

Virological and Histological Profiles of Chronic Hepatitis B Virus Carriers with Pre-core Mutations: A Cross-sectional Study in Four Hospitals in Douala, Cameroon

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ABSTRACT

Introduction: Chronic hepatitis B virus (CHB) infection is a major public health concern and a leading cause of cirrhosis and hepatocellular carcinoma (HCC). The aim of this study was to describe the virological and histological profiles of CHB virus carriers with pre-core mutations.

Methods: A cross-sectional study conducted in 4 hospitals in Douala for 10 years, from January 1st to December 31st. The study included patients diagnosed with CHB and who were HBeAg-negative. We excluded patients Co-infected with HDV, HCV, HIV and those already on treatment at the time of consultation. Data collected was sociodemographic, clinical, biological, histological and morphological variables. Liver fibrosis was assessed using transient elastography, FIB-4 and APRI score. The degree of fibrosis was separated into significant and non-significant fibrosis. Statistical analyses were performed using SPSS software version 26.0. Logistic regression by univariate then multivariate analysis were used to identify associated factors with significant fibrosis. A p-value <0.05 was considered statistically significant.

Results: A total of 1082 patients were included, with a mean age of 35.9 ± 9.9 years. The male gender represented 59.3% (n=642). The main comorbidity was alcohol consumption 62.2% (n=659), and the dominant risk factor was unprotected sex 92% (n=977). The prevalence of pre-core mutations was 36.8% (N=1082/2937). ALAT was greater than normal in 19.2% (n=205) and ASAT greater than normal in 14.4% (n=153). High viral load was found in 30.2% (n=327) and low quantitative HBsAg was found in 77.1% (n=229) of the study population. The median of HBV DNA was 533IU/mL (IQR=95-3036) and that of quantitative HBsAg was 5160 IU/mL (IQR=1161-12515). On transient elastography, 17.7% (n=77) showed significant fibrosis. FIB-4 and APRI score showed 4.9% (n=49) and 6.3% (n=63) of patients with significant fibrosis respectively. Cirrhosis and HCC were present in 1.3% (n=14) and 0.2% (n=2) respectively. Significant fibrosis on transient elastography was independently associated with the male gender ($p=0.033$, $OR=1.954$, 95% CI: 1.056-3.615) and ASAT greater than the normal value ($P=0.027$, $OR=2.659$, 95% CI: 1.118 – 6.323).

Conclusion: pre-core mutations were predominantly found in male patients. The most common comorbidity and risk factors were alcohol consumption and unprotected sexual activity. A great proportion presented with significant fibrosis and high viral load. The male gender and ASAT were associated with significant fibrosis.

Keywords

CHB, HBeAg-negative, Pre-Core mutation, Douala.

List of Abbreviations

ACG: American College of Gastroenterology; ALP: Alkaline Phosphate; ALAT: Alanine Aminotransferase; APRI: Aspartate Aminotransferase to Platelet Ratio Index; ASAT: Aspartate Aminotransferase; AASLD: American Association for the Study of Liver Disease; CCCDNA: Covalently Closed Circular Deoxyribonucleic Acid; CHB: Chronic Hepatitis B; CLIA: Chemiluminescence Immuno Assay; DGH: Douala General Hospital; DLH: Douala Laquintinie Hospital; EASL: European Association for the Study of Liver Disease; ECLIA: Electro Chemiluminescence Immuno Assay; ELISA: Enzyme-Linked Immuno Assay; FIB-4: Fibrosis Biomarker 4; FMPS-UD: Faculty of Medicine and Pharmaceutical Sciences of the University of Douala; GGT: Gamma Glutamyl Transferase; G/L: Giga per Litter; g/dL: Gram per Decilitter; g/L: Gram per Litter; HBeAg: Hepatitis B e Antigen; HBsAg: Hepatitis B Surface Antigen; HBV: Hepatitis B virus; HBV DNA: Hepatitis B virus Deoxyribonucleic Acid; HCV: Hepatitis C virus; HCC: Hepatocellular Carcinoma; HDV: Hepatitis Delta Virus; HIV: Human Immune Deficiency Virus; IGM: Immunoglobulin M; IU/L: International Unit per Litter; IU/mL: International Unit per Millilitter; Mg/L: Milligram per Litter; PCR: Polymerase Chain Reaction; WHO: World Health Organization.

