

## Original Research Article

<https://doi.org/10.20546/ijcmas.2017.604.261>

## Multidrug Resistant Gram Negative Bacilli Causing Surgical Site Infections: Isolation and Antimicrobial Susceptibility

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### ABSTRACT

Surgical site infections (SSI) are the third most commonly reported nosocomial infections. They have been responsible for the increasing cost, morbidity and mortality related to surgical operations. SSI rate has varied from a low of 2.5% to a high of 41.9%. The common organisms encountered in post-operative wound infections are *Staphylococcus aureus*, Coagulase-negative staphylococci, *Escherichia coli*, *Enterococcus*, *Proteus*, *Pseudomonas* and *Klebsiella* species. A working knowledge of the most likely causative organism and the prevailing antibiotic sensitivity/resistance pattern will be of great help to treating physician and patient. The objective of this study was to isolate and identify various gram negative bacilli from surgical site infected cases and determine their antimicrobial susceptibility pattern. Out of 100 culture positive isolates, a total of 66 gram negative bacilli were isolated from infected surgical sites. All isolates were identified as per standard procedures. Antimicrobial susceptibility testing of all isolates was done by Kirby Bauer disc diffusion method as per CLSI guidelines. ESBL production by both double disc synergy test and phenotypic confirmation test recommended by CLSI was performed. Most common gram negative bacilli isolated was *P. aeruginosa* 31 (26%) followed by *K. pneumoniae* 10 (8.40%), *E. coli* 9 (7.56%), *C. freundii* 6 (5.05%), *K. oxytoca* 5 (4.20%), *Acinetobacter sp.* 3 (2.52%) and *C. koseri* 2 (1.68%). All isolates were sensitive to imipenem. The emergence of Gram-negative bacterial species with acquired resistance to various broad spectrum betalactams is becoming a worldwide clinical problem.

#### Keywords

Surgical site infection,  
Extended spectrum beta lactamases,  
*Pseudomonas aeruginosa*,  
post-operative wound infection.

#### Article Info

Accepted:  
20 March 2017  
Available Online:  
10 April 2017

### Introduction

Joseph Lister (1827–1912) made one of the great contributions to surgery by demonstrating that antiseptics could prevent infection. The antiseptic principle or Listerian method emphasized antiseptic treatment of wounds after the operation (Howard *et al.*, 2010).

Wound infection and other postoperative infections continue to be a problem even

though antibiotics have reduced their risk. The widespread use of antibiotics has even led to the emergence of strains of antibiotic-resistant bacteria. The nature of postoperative infections has also changed because of the many patients (debilitated, elderly, cancer patients) being operated on who have compromised host defenses or who are given drugs that inhibit host defenses (cancer chemotherapy agents, immunosuppressants to

prevent organ transplant rejection) (Howard *et al.*, 2010). About 77% of the deaths of surgical patients are related to surgical wound infection (Goswami *et al.*, 2011).

When there is a decrease in integrity and protective function of the skin, large number of different pathogens will enter into the wound and initiate an inflammatory response characterized by the classic signs of redness, pain, swelling, raised temperature and fever. This process ultimately aims to restore homeostasis. Most post-operative wounds are endogenous and are acquired from the skin, mucous membranes or gastrointestinal tract of the patient. Exogenous infections are mainly acquired from the nose or skin flora of the operating team and transmitted on the hands of the surgeon or through the air directly or indirectly from instruments (Sanjay, 2010).

Whichever organism(s) is involved depends on the locations of the wound, exposure of the patients and the hospital hygiene. Nosocomial infection poses a great threat to surgical wound management especially when the microbe involved is resistant to conventional antibiotics in wound management (Adegoke *et al.*, 2010).

Incidence of SSI in India reported to vary from 3.6% to 22.5% (Jain *et al.*, 2014).

