

Screening at Admission for Carrier Prevalence of Multidrug Resistant Organisms: A Hospital Based Observational Study

S. Deepa, S. Vandana and D. Venkatesha

Department of Microbiology, Mysore Medical College & Research Institute, Mysore - 570 001, India.

[dx.doi.org/10.13005/bbra/1273](https://doi.org/10.13005/bbra/1273)

(Received: 02 March 2014; accepted: 04 April 2014)

“Relentless and dizzying rise of antimicrobial resistance” has contributed in a large measure to the persistence of infections as a major cause of morbidity and mortality. The purpose of this study was to catalog the carrier prevalence of MDR bacteria at the community level. It was mainly focused on isolating carriers from anterior nares and the gut and to determine their antimicrobial susceptibility pattern at admission. A total of 100 patients were screened for carriage prevalence of MDR bacteria in anterior nares and gut. Of the 158 isolates obtained from the stool samples, 48 (30.3%) were MDR, 34 (21.5%) were XDR and 3 (1.8%) were PDR. One more point worth noting was the pan-drug resistant organisms in the gut of 3 (03%) patients. 2 (66.6%) of these isolates were *E.coli* and the other was *Enterococci*. On the contrary, of 83 isolates obtained from nasal samples 18 (21.6%) were MDR and 7 (8.43%) were XDR. (26.4%) of the *Staphylococci* obtained were multi-drug resistant. 20 (29.4%) of the *Staphylococci* were MRSA. Of these 14 (70%) could be CA-MRSA. 33 (24.6%) and 30 (22.3%) of the GNBs obtained from the stool samples were ESBL and MBL producers respectively. Thirteen (54.1%) of the isolates of *Enterococci* were multi-drug resistant. 10 (41.6%) of the *Enterococci* were resistant to vancomycin. In this situation, it is perhaps better for all the health care institutions to come up with their own screening strategy, in order to curb the evolution of these multi-drug resistant bacteria to a destructive rate.

Key words: Screening, Antimicrobial resistance.

Bacterial antimicrobial drug resistance is a worldwide problem that is exacerbated by the diminishing number of new antimicrobial drugs in the pharmaceutical pipeline¹. Recent concern has led to plethora of govt. and agency reports advocating less antibacterial use, better infection control and development of new antibacterials². The rapid emergence of resistance to antibiotics amongst pathogens generates visions of the ‘potential post-antibiotic era threatening present

and future medical advances’. Overall burden of bacterial infection is rising, largely fuelled by antimicrobial resistant organisms³.

Certain areas in hospitals like ICUs and areas with immunocompromised and debilitated patients as well as treatment modalities like topical and prophylactic use of antibiotics are foci of generation of multidrug resistant (MDR) bacteria. Gradual dissemination to the community through population interactions spreads the organism widely.⁴ This spread represents the convergence of various factors including mutations, development of selection pressure in hospitals and in the community and inability of some laboratory testing methods to detect emerging resistant phenotypes.^{5,6,7} Expanded use of antimicrobial agents in hospitals and outside the hospital increases the selective pressure for these organisms⁸.

* To whom all correspondence should be addressed.
Mob.: +91-9686229737;
E-mail: drdeepa_intel@yahoo.co.in

The correlation between invitro resistance and treatment failure is imperfect, but resistance undoubtedly increases mortality, morbidity and costs in many settings². While newer drugs have kept the problem under control, the poorer communities are squeezed between the rampant resistance and inadequate resources. Thus the contribution of infectious disease to overall mortality is greater in impoverished societies.³ Thus it is very much necessary to track the misuse of antibiotics. MDR strains not only complicate anti-infective therapy, but it also heightens the need for effective control policies to prevent the spread of MDR organisms to other hospitalized patients.^{9,10} Screening for MDR organisms is one of the many approaches needed to deal with the very major clinical problem concerning drug resistance.

A sensitive, specific and cost effective screening test may help the clinicians in the choice of appropriate antimicrobial therapy, provide baseline data about the epidemiology of MDR pathogen, to guide policy recommendations and help in infection control by early identification of patients, thereby facilitating an informed decision about infection control interventions.

