3), 72-83.
43. European Association for the Study of the Liver. (2012). EASL clinical practical guidelines: management of chronic hepatitis B virus infection. Journal of Hepatology, 58(1): 201.
44. Falade-Nwulia, O., &Thio, C. L. (2011). Liver disease, HIV and aging. SexualHealth8(4), 512–520.
45. Faria, N.R., Rambaut, A., Suchard, M.A., Baele, G., Bedford, T., Ward, M.J., Tatem, A.J., Sousa, J.D., Arinaminpathy, N., Pepin, J., Posada, D., Peeters, M., Pybus, O.G., andLemey, P. (2014). HIV epidemiology. The early spread and epidemic ignition of HIV-1 in human populations. Science, 346(6205): 56–61.
46. Feld, J.J., Ocama, P., and Ronald, A (2015). The liver in HIV in Africa. Antiviral Therapy,10: 953-965.
47. Forrester, J.E., Rhee, M.S., Mcgovern, B.H., Sterling, R.K., Knox, T.A., andTerrin, N. (2012). The association of HIV viral load with indirect markers of liver injury. Journal of Viral Hepatitis, 19(2), 202-211.
48. Fromontin, R. and Chomont, N. (2020). HIV persistence in subsets of CD4+ T cells: 50 shades of reservoirs. Seminar of Immunology, 10, 1016.
49. Gandhi, R.T., Wurcel, A., Lee, H., Mcgovern, B., Shopis, J., Geary, M.,Sivamurthy, R., Sax, P.E., andUkomadu, C. (2005). Response to hepatitis B vaccine in HIV-1-positive subjects who test positive for isolated antibody to hepatitis B core antigen: implications for hepatitis B vaccine strategies. The Journal of Infectious Diseases, 191(9), 1435–1441.
50. Gilson, R.J., Hawkins, A.E., Beecham, M.R., Ross, E., and Waite, J. (1997). Interactions between HIV and hepatitis B virus in homosexual men: effects on the natural history of infection. AIDS, 11: 597-606.
51. Giordano, T.P., Kramer, J.R., Souchek, J., Richardson, P. and El-Serag, H.B. (2004). Cirrhosis and hepatocellular carcinoma in HIV-infected veterans with and without the hepatitis C virus: a cohort study, 1992-2001. Archives of Internal Medicine, 164(21), 2349-2354.
52. Gish, R., Jia, J.D., Locarnini, S., andZoulim, F. (2012). Selection of chronic hepatitis B therapy with high barrier to resistance. The Lancet, Infectious Diseases, 12(4), 341–353.

53. Glebe, D., and Urban, S. (2007). Viral and cellular determinants involved in hepadnaviral entry. World Journal of Gastroenterology, 13(1), 22–38.
54. Goodsell, D.S. (2015). Illustrations of the HIV life cycle. Current Topics in Microbiology and Immunology, 38(9), 243–252.
55. Götz, H. M., Van Doornum, G., Niesters, H. G., Den Hollander, J. G., Thio, H. B., and De Zwart, O. (2005). A cluster of acute hepatitis C virus infection among men who have sex with men–results from contact tracing and public health implications. AIDS19, 969–974.
56. Hagan, H., Pouget, E.R., and Des Jarlais, D.C. (2011). A meta-analysis and systematic review of interventions to prevent hepatitis C virus infection in people who inject drugs. TheJournal of Infectious Diseases, 204(1), 74-83.
57. Hagan, L.M., andSchinazi, R.F. (2013). Best strategies for global HCV eradication. Liver International,33(1), 68-79.
58. Hamza, M. A., Samaila, A., Yakassai, A., Babashani, M., Borodo, M. M. and Habib, A. G. (2013). Prevalence of Hepatitis B and C virus infection among HIV infected individuals in tertiary hospital in Northen western Nigeria, Nigeria. Journal of Basic Clinical Sciences,10: 76-81.
59. Hawkins, C., Grant, J., Ammerman, L. R., Palella, F., Mclaughlin, M., Green, R., Mcgregor, D., andStosor, V. (2016). High rates of hepatitis C virus (HCV) cure using directacting antivirals in HIV/HCV-coinfected patients: a real-world perspective. The Journal of Antimicrobial Chemotherapy, 71(9), 2642–2645.
60. Health policy project (2010). 2014-2018 National HIV program funding advocacy campaign.
61. Hernandez-Ramirez, R., Meredith, S.S., Robert, D. and Eric, A.E. (2017). Cancer risk in HIV- infected people in the USA from 1996 to 2012: a population-based registry-linkage study. The Lancet.
62. Herrscher, C., Pastor, F., Burlaud-Gailard, J., Dumans, A., Seigneuret, F., Moreau, A, Patient, R., Eymieux, S., De Rocquigny, H., Hourioux, C., Roingeard, P., and Blanchard, E. (2020). Hepatitis B virus entry into HepG2-NTCP cells requiresmclanthrin-mediated endocytosis. Cell Microbiogy, 22(8), 13205.
63. Hu, J., and Liu, K. (2017). Complete and Incomplete Hepatitis B Virus Particles: Formation, Function, and Application. Viruses, 9(3), 56-61.
64. Hu, J., Lin, Y.Y., Chen, P.J., Watashi, K., andWakita, T. (2019). Cell and Animal Models for Studying Hepatitis B Virus Infection and Drug Development. Gastroenterology, 156(2), 338–354.
65. Hundie, G. B., Raj, V. S., GebreMichael, D., Pas, S. D., andHaagmans, B. L. (2017) "Genetic diversity of hepatitis C virus in Ethiopia," PLoS One, 12(12), 23-35.
66. Isa, I., Aminu, M., Abdullahi, S.A., Sani, M.A., and Esona, M.D. (2015). Seroprevalence of hepatitis B virus in a tertiary institution in North Western Nigeria. African Journal of Microbiology Research, 9(3), 171-179.
