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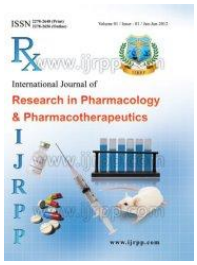

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Emergence of drug resistance of Acinetobacter at tertiary care hospital

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ABSTRACT

BACKGROUND & OBJECTIVE

Acinetobacter, although saprophytic, the emergence and spread of Acinetobacter in hospital environment is a major area of concern. . It has been associated with a wide variety of illnesses in hospitalized patients, especially patients in the intensive care units. These infections are often difficult to treat, because of the widespread antibiotic resistance**.** This study is conducted to know the prevalence and drug resistant pattern of Acinetobacter in tertiary care hospital.

MATERIAL & METHODS

950 isolates of Acinetobacter were obtained from various clinical samples, and subjected to identification by different biochemical tests and antibiotic susceptibility testing by disc diffusion testing by Kirby Baurer method.

RESULT

Out of 950 isolates of Acinetobacter, 82% were Acinetobacter. calcoaceticus baumannii complex remained 18% were other Acinetobacter spp. Acinetobacter isolates were resistant to important groups of antibiotics tested, including amikacin (50%), gentamycin (65%), tobramycin (38%), ceftriaxone (92%), ceftazidime (80%), cefepime (88%), ampicillin-sulbactum (44%), piperacillin-tazobactum (38%),ciprofloxacin (81%) levofloxacin(82%), imipenem (10%) and trimethoprim-sulfamethoxazole (65%), meropenem (15%), polymixin B (2.2%), colistin (2%).

CONCLUSION

Early detection of MDR Acinetobacter and infection control practices are the best defense against these organisms. Rational use of antimicrobials is an important aspect to delay the emergence of XDR and PDR Acinetobacter spp.

KEY WORDS: Acinetobacter, MDR (Multi Drug Resistance), XDR (Extreme Drug Resistance), PDR (Pan Drug Resistance.

INTRODUCTION

Hospitalized patients always remain in the environment of potential pathogens. Among these silent invaders of hospital environment is Acinetobacter spp. It has been associated with a wide variety of illnesses in hospitalized patients, especially patients in the intensive care units.[1] These infections are often difficult to treat, because of the widespread antibiotic resistance. Further, this bacteria survives for a long time in the hospital environment, with enhanced opportunities for transmission between patients.[2] It also contributes to outbreaks in the hospitals. So identification and analysis of its drug resistance is up most important to minimize such nosocomial infections and outbreaks. Acinetobacter is Non motile, Oxidase Negative, and Gram Negative Coccobacilli. It is generally saprophytic, but cause opportunistic infections in seriously ill, hospitalized and immune compromised patients.[3] This study is conducted to know the prevalence and drug resistant pattern of Acinetobacter in tertiary care hospital.

