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Prevalence of Specific and Non-Specific Markers and Factors Associated with Hepatitis B Virus in Health Science Students and Healthcare Workers in the City of Conakry, Guinea

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Abstract

Introduction: Hepatitis B virus (HBV) is a DNA virus that causes inflammation of the liver. It is transmitted through unprotected sexual intercourse, contact with infected blood, and vertically from mother to child. The risk of infection is very high in healthcare settings, especially for under-equipped and less experienced staff. The objective of this study was to determine the prevalence of specific and non-specific markers and factors associated with hepatitis B among health science students and healthcare personnel in the city of Conakry. **Methods:** This was a prospective cohort study of staff at national hospitals and municipal medical centers, as well as health science students at public and private universities and health schools in the city of Conakry, covering 24 institutions. The data collected included sociodemographic information and 2 ml of blood in a tube without anticoagulant, which was tested for specific and non-specific markers were detected using the chemiluminescence method with the Architect 1000 i SR analyzer (Abbott Industries) and non-specific markers using the URIT 8021A analyzer, which uses a monochromatic light source to measure the absorbance of light passing through a reaction mixture

containing the sample and reagents at the immuno-serology laboratory of the National Institute of Health. Statistical analyses were performed using R software version 4.4.0. The p-value was calculated to determine the existence of a statistically significant link between the study variables. **Results:** A total of 3,041 healthcare workers and students participated in the study, of whom 257 were HBsAg carriers (8.5%). All of these HBsAg-positive participants also carried total anti-HBc antibodies, 2.3% were HBeAg carriers, and 1.2% carried anti-HBs antibodies. **Conclusion:** The prevalence of hepatitis B among healthcare workers and health science students, comparable to that of the general population, reveals a striking reality: hepatitis B knows no professional boundaries.

Keywords

Hepatitis B, Health Science Students, Healthcare Personnel, Conakry, Guinea

1. Introduction

The hepatitis B virus (HBV) belongs to the genus Orthohepadna of the family Hepadnaviridae and has a unique replication strategy in which it replicates its 3.2 kb DNA genome using an RNA intermediate via reverse transcription [1]. HBV infects hepatocytes to cause liver disease in the form of acute or chronic infection. HBV was classified by a physician, MacCullum, as responsible for serum hepatitis in 1947 transmitted through blood [2]. However, the discovery of an antigen associated with the virus occurred in an Australian Aboriginal patient while studying protein polymorphisms and was named the Australia antigen by Dr. Baruch S. Blumberg in 1965 [2] [3]. In 1970, the complete infectious virus was described by Dane using electron microscopy [4]. The virus completes its life cycle in the host hepatocyte, and its tropism is limited to humans, chimpanzees, and tupaia (tree shrews) [5]. The hepatitis B virus causes acute or chronic infection in humans, with long and variable incubation periods ranging from 8 weeks to 6 months [6].

HBV infects hepatocytes to cause liver disease in the form of acute or chronic infection. HBV was classified by a physician, MacCullum, as the cause of serum hepatitis in 1947, transmitted by the acute infection can be characterized by the presence of HBV surface antigen (HBsAg), secreted viral protein (HBeAg), and alanine and aspartate aminotransferase in serum. This is followed by the appearance of antibodies against the core antigen of HBV (HBcAg), followed by HBeAg and HBsAg in the serum, which helps the patient recover and eliminate the HBV infection [7]. Acute infection remains asymptomatic in many patients, while others experience symptoms such as nausea and hepatitis [8]. Chronic infection develops similarly to acute infection, but the patient does not recover from HBV infection because high levels of HBV DNA and HBsAg in serum persist long after exposure to HBV [9]. The risk of chronic HBV infection is higher in infants infected during the perinatal period and in the elderly [10].

The HBV life cycle begins when the virus binds to its receptor on the surface of hepatocytes. This receptor remained undefined for HBV for a long time, but it has recently been observed that the sodium taurocholate cotransporter polypeptide (NTCP) acts as one of the receptors for HBV [11]. Hepatitis B virus is mainly transmitted through blood (needlestick or wound injuries with contaminated blood, transfusions, transplants), unprotected sexual intercourse, and from mother to child during childbirth) [12]. Mother-to-child transmission remains the major cause of continued endemicity in areas of high prevalence (more than 8%), including sub-Saharan African countries [13]. HBV infection increases the risk of premature death from hepatocellular carcinoma (HCC) or cirrhosis by 15 to 25% [14]. Worldwide, 2 billion people have been exposed to the virus and 400 million are chronic carriers [15].

