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Impact of hepatitis B virus infection in patients with plasmodium parasites in selected health facilities in Accra, Ghana

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Abstract

Background The severity of malaria is often exacerbated by concurrent health issues, including coinfections with hepatitis B virus (HBV). Recent research suggests that the shared pathophysiological characteristics of HBV and malaria influence patient outcomes. This study aimed to determine the prevalence of HBV among patients infected with *Plasmodium* species and to evaluate the associated risk factors and clinical outcomes in individuals attending various health facilities.

Method A cross-sectional study was conducted at Korle-Bu, Mamprobi, and Ashaiman Polyclinics in the Greater Accra Region. Patients presenting with malaria-like symptoms at the outpatient department (OPD) were recruited. Participants who tested positive for malaria via a rapid diagnostic test (RDT) and provided written consent were included. Demographic and clinical data were obtained through questionnaires. Blood samples (4 mL) were collected into a serum separator and EDTA tubes. The HBV surface antigen (HBsAg) and malaria parasites were detected via an HBV rapid diagnostic test and microscopy, respectively. Polymerase chain reaction (PCR) was also used to confirm the presence of the *Plasmodium* parasite. Liver function and hematological parameters were evaluated via a chemistry analyser and hematology analyser, respectively.

Results In total, 174 participants were recruited, comprising 99 (56.90%) from Korle-Bu Polyclinic, 50 (28.74%) from Mamprobi Polyclinic, and 25 (14.37%) from Ashaiman Polyclinic. Malaria diagnostics revealed that 52.87% (92/174) of the samples tested positive for malaria parasite via microscopy, and 73.56% (128/174) of the samples tested positive via PCR. The majority of participants were females (56.89%) and were predominantly aged 18–30 years. Common symptoms included headache, loss of appetite, and fever. HBV and malaria coinfection was observed in 9.19% of the patients. Increased alanine transaminase (ALT) levels and basophil counts were significantly associated with HBV and malaria coinfection ($\boldsymbol{p}$ = **0.029**).

Conclusion This study revealed a slightly greater prevalence of HBV and malaria coinfection than previously reported. These findings highlight the importance of incorporating routine coinfection diagnostics and improving HBV vaccination efforts to improve patient outcomes.

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Keywords Hepatitis B virus (HBV), Malaria coinfection, *Plasmodium* Sp., Risk factors, Clinical outcomes, Liver function, Hematological parameters

Introduction

Malaria endemicity continues to persist in certain regions, especially in Africa and Southeast Asia [1]. The most recent World Malaria Report estimates that there were 263 million cases and 597 000 malaria deaths worldwide in 2023, with children under five accounting for the majority of these deaths (67%) [2]. The African nations with the highest rates of malaria cases include Nigeria (25%), the Democratic Republic of the Congo (12%), Uganda (5%), Côte d'Ivoire (4%), Mozambique (4%) and Niger (4%) [1].

Hepatitis B (HB) is a liver disease caused by the hepatitis B virus (HBV). It can lead to serious complications, including cirrhosis, hepatocellular carcinoma, and various other clinical conditions [3]. Although vaccination against HB has been rolled out globally, infection from HBV causes approximately 1.2 million infections annually according to a report by WHO in 2015 [4]. Ghana is not exempt from the shared burden of HBV. The prevalence of hepatitis B virus (HBV) infections is high in the general population, with recent systematic reviews estimating rates of 12.3% and 14.4% [5, 6], classifying the country as highly endemic.

People who live in areas where *Plasmodium* spp. and HBV infections are endemic are at increased risk of coinfection due to the geographic overlap of these diseases. In the context of *Plasmodium* and HBV coinfection, the liver, which serves as the primary site for the development of both pathogens, is particularly vulnerable. This dual infection can lead to significant liver pathology, further increasing the risks of morbidity and mortality [1]. The impact of coinfection may include a cocktail of activities, including pro-inflammatory cytokine reactions [7–10], a reduction in red blood cell (RBC) counts by *P. falciparum*, which results in anaemia, the promotion of erythropoietin production from newly formed liver cells by HBV, and increased levels of haemoglobin (Hb) [11–13]. Therefore, it is reasonable to anticipate that in the case of coinfection, HBV may mitigate the impact of *P. falciparum* on haemoglobin (Hb) levels [14, 15] and may result in health complications [16]. The impact on haemoglobin levels is complex and can be influenced by many factors, including the severity of each infection and an individual's overall health and immune response [17].