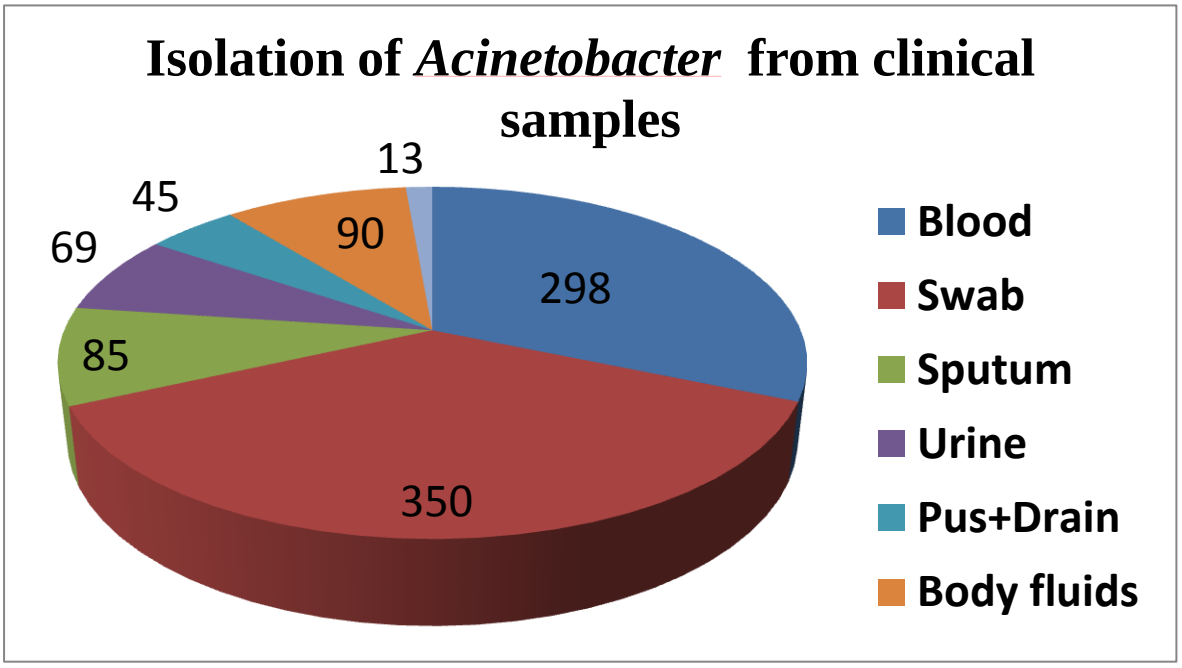
MATERIALS AND METHODS

Total of 950 clinical isolates of Acinetobacter spp. were isolated from different patients admitted to tertiary care hospital, Gujarat, India during 2011-2012.The Acinetobacter isolates were obtained from different clinical specimens including pus, respiratory fluids, blood, tracheal asp, abdominal fluid, wound swabs, sputum and urine. All the clinically isolated samples were identified as Acinetobacter by routine biochemical methods and antimicrobial susceptibility was determined by the Kirby Bauer disc diffusion method as per CLSI criteria. The antibiotics incorporated were: amikacin (30 µg), gentamycin (10 µg), tobramycin (10µg), ceftriaxone (30 µg), ceftazidime (30 µg), cefepime (30 µg), ampicillin-sulbactum (10/10µg), piperacillin/tazobactum (100/10 µg), ciprofloxacin (5 µg) levofloxacin (5 µg), imipenem (10 µg) and trimethoprim-sulfamethoxazole (1.25/23.75 µg), meropenem (10), polymixin B, colistin (E test). Zone interpretation of different drugs was done as per CLSI guidelines (2012).

RESULTS

A total of 950 Acinetobacter spp. were isolated in this study from various clinical samples which is shown below in Chart 1.

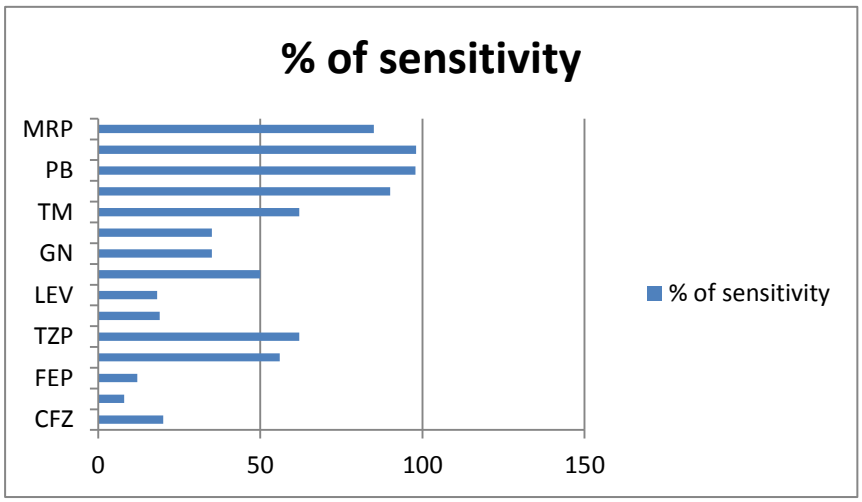
Chart 1.Sample wise distribution of Acinetobacter spp.