These interventions may include notification to the concerned clinical and nursing team, infection control precautions such as isolation or cohort nursing of such patients, use of decolonization regimens, use of antibacterial prophylaxis during surgery or other invasive interventions or use of an appropriate agent for empirical antibiotic therapy in case of unconfirmed infection in a colonized patient¹¹.

This approach is wise as suggested by Berkelmen and colleagues, who wrote, "history has shown us repeatedly, in terms of both human suffering and economic loss, that the cost of preparedness through vigilance are far more lower than those needed to respond to unanticipated public health crises"¹².

Also in the Indian scenario, there is a very low coverage of surveillance, poor data management, lack of intersectoral co-operation. With this background, this study was taken up to know the carriage prevalence of multi-drug resistant bacteria and to emphasize on the burden of antimicrobial resistance in the society.

MATERIALS AND METHODS

The present observational study was carried out in the Department of Microbiology, Mysore Medical College and Research Institute, Mysore and its attached hospitals during 2013.

Inclusion criteria

Patients at admission without any past history of long hospital stay or any other hospital admissions.

Exclusion criteria

Patients should not be a health worker, should not have any history of immunocompromised status.

Sample collection

2 nasal swabs and 1 stool sample were collected from each patient. Nasal swabs were taken from anterior nares with a sterile cotton swab. Stool samples were collected in the sterile universal container. Total transit time to the laboratory was within 30 minutes. Samples were processed as per the standard protocols¹³.

Nasal samples

Microscopy and culture of nasal swabs was performed. The (isolates from the nasal swabs were identified with standard biochemical reactions¹⁴.

Antibiotic susceptibility test (AST) was performed using Kirby-Bauer's disk (diffusion test as per CLSI guidelines. Ampicillin-10mcg, erythromycin-15 mcg, clindamycin-2 mcg, cefoxitin-30 mcg, vancomycin-30 mcg, tetracycline- 30 mcg, chloramphenicol-30 mcg, cotrimoxazole -23.75 mcg disks were used to test the antimicrobial susceptibility. Cefoxitin 30 mcg disk was used to identify methicillin resistance.¹⁵ Based on coagulase test and cefoxitin disk screen test, strains were identified as methicillin sensitive CoNS (MS- CoNS), methicillin resistant CoNS (MR-CoNS), methicillin sensitive *Staphylococcus aureus* (MSSA) and methicillin resistant *Staphylococcus aureus* MRSA.

Erythromycin and clindamycin disk were placed at a distance of 21mm to identify D-zone indicating the development of inducible resistance.¹⁵

Stool samples

Stool microscopy was done and samples were cultured and the isolates were identified by standard biochemical reactions.¹⁴ AST was

performed for each isolated bacteria. Ampicillin-10 mcg, tetracycline- 30 mcg, gentamicin-10 mcg, ciprofloxacin-5 mcg, ceftazidime-30 mcg, ceftazidime/clavulanic acid-30/10 mcg, co-trimoxazole- 23.75 mcg and meropenem-10 mcg disks were used to test the susceptibility in the gram-negative bacteria (GNB). A zone diameter of < 16mm around the meropenem disk was presumed to be as MBL-producing organism. A > 5mm increase in zone diameter for ceftazidime in combination with clavulanic acid-30/10 mcg versus ceftazidime-10mcg zone tested alone is taken as (ESBL-producing organism). To test the susceptibility pattern among the *Enterococci*, penicillin-10 units, erythromycin-15 mcg, clindamycin-2mcg, ciprofloxacin-5 mcg, ceftriaxone-30 mcg, vancomycin-30 mcg, linezolid-30 mcg, gentamicin-10 mcg, chloramphenicol-30 mcg co-trimoxazole -23.75 mcg disks were used.

RESULTS

A total of 100 patients were screened at admission or/ within 48 hours of admission.

The age of the population studied ranged between 16-88 years. The mean age was 52 years.

100 nasal swabs yielded 83 isolates. Of these, 56 patients were monobacterial carriers and

15 patients were polybacterial carriers. 29 samples yielded no growth. Most commonly isolated bacteria from the anterior nares were MS-CoNS (33.7%), followed by MRSA (20.4%) and MSSA (15.6%).

