JOURNAL OF CLINICAL MICROBIOLOGY, Nov. 2000, p. 3926–3931 Vol. 38, No. 11
0095-1137/00/$04.00+0

Community Acquisition of Gentamicin-Sensitive Methicillin-Resistant *Staphylococcus aureus* in Southeast Queensland, Australia

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Received 12 April 2000/Returned for modification 28 June 2000/Accepted 18 August 2000

Community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) susceptible to gentamicin has been reported in a number of countries in the 1990s. To study the acquisition of gentamicin-sensitive MRSA (GS-MRSA) in southeast Queensland and the relatedness of GS-MRSA to other strains of MRSA, 35 cases of infection due to GS-MRSA from October 1997 through September 1998 were examined retrospectively to determine the mode of acquisition and risk factors for MRSA acquisition. Thirty-one isolates from the cases were examined using a variety of methods (antibiotyping, phage typing, pulsed-field gel electrophoresis [PFGE] fingerprinting, and coagulase typing by restriction analysis of PCR products) and were compared with strains of local hospital-acquired gentamicin-resistant MRSA (GR-MRSA) and of Western Australian MRSA (WA-MRSA). Only 6 of 23 cases of community-acquired GS-MRSA had risk factors for MRSA acquisition. Twenty of 21 isolates from cases of community-acquired infection were found to be related by PFGE and coagulase typing and had similar phage typing patterns. Hospital- and nursing home-acquired GS-MRSA strains were genetically and phenotypically diverse. Community-acquired GS-MRSA strains were not related to nosocomial GR-MRSA or WA-MRSA, but phage typing results suggest that they are related to GS-MRSA previously reported in New Zealand.

Methicillin (oxacillin)-resistant *Staphylococcus aureus* (MRSA) has proven to be one of the more widespread and durable nosocomial pathogens of the late 20th century (1, 34). In eastern Australia the appearance of MRSA was documented as early as 1965 (26) and was followed by an epidemic of gentamicin-resistant MRSA (GR-MRSA) in the late 1970s and early 1980s (22). MRSA has remained endemic in eastern Australian states in the 1980s and 1990s, and the majority of isolates have been resistant to gentamicin and multiple other non-beta-lactam antimicrobials (30, 31). Throughout this period GR-MRSA did not become established as an endemic problem in the state of Western Australia (25). However, in the late 1980s strains of gentamicin-sensitive MRSA (GS-MRSA) began to cause community-acquired infections in remote Aboriginal communities in northern Western Australia and subsequently spread to the Perth metropolitan area in the south, causing both community-acquired and nosocomial infection (20, 24). These strains have been referred to as WA-MRSA. Further spread of WA-MRSA to the Northern Territory has since been documented (16).

The emergence of GS-MRSA, as either a nosocomial or community-acquired infection phenomenon, is now worldwide. GS-MRSA with increased susceptibility to other antimicrobials has recently been reported in six widely dispersed hospitals in France and one in the West Indies (15). In the United States an increase in the incidence of community-acquired MRSA infections in children in Chicago has been observed (12). Many children had no identified risk factors for MRSA infection, and 14 of 15 isolates from such children were gentamicin susceptible and were more likely to be susceptible to other antimicrobials than nosocomially acquired isolates. In the southwest Pacific region, community-acquired infections due to GS-MRSA have been reported in the mid-1990s in Auckland, New Zealand. The majority of strains involved belong to the Western Samoan phage patterns (WSPP), and infections are particularly common among the Polynesian population (17, 24). The emergence of community-acquired GS-MRSA infections has also been observed in Brisbane, Sydney, Canberra, and Melbourne in eastern Australia in the late 1990s (5).

The observation in our laboratory that GS-MRSA was being isolated de novo from patients attending hospital emergency departments and outpatient clinics prompted a prospective collection of all GS-MRSA isolates from clinical specimens from October 1997 through September 1998 and a subsequent retrospective survey of associated clinical and epidemiological data. We wished to determine the mode of acquisition associated with GS-MRSA, the spectrum of infection associated with it, genetic relationships within GS-MRSA strains, and relatedness to local strains of GR-MRSA. We also hoped to determine the relationship of these isolates to WA-MRSA and to the MRSA reported in the southwest Pacific region (SWP-MRSA).

