

# CHAPTER ONE

## INTRODUCTION AND OBJECTIVES

### 1.1. Introduction

Hepatitis B virus (HBV) is a human pathogen that infects the liver and can cause both acute and chronic disease (Williams, 2006). HBV infection is one of the major global health problems (Rachna *et al.*, 2008). More than 350 million people worldwide have chronic HBV, out of some 2 billion exposed, leading to more than 600 000 deaths per year (Franco *et al.*, 2012).

HBV transmission occurs as a result of exposure to infected human blood or body fluids (Habib *et al.*, 2014). Hepatitis B virus is the most commonly transmitted blood borne virus in the health-care setting. Transmission generally occurs from patient to patient or from patients to healthcare personnel via contaminated instruments or accidental needle-stick (NSIs) or sharps injuries (Elduma and Saeed, 2011).

The virus can be transmitted directly through body fluids to mucous membranes, cutaneous scratches, abrasions, burns or other lesions. Indirect transmission can occur from surfaces contaminated with blood or body fluids to mucous membranes. HBV has been shown to survive in dried blood on surfaces at room temperature for at least a week (Elduma and Saeed, 2011).

The risk of HBV infection among health-care workers is 3–5 times higher than in the general population; in particular, surgeons, pathologists, physicians, laboratory staff, domestic staff and nurses have the highest risk of infection (Elduma and Saeed, 2011). HBV has long been recognized as an occupational risk for health-care personnel (HCP), including HCP trainees. The virus remains infectious for prolonged periods on environmental surfaces and is transmissible in the absence of visible blood (CDC, 2013).

HCP don't recognize all exposures to potentially infectious blood or body fluids and even if exposures are recognized, often don't seek post exposure prophylactic management (CDC, 2013).

World Health Organization (WHO) Reported in 2002, that 35 million health-care workers, 2 million experience percutaneous exposure to infectious diseases each year. Around 37.6% of hepatitis B health-care workers around the world are due to NSIs (Laishram *et al.*, 2013).

The risk of developing serological evidence of hepatitis B is high (32–67%) when blood is both hepatitis B surface antigens (HBsAg) and envelop antigen (HBeAg) positive. It reduces (23–37%) with HBsAg-positive but HBeAg-negative blood (Gorar *et al.*, 2014).

Prevention of HBV is a public health priority and immunization with hepatitis B vaccine is the most effective means of preventing hepatitis B infection and its consequences (Mohammad *et al.*, 2014).

The Advisory Committee on Immunization Practices (ACIP) recommends hepatitis B vaccine for everyone 18 years of age and younger as well as for adults over 18 years of age who are at risk of hepatitis B infection. Adults who are at increased risk of infection and who should receive vaccination include persons at occupational risk of infection (Mohammad *et al.*, 2014).

Immunizing healthcare workers (HCWs) against hepatitis B prevents nosocomial transmission of the virus from HCWs to patients and from patient to HCWs (Jobran *et al.*, 2014).

## **1.2. Rationale**

Hepatitis B Virus infection remains a major health problem causing considerable morbidity and mortality. Sudan is classified among the countries with high hepatitis B virus seroprevalence. Exposure to the virus varied from 47%–78%, with a hepatitis B surface antigen (Hatim, 2008). Hepatitis B virus is the agent of the most common blood-borne virus infection. Hospital personnel are often victims of occupational injuries by contaminated sharps or projection of contaminated fluids into mucous membranes (Hatim, 2008).

This is usually due to the unique nature of their occupation which involves working with exposure prone procedures during healthcare delivery (Elduma and Saeed, 2011). This study was conducted to detect the prevalence of HBV infection among hospital personnel working in Khartoum State hospitals.

### **1.3. Objectives**

#### **1.3.1. General objective**

To detect Hepatitis B virus infection among hospital personnel.

#### **1.3.2. Specific objectives**

- To determine the prevalence of hepatitis B virus among hospital personnel.
- To evaluate the risk of blood-borne infection among hospital personnel.
- To assess the knowledge of occupational exposure of blood borne infection by HBV and vaccination status among hospital personnel.

## **CHAPTER TWO**

### **LITERATURE REVIEW**

#### **1. Background**

Hepatitis B is an infectious disease, which is responsible for an estimated 350 million chronic infection worldwide (Seeger *et al.*, 2007). Although cases of hepatitis transmitted from blood and body fluids were noted in the 19th and early 20th centuries, widespread acceptance of this notion did not occur until the 1930s and 1940s (Seeger *et al.*, 2007).

Recognition of this form of blood-borne hepatitis came following investigations of outbreaks of hepatitis in people vaccinated for measles, mumps and yellow fever. All these vaccines contained serum or plasma that was added as “stabilizer” (Seeger *et al.*, 2007). This led MacCallum and Baur to introduce the term hepatitis B for the “serum” hepatitis, differentiating it from epidemiologically distinct hepatitis A in 1947 (Hovart and Tegtmeier, 2011).