100 stool samples yielded 158 isolates. 54 patients were monobacterial carriers and the rest of 46 were polybacterial carriers. The isolates resistant to 3 or > 3 categories of antimicrobials used for testing antimicrobial susceptibility pattern in GNBs were considered as MDR. Those resistant to all the categories except 1 or 2 categories were considered as XDR and those resistant to all the drugs in all the 6 categories of antimicrobials were

Table 1. Isolates obtained from nasal and stool samples

Organisms	Nasal sample n=100	Stool specime n=100
<i>Eschericia coli</i>	08	82
<i>Klebsiella spp.</i>	05	30
<i>Enterobacter</i>	01	12
<i>Citrobacter</i>	00	07
<i>Providencia</i>	01	03
<i>Enterococci</i>	00	24
<i>CoNS</i>	35	0
<i>Staphylococcus aureus</i>	33	0
Total	83	158

Table 2. Antimicrobial resistance pattern among the Staphylococci obtained from nasal samples.

Antimicrobials	MS-CoNS n= 28	MR-CoNS n= 07	MSSA n= 13	MRSA n= 20	Total n=68
Ampicillin	12 (42.8%)	6 (85.7%)	7 (53.8%)	19 (95%)	44 (64.7%)
Erythromycin	11 (39.2%)	1 (14.2%)	4 (30.7%)	5 (25%)	21 (30.8%)
Clindamycin	9 (32.1%)	3 (42.8%)	5 (38.4%)	5 (25%)	22 (32.3%)
Cefoxitne	0 (0%)	7 (100%)	0 (0%)	20 (100%)	27 (39.7%)
Ciprofloxacin	8 (28.5%)	1 (14.2%)	4 (30.7%)	4 (30.7%)	17 (25%)
Vancomycin	3 (10.7%)	2 (28.5%)	3 (23%)	3 (15%)	11 (16.1%)
Tetracycline	4 (14.2%)	1 (14.2%)	5 (38.4%)	1 (50%)	20 (29.4%)
Chloramphenicol	1 (3.5%)	1 (14.2%)	3 (23%)	1 (5%)	6 (6%)
Co-trimoxazole	8 (28.5%)	1 (14.2%)	3 (23%)	4 (20%)	16 (23.5%)

considered as PDR.

- 1) Inducible resistance was seen among 19 (27.9%) isolates. Inducible resistance was seen among 10(28.5%) of the CoNS and among 9(27.2%) of the *Staphylococcus aureus*.
- 2) Among 35 isolates of CoNS, 8 (22.8%) were MDR and 2 (5.7%) were XDR. Among 33 isolates of *Staphylococcus aureus*, 5 (15.15%) were MDR and 3 (9%) were XDR.
- 3) Of the 20 MRSA isolates obtained, 4 (20%)

were MDR and 2 (10%) were XDR. The other 14 (70%) isolates were considered as community acquired MRSA (CA-MRSA). Isolates which were showing resistance to methicillin but sensitive to most of the commonly used antibiotics were considered as possible cases of CA-MRSA.

Of the 15 isolates, 7 (46.6%) isolates were showing resistance to multiple drugs⁵. (33.3%) strains were MDR and 2 (13.33%) strains were

Table 3. Antimicrobial resistance pattern among the GNBs obtained from nasal samples

Antimicrobials	<i>E.coli</i> n= 8	<i>Klebsiella</i> n= 5	<i>Enterobacter</i> n= 1	<i>Providencia</i> n= 1	Total n= 15
Ampicillin	8 (100%)	5 (100%)	1 (100%)	1 (100%)	15 (100%)
Tetracycline	6 (75%)	4 (80%)	0 (0%)	1 (100%)	11 (73.3%)
Gentamicin	3 (37.5%)	0 (0%)	0 (0%)	0 (0%)	3 (20%)
Ciprofloxacin	2 (25%)	3 (60%)	1 (100%)	0 (100%)	6 (40%)
ceftazidime	2 (25%)	1 (20%)	1 (100%)	0 (0%)	4 (26.6%)
Ceftazidime/ clavulanate	5 (62.5%)	1 (20%)	0 (0%)	1 (100%)	7 (46.6%)
Co- trimoxazole	4 (50%)	1 (20%)	0 (0%)	0 (0%)	5 (33.3%)
Meropenem	0 (0%)	1 (20%)	0 (0%)	0 (0%)	1 (6.6%)

