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Spontaneous Infection of Ascites Fluid at the National and University Hospital Hubert Koutoukou Maga in Cotonou: Prevalence and Associated Factors

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Abstract

Background: Spontaneous ascites fluid infection (SAFI) is an extremely serious and frequently encountered complication in cirrhotic patients. We aimed to determine the prevalence of SAFI and the factors associated with it in the largest hospital in Cotonou. **Methods:** This was a retrospective descriptive and analytical study conducted from January 2013 to July 2019, at the National and University Hospital Hubert Koutoukou Maga (CNHU-HKM) in Cotonou, Benin. All patients followed in the University Clinic of Hepato-Gastroenterology and diagnosed with SAFI were included. **Results:** Eighty-two patients were included, predominantly males (69.5%), with a mean age of 51.5 ± 14.5 years. Among them, 32 had SAFI, *i.e.*, a prevalence of 39%. Of the 32 cases of SAFI, the culture of ascites fluid was positive in 6 cases (18.7%). The most frequent germ found in SAFI was *Escherichia coli* (5 patients, 83.3%). The factors associated with SAFI in this study were: abdominal pain ($p = 0.004$), increased bilirubinemia ($p = 0.009$), decreased prothrombin level <50% ($p = 0.007$), cloudy macroscopic appearance of the fluid ($p < 0.001$), ascites protide level <15 g/L ($p = 0.001$), and severe cirrhosis, with a high Child Pugh C score or MELD score >20 ($p = 0.001$). **Conclusion:** SAFI was common in cirrhotic patients in the department. Certain clinical and paraclinical factors were associated with it, as was the severity of cirrhosis. Early diagnosis and aetiological management of cirrhosis could reduce its frequency.

Keywords

Ascites, Infection, Cirrhosis, Cotonou

1. Introduction

Spontaneous ascites fluid infection (SAFI) is a formidable complication during cirrhosis, occurring in approximately 20% - 35% of patients hospitalised with ascites, especially those with cirrhosis [1] [2]. SAFI results from the seeding of ascites fluid by bacteria from the digestive tract; this is favoured by abnormal digestive permeability associated with an increase in microbial proliferation [3]. Patients with reduced antimicrobial activity and reduced opsonising capacity of ascites fluid proteins are more likely to develop spontaneous bacterial peritonitis [4]. The germs most likely to induce SAFI are gram-negative bacilli (especially enterobacteria), probably due to their high adhesion capacity; and gram-positive cocci [3] [5] [6].

SAFI has a poor prognosis, as it is responsible for a worsening of the disease at the origin of the ascites, with a mortality rate of between 10% and 50% [7] [8]. In the West African sub-region, few studies have been carried out on ISLA. In Benin, work by Kom Mogto [3] in 1999 showed that the prevalence of ISLA was 33.3% and that cirrhosis was the most common aetiology among patients with ISLA, accounting for 81.8% of cases. The lack of recent studies in Benin on this serious pathology in cirrhotics justifies the interest of this work.

The aim of this study was to determine the prevalence of SAFI and the germs involved, as well as the clinical and biological characteristics and factors associated with its occurrence.

2. Methods

This was a retrospective descriptive and analytical study conducted from January 2013 to July 2019 (80 months). All patients with ascites who consulted the Hepato-Gastroenterology University Clinic of the CNHU-HKM of Cotonou, aged 15 years or more and with a medical record including a cytobacteriological examination (CBE) of the ascites fluid, were included. Of the 3787 patients seen in the department during this period, 150 presented with ascites. Sixty-eight cases were excluded from this study: those with tuberculosis or peritoneal carcinosis, and incomplete records (absence of CBE of ascites fluid, protein levels in ascites fluid and/or clinical signs).

The dependent variable was spontaneous ascitic fluid infection.

Positive diagnosis of SAFI was retained if the number of neutrophils in ascites was 250/mm^3 with positive ascitoculture (Spontaneous Bacterial Peritonitis: SBP) or negative (neutrocytic ascites with negative culture: ANCN); or the number of neutrophils < 250/mm^3 and bacteriascity. Thus, we had 32 cases of spontaneous infection of ascites fluid.

The independent variables are sociodemographic, variables relating to antecedents, clinical, paraclinical, therapeutic and evolutionary data. Data was collected using a standardised questionnaire.