MATERIALS AND METHODS

Setting. The study was performed at the microbiology laboratory at Princess Alexandra Hospital. This laboratory serves a 900-bed university hospital and three community hospitals (400 beds in all) within the cities of Brisbane and Logan and the shire of Redland, all of which fall within the Brisbane metropolitan area in southeast Queensland. A total of 820,000 people live within the area served by these institutions, although another three laboratories also provide services within the same area.

Study design. We conducted a retrospective analysis of all new unique clinical isolates of GS-MRSA and cases associated therewith from October 1997 through September 1998. Medical records of all cases were reviewed and patients were interviewed by phone where possible to determine type of infection, acquisition

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status (community, hospital, or nursing home), ethnicity, and outcome of infection. Classification of infections as community acquired or nosocomial (hospital or nursing home) was in accordance with Centers for Disease Control and Prevention definitions (9). In addition, ascertainment of acquisition status included searching the medical record and questioning during the interview for evidence of contact with health care institutions (including nursing homes) within the preceding 12 months, previous surgery, underlying chronic disorder, or a household member with contact with health care institutions; cases of community-acquired infection were subclassified as either having or not having risk factors for prior MRSA acquisition.

Identification. *S. aureus* was identified by the presence of clumping factor and detection of the *nuc* gene, and oxacillin (methicillin) resistance was confirmed by detection of the *mec* gene. The multiplex PCR procedure used was based on a modification of the method by Unal et al. (32); the *mecA* primers were described by Murakami and Minamide (19), and the *nuc* primers were described by Brakstad et al. (4). The 25-μl reaction mixture consisted of 10 μl of lysate, 100 μM (each) deoxynucleoside triphosphate, 0.2 μM (each) primer, 0.5 U of DyNAzyme II DNA polymerase (Finnzymes Oy, Espoo, Finland) in 10× PCR buffer (1× is 10 mM Tris-HCL [pH 8.8], 50 mM KCl, 1.5 mM $MgCl_2$, 0.1% [wt/vol] Triton X-100). DNA amplification consisted of an initial cycle of 94°C for 5 min, 55°C for 30 s, and 72°C for 2 min; this was followed by 29 cycles of 94°C for 30 s, 55°C for 30 s, and 72°C for 2 min. PCR products were visualized on 2% agarose gels stained with ethidium bromide.

Control and comparator strains. Control strains used for coagulase typing were *Staphylococcus epidermidis* ATCC 12228, *S. aureus* ATCC 29213, and MRSA ATCC 49476. Control strains used for *mec* and *nuc* gene detection were *S. aureus* ATCC 29213, MRSA ATCC 49476, *S. epidermidis* ATCC 14990, and *S. epidermidis* (wild, *mecA* positive) PA2E16433. For the purpose of comparison, WA-MRSA B8-10 and B8-31 (Pathcentre, Perth, Western Australia, Australia) and six local isolates of hospital-acquired GR-MRSA selected to represent prevalent antibiograms (results not shown) and phage types were also tested. All isolates were stored on Protect bacterial preservers (Technical Service Consultants Ltd., Lancashire, United Kingdom) at −80°C.

Antimicrobial susceptibility tests. Tests for susceptibility to fusidic acid, rifampin, tetracycline, erythromycin, and ciprofloxacin were performed by the Vitek IMS using the GPS-IX card (bioMerieux Vitek, Hazelwood, Mo.). The production of β-lactamase was determined by the use of a Cefinase disc (Becton Dickinson, Cockeysville, Md.). Heteroresistance to oxacillin was detected by a disc method (7). Briefly, a 5-μg oxacillin disc was added to a Mueller-Hinton agar plate containing no NaCl using an inoculum of 10^8 CFU/ml. The plate was incubated for 48 h at 30°C. A zone diameter of <20 mm indicated a resistant isolate. Heterogeneously resistant isolates exhibited partial growth or microcolonies within the inhibition zone.

