

**Epidemiological study of hepatitis B virus among immigrants in Khartoum state- Sudan 2021-2022**Wafa Mohammed Hussein Abuelgasim<sup>1\*</sup>, Ayman Anwar Mohammed osman<sup>2</sup>, Muslih Haroun Elhussein Gamea<sup>3</sup><sup>1</sup>Assistant professor of Medical microbiology, Faculty of Medical Laboratory Sciences, National Ribat University  
Email: [dr.wafa1980@gmail.com](mailto:dr.wafa1980@gmail.com)<sup>2</sup>Institute of Forensic Sciences, National Ribat University<sup>3</sup>Sudan International University, Register of the National Council for Medical and Health Professions, SudanCorresponding Author: Wafa Mohammed Hussein Abuelgasim | Email: [dr.wafa1980@gmail.com](mailto:dr.wafa1980@gmail.com)**Received:** 18.10.2025 | **Revised:** 23.12.2025**Accepted:** 29.01.2026 | **Published:** 28.02.2026

**Abstract: Summary:** Simple, rapid and accurate assays for hepatitis B surface antigen (HBsAg) and hepatitis B e antigen (HBeAg) are helpful for clinical diagnosis and field epidemiological surveys. A commercially developed, rapid immunochromatographic test for simultaneous detection of HBsAg, and HBeAg was evaluated using a total of 250 selected samples. Results of the rapid test were compared with standard enzyme immunoassay (EIA) methods for HBsAg and HBeAg detection. The overall sensitivity and specificity for the detection of HBsAg were 95 and 100%, and the corresponding positive and negative predictive values were 100 and 99.7%, respectively. The sensitivity and specificity for the detection of HBeAg were slightly less than that for HBsAg, and were 80 and 98%, with positive and negative predictive values of 91 and 94%, respectively. Thus, compared with the EIA method, the rapid test was highly sensitive and accurate for the detection of HBsAg although somewhat less sensitive and specific for detection of HBeAg. Because of its speed, simplicity and flexibility, the rapid test is ideally suited for HBsAg and HBeAg screening in population-based epidemiological studies and in low-risk populations, particularly in regions of the world where hepatitis B is endemic.

**Keywords:** HBsAg, epidemiological studies, EIA, antigen.**Citation:** Wafa Mohammed Hussein Abuelgasim *et al.* Epidemiological study of hepatitis B virus among immigrants in Khartoum state- Sudan 2021-2022. Grn Int J Apl Med Sci, 2026 Jan-Feb 4(1):19-33.**INTRODUCTION**

Hepatitis B virus (HBV) infects more than 300 million people worldwide and is a common cause of liver disease and liver cancer. HBV, a member of the *Hepadnaviridae* family, is a small DNA virus with unusual features similar to retroviruses.

HBV replicates through an RNA intermediate and can integrate into the host genome. The unique features of the HBV replication cycle confer a distinct ability of the virus to persist in infected cells. Virological and serological assays have been developed for diagnosis of various forms of HBV-associated disease and for treatment of chronic hepatitis B infection. HBV infection leads to a wide spectrum of liver disease ranging from acute (including fulminant hepatic failure) to chronic hepatitis, cirrhosis, and hepatocellular carcinoma.

Acute HBV infection can be either asymptomatic or present with symptomatic acute hepatitis. Most adults infected with the virus recover, but 5%-10% are unable to clear the virus and become chronically infected. Many chronically infected persons have mild liver

disease with little or no long-term morbidity or mortality. Other individuals with chronic HBV infection develop active disease, which can progress to cirrhosis and liver cancer. These patients require careful monitoring and warrant therapeutic intervention.

Extrahepatic manifestations of HBV infection are rare but can be difficult to diagnose and manage. The challenges in the area of HBV-associated disease are the lack of knowledge in predicting outcome and progression of HBV infection and an unmet need to understand the molecular, cellular, immunological, and genetic basis of various disease manifestations associated with HBV infection.

**Structure of Virus**

The hepatitis B virus (HBV) is a small DNA virus with unusual features similar to retroviruses [1,2] it is prototype hepadnaviridae family. Related viruses are found in woodchucks, ground squirrels, tree squirrels, Peking ducks, and herons. Based on sequence comparison, HBV is classified into eight genotypes, A to H. Each genotype has a distinct geographic distribution. Three types of viral particles are visualized

in infectious serum by electron microscopy. Two of the viral particles are smaller spherical structures with a diameter of 20 nm and filaments of variable lengths with a width of 22 nm. The spheres and filaments are composed of hepatitis B surface antigen (HBsAg) and host-derived lipids without viral nucleic acids and are therefore noninfectious [3].

The infectious HBV virion (Dane particle) has a spherical, double-shelled structure 42 nm in diameter, consisting of a lipid envelope containing HBsAg that surrounds an inner nucleocapsid composed of hepatitis B core antigen (HBcAg) complexed with virally encoded polymerase and the viral DNA genome. The genome of HBV is a partially double-stranded circular DNA of about 3.2 kilobase (kb) pairs. The viral polymerase is covalently attached to the 5' end of the minus strand [4].

The viral genome encodes four overlapping open reading frames (ORFs: S, C, P, and X) [1,2]. The S ORF encodes the viral surface envelope proteins, the HBsA, and can be structurally and functionally divided into the pre-S1, pre-S2 and S region. The core or C gene has the precore and core regions. Multiple in-frame translation initiation codons are a feature of the S and C genes, which give rise to related but functionally distinct proteins. The C- ORF encodes either the viral nucleocapsid HBcAg or hepatitis B e antigen (HBeAg) depending on whether translation is initiated from the core or precore regions, respectively. The core protein has the intrinsic property to self-assemble into a capsid-like structure and contains a highly basic cluster of amino acid.

