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Original article

Prevalence and risk factors for the occurrence of spontaneous ascitic fluid infection (SLAI) in cirrhosis

Prévalence et facteurs de risque de survenue d'une infection spontanée du liquide d'ascite (ISLA) au cours de la cirrhose

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Résumé

Introduction : L'infection spontanée du liquide d'ascite est une complication fréquente au cours de la cirrhose et associée à un mauvais pronostic.

Méthodologie : Il s'agissait d'une étude prospective, transversale portant sur tous les patients hospitalisés dans le service d'hépato-gastro-entérologie durant la période d'étude allant du 1er Janvier 2021 au 31 Décembre 2021.

Résultats : Sur un total de 707 patients hospitalisés durant la période d'étude dont 70 patients cirrhotiques retenus avec 28 cas de patients ISLA. La prévalence de l'ISLA chez les cirrhotiques dans notre série était de 40%. L'âge moyen de nos patients était de 43 ans. Le sex-ratio H/F était de 2,11. Les principaux signes cliniques étaient : la fièvre (78,57%), la douleur abdominale (67,86%) et l'hémorragie digestive (35,71%). L'aspect macroscopique du liquide d'ascite était trouble dans la moitié des cas (50%) et citrin dans 35,71% des cas. En analyse univariée, il existe un lien significatif entre la douleur abdominale p=0,01 ; la fièvre p= 0,00 ; l'aspect trouble du liquide d'ascite p=0,00 et l'ISLA. Les principaux facteurs de risque de survenue de l'ISLA sont : le taux de protéines dans LA < 15 g/l (60,71%), Bilirubine totale > 30 mg/l (50%), des plaquettes < 98 G/L (42,86%), une ponction du liquide d'ascite (35,71%), une créatininémie > 12 mg/l (25%) et une hémorragie digestive (17,86%). Sur le plan thérapeutique, (46,43%) et (21,43%) de nos patients avaient reçu respectivement de la ceftriaxone et de la norfloxacine. L'évolution sous traitement était favorable dans plus de la moitié des cas (60,71%) et défavorable avec décès des patients dans 39,29% des cas.

Conclusion : L'ISLA est un événement potentiellement grave au cours de la cirrhose et est et est associée à une mortalité élevée.

Mots-clés : infection spontanée du liquide d'ascite, cirrhose, facteurs de risque, antibiotique, Conakry.

Abstract

Introduction: Spontaneous infection of ascitic fluid is a frequent complication during cirrhosis and associated with a poor prognosis.

Methodology: This was a prospective, cross-sectional study covering all patients hospitalized in the hepato-gastroenterology department during the study period from January 1, 2021 to December 31, 2021.

Results: Out of a total of 707 patients hospitalized during the study period including 70 cirrhotic patients included with 28 cases of ISLA patients. The prevalence of ISLA among cirrhotics in our series was 40%. The average age of our patients was 43 years. The M/F sex ratio was 2.11. The main clinical signs were: fever (78.57%), abdominal pain (67.86%) and digestive bleeding (35.71%). The macroscopic appearance of the ascites fluid was cloudy in half of the cases (50%) and citrine in 35.71% of cases. In univariate analysis, there is a significant link between abdominal pain p=0.01; fever p = 0.00; the cloudy appearance of the ascitic fluid p=0.00 and the ISLA. The main risk factors for the occurrence of ISLA are: protein level in LA < 15 g/l (60.71%), total bilirubin > 30 mg/l (50%), platelets < 98 G/L (42.86%), ascitic fluid puncture (35.71%), serum creatinine > 12 mg/l (25%) and digestive bleeding (17.86%). Therapeutically, (46.43%) and (21.43%) of our patients had received ceftriaxone and norfloxacin respectively. The outcome under treatment was favorable in more than half of the cases (60.71%) and unfavorable with patient death in 39.29% of cases.

Conclusion: ISLA is a potentially serious event during cirrhosis and is associated with high mortality.

Keywords: spontaneous ascitic fluid infection, cirrhosis, risk factors, antibiotic, Conakry.

Introduction

Spontaneous ascites fluid infection (S.E.F.I.) is defined as the occurrence of non-tuberculous bacterial peritonitis without an apparent intra-abdominal septic focus. It is a frequent, recurrent and severe complication, reflecting a pejorative evolutionary modality of the underlying liver disease [1, 2].

Management has improved significantly over the past 30 years, and in-hospital mortality has fallen from 90 % to around 20 % [3].

