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Original Research Article

Peritoneal fluid culture and its antibiotic sensitivity in perforative peritonitis patients – a prospective observational study from Southern India

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ABSTRACT

Background: Multiple microorganisms, including anaerobic bacteria, gram positive and gram-negative bacteria, enter the peritoneal cavity during peritonitis. We studied the microbiological pattern in peritoneal fluid cultures and t the organisms' antibiotic susceptibility and resistance pattern in perforative peritonitis.
Methods: This study was a cross-sectional study conducted for 12 months with 50 patients. The patients presenting with features of perforation peritonitis aged above 18 years were included in the study. Emergency laparotomy done using midline incision and peritoneal fluid was obtained from confirmed the non-traumatic cases and sent for aerobic microbiological culture. Peritoneal fluid culture reports were followed up and the antibiotics were changed according to the sensitivity pattern of organism.
Results: Mean age of the study participants was 42.6±15.1 years. Study participants were predominantly male 36 (72%). Nine (58%) does not have any comorbidities. All patients presented with 50 (100%) abdominal pain followed by predominant symptom was vomiting 44 (88%), nausea 29 (58%), oliguria 7 (14%) and anorexia 3 (6%). Among patients without comorbidities, *E. coli* was the pre-dominant organism isolated 16 (55.2%) followed by *Klebsiella* 9 (31%), *Proteus* 3 (10.3%) and *Pseudomonas* 1 (3.4%). Maximum number of patients were sensitive to ceftriaxone 47 (94%) followed by ciprofloxacin 41 (82%). Maximum number of patients were resistant to higher end antibiotic like ampicillin 46 (92%) followed by co-trimoxazole 45 (90%).
Conclusions: Perforation most commonly seen in antral followed by duodenum. Common etiology found out was peptic ulcer disease. In this study, all organisms showed maximum sensitivity to ceftriaxone followed by ciprofloxacin and amikacin. Hence its recommended to administer empirical therapy with combination of cephalosporins/ fluroquinolones/ aminoglycosides and metronidazole.

Keywords: Perforative peritonitis, Antibiotics, Sensitivity

INTRODUCTION

Perforative peritonitis is one of the most prevalent conditions that surgeons treat in emergency rooms across the globe, especially in developing countries.[1] Mortality rates for small bowel perforations range from 20-38%, large bowel perforations from 20-45%, and gastroduodenal perforations from 20-38%.[2] Contamination from intraperitoneal organs inside the peritoneal cavity results in secondary peritonitis. An intestinal perforation, such as a ruptured appendix or colonic diverticulum, can introduce an infection into the sterile peritoneal environment and result in peritonitis.[3]

Of the three varieties of peritonitis, secondary peritonitis is the most prevalent, occurring from intestinal diseases like ischaemia or perforation.[4]

Multiple microorganisms, including anaerobic bacteria, gram positive and gram-negative bacteria, enter the peritoneal cavity during peritonitis.[5] It is said that the most often isolated pathogen is *E. coli*. These pathogens contribute to inflammation, which is then followed by the complement cascade being activated. They also result in bacteraemia, which can induce sepsis and its aftereffects. Patients who present with sepsis are connected with a higher mortality rate.[6] The most prevalent Gram-negative bacteria was *Klebsiella*, which was discovered in 52% of the cases. *E. coil* was identified in 36% of the cases, and both were found combined in 5% of the cases. Proteus and Pseudomonas were found in the remaining cases.[7]

Poor prognosis in secondary peritonitis is caused by improper antibiotic dosing, comorbidities, and inadequate source control.[8-10] Mortality is frequently the result of resistant bacteria's inability to react to the first empirical antibacterial treatments. In cases of secondary peritonitis, prompt surgical intervention and suitable antibiotic therapy are essential for a positive result.[11,12] Starting a specific course of antibiotic medication, which often consists of broad-spectrum antibiotics covering gram positive, gram negative, and anaerobe pathogens, makes the treatment process simple. But the current issue is that these medicines are becoming resistant, which leads to significant treatment failure rates.[1,13]