### Replication of Virus

The HBV replication pathway has been studied in great detail. The initial phase of HBV infection involves the attachment of mature virions to host cell membranes, likely involving the pre-S domain of the surface protein [5]. Various cellular factors have been proposed to be the viral receptors, but only carboxypeptidase D has been shown to play an essential role in viral entry for the duck HBV [6].

Mechanisms of viral disassembly and intracellular transport of the viral genome into the nucleus are not well understood and probably involve modification of the nucleocapsid core protein [7]. After entry of the viral genome into the nucleus, the single-stranded gap region in the viral genome is repaired by the viral pol protein, and the viral DNA is circularized to the covalently closed circular (cccDNA) form [8]. This form of HBV DNA serves as the template for transcription of several species of genomic and sub genomic RNAs and is the stable component of the replication cycle that is relatively resistant to antiviral action and immune clearance. The transcripts from the cccDNA are unspliced, polyadenylated and possess a 5' cap structure. The 3.5-kb genomic transcripts consist of two species

with different 5' ends: the pregenomic and the precore RNAs. The pregenomic RNA (pgRNA) serves as the template for reverse transcription and the messenger RNA for core and polymerase; the precore RNA directs the translation of the precore gene product. The polymerase translation as a result of a ribosomal scanning mechanism [9].

The large HBsAg (L-HBsAg) protein is translated from the 2.4-kb subgenomic RNA, the middle (M-HBsAg) and small HBsAg (S-HBsAg) proteins from the various forms of 2.1-kb RNAs, and the HBxAg protein from the 0.7-kb RNA. The S-HBsAg is the major S gene product and the L and M proteins are the minor species. Each surface protein has a glycosylation site in the S domain. Additional modifications of the L and M proteins occur at the pre-S2 domain with an N-linked oligosaccharide and a myristic acid at the amino-terminal glycine residue of the pre-S1 domain [10].

The distribution of the three envelope glycoproteins varies among the types of viral particles, with little or no L and M protein in the 20-nm particles but relatively more L protein in the Dane particles.

Replication of HBV begins with encapsidation of the genome. The packaging signal is a *cis*-acting element referred to as epsilon, which contains a stem-loop structure [11]. The terminal protein of the pol interacts with the epsilon and in concert with the core protein forms the nucleocapsid. After encapsidation, the pol mediates the reverse transcription of the pg RNA to minus-strand DNA and subsequent positive-strand synthesis. The circular form of the DNA is completed through several complicated steps of strand transfer [12]. The nucleocapsid then interacts with the envelope proteins in the endoplasmic reticulum to assemble into mature virion, which are then secreted into the extracellular milieu.

### Transmission risks

The hepatitis B virus can survive outside the body for at least 7 days [13]. Several factors influence the risk of transmission of HBV infection, including the viral load of the source.

In a healthcare occupational context, the level that is regarded as "high" for a viral load differs in various regions. In America and Ireland, HCWs who are infected with HBV but have a circulating viral burden <104 genome equivalents/ml are allowed to continue working unrestricted [14,15]. Transmission of HBV via a percutaneous route is considered unlikely at HBV DNA levels below 107 genome equivalents/ml [16].

### Needle sticks injuries

Those who are e antigen positive generally have higher viral loads, and the transmission rate of HBV following a needle sticks injury from a source who is e antigen positive is estimated to be between 30% and 62% [4,



22]. The same injury with exposure to blood from a source who is e antigen negative is associated with 6-37% risk of serological evidence of HBV infection in the recipient [4, 22]. Some patients are infected with pre-core mutant viruses. This is associated with a high viral load in the absence of the e antigen, and thus is also associated with a high risk of HBV transmission. The risk from needle stick injuries in the community is more difficult to estimate and the exact incidence of needle sticks injuries and the transmission rate is unknown. The limited published case reports [17,18] would indicate that there is a very low risk of HBV transmission associated with community acquired needle stick injuries.

### Other healthcare setting exposures

Spring loaded lancets have been implicated in the transmission of HBV to patients [19], as have reusable sub-dermal EEG electrodes [20]. There is a report of transmission of HBV to a patient during an endoscopic procedure, although no biopsies were taken, but bleeding gastric ulceration was identified. The presumed source was HBeAg positive [21]. Cleveland *et al* report that HBV infection prevalence in dentists increases with longer duration in practice [22]. Although rates in a reference control population were not included in this report, increasing prevalence with longer duration of practice indicates that there is potential for transmission to dentists during their work.

### Other percutaneous exposures

There are case reports documenting the transmission of HBV among butchers [23,24]. These are attributed to small hand cuts, and sharing knives, which can carry the virus on the handle. It is also thought that HBV can be transmitted via small cuts acquired in barber shops [25].

### Body fluid exposures

HBV DNA has been detected in body fluids apart from blood, including saliva, urine, nasopharyngeal fluid, semen, cervicovaginal fluids and tear (26). HBV transmission can occur following exposure to non-intact skin and mucous membranes. A case report describes transmission of HBV via broken skin, following contact with saliva and nasopharyngeal fluids from the source [27].

### Human bites

Case reports have documented HBV virus transmission via a human bite, when associated with the skin being broken [28,29].