Background

Hepatitis B virus (HBV) infection is a major public health concern and it is the most common cause of viral hepatitis, a leading cause of end-stage liver disease worldwide [1]. Chronic hepatitis B (CHB) virus infection is defined by the presence of hepatitis B surface antigen (HBsAg) in the serum for at least 6 months [2].

About 30 million people worldwide are newly infected yearly and around 296 million persons currently live with CHB [1]. The world health organization (WHO) in 2022 reported that about 1.1 million people worldwide die from complications of CHB such as cirrhosis and hepatocellular carcinoma (HCC) every year [3]. Despite the existence of an effective vaccine recommended by WHO, the burden of CHB remains high, particularly in the Middle East, sub-Saharan Africa and Asia [4-6]. Recent WHO from 2023 estimate a 6.1% prevalence of HbsAg in Africa [7,8]. A meta-analysis in Sierra Leone in 2022 found a pooled HBV seroprevalence of 13.0% [9]. The prevalence of HCC in sub-saharan Africa as reported by Kedar mukthi nuthalapati et al. in 2021 is 2.3% yearly [10]. Meda et al. in Burkina Faso in 2018, found a seroprevalence of 9.1% of HbsAg [11]. The prevalence of HBsAg in Egypt is about 2-8% [12]. In 2022 in Ghana, Madison et al. found a HBV prevalence of 4.3% [13]. Tanzania has a prevalence of HbsAg of 6% found by Kilonzo et al. in 2018 [14]. A meta-analysis in 2017 showed that HBV is endemic in Cameroon with 11.2%, 7.1% in the general population, 10.5% in blood donors, and 9.5% in pregnant women

[15]. The mean age were 45 ± 13 years, 79.3 % males and 20.7 % females [4]. In Cameroon, Ankouane F et al. found 55.9% cirrhosis and 14.75, 47.75, 20.6%, 11.8% and 5.9% amongst those below 30, 30-39, 40-49, 50-59 and 60 years and above respectively [16]. Ndjitoyp Ndam AW et al. in 2022 in Cameroon found 23.4% with cirrhosis and 6.2% with HCC in patients with CHB [17].

Several HBV mutations have been associated with different clinical outcome, including: pre- core(pre-C), core promoter (CP), pre-surface(pre-S), Surface(S) deletion mutations [15,18,19]. The pre-C mutation can be defined as a common genomic alteration that does not produce hepatitis B e antigen (HBeAg) [20]. This mutation introduces a premature stop codon in the pre- C region of the HBV genome, thereby inhibiting HBeAg synthesis [20]. The prevalence of pre- C mutations varies worldwide, ranging from 7–30% [21]. Studies from different regions report varying rates: Malik and al. in 2016 in India found a 48.1% prevalence in CHB carriers, 60% with liver cirrhosis and 94.4% with HCC [20]. According to Wang et al. in 2016, the rate of Pre-C mutations in china was 96.2% in HBeAg negative patients [21]. Angouda BM et al. in 2016 in Congo found a 15.3 % prevalence of HBeAg-negative [4]. Some EN et al. in Burkina Faso in 2021 found a 28.7% prevalence of patients with HBeAg-negative [22]. Ankouane F et al. in 2015 in Cameroon found a Prevalence of 92.1% HBeAg-negative with 6.9% suffering from cirrhosis [16]. According to some EN et al. in Burkina Faso in 2021, the mean age was 38.7 years with a male predominance of 60% and the 25- 44-year-old age group mostly represented [22]. From a virological perspective, this mutation enhances HBV replication by promoting covalently closed circular DNA (cccDNA) stability and resulting in fluctuating viral loads [23]. Histological assessments reveal active liver injury, including inflammation, fibrosis, cirrhosis and progression to HCC [21,23]. This mutation alters the natural history of chronic HBV infection, thus influencing the efficacy of antiviral therapy and highlight the need for mutation- specific clinical management strategies [21].

