



Comparative Study between the Efficacy of Oral Gemifloxacin and Intravenous Cefotaxime in Treatment of Spontaneous Bacterial Peritonitis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background and Aims: Spontaneous bacterial peritonitis (SBP) is defined as a bacterial infection of the ascitic fluid (AF) that arises in individuals without an intra-abdominal source of infection that is surgically curable. SBP is a unique complication in cases with cirrhosis-linked ascites, and it can be so subtle that it is only reported by chance when paracentesis is done. SBP is handled with any of a variety of cephalosporins, such as cefotaxime or quinolones, such as Gemifloxacin. The aim was comparing the effect of oral Gemifloxacin versus intravenous cefotaxime in 60 individuals with cirrhosis who had SBP.

Patients and Methods: The randomized controlled research involved 60 cirrhotic ascitic individuals suffering from SBP admitted to Tropical Medicine Department, Tanta University Hospital. Two groups of patients were investigated: group I included 30 cirrhotic ascitic patient suffering from SBP treated with IV cefotaxime 2gm|8 hours for 7 days and group II included 30 cirrhotic ascetic patient suffering from SBP and treated with oral Gemifloxacin 320 mg once daily for 7 days.

Results: No substantial difference was noted among both groups regarding age, sex, or symptoms, or in laboratory tests such as hemoglobin, total leukocytic count, platelet, serum bilirubin, ALT, AST, albumin, prothrombin activity, INR, creatinine, urea, sodium, and potassium. Also, the outcome was

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comparable in both groups as regarding the number of treated patients ($p=0.781$); 20 (66.67%) cases were cured in group I and 21 (70%) cases were cured in group II and in AF analysis after treatment. By using gram stain, gram -ve organism were the predominant in group I 23 (76.7%) and group II 24 (80%) while gram +ve was detected in 7 (23.3%) and 6 (20%) in group I and II respectively. Furthermore, no substantial difference was noted among both groups regarding treatment response based on culture prior to treatment.

Conclusions: Our research is the first to focus on Gemifloxacin use in the management of SBP rather than in the prevention of SBP. Since these agents' relative effectiveness is identical, cost should be the deciding factor. In conclusion, our findings indicate that cefotaxime or Gemifloxacin can be used as a first-line therapy.

Keywords: Cirrhosis; ascitic fluid; resistance.

1. INTRODUCTION

Spontaneous bacterial peritonitis (SBP), which is described as an infection of the ascitic fluid without local infection of abdominal cavity, is one of the major severe and potentially fatal complications of cirrhosis. SBP should be diagnosed and treated initially to improve the patient's chances of survival [1].

Gram-negative aerobic species are the most common source of SBP (75 percent). The remaining cases are occurred with Gram-positive aerobic species, the most common of which are *Streptococcus pneumoniae* or *Viridians* community streptococci [2]. SBP is present in as many as 25% of cases with ascites, resulting in a 20% death rate [3].

Prior to the investigation of SBP, a total nuclear leukocyte count of at least 250 cells/mm ($0.25 \times 10^9/L$) is needed, along with the identification of fluid bacteria without an intra-abdominal pathogen origin) in ascitic fluid [4].

The antibiotic treatment (Third generation cephalosporins) has a high overall efficacy against SBP episodes is the empiric alternative, which is believed to be successful in 80% of patients, as opposed to the ampicillin and's overall medication (Tobramycin) in 56% of the patients The recommended dosage is two grams a day for at least five days out of each week for five weeks to have a maximum impact [5].

Gemifloxacin is an orally administered fluoroquinolone that is used to treat moderate to serious respiratory infections. An extensive number of aerobic gram-positive and gram-negative bacteria are susceptible to Gemifloxacin. It works by inhibiting type II DNA topoisomerases (gyrases), which are necessary

for bacterial mRNA replication and transcription [6]. A 320 mg once daily for 5 to 7 days is the maximum dosage. Furthermore, Gemifloxacin's pharmacological and microbiological properties make it an appealing treatment choice for serious bacterial infections [7]. The aim of the study were to compare the effect of oral Gemifloxacin versus intravenous cefotaxime in 60 cirrhotic ascitic cases with SBP.

2. PATIENTS AND METHODS

This was a randomized controlled study which carried out on 60 cirrhotic ascetic cases suffering from SBP admitted to Tropical Medicine Department, Tanta University Hospital. The research ethics committee of Tanta University's faculty of medicine authorized this study, and each participant provided written informed permission. This research proposal conforms to the accepted ethical standard. Approval code: 32346 This /05/18 .The study period from 10/2018 to 11/2019.