### Sexual exposures

HBsAg has been found in seminal fluid and vaginal secretions; although concentrations in these fluids are lower than in blood [41]. The risk of transmission of HBV following sexual exposure depends on the type of exposure, the viral load of the source, and the presence of sexually transmitted infections [42]. The prevalence

of HBV in heterosexuals increased in those with multiple sexual partners [42-44], and those who have markers for HIV or syphilis [45]. An infection rate of [18-44] 2% is seen in regular heterosexual partners of HBV infected patients [46-48]. In addition, female commercial sex workers with a history of having anal intercourse had an increased risk of HBV infection [30].

The risk of developing HBV infection is particularly high among men who have sex with men. For men who have sex with men, the prevalence of HBV infection is increased in those who have a history of an ulcerative sexually transmitted infection, chlamydia, gonorrhoea, commercial sex work, or multiple partners. There is also a significant risk associated with unprotected insertive anal intercourse [31].

### Diagnosis and Serology

HBV infection leads to a wide spectrum of liver disease ranging from acute hepatitis (including fulminant hepatic failure) to chronic hepatitis, cirrhosis, and hepatocellular carcinoma (HCC) [2]. The diagnosis of HBV infection and its associated disease is based on a constellation of clinical, biochemical, histological, and serologic findings. A number of viral antigens and their respective antibodies can be detected in serum after infection with HBV, and proper interpretation of the results is essential for the correct diagnosis of the various clinical forms of HBV infection. HBV DNA followed shortly afterward by HBsAg and HBeAg are the first viral markers detected in serum [32].

HBsAg may be detected as early as 1-2 weeks or as late as 11-12 weeks after exposure, and its persistence is a marker of chronicity. HBeAg correlates with the presence of high levels of HBV replication and infectivity [33]. Within a few weeks of appearance of viral markers, serum alanine and aspartate aminotransferase (ALT, AST) levels begin to rise and jaundice may appear. HBeAg is usually cleared early, at the peak of clinical illness, whereas HBsAg and HBV DNA usually persist in the serum for the duration of clinical symptoms and are cleared with recovery. Antibodies to the HBV proteins arise in different patterns during acute hepatitis B. Antibody to HBcAg (anti-HBc) generally appears shortly before onset of clinical illness, the initial antibody being mostly immunoglobulin M (IgM) class, which then declines in titer as levels of IgG anti-HBc arise. Antibody to HBeAg (anti-HBe) usually appears shortly after clearance of HBeAg, often at the peak of clinical illness. Thus, loss of HBeAg and appearance of anti-HBe is a favorable serological marker during acute hepatitis B, indicating the initiation of recovery.

Antibody to HBsAg arises late during infection, usually during recovery or convalescence after clearance of HBsAg. Anti-HBs persists after recovery, being the antibody associated with immunity against HBV. However, between 10% and 15% of patients who



recover from hepatitis B do not develop detectable anti-HBs and have anti-HBc alone as a marker of previous infection. For this reason, anti-HBc testing is the most reliable means of assessing previous infection with HBV, whereas anti-HBs testing are used to assess immunity and response to HBV vaccine [34].

Patients who develop chronic hepatitis B have a similar initial pattern of serological markers with appearance of HBV DNA, HBsAg, HBeAg, and anti-HBc. In these persons, however, viral replication persists and HBsAg, HBeAg, and HBV DNA continue to be detectable in serum, often in high titers. The subsequent course of chronic hepatitis B is quite variable. Most persons remain HBsAg-positive for years if not for life and have some degree of chronic liver injury (chronic hepatitis) that can lead to significant fibrosis and cirrhosis. Persons with chronic HBV infection are also at high risk to ultimately develop HCC.

The diagnosis of acute hepatitis B is reliably made by the finding of IgM anti-HBc in serum, particularly in a patient with HBsAg and signs, symptoms, or laboratory features of acute hepatitis. Nevertheless, in some instances, HBsAg is cleared rapidly from the serum, and IgM anti-HBc is the only marker detectable when the patient presents with hepatitis. Testing for anti-HBc (total) and anti-HBs are not useful in diagnosis, and testing for HBeAg and anti-HBe should be reserved for persons who test positive for HBsAg. The finding of HBsAg without IgM anti-HBc suggests the presence of chronic hepatitis B, but this diagnosis generally also rests upon finding of persistence of HBsAg for at least 6 months [35].

HBV DNA testing can also be helpful in the assessment of level of viral replication and possibly helpful in assessing prognosis and need for antiviral therapy. Assays for HBV DNA level have improved substantially over the years [36].

The current real-time polymerase chain reaction– based assay (TaqMan) has a lower limit of detection of 5-10 HBV DNA copies/mL and can accurately quantify a wide range of levels. With this degree of sensitivity, HBV DNA can be detected early during the course of infection, arising before the appearance of other serological markers, such as HBsAg or anti-HBc. As a consequence, testing for HBV DNA has emerged as a primary approach in the diagnosis and management of HBV infection.

HBV DNA testing has now become routinely used in blood product screening (nucleic acid testing) [37] and monitoring of patients with HBV during treatment [38]. Persistently high levels of HBV DNA following resolution of hepatitis may be indicative of a failure to control the infection and an evolution into chronic infection.

### Acute Hepatitis B

About two-thirds of patients with acute HBV infection have a mild, asymptomatic and subclinical illness that usually goes undetected [39]. Approximately one-third of adults with acute HBV infection develop clinical symptoms and signs of hepatitis, which range from mild constitutional symptoms of fatigue and nausea, to more marked symptoms and jaundice, and rarely to acute liver failure.