Africa, particularly sub-Saharan Africa, with an estimated prevalence rate of between 8% and 18%, is a highly endemic area [16].

In Guinea, no nationwide study has been conducted to determine the national seroprevalence of viral hepatitis. However, a few fragmented studies conducted over the past two decades show that HBsAg prevalence ranges from 8% to 16% [17].

A study conducted by a dermatology team at Donka University Hospital on HIV/HBV co-infection recorded 8.49% of 306 HIV-positive cases [18].

Healthcare professionals are at a much higher risk of HBV infection than the general population due to their exposure to blood and bodily fluids.

The results can be used as a basis for establishing or updating routine vaccination and booster programs for health students and healthcare workers.

They can also inform national hepatitis control strategies (needlestick injury prevention standards, screening and follow-up protocols) by providing recent local data from this study.

Few Guinean studies simultaneously document health science students and healthcare workers, even though these two groups are at the heart of the healthcare system and potential transmission.

By targeting these populations and specifically analyzing the risk factors for transmission and the control of these risks, our work fills a gap in the national literature and complements the data produced in other countries in the sub-region.

The objective of this study is to contribute to improving knowledge, protecting health, and controlling HBV transmission risk factors among health science students and healthcare workers in the city of Conakry.

2. Materials and Methods

2.1. Study Setting

This was a cross-sectional observational study of health science students at public universities (medicine, pharmacy, dentistry, laboratory technology) and private universities (Source, Koffi Annan), health schools in Conakry (Nelson Mandela, King Hassan II, Dabompa Professional Institute for Health Worker Training),

health personnel from three national hospitals, and municipal medical centers in the city of Conakry. The study population consisted of all students and health personnel present in the targeted facilities (24 sites) at the time of our visit.

Data collection consisted of administering a questionnaire covering the socio-demographic characteristics of the participants, after which a capillary blood sample was collected for HBV surface antigen testing using a rapid test. Next, at least 2 ml of whole blood was collected in a dry tube ordinary without anticoagulant and transported to the National Institute of Public Health laboratory for storage at −80˚C after centrifugation and decantation, followed by biological analysis after one month using the chemiluminescence method with the Architect®1000 i SR automated system (Abbott Industries) and non-specific markers with the URIT 8021A®, which uses a monochromatic light source (halogen lamp) to measure the absorbance of light passing through a reaction mixture containing the sample and reagents. The variation in this absorbance is correlated with the concentration of the target enzyme (GOT or GPT) via a specific enzymatic reaction.

2.2. Methods

Data collection:

We collected socio-demographic data from all participants (gender, age, level of education, occupation, residence). Next, a 2 ml blood sample was taken from a vein into a dry tube. The AgHbs were immediately tested using a rapid immunochromatography diagnostic test. The samples were transported to the INSP immuno-serology laboratory and then decanted for storage at −80˚C until biological analysis.

Sampling and size:

To calculate this size, we applied Schwartz's formula.

This calculation ensured that the selected sample (499 people) would be large enough for the estimate of the proportion to have acceptable statistical accuracy, with a 95% probability that the true proportion would be within ±5% of the estimate obtained.

Biological analysis of specific and non-specific markers of the hepatitis B virus:

The samples were removed from the freezer and left at room temperature for 2 hours to thaw. We then centrifuged them at 2000 rpm for 10 minutes. All samples were analyzed using the Architect i 1000SR chemiluminescence immunoassay (CMIA) for specific markers (AgHbs, Ac-antiHbc, AgHbe, Ac-antiHbs), the principle of which consists in labeling antibodies with chemiluminescent compounds capable, in the presence of acridinium carboxamide, of producing light in proportion to the antigen concentration. In practice, monoclonal antibodies directed against HBsAg are attached to magnetic microparticles and incubated with the patient's serum. Monoclonal antibodies directed against HBsAg labeled with acridinium carboxamide are added to the reaction medium. The wells of the microplate are exposed to a magnetic field, which separates the microparticles from the