2.1 Two Groups of Patients were Created

Group I: 30 cirrhotic ascetic patients suffering from spontaneous bacterial peritonitis, were treated with IV cefotaxime 2gm|8 hours for 7 days.

Group II: 30 cirrhotic ascetics patients suffering from spontaneous bacterial peritonitis, were treated with oral Gemifloxacin 320 mg |once daily for 7 days.

2.2 The Cases Underwent to

Consent of the patient.

All data king.

Whole clinical exam.

2.3 Routine Laboratory Investigation Including

1. Complete urine and stool exam.
2. Complete blood count.
3. Fasting and 2 hours post-prandial blood sugar.
4. Blood urea and creatinine.
5. Liver function tests (total bilirubin, serum ALT, serum AST, serum total protein, serum albumin, serum alkaline phosphatase and prothrombin time and activity).
6. Viral markers HBsAg and HCV Abs.

2.3.1 Inclusion criteria

1. Adult male and non-pregnant female (more than 18 years).
2. Patients who diagnosed as spontaneous bacterial peritonitis by paracentesis (ascetic fluid (AF) polymorph nuclear cell count is more than $250/\text{mm}^3$).
3. Lack of allergy to any ingredient in Gemifloxacin or to any other quinolone antibiotic.
4. There is no surgically treatable cause of infection in the abdomen.

2.3.1 Exclusion criteria

1. Pregnant or breast feeding female.
2. Allergy to any ingredient in Gemifloxacin or to any other quinolone antibiotic.
3. Cardiac arrhythmias.
4. Bacterial peritonitis secondary to the first (presence of surgically treatable abdominal source of infection).

2.4 Statistical Analysis

SPSS version 25 was used to conduct the statistical analysis (IBM Inc., Chicago, IL, USA). To test the distribution of quantitative variables, the Shapiro-Wilks normality test and histogram were used to decide if parametric or nonparametric statistical testing should be used.

RESULTS

As presented in Table (1), the age between the two studied groups was insignificantly different. Most of the case were males (63.33%) and (60%) in Group I and Group II respectively. As regarding to lab investigations as HB, TLC, platelet, TSB, DSB, ALT, AST, albumin, PA, INR, creatinine, urea, sodium and potassium, there was insignificantly different between the studied groups as shown in Table (2,3).

Table 1. Patients' characteristics in studied groups

		Group I (n = 30)	Group II (n = 30)	P value
Age (y)	Mean \pm SD	56.7 \pm 9.30	53.77 \pm 7.21	0.178
	Range	32-75	30-64	
Sex	Male	19 (63.33%)	18 (60%)	0.791
	Female	11 (36.67%)	12 (40%)	

Age and sex were insignificantly different between studied groups (n means number)

Table 2. CBC parameters in studied groups

		Group I (n = 30)	Group II (n = 30)	P value
Hb (gm/dL)	Mean \pm SD	10.07 \pm 1.89	9.78 \pm 1.81	0.538
	Range	6.4 – 15.4	6.7 – 13.8	
TLC (/mm ³)	Median	6750	5490	0.174
	Range	2900 – 19700	1500 – 17100	
Platelets (/mm ³)	Median	120500	90000	0.268
	Range	27000 – 379000	31000 – 390000	

There was an insignificant difference between both groups as regards CBC parameters

Table 3. Liver and kidney function tests and electrolytes in studied groups

		Group I (n = 30)	Group II (n = 30)	P value
TSB (mg/dL)	Median	2.95	2.45	0.442
	Range	0.6 – 30.9	0.5 – 21.3	
DSB (mg/dL)	Median	1.75	1.15	0.249

		Group I (n = 30)	Group II (n = 30)	P value
ALT (U/L)	Range	0.2 – 20.3	0.2 – 15.3	0.865
	Median	35.5	27	
AST (U/L)	Range	11 – 82	6 – 211	0.636
	Median	51.5	48.5	
Albumin (gm/dL)	Mean ± SD	2.71 ± 0.50	2.56 ± 0.55	0.298
	Range	1.8 – 3.6	1.7 – 3.9	
PA (%)	Mean ± SD	29.93 ± 18.57	52.16 ± 21.21	0.139
	Range	29 – 100	15 – 93	
INR	Mean ± SD	1.52 ± 0.36	1.85 ± 0.87	0.059
	Range	1 – 2.59	1 – 4.78	
Creatinine (mg/dL)	Median	1.30	1.04	0.301
	Range	0.5 – 1.3	0.42 – 2.5	
Urea (mg/dL)	Median	68.5	62	0.584
	Range	18 – 198	16 – 140	
Sodium (mEq/L)	Mean ± SD	132.92 ± 7.75	132.63 ± 5.56	0.865
	Range	116.1 – 148.5	116.1 – 142	
Potassium (mEq/L)	Mean ± SD	4.04 ± 0.83	4.26 ± 0.93	0.343
	Range	2.95 – 6.45	2.8 – 6.54	

