

Original Research Article

DOI: <https://dx.doi.org/10.18203/2349-2902.ijssurgery20260133>

A prospective study of exploratory laparotomy and their correlation with microbiological profile in view of anatomical site of perforation peritonitis

Mudavathu Poolchend Naik, Zil Desai, Vishal Desai, Ankit Rathod, Sarvagya Jha*

Department of Surgery, GMERS Medical College, Sola, Ahmedabad, Gujarat, India

Received: 03 December 2025

Revised: 07 January 2026

Accepted: 20 January 2026

*Correspondence:

Dr. Sarvagya Jha,

E-mail: sarvagyajha@icloud.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Surgical peritonitis is a common and life-threatening emergency in tertiary care hospitals in India. Secondary peritonitis, most often due to gastrointestinal perforation or ischemia, constitutes the majority of intra-abdominal infections and usually presents late, resulting in high morbidity and mortality. Outcomes vary with the site of perforation and the causative organisms. These infections are typically polymicrobial, involving both community- and hospital-acquired pathogens. The growing problem of antimicrobial resistance further complicates management. Identifying the microbial profile and antibiotic sensitivity patterns in relation to perforation site is crucial for appropriate empirical therapy. This study evaluates the spectrum of community-acquired acute bacterial peritonitis and the role of microbiological cultures in its management.

Methods: A prospective study was conducted on 100 patients undergoing emergency laparotomy for perforation peritonitis at GMERS Medical College and Hospital, Sola, Ahmedabad. Intraoperative peritoneal fluid and postoperative wound discharge samples were collected using sterile techniques. Isolates were identified by Gram staining and culture, followed by in-vitro antibiotic susceptibility testing.

Results: Males predominated (male:female ratio 3.2:1), with the highest incidence in the 18–30-year age group (41%). The ileum was the most common site of perforation (31%), followed by the stomach (21%) and appendix (17%). Culture positivity was seen in 74% of cases. *Escherichia coli* was the most common isolate (92%), followed by *Klebsiella* spp. (42%), *Citrobacter* (8%), and *Acinetobacter* (5.4%). Culture positivity increased distally along the gastrointestinal tract. *E. coli* showed high sensitivity to amikacin (85.3%) and moderate sensitivity to meropenem (37%), while resistance to ampicillin (91%) and piperacillin-tazobactam (87%) was high. Although anaerobes were not isolated, empirical anaerobic coverage remained clinically relevant.

Conclusions: *E. coli* was the predominant pathogen irrespective of perforation site, highlighting discordance between expected gut flora and actual isolates. Rising resistance to third-generation cephalosporins underscores the need for rational antibiotic use. Early empirical therapy with agents such as amikacin, guided by culture and sensitivity results, along with prompt surgical source control, is essential for improving outcomes in perforation peritonitis.

Keywords: Perforation peritonitis, Exploratory laparotomy, Microbiological profile, Anatomical site of perforation, Surgical site infection, Peritoneal contamination

INTRODUCTION

Surgical peritonitis is among the most frequently encountered emergencies in tertiary care hospitals across

India. Intra-abdominal infections, particularly secondary peritonitis resulting from bowel perforation or ischemia, are common and often present at an advanced stage. Mortality rates differ based on the location of the

perforation, ranging from 3-28% for gastroduodenal, 20-38% for the small intestine, and 20-45% for the large intestine. Managing these infections continues to be a major challenge in surgical practice, particularly in high-volume tertiary care centers.¹ Intra-abdominal infections (IAIs) are widely prevalent and associated with significant mortality. They are commonly caused by pathogens such as *Bacteroides fragilis* and *E. coli*. In cases of hospital-acquired infections, microorganisms like *Pseudomonas*, *Enterococcus*, *Staphylococcus*, and certain fungi are frequently identified. Across the globe, bacterial resistance to medications has been increasing, impacting both hospitalized and community-based patients.²

Peritonitis is categorized into primary, secondary and tertiary types, with secondary peritonitis being the most common form of intraperitoneal infection. It usually results from bowel damage caused by perforation, strangulation, or infection. Complicated intra-abdominal infections (cIAIs) occur when the infection extends beyond the original injury site into the peritoneal cavity, often leading to abscess formation or widespread peritonitis. The widely accepted treatment approach for secondary peritonitis due to hollow viscus perforation involves stabilizing the patient, promptly eliminating the source of contamination, and administering appropriate antimicrobial therapy.⁴