It is crucial to understand the microbial distribution in relation to the anatomical site of perforation peritonitis. This information can be acquired by culturing peritoneal fluid that is taken after surgery. The bacterial flora in the stomach has been shown to be nearly non-existent because of the low pH; the bacterial count in the duodenum is 103-106/gram, in the jejunum and proximal ileum, it is 105-108/gram, in the lower ileum and caecum, it is 108-1010/gram, and in the colon, it is 1011/gram.[11] This demonstrates that the load of bacteria rises in the gastrointestinal tract from the proximal to the distal regions.[11] The most frequent organism found isolated from stomachs was *E. coli*. *Enterobacteriaceae* (Gram negative bacilli) are predominant in the distal ileum and caecum. Anaerobes in the colon predominate (96-99%) of which Bacteroides spp. is most common.[14]

When secondary peritonitis is treated with antibiotics inappropriately, patient outcomes may be poor.[5,15,16] A growing trend of antibiotic resistance has been observed in some of the microorganisms that cause secondary peritonitis.[17,18] More than 87% of gram-negative bacteria were found to be sensitive to ceftriaxone, ciprofloxacin, and amikacin, although ampicillin and cotrimoxazole exhibited resistance. Minocycline and linezolid were the other antibiotics that demonstrated sensitivity to microorganisms in 76% of cases. Methicillin-resistant or methicillin-sensitive Staphylococcus aureus was present in around 8% of the fluid; both strains were susceptible to linezolid and minocycline but resistant to penicillin, and erythromycin.[7] Antibiotic stewardship is therefore essential in combating the emergence of resistance.

Controversial topics include the function of peritoneal cultures and the use of antibiotics that are effective against culture results. Previous research has shown that culture outcome data rarely made meaningful contribution to the patient's recovery following surgery. Recent research indicates that the administration of antibiotics improves patient outcomes based on sensitivity and culture. Antibiotic medication that is started during surgical intervention and prior to availability of culture results is known as empirical therapy, and it is intended to be effective against probable isolates.[19] Purpose of study to understand microbiological pattern in the peritoneal fluid cultures and to study the organisms' antibiotic susceptibility and resistance pattern of individuals exhibiting perforative peritonitis.

METHODS

This study was a cross-sectional study conducted for 12 months (April 2023 to April 2024) in the Department of General Surgery, Government Tiruvannamalai Medical College, after obtaining ethical approval from the Institute’s Ethical Committee. Inclusion criteria: Patients presenting with features if perforation peritonitis aged above 18 years. Exclusion criteria: Patients with primary peritonitis and peritonitis due to trauma. Based on the study by Gauzit et al prevalence of small bowel peritonitis 13%, and the sample size calculated was 45.2, rounded off to 50.[20] Purposive sampling method (non-probability sampling method): 50 patients attending the General Surgery Department presented with features of perforation peritonitis who fulfilled the eligible criteria are included in the study are evaluated clinically and confirmed by X-ray abdomen and chest erect position as per the designed proforma.

Data collection

Data were collected using questionnaire which were divided into 8 parts: Socio-demographic details of the patients like age, residence, gender etc., Symptoms and signs and its duration, Possible causes-peptic ulcer, diverticulitis, Crohn’s disease etc., Associated/co-morbid conditions, Physical examination were General/Systemic and local examination and pre-op investigations and their reports-X-ray chest and abdomen erect, CBC, Blood urea and serum creatinine, electrolytes and blood sugar, ECG

Pre-op preparation

Patient confirmed with diagnosis of perforation peritonitis were resuscitated with intravenous fluid and stabilising the patient vitals were planned for emergency laparotomy and taken up for surgery after getting consent from the patient and his/ her attenders.

Intra-operative procedure

Emergency laparotomy done using midline incision and peritoneal fluid was obtained from confirmed non

traumatic cases and sent for aerobic microbiological culture. Following which perforation closure is done using vicryl with live omental patch and abdomen is closed after keeping abdominal drains.