Microbial contamination of the surgical site is a necessary precursor of SSI. Quantitatively it has been shown that, if a surgical site is contaminated with  $>10^5$  microorganisms per gram of tissue, the risk of SSI is markedly increased. However the dose of contaminating microorganisms required for producing infection may be much lower when foreign material is present at the site (Mangram *et al.*, 1999).

Sources of SSI can include the patient's own normal flora or organisms present in the

hospital environment. The common organisms encountered in post-operative wound infections are *Staphylococcus aureus*, Coagulase-negative *Staphylococci*, *Enterococci*, *Proteus*, *Pseudomonas*, *Escherichia coli* and *Klebsiella* species. In the case of wound infections following appendectomy or other lower bowel surgery, indigenous flora of the lower gastrointestinal tract like *Escherichia coli* are involved (Forbes *et al.*, 2002).

Each hospital has its own unique bacterial flora to which patients are at risk for acquiring infection during hospitalization.

In such situations microorganisms exhibit unique pattern of antimicrobial activity during a certain period of time. Only when such epidemiological data are available can the surgeon employ a logical approach towards surgical site infection control.

## **Materials and Methods**

This study included all cases of post operative wound infections occurring in surgeries of different specialties of rural teaching hospital, during the study period.

Informed consent was taken from all the patients.

Pus specimen was collected from all infected surgical sites under aseptic precautions from the depth of the wound by using 2 sterile cotton swabs.

The specimens were brought to the laboratory within 2 hrs of collection and they were subjected to Gram stain on direct smear and culture on 5% sheep blood agar and MacConkey agar. The culture plates were incubated at 37°C for 18 to 24 hrs. Aerobic bacteria grown were identified by conventional methods.

Antibiotic sensitivity of aerobic bacterial isolates was performed on Mueller Hinton agar (MHA) plates by standardized Kirby Bauer disc diffusion technique as per the CLSI guidelines.

### **Detection of extended spectrum beta lactamase in Gram negative bacilli**

Isolate that showed resistance to at least one of the third generation cephalosporins (ceftazidime, cefotaxime, ceftriaxone, cefpodoxime) were tested for ESBL production by both double disc synergy test and phenotypic confirmation test recommended by CLSI.

### **Double disc synergy test (Jarlier *et al.*, 1988) (DDST)**

The inoculum for the test was adjusted to a turbidity of McFarland 0.5 standard ( $1.5 \times 10^8$  CFU/ml) standard. 3rd generation cephalosporins and augmentin (amoxicillin/clavulanic acid) discs were placed at 15-30mm apart from centre to centre of the discs on lawn culture of test strain, on Mueller- Hinton agar (MHA). The increase in size of zone of inhibition of cephalosporin towards augmentin disc is considered positive. This test is easy to perform and interpret. But this test is not specific for ESBLs and is technically difficult. There is no standard recommendation for distance between the two discs and for interpretation of increase in zone of inhibition size for positivity.

### **Phenotypic confirmatory disc diffusion test (PCDDT) (CLSI phenotypic confirmatory disc diffusion test/ phenotypic confirmatory test) (CLSI, 2010)**

On the lawn culture of the organism, 4 discs of ceftazidime (30 $\mu$ g), ceftazidime/clavulanic acid (30/10 $\mu$ g), cefotaxime (30 $\mu$ g),

cefotaxime/clavulanic acid (30/10 $\mu$ g) were placed at 20mm distance from centre to centre. If the strain was resistant to plain cephalosporin, but showed increase in zone of inhibition of > 5mm at the clavulanate potentiated disc of cephalosporins, the test was considered positive.

All statistical analysis was performed using SPSS 11.5 version software. The association between different variables was tested using non-parametric tests.

P value < 0.05 was considered as significant association between the variables tested.

Chi-square test / Fischer's Exact test has been used to find the significance of study parameters on categorical scale between two groups.