Secondary peritonitis, often associated with polymicrobial infections, occurs due to a breach in abdominal integrity. It accounts for about 1% of emergency hospital admissions and is the second leading cause of sepsis, with a global mortality rate of around 6%. In India, it remains a common surgical emergency, with recent studies indicating mortality rates between 9% and 16%.⁵

Peptic ulcer disease primarily affects the stomach and the upper section of the duodenum. In the United States, it is most commonly caused by *Helicobacter pylori* infection and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs). Typical symptoms include epigastric pain, which may improve after eating or taking antacids, along with discomfort at night or between meals. Other possible symptoms include loss of appetite and unintentional weight loss.⁶

In Ethiopia, data on the characteristics of perforated peptic ulcers is scarce. Delayed diagnosis was noted in 95% of cases. Most patients had perforations in the duodenum, with nearly 78% showing purulent peritonitis during laparotomy. During hospitalization, 14 patients succumbed to the condition. Enhancing early access to surgical care facilities may help lower the morbidity and mortality rates associated with peptic ulcer perforation.⁷ Ileal perforation due to typhoid fever is a serious and potentially life-threatening complication. At Komfo Anokye Teaching Hospital (KATH), typhoid ileal perforation (TIP) ranks as the second most common reason for acute surgical admissions related to abdominal

pain in adults. Over the past three decades, mortality rates from TIP in the West African subregion have significantly decreased, falling from previous levels of 40-50% to around 10-15%. This decline is largely due to improvements in aggressive resuscitation methods and timely surgical interventions.⁸ Understanding the microbial distribution based on the anatomical site of perforation is crucial for selecting the most effective empirical antibiotic treatment. Identifying the bacterial profile in different regions helps optimize antibiotic choices for affected patients. This information can be obtained through cultures of peritoneal fluid collected during surgery. While multiple guidelines exist for the empirical treatment of intra-abdominal infections, most research on causative bacteria was conducted before the 2000s.⁶⁻⁸

The surgical complications of typhoidileitis, such as ileal perforations (TIP), continue to result innumerable deaths, particularly in countries with inadequate sanitation and limited healthcare resources. This study aimed to explore the spectrum of community-acquired acute bacterial peritonitis, assess the role of microbiological culture in its management, and identify other factors influencing its outcomes. Understanding the microbial profile and antibiotic sensitivity of peritoneal fluid cultures, in relation to the anatomical site of perforation peritonitis, can facilitate the early initiation of appropriate antibiotic therapy during the postoperative period.

Aim

To study the microbiological pattern of the peritoneal fluid in culture and sensitivity and to identify the pattern of antibiotic sensitivity against those organisms and the causative organisms

Objectives

Primary objective

To study the microbiological pattern of the peritoneal fluid in culture and sensitivity and to identify the pattern of antibiotic sensitivity against those organisms and the causative organisms

Secondary objective

To estimate the relative frequency of anatomical site of perforation. To enlist the mode of presentation of perforation cases. To know the usefulness of investigative procedures in diagnosis. To study the outcome of conservative and medical management of perforative peritonitis.

METHODS

Study design

A prospective study was conducted on 100 patients undergoing emergency laparotomy for perforation

peritonitis. Samples from peritoneal fluid intraoperatively and discharge from infected post operative wound collected using a sterile swab and studied for identification of isolates by Gram stains and culture growth followed by invitroantibiotic susceptibility testing.

Study population

All patients who underwent exploratory laparotomy because of perforation peritonitis from April 2025 to August 2025 were part of the study population at GMERS Medical College and Hospital, Sola, Ahmedabad.

Sample size calculation

The study had a sample size of 100 participants.

Inclusion criteria

Adult patients of either sex presenting with abdominal sepsis 18-80 years age group. Patients who had confirmed hollow viscous perforation by X-ray, ultrasonography or computed tomography (CT) scan. Patients in whom the samples were collected and cultured from the abdomen during surgery and post operative period

Exclusion criteria

Children <18 years of age, patients who had abdominal sepsis without hollow viscous perforation.

Study methodology

After the application of inclusion and exclusion criteria, patients undergoing exploratory laparotomy for perforation peritonitis are included in the study. Intraoperative findings are confirmed. During routine follow-up visits at two weeks and one month, culture sensitivity reports are recorded. All the data have been recorded on pre-approved case record forms. Data has entered in a Microsoft Excel sheet (Microsoft Corp., Redmond, WA) and analyzed to produce results.