Post-operative care

Following surgery patient were given routine postoperative care with intravenous fluids and antibiotics. Peritoneal fluid culture reports were followed up and the isolated organisms were tested for antimicrobial sensitivity by Kirby-Bauer disc diffusion method using ampicillin, amikacin, ciprofloxacin, ceftriaxone and cotrimoxazole and the culture reports were obtained. Antibiotics were changed according to the sensitivity pattern of organism grown in the culture.

Statistical analysis

Descriptive statistics were used for baseline comparison. For categorical variables, chi-square tests or fisher's exact tests (if cell frequency <5) were used. A p value of less than 0.05 was taken as significant. The relationship between various parameters was analysed with bivariate analysis, and spearman's correlation coefficient (r) was derived.

RESULTS

A total of 50 patients were enrolled, mean age of the study participants was 42.6±15.1 years with range from 19 to 75 years (Figure 1). The study participants were predominantly male 36 (72%). The predominant study participants 29 (58%) did not have any comorbidities. 14 (28%) of the study participants are diabetic followed by 6 (12%) are both diabetic and hypertensive. Only 1 (2%) participant had hypertension. All patients presented with 50 (100%) abdominal pain. The other symptoms noted were vomiting 44 (88%), nausea 29 (58%), oliguria 7 (14%) and anorexia 3 (6%). Mean duration of symptoms was 2.9±0.7 days with range from 2 to 4 days. Peptic ulcer 27 (54%) was the commonest cause of perforation peritonitis followed by 15 (30%) infective and malignancy 8 (16%).

The maximum number of patients had perforation in the antrum 15 (30%) followed by duodenum 14 (28%), ileum and appendix 10 (20%) and sigmoid 1 (2%). *E. coli* 24 (48%) was the commonest organism isolated from maximum number of patients followed by *Klebsiella* 18 (36%), *Proteus* 6 (12%) and *Pseudomonas* 2 (4%). Maximum number of patients were sensitive to ceftriaxone 47 (94%) followed by ciprofloxacin 41 (82%). Maximum number of patients were resistant to higher end antibiotic like ampicillin 46 (92%) followed by cotrimoxazole 45 (90%).

Among patients presented at 2nd day of symptom, predominant organisms isolated was *E. coli* 14 (87.5%) followed by *Klebsiella* and *Proteus* 1 (6.3%). Among patients presented at 3rd days of symptoms, predominant organisms isolated was *Klebsiella* 13 (52%) followed by *E. coli* 7 (28%), *Proteus* 3 (12%) and *Pseudomonas* 2 (8%). Among patients presented at 4th days of symptoms, predominant organisms isolated was *Klebsiella* 4 (44.4%) followed by *E. coli* 3 (33.3%), *Proteus* 2 (22.2%). This is found to be statistically significant with p<0.05 (Table 1).

Among patients with antral perforation, *E. coli* 8 (53.3%) was the predominant organism followed by *Klebsiella* 6 (40%) and *Pseudomonas* 1 (6.7%). Among patients with appendix *E. coli* 5 (50%) was the predominant organism followed by *Klebsiella* 3 (30%) and *Proteus* 2 (20%). Among patients with duodenal perforation, *E. coli* 7 (50%) was the predominant organism followed by *Klebsiella* 6 (42.9%) and *Pseudomonas* 1 (7.1%). Among patients with ileal perforation, *Proteus* 4 (40%) was the predominant organism followed by *E. coli* and *Klebsiella* 3 (30%). *E. coli* was the 1 (100%) predominant in sigmoid perforation (Table 2).