67. Jaquet, A., Odutola, M., Ekouevi, D.K., Tanon, A., Oga, E., Akakpo, J., Charurat, M., Zannou, M. D., Eholie, S. P., Sasco, A. J., Bissagnene, E., Adebamowo, C., Dabis, F., &IeDEA West Africa Collaboration. (2015). Cancer and HIV infection in referral hospitals from four West African countries. Cancer Epidemiology, 39(6), 1060-1065.
68. Jaquet, A., Tchounga, B., Tanon, A., Bagny, A., Ekouevi, D. K., Traore, H. A., Sasco, A. J., Maiga, M. and Dabis, F. (2018). Etiology of hepatocellular carcinoma in West Africa, a case-control study. International Journal of Cancer, 143: 869-77.
69. Jaroszewicz, J., Reiberger, T., Meyer-Olson, D., Mauss, S., Vogel, M., Ingiliz, P., Payer, B. A., Stoll, M., Manns, M. P., Schmidt, R. E., Flisiak, R., Wedemeyer, H., Peck-Radosavljevic, M., Rockstroh, J. and Cornberg, M. (2012). Hepatitis B surface antigen concentrations in patients with HIV/HBV co-infection. PLoS One,7(8), 31-43.
70. Jelagat, M. K., Fatuma Faraj, S., Hellen IrusaLukhaka, D., Eric Wang'welo, D., Laban Kipkemei, M., and Adrian, G., 2020. Burden of hepatitis B infection among high-risk populations in Western Kenya. Journal of Infectous Disease Epidemiology, 6(3), 121-143.
71. Joseph, C.F., Puddy, M.A. and Campo, D.S. (2012). Epidemic history of hepatitis C infection in two remote communities in Nigeria, West Africa. Journal of General Virology, 93(7):1410-1421.
72. Joshi, D., O'grady, J., Dieterich, D., Gazzard, B., and Agarwal, K. (2011). Increasing burden of liver disease in patients with HIV infection. The Lancet, 377(9772), 1198-1209.

73. Kabarambi, A., Balinda, S., Abaasa, A., Cogill, D., andOrrell, C. (2022). Determinants and reasons for switching anti-retroviral regimen among HIV-infected youth in a large township of South Africa (2002-2019). AIDS Research and Therapy, 19: 32-40.
74. Kao, J.H. (2008). Diagnosis of hepatitis B virus infection through serological and virological markers. Expert Reviews in Gastroenterology and Hepatology, 2(1), 553–562.
75. Kaur, R., Pooja, S., Gupta, K.G., Fidele, N.K., and Dinesh, K. (2020). Structure-activity-relationship and mechanistic insights for anti-HIV natural products. Molecules, 25(9), 2070.
76. Kazuhide, T., Eri, Y., Satoshi S., Yoshinari, U., Hiroma, F., Ryo, Y., and Fumihito, H. (2020). Horizontal transmission of hepatitis B virus genotype C among members of a wrestling club in Japan. American Journal of Case Reports,21: 925044-1-925044-6.
77. Kenfack-Momo, R., Kenmoe, S., Takuissu, G.R., Ebogo-Belobo, J.T., Kengne-Nde,´ C., Mbaga, D.S., Tchatchouang, S., Oyono, M. G., Kenfack-Zanguim, J., LontuoFogang, R., MbongueMikangue, C. A., Zeuko'oMenkem, E., NdzieOndigui, J. L., Kame-Ngasse, G. I., Magoudjou-Pekam, J. N., Taya-Fokou, J. B., Bowo-Ngandji, A., NkieEsemu, S., KamdemThiomo, D., MoundipaFewou, P., Ndip, L., andNjouom, R. (2022). Epidemiology of hepatitis B virus and/ or hepatitis C virus infections among people living with human immunodeficiency virus in Africa: A systematic review and meta-analysis. PLoS ONE, 17(5), 0269250
78. Kilonzo, S. B., Gunda, D. W., Mpondo, B. C. T., Bakshi, F. A. and Jaka, H. (2018). Hepatitis B virus infection in Tanzania: current status and challenges. Journal of Tropical Medicine, 4(23), 46-96.
79. Kilonzo, S.B., Gunda, D.W., Kashasha, F., andMpondo, B.C. (2017). Liver fibrosis and hepatitis B coinfection among art naïve HIV-infected patients at a tertiary level hospital in northwestern Tanzania: a cross-sectional study. Journal of Tropical Medicine,5(62), 9-130.
80. Kim, A. (2016). Hepatitis C virus. Annals of Internal Medicine, 165(5), 33-48.
81. Kim, A. Y., Chung, R. T., and Polsky, B. (2000). Human immunodeficiency virus and hepatitis B and C coinfection: pathogenic interactions, natural history, and therapy. AIDS Clinical Reviews, 12(1), 263–306.
82. Kim, C.W., and Chang, K.M. (2013). Hepatitis C virus: virology and life cycle. Clinical Molecular Hepatology,19(1), 17-25.
83. Kirk, G. D., Mehta, S. H., Astemborski, J., Galai, N., Washington, J., Higgins, Y., Balagopal, A., and Thomas, D. L. (2013). HIV, age, and the severity of hepatitis C virus-related liver disease: a cohort study. Annals of Internal Medicine, 158(9), 658–666.
84. Klein, R.A., Vianello, M., Hasselman, F., Adams, B.G., Adams, R.B., Alper, S., Aveyard, M., Babalola, M., Bahnik,S., Batra, R., Berkics, M., Bernstein, M.J., Berry, D.R., Kovacs, C. and Kurapov, G. (2018). Many labs 2: Investigating variation in replicability across samples and settings. Advances in Methods and Practices in Psychological Science, 1(4): 443-490.