Table 4. Antimicrobial resistance pattern among the GNBs isolated from stool samples

Antimicrobials	<i>E.coli</i> n= 82	<i>Klebsiella</i> n= 30	<i>Enterobacter</i> n= 12	<i>Citrobacter</i> n= 7	<i>Providencia</i> n= 3	Total n= 134
Ampicillin	57 (69.5%)	20 (66.6%)	9 (75%)	7 (100%)	1 (33.3%)	94 (70.1%)
Tetracycline	56 (68.2%)	14 (46.6%)	2 (22.2%)	6 (85.7%)	2 (66.6%)	80 (59.7%)
Gentamicin	26 (31.7%)	7 (23.3%)	3 (25%)	3 (42.8%)	0 (0%)	39 (29.1%)
Ciprofloaxacin	32 (39%)	7 (23.3%)	5 (41.6%)	2 (16.6%)	2 (66.6%)	48 (35.8%)
Ceftazidime	27 (32.9%)	11 (36.6%)	4 (33.3%)	2 (28.5%)	2 (66.6%)	46 (34.3%)
Ceftazidime/ Clavulanate	36 (43.9%)	12 (40%)	6 (50%)	5 (71.4%)	1 (33.3%)	60 (44.7%)
Co-trimoxazole	37 (45.1%)	7 (23.3%)	3 (25%)	3 (42.8%)	1 (33.3%)	41 (30.5%)
Meropenem	19 (23.1%)	6 (20%)	2 (16.6%)	2 (42.8%)	1 (33.3%)	30 (22.3%)

XDR. Two isolates (13.3%) of them were ESBL producers and 1 (6.6%) was an MBL producer.

Of the 134 isolates, 72 (53.7%) isolates were showing resistance to multiple drugs. 38 (28.3%) of them were MDR, 32 (23.8%) of them were XDR. Among 134 isolates, 33 (24.6%) were ESBL producers and 30 (22.3%) were MBL producers.

Of the 24 isolates of *Enterococci*, VRE 10 (41.6%), 13 (54.1%) were showing resistance to multiple drugs. 10 (41.6%) were MDR and 2 (8.3%) were XDR and 1 (4.1%) was PDR.

Among 100 patients screened, 14 of them were of the age group of ≤ 60 years. Among this age group,

- 1) 2 (14.2%) of them had acquired MRSA.
- 2) 7 (50%) MDR strains and 3 (21.4%) XDR strains could be isolated from their gut.
- 3) 4 (28.5%) of them were found to colonise ESBL producing bacteria in their gut.
- 4) 6 (42.8%) of them were found to colonise MBL producing bacteria in their gut.

Table 5. Antimicrobial resistance among the *Enterococci* species isolated from stool samples

Antimicrobials	VRE n= 10	Non-VRE n= 14	Total n= 24
Penicillin	10 (100%)	6 (42.8%)	16 (66.6%)
Erythromycin	4 (40%)	7 (20%)	11 (45.8%)
Clindamycin	5 (50%)	8 (57.1%)	13 (54.1%)
Ciprofloxacin	2 (20%)	4 (28.5%)	6 (25%)
Ceftriaxone	0 (0%)	3 (21.4%)	3 (12.5%)
Vancomycin	10 (100%)	0 (0%)	10 (41.6%)
Linezolid	0 (0%)	3 (21.4%)	3 (12.5%)
Gentamicin	5 (50%)	2 (14.2%)	7 (29.1%)
Co-trimoxazole	3 (30%)	3 (21.4%)	6 (25%)