With both pathogens prevalent in Ghana, coinfection is common [18]. Epidemiological data show increased asymptomatic carriage of the *Plasmodium* parasite among the adult population [19, 20]. This contradictory information also indicates that the simultaneous presence of these pathogens might not affect each other because repeated exposure leads to a state of immune tolerance [15, 21]. These observations suggest the need for further investigation to understand the differences in clinical response among coinfected individuals and individuals with mono-infection with *Plasmodium spp*. This study therefore sought to understand the impact of HBV infections in individuals with *Plasmodium* spp. in adult populations from three primary healthcare facilities with malaria-like symptoms.

Methodology

Study sites and population

This cross-sectional study was conducted at the Korle-bu Polyclinic, Mamprobi Polyclinic, Ashaiman polyclinic, which are all in the Greater Accra Region of Ghana. These health facilities are primary healthcare facilities designed to address basic health care needs within different communities. All consenting individuals who were positive for *the* malaria Rapid Diagnostic Test (RDT) (SD Bioline, St. Louis, USA; Lot. No. 05FK50) were included in the study. Administered questionnaires were used to collect sociodemographic data and medical history from each participant. Each participant was assigned a unique study-specific identifier, which was cross-referenced with their hospital identification number. All data collection instruments and sample containers, including questionnaires, consent forms, and blood collection tubes, were labelled with this study-specific identifier to maintain consistent reference and ensure traceability. The hepatitis B surface antigen (HBsAg) was screened via the Determine™ HBsAg 2 rapid test (Abbott Diagnostics Medical Co., Ltd., Matsudo, Chiba, Japan). Patients with both malaria parasites and HBsAg were considered to have coinfection. This study was carried out between October 2023 and November 2023.

Inclusion criteria

Individuals who were confirmed to be RDT positive for malaria infection (aged 18 years and above).

Exclusion criteria

In addition to mothers, patients with known clinical conditions other than malaria and/or HBV infection were not allowed to participate in the study. These conditions included cancer, sickle cell disease, diabetes mellitus, hypertension, renal disorders, and chemotherapy. Information from administered questionnaires was used to either recruit or exclude participants in this study.

Sample size/sample determination

The minimum sample size for the study was determined via Cochran's formula, $n = Z^2P(1 - P)/d^2$ with a 95% confidence level, a 5% margin of error, and an anticipated prevalence rate. In this case, **Z** is the Z score for a 95% confidence level (1.96), **P** is the estimated prevalence of HB infections from previous studies (8.36%) [6], and **d** is the margin of error set at 5% (0.05). Therefore, the sample size was calculated as $n = (1.96)^2 \times 0.0836(1 - 0.0836)/(0.05)^2 \approx 118$. Thus, the minimum sample size for this study was 118 participants.

Data collection and laboratory methods

Sample collection and procedure

Venous blood (4 *mL*) in both EDTA and serum separator tubes was drawn from each study participant. This was drawn by inserting a hollow needle through the skin and into a superficial vein (typically in the cubital fossa of the forearm). Thin and thick blood smears were prepared on slides for all RDT-positive malaria patients and examined for parasites via light microscopy (LM) [22]. For molecular analysis, whole-blood samples (100 *µL*) were used to generate blood films and spotted onto 3-mm Whatman 903 filter paper (Merck KGaA, Darmstadt, Germany), which was then air-dried and placed individually in plastic bags with desiccant for transportation to the Clinical Virology Laboratory of the Department of Medical Microbiology, University of Ghana Medical School. Blood samples were aspirated and analysed via a hematology analyser within 2 h of collection.

Malaria parasitaemia by microscopy

Laboratory analyses for detecting malaria parasites were carried out at the laboratories of the designated health facilities under the supervision of highly trained laboratory professionals. Thick and thin blood films were prepared on a single slide and stained with Giemsa solution to detect and quantify malaria parasites, following the Ministry of Health (MOH) Standard Malaria Diagnosis and Treatment Guidelines (2004). To determine parasite density, 200 fields (equivalent to 0.5 *µL* of thick blood film) were examined. Specifically, the parasite density was calculated per microliter (*µL*) of blood, assuming 8000 leukocytes per microliter. In brief, a thick film with evenly distributed white blood cells was selected. Using the oil immersion objective, 200 white blood cells 200wbc if parasites are more than 100 and 500wbc if parasites are less than 100 were systematically counted, simultaneously tallying the number of parasites (asexual forms only) in each field. The parasite count per microlitre of blood was then derived by multiplying the number of parasites counted by 200 (representing the fields examined), dividing this product by 200, and then multiplying by 8000 (the assumed number of leukocytes per microlitre of blood). Thin smears were used to identify the malaria parasite species. The parasite density was then estimated from the thick blood smears.