The discovery of Australia antigen, which is detected in the sera of both acutely and chronically infected patient of hepatitis B virus, enabled the identification and characterization of hepatitis B virus. The Australia antigen, discovered by Blumberg and his colleges in 1965, is now known as Hepatitis B surface antigen (Seeger *et al.*, 2007).

HBV, a member of the Hepadnaviridae family, is a small DNA virus with unusual features similar to retroviruses. Since it possesses reverse transcriptase enzyme, it replicates through an RNA intermediate (Jake, 2009).

HBV genotypes have a distinct geographical distribution with genotypes A and D predominant in Europe, Middle East, Central Asia, Siberia and America. Genotypes B and C are predominant in East Asia while genotype E is more predominant in Africa (Panessa *et al.*, 2008). Genotype F has been reported in Central America. Also, genotype G has been reported in the United States and France (Chu *et al.*, 2003).

There are three envelope polypeptides that come under the designation HBs Ag (hepatitis B surface antigen), HBc Ag (hepatitis B core antigen), and HBe Ag (hepatitis B e antigen) (Washington *et al.*, 2009).

## **2.2. Structure and Composition**

Electron microscopy of HBsAg-positive serum reveals three morphologic forms. The most numerous are spherical particles measuring 22 nm in diameter. These small particles are made up exclusively of HBsAg—as are tubular or filamentous forms, which have the same diameter but may be over 200 nm long—and result from overproduction of HBsAg. Larger, 42-nm spherical virions (originally referred to as Dane particles) are less frequently observed (Geo *et al.*, 2010).

The outer surface, or envelope, contains HBsAg and surrounds a 27-nm inner nucleocapsid core that contains HBcAg. The variable length of a single-stranded region of the circular DNA genome results in genetically heterogeneous particles with a wide range of buoyant densities (Geo *et al.*, 2010).

### **2.3. Replication**

The replication of HBV is unique for several reasons. First, HBV has a distinctly defined tropism for the liver. Its small genome also necessitates economy, as illustrated by the pattern of its transcription and translation.

In addition, HBV replicates through an RNA intermediate produces and release antigenic decay particles (Murray *et al.*, 2002). HBV attachment to a receptor on the surface of hepatocytes occurs via a portion of the pre-S region of HBsAg. After uncoating of the virus, unidentified cellular enzymes convert the partially double-stranded DNA to covalent closed circular (ccc) DNA that can be detected in the nucleus (Murray *et al.*, 2002). The cccDNA serves as the template for the production of HBV mRNAs and the 3.5-kb RNA pregenome. The pregenome is encapsidated by a packaging signal located near the 5' end of the RNA into newly synthesized core particles, where it serves as template for the HBV reverse transcriptase encoded within the polymerase gene (Geo *et al.*, 2010).

An RNase H activity of the polymerase removes the RNA template as the negative-strand DNA is being synthesized. Positive-strand DNA synthesis does not precede to completion within the core, resulting in replicative intermediates consisting of full-length minus-strand DNA plus variable-length (20–80%) positive-strand DNA. Core particles containing these DNA replicative intermediates bud from pre-Golgi membranes (acquiring HBsAg in the process) and may either exit the cell or reenter the intracellular infection cycle (Geo *et al.*, 2010).

#### **2.4. Transmission**

Transmission of hepatitis B virus results from exposure to infectious blood or body fluids containing blood. Possible forms of transmission include sexual contact, blood transfusions and transfusion with other human blood products and vertical transmission from mother to child during childbirth (Fairley and Read, 2012; Buddeberg *et al.*, 2008).

#### **2.5. Epidemiology**

HBV infection is prevalent worldwide, representing a global public problem and causing chronic hepatitis, liver cirrhosis and HCC (Hou *et al.*, 2005; Hovart and Tegtmeier, 2011).



It is estimated that about 2 billion people have been infected by HBV, representing approximately 1/3 of the world's population, while more than 350 million are chronic carriers of HBV (Hou *et al.*, 2005).

The prevalence of HBV infection varies greatly in different parts of the world; it is highly endemic in developing region with large population such as south East Asia, China, sub-Saharan Africa, and the Amazon basin, where at least 8% are HBV chronic carriers (Hou *et al.*, 2005).

HBV is moderately endemic in the part of Eastern and Southern Europe, the Middle East, Japan and part of South America, where 2-7% are chronically infected. In the developed world, HBV has low endemicity. In North America, Northern and Western Europe and Australia, HBV is chronically present among 0, 5-2% of the population residing there (Hou *et al.*, 2005).

The prevalence of the infection in HCWs, a high risk group for acquiring infection with blood borne pathogens due to occupational contact with infected body fluids, depends upon HBV prevalence in the general population.

In India, an intermediate endemic zone where the estimated prevalence rate of HBV in the healthy general population is around 4.7%, a recent study showed 5% HBs Ag positivity in HCWs, but a highest seropositivity of around 40% among laboratory technicians (Kosgerogluet *al.*, 2004). In Taiwan, among HCWs who were exposed to high risk patients, nearly 16% had HBV (Pereira *et al.*, 2009).

