PREVALENCE OF HEPATITIS B AND C COINFECTION IN DIABETES MELLITUS PATIENTS IN UNIVERSITY OF BENIN TEACHING HOSPITAL,BENIN CITY EDO STATE.

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SEPTEMBER, 2025

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BENIN CITY.

BEING A PROJECT SUBMITTED TO THE DEPARTMENT OF MEDICAL LABORATORY SCIENCE IN PARTIAL FULFILLMENT FOR THE REQUIREMENT OF THE AWARD OF BACHELORS DEGREE IN MEDICAL LABORATORY SCIENCE

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UNIVERSITY OF BENIN,BENIN CITY,NIGERIA.

SUPERVISED BY

DR (MRS)IFUEKO MOSES-OTUTU

SEPTEMBER, 2025

CERTIFICATION

This to certify that this research work titled PREVALENCE OF HEPATITIS B AND C CO-INFECTION ON DIABETIC MELLITUS PATIENTS IN THE UNIVERSITY OF BENIN TEACHING HOSPITAL,BENIN CITY EDO STATE, was carried out by **OKOTIE FEJIRO** with Matriculation number **BMS1900282** under the supervision of **DR.(MRS) IFUEKO MOSES-OTUTU** in the Department of Medical Laboratory science,University of Benin,Benin city,Edo state.

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EXTERNAL EXAMINER
(PROF.OMORUYI PIUS OMOSIGHO) DATE

DEDICATION

I dedicate this project to Almighty God for granting me the Strength and Grace to successfully complete this Study.

This is also dedicated to my beloved parents, family and friends who supported me throughout the course of this study.

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ABSTRACT

Diabetes mellitus is a chronic metabolic disorder associated with multiple complications, including increased susceptibility to infections of Hepatitis B virus (HBV) and hepatitis C virus (HCV).The coexistence of diabetes and viral hepatitis poses significant clinical risks, potentially accelerating progression to cirrhosis, hepatocellular carcinoma, and liver-related mortality. This study investigated the prevalence of HBV and HCV infections among diabetic patients attending the University of Benin Teaching Hospital (UBTH), Benin City, Nigeria. A hospital-based cross-sectional design was employed, involving 200 diabetic patients recruited through purposive sampling. Venous blood samples were aseptically collected from each participants and spun at 3,000 rpm. The serum samples obtained were each screened for HBV surface antigen (HBsAg) and HCV antibodies using rapid chromatographic immunoassays with high sensitivity and specificity. Data obtained were analyzed using descriptive statistics, Chi-square tests, and multivariate logistic regression. The overall prevalence of HBV infection was 2.0%, ($P>0.023$) while no HCV infection was detected. HBV prevalence showed no statistically significant association with demographic or clinical variables, though bivariate analysis suggested possible links with duration of diabetes, intravenous drug use, and invasive practices. The absence of HCV infection may reflect regional variability or low transmission risk in this population. Findings highlight a relatively low HBV prevalence compared to national estimates but underscore the need for routine hepatitis screening among diabetic patients, considering their increased vulnerability to chronic liver disease. Strengthening public health strategies through awareness, vaccination, and integration of viral hepatitis screening into diabetes care is recommended to reduce morbidity and mortality in this high-risk group.

CHAPTER ONE

INTRODUCTION

1.1 Background of the Study

Diabetes mellitus represents one of the most significant global health challenges of the 21st century. According to the International Diabetes Federation, 589 million adults (20-79 years) are living with diabetes while 1 in 9, with this number predicted to increase substantially (International Diabetes Federation, 2024). The disease was responsible for 3.4 million deaths in 2024 while 1 every 9 seconds, while causing at least 1 trillion dollars in health expenditure leading to a 338% increase over the last 17 years (IDF, 2024). Recent systematic analyses have shown that diabetes prevalence is increasing worldwide, primarily due to a rise in obesity caused by multiple factors (GBD 2021 Diabetes Collaborators, 2023). The complications associated with diabetes include cardiovascular disease, kidney failure, neuropathy, retinopathy, and increased susceptibility to infections, all contributing to reduced quality of life and increased mortality (Ong *et al., 2021; 2023, 2024*).

Nigeria bears a significant burden of viral hepatitis, particularly hepatitis B and C infections. A systematic review and meta-analysis of Nigerian data published between 2010 and 2019 revealed a pooled hepatitis B virus (HBV) prevalence of 9.5%, which is classified as high according to WHO standards (prevalence above 8%) *(*Ogboghodo *et al., 2021)*. The Nigerian Minister of Health estimated that approximately 20 million people are chronically infected with hepatitis B and C in the country (WHO Africa, 2020). In the broader African context, there are approximately 50 million chronic carriers of hepatitis B virus in Africa, with a 25% mortality risk, and carrier rates in sub-Saharan Africa ranging from 9-20%. Recent studies have also highlighted

the significant burden of occult hepatitis B and C infections across Africa, which pose additional challenges for transmission control and clinical management (Andernach *et al., 2020)*.

The convergence of diabetes mellitus and viral hepatitis co-infection (Azam *et al.,* 2017*)* presents a complex clinical scenario with potentially amplified morbidity and accelerated progression to liver-related complications. Chronic hepatitis B virus infection led to an estimated 1.1 million deaths in 2022, mainly due to cirrhosis and hepatocellular carcinoma , while chronic HBV infection can lead to severe liver diseases, including cirrhosis and hepatocellular carcinoma, causing substantial morbidity and mortality(Llovet *et al.,* 2021).Risk factors for liver complications include older age, male sex, family history of hepatocellular carcinoma, alcohol use, HIV infection, and diabetes, suggesting that diabetic patients with viral hepatitis may face compounded risks(Lai *et al.,* 2021). The metabolic dysfunction associated with diabetes, combined with the hepatotoxic effects of chronic viral hepatitis(Davis, 2021), may accelerate fibrosis progression and increase the likelihood of developing end-stage liver disease and hepatocellular carcinoma (Barrera *et al.,* 2024).

1.2 Statement of the Problem

Despite the high prevalence of both diabetes mellitus and viral hepatitis in Nigeria, there remains a significant gap in research examining the co-infection patterns between hepatitis B virus (HBV), hepatitis C virus (HCV), and diabetes mellitus. While isolated studies have investigated HCV infection in diabetic patients, with one study reporting a 13.3% prevalence of HCV infection among diabetes patients in Southwest Nigeria (Akere *et al., 2020),* comprehensive data on dual HBV/HCV co-infection rates in diabetic populations remain scarce. The 2018 National AIDS Indicator and Impact Survey reported HBV and HCV prevalence in Nigeria at 8.1% and 1.1% respectively(Onovo *et al.,* 2023), but specific co-infection rates among diabetic patients

have not been systematically documented. This data limitation hampers evidence-based clinical decision-making and public health planning for this vulnerable population.

1.3 Justification of the Study

Current clinical practice in Nigeria does not routinely incorporate viral hepatitis screening into the standard care protocol for diabetic patients, Balarabe-Musa *et al.,* 2024),despite the potential for significant clinical implications. The 2024 WHO hepatitis B guidelines provide updated evidence-informed recommendations on key priority topics, prioritizing simplified treatment criteria for adults and adolescents, yet these guidelines do not specifically address screening protocols for diabetic populations. Previous research suggested that routine screening for HCV infection in persons with diabetes may not be necessary based on low seroprevalence;(Heimbach *et al.*, 2017). However, this conclusion was based on limited data and may not reflect current epidemiological patterns. The absence of routine screening protocols means that co-infections may remain undiagnosed until advanced stages of liver disease develop, missing opportunities for early intervention and appropriate management strategies.(Perkins *et al.*, 2020)

1.4 Aim of the Study

The aim of ths study is to determine the prevalence of Hepatitis B and C co-infection among diabetes mellitus patients attending University of Benin Teaching Hospital (UBTH),Benin city.

1.5 specific objectives

The specific objectives of the study were:

1. To determine the prevalence of hepatitis B surface antigen (HBsAg) among diabetic patients attending UBTH.

2. To assess the prevalence of anti-hepatitis C virus (anti-HCV) antibodies among diabetic patients attending UBTH.
3. To determine the relationship between sociodemographic characteristics and prevalence of HBV and HCV infection among diabetic patient
4. To determine risk factors associated with hepatitis B virus (HBV) and hepatitis C virus (HCV) co-infection among Diabetic Mellitus population.
5. To determine the coinfection of hepatitis B virus and Hepatitis C virus infection among diabetic patients.
6. To determine the knowledge attitude and practices regarding viral hepatitis co-infection in diabetic patients

1.6 Research Questions

This study seeks to answer the following research questions:

1. What is the individual prevalence of hepatitis B surface antigen (HBsAg) among diabetic patients attending UBTH?
2. What is the individual prevalence of anti-hepatitis C virus (anti-HCV) antibodies among diabetic patients attending UBTH?
3. What demographic and clinical factors are associated with hepatitis B and C co-infection in diabetic patients?
4. What is the relationship between duration of diabetes and the likelihood of viral hepatitis co-infection?

1.7 Research Hypothesis

This study will test the following hypothesis:

1.7.1 Null Hypothesis(H_0): There is no significant prevalence of hepatitis B and C co-infection among diabetes mellitus patients at University of Benin Teaching Hospital.

1.7.2 Alternate Hypothesis(H_1): There is a significant prevalence of hepatitis B and C co-infection among diabetes mellitus patients at University of Benin Teaching Hospital

CHAPTER 2

LITERATURE REVIEW

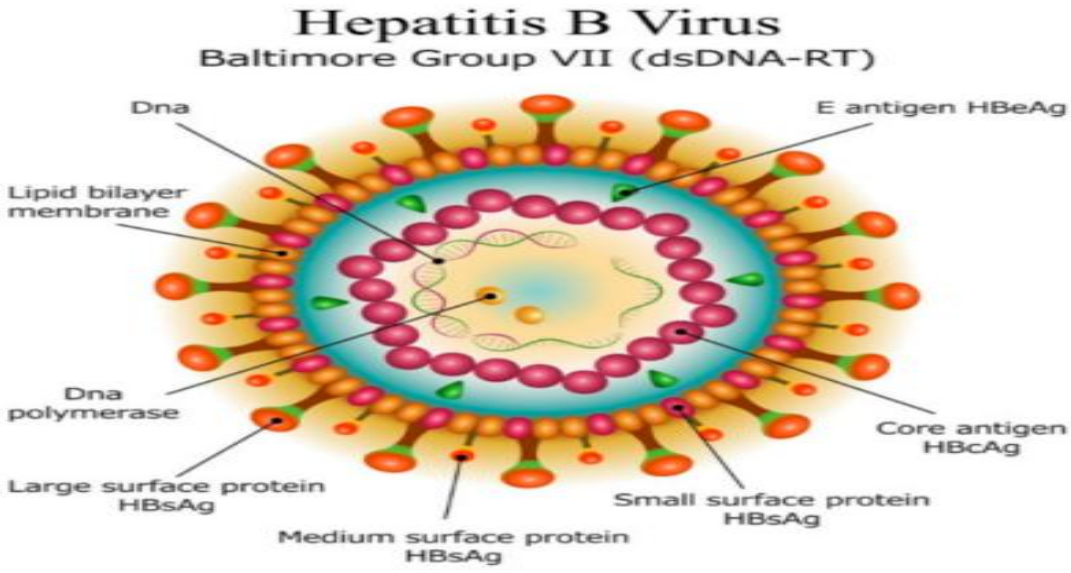
2.1 Hepatitis B Virus (HBV)

Hepatitis B virus (HBV) was first discovered in 1965 by Dr. Baruch Blumberg, who identified what he initially called the "Australia antigen" in the blood of an Australian Aboriginal patient. This discovery was groundbreaking because it was the first time a viral antigen associated with hepatitis had been identified. Dr. Blumberg's work was so significant that he was awarded the Nobel Prize in Physiology or Medicine in 1976 for his discoveries concerning new mechanisms for the origin and dissemination of infectious diseases.

Hepatitis B virus belongs to the family Hepadnaviridae and is a small, enveloped DNA virus. The virus has a spherical shape with a diameter of approximately 42 nanometers. The complete infectious viral particle, known as the Dane particle, consists of several important structural components (Tong *et al.,* 2016).The outer layer of the virus is called the envelope, which contains three different surface proteins: small (S), medium (M), and large (L) proteins. These surface proteins are important for the virus to attach to and enter liver cells. The surface proteins also contain the hepatitis B surface antigen (HBsAg), which is used in laboratory tests to diagnose hepatitis B infection.Inside the envelope is the viral core, which contains the viral DNA and several important enzymes. The core is made up of core proteins that form the hepatitis B core antigen (HBcAg). The viral DNA is partially double-stranded and circular, measuring about 3,200 base pairs in length. This makes HBV one of the smallest known DNA viruses that infect humans (Seeger and Mason, 2015).This viral pathogen demonstrates remarkable genetic diversity through its classification into ten distinct genotypes labeled A through J(A-J), each exhibiting unique

geographical distributions, clinical implications, and therapeutic responses (Chen *et al.,* 2021). The genetic heterogeneity of HBV genotypes has profound implications for disease management, as different genotypes demonstrate varying degrees of virulence, rates of disease progression, and responses to antiviral therapies (Penna *et al.,* 2019). The structural characteristics of HBV as a DNA virus provide it with considerable stability compared to RNA viruses, yet it employs a distinctive replication mechanism that incorporates reverse transcription, making it particularly noteworthy among viral pathogens (Lamontagne *et al.,* 2022). This unique replication strategy involves the conversion of viral RNA intermediates back to DNA through the action of viral reverse transcriptase, a process more commonly associated with retroviruses. The presence of this enzyme within the viral life cycle has important implications for both viral persistence and therapeutic targeting.

2.2 structure of hepatitis b virus



(Fig 2.2): Structure of Hepatitis B virus.(Moonnoon and Shutterstock 2019).