The parasite density was estimated as follows:

Parasite density = Number of parasitized RBCs × X/200 WBCs (white blood cells).

where *X* = total WBC count/200; WBC: white blood cell; RBC: red blood cell [23].

Plasmodium species identification via nested PCR

The genomic DNA of the parasites was extracted via the Chelex method as previously described [24, 25]. To identify *Plasmodium* species from extracted DNA, nested PCR was utilized with primers specific for *Plasmodium* species that target the 18 S rRNA genes of *P. ovale*, *P. malariae*, *and P. falciparum* [26]. In the first round of amplification, the genus-specific primers rPLU5 and rPLU6 were used to extend the DNA initially extracted from dried blood spots in a reaction supplemented with buffer, deoxynucleotide triphosphates (dNTPs), magnesium chloride ($MgCl_{2)}$, and Taq polymerase. Using species-specific primers, rFAL1/rFAL2 (specific to *P. falciparum*), rMAL1/rMAL2 (specific to *P. malariae*), and rOVA1/rOVA2 (specific to *P. ovale*) were added to a reaction mixture that was similar to that used for the first amplification. The resulting product was then used as a template for the second amplification.

HBV serology

Sera were obtained by centrifuging the blood samples at 3,000 rpm for 15 min, after which they were frozen in aliquots at -20 °C until use. A commercial HBsAg test kit Determine™ HBsAg 2 rapid test (Abbott Diagnostics Medical Co., Ltd., Matsudo, Chiba, Japan) was used for the qualitative detection of HBsAg [27].

Liver function tests

Liver function was assessed via liver enzymes, mainly aspartate amino transferase (AST), alanine amino transferase (ALT), alkaline phosphatase (ALP), and albumin. A chemical analyser (Mindray Biomedical Electronics Co., Ltd., Shenzhen, China) was used to assess each of the above listed parameters.

Statistical analysis

A frequency distribution table in the Statistical Package for the Social Sciences (SPSS) vs. 26.0 (IBM Corp., Armonk, NY, USA) was used to ascertain the percentage and severity of the disease in individuals who also had coinfections compared with those with only malaria. Chi-square tests for associations were used to determine the impact of mono-infection or coinfection on LFTs. The Kruskal–Wallis test was used to determine the relationships between test parameters in non-distributed