Cameroon remains a high endemic area for CHB with 11.2% prevalence. Despite this, data on HBV mutations especially the pre-core variant are scarce. This study relies on real-world clinical data to highlight patterns that are already known to clinicians but not formally documented, potentially leading to inadequate clinical monitoring and delayed management. This will bridge the gap between routine clinical practice and research. Therefore, this study aims to describe the virological and histological profiles of chronic HBV carriers with pre-core mutations in Douala.

Methods

Study Design, study Period and study sites

This was a cross-sectional study conducted over ten years, from January 1st to December 31st. The research was carried out from November 1st, 2024, to June 30th, 2025. The study was conducted in the gastroenterology units of four hospitals in Douala, Cameroon.

Two public hospitals (the Douala General Hospital (DGH), Douala Laquintinie Hospital (DLH)) and two private hospitals both equipped with the transient elastography apparatus (Polyclinic Marie O and Centre des Maladies de l'Appareil Digestif (CMAD)). These sites were chosen because they have the same clinical practice, the same routine follow-up of patients and are in the same authorized treatment center (ATC). ATC is a structure established by the Cameroon Ministry of public health for the comprehensive, standardized and specialized care of CHB patients. This structure brings together and coordinates specialist care delivery, primarily involving hepatologists and multidisciplinary teams (including pharmacists and nurses) from the health facilities that make up the ATC. They work together to ensure the availability of advanced diagnosis and approved antiviral therapies. The DGH is one of the centers where hepatologists come together to plan and follow-up patients and ensure adherence to national treatment guidelines.

Study Population and Sampling

The study population consisted of patients diagnosed with CHB. Inclusion criteria were HBeAg-negative patients with complete medical files. Exclusion criteria were co-infection with HDV, HCV, or HIV, and being on treatment at the time of consultation. A consecutive and exhaustive sampling method was used, with the sample size corresponding to all eligible patients identified through a review of medical files during the study period.

Data Collection

Data were collected retrospectively from patient files using questionnaires. The variables studied included:

- Sociodemographic data: age, gender, residence, occupation, marital status, education, and country of origin, risk factors of HBV transmission.
- Past medical history, complications and risk factors: past medical history such as (hypertension, diabetes, alcohol consumption, chronic kidney disease), complications such as (cirrhosis and hepatocellular carcinoma) and risk factors for HBV transmission such as (unprotected sex, blood transfusion, scarifications, dental care).
- Clinical presentations: signs and symptoms such as (asthenia, abdominal pain, jaundice, nausea, anorexia, vomiting, diarrhoea, dark urine), and physical signs such as (general state, ascites, splenomegaly).

General state:

- General biological data: full blood count values were obtained through analysis of the patient's blood sample using electric impedance and colorimetric photometry with the pentra analyser. Haemoglobin levels were expressed in g/dL with a normal range of 12-16g/dL. Platelet standard was [150-400] G/L with 3 groups >400000, 150000-400000, <150000 per mm³. White blood cells were express in G/L with a normal range of [4-10].
- Liver enzymes: levels of ASAT and ALAT (expressed in IU/L with normal values \leq 40 IU/L), which was represented as less than or equal to normal value or greater than normal value. Total and conjugated bilirubin which was either less than

12 or greater than or equal to 12 and less than 2 or greater than or equal to 2 respectively (expressed in mg/L and whose standards was less than 12mg/L, for total bilirubin and less than 2mg/L for conjugated bilirubin), GGT was either less than or equal to 45 or greater than 45IU/L (expressed in IU/L and whose standard was \leq 45IU/L) and ALP was either less than 140 or greater than or equal to 140IU/L (expressed in IU/L and whose standard was less than 140IU/L). These values were obtained through enzymatic kinetic analysis using BIOLABO single reagent kits on the VIDAS multiparametric immunoanalytical machine.