DISCUSSION

Survival of the fittest holds good for the men as well as the bacteria. Some of the non-pathogenic bacteria in nature live as commensals in our body. A limited population of bacteria, which have become pathogenic, were also sensitive to antibiotics to start with. It is the man made antibiotic pressure, which has led to the emergence and spread of resistant genes among the bacteria. In the present study randomly 100 patients at admission or within 48 hours of admission were screened for various drug resistance patterns. Nasal samples yielded 35 (42.16%) of CoNS & 33 (39.75%) of *Staphylococcus aureus*. 25 (30.1%) isolates obtained from the anterior nares were found to be multi-drug resistant. The prevalence of MRSA was 60.60% & MR-CoNS was 20% in our study. Among 20 isolates of MRSA, 14 isolates that amounts to 70% strains were considered to be CA-MRSA as they were commonly sensitive to most categories of antibiotics except few.

In a study by S. Saxena *et al.*, 16 in 2002, of the 317 nasal swabs from healthy individuals in the community, 94 (29.6%) yielded growth of *Staphylococcus aureus* and 17 (18%) of them were MRSA strains. In another observational study by K.E. Vandana, 17 nine (4.28%) nasal swabs from patients 210 collected at the time of admission grew MRSA.

Initially MRSA strains were associated with nosocomial infections, but later they were disseminated into the community. The development of methicillin resistance among *Staphylococcus aureus* in the community may be due to horizontal acquisition of *mecA* gene from the hospital settings. Also health care workers are important reservoirs for transfer of these resistant bacteria through anterior nares. It is very difficult to know the extent of spread of resistant bacteria at the community level except by surveillance studies. However, whether acquired nosocomially or through community, these strains go unrecognized unless the clinical infection develops. This high

carriage of multi drug resistant organisms in nares is definitive risk factor for further cross infections & treatment at the hospital level.

In our study, a total of 33 (24.6%) ESBL producers & 30(22.30%) isolates of MBL producers were obtained. Also among 14 patients ≤ 60 years, four (28.5%) patients were ESBL producers & six (42.8%) patients were found to colonize with MBL producers in their gut. Among the nasal gram negative isolates isolated, 1(6.66%) isolate was an MBL producer.

A study by A.A. Kader and K.A. Kamath¹⁸, regarding the fecal carriage of ESBL producing bacteria in a community in Saudi Arabia isolated 91 (12.7%) of ESBL producers from 716 faecal specimens of which 85 (95.6%) were *E. coli* and 4 (4.4%) were *Klebsiella pneumonia*. In comparison to the study by K.E. Vandana¹⁷, where 21 (18.58%) ESBL producers were isolated from 113 stool samples, in our study 33 (33%) ESBL producers were obtained from 100 stool samples.

Prevalence of these drug resistance patterns is the point of discussion everywhere. Antibiotic use creates a selective pressure on the host bacteria in the large bowel which leads to the emergence of antimicrobial resistant organisms, which in turn increases the number of carriers and enhances the opportunity for these bacteria to cause infections. Without knowing the susceptibility pattern, these resistant bacteria are very difficult to treat, causing treatment failures. Many laboratories fail to detect these resistant patterns. Clinicians should have proper knowledge about the ESBL and MBL producers for choosing better alternatives to avoid unnecessary empirical treatment.

Older age is always a risk factor for any complications. Probably patient compliance with drugs, frequent exposure to antibiotics, cross infections, complications like diabetes mellitus etc can be few reasons for higher levels of MDR bacteria in older age groups.

In our study we isolated²⁴ *Enterococci* from 100 faecal specimens, of which 10 (41.64%) were found to be vancomycin resistant. These findings mirrors those of a study by I. Klare *et al.*,¹⁹ who isolated 12 (12%) faecal glycopeptides resistant (van A type) *Enterococcus faecium* from 100 non hospitalized patients and 5 (38.4%) van A type *E. faecium* from 13 samples of raw minced meat of pigs from different butcher shops. But

in contrast to the study by K.E. Vandana¹⁷, where 3 (6.25%) VRE were obtained from 113 stool samples, in our study the VRE (24%) isolates obtained were higher.

