Resistance Pattern of Aerobic Gram Negative Isolates from Wound Infections in a Teaching Hospital

Joshy M. Easow^b, Yogitha Jalan^b, Godwin Wilson^b, Sangeeta Dey^b, Simantee Guha^b & Shivananda P.G^a

Abstract

Introduction	A retrospective analysis of reports from blood cultures was done to determine the spectrum of bacterial isolates and their antibiograms. Altogether, 1050 blood cultures were performed in the clinical microbiology laboratory of the Teaching Hospital of Manipal Medical College of Medical Sciences during the period January 1, 2002 to December 3, 12.2002.
Objectives	To study the pattern of Gram Negative bacterial isolates in wound infection and their antibiogram so that recommendations can be made for empirical antibiotic treatment.
Methods	This study was carried out at Department of Microbiology, Manipal Teaching Hospital, Pokhara, Nepal. A total of 416 specimens were received from various cases of wound infection for a period of 1 year from January 2002 to December 2002. They were processed by standard microbiological methods and antimicrobial testing was performed by the Kirby-Bauers disc-diffusion method as per NCCLS recommendations.
Results	Of 416 samples cultured, 95 (22.84%) grew Gram negative organisms. The most frequent offender was <i>Escherichia Coli</i> (25.26%) followed by <i>Klebsiella spp.</i> (24.21%), <i>Proteus</i> (17.89%), <i>Pseudomonas</i> (15.79%) and other less frequent isolates. Resistance was frequently seen in commonly prescribed antibiotics.
Conclusion	The emergence of multiple resistance among pathogens is increasingly recognized. The varying microbiological pattern of wound infection warrants the need for ongoing review of causative agents and their antibiotic sensitivity patterns. Judicious use of antimicrobials and education among the medical community and lay public should be undertaken to increase the awareness of emergence of resistant pathogens.
Keywords	Wound infection, Gram negative bacteria, Drug resistance

Introduction

Wounds have been with humankind from the prehistoric beginning. Wound infection has been defined as the emergence of pus from a wound irrespective of results from a subsequent culture. Wound infections occur as complications of surgery, trauma or disease that may interrupt a mucosal or skin surface. The nature of the infecting organism depends on the underlying condition and location of the infective process. Gram negative

bacterial plays an important role in wound infection², most of them being members of the family Enterobacteriaceae. The treatment and healing of wounds is an art as old as humanity³. Despite many advances the deleterious effects of the seemingly indiscriminate and widespread community distribution of broad-spectrum antibiotics has resulted in increasing number of resistant pathogens⁴. Irrespective of etiology, the

^a Correspondence Author: Dr. P.G. Shivananda, Professor and Head, Department of Microbiology, Manipal College of Medical Sciences, PO Box NO. 155, Deep Heights, Pokhara, Nepal

^b Department of Microbiology, Manipal College of Medical Sciences, PO Box NO. 155, Deep Heights, Pokhara, Nepal

choice of antibiotic should be based on culture and sensitivity reports. Empirical therapy can be started based on the Antimicrobial surveillance report of that region.

The present study was done to analyze the spectrum of gram negative organisms in wound infection and their resistance pattern.

Materials and methods

The study was conducted at Department of Microbiology, Manipal Teaching Hospital, Pokhara, Nepal. A total of 416 specimens were received from cases of wound infection during the period January 2002 to December 2002. From each case two swabs were taken; one for smear studies and the other for culture. The medias' used were Blood Agar, Chocolate Agar, MacConkey's Agar and Mueller Hinton Agar. Any growth was identified by colony characteristics and standard biochemical tests⁵. Antimicrobial testing was performed by the Kirby-Bauers disc-diffusion method as per NCCLS recommendations.

Results

Of 416 samples cultured, 95 (22.84%) grew Gram negative organisms. The most frequent offender was Escherichia coli (25.26%) followed by Klebsiella species. (24.21%), Proteus (17.89%), Pseudomonas (15.79%) and other less frequent isolates as shown in Table I.

Table I Gram negative isolates						
Organism 7	otal isolates	%				
Escherichia coli	24	25.26				
Klebsiella species	23	24.21				
Proteus species	17	17.89				
Pseudomonas spec	ies 15	15.79				
Enterobacter speci	es 8	8.42				
Acinetobacter spec	ies 3	3.16				
Morganella morga	nii 3	3.16				
Citrobacter species	s 1	1.05				
Chryseobacterium		1.05				
Total	95	100				

The drug resistances of these organisms are as shows in Table II.