We searched for possible statistical associations between SAFI (dependent variable) and several other independent variables using the Pearson Chi^2 test or the Fisher test depending on the case or an ANOVA model.

The accepted statistical significance threshold was 5%. If necessary, the student test, the Odds ratio was used with a 95% confidence interval.

The information collected in the medical records was treated in strict confidentiality. Anonymity has been respected in the processing of data.

3. Results

3.1. Characteristics of the Study Population

Overall, the population consisted of 82 patients with ascites. There was a male predominance (69.5%) with a sex ratio of 2.3. The mean age was 51.5 ± 14.5 years. The majority were Fon and related ethnic groups (70.7%). Shopkeepers and the like were the most represented with a percentage of 24.4%.

3.2. SAFI Prevalence and Sociodemographic Characteristics of Patients

Of the 82 patients included, 32 had SAFI, *i.e.* a prevalence of 39%.

The mean age of patients with SAFI was 51.9 18.0 years (with extremes of 19 years and 80 years). Males predominated in this sample of patients with SAFI with a sex ratio of 3.5. Shopkeepers and the like accounted for the majority of the population with SAFI, with a percentage of 31.3%.

3.3. Clinical, Biological and Evolutionary Characteristics of Patients with SAFI

The history of viral hepatitis B (25%, n = 8) and C (15.6%, n = 5) and chronic alcoholism (21.9%, n = 7) are the most represented in the group of patients with SAFI.

The main reason for consultation was ascites (97.5%, n = 80). The associated functional signs were dominated by abdominal pain (78.1%, n = 25), followed by diarrhea (18.8%, n = 6). As general signs, most of the patients had a fever (34.4%, n = 11), with a WHO Performance Status Index of 3 (25.6%, n = 21). Physical examination revealed jaundice in 34.4% (n = 11) of cases and hepatomegaly in 48.8%. Ascites fluid was cloudy in 40.6% (n = 15) of cases; and 86.7% (n = 13) of subjects with a cloudy ascites fluid had infected ascites. Cirrhosis was the main causal disease of ascites in patients (81.2%, n = 26).

Ascites fluid culture was positive in 6 cases. The germs identified were *Escherichia coli* (*E. coli*) in 5 cases and *Staphylococcus aureus* in 1 case. Patients with SAFI had neutrocyte ascites with positive culture (*Spontaneous bacterial peritonitis*), 3.1% had neutrocyte-free monomicrobial bacteriology, and 81.3% of pa-

tients had neutrocyte ascites with negative culture (NANC) (Table 1).

As other biological abnormalities, total bilirubin averaged 54.6 53.4 mg/L, prothrombin 51.1% ± 16.3%, Gamma GT 427.1 ± 794.5 IU/L, AST 220.2 ± 352.5 IU/L, creatinine 16.3 ± 18.2 mg/L, intra ascitic protein 14.6 ± 13.5 g/L and albumin 35.6 ± 3.6 g/L.

Furthermore, 68% (n = 17) of patients had a Child Pugh B score and 71.4% (n = 10) of those with a Child Pugh C score developed SAFI. This was also observed in 81.8% (n = 9) of patients with a Meld Score greater than 20.

At the evolutionary level, SAFI was treated with ceftriaxone in all cases. Cure was obtained in 37.5% (n = 12) of subjects. Among the 32 patients with SAFI, 13 patients (40.6%) died and 11 (34.4%) were lost to follow-up.

Comparatively, there are more deaths in the group of patients with SAFI than in the group of patients without SAFI: 40.6% (n = 13) in the group of SAFI versus 20% (n = 10) in the group without SAFI with p-value = 0.049.

Among patients with SAFI, 40.6% died at the first episode of ascitic fluid infection. However, the exact cause of death could not be proven in this work.

3.4. Factors Associated with SAFI

In univariate analysis, the independent variables associated with SAFI were the existence of abdominal pain (p = 0.004), the risk being increased by 4.2 times compared to an ascitic subject without SAFI (Table 2); ascites cloudy fluid (p = 0.001), increased bilirubinemia (p = 0.009) or Aspartate aminotransferase (ASAT) (p = 0.018), prothrombin rate (PR) less than 50% (p = 0.007), albumin less than 35 g/L (p < 0.001), and ascites fluid protide less than or equal to 15 g/L (p = 0.001) (Table 3). Cirrhosis was also associated with SAFI (p < 0.001), increasing the risk by 8.4 times; 60.5% of cirrhotic subjects had a SAFI against

Table 1. Cytobacteriological characteristics of ascites fluid from patients at the HGE university clinic of CNHU-HKM (2013-2019).