- Liver function tests: Prothrombin time, which was either less than 70% or greater than or equal to 70% (expressed in % and whose standard varied from 70 to 100%). Albuminemia which was divided into 2 groups: less than or equal to 35g/L and greater than 35g/L (expressed in g/L and whose standard was 28-35g/L) were obtained after analysis of the blood sample by spectrophotometry with Bromocresol green on a VIDAS multiparametric immunoanalytical machine.
- Virological parameters: HBeAg was obtained by analysis of the blood sample by enzyme immunoassays (ELISA).
- HBsAg quantification was measured using automated chemiluminescent Microparticle immunoassay (CMIA). Results were expressed in IU/mL and the detectability threshold was >0.05 IU/mL. Values <1000 were considered low and those ≥ 1000 were considered high.
- Hepatitis B viral DNA was expressed in IU/mL. The detection threshold was >10 IU/ml. The viral load was measured by analysis of the blood sample using real-time polymerase chain reaction (RT- PCR). The method used was the COBAST TaqMan HBV test. Values <2000 were considered low viral load and those ≥ 2000 were considered high viral load.
- Histological data: non-invasive methods were used to assess liver fibrosis, including transient elastography, APRI score, and FIB-4 score, which were correlated with the METAVIR score. Transient elastography is a non-invasive, painless test which is like an ultrasound. It allows the gastroenterologist performing the procedure to assess if there is any scarring or stiffness in the liver. It works by measuring shear wave velocity. It has a probe which has a transducer at the end that measures the velocity as it passes through the liver.

The METAVIR score is a standard histological grading system used to assess liver injury in patients with chronic liver disease by assessing the fibrosis and necro-inflammatory activity. Fibrosis is classified from F0-F4 as follows: F0-F1: absent or non-significant fibrosis, F2 or significant fibrosis, F3 or severe fibrosis, F4 or cirrhosis [18].

Operational definition of terms

Chronic hepatitis B (CHB) virus infection: the presence of hepatitis B surface antigen (HBsAg) in the serum for at least 6 months.

- Pre-core mutation can be defined as a specific genetic alteration in the pre-core region of the HBV genome, which

introduces a premature stop codon and prevents the synthesis of HBeAg, with the persistence of HBsAg, with or without ongoing viral replication

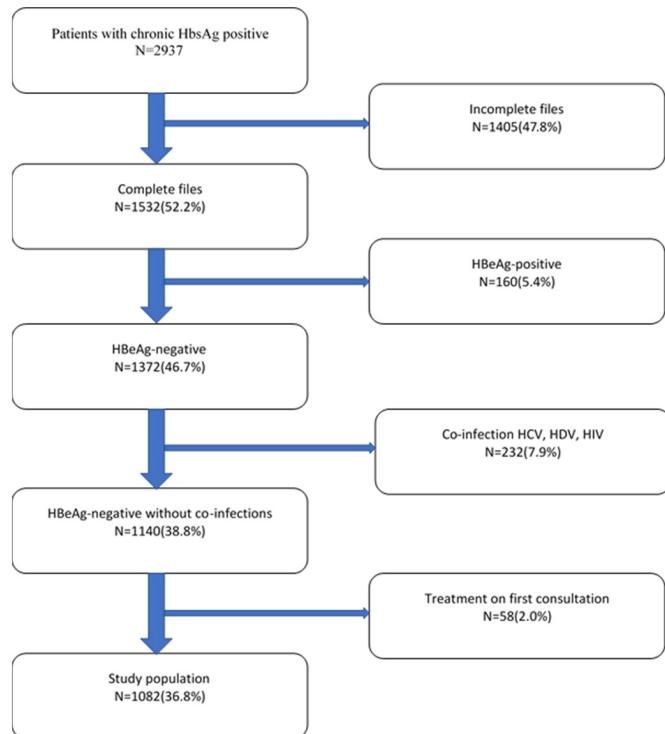
- Incomplete files: files not including age, sex, HBeAg, HBV viral load, fibrosis score.
- Pre-core mutations: patients with HBeAg-negative.
- Virological profile: HBV-DNA.
- Low viral load: HBV-DNA <2000
- High viral load: HBV-DNA ≥2000
- Low HBsAg quantification: HBsAg<1000.
- High HBsAg quantification: HBsAg≥1000.
- Non-significant fibrosis: defined as a METAVIR score <F2.
- Significant fibrosis: defined as a METAVIR score ≥F2.