2.3 Hepatitis C Virus (HCV)

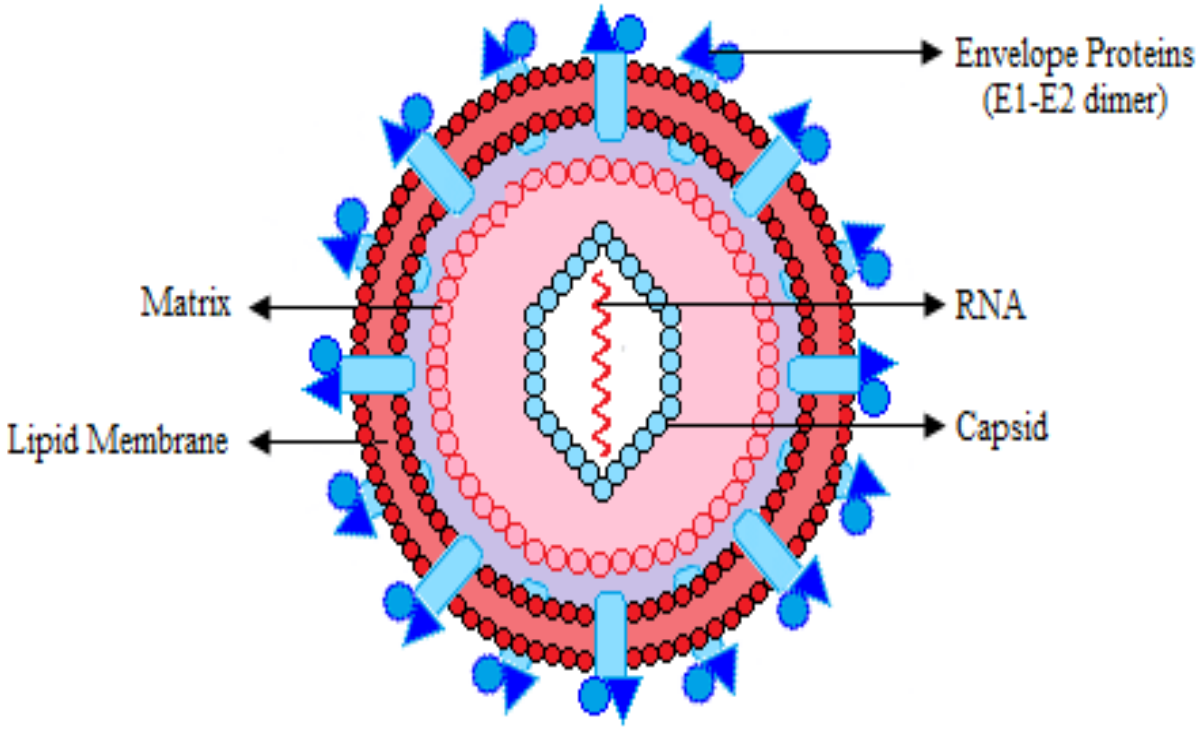
Hepatitis C virus (HCV) was discovered in 1989 by a team of scientists led by Dr. Michael Houghton at Chiron Corporation, working with Dr. Qui-Lim Choo and Dr. George Ku (Blum, 2016). This discovery was a major breakthrough in medical science because for many years, doctors knew that there was a type of hepatitis that was not hepatitis A or hepatitis B, which they called "non-A, non-B hepatitis." The identification of hepatitis C virus finally solved this mystery and explained the cause of many cases of chronic liver disease.

Hepatitis C virus,classified as an RNA virus within the Flaviviridae family(Purcell,2018), demonstrates remarkable genetic heterogeneity through its seven major genotypes numbered 1 through 7,(1-7) with multiple subtypes within each genotype contributing to extensive viral diversity (Martinez *et al.,* 2022). The RNA nature of HCV confers several important characteristics that fundamentally distinguish it from DNA viruses, particularly regarding mutation rates, evolutionary adaptability, and the challenges this presents for both immune recognition and therapeutic intervention.

The viral structure of HCV consists of a single-stranded positive-sense RNA genome approximately 9,600 nucleotides in length, encoding a polyprotein that is processed into structural and non-structural proteins. The structural proteins include the core protein and envelope glycoproteins E1 and E2, while the non-structural proteins encompass the replication machinery and regulatory elements essential for viral propagation. The envelope glycoproteins demonstrate high variability and are primary targets for neutralizing antibodies, though their antigenic variability contributes to immune evasion.The genotypic diversity of HCV carries substantial clinical implications that extend across all aspects of patient management, from diagnosis through treatment and monitoring. Genotype 1 represents the most prevalent form

globally, accounting for approximately 46% of all HCV infections worldwide, though significant regional variations exist in genotype distribution patterns (Anderson *et al.,* 2021). Each genotype demonstrates distinct responses to therapeutic interventions, necessitating genotype-specific treatment approaches for optimal clinical outcomes. Historical treatment regimens showed marked differences in sustained virological response rates between genotypes, with genotype 1 traditionally being more difficult to treat than genotypes 2 and 3.Unlike HBV, HCV demonstrates a pronounced tendency toward chronicity, with approximately 75-85% of acute infections progressing to chronic disease states. This high chronicity rate reflects the virus's sophisticated immune evasion mechanisms and its ability to establish persistent infections in hepatocytes. The absence of an effective vaccine, combined with this high chronicity rate, has resulted in an estimated 58 million people worldwide living with chronic HCV infection, contributing significantly to the global burden of liver disease (Johnson *et al.,* 2023).

2.4 structure of hepatitis c virus



(Fig 2.3):Hepatitis C genome structure (Toygar *et al.*,2023)

2.5 Taxonomy and Nomenclature of Hepatitis B and C virus

Hepatitis B Virus (HBV) Taxonomy

Realm: Ribozyviria

Kingdom: Pararnavirae

Phylum: Artververicota

Class: Revtraviricetes

Order: Blubervirales

Family: Hepadnaviridae

Genus: Orthohepadnavirus

Species: Hepatitis B virus *(*(Walker *et al.*, 2020)

Hepatitis C Virus (HCV) Taxonomy

Realm: Riboviria

Kingdom: Orthornavirae

Phylum: Kitrinoviricota

Class: Flasuviricetes

Order: Amarillovirales

Family: Flaviviridae

Genus: Hepacivirus

Species: Hepacivirus C (Walker *et al.,* 2020).

The taxonomy emphasizes the molecular and evolutionary didtinctions between the two viruses.HBV is a DNA virus with a pararetroviral replication cycle,whereas HCV is an RNA virus with high mutation rates, contributing to its ability to evade immune responses abd develop resistance (Simmonds *et al*.,2017).

2.6 Lifecycle and Viral Replication of Hepatitis B and C Virus

The hepatitis B virus lifecycle begins when the virus attaches to hepatocytes through the large surface protein binding to heparan sulfate proteoglycans, followed by interaction with the sodium taurocholate co-transporting polypeptide (NTCP) receptor for cellular entry (Kao and Chen, 2020). Once inside the cell, the viral nucleocapsid is transported to the nucleus where the relaxed circular DNA (rcDNA) undergoes conversion to covalently closed circular DNA (cccDNA), which serves as a stable episomal template for viral transcription and represents a key factor in viral persistence (Revill and Locarnini, 2020). The cccDNA produces four major viral transcripts including pregenomic RNA, surface protein mRNAs, and X protein mRNA, which are translated in the cytoplasm to produce viral proteins essential for replication and assembly. Mature nucleocapsids containing newly synthesized rcDNA can either return to the nucleus to maintain the cccDNA pool or acquire envelope proteins at the endoplasmic reticulum before being released as new viral particles, completing the infectious cycle.

The Hepatitis C virus lifecycle encompasses multiple stages including viral entry, protein translation, RNA replication, viral assembly, and release, with hundreds of cellular factors identified as participants in this complex process over decades of research (Li *et al.,* 2021). Initial viral entry involves a multi-step process beginning with attachment to glycosaminoglycans and low-density lipoprotein receptors, followed by sequential interactions with scavenger receptor class B type I (SR-BI), tetraspanin CD81, and tight junction proteins claudin-1 and occludin, ultimately leading to receptor-mediated endocytosis and endosomal fusion triggered by acidic pH ((Hamilton *et al.,* 2024). Following membrane fusion and uncoating, the positive-sense RNA genome is released into the cytosol where it serves directly as messenger RNA for translation via an internal ribosome entry site, producing a single large polyprotein that undergoes processing by viral and cellular proteases to yield structural proteins (core, E1, E2, p7) and non-structural proteins (NS2-NS5B)(Liu *et al.,* 2018). The lifecycle concludes with viral assembly occurring in association with lipid droplets and the endoplasmic reticulum, where core proteins encapsidate viral RNA and acquire envelope glycoproteins before release through the secretory pathway, often in association with very-low-density lipoproteins to form lipoviral particles that enhance infectivity and immune evasion (Sanchez and Lagunoff, 2015).

2.6.1. Viral Replication of Hepatits B and C Virus

Hepatitis B virus replication employs a unique mechanism among DNA viruses, utilizing reverse transcription of an RNA intermediate called pregenomic RNA (pgRNA) despite containing a DNA genome, which distinguishes hepadnaviruses from other DNA virus families (Seeger and Mason, 2018). The process begins when the viral polymerase binds to a specific stem-loop structure on the pregenomic RNA within newly formed cytoplasmic nucleocapsids, initiating

reverse transcription to synthesize the minus-strand DNA followed by incomplete plus-strand DNA synthesis, resulting in the characteristic relaxed circular DNA with a gap in the plus strand (Wang et al., 2021). This replication strategy involves the hijacking of host cellular mechanisms, with host proteins serving as both restriction factors that can impede viral replication and essential cofactors that facilitate various steps in the viral lifecycle (Kumar et al., 2024). The reverse transcription process occurs entirely within the cytoplasmic nucleocapsid core, where the compact viral genome maximizes its coding capacity through overlapping reading frames, allowing efficient production of all necessary viral proteins from the limited 3.2 kilobase genome while maintaining the ability to establish chronic infection through cccDNA persistence in the nucleus.

Hepatitis C virus replication occurs exclusively in the cytoplasm within specialized membrane structures called replication organelles, formed through extensive rearrangement of endoplasmic reticulum membranes by non-structural proteins (Pan *et al.,* 2021). The replication process begins after polyprotein processing releases non-structural proteins that form a replication complex containing NS3 protease/helicase, NS4A cofactor, NS4B membrane protein, NS5A regulatory protein, and NS5B RNA-dependent RNA polymerase, which coordinately synthesize viral RNA through a minus-strand intermediate (Bartenschlager *et al.,* 2017). This replication machinery first uses the positive-strand genomic RNA as a template to produce complementary minus-strand RNA, which then serves as a template for generating multiple copies of new positive-strand genomic RNA, with the entire process tightly regulated by viral and host factors to balance RNA synthesis with virion assembly requirements(Tsai et al., 2017). The high error rate of the NS5B polymerase, lacking proofreading activity, generates extensive genetic diversity within infected individuals, creating quasispecies populations that contribute to viral persistence

and immune evasion while presenting challenges for therapeutic intervention and vaccine development (Chen *et al.,* 2024).

2.7 Mode of Transmission

Hepatitis B and C viruses spread through contact with infected blood and body fluids, with different transmission patterns affecting millions of people worldwide. Hepatitis C virus infection affects over 58 million individuals and is responsible for 290,000 annual deaths, while hepatitis B remains highly infectious and can be transmitted through various routes (WHO 2021). Understanding how these viruses spread helps prevent new infections and protect vulnerable populations, especially in Nigeria where hepatitis is common (Nigerian Federal Ministry of Health, 2021).

Routes of Hepatitis B Transmission

1. Blood Contact: HBV is highly infectious and can be transmitted by percutaneous, mucosal, or nonintact skin exposure to infectious blood, semen, and other body fluids. This includes sharing needles, razors, or toothbrushes with infected people (CDC, 2024).
2. Sexual Contact: The virus spreads through unprotected sex with infected partners, as hepatitis B is present in semen and vaginal fluids. Multiple sexual partners significantly increase infection risk (WHO, 2024).
3. Mother to Baby: Perinatal transmission from HBsAg-positive mothers is a major mode of infection in Nigeria when preventive measures are not implemented.
4. Medical Procedures: Body piercing, tattooing, acupuncture, and even nail salons are potential routes of infection unless sterile needles and equipment are used. Poor sterilization in healthcare settings also contributes to transmission (WHO, 2024).

5. Household Items and Traditional Practices: Sharing razors, toothbrushes, or nail clippers with infected family members, as well as cultural practices such as tribal marks, circumcision, and scarification using shared instruments, can also spread hepatitis.

Routes of Hepatitis C Transmission

1. Injection Drug Use: Sharing needles, syringes, or other drug equipment is the most common way hepatitis C spreads in developed countries, and it plays an increasingly important role worldwide (Polilli *et al., 2*024).
2. Medical Procedures: In developing countries, including Nigeria, iatrogenic transmission due to unsterilized medical equipment remains a concern, similar to patterns seen historically in Cameroon, Central African Republic, and Egypt.
3. Blood Transfusion: HCV is efficiently transmitted through transfusion of infected blood or transplantation of infected organs, though screening has reduced this risk in major Nigerian cities.
4. Mother to Child: HCV can be passed from an infected mother to her baby, although this occurs less frequently than hepatitis B transmission.
5. Sexual Contact: Sexual practices involving exposure to blood (e.g., multiple sexual partners, men who have sex with men) can spread hepatitis C, though this is relatively rare .
6. Household Items: Sharing razors, toothbrushes, or nail clippers with infected family members can transmit the virus through small amounts of blood (Okwu *et al.*, 2020).

