SENSITIVITY PATTERNS IN PATIENTS OF ACUTE SECONDARY BACTERIAL PERITONITIS, AND SUGGESTED EMPIRICAL TREATMENT

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ABSTRACT

**Objective:** The objective of this study was to determine the common pathogens in acute secondary bacterial peritonitis patients in tertiary care hospitals and their sensitivity to the antibiotics commonly prescribed for acute bacterial peritonitis.

**Material and Methods:** We conducted culture sensitivity studies on 25 consecutive laprotomies for secondary peritonitis presented to our Surgical-A ward through Accident and Emergency Department without any advance knowledge of their prior antibiotic therapy or culture sensitivity of the pathogens involved.

**Results:** We were able to get the results for 19 out of 25 cases. They yielded mixed cultures of Gram negative rods and staphylococci. There were three cases of MRSA. They were tested against various antibiotics. Resistance of the Gram negative rods to various antibiotics was also high.

**Conclusion:** Our study revealed a very disturbing trend of occurrence of high resistance strains in patient coming directly from the community. We suggest empirical therapies in view of our results and current medical literature. The high bacterial resistance encountered necessitates use of broad-spectrum and expensive antibiotics.

**Key words:** Acute Secondary Bacterial Peritonitis, Culture sensitivity, Gram negative rods, Staphylococci.

INTRODUCTION

Acute generalized bacterial peritonitis is one of the most serious emergencies in surgical patients. The common causes are acute appendicitis, diverticulitis, enteric and tubercular perforations, and trauma. Iatrogenic injuries, ischemic perforations, in-
flammatory bowel diseases are less common causes in this part of the world.\textsuperscript{1,2,3,4,5} We conducted a study on 25 consecutive patients who underwent laparotomy without having advance knowledge of the original pathology or history of antibiotic therapy to see the sensitivity pattern with the aim to have some idea of effectiveness of the empirical antimicrobial therapy in these patients. The empirical therapy was started while waiting for the culture results or in the areas where the facilities are not available.

**MATERIAL AND METHODS**

Aerobic cultures and sensitivity tests of pus from 19 cases of peritonitis due to different gut pathologies and trauma was done on the cases admitted via A & E department into the Surgical-A ward of the Lady Reading Hospital, Peshawar from the 18\textsuperscript{th} of November, 2000, till the 31\textsuperscript{st} of March, 2001. After admission, laparotomy was done, and samples were taken. The anaerobic cultures were not done because of non availability of the transport medium and culture facilities.

In this study amikacin, aztreonam, cephperazone, cephoxime, cephtriazone, cephradine, ciprofloxacin, clarithromycin, co-amoxiclav, co-trimoxazole, enoxacin, erythromycin, gentamicin, imipenem, maxifloxacin, ofloxacine, sparaxin, targcid and vancomycin were tested.

**RESULTS**

- There were 12 males and 7 females in the study groups (Figure 1).
- Four were up to the age of 20 years, 5 were from 21 to 40 years of age, 7 were from 41 to 60 years of age, 3 were above 60. (Figure 2)
- In these 19 patients, there were two (10.53\%) with no pathogens reported and 17 (89.47\%) samples yielded growth of bacteria. There were 21 bacterial isolates obtained in all. (Figure 3)
  - Two had no growth
  - Four had mixed growth of *E.coli* and other Gram negative rod, such as *Proteus* (1), *Morganella* (1), and *Pseudomonas aeruginosa* (2).
  - Two had pure growth of *E.coli* (total were 6), one of *Enterobacter*.
  - Eight had *Staphylococcus aureus*, of which 3 were of the notorious MRSA strain.
  - Two had pure growth of *Pseudomonas aeruginosa*.

These were tested against different antibiotics and therapeutic agents. All three MRSA staphylococci were sensitive to Glycopeptides (i.e., Vancomycin, and Teicoplanin). They were not tested against carbapenems. (Figure 4)

Non MRSA staphylococci were present in five cases, out of which 4 were sensitive
Incidence of bacteria in the study

![Bar chart showing incidence of various bacteria](chart1)

**Fig. 3**

Sensitivity of MRSA

![Bar chart showing sensitivity of different antibiotics](chart2)

**Fig. 4**
Sensitivity of pathogens to 3rd Generation Cephalosporins

![Graph showing sensitivity of pathogens to 3rd Generation Cephalosporins.]

- **Incidence**
- **No. of Tests**
- **Sensitivity to 3rd Generation Cephalosporin (Partial or Full)**

Pathogens

Fig. 5

Sensitivity of pathogens to 4-fluoro-quinolones

![Graph showing sensitivity of pathogens to 4-fluoro-quinolones.]

- **Incidence**
- **No. of Tests**
- **Sensitivity to Quinolones**

Pathogens

Fig. 6
Sensitivity Patterns in Patients of Acute Secondary Bacterial Peritonitis, and Suggested Empirical Treatment

Sensitivity of pathogens to Carbapenem

![Graph showing sensitivity of pathogens to Carbapenem]

- Staph aureus
- E coli
- Pseudomonas aeruginosa

<table>
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<th>Pathogen</th>
<th>Incidence</th>
<th>No. of Tests</th>
<th>Sensitivity to Carbapenem</th>
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<td>Staph aureus (MRSA)</td>
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<td>4</td>
</tr>
<tr>
<td>E coli</td>
<td>5</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
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</table>

Fig. 7

Sensitivity of pathogens to Amikacin and Gentamicin

![Graph showing sensitivity of pathogens to Amikacin and Gentamicin]

- Staph aureus
- E coli
- Proteus vulgaris
- Pseudomonas aeruginosa
- Morganella morganii

<table>
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<tr>
<th>Pathogens</th>
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<th>No. of Tests of Amikacin</th>
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<td>E coli</td>
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<td>Proteus vulgaris</td>
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<td>Pseudomonas aeruginosa</td>
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<tr>
<td>Morganella morganii</td>
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Fig. 8
to 3rd generation cephalosporin, 2 out of 5 were sensitive to 4-fluoroquinolones. 3 out of 5 were tested against imipenem, which was efficacious in all cases. All the cases were tested against aminoglycosides (amikacin and gentamycin), and only one cases was sensitive to amikacin. Sensitivity of different types of 3rd generation cephalosporin is shown in Figure 5.