Liver and kidney function tests and electrolytes were insignificantly different between studied groups

Table 4. Ascitic fluid analysis before treatment in studied groups

		Group I (n = 30)	Group II (n = 30)	P value
Protein (gm/dL)	Median	1.45	1.7	0.340
	Range	0.1 – 4.8	0.4 – 6.5	
Glucose (mg/dL)	Median	125	150.5	0.340
	Range	10 – 375	78 – 400	
TLC (/mm ³)	Median	775	745	0.196
	Range	570 – 2900	550 – 2500	
Neutrophils (/mm ³)	Mean ± SD	661 ± 155.9	726.66 ± 120.9	0.073
	Range	350 – 900	500 – 900	

There was an insignificant difference between both groups as regards ascitic fluid analysis before treatment. (n means number)

Table 5. Ascitic fluid analysis after treatment in studied groups

		Group I (n = 30)	Group II (n = 30)	P value
Protein (gm/dL)	Median	1	1	0.882
	Range	0.4 – 4.8	0.5 – 4	
Glucose (mg/dL)	Median	100.5	100	0.684
	Range	8 – 362	43 – 376	
TLC (/mm ³)	Median	200	210	0.333
	Range	75 – 2200	50 – 1110	
Neutrophils (/mm ³)	Mean ± SD	65.6 ± 22.49	56.17 ± 15.41	0.063
	Range	15 – 90	30 – 90	

There was an insignificant difference between studied groups according to ascitic fluid analysis after treatment. (n means number)

Table 6. Culture before treatment in studied groups

	Group I (n = 30)	Group II (n = 30)
G +ve	7 (23.3%)	6 (20%)
G -ve	23 (76.7%)	24 (80%)
P value	0.754	

Results of the culture before treatment were insignificantly different between studied groups as regards. (n means number)

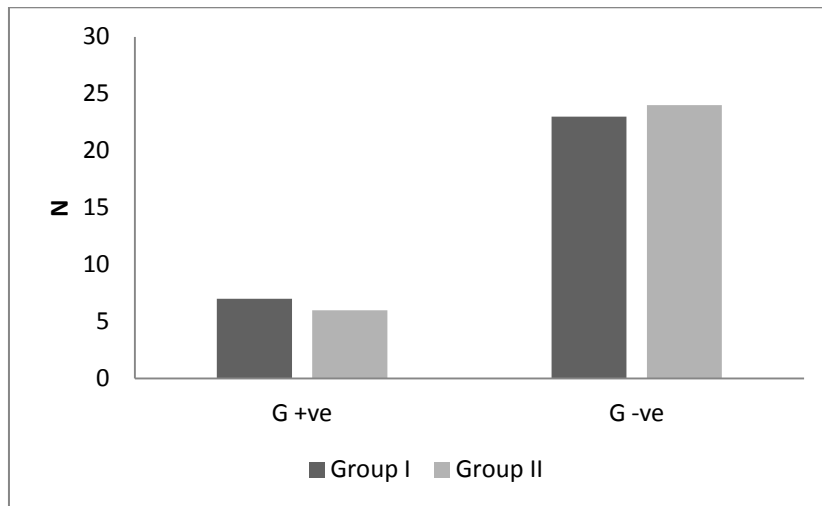


Fig. 1. Culture before treatment in both groups

Table 7. Number of cured patients in studied groups

	Group I (n = 30)	Group II (n = 30)
Cured	20 (66.67%)	21 (70%)
Not cured	10 (33.33%)	9 (30%)
P value	0.781	

There was an insignificant difference between studied groups as regards number of cured patients; 20 (66.67%) cases were cured in group I and 21 (70%) cases were cured in group II. (n means number)

Table 8. Response of treatment according to the culture before treatment in both groups

	Group I (n = 20)	Group II (n = 21)	Total
G +ve	2 (28.57%)	3 (50%)	5
G -ve	18 (78.26%)	18 (75%)	36
P value	0.754		-----

%; percent of the response from the total before treatment

There was an insignificant difference between both groups as regards the response of treatment according to the culture before treatment. (n means number).