The clinical incubation period of acute hepatitis B averages 2-3 months and can range from 1-6 months after exposure, the length of the incubation period correlating, to some extent, with the level of virus exposure [40]. The incubation period is followed by a short preicteric or prodromal period of constitutional symptoms such as fever, fatigue, anorexia, nausea, and body aches. During this phase, serum ALT levels rise and high levels of HBsAg and HBV DNA are detectable. The preicteric phase lasts a few days to as long as a week and is followed by onset of the detection limits and dynamic ranges of assays for HBV DNA. Jaundice or dark urine. The icteric phase of hepatitis B lasts for a variable period averaging 1-2 weeks, during which viral levels decrease. In convalescence, jaundice resolves but constitutional symptoms may last for weeks or even months. During this phase, HBsAg is cleared followed by the disappearance of detectable HBV DNA from serum.

Acute liver failure occurs in approximately 1% of patients with acute hepatitis B and jaundice [41]. The onset of fulminant hepatitis is typically marked by the sudden appearance of fever, abdominal pain, vomiting, and jaundice, followed by disorientation, confusion, and coma. HBsAg and HBV DNA levels generally fall rapidly as liver failure develops, and some patients are HBsAg-negative by the time of onset of hepatic coma.

Patients with acute liver failure due to hepatitis B require careful management and monitoring and should be referred rapidly to a tertiary medical center with the availability of liver transplantation [42].

### Chronic Hepatitis B

Chronic hepatitis B has a variable and dynamic course. Early during infection, HBeAg, HBsAg, and HBV DNA are usually present in high titers, and there are mild to moderate elevations in serum aminotransferase levels. With time, however, the disease activity can resolve either with persistence of high levels of HBeAg and HBV DNA (the “immune tolerance phase”) or with loss of HBeAg and fall of HBV DNA to low or undetectable levels (“inactive carrier state”). Other patients continue to have chronic hepatitis B, although some lose HBeAg and develop anti-HBe (HBeAg-negative chronic hepatitis B).

The course and natural history of hepatitis B are discussed in detail elsewhere in these proceedings [43].



The overall prognosis of patients with chronic hepatitis directly related to the severity of disease. For those with severe chronic hepatitis and cirrhosis, the 5-year survival rate is about 50% [44].

Among patients with evidence of chronic hepatitis (elevated ALT and inflammation and/or fibrosis on liver biopsy), many are asymptomatic or have non specific symptoms, such as fatigue and mild right upper quadrant discomfort.

Patients with more severe disease or cirrhosis may have significant constitutional symptoms, jaundice, and peripheral stigmata of end-stage liver disease including spider angiomas, palmar erythema, splenomegaly, gynecomastia, and fetor hepaticus. Ascites, peripheral edema, encephalopathy, and gastrointestinal bleeding are seen in patients with more advanced cirrhosis. ALT and AST are often elevated but may not correlate well with severity of liver disease. Bilirubin, prothrombin time, and albumin often become abnormal with progressive disease. Decreasing platelet count is often a poor prognostic sign. Patients with chronic hepatitis may develop acute exacerbations with markedly elevated serum ALT. This scenario is more frequently described in those with HBeAg negative chronic hepatitis B. To distinguish between acute hepatitis B and chronic hepatitis B with a flare, anti-HBc IgM is a useful marker, as described in the previous section. However anti-HBc of the IgM class can be detected occasionally in patients with chronic hepatitis B with exacerbation. Alpha-fetoprotein (AFP), used as a marker for HCC, is often elevated in parallel with ALT during acute exacerbation [45].

However, it is unlikely to exceed 400 ng/mL. In patients with AFP much greater than this level, development of HCC should be suspected [46].

An estimated one-third of persons with chronic HBV infection will ultimately develop a long-term consequence of the disease, such as cirrhosis, end-stage liver disease, or HCC. The determinants of outcome of chronic hepatitis B appear to be both viral (HBV DNA levels, HBV genotype, some HBV mutation patterns) and host-specific (age, gender, genetic background, immune status) [46].

### Rationale

The HBV is one of the most dangerous diseases in the world and cause high rate of mortality because of that we decided to talk about it but in specific way in immigrants in Sudan because they carry the disease by high rate and they transfer it to the Sudanese people by different way.

## OBJECTIVES

### General objective

Epidemiological study of hepatitis B among an immigrant in Sudan.

### Specific objectives

- To detect the presence of Hepatitis B antigen in an immigrant's serum by ICT and ELISA.
- To detect percentage of immigrants were infected with disease in Sudan.

## LITERATURE REVIEW

The 69th World Health Assembly endorsed the Global Health Sector Strategy including a goal to eliminate viral hepatitis infection as a public health threat by 2030 [46], and the World Health Organization (WHO) introduced the global targets for the care and management of hepatitis [47]. An accurate and updated estimate of hepatitis B virus (HBV) infection prevalence in Western countries is needed to support the hepatitis B elimination efforts along with the populations that need to be targeted for screening. In addition, the disease burden from HBV infection in these countries needs to be updated for the public health planning and appropriate allocation of healthcare and financial resources. However, to track progress against the WHO targets, a realistic estimate of HBV infection disease burden is required [47].

In 2020 Razavi-Shearer *et al.* was found, 76% (1.4 million out of 1.8 million total infections) of all living chronic HBV-positive immigrants were born in 20 countries, with almost 40% (554,700 HBV + immigrants from these three countries out of 1.4 million total infections among immigrants) coming from the Philippines, China, and Vietnam. When Global Burden of Disease regions are examined, 74% (1,039,900 from these regions out of 1.4 million total infected immigrants) of all cases among immigrants are from 5 regions: Asia Southeast, Asia East, Caribbean, Sub-Saharan Africa West, and Asia South [48].