85. Ko, H.M., Hernandez-Prera, J.C., Zhu, H., Dikman, S.H., Sidhu, H.K., Ward, S.C., andThung, S.N. (2012). Morphologic features of extrahepatic manifestations of hepatitis C virus infection. Clinical and Developmental immunology, 7(40), 138
86. Kovacs, A., Karim, R., Mack, W. J., Xu, J., Chen, Z., Operskalski, E., Frederick, T., Landay, A., Voris, J., Spencer, L. S., Young, M. A., Tien, P. C., Augenbraun, M., Strickler, H. D., and Al-Harthi, L. (2010). Activation of CD8 T cells predicts progression of HIV infection in women coinfected with hepatitis C virus. TheJournal of Infectious Diseases, 201(6), 823–834.
87. Kowdley, K.V., Wang, C.C., Welch, S., Roberts, H., andBrosgart, C.L. (2012). Prevalence of chronic hepatitis B among foreignborn persons living in the United States by country of origin. Hepatology, 56(2), 422–433.
88. Kramvis, A., and Clements, C.J. (2010). Implementing a birth dose of hepatitis B vaccine for home deliveries in Africa—Too soon? Vaccine, 28(6), 6408–6410.
89. Krassenburg, L.A.P., Maan, R., Ramji, A., Manns, M.P., Cornberg, M., Wedemeyer, H., de Knegt, R.J., Hansen, B.E., Janssen, H.L.A., de Man, R.A., Feld, J.J., and van der Meer, A.J. (2021). Clinical outcomes following DAA therapy in patients with HCV-related cirrhosis depend on disease severity. Journal of Hepatology, 74(5), 1053–1063.
90. Kumar, M., Pahuja, S., Khare, P., and Kumar, A. (2023). Current Challenges and Future Perspectives of Diagnosis of Hepatitis B Virus. Diagnostics, 13(3), 368 1-12.

91. Kwon, H. and Lok, A.S. (2011). Hepatitis B Therapy. Nature Reviews Gastroenterology and Hepatology, 8(5), 275–284.
92. Lackner, C., Struber, G., Liegl, B., Leibl, S., Ofner, P., Bankuti, C., Bauer, B., andStauber, R.E. (2005). Comparison and validation of simple noninvasive tests for prediction of fibrosis in chronic hepatitis C. Hepatology,41(6), 1376–1382.
93. Lam, P.J., Omish, M.C., and Burns, S. (1993). Infrequent vertical transmission of hepatitis C virus. Journal of Infectious Diseases, 167: 572-576.
94. Lamontagne, R.J., Bagga, S., and Bouchard, M.J. (2016). Hepatitis B virus molecular biology and pathogenesis. Hepatoma Research, 2(12), 163–186.
95. Lar, P.M., Pam, V.K., Christopher, P.B., Gwamzhi, I., and Mawak, T. (2013). Prevalence and immune status of HIV/AIDS coinfected pregnant women. African Journal of Experimental Microbiology, 14: 120-126
96. Lavanchy, D. (2004). Hepatitis B virus epidemiology, disease burden, treatment, and current and emerging prevention and control measures. Journal of Viral Hepatitis, 11(2), 97–107.
97. Leumi, S., Bigna, J. J., Amougou, M. A., Ngouo, A., Nyaga, U. F., andNoubiap, J. J. (2020). Global Burden of Hepatitis B Infection in People Living With Human Immunodeficiency Virus: A Systematic Review and Meta-analysis. Clinical Infectous Disease, 71(2), 799-806.
98. Li, K.W., Kramvis, A., Liang, S., He, X., Chen, Q. Y., Wang, C., Yang, Q. L., Hu, L. P., Jia, H. H., and Fang, Z. L. (2017). Higher prevalence of cancer related mutations 1762T/1764A and PreS deletions in hepatitis B virus (HBV) isolated from HBV/HIV co-infected compared to HBV-mono-infected Chinese adults. Virus Research, 2(27), 88-95.
99. Liang, X., Bi, S., Yang, W., Wang, L., Cui, G., Cui, F.,Zhang, Y., Liu, J., Gong, X., Chen, Y., Wang, F., Zheng, H., Wang, F., Guo, J., Jia, Z., Ma, J., Wang, H., Luo, H., Li, L., Jin, S., Hadler, S.C. and Wang, Y. (2009). Epidemiological serosurvey of hepatitis B in China—Declining HBV prevalence due to hepatitis B vaccination. Vaccine,27(47), 6550–6557.
100. Lin, C.L., and Kao, J.H. (2015). Hepatitis B virus genotypes and variants. Cold Spring Harbour Perspective in Medicine,5(5), 123-142.
101. Lindenbach, B.D., and Rice, C.M. (2003). Molecular Biology of flaviviruses. Advances in Virus Research, 59: 23-61.
102. Little, J.W., Falace, D.A., Miller, C. and Rhodus, N.L. (2013). Dental Management of the Medically Compromised Patient, 151.
103. Lo Re, V.III., Kallan, M. J., Tate, J. P., Localio, A. R., Lim, J. K., Goetz, M. B., Klein, M. B., Rimland, D., Rodriguez-Barradas, M. C., Butt, A. A., Gibert, C. L., Brown, S. T., Park, L., Dubrow, R., Reddy, K. R., Kostman, J. R., Strom, B. L., and Justice, A. C. (2014). Hepatic decompensation in antiretroviral-treated patients co-infected with HIV and hepatitis C virus compared with hepatitis C virus-monoinfected patients: a cohort study. Annals of Internal Medicine, 160(6), 369–379.
104. Locarnini, S., Hatzakis, A., Chen, D. S., and Lok, A. (2015). Strategies to control hepatitis B: Public policy, epidemiology, vaccine and drugs. Journal of Hepatology, 62(21), 76–86.
105. Lok, A.S., & McMahon, B.J. (2009). Chronic hepatitis B: update. Hepatology 50(3): 661–662.
106. Mabeya, S. N., Ngugi, C., Lihana, R. W., Khamadi, S. A., andNyamache, A. K. (2017). Predominance of Hepatitis B Virus Genotype A Among Treated HIV Infected Patients Experiencing High Hepatitis B Virus Drug Resistance in Nairobi, Kenya. AIDS research and human retroviruses, 33(9), 966–969.