Enterococci are normal constituents of the gut flora of humans and majority of the animals. Despite the high standards of hygiene during the slaughtering process, contamination of the meat and the meat products by the intestinal flora of the slaughtered animals cannot be excluded. Furthermore, the use of antibiotics as feed additives for growth enhancement in animals may be associated with the emergence of VRE. *Enterococci* can reach human consumers via the food chain. However it remains to be elucidated whether VRE of the probable animal sources takes a short passage through the human intestine after food consumption or if they become permanent residents.

Another classical example of emergence of resistance due to abuse of antibiotics is the extensive use of vancomycin. As the infections due to MRSA in hospitals all over the world increased, vancomycin became the drug of the choice to treat these infections. This led to the selection of VRE present as normal flora in the gut of the patient and possibly contributed to the emergence of VRE besides other factors.

One more point worth noting was the pan-drug resistant organisms in the gut of three patients. Two of these isolates were *E. coli* and the other was *Enterococci*. This may be due to extensive exposure of the gut to the antibiotics rather than the anterior nares or the breathing airways.

Various contributing factors other than these included like the combination of poverty and ignorance making the ground perfect for the development of resistance. Community awareness of the issues involved in antibiotic therapy is poor and this is compounded by over the counter drug availability & self-medication. The commonly used drugs are generally inexpensive and popular broad-spectrum agents. Patient's pressure, aggressive marketing by pharmaceutical company, lack of uniformity among physicians to follow antibiotic policy, inadequate intake causing the recurrence of the disease also have added to this practice.

Gene events that cause sensitive bacteria to become resistant could be intrinsic like point mutations or extrinsic like horizontal transfer of resistant genes between the bacteria by the

dissemination of transposons, integrons, plasmids, that give rise to so called gene epidemics. The reasons for this epidemic success of bacteria may be due to increased adherence to host cells, greater tolerance to disinfectants and desiccation, faster growth rates, lack of hygiene in hospitals and disregard to isolation precautions in most of the busy hospitals with limited resources. Also, community acquired resistant strains on admission to hospitals exchange genetic information with nosocomial isolates resulting in 'superbugs' that could cause difficult-to-treat infections.

Antibiotics are widely used in agriculture and aquaculture for therapeutic, prophylactic and growth promoting purposes. Antibiotics in flesh of such animals may enter the human intestine via food chain. Antimicrobial resistant bacteria can also be found on fruits and vegetables due to the spreading of sewage sludge on farmland and direct use of antibiotics on the fruits. Presence of antimicrobial resistant bacteria has also been reported in the fresh water sources. The antibacterial substances added to diverse household cleaning are similar to antibiotics in many ways²⁰. Release of fabric conditioning chemicals into a reed bed system has recently shown to strongly select for Class I integron carriage, which is a key molecular mechanism for the spread of antibiotic resistant genes by horizontal transfer²¹.

The situation is alarming. This emerging threat has to be tackled at the initial phase itself, which could be done through active surveillance of antimicrobial resistance in the community.

In essence, some strategies also aim in optimizing the antibiotic stress in the environment, using decolonization regimens for those harboring resistant bacteria, rotating antibiotics, decrease unintended interaction between antibiotics and pathogens, restrict the spread of resistant organisms and treat infections with minimum amount of antibiotics necessary to effect the cure. Education of professionals and public, accessibility of the microbiological investigation and its results to the general practitioner in an effort to rationalize the choice of antimicrobial therapy, regulatory issues with central prescribing restrictions and advertising restrictions, closer international co-operation is also required to combat this problem. Equally important is the co-ordination of surveillance of antibiotic resistance in human and animal health

sector along with regulating antibiotic use in both sectors, restriction of antibiotic use as growth promoters in animals.

For all this to be successful, the initiative lies in screening the patients at an early stage. Epidemiological surveillance of antimicrobial resistance, is indispensable for empirically treating infections, implementing resistance control measures and preventing spread of antimicrobial organisms in the healthy community.

CONCLUSION

A screen-isolate-destroy strategy is very effective for the control of MDR. Information to the doctors regarding drug susceptibility through routine screening of the patients before admission would be an effort to rationalize the judicious use of antibiotics and to implement the antibiotic policy. Continuously updated data through epidemiological surveillance on antimicrobial resistance will continue to be essential to ensure effective infection control & patient care.