Ascites fluid infection (EFI) is an extremely serious complication, occurring in around 20- 30% of cirrhotic patients, especially when the level of protides in the fluid is below 10 g /l [4, 5, 6].

This infection has a poor prognosis, as it is responsible for worsening hepatocellular insufficiency, deteriorating renal function and increased portal pressure, with a high risk of ruptured esophageal varices [4, 7].

This seriousness calls for particular vigilance in cirrhotic patients with ascites, and early, appropriate treatment in cases of suspected infection, even in the absence of biological criteria. Primary antibiotic prophylaxis may be indicated in subjects at high risk of infection [4, 7].

The diagnostic difficulty and severity of ISLA means that it must be systematically investigated in the presence of ascites, particularly in cirrhotic patients [4, 7].

Worldwide, there are numerous publications on cirrhosis, and in particular on its complications, such as oedemato-ascitic decompensation. In sub-Saharan Africa, work on ISLA in cirrhotic patients is rare.

In Côte d'Ivoire, the only publication available on this subject is that of Attia, based on a sample of 12 cases [8]. Although decompensated cirrhotic patients are frequently hospitalized in our department, publications on ISLA involving a large sample of cirrhotic patients are not available. In Guinea, to our knowledge, there are no data available to date to motivate this study.

The aim of this work was to determine the prevalence of ISLA and the factors associated with the occurrence of ISLA in cirrhotic patients.

Methodology

This was a prospective, cross-sectional, analytical study of all patients hospitalized in the hepato-

gastroenterology department of the Donka National Hospital from January 1er 2021 to December 31, 2021.

We included all cirrhotic patients with spontaneous ascites fluid infection (defined as a neutrophil count greater than 250 elements/mm3 in ascites fluid).

We excluded all non-cirrhotic patients, cirrhotic patients with ascites of other etiologies (tubercular, carcinomatous, cardiac, renal), cirrhotic patients with unexplored ascites, cirrhotic patients with incomplete medical records and those who had undergone an abdominal operation in the forty days preceding the diagnosis of ISLA. Data collected were: socio-demographic (age, sex, lifestyle, alcohol consumption), clinical (reason for consultation or hospitalization, patient's personal history, functional, physical signs: vital constants); macroscopic appearance of ascites fluid (citrine, cloudy or hematic) biological data: Hemogram, transaminases (ALAT, ASAT), total bilirubinemia, prothrombin rate, creatinemia, protidogram (total proteins, albuminemia, search for beta gamma block), analysis of ascites fluid (cytology, leukocyte and lymphocyte count, protide level assay), cytobacteriological examination of urine; virological data (HBV, HCV) for etiological research.

The following elements were considered as risk factors:

- Digestive hemorrhage
- Iterative aspiration of ascites fluid
- Platelet count below 98,000/mm3
- Total bilirubin over 30mg/L
- Ascites fluid protide level below 15g/L
- Creatinine levels above 12mg/L

Radiological parameters: abdominal ultrasound or CT scan to assess intra-abdominal organs, especially to look for hepatic dysmorphia, signs of portal hypertension (ascites, splenomegaly, intra-abdominal collateral circulation), nodules suspicious of hepatocellular carcinoma (HCC) and HCC kinetics: hypodense nodule without contrast injection and hypervascularization at arterial time, with drop in contrast at portal time; this is known as "wash out".

A chest X-ray to look for a pulmonary infection or pleural effusion.

Oeso-Gastroduodenal fibroscopy to search for esophageal varices, gastric varices, portal hypertension gastropathy, antral vascular ectasia and other lesions (gastro6duodenal ulcer, gastropathy).

For prognosis, the CHILD-PUGH score was used. Treatment consisted of antibiotics alone or in combination with human albumin 20% for patients who could obtain it. The course was favorable, with improvement in clinical signs, and unfavorable, with the patient's death.

Data entry was carried out in EPI-INFO VERSION 7.54.0

The tests used to analyze the data were Pearson's Chi-square test, the corrected Chi-square test and Fisher's exact test.

The alpha threshold has been set at 5%.

Results

Seventy cirrhotic patients were selected, including 28 cases of ISLA. The prevalence of ISLA in cirrhotics in our series was 40%. The mean age was 43 years, with extremes of 22 and 72 years, and the sex ratio was 2.11 in favor of men.