## **2.6. Pathogenesis**

HBV disease is a non-inflammatory liver disease with variable severity, with hepatocytes representing the primary target cells for HBV (Chisari *et al.*, 2009; Hovart and Tegtmeier, 2011). Infection leads either to acute disease that resolve or chronic one lasting for years. Persistent infection by HBV is often associated with chronic liver disease that can lead to development of cirrhosis and hepatocellular carcinoma. It is suggested that HBV that is not directly cytopathic for the infected hepatocyte (Chisari *et al.*, 2009). Rather, viral clearance and disease pathogenesis are largely mediated by adaptive immune response in HBV infection. It is widely believed that cytotoxic T-lymphocytes are the cells that clearer viral infections by killing HBV-infected cells. This is likely to occur in adult patient, unlike in neonates, where neonatal tolerance to HBV is responsible for viral persistence following mother-infant transmission. This leading to high rates of chronic infections leading to liver cirrhosis and HCC (Chisari *et al.*, 2009).

Multifactorial mechanisms contribute to development of HCC is chronic HBV infection. Most tumors in HBV-associated HCC result from random integration of HBV DNA in host cell DNA resulting in down-regulation of cellular growth control mechanisms. Also, certain HBV proteins may directly participate in HCC development. For example, HBV x gene product transactivates cellular genes that control cellular growth (Chisari *et al.*, 2009).

## **2.7. Clinical significance**

HBV is found in highest concentrations in blood and in lower concentrations in other body fluids (e.g., semen, vaginal secretions, and wound exudates). The incubation period from the time of exposure to onset of symptoms is 6 weeks to 6 months. HBV infection can be self-limited or chronic (CDC, 2012).

The clinical course of acute hepatitis B is indistinguishable from that of other types of acute viral hepatitis. The incubation period ranges from 45 to 160 days (average, 120 days). Clinical signs and symptoms occur more often in adults than in infants or children, who usually have an asymptomatic acute course. However, approximately 50% of adults who have acute infections are asymptomatic. Most acute HBV infections in adults result in complete recovery with elimination of HBsAg from the blood and the production of anti-HBs, creating immunity to future infection (CDC, 2012).

Chronic infection with hepatitis B virus either may be asymptomatic or may be associated with a chronic inflammation of the liver (chronic hepatitis), leading to cirrhosis over a period of several years. This type of infection dramatically increases the incidence of hepatocellular carcinoma (El-Serag and Rudolph, 2007).

Chronic carriers of HBsAg may or may not have demonstrable evidence of liver disease. Persistent (unresolved) viral hepatitis, a mild benign disease that may follow acute hepatitis B in 8–10% of adult patients (Geo *et al.*, 2010).

## **2.8. Laboratory diagnosis**

Diagnosis is based on clinical and laboratory findings. It is impossible to differentiate HBV infection on clinical ground alone; so, differentiate diagnosis should be established on the results of laboratory testing. Both serological and molecular methods are available and used to distinguish between acute and chronic infections (CDC, 2012).

### **2.8.1. Specimens**

Serum or plasma can be used for the detection of serologic and molecular markers of HBV infection. Plasma is separated from blood collected in containers with EDTA or citrate dextrose as anticoagulant. Heparinized plasma is unacceptable for nucleic acid analysis as heparin interferes with Taq polymerase in PCR (Hovart and Tegtmeier, 2011).

## **2.8.2. Direct detection**

### **2.8.2.1. Antigen detection**

Several laboratory assays are available to detect HBV antigens in the patient serum after infection. In general, HBV antigens are detected using highly sensitive techniques that used either solid-phase immune assays or microparticles to capture the protein (Hovart and Tegtmeier, 2011). By definition, HBsAg persist for more than 6 months in the presence of HBe Ag or anti-HBc antibodies (Hovart and Tegtmeier, 2011).

### **2.8.2.2. Nucleic acid detection**

Detection and / or quantitation of HBV DNA are useful in the initial characterization of HBV infection and monitoring of chronic infection, especially in patients on antiviral therapy. Many of the assays that detect HBV DNA use oligo primers that recognize a conserved sequence within the HBV precore / core gene. Conventional PCR and real-time PCR are commonly used to detect and quantify HBV DNA, respectively (Hovart and Tegtmeier, 2011).

### **2.8.2.3. Serological tests**

Several commercial assays are available to detect HBV-specific antibodies, which determine the stage of the disease and immunity due to vaccination (Hovart and Tegtmeier, 2011).

IgM antibody to HBcAg (IgM and anti-HBc) persists for several weeks to months, and its presence indicates recent, acute infection of less than 6 months duration (Hovart and Tegtmeier, 2011).

Also, its absence does not rule out of chronic infection. After IgM anti-HBc Ag disappears, total antibody to HBcAg remains positive indefinitely and is the best serological marker for documenting post infection with HBV. This marker should not be present in vaccinated individuals unless they were infected with HBV prior to vaccination (Hovart and Tegtmeier, 2011).

