**ORIGINAL RESEARCH**

# Study of organism profile in relation to site of perforation in Peritoneal fluid in patients of peritonitis

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**ABSTRACT**

**Background:** Peritonitis is inflammation of the lining of the abdominal cavity. The present study was conducted to study of organism profile in relation to site of perforation in peritoneal fluid in patients of peritonitis. **Materials & Methods:** 60 patients with a diagnosis of peritonitis were assessed for site of perforation and peritoneal fluid culture and sensitivity report of every patient was sent to the department of microbiology. Patient was reviewed till the day of discharge. Peritoneal, aerobic culture and sensitivity report was followed, and antibiotics were changed as per the sensitivity pattern of the organism grown in the culture. **Results:** The majority of the patients were between the age group of 18-29 years [20(33.33%)], followed by 30-44 years [19 (31.67%)]. Male preponderance was observed among patients [46(76.67%)]. The majority of the patients were delayed by 0-2 days [29(48.33%)], followed by a delay of 3-5 days [27(45.00%)]. The number of smokers [31(51.67%)] were more than non-smokers [29(48.33%)] among the enrolled patients. Statistically, insignificant difference was observed among patients. The majority of the patients had perforated appendix [17(28.33%)], followed by perforated gastric[11(18.33%)], perforated ileum [10(16.67%)]. Most of the patients had E. coli-in their peritoneal fluid [25(41.67%)], followed by Enterococcus Faecalis [6 (10.00%)] and so on. At the same time, no-aerobic growth was observed in [18(30.00%)] patients. The majority of the patients showed no post operative complications [33(55.00%)], while [18(30.00%)] patients had SSI. Respiratory failure was seen inonly[1(1.67%)] patient. A total of 25 patients had E. coli growth in the appendix [n=12], followed by the ileum [n=5]. A total of 6 patients had Enterococcus faecalis growth at the duodenum [n=3], followed by ileum [n=2]. Pseudomonas aeuroginos a growth was observed at the duodenum [n=1]. **Conclusion:** The most common site of perforation was Appendix, followed by gastric and duodenum. Peritonitis was most commonly due to E. coli, followed by Enterococcus faecalis. E. coli has also emerged as the predominant organism implicated in the pathogenesis, even if we consider it a site-specific culture. E. coli was sensitive to Amikacin, Tigecycline, Gentamycin, Imipenem, Meropenem and Cefoperazone-sulbactam.At the same time E. coli was resistant to Cefipime, Ceftriaxone and Ciprofloxacin. Surgical site infection was the most common postoperative complication.

**Key words:** Peritonitis, Enterococcus faecalis, Pseudomonas aeuroginosa



## INTRODUCTION

Peritonitis is inflammation of the lining of the abdominal cavity. Although the etiology of peritonitis varies, the outcome is the same in all cases. The most common peritonitis encountered in surgery is perforating peritonitis of infectious or traumatic etiology or postoperative perforating peritonitis after an anastomotic leak. Peritonitis is of three types. Primary, secondary and tertiary. Perforating peritonitis is one of the most common emergency surgeries in all hospitals. Despite many recent advances in medicine, perforation peritonitis remains a major threat to surgeons.[1]

The biggest problems facing surgeons are delayed presentation of patients and the emergence of resistant strains of bacteria that cause peritonitis and sepsis. This problem needs to be addressed quickly as the risk of emergence of drug resistance to antibiotics increases.[2] From an etiological point of view, acute pancreatitis complicated by simple duodenal perforation, traumatic perforation, appendicular perforation, or pancreatic abscess remains a major cause of morbidity and mortality. Only in recent decades has there been significant improvement in the treatment of peritonitis, both through the use of antibiotics and through surgery. Peritonitis is usually

secondary peritonitis, although it often occurs in emergencies. Another fact that makes peritonitis more dangerous is due to the very massive contamination of the abdominal cavity with certain deadly organisms belonging to the Enterobacteriaceae family. These include Escherichia coli, Klebsiella, Proteus, and Enterococcus species. These organisms, either directly or through their toxins, cause specific effects leading to the development of SIRS.[3]

With the use of antibiotics, aerobic-targeted therapy was associated with lower mortality and higher residual abscess formation, whereas anaerobe-targeted therapy was associated with lower abscess formation and no change in mortality. Therefore, treatment was considered optimal when a combination is used.[4] Treatment can be accomplished by initiating specific antibiotic therapy, which usually includes broad-spectrum antibiotics covering Gram- positive, Gram-negative, and anaerobes. However, a current problem is the development of resistance to these antibiotics, leading to high rates of treatment failure.[5] In this study, we correlated various aerobic microbes growing in peritoneal fluid cultures and antibiotics sensitive or resistant to each microbiological pattern with perforation sites in patients exhibiting features of peritonitis from any cause, and determined the appropriate perforation site. Allow for early detection. Possible Initiation of antibiotic therapy is possible in patients presenting with peritonitis preoperatively and to reduce patient morbidity and mortality.