107. MacLachlan, J.H., and Cowie, B.C. (2012). Liver cancer is the fastest increasing cause of cancer death in Australians. The Medical Journal of Australia, 197(9), 492–493.
108. Magoro, T., Gachara, G., andMavhandu, L., (2016). Serologic and genotypic characterization of hepatitis B virus in HIV-1 infected patients from Southwest and Littoral Regions of Cameroon. Virology Journal, 13(1), 78-101.
109. Mahoney, F.J. (1999). Update on diagnosis, management, and prevention of hepatitis B virus infection. Clinical Microbiology Reviews, 12(2), 351–366.
110. Manka, P., Jens, V., Guido, G., and Ali, C. (2016). Liver failure due to acute viral hepatitis (A-E). Visceral Medicine, 32(2), 80-85.
111. Manns, M.P., Buti, M., Gane, E., Pawlotsky, J. M., Razavi, H., Terrault, N., andYounossi, Z. (2017). Hepatitis C virus infection. Nature Review Disease Primers, 3, 17006.

112. Maponga, T. G., Glashoff, R. H., Vermeulen, H., Robertson, B., Burmeister, S., Bernon, M., Omoshoro-Jones, J., Ruff, P., Neugut, A. I., Jacobson, J. S., Preiser, W. and Andersson, M. I. (2020). Hepatitis B virus-associated hepatocellular carcinoma in South Africa in the era of HIV. BMCGastroenterology, 20(1),226.
113. Marks, K.M., Clarke, R.M., Bussel, J.B., Talal, A.H., andGlesby, M.J. (2009). Risk factors for thrombocytopenia in HIV-infected persons in the era of potent antiretroviral therapy. Jounal of Acquired Immune Deficiency Syndromes, 52(5), 595–599.
114. Martinez-Sierra, C., Arizcorreta, A., Díaz, F., Roldán, R., Martín-Herrera, L., Pérez-Guzmán, E., and Girón-González, J. A. (2003). Progression of chronic hepatitis C to liver fibrosis and cirrhosis in patients coinfected with hepatitis C virus and human immunodeficiency virus. Clinical Infectious Diseases,36(4), 491–498.
115. Mbaawuga, E., Iroegbu, C., and Ike, A. (2014). Hepatitis B virus serological patterns in Benue state, Nigeria. Open Journal of Medical Microbiology, 4, 1-10.
116. Mei, J., and Zhao, J. (2018). Prediction of HIV-1 and HIV-2 proteins by using Chou's pseudo amino acid compositions and different classifiers. Scientific Reports, 8(1), 23-59.
117. Messina, V., Lorenzo, O., Giovanni, D.C., Ernesto, C., Vincenzo, L., Antonio, R., Valerio, R., Angella, S., Riccardo, N., Filomena, S., Fabio, C., Pisaturo, M. and Nicola, C. (2021). Directly Acting Antiviral-based Treatment for HCV-infected people who inject drugs: A multicenter real-life study. Life, 11(1): 17.
118. Miailhes, P., Trabaud, M. A., Pradat, P., Lebouché, B., Chevallier, M., Chevallier, P., Zoulim, F., andTrepo, C. (2007). Impact of highly active antiretroviral therapy (HAART) on the natural history of hepatitis B virus (HBV) and HIV coinfection: relationship between prolonged efficacy of HAART and HBV surface and early antigen seroconversion. Clinical infectious diseases: an official publication of the Infectious Diseases Society of America, 45(5), 624–632.
119. Moir, S., Chun, T.W., andFauci, A.S. (2011). Pathogenic mechanisms of HIV disease. Annual Review of Pathology, 6(5), 223–248.
120. Monaco, S., Sergio, F., Alberto, G., Gianlugi, Z. and Sara, M. (2012). HCV-related nervous system disorders. Journal of Immunology Research, 1:1155.
121. Morozov, V. A., andLagaye, S. (2018). Hepatitis C virus: Morphogenesis, infection and therapy.World journal of hepatology, 10(2), 186–212.
122. Moyer, V.A. (2013). Screening for hepatitis C virus infection in adults: U.S Preventive Services Taskforce recommendation statement. Annals of Internal Medicine, 159(5), 349-357.
123. Murray, A.J., Kwon, K.J., Farber, D.L., andSiliciano, R.F. (2016). The Latent Reservoir for HIV-1: How Immunologic Memory and Clonal Expansion Contribute to HIV-1 Persistence. Journal of Immunology, 197(2): 407–417.
124. Naggie, S., Cooper, C., Saag, M., Workowski, K., Ruane, P., Towner, W. J., Marks, K., Luetkemeyer, A., Baden, R.P., Sax, P.E., Gane, E., Santana-Bagur, J., Stamm, L.M., Yang, J.C., German, P., Dvory-Sobol, H., Ni, L., Pang, P.S., McHutchison, J.G., and Stedman, C. A. (2015). Ledipasvir and sofosbuvir for HCV in patients coinfected with HIV-1. The New England Journal of Medicine, 37(8): 705–713.
125. Naing, L., Winn, T. and Rusli, B.N. (2006). Practical issues in calculating the sample size for prevalence studies. Arch Orofacial Science, 1: 9-14.
126. Naito, H., Hayashi, S. and Abe, K. (2001). Rapid and specific genotyping system for hepatitis B virus corresponding to six major genotypes by PCR using type-specific primers. Journal of Clinical Microbiology, 39(1): 362-364.
127. Ndako, J.A., Echeonwu, G.O.N., Nwakiti, O.O., and Onovoh, E.M. (2012). Hepatitis B virus seroprevalence among pregnant females in Northern Nigeria. Research Journal of Medical Sciences, 6(3): 129-133.
128. Ndifontiayong, A. N., Ali, I. M., Sokoudjou, J. B., Ndimumeh, J. M., andTume, C. B. (2021). The effect of HBV/HCV in response to HAART in HIV patients after 12 months in Kumba Health District in the Southwest Region of Cameroon. Tropical medicine and infectious disease, 6(3), 150.