Among patients without comorbidities, *E. coli* was the pre-dominant organism isolated 16 (55.2%) followed by *Klebsiella* 9 (31%), *Proteus* 3 (10.3%) and *Pseudomonas* 1 (3.4%). It indicates that *E. coli* equally affect irrespective of presence of comorbidities. In patients having both diabetes and hypertension, *Proteus* 3 (50%) is the common organism isolated followed by *Klebsiella* 7 (50%) and *E. coli* 1 (16.7%). Among diabetic patients, 7 (50%) of *E. coli* and *Klebsiella* has been isolated. (Table 3)

E. coli showed maximum sensitivity to ceftriaxone 22 (91.7%) followed by ciprofloxacin 20 (83.3%) and maximum resistance to co-trimoxazole 23 (95.8%) and ampicillin 22 (91.7%). *Klebsiella* showed maximum sensitivity to ceftriaxone 17 (94.4%) followed by ciprofloxacin and amikacin 14 (77.8%) and maximum resistance to co-trimoxazole and ampicillin 17 (94.4%). *Proteus* showed 6 (100%) sensitivity to ceftriaxone, ciprofloxacin and amikacin and maximum resistance to ampicillin 5 (83.3%) and co-trimoxazole 4 (66.7%). *Pseudomonas* showed 2 (100%) sensitivity to ceftriaxone and amikacin and maximum resistance to ampicillin 2 (100%) followed by co-trimoxazole and ciprofloxacin 1 (50%) (Table 4).

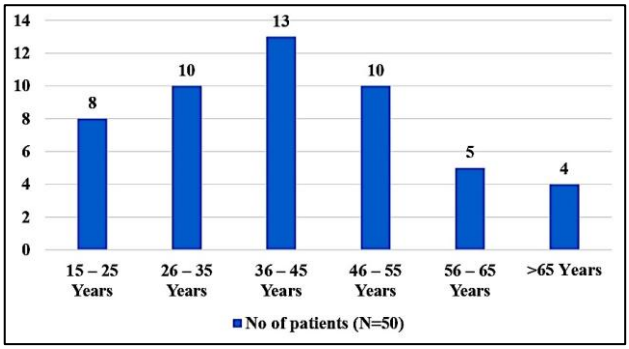

Figure 1: Age wise distribution of study participants.

Table 1: Association of duration of symptoms with organisms isolated among study participants.

Duration of symptoms	*E. coli*, N (%)	*Klebsiella*, N (%)	*Proteus*, N (%)	*Pseudomonas*, N (%)	Chi-square value (df)	P value
2 days	14 (87.5)	1 (6.3)	1 (6.3)	0 (0)	16.80 (6)	0.01
3 days	7 (28)	13 (52)	3 (12)	2 (8)		
4 days	3 (33.3)	4 (44.4)	2 (22.2)	0 (0)		
Total	24 (48)	18 (36)	6 (12)	2 (4)		

Table 2: Association of site of perforation with organisms isolated among study participants.

Site of perforation	*E. coli*, N (%)	*Klebsiella*, N (%)	*Proteus*, N (%)	*Pseudomonas*, N (%)	Chi-square value (df)	P value
Antrum	8 (53.3)	6 (40)	0 (0)	1 (6.7)	14.27 (12)	0.28
Appendix	5 (50)	3 (30)	2 (20)	0 (0)		
Duodenum	7 (50)	6 (42.9)	0 (0)	1 (7.1)		
Ileum	3 (30)	3 (30)	4 (40)	0 (0)		
Sigmoid	1 (100)	0 (0)	0 (0)	0 (0)		
Total	24 (48)	18 (36)	6 (12)	2 (4)		

Table 3: Association of comorbidities with organisms isolated among study participants.

Comorbidities	*E. coli*, N (%)	*Klebsiella*, N (%)	*Proteus*, N (%)	*Pseudomonas*, N (%)	Chi-square value (df)	P value
Both	1 (16.7)	2 (33.3)	3 (50)	0 (0)	36.31 (9)	0.00
Diabetes mellitus	7 (50)	7 (50)	0 (0)	0 (0)		
Hypertension	0 (0)	0 (0)	0 (0)	1 (100)		
No comorbidities	16 (55.2)	9 (31)	3 (10.3)	1 (3.4)		
Total	24 (48)	18 (36)	6 (12)	2 (4)		

Table 4: Antibiotic sensitivity to organisms isolated among study participants.