A positive anti-HBcAg total may indicate an acute (HBsAg positive, IgM anti-HBc positive, resolved HBsAg negative). A positive anti-HBsAg result is indicative of immunity to HBV as a result of resolved infection, or from effective vaccination, furthermore, this marker is used to monitor vaccine success. Both WHO and the CDC recognize levels of 10 mIU/ml of anti-HBs Ag as protective. Presence of anti-HBe Ag indicates the resolution of acute infection and is associated with a decrease in viral replication. Patients who have recovered from acute HBV infection will have detectable anti-HBe Ag, anti-HBc Ag and anti-HBs Ag (Hovart and Tegtmeier, 2011).

## **2.9. Prevention of HBV infection**

Viral hepatitis is preventable with effective vaccines, which is available since 1982 and have proven safe to both adults and children but its use among HCW in the developing world is low (Dannetun *et al.*, 2006; Abdhahah *et al.*, 2010).

Any healthcare worker who performs tasks involving contact with blood, blood-contaminated body fluids, other body fluids, or sharps should be vaccinated or have serologic evidence of immunity due to natural infection (Alter, 2005).

Blood-borne infections have been recognized as an occupational hazard for nearly 50 years (Alter, 2005). However, it is only in the last 20 years that there has been a widespread recognition of the specific risk posed to health care workers by blood-borne viruses such as hepatitis B virus (HBV), hepatitis C virus (HCV) and human immunodeficiency virus (HIV) (Alter, 2005).

## **2.10. Treatment**

Acute hepatitis B infection does not usually require treatment and most adults clear the infection spontaneously. Early antiviral treatment may be required in fewer than 1% of people, whose infection takes a very aggressive course (fulminant hepatitis) or who are immunocompromised. On the other hand, treatment of chronic infection may be necessary to reduce the risk of cirrhosis and liver cancer (Hollinger and Lau, 2006).

Although none of the available drugs can clear the infection, they can stop the virus from replicating, thus minimizing liver damage. As from 2008, there are seven medications licensed for treatment of Hepatitis B infection in the United States antiviral drugs Lamivudine (Epivir), Adefovir (Hepsera), Tenofovir (Viread), Telbivudine (Tyzeka) and Entecavir (Baraclude), and the two immune system modulators interferon Alpha-2a and PEGylated Interferon Alpha-2a (Pegasys)(Albert and Caporaso, 2011).

The use of interferon, which requires injections daily or thrice weekly, has been substituted by long-acting PEGylated interferon, which is injected only once weekly (Albert and Caporaso, 2011).

### **2.11. Previous study**

Many studies have been published in the last two decades addressing various aspects of HBV infection in Sudan, such as its prevalence among blood donors, health care workers, pregnant women, virus genotypes and its relation to hepatocellular carcinoma. These studies indicate that the disease is endemic in the Sudan and of major public health importance (Elduma and Saeed, 2011).



Hospital personnel are often victims of occupational injuries by contaminated sharps or projection of contaminated fluids into mucous membranes. Therefore, several studies for screening of HBV among hospital personnel were conducted in Khartoum State hospitals. In a study was done to detect the seroprevalence of HBV markers among HCWs in public teaching hospitals in Khartoum State, they found that 6% were HBsAg seropositive (Elmukashfi *et al.*, 2012).

Another study was conducted among HCWs in Khartoum HBsAg was detected in 4.4 % and the study proved that it was high in males (Abdalwahab and Nafi, 2014). In a study for Seroprevalence of Hepatitis B and C among HCWs in Omdurman, Sudan; the occupation risk of HBV infection among the HCW in this study was high for the nurses and cleaning staff (Nail *et al.*, 2008). In study conducted during November 2007 on the HCW of Tropical Diseases Teaching Hospital in Omdurman city, central Sudan. The seroprevalence of HBV among HCW of Tropical Diseases Teaching Hospital in central Sudan was 2.4% (Elmukashfi *et al.*, 2012).

There is evidence among some groups of HCWs, such as dentists, that rates of exposure are decreasing over time, temporally associated with increased awareness and compliance with the practice of standard precautions (Beltrami *et al.*, 2000).

Hepatitis B is a well documented occupational hazard for HCWs, including both laboratory and nursing personnel (Sheikh *et al.*, 2007).

Apart from lack of hepatitis B vaccination, nurses and non-professional staff on their own were found to be significantly more susceptible to HBV infection than others (Shrestha and Bhattarai, 2006). In Sana, Yemen the prevalence in HCWs was 9.9% (AlHurabiet *al.*, 2004). Among HCWs in Korea another study among HCWs the prevalence was 2.4% (Shin, 2006).

## **CHAPTER THREE**

### **MATERIALS AND METHODS**

#### **3.1 Study design**

##### **3.1.1. Types of study**

This is a descriptive cross- sectional study conducted to detect Hepatitis B virus infection among hospital personnel in Khartoum State hospitals.

##### **3.1.2. Study area**

Hospital personnel working in Omdurman Teaching Hospital, AL-Naow Teaching Hospital, AL-Dosogi specialized Hospital, Khartoum Teaching Hospital, Dar-ELelage Hospital and Omdurman Military Hospital were sampled. The practical part of this study was carried out in the Research Laboratory, College of Medical Laboratory Science, Sudan University of Science and Technology.