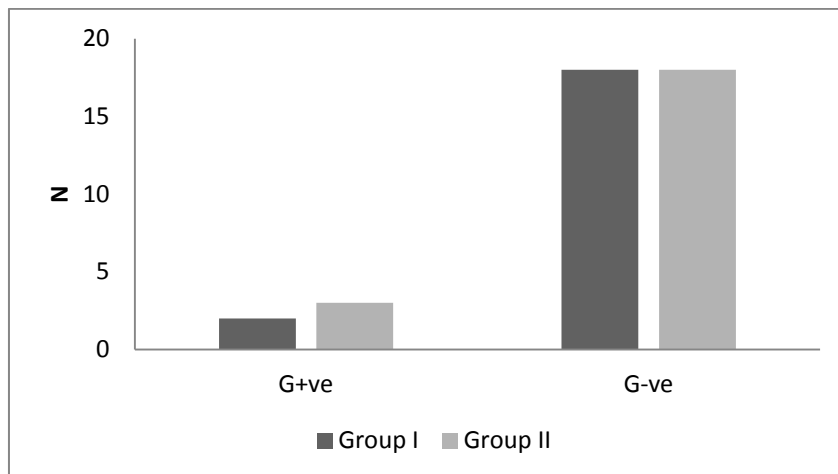


Fig. 2. Response of treatment according to the culture before treatment in both groups

4. DISCUSSION

As regarding to ascitic fluid protein, in group I was 1.45 and 1.7 in group II before the treatment which after treatment changed to 1 in both. In terms of ascetic fluid (AF) protein, before and after treatment was insignificant different between the studied groups. The absence of endogenous antibacterial action in AF with low protein concentrations clarifies minimum in apart, the efficacy of the ascitic fluid protein content as a marker of probability to "spontaneous" ascitic fluid infection. AF's opsonic activity is closely linked to its protein levels and dilution of opsonic proteins below a certain level tends to eliminate the fluid's antibacterial properties [8].

As regarding to AF, glucose in group I was 125 mg/dl and 150.5 mg/dl in group II before the treatment and after treatment changed to 100.5&100 mg/dl. An insignificant difference between both groups as regards AF glucose before and after treatment. Since glucose diffuses easily across membranes, unless bacteria or fluid white blood cells are metabolizing it, the concentration in AF will reflect the plasma concentration.

Estakhri et al., found that SBP patients had significantly lower ascetic glucose levels [9]. Also, reduced AF glucose concentration has been identified in SBP, and This, according to several reports, has proved useful in separating infection and malignant ascites from other causes [10]. However, according to Akriadias and Runyon, the AF glucose was found to be close to that of antiseptic fluid in early SBP, and total glucose presents to have poor analytic sensitivity and specificity, restricting its regular usage [11].

In the present study, before the treatment TLC & Neutrophils was 775& 661 ± 155.9 in group I and 745&726.66 ± 120.9 in group II which decreased to 200& 65.6 ± 22.49 in group I and to 210&56.17 ± 15.41 in group II after treatment. There was an insignificant difference between both groups as regards ascetic fluid analysis (TLC and neutrophils) before and after treatment.

Regarding most recommendations and studies, as suggested by medical experts, the PMN cell count in AF is a diagnostic criterion for SBP, if that gives a reliable indication of the best treatment is as well as the management [9,12].

AF paracentesis and PMN calculation or culture are suitable methods for SBP diagnosis. Even if the culture for bacteria is negative, PMN counts of 250/L in ascites have [13].

A PMN cell count of 250 cells/mm³ or above is significantly predictive with SBP and should prompt the start of empirical antibiotic therapy. Just a small percentage of SBP patients may experience common peritoneal infection symptoms including fever, abdominal pain, and a high blood leukocyte count [14].

By using gram stain, gram -ve organisms were the predominant in group I 23 (76.7%) and group II 24 (80%) while gram +ve were detected in 7 (23.3%) & 6 (20%) in group I&II respectively. There was an insignificant difference between both groups as regards results of the culture before treatment.

on various studies, Escherichia coli, as well as Gram-positive cocci (predominantly Streptococcus species) are commonly isolated in patients with SBP has been found, have been found, were seen in several of patients that have already been found, have already, and have been detected in many of patients with SBP. About 70% of all SBP occurrence are due to These organisms [15].

E. Coli was the microorganism that is accountable (37.0%) of the culture positive patients examined by Cekin et al, staphylococci that lack coagulase in six (22.2%), enterococci in three (11.1%), and ESBL negative E. coli in two patients (7.4 percent) [16].

According to Kim et al., the most frequently cultured microorganism in culture positive cases (n=27) was E. Coli (37.0 percent), followed by staphylococci that lack coagulase (22.2 percent), enterococci (22.2 percent), and coagulase positive staphylococci (22.2 percent) (11.1 percent) [17].

Moreover, according to many studies, the most common microorganism isolated from SBP cases is E. coli [18].