antibodies. The solution is then alkalized, which induces light emission by the chemiluminescent compound. The light measured is proportional to the concentration of HBsAg in the solution. The test calculates the result based on the E/VS ratio. E/VS = URL (Reduced Light Unit) of the Sample/URL Threshold Value. Samples with an E/VS value below 1.00 are considered negative. Samples with an E/VS value greater than or equal to 1.00 are considered reactive and the non-specific parameters (GOT and GPT) have been analyzed by the URIT 8021A biochemical analyzer, the procedure for which is as follows: Initialization: The machine mechanism starts up and performs instrument checks. Cleaning the cuvettes: An eight-step washing system automatically cleans the 120 cuvettes used for reactions, unless the "clean before test" option is not selected. Reagent aspiration: The machine moves the clean cuvette under the reagent aspiration position. The aspiration arm descends into the reagent bottle and draws the exact amount, then injects it into the cuvette. Preheating: The reagent in the cuvette is preheated for several cycles. Sample aspiration: The aspiration arm moves to the tube or cup containing the sample, aspirates the defined amount, and then injects it into the cuvette. Mixing: The cuvette containing the reagent and sample is moved to the mixing position where a stirrer homogenizes the solution. Multiple reagent management (if applicable): Constant speed mode: After a certain amount of time, the cuvette is rotated to a position for aspirating and injecting a second reagent, followed by mixing. Mixed mode: After the incubation time for the first reagent, the second reagent is aspirated and injected, and then the solution is mixed. Photometric measurement: During the process, the cuvette is automatically moved to the light detection position where the machine measures the absorbance at different wavelengths, allowing the analytical concentrations to be calculated. Final cleaning: After the tests are complete, the cuvettes are automatically washed to prepare the next cycle.

Interpretation of the results on the URIT-8021A requires a good knowledge of analytical methods, rigorous quality control, consultation of reference ranges, and clinical perspective of the data.

Statistical analyses:

Our data was collected using a pre-established survey form incorporated into the KOBOCOLLECT application, version v1.29, and exported to R software version 4.4.0 for analysis.

The p-value was calculated (5%) to determine the existence of a statistically significant link between the study variables.

3. Results

There were 257 positive subjects for HBsAg, who also had positive total anti-HBc antibodies, confirming exposure to the virus and the persistence of the immune response against the nucleocapsid. Only 6 subjects tested positive for HBeAg, suggesting strong viral replication in a minority of them, while the majority tested negative for this marker. Only 3 subjects had anti-HBs antibody levels above 10 IU/L, indicating rare immunization ("seroconversion") in this group (**Figure 1**).

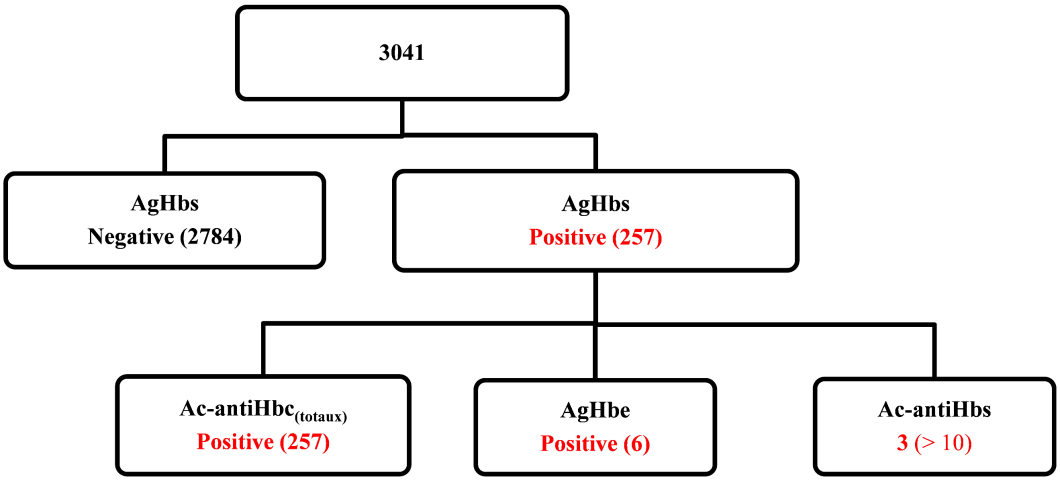

Figure 1. Flow chart of hepatitis B (HBsAg positive) among health science students and health personnel in the city of Conakry.

Table 1. Frequencies of serological markers (specific) for HBV among participants who were HBsAg-positive carriers.

Feature	N = 257[1]	95% CI[2]	N
AgHbe	24.87 (159.20) [0.23 - 1096.20]	[5.3 - 44]	257
AgHbe			257
NEGATIF	251 (98%)	[95 - 99]	
POSITIF	6 (2.3%)	[0.95 - 5.3]	
Ac_antiHbc	8.62 (0.93) [3.27 - 9.84]	[8.5 - 8.7]	257
Ac_antiHbc			257
POSITIF	257 (100%)	[98 - 100]	
Ac_anti_Hbs	0.19 (1.76) [0.00 - 19.20]	[−0.02 - 0.41]	257
Ac_anti_Hbs			257
NEGATIF	254 (99%)	[96 - 100]	
POSITIF	3 (1.2%)	[0.30 - 3.7]	

[1]Mean (SD) [Min − Max]; n (%); [2]CI = confidence interval.

