ORIGINAL ARTICLE

A Decade of an Underestimated Nosocomal Pathogen- Acinetobacter In a Tertiary Care Hospital in Punjab

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Abstract

The present study was designed to know the prevalence of Acinetobacter in various clinical samples, their characterization, antibiotic susceptibility pattern and relevance in various clinical samples in Christian Medical College and Hospital, Ludhiana. Out of 3024 samples including pus, body fluids, blood, urine, drain tips, catheter tips and other appropriate samples, Acinetobacter was isolated in 255 (8.4%) samples. 66.6% isolates were sensitive to amikacin and 95% to cefaperazone/ sulbactam combination. This drug resistant nosocomial infection can be minimized to some extent by judicial use of antibiotics and adopting proper infection control measures.

Key Words

Acinetobacter, Nosocomal, Infection, Antibiotic

Introduction

Acinetobacter are opportunistic pathogens that readily colonize patients with compromised host defences. Acinetobacter calcoaceticus, the species usually involved in human infection, causes disease chiefly in a hospital setting usually associated with respiratory therapy equipment and indwelling catheters. Sepsis, pneumonia and urinary tract infections are the most frequent manifestations (1).

Excluding enterobacteriaceae, Acinetobacter species and Stenotrophomonas maltophilia are the second and third most common gram negative bacilli respectively encountered in clinical specimens (2). This genus contains strictly aerobic, short, often capsulate, nonmotile, Gram negative (or gram variable) bacilli or coccobacilli (often diplo coccobacilli) that grows well on simple media. These organisms occur frequently as components of the commensal flora of man and animals and are therefore regular contaminants of the hospital enviornment (3).

The number of nosocomial infections caused by Acinetobacter species has increased in recent years. These gram negative bacilli are ubiquitous in nature and are highly resistant to various anti microbial agents (4,5). The present study is aimed to know the prevalence of Acinetobacter in various clinical samples, their characterization, antibiotic susceptibility pattern and to know their relevance in various clinical samples.

Material and Methods

The present study was conducted on 3024 clinical samples received from patients admitted in various

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departments of Christian Medical College and Hospital, Ludhiana. Samples such as pus, body fluids, blood, urine, drain tips, catheter tips and other appropriate samples were collected.

The samples were inoculated on Blood agar and MacConkey's agar medium. Catalase positive, oxidase negative colonies were inoculated on triple sugar, Iron medium to see the fermentation/non fermentation of sugar and in Hugh and Leifson medium to see if the organisms is oxidizer or non oxidizer (2). Antibiotic sensitivity test were performed using Cephalexin (30 micro g), Cefotaxime (30 micro g), Amikacin(30 micro g), Ceftazidime(30 microg), Peflox (5micro g), Meropenem(10 micro g), Piperacillin(30 micro g) and Cefaperazone / Sulbactam (30/75micro g) discs.

Restults

Out of 3024 samples processed, Acinetobacter were isolated in 255 (8.4%) samples. Maximum number (36.8%) of samples were from pus (1112/3024) followed by urine 32.5% (983/3024) and blood 36.7% (1109/3024).Isolation of Acinetobacter was maximum 86.2% (220/255) from pus, followed by urine 8.23% (21/255) and blood 5.4% (14/255).Various Predisposing factors in these patients are shown in *table 1*. Among all the Isolates, 67% of isolates were Acinetobacter saccharolytic nonhaemolytic, 5% were Acinetobacter asaccharolytic non haemolytic, 28% were Acinetobacter asaccharolytic non haemolytic and none was Acinetobacter asacchrolytic haemolytic.

Sixty six point six % isolates were sensitive to Amikacin and 95% to Cefaperazone /Sulbactam combination as shown in *table 2*. Most of the isolates (90%) were resistant to Cephalaxin and Ceftazidime. Antibiotic susceptibility patterns of various isolates are shown in *table 2*.

Discussion

Acinetobacter organisms have gained nosocomial importance, cause a broad range of clinical syndrome especially in patients with factors that impair normal host resistance. Out of 3024 samples from indoor patients, Acinetobacter were isolated in 255 (8.4%) samples in present study while in the study of Roussel *et al* (6) and

Table-1:Various Predisposing Factors and
Acinetobacter Isolation

S.No.	Predisposing Factors	% Isolation
1	Antibiotics intake >72 hrs	80
2	I/V lines >48 hours	76
3	Post operative	40
4	Burns	30
5	Urinary catheterization	25
6	Malignancy	10
7	Renal transplant	5
8	Chest tube	1-2

Table-2: Antibiotic Susceptibility Patterns

S.No	Antibiotic	No. of Sensitive Isolates n(%)
1	Cephalaxin	26(10)
2	Cefotaxime	38(15)
3	Amikacin	165(64.7)
4	Ceftazidime	26(10)
5	Meropenem	210(82.3)
6	Peflox	153(60)
7	Piperacillin	153(60)
8	Cefaperazone /Sulbactam	240(94)

Sakata *et al* (7), incidence of Acinetobacter was 15.2% and 19% respectively.

Maximum number of Acinetobacter isolates were from pus 86.2% (220/255) followed by urine 8.23% (21/255) and blood 5.4% (14/255) respectively. In the study of Mishra *et al* (8) 1986, also maximum isolates were from pus, followed by blood 35/75 and 16/75 while in the study of Pedersen et al9 1970, maximum isolates were from sputum 19/72, and from urine 16/72. The rate of infection varies according to the patient, duration of stay in hospital and the type of infection.