	SIFA		Univariate Analysis		
	Yes	No	OR	CI (95%)	p-value
Neutrophils (/mm^3)					
≥250	32 (100%)	0 (0%)	-	-	<0.001*
<250	0 (0%)	50 (100%)	-	-	-
Culture					
Positive	6 (100%)	0 (0%)	-	-	0.0013*
Negative	26 (34.7%)	49 (65.3%)	1	-	-
Macroscopic Aspect					
Citrine Yellow	10 (25%)	30 (75%)			
Brown	0 (0%)	5 (100%)			
Trouble	13 (86.7%)	2 (13.3%)			
Purulent	0 (0%)	3 (100%)			
Hematic	9 (47.4%)	10 (52.6%)			0.001*

*Statistically significant link.

Table 2. Relationship between spontaneous ascitic fluid infection and clinical characteristics of patients at the CNHU-HKM HGE university clinic (2013-2019).

	SAFI		Univariate analysis		
	Yes	No	OR	CI (95%)	p-value
Mean age (years)	51.9	50.4			0.073
Gender					
Male	25 (43.9%)	32 (56.1%)	-		0.222
Female	7 (28%)	18 (72%)	-	-	
Fever					
Yes	18 (66.3%)	18 (36%)	2.28	0.92 - 5.65	0.11
No	14 (43.8%)	32 (64%)	1		
Abdominal pain					
Yes	25 (78.1%)	23 (46%)	4.19	1.53 - 11.46	0.004*
No	7 (21.9%)	27 (54%)	1		
Jaundice					
Yes	11 (34.4%)	15 (30%)	1.22	0.47 - 3.15	0.808
No	21 (65.6%)	35 (70%)	1		
Hepatic Encephalopathy					
Yes	4 (12.5%)	2 (4%)	-	-	0.28
No	28 (87.5%)	47 (94%)	1		
Cirrhosis					
Yes	26 (60.5%)	17 (39.5%)	8.41	2.90 - 24.35	0.000*
No	6 (15.4%)	33 (84.6%)	1		

*Statistically significant link.

Table 3. Relationship between spontaneous ascitic fluid infection and biological characteristics of patients at the HGE University Clinic of CNHU-HKM (2013-2019).

	SAFI		Univariate analysis		
	Yes	CI (95%)	No	CI (95%)	p-value
Leukocyte in ascites fluid	2438.03	1221.73 - 3654.32	257.58	197.66 - 317.49	0.001*
Intra-ascitic proteins	14.59	9.71 - 19.48	24.81	20.98 - 28.65	0.001*
Total Bilirubin	54.66	35.39 - 73.93	26.22	17.28 - 35.15	0.009*
Prothrombin rate	51.14	45.26 - 57.01	62.96	56.60 - 69.31	0.007*
ASAT	220.24	93.14 - 347.33	62.38	38.94 - 85.81	0.018*
Albumin	35.65	34.27 - 37.02	30.85	30.08 - 31.61	0.000*
Proteinemia	69.56	67.30 - 71.82	61.14	59.42 - 62.85	0.000*
MELD score					
<20	16 (35.6%)	29 (64.4%)	-	-	
>20	9 (81.8%)	2 (18.2%)	-	-	0.005*
No information	7 (26.9%)	19 (73.1%)	-	-	
Child Pugh					
Class A	1 (12.5%)	7 (87.5%)	-	-	
Class B	17 (68%)	8 (32%)	-	-	0.001*
Class C	10 (71.4%)	4 (28.6%)	-	-	
No information	4 (21.1%)	15 (78.9%)	-	-	

*Statistically significant link.

15.4% of non-cirrhotic subjects (Table 2). Patients with a Child Pugh C and B score were more affected by SAFI than classes A, thus showing correlation with cirrhosis severity (p = 0.001). The same was true for patients with a MELD score greater than 20 (p = 0.005) (Table 3).