In Table 1, among the 257 HBsAg-positive participants: HBe antigen (HBeAg) was positive in 2.3% of cases, Anti-HBc antibody (anti-HBcAb) was present in 100% of carriers, Anti-HBs antibody (anti-HBsAb) was detected in only 1.2%.

Table 2. Non-specific markers of hepatitis B (GOT).

Feature	**Normal** N = 254[1]	**Pathological** N = 3[1]	N
GOT_ASAT	19.92 (4.91) [10 - 31]	46.33 (11.37) [37 - 59]	257

[1]Mean (SD) [Min − Max].

An elevation in GOT (ASAT) in 12% of positive subjects with a mean pathological value of 46.33 IU/L (Table 2).

Table 3. Nonspecific markers of hepatitis B (GPT).

Feature	Normal N = 256[1]	Pathological N = 1[1]	N
GPT_ALAT	9.72 (3.03) [4 - 22]	40.00 (NA) [40 - 40]	257

[1]Average (SD) [Min – Max].

An increase in GPT (ALAT) in 4% of positive cases, with an average value of 40 IU/L (Table 3).

Table 4. Bivariate analysis of participants' sociodemographic characteristics with the results of biological analyses.

Feature	NEGATIVE N= 2 784[1]	POSITIVE N = 257[1]	p-value[2]
AGE GROUPS			0.028
18 - 27 years old	1318 (47.34)	114 (44.36)	
28 - 37 years old	887 (31.86)	105 (40.86)	
38 - 47 years old	348 (12.50)	21 (8.17)	
48 - 57 years old	146 (5.24)	10 (3.89)	
58 years old and older	85 (3.05)	7 (2.72)	
SEX			<0.001
Feminine	1679 (60.31)	120 (46.69)	
Masculine	1105 (39.69)	137 (53.31)	
PROFESSION			0.050
Biologist	297 (10.67)	20 (7.78)	
Student	595 (21.37)	68 (26.46)	
Nurse	1082 (38.86)	103 (40.08)	
Doctor	771 (27.69)	59 (22.96)	
Pharmacist	39 (1.40)	7 (2.72)	
MARITAL STATUS			0.024
Single	1373 (49.32)	147 (57.20)	
Divorced	3 (0.11)	0 (0.00)	
Married	1395 (50.11)	107 (41.63)	
Widowed	13 (0.47)	3 (1.17)	
RESIDENCE			>0.9
Coyah	88 (3.16)	9 (3.50)	
Dixinn	220 (7.90)	20 (7.78)	
Dubreka	104 (3.74)	7 (2.72)	
Kaloum	106 (3.81)	9 (3.50)	
Matam	118 (4.24)	14 (5.45)	
Matoto	733 (26.33)	69 (26.85)	
Ratoma	1415 (50.83)	129 (50.19)	

[1]n (%); [2]Chi-square test of independence; Fisher's exact test.

In Table 4, the bivariate analysis highlights a statistically significant association between HBsAg positivity and certain sociodemographic characteristics:

Age ($p = 0.028$): young adults aged 18 - 27 (44.36%) and 28 - 37 (40.86%) is the most affected, probably due to increased exposure to occupational risks (handling blood, medical practices) and low vaccination coverage.

Gender ($p < 0.001$): men (53.31%) are significantly more infected than women (46.69%), which could be explained by greater occupational or behavioral exposure.

Occupation ($p = 0.050$): Nurses and students are the most affected, reflecting frequent exposure to biological fluids and inadequate protective measures (vaccination, gloves, hygiene).

Marital status ($p = 0.024$): single people (57.2%) are more often positive, possibly due to more frequent risky behavior in this category.

However, place of residence was not associated with VHB positivity ($p > 0.9$).