## MATERIALS & METHODS

The study was conducted on 60 patients of more than 18 years of age with a diagnosis of peritonitis in the department of surgery in Shri Guru Ram Rai Institute of Medical and Health Sciences and associated Shri Mahant Indiresh Hospital over a time period of 18 months. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. A detailed history with special reference on the onset and days of the presentation was taken. A complete General physical and systemic Examination was done. All patients were subjected to X-Ray Abdomen (erect) for free air under Diaphragm or ultrasonography or computed tomography, followed by a complete Haemogram. Intra-operatively Site of perforation was noted, and peritoneal fluid culture and sensitivity report of every patient was sent to the department of microbiology. Patient was reviewed till the day of discharge, peritoneal Aerobic culture and sensitivity report was followed, and antibiotics were changed as per the sensitivity pattern of the organism grown in the culture. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

## RESULTS

**Table I: Age-wise distribution of patients**

| Age[years] | N | % |
|---|---|---|
| 18-29 | 20 | 33.33% |
| 30-44 | 19 | 31.67% |
| 45-59 | 12 | 20.00% |
| ≥60 | 9 | 15.00% |
| **Total** | **60** | **100.00%** |

The majority of the patients were between the age group of 18-29 years[20(33.33%)],followedby30-44years[19 (31.67%)].

**Table II: Gender-wise distribution of patients**

| Gender | N | % |
|---|---|---|
| **Female** | 14 | 23.33% |
| **Male** | 46 | 76.67% |
| **Total** | **60** | **100.00%** |

Table II shows that male preponderance was observed among patients [46(76.67%)].

**Table II: Delay in the presentation of patients**

| Delay in presentation | N | % |
|---|---|---|
| 0-2days | 29 | 48.33% |
| 3-5days | 27 | 45.00% |
| 6-8days | 4 | 6.67% |
| **Total** | **60** | **100.00%** |

Themajorityofthepatientsweredelayedby0-2days[29(48.33%)],followedbyadelayof 3-5days[27(45.00%)].

**Table III: Number of smokers among patients**

| Smokers | N | % | P value |
|---|---|---|---|
| **No** | 29 | 48.33% | |

| | | | |
|---|---|---|---|
| **Yes** | 31 | 51.67% | |
| **Total** | **60** | **100.00%** | =0.03334p=0.8551 |

The number of smokers [31(51.67%)] were more than non-smokers [29(48.33%)] among the enrolled patients. Statistically, in significant difference was observed among patients.

**Table IV: Site of perforation in patients**

| Site of perforation | N | % |
|---|---|---|
| Ileum | 10 | 16.67% |
| Appendix | 17 | 28.33% |
| Ascending Colon | 1 | 1.67% |
| Caecum | 2 | 3.33% |
| CBD | 1 | 1.67% |
| Cystic Duct Stump | 1 | 1.67% |
| Duodenum | 8 | 13.34% |
| Gall Bladder | 3 | 5.00% |
| Gastric | 11 | 18.33% |
| Hepatic Duct | 1 | 1.67% |
| Jejunum | 1 | 1.67% |
| Sigmoid Colon | 4 | 6.67% |
| **Total** | **60** | **100.00%** |

The majority of the patients had perforated appendix [17(28.33%)], followed byperforatedgastric[11(18.33%)], perforated ileum [10(16.67%)].

**Table V: Aerobic organism present in peritoneal fluid of patients**

| Aerobic organism | N | % | P value |
|---|---|---|---|
| E.coli | 25 | 41.67% | X=33.25 **p<0.0001*** |
| Enterococcusfaecalis | 6 | 10.00% | |
| Pseudomonasaeuroginosa | 1 | 1.67% | |
| Klebsiellapneumoniae | 3 | 5.00% | |
| EnterobactercloacaeComplex | 5 | 8.33% | |
| StreptococcussanguinisGp | 2 | 3.33% | |
| Citrobacterfreundii | 1 | 1.67% | |
| AcinetobacterbaumaniiComplex | 1 | 1.67% | |
| NoAerobicGrowth | 18 | 30.00% | |

Most of the patients had E. coliin their peritoneal fluid [25(41.67%)], followed by Enterococcus Faecalis [6(10.00%)] and so on. At the same time, noaerobic growth was observed in[18(30.00%)] patients.