Statistical analysis

Data was entered in Microsoft Excel spread sheet. Data analysis was done using SPSS software licensed version 21.0. Appropriate tests of significance like test of significance between two proportions and means have been used to compare between groups (chi square, fisher's exact etc.) wherever applicable. $P < 0.05$ was taken as significant. Appropriate graphs and charts were prepared to represent the data. Bivariate and multivariate regression analysis were performed to examine the association between disability and various independent variables. Bivariate analysis explored individual relationships, while multivariate analysis accounted for

potential confounders by analysing all variables simultaneously to assess their combined effects.

RESULTS

The highest incidence of cases was observed in the 18-30 age group, accounting for 41% (41 out of 100), followed by the 31-40 age group at 25% (25 out of 100). The lowest occurrence of perforation was recorded in individuals aged 51-60 years. Males accounted for 76% (76 out of 100) of the cases, while females made up 24% (24 out of 100). The study population had a male-to-female ratio of 3.2:1. The most frequently affected site of perforation was the ileum, accounting for 31% (31 out of 100) of cases, followed by the stomach at 21% (21 out of 100) and the appendix at 17% (17 out of 100). The rectum had the lowest occurrence, with only 2% (2 out of 100) of cases. Out of the 100 cases analysed, 74% (74 out of 100) showed positive culture results, while no bacterial growth was observed in 26% (26 out of 100) of the cases.

Among the 21 cases of stomach perforation, 57% (12 out of 21) showed positive culture results, while 43% (9 out of 21) had no bacterial growth. Among the culture-positive cases, *E. coli* was identified in 8 out of 12 cases, *Acinetobacter* species in 3 cases and *Klebsiella* species in 1 case. Among the 11 cases of duodenal perforation, 74% (8 out of 11) showed positive culture results, while 26% (3 out of 11) had no bacterial growth. Among the culture-positive cases, *Escherichia coli* was detected in 7 out of 8 cases, *Klebsiella* species in 5 cases and *Enterococcus* in 2 cases. Among the 8 cases of jejuna perforation, 87% (7 out of 8) tested positive for bacterial culture. *E. coli* was identified in all 7 culture-positive cases, while *Klebsiella* species were present in 3 cases. Among the 31 cases of ileal perforation, 84% (26 out of 31) showed positive culture results, while 16% (5 out of 31) had no bacterial growth. Among the culture-positive cases, *E. coli* was identified in 24 cases, *Klebsiella* species in 13 cases, and *Citrobacter* in 6 cases. Among the 5 cases of caecal perforation, 71% tested positive for bacterial culture. *E. coli* was identified in all culture-positive cases, while *Klebsiella* species were found in one case. Among the 17 cases of appendicular perforation, 87% (15 out of 17) showed positive culture results, while 13% (2 out of 17) had no bacterial growth. Among the culture-positive cases, *E. coli* was detected in 12 cases, and *Klebsiella* species in 5 cases.

Responses are not mutually exclusive

Both cases of colonic perforation tested positive for bacterial culture. *E. coli* was identified in both cases, while *Klebsiella* species were found in one case. The only case of rectal perforation tested positive for bacterial culture, with *E. coli* identified. Among the 74 culture-positive cases, *Escherichia coli* was the most frequently detected organism, present in 92% (68 out of 74) of cases. *Klebsiella* species were identified in 42% (31 out of 74) of cases, while *Enterococcus* was found in only

2.7% (2 out of 74) of the culture *E. coli* was identified in 68 cases. Among these, it exhibited sensitivity to ampicillin in 6 cases, while resistance was observed in 62 cases. Sensitivity to amikacin was noted in 58 cases, whereas ciprofloxacin showed effectiveness in 17 cases. Of the 52 cases tested for ceftriaxone, 21 demonstrated sensitivity. Cotrimoxazole sensitivity was detected in 13 out of 31 tested cases. Meropenem sensitivity was observed in 25 out of 45 cases, and piperacillin-tazobactam was effective in 9 out of 14 tested cases. *Klebsiella* was detected in 31 cases. Among the 13 cases tested for ampicillin, 4 were sensitive, while 9 exhibited resistances. Sensitivity to amikacin was observed in 11 cases, and ciprofloxacin was effective in 4 out of 13 cases. Of the 10 cases tested for ceftriaxone, 6 showed sensitivities. Cotrimoxazole sensitivity was identified in 3 out of 10 tested cases. Meropenem was effective in 4 out