## **Results and Discussion**

A total of 119 bacterial species were isolated from 100 SSI culture positive cases. *Staphylococcus aureus* was the most frequent bacterial species (28.57%) isolated followed by *P. aeruginosa* (26%), Coagulase negative *Staphylococci* (10.92%), *K. pneumoniae* (8.4%), *E. coli* (7.56%), *Enterococcus spp* (5.05%), *C. freundii* (5.05%), *K. oxytoca* (4.2%), *Acinetobacter spp* (2.52%) and *C. koseri* (1.68%).

A total of 66 (55.46%) isolates were Gram negative bacilli with *P. aeruginosa* 31 (26%) being the predominant followed by *K. pneumoniae* 10 (8.40%), *E. coli* 9 (7.56%), *C. freundii* 6 (5.05%), *K. oxytoca* 5 (4.20%), *Acinetobacter sp.* 3 (2.52%) and *C. koseri* 2 (1.68%) (Table 1). Out of 66 Gram negative bacteria subjected to ESBL screening test, 47 were found to be positive. Confirmatory test was then done by two methods, namely, the PCDDT and DDST.

**Table.1** Frequency of isolation of Gram negative bacilli in SSI cases

<b>Organism isolated</b>	<b>No. of cases</b>	<b>Percentage</b>
<i>Pseudomonas aeruginosa</i>	31	26
<i>Klebsiella pneumoniae</i>	10	8.40
<i>Escherichia coli</i>	9	7.56
<i>Citrobacter freundii</i>	6	5.05
<i>Klebsiella oxytoca</i>	5	4.20
<i>Acinetobacter spp</i>	3	2.52
<i>Citrobacter koseri</i>	2	1.68
Total	66	55.46

**Table.2** Bacterial species associated with polymicrobial SSI cases (n=100)

<b>S.no.</b>	<b>Organisms</b>	<b>No. of cases</b>
1	<i>P. aeruginosa</i> and <i>S. aureus</i>	4(25%)
2	<i>P. aeruginosa</i> and <i>K. pneumoniae</i>	2(12.5%)
3	<i>P. aeruginosa</i> and <i>CONS</i>	2(12.5%)
4	<i>Enterococcus spp.</i> and <i>K. pneumoniae</i>	2(12.5%)
5	<i>Enterococcus spp.</i> and <i>K. oxytoca</i>	1(6.25%)
6	<i>C. koseri</i> and <i>K. oxytoca</i>	1(6.25%)
7	<i>C. koseri</i> and <i>E. coli</i>	1(6.25%)
8	<i>C. freundii</i> , <i>P. aeruginosa</i> and <i>Enterococcus spp</i>	1(6.25%)
9	<i>C. freundii</i> , <i>K. oxytoca</i> and <i>CONS</i>	1(6.25%)
10	<i>C. freundii</i> , <i>S. aureus</i> and <i>K. pneumoniae</i>	1(6.25%)
	Total	16(100%)

**Table.3** Antibiotic resistance pattern of GNBs

Antibiotics	<i>P.aeruginosa</i>	<i>K.pneumoniae</i>	<i>E. coli</i>	<i>C.freundii</i>	<i>K.oxytoca</i>	<i>Acinetobacter spp.</i>	<i>C.koseri</i>
Amoxyclav	90.32%	70%	55.55%	100%	80%	33.33%	50%
Ciprofloxacin	64.52%	20%	44.44%	16.67%	80%	0%	0%
Amikacin	41.93%	0%	11.11%	16.67%	0%	0%	50%
Gentamicin	67.74%	30%	44.44%	66.66%	40%	0%	0%
Cotrimoxazole	87.09%	60%	88.88%	50%	60%	66.66%	100%
Ceftriaxone	61.29%	70%	66.66%	66.66%	80%	100%	50%
Cefotaxime	67.74%	70%	66.66%	66.66%	80%	100%	50%
Ceftazidime	58.06%	70%	77.77%	33.33%	80%	33.33%	0%
Sparfloxacin	61.29%	50%	77.77%	50%	100%	0%	50%
Cefoperazone-sulbactam	77.42%	40%	22.22%	0%	60%	0%	0%
Ceftriaxone-tazobactam	67.74%	40%	22.22%	0%	60%	33.33%	0%
Piperacillin-tazobactam	48.39%	20%	22.22%	0%	0%	0%	0%
Tobramycin	51.61%	10%	22.22%	0%	20%	0%	0%
Meropenem	41.93%	10%	22.22%	0%	20%	0%	0%
Imipenem	0%	0%	0%	0%	0%	0%	0%