**Table VI: Postoperative complications inpatients**

| Post operative complication | | N | % |
|---|---|---|---|
| NO | | 33 | 55.00% |
| YES | SSI | 18 | 30.00% |
| | Wound Dehiscence | 4 | 6.67% |
| | Respiratory Failure | 1 | 1.67% |
| | Deceased | 4 | 6.67% |

The majority of the patients showed no post operative complications [33(55.00%)], while [18(30.00%)] patients had SSI. Respiratory failure was seen in only[1(1.67%)]patient.

**Table VII: Organism growth with respect to the site of perforation**

| Site of Perforation | E.coli | erococcusfaecalis | seudomonasaeuroginosa | Klebsiellapneumoniae | EnterobacterCloacaeComplex | Streptococcus | Citrobacterfreundii | AcinetobacterbaumaniiComplex | NoAerobicGrowth |
|---|---|---|---|---|---|---|---|---|---|
| Ileum | 5 | 2 | 0 | 1 | 1 | 0 | 0 | 0 | 2 |
| Appendix | 12 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 4 |
| AscendingColon | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Caecum | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 |
| CBD | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| CysticDuctStump | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Duodenum | 2 | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 2 |

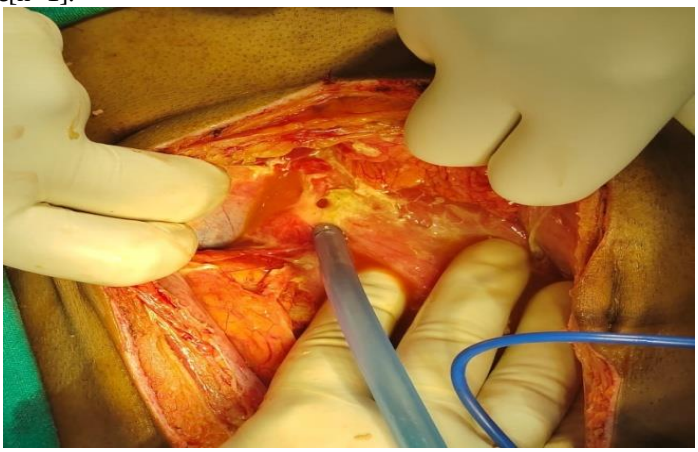
| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| GallBladder | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 1 |
| Gastric | 2 | 0 | 0 | 1 | 1 | 1 | 0 | 1 | 5 |
| HepaticDuct | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Jejunum | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| SigmoidColon | 1 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 2 |
| **Grand Total** | **25** | **6** | **1** | **3** | **5** | **2** | **1** | **1** | **18** |

A total of 25 patients had E. coli growth in the appendix [n=12], followed by the ileum [n=5]. A total of 6 patients had Enterococcus faecalis growth at the duodenum [n=3], followed by ileum [n=2]. Pseudomonas aeuroginosa growth was observed at the duodenum [n=1]. Citrobacter freundii growth was observed at the caecum [n=1], and Acine to bacter baumanii complex growth was observed at the gastric [n=1]. A total of 3 patients had Klebsiellapneumoniae growth, and 5 patients had Enterobactercloacae complex growth. Noaerobic growth was observed in [n=18]patients.

**Table VIII: Antibiotic sensitivity pattern with respect to organisms grown**

| **ANTIBIOTICSSENSITIVE** | **E.coli** | **Enterococ** | **Pseudomonas aeur** | **Klebsiella pneumoni** | **Enterobacterc** | **Streptococcu** | **Citrobacter freu** | **Acinetobacter bau** | **NoAerobic** |
|---|---|---|---|---|---|---|---|---|---|
| Benzylpenicillin | **0** | 4 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Erythromycin | **0** | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Linezolid | **0** | 1 | 0 | 0 | **0** | 0 | 0 | 0 | 0 |
| Vancomycin | **0** | 2 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Cefipime | 7 | 0 | 1 | 2 | 1 | 0 | 0 | 0 | 0 |
| Ceftriaxone | 6 | **0** | 0 | **1** | 2 | 0 | 0 | 0 | 0 |
| Amikacin | 24 | 5 | 0 | 2 | 4 | 0 | 1 | 0 | 0 |
| Gentamycin | 19 | 2 | 0 | 1 | 2 | 1 | 0 | 0 | 0 |
| Ciprofloxacin | 2 | **0** | 0 | 0 | 1 | 1 | 0 | 0 | 0 |
| Imipenem | 19 | 3 | 0 | 1 | 4 | 1 | 1 | 0 | 0 |
| Meropenem | 19 | 3 | 0 | 1 | 4 | 1 | 1 | 0 | 0 |
| Piperacillin-Tazobactam | 8 | 2 | 0 | 1 | 2 | 1 | 1 | 0 | 0 |
| Cefoperazone-Sulbactam | 14 | 2 | 1 | 1 | 4 | **0** | 0 | 0 | 0 |
| Amoxycillin-Clavunate | 10 | 0 | 0 | 1 | 2 | 1 | 0 | 0 | 0 |
| Trimethoprime-Sulfamethoxazole | 11 | 2 | 0 | 1 | 1 | 2 | 0 | 0 | 0 |
| Colistin | 13 | **0** | 1 | 2 | 4 | **0** | 1 | 0 | 0 |
| Minocycline | 4 | **0** | 0 | 0 | 1 | 1 | 0 | 1 | 0 |
| Tigecycline | 23 | **0** | 0 | 2 | 5 | **0** | 1 | 0 | 0 |