##### **3.1.3. Study duration**

The study was conducted during the period from January to May 2015.

##### **3.1.4. Study population**

Hospital personnel working in Khartoum State hospital.

### **3.2. Sample size and Sampling technique**

A total of ninety (n=90) hospital personnel were enrolled in this study. Five ml of blood samples were collected.

### **3.3. Ethical consideration**

Approval to conduct this study was obtained from the College Ethics Committee of the Sudan University of Sciences and Technology (SUST). After explaining the study and its goal, a verbal consent was taken from the study recruits before proceeding with the study and collecting blood samples.

### **3. 4. Sample collection**

Blood samples were collected from each person (hospital personnel) after their consented. The venipuncture technique were used for collection; the suitable vein was located, then skin was cleaned by 70 %(v/v) ethanol, sterile syringe (5 ml) was used to collect 5 ml of blood, then the blood was dispensed in a sterile plain blood container (without anticoagulant).

### **3.5. Laboratory work**

#### **3.5.1. Preparation of specimens**

Blood samples were allowed to clot and then centrifuged at 3000 rpm for 5-10 minutes to obtain serum. Then, obtained sera were preserved at -20 °C until the serological analysis.

#### **3.5.2. Sample analysis**

The samples were analyzed for the presence of HBsAg by a commercially available enzyme -linked immunosorbent assay “HBsAg ELISA” kit (Fortress Diagnostics Limited, unit 2C Antrim technology park, Antrim, BT4I IQS United Kingdom).The assays were performed following the instructions of the manufacturer. Positive and negative controls were included in each assay. According to the information included in the kit’s insert, the immunoassay used has specificity 99.94%.

### **3.5.3. Principle of the assay**

The test is an enzyme-immunoassay based on a sandwich principle. Polystyrene micro titer strip wells have been coated with monoclonal anti-HBs (antibody to HBsAg). Patients' serum sample is added to the micro wells. During incubation, the specific immune-complex formed in the case of presence of HBsAg in the sample, is captured on the solid phase. After washing to remove sample serum proteins, second antibody conjugated to the enzymes HRP and directed against different epitope of HBsAg is added to the wells. During the second incubation steps, these HRP conjugated antibodies will be bound to any anti-HBs-HBsAg complexes previously formed during the first incubation, and the unbound HRP conjugate is then removed by washing. After washing to remove unbound HRP conjugate, chromogen solutions containing TMB and urea peroxidase are added to the wells. In the presence of the antibody-antigen-antibody HRP sandwich immune-complex, the colorless chromogens are hydrolyzed by the bound HRP conjugate to a blue colored product. The blue color turns yellow color after stopping the reaction using the stop solution. The colour was read as optical density in order to determine the result of the test. Wells containing samples negative for HBsAg remain colorless.

#### **3.5.4. Procedure**

All reagents and specimens were settled to reach room temperature, 20ul of specimen diluent was added to each well except the blank, then 100ul of positive control, negative control and specimen were added to their respective wells. The plate was covered with plate cover and incubated for 60 minutes at 37°C. At the end of incubation period, 50ul of HRP-conjugate was added to each well except the blank; the plate was covered and incubated for 30 minutes at 37°C. By the end of incubation period each well was washed 5 times with diluted wash buffer. Finally 50ul of chromogen A and chromogen B solutions were added to each well including blank, then the plate was incubated at 37°C for 15 minutes and stop solution was added.

#### **3.5.5. Quality control and calculation of the results**

Reagent, standard and control were checked for storage, stability and preparation before starting work. Each microplate was considered separately when the results was calculated and interrelated; the results were calculated by relating each specimen absorbance (A) to the cut off (c.o.) of the plate.

Calculation of cut off value (C.O.) =  $NC \times 2.1$  (NC is mean of the three negative controls).

The OD value of the blank well must be less than 0.080 at 450nm.

The OD value of the positive control must be more than 0.80 at 450nm.

The OD value of the negative control must be less than 0.1 at 450 nm.

### **3.5.6. Interpretation of results**

Positive more than cut of value.

Negative less than cut of value.



## **CHAPTER FOUR**

### **RESULTS**

A total of 90 hospital personnel were participated in this study. Blood samples were collected from hospital personnel working in Omdurman Teaching Hospital, AL-Naow Teaching Hospital, AL-Dosogi specialized Hospital, Khartoum Teaching Hospital, Dar-ELelage Hospital and Omdurman Military Hospital (Table 1). Males were predominant 59 (65.6%); while females were 31(34.4%) (Table 2).Considering the occupation of the study subjects, the most frequent were laboratory technicians 55 (66.1%), followed by nurses20 (22.2%) and 15 (16.6%) physician (Table 3).

Only 16(17.8%) of the study subjects were positive for HBsAg, while 74 (82.2%) were negative (Table 4). Among the positive HBsAg subjects laboratory technologist had the highest frequency of the infection 13 (14.4%), followed by nurses 3(3.3%) (Table5).

Among hospital personnel, males more infected than females, 14 (15.6%) and 2 (2.2%) respectively (Table 6). Only 25 (27.8%) participant had had full dose of hepatitis B vaccination, while 65 (72.2%) had no history of vaccination (Table 7).