Significant numbers of isolates (48%) were obtained from patients admitted in the intensive care units (ICUs).This was followed by patients admitted in surgical (20%), medical (22%) and

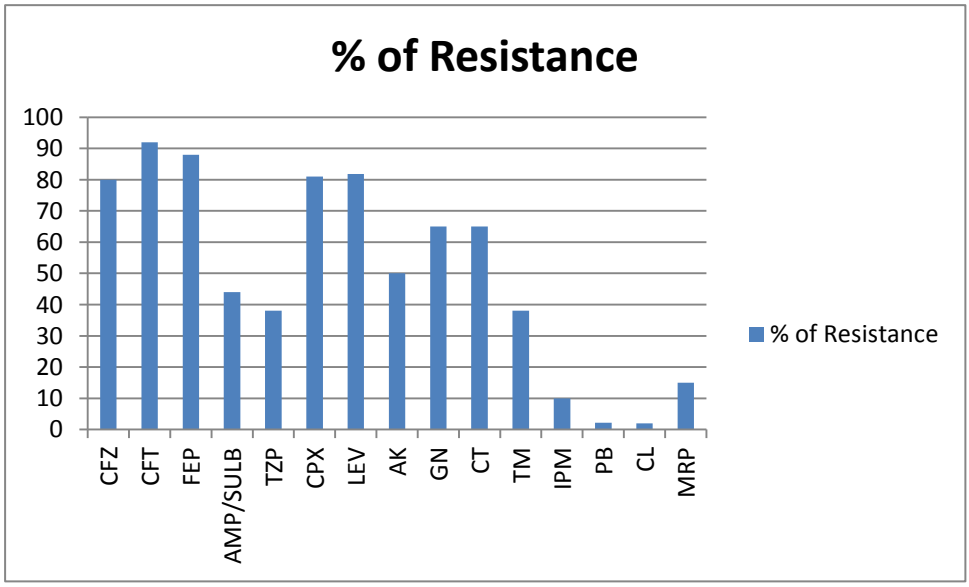
burns ward (10%). Based on the results of the biochemical and carbon assimilation tests, 82% were Acinetobacter. calcoaceticus baumannii complex remained 18% were other Acinetobacter spp. The antimicrobial susceptibility testing revealed that Acinetobacter species were sensitive to following antibiotics tested shown in Chart 2 and the resistant pattern of Acinetobacter spp. to different antibiotics shown in Chart 3.

Chart 2. Percentage of sensitivity to Acinetobacter spp.



[CFZ-Ceftazidime, CFT-Cetriaxone, FEP-Cefepime, AMP/SULB- mpicillin/Sulbactum,TZP-Piptaz,CPX-Ciprofloxacin,LEV-Levofloxacin,AK-Amikacin,GN-Gentamycin,CT-trimethoprim-sulfamethoxazole,TM-Tobramycin,IPM-Imipenem,MRP-Meropenem,PB-Polymyxin b,CL-Colistin]

Chart 3. Percentage of resistant to Acinetobacter spp.



DISCUSSION

Acinetobacter is currently considered one of the most important nosocomial pathogen.[4] Several studies have shown that some A. baumannii strains can adhere to human cells and form biofilm on abiotic surfaces. A. baumannii survives on fingertips and inanimate objects such as glass, plastic, and other environmental surfaces, even