The findings of Wiest et al. are in accordance with the authors' contention that Klebsiehmman et al. as well as the other Enterobacteriaceae are generally known as translocating bacteria responsible for SBP, since they are in the mesenteric lymph nodes [19].

In contrast, Cholongitas et al. showed their outcomes from Athens. Gram-positive bacteria have been shown to be more commonly responsible for culture positive SBP in cirrhotic patients [20].

Also, Campillo et al., Gram-positive pathogens were found to be the most widespread among separates from AF, cultures given from cirrhotic individuals hospitalized with nosocomial SBP, according to the researchers [21].

Jain et al. found that Coagulase-positive *S. aureus* was the organism that was isolated the most often (44 percent), followed by *E. coli* (22 percent) [22].

Fernández et al., found that in cirrhosis, infections caused by Gram-positive cocci had risen dramatically. This might be a phenomenon linked to cirrhotic patients' recent increased level of instrumentation [23].

In the current study, the outcome was comparable in both groups as regarding number of cured patients 20 (66.67%) cases were cured in group I and 21 (70%) cases were cured in group II.

After treatment in group I, 20 (66.67%) were cured, 18 (78.26%) of them were Gram –ve organism and 2 (28.57%) were Gram +ve. While in group II, 21 (70%) were cured, 18 (75%) of them were Gram –ve organism and 3 (50%) Gram +ve.

It is appropriate to administer an antibiotic when SBP test as a suspect colonization occurs, rather than awaiting confirmation of the pathogen or in the results of an in vitro susceptibility test. according to Rimola et al., Cefotrim is the antibiotic that has the best track record of bacterial fluid isolation in patients with SBP, who claims that it protects 95% of flora, and is shown to get into the antibiotic-containing fluid at elevated levels during care and into AF-to-enema concentrations at the moment of administration [24].

Cefotaxime has a broad therapeutic-to-toxic dosage range, and patients with severe infections will receive very high doses of this antibiotic without experiencing any side effects [25].

Many studies have shown that intravenous cefotaxime (2 g per 8 hours) or as like

cephalosporin third generation (for a total course of 5 days) is the most effective therapy for SBP since it removes the most often occurring causal microorganisms: *E. coli*, *Klebsiella pneumoniae*, and *Streptococcus pneumoniae*. Seftinox Cefotaxime has proven to be effective in helping to decrease SBP in the range of 77% to 98% of the cases [26].

Sheer and Runyon have discovered that cefotaxime is the best-studied antibiotic for SBP treatment and is very effective at penetrating ascites without causing nephrotoxicity [27].

The standard treatment for SBP is a 5-day course of third-generation cephalosporin [28]. However, due to the increase in bacterial resistance, its effectiveness has dwindled. Furthermore, it was no longer sufficient for enterococci, which has become a more popular cause of SBP [29]. Failure of first-line treatment is closely related to poor survival outcomes [30].

According to current recommendations for empirical antibiotic therapy of SBP, a third-generation cephalosporin should be begun promptly upon diagnosis of SBP. Until around ten years ago, the usage of third-generation cephalosporins was reported to be very beneficial in the management of SBP, when the majority of the outbreaks and infections were classified into community-acquired were due to Gram-ve bacteria, health care–linked and nosocomial infections was not a term that was often used in clinical practise when admitting to SBP (Setoyama et al., 2019).

However, when comparing community acquired, health care–acquired, and nosocomial SBP in individuals with cirrhosis, the epidemiology of SBP varies (Ariza et al., 2012). It has been observed that patients with nosocomial SBP have a high prevalence of multidrug resistant (MDR) bacteria and fail to respond to third-generation cephalosporins in up to 33%-75% of cases (Fernández et al., 2012).

Our results was supported by *Koulaouzidis et al.*, found that, Third-generation cephalosporins have been the first-line therapy option for SBP; oral quinolones are another alternative (Koulaouzidis et al., 2009).

Quinolones have been used to prevent bacterial infections in cirrhotic and nosocomial prophylaxis at our institution for a long period of time, as they demonstrated a significant decrease in the

prevalence of nosocomial bacterial infections without the expansion of opportunistic infections or significant adverse effects. At the beginning, while norfloxacin, ciprofloxacin, and ofloxacin are all effective antibiotics for the inhibition of bacterial infections in cirrhotic patients, they have a broader antimicrobial spectrum and a higher systemic absorption profile, which may increase the danger of developing infections resulted from gram +ve cocci or drug resistant gram-ve bacilli during long-term therapy. Additionally, the infections that cirrhotic patients get while on quinolone prophylaxis are often due to gram +ve cocci, and a significant prevalence of infections due to gram-negative bacteria resistant to norfloxacin (mostly E. coli) and enterococci has been recorded in long-term treated cases. (Zhang et al., 2010)

Our results were supported by Sader et al., found the fluoroquinolones (ciprofloxacin and ofloxacin) and the cephalosporin cefpirome (fourth generation) were the most active drugs against Gram-negative bacteria. Ofloxacin (98 percent susceptibility) had the broadest breadth of action against SBP isolates of all fluoroquinolones. Cefpirome and the 2:1 cefotaxime-DES-CTX combination displayed the broadest breadth of

efficacy among the β -lactams (93 percent susceptibility).) (Sader et al., 1995).

