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Seroprevalence of HBsAg among HIV Infected Sudanese Patient in Khartoum State

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Abstract

The rate of progression and complications from viral hepatitis are accelerated in patients with HIV co-infection.

The aim of this study is to determine the seroprevalence of HBsAg in HIV positive infected patient in Khartoum state. This is a cross-sectional study conducted at the VCT centers in Khartoum state during the period from January to May 2018. A total of 92 subjects were recruited to participate in this study.

Blood samples were collected from each patient. The samples were screened for HBsAg using ELISA (Acon Laboratories, Inc.), Results of this study showed a high prevalence of HBsAg infection (7.6%). HBsAg infection was higher among males than females, in the age group of 20-40 years and among patients on treatment.

Also, results show a significant association between abnormal Total protein (T. protein) and taking HIV treatment. The study concluded that there was a high prevalence rate of HBsAg among HIV infected patients in Khartoum state.

Keywords: Hepatitis; HBsAg; T. protein; Urinary tract infections

Introduction

Hepatitis is an infection of the liver caused by several viruses, the most common of which are hepatitis A, B, and C. Both Hepatitis B Virus (HBV) and Hepatitis C Virus (HCV) are spread mainly through contaminated blood and blood products, sexual contact and contaminated needles [1].

HBV and hepatitis C virus are hepatotropic virus whose primary replication occurs in the liver [2]. The natural history and clinical course of HBV differ from that of HCV infection. However, similarities may be drawn in the context of chronic disease and public health burden. Chronic infection by these viruses leads to slow progressive liver disease that over a period of up to 30 years may result in cirrhosis, chronic liver failure and Hepatocellular Carcinoma (HCC) [3].

Liver disease is the single greatest cause of non-AIDS-related death in patients with HIV disease, accounting for a greater proportion of deaths than non-AIDS-related cancers. HIV, HBV, and HCV infection share similar transmission routes and therefore co-infection is common. In patients co-infected with HIV plus HBV or HCV, fibrosis rates are accelerated compared with those infected with HBV or HCV alone, leading to faster progression to end-stage liver disease. Overall, the signs of end-stage liver disease in patients with HIV infection seem to be similar to the signs in patients without HIV infection, but survival after the first episode of decompensation is reduced [4].

Material and Methods

Methods

- **Study design:** This study is a cross-sectional study
- **Study area:** This study was carried out at the VCT center Omdurman teaching hospital at Khartoum State
- **Study population:** A total of 92 of HIV infected Sudanese patients were recruited in this study
- **Inclusion criteria:** Patients diagnosed as having HIV by using several diagnostic parameters including ELISA and PCR
- **Exclusion criteria:** Patients positive for HIV infection, but have other liver diseases
- **Sampling technique:** The study was based on a simple random selection technique during admission to VCT center at Omdurman teaching hospital
- **Data collection:** A questionnaire was designed to get informative data about the history of each patient these include (taking treatment, clinical situation of the patient, age, gender, residence, duration of illness and weight)
- **Sample size:** Calculation of the sample size for Simple Random Selection (SRS) was done based on the following Steven Sampson equation

$$n = \frac{M}{\left[\left(S^2 \times (M-1)\right) \div pq\right] + 1}$$

Where, n=sample size; M=confidence level; P=prevalence=50%=0.5; Q=1-P=1-0.5=0.5; s=degree of precision

According to Steven Sampson equation, the calculated sample size was 90 HIV patients.

- **Specimen collection:** Five ml of venous blood was collected into EDTA vacationers under aseptic technique from each patient under study after having his/her written consent to participate in this study. The specimens were centrifuged at 2000 rpm and the plasma separated into plain containers.
- **Data analysis:** The Statistical Package for Social Science (SPSS) version 21 was used for statistical analysis. The significance of difference was determined using the chi-square test.
- **Ethical consideration:** Permission to carry out this study was taken from Faculty of Medical Laboratories Sciences, Omdurman Islamic University. Permission for sample collection was taken from director at of preventive medicine, the ministry of health, Khartoum State. The verbal consent was taken from patients before the samples collection.

Assay Procedures of ELISA

Step 1: Reagent preparation

- Allow the reagent and samples to reach room temperature (18-30°C) for at least 15-30 min
- Check the wash buffer concentrate for the presence of salt crystals
- If crystals have formed in the solution, resolubilize by warming at 37°C until crystal dissolve
- Dilute the stock, wash buffer 1 to 20 with distilled or deionized water, use only clean vessels to dilute the buffer

Step 2: Numbered wells

- Set the strips needed in strip-holder and a sufficient number of wells included three negative control (e.g., B1, C1, D1), two positive control (e.g., E1, F1) and one Blank (e.g., A1); neither samples nor HRP Conjugate should be added into the blank well)
- If the results are determined by a used dual wavelength plate reader, the requirement for use of the blank well could be omitted
- Use only the number of strips required for the test

Step 3: Added sample diluent

- Add 20 µl of sample diluents to each well except the Blank, and mix by taped the plate gently

Step 4: Added sample

- 100 µl of the positive control, negative control and specimen were added into their respective wells
- Note: A separate disposable tip for each specimen, negative control, and positive control was used to avoid cross-contamination

Step 5: Incubation

- The plate was covered with plate cover and incubated for 60 min at 37°C
- Thermostat-controlled water tank use was recommended to assure the temperature stability and humidity during the incubation
- Also sometimes dry incubator was used and the door was not opened frequently

Step 6: Added HRP conjugate

- 50 µl HRP Conjugate was added to each well except the Blank and mixed by tapping the plate gently

Step 7: Incubation II

- The plate was covered with the plate cover and incubated for 30 min for 37°C as in Step 5