**Table1. Distribution of hospital personnel according to hospital.**

Hospitals	Frequency	%
Omdurman Teaching Hospital	30	33.3
AL-Naow Teaching Hospital	20	22.2
AL-DosogiEspicialized hospital	14	15.6
Khartoum Teaching Hospital	12	13.3
Dar-ELelage	9	10.0
Omudurman Military Hospital	5	5.6
Total	90	100.0

**Table2. Number and percentage of participant according to the gender.**

Gender	Frequency	%
Male	59	65.6
Female	31	34.4
Total	90	100.0

**Table3. Number and percentage of samples according to occupation.**

Occupation	Frequency	%
Physicians	15	16.7
Nurses	20	22.2
Laboratory technicians	55	61.1
Total	90	100.0

**Table4. Number and percentage of seropositivity of HBsAg among hospital personnel**

Results	Frequency	%
Negative	74	82.2
Positive	16	17.8
Total	90	100.0

**Table5. Detection of HBsAg according to occupation**

Occupation		Sample		Total
		Negative	Positive	
Physicians	Count	15	0	15
	% of Total	16.7%	0	16.7%
Nurses	Count	17	3	20
	% of Total	18.9%	3.3%	22.2%
Laboratory Technologists	Count	42	13	55
	% of Total	46.7%	14.4%	61.1%
Total	Count	74	16	90
	% of Total	82.2%	17.8%	100%

**Table6. Detection of HBsAg according to gender**

Gender		Sample		Total
		negative	positive	
Male	Count	45	14	59
	% of Total	50.0%	15.6%	65.6%
Female	Count	29	2	31
	% of Total	32.2%	2.2%	34.4%
Total	Count	74	16	90
	% of Total	82.2%	17.8%	100.0%

**Table 7. Detection of HBsAg according to vaccination status**

Vaccination status		Sample		Total
		Negative	positive	
Yes	Count	25	0	25
	% of Total	27.8%	.0%	27.8%
No	Count	49	16	65
	% of Total	54.4%	17.8%	72.2%
Total	Count	74	16	90
	% of Total	82.2%	17.8%	100.0%

## CHAPTER FIVE DISCUSSION

### 5.1. Discussion

Hospital personnel are often victims of infection by nosocomial pathogens like HBV. Blood exposure incidents pose a risk for transmission of blood borne pathogens for both hospital personnel and public health. The present study aimed at detection of HBV among hospital personnel in Khartoum State hospitals.

The study revealed that the prevalence of HBsAg among hospital personnel was 17.8%. This result is higher to that obtained by Djeririet *al.*, (2008) and Shin, (2006) who reported that prevalence of hepatitis B among Moroccan and Korean health care workers was 1% and 2.4% respectively.

This may be due to difference in implementation of universal precautions and lack of awareness of personal prophylaxis and probably no or little vaccination option available to Sudanese hospital personnel.

The seropositivity of HBsAg in this study was more among males than females. Similar result was found in a study conducted among HCWS at Omdurman and Wad Madani Teaching hospital (Nail *et al.*, 2008; Gasmelseed *et al.*, 2013).

In the present study the occupational risk of HBV infection among hospital personnel was reported to be highest among the laboratory technicians, followed by nurses. The same result was found in a study conducted among HCWCs from different hospital in Khartoum done by Abdalwhab *et al.*, (2014).



This may be due to technicians and nurses were at high risk of HBV infection as they interact first-hand with the patients. Prevalence of HBV might associate with their low socioeconomic status as it is the disease of poverty. Similar to Sarwar *et al.*, (2008) none of the physicians were found positive, but not in accordance to another study done by Belo, (2000) who reported that among HCWs, surgeons/physicians have the highest risk of HBV infection from their patients .

In a study conducted to evaluate vaccination coverage among HCWCs in Pakistan, 65% of the participant had received a full dose of hepatitis B vaccine (Memon *et al.*, 2007). This is lower to our finding, only 27% of the participant had received a full dose of hepatitis B vaccine.

This study also indicated that in Sudan, the surveyed hospitals did not have a policy for hepatitis B vaccination. In fact, vaccination for HBV was obtained by the individuals themselves independent of the hospitals.

## **5.2. CONCLUSION**

The study concluded that the prevalence rate of HBV among hospital personnel in Khartoum State was moderate. Laboratory technicians had the highest frequency of infection followed by nurses.

### **5.3. RECOMMENDATIONS**

- To eradicate the infection among hospital personnel a vaccination program must be set place by the Ministry of Health for all of HCWs, Health care facilities must be improved, seminars and workshops on laboratory safety and laboratory management should be established by the Ministry of Health.
- Hospital personnel should be considered as a matter of policy. For example vaccination against HBV infection could be made mandatory for preclinical medical and nursing students.
- The Ministry of Health could consider offering subsidized or free Hepatitis B vaccination to HCWs. In addition education on infection control and other strategies for infection control need to be strengthened.

