METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS (MRSA) IN BURN PATIENTS

ZULFIQAR A. NAQVI, KHURSHEED HASHMI* AND SALEEM A. KHARAL**
Department of Pathology, Fatima Jinnah Dental College, Karachi
*Department of Pathology, Sindh Medical College, Dow University of Health Sciences, Karachi
**Department of Microbiology, Basic Medical Sciences Institute, Jinnah Post Graduate Medical Centre, Karachi

ABSTRACT
Burns remain a significant public health problem in terms of morbidity, long term disability and mortality throughout the world, especially in economically developing countries. Thermal injury destroys the skin barriers that normally prevent invasion by microorganisms. Burn wound infection is a major complication in burn patients after initial period of shock. More than 70 % mortality in burn patients is attributed to infection. Among the Gram-positive cocci, Methicillin resistant Staphylococcus aureus (MRSA) is the most important nosocomial pathogen. Drug sensitivity of MRSA to only a few antibacterial agents limits therapeutic options and poses a threat to the life of the burn victim.

This study was conducted from November 2002 to February 2003 at the Department of Microbiology, Basic Medical Sciences Institute, Jinnah Postgraduate Medical Centre, Karachi, Pakistan. A total of 52 infected patients irrespective of age, sex, duration of hospital stay, percentage and degree of burn, admitted in burn unit of three Government Hospitals of Karachi were included in this study. A total of 170 burn wound swab specimen were collected from 52 patients. Forty one isolates of Staph. aureus were confirmed by biochemical reactions and coagulase test. Antimicrobial sensitivity and Methicillin resistance of 10 strains of MRSA was seen by disc diffusion Kirby Bauer’s method as per National Committee of Clinical Laboratory standard (NCCLs) 1998. MRSA infection was common in patients >12 years of age and TBSA >15% All (100%) isolates were sensitive to Vancomycin and Chloramphenicol, were resistant to Ciprofloxacin, whereas 70-90% strains were resistant to Clindamycin, Gentamicin, Clarithromycin, and Amikacin. Methicillin resistant Staphylococcus aureus (MRSA) were considered resistant to all β-lactam drugs.

Keywords: Burn wound, infection, MRSA.

INTRODUCTION
Burns remain a significant public health problem in terms of morbidity, long-term disability and mortality throughout the world; especially in economically developing countries. Thermal injury destroys the skin barriers that normally prevent invasion by micro-organisms (Singh et al., 2003; Barret et al., 1999; Murray and Finegold 1984; Lari AK et al., 1998; Nasser et al., 2003). Burn patients become susceptible to infection due to the loss of this protective barrier and decreased cellular and humoral immunity (Wong et al., 2002). Burn infection remains a major complication in burn patients after initial period of shock and the chance of infection persist until complete wound healing (Kaushik et al., 2001; Pallua et al., 1999 and Gang et al., 1999). On the other hand extensive full thickness burn wounds specially have high incidence of sepsis and other physical, pathological and psychological complications. It is estimated that infection in burn patients is responsible for more than 70% of the mortality whereas
the remaining 30% is attributable to other causes (Singh et al., 2003; Nasser et al., 2003; Kaushik et al., 2001; Gang et al., 1999; Tang et al., 1999; Bang et al., 1999; Oraloncul, et al., 2002; Nakae and 2000). Gram-positive bacteria in the depth of sweat glands and hair follicles may survive the heat of initial injury. Following colonization, these organisms of the surface start to penetrate the burn eschar to available extent and viable sub eschar tissues become invaded (Nasser et al., 2003 and Pallua et al., 1999).

