EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 4
Issue: 8
116-119
Year: 2017

ANTIBIOTICS SUSCEPTIBILITY PATTERNS OF STAPHYLOCOCCUS AUREUS IN CLINICAL ISOLATES FROM MAITAMA DISTRICT HOSPITAL, ABUJA, NIGERIA

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Article Received on 14/06/2017 **Article Revised on 04/07/2017** **Article Accepted on 25/07/2017**

ABSTRACT
Staphylococcus aureus is a major pathogen causing a variety of infections ranging from mild skin infections to life threatening systemic illness. Antibiotic resistance by Methicillin Resistant Staphylococcus Aureus (MRSA) has grown to be common, and resistant to almost all antibiotics. The objective of the study was to see the antimicrobial susceptibility pattern of S. aureus isolates from different clinical samples against various brands of antibiotics used in Maitama District Hospital, Abuja, Nigeria. A cross-sectional study was conducted from November 2014 to April 2015 among patients attending Maitama District Hospital, Abuja, Nigeria. A total of 118 non-duplicated suspected S. aureus isolates were collected from Maitama District Hospital, Abuja, Nigeria. Using conventional biochemical analysis, 79(66.9%) of the isolates were observed to ferment Mannitol salt agar, of which 60(76.9%) were Coagulase-positive and 19(31.7%) were Coagulase-negative. Further evaluation using Microgene Staph Identification kit 56(93.3%) were confirmed as S. aureus, 3(5%) as S. xylosus and 1(1.7%) as S. hyicus. The resistance patterns of this isolates were as follows: 8(14.3%) Gentamycin (10µg), 21(37.5%) Ciprofloxacin (5µg), 46(82.1%) Cefoxitin (30µg), 11(19.6%) Vancomycin (30µg), 26(46.4%) Erythromycin (15µg), 40(70.4%) Tigercycline (15µg), 37(66.1%) Clindamycin (2µg), 27(48.2%) Trimethoprim/Sulphamedazol (25µg), 27(48.2%) Chloramphenicol (30µg), 21(37.5%) Linezolid (10µg), 30(53.6%) Amoxicillin/Clavulanic acid (20µg /10µg). The rate of antimicrobial resistance to conventional antibiotics is very high, continuous surveillance on antimicrobial susceptibility testing of S. aureus is essential for the detection of emerged resistant strains of S. aureus especially MRSA.

KEYWORDS: S. aureus, Resistance, Antibiotics.

INTRODUCTION
Staphylococcus aureus is a dangerous pathogen, responsible for a multitude of human infections around the world.[1] Many S. aureus infections present as moderately severe infections of the skin or respiratory tract, but S. aureus may also cause more dramatic forms of disease that may be life-threatening, such as necrotizing fasciitis or necrotizing pneumonia. Considerable efforts have been undertaken to decipher the importance that specific molecular determinants have in defining S. aureus virulence and interaction with the host. From a clinical point of view, a major problem that physicians have to face when treating S. aureus infections is antibiotic resistance. Resistance to the first antibiotic, penicillin emerged in the 1940s.[2] In 1942, penicillin resistant S. aureus was detected.[3] Mechanistically, resistance to penicillin is due to an enzyme called penicillinase, which was found even before the introduction of penicillin into clinical use.[4] Penicillinase cleaves the β-lactam ring that is characteristic of β-lactam antibiotics such as penicillin and its derivatives. Already in the 1950s, penicillinase-containing strains of S. aureus were pandemic in hospitals and the community.[5] Nowadays, most infectious S. aureus isolates are resistant to penicillin.

