

THE ANTIBIOGRAM OF *STAPHYLOCOCCUS AUREUS* ISOLATED FROM BLOOD, AND WOUND INFECTIONS OF PATIENTS AT JOS UNIVERSITY TEACHING HOSPITAL

Y. T. KANDAKAI - OLUKEMI Ph.D*
Lecturer

C. S. S. BELLO MD*
Professor

J. D. MAWAK MSc*
Assistant Lecturer

M. A. OLUKEMI Ph.D*
Lecturer

*Department of Medical Microbiology, Faculty of Medical Sciences

** Department of Pharmaceutics and Pharmaceutical Technology,
Faculty of Pharmaceutical Sciences,
University of Jos, P. M. B. 2088, Jos,
Nigeria.

Correspondence to:

PROFESSOR C. S. S. BELLO

ABSTRACT

A total of fifty *Staphylococcus aureus* isolates consisting of twenty-five each from blood and wound specimens collected from the Jos University Teaching Hospital were used in this study. In Vitro antibiotic sensitivity testing was carried out on the isolates. Of the fifty *Staphylococcus aureus* isolates tested 26 (52%) were sensitive to erythromycin, 20 (40%) cloxacillin, 20 (40%) tetracycline, 17 (34%) gentamycin, 13 (26%) streptomycin and 12 (24%) chloramphenicol. All isolates were 100% resistant to penicillin G. Isolates from blood specimens were found to be more resistant to all of the antibiotics tested than those from wound. This difference was statistically significant ($p < 0.05$). It is recommended that periodic invitro antibiotic susceptibility testing of clinical isolates be carried out to know the current predominant strains of bacteria within a given area and their antibiograms as well.

Key words: Antibiogram, Staphylococcus aureus, Blood, Wound Infections.

INTRODUCTION

Staphylococcus aureus are among the most resistant of the pathogenic bacteria and are responsible for many nosocomial infections. They produce a large number of toxins and enzymes that act locally, primarily to help them withstand phagocytosis by neutrophils. For this reason, they cause more frequent and more varied diseases than any other human pathogen. They have been isolated frequently from wound^{2, 3} and blood⁴ infections. They have also been found to be the most prevalently isolated pathogen from neonatal septicemia in Nigeria⁵⁻⁷.

It has been observed that bacterial isolates and their antibiotic susceptibility patterns change from time to time⁷⁻⁸ hence, periodic study of prevailing pathogens and their antibiograms are necessary for effective therapy. Thus, the present study was undertaken to ascertain the antibiotic sensitivity pattern of *Staphylococcus aureus* strains isolated from blood and wound infections of patients attending the Jos University Teaching Hospital (JUTH).

MATERIALS AND METHODS

A total of fifty *Staphylococcus aureus* isolates were obtained from Jos University Teaching Hospital Microbiology Laboratory, Twenty-five each from blood and wound specimens. The isolates were identified using standard biochemical tests.⁹ Antibiotic sensitivity testing was carried out using the Bauer-Kirby disc diffusion method.¹¹ Antibiotics tested included cloxacillin (5Mcg) erythromycin (15 Mcg), penicillin G (1.2 iu), tetracycline (10 Mcg), gentamycin (10 Mcg), streptomycin (10 Mcg) and chloramphenicol (10 Mcg).

Staphylococcus aureus test isolates/organisms and control (NCTC 6571) were grown overnight at 37°C in nutrient broth. The overnight cultures were then diluted with sterile saline (0.85% NaCl) to yield approximately 10⁵ colony-forming Units (cfu) per ml. Suspensions used as inocula were compared with a standardised barium sulfate suspension according to the method of Vandepitte et al¹². Muller - Hinton agar plates were inoculated with test isolates and control strain according to the method of Scott¹³. The prepared discs were applied onto inoculated plates and incubated overnight at 37°C. The diameter of the zone of inhibition for each test antibiotic was measured and sensitivity or resistance estimated by comparing with zone-diameter interpretive standard¹⁴. The chi-square was used to compare the relatedness or otherwise of sensitivity pattern of isolates from wounds and those from blood specimens.

RESULTS

The susceptibility pattern of *Staphylococcus aureus* strains isolated from wound specimens was as follows: cloxacillin 18 (72%), erythromycin 16 (64), tetracycline 12 (48%), gentamycin 10 (40%), chloramphenicol 8(32%) and streptomycin 8 (32%). All isolates from wound specimens were resistant to penicillin G (Table 1).

Table 1:

In vitro antibiotic sensitivity pattern of *Staphylococcus aureus* isolated from wound and blood specimens.

Antibiotic tested	Number <i>Staphylococcus aureus</i> Sensitive (%)	
	Wound	Blood
Cloxacillin	18 (72)	2 (8.)
Erythromycin	16 (64)	10 (40)
Tetracycline	12 (48)	8 (32)
Gentamycin	10 (40)	7 (28)
Streptomycin	8 (32)	5 (20)
Chloramphenicol	8 (32)	4 (16)
Penicillin G	0 (0)	0 (0)

Table 2:

Total *Staphylococcus aureus* isolated from sensitive to the various antibiotics.