Data analysis

The data was collected and analysed using SSPS version 26.0. During descriptive analysis, continuous variables were summarized using means and standard deviations or medians and interquartile ranges, as appropriate. Categorical variables were summarized using frequencies and proportions then presented in tables and charts. The chi square test was used to test association between dependent variables and independent variables. Factors associated with significant fibrosis were identified using a univariate, then multivariate binary logistic regression model. The strength of the association was expressed using odds ratio and a two-tailed p-value less than 0.05 was considered statistically significant.

Results

Figure 1: Recruitment flow chart.



We reviewed 2937 files. Overall 28.6%(N=1082) patients were

HBeAg-negative and met the inclusion criteria. The mean age was 35.9 ± 9.9 years, the median was 34 years and the most represented age group 28.6% (n=309) was 34–41 years. The male gender represented 59.3% (n=642) of the study population. The sex ratio M/F was 1.46:1. The main comorbidity was alcohol consumption 62.2% (n=659), and the dominant risk factor was unprotected sex 92.2% (n=977). The most common presenting symptoms was abdominal pain 7.2% (n=77). Complications like cirrhosis and HCC were rare, found in 1.3% (n=14) and 0.2%(n=2) of patients, respectively. Virologically, 30.2% (n=327) of patients had HBV DNA levels ≥ 2000 IU/mL, and 77.1%(n=297) had HBsAg levels ≥ 1000 IU/mL. The median of quantitative HBsAg concentration from 297 patients was 5160 IU/mL (range: 0.01 IU/mL -399000 IU/mL). The median viral load from all participants was 533 IU/mL (range: 0.0-24033780IU/mL). HBV DNA was detectable (greater than 10IU/ml) in 88.4%(n=956) of patients.

Table 1: Characteristics of the general study population.

Variable	Modality	N	Value
Age(years) ±SD		1082	35.9±9.9
Male gender		1082	642(59.3%)
Risk factor	Born to HBV mother	1082	3(0.3%)
Comorbidities	Family history of HBsAg	1082	77(7.1%)
	Alcohol consumption	1082	659(60.9%)
	Diabetes	1082	26(2.4%)
	CKD	1082	8(0.7%)
	Hypertension	1082	49(4.5%)
Clinical characteristics	Jaundice	1082	19(1.8%)
	General state<2	1082	1051(97.1%)
	Cirrhosis	1082	14(1.3%)
	HCC	1082	2(0.2%)
Biological data	Hemoglobin	1082	13.6±4.0
	Platelets	1082	209144.7±84806.4
	ALAT(IU/L)	1082	35.6±59.2
	≤40		863(79.8%)
	>40		205(18.9%)
	ASAT(IU/L)	1082	34.1±52.1
	≤40		913(84.4%)
	>40		153(14.1%)
	GGT (IU/L)	392	37.5±52.2
	Alkaline phosphate (IU/L)	131	170.7±268.1
	Prothrombin time (%)	412	85.4±18.7
	Albumin (g/l)	123	42.4±9.0
	AFP (ng/mL)	132	625.4±7313.1
HBV DNA(IU/mL)	(IQR)	1082	5331(95-3036)
	≥2000		327(30.2%)
	Quantitative HBsAg (IU/mL) (IQR)	297	51601(1161-12515)
	≥1000		229(77.1%)

ALAT*: alanine aminotransferase, ASAT*: aspartate aminotransferase, HBsAg*: hepatitis b surface antigen, CKD*: chronic kidney disease, GGT*: gamma glutamyl transferase, HCC*: hepatocellular carcinom.

Histologically, 22.5% of patients showed significant fibrosis ($\geq F2$)

on transient elastography. Low risk of fibrosis was found in 75.8% and 73.5% of patients using FIB-4 and APRI scores, respectively (Table 1).

Multivariate analysis identified male gender and ASAT greater than normal as factors independently associated with significant fibrosis (Table 2).

Table 2: Factors associated with significant fibrosis using transient elastography.

Variables	Modalities	Transient elastography		OR (CI at 95%)	p value
		$\geq F2$	$< F2$		
Sex	Male	58	213	1.954 [1.056 – 3.615]	0.033
	Female	19	144		
Hypertension	Yes	7	13	2.880 [0.990 – 8.380]	0.052
	No	69	336		
ALAT(IU/L)	\leq Normal	55	290	1.141 [0.523 – 2.492]	0.740
	>Normal	20	57		
ASAT(IU/L)	\leq Normal	55	309	2.659 [1.118 – 6.323]	0.027
	>Normal	20	36		

Table 3: Prevalence of significant fibrosis with respect to various non-invasive markers.