of 5 cases, while piperacillin-tazobactam sensitivity was noted in 6 out of 10 cases. *Citrobacter* was identified in six cases. Among these, one case showed sensitivity to ampicillin, while five were resistant. All six cases exhibited sensitivity to amikacin, while ciprofloxacin was effective in three cases. Of the six cases tested for imipenem, four demonstrated sensitivities. *Acinetobacter* was detected in four cases. Sensitivity to amikacin and imipenem was observed in one case, while resistance was noted in three cases. All four cases exhibited resistance to piperacillin. However, colistin was effective in all cases. Tigecycline showed sensitivity in two cases, while the remaining two were resistant. *Enterococcus* was the only gram-positive organism isolated. The isolated organism showed resistance to penicillin and sensitivity to gentamycin, vancomycin, teicoplanin, and linezolid.

Table 1: Distribution of study participants according to age (n=100).

S. No.	Age (in years)	Gender		Number (%)
		Male	Female	
1	18-30	30 (39.5%)	11 (45.8%)	41 (41)
2	31-40	19 (25%)	6 (25%)	25 (25)
3	41-50	10 (13%)	4 (16.7%)	14 (14)
4	51-60	8 (10.5%)	1 (4.2%)	9 (9)
5	≥60	9 (11.8%)	2 (8.3%)	11 (11)
Total		76 (57%)	24 (24%)	100 (100)

Table 2: Distribution of study participants according to gender (n=100).

Gender	Frequency	(%)
Male	76	76
Female	24	24
Total	100%	100

Table 3: Distribution of study participants according to sites of perforation (n=100).

S. No.	Site of perforation	Frequency	(%)
1	Stomach	21	21
2	Duodenum	11	11
3	Jejunum	8	8
4	Ileum	31	31
5	Ceacum	7	7
6	Appendix	17	17
7	TransverseColon	3	3
8	Rectum	2	2
Total		100	100

Table 4: Distribution of study participants according to culture reports of cases with stomach perforation (n=21).

S. No.	Organism cultured	Frequency	(%)
1	Culture-Positive	12	57
2	Culture-negative	9	43
Organisms detected*			
1	<i>Escherichia coli</i>	10	83
2	<i>Acinetobacter</i> species	4	34
3	<i>Klebsiella</i> species	3	25

*Responses are not mutually exclusive.

Table 5: Distribution of study participants according to culture reports of cases with duodenum perforation (n=11).

S. No.	Organism cultured	Frequency	(%)
1	Culture-positive	8	74
2	Culture-negative	3	26
Organisms detected*			
1	<i>Escherichia coli</i>	7	87
2	<i>Klebsiella</i> species	5	62
3	<i>Enterococcus</i>	2	25

*Responses are not mutually exclusive.

Table 6: Distribution of study participants according to culture reports of cases with jejunum perforation (n=8).

S. No.	Organism cultured	Frequency	(%)
1	Culture-positive	7	87
2	Culture-negative	1	13
Organisms detected*			
1	<i>E. coli</i>	7	100
2	<i>Klebsiella</i> species	3	43

*Responses are not mutually exclusive.

Table 7: Distribution of study participants according to culture reports of cases with ileum perforation (n=31).

S. No.	Organism cultured	Frequency	(%)
1	Culture-positive	26	84
2	Culture-negative	5	16
Organisms detected*			
1	<i>E. coli</i>	24	92
2	<i>Klebsiella</i> species	13	50
3	<i>Citrobacter</i>	6	23

Table 8: Distribution of study participants according to culture reports of cases with caecum perforation (n=7).

S. No.	Organism cultured	Frequency	(%)
1	Culture-positive	5	71
2	Culture-negative	2	29
Organisms detected*			
1	<i>E. coli</i>	5	100
2	<i>Klebsiella</i> species	1	20

*Responses are not mutually exclusive.

Table 9: Distribution of study participants according to culture reports of cases with appendix perforation (n=17).

S. No.	Organism cultured	Frequency	(%)
1	Culture-positive	13	87
2	Culture-negative	4	13
Organisms detected*			
1	<i>E. coli</i>	12	92
2	<i>Klebsiella</i> species	5	38

*Responses are not mutually exclusive.