2.8 Diabetes Mellitus

Diabetes mellitus is a complex, chronic metabolic disorder characterized by persistent hyperglycemia, or elevated blood glucose levels, resulting from defects in insulin secretion,

insulin action, or both (Yameny, 2024). Insulin, a hormone produced by the pancreas, plays a crucial role in regulating blood glucose by facilitating its uptake into cells.When the body either does not produce enough insulin or cannot effectively use the insulin it produces, glucose accumulates in the bloodstream, leading to various short-term and long-term complications .The global prevalence of diabetes has risen dramatically in recent decades, with an estimated 537 million adults aged 20-79 living with the condition worldwide in 2021, and projections indicating this number could reach 783 million by 2045 if current trends persist (IDF,Yameny, 2024).This escalating burden is largely attributed to factors such as population growth, aging, urbanization, increasing rates of obesity, and physical inactivity, with a disproportionately high prevalence in low- and middle-income countries (WHO, 2024).There are several main types of diabetes. Type 1 diabetes mellitus (T1DM), an autoimmune condition, accounts for 5% to 10% of all cases and involves the body's immune system mistakenly attacking and destroying the insulin-producing beta cells in the pancreas, leading to an absolute insulin deficiency (StatPearls,2023). This type is typically diagnosed in childhood or adolescence, though it can occur at any age. Type 2 diabetes mellitus (T2DM) is the most common form, accounting for approximately 90% of cases. It is characterized by insulin resistance, where the body's cells do not respond effectively to insulin, and over time, the pancreas may also lose its ability to produce sufficient insulin. T2DM is strongly associated with aging, obesity, physical inactivity, and unhealthy lifestyle factors (StatPearls, 2023). Gestational diabetes mellitus (GDM) is another form that develops during pregnancy, characterized by hyperglycemia with blood glucose values above normal but below those diagnostic of overt diabetes (WHO, 2024). Other specific types of diabetes can be caused by genetic defects, diseases of the exocrine pancreas, or certain drug-induced conditions .Common symptoms of diabetes include increased thirst (polydipsia), frequent

urination (polyuria), extreme hunger (polyphagia), unexplained weight loss, and blurred vision (WHO, 2024). If left untreated or poorly managed, chronic hyperglycemia can lead to severe and life-threatening complications affecting various organ systems. These complications are broadly categorized into microvascular (damage to small blood vessels) and macrovascular (damage to large blood vessels). Microvascular complications include diabetic occulopathy, which can cause vision loss; diabetic nephropathy, leading to kidney failure; and diabetic neuropathy, affecting nerve function and potentially leading to foot ulcers and amputations (Yameny, 2024). Macrovascular complications significantly increase the risk of cardiovascular diseases such as heart attack and stroke (WHO, 2024). Diabetes also increases the risk of cognitive decline and certain cancers (Wang *et al.,* 2023).

2.9 Hepatitis B and C Co-infection in Diabetic Patients

Hepatitis B (HBV) and Hepatitis C (HCV) co-infection in diabetic patients represents a significant clinical challenge due to the complex and often synergistic interactions between these conditions, leading to exacerbated disease progression and poorer outcomes(Supram *et al.,* 2015). Recent studies have highlighted varying prevalences of these co-infections in diabetic populations, emphasizing the need for targeted screening and integrated management. For instance, a recent study from Golestan Province in northeastern Iran found HBsAg (a marker for HBV infection) positivity in 2.52% of diabetic individuals and anti-HCV antibody positivity in 0.53%. The mechanisms of association between viral hepatitis and diabetes are complex and bidirectional. Chronic viral hepatitis, particularly HCV, can directly contribute to the development and progression of diabetes. HCV core protein and NS5A are known to interfere with insulin signaling pathways within hepatocytes, leading to both peripheral and hepatic insulin resistance (Abdel-Malek *et al.,* 2022). This interference involves impaired insulin receptor

substrate (IRS) phosphorylation, activation of suppressor of cytokine signaling (SOCS) proteins, and the induction of endoplasmic reticulum stress, all of which disrupt glucose metabolism. Chronic HBV infection, though less consistently linked than HCV, can also impact glucose metabolism, with its X protein (HBx) being implicated in affecting gluconeogenesis and potentially leading to hyperglycemia (MDPI, 2024; Chen *et al.,* 2023). Both HBV and HCV infections trigger chronic liver inflammation and oxidative stress, leading to the release of pro-inflammatory cytokines such as TNF-α and IL-6. These mediators are well-known to induce insulin resistance and beta-cell dysfunction, thereby contributing to diabetes development. Furthermore, HCV, particularly genotype 3, can induce hepatic steatosis, and fatty liver disease itself is a strong driver of insulin resistance and T2DM (Poustchi *et al.,* 2021).

Conversely, diabetes can exacerbate the course of viral hepatitis. Diabetes-associated immune dysfunction, characterized by impaired T-cell function and reduced phagocytic activity, can compromise the host's ability to clear viral infections, potentially contributing to chronic persistence or increased susceptibility to infection (Oh *et al.,* 2024;).The metabolic environment of diabetes, including hyperglycemia, dyslipidemia, and increased oxidative stress, can accelerate the progression of liver fibrosis and inflammation in patients with chronic hepatitis, leading to more rapid advancement to cirrhosis and hepatocellular carcinoma (Luo *et al.*, 2023). There is also evidence suggesting shared genetic predispositions and common lifestyle and socioeconomic factors, such as unhealthy diets, sedentary lifestyles, obesity, and shared healthcare exposures, which can concurrently increase the risk of both conditions (ResearchGate, 2025). For instance, frequent medical procedures and blood glucose monitoring, common in diabetic care, have been linked to HBV and HCV outbreaks in healthcare settings, highlighting the risk of iatrogenic transmission (MDPI, 2024; Hussein *et al.,* 2025). This bidirectional

relationship underscores the importance of a holistic approach to patient care, recognizing that each condition can profoundly influence the other's onset, progression, and management(Wang *et al., 2*023).

2.10 Risk Factors

- Healthcare Workers: Nigerian doctors, nurses, and lab workers face higher infection risks due to needle stick injuries and poor safety equipment in many facilities.
- Diabetic Patients: Studies on diabetic patients attending a tertiary health-care facility South-west Nigeria show increased hepatitis risks due to frequent medical procedures and blood testing.
- Blood Recipients: People who received blood transfusions before proper screening became common in Nigeria are at higher risk for both hepatitis B and C.
- Traditional Medicine Users: Patients visiting traditional healers who use unsterilized cutting tools for scarification or bloodletting procedures.

Social Risk Factors

- Poverty: Poor Nigerians cannot afford safe medical care and may visit unqualified practitioners who reuse needles and equipment.
- Low Education: Lack of knowledge about hepatitis transmission leads people to engage in risky behaviors like sharing personal items.
- Cultural Practices: Traditional ceremonies involving blood contact, communal shaving, and group circumcision without sterile tools increase transmission risks.

- Urban Crowding: Overcrowded living conditions in Nigerian cities increase chances of household transmission through sharing personal items.

2.11 Pathogenesis of Hepatitis B and C Co-infection in Diabetes Mellitus

The pathogenesis of HBV infection in diabetic patients is significantly influenced by the altered immune landscape inherent to diabetes. Diabetes, particularly chronic hyperglycemia, fosters a state of low-grade chronic inflammation and impaired immune function, affecting both innate and adaptive immune cells. For example, diabetic individuals often exhibit T-cell exhaustion and altered cytokine profiles, including reduced interferon-gamma production, which collectively compromise the body's ability to mount an effective antiviral response against HBV (Oh *et al.,* 2024; Wang *et al.*, 2023). This weakened immune surveillance can lead to higher rates of chronic HBV infection, as diabetic patients may be less likely to spontaneously clear acute HBV infection, thereby increasing the propensity for chronicity (Chen *et al.,* 2023). Furthermore, for diabetic patients with resolved HBV infection who possess anti-HBs antibodies but carry latent viral DNA, factors such as long-term immunosuppressive therapy (e.g., for diabetic nephropathy or autoimmune comorbidities) or even the metabolic stress of diabetes itself can trigger HBV reactivation, resulting in viral replication and potentially severe liver inflammation (He *et al.,* 2022). The underlying immune dysfunction in diabetes might also theoretically influence the long-term effectiveness of antiviral therapies for HBV, though this effect is more pronounced with interferon-based regimens, which are now less commonly used for HBV. In diabetic patients, chronic HBV infection may also exhibit higher viral loads and potentially faster progression of liver fibrosis due to the additive effects of viral-induced inflammation and metabolic stress on the liver (Luo *et al.,* 2023). The sustained high glucose environment can further promote HBV replication and persistence (Zhang *et al.,* 2024).

HCV pathogenesis in diabetic patients is particularly marked by a strong and direct exacerbation of insulin resistance and accelerated hepatic steatosis, significantly contributing to faster liver disease progression. HCV infection is a well-established cause of insulin resistance, independently of obesity or liver cirrhosis. In diabetic patients, HCV profoundly worsens existing insulin resistance through multiple mechanisms (Abdel-Malek *et al.*, 2022). Direct viral proteins such as HCV core protein, NS5A, and NS5B interfere with key steps in the insulin signaling pathway, specifically by inhibiting insulin receptor substrate-1 (IRS-1) phosphorylation and promoting its degradation. Moreover, HCV replication induces oxidative stress and endoplasmic reticulum (ER) stress within hepatocytes. Both of these stress pathways activate inflammatory kinases (e.g., JNK, IKKβ) that phosphorylate IRS-1 at serine residues, leading to its functional inhibition and promoting insulin resistance (Poustchi *et al.,* 2021). HCV infection also stimulates the release of pro-inflammatory cytokines like TNF-α and IL-6, which are potent inhibitors of insulin signaling. Additionally, HCV may affect adipose tissue function, promoting lipolysis and the release of free fatty acids, further contributing to insulin resistance in the liver and muscle. HCV infection, especially genotype 3, directly induces hepatic steatosis by interfering with lipid metabolism. In diabetic patients, pre-existing metabolic dysregulation, such as dyslipidemia or obesity-induced fatty liver, synergizes with HCV-induced steatosis, leading to more severe and rapidly progressing fatty liver disease (Chekina *et al.,* 2023). Hepatic steatosis itself is a strong driver of insulin resistance and can accelerate liver fibrosis progression, creating a vicious cycle in co-infected individuals.

2.12 Immune response to Hepatis B and C Co-infection

The immune system's response to hepatitis B and C viral infections becomes significantly compromised in individuals with diabetes mellitus, creating a complex interplay between metabolic dysfunction and viral persistence that substantially impacts clinical outcomes. Diabetes mellitus fundamentally alters the host immune landscape through chronic hyperglycemia-induced inflammatory states, insulin resistance, and impaired cellular immune functions, which collectively compromise the body's ability to mount effective antiviral responses against hepatitis B and C infections (Li *et al.,* 2020). The bidirectional relationship between diabetes and viral hepatitis involves direct viral effects on pancreatic beta cells, systemic inflammatory cascades mediated by proinflammatory cytokines, and immune-mediated mechanisms that contribute to both insulin resistance development and impaired viral clearance (Huang *et al.*, 2021). Recent studies demonstrate that diabetic patients exhibit significantly reduced hepatitis B vaccine response rates, with only 50-70% achieving protective antibody levels compared to 90-95% in healthy individuals, indicating fundamental defects in adaptive immune responses (Ahmed *et al.,* 2019).

The cellular immune dysfunction observed in diabetic patients with hepatitis coinfection manifests through multiple pathways including altered T-cell responses, impaired natural killer cell function, and dysregulated cytokine production patterns that favor viral persistence and chronic inflammation(ADA, 2017). Chronic hepatitis B infection in diabetic individuals demonstrates significantly suppressed host immune responses characterized by increased CD4+ CD25high regulatory T cells (Tregs), elevated interleukin-10 production, and decreased interferon-gamma secretion, creating an immunosuppressive environment that facilitates viral

replication and reduces treatment efficacy (Wang *et al.,* 2022). Similarly, hepatitis C coinfection in diabetic patients results in enhanced viral quasispecies diversity due to impaired immune surveillance, with studies showing that the processes through which chronic hepatitis C infection associates with type 2 diabetes involve direct viral effects, insulin resistance, proinflammatory cytokines, chemokines, and other immune-mediated mechanisms that create a self-perpetuating cycle of metabolic and immunological dysfunction (Zhang *et al.,* 2018). The immunometabolic alterations in type 2 diabetes mellitus, as revealed by single-cell RNA sequencing studies, demonstrate profound changes in immune cell populations including altered monocyte phenotypes, impaired dendritic cell maturation, and modified B-cell responses that collectively compromise antiviral immunity and increase susceptibility to chronic viral infections (Chen and Liu, 2024).

2.13 Epidemiology

The co-infection of viral hepatitis (Hepatitis B Virus - HBV and Hepatitis C Virus - HCV) with diabetes mellitus represents a substantial global health burden, characterized by distinct prevalence patterns and evolving temporal trends. Globally, an estimated 254 to 296 million people live with chronic HBV infection, while approximately 537 million people worldwide have diabetes, with a global prevalence of around 10.5% (Oh *et al.,* 2024; WHO, 2022; Frontiers, 2023). HCV infection and T2DM show a strong association, with meta-analyses indicating a 1.7-fold increased risk of diabetes in HCV-infected individuals, and conversely, T2DM patients facing an approximately 3-fold increased risk of acquiring HCV infection (Frontiers, 2023). Local studies highlight this variability, such as an HCV seroprevalence of 17.6% in diabetic patients versus 5.5% in non-diabetics in a sub-Saharan African population, and HBV/HCV seroprevalence among diabetic patients in Northern Tanzania reported at 2.1% and 0.5% respectively (Frontiers,

2023; Eliah *et al.,* 2025). The overall prevalence of HBsAg among diabetic patients in Ethiopia was 7.4% (Frontiers, 2025). Diabetic patients undergoing dialysis also show a notably higher prevalence of both HBV and HCV, with HCV prevalence in hemodialysis patients globally ranging from 1% to 90%, including a high reported rate of 45.13% in Egypt (Deng *et al.,* 2023; International Journal of tropical disease and Health, 2025).

