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Epidemiological, Clinical and Etiological Aspects of Adult Hepatomegaly at the Gabriel Touré University Hospital of Bamako in 2023

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Abstract

Summary: Hepatomegaly is one of the clinical signs commonly encountered in chronic liver disease. The aim of our study was to study the epidemiological, clinical and etiological aspects of hepatomegaly in the hepato-gastroenterology department (HGE) of the Gabriel Touré university hospital. This was a cross-sectional study that took place from September 2021 to October 2022 in the department. All patients aged 18 years and older with hepatomegaly, hospitalized or examined by physicians were included. We collected 100 cases of hepatomegaly, representing 3.7% of 2661 patients examined or hospitalized in the HGE department during the study period. These patients had a mean age was 46.7 ± 16.10 years with extremes of 18 and 76 years. The sex ratio was 2.7. Jaundice was the most commonly reported history with a frequency of 26%. Pertaining to patients' lifestyle, alcohol consumption was the most common feature with 6%. The most common associated clinical signs were jaundice, ascites, and edema of the lower limbs. Painful hepatomegaly (86%), with a sharp lower edge (74%), a firm consistency (69%), and an irregular surface (52%) was frequently observed. Cytolysis (75.5%), increased alphafetoprotein levels (70%), microcytic anemia (37%), hyperleukocytosis (54.3%), and biological hepatocellular insufficiency syndrome (low albumin with 30.4%, high bilirubinemia with 61.2% and low platelets with 33.9%) were the most common observed laboratory abnormalities. Hepatitis B virus markers (61%) were the most frequently found in the study patients. At ultrasound examination, heterogeneous hepatomegaly was observed in 87.3% of the patients. Esophageal varices (43.1%) were more commonly seen than other varices during upper gastrointestinal endoscopy. The

dominant etiology was hepatocellular carcinoma on cirrhosis with 66% followed by cirrhosis. **Conclusion:** Painful hepatomegaly was quite frequently in our urban setting hospital with several etiologies. HCC was the most common etiology, therefore measures to prevent it in the community need to be established by the concerned stakeholders to improve adult population health in Bamako.

Keywords

Hepatomegaly, Epidemiology, Clinical, Etiology, Gabriel Toure University Hospital, Bamako

1. Introduction

Hepatomegaly is an increase in the size of the liver (localized or diffuse) with a height of the hepatic arrow greater than or equal to twelve centimeters on the right midclavicular line and/or three centimeters on the xypho-umbilical line [1]. It reflects primary or secondary liver damage [2]. Imaging techniques, particularly abdominal ultrasound, abdominal CT scan and MRI, can highlight the increase in liver size that is clinically suggested, and point to an etiological diagnosis [3]. The etiologies of hepatomegaly are diverse, and can be infectious, immune, metabolic, or toxic [2] [4]. In Mali, the hospital frequency is 3.6% [3]. Given the age of these studies, we felt it necessary to update the data on hepatomegaly. It is in this context that we initiated this work, the general objective of which was to study the epidemiological, clinical and etiological aspects of adult hepatomegaly in the Hepato-Gastro-enterology Department of the Gabriel Toure University Hospital.

2. Patients and Methods

This was a cross-sectional study that took place from September 2021 to October 2022 in the HGE department of the Gabriel Toure University Hospital in Bamako. All patients aged 18 years and older with hepatomegaly, hospitalized or examined by a physician were included. The variables studied were age, sex, occupation, personal history, associated clinical signs, hepatomegaly characteristics, associated biological abnormalities, ultrasound and digestive endoscopy results, etiologies. All patients were informed of the nature of the study and their verbal consents were essential for inclusion.

A recruitment of all patients meeting the inclusion criteria and corresponding to the study periods was carried out. Formula: $n = z^2 \times p \times (1 - p)/m^2$; for $p = 0.036$, $z = 1.96$, $m = 0.05$ n = sample size Z: confidence level according to the reduced centered normal law (for a confidence level of 95%, $z = 1.96$, for a confidence level of 99%, $z = 2.575$) p = estimated proportion of the population that has the characteristic $1 - p$ = expected proportion in the population m = tolerated margin of error, after calculation we obtained as sample size n = 53.

The data were collected on a survey sheet and analyzed with software to compare the results, which were significant for a $p < 0.05$.