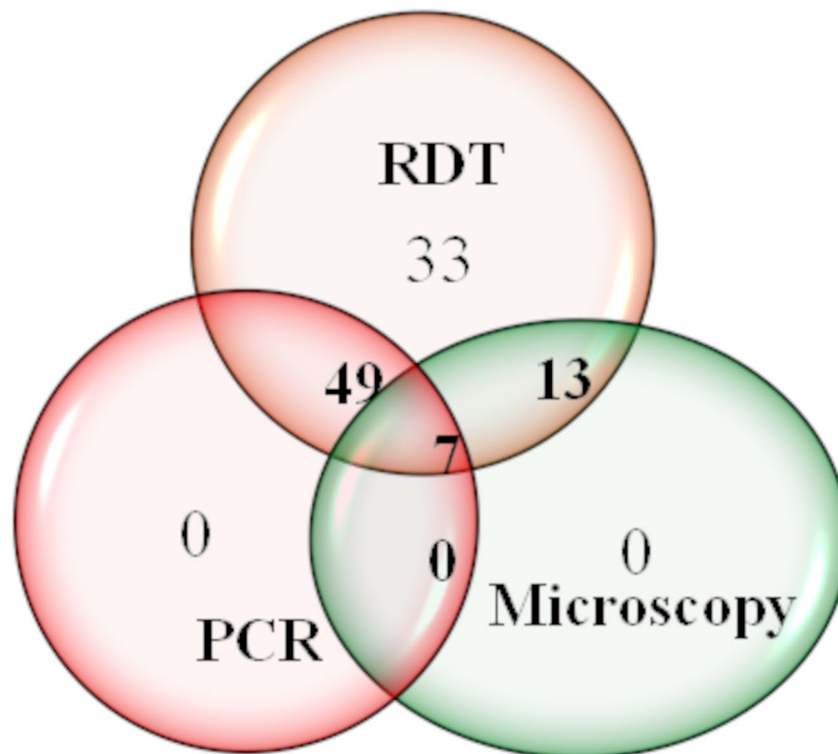

Fig. 1 Malaria diagnostic test results among study participants. All tested samples were RDT positive. The figure illustrates the proportions of participants with RDT/microscopy positivity (7.4%), RDT/PCR positivity (28.2%), and RDT/PCR/microscopy positivity (45.4%). It also shows the 18.9% of participants who were RDT positive but negative by PCR and microscopy

data. All the statistical analyses were conducted with a p value less than 0.05 indicating statistical significance.

Ethical considerations

Written consent was obtained from the participants in this study. In instances where patients encountered language barriers, an interpreter was present throughout the consent process to ensure comprehension. All the data were stored anonymously on a secured computer with members of the research team having sole access to this information.

Results

Demographics and clinical characteristics of malaria patients

In total, 174 study participants presenting with malaria were included in this study. A total of 99 (56.90%) patients were recruited from the Korle-bu polyclinic, 50 (28.74%) from the Mamprobi polyclinic and 25 (14.37%) from the Ashaiman polyclinic. All the samples tested were RDT positive. Malaria diagnostic test results indicate that 7.4% (13/174) were RDT/microscopy positive, 28.2% (49/174) were RDT/PCR positive, 45.4% (79/174) were RDT/PCR/microscopy positive, and 18.9% (33/174) were negative according to PCR and microscopy (Figure 1). Most of the participants in this study were females (56.89%). The majority of the participants in this study were in the 18–30-year age class, and most of the study participants were from Korle Gonno, Dansoman, Mamprobi and its environs (Table 1).

Table 1 Socio-demographic characteristics of study population

Demographics	Categories	Result (out of $N=174$, n%)
Study Sites	Korle-Bu polyclinic	99 (56.90)
	Mamprobi polyclinic	50 (28.74)
	Ashaiman polyclinic	25 ((14.37)
Age	18–30	89 (51.14)
	31–40	25 (14.36)
	41–50	30 (17.24)
	51–60	13 (7.47)
	> 60	17 (9.77)
Gender	Females	99 (56.89)
	Males	75 (43.10)

Prevalence of HB/malaria and associated risk factors

Among the 174 study participants who were positive for malaria, 16 were positive for HBV, for a proportion of 9.19%. Among the 16 coinfected individuals, the majority were from the Korle-bu polyclinic (8, 50%), and the

minority were from the Mamprobi polyclinic (3, 18.8%). Additionally, more males (9, 56.2%) than females (7, 43.8%) were coinfected. The 18–30-year age group (8, 50%) had the highest number of coinfected individuals. No coinfection was recorded for individuals who were 60 years old or older. Most individuals who had coinfection were found to have formal education (10 (62.5%)). Additionally, the majority of individuals who experienced coinfection were found to be single (9. 56.3%). These factors did not significantly affect the HB or malaria status of individuals (Table 2).

Clinical characteristics and outcomes of patients with HBV/Malaria

The symptoms observed in this study were fever (31.6%), headache (36.8%) and loss of appetite (15.5%). The majority of the study participants recorded normal levels of each of the hematological parameters (Table 3). Similar results were obtained for the parameters of the LFT test. In terms of clinical symptoms, 9 study participants reported coinfected with headache, representing 56.3% of the coinfected population in the study. The majority of individuals with coinfection were also found to have low WBC (10, 62.5%), neutrophil (9, 56.3%) and HB (11, 68.8%) counts. However, normal levels of lymphocytes (14, 87.8%), monocytes (13, 81.3%), eosinophils (14, 87.5%), basophils (11, 68.8%), RBCs (13, 81.3%) and platelets (12, 75%) were found among the study participants (Table 3). Liver function test biomarkers were observed to be at normal levels among the majority of the study participants with coinfection, except for high ALT levels, which were found among 10 (62.5%) out of 16 coinfected individuals. No association was found between the clinical presentation of a patient and coinfection ($p>0.05$) (Table 4). However, an association was found between basophil levels and coinfection ($p=0.000$). For the LFT parameters, high levels of ALT were found to be associated with HBV/*Plasmodium* sp. coinfection ($p=0.029$).