**Table.4** ESBL detection in GNBs

Organism	ESBL	Non ESBL
<i>P. aeruginosa</i> (31)	11(35.48%)	20(64.52%)
<i>Klebsiella spp.</i> (15)	4(26.66%)	11(73.34%)
<i>E. coli</i> (9)	2(22.22%)	7(77.78%)
<i>Citrobacter spp.</i> (8)	0	8(100%)
<i>Acinetobacter spp.</i> (3)	0	3(100%)
Total(66)	17(25.76%)	49(74.24%)

**Table.5** Comparison of methods of ESBL detection

Test	Positive		Negative	
	No.	%	No.	%
DDST	4	8.51%	43	91.49%
PCDDT	17	36.17%	30	63.83%

(p<0.01)

Sensitivity

= 80.95%, 95% CI (58.08% to 94.44%)

Specificity

= 58.90%, 95% CI (46.77% to 70.29%)

**Table.6** Antibiotic sensitivity of ESBL and Non ESBL producers

Antibiotics	ESBL(n=17)		Non ESBL(n=49)	
	No.	%	No.	%
Amoxyclav	0	0%	16	32.65%
Ciprofloxacin	2	11.76%	33	67.35%
Amikacin	10	58.82%	39	79.59%
Gentamicin	2	11.76%	30	61.22%
Cotrimoxazole	0	0%	15	30.61%
Ceftriaxone	2	11.76%	20	40.82%
Cefotaxime	0	0%	20	40.82%
Ceftazidime	2	11.76%	25	51.02%
Sparfloxacin	2	11.76%	24	48.98%
Cefoperazone-sulbactam	0	0%	33	67.35%
Ceftriaxone-tazobactam	0	0%	35	71.43%
Piperacillin-tazobactam	6	35.29%	41	83.67%
Tobramycin	6	35.29%	40	81.63%
Meropenem	8	47.06%	41	83.67%
Imipenem	17	100%	49	100%

**Table.7** Incidence of ESBL in various studies

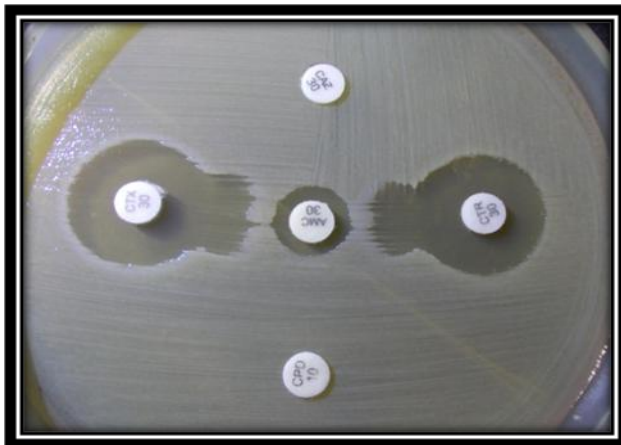
Study	Year	ESBL %
Sanjay <i>et al.</i> ,	2010	23.14%
Mawalla <i>et al.</i> ,	2011	70.83%
Malik <i>et al.</i> ,	2011	67.10%
Sharan <i>et al.</i> ,	2012	30.77%
Wassef <i>et al.</i> ,	2012	41.80%
Present study	-	25.76%