3. Results

We collected 100 cases of hepatomegaly out of 2661 patients who consulted or were hospitalized in the hepatogastroenterology department, *i.e.* a frequency of 3.7%. The mean age was 46.7 ± 16.10 years with extremes of 18 and 76 years (Figure 1). The sex ratio was 2.7. Jaundice was the most common antecedent with 26%. As a way of life, alcoholism was more common with 6%. The most frequently encountered associated clinical signs were jaundice, ascites and edema of the lower limbs (Table 1, Figure 2). Painful hepatomegaly (86%), with a sharp lower edge (74%), a firm consistency (69%), and an irregular surface (52%) were more frequent (Table 2). Cytolysis (75.5%), increased alphafetoprotein levels (70%), microcytic anemia (37%), hyperleukocytosis (54.3%), and biological hepatocellular insufficiency syndrome (low albumin with 30.4%, high bilirubinemia with 61.2% and low platelets with 33.9%) were the most common laboratory abnormalities. Markers of the hepatitis B virus were the most represented with 61%. On ultrasound, heterogeneous hepatomegaly was common with 87.3%. Esophageal varices were more represented on upper gastrointestinal endoscopy with 43.1%. The dominant etiology was HCC over cirrhosis with 66% followed by cirrhosis (Figure 3). There was a statistically significant association between age group and dominant etiological diagnosis with p = 0.0044. There was a significant difference between dominant etiology (hepatocellular carcinoma "HCC") and sex with p = 10^{-7} (OR = 9.76, IC = [4.40 - 21.6]).

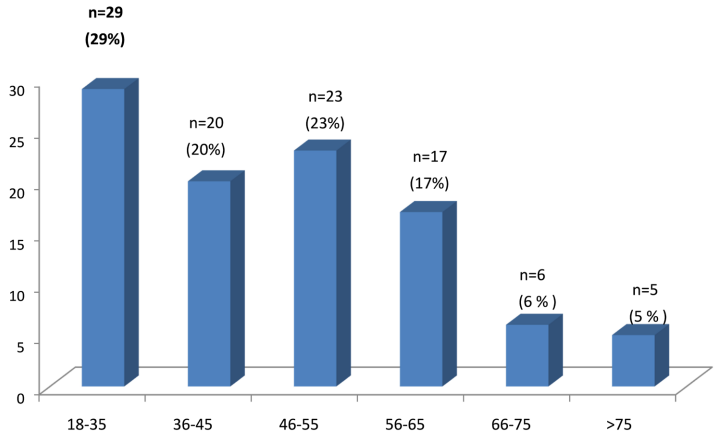


Figure 1. Age group.

Table 1. Character of hepatomegaly.

Characteristics of hepatomegaly		Effective n = 100	%
Consistency	**Firm**	**69**	**69**
	Hard	**31**	**31**
Surface	**Regular**	**48**	**48**
	Irregular	**52**	**52**
	painless	**14**	**14**

Continued

Sensitivity	painfull	86	86
Bottom edge	Sharp	74	74
	Mousse	26	26

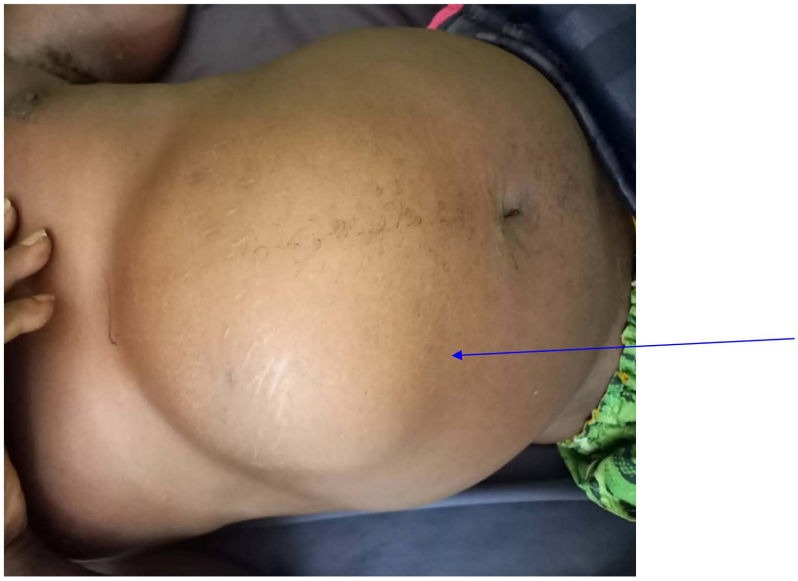
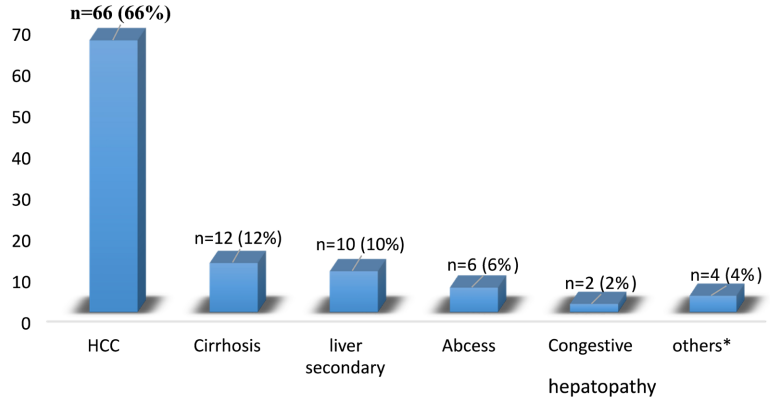


Figure 2. Hepatomegaly (arrow).