The association between viral hepatitis, particularly HCV, is overwhelmingly stronger with Type 2 Diabetes Mellitus (T2DM), which constitutes about 95% of all diabetes cases, reflecting the direct metabolic interplay (Poustchi *et al.*, 2021;Popescu *et al.,*2024). The epidemiology is dynamic, influenced by global hepatitis elimination efforts and shifts in diabetes prevalence. Significant geographic variations exist; for instance, anti-HCV seroprevalence among diabetics in Taiwan ranged from 1.01% to 12.77% across different townships. The relationship is demonstrably bidirectional: HCV can directly induce insulin resistance, contributing to T2DM development (Abdel-Malek *et al.,* 2022), with an approximately 1.5-fold excess risk of new-onset T2DM in HCV-infected individuals (Eliah *et al.,* 2025). HBV also influences glucose metabolism through chronic liver inflammation (Chen *et al.,* 2023). Conversely, diabetes can exacerbate chronic hepatitis, leading to accelerated liver fibrosis, higher rates of cirrhosis, increased HCC risk (Lu*o et al.,* 2023), and elevated risk of HBV reactivation in resolved cases (He *et al.,* 2022).

National Epidemiology

Nigeria faces a particularly severe dual epidemic of diabetes mellitus and viral hepatitis, with epidemiological data revealing substantial disease burdens that intersect to create complex public health challenges requiring targeted intervention strategies. The Nigerian diabetic population, estimated at 11.2 million individuals representing 5.8% of the adult population, demonstrates disproportionately high prevalence of viral hepatitis coinfection compared to non-diabetic

cohorts (Nigerian Diabetes Association, 2023). National studies indicate that hepatitis B surface antigen positivity among diabetic Nigerians ranges from 18.4% to 24.7% across different regions, substantially exceeding the general population prevalence of 12.2%, with northern states showing particularly elevated coinfection rates attributed to limited healthcare infrastructure and cultural practices influencing transmission patterns (Adelani *et al.,* 2021).

2.14 Clinical Manifestations

The co-existence of viral hepatitis and diabetes mellitus often leads to altered clinical presentations, diagnostic challenges, and accelerated disease progression due to their complex interplay.Hepatitis symptoms in diabetic patients can present acutely or chronically, often with subtle or atypical features. Acute hepatitis, characterized by jaundice, fatigue, nausea, and abdominal pain, might be masked or complicated by diabetic symptoms. In chronic hepatitis, common manifestations like fatigue and non-specific malaise can be easily attributed to poorly controlled diabetes, leading to delayed diagnosis. Furthermore, both HBV and HCV can cause various extrahepatic manifestations, such as skin rashes, arthralgia, or cryoglobulinemia (for HCV), which may be more pronounced or altered in the context of a dysregulated diabetic immune system (Abdel-Malek *et al.,* 2022).Conversely, diabetes symptoms in hepatitis patients can range from the insidious development of new-onset diabetes to acute diabetic decompensation. Chronic HCV infection is a well-recognized cause of new-onset Type 2 Diabetes Mellitus (T2DM), where patients may initially present with typical diabetic symptoms like polyuria, polydipsia, and unexplained weight loss, often coinciding with or after their hepatitis diagnosis (Poustchi *et al.,*Popescu *et al.*,2024). In patients with pre-existing diabetes, hepatitis infection, particularly with liver inflammation or advanced liver disease, can worsen glycemic control, leading to frequent episodes of hyperglycemia or, paradoxically, increased risk

of hypoglycemia due to impaired hepatic glucose regulation, requiring significant adjustments in diabetes management(Poustchi *et al.*, 2021).

Co-infection patterns frequently involve overlapping symptoms and atypical presentations, posing significant diagnostic challenges for clinicians. The non-specific nature of many chronic hepatitis symptoms can be mistakenly attributed to diabetes complications, delaying crucial hepatitis diagnosis and antiviral treatment. This diagnostic ambiguity necessitates a high index of suspicion and routine screening for viral hepatitis in diabetic populations (Chen *et al., 2*023).

The most concerning aspect of co-infection is the acceleration of complications, leading to faster disease progression and increased morbidity. Diabetic patients with chronic viral hepatitis experience accelerated liver fibrosis, leading more rapidly to cirrhosis and its associated complications, such as ascites and hepatic encephalopathy (Luo *et al.,* 2023). The risk of hepatocellular carcinoma (HCC) is also significantly amplified in these co-infected individuals compared to those with either condition alone, highlighting the synergistic adverse effects on liver health (Luo *et al.,* 2023). Moreover, metabolic derangements induced by hepatitis can worsen pre-existing diabetic complications like cardiovascular disease and nephropathy, further increasing overall morbidity and mortality (Popescu *et al*., 2024).

2.15 Laborartory Diagnosis

Laboratory diagnosis serves as the cornerstone for investigating hepatitis B and C infections, providing essential information that enables accurate disease identification, staging, and management decisions in clinical practice. Laboratory testing plays a crucial role in distinguishing between acute and chronic infections, determining viral activity levels, assessing

immune status, and monitoring treatment responses throughout the course of disease management (Terrault *et al.,* 2018).

Serological Testing of Hepatitis B

Hepatitis B serological testing involves detection of viral antigens and host antibodies that appear at different stages of infection, providing comprehensive assessment of infection status and immune responses. The primary serological marker, hepatitis B surface antigen (HBsAg), indicates active infection and appears earliest during the acute phase, with persistence beyond six months confirming chronic infection status (Sarin *et al., 2*016). Hepatitis B surface antibody (anti-HBs) detection demonstrates protective immunity either from vaccination or resolved natural infection, serving as a crucial marker for assessing vaccination effectiveness and determining need for booster doses. The hepatitis B core antibody total (anti-HBc total) provides evidence of current or previous infection exposure, while the IgM component (anti-HBc IgM) specifically indicates acute or recent infection phases(Rheinlände *et al.,* 2018). Additional markers include hepatitis B e antigen (HBeAg), which reflects high viral replication and infectivity levels, and its corresponding antibody (anti-HBe), suggesting seroconversion and reduced viral activity, together providing comprehensive assessment of disease phase and transmission risk.

Molecular Testing of Hepatitis B

Molecular testing for hepatitis B directly detects and quantifies viral DNA using polymerase chain reaction amplification, providing precise measurements of viral load that correlate with disease activity and treatment response. Quantitative HBV DNA testing serves as the gold standard for assessing viral replication levels, with values above 2,000 IU/mL in HBeAg-positive patients or 200 IU/mL in HBeAg-negative patients indicating significant viral activity requiring

treatment consideration (Terrault *et al.,* 2018). Genotyping analysis identifies specific viral strains (genotypes A through J) that influence treatment response patterns, disease progression rates, and geographical distribution, with genotypes A and B generally showing better response to interferon therapy compared to genotypes C and D(Ghany *et al.,* 2020). Drug resistance mutation testing becomes essential when patients experience treatment failure, identifying specific genetic changes that confer resistance to nucleos(t)ide analogs such as lamivudine, adefovir, or entecavir, enabling optimization of therapeutic regimens. Advanced molecular techniques include quantitative hepatitis B surface antigen measurement and hepatitis B core-related antigen detection, serving as alternative biomarkers for monitoring treatment response and predicting functional cure achievement(Leowattana *et al,* 2024).

Serological Testing of Hepatitis C

Hepatitis C serological testing primarily focuses on antibody detection as the initial screening approach, though antibodies indicate exposure rather than active infection status. The anti-hepatitis C virus antibody test (anti-HCV) using third-generation enzyme immunoassays demonstrates high sensitivity and specificity for detecting exposure to hepatitis C virus, though positive results require confirmatory testing since antibodies persist following viral clearance (Ghany *et al.,* 2020). Signal-to-cutoff ratios from antibody testing provide additional interpretive value, with higher ratios increasing the likelihood of true positive results and active infection. Hepatitis C core antigen testing represents an alternative serological approach that may correlate better with viral replication compared to antibody detection, particularly useful in immunocompromised patients who may have delayed or absent antibody responses. The major limitation of hepatitis C serological testing lies in its inability to distinguish between active and

resolved infections, necessitating molecular confirmation for definitive diagnosis and treatment decisions.

Molecular Testing of Hepatitis C

Molecular testing for hepatitis C involves detection and quantification of viral RNA using polymerase chain reaction techniques, providing definitive confirmation of active infection and essential information for treatment planning(Vogel *et al.,* 2018). Qualitative HCV RNA testing confirms the presence of replicating virus, distinguishing active infection from resolved disease in antibody-positive individuals, with detectable RNA indicating need for clinical evaluation and potential treatment intervention (AASLD-IDSA, 2019)(Wilkins *et al.,* 2024). Quantitative HCV RNA measurement provides baseline viral load assessment and enables monitoring of treatment response, though baseline levels do not predict treatment outcomes in the direct-acting antiviral era(Prince *et al.,* 2021).Genotyping and subtyping analysis identifies specific viral strains (genotypes 1-7 with multiple subtypes) that directly influence treatment regimen selection, duration, and monitoring requirements, with genotype 1 historically requiring longer treatment courses though modern therapies show high efficacy across all genotypes(Ghany *et al.,* 2020). . Resistance-associated substitution testing may be performed in patients with prior treatment failure or specific genotype patterns, identifying mutations that could impact direct-acting antiviral effectiveness and guiding selection of optimal therapeutic combinations.

2.16 Prevention and Control of Hepatitis B and C

Prevention and control of hepatitis B and C infections in Nigeria require targeted strategies addressing the high disease burden and specific transmission patterns in the country. Nigeria has one of the world's highest hepatitis B prevalence rates at 12.2% and faces significant challenges

in implementing effective prevention programs due to limited healthcare infrastructure and resources (WHO, 2024).

(A) Prevention of Hepatitis B

1. Universal Vaccination Programs: Nigeria implemented the hepatitis B vaccine into its routine immunization schedule in 2004, with WHO recommending birth dose vaccination within 24 hours for all newborns (Hepatitis B Foundation, 2024). However, vaccination coverage remains suboptimal at 65% nationally, with rural areas showing lower rates due to healthcare access barriers and vaccine supply chain challenges.

2. Healthcare Worker Protection: Nigerian healthcare workers have hepatitis B infection rates of 8.9-15.2%, necessitating mandatory vaccination programs and improved occupational safety measures (Spearman *et al., 2*017). Many healthcare facilities lack adequate vaccination protocols and post-exposure prophylaxis, contributing to ongoing occupational transmission.

3. Prevention of Mother-to-Child Transmission: In Nigeria, 85% of pregnant women with hepatitis B transmit the virus to their babies without intervention (Nigerian Federal Ministry of Health, 2019). Implementing routine antenatal screening and birth dose vaccination could prevent most vertical transmissions, but current programs reach less than 30% of pregnant women.

(B) Prevention of Hepatitis C

1. Harm Reduction for High-Risk Groups: Nigeria's hepatitis C prevention focuses on reducing transmission among people who inject drugs and recipients of unsafe medical procedures,

particularly in northern regions where prevalence is highest (Forbi e*t al., 20*18). Limited harm reduction programs exist, with most prevention efforts targeting healthcare settings rather than community-based interventions.

2. Healthcare Infection Control: Unsafe injection practices and poor sterilization contribute significantly to hepatitis C transmission in Nigerian healthcare facilities (CDC, 2024). Strengthening infection prevention protocols, ensuring single-use medical devices, and training healthcare workers on safe practices are critical for reducing healthcare-associated transmission.

3. Screening and Treatment Programs: Nigeria's National Strategic Plan aims to screen 30% of the population for hepatitis C by 2030, though current screening rates remain below 10% (Nigerian Federal Ministry of Health, 2020). Expanding access to direct-acting antivirals and implementing treatment as prevention strategies could reduce community transmission, but high drug costs and limited healthcare infrastructure remain major barriers.

(C) Infection control in Diabetic patients

1. Vaccination Programs: All diabetic patients should receive hepatitis B vaccine with booster doses when antibody levels drop below protective levels (WHO, 2024)

2. Pre-exposure Prophylaxis: High-risk diabetic patients may require additional protective measures before medical procedures (CDC, 2020)

3. Immune Status Monitoring: Regular testing of hepatitis B antibody levels to ensure continued protection (ADA, 2023)

4. Risk Assessment: Identify and minimize exposure risks through patient history and lifestyle evaluation (ADA, 2023)

CHAPTER THREE

MATERIALS AND METHODS

3.1 Study Design

The study design for this research was cross sectional to determine the prevalence of Hepatitis B and C coinfection among diabetic patients in University of Benin Teaching Hospital, Benin City Edo state.The study included blood collection and baseline data collection.

3.2 Study Area

The study was conducted at the University of Benin Teaching Hospital (UBTH), Benin City, Edo State, Nigeria.University of Benin Teaching Hospital has an elevation of about 90 metres (295 feet above sea level),it coordinates 6.3886° N, 5.6001° E .UBTH serves as a tertiary healthcare facility providing comprehensive medical services to a diverse patient population across Edo State and neighboring regions in South-South Nigeria.

3.3 Study Population

The study population comprised adult patients aged 18-75 years diagnosed with diabetes mellitus (Type 1 or Type 2) attending the endocrinology clinic at UBTH. Patients included those with newly diagnosed diabetes (within six months of diagnosis) and those with established diabetes of varying duration.

3.4 Inclusion Criteria

Participants meeting the following criteria were included in the study:

- Patients with confirmed diagnosis of diabetes mellitus (Type 1 or Type 2)
- Adults aged 18 years and above

- Willingness to give informed consent.