after exposure to dry conditions and nutrient starvation during extended periods of time.[5,6] These all factors contributes to its survival and development of drug resistance. The mechanisms involved in resistance by A. baumannii typically include: (i) enzymatic mechanisms or production of hydrolyzing enzymes like β-lactamases, ESBL, Amp C, Carbapenemase, Metallo beta lactamase and (ii) non-enzymatic mechanisms that involve modification of membrane permeability by either the loss of or decrease in the expression of OMPs or an increased expression of efflux pumps as well as sequence variation of PBPs, expression of modifying enzymes specially in case of aminoglycosides or by point mutations in amino acid substitutions in case of quinolones. [4,7,8,9] The emergence of bacteria resistant to most of the antibiotics available has lead to the appearance of different terms concerning resistance. [4] MDR may be referred when the strain is non-susceptible to ≥1 antimicrobial agent in ≥3 antimicrobial categories; an increase in resistance now refers to extensive drug resistance (XDR) when non-susceptible to ≥1 antimicrobial agents in all but ≤2 categories (i.e., bacterial isolates remain susceptible to only one or two categories), and pan-drug resistance (PDR) is considered when the microorganism is non-susceptible to all the antimicrobial agents in all antimicrobial categories.[9] Present study revels 950 clinical isolates of Acinetobacter from various clinical samples.37% of Acinetobacter spp. was isolated from swabs followed by 31% from blood stream infections. These isolation rate reflects the more prevalence of Acinetobacter in ICUs and surgical units. 48% of total Acinetobacter isolates were obtained from patients admitted in the intensive care units (ICUs).This was followed by patients admitted in surgical (20%), medical (22%) and burns ward (10%).As shown in Chart 2,Acinetobacter shows varied sensitivity towards different antibiotics groups. Acinetobacter shows 98% sensitive to Polymyxins followed by 87% to carbapenems, 58% to b lactams /Blactam inhibitors, 65% to aminoglycosides, 18% to Floroquinolones and 20% to b lactams. Low sensitivity pattern to different groups of antibiotics can be comparable with other study done by Dibyendu et al, Rahbar et al, Mindolli et al. Resistance patterns among nosocomial bacterial pathogens may vary widely from country to country at any given point and within the same country over time. Acinetobacter isolates were resistant to important groups of antibiotics tested, including amikacin (50%), gentamycin (65%), tobramycin (38%), ceftriaxone (92%), ceftazidime (80%), cefepime (88%), ampicillin-sulbactum (44%), piperacillin-tazobactum (38%), ciprofloxacin (81%) levofloxacin(82%), imipenem (10%) and trimethoprim-sulfamethoxazole (65%), meropenem (15%), polymixin B (2.2%), colistin (2%). Carbapenems have a broad spectrum activity and they are stable to hydrolysis by most of the β-lactamases, including the extended spectrum β lactamases (ESBLs) and the Amp C, thats why they are used as the last resort for treating MDR Gram negative infections in any noscomial setting. For Metallo betalactamase producing strains (MBLs), limited treatment options are available and combination therapy or polymyxins are often employed in treatment of multidrug-resistant Acinetobacter spp. Our study shows that the Sulbactam was superior to tazobactam which is correlated with study done by Paul GH et al, so it represents an alternative treatment option for infections due to multiresistant Acinetobacter spp.[9,11,12]

CONCLUSION

There has been an alarming increase in the reports on exremely resistant Acinetobacter spp. over the last decade. Rational use of antimicrobials is an important aspect to delay the emergence of XDR and PDR *Acinetobacter* spp. This can be achieved using an effective antimicrobial stewardship program by placing antibiotic policy, educational training regarding the stewardship program, and monitoring of the program. Inadequate hand hygiene is significant factor in the transmission of pathogens in the hospital. Cross-transmission of MDR *A. baumannii* occurs via direct contact from hands and gloves from healthcare professionals to patients. Early detection and infection control practices are the best defense against these organisms; therefore systematic surveillance to detect drug resistance should be carried out promptly to minimize such spread of drug resistance. Limitations: We couldn't see the clinical outcome & characterization by molecular methods that are gold standard for detection of drug resistance genes.

Future Perspectives

- Routine surveillance of MDR Acinetobacter spp. isolates and their typing and characterization by reference centers.
- Awareness & Training of clinicians for use of combination therapy for MDR Acinetobacter spp. and analysis of their clinical outcome.

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