Variable	Modality	n (%)
Transient Elastography	$< F2$	357(82.3)
	$\geq F2$	77(17.7)
APRI score	$< F2$	944(93.7)
	$\geq F2$	63(6.3)
FIB 4	$< F2$	957(95.10)
	$\geq F2$	49(4.9)

Discussion

Sociodemographic characteristics

The most represented age group was 34–41 years (28.2%). This age group is closer to the findings of Some EN et al., in Burkina Faso in 2021 who found 25–44 years [22], Eloumou et al., in 2016 (26–40) [23] and to that of Kamali et al., in Rwanda in 2021 with the 25–54 age group mostly represented [24]. This might be explained by the fact that this group is typically sexually active and most of them might have been contaminated during childhood given that fibrosis takes time to establish itself [23]. The mean age was 35.9 ± 9.9 years. This mean age was similar to the findings of Fouad R et al., in Egypt in 2020 who found a mean age of 33.7 ± 10.6 years [12] and to that of Ankouane et al., in Yaounde, Cameroon in 2015 with a mean age of 35.3 ± 9.8 [16]. This mean age was smaller compared to the findings of Angounda BM et al., in 2016 in Congo who found a mean age of 45 ± 13 years [4]. This mean age could be explained by the fact that CHB is mostly common amongst the youths. Patients are usually younger, as transmission is mostly perinatal and in early childhood [22].

There was a male predominance of 59.3% with a sex ratio of 1.46:1. This is similar to the findings of some EN et al., in Burkina Faso in 2021 who found a male predominance of 60% [22] and differed from that of Kamali et al., in Rwanda in 2021 with a female predominance of 53.8% [24]. This could be due to the fact that males are generally more exposed to the risk factors, mostly the working group, thus available resources for regular monitoring. Also, genetic factors could favor viral persistence in men. Hormonal factors could play a role in women eliminating HBV more than men [22].

Clinical characteristics

The most common comorbidity was alcohol consumption (60.9%), followed by family history of HBsAg (7.1%). This finding was similar to that of Victoria et al., in 2008 in the USA (36.4%) [25]. Alcohol is a risk factor in fibrosis progression when consumption exceeds 50g per day and majority of our patients were occasional consumers as alcohol is known to exacerbate liver damage in CHB virus carriers [22].

The major risk factors included unprotected sex (92.2%) and scarification (16.5%). These findings were similar to that of Silva KM et al., in Alagoas in 2022 [7]. These modes of transmission correspond with those identified by WHO and supported by Ankouane F et al., in 2015.

The most common symptoms were abdominal pain (7.1%) and asthenia (1.8%). This study was in accordance to the findings of Victoria et al. in USA in 2008 (38.1%) asthenia [25]. This could be due to the fact that many patients are diagnosed only when symptoms emerge often prompting consultation. Also, ongoing liver inflammation may arise due to the abolition of HBeAg by mutations.

Physical signs included ascites (1%) and edema (0.5%). This is a characteristic of CHB infection which often remains asymptomatic until advanced liver disease develops. This could be explained by the late diagnosis and poor access to care.

Cirrhosis and HCC were present in 1.3% and 0.2% respectively. This was lower to the studies Fouad R et al., in 2020 (3%HCC) [12] and to that of Ankouane F et al., in 2015 6.1% Cirrhosis [26]. This can be due to the fact that majority of patients were in the inactive phase and mostly young people or that advanced cases remain underdiagnosed [12].

Paraclinical characteristics

The mean of aminotransferase values was 35.6 ± 59.9 and 34.2 ± 52.3 for ALAT and ASAT respectively. 19.2% of patients had ALAT $> N$ and 14.4% of patients had ASAT $> N$. This was lower to that of Ankouane F et al., (32.8% with elevated ALAT) [16]. This is consistent with literature that says many patients are in the inactive phase of the disease whereby ALAT and ASAT fluctuations are not always high. The elevated transaminase levels reflect ongoing liver injury. The mean of albuminemia was 41.9 ± 9.0 with 13.3% in the normal range. That of GGT was 36.8 ± 50.9 with 17.9% ≥ 45 IU/L.