**Table.8** Comparison of different methods of ESBL detection

Authors	PCDDT	DDST
Metri <i>et al.</i> , (2011)	46.40%	42.90%
Dalela <i>et al.</i> , (2012)	58.10%	51.20%
Chugh <i>et al.</i> , 2012	74.04%	40.70%
Oberoi <i>et al.</i> , 2013	33.80%	33.80%
Sanjay <i>et al.</i> , (2010)	-	23.14%
Present study	36.17%	8.51%

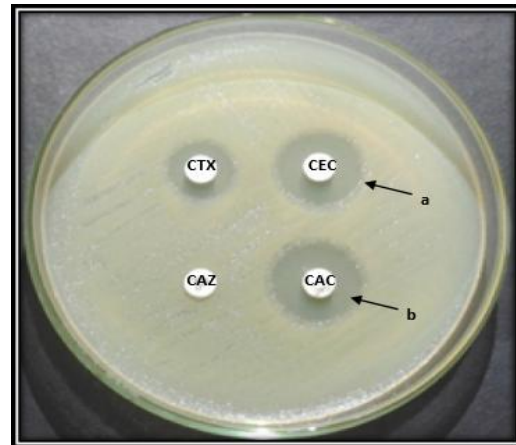
**Fig.1** Double disc synergy test



The results obtained as in table 5. From the above table 5, it can be seen that, the screening test was evaluated for validity (accuracy) by comparing with both the confirmatory tests PCDDT and DDST. It was found that the ability to identify true positives (sensitivity) was found to be 80.95% and the ability to identify true negatives (specificity) was found to be 58.90% when screening test was compared with PCDDT. It was found to be statistically significant by Fischer's exact test ( $p < 0.01$ ).

Infections of the surgical sites are now referred to as surgical site infection (SSI) (Barie *et al.*, 2005; Horan *et al.*, 1992).

**Fig.2** Phenotypic confirmatory disc diffusion test (PCDDT). a & b showing zone of enhancement  $> 5$ mm with  $\beta$ -lactamase inhibitor. CAZ – ceftazidime, CAC – ceftazidime + clavulanic acid, CTX – cefotaxime, CEC - cefotaxime + clavulanic acid



Before mid 19<sup>th</sup> century SSI were termed 'irritable fever' which was followed by purulent discharge from incisions, overwhelming sepsis and often death. It was not till 1865, after Joseph Lister introduced antiseptics that post operative infection morbidity decreased substantially. Subsequently antiseptic surgery was replaced by aseptic surgery.

SSI accounts for approximately a quarter of all nosocomial infections (Barie *et al.*, 2005).

SSI is recognized as a common surgical complication. Potential complications include tissue destruction, failure or prolongation of

proper wound healing, incisional hernias, occasional bacteremia, recurrent pains, disfiguring and disabling scars. SSI also results in substantial morbidity, prolonged hospital stays and increased direct patient costs. SSI continues to be a major problem even in hospitals with most modern facilities and standard protocols of preoperative preparation and antibiotic prophylaxis (Wassef *et al.*, 2012; Yalcin *et al.*, 1995).

Surgical site infection rate varied from 2.5% to as high as 41.9% (Anvikar *et al.*, 1999). In this study we recorded overall 6.97% SSIs.

Indian reports vary from 4% to 30% (Bandaru *et al.*, 2012). Our finding correlates with some of the earlier reports (Shahane *et al.*, 2012; Jain *et al.*, 2013; Reddy *et al.*, 2014) who reported 6.0%, 6.97% and 6.8% respectively.

Microbiological study of the samples obtained from SSI cases revealed that 84% cases were monomicrobial infections. Only 16% of the SSI were polymicrobial infections. 55% to 75% monomicrobial and 25% to 45% polymicrobial infections of surgical wound were reported by others (Jain *et al.*, 2013; Mahesh *et al.*, 2010).