To overcome the problem with penicillin-resistant S. aureus, the semi synthetic antibiotic methicillin was developed, which is derived from penicillin, but resistant to β-lactamase inactivation. Methicillin was introduced by Beecham in 1959; but already about 1 year later, methicillin-resistant S. aureus was detected in the UK.[6]

Unlike in the case of resistance to penicillin, the mechanism underlying methicillin resistance protects the bacteria from the entire class of β-lactam antibiotics including penicillins, cephalosporins and carbapenems. Staphylococcus aureus epidemics occur in waves of antibiotic resistance.[7] The first epidemic penicillin-resistant strains were replaced by the so-called 'archaic' methicillin-resistant S. aureus (MRSA) strains first found in the UK. This epidemic was largely restricted to Europe. Starting in the 1980s, novel lineages of MRSA emerged, leading to a worldwide pandemic of MRSA that is still ongoing. Nowadays, many industrialized countries report that methicillin-resistant strains account for at least 25–50% of infectious S. aureus isolates in hospitals.[8] In contrast, some countries such as The Netherlands and the Scandinavian countries historically have low MRSA infection rates (often < 1%), most likely owing to rigid search-and-destroy and surveillance policies, as well as restraint in antibiotic prescription. In fact, a recent Japanese study indicates that high antibiotic consumption rates lead to increased MRSA burden over time.[9] While for a long time MRSA infections were limited to hospitalized patients, the most recent epidemic MRSA wave, beginning in the mid- to late 1990s, is characterized by the emergence of community associated MRSA (CA-MRSA) with the capacity to infect otherwise healthy individuals.

MATERIALS AND METHODS

Sample collection

Total of 118 consecutive, non-duplicate suspected S. aureus isolates were collected from Maitama General Hospital, Abuja, between November 2014 to April 2015 after the ethical clearance has been obtained from the FCTA Hospital Management Board. Epidemiological information collected with bacterial isolates includes, age, sex of the patient, type and source of clinical specimens. The sources of clinical specimen were classified into inpatient, of the patients on admission on the wards and outpatient, of those patients seen on outpatient basis.

Biochemical Characterization

Identification of isolates was carried out using standard microbiological procedures.[10] S. aureus was identified on the basis of colony and microscopic morphology, Latex agglutination (coagulase), catalase and Microgen staph identification kit.

Antibiotics Identification Tests

Antibiotic susceptibility testing of the S. aureus was determined by Kirby-Bauer disc diffusion method according to EUCAST, 2015 guidelines, using the following antibiotics, Gentamycin(CN) (10µg), Ciprofloxacin(CIP) (5µg), Cefoxitin(FOX) (30µg), Vancomycin(VAN) (30µg), Erythromycin(E) (15µg), Tigercycline(TGC) (15µg), Clindamycin(DA) (2µg), Trimethoprim/ Sulphamedazol (SXT) (25µg), Chloramphenicol(C) (30µg), Linezolid(LZD) (10µg), Amoxicillin/Clavulanic acid(AMC) (20µg /10µg).

RESULTS

The biochemical characteristics results of the 118 consecutive, non-duplicate isolates from sputum, urine, abscess, swab, semen, vaginal swab, wound and blood samples showed that only 79(66.9%) fermented Mannitol salt agar, 60(76.7%) were Coagulase-positive, 19(31.6%) Coagulase-negative, 56(93.3%) of the isolates were S. aureus as shown in table 1, 3(5%) were S. xylosus, S. hyicus. 1(1.7%). Demographic information of the S. aureus strains (Table 1), Antibiotics susceptibility testing information of S. aureus strain (Table 2).

Table 1: Demographic Data of S. aureus Isolates (n=118).

A. Distribution of the Isolates within Clinical Specimen Analyzed

Isolate site	isolate size	confirmed s. Aureus	percentage (%)
Sputum	25	14(25%)	(21.2%)
Abscess	29	16(28.6%)	(24.6%)
Blood	3	3(5.4%)	(2.5%)
Urine	24	10(17.9%)	(20.3%)
Swab	15	3(5.4%)	(12.7%)
Semen	2	1(1.8%)	(1.7%)
Wound	12	6(10.7%)	(10.2%)
Others	8	3(5.4)	(6.8%)

B. Distribution within age-group

Age	Size	Percentage (%)
≤ 10 Years	1	(0.8%)
10-19	3	(2.5%)
20-29	34	(28.8%)
30-39	38	(32.2%)
40-49	26	(22.0%)
≥ 50 Years	16	(13.6%)

C. Source of S. aureus isolates

Source	Size	Percentage (%)
In-patients	53	(44.9%)
Out-patients	65	(55.1%)

D. Distribution within sex

Sex	Size	Percentage (%)
Male	57	(48.3%)
Female	61	(51.7%)

Table 2: Percentage Distribution of antibiotic resistant pattern.