Table 10: Distribution of study participants according to organisms detected in culture reports (n=74).

S. No.	Organism	Frequency	(%)
1	<i>E. coli</i>	68	92
2	<i>Klebsiella</i> species	31	42
3	<i>Acinetobacter</i> species	4	5.4
4	<i>Enterococcus</i>	2	2.7
5	<i>Citrobacter</i>	6	8

DISCUSSION

This study evaluated the microbiological profile of peritoneal fluid cultures in patients with perforation peritonitis and analyzed variations based on the anatomical site of perforation. It also assessed antibiotic sensitivity patterns of the isolated microorganisms. By correlating microbial isolates with their antimicrobial susceptibility, the study aimed to generate evidence to guide effective empirical and targeted antibiotic therapy in perforation peritonitis. Understanding pathogen distribution and resistance patterns is crucial for optimizing treatment strategies and improving patient outcomes.

Age

The most commonly affected age group in the present study was 18–30 years, accounting for 41% of cases. This finding is comparable to the study by Yadav et al, where the mean age was 26.38 years, closely aligning with our results.⁹ Increased susceptibility in this younger age group may be related to dietary habits, lifestyle factors, occupational stress, and a higher incidence of infective and inflammatory gastrointestinal conditions. Similar age distribution has also been reported by Lohith et al, supporting the observed trend.

Gender

A clear male predominance was noted, with a male-to-female ratio of 3.2:1. This finding is consistent with previous studies, including Yadav et al who reported a ratio of 4:1.⁹ In India, perforation peritonitis is more common in males, likely due to higher prevalence of risk factors such as smoking, alcohol consumption, and occupational exposure. Additionally, gastrointestinal perforation secondary to abdominal tuberculosis a leading cause of perforation peritonitis in India shows higher incidence among males, which may further explain this gender disparity.

Sites of perforation

In our study, the ileum was identified as the most common site of perforation, consistent with the findings of Lohith et al 1 (32%). This observation maybe linked to the higher prevalence of ileocecal tuberculosis in the region. However, studies by More et al and Ravishankar et al reported the gastroduodenal region as the most frequently perforated site, with rates of 51% and 94%, respectively.^{10,11} This discrepancy is likely due to the significant incidence of peptic ulcer disease, which predominantly affects the gastroduodenal area.

Culture sensitivity

In patients with perforation peritonitis the peritoneal fluid culture did not reflect the major differential normal flora according to the region of the gastro-intestinal tract.

Consistent with the findings of Lohith et al *E. coli* was the most commonly isolated organism in gastric perforation cases, accounting for 44.4% of instances.¹ The high acidity of the stomach, which creates an inhospitable environment for most microorganisms, likely contributes to the significant rate of culture negativity observed in gastric perforations. This suggests that while *E. coli* can survive in such conditions, many other organisms cannot, leading to a lower diversity of microbial growth in cultures from gastric perforation cases. These findings highlight the unique microbiological profile associated with gastric perforations compared to other sites.

In duodenal perforations, the culture positivity rate was 80%, with *E. coli* being the most commonly isolated organism, followed by *Klebsiella*. Gram-positive *Enterococcus* was identified in one sample. For jejuna perforations, *E. coli* was the predominant organism, with a 100% culture positivity rate. In ileal perforations, the culture positivity rate was 83.3%, with *E. coli* as the most frequent isolate, followed by *Klebsiella* and *Citrobacter*.

Notably, jejunal perforations exhibited a significantly higher culture positivity rate compared to other sites. Gram-negative bacilli, particularly *Enterobacteriaceae*, which are naturally abundant in the jejunum and ileum, were also the primary isolates in perforation peritonitis cases from these regions, with *E. coli* being the dominant pathogen in both jejunal and ileal perforations.^{1,12} These findings reflect the typical microbial flora of the small intestine and its role in perforation-related infections. In cases of caecal perforation, *E. coli* was the most frequently isolated organism, with a 100% culture positivity rate.