4. Discussion

SAFI is the second leading cause of bacterial infection in cirrhotics (after urinary tract infection) in western countries [9]. In developing countries, especially in black Africa, there are very few published data available on the subject. The prevalence of SAFI in the University Clinic of Hepato-Gastroenterology during the period of this study was 39%. This result is similar to those of Kom Mogto [3] in Benin in 1999 and Diarra [10] in Mali in 2007, which were 33.3% and 35.9% respectively. It is also close to that of Rimola *et al*. [11] in Barcelona which was 46%. In addition, other studies were conducted in hospitalized ascitic patients where the frequency of occurrence of SAFI was much lower than that found in our study. Thus, the prevalence of SAFI in our study is higher than those found by Laah Njoho [12] in a prospective study in Yaoundé in 1998 which was 17.9%. Our result is higher than that reported in several European studies which is 10% to 30% [8] [13] [14]. This difference in results is related to the fact that patients in our study population had several risk factors for ascites fluid infection that are: advanced age (54% of patients were older than 50 years), severe hepatocellular insufficiency (mean prothrombin rate = 51%, mean albuminemia = 35 g/L), low concentration of intra-ascitic proteins equal to 14 g/L, increased mean bilirubinemia (54 mg/L) and impaired renal function with mean creatinine at 16.36 mg/L.

In our study the factors associated with SAFI were abdominal pain, increased bilirubinemia, a decrease in TP < 50%, a macroscopic cloudy appearance of the fluid, a protide level in ascites < 15 g/L, and a severe cirrhosis score of Child Pugh C or a high Meld score. SAFI is often pauci symptomatic and systematic discovery by biological examinations of ascites fluid [15]. The clinical signs of SAFI are important to consider when they are present but the diagnosis is primarily paraclinical (cytological and bacteriological examination of ascites fluid), especially in asymptomatic patients. Abdominal pain is the only clinical sign associated with SAFI (p = 0.004) in our study. Among subjects with SAFI, 78.1% had abdominal pain. This result is superimposed on that of Bouzaidi *et al*. [16] who found as clinical signs associated with SAFI abdominal pain and thermoregulatory disorders (hypo or hyperthermia) in a respective proportion of 84% and 66%. In our study, the macroscopic aspect of ascites fluid was associated with the occurrence of SAFI (p = 0.001). Indeed, 86.7% of subjects with ascites fluid disorder had infected ascites and among patients with SAFI 40.6% had a cloudy fluid. This result is comparable to that of Maïga *et al*. [17] and that of Diarra [10] who reported respectively a frequency of 45.5% and 42.9% of cloudy fluid in their population of patients with SAFI. However, our result is lower than that of

Attia *et al*. [18] in Abidjan who found a frequency of 66.7%. However, a citrine yellow liquid does not rule out SAFI.

Thus, in our group of patients with SAFI, 81.2% of patients had cirrhosis as the cause of ascites and statistically a cirrhotic subject has 8.4 times the risk of developing SAFI compared to a non-cirrhotic subject. This result is similar to those of Maïga *et al*. [17] and Kom Mogto [3] who noted 87.5% and 81.8% of cirrhotic patients in the group of patients with SAFI, respectively. In cases of cirrhosis, hepatocyte functions are impaired and the body's defense capacity against germs is reduced; this explains the high frequency of SAFI in cirrhotic patients [7]. The group of patients free from SAFI in our study also consists mainly of cirrhotics with a percentage of 34%, we can conclude that SAFI can occur in any patient with ascites, but more particularly in cirrhotics.

The plasma biological disturbances observed in our series are more significant in the group of patients with SAFI than in the group with sterile ascites and there is a statistically significant link between prothrombin rate and SAFI ($p = 0.007$), between albuminemia and SAFI ($p < 0.001$) then between ASAT and SAFI ($p = 0.018$). In patients with SAFI, the mean prothrombin rate value is 51.1% ± 16.3% and the mean serum albumin is 35.6 ± 18.2 g/L. These results are superimposable to those of Kom Mogto [3] in Benin and those of Diarra [10] in Mali who found in their series respectively for average prothrombin rate 41.19% ± 19% and 50.21% ± 14.7% and for mean albuminemia respectively 28.6 ± 7 g/L and 34.8 ± 16.9 g/L. Furthermore, Attia *et al*. [18] in Ivory Coast found that 83.3% of patients with SAFI had a prothrombin rate less than 50% and that 75% of these patients had a serum albumin less than 20 g/L. This shows that ascitic patients with hepatocellular insufficiency have a high risk of developing SAFI, as confirmed by the work of Dever *et al*. [7]. In our series, the average ASAT level in patients with SAFI of 220.2 IU/L is higher than that of patients without SAFI which is 62.4 IU/L. This result is similar to that of Kom Mogto [3] who found that the average ASAT level in patients with infected ascites is higher than that of patients without SAFI (178 IU/L in patients with SAFI compared to 76 IU /L in patients without SAFI). This proves that ascitic patients with hepatic dysfunction are more susceptible to the development of SAFI. According to our study, there was a correlation between the severity of cirrhosis and SAFI. High Child-Pugh and MELD scores are a factor favoring the development of SAFI. Thus, 68% of cirrhotic patients classified as Child-Pugh B and 71.4% of those classified as Child-Pugh C had developed SAFI. In addition, 81.8% of patients with a MELD score greater than 20 had SAFI. These results are in agreement with those of Amoako *et al*. [19] in Accra who found in their study that 80% of patients with infected ascites were classified Child-Pugh C and had a MELD score greater than 15. Furthermore, a study conducted in Portugal by Cristina *et al*. [20] in 2019 also showed that the majority of cirrhotics who developed an ascitic fluid infection were classified Child-Pugh B or C and these patients had a high MELD score (greater than 20). Indeed, patients with high Child-Pugh