129. Nelson, P.K., Mathers, B.M., Cowie, B., Hagan, H., Des Jarlais, D., Horyniak, D., and Degenhardt, L. (2011). Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: Results of systematic reviews. Lancet, 378(9791), 571 –583.

130. Ni, Y., Florian, A.L., Stefan, M., Ralf, K., Holger, S. and Stephan, U. (2014). Hepatitis B and D viruses exploit sodium taurocholate co-transporting polypeptide for species-specific entry into hepatocytes. Journal of Gastroenterology, 146(4):1070-1083.
131. Nnakenyi, I.D., Uchechukwu, C., and Nto-ezimah, U. (2020). Prevalence of hepatitis B and C virus co-infection in HIV positive patients attending a health institution in southeast Nigeria. Africa Health Sciences, 20(2): 579-586.
132. Ocama, P., Seremba, E., Apica, B., andOpio, K. (2015). Hepatitis B and HIV co-infection is still treated using lamivudine-only antiretroviral therapy combination in Uganda. Africa Health Science,15(3) 28-33.
133. Ochoa-Callejero, L., Pérez-Martínez, L., Rubio-Mediavilla, S., Oteo, J.A., Martínez, A., and Blanco, J.R. (2013). Maraviroc, a CCR5 antagonist, prevents development of hepatocellular carcinoma in a mouse model. PLoS One, 8(1), 53992.
134. Ochwoto, M., Kimotho, J.H., andOyugi, J. (2016). Hepatitis B infection is highly prevalent among patients presenting with jaundice in Kenya. BMC Infectous Disease 16(101), 452-491.
135. Ogunfemi, M.K., Olawumi, H.O., Abdulfatai, B.O., Modu, B.K., Sikiru, A.B., Kabir, A.D., Idayat, A.D., and Akeem, O.S. (2017). Prevalence of hepatitis B surface antigen-negative blood donors in Ilorin, Nigeria: A cross-sectional study. Malawi Medical Journal, 29(1), 32-36.
136. Okechukwu, N., Godwin, M., Desmond, F.E., and Patrick, O. (2014). Seroprevalence of hepatitis viral infections in HIV tested positive individuals in Owerri, Imo state, Nigeria. Journal of AIDS and Clinical Research, 5: 272-27.
137. Okeke, E., Mark Davwar, P., Mullen, B., Duguru, M., Agbaji, O., Sagay, A., Murphy, R., and Hawkins, C. (2021). The impact of HIV on hepatocellular cancer survival in Nigeria. Tropical Medicine and International Health, 26(3), 335-342.
138. Operskalski, E. A., and Kovacs, A. (2011). HIV/HCV co-infection: pathogenesis, clinical complications, treatment, and new therapeutic technologies. Current HIV/AIDS Reports, 8(1), 12–22.
139. Owens, D.K., Davidson, K.W., Krist, A.H., Barry, M.J., Cabana, M., andCaughey, A.B. (2020). “Screening for hepatitis C virus infection in adolescents and adults: US Preventive Taskforce Recommendation Statement”. Journal of Advanced Medicine in America,323(10), 970-975.
140. Patient, R., Hourioux, C., andRoingeard, P. (2009). Morphogenesis of hepatitis B virus and its subviral envelope particles. Cellular Microbiology, 11(11), 1561–1570.
141. Pawlotsky, J.M. (2014). New hepatitis C therapies: the toolbox, strategies, and challenges. Gastroenterology, 146(5), 1176–1192.
142. Pecoraro, V., Banzi, R., Cariani, E., Chester, J., Villa, E., D’Amico, R., Bertele, V., andTrenti, T. (2019). New direct-acting antivirals for the treatment of patients with hepatitis C virus infection: a systematic review of randomized controlled trials. Journal of Clinical andExperimental Hepatology, 9(4), 522-538.
143. Penin, F., Dubuisson, J., Rey, F. A., Moradpour, D., andPawlotsky, J. M. (2014). Structural biology of hepatitis C virus. Hepatology, 39, 5–19.
144. Pennap, G.R.I., Nwachukwu, O., Ishaleku, D., and Ombugadu, R.J. (2011). Hepatitis B virus carriage among students of a Nigerian tertiary institution: a cohort of eligible blood donors. Research Journal of Medicine, 5: 903.
145. Pfaender, S., Von Hahn, T., Steinmann, J., Ciesek, S., and Steinmann, E. (2016). Prevention strategies for blood-borne viruses-in the era of vaccines, direct acting antivirals and antiretroviral therapy. Reviews in Medical Virology, 26(5), 330–339.
146. Pierra-Rouviere, C., Dousson, C.B., and Tavis, J.E. (2020). HBV Replication Inhibitors. Antiviral Research, 179, 104815.
147. Pinato, D.J., Dalla Pria, A., Sharma, R., and Bower, M. (2017). Hepatocellular carcinoma: an evolving challenge in viral hepatitis and HIV coinfection. AIDS, 31(5), 603-611.
148. Plymoth, A., Viviani, S. and Hainaut, P. (2009). Control of hepatocellular carcinoma through hepatitis B vaccination in areas of high endemicity: Perspectives for global liver cancer prevention. Cancer Letters, 286(1), 15–21.
149. Pollicino, T., Cacciola, I., Saffioti, F. and Raimondo, G. (2014). Hepatitis B virus PreS/S gene variants: pathobiology and clinical implications. Journal of Hepatology, 61(2), 408–417.

150. Ponde, R.A. (2011). Hidden hazards of HCV transmission. Medical Microbiology and Immunology,200(1), 7-11.
151. Previsani, N., andLavanchy, D. (2002). Hepatitis B. Department of Communicable Diseases Surveillance and Response, World Health Organisation, Geneva.
152. Prifti, G.M., Moianos, D., Giannakopoulou, E., Pardali, V., Tavis, J.E., andZoidis, G. (2021). Recent Advances in Hepatitis B Treatment. Pharmaceuticals, 14(5), 417.