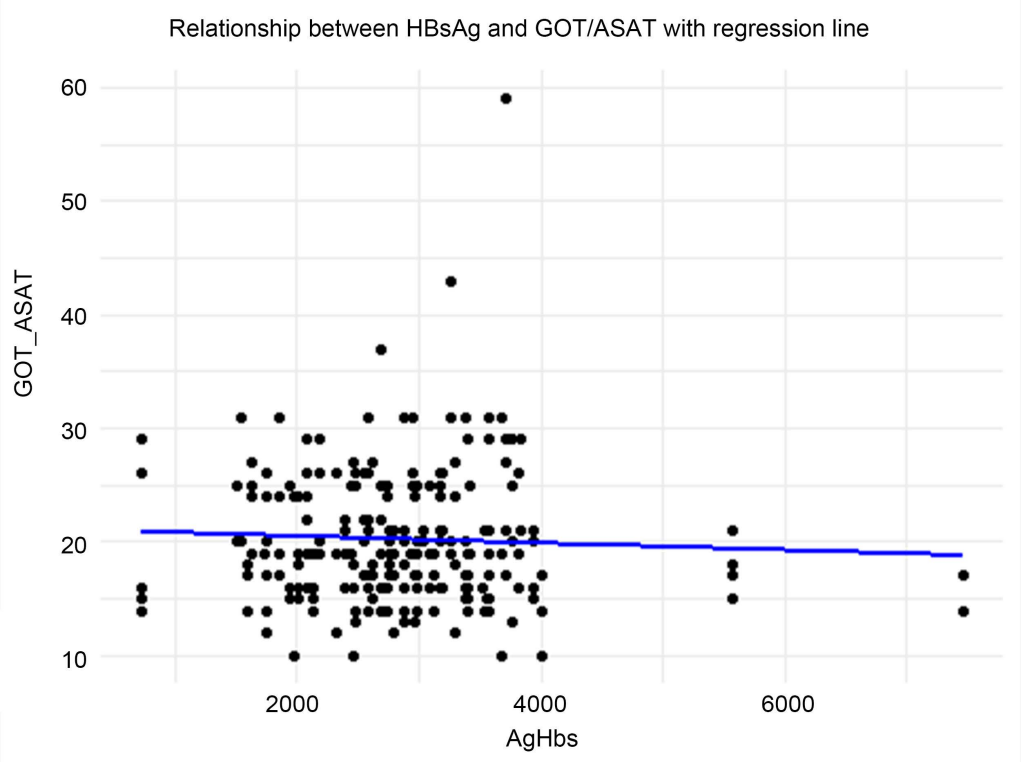

Figure 2. Correlation between HBsAg and GOT_ASAT.

There is a correlation between HBsAg and GOT/ASAT, and the correlation between HBsAg and transaminases (GOT/ASAT) confirms this biological association between HBV positivity and enzyme disruption (Figure 2).

This figure also shows a comparable correlation with GPT/ALAT levels, illustrating the relationship between HBsAg and transaminases, which confirms the biological association between HBV positivity and liver enzyme abnormalities (Figure 3).

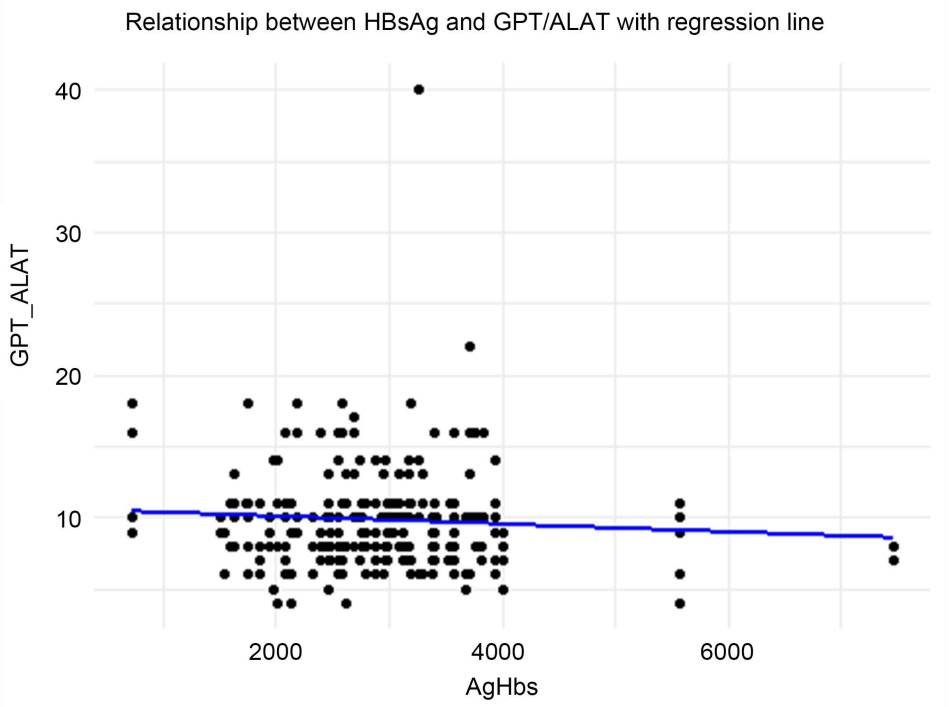

Figure 3. Correlation between HBsAg and GPT_ALAT.