In this study, polymicrobial infections included both Gram positive and Gram negative bacteria which is similar to previous reports (Table 2) (Bhatia *et al.*, 2003).

Similar to earlier reports, Gram negative bacilli were more associated with SSI in our study. It is disheartening to note that *P. aeruginosa* was most commonly isolated organism (56.25%) in polymicrobial SSI, because it is always difficult to treat *Pseudomonas* infection due to high level intrinsic and acquired drug resistance. In our study we found 4 (25%) cases of *S. aureus* and *P. aeruginosa* together out of 16 polymicrobial infections. Similarly Onche *et*

*al.*, (2004) reported 3(21.4%) cases were polymicrobial infections with *S. aureus* and *Pseudomonas spp.* out of 15 cases of mixed infections in their study.

Gram negative bacteria are, as already mentioned, intrinsically more resistant to antibiotics than Gram positive bacteria by virtue of their outer membrane porins. The acquired resistance in enteric bacteria are attributed to the wide spread transmission of resistance plasmids among different genera.

*P. aeruginosa*, the second most common pathogen (26%) isolated in our study showed high resistance to amoxyclav (90.32%), cotrimoxazole (87.09%), cefoperazone-sulbactam (77.42%), cefotaxime (67.74%) and gentamicin (67.74%), ciprofloxacin (64.52%), ceftazidime (61.29%). Below 50% resistance was seen to piperacillin-tazobactam (48.39%), amikacin (41.93%) and meropenem (41.93%). The resistant patterns observed by other workers to the drugs are shown in table 7. Unlike other reports, Malik *et al.*, (2011) and Jain *et al.*, (2014), *P. aeruginosa* isolated in our study was completely susceptible to imipenem (Table 3).

Gram negative bacteria in general show relatively high antibiotic resistance by virtue of outer membrane porins which are less permeable to larger antibiotic molecules. *P. aeruginosa* in particular, is extremely resistant to antibiotics as its outer membrane is 100 times less permeable than that of *E. coli* (Brooks *et al.*, 2004).

The recommendation is that clinically significant infections with *P. aeruginosa* should not be treated with single drug therapy because of two reasons:

1. Low success rate with such therapy.
2. Rapid development of resistance when single drugs are employed.



Drug combinations such as ticarcillin/piperacillin and aminoglycoside (tobramycin), aztreonam ± aminoglycosides, ciprofloxacin ± ceftazidime, imipenem/meropenem ± aminoglycoside are used in the therapy (Brooks *et al.*, 2004). The susceptibility patterns of *P. aeruginosa* vary geographically and performance of susceptibility tests should be done as an adjunct to selection of antimicrobial therapy (Brooks *et al.*, 2004).

All *Klebsiella spp* isolates were sensitive to imipenem and amikacin. High resistance (70%) was exhibited to all tested third generation cephalosporins *i.e.*, ceftriaxone, cefotaxime and ceftazidime except cefoperazone-sulbactam (40% vs. 15.38%). Cotrimoxazole resistance was less (60%) as compared to previous reports (83.33%) and 100% (Lakshmidevi, 2009).

When compared to previous reports *Klebsiella* isolates of our study exhibited low resistance to some of the antibiotics used as alternatives to cephalosporins in the treatment of *Klebsiella* infections: gentamicin (30% vs. 35% to 83.33%), Ciprofloxacin (20% vs. 50% to 83.33%), piperacillin-tazobactam (20% vs. 19.23% and 75%), tobramycin (10% vs. 19.23% and 62.5%), meropenem (10% vs. 15.38%).

In the present study, ESBL production was detected in 25.76% among all the Gram negative organisms. This was similar to study done by Sanjay *et al.*, (2010).

In our study, 35.48% of *P. aeruginosa* were ESBL producer which was similar to incidence reported by Wassef *et al.*, (2012) (40% of *P. aeruginosa*).