Discussion

The coinfection of *Plasmodium* sp. and hepatitis B virus (HBV) is known to affect the progression of either or both diseases [28–30], despite the implementation of control programs, such as vaccinations, aimed at mitigating their impact [14]. Previous studies have demonstrated significant epidemiological interactions between malaria and hepatitis B virus (HBV), which are believed to influence patient prognosis [21, 31, 32]. However, the extent to which both diseases may impact the clinical outcome is still unknown. Despite vaccination rollout in the country, the high prevalence of HBV infections in Ghana still provides a clinical environment where coinfection is common.

Variations in malaria diagnostic results were observed among the 174 participants. The study employed three diagnostic methods: rapid diagnostic tests (RDTs), microscopy, and polymerase chain reaction (PCR). While microscopy is considered the gold standard for malaria diagnosis, its accuracy can be affected by factors such as parasite clearance through the immune response or the administration of antimalarial drugs [33]. PCR is known for its high sensitivity and specificity and serves as a valuable alternative for detecting low-level infections. The discrepancies between the diagnostic methods reflect the varying sensitivities of each technique, highlighting the

Table 2 Demographic distribution of study participants

Characteristics	Malaria infection *n*/174 (%)	HBV/Malaria co-infection *n*/16 (%)	*p*-value
Study sites			
Korle-bu polyclinic	97 (55.7)	8 (50)	**0.877**
Ashaiman polyclinic	50 (28.7)	5 (31.2)	
Mamprobi polyclinic	27 (15.5)	3 (18.8	
Gender			
Male	75 (43.1)	9 (56.2)	**0.265**
Female	99 (56.9)	7 (43.8)	
Age group			
18–30	89 (51.14)	8 (50)	**0.432**
31–40	25 (14.36)	2 (12.5)	
41–50	30 (17.24)	5 (31.2)	
51–60	13 (7.47)	1 (6.3)	
> 60	17 (9.77)	0 (0)	
Educational background			
Formal education	115 (66.1)	10 (62.5)	**0.750**
Informal education	59 (33.9)	6 (37.5)	
Marital status			
Single	101 (58.0)	9 (56.3)	0.820
Married	72 (41.38)	7 (4)	
Divorce	1 (0.6)	0 (0)	

Table 3 Association between the clinical characteristics (clinical symptoms and haematological parameters) and HBV/Malaria coinfection

Clinical characteristics	Range	Malaria $n = 174$(%)	HBV/Malaria coinfection. $n = 16$(%)	p-value
Symptom				
Fever		55 (31.6)	3 (18.8)	0.232
Headache		64 (36.8)	9 (56.3)	0.708
Loss of appetite		27 (15.5)	3 (18.8)	0.246
Hematological parameters				
WBC				0.436
Normal	4.00–10.00	131 (75.3)	5 (31.3)	
Low	< 4.00	34 ((19.5)	10 (62.5)	
High	> 10.00	9 (5.2)	1 (6.3)	
Neutrophils				0.349
Normal	2.00–7.00	118 (67.8)	7 (43.8)	
Low	< 2.00	51 (29.3)	9 (56.3)	
High	> 7.00	5 (2.9)	0 (0)	
Lymphocytes				0.221
Normal	0.80-4.00	145 (83.3)	14 (87.8)	
Low	< 0.8	26 (14.9)	1 (6.3)	
High	> 4.00	3 (1.7)	1 (6.3)	
Monocytes				0.566
Normal	0.12–1.20	152 (87.4)	13 (81.3)	
Low	< 0.12	11 (6.3)	1 (6.3)	
High	> 1.20	11 (6.3)	2 (12.5)	
Eosinophils				0.604
Normal	0.02–0.5	136 (78.2)	14 (87.5)	
Low	< 0.02	35 (20.1)	2 (12.5)	
High	> 0.5	3 (1.7)	0 (0)	
Basophils				0.000
Normal	0.00-0.10	169 (97.1)	11 (68.8)	
High	> 0.10	5 (2.9)	3 (18.8)	
RBC				0.578
Normal	4.00-5.50	137 (78.7)	13 (81.3)	
Low	< 4.00	23 (13.2)	1 (6.3)	
High	> 5.50	14 (8.0)	2 (12.5)	
Platelet				0.574
Normal	100–300	126 (72.4)	12 (75)	
Low	< 100	38 (21.8)	4 (25)	
High	> 300	10 (5.7)	0 (0)	
HB				0.425
Normal	12.0–16.0	134 (7.7)	2 (12.5)	
Low	< 12.0	35 (20.1)	11 (68.8)	
High	> 16.0	5 (2.9)	5 (31.3)	