4. Discussion

Out of a total of 257 samples positive for surface antigen (HBsAg): HBe antigen (HBeAg) was positive in 2.3% of cases, anti-HBc antibody was present in 100% of carriers, and anti-HBs antibody was detected in only 1.2%.

Evaluation of non-specific liver markers revealed: Elevated GOT (AST) in 12% of positive subjects (mean pathological value: 46.33 IU/L), an elevation of GPT (ALAT) in 4% of positive cases, with an average of 40 IU/L, univariate analysis shows a statistically significant association between HBsAg positivity and certain sociodemographic characteristics:

Age ($p = 0.028$): young adults aged 18 - 27 (44.36%) and 28 - 37 (40.86%) are the most affected, probably due to increased exposure to occupational risks (handling blood, medical practices) and low vaccination coverage.

Gender ($p < 0.001$): Men (53.31%) are significantly more infected than women (46.69%), which could be explained by greater occupational or behavioral exposure.

Occupation ($p = 0.050$): Nurses and students are the most affected, reflecting frequent exposure to biological fluids and inadequate protective measures (vaccination, gloves, hygiene).

Marital status ($p = 0.024$): single individuals (57.2%) are more often positive, possibly due to more frequent risky behaviors in this category.

On the other hand, place of residence was not associated with seropositivity ($p > 0.9$).

There is a correlation between HBsAg and GOT/ASAT, representing the corre-

lation between HBsAg and transaminases (GOT/ASAT), which confirms this biological association between HBV positivity and enzyme disruption. There is also a comparable correlation with GPT/ALAT levels, illustrating the relationship between HBsAg and transaminases, which confirms the biological association between HBV positivity and liver enzyme alteration.

Our study reported a seroprevalence of 8.5% of HBsAg and anti-Hbc antibody carriers among participants. These results corroborate data from the literature on the region according to the World Health Organization, which places Guinea in a zone of high prevalence varying between 8% and 16% [19]. Other authors reported lower results than ours in a review of 25 studies conducted in Africa and Asia covering more than 10,000 healthcare workers in 11 countries, with an overall prevalence of 5.0% for Africa, slightly higher than that for Asia [20].

Other superior results were reported in 2010 by the Society for Epidemiology in the United States, which revealed a seroprevalence of hepatitis B among healthcare workers that was two to four times higher than that of blood donor controls [21]. In Burkina Faso, a study conducted on a sample of 157 healthcare workers in 2008 noted a seroprevalence of 12.1% for HBsAg and 63.7% for at least one of the HBV markers [22]. A study of blood donors in the N'Zérékoré region reported a prevalence of 13.4% for HBsAg carriage. Although this result is higher than ours, it corroborates most of the data in the literature [23].

Our data were collected using a survey form.

Anti-HBs antibodies were detected in only 1.2% of cases, indicating that almost all HBsAg-positive individuals are not immune (absence of anti-HBs), reflecting active infection, with a small percentage of atypical cases. This highlights a high risk of transmission and a need for monitoring or preventive intervention. In a Chinese study, the "HBsAg positive + anti-HBs" profile (*i.e.*, both simultaneously) was observed in 2.93% of HBsAg carriers [24].

Transaminase testing showed that 12% of participants had elevated GOT-ASAT levels, compared with 4% for GPT-ALAT. Transaminase spikes are often correlated with HBV genome reduction, HBeAg seroconversion, and ccDNA inhibition/reduction, which characterize the degree of liver function impairment and appear to be a marker for establishing functional cure [25].

A prevalence of 8.5% of chronic HBV infection places the study population well above the 2% threshold used by the WHO to define areas of intermediate to high endemicity, where systematic screening and management are recommended. In such a context, healthcare settings become major sites of transmission, particularly for healthcare professionals and students during clinical activities [26].

This study is limited in that it focuses on the situation of staff in Conakry and should be extended to the whole country in order to accurately measure the extent of hepatitis B among healthcare professionals and students in Guinea.

Continuing education on infection prevention and control procedures and mandatory vaccination should be organized by the authorities to reduce the impact of infection.