3.5 Exclusion Criteria

Participants excluded from the study were:

- non diabetic Patients
- diabetic patients previously diagnosed with hepatitis B or C virus
- Indidviduals unable to provide consent
- Pregnant women

3.6 Sample Size Determination

Sample size calculation was performed using the formula for cross-sectional studies investigating prevalence estimates (Lwanga and Lemeshow, 1991):

$n= Z^2p(1-p)/d^2$

Where:

• (n) = minimum required sample size

Z = Z score for 95% confidence level (1.96)

• p = expected prevalence of hepatitis B and C co-infection in diabetic patients (14% Ndako *et al.,*2020)

• d = precision or margin of error (5% or 0.05)

Calculating: $n = (1.96)^2 \times 0.14 \times 0.86 / (0.05)^2 = 159.12$

n~200

3.7 Ethical Considerations

Ethical approval for this research was obtained from the Health Research Committee of the University of Benin Teaching Hospital, Benin City with Approval Number(ADME/22/A VOL.VII/1486549125533).This study also adhered to ethical principles outlined in the Declaration of Helsinki. Informed consent was gotten from all participants, ensuring they understood the study's purpose, proceDure, potential risks and benefits. A strustured questionnaire was administered to collect bio-data and medical diagnosis of the study participants. The participants confidentiality was maintained with data anonymized and securely stored.

3.8 Specimen Collection and Processing

About 2ml venous Blood samples were collected from each participants with the aid of sterile needles and syringes in accordance with ethical and safety standard. The samples were then dispensed into sterile plain containers and allowed to clot. The samples were centrifuge at 3,000rpm for 15 minutes .The serum obtained were stored at -20C before the saples were analyzed.

Procedure:

1) Participant recuitment and consent
2) Administartion of questionnaire
3) Collection of 2ml of venous blood samples from the forearm of diabetic patients into a sterile plain container
4) HBsAg and HCV kits were used for the serological testing

Sample Processing.

Hepatitis B surface antigen (HBsAg) and Hepatitis C virus (HCV) were detected using commercially available rapid chromatographic immunoassay for qualitative detection. The HBsAg and HCV kits used was (TELL) rapid kits having sensitivity, specificity and accuracy of >99.5%,97% and 98.5% respectively. The qualitative assays were performed using one step strips for detection HBsAg and HCV. The immunochromatographic reaction was allowed to take place within a few minutes a nd the result read at eaxctly 15 minutes after.Only clear non haemolyzed serum samples were used. The HBsAg and HCV has maufactured diagnostic specificity,sensitivity, and accuracy of >99.5%,97%,and 98.5% respectively.

3.8.1 Interpretation

The results of the test were reported as positive and negative respectively.The presence of double lines i.e the control line and the test line indicates a positive result and is reported as positive.The presence of one line on the test kit i.e only the control line present indicates a negative result and is reported negative.

3.9 Statistical Analysis

Data analysis was carried out using SPSS version 27. Descriptive measures such as mean, standard deviation, frequency, and percentage were applied to summarize participants' demographic and clinical characteristics. Results were illustrated in tables and figures. Associations between HBV infection and selected demographic or clinical variables were assessed using the Chi-square test, while multivariate logistic regression was performed to determine independent predictors of HBV infection. A probability value of less than 0.05 was

considered statistically significant, whereas values greater than 0.05 were regarded as not significant.

CHAPTER FOUR

RESULTS

Table 4.1: Demographic Characteristics of Diabetic Patients attending UBTH

Table 4.1 shows that the largest age group comprised participants aged 46–55 years (35.0%), followed by those aged 56–65 years (23.5%) and 66–75 years (26.5%). Participants aged 36–45 years accounted for 13.5%, while the smallest group was those aged 26–35 years (1.5%). The mean age of the participants was 57.57 ± 12.16 years. With respect to gender, females constituted the majority (66.5%), while males represented 33.5%. In terms of marital status, the majority of participants were married (87.0%), while 8.5% were widowed, 3.0% single, and 1.5% divorced. Regarding educational status, more than half of the participants had tertiary education (54.5%), followed by those with secondary education (27.0%) and primary education (14.5%), while a small proportion (4.0%) had no formal education.

Table 4.1: Demographic Characteristics of Diabetic Patients attending UBTH

Variables	Frequency	Percent
Age Range		
26–35 Years	3	1.5
36–45 Years	27	13.5
46–55 Years	70	35.0
56–65 Years	47	23.5
66–75 Years	53	26.5
Total	200	100.0
Mean Age ± SD	57.57 ± 12.16	
Gender		
Female	133	66.5
Male	67	33.5
Total	200	100.0
Marital Status		
Single	6	3.0
Married	174	87.0
Divorced	3	1.5
Widowed	17	8.5
Total	200	100.0
Educational Status		
No formal education	8	4.0
Primary	29	14.5
Secondary	54	27.0
Tertiary	109	54.5
Total	200	100.0

Table 4.2: Prevalence of HBV and HCV among Diabetic Patients attending UBTH

Table 4.2 presents the prevalence of hepatitis B surface antigen (HBsAg) among diabetic patients attending UBTH. Out of the 200 participants, 4 (2.0%) tested positive for HBsAg, while the vast majority, 196 (98.0%), were negative. This indicates a relatively low prevalence of hepatitis B infection in the study population.For hepatitis C virus (HCV), none of the participants tested positive, and consequently, there were no cases of HBV and HCV coinfection among the diabetic patients studied.

Table 4.2: Prevalence of HBV and HCV Among Diabetic Patients Attending UBTH

Serology	Frequency	Percentage (%)
HBV		
Positive	04	2.0
Negative	196	98.0
HCV		
Positive	00	0
Negative	200	100.0

Table 4.3 Prevalence of HBV Infection Among Diabetic Patients attending UBTH in Relation to Age

Table 4.3 presents the prevalence of HBV infection among diabetic patients attending UBTH in relation to age. The results show a statistically significant association between age and HBV status ($p = 0.023$). All HBV-positive cases (100%) were recorded among participants aged above 65 years, while no positive cases were found in the younger age groups.

Table 4.3 Distribution of HBV Infection Among Diabetic Patients attending UBTH in Relation to Age

Age (Years)	Sample size	No. of sero positive	% sero positive	p-value	Odds Ratio	95% CI
26–35	3 (1.5)	0	0.0	0.023	–	–
36–45	27 (13.5)	0	0.0			
46–55	70 (35.0)	0	0.0			
56–65	47 (23.5)	0	0.0			
66–75	53 (26.5)	4	100.0			
Total	**200 (100.0)**	**4**				

Table 4.4 Prevalence of HBV Infection Among Diabetic Patients attending UBTH in Relation to Gender

Table 4.4 presents the prevalence of HBV infection among diabetic patients attending UBTH in relation to gender. Gender showed no significant impact on HBV prevalence in diabetic mellitus patients (p=0.716). However, 3 HBV-positive cases (75.0%) were recorded among females and 1 case (25.0%) among males.

Table 4.4 Gender Distribution of HBV Infection Among Diabetic Patients attending UBTH

Gender	Sample size	No. of sero positive	% sero positive	p-value	Odds Ratio	95% CI
Female	133 (66.5)	3	75.0	0.716	0.657	0.067 – 6.435
Male	67 (33.5)	1	25.0			
Total	200 (100.0)	4				

Table 4.5 Distribution of HBV Infection Among Diabetic Patients attending UBTH in Relation to Marital Status

Table 4.5 presents the prevalence of HBV infection among diabetic patients attending UBTH in relation to marital status. Marital status showed no significant impact on HBV prevalence in diabetic mellitus patients (p=0.894). However, all 4 positive cases (100%) occurred among married participants.

Table 4.5 Distribution of HBV Infection Among Diabetic Patients attending UBTH in Relation to Marital Status

Marital Status	Sample size	No. of sero positive	% sero positive	p-value	Odds Ratio	95% CI
Single	6 (3.0)	0	0.0	0.894	–	–
Married	174 (87.0)	4	100			
Divorced	3 (1.5)	0	0.0			
Widowed	17 (8.5)	0	0.0			
Total	**200 (100.0)**	**4**				

Table 4.6: Prevalence of HBV Infection Among Diabetic Patients attending UBTH in Relation to Educational Level

Table 4.6 presents the prevalence of HBV infection among diabetic patients attending UBTH in relation to educational level. Educational level showed no significant impact on HBV prevalence in diabetic mellitus patients (p=0.185). However, 2 positive cases (50.0%) were found among participants with primary education and 2 (50.0%) among those with tertiary education. No positive cases were recorded among participants with secondary education or no formal education.

Table 4.6: Prevalence of HBV Infection Among Diabetic Patients attending UBTH in Relation to Educational Level

Educational Level	Sample size	No. of sero positive	% sero positive	p-value	Odds Ratio	95% CI
No formal education	8 (4.0)	0	0.0	0.185	0.685	0.251 – 1.873
Primary	29 (14.5)	2	50.0			
Secondary	54 (27.0)	0	0.0			
Tertiary	109 (54.5)	2	50.0			
Total	**200 (100.0)**	4				

Table 4.7: Clinical History of Diabetic Patients attending UBTH

Table 4.7 presents the clinical history of diabetic patients attending UBTH. The results show that the majority of participants had been living with diabetes for 1–5 years (37.0%), followed by those with 6–10 years (32.5%) and more than 10 years (25.0%). Only a small proportion (5.5%) had been diagnosed for less than 1 year. With respect to the type of diabetes, most participants reported Type 2 diabetes (65.5%), while 34.5% were unsure of their type. In terms of blood transfusion history, the vast majority (93.5%) had never received a transfusion, while 6.5% reported a history of transfusion. Similarly, most participants (95.5%) had never been previously tested for hepatitis, while only 4.5% had undergone testing.

Concerning risk behaviours, 18.5% of participants reported intravenous drug use, while the majority (81.5%) did not. Regarding sexual practices, 42.5% of participants reported not practicing safe sex, 20.0% reported occasional practice, and 37.5% consistently practiced safe sex. For invasive medical and lifestyle practices, 13.0% of participants reported a history of surgical procedures, while 8.5% admitted to sharing needles. Tattoos and piercings were reported by 7.0% of participants, while the majority (93.0%) did not have any.

Table 4.7: Clinical History of Diabetic Patients attending UBTH

Variables	Frequency	Percent
Duration of Illness		
– <1 Year	11	5.5
– 1–5 Years	74	37.0
– 6–10 Years	65	32.5
– >10 Years	50	25.0
Total	200	100.0
Type of Diabetes		
– Not sure	69	34.5
– Type 2	131	65.5
Total	200	100.0
History of Blood Transfusion		
– No	187	93.5
– Yes	13	6.5
Total	200	100.0
Previously Tested for Hepatitis		

– No	191	95.5
– Yes	9	4.5
Total	200	100.0
Intravenous Drugs		
– No	163	81.5
– Yes	37	18.5
Total	200	100.0
Safe Sex		
– No	85	42.5
– Sometimes	40	20.0
– Yes	75	37.5
Total	200	100.0
Surgical Procedures		
– No	174	87.0
– Yes	26	13.0
Total	200	100.0
Sharing of Needles		

– No	183	91.5
– Yes	17	8.5
Total	200	100.0
Tattoos & Piercings		
– No	186	93.0
– Yes	14	7.0
Total	200	100.0

Table 4.8 Prevalence of HBV Infection Among Diabetic Patients attending UBTH in Relation to Clinical Characteristics

Table 4.8 presents the prevalence of HBV infection among diabetic patients attending UBTH in relation to clinical characteristics. The results show a statistically significant association between duration of illness ($p = 0.001$), intravenous drug use ($p = 0.000$), surgical procedures ($p = 0.000$), sharing of needles ($p = 0.000$), and tattoos/piercings ($p = 0.000$) with HBV infection in diabetic mellitus patients. Half of the HBV-positive cases (50.0%) were observed among participants with illness duration less than one year, while the remaining cases were distributed among those with 6–10 years (25.0%) and more than 10 years (25.0%) of illness. All HBV-positive participants (100%) reported intravenous drug use, needle sharing, and tattoos/piercings, while 75.0% had a history of surgical procedures. Among the HBV-positive participants, 3 (75.0%) had type 2 diabetes and 1 (25.0%) were unsure of their diabetes type. With respect to blood transfusion, 3 cases (75.0%) occurred among those without transfusion history, while 1 case (25.0%) was recorded among those who had received transfusion. Similarly, all HBV-positive cases (100%) were found among participants who had never been tested for hepatitis. Regarding safe sex practices, 3 cases (75.0%) occurred among participants who did not practice safe sex and 1 case (25.0%) among those who practiced it sometimes, while no positive cases were recorded among those who consistently practiced safe sex. However, type of diabetes, history of blood transfusion, prior hepatitis testing, and practice of safe sex showed no statistically significant association with HBV status ($p > 0.05$).