The reasons for consultation were: oedemato-ascitic syndrome (60.71%), abdominal pain (21.43%), digestive haemorrhage (35.71%), hepatomegaly (7.14%), disturbed consciousness (3.57%), abdominal pain associated with fever and digestive haemorrhage (3.57%).

The main clinical signs were: fever (78.57%), abdominal pain (67.86%), digestive haemorrhage (35.71%), jaundice (46.43%), hepatic encephalopathy (32.14%), splenomegaly and hypothermia with frequencies of 32.14% and 3.57% respectively (Table I).

The macroscopic appearance of ascites fluid was cloudy in half the cases (50%), citrine in 35.71% and hematic in 14.29%. The neutrophil count was between 300 and 1000 elements/mm3 in 64.28% of cases. The mean leukocyte count was 2911/mm3 with extremes of 270 and 9500/mm3 . The mean neutrophil count in

ascites fluid was 4742/mm3 with extremes of 270.1 and 9215/mm3 .

In univariate analysis, there was a statistically significant association between abdominal pain p=0.01; fever p= 0.00; cloudiness of ascites fluid p=0.00 and ISLA. The main risk factors for the occurrence of ISLA were: protein level in ascites fluid < 15 g/l (60.71%), hyperbilirubinemia > 30 mg/l (50%), platelets < 98 G/L (42.86%), iterative puncture of

ascites fluid (35.71%), creatinemia > 12 mg/l (25%) and digestive hemorrhage at 17.86% (Table II).

In our patients, the progressive stage of cirrhosis was severe: Child-Pugh B at 50% including 10.71% B7, B8(21.43%), B9 (17.86%) and Child-Pugh C at (50%) with C10 (25%), C11

10.71%), C12(10.71%) and C13 (3.57%). Half of our patients had prothrombin levels below 50% and 60.71% had ascites fluid protide levels less than or equal to 15g/L. Urine cytobacteriological examination (UCE) was performed in 14.08% of cases, with germ absence in 10.71% and Escherichia coli in 3.57%. UCE was not performed in 85.71% of cases.

The etiologies of cirrhosis were as follows: hepatitis B virus (HBV) in 57.14% of cases, hepatitis C virus (HCV) in 10.71% of cases, alcohol + HBV in 10.71%, HBV + HCV in 3.57% and undetermined in 17.86% of cases (autoimmune diseases not investigated).

In terms of treatment, (46.43%) and (21.43%) of our patients had received ceftriaxone and norfloxacin respectively, 10.71% albumin 20% and ceftriaxone, norfloxacin + albumin 20% in 7.14%; amoxicillin + clavulanic acid in 14.29% and norfloxacin alone in 21.43%.

Progression under treatment was favorable in more than half the cases (60.71%, n= 17) and unfavorable with death in 39.29% of cases (five cases of hepatic encephalopathy, four cases of digestive hemorrhage and two cases of hepatorenal syndrome; Figure 1).

Table I: Distribution of patients according to clinical signs

Clinical signs (n=28)	Workforce	Percentage (%)
Fever	22	78,57
Abdominal pain	19	67,86
jaundice	13	46,43
Digestive hemorrhage	10	35,71
Impaired general condition	9	32,14
Hepatic encephalopathy	9	32,14
Splenomegaly	9	32,14
Vomiting	7	25
Diarrhea	4	14,28
Hypothermia	1	3,57
Constipation	1	3,57
Dehydration folds	1	3,57

Table II: Distribution of patients according to risk factors for ascites fluid infection

Risk factor	Workforce	Percentage (%)
Digestive hemorrhage	5	17,86
Total bilirubin > 30 mg/l	14	50
Platelets <98000/mm^3	12	42,86
Protein LA < 15 g/l	17	60,71
Creatininemia > 12mg/l	7	25
Ascites fluid puncture	10	35,71
Total	28	100