Richter C found in 2014 Migrants born in hepatitis B virus (HBV) and hepatitis C virus (HCV) endemic countries are at increased risk of being infected with these viruses. The first symptoms may arise when liver damage has already occurred. The challenge is to identify these infections early, since effective treatment has become available. In 2011 we conducted a screening project in first-generation migrants (FGMs) born in Afghanistan, Iran, Iraq, the former Soviet Republics, and Vietnam and living in Arnhem and Rheden. All participants were offered free blood screening for HBV and HCV. In total 959 participants were tested, with the country of origin known for 927, equating to 28.7% of all registered FGMs from the chosen countries. Nineteen percent (n = 176) had serological signs of past or chronic HBV infection and 2.2% (n = 21) had chronic HBV infection. The highest prevalence of chronic HBV infection was found in the Vietnamese population (9.5%, n = 12). Chronic HCV was found in two persons from the former Soviet Republics and one from Vietnam. Twenty-four percent (n = 5) of the newly identified patients with chronic



HBV and one of the three patients with chronic HCV received treatment. Three of the patients, two with HCV and one with HBV, already had liver cirrhosis. The highest (9.5%) HBV prevalence was found in FGMs from Vietnam, indicating a high need for focusing on that particular immigrant population in order to identify more people with silent HBV infection. The fact that three patients already had liver cirrhosis underlines the necessity of early identification of HBV and HCV infection in risk groups [49].

Pablo Rivas found in 2013 Hepatitis B, C, and D and HIV Infections among Immigrants from Equatorial Guinea Living in Spain. A total of 1,220 subjects from Equatorial Guinea living in Spain (median age = 41 years; 453 male and 767 female) was examined for antibodies to human immunodeficiency virus (HIV) and Hepatitis B (HBV), C (HCV), and D (HDV) viruses. Extracted RNA and DNA from the positive samples were used to quantify viral load. The prevalence of HIV antibodies, HCV RNA, and HBV surface antigen (HBsAg) was 10.8% ( $N = 132$ ), 11.6% ( $N = 141$ ), and 7.9% ( $N = 96$ ), respectively. The most prevalent HIV variant was CRF02\_AG (38.5%;  $N = 40$ ). HCV genotype 4 (60%;  $N = 36$ ) and HBV genotype A3 (32%;  $N = 8$ ) were the hepatitis variants most frequently found. Super infection with HDV was seen in 20.9% ( $N = 24$ ) of HBsAg carriers. A control group of 276 immigrants from other sub-Saharan countries showed similar rates of HIV and HBsAg, although no HCV cases were found. Immigrants constitute a major source of HIV and hepatitis viruses in Spain; therefore, it is important that control measures are intensified [51].

Zuure FR in 2013 done Screening for hepatitis B and C in first-generation Egyptian migrants living in the Netherlands. Egypt has high prevalence of hepatitis C virus (HCV) infection and intermediate prevalence of hepatitis B virus (HBV) infection; however, infection prevalence among Egyptian migrants is unknown. Considering the asymptomatic onset and development of disease in chronically-infected patients, many may remain undiagnosed [50].

Carmine Rossi in 2012 found International migrants experience increased mortality from hepatocellular carcinoma compared to host populations, largely due to undetected chronic hepatitis B infection (HBV). We conducted a systematic review of the seroprevalence of chronic HBV and prior immunity in migrants arriving in low HBV prevalence countries to identify those at highest risk in order to guide disease prevention and control strategies [51].

Mark H.Eckman in 2011 found Hepatitis B virus (HBV) continues to cause significant morbidity and mortality in the United States. Current guidelines suggest screening populations with a prevalence of  $\geq 2\%$ . Our objective was to determine whether this screening threshold is cost-effective and whether

screening lower-prevalence populations might also be cost-effective [54].

Wong ww in 2011 found the prevalence of chronic hepatitis B (CHB) infection among the immigrants of North America ranges from 2 to 15%, among whom 40% develop advanced liver disease. Screening for hepatitis B surface antigen is not recommended for immigrants [52].

Christian Greenaway in 2011 found the Canadian Collaboration for Immigrant and Refugee Health. The foreign-born population bears a disproportionate health burden from tuberculosis, with a rate of active tuberculosis 20 times that of the non-Aboriginal Canadian-born population, and could therefore benefit from tuberculosis screening programs. We reviewed evidence to determine the burden of tuberculosis in immigrant populations, to assess the effectiveness of screening and treatment programs for latent tuberculosis infection, and to identify potential interventions to improve effectiveness [53].

Milionis C in 2010 found Greece is a place of settlement for a large number of immigrants, particularly from Albania, which constitute special community groups for public health policies. This study was designed to assess the seroprevalence of serological markers for Hepatitis B and C among juvenile immigrants from Albania settled in Greece [54].

Museruol in 2010 found Hepatitis B virus infection (HBV) remains highly endemic in many parts of the world. Refugees resettling in their host countries may carry a significant burden of disease due to HBV and may require long-term medical care. A retrospective descriptive study was conducted to assess the epidemiology of HBV and entry into medical care in refugee communities resettled in the State of Georgia over a five-year period: 2003-2007. Among 6,347 refugees (89.7% of those resettled) screened for HBV infection, six hundred and eighty (10.7%) were found to be HBsAg seropositive. Those between the ages of 10-39 years of age contributed to the majority of cases; and most originated from Africa (71%). All HBsAg positive cases were adequately referred to a primary care physician for further management but there are no formal feedback mechanisms in place to learn if those who tested positive for HBsAg accessed the primary healthcare system. HBV infection is a frequent infection among refugees resettled in the US. But their entry into healthcare to treat those with chronic infection is often unknown. Further efforts are required to assure their entry into the healthcare system. Primary care physicians caring for refugee patients should think about verifying HBV-infection status as part of health maintenance protocols [55].