Antibiotics	Percentage
Fox	46(82.1%)
Cn	8(14.3%)
Lzd	21(37.5%)
Van	11(19.6%)
Amc	30(53.6%)
Tgc	40(71.4%)
C	27(48.2%)
Cip	21(37.5%)
E	26(46.4%)
Da	37(66.1%)
Sxt	27(48.2%)

Keys: Cefoxitin (FOX), Gentamycin (CN), Amoxicillin-Clavulanic acid (AMC), Linezolid (LZD), Vancomycin (VAN), Tigercycline (TGC), Chloramphenicol (C), Ciprofloxacin (CIP), Erythromycin (E), Clindamycin (DA), Trimethoprim/Sulphamedazol (TRI/SUL).

DISCUSSION

Staphylococcus aureus has long been recognized as an important pathogen in human disease and is the most common cause of both community and hospital acquired infections.[11] The prevalence of Staphylococcus aureus 56(47.5%) observed among the suspected S. aureus isolates from hospital samples in this study shows the versatility of this organism amongst other staphylococci pathogen makes in clinical settings.[12,13,14] The highest prevalence of S. aureus was in found in abscess (25%) and sputum (25%), a finding that is consistent with other reports[15] and is in contrast with the observation of Orji et al., 2012.[16] The high prevalence of the isolates in abscess and sputum could be attributed to poor personal hygiene and exposure of the wounds, which might have made it more prone to contamination and infection.[13]

The treatments of infections caused by S. aureus have always been limited nowadays due to the alarming rate of resistance to the current conventional antibiotics. These organisms are capable of producing or acquiring some resistant gene that code for enzymes that are able to destroy or inactivate some antibiotics. Greater than 40% resistance was observed to Cefoxitin, Amoxicillin/clauvulanic acid, Tigercyclin, Clindamycin, Chloramphenicol, Erythromycin and Trimethroprime/ Sulphamedazol among the S. aureus isolates in this study. The relative susceptibility to Vancomycin has been commonly noted among the S. aureus isolated at different hospitals worldwide.[17,18]

This study provides important data on current antimicrobial resistance, including methicillin resistance, from a recent collection of clinical isolates of S. aureus from Maitama District Hospital, Abuja. A total 82.1% prevalence rate of methicillin (Cefoxitin) resistance was observed among the isolates this compares with prevalence rates of 54% in Japan.[19] 43% from a study in the USA.[20] and 30% in European countries.[21] This observed difference could be due in part to the increased antimicrobial resistance associated with district hospitals, possibly due to increased selective pressure arising from widespread antimicrobial use, as well as high density patient population in contact with healthcare staff and the attendant risk of cross infection.[22]

CONCLUSION

Many of the drivers of antimicrobial resistance have a common origin in inappropriate use of antimicrobials in human and animal health care or in agriculture, or from environmental contamination. Although our understanding of antimicrobial resistance is far from complete, the existing evidence base is sufficient to allow targeted policies to be developed in several areas. Such strategies to reduce antimicrobial resistance should consider the role and effect of many factors, including the resistance mechanisms, species of microorganism, the particular antimicrobial, as well as the setting and context. So far, all evidence suggests that no single solution exists and several overlapping and synergistic approaches will be needed.

ACKNOWLEDGMENT

We thank Prof. J.A Onaolapo for sharing his expertise in antimicrobial susceptibility testing and the Maitama District Hospitals that permitted us to collect isolates from their Microbiology Laboratory.

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