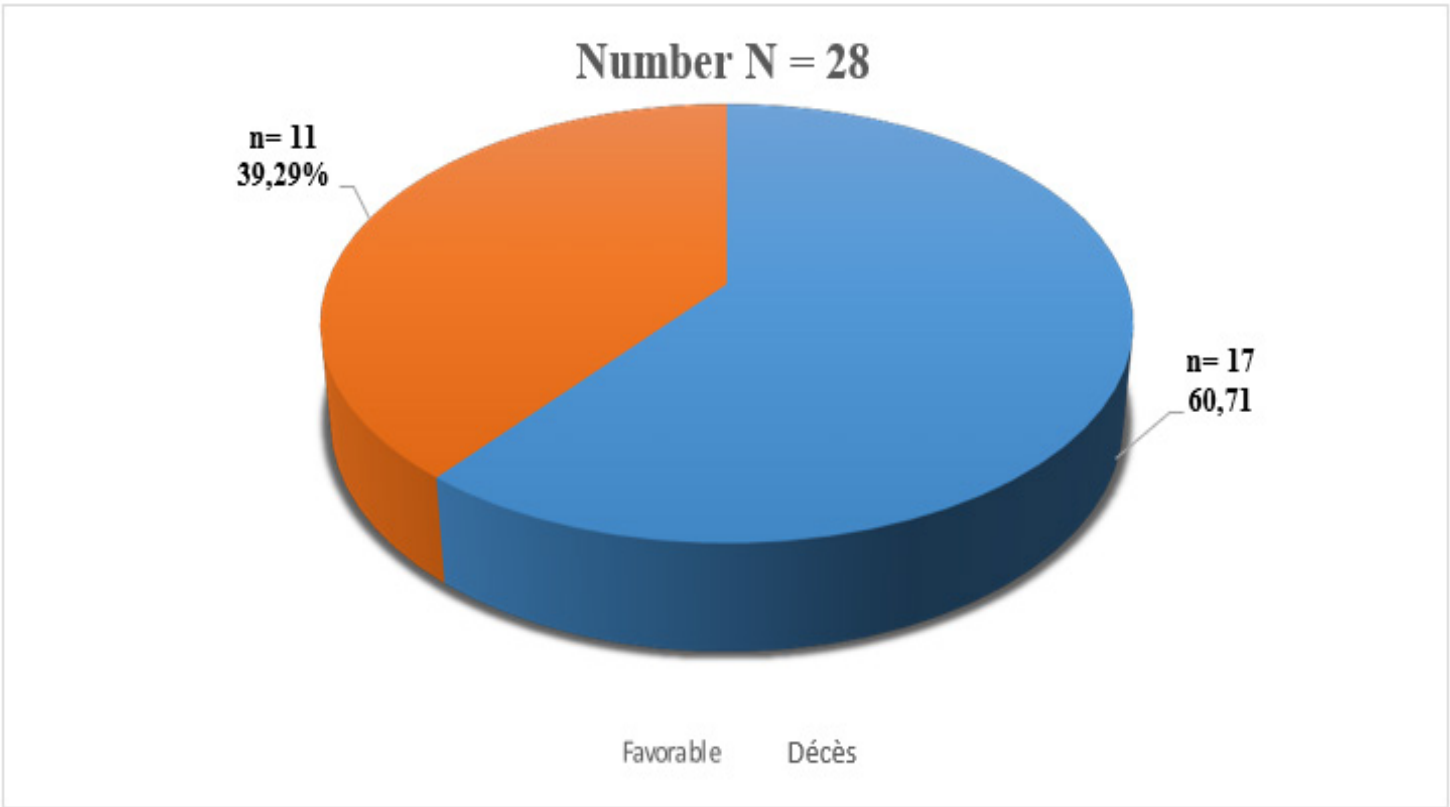


Figure 1: Distribution of patients according to progress on treatment

Discussion

The prevalence of ISLA in cirrhotic patients in our series was by 40%.

Our results are higher than those reported in Dakar [9] and Côte d'Ivoire [8], which were 27.3% and 28% respectively. This may be explained by our larger sample size.

The prevalence of ISLA, which affects around a quarter of cirrhotic patients with ascitic decompensation, and its detrimental effect on prognosis explain the recommendation for its systematic investigation [10].

The sex ratio was 2.11 in favor of men, comparable to those reported in Mali [11] and Senegal

[10], which found a sex ratio of 3 and 1.89 in favor of men respectively.

But in an Ivorian study [8], the sex ratio was 1, and there was no statistically significant difference in the distribution of ISLA according to age and sex.

The mean age of our patients was 43 years; identical to those found by MAIGA [11] and Dia [10] who were 42; 43 years respectively. We note that our cirrhotic patients are young. Younger subjects are more exposed to the risk of HBV and HCV infection, and in Sub-Saharan Africa, hepatitis B infection appears much earlier (around 40 years), which could explain the predominance of this age group [8 ; 9].