Mccarthy AE in 2009 found increasing international migration may challenge healthcare providers



unfamiliar with acute and long latency infections and diseases common in this population. This study defines health conditions encountered in a large heterogeneous group of migrants [56].

Gaetano Scotto in 2009 done this study aims to determine the distribution and clinical features of HBV-genotypes in a population of immigrants affected by HBV-infection. *Methods.* Between 01/2003 and 03/2009, 1623 immigrants were tested for HBV-infection. Biochemical and virological activities were determined in HBsAg-positive patients; HBV-genotypes were determined, by the INNO-LiPA HBV Genotyping, in the subjects with HBV DNA detectable. In every patient we evaluated the stage and classified the infection as inactive carrier, mild or moderate/severe chronic hepatitis, cirrhosis, and/or HCC. *Results.* Among the tested subjects, 191 (11.7%) resulted HBsAg-positive, and in 144/191 (75.4%) serum HBV-DNA was detectable. The genotype distribution was as follows: 45,13% genotype E, 18,1% genotype D, 15,3% genotype B, 13,2% genotype C, 4,9% genotype A, 3,5% mixed genotypes (A–D). The evaluation of liver disease degree showed that 24.6% patients were inactive carriers of HBV infection, 19.4% presented immunotolerance phase, 34.5% had mild chronic hepatitis, 13.6% had a moderate/severe chronic hepatitis, 6.3% had cirrhosis, and 1.6% presented HCC. *Conclusions.* Our study evidences a high prevalence of HBV-infection in immigrants, and the potentiality of migratory flow in the introduction of genotype non-D hepatitis B virus. The Hepatitis B virus genotypes presented significant differences in epidemiological and clinical characteristics [57].

Silvia majori in 2008 found In Italy, about 5% of the population is represented by immigrants. The epidemiology of hepatitis A virus (HAV), hepatitis B virus (HBV), and hepatitis C virus (HCV) infection in Africa is very different from Europe; the present study aimed to assess the seroprevalence of viral hepatitis infections in sub-Saharan African immigrants living in Verona [58].

Hislop TG in 2007 Done Hepatitis B knowledge, testing and vaccination levels in Chinese immigrants to British Columbia, Canada.

Little is known about hepatitis B (HBV) and liver cancer control in Chinese in Canada. Liver cancer, a significant health problem in Asia, is preventable and can be controlled through HBV blood testing, vaccination, and community education about HBV [59].

Lin SY in 2006 done in Chronic hepatitis B virus (HBV) infection is a serious liver disease that, if left undiagnosed or without appropriate medical management, is associated with a 25% chance of death from cirrhosis or liver cancer. To study the demographics and prevalence of chronic HBV infection

and HBV vaccination in the Asian American population, we provided free HBV serological screening and administered a survey to 3163 Asian American adult volunteers in the San Francisco Bay Area between 2001 and 2006. Of those screened, 8.9% were chronically infected with HBV. Notably, one-half to two-thirds (65.4%) of the chronically infected adults were unaware that they were infected. Of those who were not chronically infected, 44.8% lacked protective antibodies against HBV and were likely susceptible to future infection. Men were twice as likely as women to be chronically infected (12.1% versus 6.4%). Asian Americans born in East Asia, Southeast Asia, or the Pacific Islands were 19.4 times more likely to be chronically infected than those born in the United States. Self-reporting of prior [60].

Levy V in 2004 found despite an effective vaccine, 60,000 new HBV infections were reported in the US in 2004; 95% in adults. We evaluate HBV seroprevalence, risk behaviors and self-reported vaccination among Latino immigrant, Asian immigrant and US born low income men in five northern California counties [61].

## MATERIAL AND METHODS

### Study approach

Qualitative approach.

### Study design

This study was a cross-sectional study.

### Study area

Khartoum state in Sudan republic in Alliance Affairs Department.

### Study population

Immigrants in Sudan were admitted to Alliance Affairs Department.

### Inclusion criteria

Adult male and female of immigrants in Sudan.

### Exclusion criteria

Children and pregnant women.

### Sample size

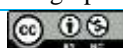
250 blood samples were collected.

### Method of data collection

Data was collected through an interviewed Questionnaire (Appendix).

### Specimen Collection

Under sterile condition, 3ml of venous blood were collected in sterile plain containers. The sera were obtained by centrifugation of the blood at 3000 rpm for 5minutes. The serum was separated from the clot and transferred into new sterile labeled plain containers and stored at -20°C until used.



## Laboratory Methods

### ICT technique for detection of HBV

Rapid immune chromatographic assay was used to detect anti HBV IgM antibody.

#### Principle of test

RapiGEN BIOCREREDIT HBsAg Test is adopted dual color system, contains a membrane strip pre-coated anti-Hepatitis B surface antigen antibody on test band.

#### Procedure

##### PREPARATION

\*Bring the test components and patient samples to room temperature before testing.

\*Do not break the seal of the foil pouch until ready to perform the test.

\*Add 100 ul of serum into the sample well using micropipette

\*Read the result within 10-15 minutes.

\*Don't read the result after 15 minutes.

#### Negative result

The presence of only one purple/red color band within the result window indicates a negative result.

#### Positive result

The presence of two color bands (C band and T band within the result windows, no matter which band appears first, indicates a positive result.

### ELISA technique for detection of HBV:

Enzyme-Linked Immunosorbent Assay (ELISA) was used to detect anti-HBV IgM antibody (Wantai, China).