Sensitivity of 4-fluoroquinolones is shown in Figure 6.

Sensitivity to Carbapenems are the best but unfortunately not tested against the MRSA. (Figure 7)

Sensitivity of aminoglycosides is shown in Figure 8. Aminoglycosides still has shown good results with gram negative rods.

**DISCUSSION**

Acute bacterial peritonitis is infection of the peritoneal cavity by bacteria. It can be primary, spontaneous or secondary to injury. Primary peritonitis is a pediatric condition in which the infant has peritonitis due to infection by pneumococcus, Streptococcus pyogenes, or H influenzae, with no preceding injury. Spontaneous Peritonitis may be associated with ascites, in which colonic flora, may relocate into the peritoneum and cause peritonitis with no antecedent injury or operation. Acute secondary bacterial peritonitis is bacterial infection secondary to injury to the abdominal wall or the gastrointestinal tract or both, due to trauma, operation or perforation due to disease process. Most commonly the infection is dominated by coliforms, which maybe the primary or secondary colonizers. Depending on the site of rupture, the clinical picture may be influenced by the presence or absence of chemical injury. For example, in perforated peptic ulcer, the onset of the symptoms of peritonism may precede real bacterial infection by hours.

Peritonitis can be localized or generalized, depending upon location, drainage of pus, presence or absence of omentum and other loops of intestine, and the general immunity of the patient. Later on, the spread of the peritonitis and progression to septicemia also depends upon species, virulence, load of bacteria and its sensitivity to antimicrobial agents.

Usually, the culture is dominated by a mixed growth of aerobes and anaerobes of colonic origin. In the early stages, the coliform aerobes are more important, and prepare the site for colonization by anaerobes, which cause abscess formation in later stages of the disease. Rarely, especially in patient with hydrocephalous who have a VP shunt, the peritonitis may be caused by Staphylococcus aureus.

If one knows the particular bacterial species involved and their culture sensitivities, one may tailor treatment accordingly. However, ground realities and clinical imperatives do not allow such cultural studies to be done routinely, and most treatment has to be empirical. However, one must not disregard surgical management of acute secondary bacterial peritonitis (i.e., drainage and lavage as needed) which always will remain the cornerstone of peritonitis management.

In this study, there was, as one may expect, mixed growth. The biggest group of Gram negative rods was E coli, closely followed by Pseudomonas. There were five cases of simple Staphylococcus aureus. However, one perturbing feature was the presence of three cases of MRSA out of nineteen patients coming from the community. This is a very high percentage community acquired MRSA, and may hypothesize that this is due to misuse of antibiotics in the community. Academically, MRSA is considered to be a nosocomial infection, and a serious complication of hospitalization.
The focus of this study was on the major groups of antimicrobial agents routinely used in peritonitis: aminoglycosides, 4-fluoroquinolones, and 3rd generation cephalosporin. Some tests were done on carbenems; however, due to paucity of supplies, a more complete testing for this group was not possible. Also, a comparison of amikacin and gentamycin was done.

*E. coli* is considered to be the predominant coliform, and was the biggest group of Gram negative rods in our study, as expected. *E. coli* showed 67% sensitivity to 4-fluoroquinolones, gentamycin and third generation cephalosporin, and 100% sensitivity to amikacin. 2 cases were tested with carbenems, giving 100% sensitivity.

The next major group was the *Pseudomonas* (4), which shown 75% sensitivity to 3rd generation cephalosporin, 4-fluoroquinolones, and gentamycin, and 100% sensitivity to amikacin. Four cases were tested with carbenems, giving 100% sensitivity.

Methicillin sensitive *Staphylococcus aureus* (5) shown 80% sensitivity to 3rd generation cephalosporin, 40% to 4-fluoroquinolones, 25% to amikacin (4 cases), and 100% (3 cases) to carbenems. Gentamycin was not tested.

MRSA was sensitive only to vancomycin and teicoplanin.

**CONCLUSION**

In the light of this study, it is apparent that according to sensitivity, the appropriate empirical regimes for acute secondary bacterial peritonitis include aminoglycoside (preferably large single dose of amikacin or gentamycin) plus a broad-spectrum penicillin with beta-lactamase inhibitor (e.g., co-amoxiclav). Anaerobe cover may be necessary, but is beyond the scope of this study. Alternatives include 4-fluoroquinolone with anti-anaerobes, or high dose 3rd generation cephalosporin (e.g., 2g tds cefotaxime for 5 days) with anti-anaerobes. This regimen is preferred because of its safety. Carbapenems are very broad-spectrum drugs, effective, but can lead to super-infection of the colon, which along with its cost, tempers its use in common clinical practice. Aztreonam/clindamycin combination had a similar efficacy to that of tobramycin/clindamycin in randomized trials, and thus can serve as a substitute for aminoglycosides. Aztreonam can also replace penicillins or cephalosporin in sensitive patients, but cross-sensitivity has been known to occur. For MRSA, vancomycin or teicoplanin can be used, as is evident from the results of this study.

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