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Methicillin Resistant Staphylococcus Aureus: A Problem in the Burns Unit

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ABSTRACT

Methicillin-resistant staphylococcus aureus (MRSA) has become a frequent cause of nosocomial infection. It causes significant morbidity and mortality to the burn patients who have been shown to become colonized and infected more readily than other patient groups. Extensive burn injuries are particularly susceptible to infection as a result of the disruption of the normal skin barrier and accompanying depression of immune responses. Extended hospitalization and antibiotic therapy have been identified as additional risk factors for MRSA carriage and infection. The aim of this study was to know the incidence of MRSA infection and the effect of patients colonization by MRSA on the incidence of graft infection in the Burns Unit at Mansoura University Hospitals in the period from January 2001 to January 2002. Sterile swabs were taken from the nostrils, hair, hand skin and skin graft of the patients. The swabs were identified by morphological characteristics, Gram staining, coagulase production and sensitire identification system. The results revealed that colonization of patients skin with MRSA increased the risk of graft infection while nostril and hair colonization didn't significantly increase the incidence of graft infection. The production of enterotoxins A,B,C and D were evaluated in the isolated strains by reversed passive latex agglutination. Enterotoxin A was the most prevalent in the examined isolates. MRSA may have contributed to graft breakdown. We recommend regular screening of burns patients to give an early warning of the presence of MRSA and allow the assessment of barrier and infection control techniques.

INTRODUCTION

Staphylococcus aureus is recognized as one of the most important bacterial pathogens seriously contributing to the problem of hospital infections all over the world [1].

Penicillin was produced in large quantities in the 1940s and many lives were saved. However, the success was short-lived. It was found that some strains of staphylococcus aureus quickly developed resistance to penicillin by producing an enzyme (β-lactamase) which could break down the penicillin molecule. A number of synthetic derivatives of penicillin, resistant to the ß-lactamase enzyme, were developed. Of these, methicillin became the standard treatment for staphylococcus aureus. In 1961, the first methicillin-resistant strains of staphylococcus aureus (MRSA) were isolated in Europe. They were first reported in Australia in 1966 in the eastern states and in the United States in 1968. As well, other strains were identified that had a broad pattern of resistance, not only to methicillin, but also to the aminoglycosides and cephalosporins. In the 1970s only a small number of the Methicillin-resistant strains of Staphylococcus aureus were isolated (< 2%). However, in 1979, a survey in Victoria reported an increase in MRSA infections to 20-40% of all Staphylococcal isolates in six of the large teaching hospitals [2].

MRSA continues to be a major cause of serious infection to man, both in hospitals and in the community [3]. Until the early 1980s MRSA reports consisted of isolated cases, later in 1982 epidemic MRSA strains (EMRSA) were described as multi-resistant strains with special capacity to colonize patients and staff and cause widespread outbreaks of infections. These epidemic MRSA strains have subsequently spread to various parts of the world [4].

β-lactam antibiotics such as methicillin inactivate penicillin binding proteins 1,2 and 3 (PBPs 1,2 and 3) by the acylation of the catalytic site of the PBP. The PBPs occur in the bacterial cell wall and have an enzymatic role in the

synthesis of peptidolgycan. PBPs normally possess a high affinity for ß-lactam antibiotics; in MRSA this affinity is reduced resulting in antibiotic resistance. MRSA carry the mec A gene which encodes an additional low-antibiotic affinity penicillin binding protein, Known as PBP2a [5,6].

MRSA hospital infection have been reported from many countries [7-11]. The concern about MRSA infection in burns units has been increasing since the late 1980s [12-17].

The organism is now a common cause of nosocomial infection, spread by hand and airborne particles and via environmental routes. Wound sepsis remains a major cause of death in burn patients and colonization with MRSA of the surface of burn wounds may go on to cause systemic infection and serious clinical complications. MRSA colonization and infection have significant implications for patient nursing, treatment and recovery, providing a reservoir of MRSA within the burn unit, increasing the risk of infection for other patients [18].

The aim of this study was to assess the incidence of MRSA in the Burns Unit at Mansoura University Hospitals and the effect of patients colonization by MRSA on the incidence of graft loss.

SUBJECTS AND METHODS

Patients:

This work was performed on 62 patients (34 males and 28 females) admitted to the Burns Unit at Mansoura University Hospital from January 2001 to January 2002. Their ages ranged from 13 to 50 years (mean 28.6±9.9 years). The % TBSA burn ranged from 25 to 60% (mean 41±9.9%). The 3rd˚ burn ranged from 8 to 30% (mean 17.8±6.9). All patients suffered flame burns. The length of hospital stay was 52.6±19.3 days (Table 1).