Table 2. Clinical signs.

Clinical signs	Effective n = 100	%
General signs		
Pallor	23	23
Febricle/fever	18	18
Weight loss	3	3
Functional signs		
Lower limb edema	47	47
Pruritus	3	3
Transit disorder	3	3
Digestive hemorrhage	10	10
Physical signs		
Jaundice	49	49
Ascite	48	48
Collateral venous circulation	16	16
Splenomegoly	10	10
Hepatojugular reflux	1	1



OR = 9.76; IC = [4.40 - 21.6]; $p = 10^{-7}$.

Figure 3. Etiology.

There was a significant association between the associated clinical signs and the dominant etiology (hepatocellular carcinoma) with $p = 10^{-7}$. Irregular surface, painful tenderness, and sharp liver lower edge were significantly associated with dominant etiology (HCC) with $p = 10^{-8}$. There was a significant link between the ultrasound aspects of heterogeneous hepatomegaly and the dominant etiological diagnosis (HCC) with $p = 10^{-8}$ (OR = 7.26, IC = [3.69 - 14.29]).

4. Discussion

As a limit of the study, the limited financial resources of the patients and sometimes the poor technical plateau did not make it possible to carry out desired and appropriate complementary examinations in all patients such as the lack of liver biopsy puncture in the service.

The mean age was 46.7 ± 16.10 years with extremes of 18 and 76 years. Our results are similar to those of Sang so Lee [5] in 2010 in Korea, with a mean age of 49.3 ± 14.2 years, Bouglouga O in Togo [6] who had found an average age of 44 ± 16 years with extremes of 17 and 80 years, and those of Yassibanda in Mali in 2004. with an average age of 42 years [7].

In our series the sex ratio was 2.7, similar to the Bouglouga O study in 2011, which had a sex ratio of 2.5 [8], Yassibanda with a sex ratio of 1.7 [9], Maiga with 1.63 [10]. This male predominance is explained by the high exposure of men to risk factors such as alcohol consumption, chronic HBV infection or HCV. Estrogen, through its inhibitory effect on the secretion of interleukin 6, also contributes to better protection in women [11].

Our patients' histories were dominated by jaundice with 26% of cases. Yassibanda also had jaundice as a dominant antecedent in 48.6% of cases [7] on the other hand Bouglouga O had jaundice in 3.8% of cases [6].

In our study, the frequency of hepatomegaly was 3.7% of all consultations and hospitalizations. This frequency is comparable to that of Konate A [3], which had regained a frequency of 3.6% of consultations. However, it seems to be lower than the data in the African literature [6] [7] [12]-[14] with 24.14%, 16.45%, 9.6%, 14.3%

and 36.60% respectively. These differences in the frequencies of hepatomegaly could be due to selection bias related to variability in the work methodology.

In our series, hepatomegaly was painful in 86% of cases, this painfulness would be due to the dominant etiology in our study. This would be similar to those of Konaté A [3] and Klotz F [13] in Gabon who had found painful hepatomegaly in 71.8% and 70% of cases respectively. On the other hand, painless hepatomegaly was found in 33.9% of cases in Togo [6]. It was firm in 69% of our cases. This result was lower than that of Klotz F [13] in Gabon, where hepatomegaly was firm in 88.1% of cases, unlike the Togo series [6] which had regained the hard consistency of hepatomegaly in 26.3% of cases. Hepatomegaly in our series was irregular in 52% of cases. This result was similar to that of Konaté A [3] who also found irregular surface hepatomegaly in 59.1% of cases, different from that of Klotz F [13] in Gabon in which hepatomegaly was smooth regular in 71.8% of cases. The appearance of the sharp lower edge of hepatomegaly was found in 74% of cases in our series, higher than the results of Konaté A and Klotz F which had found 57.2% and 59.1% of cases respectively [3] [13]. These differences found according to the characteristics of hepatomegaly could be explained by the etiological variability found in each series of studies.

The clinical signs frequently encountered were: ascites (48%), oedema of the lower limbs (47%), Yassibanda [7] had frequently found ascites in (32.8%), and oedema of the lower limbs in (28%). Bouglouga O had found an even higher frequency of ascites with (66%) [6].