The majority of the patients who had *E.coli* growth also had maximum antibiotic sensitivity to Amikac in [n=24], followed by Tigecycline [n=23], and only [n=2] patients were sensitive to Ciprofloxacin. The patients who had *Enter ococcus faecalis* growth were sensitive to Amikacin [n=5], Bezyl penicillin [n=4] and so on. Patients who had *Pseudomonas aeuroginosa*growth were sensitive to Cefipime [n=1], Cefoperazone-Sulbactam [n=1] and Colistin [n=1]. Patients who had *Klebsiella pneumoniae* growth were sensitive to Cefipime [n=2], Amikacin [n=2], Colistin [n=2] and Tigecycline [n=2].Patients with *Enterobacter cloacae c*omplex growth were also sensitive to Tigecycline [n=5], Amikacin[n=4], Imipenem [n=4], Meropenem [n=4] andsoon.PatientswiththegrowthofStreptococcussanguinisGpweresensitivetoTrimethoprime-Sulfamethoxazole[n=2],Imipenem[n=1]andsoon.Patients with Citrobacter freundii growth were sensitive to Amikacin [n=1], Imipenem [n=1] and so on. Patients with Acinetobacter baumanii complex growth were only sensitive to Minocycline[n=1].



## DISCUSSION

Peritonitis is an inflammation of the peritoneum, the membrane that lines the abdominal cavity. The aetiology of peritonitis may vary, but its conclusion is always the same.[6] The most frequent peritonitis encountered in surgical practice is perforation peritonitis of infectious, traumatic, or postoperative anastomotic leak origin. There are three forms of peritonitis: primary, secondary, and tertiary.[7] Whether the cause is a simple duodenal perforation, traumatic perforation, appendicular perforation, or acute pancreatitis accompanied by a pancreatic abscess, it continues to be a leading source of morbidity and mortality. Antibiotics and surgery for the treatment of peritonitis have undergone significant development only in the last few decades. A second factor that makes peritonitis more hazardous is the extremely high level of infection of the peritoneal cavity by certain lethal Enterobacteriaceae species.[8] These include species of E. coli, Klebsiella, Proteus, and Enterococci. These organisms, directly or by their toxins, cause certain effects that contribute to the development of SIRS.[9] Current treatment for peritonitis focuses on correcting the underlying cause, administering systemic antibiotics, and supporting supportive therapy.[10] Therefore, the purpose of this research was to study bacteriological patterns, antibiotic sensitivity and resistance in relation to the site of perforation in the peritoneal fluid culture of the patients presenting with peritonitis.

We found that the majority of the patients were between the age group of 18-29 years [20(33.33%)],followedby30-44years[19 (31.67%)]. We found that male preponderance was observed among patients [46 (76.67%)]. Themajorityofthepatientsweredelayedby0-2days[29 (48.33%)] followed by adelay of 3-5days[27(45.00%)]. In study conducted by Mutibwa et al[11]in their study on aerobic bacterial causes of secondary peritonitis and their antibiotic sensitivity pattern in secondary small bowel perforation, demonstrated that in order to guide in choice of antimicrobial therapy, peritoneal fluid culture in perforation peritonitis is necessary. In their study most of patient had Klebsiella Species (37.9%) followed by E. coli (26.4%) and 13.8% had no growth. Most organisms were susceptible to ceftriaxone followed by ciprofloxacin and gentamycin.