Though we noted 26.66% of *E. coli* and 22.22% of *K. pneumoniae* as ESBL producers in our study, other studies such as Malik *et al.*, (2011) (72% of *E. coli*, 73% of *Klebsiella spp.*), Sharan *et al.*, (2012) (50% of *E. coli*,

60% of *K. pneumoniae*), Sanjay *et al.*, (2010) (24% of *E. coli*, 34.78% of *Klebsiella spp.*), Jain *et al.*, (42.8% of *E. coli*) and Wassef *et al.*, (2012) (61.1% of *E. coli*, 60.7% of *K. pneumoniae*), have reported a higher incidence.

Beta lactamase production by several Gram negative and Gram positive organisms is perhaps the most important single mechanism of resistance to penicillins and cephalosporins (Robert *et al.*, 2009).

The resistant organisms can be found in a variety of Enterobacteriaceae species, however, the majority of ESBL producing strains are *K. pneumoniae*, *K. oxytoca* and *E. coli*. Other organisms reported to harbor ESBLs include *Enterobacter*, *Salmonella*, *Morganella morganii*, *Proteus mirabilis*, *Serratia marcescens* and *Pseudomonas aeruginosa*. However, the frequency of ESBL production in these organisms is low (Nathisuwan *et al.*, 2001). A comparison of the antibiogram of ESBL producers and non ESBL producers revealed that ESBL producers are much more resistant to many of the commonly used antibiotics than non ESBL producers (Table 6). As reported by Sanjay *et al.*, (2010) ESBL producing organisms exhibit co-resistance to many other classes of antibiotics resulting in limitation of therapeutic option. The emergence of Gram-negative bacterial species with acquired resistance to various broadspectrum betalactams is becoming a worldwide clinical problem (Table 4).

### **Comparison of different methods of ESBL detection**

Gram negative isolates, which were resistant to at least one of the third generation cephalosporins, were processed for ESBL production detection by double disc synergy test (DDST) (Fig. 1) and phenotypic confirmatory disc diffusion test (PCDDT or

CLSI phenotypic confirmatory test) (Fig. 2). In this study, out of 47 ESBL screening positive Gram negative bacteria, 4(8.51%) were positive with DDST and 17(36.17%) were positive with PCDDT and it was statistically significant ( $p < 0.01$ ) using Fischer's exact test. The findings are comparable to the report of Chugh *et al.*, (2012) (Table 8).

### **Antibiotic sensitivity of ESBL and Non ESBL producers**

Isolates that exhibited ESBL production were multidrug resistant and showed co-resistance to various drugs such as gentamicin, amikacin, ceftriaxone, ceftazidime, piperacillin-tazobactam, tobramycin and meropenem. Wassef *et al.*, (2012) also reported co-resistance of ESBL isolates to sulfamethoxazole-trimethoprim, gentamicin, fluoroquinolones, amikacin and carbapenems. Sanjay *et al.*, (2010) also reported co-resistance among ESBL isolates for amoxycylav, ceftazidime, cefotaxime, ciprofloxacin, gentamicin, piperacillin-tazobactam and meropenem. The Non-ESBL isolates were less resistant to commonly used drugs when compared with ESBL isolates which was statistically significant ( $p < 0.05$ ).

In conclusion SSI have been responsible for the increasing cost, morbidity and mortality related to surgical operations and continues to be a major problem even in hospitals with most modern facilities and standard protocols of preoperative preparation and antibiotic prophylaxis. Multidrug resistant gram negative bacilli need to be extensively identified and actively treated.

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**How to cite this article:**

Ashish Bajaj and Sneha Kukanur. 2017. Multidrug Resistant Gram Negative Bacilli Causing Surgical Site Infections: Isolation and Antimicrobial Susceptibility. *Int.J.Curr.Microbiol.App.Sci.* 6(4): 2244-2255. doi: <https://doi.org/10.20546/ijcmas.2017.604.261>