In the present study, Acinetobacter saccharolytic non haemolytic (previously named as A.baumanii, A.anitratus) were dominant (67%) while Acinetobacter asacchrolytic non haemolytic (A.lwofii) were 28% and Acinetobacter asaccharolytic haemolytic were nil. Pedersen et al in 1970 (9) isolated Acinetobacter antitratus in 72,



Acinetobacter lwofii in 42 cases. Gulati *et al* (10) in 1999 reported that Acinetobacter baumanii were associated with disease more frequently while Acinetobacter lwoffi as environmental contaminant Smego *et al* (11) in 1985 found that 16/25 isolates of A.anitratus to be hospital acquired and disease associated and Acinetobacter lwofii in only two cases of bacteremia that was also community acquired.

In the present study, all 255 isolates were from patients having some predisposed conditions like antibiotics intake >72 hours, I/V lines 48 hours, post operative, burns, urinary catheterization, malignancy, renal transplant, chest tube etc. This finding is supported by other workers also (12-14). In the present study 82% of isolates were sensitive to Meropenem, 64.7% to Amikacin and 94.1% to Cefaperazone/ Sulbactam while Cephalexin showed least susceptibility. This finding is nearly comparable with Smego 1985 (12) who found Amikacin to be 100% sensitive. However despite such resistance, combination therapy using a third generation Cephalosporin and Amikacin could be the best choice for treating Acinetobacter infections in our set up (15). **Conclusion**

It can be concluded from the study that Acinetobacter occurs as colonizer and contaminant in clinical samples of hospitalized patients. The increasing trends towards antibiotic resistance reflect the extensive usage of antibiotics in hospitals which in turn exerts selective pressure on Acinetobacter in hospital environment. The infections caused by these organisms are becoming difficult to treat day by day. So, this drug resistant nosocomial infection can be minimized to some extent by judicial use of antibiotics and adopting other methods of infection control.

References

- 1. Jawetz, Melnick & Adelberg's Medical Microbiology, 20th Edition 1995.pp. 221.
- Forbes BA, Sahm DF, Weissfeld AS. Acinetobacter chryseomonas, Flavimonas and Stenotrophomonas. In Bailey and Scott's (ed.) Diagnostic Microbiology, Mosby St. Louis. (Edt). 14th, 1998.pp. 502-508

- Collee JG, Fraser AG, Marmion BP, Simmons A, Mackie ,McCartney. Practical Medical Microbiology, 14th ed. 1996.pp. 294.
- 4. Bergogne-Berezin E ,Joly-Guillou ML. An under estimated nosocomical pathogen, Acinetobacter calcoaceticus. *J Antimicrob Chemo* 1985; 16: 535-38.
- Seifert H, Baginski R, Schulze A, Pulverer G. Antimicrobiol susceptibility of Acinetobacter species. *Antimicrobial Agents Chemother* 1993; 37(4): 750-53.
- Roussel Delvallez M, Wallet F, Delpierre F, Ciurcol RJ. In vitro bactericidal effect of a betalactam and aminoglycoside combination against multiresistant pseudomonas aeruginosa and Acinetobacter baumannii. J Chemother 1996; 8 (5): 365-68.
- 7. Sakata H, Fujita K, Maruyama S, Kakehashi H, Mori Y, Yoshioka H. Acinetobacter calcoaceticus biovar antiratus septicemia in a neonatal intensive care unit, epidemiology and control. *J Hosp Infect* 1998;14: 15-22.
- 8. Mishra B, Bhujwala RA, Shriniwas 1986. Nonfermenters in human infections. *Ind J Med Res* 83: 561-566.
- 9. Pedersen MM, Marso E, Picket MJ. Non fermentative bacilli associated with man III pathogenecity and antibiotic susceptibility. *Am J Clin Pathol* 1970; 54: 178.
- Gulati S, Kapil A, Goel V, Das B, Dwivedi SN, Mahapatra AK. Biotyping of Acinetobacter species isolated from clinical samples. *Ind J Med Res* 1999; 110: 160-63.
- Smego RA. Endemic nosocomial Acinetobacter calcoaceticus bacteremia clinical significance, treatment and prognosis. Archives Internal Med 1985; 145: 2174-79
- French GL, Casewell MW, Ronocoroni AJ, Knight AJ, Phillips I. A hospital outbreak of antibiotic resistant Acinetobacter anitratus epidemiology and control. *J Hosp Infect* 1980; 1: 125-31.
- 13. Hoffmann S, Mabeck CE, Vejlsgarod R. Bacteriuria caused by Acinetobacter calcoaceticus biovars in a normal population and in general practice. *J Clinical Microbiol* 1982; 16: 443-41.
- Holton J. A report of a further hospital outbreak caused by a multiresistant Acinetobacter anitratus. *J Hosp Infec* 1982 3: 305-09.
- Prashanth K, Badrinath S. In vitro susceptibility pattern of Acinetobacter species to commonly used Cephalosporins, Quinolones and Aminoglycosides. *Ind J Med Microbiol*. 2004,22 (2): 97-103.