scores (B and C) and MELD present a severe alteration of hepatocyte functions with a reduction in the secretion of proteins (Immunoglobulins and albumin) involved in the body's defense against germs [19] [20]; which explains the high risk of occurrence of SAFI in these patients.

The average intra-ascitic protein level in patients with infected ascites is 14.6 g/L, lower than the average in patients with sterile ascites which is 24.8 g/L. In addition, there is a statistically significant link between the intra-ascitic protein level and SAFI ($p = 0.001$). Subjects with low intra-ascitic protein levels are more likely to develop SAFI. This finding is consistent with the literature [1] [2] [20] [21] [22]. For most authors, the risk of SAFI is very high if the protein level in the ascitic fluid is less than 10 g/L.

All patients with SAFI in our series received a 3rd generation cephalosporin (Ceftriaxone). This antibiotic has proven sensitivity to almost all germs that frequently induce ascitic fluid infections according to literature data [8] [23] [24]. But Piroth *et al*. [25] suggested that the combination of 3rd generation cephalosporins + Amoxicillin-clavulanic acid, Cotrimoxazole + Amoxicillin-clavulanic acid are effective options in cases of severe infection or lack of improvement after 48 hours antibiotic therapy.

Among patients with SAFI, 40.6% died at the first episode of ascitic fluid infection. This result is compatible with that found by the European Association for the Study of the Liver in 2010 which was 10% to 50% [8]. Our result is, however, lower than those of Kom Mogto [3], Diarra [10] and Maïga *et al*. [17] who respectively found 90.9% in their series; 69.6% and 75% deaths.

This difference in results could be linked to the fact that 34.4% of patients in our study population were lost to follow-up. This significant rate of loss to follow-up could be explained by the lack of financial means to honor care by patients with the possibility of resorting to traditional medicine. Comparatively, there are more deaths in the group of patients with SAFI than in the group of patients without SAFI (40.6% in the SAFI group versus 20% in the group without SAFI with p-value = 0.049). SAFI therefore worsens the prognosis of the disease-causing ascites with an increased risk of mortality.

5. Conclusion

The prevalence of SAFI is high in our series 39%, with *Escherichia coli* as the most common germ. It would be associated with a low protein level in ascites and severe underlying cirrhosis. The puncture with cytobacteriological examination of the ascitic fluid, which represents the only reliable diagnostic method for SAFI, must be systematic in any patient with ascites; it must be followed by an ascites-culture. The worsening of SAFI seriously impacts the prognosis of cirrhotic patients. Therefore, there is a need for early antibiotic therapy in suspected cases of ascitic fluid infection. Antibiotic prophylaxis must be systematic in cirrhotic patients at high risk of developing SAFI, especially if the protein level in ascites is less than 15 g/L. Prevention and early treatment could reduce mor-

tality in cirrhosis.

Authors' Contributions

All authors participated in the active writing and editing of the article. All authors read and approved the final version of the manuscript.

Limitations of the Study

This was a retrospective study, so a lot of information was missing from the files used. The collection was done using a questionnaire with 7 main sections, there could be information bias. The study was carried out in the largest university hospital in the city of Cotonou; however, the data cannot be generalized on a national scale.

Conflicts of Interest

The authors declare no conflict of interest.

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