5. Conclusion

The prevalence of hepatitis B among healthcare workers and health science students, comparable to that of the general population, reveals a striking reality: hepatitis B knows no professional boundaries. This finding challenges Guinean society as a whole and recognizes the universal nature of the risk. This is a valuable step forward in understanding the dynamics of infection in Guinea, which should encourage collective action for prevention, vaccination, and research.

Authors' Contributions

Study design: **ASB, AC,** Biological analysis of samples: **ASB, AC, AK, ATT, NT, AHD, MCD;** Analysis of results and drafting of manuscript: **ASB, AC, KK, TID, AW.**

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Our sincere thanks go to the late Professor Naby Moussa Baldé, the main promoter of the study, who was taken from us (may he rest in peace).

Ethical Aspects

The protocol was approved by the scientific committee of Gamal Abdel Nasser University in Conakry. The data were collected anonymously and will only be used for the purposes of the study, in accordance with confidentiality requirements. The informed consent of all participants was obtained prior to their inclusion. The principle was in accordance with the Declaration of Helsinki.

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Gerlich, W.H. (2013) Medical Virology of Hepatitis B: How It Began and Where We Are Now. *Virology Journal*, **10**, Article No. 239. https://doi.org/10.1186/1743-422x-10-239

[2] Blumberg, B.S. (1965) A "New" Antigen in Leukemia Sera. *JAMA*: *The Journal of the American Medical Association*, **191**, 541-546. https://doi.org/10.1001/jama.1965.03080070025007

[3] Blumberg, B.S. (1977) Australia Antigen and the Biology of Hepatitis B. *Science*, **197**, 17-25. https://doi.org/10.1126/science.325649

[4] Dane, D.S., Cameron, C.H. and Briggs, M. (1970) Virus-Like Particles in Serum of Patients with Australia-Antigen-Associated Hepatitis. *The Lancet*, **295**, 695-698. https://doi.org/10.1016/s0140-6736(70)90926-8

[5] Walter, E., Keist, R., Niederöst, B., Pult, I. and Blum, H.E. (1996) Hepatitis B Virus

Infection of Tupaia Hepatocytes *in Vitro* and *in Vivo*. *Hepatology*, **24**, 1-5. https://doi.org/10.1002/hep.510240101

[6] Barker, L.F. and Murray, R. (1972) Relationship of Virus Dose to Incubation Time of Clinical Hepatitis and Time of Appearance of Hepatitis' Associated Antigen. *The American Journal of the Medical Sciences*, **263**, 27-33. https://doi.org/10.1097/00000441-197201000-00005

[7] Rehermann, B. and Nascimbeni, M. (2005) Immunology of Hepatitis B Virus and Hepatitis C Virus Infection. *Nature Reviews Immunology*, **5**, 215-229. https://doi.org/10.1038/nri1573

[8] Liang, J.T. (2009) Hepatitis B: The Virus and Disease. *Hepatology*, **49**, S13-S21. https://doi.org/10.1002/hep.22881

[9] Brian, J.M. (2008) Natural History of Chronic Hepatitis B—Clinical Implications. *The Medscape Journal of Medicine*, **10**, Article 91.

[10] Schweitzer, I.L., Dünn, A.E.G., Peters, R.L. and Spears, R.L. (1973) Viral Hepatitis B in Neonates and Infants. *The American Journal of Medicine*, **55**, 762-771. https://doi.org/10.1016/0002-9343(73)90257-x

[11] Yan, H., Zhong, G., Xu, G., He, W., Jing, Z., Gao, Z., *et al.* (2012) Sodium Taurocholate Cotransporting Polypeptide Is a Functional Receptor for Human Hepatitis B and D Virus. *eLife*, **1**, e00049. https://doi.org/10.7554/elife.00049

[12] Li, H., Zhuang, Q., Wang, Y., Zhang, T., Zhao, J., Zhang, Y., *et al.* (2014) HBV Life Cycle Is Restricted in Mouse Hepatocytes Expressing Human NTCP. *Cellular & Molecular Immunology*, **11**, 175-183. https://doi.org/10.1038/cmi.2013.66

[13] Schmitz, A., Schwarz, A., Foss, M., Zhou, L., Rabe, B., Hoellenriegel, J., *et al.* (2010) Nucleoporin 153 Arrests the Nuclear Import of Hepatitis B Virus Capsids in the Nuclear Basket. *PLOS Pathogens*, **6**, e1000741. https://doi.org/10.1371/journal.ppat.1000741