Coagulase typing by PCR. Molecular typing on the basis of coagulase gene polymorphisms was performed by a modification of the method of Goh et al. (10). Overnight broth cultures (1 ml of tryptic soy; 35°C) were washed by centrifugation (1,000 × *g*) in 1 ml of 50 mM Tris-EDTA (TE) buffer (Sigma, St. Louis, Mo.). Pellets were resuspended in 500 μl of TE containing 15 U of lysostaphin (Sigma)/ml. Suspensions were heated to 37°C for 1 h. One milliliter of lysing buffer (0.45 μl of Igepal CA-630 [Sigma], 0.45 μl of Tween 20 [Sigma], 6 μl of proteinase K [Sigma], and 1 ml of PCR buffer [50 mM KCL, 10 mM Tris-HCL, 1.5 mM MgCl]) was added before the samples were heated for 1 h at 56°C. Samples were heated at 95°C for 10 min and centrifuged, and the supernatant was frozen at −80°C for subsequent use. The 3′-end region of the coagulase gene was amplified using primers COAG2, 5′CGAGACCAAGATTCAA CAAG3′, and COAG3, 5′AAAGAAAACCACTCACATCA3′, which hybridize to sites 1632 to 1651 and 2589 to 2608, respectively. PCR amplifications were performed by adding the cell lysate (10 μl) to a 40-μl PCR mixture with the addition of 1.5 mM $MgCl_2$, as described in detail by Goh et al. (10). Seventeen microliters of the PCR product was digested for 15 min at 37°C with 6 U of the restriction enzyme *Hae*III (Sigma) in 1.9 μl of buffer provided as described by Lawrence et al. (14). The *Hae*III digests were visualized on 2% agarose gels stained with ethidium bromide. Isolates were allocated to types based on the sizes of their PCR products and to subtypes based on restriction fragment length polymorphisms (RFLPs) of the digested product. Subtypes were determined by the number of bands present and their sizes.

Fingerprinting by PFGE. Pulsed-field gel electrophoresis (PFGE) of chromosomal DNA was performed using the enzyme *Sma*I. DNA was separated on a GenePath system (Bio-Rad, Hercules, Calif.) using the GenePath group 1 reagent kit (Bio-Rad) with initial pulse times of 5.3 and 34.9 s at the end of the 20-h run. Gels were stained with ethidium bromide and were photographed under UV illumination. The patterns were compared visually using the criteria of Tenover et al. (28) and were analyzed with GelCompar software (Applied Maths, Kortrijk, Belgium). Results were analyzed using the unweighted pair group method for arithmetic averages and the Dice coefficient (6) with 1.2% band tolerance.

Phage typing. Phage typing was performed using the method of Blair and Williams (2). The 23 phages of the Basic International Set of Typing Phages were supplemented by 10 phages of the International Set of Experimental Phages for MRSA (23), by two experimental phages issued by the International Centre at Colindale, London, United Kingdom, and by eight experimental phages isolated at the Royal Prince Alfred Hospital, Sydney, Australia (33). All phages were used at 100 × routine test dilution.

TABLE 1. Epidemiological and clinical characteristics of 35 cases of GS-MRSA infection analyzed according to mode of acquisition[b]

Characteristic	No. of cases of infections that were:				P[a]
	Community acquired		Health care facility acquired		
	No risk factors	With risk factors	Hospital	Nursing home	
Total no. of cases	17	6	10	2	
Sex					0.19
Male	11	2	4		
Female	6	4	6	2	
Ethnicity					
Aboriginal	1				
Caucasian	5	1	8	2	0.001
Polynesian	11	5	2		
Nature of infection					
Abscess	13	3			<0.0001
Cellulitis	3	1	2	1	0.58
Surgical wound			5		
Decubitis ulcer				1	
Hydradenitis suppurativa		1			
Impetigo		1			
Pneumonia			1		
Septicemia			1		
Septicemia and osteomyelitis	1				
Urinary catheter			1		

[a] Statistical tests compare combined results for community-acquired infections with those for health care facility-acquired infections.

[b] Median ages (confidence intervals) of patients with community- and health care facility-acquired infections, 29 (24 to 45 years) and 67 years (43 to 28 years), respectively ($P = 0.0002$).

Statistical analysis. Categorical data were analyzed by comparing differences in proportions. Medians were compared using the Mann-Whitney rank sum test. The significance level was set at 0.05. Rank sum and confidence interval calculations were performed using Graphpad Prism, version 3.00 (GraphPad Software Inc., San Diego, Calif.) and C.I.A., version 1, 1989 (BMA Publishing, London, United Kingdom), respectively.