These signs indicate a late course of the causative disease [2] [15]. They can also reflect the fact that patients came late to the consultation, or the fact that there was a delay in diagnosis for better care. In our study, cytolysis was found in 75.5% of the patients, this frequency is higher than the one reported by Bouglouga O [6] who had also found cytolysis syndrome as a dominant biological abnormality in 52.6%. In our study, hypoalbuminemia was observed in 30.4% of patients, and low platelets in 33.9% of them. Pertaining to hypoalbuminemia, Bouglouga O [6] reported a similar result with a 35.9% frequency within his study patients as well as a relatively low frequency of PT (27.6%). All these biological signs could reflect the late evolution of the etiologies found in our study.

In our series, ultrasound aspects of heterogeneous hepatomegaly were frequent with 87.3% higher than that of Yassibanda S [7] who had also found a predominance of heterogenous hepatomegaly in 69% of the patients. This heterogenous hepatomegaly feature could be due to the manifestations of the different dominant causes found in our various studies [3] [10].

In this study, the etiologies of hepatomegaly were dominated by hepatocellular carcinoma in (66%) of cases. This result is comparable to that of Samake S [16] which also found hepatocellular carcinoma as the dominant cause. In the Lohoues Kouakou M.J series [17], and Bouglouga O [6], hepatic cirrhosis not degenerated into HCC was the leading cause of hepatomegaly in 35.2% and 53.2% of cases, respectively. Yassibanda S also found hepatic cirrhosis at the decompensated stage

as the most common cause of liver disease (50.49% of the patients) [18].

5. Conclusion

Painful hepatomegaly was frequently encountered. The etiological diagnosis requires a careful approach to the diversity of causes. Therefore, given the large range of etiologies dominated by HCC. Universal vaccination of children at birth can significantly reduce the incidence of this cause.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix N

Survey card: No.

I- Identity of the patient:

1- Name:.....

2- First name: .....

3- Age: / ___ / ___ / 4- Sex: / ___ / (1 = masculine, 2 = female)

5- Occupation: .....

6- Address / Contact: .....

II- HOSTENCE (Examination): 1 = Yes, 0 = no

7- Jaundice: []

8- Transfusion: []

9- Alcohol: []

10- Bilharziosis: []

11-Other: .....

III- Characters of hepatomegaly (Physical exam):

12- Size: / _____ / cm (LMC or LXO)

13- Flasted consistency: / ___ / (1 = farm, 2 = hard, 3 = soft)

14- SurfaceRegular smooth: [] (1 = yes, 0 = not)

15- Sensitivity:

- Dollore: [] (1 = yes, 0 = not)

- Pain: [] (1 = Yes, 0 = not)

16- Lower edge:

- Tranging: [] (1 = Yes, 0 = not)

- Foam: [] (1 = Yes, 0 = not)

17- Breath: [] (1 = Yes, 0 = not)

18- RHJ: [] (1 = Yes, 0 = not)

19- EXPANsystolic: [] (1 = yes, 0 = not)

Iv- signs:

20- Jight: [] (1 = Yes, 0 = not)

21- ascites: [] (1 = yes, 0 = not)

22- OMI: [] (1 = Yes, 0 = not)

23- CVC: [] (1 = yes, 0 = not)

24- Splenomegaly: [] (1 = Yes, 0 = not)

25- Temperature: []

26- Pruritus: [] (1 = Yes, 0 = not)

27-Big vesicle: [] (1 = yes, 0 = not)

28-Troubles of the transit to be specified .....

29- Loss (weight loss): / ___ / ___ / (kg)

30- Paleter: [] (1 = Yes, 0= not)

31-TR: .....

32- Other to specify: .....

V- ExaminationsPara clinics:

33-Transaminases: ASAT:/ ..... / Alt: / ..... /

34A-blood albumin: / ..... / 34B-βγ: /..... /
34C-GGT: /. ..... /
35- Alkaline phosphatase: .....
36A- Bilirubin Total: / ..... /36B- Bilirubin conjugate: /
+ ..... /
37- Ascites liquid: .....
38has-Tp/ ...../ 38b-factorv: / ..... /
39- Hemogram: HB: / .... / VGM:/...../Plaquettes:/...../
GB: / ..... / lymphocytes:/...../
40- AMBIAN serology: .....
41- Viral markers: .....
42-EchographieAbdominal and / or abdominal TDM: .....
43-Endoscopy digestivehigh: .....
44-Cytology or histology .....
45-Other to specify: .....

VI-ETIOLOGICAL DIAGNOSIS:

....