[14] Guo, H., Jiang, D., Zhou, T., Cuconati, A., Block, T.M. and Guo, J. (2007) Characterization of the Intracellular Deproteinized Relaxed Circular DNA of Hepatitis B Virus: An Intermediate of Covalently Closed Circular DNA Formation. *Journal of Virology*, **81**, 12472-12484. https://doi.org/10.1128/jvi.01123-07

[15] Seeger, C. and Mason, W.S. (2015) Molecular Biology of Hepatitis B Virus Infection. *Virology*, **479**, 672-686. https://doi.org/10.1016/j.virol.2015.02.031

[16] Tan, Z., Pionek, K., Unchwaniwala, N., Maguire, M.L., Loeb, D.D. and Zlotnick, A. (2015) The Interface between Hepatitis B Virus Capsid Proteins Affects Self-Assembly, Pregenomic RNA Packaging, and Reverse Transcription. *Journal of Virology*, **89**, 3275-3284. https://doi.org/10.1128/jvi.03545-14

[17] Souare, O.I., Huws, J., Djenabou, D. and Kadiatou, D. (2024) Epidemiology of Viral Hepatitis B and C in Guinea: Scooping Review. *Health Research in Africa*, **2**, 1-10.

[18] Keita, M., Fadiga, A.G., Soumah, M.M., Sylla, D., Traore, F.A., Biané, B., *et al.* (2014) Co-infection VIH et virus de l'hépatite B au service d'hématologie de l'hôpital national Ignace Deen (Guinée-Conakry). *Annales de Dermatologie et de Vénéréologie*, **141**, S442. https://doi.org/10.1016/j.annder.2014.09.479

[19] ONU (2022) Plus de 90 millions d'Africains infectés par l'hépatite B ou C (OMS). ONU Info 2022. https://news.un.org/fr/story/2022/07/1124552

[20] Maamor, N.H., Muhamad, N.A., Mohd Dali, N.S., Abdul Mutalip, M.H., Leman, F.N., Aris, T., *et al.* (2022) Seroprevalence of Hepatitis B among Healthcare Workers in Asia and Africa and Its Association with Their Knowledge and Awareness: A Systematic Review and Meta-Analysis. *Frontiers in Public Health*, **10**, Article 859350.

https://doi.org/10.3389/fpubh.2022.859350

[21] Helcl, J., Cástková, J., Benes, C., Novotna, L., Sepkowitz, K.A. and DeHovitz, J.A. (2000) Control of Occupational Hepatitis B among Healthcare Workers in the Czech Republic, 1982 to 1995. *Infection Control & Hospital Epidemiology*, **21**, 343-346. https://doi.org/10.1086/501771

[22] Pietra, V., Kiema, D., Sorgho, D., Kabore, S.P.C.G., Mande, S., Castelli, F., *et al.* (2008) Prévalence des marqueurs du virus de l'hépatite B et des anticorps contre le virus de l'hépatite C parmi le personnel du District Sanitaire de Nanoro, Burkina Faso [Prevalence of Hepatitis B Virus Markers and Hepatitis C Virus Antibodies in Health Personnel in the District of Nanoro, Burkina Faso]. *Sciences de la Santé*, **31**, 53-59.

[23] Camara, A., Thea, E., Haba, I.N., Youla, Y., Diallo, I.S., Diallo, M.S., *et al.* (2024) Seroprevalence of Human Immunodeficiency Virus and Hepatitis B in Blood Donors at the N'Zérékoré Regional Blood Transfusion Centre in Guinea. *Open Journal of Immunology*, **14**, 33-45. https://doi.org/10.4236/oji.2024.142004

[24] Pu, Z., Li, D., Wang, A., Su, H., Shao, Z., Zhang, J., *et al.* (2015) Epidemiological Characteristics of the Carriers with Coexistence of HBsAG and Anti-HBs Based on a Community Cohort Study. *Journal of Viral Hepatitis*, **23**, 286-293. https://doi.org/10.1111/jvh.12492

[25] Vaillant, A. (2021) Transaminase Elevations during Treatment of Chronic Hepatitis B Infection: Safety Considerations and Role in Achieving Functional Cure. *Viruses*, **13**, Article 745. https://doi.org/10.3390/v13050745

[26] Tadesse, S., Munshea, A., Gelaw, B., Peshu, N., Tesfa, E., Mekonnen, F., *et al.* (2025) Prevalence of Hepatitis B Virus Infection and Its Associated Factors in Ethiopia: A Recent Systematic Review and Meta-Analysis. *BMC Infectious Diseases*, **25**, Article No. 749. https://doi.org/10.1186/s12879-025-11150-8