MRSA is the most important pathogen among Gram-positive cocci (Oraloncul et al., 2002). Patients with extensive burn injuries are especially susceptible to infection with this organism. Infections with MRSA require special management. The percentage of these patients with proven microbiological MRSA infection in industrial countries has increased dramatically (Fuchs et al., 2002). Burn units within hospital have become major reservoir for MRSA that have special characteristic of spreading quickly in hospital environment (Mokaddas et al., 1999). Therefore this organism is considered as nosocomial pathogen and causes out breaks of infection that result in serious problem in the management of burn patients because of many strains becoming multi resistant to several classes of antibiotics (Gang et al., 1999 and Mokaddas et al., 1999).

MATERIAL AND METHODS

This study was conducted from November 2002 to February 2003 at the Department of Microbiology, Basic Medical Sciences Institute, Jinnah Postgraduate Medical Centre, Karachi, Pakistan.

Patients

A total 52 infected patients irrespective of age, sex, duration of hospital stay, percentage and degree of burn, admitted in burn unit of three Government Hospitals of Karachi were included in this study.

Material

Wound swabs were taken from all patients registered in the study. Swabs were immersed in Stuart’s transport medium.

Methods

A total of 170 burn wound swabs were collected from 52 patients. Swabs were collected from infected wound following cleansing of any remnant ointment. After collection, all swabs were inoculated on Blood agar, Mac-Conkey’s agar and nutrient agar and incubated at 37°C for 24 hours. Morphological Examination of pus smear and culture smear, colonial morphology, production of β hemolysis on blood agar and production of pigmentation on Nutrient agar revealed 41 isolates of Staphylococcus that were confirmed to be Staphylococcus aureus by biochemical tests like catalase, coagulase Manitol fermentation and Novobiocin sensitivity. Antimicrobial susceptibility test was was performed by disc diffusion method as per National Committee of Clinical Laboratory standards (NCCLs).

RESULTS AND DISCUSSION

Heavy bacterial wound colonization is more likely to lead to wound sepsis that may reflect the current status of the wound. This colonization may occur more rapidly when the condition of wound is poor. Poor socio-economic status of the patient may also be a very important predisposing factor in burn wound infection (Ozumba et al., 2000).

Total burn surface area has been found to be most important risk factor for nosocomial infection (Oraloncul et al., 2002). Infection is common at extreme of age (Edwards 2003). All patients in present study were the victim of infection therefore this observation is not significant in theses patients.

Staphylococcus aureus is a versatile human pathogen. It was the predominant cause of burn wound infection in pre antibiotic era and still persists as an important pathogen, strongly considered as a major cause of
nosocomial infection. Interestingly the frequency of infection has increased during last three decades. Burn units have become major reservoir for Methicillin resistant Staph. aureus that has the special characteristics for spreading quickly in a hospital environment. In recent years, marked increase in number of hospital acquired infections due to MRSA has been reported from many countries. Infection with MRSA varies widely from one geographical location to another, from hospital to hospital and over time (Mokaddas et al. 1999; Gang et al., 2000; Lari and Alaghebandan 2000; Prasanna and Thomas 1999; Muir et al., 1987). This observation is also reflected in present study, where the prevalence of MRSA infection was greater in one hospital to other and was not found in another hospital located in the same city. However, due to small number of cases included in the study, further comment on this point may not be relevant.

The prevalence of MRSA infection in the present study was 24.6%. It is less than that reported by Mokaddas et al 1996 i.e., 74.6% and other studies conducted in Guru Teg Bahadur Hospital in New Delhi from 1997 to 2002 (Singh et al., 2003).

### Table 1

<table>
<thead>
<tr>
<th>Patients</th>
<th>Cultures</th>
<th>Isolates Recovered</th>
<th>Coagulase +ve S. aureus</th>
<th>MRSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>52</td>
<td>170</td>
<td>190</td>
<td>41</td>
<td>10 (24.6%)</td>
</tr>
</tbody>
</table>