Table 4.8: Prevalence of HBV Infection Among Diabetic Patients attending UBTH in Relation to Clinical Characteristics

Variables	Positive	Negative	X^2	df	p-value
Duration of Illness					
– <1 Year	2 (50.0%)	9 (4.6%)	16.28	3	0.001*
– 1–5 Years	0 (0.0%)	74 (37.8%)			
– 6–10 Years	1 (25.0%)	64 (32.7%)			
– >10 Years	1 (25.0%)	49 (25.0%)			
Type of Diabetes					
– Not sure	1 (25.0%)	68 (34.7%)	0.16	1	0.686
– Type 2	3 (75.0%)	128 (65.3%)			
Blood Transfusion					
– No	3 (75.0%)	184 (93.9%)	2.30	1	0.129
– Yes	1 (25.0%)	12 (6.1%)			
Previously Tested for Hepatitis					
– No	4 (100.0%)	187 (95.4%)	0.19	1	0.661
– Yes	0 (0.0%)	9 (4.6%)			
Intravenous Drugs					
– No	0 (0.0%)	163 (83.2%)	17.98	1	0.000*
– Yes	4 (100.0%)	33 (16.8%)			
Safe Sex					
– No	3 (75.0%)	82 (41.8%)	2.60	2	0.273

– Sometimes	1 (25.0%)	39 (19.9%)			
– Yes	0 (0.0%)	75 (38.3%)			
Surgical Procedures					
– No	1 (25.0%)	173 (88.3%)	13.87	1	0.000*
– Yes	3 (75.0%)	23 (11.7%)			
Sharing of Needles					
– No	0 (0.0%)	183 (93.4%)	43.94	1	0.000*
– Yes	4 (100.0%)	13 (6.6%)			
Tattoos & Piercings					
– No	0 (0.0%)	186 (94.9%)	54.23	1	0.000*
– Yes	4 (100.0%)	10 (5.1%)			

Table 4.9: Risk Factors Associated with HBV Infections among Diabetic Patients attending UBTH

Table 4.9 presents the potential risk factors associated with HBV infection among diabetic patients attending UBTH using multivariate logistic regression. The results show that none of the variables assessed, including age, gender, marital status, educational status, duration of illness, type of diabetes, blood transfusion history, previous hepatitis testing, intravenous drug use, safe sex practices, surgical procedures, sharing of needles, and tattoos/piercings, were significant predictors/risk factors of HBV infection ($p > 0.05$).

The bivariate analysis (Chi-square test) showed that age ($p = 0.23$), duration of illness ($p = 0.001$), intravenous drug use ($p = 0.000$), surgical procedures ($p = 0.000$), sharing of needles ($p = 0.000$), and tattoos/piercings ($p = 0.000$) were significantly associated with HBV infection. However, in multivariate logistic regression, none of these variables remained statistically significant ($p > 0.05$), indicating that the observed associations were not independent predictors after adjusting for potential confounders. If significant in Chi-square but not in Logistic regression, this means that the factor shows an association with HBV on its own, but after adjusting for other risk factors, the association is not independent.

Table 4.9: Risk Factors Associated with HBV Infections among Diabetic Patients attending UBTH

Variables	p-value	OR (Exp(B))	95% C.I. for OR	
			Lower	Upper
Age Range	1.000	1.95	0.000	-
Gender	1.000	2.83×10^{5}	0.000	-
Marital Status	1.000	1.54×10^{3}	0.000	-
Educational Status	0.999	1.70×10^{4}	0.000	-
Duration	0.999	0.04	0.000	-
Type	0.997	1.23×10^{12}	0.000	-
Blood Transfusion	1.000	9.73×10^{4}	0.000	-
Previously Tested for Hepatitis	1.000	6.65×10^{5}	0.000	-
Intravenous Drugs	0.999	8.43×10^{5}	0.000	-
Safe Sex	0.999	1.59×10^{5}	0.000	-
Surgical Procedures	0.999	2.17×10^{6}	0.000	-
Sharing of Needles	0.998	5.48×10^{15}	0.000	-
Tattoos & Piercings	0.999	6.41×10^{15}	0.000	-
Constant	0.992	0.00	-	-

CHAPTER FIVE

DISCUSSION, CONCLUSION AND RECOMMENDATION

5.1 DISCUSSION

This study investigated the prevalence of hepatitis B and C infections among diabetic patients attending University of Benin Teaching Hospital and examined the clinical and demographic factors associated with infection. The overall prevalence of hepatitis B surface antigen (HBsAg) in the study population was 2.0%.While no cases of hepatitis C virus (HCV),there was also no co-infection detected in the study.

This prevalence of HBV in this cohort appears lower than the national average prevalence in Nigeria, which has been estimated between 8.1% and 11% (with an average of 9.5%) (Ajuwon *et al.,* 2021). Also, when comparing with similar studies, the HBV prevalence found here among diabetic patients is notably lower than that reported among diabetic patients in Jigawa (4.7% by Hauwa *et al.,* 2018), Ghana (5.5% by Ephraim *et al.,* 2014), 3.4% and 3.9 % in Congo and in Ethiopia by (Belete *et al.,*2023).One possible explanation for this difference may be the increasing adoption of HBV vaccination, particularly among younger age groups, and heightened awareness of blood safety measures in healthcare facilities. Such differences may stem from variations in study population size, diagnostic methods, or socio-behavioral characteristics. These findings challenge the assumption that diabetes predisposes individuals to HBV infection, suggesting that other factors induce risk more than diabetes itself, given that the prevalence observed in this study was lower than the national average.

No cases of Hepatitis C virus (HCV) infection were detected in this study. The absence of hepatitis C infection in the study population is important to note, particularly given that similar studies in Nigeria and other African countries have reported variable prevalence rates, ranging from 0.5% to 17.6% among diabetic patients (Eliah *et al*., 2025). Hauwa *et al.* (2018) also reported a 1.56% HCV prevalence among type 2 diabetic patients in Jigawa State, while Ndako *et al*.(2019) reported an HCV prevalence of 13.3% in southwestern Nigeria, while an earlier study by Ndako *et al.* (2011) documented a 5.0% prevalence among 180 individuals with diabetes.One possible explanation is that there may be a genuinely low circulation of HCV in this region,thereby reducing the likelihood of detecting the cases within the study sample.

The absence of HBV/HCVco-infection in this study is consistent with earlier reports that have documented no HBV/HCV co-infection prevalence among diabetic populations in sub-Saharan Africa (Eliah *et al*.,2025). The finding may reflect true absence due to low circulation of HCV in this region, and can also be influenced by the sample size. It is also possible that awareness and avoidance of common HCV risk factors are higher in this population.

Analysis of demographic factors revealed that all HBV-positive cases occurred in participants aged 65 years and above. However, contrary findings have been reported by (Onubi *et al.,* 2023), who observed higher HBV prevalence (10.2%) among younger adults in North Central Nigeria. Gender differences in HBV prevalence were not statistically significant, although females accounted for 75% of positive cases. This finding contrasts with the male predominance (80%) reported by (Eliah *et al.,* 2025). In this case, the small number of HBV-positive cases may limit meaningful gender comparisons, yet the pattern raises questions about possible underexplored vulnerabilities among women, such as informal medical practices during childbirth or cosmetic

procedures. Such inconsistencies could reflect generational differences in vaccination coverage and lifestyle practices.

Educational status and marital status also showed no significant association with HBV prevalence, although all positive cases were among married individuals and half had tertiary education. While education is often expected to reduce infection risk through improved health literacy, this was not evident here. Similar cases have been documented in earlier Nigerian studies where higher education did not always translate into safer health-seeking practices (Jama *et al.,* 2025).

Clinical characteristics presented more compelling associations. Participants who had been living with diabetes for less than one year accounted for half of the HBV-positive cases, and duration of illness showed a statistically significant association with HBV infection. This observation appears counterintuitive, as one might expect longer duration of diabetes, with its increased hospital visits and procedures, to have higher risk. Risk behaviors including intravenous drug use, sharing of needles, and having tattoos or piercings were all significantly associated with HBV infection in chi-square analysis, with every HBV-positive participant reporting such practices. This is consistent with the well-documented role of these behaviors in HBV transmission as stated by WHO (2024). Invasive medical procedures, including surgery, also showed strong associations, reflecting the enduring problem of inadequate infection control in some healthcare settings. These findings echo those of Ajayi *et al*.(2021) and Orabueze *et al.* (2024) who highlighted the persistence of unsafe injection and surgical practices as key drivers of HBV in Nigeria.

However, none of these associations remained significant in multivariate logistic regression, suggesting that the relationships observed in chi-square analysis were not independent predictors

once other variables were considered. This points to possible confounding, where the same individuals may be exposed to multiple overlapping risk factors. For instance, someone who injects drugs may also share needles and undergo unregulated tattooing, making it difficult to isolate a single independent predictor. The lack of independent risk factors may also reflect the small number of HBV-positive cases, which reduces statistical power and hides true associations. Nevertheless, this finding aligns with the work of Hussein *et al*.(2025), who reported that after adjusting for confounders, most behavioral and clinical factors lost their statistical significance in predicting HBV infection among high-risk populations.

5.2 CONCLUSION

This study examined the prevalence of hepatitis B (HBV) and hepatitis C (HCV) among diabetic patients at UBTH, as well as the demographic and clinical factors that might be linked to infection. The results showed a low HBV prevalence of 2.0% and no cases of HCV. The absence of HCV also points to possible regional differences in circulation and transmission. While factors such as older age, invasive medical procedures, and certain risk behaviors showed significant associations in the initial analysis, none remained significant after adjusting for confounders. This outcome may be related to overlapping exposures or the small number of positive cases. Overall, the findings indicate that diabetes by itself may not substantially increase the risk of HBV or HCV infection; instead, broader healthcare practices and behavioral patterns may play a more deliberate role.

5.3 RECOMMENDATION

1. Expanded Surveillance: Multi-center studies with larger sample sizes are needed to better capture the true burden of HBV and HCV among diabetic patients in Nigeria.

2. Molecular Characterization: Future research should incorporate polymerase chain reaction (PCR) and genotyping to provide deeper insights into viral dynamics and chronicity.

3. Vaccination Campaigns: Strengthening HBV vaccination programs, especially targeting older adults and high-risk groups, remains critical to reducing prevalence.

4. Improved Infection Control: Healthcare facilities should reinforce safe surgical and injection practices to minimize iatrogenic transmission.

5. Behavioral Interventions: Targeted health education addressing needle sharing, unregulated cosmetic procedures, and other high-risk behaviors should be prioritized among diabetic patients.

6. Longitudinal Studies: Prospective designs are needed to establish causal relationships and evaluate the long-term impact of diabetes on HBV and HCV susceptibility.

5.4 LIMITATION

1. Small number of positive cases: The low prevalence of HBV and absence of HCV reduced statistical power, limiting the ability to detect independent predictors.

2. Single-center study: Conducted at UBTH, the findings may not be generalizable to other populations or regions in Nigeria.

3. Self-reported risk behaviors: Data on practices such as intravenous drug use, needle sharing, and tattoos were based on self-report, which may be subject to recall or social desirability bias.

4. Cross-sectional design: The study design does not allow causal inference between risk factors and HBV infection.

5. Diagnostic limitation: Only serological markers were used, without molecular testing to distinguish between acute, chronic, or resolved infections.

REFERENCES

AASLD-IDSA. (2019). HCV guidance: Recommendations for testing, managing, and treating hepatitis C. American Association for the Study of Liver Diseases and the Infectious Diseases Society of America.

Abdel-Malek, W., Abdel-Rahman, M., and Ahmed, S. (2022). Hepatitis C Virus proteins and their role in insulin resistance and diabetes development. *Frontiers in Endocrinology*, 13, 954321.

Adelani, I. B., Adepoju, O. K., and Adeyemi, A. B. (2021). Prevalence of hepatitis B and C co-infections among diabetic patients in Nigeria*:* A multicenter study. *African Health Sciences,* 21(2), 789–798

Ahmed, M., Khan, S., and Malik, R. (2019). Impaired hepatitis B vaccine response in patients with type 2 diabetes mellitus. *Human Vaccines & Immunotherapeutics,* 15(7–8), 1765–1772.

Ajayi, B. B., Adebolu, T. T., and Oladunmoye, M. K. (2021). Sero-prevalence and associated risk factors of Hepatitis B among adults attending selected government hospitals in Nigeria. *Micromedicine,* 9(1), 27–36

Ajuwon, B. I., Yujuico, I., Roper, K., Richardson, A., Sheel, M., and Lidbury, B. A. (2021). Hepatitis B virus infection in Nigeria: a systematic review and meta-analysis of data published between 2010 and 2019. *BMC infectious diseases,* 21(1), 1120.

Akere, A., Adepeju, A. A., Solagberu, B. A., and Otegbayo, J. A. (2020). Prevalence of hepatitis C virus infection among patients with diabetes mellitus in Southwest Nigeria. Nigerian *Journal of Clinical Practice*, 23(4), 527–532.

American Diabetes Association. (2017). Standards of medical care in diabetes—2017. *Diabetes Care,* 40(Suppl. 1), S1–S135.

American Diabetes Association. (2023). Standards of medical care in diabetes—2017. *Diabetes Care, 40*(Suppl. 1), S1–S135.

Andernach, I. E., Hübschen, J. M., and Muller, C. P. (2020). Hepatitis B virus*: The genotype G story. Virus* Research, 285, 198021.

Anderson, E. S., Shao, Y.,and Houghton, M. (2021). Global distribution and clinical significance of hepatitis C virus genotypes. *Journal of Hepatology*, 75(2), 294–306.

Azam, G., Alam, S., Khan, A. S., Giasuddin, R. S., and Khan, M. (2017). High prevalence of diabetes mellitus among adult patients with viral hepatitis C than hepatitis B. International. *Journal of Life-Sciences* Scientific Research, 3(5), 1365–1369.

Balarabe-Musa, B., Olaomi, O., and Okoye, G. (2024). Gaps in viral hepatitis screening among diabetic patients in Nigeria. *Nigerian Journal of Clinical Practice*, 27(12), 1345–1350.

Barrera, F., Uribe, J., Olvares, N., Huerta, P., Cabrera, D., and Romero-Gómez, M. (2024). The Janus of a disease: Diabetes and metabolic dysfunction-associated fatty liver disease. *Annals of Hepatology*, 29(4), 101501

Bartenschlager, R., Lohmann, V., and Penin, F. (2017). The molecular and structural basis of advanced antiviral therapy for hepatitis C virus infection. *Nature Reviews Microbiology,* 15(8), 482–496.