Resuscitation and wound care:

All patients were resuscitated based on the Parkland formula guidelines using crystalloids, except in a few cases where there was either delayed or difficult resuscitation and colloids were used earlier than 24h post-burn. The deep partial-thickness and full-thickness wounds were dressed by bovidone-iodine bathing followed by application of 1% silver sulphadiazine (Dermazin) and 0.2% chlorhexidine digluconate cream, with melolin for open wounds and tulle gras with 0.5% chlorhexidine for virtually healed wounds or skin grafts. Second-degree superficial burns were dressed with sofra-tulle (1% framycetin in paraffin and anhydrous lanolin). Nutritional care was provided using a 1 cal/ml. mixture and gradually increased to the full amount and continued until healing was complete. Primary excision and skin grafting with autografts were done in some of the deep partial-thickness and all the full-thickness burn patients as a routine. Burn areas of 15% were dealt with during the first and the subsequent sessions of surgery. The first session was performed on the 3rd postburn week. The subsequent surgery was performed as soon as the previous grafts were stable and the donor area was again available for harvest. Antibiotics were used as per susceptibility reports of the isolates. All patients received a tetanus toxoid booster on admission. Informed consent was obtained from all patients.

Microbiology:

Sterile swabs were collected from the nostrils, hair, hand skin, catheter sites and skin grafts of the patients. Swabs were cultured on blood agar and mannitol salt agar (MSA) (bioMerieux) and incubated at 37ºC for 24 hours and 48 hours respectively. The resulting growth was identified by sensitite system. Staphylococcus aureus colonies were identified based on growth characteristics on MSA plates (yellow colonies), Gram stain and positive results for coagulase tube test and catalase test. Identification was confirmed by sensitite identification system following the manufacturer's instructions.

Antibiotic-resistance:

The isolated staphylococcus strains were tested for resistance to antimicrobial agents by performing disc diffusion method using commercial discs (bioMerieux) according to the guidelines of the national committee for clinical laboratory standard [9] with a 1ug oxacillin disc and muller hinton agar. The plates were incubated in ambient air at 35ºC for 24 hours. Any growth within 12 mm diameters zone around the disc was indicative of resistance. Other tested antibiotics included amoxacillin (20 ug) + clavulanic acid (10 ug), ciprofloxacin (5 ug), clindamycin (2 ug), penicillin (10 units), gentamycin (10 ug), penicillin (10 units), rifampicin (30 ug), trimethoprim-sulfamethoxapole (1.25 ug + 23.75 ug) and vancomycin (30 ug).

Enterotoxin production:

Isolated staphylococcus aureus strains were also subjected to screening for enterotoxin production by reversed passive latex agglutination (oxoid).

Principle of reversed passive latex agglutination:

Isolated organisms were inoculated into tryptone soya broth and incubated at 37ºC for 24 hours. After growth, the test sample was extracted by means of centrifugation. The test is performed in V-well microtitre plates. Latex particles are sensitized with purified staphylococcal enterotoxins A,B,C and D. These latex particles will agglutinate in the presence of the corresponding enterotoxin, forming a clearly visible lattice structure. If staphylococcal enterotoxins are absent or present at concentrations below the assay detection level, no such lattice structure is formed and the latex particles settle in a tight button at the base of the well.

Statistical analysis:

Data were analyzed using SPSS (Statistical Package for Social Sciences, Version 9). Data were presented as number and percent. Chi-square and Fisher's exact tests were used to test the difference between groups when required. Agreement was measured by Kappa coefficient. *p*-value ≤ 0.05 was considered statistically significant.

RESULTS

Staphylococcus aureus was present in 37.1% of patients nostrils (20.96% MSSA and 16.3% MRSA), in 25.8% of patients hair (14.52% MSSA and 11.29% MRSA), in 41.9% of patients hand skin (22.58% MSSA and 19.35% MRSA) and in 32.3% of graft infections (12.9% MSSA and 19.35% MRSA). Gram-negative bacilli represented 40.3% of organisms causing graft infections. Pseudomonas aeruginosa was the most common among gram negative bacilli representing 48% followed by E.Coli (24%), Klebsiella pneumonia (24%) and Proteus mirabilis (4%) (Table 2).