153. Puoti, M., Bruno, R., Soriano, V., Donato, F., Gaeta, G. B., Quinzan, G. P., Precone, D., Gelatti, U., Asensi, V., Vaccher, E., andHIV HCC Cooperative Italian-Spanish Group. (2004). Hepatocellular carcinoma in HIV-infected patients: epidemiological features, clinical presentation and outcome. AIDS, 18(17), 2285-2293.
154. Raina, D., Wilkins, T., Malcolm, J.K. and Schade, R.R. (2010). Hepatitis C: diagnosis and treatment. American Family Physician, 81(11), 1351-1357.
155. Ray-Saraswati, L., Sarna, A., Sebastian, M. P., Sharma, V., Madan, I., Thior, I., Pulerwitz, J., and Tun, W. (2015). HIV, hepatitis B and C among people who inject drugs: high prevalence of HIV and Hepatitis C RNA positive infections observed in Delhi, India. BMC Public Health,15, 726.
156. Ripoli, M., andPazienza, V. (2011). Impact of HCV genetic differences on pathobiology of disease. Expert Review of Anti-Infective Therapy, 9(5), 747–759.
157. Rodrigo, L. (2020). Hepatitis B and C. IntechOpen, London. pp 112.
158. Rossotti, R., Merli, M., Mazzarelli, C., De Carlis, R. M., Travi, G., Vecchi, M., Viganò, R., Lauterio, A., Raimondi, A., Belli, L. S., De Carlis, L. G., andPuoti, M. (2022). Similar survival but higher and delayed hepatocellular carcinoma recurrence in HIV-positive compared to negative cirrhotics undergoing liver transplantation. Digestive Liver Disease: official journal of the Italian society of Gastroenterology and the Italian Association for the study of the Liver, 55(2), 268-275.
159. Rotman, Y., and Liang, T. J. (2009). Coinfection with hepatitis C virus and human immunodeficiency virus: virological, immunological, and clinical outcomes. Journal of Virology, 83(15), 7366–7374.
160. Ryom, L., Lundgren, J.D., De Wit, S., Kovari, H., Reiss, P., Law, M., El-Sadr, W., Monforte, A. D., Mocroft, A., Smith, C., Fontas, E., Dabis, F., Phillips, A., Sabin, C., and D:A:D Study Group. (2016). Use of antiretroviral therapy and risk of end-stage liver disease and hepatocellular carcinoma in HIV-positive persons. AIDS, 30(11), 1731-1743.
161. Sagnelli, E., Sagnelli, C., Pisaturo, M., Macera, M., and Coppola, N. (2014). Epidemiology of acute and chronic hepatitis B and delta over the last 5 decades in Italy. World Journal of Gastroenterology,20(24), 7635–7643.
162. Schweitzer, A., Horn, J., Mikolajczyk, R.T., Krause, G., and Ott, J.J. (2015). Estimations of worldwide prevalence of chronic hepatitis B virus infection: A systematic review of data published between 1965 and 2013. The Lancet, 386(10003), 1546–1555.
163. Shahani, L. and Hamill, R. J. (2016). Therapeutics targeting inflammation in the immune reconstitution inflammatory syndrome. Translational Research: the journal of laboratory and clinical medicine, 167(1): 88–103.
164. Shahriar, S., Araf, Y., Ahmad, R., Kattel, P., Sah, G.S., Rahaman, T.I., Sadiea, R.Z., Sultana, S., Islam, M.S., Zheng, C., and Hossain, M.G. (2022). Insights Into the Coinfections of Human Immunodeficiency Virus-Hepatitis B Virus, Human Immunodeficiency Virus-Hepatitis C Virus, and Hepatitis B Virus-Hepatitis C Virus: Prevalence, Risk Factors, Pathogenesis, Diagnosis, and Treatment. Frontiers in Microbiology, 12, 780887.
165. Shapatava, E., Nelson, K. E., Tsertsvadze, T. and Del Rio, C. (2006). Risk behaviors and HIV, hepatitis B, and hepatitis C seroprevalence among injection drug users in Georgia. Drug and Alcohol Dependence, 82(1), 35–38.
166. Shi, P., Chen, Z., Meng, J., Su, M., Yang, X., Fan, W., Shi, H., Gao, Y., and Lu, X. (2021). Molecular transmission networks and pre-treatment drug resistance among individuals with acute HIV-1 infection in Baoding, China. PLoSONE, 16(12), 26-70.
167. Siebers, A., and Finlay, B. B. (1996). M cells and the pathogenesis of mucosal and systemic infections. Trends in Microbiology, 4(1), 22–29.
168. Singal, A.K., and Anand, B.S. (2009). Management of hepatitis C virus infection in HIV/HCV co-infected patients: clinical review. World Journal of Gastroenterology, 15(30), 3713–3724.

169. Singh, K.P., Crane, M., Audsley, J., Avihingsanon, A., Sasadeusz, J., and Lewin, S.R. (2017). HIV-hepatitis B virus coinfection: epidemiology, pathogenesis, and treatment. AIDS, 31(15), 2035-2052.
170. Slogrove, A.L. (2021). It is a question of equity: Time to talk about children who are HIV-exposed and "HIV-free". Journal of the International AIDS Society, 24(11), 25-50.
171. Soriano, V., Puoti, M., Sulkowski, M., Cargnel, A., Benhamou, Y., Peters, M., Mauss, S., Bräu, N., Hatzakis, A., Pol, S., andRockstroh, J. (2007). Care of patients coinfected with HIV and hepatitis C virus: 2007 updated recommendations from the HCV-HIV International Panel. AIDS,21(9), 1073–1089.
172. Spearman, C.W., Afihene, M., Ally, R., Apica, B., Awuku, Y., Cunha, L., Dusheiko, G., Gogela, N., Kassianides, C., Kew, M., Lam, P., Lesi, O., Lohouès-Kouacou, M. J., Mbaye, P. S., Musabeyezu, E., Musau, B., Ojo, O., Rwegasha, J., Scholz, B., Shewaye, A. B., Tzeuton, C., Sonderup, M.W., and Gastroenterology and Hepatology Association of sub-Saharan Africa (GHASSA). (2017). Hepatitis B in sub-Saharan Africa: strategies to achieve the 2030 elimination targets. The Lancet Gastroenterology & Hepatology, 2(12), 900-909.