Belete, D., Kassaw, D., and Andualem, T. (2023). Prevalence of Hepatitis B and Hepatitis C Viral Infections and Their Associated Factors among Diabetic Patients Visiting Debre Tabor Referral Hospital, Northwest Ethiopia, 2021:A Cross-Sectional Study. *Canadian Journal of Gastroenterology and Hepatology, 2*023(1):1-7

Blum, H. E. (2016). History and global burden of viral hepatitis. Digestive Diseases, 34(4), 293–302.

Centers for Disease Control and Prevention. (2024). Hepatitis B.

Chen, C. J., Yang, H. I., and Iloeje, U. H. (2021). Hepatitis B virus genotypes: Epidemiological and clinical relevance. *Journal of Hepatology,* 74(3), 460–472.

Chen, L., Zhang, Q., and Wang, H. (2024). Progress of infection and replication systems of hepatitis B virus. *ACS Pharmacology and Translational Science,* 7(4), 1147–1160.

Chen, Y., Zhao, L., and Xu, D. (2023). Hepatitis B virus X protein and its role in glucose metabolism dysregulation. *Journal of Medical Virology,* 95(1), e28134.

Chen, Z.,and Liu, Y. (2024). Immunometabolic alterations in type 2 diabetes mellitus revealed by single-cell sequencing. *Frontiers in Immunology,* 15, 1187654.

Chekina, N., Petrova, A., and Ivanov, V. (2023). Hepatitis C virus genotype 3 and its role in hepatic steatosis: A clinical perspective. *Hepatology Research,* 53(4), 421–430.

De Luca, A., Bianco, C., and Parisi, S. (2018). Immune response to hepatitis B vaccination in patients with type 2 diabetes mellitus: *A systematic review and meta-analysis*. *Vaccine,* 36(27), 3774–3781.

Deshpande, A., Mistry, F., and Kumar, S. (2019). Hepatitis C virus infection and diabetes mellitus: Pathophysiology and clinical implications. *Journal of Clinical and Translational Hepatology*, 7(1), 42–48.

Duggal, P., Thio, C. L., and Ray, S. C. (2020). Genetic determinants of host susceptibility to hepatitis B and C virus infections. *Nature Reviews Gastroenterology and Hepatology,* 17(1), 40–55.

El-Serag, H. B., and Kanwal, F. (2014). Epidemiology of hepatocellular carcinoma in the United States: Where are we? Where do we go? *Hepatology, 60(5),* 1767–1775.

Ephraim, R., Nsiah, P., Osakunor, D., Adoba, P., Sakyi, S., and Anto, E. (2014). Seroprevalence of Hepatitis B and C Viral Infections among Type 2 Diabetics: A Cross-sectional Study in the Cape Coast Metropolis. *Annals of medical and health sciences research,* 4(5): 719–722.

Esmaeilzadeh, A., Ghasemi, E., & Karimzadeh, H. (2022). Hepatitis C virus and insulin resistance: Molecular mechanisms and clinical impact. *World Journal of Hepatology,* 14(6), 1128–1141

Eliah, D. T., Chamba, N. G., Sadiq, A. M., Mwasamwaja, A. O., Kimondo, F. C., Matay, C. D., Nziku, E. B., Mirai, T. E., Muhina, I. A. I., Said, F. H., Gharib, S. K., Lyamuya, F. S., Mkwizu, E. W., Kilonzo, K. G., Maro, V. P.,and Shao, E. R. (2025). Seroprevalence and associated factors for hepatitis B and hepatitis C viral infection among patients with diabetes mellitus in Northern Tanzania. *PloS one,* 20(2): 11-16.

Feng, J., Chen, G., and Liu, Y. (2023). The role of hepatitis B virus preS/S gene mutations in disease progression and vaccine escape. *Viruses,* 15(2), 445.

Ghany, M. G., Morgan, T. R., and AASLD-IDSA Panel. (2020). Hepatitis C guidance 2020 update: Recommendations for testing, managing, and treating hepatitis C virus infection. *Hepatology*, 71(2), 686–721.

Gower, E., Estes, C., Blach, S., Razavi-Shearer, K., and Razavi, H. (2014). Global epidemiology and genotype distribution of the hepatitis C virus infection. *Journal of Hepatology*, 61(1 Suppl), S45–S57.

Gupta, V., Singla, R., and Sinha, S. (2018). Prevalence of hepatitis B and C infections in patients with diabetes mellitus in India: A cross-sectional study. *Indian Journal of Endocrinology and Metabolism,*22(3),389–394.

Harrison, T. J., & Dusheiko, G. M. (2019). Hepatitis B virus: Molecular virology and common mutants. Seminars in Liver Disease, 39(1), 18–32.

Hauwa, B. M, Salisu, B. D., Sani, U. A., Mohammad, B. Iliya, S. J., Noah, O., Aminu, S., and Banya, R. J. (2018). Seroprevalence of Hepatitis B and C Viruses Amongst Type 2

Diabetic Patients in Dutse, Jigawa State*, Nigeria. Journal of Advances in Medicine and Medical Research*, 27 (9):1–7.

Hou, J., Wang, Z., Cheng, J., and Lin, Y. (2022). Hepatitis B virus genotypes and clinical outcomes: The Asian perspective. *Hepatology International,* 16(2), 233–245.

Hu, Y., Wu, Z., and Zhou, Y. (2021). Hepatitis B virus and metabolic disorders: The emerging interplay. *Frontiers in Cellular and Infection Microbiology*, 11, 738307.

Hussein, N. R., Abozait, H. J., Naqid, I. A., Ibrahim, N. M., Khalid, F. K., Musa, D. H., and Saleem, Z. S. (2025). Risk Factors of Hepatitis B Virus Infection in the Kurdistan Region of Iraq: A Cross-Sectional Study. *Mediterranean journal of hematology and infectious diseases,* 17(1): e2025018

Ibrahim, A. O., Musa, B. M., and Adamu, U. (2019). Prevalence of hepatitis B and C virus infections among patients with diabetes mellitus in Kano, Nigeria. *Pan African Medical Journal*, 34, 62.

International Diabetes Federation. (2024). *IDF Diabetes Atlas* (11th ed.). Brussels, Belgium: International Diabetes Federation.

Jama, D. M., Mahdi, M. W., Mohammed, A. M., Muhammad, A. S., and Kabiru, M. Y. (2025). Exploring Health-Seeking Patterns Among Abuja Elites: Institutional And Socio-Cultural Driver. *International Journal of Social Health and Medical Research*, 8(3).

Lemoine, M., Thursz, M.,and Njie, R. (2020). Hepatitis B in Africa: The current state of knowledge and perspectives for the future. *Clinical Liver Disease,* 15(1), 1–6.

Liang, T. J., and Block, T. M. (2015). Molecular biology of hepatitis B virus: Implications for pathogenesis and therapy. *Hepatology,* 62(2), 432–443.

Lok, A. S. F., and McMahon, B. J. (2009). Chronic hepatitis B: Update 2009. *Hepatology*, 50(3), 661–662.

Mak, L. Y., Seto, W. K., and Yuen, M. F. (2020). Hepatitis B core-related antigen (HBcrAg): An emerging marker for chronic hepatitis B virus infection. *Alimentary Pharmacology and Therapeutics, 5*2(12), 1740–1751. .

Mohd Hanafiah, K., Groeger, J., Flaxman, A. D., and Wiersma, S. T. (2013). Global epidemiology of hepatitis C virus infection: New estimates of age-specific antibody to HCV seroprevalence. *Hepatology,* 57(4), 1333–1342.

Ndako, J.A., Owolabi, A.O., and Olisa, J.A. (2020). Studies on the prevalence of Hepatitis C virus infection in diabetic patients attending a tertiary health-care facility South-west Nigeria. *BMC Infect Disease,* 20: 664.

Ndako, J., Nwankiti, O., Okeke, C., Adekeye, A., Choji, T., Alesa, M., and Alarape, A. (2011). Screening Response to Hepatitis C Virus Antibodies among Diabetic Patients Attending UITH Nigeria. *Curr. Res. J. Biol. Sci.,* 3(6): 542-546.

Nguyen, M. H., Wong, G., and Gane, E. (2020). Hepatitis B virus: Advances in prevention, diagnosis, and treatment. *Clinical Microbiology Reviews,* 33(2), e00046-19.

Ong, J. J., Fu, H., Smith, M. K., Tucker, J. D., Baggaley, R. C., and Easterbrook, P. J. (2021). Global epidemiology of HIV, hepatitis B, and hepatitis C coinfection: A systematic review. *The Lancet Infectious Diseases, 21*(6), 795–808.

Olokoba, A. B., Aderibigbe, S. A., Kayode, O. O., and Olokoba, L. B. (2012). Hepatitis B virus infection in Nigeria: A review. *Nigerian Medical Journal,* 53(2), 120–122.

Oluwole, O. F., Adejumo, O. E., & Oke, O. (2016). Prevalence of hepatitis B virus and hepatitis C virus among diabetic patients in Ekiti State, Nigeria. *International Journal of Tropical Disease & Health,* 19(2), 1–7.

Orabueze, I.N., Ike, A.C., and Aniche, O.M. (2024). Hepatitis B virus infection among illegal drug users in Enugu State, Nigeria: prevalence, immune status, and related risk factors. *BMC Public Health* 24: 1203.

Ott, J. J., Stevens, G. A., Groeger, J., and Wiersma, S. T. (2012). Global epidemiology of hepatitis B virus infection: New estimates of age-specific HBsAg seroprevalence and endemicity. *Vaccine,* 30(12), 2212–2219.

Papatheodoridis, G. V., Dalekos, G. N.,and Yurdaydin, C. (2016). Similar risk of hepatocellular carcinoma during long-term entecavir or tenofovir therapy in Caucasian patients with chronic hepatitis B. *Journal of Hepatology,* 64(6), 1210–1216.

Polaris Observatory HCV Collaborators. (2017). Global prevalence and genotype distribution of hepatitis C virus infection in 2015: A modelling study. *Lancet Gastroenterology & Hepatology*, 2(3), 161–176.

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. *Lancet*, 386(10003), 1546–1555.

Stanaway, J. D., Flaxman, A. D., Naghavi, M., Fitzmaurice, C., and Vos, T. (2016). The global burden of viral hepatitis from 1990 to 2013: Findings from the Global Burden of Disease Study 2013. *Lancet,* 388(10049), 1081–1088.

Umoh, N. J, Akpan, U. P, and Ekong, E. E. (2018). Seroprevalence of hepatitis B and C virus among diabetic patients in Uyo, South-South Nigeria. *African Journal of Clinical and Experimental Microbiology*, 19(1), 34–41.

Wang, Q., Li, M., and Zhou, Y. (2023). Diabetes mellitus and its association with viral hepatitis: Pathophysiology and clinical outcomes. *Frontiers in Medicine,* 10, 1123456.

World Health Organization. (2017). *Global hepatitis report 2017.* World Health Organization.

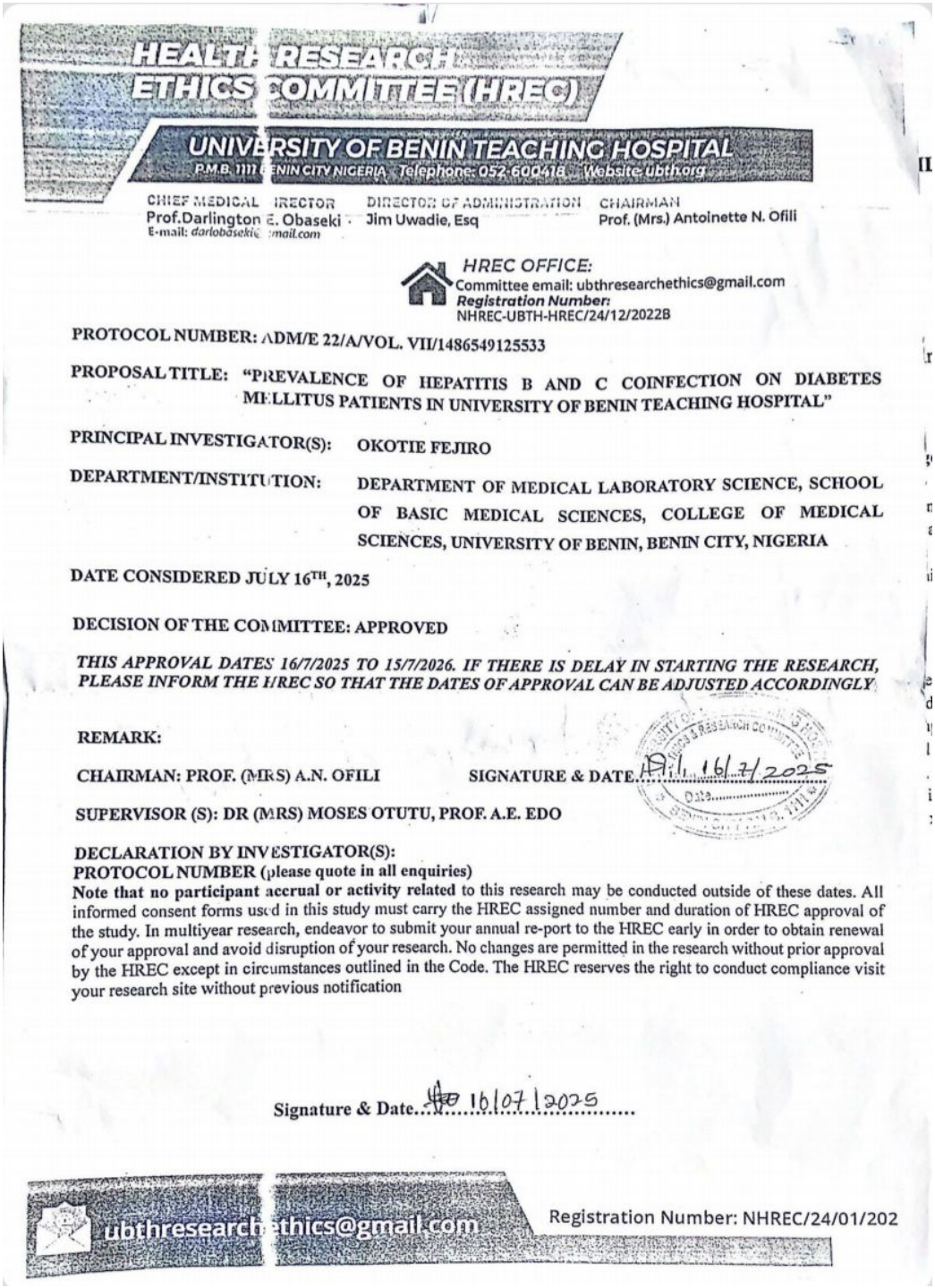
World Health Organization. (2021). Global progress report on HIV, viral hepatitis and sexually transmitted infections, 2021. World Health Organization.