RESULTS

Thirty-five cases of infection due to GS-MRSA were identified in 35 patients. The majority of cases were community-acquired infections, and most of these occurred in Polynesians (Table 1). Six (26%) of 23 cases of community-acquired infection had risk factors for MRSA: one Polynesian patient worked in a hospital in a non-patient contact position, and two of her family household members were also patients in the study; another Polynesian patient was a domestic worker in a nursing home; the remaining two had previous hospital contact as patients. All resided in the laboratory's service area with the exception of one who lived in Sydney. The predominant types of infection were soft-tissue abscesses in community-acquired infections and surgical wound infection in hospital-acquired infections (Table 1). Three Caucasian patients died following hospital-acquired infection. One 87-year-old patient died of GS-MRSA septicemia with no primary focus identified. Two other patients (aged 86 and 56) died of other causes. Five cases of community-acquired infection occurred in two Polynesian families (two in one and three in the other).

Isolates were available for study in 21 of 23 community-acquired infections, 8 of 10 hospital-acquired infections, and 2 of 2 nursing home-acquired infections (P, 0.6). All isolates were positive for *nuc* and *mecA* gene products, and all produced β-lactamase. Resistance to other antimicrobials was rare

TABLE 2. Results of oxacillin resistance phenotyping, susceptibility testing, coagulase gene RFLP by PCR, PFGE, and phage typing

Isolate	Acquisition	Ethnicity	OR[b] phenotype	Susceptibility to[c]:					RFLP type	PFGE type	Phage type at 100 × RTD[d]	Experimental set
				FD	RD	TC	E	CIP				
F810539	Community	Caucasian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/79/80/3A/42E/47/53/54/77/81/95	56A
D817541	Community	Caucasian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/79/80/3A/3C/6/42E/47/53/54/77/84/81/94/95	56A
A830538	Community	Caucasian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/5A/79/80/3A/42E/47/53/54/77/81	56A
I825560	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	6/42E/47/53/54/77/81	MR12
B826559	Community	Polynesian	Heterogeneous	S	S	S	S	S	AII	A0	29/80/42E/47/53/54/75/77/81	56A/67R/M5/F33/F38
I816601	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/79/80/3A/6/42E/47/53/54/77/84/81/95	56A/MR12
H823537	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/79/80/3A/6/42E/47/53/54/77/81/95	MR12
G823530	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/80/3A/6/42E/47/53/54/77/81/95	47T/MR12
D828570	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/80/3A/3C/47/53/54/77/95	Not typeable
A803355	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/79/80/3A/42E/47/53/54/77/81/94	56A
A806533	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/79/80/3A/3C/42E/47/53/54/77/81/94	56A
E803543	CommunityR[a]	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	29/52/52A/80/3A/3C/6/42E/47/53/54/77/81/95	56A/1648/F38
E822547	CommunityR	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	42E/47/54/77/81	56A
F809718	CommunityR	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	6/42E/47/53/54/77/81	56A
F809715	CommunityR	Polynesian	Heterogeneous	S	S	S	S	S	AI	A0	6/42E/47/53/54/75/77/84/81	56A/67R/87M/F38
A823547	Community	Aboriginal	Heterogeneous	S	S	S	S	S	AI	A1	29/52/52A/79/80/3A/47/54/77/95	Not typeable
C810534	Community	Caucasian	Heterogeneous	S	S	S	S	R	AII	A1	29/52/52A/79/80/3A/42E/54/77	F38
G821561	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A1	29/52/52A/80/3A/42E/47/54/77/84/95	56A/MR12
D821552	CommunityR	Polynesian	Heterogeneous	S	S	S	S	S	AI	A2	29/52/52A/79/80/3A/3C/6/42E/47/53/54/77/81/94/95/96/88	56A/1648/F38
E802537	Community	Polynesian	Heterogeneous	S	S	S	S	S	AI	A3	29/52/52A/79/80/3A/47/53/54/77	Not typeable
I802552	Hospital	Polynesian	Heterogeneous	S	S	S	S	S	AI	A4	29/80/42E/54/81/95	MR12
E822485	Hospital	Caucasian	Homogeneous	S	S	S	R	R	D	B	52/77/83A/95	Not typeable
J710566	Nursing home	Caucasian	Heterogeneous	S	S	S	S	S	AIV	C	83A	M5/F30/F38
F829549	CommunityR	Caucasian	Homogeneous	S	S	S	R	S	C	D	6/47/53/54	56B/56C
C801535	Hospital	Caucasian	Homogeneous	S	S	S	S	S	C	D	29/52/52A/79/80/6/47/53/54/95	56B/56C/M3/M5/F33/F38
D828354	Hospital	Caucasian	Heterogeneous	R	S	R	S	S	BII	E	83A	Not typeable
B827549	Nursing home	Caucasian	Heterogeneous	R	S	R	S	S	BI	E	52A/42E/53/54/83A	MR25
I823541	Hospital	Caucasian	Homogeneous	S	S	R	R	S	AI	G2	83A/85/88	47T/90A/87M/MR12/MR25
E804531	Hospital	Caucasian	Heterogeneous	S	S	S	S	S	AIII	I	53/81	56A/67R/M3
E812560	Hospital	Caucasian	Homogeneous	S	S	R	R	S	AI	J	Not typeable	47T/56A/1648/F38/MR25
D808118	Hospital	Caucasian	Heterogeneous	R	S	S	S	S	BI	L	42E/47/53/54/83A/85	56B/56C/F38/MR25