### Table 2

<table>
<thead>
<tr>
<th>Drug</th>
<th>Disc potency</th>
<th>Sensitive</th>
<th>Intermediate</th>
<th>Resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vancomycin</td>
<td>30 µg</td>
<td>10 (100%)</td>
<td>00</td>
<td>00</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>30 µg</td>
<td>10 (100%)</td>
<td>00</td>
<td>00</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>02 µg</td>
<td>02 (20%)</td>
<td>01 (10%)</td>
<td>07 (70%)</td>
</tr>
<tr>
<td>Amikacin</td>
<td>30 µg</td>
<td>-</td>
<td>01 (10%)</td>
<td>09 (90%)</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>05 µg</td>
<td>-</td>
<td>-</td>
<td>10 (100%)</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>10 µg</td>
<td>02 (20%)</td>
<td>00</td>
<td>08 (80%)</td>
</tr>
<tr>
<td>Clarithromycin</td>
<td>15 µg</td>
<td>02 (20%)</td>
<td>-</td>
<td>08 (80%)</td>
</tr>
</tbody>
</table>

In Muscat, Oman the prevalence of MRSA was about 95% during 1995-96 (Prasanna and Thomas 1999). In another study all isolates of S. aureus were resistant to Methicillin at Gulhane Military Medical Academy Istaumbul Turkey (Oraloncul et al., 2002). Authors of this study suggested many factors that may account for increased incidence of MRSA colonization and infection. These factors included use of broad spectrum antibiotics, average length of hospital stay and poor hospital infection control practices. A similar picture is also reflected in the present study.

The prevalence of MRSA infection in the present study was slightly higher than other studies. In a retrospective analysis of out break of MRSA at burn units of Pauwelstrasse Aachen, Germany during 2000-2001 (Fuchs et al., 2002) and Karolinska hospital Stockholm, Sweden, during 1993-95 (Cook, 1998), it was 16% and 18% respectively. Where as prevalence of MRSA was 31.6% in a study of government Medical College and hospital Chandi Garh India (Kaushik et al., 2001). In the study of Naseer et.al, conducted during 1999-2001 at Ain Shams University hospital Cairo Egypt no isolate of MRSA was found.
Methicillin Resistant S. Aureus in Burn Patients

(Nasser et al., 2003). Until the early 1980 MRSA reports consisted of isolated cases. In 1982 epidemic MRSA strains were described as multi-resistant strains that pose a serious problem in management of patients. Over time the susceptibility pattern of MRSA has changed with many strains becoming multi-resistant to several classes of antibiotics including Aminoglycosides, Macrolides, Quinolones and Tetracyclines (Mokaddas et al 1999; Gang et al., 2000; Prasanna and Thomas 1999 and Muir et al., 1987). Increasing prevalence of MRSA has been posing serious therapeutic and infection control program challenges with in the hospital environment (Cook, 1998). In a study conducted by Mokaddas et al. (1999) vast majority of the S. aureus, especially MRSA were resistant to Gentamicin (Mokaddas et al., 199). The author stated that MRSA were considered clinically resistant to all β-lactam drugs. Another study from Khoul hospital Muscat Oman in 1996 reported the resistance of the MRSA against Erythromycin, Gentamicin, Amikacin and Ciprofloxacin. Author further added that the susceptibility of MRSA to Ciprofloxacin has dropped from 76% in 1995 to 59% in 1996. The author quoted a reference according which in Australia 5.7% MRSA strains were resistant to this drug from 1964 to 1967 later became more resistant i.e., 50% by 1980. In a Chinese report by Xu et al. (1998), about 30% of MRSA were resistant to Ciprofloxacin (Xu et al., 1998). Similar trend of multi-resistance has been observed in the present study.

CONCLUSION

MRSA is a multi drug resistant strain. It may interfere with wound healing and infection may occur any time until complete wound healing. Therefore it is suggested that the risk of this infection may be minimized by forceful implementation of infection control measures. This may not only reduce the diagnostic and therapeutic cost but it will also help in delaying the process of development of drug resistance in clinical isolates.

REFERENCES

Muir I.F.K., Barclay T.L. and Settle J.A.D.

(continued)


