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Research Article

Assessment of viral load and liver enzymes levels among infected individuals with hepatitis B virus in Kassala State, Sudan

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Background: Hepatitis B Virus (HBV) is a major global health issue, affecting over 296 million people worldwide. This study aimed to assess the relationship between viral load and liver enzyme levels among HBV-infected individuals in Kassala State, Sudan.

Methods: A cross-sectional study was conducted from August to November 2024 in Kassala State. Data were collected from 50 HBV-positive individuals using a structured questionnaire. Viral load was measured via quantitative PCR, and liver enzyme levels (ALT, AST, and ALP) were analyzed using standard biochemical assays. Data analysis was performed using SPSS version 22.

Results: The study population consisted of 50% males and 50% females. Significant correlations were found between viral load and liver enzymes: ALT (r=0.833, p=0.000), AST (r=0.733, p=0.000), and ALP (r=0.586, p=0.000). Gender analysis revealed higher viral loads in females (p=0.044). No significant associations were observed between viral load and marital status (p=0.905), age (p=0.985), or duration of infection (p=0.960).

Conclusion: Elevated viral load was associated with increased liver enzyme levels, particularly in females. Routine viral load and liver enzyme monitoring are recommended to improve HBV management.

Keywords: *Hepatitis B Virus (HBV), Viral Load, Liver Enzymes, Sudan*

Introduction

Hepatitis B virus (HBV) is a significant global public health concern, with an estimated 296 million people living with chronic HBV infection as of 2019, leading to nearly 820,000 deaths annually (Tekeste et al., 2017; WHO, 2024). HBV is a partially double-stranded DNA virus belonging to the Hepadnaviridae family and primarily targets hepatocytes,

causing both acute and chronic liver diseases. Transmission occurs through percutaneous or mucosal exposure to infected blood and body fluids, with common routes including vertical transmission from mother to child, unprotected sexual contact, and unsafe injection practices (MacLachlan & Cowie, 2015). Chronic HBV infection is a leading cause of liver cirrhosis and hepatocellular carcinoma (HCC), accounting for approximately 50% of HCC cases worldwide (Schweitzer et al., 2015). A number of variables, including age at infection, host immune response, and viral factors, might affect the natural course of an HBV infection. Perinatal or early childhood infections have a higher chance of turning into chronic illnesses; 90% of newborns infected with HBV are thought to develop chronic HBV, compared to less than 5% of adults (Croagh & Lubel, 2014). HBV is still widespread in many areas even with the availability of efficient vaccinations and antiviral treatments, especially in low- and middle-income nations with limited access to treatment and vertical transmission prevention initiatives (Lemoine et al., 2015). Monitoring the course of the disease and the effectiveness of treatment has been made easier by developments in molecular diagnostics, such as the measurement of HBV DNA (viral load). A vital indicator for determining the likelihood of liver damage and HCC as well as for directing therapy choices is viral load (Terrault et al., 2018). A vital biomarker for comprehending the replication activity and course of HBV infection is the hepatitis B virus (HBV) viral load, which is measured by the amount of HBV DNA in the blood. Since viral load evaluation is highly correlated with disease activity, risk of liver damage, and responsiveness to treatment, it is essential for the diagnosis, monitoring, and management of HBV (Terrault et al., 2018). Persistent high HBV DNA levels are associated with an increased risk of cirrhosis and hepatocellular carcinoma (HCC), highlighting its prognostic value in chronic HBV infection (Tseng et al., 2012). As the infection progresses, the interaction between the virus and the host immune system affects the levels of HBV DNA. Both normal liver function tests and high viral loads coexist during the immunological tolerance period, which is usually seen in perinatally infected patients. On the other hand, an active immune response during the immunological clearance phase causes hepatocyte damage, which raises liver enzymes and causes variable viral loads (Croagh & Lubel, 2014). Because of these dynamic variations, routine viral load monitoring is crucial for efficient clinical therapy. Polymerase chain reaction (PCR)-based assays and other developments in molecular diagnostics have made it possible to precisely quantify HBV DNA, which has improved our knowledge of viral dynamics and illness progression (Lampertico et al., 2017). Clinicians can use the classification of viral load into low, moderate, and high levels as a framework to evaluate the severity of the disease and choose the best course of treatment (Spengler et al., 2012).