[a] CommunityR, community acquired with risk factors.
[b] OR, oxacillin resistance.
[c] S, susceptible; R, resistant. FD, fusidic acid; RD, rifampin; TC, tetracycline; E, erythromycin; CIP, ciprofloxacin.
[d] RTD, routine test dilution.

in community-acquired isolates but was common in isolates acquired in a hospital or nursing home: two community-acquired isolates had single-agent resistance, while five hospital- or nursing home-acquired isolates had two-agent resistance and one had single-agent resistance (Table 2). Expression of oxacillin resistance was homogeneous in only one community-acquired isolate and in four hospital-acquired isolates (P, 0.01). All of these isolates were from Caucasian patients, and the patient with the community-acquired isolate had a risk factor for MRSA acquisition (Table 2). All local GR-MRSA strains tested expressed homogeneous resistance (Table 3).

Coagulase gene RFLP patterns of the 31 GS-MRSA isolates were divided into four types (A to D), with types A and B being further divided into four (I to IV) and two (I and II) closely related subtypes, respectively (Fig. 1 and Table 2). All but one of the community-acquired isolates fell into subtypes AI and AII, and conversely only three of the health care facility-acquired isolates belonged to subtype AI. Three additional subtypes for the control and WA-MRSA strains were described (Table 3).

The 31 study isolates were divided into nine pulsotypes (A to E, G, I, J, L) by PFGE (Fig. 2, Table 2). There were five closely related subtypes (A0 to A4) within type A. Isolates from all 16 Polynesian cases, the 1 aboriginal case, and 4 of the 14 Caucasian cases fell within type A. Pulsotype A subtypes accounted for all community-acquired isolates but one. The pulsotypes of all but one of the GS-MRSA isolates tested differed from those of the GR-MRSA isolates (Fig. 2, Table 3). GS-MRSA isolate I823541 belonged to pulsotype G2, which was related to two GR-MRSA isolates (Fig. 2).

Twenty of the community-acquired GS-MRSA isolates appeared to be closely related to the isolates of the WSPP as described by Heffernan et al. (11) (Table 2). Fifteen of these isolates were related to WSPP1, and five were related to WSPP2, the latter isolates showing lysis with phage 81 but none with phages 52, 52A, 3A, or 95. However, within the isolates related to WSPP1 and WSPP2 there were several distinct phage typing patterns. The only one of the community-acquired isolates which did appear not to be related to the WSPP strains of MRSA was F829549; this isolate also differed from other community-acquired isolates in PFGE pulsotype and coagulase RFLP. The hospital- and nursing home-acquired isolates are a varied group, all having different phage typing patterns. The GR-MRSA isolates with PFGE pulsotypes G and F had closely related or identical phage types (Table 3).