Patients colonized in the skin showed a significant increase in graft infection more than Staphylococcal colonization in the nostrils and hair (Kapp = 1.0 and $p = < 0.001$) (Table 3). A significant increase in graft loss was observed in patients with Staphylococcal colonization in the hand skin ($p = 0.036$) in contrary to those in the nostrils and hair ($p = 0.57$ and $p = 1.0$ respectively) (Table 4). There was a highly significant increase in graft loss in patients grafts infected with MRSA compared to patients with no MRSA infection in their grafts ($p = 0.009$) (Table 5).

The resistance of MRSA and MSSA to antibiotics was: oxacillin (100%, 0.0% respectively), clindamycin (100%, 12.5%), pencillin (100%, 25%), gentamycin (58.3%, 50%), erythromycin (33.3%, 25%), ciprofloxacin (33.3%), 25%), tetracycline (25%, 25%), trimethoprim + sulfamethoxazole (25%, 25%), rifampicin (16.7%, 12.5%) while vancomycin showed no resistance (Table 6).

About 93% of MRSA produced enterotoxins while 75% of MSSA produced enterotoxins. Enterotoxins A was the most common representing 85.7% and 75% of MRSA and MSSA isolates respectively (Table 7).

Table (1): Patient population.

	Mean ± S.D.
Total number	62
Sex:	
Male	34
Female	28
Age (years)	28.6±9.9
TBSA burn (%)	41±9.9
3rd^{o} burn (%)	17.8±6.9
E/G per patient	1.5±0.5
Length of hospital stay (days)	52.6±19.3

E/G: Excision and skin graft.

Table (2): Distribution of organisms causing infections.

	Staphylococcal infection						Gram negative bacilli	
	MSSA		MRSA		Total			
	No.	%	No.	%	No.	%	No.	%
Nostril	13	20.96	10	16.3	23	37.1		
Hair	9	14.52	7	11.29	16	25.8		
Skin	14	22.58	12	19.35	26	41.9		
Graft infection	8	12.9	12	19.35	20	32.3	25	40.3

Table (3): Effect of staphylococcal colonization on graft infection.

			Graft infection			Kappa coefficient
			MSSA	MRSA	Total	
Nostril	MRSA	Count	1	3	4	
		%	20.0%	75.0%	100.0%	
	MSSA	Count	1	0	1	$p = 0.17$
		%	100.0%	0.0%	100.0%	
Hair	MRSA	Count	2	1	3	
		%	66.7%	33.3%	100.0%	
Skin	MRSA	Count	0	10	10	
		%	66.7%	100.0%	100.0%	
	MSSA	Count	4	0	4	$p = 0.001$
		%	100.0%	0.0%	100.0%	

Table (4): Effect of staphylococcal colonization on graft loss.

			Graft loss			Fisher's exact
			No	Yes	Total	
Nostril	-ve	Count	34	5	39	
		%	87.2%	12.8%	100.0%	
	MRSA	Count	10	-	10	$p = 0.57$
		%	100.0%		100%	
Hair	-ve	Count	41	5	46	
		%	89.0%	10.9%	100%	
	MRSA	Count	7	7		$p = 1.0$
		%	100%		100%	
Skin	-ve	Count	34	2	36	
		%	94.4%	5.6%	100.0%	
	MRSA	Count	9	4	13	$p = 0.036$
		%	69.2%	30.8%	100.0%	

-ve: No infection.

Table (5): Effect of graft infection with MRSA on graft loss.

			Graft loss		Total	Fisher's exact
			No	Yes		
Graft	-ve	Count	17		17	
		%	100.0%	0	100.0%	$p = 0.009$
	MRSA	Count	8	5	13	
		%	61.5%	38.5%	100.0%	

Table (6): Antibiotic susceptibility of staphylococcus aureus.