Antibiotic tested	Number isolates Sensitive (%)
Erythromycin	26 (52)
Cloxacillin	20 (40)
Tetracycline	20 (40)
Gentamycin	17 (34)
Streptomycin	13 (26)
Chloramphenicol	12 (24)
Penicillin G	0 (0)

Most of the isolates from blood specimens were found to be resistant to many of the antibiotics tested. The susceptibility pattern was as follows; erythromycin 10 (40%), tetracycline 8(32%), gentamycin 7 (28%), streptomycin 5 (20%), chloramphenicol 4 (16%) and cloxacillin 2(8%). All isolates from blood were also resistant to penicillin G.

The combined susceptibility pattern of all *Staphylococcus aureus* isolates to the various antibiotics tested were as follows; erythromycin 26 (52%), cloxacillin 20 (40%), tetracycline 20(40%), Gentamycin 17 (34%) streptomycin 13 (26%) and chloramphenicol 12 (24%), (Table 2).

Isolates from blood specimens were more resistant to all of the antibiotics tested than those from wound

specimens. Statistical analysis showed that the difference was significant. ($X^2 = 0.05$, $df = 6$, $p < 0.05$).

DISCUSSION

Bacterial infection are still an important cause of morbidity and mortality in Nigeria. Drug abuse has been implicated as a cause of resistance to antibiotics such as ampicillin, cloxacillin, chloramphenicol and tetracycline.³ This may be a probable reason for resistance to these antibiotics by *staphylococcus aureus* encountered in this study. However, since, relevant data pertaining to patients from which specimens were collected are lacking definitive conclusion linking resistance to indiscriminate use of antibiotics can only be inferred. The rate of resistance of *Staphylococcus aureus* to the beta-lactamase stable penicillin, cloxacillin in this study (40% sensitivity is higher than the findings of Egri-Okwaji et al⁷ and Egah¹⁵ where 83.33% and 70.1% respectively of their *Staphylococcus aureus* strains were sensitive to this antibiotic. A probable reason which may account for the differences in results is that since the *staphylococcus aureus* strains from the various studies are different their susceptibility pattern could also vary. Resistance to beta lactamase stable penicillin is due to the intrinsic insensitivity of the bacterium to the antibiotic or to the instability of the antibiotic to *Staphylococcal* beta lactamase produced¹⁶. Also, in some hospitals in the United States the percentage of *Staphylococcus aureus* strains resistant to beta-lactamase Stable penicillins now exceed 50% and it was observed that increase in incidence of resistance to this group of antibiotics may occur abruptly⁴. In Nigeria it has been found that the frequency of resistance to penicillin varies so much from hospital to hospital that country wide comparison is not helpful.³

The finding that 24% of *Staphylococcus aureus* isolates were sensitive to chloramphenicol agrees with the findings of Egri-Okwaji et al⁷. They observed that only 29.2% of their *Staphylococcus aureus* strains were sensitive to chloramphenicol. Bacterial resistance to chloramphenicol is higher in Nigeria than Europe and it has been suggested that this reflects the greater use of the antimicrobial agent in the treatment of enteric fevers in this country³. Which illustrates that antibiotics will affect microbial population other than the ones they are specifically directed at.

A high degree of resistance to erythromycin was encountered in this study, which is contrary to other findings.^{7,15} Resistance of gram-positive bacteria to the macrolide group of antibiotics like erythromycin probably occurs as a result of induction or constitutive production of an enzyme which methylate the ribosome binding site of the antibiotic molecule.¹⁷⁻¹⁹ The rates of resistance to erythromycin have been found to decrease with less usage

of the antibiotic.²⁰ Therefore it may be inferred that variation in usage of the antibiotic by patients is probably a contributing factor for differences in the susceptibility pattern of staphylococcal isolates observed by various workers.

When comparing *Staphylococcus aureus* isolates from wound and blood specimens it was observed that isolates from blood displayed a greater degree of resistance to all of the antibiotics tested than those from wounds. This difference is particularly striking with Cloxacillin ($p < 0.05$). Eighteen (72%) of isolates from wounds were sensitive to this penicillin while only 2 (8%) of those from blood were sensitive.

The blood is a relatively inhabitable part of the body from the microorganisms viewpoint since it contains potent antimicrobial systems including leukocytes, immunoglobulins and complement. These systems act in clearing microbes more rapidly from the blood than from other organs. However, use of high doses and long course of multiple potent antibiotics may promote selection of resistant organisms which is one means by which host defenses may be weakened or interfered with and highly resistant organisms are then able to establish themselves in the blood. There is therefore a greater, likelihood of progression to bacteraemia in patients infected with highly resistant organisms. Whatever the cause, infections involving the blood tend to be serious and often life threatening. The problem of multiple drug resistant *Staphylococcus aureus* in the clinical setting presents cause for concern especially in developing countries where bacterial infections are still an important cause of illness and death. It is therefore recommended that careful surveillance for the emergence of resistant bacterial strains be undertaken to prevent further spread of these organisms. This would entail periodic in vitro antibiotic susceptibility testing of clinical isolates to know the current predominant strains of bacteria within a given area and their antibiograms.