TABLE 3. Results of *nuc* and *mecA* PCR, oxacillin resistance expression, coagulase gene RFLP by PCR, PFGE, and phage typing for control and comparator strains

Description	Identification	Origin	Presence of:		OR[b] expression	RFLP type	PFGE type	Phage type	Experimental set
			nuc	*mecA*					
MSSA[c]	ATCC 29213	Type collection	Positive	Negative	NT[a]	AIII	NT	NT	NT
MRSA	ATCC 49476	Type collection	Positive	Positive	NT	AI	NT	NT	NT
WA-MRSA	B8-10	Pathcentre	Positive	Positive	NT	BIII	H	52A/80/6/42E/47/53/54/75/81/88	56B/56C/67R/87M/13M/M5/F30/F33/F38
WA-MRSA	B8-31	Pathcentre	Positive	Positive	NT	AV	K	54/95	Not typeable
GR-MRSA	K703484	Local	Positive	Positive	Homogeneous	AI	G1	88	(M5)/(F30)/(F38)/MR12/MR25
GR-MRSA	K704540	Local	Positive	Positive	Homogeneous	AI	F	83A/85/88	56B/56C/(67R)/87M/M3/F30/(F38)/MR12/MR25
GR-MRSA	K705613	Local	Positive	Positive	Homogeneous	AI	F2	83A/85/88	56B/56C/(67R)/87M/M3/F30/MR12/MR25
GR-MRSA	K711532	Local	Positive	Positive	Homogeneous	AI	F3	83A/85/88	(56A)/56B/56C/1648/67R/87M/M3/(M5)/F30/F38/MR12/MR25
GR-MRSA	K714372	Local	Positive	Positive	Homogeneous	AI	F1	83A/85/88	56B/56C/87M/M3/F30/F38/MR12/MR25
GR-MRSA	K722538	Local	Positive	Positive	Homogeneous	AI	G	88	87M/(M3)/F30/F38/MR12/MR25

[a] NT, not tested.
[b] OR, oxacillin resistance.
[c] MSSA, methicillin-sensitive *S. aureus*.

DISCUSSION

The classification of acquisition status in the study of community-acquired MRSA remains controversial. Previous studies have shown that contact with a health care institution in the 12 months prior to admission is the most common risk factor for MRSA carriage (21, 27). The need to document risk factors for MRSA infection and especially contact with health care institutions and not to rely on an arbitrary time-related definition when determining acquisition has been canvassed previously (3). One study in southern Texas dealt with this issue by performing a case control study comparing community-acquired MRSA and community-acquired methicillin-sensitive *S. aureus* infections (18). They found no significant difference when risk factors for MRSA within the preceding 6 months were compared. We have endeavored to overcome this difficulty by subdividing apparently community-acquired cases into those with and those without risk factors for MRSA acquisition. The presence of risk factors for MRSA in only 6 of 23

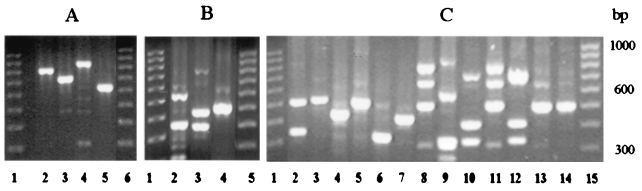

FIG. 1. Electrophoresis of PCR coagulase gene products and *Hae*III-digested products of representative strains. (A) Lanes 1 and 6, size markers; lanes 2 to 5, products A to D, respectively. (B) Lanes 1 and 5, size markers; lanes 2 to 4, RFLP patterns BIII, BII, and AI, respectively. (C) Lanes 1 and 15, size markers, lanes 2 to 14, RFLP patterns AIII, AI, AV, AI, BIV, AIV, AII, C, BI, AII, BI, AI, and AI, respectively.

cases of apparently community-acquired infection suggests that the majority were truly community acquired.

The PFGE results demonstrate that all of the isolates from Polynesians and all except one (F829549) of the other community-acquired isolates were closely or possibly related (pulsotype A). Both coagulase RFLP and phage typing results also support this conclusion. It is noteworthy that the one exception was isolated from a Caucasian patient with previous hospital contact. In addition, the only hospital-acquired Polynesian isolate was recovered from a postappendectomy wound. As the procedure was performed on the day of admission, it is likely that infection was caused by the patient's endogenous flora. WA-MRSA isolates have been shown to be distinct from GR-MRSA isolates endemic in eastern Australia (20). Results for the two WA-MRSA strains examined confirm this finding and demonstrate that they are unrelated to any of the other GS-MRSA strains studied. The hospital-acquired GR-MRSA isolates examined fell into two related groups, one of which appeared to be related to a hospital-acquired GS-MRSA isolate (I823541). Members of the other hospital-acquired GS-MRSA group were genotypically and phenotypically quite diverse, with the exception that the discrepant community-acquired isolate (F828549) appeared closely related to hospital-acquired isolate C801535.