Drugs			Organism			Fisher's exact
			MSSA	MRSA	Total	
Amoxicillin + clavulamic acid	R	Count	12	2	14	
		%	100%	25%	70%	$p < 0.001$
	S	Count	0	6	6	
		%	0	75.0%	30.0%	
Rifampine	R	Count	2	1	3	
		%	16.7%	12.5%	15.0%	$p = 1.0$
	S	Count	10	7	17	
		%	83.3%	87.5%	85.0%	
Tetracylcin	R	Count	3	2	5	$p = 1.00$
		%	25.0%	25.0%	25.0%	
	S	Count	9	6	15	
		%	75.0%	75.0%	75.0%	
Trimethoprin + sulfamethoxazole	R	Count	3	2	5	
		%	25%	25.0%	25.0%	$p = 1.0$
	S	Count	9	6	15	
		%	75.0%	75.0%	75.0%	
Ciprofloxacin	R	Count	4	2	6	
		%	33.3%	25.0%	30.0%	$p = 1.0$
	S	Count	8	6	14	
		%	66.7%	75.0%	70.0%	
Clindamycin	R	Count	12	1	13	$p = < 0.001$
		%	100.0%	12.5%	65.0%	
	S	Count	0	7	7	
		%	0	8.5%	35.0%	
Erythromycin	R	Count	4	2	6	$p = 1.0$
		%	33.3%	25%	30.0%	
	S	Count	8	6	14	
		%	66.7%	75.0%	70.0%	
Gentamycin	R	Count	7	4	11	
		%	58.3%	50.0%	55.0%	$p = 1.0$
	S	Count	5	4	9	
		%	41.7%	50.0%	45.0%	
Oxacillin	R	Count	12	0	12	
		%	100.0%	0	60.0%	$p = < 0.001$
	S	Count	0	8	8	
		%	0	100%	40.0%	
Penicillin	R	Count	12	7	19	
		%	100%	87.5%	95.0%	
	S	Count	0	1	1	
		%	0	12.5%	5.0%	$p = 0.4$
Vancomycin	R	Count	12	8	20	
		%	100%	100%	100%	

R = Resistance. S = Sensitivity.

Table (7): Enterotoxin production by staphylococci.

Toxin	MRSA		MSSA	
	N	%	N	%
A	1	85.7	6	75
A + B	1	7.1	0	0.0
A + (A + B)	13	92.9	6	75
Negative	1	7.1	2	25
Total	14	100	8	100

DISCUSSION

The burn wound is particularly susceptible to bacterial colonization and infection due to the physical disruption of the normal skin barrier and the accompanying reduction in cell-mediated immunity. It has been demonstrated that a 30% burn injury induced a high level of immunosuppression in mice in terms of cell-mediated immunity which in turn could be prevented by early wound excision and grafting **[20]**. Furthermore, a significant correlation be-

tween immunological status and mortality was observed in burn patients. Immunosuppression was present in 70.1% of such patients who died [21]. Experiments in mice suggest that MRSA appears to be less virulent in the normal subject but equally virulent in the immunocompromised [22]. Staphylococci can survive intracellularly in polymorphonuclear leukocytes (PMNs). However, in burn patients, PMNs bactericidal function is decreased allowing the organism to survive longer. No enhanced bactericidal activity of PMNs taken from burn patients could be demonstrated even by the addition of teicoplanin, which is known to be concentrated in PMNs and has been extensively used in the treatment of MRSA infection [23].

Administration of antibiotics prior to the development of infection and extended duration of hospitalization have been linked with MRSA infection and are thought to act synergistically in promoting the acquisition of MRSA [24]. Asensio et al. identified six factors that were independently associated with MRSA infection, colonization, increasing age, ward type (particularly intensive care units), coma, previous hospitalization, invasive procedures and length of hospitalization. However, they failed to show any independent risk factor associated with antibiotic therapy [25]. Hunt et al. reported a direct correlation between the length of stay in hospital and the risk of a burn becoming infected with MRSA with 90% of colonization originating in the wound, 9% the lungs and 1% other sites. The appearance of new cases in the unit also correlated with high patient population, nursing overtime and burn size. An additional factors which could be influential in this process is the increase in community nursing, where nursing and convalescent homes which have become colonized with MRSA and act as a reservoir for infection [26].

Colonization with MRSA increases the patients risk of bacteremia, septicemia and metastatic spread with associated complications including the loss of skin grafts in burns patients [18]. MRSA colonization rates of up to 39% have been reported in burn patients [27] but proper infection control programmes have since been shown to be effective in maintaining low rates of nosocomial MRSA infection [16,28]. Investigations by the central public health laboratory showed that the incidence of MRSA isolations from blood cultures in England and Wales remained stable during 1989-1991 at 1.5% but from then until 1995 there has been an increase to 13.2% [29]. Lesseva and Hudjiski reported that 23.8% of S.aureus isolated from burn wounds were MRSA and 31.32% of blood culture isolates were MRSA, the overall MRSA infection rate being 18.8% [30]. Suzuki et al. reported multiple brain abscess formation associated with MRSA septicemia and compounded by the breakdown of the blood-brain barrier in a burn patient [31]. Gang et al. reported the development of tumor like growths on the surface of healing burn wounds which were specifically associated with the presence of MRSA; six out of ten patients affected required radical surgery and systemic antibiotic therapy [32]. Gang et al. indicated a high incidence of Staphylococcal septicemia (especially due to MRSA) in their burn unit. A surface wound is the likely source of entery to the blood stream in these immunocompromised patients. The organism could be detected as early as 48hr postburn and in as little TBSA burn as 1% [33].