The clinical and biological symptomatology of ISLA is non-specific, and signs vary from study to study. In univariate analysis, there was an association between ISLA, abdominal pain, fever and cloudy macroscopic appearance of ascites fluid. In France, abdominal pain, hypothermia or hyperthermia and hepatic encephalopathy are statistically associated with the occurrence of ISLA [12]. Clinical manifestations in ISLA are diverse, but generally represented at highly variable frequencies by fever, abdominal pain and jaundice [3, 13, 14].

In Dakar, DIA et al [9] found the main clinical signs to be abdominal pain (66.66%) and fever (26.66%).

In Côte d'Ivoire, ATTIA et al [8] reported fever (91.7%), abdominal pain (83.3%) and jaundice (41.7%) as the main clinical manifestations of ISLA.

Digestive haemorrhages are also found at high frequencies in these patients; they are not signs of ISLA, but rather a favourable circumstance for its occurrence, justifying prevention by antibiotic therapy and the use of lactulose [3, 13, 15].

Ascites fluid was macroscopically cloudy in 50% of cases and citrine in 35.71%; this result is comparable to those found by DIA et al.[9]

and ATTIA et al [8], who respectively reported cloudy ascites fluid in 53.3% and 66.7% of cases.

The cloudy appearance of ascites fluid is highly suggestive of ISLA, but clear or lemon-yellow aspects do not rule it out [13].

In univariate analysis, we found a statistically significant association between the occurrence of ISLA and the presence of abdominal pain (p= 0.014), fever (p= 0.000), cloudy ascites fluid (p= 0.000) and ISLA.

Similarly, ATTIA et al [8] found a significant association between the presence of abdominal pain (p=0.046), fever (p=0.002), cloudiness of ascites fluid (p=0.018) and ISLA.

DIA et al [9] found a significant association between cloudiness (p=0.000) and ISLA.

In our series, there was no significant relationship between diarrhea (p=0.208), encephalopathy (p=0.442), jaundice (p=0.528) and ISLA.

Our results are similar to those of ATTIA et al[8] in Côte d'Ivoire.

Reported no significant relationship between jaundice (p=0.286), hepatic encephalopathy (p=0.143) and ISLA.

In contrast, DIA et al [9] found no relationship between fever (p=0.26), abdominal pain (p=0.25), hepatic encephalopathy (p=0.63), diarrhea (p=0.51) and ISLA.

The clinical signs statistically associated with ISLA vary widely from study to study. Among these signs, abdominal pain, fever and cloudy ascites fluid are the most frequently reported. They can be considered as guiding elements in the diagnosis of ISLA.

Thus, 60.71% of our patients had a protein level in

LA ≤ 15 g/l. For most authors, the risk of ISLA is very high if the protein level in ascites fluid is below 15 g/l [4, 5, 6, 16]. A low ascites protein concentration of less than 15 g/l increases the risk of ISLA tenfold [3, 17]. Our results are lower than those reported by DIA et al [9], who found an ascites fluid protide level of 20.7 g/l.

In our series, 64.20% (n=18) patients had a neutrophil count (PNN) in ascites fluid between 250 and 1000/mm3.

None of our patients had undergone ascitoculture.

In our patients, the progressive stage of cirrhosis was severe Child B (50%) and Child C (50%), 25% of whom had Child C10.

The cirrhosis was of :

B viral in 57.14% of cases

mixed (HBV and alcohol) in 10.71% of cases

C viral in 10.71% of cases

and of undetermined cause in 17.86% of cases.

Our results are identical to those reported by DIA et al [9], who found 67% of B viral etiology, 2% of alcoholic origin and 31% of undetermined cause, and ATTIA et al [8], who noted 45.3% of B viral etiology, 10% of C viral etiology and 20.9% of undetermined cause.

In uni-variate analysis, in our series, we found no significant relationship between digestive hemorrhage (p= 0.107); protein level in ascites fluid < 15 g/l (p=0.349); thrombocytopenia < 98000/mm3 (p=0.308) ; recent puncture of ascites fluid (p= 0.180); bilirubin >30 mg/l (p= 0.108) and creatinemia >12 mg/l (p=0.742).

This lack of relationship could be explained by the small size of our sample, which could affect the power of the statistical tests.

In addition, all patients with GI bleeding or protein levels below 15g/l were systematically put on prophylactic antibiotic therapy, a practice in line with international recommendations [3, 13, 15].