Antibiotics	Sensitivity	*E. coli*, N (%)	*Klebsiella*, N (%)	*Proteus*, N (%)	*Pseudomonas*, N (%)	Chi-square value (df)	P value
Ampicillin	R	22 (91.7)	17 (94.4)	5 (83.3)	2 (100)	0.94 (3)	0.81
	S	2 (8.3)	1 (5.6)	1 (16.7)	0 (0)		
	Total	24 (100)	18 (100)	6 (100)	2 (100)		
Ciprofloxacin	R	4 (16.7)	4 (22.2)	0 (0)	1 (50)	2.95 (3)	0.39
	S	20 (83.3)	14 (77.8)	6 (100)	1 (50)		
	Total	24 (100)	18 (100)	6 (100)	2 (100)		
Ceftriaxone	R	2 (8.3)	1 (5.6)	0 (0)	0 (0)	0.75 (3)	0.86
	S	22 (91.7)	17 (94.4)	6 (100)	2 (100)		
	Total	24 (100)	18 (100)	6 (100)	2 (100)		
Cotrimoxazole	R	23 (95.8)	17 (94.4)	4 (66.7)	1 (50)	8.48 (3)	0.03
	S	1 (4.2)	1 (5.6)	2 (33.3)	1 (50)		
	Total	24 (100)	18 (100)	6 (100)	2 (100)		
Amikacin	R	7 (29.2)	4 (22.2)	0 (0)	0 (0)	2.97 (3)	0.39
	S	17 (70.8)	14 (77.8)	6 (100)	2 (100)		
	Total	24 (100)	18 (100)	6 (100)	2 (100)		

*R-Resistant; S-Sensitive

DISCUSSION

In our study, mean age of the study participants was 42.6±15.1 years with range from 19 to 75 years. Maximum number of study participants in the age group 36 to 46 years 13 (26%) followed by 10 (20%) in 26 to 35 years and 46-55 years. Only 4 (8%) in above 65 years. Similar findings were reported by Kishore et al as maximum participants in 35-45 years.[21] Our study findings are comparable to the study by Bharathi et al maximum participants in 45% in 21-40 years, Manasa et al as mean age was 46.5±10.8 years, Kumar et al as mean age was 41.3±11.5 years.[11,22,23]

But Srivastava et al reported mean age was 36.2 years and maximum participants in 20-30 years.[7] Lohith et al reported mean age was 32.9±14.2 years and maximum participants in 18-27 years.[2] Samiuddin et al reported mean age as 35.2 years.[1] This difference in age may be due to difference in sample size, different causes of perforation as each common in different age groups.

In our study, participants were predominantly male 36 (72%) followed by female 14 (28%). Similar male predominance reported by Srivastava et al 75 (75%), Kishore et al 83.1%, Lohith et al 86%, Bharathi et al 70%, Manasa et al 96.6%, Samiuddin et al 87.5%, Ashok Kumar et al 66%, Choudhuri et al.[1,2,7,11,21-23] This male predominance may be due to smoking and alcoholism were more common among males in our setup which is predisposing factor for peptic ulcer perforation.

In our study, maximum number of study participants 29 (58%) does not have any comorbidities. 14 (28%) of the study participants are diabetic followed by 6 (12%) are both diabetic and hypertensive. Only 1 (2%) participant had hypertension. Similar findings reported by Jayaprakash et al as maximum no comorbidities may be due majority of study participants were below <45 years in our study.[6]

In our study, all patients presented with 50 (100%) abdominal pain followed by predominant symptom was vomiting 44 (88%), nausea 29 (58%), oliguria 7 (14%) and anorexia 3 (6%). Srivastava et al also reported the same findings 100% has abdominal pain followed by vomiting 59%.[7] Jayaprakash et al also reported the same as abdominal pain 98.7%.[6] In our study, mean duration of symptoms was 2.9±0.7 days with range from 2 to 4 days. Half of the 25 (50%) of the study participants were presented at 3rd day of symptoms followed by 16 (32%) at 2nd day and 9 (18%) at 4th day of symptoms. Similar findings reported by Samiuddin et al and also by Choudhury et al 80% in 2-4 days.[1,24]