Our work revealed that Staphylococcus aureus was present in 37.1% of patients nostrils (20.96% MSSA and 16.3% MRSA), in 25.8% of patients hair (14.52% MSSA and 11.29% MRSA), in 41.9% of patients skin (22.6% MSSA and 19.4% MRSA) and in 32.3% of graft infections (12.9% MSSA and 19.4% MRSA). There was a significant increase in graft infections in patients colonized in the skin (Kappa = 1.0 & p = < 0.001), while Staphylococcal colonization in the nostrils and hair didn't increase the incidence of graft infection significantly. Also, there was a significant increase in graft loss in patients colonized in the skin (p = 0.036). In contrary, nostril and hair colonization didn't significantly increase the incidence of graft loss (p = 0.57 & p = 1.0 respectively).

The significant increase in graft infections in patients colonized in the skin (p = 0.001) may be attributed to the more and easy exposure of burn grafts to colonized skin. It is evident that MRSA colonized in the skin also affected the incidence of graft loss. This may be explained by the increased incidence of graft infections caused by MRSA colonization of the skin. There was a highly significant increase in graft loss in patients grafts infected with MRSA compared to patients with no MRSA infection in their grafts (p = 0.009) Garrouste-Orgeas et

al. reported 5.1% patients were colonized with MRSA on admission and 4.9% acquired MRSA colonization in the ICU. MRSA colonization increased the risk of S.aureus infection [34]. Environmental contamination is a potential source of nosocomial MRSA. MRSA may have contributed to skin graft breakdown in one case and delayed wound healing in others [35].

The dead tissue which constitutes the burn eschar can not reliably be reached by antibiotics administered systemically. Careful selection of topical agents for prophylactic treatment of burn wounds can achieve adequate concentrations penetrating through the damaged tissue to the underlying healthy tissue, thereby limiting bacterial colonization, but wound care remained the factor of primary importance. Meticulous attention to hand washing and wound care are likely to be the most effective protection against nosocomial spread of MRSA in the burn unit [18]. Additional treatment of chlorhexidine bathing followed by application of 1% silver sulphadiazine and 0.2% chlorhexidine digluconate cream preparations with melolin for open wounds or tulle gras with 0.5% chlorhexidine for virtually healed wounds or skin grafts had been recommended [36]. Hansbrough indicated that the use of ointment containing collagenase Clostridium histolyticum for the removal of the necrotic tissue resulted in earlier healing of the burn sites than the use silver sulphadiazine and was less painful for the patient [37]. Mupirocin is extensively used for the eradication of MRSA carriage in hospital personnel with great success and has been used as a topical antibacterial cream to eradicate MRSA in burn wounds with good effect [18]. Mupirocin, a type A pseudomonic acid produced by Pseudomonas fluorescens, inhibits protein synthesis by competing for the isoleucine binding site on bacterial isoleucyl tRNA synthetase [39]. Smoot et al. suggested that in vitro susceptibility testing of topical antibacterial agents is relevant in selecting appropriate therapy for MRSA. In vitro susceptibility testing of MRSA isolated demonstrated increased resistance to mafenide acetate and silver sulphadiazine when extensively used in burn units [40]. Plasmid-mediated resistance to many commonly used biocides had been discussed, including the resistance to chlorhexidine and povidone-iodine in MRSA [41]. The need to monitor antibacterial resistance profiles is clear particularly when there is persistent use of antibacterial agents and the associated risk of evolution or selection of resistance. In our burns unit, no topical sensitivity test is carried out.

Vancomycin remains the mainstay of treatment for serious MRSA infection. Its efficacy is well recognized, but with more extensive use of this antibiotic the likelihood of resistance emerging increases. Philips et al. demonstrated plasmid transfer of antibiotic resistance from both MRSA and antibiotic resistant enterococci to a strain of MSSA, at a frequency of up to 80% between Staphylococci and 30% between enterococci and Staphylococci. This in vitro demonstration of transfer of resistance highlights the need for strategies to prevent the emergence of resistant organisms by effective and properly controlled use of antibiotics, in particuar vancomycin [15].