173. Stabinski, L., O'Connor, S., Barnhart, M., Kahn, R.J., and Hamm, T.E. (2015). Prevalence of HIV and hepatitis B virus co-infection in sub-Saharan Africa and the potential impact and program feasibility of hepatitis B surface antigen screening in resource-limited settings. Journal of Acquired Immune Deficiency Syndrome, 68(3), 274-285.
174. Sule, W.F., Okonko, I.O., Yumusa, I.P., Odu, N.N., and Frank-Peterside, N. (2011) Hepatitis B surface antigen HBsAg) and risk factors of transmission among patients attending hospital in Ankpa, Kogi State, Nigeria. Nature and Science, 9:37- 41.
175. Sulkowski, M.S., Eron, J.J., Wyles, D., Trinh, R., Lalezari, J., Wang, C., Slim, J., Bhatti, L., Gathe, J., Ruane, P.J., Elion, R., Bredeek, F., Brennan, R., Blick, G., Khatri, A., Gibbons, K., Hu, Y.B., Fredrick, L., Schnell, G., Pilot-Matias, T., Tripathi, R., Da Silva-Tillmann, B., McGovern, B., Campbell, A.L., andPodsadecki, T. (2015). Ombitasvir, paritaprevir co-dosed with ritonavir, dasabuvir, and ribavirin for hepatitis C in patients co-infected with HIV-1: a randomized trial. The Journal of American Medical Association, 313(12), 1223–1231.
176. Sulkowski, M.S., Gardiner, D.F., and Rodriguez – Torres M. (2014). Daclatasvir plus sofosbuvir for previously treated or untreated chronic HCV infection. New England Journal ofMedicine,37(2), 11-21.
177. Tassachew, Y., Abebe, T., Belyhun, Y., Teffera, T., Shewaye, A. B., Desalegn, H., Andualem, H., Kinfu, A., Mulu, A., Mihret, A., Howe, R., andAseffa, A. (2022). Prevalence of HIV and Its Co-infection with hepatitis B/C virus among chronic liver disease patients in Ethiopia. Hepatic Medicine: evidence and research, 14, 67-77.
178. Thio, C. L., Seaberg, E. C., Skolasky, R. Jr., Phair, J., Visscher, B., Muñoz, A., Thomas, D. L., and Multicenter AIDS Cohort Study. (2002). HIV-1, hepatitis B virus, and risk of liver-related mortality in the Multicenter Cohort Study (MACS). Lancet, 360(9349): 1921–1926.
179. Thomas, J., Ruggiero, A., Paxton, W.A., andPollakis, G. (2020). Measuring the success of HIV-1 cure strategies. Frontiers in Cellular and Infection Microbiology, 10(5), 134-145.
180. Thompson, N.D., Perz, J.F., Moorman, A.C. and Holmberg, S.D. (2009). Nonhospital health care-associated hepatitis B and C virus transmission: United States, 1998–2008. Annals of Internal Medicine, 150(1), 33–39.
181. Thornton, A.C., Jose, S., Bhagani, S., Chadwick, D., Dunn, D., Gilson, R., Main, J., Nelson, M., Rodger, A., Taylor, C., Youssef, E., Leen, C., Gompels, M., Kegg, S., Schwenk, A., Sabin, C., and UK Collaborative HIV cohort (UK CHIC) steering committee. (2017). UK Collaborative HIV cohort (UK CHIC) steering committee. Hepatitis B, hepatitis C, and mortality among HIV-positive individuals. AIDS, 31(18), 2525-2532.
182. Tre'po, C., Chan, H.L., and Lok, A. (2014). Hepatitis B virus infection. Lancet, 384(9959), 2053-2063.
183. Tuyama, A. C., Hong, F., Saiman, Y., Wang, C., Ozkok, D., Mosoian, A., Chen, P., Chen, B. K., Klotman, M. E., and Bansal, M. B. (2010). Human immunodeficiency virus (HIV)-1 infects human hepatic stellate cells and promotes collagen I and monocyte chemoattractant protein-1 expression: implications for the pathogenesis of HIV/hepatitis C virus-induced liver fibrosis. Hepatology52(2), 612–622.
184. Tyas, A.A., Raeni, S.F., Sakti, S.P., andSabarudin, A. (2021). Recent Advances of Hepatitis B Detection towards Paper-Based Analytical Devices. The Scientific World Journal, 6(6), 43-57.

185. Van de Laar, Thijs, J.W., Mathews, G.V., Prins, M. and Danta, M. (2010). Acute hepatitis C in HIV-infected men who have sex with men: an emerging sexually transmitted infection. AIDS,24(12): 1799-1812.
186. Venkataramani, M., Hutton, N., Colombani, P., Anders, R.A. and Agwu, A.L. (2010). Hepatocellular carcinoma in a teenager with perinatally acquired HIV Infection without hepatitis B or C coinfection: a case report. AIDS Patient Care and STDs, 24(11), 693-696.
187. Vidya Vijayan, K., Karthigeyan, K.P., Tripathi, S.P., and Hanna, L.E. (2017). Pathophysiology of CD4 T-cell depletion in HIV-1 and HIV-2 infections. Frontiers in Immunology, 8(5), 580-601.
188. Vu, L.N-C., Patricia, M.D., Gotsch, M.D. and Robert, C. (2010). Caring for women and newborns with hepatitis B or C. American Family Physician, 82(10): 1225-1229.
189. Wada N., Jacobsen, L.P., Cohen M., French A.P., and Munoz A. (2014). Cause-specific mortality among HIV-infected individuals, by CD4(+) cell count at HAART initiation, compared with HIV-uninfected individuals. AIDS, 282(5), 765.