In our study, peptic ulcer 27 (54%) was the commonest cause of perforation peritonitis followed by 15 (30%) infective and malignancy 8 (16%). In our study, maximum number of patients had perforation in the antrum 15 (30%) followed by duodenum 14 (28%), ileum and appendix 10 (20%) and sigmoid 1 (2%). Kishore et al also reported similar findings as stomach 35.3% followed by duodenum 26.1%.[21] Akulwar et al also reported the same findings as maximum had stomach 43.3%.[25] Jayaprakash et al also reported the same as gastric 52.6% followed by ileum 17.1%.[6]

But Srivastava et al also reported as commonest site of perforation was duodenum 55% followed by ileum 20%.[7] Lohith et al reported ileum 32% followed by stomach and appendix 18%.[2] Bharathi et al reported Appendix 63% followed by stomach 16%.[11] Samiuddin et al reported duodenum 50% followed by gastric 36.5%.[1] Ashok et al reported as duodenum 48% followed by gastric 40%.[23] Choudhury et al reported duodenum 50% followed by gastric 37.5%.[24] This difference in site of perforation may due to difference in causes, age at presentation.

In our study, all patients were isolated with monomicrobial growth. Same findings reported by Srivastava et al 80%. In our study, *E. coli* 24 (48%) was the commonest organism isolated from maximum number of patients followed by *Klebsiella* 18(36%), *Proteus* 6 (12%) and *Pseudomonas* 2 (4%). Similar findings reported by Kishore et al as *E. coli* 27.6% followed by *Klebsiella* 18.4% same by Lohith et al as *E. coli* 82.4% followed by *Klebsiella* 14.7%.[2,21] Similar findings reported by Bharthi et al also reported as *E. coli* 53.4% followed by *Klebsiella* 27.5%.[11] Manasa et al also reported the same findings as *E. coli* 48.9%. Akulwar et al and Jayaprakash et al also reported the same.[6,22,25]

But Srivastava et al reported as Klebsiella 52% was the commonest organism isolated from maximum number of patients followed by *E. coli* 36%.[7] Samiuddin et al reported *Klebsiella* 50% was the commonest organism isolated from maximum number of patients followed by *E. coli* 37.5%.[1] Kumar et al reported as *Klebsiella* 34% followed by *E. coli* 28%.[23] Choudhury et al also reported as Klebsiella 50% followed by *E. coli* 20%.[26]

In our study, maximum number of patients were sensitive to ceftriaxone 47 (94%) followed by ciprofloxacin 41 (82%). Maximum number of patients were resistant to higher end antibiotic like ampicillin 46 (92%) followed by co-trimoxazole 45 (90%). Similar findings reported by Srivastava et al as maximum sensitivity to ceftriaxone and ciprofloxacin 87% and maximum resistance to ampicillin and co-trimoxazole.[7]

In our study, among patients presented at 2nd day of symptom, predominant organisms isolated was *E. coli* 14 (87.5%) followed by *Klebsiella* and *Proteus* 1 (6.3%). Among patients presented at 3rd days of symptoms, predominant organisms isolated was *Klebsiella* 13 (52%) followed by *E. coli* 7 (28%), *Proteus* 3 (12%) and *Pseudomonas* 2 (8%). Among patients presented at 4th days of symptoms, predominant organisms isolated was *Klebsiella* 4 (44.4%) followed by *E. coli* 3 (33.3%), *Proteus* 2 (22.2%). This is found to be statistically significant with $p<0.05$. Similar findings reported by Chakravarthi et al.[13] This difference may due to difference in predominant organisms in different studies.