Angeloni et al. studied in 2008, there were 38 cases of SBP in 32 cases. As an observational procedure, patients were given cefotaxime at a dose of 2 g every eight hours for five days. Patients who did not react to therapy were shifted due to the cultural or observational influences. In 59 percent of cases, cefotaxime treatment was successful, but 41% of episodes needed a change in antibiotic therapy due to a decrease in ascetic PMN count of lower than 25% at 48 hours. In 87 percent of cases, changing antibiotic treatment resulted in the infection being resolved [31].

This gives the rise to conducted more clinical study to search a new safe treatment for SBP. No previous study evaluated the effect of Gemifloxacin broad spectrum quinolone on treatment of SBP.

Our findings were supported by Koulaouzidis et al., who discovered that third Cephalosporins of the first generation are the first-line therapy for SBP.; oral quinolones are another alternative [32].

Chart 1.

SBP therapy and special considerations		
Special Considerations	Antibiotic Therapy	Reasonable Alternative
Standard therapy	Cefotaxime 2 g IV q8 h \times 5 d	Ceftriaxone 1 g IV q12 h or 2 g IV q24 h \times 5 d
Uncomplicated SBP ^a	Ofloxacin 400 mg PO bid \times 8 d is an option	Similar widely bioavailable fluoroquinolone (eg, ciprofloxacin 500 mg PO bid or levofloxacin 500 mg PO q24 h)
Nosocomial SBP	Extended spectrum antibiotics (eg, carbapenems, piperacillin/tazobactam)	Depends on local resistance patterns
Fluoroquinolone or trimethoprim/sulfamethoxazole SBP prophylaxis	Cefotaxime 2 mg IV q8 h \times 5 d	Similar third-generation cephalosporin (eg, ceftriaxone 1–2 g IV q24 h)
β -Lactam hypersensitivity	Ciprofloxacin 400 mg IV q12 h	Levofloxacin 750 mg IV q24 h
Advanced liver or renal failure: serum creatinine greater than 1 mg/dL, blood urea nitrogen greater than 30 mg/dL, or total bilirubin greater than 4 mg/dL	IV cefotaxime 2 g IV q8 h \times 5 d plus IV albumin 1.5 g/kg given on day 1 and 1.0 g/kg given on day 3	—

Abbreviations: bid, twice a day; IV, intravenous; PO, by mouth; q, every.

^a Community-acquired SBP with absence of shock, ileus, gastrointestinal hemorrhage, greater than grade 2 HE, and serum creatinine greater than 3 mg/dL.

Data from Runyon BA, AASLD. Introduction to the revised American Association for the Study of

Quinolones have been used at our institution for a long time to avoid bacterial infections in cirrhotic and nosocomial prophylaxis, as they have shown a considerable reduction in the prevalence of nosocomial bacterial infections without causing opportunistic infections or causing considerable side effects. First, though the antibiotics norfloxacin, ciprofloxacin, and ofloxacin are effective [33].

Furthermore, gram-positive cocci are the most common cause of infections in cirrhotic patients on quinolone prophylaxis, and infections resulted from gram-negative bacilli immune to norfloxacin (primarily *E. coli*) and enterococci have been identified in long-term treated patients [34].

According to Strauss and Caly, Cefotaxime or another third generation cephalosporins are the first-line empirical antibiotics in cirrhotic cases with SBP., and they are effective in around 90% of patients. Broad-spectrum quinolones are recently utilized for oral therapy of uncomplicated SBP because they are completely absorbed upon oral administration and quickly dispersed throughout (AF) [35].

The European Association for the Study of the Liver (EASL) advises empiric antibiotic therapy for SBP with a third-generation cephalosporin, cefotaxime, at a dose of 2.0 gm every 12 hours or every 8 hours for a minimum of 5 days. Alternatives include ciprofloxacin/ofloxacin, but not for cases taking quinolones prophylactically or from areas where fluoroquinolone resistance is common. The American Association for the Study of Liver Diseases' (AASLD) recommendations take the source of the infection into account, as well as any prior antibiotic medication. If there has been no quinolone exposure, vomiting, shock, grade II or elevated encephalopathy, or a creatinine rise of more than 3 mg/dl, oral ofloxacin (400 mg per 12 hours) is prescribed as a second-line medication [36].