Materials and Methods

A cross-sectional study conducted in Kassala City between August to September 2024. Fifty adults (aged ≥18 years) who tested positive for HBV by HBsAg and viral load were included in the study. Individuals co-infected with HCV or HIV or undergoing antiviral therapy was excluded. Whole blood samples were collected under hygienic conditions, and sociodemographic data were obtained via a structured questionnaire. Ethical approval was granted by Gharb University College, and informed consent was obtained from all participants. Data analysis was performed using SPSS version 22, applying descriptive and inferential statistics to assess relationships between viral load and other variables.

Results

The study included a total of 50 patients who tested positive for HBV by HBsAg and viral load. Among these, 50% were male and 50% were female. About 62% are married and 40% have high HBsAg viral load (Table 1). However, statistically significant variation in viral load between gender (Table 4, 8). Additionally, ALP statistically significantly difference between age groups (P = 0.000) (Table 5); ALT statistically significantly differences between age groups (P = 0.032) and viral load (P = 0.001) (Table 6); and AST statistically significantly difference between viral load (P = 0.001) (Table 7). HBsAg viral load significantly correlated with liver enzymes (positive correlation) (P = 0.000, 0.000, and 0.000 respectively) (Table 3).

Table 1. Sociodemographic characteristic of study population

Category	Range	Frequency	Percent %
Gender	Male	25	50.0
	Female	25	50.0
Marital Status	Single	19	38.0
	Married	31	62.0
Age	17 – 27 Years	18	36.0
	28 – 37 Years	18	36.0
	Above 37 Years	14	28.0
Duration	New case	8	16.0

	< 5Years	25	50.0
	≥ 5Years	17	34.0
Viral Load	Low (<2,000 IU/mL)	12	24.0
	Moderate (2,000–20,000 IU/mL)	18	36.0
	High (>20,000 IU/mL)	20	40.0

Table 2. Means of study parameters

	Viral load	**ALP**	**AST**	**ALT**
Mean	1037668.76	62.76	34.20	39.02
N	50	50	50	50
Std. Deviation	2392341.93	31.29	29.41	35.91
Minimum	190	28	10	12
Maximum	8870620	155	108	149

Table 3. Correlation between viral load and liver enzymes

Viral Load		
Parameter	**Correlation R**	**P. Value**
ALP	0.586**	**0.000**
AST	0.733**	**0.000**
ALT	0.833**	**0.000**

Table 4. Compare of viral load with sociodemographic data

Viral Load				
Category	**Groups**	**Number**	**Mean Rank**	**P. Value**
Gender	Male	25	21.34	**0.044**
	Female	25	29.66	
Marital Status	Single	19	25.18	0.905
	Married	31	25.69	
Age	17 – 27 Years	18	25.31	0.985
	28 – 37 Years	18	25.25	
	Above 37 Years	14	26.07	
Duration	New case	8	24.19	0.960
	< 5 Years	25	25.62	
	≥ 5 Years	17	25.94	

Table 5. Compare of ALP with sociodemographic data and viral load

ALP					
Groups	**Range**	**No**	**Mean**	**SD**	**P. Value**
Gender	Male	25	59.48	27.64	0.464
	Female	25	66.04	34.82	
Marital Status	Single	19	53.79	20.15	0.073
	Married	31	68.26	35.68	
Age	17 – 27 Years	18	56.44	22.43	**0.000**
	28 – 37 Years	18	49.00	15.00	
	Above 37 Years	14	88.57	41.27	
Duration	New case	8	83.38	47.26	0.126
	< 5 Years	25	59.04	31.75	
	≥ 5 Years	17	58.53	16.00	
Viral Load	Low (<2,000 IU/mL)	12	54.50	21.03	0.286
	Moderate (2,000–20,000 IU/mL)	18	58.94	21.62	
	High (>20,000 IU/mL)	20	71.15	41.47	