Similarly, for appendicular perforations, *E. coli* was the predominant organism, with an 87.5% culture positivity rate. In colonic and rectal perforations, *E. coli* was also the most common isolate, achieving a 100% culture positivity rate. This high rate of culture positivity can be attributed to the abundant microbial flora present in the large intestine. As *E. coli* is a dominant organism in the aerobic flora of the gut, its prevalence aligns with our study's findings, where it was the primary isolate from peritoneal fluid cultures in colonic perforations.^{13,14} These results highlight the consistent role of *E. coli* in infections related to perforations of the lower gastrointestinal tract.

The bacterial count in the gastrointestinal tract varies significantly across regions, the duodenum contains approximately 103–106 bacteria per gram, the jejunum and proximal ileum have 105–108 per gram, the lower ileum and caecum host 108–1010 per gram, and the colon has the highest concentration at 1011 per gram. In contrast, the stomach has minimal bacterial flora due to its low pH. This gradient illustrates that microbial density increases progressively from the proximal to the distal regions of the gastrointestinal tract. The study's findings align with this pattern, showing a rise in culture positivity

as the site of perforation shifts distally from the stomach to the rectum.^{1,14} This correlation underscores the relationship between microbial load and infection rates in perforation peritonitis.

In the study, amikacin and meropenem emerged as the most effective antibiotics overall. Amikacin showed sensitivity in 85.18% of *E. coli* cases and 84.6% of *Klebsiella* cases, while meropenem was effective in 76.9% of *E. coli* and 80% of *Klebsiella* cases. Other antibiotics with notable sensitivity included piperacillin+tazobactam (64.2% for *E. coli* and 60% for *Klebsiella*) and ceftriaxone (57% for *E. coli* and 60% for *Klebsiella*).

A study by More et al reported that most *E. coli* isolates were sensitive to amikacin (94%) and cefazidime (91%).¹⁰ Additionally, some *Klebsiella* species demonstrated sensitivity to ciprofloxacin, aminoglycosides, and cephalosporins. Ravishankar et al found that approximately 87.5% of *E. coli* were sensitive to ceftriaxone, while 81.25% were sensitive to ciprofloxacin and amikacin.¹¹ For *Klebsiella*, sensitivity to ceftriaxone was 91.07%, followed by amikacin (78%) and ciprofloxacin (73.9%). Both *E. coli* and *Klebsiella* exhibited high resistance to ampicillin and cotrimoxazole. Generally, these organisms were most susceptible to ceftriaxone, ciprofloxacin, and amikacin, in that order. In the study, anaerobic organisms were not isolated from any sites, aligning with findings from Ramakrishnaiah et al.¹⁵ This is likely because anaerobes such as *Bacteroides* spp, which are predominant in the colon, are difficult to culture due to their fastidious nature and requirement for strict anaerobic conditions.^{13,16}

There is general agreement that antibiotic therapy should include coverage for anaerobes in most cases.¹⁷ Agents lacking anaerobic activity are often combined with antibiotics like metronidazole. Dosages should be tailored based on renal function and hemodynamic status, using antibiotics with proven efficacy in susceptibility tests. While therapy duration should theoretically follow a standard protocol, it should be individualized for each patient.

Limitations

The study has certain limitations. It was conducted at a single tertiary care center with a relatively small sample size, which may limit the generalizability of the findings. Anaerobic cultures were not performed, potentially underestimating the role of anaerobic organisms in perforation peritonitis. Prior antibiotic use before hospital admission may also have influenced culture positivity and sensitivity patterns. Larger multicenter studies incorporating anaerobic cultures and molecular diagnostic techniques are recommended to obtain a more comprehensive understanding of the microbiological spectrum and resistance patterns in perforation peritonitis.

CONCLUSION

The peritoneal fluid culture in patients with perforation peritonitis did not align with the typical normal flora of the gastrointestinal tract region. Instead, *E. coli* was the most commonly isolated organism across all perforation sites. The antibiotic sensitivity profile revealed increasing resistance to third-generation cephalosporins, which are often used empirically. However, aminoglycosides, such as amikacin, maintained a high sensitivity profile. Other effective antibiotics included piperacillin-tazobactam, meropenem, and colistin, which demonstrated significant activity against pathogens isolated from these patients.

The primary treatment for perforation peritonitis is source control, which includes procedures like appendectomy, perforation closure, resection of gangrenous bowel, and abscess drainage. Systemic antibiotic therapy serves as the secondary main stay of treatment. Early administration of antibiotics, preferably preoperatively, is crucial as it significantly reduces the concentration and growth rates of viable bacteria in the peritoneal fluid. Delayed antibiotic therapy is less effective, particularly in advanced stages of infection.

