

## Laboratory and Epidemiology Communications

# Molecular Epidemiology of Methicillin-Resistant *Staphylococcus aureus* in a Community Hospital in Hiroshima

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Methicillin-resistant *Staphylococcus aureus* (MRSA) is a major nosocomial pathogen in hospitals, including community hospitals with relatively small number of beds (1). Restriction fragment length polymorphism analysis of genomic DNA using pulsed-field gel electrophoresis (PFGE) is a powerful tool for assessment of hospital infection controls (2).

The present study was conducted in a hospital in Hiroshima with two wards and 100 beds. In August 2000, MRSA was isolated from five patients' sputa in ward I and three patients' sputa and one patient's stool specimen in ward II. In December, MRSA was isolated from five patients' sputa and one patient's pus in ward I and two patients' sputa in ward II. At the time, a MRSA survey of the hospital personnel was performed and five carriers were detected, including a doctor and four nurses, among eight doctors and forty-three nurses. The isolates from the patients and the carriers were tested for chromosomal DNA typing by using a contour-clamped homogeneous electric field system (CHEF Mapper™, Bio-Rad Laboratories, Hercules, Calif., USA), enterotoxin serotyping (SET-RPLA, Denka Seiken Co., Tokyo), toxic shock syndrome toxin-1 (TSST-1) production (TST-RPLA, Denka Seiken), and coagulase serotyping (Denka Seiken).

Fifteen different PFGE patterns of *Sma*I DNA digests were detected (Fig. 1a). Band-based cluster analysis of these patterns (Molecular Analyst™, Bio-Rad) revealed five clusters A to E (Fig. 1b) (patterns sharing a similarity of 70% or higher were grouped into a cluster). Patterns B and C exhibited more than 60% similarity to patterns A and D. E shared less similarity with the others (Fig. 1b). MRSA isolates with PFGE pattern A (Nos. 395, 396, 397, 399, 400, 401, 402, 624, 625, 626, 627, 629, 633, 634, 635, 636, and 641), and isolates with patterns B (No. 623) and C (No. 630) produced enterotoxin type C, TSST-1, and coagulase type II (Table 1). Isolates with PFGE pattern D (Nos. 398 and 404) produced enterotoxin type B and coagulase type II, but did not produce TSST-1 (Table 1). An isolate with pattern E (No. 628) produced enterotoxin D and nontypable enterotoxin other than types A, B, C, or D (Table 1).

All of the five MRSA isolates from ward I in August were of cluster type A and producers of enterotoxin type C, TSST-1, and coagulase type II. Four of six isolates from the same ward in December were of the same type, and all the clinical

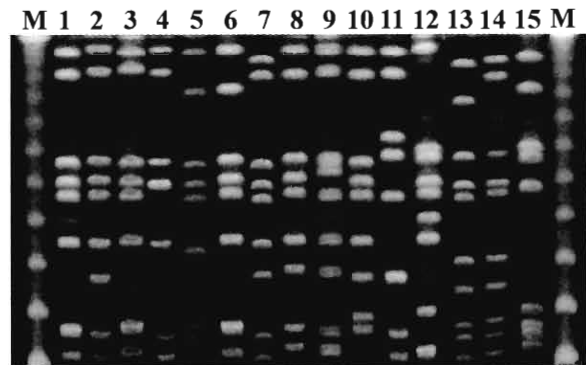


Fig. 1a. Pulsed-field gel electrophoresis of genomic DNA from MRSA isolates. Upper panel: *Sma*I-digested genomic DNA. Lane 1: MRSA isolate No. 395, lane 2: No. 397, lane 3: No. 635, lane 4: No. 401, lane 5: No. 399, lane 6: No. 627, lane 7: No. 396, lane 8: No. 636, lane 9: No. 629, lane 10: No. 634, lane 11: No. 623, lane 12: No. 630, lane 13: No. 398, lane 14: No. 404, lane 15: No. 628. M: low range PFGE Marker.

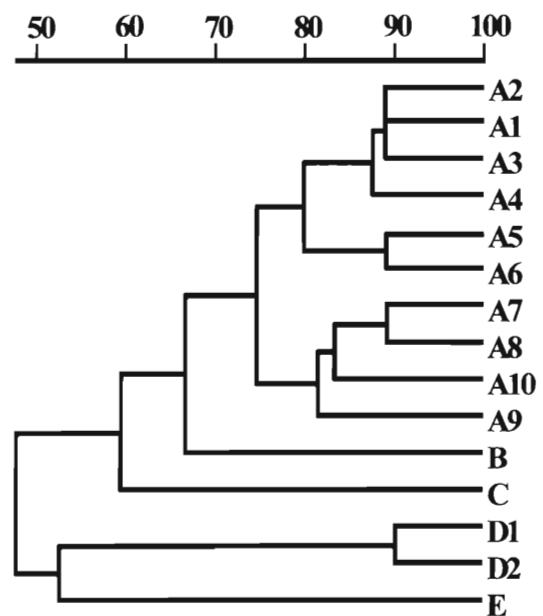


Fig. 1b. Cluster analysis of MRSA isolates based on PFGE patterns of *Sma*I-digested genomic DNA.

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Table 1. Phenotypic and genotypic characterization of the *S. aureus* isolates

Isolate month 2000	Ward	Isolate No.	Patient(P)/ Medical personnel(M)	Isolation part	PFGE pattern	Enterotoxin	TSST-1	Coagulase type
August	I	395	P1	sputum	A1	C	+	II
	I	399	P2	sputum	A5	C	+	II
	I	400	P3	sputum	A1	C	+	II
	I	401	P4	sputum	A4	C	+	II
	I	402	P5	sputum	A1	C	+	II
	II	396	P6	sputum	A7	C	+	II
	II	397	P7	feces	A2	C	+	II
	II	398	P8	sputum	D1	B	-	II
	II	404	P9	sputum	D2	B	-	II
December	I	623	P10	sputum	B	C	+	II
	I	624	P11	sputum	A1	C	+	II
	I	625	P12	sputum	A1	C	+	II
	I	626	P13	sputum	A1	C	+	II
	I	627	P14	sputum	A6	C	+	II
	I	628	P15	pus	E	D	-	NT
	II	629	P16	sputum	A9	C	+	II
	II	630	P17	sputum	C	C	+	II
	I	633	M1	nasal cavity	A1	C	+	II
	I	634	M2	nasal cavity	A10	C	+	II
	I	635	M3	nasal cavity	A3	C	+	II
	II	636	M4	nasal cavity	A8	C	+	II
	-	641	M5	nasal cavity	A1	C	+	II

NT: nontypable

personnel (M1-M5) carried MRSA of the same type. MRSA of the same type was isolated in ward II in August (Nos. 396 and 397) and December (No. 629). The above observation appears to suggest that the main MRSA infections during the observation period were initiated by a single MRSA strain which mainly spread in ward I through medical personnel or other vehicles.

#### REFERENCES

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