#### Principle

The HBV antibody EIA test kit is a solid phase qualitative enzyme immunoassay based on a sandwich principle for the detection of total antibodies [IgG and IgM] to HBV in human serum or plasma, the micro well plate was coated with HBV recombinant antigens. During testing the specimens will add to the antigen coated micro well plate and then incubate. If the specimens contain antibodies to HBV it will bind to the antigens coated on the micro well plate to form immobilized antigen- HBV antibodies to HBV the complexes will not be formed. After initial incubation, the micro well plate should wash to remove unbound materials. The enzyme conjugated recombinant HBV antigen was added to the micro well plate and then incubated. The enzyme conjugated HBV antigens was bind to the immobilized antigen- HBV antibody complexes present. After the second incubation the micro well plates washed to remove unbound materials substrate A and substrate B was added and then incubated to produce blue color indication the amount of HBV antibodies presents in the specimens. Sulfuric acid solution will add to the micro well plates to stop the reaction producing a color change from blue to yellow. The color intensity, which corresponds to the

amount of HBV antibodies present in the specimens, was measured with a micro plate reader.

#### Assay procedure

All reagents and specimens to room temperature [15-30c] prior to testing. And procedure must be strictly followed.

#### Steps

Working wash buffer will prepare by diluting the concentrate wash buffer 1.25. Add each 100gl of ready to use controls into the appropriate wells of micro well plate .as: Leave well A1 as blank well. Well, B1 and C1 for negative control. Well, D1 and E1 for HBV positive control. Starting from well H1 for specimen 1.....

- Mix gently and cover the micro well plate with the plate sealer and incubate at 37c for 30 min.
- Remove the plate sealer and wash each well 5 time with the working wash buffer 1 by automated washer and turn the micro well plate upside down on absorbance tissue.
- Add 100gl of conjugate to each well except for the blank well.
- Cover the micro well plate with the plate sealer and incubate at 37C for 20 min.
- After incubation wash all well with working buffer.
- Add 50gl of substrate A and B to each well. Mix then cover micro well plate with plate sealer and incubate at 37C<sup>o</sup>for 10 min. Remove the plate sealer and add 50ql of stop solution to each well.
- Read absorbance optical density by micro well reader of each well at 450nm within 30 min.

#### Data analysis

Data analysis was been done manually and by a computer system using Statistical package for social science (SPSS) version 21.

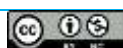
#### Ethical Consideration

Approval will take from college management and medical centers authority from which sample were been collected, consent also was taken from patients before sampling.

#### RESULTS

A total of 250 serum samples were obtained from an immigrants were admitted to Alliance Affairs Department in Khartoum state during the of January 2021 to March 2022.

The prevalence of HBV is 20% using ICT, while it was been 10% using ELISA. In population of (72% male). There is no association between gender and infection by HBV virus.



Frequencies of positive were 50 and percent (20%) and frequency of negative were 200 and percent (80%). Total of frequency 250 and percent (100%) (Table-1).

According to gender positive male were 36, negative 144 positive female 14, negative 56. Total positive 50, negative 200 (Table-4).

Frequency of the male 180 and percent (72%) and frequency of female 70 and percent (28%). Total of frequency 250 and percent (100%) (Table-2).

Frequency of the male positive were 20 and negative were 160 and total 180. And frequencies of the female positive were 5 and negative were 65 and total 70. Frequencies of the total positive were 25 and negative were 225 and total 250 (Table-5).

Frequency of positive was 25 and percent (10%) and frequency of negative were 25 and percent (10%). Total of frequency 250 and percent (100%) (Table-3).

**Table-1: Frequency and percentages of males and females**

	Frequency	Percent
Male	180	72%
Female	70	28%
Total	250	100%

**Table-2: Frequency and percentages of positive and negative results from total population**

	Frequency	Percent
Positive	50	20%
Negative	200	80%
Total	250	100%

**Table-3: Frequency and percentages of ICT results according to gender**

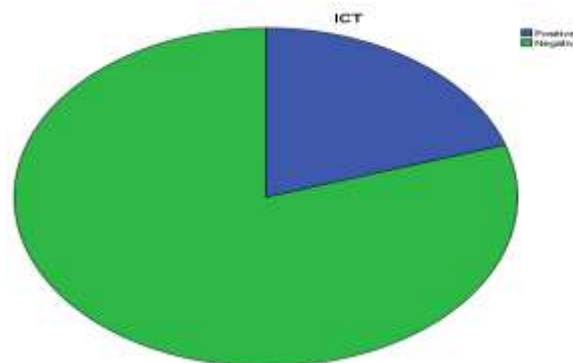
		ICT		Total
		Positive	Negative	
Gender	Male	36	144	180
	female	14	56	70
Total		50	200	250

**Table-4: Frequency and percentages of ELISA results**

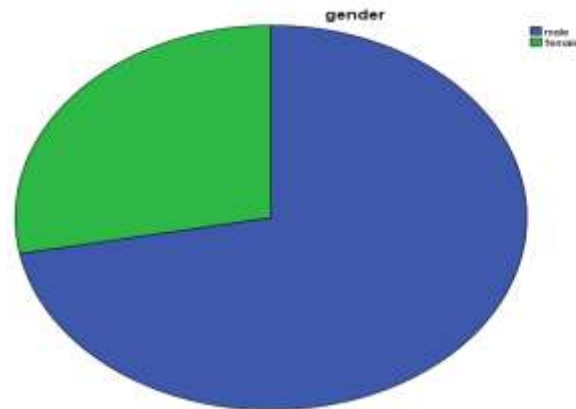
	Frequency	Percent
Positive	25	10.0
Negative	25	10.0
Total	50	20.0
Total	250	100.0

**Table-5: Frequency and percentages of ELISA results according to gender**

		ELISA		Total
		Positive	Negative	
Gender	Male	20	160	180
	Female	5	65	70
Total		25	225	250



**Figure-1: Percentage of ELISA positive and negative results by ICT**



**Figure-2: Percentage of ELISA positive and negative results according to age**

## DISCUSSION

Hepatitis B virus (HBV) is a common viral pathogen that currently infects an estimated 4 billion people worldwide, including 400 million who have chronic infection. Persons with chronic HBV infection are at a lifelong risk of developing hepatocellular carcinoma (HCC) or cirrhosis, or both. HBV is vaccine preventable; the rate of prevalence varies from region to region and among different population segments.