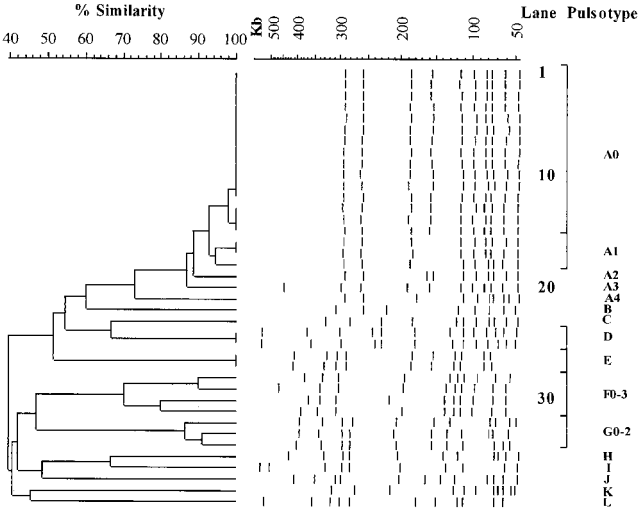

FIG. 2. Schematic representation of PFGE pulsotypes of 31 study isolates (lanes 1 to 31), 6 nosocomial GR MRSA isolates (lanes 31 to 37), and 2 WA-MRSA isolates (lanes 38 and 39), together with a dendrogram showing percent similarities of patterns and nomenclature of pulsotypes. Letters, pulsotypes (seven or greater band differences); numerals, subtypes (one to six band differences).

Phage typing results suggest that community-acquired GS-MRSA strains being isolated in southeast Queensland are related to SWP-MRSA strains reported in Auckland, New Zealand, where infections with these organisms are also predominantly community acquired and mainly seen in the Pacific Island patients (17, 24). There was substantial migration of New Zealanders (including Polynesians) to Australia in the 1980s and 1990s (*Australia Now—A Statistical Profile*, Australian Bureau of Statistics, Commonwealth of Australia, 2000 [http//www.abs.gov.au]). The predominantly Polynesian ethnicity of cases in southeast Queensland and the earlier appearance of these strains in Auckland supports the view that their introduction to Australia was from Polynesia via New Zealand. Confirmation by direct comparison of these geographically diverse strains is awaited.

The range and severity of infections caused by these GS-MRSA strains are in keeping with those reported previously (5). The appearance of these strains in the community and their potential for further spread are of public health importance. The prevalence of methicillin resistance in community-acquired *S. aureus* should be monitored, as a significant increase would necessitate changes to prescribing guidelines for community-acquired staphylococcal infections. The currently recommended first-line agents for common staphylococcal infections, isoxazolyl penicillins and cephalosporins (D. N. Gilbert, R. C. Moellering, and M. A. Sande (ed.), The Sanford guide to antimicrobial therapy, 30th ed., Antimicrobial Therapy Inc., Hyde Park, Vt.), will not be effective, and selection of alternative agents will be dependent on local susceptibility patterns.

Lack of resistance to the other antimicrobials tested was also quite uniform in the community-acquired isolates, with only one expressing resistance to ciprofloxacin. The phenotypic expression of resistance to oxacillin in pulsotype A was uniformly heterogeneous. Furthermore, the six hospital-acquired GR-MRSA isolates expressed resistance homogeneously, as did F829549, the genotypically unrelated community-acquired isolate, and four of eight hospital-acquired GS-MRSA isolates. Phenotypic expression of methicillin resistance in *S. aureus* has been shown to be stable (29), and reemergence of heterogeneous expression has also been noted in France with the reappearance of GS-MRSA since 1993 (8). The relationship of the heterogeneous phenotype to expression of gentamicin resistance is uncertain but may be related to genes other than *mecA* such as the regulatory genes *mecI* and *mecRI* and the *mec* promoter region (13, 35). Sequence analysis of the *mec* regulatory and promoter regions of GR-MRSA and GS-MRSA may provide an explanation.

ACKNOWLEDGMENTS

We thank the staffs of the Departments of Microbiology and Infection Control, Princess Alexandra Hospital, and of the Brisbane Southside Public Health Unit for their assistance in isolate and data collection and A. Morton for assistance in statistical analysis.

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