Yameny, A. (2024). *Diabetes mellitus: Pathophysiology, complications, and manage*Abdel-Malek, W., Abdel-Rahman, M., and Ahmed, S. (2022). Hepatitis C virus proteins and their role in insulin resistance and diabetes development. *Frontiers in Endocrinology, 13,* 954321.

Yuen, M. F., Chen, D. S., Dusheiko, G. M., Janssen, H. L. A., and Locarnini, S. A. (2018). Hepatitis B virus infection. *Nature Reviews Disease Primers,* 4, 18035.

Zhang, Z., Li, X., and Liu, J. (2020). Hepatitis B virus and metabolic disease: A clinical overview. *Journal of Clinical and Translational Hepatology*, 8(3), 265–272.

APPENDIX I



HEALTH RESEARCH ETHICS COMMITTEE (HREC)

UNIVERSITY OF BENIN TEACHING HOSPITAL

P.M.B. 1111 BENIN CITY NIGERIA Telephone: 052-600418 Website: ubth.org

CHIEF MEDICAL DIRECTOR Prof. Darlington E. Obaseki E-mail: darlobaseki@gmail.com

DIRECTOR OF ADMINISTRATION Jim Uwadie, Esq

CHAIRMAN Prof. (Mrs.) Antoinette N. Ofili

HREC OFFICE:
Committee email: ubthresearchethics@gmail.com
Registration Number:
NHREC-UBTH-HREC/24/12/2022B

PROTOCOL NUMBER: ADM/E 22/A/VOL. VII/1486549125533

PROPOSAL TITLE: "PREVALENCE OF HEPATITIS B AND C COINFECTION ON DIABETES MELLITUS PATIENTS IN UNIVERSITY OF BENIN TEACHING HOSPITAL"

PRINCIPAL INVESTIGATOR(S): OKOTIE FEJIRO

DEPARTMENT/INSTITUTION: DEPARTMENT OF MEDICAL LABORATORY SCIENCE, SCHOOL OF BASIC MEDICAL SCIENCES, COLLEGE OF MEDICAL SCIENCES, UNIVERSITY OF BENIN, BENIN CITY, NIGERIA

DATE CONSIDERED JULY 16TH, 2025

DECISION OF THE COMMITTEE: APPROVED

THIS APPROVAL DATES 16/7/2025 TO 15/7/2026. IF THERE IS DELAY IN STARTING THE RESEARCH, PLEASE INFORM THE HREC SO THAT THE DATES OF APPROVAL CAN BE ADJUSTED ACCORDINGLY

REMARK:

CHAIRMAN: PROF. (MRS) A.N. OFILI **SIGNATURE & DATE.** 16/7/2025

SUPERVISOR (S): DR (MRS) MOSES OTUTU, PROF. A.E. EDO

DECLARATION BY INVESTIGATOR(S):
PROTOCOL NUMBER (please quote in all enquiries)
Note that no participant accrual or activity related to this research may be conducted outside of these dates. All informed consent forms used in this study must carry the HREC assigned number and duration of HREC approval of the study. In multiyear research, endeavor to submit your annual re-port to the HREC early in order to obtain renewal of your approval and avoid disruption of your research. No changes are permitted in the research without prior approval by the HREC except in circumstances outlined in the Code. The HREC reserves the right to conduct compliance visit your research site without previous notification

Signature & Date. 16/07/2025

ubthresearchethics@gmail.com Registration Number: NHREC/24/01/202

APPENDIX II

INFORMED CONSENT

STUDY TITLE: PREVALENCE OF HEPATITIS B AND C COINFECTION IN DIABETES MELLITUS PATIENTS IN UNIVERSITY OF BENIN TEACHING HOSPITAL

Principal Investigator: OKOTIE FEJIRO

Institution: UNIVERSITY OF BENIN (500 Level Student, Medical Laboratory Science)

INTRODUCTION

You are invited to participate in a research study aimed at determining the prevalence of hepatitis B and C co-infection among patients living with diabetes mellitus in Benin City, Nigeria. The study seeks to understand how common these infections are among diabetic patients and contribute to strategies for improving their care. Your participation is voluntary, and this form contains important information to help you decide whether to take part.

PURPOSE OF THE STUDY

- Determine the prevalence of Hepatitis B (HBV) and Hepatitis C (HCV) Co-infection among individuals with diabetes mellitus.

- Identify potential risk factors for infection within this population.

- Contribute to public health awareness and improve preventive healthcare strategies.

PROCEDURE

If you agree to participate, the following will occur:

Sample Collection:

- A trained healthcare professional will collect approximately 5–10 mL of blood from a vein in your arm using standard venipuncture techniques in a sterile and safe environment.

Laboratory Testing:

- Your blood will be tested in a certified laboratory for:

  - Hepatitis B surface antigen (HBsAg)

  - Hepatitis C virus antibodies (anti-HCV)

  using standard rapid diagnostic test kits.

Medical Record Review:

- Information such as your age, gender, diabetes type, and duration of illness may be reviewed from your medical records to support the study analysis.

DURATION

The blood collection will take about 5–6 minutes. The overall study duration may last up to 3 months, but your active participation will be limited to the sample collection and brief data recording.

OPTIONAL FOLLOW-UP

You may choose to be contacted later for follow-up on your results or to participate in future related studies.

RISKS AND DISCOMFORT

- Physical Risks: Minor discomfort, bruising, or swelling may occur at the blood draw site. Fainting or infection is rare.

- Emotional Risks: Testing positive for hepatitis B or C may cause emotional stress. Counseling and referral will be offered.

- Privacy Risks: There is a small chance of unintended data disclosure, but strong confidentiality measures will be in place.

BENEFITS

- Direct Benefit: You will receive free hepatitis B and C testing. If you test positive, you will be referred for medical care and support.

- Indirect Benefit: Your participation will help researchers understand co-infection risks in diabetic patients, potentially improving healthcare approaches.

CONFIDENTIALITY

- Your name will be replaced with a study code.

- Only approved study personnel will access your data.

- Results will be reported in group format only.

- If you test positive, results will be shared confidentially with you and, with your permission, with your doctor.

- Positive test results may be reported to public health authorities, but your identity will remain protected.

VOLUNTARY PARTICIPATION AND RIGHT TO WITHDRAW

Participation is completely voluntary. You can refuse or withdraw at any time without affecting your medical care or benefits. If you withdraw, any data or samples collected will be discarded unless you allow otherwise.

WHAT HAPPENS TO YOUR SAMPLES AND DATA?

- Your sample will be used only for the hepatitis B and C tests.

- Remaining samples may be stored for related future studies only with your consent.

- Data may be shared with other researchers in a de-identified (anonymous) form.

IF YOU TEST POSITIVE FOR HEPATITIS B OR C

- You will be informed confidentially.

- You will receive counseling and referral to a healthcare provider for further care and treatment.

- You will receive information about hepatitis, its management, and prevention.

CONSENT TO PARTICIPATE

I have read (or had read to me) this informed consent form. I understand the purpose, procedures, risks, and benefits. I have had the opportunity to ask questions and received clear answers. I voluntarily agree to participate in this study.

Participant's Name: _____

Signature: _____ Date: _____

Researcher's Name: _____

Signature: _____ Date: _____

APPENDIX III

QUESTIONNAIRE

Title: Prevalence of Hepatitis B and C Co-Infection in Diabetes Mellitus Patients

Instructions: Please answer the following questions as accurately as possible. All responses will be kept confidential and used only for research purposes.

Section A: Socio-Demographic Information

1. Age: _____

2. Gender:

[ ] Male

[ ] Female

3. Marital Status:

[ ] Single

[ ] Married

[ ] Divorced

[ ] Widowed

4. Educational Level:

[ ] No formal education

[ ] Primary

[ ] Secondary

[ ] Tertiary

5. Occupation: _____

Section B: Clinical Information

6. Duration of Diabetes Diagnosis:

[ ] <1 year

[ ] 1–5 years

[ ] 6–10 years

[ ] >10 years

7. Type of Diabetes:

[ ] Type 1

[ ] Type 2

[ ] Don’t know

8. Are you on any treatment for diabetes?

[ ] Yes

[ ] No

If yes, specify: _____

9. Have you been hospitalized for diabetes complications in the past year?

[ ] Yes

[ ] No

Section C: Risk Factors for Hepatitis B and C

10. Have you ever received a blood transfusion?

[ ] Yes

[ ] No

11. Have you ever undergone any surgical procedure?

[ ] Yes

[ ] No

12. Do you share sharp objects (razors, needles, etc.)?

[ ] Yes

[ ] No

13. Have you ever had a tattoo or body piercing?

[ ] Yes

[ ] No

14. Have you ever been tested for hepatitis B or C before?

[ ] Yes

[ ] No

If yes, what was the result?

[ ] Negative

[ ] Positive

[ ] Don’t remember

15. Do you use intravenous drugs?

[ ] Yes

[ ] No

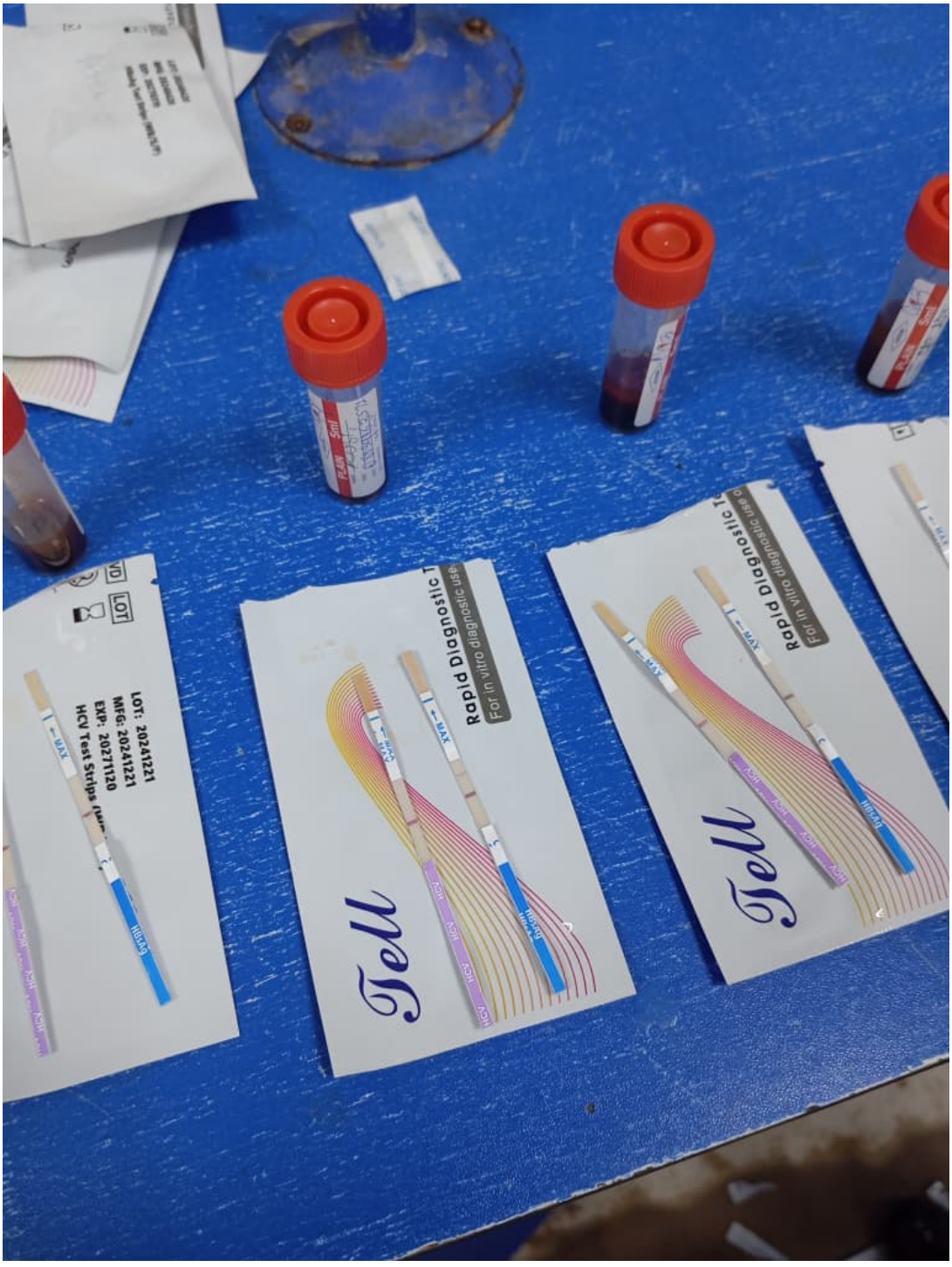
16. Do you practice safe sex (use of condoms)?

[ ] Always

[ ] Sometimes

[ ] Never

APPENDIX IV


LOT
LOT: 20241221
MFG: 20241221
EXP: 20271120
HCV Test Strips
Rapid Diagnostic
For in vitro diagnostic use
Tell
MAX
HCV
Ab
Human