Step 8: Washing

- At the end of the incubation, the plate cover was removed and discarded
- Each well was washed 5 times with diluted wash buffer
- Each time, the microwells were soaked for 30-40 seconds
- After the final washed cycle, the plate was turned down onto blotting paper or a clean towel and tops it to remove any remainders

Step 9: Colouring

- 50 µl of chromogen A and 50 µl of chromogen B solution were dispensed into each well including the Blank and were mixed by tapping the plate gently
- The plate was incubated at 37°C for 15 min where the light was avoided
- The enzymatic reaction between the chromogen solution and the HRP-Conjugate produced a blue color in positive control and HBsAg positive sample wells

Step 10: Stopping reaction

- 50 µl stop solution was added using the multichannel pipette or was added manually into each well and mixed gently
- An intensive yellow color developed in positive control and HBsAg positive sample wells

Step 11: Measuring the absorbance

- The plate reader was calibrated with the blank well and the absorbance was checked at 450 nm.
- Also, the dual filter instrument was used and the reference wavelength was set at 630 nm
- The Cut-off value was calculated and evaluated the results
- Note: Read the absorbance within 5 min after stopping the reaction
- LFT test was done by using Full automated Mindray BS_200

Result

The present study was conducted to determine the seroprevalence of HBV among HIV infected patients who attended VCT centers in Khartoum State. A total of 92 HIV infected patients were included in this study (50 males and 42 females). The frequency of HBsAg among HIV positive Sudanese patients was 7.6%.

In term of gender, the seroprevalence of HBsAg was statistically insignificantly higher (P value=0.085) among male (12%) than female (2.4%) (Table 1).

Gender	Number of tested	HBsAg +ve		P value
		Frequency	%	0.085
Male	50	6	12	
Female	42	1	2	
Total	92	7	8	

Table 1: Distribution of HBsAg positive HIV patients according to gender.

HBsAg was insignificantly higher (P value=0.877) among the age group of 20-40 years 8%, while 7.1% among the age group of 40-72 years (Table 2).

Age	Number of tested	HIV/ HBsAg +ve		P value
		Frequency	%	0.877
Up to 40 years	50	4	8%	
More than 40 years	42	3	7.1%	
Total	92	7	7.6%	

Table 2: Age distribution of the HBsAg positive HIV patients.

HBsAg detection was statistically insignificantly higher (P value=0.878) among patients on treatment (18.8%) (Table 3).

Treatment	Number of tested	HIV/HBsAg +ve		P value
		Frequency	%	0.878
On treatment	78	6	7.7%	
Not on treatment	14	1	7.1%	
Total	92	7	7.6%	

Table 3: Distribution of HBsAg positive HIV infected patients according to treatment.

LFT (Albumin, T. protein, ALT and AST) was statistically insignificant (P value=0.261, 0.2, 0681, 0.259, respectively) among HBsAg positive patients (Table 4).

Parameters	HIV (Mean ± SD)	HIV/HBsAg +ve (Mean ± SD)	P value
Albumin	1.23 ± 0.42	1.42 ± 0.53	0.261
T. protein	1.67 ± 0.47	1.42 ± 0.53	0.2
ALT	1.09 ± 0.29	1.14 ± 0.37	0.681
AST	1.12 ± 0.33	1.28 ± 0.48	0.259

Table 4: Assessment of LFT among HBsAg positive HIV patients.

There was statistical significance (P value=0.045) between T. protein and taking HIV treatment (Table 5).

Parameters	Mean ± SD of HIV/HBV	Mean of HIV	P value
T. protein	1.42 ± 0.53	1.94	0.045
Albumin	1.71 ± 0.95	1.75	0.924
AST	1.28 ± 0.48	1.14	0.46
ALT	1.14 ± 0.37	1.11	0.826

Table 5: The effect of HIV treatment on liver status among HBsAg positive HIV patients.

Discussion

Out of the total number of HIV patients examined the seroprevalence HBsAg was 7.6%. A similar study conducted in Gabon reported 8.8% of the studied population positive for HBsAg [5]. Studies done in Nigeria revealed high seroprevalence of HBsAg (29.7% and 12.3% respectively) [6,7]. This might be due to smaller sample size, different population area, immunity, life style, and ethnicity.

In the present study, males showed insignificantly higher (P value=0.085) seroprevalence of HBsAg. This result disagreed with study done in Nigeria which showed a higher prevalence of this co-infection in men he hypothesized that male preponderance in HIV/HBV co-infection on probably result from the fact that generally, boys were more prone to aggressive sport and play that might result in injury with bleeding, predisposing more to horizontal HBV transmission [8,9].

In this study, the seroprevalence of HBsAg was insignificantly higher (P value=0.877) among the age group of 20-40 years. This is in agreement with a study done in Nigeria [10].

Also, this study showed a significant association (P value=0.045) between taking HIV treatment and abnormal (T. protein). This result in agreement with a study conducted in Tanzania which showed that HIV/HBV co-infection individuals were more likely to have liver fibrosis and cirrhosis than HBV-uninfected patients [11].

Conclusion

The study concluded that there was a low prevalence rate of 7.6% of HBsAg among HIV infected patients in the VCT center in Omdurman locality, Sudan.

In term of gender, the seroprevalence of HBsAg was statistically insignificantly higher (P value=0.085) among male (12%) than female (2.4%). HBsAg was insignificantly higher (P value=0.877) among the age group of 20-40 years 8%, while 7.1% among the age group of 40-72 years.

HBsAg detection was statistically insignificantly higher (P value=0.878) among patients on treatment (18.8%).

LFT (Albumin, T. protein, ALT and AST) was statistically insignificant (P value=0.261, 0.2, 0681, 0.259 respectively) among HBsAg positive patients.

There was statistical significance (P value=0.045) between T. protein and taking HIV treatment.

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