NAUSBAUM JB [3]; DEVER JP et al.[18] had described as the main risk factors involved in the occurrence of ISLA: severe hepatocellular insufficiency at Child's stage C, low ascites concentration in protides < 15 g/l (x 10 the risk); hyperbilirubinemia>30 mg/l; thrombocytopenia < 98000/mm3 .

In the study by TERG R et al [19], there was no significant difference between the low-protein (<15 g/l) group and ISLA.

These studies show that the risk factors statistically associated with ISLA vary widely from one study to another. Many authors do not find a statistically significant link between these parameters and the occurrence of ISLA, but recommend antibiotic prophylaxis in view of its severity and poor prognosis [3, 13, 19, 20].

The main antibiotics administered to our patients were: Ceftriaxone 2g intravenously per day (46.43%); Norfloxacin (21.43%) 400mg x 2 per day per os; and Amoxicillin-clavulanic acid (14.29%) 1g x 3 per day per os.

Five of our patients had benefited from infusion of 20% human albumin in combination with antibiotics: Ceftriaxone + albumin in three cases (10.71%) and Norfloxacin + Albumin in two cases (7.14%).

These treatments were in line with those of consensus conferences and the recommendations of certain learned societies (European and North American), which advocate the empirical use of third-generation cephalosporin antibiotics as a first-line treatment; an alternative is the combination of Amoxicillin and clavulanic acid, or the use of quinolones such as Norfloxacin, Ofloxacin or Ciprofloxacin [3, 13, 21, 22, 23].

Cytological evolution was assessed after 72 to 96 hours in four patients, with a reduction in neutrophils of at least 50%. The remaining 24 patients did not undergo follow-up puncture.

This low rate of cytological control can be explained by the absence of its systematic prescription by practitioners in our department.

Clinical evolution under treatment was judged by general condition, apyrexia and regression or disappearance of abdominal pain.

The course of treatment in our patients was favourable in 60.71% of cases (n=17) and unfavourable in

39.29% (n=11), resulting in death. This mortality was comparable to that reported by RIMIOLA A et al. who found a mortality of between 10% and 46% [24]. According to SHEER TA et al [25], mortality has fallen from 100% in 1960 to 60-70% between 1970 and 1980, and 30% over the last ten years.

This is due to rapid diagnosis and effective treatment, but above all to the identification of risk factors justifying antibiotic prophylaxis.

While the infectious episode is usually cured, in-hospital mortality is still around 30% for causes not directly infectious: hepatorenal syndrome, digestive haemorrhage, end-stage hepatocellular failure or hepatocellular carcinoma [20].

Conclusion

The prevalence of ISLA in cirrhotic patients in our series is 40%. This study enabled us to identify the main risk factors associated, with or without a causal link, with the occurrence of ISLA. The clinical picture was polymorphous, and abdominal pain, fever and cloudiness of ascites fluid were strongly suggestive of ISLA in cirrhotic patients, with a statistically significant association. ISLA is a potentially serious event in cirrhotic patients, despite significant progress in diagnostic and therapeutic management; mortality in our series was 39.29%.

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References

[1] Becq Giraudon B, Breux Jp, Silvain C, Cazenave Roblot F, Morichau Beachant M. Les infections spontanées du liquide d'ascite chez le cirrhotique .Médecine et maladies infectieuses 1988 ; 8(9) : 375-84.

[2] Zeni F, Tardy B, Comte T, Bertrand JC. Antibiothérapie de l'infection spontanée du liquide d'ascite chez le cirrhotique. Réan Urg 1993 ; 2 (5) : 544-48.

[3] Nousbaum JB. Infection spontanée du liquide d'ascite au cours de la cirrhose. Press Med 2015, https : //dx.doi.org/10.1016/j./pm 2015.07.017.

[4] Grange JD. Infection au cours de la cirrhose. Gastroenterol Clin Biol 2006 ; 30 :891-8.

[5] Guaner C, Solar M, Soriano G, Andreu M, Novela Mt, Villa MC, Sabat M,et al. Risk of first community- acquired spontaneous bacterial peritonitis in cirrhotic with low ascetic fluid protein levels. Gastroenterol Clin Biol 1999 ; 117 :414-9.

[6] Andreu M, Solar M, Stiges- Serra A, Alia C, Gallen M, Villa MC, Coll S, et al. Risk factors for spontaneous bacterial peritonitis in cirrhoses patients with ascites. Gastroenterol Clin Biol 1993 ; 104 :113-8.