REFERENCES

1. Okonko IO, Soley FA, Amusan TA *et al.* Incidence of Multi-Drug Resistance (MDR) Organisms in Abeokuta, Southwestern Nigeria. *Global J of Pharmacol*, 2009; **3**(2): 69- 80.
2. Dr. David ML. Bacterial resistance: Origins, epidemiology and impact. *Clinical infectious diseases*. 2003; **36**(1): 11-23.
3. D Raghunath. Emerging antibiotic resistance in bacteria with special reference to India. *J. Biosci.* 2008; **33**(4): 593-603.
4. Strulens MJ. RG. The problem of resistance; in Antibiotic and chemotherapy, Chapter 3. Finch, D Greenwood, SR Norrby, *et al.* (Edinburgh: Churchill Livingstone). 8th edition. 2003; 25-47.
5. Tenover FC, Hughes JM. The challenges of emerging infectious diseases: development and spread of multiply resistant bacterial pathogens. *JAMA*. 1996; **275**: 300-304.
6. Doern GV, Brueggemann AB, Blocker M, *et al.* Clonal relationships among high-level penicillin-resistant *Streptococcus pneumoniae* in the United States. *Clin Infect Dis* 1998; **27**: 757-761.
7. Levin BR, Lipsitch M, Perrot V, *et al.* The population genetics of antibiotic resistance. *Clin Infect Dis* 1997; **24**(1): 9-16.
8. Fred C. Tenover. Development and spread of bacterial resistance to antimicrobial agents: an

- overview. *Clinical infectious diseases*. 2001; **33**(3): 108-15.
9. McGowan JE, Tenover FC. Control of antimicrobial resistance in the health care system. *Infect Dis Clin North Am*. 1997; **11**:297–311.
10. Stosor V, Kruszynski J, Suriano T, et al. Molecular epidemiology of vancomycin-resistant enterococci: a 2-year perspective. *Infect Control Hosp Epidemiol* 1999; **20**:653–9.
11. Bhattacharya S. Is screening patients for antibiotic-resistant bacteria justified in the Indian context? *Indian J Med Microbiol*. 2011; **29**:213-7.
12. Berkelman RL, Bryan RT, Osterholm MT, et al. Infectious disease surveillance: a crumbling foundation. *Science*. 1994; **264**: 368-70.
13. Collee GJ, Fraser AG, Marmion BP. Mackie and McCartney's practical medical microbiology, Churchill livingstone, New York, 14th edition; 1996.
14. Forbes BA, Sahm DF, Weissfeld AS. Bailey and Scott's diagnostic microbiology. Elsevier pub, St. Louis 12th edn; 2007: 216-254.
15. Wayne PA, CLSI. Jan 2010. Performance standards for antimicrobial susceptibility testing. CLSI approved standard M02-A10. Clinical and Laboratory Standards Institute, CLSI, Jan 2010.
16. Dr. S Saxena. Prevalence of Methicillin-resistant *Staphylococcus aureus*, Colonization among Healthcare Workers and Healthy Community Residents. *J health popul nutr*:2002 **20**(3): 279-280.
17. Vandana KE, G Varghese, S Krishna et al. screening at admission for carrier prevalence of multi-drug resistant organisms in resource-constrained settings. *Journal of hospital infection*. 2010; **76**(2):180-1.
18. A.A. Kader, K.A. Kamath. Faecal carriage of extended-spectrum β -lactamase-producing bacteria in the community. *Eastern Mediterranean Health Journal*. 2009; **15** (6): 1365-1370.
19. I Klare, H Heier, H Claus et al. Enterococcus faecium strains with van-A mediated high level glycopeptide resistance isolated from animal foodstuffs and faecal samples of humans in the community, *microbial drug resistance*, 1995; **1**(3): 265-272.
20. Levy SB. Antibacterial household products: cause for concern. *Emerg Infect Dis* 2001; **7**(3): 512-515.
21. Gaze WH, Abdoulsalam N, Hawkey PM *et al*. Incidence of class 1 integrons in a quaternary ammonium compound-polluted environment. *Antimicrob Agents Chemother*. 2005; **49**: 1802–1807.