In our study, among patients with antral perforation, *E. coli* 8 (53.3%) was the predominant organism followed by *Klebsiella* 6 (40%) and *Pseudomonas* 1 (6.7%). Among patients with appendix *E. coli* 5 (50%) was the predominant organism followed by *Klebsiella* 3 (30%) and *Proteus* 2 (20%). Among patients with duodenal perforation, *E. coli* 7 (50%) was the predominant organism followed by *Klebsiella* 6 (42.9%) and *Pseudomonas* 1 (7.1%). Among patients with ileal perforation, *Proteus* 4 (40%) was the predominant

organism followed by *E. coli* and *Klebsiella* 3 (30%). *E. coli* was the 1 (100%) predominant in sigmoid perforation. Similar findings reported by Lohith et al.[2] as E. coli was the predominant organism isolated in all sites of perforation. Bharathi et al also reported *E. coli* was the predominant organism isolated in all sites of perforation except in duodenum where *Klebsiella* was predominant.[11] Ashok Kumar et al reported *E. coli* predominant in duodenal and *Klebsiella* in gastric perforation.[23]

In our study, *E. coli* shows maximum sensitivity to ceftriaxone 22 (91.7%) followed by ciprofloxacin 20 (83.3%) and maximum resistance to co-trimoxazole 23 (95.8%) and ampicillin 22 (91.7%). *Klebsiella* shows maximum sensitivity to ceftriaxone 17 (94.4%) followed by ciprofloxacin and amikacin 14 (77.8%) and maximum resistance to co-trimoxazole and ampicillin 17 (94.4%). *Proteus* shows 6 (100%) sensitivity to ceftriaxone, ciprofloxacin and amikacin and maximum resistance to ampicillin 5 (83.3%) and co-trimoxazole 4 (66.7%). *Pseudomonas* shows 2 (100%) sensitivity to ceftriaxone and amikacin and maximum resistance to ampicillin 2 (100%) followed by co-trimoxazole and ciprofloxacin 1 (50%).

Similar findings reported by Chaudhari et al as *E. coli* has maximum sensitivity to ceftriaxone, ciprofloxacin and amikacin in order, *Klebsiella* to ceftriaxone, amikacin and ciprofloxacin.[26] Our study findings are comparable to study by Ashok Kumar et al reported *E. coli* has maximum sensitivity to amikacin and ciprofloxacin, Klebsiella to amikacin and ciprofloxacin.[23] Lohith et al reported as *E. coli* has maximum sensitivity to amikacin 100%, *Klebsiella* to amikacin 40%.[2] Bharathi et al also reported *E. coli* has maximum sensitivity to amikacin 87%, *Klebsiella* to amikacin 56%.[11] Manasa et al reported *E. coli* has maximum sensitivity to amikacin 73.3%, *Klebsiella* to amikacin, ceftriaxone and ciprofloxacin 57.1%.[22]

This difference in antibiotic sensitivity may due to difference in culture and sensitivity to antibiotic included in the study. In our study, we have included commonly used antibiotics only like cephalosporins, amikacin, ciprofloxacin etc., higher antibiotics like imipenem, meropenem are not included in our study. In the current era of growing antibiotic resistance, optimizing empirical therapy is necessary to improve clinical results and limit overuse. Antibiotic usage might be seen of as an adjunct to surgical intervention, but it's crucial to utilize them appropriately for patients with PA. Consequently, Antibiotic susceptibility patterns should be learned by doing intraoperative cultures in a systematic manner. Early antibiotic initiation and switching to sensitive antibiotics based on culture sensitivity may lower morbidity, mortality, and surgical complications. Antibiotic resistance patterns across different species vary geographically, thus it's wise to have local surveillance which enable us to prescribe the right antibiotics.

Limitations

Smaller sample size of the study. Single institutional study. Multi institutional study with large sample size needed to generalize the results. Short duration of the study as antibiotic resistance pattern will change over the time.

CONCLUSION

Most common organisms for perforation peritonitis due to *E. coli* followed by *Klebsiella* and rarely by *Proteus* and *Pseudomonas*. Normal pattern of flora in different parts of gastro-intestinal tract not reflected in this study. *E. coli* is the most common organisms isolated in all sites of perforation. All organisms isolated showed maximum sensitivity to ceftriaxone followed by ciprofloxacin and amikacin. Hence, it's recommended that to administer empirical therapy with combination of cephalosporins or fluroquinolones/aminoglycosides + metronidazole.

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