[7] Andreu M, Solar M, Stiges- Serra A, Alia C, Gallen M, Villa MC, Coll S, et al. Risk factors for spontaneous bacterial peritonitis in cirrhoses patients with ascites. Gastroenterol Clin Biol 1993 ; 104 :113-8.

[8] Attia KA, N'Dri Yoman T, Sawadogo A,

Mahassadi AK, Bathaix-Yao F, Serme K, Manlan KL. L'infection spontanée du liquide d'ascite chez le cirrhotique africain. Etude descriptive à propos de 12 cas. Bull Soc Pathol Exot 2001 ; 94 (4) : 319-21.

[9] Dia D, Serme YK, Bassène ML, Halim A, Diallo S, Thioubou MA, Mbengue M, Diouf ML. Infection spontanée du liquide d'ascite à Dakar, Sénégal/ Etude der cinquante cinq cas patients cirrhotiques. Médecine et Santé Tropicales 2014 ; 24 : 55-57.

[10] Dever JB, Sheikh MY. Spontaneous bacterial peritinitis : bacterilogy, diagnosis, treatement, risk factors and prevention. Aliment Pharmacol Ther 2015 ; 41 : 1116-1131.

[11] Maiga MY, Traore HA, Dembele M, Bacoum AI, Traore AK, Diallo D, et al. L'infection du liquide d'ascite dans les services de médecine à l'hôpital national du Point G. Médecine chirurgicale digestive 1996 ; 25 : 219-22.

[12] Nousbaum JB, Cadranel JF, Nahon P, Nguyen-Khac E, Moreau R, Thévanot T, et al. Diagnostic accuracy of the Multistix 8 SG reagent strip in diagnosis of spontaneous bacterial peritonitis. Hepatology 2007 ; 45 : 1275-81.

[13] EASL. Directives de pratiques cliniques de l'EASL sur la prise en charge de l'ascite, de la péritonite bactérienne spontanée et du syndrome hépatorénal dans la cirrhose. Journal of hepatology 2010 ; 53 : 397-417.

[14] Pauwels A. Infections bactériennes et cirrhoses. Post'U 2018

[15] Restellini S, Nendaz M, Morard I. Antibioprophylaxie de la péritonite bactérienne spontanée. Rev Med Suisse 2012 ; 8 : 276-81.

[16] Pauwels A. Translocation bactérienne et cirrhose. Hépato6gastro 2008 ; 15 (1).

[17] Riggio O, Angeloni S. Ascitic Fluid analysis for diagnosis and monitoring of spontaneous bacterial peritonitis. World J Gastroenterol2009; 15 (31): 3845-50.

[18] Dever JB, Sheikh MY. Spontaneous bacterial peritonitis: bacteriology, diagnosis, treatment, risk factors and prevention. Aliment Pharmacol Ther 2015; 41: 1116-1131.

[19] Terg R, Fassio E, Guevara M, Cartier M, Longo C, Lucero R, et al. Ciprofloxacin in primary prophylaxis of spontaneous bacterial peritonitis: a randomized, placebo-controled study. J Hepatol 2008 ; 48 :774-9.

[20] Fernandez J, Navasa M, Planas R, Montonliu S, Monfort D, Soriano G, et al. Primary prophylaxis of spontaneous bacterial peritonitis delays hepatorenal syndrome and improves survival in cirrhosis. Gastro-enterology 2007 ; 133 (3) : 818-24.

[21] Runyon BA. Management of adult patients with ascites due to cirrhosis: an update. Hepatology2009; 49 (6): 2087-107.

[22] https : //www.HAS-Sante.fr/portail/ upload/ docs/application/pdf/2008-06/ prise en charge des complications des cirrhoses – argumentaire. Pdf.

[23] Ilach J, Rimola A, Navasa M, Gines P, Salmeron Jm, Gines A, et al. Incidence and predictive factors of first episode of spontaneous bacterial peritonitis in cirrhosis with ascites: relevance of ascetic fluid protein concentration. Hepatology1992; 16:724-7.

[24] Grange JD, Roulot D, Pelletier G, Pariente EA, Denis J, Ink O, et al. Norflaxacin primary prophylaxis of bacterial infections in cirrhotic patients with ascites: a double-blind randomized trial. J Hepatol1998; 29:430-6.

[25] Sheer TA, Runyon BA. Spontaneous bacterial Peritonitis. Dig Dis 2005 ; 23 : 39-46.

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