Table II: Resistance pattern of Gram negative isolates

Organisms	G	Ak	Cf	Ci	Ca	Cb	Pc
Escherichia coli	10	3	8	5			
N=24	(41.66)	(12.5)	(33.33)	(20.8)	-	_	
Klebsiella species	8	3	7	7		-	-
N=23	(34.7)	(13.0)	(30.4)	(30.4)	-	-	-
Proteus species	3	1	3	2	-	-	-
N=17	(17.6)	(5.89)	(17.6)	(11.76)			
Pseudomonas	5	1	4	2	2	2	2
Species	(33.33)	(6.67)	(26.67)	(13.3)	(13.3)	(13.3)	(20.0)
N=15							

^{*} Figures shown in table are numbers of resistant isolates.

Figures shown in parentheses indicate percentage of resistant isolates

G-gentamicin, ak-amikacin, Cf-ciprofloxacine, Ci-ceftriaxone, Ca-ceftazidime

Cb -carbenicillin, Pc-pipercillin

Discussion

The varying microbiological pattern of wound infection warrants the need for ongoing review of causative agents and their antibiotic sensitivity patterns.

Fluoroquinolones are widely used because of their broad-spectrum antimicrobial activity, bioavailability, tolerability and modest cost.

Availability in both parental and oral formulations allows their use in therapeutic as well as prophylactic settings. The resistance is due to mutation in gyr A gene, leading to an alteration at the quinolones target site DNA grase⁷ although resistance may also be mediated by barriers in diffusion through the cell wall⁸. A single operon facilitates resistance of Pseudomonas to quinolones, beta-lactams, tetracyclines via drug efflux⁹. The primary mechanism for resistance enzymes10. aminoglycosides is modifying Streptomycin and kanamycin have fallen out of clinical use due to these enzymes, however amikacin is least vulnerable to these inactivating enzymes but may be rendered ineffective by alterations in the ribosomal target11. In our study, 22.84% grew Gram negative organisms. Of these 27.36% were resistant to Gentamicin and 8.42% to Amikacine. The overall chance of treatment failure

increased if Gentamicin was preferred than Amikacin. Ceftriaxone is a good alternative to amikacin, however it should be used judiciously.

Overall resistance by these organisms towards Ciprofloxacin was 23.15% with widespread use we may witness gradual emergence of resistance with consequent loss of use of this valuable group of compounds. In our institution, Amikacin followed by ceftazidime and Carbenicillin is the drug of choice for treating infections caused by *Pseudomonas species*. 26.67% of these strains were resistant to *Ciprofloxacin* and 33.3% were resistant to gentamycin.

The emergence of multiple resistance microbe pathogens is increasingly recognized. Antibiotics should ideally be prescribed according to the local susceptibility patterns. Thus establishing a local antimicrobial surveillance center needs to be addressed. Judicious use of antimicrobials and education among the medical community and lay public should be undertaken to increase the awareness of emergence of resistant pathogens.

References

- Pollock AV: Surgical wound sepsis. Lancet, 1979(1);1283-1286
- Njoku-Obi AN, Ojiegbe GC. Resistance patterns of bacterial isolates form wound infections in a university teaching hospital. West Afr J. Med 1989 Jan-Mar, 8(1); 29-34
- Martin CR, David LS, Michael GF: Wound healing Biologic features and approaches to maximize healing trajectories. Curr Probl Surg 2001(38); 74-77.

- Mangram AJ, Horan TC, Pearson MC, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Centre for Disease Control and Prevention (CDC), Hospital Infection Control Practices Advisory Committee. Am J Infect Control 1999(27); 97-132
- Baron EJ, Finegold SM. Overview of conventional methods for bacterial identification. Chapter 13, In: Bailey and Scotts Diagnostic Microbiology (Mosby publishers, St Louis) 1994;167
- Performance standards for antimicrobial susceptibility testing. Eight Information supplement 2000. National committee for Clinical Laboratory Standards (NCCLS) M2 A7 (20) 1 & 2; Villanova, Pa.
- Woolfson JS, Hooper DC; Bacterial resistance to quinolones. Rev Infect Dis 1989(11); S960-S968
- Nikaido H: Outer membrane barrier as a mechanism of antimicrobial resistance. Antimicrob Agents Chemother 1989(33); 1831-1836
- 9. Poole K: Bacterial multi drug resistance emphasis on efflux mechanisms and Pseudomonas aeruginosa. J. Antimicrob Chemo 1994(34); 453-456
- 10. Mayer KH: Review of epidemic aminoglycoside resistance worldwide. Am J Med 1985(80); (suppl 6 B): 56-64
- Ahmad MH, Rechenmacher A, Boch A: Interaction between aminoglycoside uptake and ribosomal resistance mechanisms. Antimicrob Agents Chemother 1980(18); 798-806