importance of combining multiple approaches to obtain a more comprehensive understanding of malaria prevalence, especially in research settings where thorough assessments are critical [34, 35].

The proportion of patients with HBV/malaria coinfection in this study was 9.19%. These rates differ from previously reported rates of 4.3%, 6%, and 0.7% in Nigeria [36], Gambia [1] and Ghana [37], respectively. Different geographical settings and demographics may slightly account for this difference. Hepatitis B coinfection has significant implications for the clinical management of individuals, potentially influencing disease progression and treatment strategies [38]. Although a greater proportion of coinfections was observed in individuals aged 18–30 years, no statistically significant association was found between age and coinfections ($p = 0.432$). This trend aligns with previous reports from other studies [39–41], which reported a higher prevalence of coinfections among individuals aged 25–34 years. The high representation of participants in this age group may account for the observed results.

In this study, no statistically significant association was found between HBV/malaria coinfection and HB levels ($p = 0.425$). The association between malaria and anaemia is well established. Malaria parasites infect red blood cells, leading to their destruction and a subsequent decrease in HB levels, contributing to anaemia [42, 43]. This result contradicts the findings of [37], who reported

Table 4 Outcome of LFT test parameters in relation to HBV/Malaria coinfection

LFT parameters	Range	Malaria *n* = 174(%)	HBV/Malaria coinfection. *n* = 16(%)	*p*-value
ALT				
Normal	≤ 32.0	109 (62.6)	6 (37.5)	0.029
High	> 32	65 (37.4)	10 (62.5)	
AST				
Normal	≤ 31	130 (74.7)	10 (62.5)	0.238
High	> 31	44 (25.3)	6 (37.5)	
ALP				
Low	< 30.00	27 (15.51)	3 (1.72)	0.214
Normal	30–120	105 (60.3)	12 (75)	
High	> 120	42 (24.14)	1 (6.3)	
Albumin				
Low	< 36	41 (23.56)	4 (25)	0.943
Normal	36.0–52.0	132 (75.86)	12 (75)	
High	> 52.0	0 (0)	0	
Total Protein				
Low	< 60	7 (4.02)	0 (0)	0.149
Normal	60.0–86.0	154 (88.5)	13 (81.3)	
High	> 86.0	13 (7.5)	3 (18.8)	
Total Bilirubin				
Low	< 3.40	0 (0)	0 (0)	0.527
Normal	3.40–22.00	130 (74.71)	12 (75)	
High	> 22.00	34 (19.5)	4 (25)	
Direct bilirubin				
Normal	≤ 5.13	122 (70.1)	11 (68.8)	0.900
High	> 5.13	52 (28.7)	5 (31.2)	

a significant association between coinfection and HB levels. Hepatitis B can also influence iron metabolism by causing liver inflammation, potentially contributing to anaemia [44]. Conversely, it is hypothesized that HBV increases HB levels by promoting erythropoietin release from hepatic tissues that are regenerating [16]. The contracting effect of both diseases may account for the lack of association observed in this study, indicating that coinfection with HBV/malaria may not influence HB levels among affected individuals.