ALP had a mean of 177.3 ± 290.9 with $47.3\% \geq 140$ IU/L. Total and conjugated bilirubin had a mean of 14.8 ± 45.7 and 7.5 ± 24.1 with $21.9\% \geq 12$ mg/L and $59\% \geq 2$ mg/ respectively. AFP had a mean of 691.2 ± 7693.9 with $2.3\% > 200$ ng/mL.

Virological characteristics

HBV DNA showed that 30.2% of patients had elevated viral loads (≥ 2000 IU/mL). This was similar to the findings of Ankouane F et al., in Cameroon in 2015 (29.7%) [16]. This variation illustrates the fluctuating viral replication potential of CHB virus carriers with HBeAg-negative. Quantitative HBsAg was elevated in 77.1%. The median of quantitative HBsAg and HBV-DNA were 5160 and 533 respectively. This could be explained by the fact that the majority of patients were inactive carriers falling in the third phase where the virus is actively replicating in the natural history of CHB. These results could explain the low rate of severe liver disease among our patients, since there is loss of HBeAg without triggering strong immune-mediated liver inflammation.

Histological characteristics

About 22.5% of the study population had significant fibrosis on transient elastography. These results are in line with Fouad R et al., in Egypt in 2022 where 30.9% had significant fibrosis [12], Eloumou et al., in 2019 (22.2%) [25] and lower to that of Some EN et al., in 2021 in Burkina Faso with 40.9% [22]. The FIB 4 score and APRI score showed that 75.8% and 73.5% had a low risk of fibrosis while 2.1% and 3.3% had a high risk of fibrosis respectively. These results could be explained by the fact that majority of the patients had normal liver biochemical values.

Prevalence of pre-core mutations amongst CHB carriers

The prevalence of pre-core mutations was 36.8%. This result was higher to that of Some EN et al., in Burkina Faso in 2021 with 28.7% and lower to that of Ankouane F et al., in Cameroon in 2015 with 91.9% prevalence [16] and Malik et al., in India in 2018 (59.5%) [20]. This difference may be due to the varying sample size. This is attributed to the natural progression of the disease where HBeAg seroconversion occurs. In our setting, many chronic carriers are infected during childhood mainly through horizontal transmission. The slow rollout of birth-dose vaccination programs in several parts of Sub-Saharan Africa also contributes to the current pattern of the disease.

Factors associated with significant fibrosis

The associated factors with significant fibrosis on univariate analysis on transient elastography were the male gender, hypertension, ALAT, ASAT. These results were in accordance with those of Some EN et al., in Burkina Faso in 2021, found the male gender and diabetes to be associated with significant fibrosis [22,26]. Ankouane F et al., in 2015 in Cameroon found the male gender and high viral load to be associated with significant fibrosis [26]. Fouad R et al., in 2020 in Egypt found elevated ASAT, conjugated bilirubin, splenomegaly, jaundice and dark urine [12]. Factors associated with significant fibrosis on multivariate analysis were the male gender, ($P=0.033$, $OR=1.954$ (95% CI: 1.056-3.615) and ASAT greater than the normal value ($P=0.027$, $OR=2.659$,

95% CI: 1.118 – 6.323).

Conclusion

CHB virus carriers with pre-core mutations are mostly found in young males with a sex ratio of 1.46/1. The mean age was 35.9 ± 9.9 years and the median was 34 years. The prevalence of CHB virus carriers with pre-core mutations was $1/3^{rd}$ of the total population. Elevated viral load and quantitative HBsAg was found in 3/10 and 7/10 patients respectively and significant fibrosis was found in 2/10 patients on transient elastography. Factors associated with significant fibrosis were the male gender and ASAT greater than the normal value.

Ethics Approval

Ethical approval from the ethics committee of the University of Douala, Cameroon, was obtained (N° 4768IEC-Udo/05/2025/T). Our study adheres to the recognized standards of the Declaration of Helsinki.

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