## REFERENCES

1. **Abdalwahab M and Nafi M.** (2014). Sero-Frequency of Hepatitis B infection among HCWs in Khartoum. *American Journal of Research Communication.* **2 2(12):** 148-154.
2. **Abdhalah K, Josephine B, Alice N, Caroline Wand Harriet M.** (2010). Seroprevalence and risk factors for hepatitis B virus infection among health care workers in a tertiary hospital in Uganda. *BMC Infectious Diseases.***10:**191.
3. **Albert A, and Caporaso N.** (2011). HBV therapy guidelines. *ItaGastroentrol.* **43(1):** 57-63.
4. **Al Huraibi MA, Shidrawi R, Alhaimi MA, Dayton R and Lyon MI.** (2004). Seroprevalence of markers of viral hepatitis in Yemen health care workers. *JMV.* **73(4):**562–565.
5. **Alter MJ.** (2005).The epidemiology of hepatitis B virus infection in healthcare workers in the West and Asia. *Hep B Annual.***2(1):**186-192.
6. **Belo AC.** (2000). Prevalence of hepatitis B virus markers in surgeons in Lagos, Nigeria. *East Afr Med J.***77(5):** 283-285.

7. **Beltrami EM, Williams IT, Shapiro CN and Chamberland ME.** (2000). Hazard and Management of Blood-Borne Infections in HCWs. *CMR*.**13(3)**: 385–407.
8. **Buddeberg F, Schimmer BB and Spahn DR.** (2008)."Transfusion-transmissible infections and transfusion-related immunomodulation".*Best Practice & Research. Clinical Anaesthesiology***22 (3)**: 503–17.
9. **CDC.**(2012).Epidemiology and Prevention of Vaccine-Preventable Diseases.Vaccines and Immunizations. P24.
10. **CDC.** (2013).CDC Guidance for evaluating health-care personnel for hepatitis B virus protection and for administering post exposure management. Morbidity and mortality weekly report.**62(10)**.
11. **Chisari FV, Isogawa M and Vieland SF.** (2009).Pathogenesis of hepatitis B virus infection.*Pathol Biol*.**58(4)**: 258-266.
12. **Chu CJ, Keeffe EB, Han SH, Perrillo RP, Min AD, Soldevila PC, Carey W, Brown RS, Luketic VA, Terrault N and Lok A.** (2003). Hepatitis B virus genotypes in the United States: results of a nationwide study.*Gastroenterology*.**125**:444-451.
13. **Dannetun E, Tegnell A, Torner A and Giesecke J.** (2006).Coverage of hepatitis B vaccination in Swedish healthcare workers.*J Hosp Infect*.**63**:201-204.

14. **Djeriri K, Laurichesse H, Merle L, Charof R, Abouyoub A, Fontana L, Benchemsi N, Elharti E, Aouad R, Chamoux A, and Beytout J.** (2008). Hepatitis B in Moroccan health care workers. <http://occmmed.oxfordjournals.org>.
15. **Elduma HA and Saeed SN.**(2011). Hepatitis B virus infection among staff in three hospitals in Khartoum, Sudan. *Eastern Mediterranean Health Journal*. **17(6)**:474-478.
16. **Elmukashfi TA, Elkhidir I M, Ibrahim O A, Bashir A A, Awad Elkarim M A.** (2012). Hepatitis B virus infection among health care workers in Public Teaching Hospitals in Khartoum State, Sudan. *Safety Science*. **50(5)**: 1215–1217.
17. **El-Serag HB and Rudolph KL.** (2007). "Hepatocellular carcinoma: epidemiology and molecular carcinogenesis". *Gastroenterology*. **132 (7)**: 2557–76.
18. **Fairley CK and Read TR.** (2012). "Vaccination against sexually transmitted infections". *Current Opinion in Infectious Diseases*. **25 (1)**: 66–72.
19. **Franco E, Bagnato B, Marino MG, Meleleo C, Serino L and Zaratti L.** (2012). Hepatitis B: Epidemiology and prevention in developing countries. *World J Hepatol*. **4(3)**:74-80.

20. **Gasmelseed N, Elhaj EY, Elsir A, Mohammed ME and Saeed OK.** (2013). Screening of hepatitis B virus among health care workers in some Wad Medani hospitals. *KMJ*.**13(3):** 941-945.
21. **Geo F B, Karen C C, Janet B S, Stephen A M and Timothy A M.** (2010). Hepatitis Viruses .In: Jawetz, Melnick and Adelberg's Medical Microbiology, 25<sup>th</sup>ed, Appleton and lange (editors) .McGraw-Hill Companies. 434-455.
22. **GorarA, Zahid A and Imrana A.**(2014). Risk factors for bloodborne viral hepatitis in healthcare workers of Pakistan: a population based case–control study. *BMJ Open* .**4(7):**1110-1136.
23. **Habib U , Muhammad W, Asad U , Muhammad I ,Zobia I, Amjad A, Faisal J, Umar J, Muhammad S, Adnan A, Razi U and Afi U.** (2014). Seroprevalence of HBV infection and viral loads in outpatients attending adistrict hospital located in Mardan, Pakistan. *International Journal of Biosciences*. **4(5):**28-34.
24. **Hatim M Y.** (2008). Epidemiology of viral hepatitis in Sudan.*Clinical and Experimental Gastroenterology*.**1:** 9-13
25. **Hollinger F., and Lau D.** (2006). HBV pathway to recovery through treatment.*Clini J Gastro*.**35(4):** 895-931.