Our study revealed a significant association between basophils and HBV/malaria coinfection. Although basophils have been poorly studied in the context of malaria, a decrease in peripheral blood basophils during *Plasmodium falciparum* infection has been reported, with this reduction correlating with symptom severity and likely reflecting the recruitment and accumulation of these cells in tissues [45]. An increase in basophil numbers in the spleen during *Plasmodium chabaudi chabaudi* infection has also been documented, with their activation and IL-4 production being linked to IL-3 stimulation around peak parasitemia [46]. Additionally, skewed type 2 and type 17 immune responses associated with basophil activation have been observed in malaria infections, highlighting their potential influence on host−pathogen dynamics [47]. These findings suggest that basophils, through their activation and recruitment, play dual roles in both systemic inflammation and local immune regulation during HBV/malaria coinfection. Our study observed elevated basophil counts in a few co-infected cases, which aligns with emerging evidence on the role of basophils in immune responses. While these findings are intriguing, they are based on a limited number of patients and should be interpreted with caution. Further studies with larger sample sizes are needed to explore the potential immunological mechanisms and clinical implications of basophilia in coinfection scenarios. Liver function biomarkers are important indices that help in the assessment of disease severity [48]. Our study revealed no major alterations in liver function indices among individuals coinfected with malaria and HBV, similar to findings reported by [49] in Nigeria. No significant difference was observed in aspartate transaminase (AST) levels between those with or without coinfection, which aligns with previous findings [16, 41]. However, a significant increase in alanine transaminase (ALT) levels was observed among coinfected individuals, indicating a direct impact on liver function. Elevated ALT levels are commonly used as a marker of liver damage or inflammation [50]. In addition, no significant changes were observed in bilirubin, AST or other liver indices in relation to coinfection, further suggesting that ALT may be the most sensitive indicator of liver dysfunction in HBV/*Plasmodium* sp. coinfection.

A key limitation of this study is that the sample size was calculated based on the prevalence of co-infections rather than for assessing their clinical or laboratory impact. As

a result, while our findings provide preliminary insights into potential associations, they may not be sufficiently powered to detect smaller effect sizes with statistical significance. Future studies with larger, appropriately powered sample sizes are needed to confirm these associations and better understand the clinical implications of co-infections.

Conclusion

This study revealed a slightly greater proportion of HBV/malaria coinfections than did previous reports and revealed diagnostic variability among RDTs, microscopy, and PCR. The combined use of these methods provides a more accurate prevalence estimate in endemic areas. While females predominated, males presented a higher coinfection rate, with most participants aged 18–30 years. Although elevated basophil counts and ALT levels were observed in co-infected patients, the small number of such cases limits the ability to draw definitive conclusions. These findings highlight possible trends that warrant further investigation in larger, more powered studies. No sociodemographic factors were significantly associated with coinfection outcomes in this study. These findings highlight the need for routine HBV screening and further research to clarify clinical predictors and optimize management strategies for coinfected individuals.

Abbreviations

ALP	Alkaline phosphatase
ALT	Alanine transaminase
AST	Aspartate aminotransferase
DNA	Deoxyribonucleic Acid
EDTA	Ethylenediamine tetracetic acid
HB	Hepatitis B
HBsAg	Hepatitis B surface antigen
HBV	Hepatitis B Virus
KBTH	Korle-bu Teaching Hospital
LFT	Liver function test
LM	light microscopy
MOH	Ministry of Health
OPD	Outpatient department
PCR	Polymerase chain reaction
RBC	Red blood cells
RDT	Rapid Diagnostic Test
rRNA	Ribosomal Ribonucleic Acid
SPSS	Statistical Package for Social Sciences
WHO	World Health Organization

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12879-025-11029-8.

Supplementary Material 1

Acknowledgements

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Author contributions

JDA, SGK, ISS, KWCS, YAA and MA-P were responsible for the study design, supervised the data collection, and contributed to the writing of the manuscript. JDA, SGK, and ISS carried out the data collection and laboratory work. JDA, SGK, KWCS, YAA and MA-P performed the data visualization and analysis. JDA, SGK, KWCS, YAA and MA-P drafted and revised the manuscript. All the authors read and approved the final manuscript.

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Data availability

The datasets produced and/or analysed in this study are available upon request from the corresponding author.

Declarations

Ethics approval and consent to participate

Ethical approval for the study was obtained from the Korle-Bu Scientific and Technical Committee (protocol ID# KBTH-STC0001182023), the Ethics Review Committee of Ghana Health Services (GHS-ERC 043/08/23), and the Ethical and Protocol Review Committee (EPRC) of the University of Ghana, College of Health Sciences (CHS-Et/M.10-P.10/2022–2023). The study adhered to the principles outlined in the Declaration of Helsinki, as well as the ethical guidelines of the respective ethics committees. Informed consent was obtained from all study participants prior to their recruitment.

Consent of publication

Not applicable.

Competing interests

The authors declare no competing interests.

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