5. CONCLUSIONS

SBP therapy is complicated by the appearance of resistant bacteria. Therefore, new antibiotic classes or antibiotic combinations must be created. Antibiotics utilized as empiric first-line therapy should be able to suppress infections that are often associated with healthcare and are frequently helped by antibiotic-resistant bacteria. It's important to consider the characteristics of bacterial infection in a specific geographical region and population. When patients are

healthy, with no bleeding and oral intake restored, cefotaxime can be transferred to oral Gemifloxacin for the treatment of SBP rather than the prevention of SBP.

As a result, the generalization of our results from a monocentric analysis warrants further investigation.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT

It's not applicable.

ETHICAL APPROVAL

The research ethics committee of Tanta University's faculty of medicine authorized this study. This research proposal conforms to the accepted ethical standard. Approval code: 32346 This /05/18.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Garcia-Tsao G. Current management of the complications of cirrhosis and portal hypertension: variceal hemorrhage, ascites, and spontaneous bacterial peritonitis. *Gastroenterology*. 2001;120:726-48.
2. Lata J, Stiburek O, Kopacova M. Spontaneous bacterial peritonitis: a severe complication of liver cirrhosis. *World Journal of Gastroenterology: WJG*. 2009;15:5505.
3. Siple JF, Morey JM, Gutman TE, Weinberg KL, Collins PD. Proton pump inhibitor use and association with spontaneous bacterial peritonitis in patients with cirrhosis and

- ascites. *Ann Pharmacother.* 2012;46: 1413-8.
4. Dever JB, Sheikh MY Review article: spontaneous bacterial peritonitis--bacteriology, diagnosis, treatment, risk factors and prevention. *Aliment Pharmacol Ther.* 2015;41:1116-31.
 5. Rimola A, Garcia-Tsao G, Navasa M, Piddock LJ, Planas R, Bernard B, et al. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document. *International Ascites Club. J Hepatol.* 2000a;32:142-53.
 6. Royhan R. Development of a simple UV spectrophotometric method for analyzing Ciprofloxacin HCl. East West University; 2017.
 7. Petri W. Sulfonamides, trimethoprim-sulfamethoxazole, quinolones, and agents for urinary tract infections. *Goodman & Gilman's The Pharmacological Basis of Therapeutics.* 12th. New York: McGraw-Hill. 2011:1463-76.
 8. Runyon BA, Canawati HN, Akriviadis EA. Optimization of ascitic fluid culture technique. *Gastroenterology.* 1988;95:1351-5.
 9. Estakhri R, Bartari L, Ghojzadeh M. Diagnostic value of serum procalcitonin level in the diagnosis of the spontaneous bacterial peritonitis. *Immunopathologia Persa.* 2020;6:e19-e19.
 10. Lee , Carlson R, Bull D. Early diagnosis of spontaneous bacterial peritonitis: values of ascitic fluid variables. *Infection.* 1987;15:232-236.
 11. Akriviadis EA, Runyon BA. Utility of an algorithm in differentiating spontaneous from secondary bacterial peritonitis. *Gastroenterology.* 1990;98:127-133.
 12. Moore KP, Wong F, Gines P, Bernardi M, Ochs A, Salerno F, et al. The management of ascites in cirrhosis: report on the consensus conference of the International Ascites Club. *Hepatology.* 2003;38: 258-266.
 13. Drobne D, Kurent T, Golob S, Svegl P, Rajar P, Terzic S, et al. Success and safety of high infliximab trough levels in inflammatory bowel disease. *Scandinavian Journal of Gastroenterology.* 2018;53:940-946.
 14. Riggio O, Angeloni S. Ascitic fluid analysis for diagnosis and monitoring of spontaneous bacterial peritonitis. *World J Gastroenterol.* 2009;15:3845-50.
 15. Garcia-Tsao G. Current management of the complications of cirrhosis and portal hypertension: variceal hemorrhage, ascites, and spontaneous bacterial peritonitis. *Gastroenterology.* 2001;120: 726-48.
 16. Cekin Y, Cekin AH, Duman A, Yilmaz U, Yesil B, Yolcular BO. The role of serum procalcitonin levels in predicting ascitic fluid infection in hospitalized cirrhotic and non-cirrhotic patients. *Int J Med Sci.* 2013;10:1367-74.
 17. Kim SU, Kim DY, Lee CK, Park JY, Kim SH, Kim HM. et al. Ascitic fluid infection in patients with hepatitis B virus-related liver cirrhosis: culture-negative neutrocytic ascites versus spontaneous bacterial peritonitis. *Journal of Gastroenterology and Hepatology.* 2010;25:122-128.
 18. Zullo A, Hassan C, Ridola L, Lorenzetti R, Campo SM, Riggio O. Rifaximin therapy and hepatic encephalopathy: Pros and cons. *World Journal of Gastrointestinal Pharmacology and Therapeutics.* 2012;3:62.
 19. Wiest R, Krag A, Gerbes A. Spontaneous bacterial peritonitis: recent guidelines and beyond. *Gut.* 2012;6:297-310.
 20. Cholongitas E, Papatheodoridis GV, Lahanas A, Xanthaki A, Kontou-Kastellanou C, Archimandritis AJ. Increasing frequency of Gram-positive bacteria in spontaneous bacterial peritonitis. *Liver International.* 2005;25: 57-61.
 21. Campillo B, Richardet JP, Kheo T, Dupeyron C. Nosocomial spontaneous bacterial peritonitis and bacteremia in cirrhotic patients: impact of isolate type on prognosis and characteristics of infection. *Clinical Infectious Diseases.* 2002;35:1-10.
 22. Jain A, Chandra L, Gupta S, Gupta O, Jajoo U, Kalantri S. Spontaneous bacterial peritonitis in liver cirrhosis with ascites. *The Journal of the Association of Physicians of India.* 1999;47:619.
 23. Fernández J, Navasa M, Gómez J, Colmenero J, Vila J, Arroyo V, et al. Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norfloxacin prophylaxis. *Hepatology.* 2002 ;35:140-148.
 24. Rimola A, Salmerón JM, Clemente G, Rodrigo L, Obrador A, Miranda ML, et al. Two different dosages of cefotaxime in the treatment of spontaneous bacterial peritonitis in cirrhosis: results of a