REFERENCES:

1. World Health Organization. Antimicrobial resistance: Report on a working group. Bull WHO 1983; 61: 61: 383 - 394.
2. Otokunfer T. V, Datubo-Brown D D. Bacteriology of wound infections in the surgical wards of a Teaching Hospital. W. Afr. J. Med. 1990; 9: 285 - 290.
3. Montefiore D; Rotimi V O and Adeyemi-Doro F A B. The problem of bacterial resistance to antibiotics among strains isolated from hospital patients in Lagos and Ibadan, Nigeria. J. Antimicrob Chemother 1989; 23: 64 - 651.
4. Fang F C, McClelland M, Guiney D G, et al. Value of Molecular epidemiologic analysis in a nosocomial methicillin-resistant *Staphylococcus aureus* outbreak. JAMA 1993; 270: 1323 - 1328.
5. Ahmed I, Fadahunsi, H O and Ogunbi O. Bacterial Spectrum in sepsis at the neonatal unit Logas University Teaching Hospital. Niger Med. J. 1974; 4: 22 - 24.
6. Amiebenamo C S, Yakubu A M, Bello C S S, Ewa B. Neonatal septicaemia in Zaria. Niger Med. J. 1989; 19: 349 - 351.
7. Egri-Okwaji M T C, Iroha, E O, kesah C N, Odugbemi, T. Bacterial pathogens causing neonatal sepsis in an out-born neonatal unit in Lagos, Nigeria. Nig. Qt. J. Hosp. Med. 1996; 6: 149.
8. Njok-Obi A N U, Ojiegbe G C, Resistance patterns of bacterial isolates from wound infections in a University Teaching Hospital. West Afr. J. Med. 1989; 8: 29-24.
9. Duguid J P. *Staphylococcus*: Cluster-forming gram-positive cocci, in Mackie and McCartney practical Medical Microbiology 13th ed., Collee J.G, Duguid J P, Fraser A G, marmion B P (eds), Churchill Livingstone, Newyork, 1989, pp 303-326.
10. Bauer A W, Kirby M M, sherris, J C, Truck M. Antibiotic susceptibility testing by a standard single disc method. Am. J. Clin pathol. 1966 45: 493-496.
11. Kandakai-Olukemi Y T., Bello C S S, olukemi M A. Isolation of cloxacillin, erythromycin and azithromycin resistant *Staphylococcus aureus* strains from Nurses at the Jos Univeristy Teaching Hospital. Niger J. Med. 1996; 5: 19-23.
12. Vandepitte J, Engbaek K, piot p, Iteuk C C, Basic Laboratory procedures in Clinical Bacteriology. WHO 1991, pp. 85.
13. Scott A C. Laboratory Control of antimicrobial therapy in Mackie and Mcartney practical Medical Microbiology 13th ed., College J C, Duguid J P, Fraser A G, Marmion B P (ed) Churchill Livingstone, New york 1989 pp. 161 - 181.
14. Sommers H M. Drug susceptibility testing in Vitro: Monitoring of antimicrobial therapy, in the Biologic and Clinical Basis of Infectious Diseases. Youmans G P, pterson P Y, Sommers H M (ed) W B Saunders Co., Philadelphia, 1980, pp 782 - 804.
15. Egah D Z. Bacterial Floral of Wound Infections and Their Antibiogram in Jos University Teaching Hospital (M.Sc Thesis) Jos: University of Jos. 1996.
16. Basker M J, Edmondson R A, Sutherland R, Comparative Stabilities of penicillin and Cephalospolins of *Staphylococcal* betal-lactamase and activities against *Staphylococcus aureus*. J. Antimicrob chemother 1980; 6: 333-341.
17. Dubnau, D. Translational attenuation: the regulation of bacterial resistance to the macrolide-Lincosamide-streptogfamin B antibiotics. Critical Reviews in Biochemistry 1984; 16: 103 - 132.
18. Weisblum B. Inducible erythromycin resistance in bacteria. Br. Med. Bull. 1984; 40: 47-53.
19. Dunkin Kt, Jones S, Howard AJ. The invitro activity of C P 62 and 993 against *Haemophilus influenzae*, *Banhanella catarrhalis*, *Staphylococci* and *streptococci*. J. Antimicrob Chemother. 1988; 22: 405 - 411.
20. Moellering R C. Introduction: Revolutionary changes in the macrolide and azalide antibiotics. Am J. Med. 1991; 91 (supp/3A): 15 - 35.