We studied all foreigners in Sudan to detect people infected with hepatitis B virus. We collected 250 samples with a total of 180 men and 70 women. This means that the ratio of men to women was 72% to 28%. Of all samples, we found the number of positive samples 50 samples and the negative samples were 200 samples, which means that the percentage of negative samples was 80% of the total number of samples and positive samples were 20% of the total number of samples, that means I most samples were negative. Consistent with many of the studies conducted on immigrants, the example is where Majori's research is also a study of migrants, where negative immigrants are found to be more than positive immigrants [64-61]. This is in line with our study. From this study we found that there is no relationship between gender and HBV infection.

Our study document to know prevalence of hepatitis B in migrant in Sudan in 2018 we done ICT and ELISA we found 20 positive male from 120 and 5 positive female from 70 sample HBV surface antigen.

Zuure done in 2013 screening hepatitis B in first generation Egyptian migrant, was found is high intermediate prevalence B virus and I deal Dr. Zuure in this study according to our study [52].

Another compared case with study of geatanoscoto to determine the distribution of HBV in population of immigrant, result among the tested 191(11.7%). HBV antigen positive with our study, we found 25(10%) positive by ELISA.

This study provides a new approach to estimating HBV prevalence in countries with a low HBV prevalence in the native population. The current approach provides the estimated HBV prevalence and associated disease burden at the national population level for all ages. Much of Western countries have a low prevalence but large immigrant populations. To develop robust strategies to reach the WHO viral hepatitis elimination targets, these countries need a strong understanding of the burden among immigrant communities. The data from said analyses would provide valuable insight that could inform programs and resource allocation.

## CONCLUSION

This study highlights the fact that HBsAg may not be an effective tool for diagnosis of HBV infections in our study population.

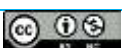
The level of occult HBV infection reported in this study clearly showed that serological markers of HBV infection should always be backed up with molecular tests to investigate possible occult HBV infection.

It also indicates the need for the identification of the virus genotypes of patients with occult HBV infection for better understanding of the clinical laboratory and the epidemiological characteristics of the infection.

Currently, a molecular test such as PCR, is not in use as a routine laboratory investigation for HBV in most of the health centers in Sudan.

Hence, PCR method as described in this study should be used as routine test for HBV infections in the hospitals in the country.

Finally, despite the current study supporting the presence of occult hepatitis B virus infection in immigrants patients, more studies need to be conducted to fully clarify the incidence of occult HBV infection in the general population in Sudan.



### Recommendation

It is recommended that hepatitis B negative health care workers should be vaccinated in order to decrease the rate of transmission of HBV among immigrants patients subjects at high-risk, especially immigrants from nations where hepatitis B is highly endemic, health care workers should be tested for HBV seromarkers and should be vaccinated.

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**Conflict of Interest:** The authors declare no conflict of interest.

**Source of Fund:** None

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Appendix

**Questionnaire**

**Epidemiological Study of Hepatitis B Virus among Immigrants in Khartoum State, Sudan**

**Please answer the following questions. All information will remain confidential and will be used only for research purposes.**

**Section A: Socio Demographic Information**

1. Age: \_\_\_\_\_ years
2. Gender:  Male  Female
3. Marital Status:  Single  Married  Divorced  Widowed
4. Country of Origin: \_\_\_\_\_
5. Duration of Residence in Sudan:  <1 year  1–5 years  >5 years
6. Educational Level:  None  Primary  Secondary  University
7. Occupation: \_\_\_\_\_

**Section B: Medical History**

8. Have you ever been diagnosed with Hepatitis B?  Yes  No  Don't know
9. Have you received Hepatitis B vaccination?  Yes  No  Not sure
10. If yes, number of doses received:  One  Two  Three
11. Have you ever had liver disease?  Yes  No
12. Do you have a family member with Hepatitis B?  Yes  No

**Section C: Risk Factors**

13. History of blood transfusion:  Yes  No
14. History of surgery:  Yes  No
15. Dental treatment history:  Yes  No
16. Sharing sharp instruments (razors, blades, needles):  Yes  No
17. Barber shop shaving with reusable blades:  Yes  No
18. Tattoo or body piercing:  Yes  No
19. Needle stick injury:  Yes  No
20. Injectable drug use:  Yes  No

**Section D: Sexual and Behavioral Risk Factors**

21. Multiple sexual partners:  Yes  No
22. Use of condoms during sexual activity:  Yes  No
23. History of sexually transmitted infection:  Yes  No

**Section E: Knowledge about Hepatitis B**

24. Have you heard about Hepatitis B before?  Yes  No
25. How can Hepatitis B be transmitted? (Select all that apply)



Blood transfusion    Sexual contact    Sharing needles

Mother-to-child transmission    Do not know

26. Can Hepatitis B be prevented by vaccination?    Yes    No    Do not know

**Section F: Laboratory Data (Completed by Researcher)**

27. ICT Result:    Positive    Negative

28. ELISA Result:    Positive    Negative