26. **Horvart RT and Tegtmeier GE.**(2011). Hepatitis B and D viruses. In: versalovic J, Carrol KC, Funke G, Jorgensen JH, Landry ML and Warnock DW (eds). *Manal of clinical microbiology*, 10<sup>th</sup> volume2, ASM Press, Washington DC, pp.1659-1676.
27. **Hou J, Liuz A and Guf M.** (2005).Epidemilogy and prevention of hepatitis B virus infection.*IntJMedsci.* **2(1)**:50-57.
28. **JakeTL.**(2009).Hepatitis B: The Virus and Disease. *NIHPAManuscripts.* 49(5):13-21.
29. **Jobran M A, Saeed A A, Ahmed A M, Awad A E and Ahmed M A.** (2014).Seroprevalence of hepatitis B and C virus infections among health students and health care workers in the Najran region, southwestern Saudi Arabia: The need for national guidelines for health students.*BMC Public Health.***14**:577.
30. **Kosgeroglu N, Ayranci U, Vardareli E and Dincer S.** (2004). Occupational exposure to hepatitis infection among Turkish nurses: frequency of needle exposure, sharps injuries and vaccination. *Epidemiol Infect.***132**:27-33.

31. **Laishram J, Keisam A, Phesao E, Tarao S, Laloo J and Sanayaima D.** (2013). Prevalence of needle stick injuries among nurses in a tertiary care hospital and their immediate response. *Nanotechnology Journal*. **3(4)**: 257-260.
32. **Memon MS, Ansari S, Nizamani R, Khathri NK, Mirza MA, Jafri W.** (2007). Hepatitis B vaccination status in health care workers of two university hospitals. *JLUMHS*. 48–51.
33. **Mohammad T Y, Rubina Qasim, Rehana Khalil, Mohammad F K, and Shafiq U R.** (2014). Hepatitis B vaccination among primary health care workers in Northwest Pakistan. *Int J Health Sci*. **8(1)**:67-76.
34. **Murray RP, Rosenthal SK, Kobayashi SG and Pfaller AM.** (2002). Hepatitis viruses. In: *Medical microbiology* 4<sup>th</sup> ed. Mosby, united state of America. Pp 591-605.
35. **Nail A, Eltiganni S and Imam A.** (2008). Seroprevalence of Hepatitis B and C among HCWs in Omdurman, Sudan. *SJMS*. 3(3): 201-206.
36. **Panessa C, Hill WD, Giles E, Yu A, Havard S, Butt G, Andonov A, Krajden M and Osiowy C.** (2008). Genotype D amongst injection drug users with acute hepatitis B virus infection in British Columbia. *J Viral Hepat*. **16**:64-73.



37. **Pereira LM, Martelli CM, Merchán-Hamann E, Montarroyos UR, Braga MC, de Lima ML, Cardoso MR, Turchi MD, Costa MA and Alencar LC.** (2009). Population-based multicentric survey of hepatitis B infection and risk factor differences among three regions in Brazil.*Am J Trop Med Hyg.***81**:240-247.
38. **Rachna B, Renu J, Krishan K, Ajay B, Nakshatra A and Dhole N.** (2008). Seroprevalence and risk factors for hepatitis B virus infection among general population in Northern India.*ArqGastroenterol.* **45(2)**: 137-140.
39. **Sarwar J, Gul N, IdrisM, Aris-ur-Rehman, Farid J, Adeel MY.** (2008) Seroprevalence of hepatitis B and hepatitis C in health care workers in Abbottabad. *J Ayub Med Coll Abbottabad.***20**: 27-29.
40. **Seeger C, Zoulim F and Masonws.**(2007). In: Kinpe DM and Howley PM (eds). *Field's virology, 5<sup>th</sup>ed, volume2.* Lippincott's Williams and Wilkins, Philadelphia, pp.2977-3028.
41. **Sheikh N H, Hasnain S, Majrooh, A, Tariq M, and Maqbool H.** (2007). Status of hepatitis B vaccination among the HCWs. *JPMA.*50: 239.
42. **Shin B.** (2006). Seroprevalence of HBV among HCWs in Korea. *JKMS.***21**: 58-62.
43. **Shrestha SK and Bhattarai MD.**(2006). Study of hepatitis B among different categories of healthcare workers. *JCPSP.***16(2)**: 108-11.

- 44. Washington C W, Stephen D, Wiliam M J, Elmer W K, Gary W P, Paul C S, Karayiannis P and Thomas HC.** (2009). Mahy BWJ, van Regenmortel MHV, ed. *Desk Encyclopedia of Human and Medical Virology*. Boston: Academic Press. p.110.
- 45. Williams R.** (2006).Global challenges in liver disease. *Hepatology (Baltimore, Md.)***44(3):**521-526.