Alternative antibiotic regimes had been advocated to treat MRSA infections, in particular, rifampicin which is highly bactericidal for Staphylococci but up to 25% of isolates had been reported as resistant to this antibiotic in some burn units [36]. Combined therapy to potentiate the effects of different antibiotics and to reduce the likely selection of resistant variants is a useful therapeutic alternative. The addition of novobiocin or trimethoprim-sulfamethoxazole to rifampicin therapy has been shown to be effective against MRSA, reducing the emergence of resistance to rifampicin [42]. Ciprofloxacin had been used for the treatment of MRSA but the emergence of resistance was rapid [43]. The resistance to ciprofloxacin in MRSA isolates rose from 9 to 82% between 1988 and 1993 [44].

Vancomycin therapy although highly effective against MRSA infection is hazardous with a risk of toxicity, nephrotoxicity, phlebitis, neutropenia and other contraindications. Vancomycin therapy in burn patient exhibits altered pharmacokinetics and specifically designed dosing regimes based on body weight, creatinine clearance and serum trough levels had been recommended to provide adequate antibiotic therapy [45]. Avoiding the use of this potentially toxic drug by preventing MRSA infections had immediate obvious advantages plus additional cost savings [18].

Our study showed the resistance of MRSA and MSSA to antibiotics as follow: oxacillin (100%, 0.0% respectively), clindamycin (100%,

12.5%), penicillin (100%, 25%), gentamycin (58.3%, 50%), erythromycin and ciprofloxacin (33.3%, 25% for each), tetracycline and trimethoprim + sulfamethoxazole (25%, 25% for each), rifampicin (16.7%, 12.5%) while vancomycin showed no resistance. This is in agreement with Udo et al. who isolated MRSA of patients in an intensive care unit and found that all isolates were susceptible to vancomycin, rifampicin and clindamycin but were resistant to methicillin and gentamycin [46]. Kumari et al. on studying antibiotic susceptibility of MRSA, reported all isolates were susceptible to vancomycin but were resistant to penicillin, methicillin, erythromycin and ciprofloxacin [47]. On studying antibiotic resistance to MRSA, Leski et al. revealed that resistance to erythromycin was 48.7%, tetracycline (40.5%), ciprofloxacin (12.7%) and trimethoprim-sulfamethoxazole (8.9%). The most active drugs were rifampicin (2.5% resistance) and vancomycin (no resistance) [1].

MRSA had been implicated in toxic shock syndrome with patients exhibiting pyrexia, anuria, diarrhoea and hypotension due to enterotoxin production [48]. EMRS A-15 is enterotoxin C positive and EMRSA-16 is enterotoxin A and toxic shock syndrome toxin 1 positive. Coia et al. studied the enterotoxin A,B,C and D production of Staphylococci by reversed passive latex agglutination and found that 87% of MRSA and 60% of MSSA were enterotoxin producers (89% of these produced enterotoxin A alone) [49]. Our study revealed about 93% of MRSA produced enterotoxins while 75% of MSSA produced enterotoxins. Enterotoxins A were the most common, representing 85.7% and 75% of MRSA and MSSA isolates respectively.

Most MRSA are said to be caused by a nosocomial infections [16]. Transmission of MRSA could occur by a combination of air-borne, transient hand-borne and environmental routes [14]. Rigorous infection control efforts have been recommended to prevent the spread of infection viz. isolation of the infected patient, barrier nursing, the use of sterile gown, gloves, cap, mask, hand washing and careful selection of antibiotics. The isolation was achieved by enlargement of the floor area per bed. Early closure of the burn wound, the primary colonized site, is an effective measure against infection [17]. Periodic "mechanical" cleaning of and the use of chemical disinfectants in the burns-center appear to be very effective control measures against MRSA [50]. Regular screening of burns patients not only gives an early warning of the presence of MRSA but should allow the efficiency of barrier and infection control techniques to be assessed. Prevention is better than cure but for those who are already affected, control is the mot likely achievable goal rather than eradication.

In conclusion, colonization with MRSA increases significantly the patients risk of skin grafts breakdown in burns patients. We recommend regular screening of burns patients to give an early warning of the presence of MRSA and allow the assessment of barrier and infection control techniques.

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