Table 6. Compare of AST with sociodemographic data and viral load

AST					
Category	**Range**	**No**	**Mean**	**SD**	**P. Value**
Gender	Male	25	32.64	27.83	0.712
	Female	25	35.76	31.41	
Marital Status	Single	19	28.42	23.60	0.281
	Married	31	37.74	32.32	
Age	17 – 27 Years	18	23.28	17.60	**0.032**
	28 – 37 Years	18	32.67	28.95	
	Above 37 Years	14	50.21	36.27	
Duration	New case	8	38.38	43.03	0.407
	< 5 Years	25	28.60	21.13	
	≥ 5 Years	17	40.47	32.72	
Viral Load	Low (<2,000 IU/mL)	12	26.83	24.46	**0.001**
	Moderate (2,000–20,000 IU/mL)	18	19.39	5.19	
	High (>20,000 IU/mL)	20	51.95	35.87	

Table 7. Compare of ALT with sociodemographic data and viral load

ALT					
Category	**Range**	**No**	**Mean**	**SD**	**P. Value**
Gender	Male	25	35.08	27.942	0.444
	Female	25	42.96	42.658	
Marital Status	Single	19	32.74	32.492	0.338
	Married	31	42.87	37.848	
Age	17 – 27 Years	18	25.11	21.573	0.109
	28 – 37 Years	18	44.44	45.328	
	Above 37 Years	14	49.93	33.591	
Duration	New case	8	36.00	35.270	0.314
	< 5Years	25	32.68	23.279	
	≥ 5Years	17	49.76	49.011	
Viral Load	Low (<2,000 IU/mL)	12	27.50	23.334	**0.001**
	Moderate (2,000–20,000 IU/mL)	18	22.61	12.059	
	High (>20,000 IU/mL)	20	60.70	45.240	

Table 8. Cross-tabulation between viral load range with sociodemographic data

Viral Load						
Category	**Groups**	**Low (<2,000 IU/mL)**	**Moderate (2,000–20,000 IU/mL)**	**High (>20,000 IU/mL)**	**Total**	**P. Value**
Gender	Male	8	12	5	25	**0.016**
	Female	4	6	18	25	
	Total	12	18	20	50	
Marital Status	Single	5	6	8	19	0.874
	Married	7	12	12	31	
	Total	12	18	20	50	
Age	17 – 27 Years	3	5	4	12	0.985
	28 – 37 Years	8	6	4	18	
	Above 37 Years	7	7	6	20	
	Total	18	18	14	50	
Duration	New case	2	6	4	12	0.893
	< 5 Years	4	8	6	18	
	≥ 5 Years	2	11	7	20	
	Total	8	25	17	50	

Discussion

The findings of this study highlight critical associations between viral load, liver enzyme activity, and sociodemographic factors in the study population (Table 1). The mean viral load among participants was high (1,037,668.76 copies/mL) (Table 2), with a broad range indicating variability among individuals. Elevated liver enzymes, including alkaline phosphatase (ALP), aspartate aminotransferase (AST), and alanine aminotransferase (ALT), were observed, consistent with the hepatic impact of viral infections (Manikat et al., 2024). The viral load was categorized as low (<2,000 IU/mL) in 24% of participants, moderate (2,000–20,000 IU/mL) in 36%, and high (>20,000 IU/mL) in 40%. This distribution underscores the significant proportion of individuals with high viral load, which is clinically concerning due to its association with greater disease severity and increased risk of complications (Spengler et al., 2012). A significant positive correlation was found between viral load and liver enzymes, with the strongest correlation observed for ALT ($r = 0.833$, $p = 0.000$). This is consistent with other studies that demonstrated the usefulness of liver enzymes, especially ALT, as accurate indicators for liver damage and inflammation in viral infections (Fan et al., 2017). The association between these enzymes and liver damage brought on by high viral replication rates is further supported by the associations with ALP ($r = 0.586$, $p = 0.000$) and AST ($r = 0.733$, $p = 0.000$) (Table 3).