- prospective, randomized, multicenter study. *Hepatology*. 1995a;21:674-679.
25. Zhao M, Lepak AJ, Andes DR. Animal models in the pharmacokinetic/pharmacodynamic evaluation of antimicrobial agents. *Bioorganic & Medicinal Chemistry*. 2016;24:6390-6400.
 26. Navasa M, Follo A, Llovet JM, Clemente G, Vargas V, Rimola A, et al. Randomized, comparative study of oral ofloxacin versus intravenous cefotaxime in spontaneous bacterial peritonitis. *Gastroenterology*. 1996;111:1011-1017.
 27. Sheer TA, Runyo BA. Spontaneous bacterial peritonitis. *Digestive Diseases*. 2005;23:39-46.
 28. European Association for The Study Of The Liver. EASL clinical practice guidelines on the management of ascites, spontaneous bacterial peritonitis, and hepatorenal syndrome in cirrhosis. *J Hepatol*. 2010;53:397-417.
 29. Reuken PA, Pletz MW, Baier M, Pfister W, Stallmach A, Bruns T. Emergence of spontaneous bacterial peritonitis due to enterococci - risk factors and outcome in a 12-year retrospective study. *Aliment Pharmacol Ther*. 2012;35:1199-208.
 30. Umgelter A, Reindl W, Miedaner M, Schmid R, Huber W. Failure of current antibiotic first-line regimens and mortality in hospitalized patients with spontaneous bacterial peritonitis. *Infection*. 2009;37:2.
 31. Angeloni S, Leboffe C, Parente A, Venditti M, Giordano A, Merli M, et al. Efficacy of current guidelines for the treatment of spontaneous bacterial peritonitis in the clinical practice. *World J Gastroenterol*. 2008 ;14:2757-62.
 32. Koulaouzidis A, Bhat S, Saeed AA. Spontaneous bacterial peritonitis. *World Journal of Gastroenterology: WJG*. 2009;15:1042.
 33. Fernández J, Tandon P, Mensa J, Garcia-Tsao G. Antibiotic prophylaxis in cirrhosis: Good and bad. *Hepatology*. 2016;63:2019-2031.
 34. Zhang JC, Gou YZ, Nie QH, Huang C, Sun L, Sun YT. Changes in the profiles of bacteria causing spontaneous bacterial peritonitis: A recent twelve-year study. *Afr J Microbiol Res*. 2010;4:527-533.
 35. Strauss E, Caly WR. Spontaneous bacterial peritonitis: a therapeutic update. *Expert Review of Anti-infective Therapy*. 2006;4:249-260.
 36. Runyon BA. Introduction to the revised American Association for the Study of Liver Diseases Practice Guideline management of adult patients with ascites due to cirrhosis 2012. *Hepatology*. 2013;57: 1651-1653.

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