Empirical antibiotic therapy should be initiated promptly, targeting common gram-negative and anaerobic organisms. Once culture and sensitivity results are available, a step-down approach should be adopted, transitioning to narrower-spectrum agents to minimize resistance and optimize treatment outcomes. This strategy ensures effective management of perforation peritonitis while addressing the growing challenge of antibiotic resistance.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Lohith P, Jindal RK, Ghuliani DPR. The anatomical site of perforation peritonitis and their microbiological profile: a cross-sectional study. Int Surg J. 2020;7(4):1251.
2. Dossantos S, Decarvalho M, Serufo J, Pintosilva R, Albuquerque W, Rosa R, et al. Antimicrobial susceptibility of microorganisms recovered from intra abdominal infections at Belo Horizonte, Brazil. American J Infect Cont. 2004;32(7):414–6.
3. Jang J Y, Lee S H, Shim H, Choi J Y, Yong D, Lee J G. Epidemiology and microbiology of secondary peritonitis caused by viscus perforation: a single-center retrospective study. Surgical Infect. 2015;16(4):436–42.
4. Nelamangala Ramakrishnaiah VP, Chandrakasan C, Dharanipragadha K. Community acquired secondary bacterial peritonitis in a tertiary hospital

of South India: an audit with special reference to peritoneal fluid culture. *TG*. 2012;33(4):275–81.

5. Ghosh A, Halder A, Sen N, Dhara A, Ghosh S, Stellone Singh K. A comparative analytical study on outcome of secondary peritonitis using Mannheim's peritonitis index in geographically diverse Indian patients. *Turk J Surg*. 2023;1;39(4):300–9.

6. Ramakrishnan K, Salinas RC. Peptic ulcer disease. *Am Fam Physician*. 2007;76(7):1005-12.

7. Ersomo T, W/Meskel Y, Kotisso B. Perforated peptic ulcer in Tikur Anbessa Hospital: a review of 74 cases. *Ethiop Med J*. 2005;43(1):9-13.

8. Oheneh-Yeboah M. Postoperative complications after surgery for Typhoid Ileal perforation in adults in Kumasi. *West African J Med*. 2007;26(1):32–6.

9. More PM, Nichkaode PB, Khatri, Bansod PY: Spectrum of perforation peritonitis: clinical presentation, imaging, biochemical, operative corelation and outcomes. *Int J Med Appl Sci*. 2015;4:71-80.

10. Ravisankar J, Venkatesan VS. A study on peritoneal fluid culture and its antibiotic sensitivity in perforative peritonitis cases. *IOSR JDMS*. 2017;16(03):34–7.

11. Punamiya AR, Chougule PG, Ahuja BR, Singh V, Mohite ST. Commonest organisms and antibiotic sensitivity in peritonitis due to duodenal ulcer perforation in Krishna Hospital, Karad. *Int J Health Sci*. 2014;(8):468.

12. Chen CY, Chen YC, Pu HN, Tsai CH, Chen WT, Lin CH. Bacteriology of acute appendicitis and its implication for the use of prophylactic antibiotics. *Surg Inf*. 2012;13(6):383–90.

13. Ananthanarayan, R. Ananthanarayananand Paniker's Textbook of Microbiology. Eleventh edition., Universities Press (India). 2020.

14. Ramakrishnaiah V P, Chandrakasan C, Dharanipragadha K, Sistla S, Krishnamachari S. Community acquired secondary bacterial peritonitis in a tertiary hospital of South India: an audit with special reference to peritoneal fluid culture. *Trop Gastroenterol*. 2012;33(4):275-81.

15. O'Connell PR, McCaskie AW, Sayers RD. *Bailey & Love's short practice of surgery*. CRC Press. 2023.

16. Blot S, De Waele JJ, Vogelaers D. *Essentials for Selecting Antimicrobial Therapy for Intra-Abdominal Infections: Drugs*. 2012;72(6):17–32.

17. Last RJ. *Anatomy: regional and applied*. 1st ed. Edinburgh: Churchill Livingstone; 1954.

Cite this article as: Naik MP, Desai Z, Desai V, Rathod A, Jha S. A prospective study of exploratory laparotomy and their correlation with microbiological profile in view of anatomical site of perforation peritonitis. *Int Surg J* 2026;13:239-46.