190. Walker, D.R., Pedrosa, M.C., Manthena, S.R., Patel, N. and Marx, S.E. (2015). Early view of the effectiveness of new direct-acting antiviral (DAA) regimens in patients with hepatitis C virus (HCV). Advances in Therapy, 32(11), 1117–1127.
191. Wan, Q., Anugwom, C., Desalegn, H., andDebes, J. D. (2022). Hepatocellular carcinoma in Hepatitis B and Human Immunodeficiency Virus coinfection in Africa: a focus on surveillance. Hepatoma Research, 2022;8:39.
192. Weber, R., Sabin, C.A., Friis-Møller, N., Reiss, P., El-Sadr, W. M., Kirk, O., Dabis, F., Law, M. G., Pradier, C., De Wit, S., Akerlund, B., Calvo, G., Monforte, A.d, Rickenbach, M., Ledergerber, B., Phillips, A. N. and Lundgren, J. D. (2006). Liver-related deaths in persons infected with the human immunodeficiency virus: the D:A:D study. Archives of Internal Medicine, 166(15), 1632-1641.
193. Westbrook, R.H., and Dusheiko, G. (2014). Natural history of hepatitis C. Journal of Hepatology, 61(1), 58-68.
194. Wing, E. J. (2017). The aging population with HIV infection. Transactions of the American Clinical Climatological Association, 128, 131–144.
195. Woo, A.S.J., Kwok, R. and Ahmed, T. (2017). Alpha-Interferon Treatment in Hepatitis B. Annals of Translational Medicine, 5(7), 159.
196. World Health Organization (WHO) (2013).consolidated guidelines on the use of antiretroviral drugs for treating and preventing HIV infection.
197. World Health Organization. (2017). Global hepatitis report.https://www.who.int/publications/i/item/9789241565455.
198. World Health Organization (WH0). (2019). Hepatitis C: Global epidemiology of hepatitis C virus; an update. World Health Journal, 584(2), 755.
199. World Health Organization. (2021). Hepatitis B key facts. https://www.who.int/newsroom/factsheets/detail/hepatitis-b.
200. Yang, R., Gui, X., Ke, H., Xiong, Y., and Gao, S. (2019). Long-term observation on hepatitis B surface antigen seroclearance in therapy experienced HIV/HBV co-infected Chinese. Journal of Viral Hepatitis, 27(2): 127-134.
201. Yang, R., Gui, X., Ke, H., Xiong, Y., and Gao, S. (2021). Combination antiretroviral therapy is associated with reduction in liver fibrosis scores in patients with HIV and HBV co-infection. AIDS Research and Therapy, 18(1), 98.
202. Yehia, B. R., Herati, R. S., Fleishman, J. A., Gallant, J. E., Agwu, A. L., Berry, S. A., Korthuis, P. T., Moore, R. D., Metlay, J. P., Gebo, K. A. and HIV Research Network. (2014). Hepatitis C virus testing in adults living with HIV: a need for improved screening efforts. PLoS One,9(7), 102-766
203. Younossi, Z.M., Stephanova, M.A., Fendy, M., and Mishra, B.P. (2013). A knowledge about infection in patients with chronic hepatitis C. Journal of Viral Hepatology,320(5), 505.
204. Yousif, M., Mudawi, H., and Hussein, W., (2014). Genotyping and virological characteristics of hepatitis B virus in HIV-infected individuals in Sudan. International Journal of Infectous Diseases,29(1), 25-32.
205. Yu, S., Yu, C., Li, J., Liu, S., Wang, H., and Deng, M. (2020). Hepatitis B and hepatitis C prevalence among people living with HIV/AIDS in China: A systematic review and Meta-analysis. Virology Journal, 17(1), 127.

206. Yu, Y., Cheng, S.T., Ren, F., Chen, Y., Shi, X.F. and Wong, V.K.W. (2021). SIRT7 restricts HBV transcription and replication through catalyzing desuccinylation of histone H3 associated with cccDNAminichromosome. Clinical Sciences, 135:1505-1522.
207. Yuan, Y., Yuan, H., Yang, G., Yun, H., Zhao, M. and Liu, Z. (2020). IFN-ά confers epigenetic regulation of HBV cccDNAminichromosome by modulating GCN5-mediated succinylation of histone H3K79 to clear HBV cccDNA. Clinical Epigenetics, 12: 135.
208. Zaman, H., Rahman, A. and Mahmuda, Y. (2018). Epidemiology of Hepatitis B virus infection in Bangladesh: Prevalence among general population, risk groups and genotype distribution. Genes, 9(11): 541.
209. Zerbato, J.M., and Lewin, S.R. (2020). A cure for HIV: How would we know? Lancet HIV, 7(5), 304–306.
210. Zhang, F., Hao, Z., Yasong, W., Zhihui, D., Yao, Z., Nora, K., Marc, B., Zunyuo, W., Ye, M., Decai, Z., Xia, L., Hua, F., Jing, L., Wei, P.C. and Hong, S. (2014). HIV, hepatitis B virus, and hepatitis C virus in patients in the China national free antiretroviral treatment program, 2010-2012: a retrospective observational cohort study. Lancet Infectious Diseases, 14(11): 1065-1072.
211. Zhang, Q., Liao, Y., and Chen, J. (2015). Epidemiology study of HBV genotypes and antiviral drug resistance in multi-ethnic regions from Western China. Scientific Report, 5(1), 74-83.
212. Zheng, Y., Lu, Y., Ye, Q., Xia, Y., Zhou, Y., Yao, Q. and Wei, S. (2011). Should chronic hepatitis B mothers breastfeed? A meta-analysis. BMC Public Health, 11: 502.
213. Zoulim, F. and Durantel, D. (2015). Antiviral therapies and prospects for a cure of chronic hepatitis B. Cold Spring Harb Perspective in Medicine, 5(4).
214. Zoulim, F., Lebossé, F., andLevrero, M. (2016). Current Treatments for Chronic Hepatitis B Virus Infections. Current Opinion in Virology, 18, 109–116.