We found that the number of smokers [31(51.67%)] were more than non-smokers [29 (48.33%)] among the enrolled patients. Statistically, in significant difference was observed among patients. The majority of the patients had perforated appendix [17(28.33%)], followed by perforated gastric[11(18.33%)], perforated ileum [10(16.67%)]. Most of the patients had E. coliin their peritoneal fluid [25(41.67%)], followed by Enterococcus Faecalis [6 (10.00%)] and so on. At the same time, noaerobic growth was observed in [18(30.00%)] patients. The majority of the patients showed nopostoperative complications [33(55.00%)], while [18(30.00%)] patients had SSI. Respiratory failure was seen in only[1(1.67%)]patient. Srivastava and Singh[12] conducted a study on Clinical evaluation of patient with perforation peritonitis and their peritoneal fluid analysis for culture and sensitivity. Total 100 cases of acute perforation peritonitis were included Male to female ratio was 3:1 and the most common age group involved was between 20 to 40 years. The most common site of perforation was found to be duodenum amounting to 55% of cases followed by ileal perforation found in 20%cases, gastric perforation was found in 10% of case. Most common microorganism among Gram negative organism was Klebsiella found in 52% cases followed by E. coli in 36% cases, both were found together in 5% cases in rest of the cases Proteus and Pseudomonas were found. Sensitivity was found to ceftrioxone, ciprofloxacillin and amikacin in more than 87% of gram negative organism while resistance was seen to ampicillin and clotrimoxazole other antibiotics that showed sensitivity to microorganism were Linizoild and minocycline in 76% cases.Around 8% fluid showed presence of methicillin resistant or sensitive Staphylococcus aureus and both were

sensitive to linezolid and minocycline and resistant to Penicillin, erythromycin and cephxatin.

A total of 25 patients had E. coli growth in the appendix [n=12], followed bythe ileum [n=5]. A total of 6 patients had Enterococcus faecalis growth atthe duodenum [n=3], followed by ileum [n=2]. Pseudomonas aeuroginosa growth was observed at the duodenum [n=1]. Citrobacter freundii growth was observed at the caecum [n=1], and Acinetobacter baumanii complex growth was observed at the gastric [n=1]. A total of 3 patients had Klebsiellapneumoniae growth, and 5 patients had Entero bactercloacae complex growth. Noaerobic growth was observed in [n=18] patients. The majority of the patients who had *E.coli* growth also had maximum antibiotic sensitivity to Amikac in [n=24], followed by Tigecycline [n=23], andonly [n=2] patients were sensitive to Ciprofloxacin. The patients who had *Enterococcusfaecalis* growth were sensitive to Amika cin[n=5], Bezylpenicillin [n=4] and so on. Patients who had *Pseudomonas aeuroginosa* growth were sensitive to Cefipime [n=1], Cefoperazone-Sulbactam [n=1] andColistin [n=1]. Patients who had *Klebsiella pneumoniae* growth were sensitive to Cefipime [n=2], Amikacin [n=2], Colistin [n=2] and Tigecycline [n=2].Patients with *Enterobacter cloacae c*omplex growth were also sensitive toTigecycline [n=5], Amikacin[n=4], Imipenem [n=4], Meropenem [n=4] and soon. Patients with the growth of *Streptococcussanguinis* Gp were sensitive to Trime tho prime-Sulfamethoxazole[n=2],Imipenem[n=1] and soon. Patients with *Citrobacter freundii* growth were sensitive to Amikacin [n=1], Imipenem [n=1] and so on. Patients with Acinetobacter baumanii complex growth were only sensitive to Minocycline[n=1].

Kamble, Jaiswal et al[13] conducted a study on prognostic factors in perforative peritonitis aimed to identify factors in patients with peritonitis which have a significant bearing on morbidity and mortality. 50 patients with perforative peritonitis presented to the emergency of Lokmanya Tilak Municipal Hospital, Mumbai were included in our study mean age of presentation 36.80 years and mortality rate was 16%. Majority of cases were male 88%. Tuberculosis was the most common co-morbidity (16%) most common site of perforation was gastroduodenal perforation (61%) [duodenum (48.9%) gastric (12.8%)] with peptic ulcer as the most common histopathology. They found most of the patients having no growth (46%) in peritoneal contamination followed by E. coli (34%) highly sensitive to amikacin. Klebsiella (16%) sensitive to amikacin, ceftriaxone and ceftazidime (75%).

The limitation the study is small sample size.

## CONCLUSION

Authors found that the most common site of perforation was Appendix, followed by gastric and duodenum. Peritonitis was most commonly due to E. coli, followed by Enterococcus faecalis. E. coli has also emerged as the predominant organism implicated in the pathogenesis, even if we consider it a site-specific culture. E. coli was sensitive to Amikacin, Tigecycline, Gentamycin, Imipenem, Meropenem and Cefoperazone-sulbactam. At the same time E. coli was resistant to Cefipime, Ceftriaxone and Ciprofloxacin. Surgical Site Infection was the most common postoperative complication.

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