Gender differences were evident in viral load, with females showing a higher mean rank than males ($p = 0.044$) (Table 4). This finding may indicate gender-based variations in immune response or viral replication, as suggested in study by (Klein & Flanagan, 2016), which reported sex differences in immunity and susceptibility to viral infections. Marital status, age, and disease duration, however, did not show significant associations with viral load ($p > 0.05$), suggesting these factors may play a less direct role in influencing viral replication or liver enzyme activity in this population. A statistically significant association was observed between gender and viral load classification ($p = 0.016$). Notably, 72% of females had a high viral load compared to 20% of males, while males were more likely to have low or moderate viral loads. These findings align with previous studies indicating that hormonal and immunological differences between genders may influence viral replication and immune response (Emery et al., 2010). Factors such as estrogen modulation of immune activity in females and differences in viral receptor expression could explain this disparity. No significant association was found between marital status and viral load ($p = 0.874$). While marital status is often explored as a proxy for psychosocial stressors and access to healthcare, its lack of impact in this study suggests that viral load variability may be primarily influenced by biological rather than social factors (Haider et al., 2021). Age also did not show a significant relationship with viral load ($p = 0.985$). Although the age groups were relatively balanced, the lack of association may indicate that age-related immune senescence plays a minimal role in this context, particularly given the younger demographic of this study (median age below 40 years). This finding contrasts with studies in older populations, where age has been linked to higher viral loads and slower viral clearance (Busse & Mathur, 2010). The duration of diagnosis did not show a statistically significant association with viral load classification ($p = 0.893$). However, it is noteworthy that participants with a diagnosis duration of ≥5 years had a relatively higher proportion of moderate and high viral loads (56%), suggesting that chronicity may contribute to viral persistence or treatment resistance. These findings align with research highlighting the challenges of managing long-term infections and their potential impact on viral load dynamics (Spengler et al., 2012). The broad range of liver enzyme values and their associations with viral load underscore the importance of monitoring liver function in patients with high viral loads. Elevated liver enzymes are a marker of hepatic stress and can progress to significant complications if left unmanaged (Zheng wt al., 2023). Early detection and intervention are critical in mitigating long-term liver damage in affected individuals.

Conclusion

This study demonstrates a significant correlation between viral load and elevated liver enzymes (ALP, AST, and ALT), highlighting the impact of viral infections on liver function. The strong association between viral load and ALT underscores its utility as a key marker for hepatic inflammation and injury. Gender differences in viral load suggest potential variations in immune responses, with females showing higher viral replication rates. However, no significant associations were observed between viral load and marital status, age, or disease duration. These findings emphasize the need for regular monitoring of liver function in patients with high viral loads to prevent long-term complications.

Author contributions

All the authors made a substantial intellectual contribution, read and approved the final version of the manuscript, and agreed to be accountable for all aspects of the work.

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AI usage declaration

The authors have declared that no AI and associated tools were used to create scientific contents and interpretation of the results in this manuscript. But authors used Grammar or style correction tools such as Grammarly, ChatGPT and QuillBot were used to improve the language and readability of the manuscript.

Conflict of interest

The author declares no conflict of interest. The manuscript has not been submitted for publication in other journal.

Ethics approval

All study procedures were approved by the Researches and Ethics Committees (REC) of Ministry of Health, Gezira State, Sudan.

Consent to publish

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/ or national research committee and the Helsinki Declaration. Informed consent was written from each participant.

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