Community-based Nasal Screening Poorly Predicts pyl Carriage and Antimicrobial Resistance in Communityacquired S.aureus Infection

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Introduction

Since 2002-3, there has been an increase in PVL toxinproducing meticillin sensitive SA community acquired infections reported from a single major city in SW England (A). This study was to assess whether this was linked to higher nasal carriage rates in this population when compared to two other cities in the North of the region (B,C) which had not recorded a similar rise. At the same time the carriage of *mecA* and other antimicrobial resistances were also compared between the three centres and also with S.aureus isolates from infections occurring in the three communities.

Materials and methods

The study was approved by the local ethics committee. In 2009-10, nasal swabs were sent to a representative sample of adults living in 3 geographically distinct cities of SW England (A-C). Following an initial invitation, a selftaken swab kit was posted to the volunteers, who completed a short questionnaire and returned the swabs by post to a central laboratory. There was no follow up of subject who failed to respond or return the postal kits. On



receipt they were cultured for SA on both selective and not selective media and identities confirmed by a latex aggultination test. These strains, along with SA from superficial infections in each communitiy, were tested for mecA and pvl by multiplex PCR; other susceptibilities were performed by BSAC standardised disc test.

Location of the three Cities in SW England

Results

1952 nose swabs were received from residents of the 3 cities (Figure): 553 vielding SA (28.3%), 13 mecA (0.7%) and 6 pvl (0.3%) with no statistically significant association to geographic locality (Table 1, Chi-squared test). In contrast, 578 SA isolates from infection yielded 47 mecA (8.1%) & 26 pvl (4.5%) with pvl carriage strongly associated with City A (Table 2, Chi-squared test).

Figure: Volunteers recruited into the study

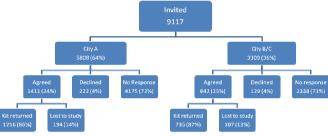


Table 1: Isolation of S.aureus, along with mecA and pvl carriage, for nasal swabs received from the volunteers.

	City A	City B	City C	Р	
Nasal Swabs	1,216	407	328	N/A	
S.aureus	360 (29.6%)	109 (26.8%)	83 (25.3%)	0.221	
mecA	7 (0.6%)	1 (0.2%)	4 (1.2%)	N/A	
pvl	4 (0.3%)	2 (0.5%)	0 (0.0%)	N/A	

Resistance was not associated (other than methicillin) with geographic locality but, after correction for mecA carriage, was associated with nasal carriage v infection for erythromycin, fusidic acid and ciprofloxacin, but not mupirocin, clindamycin or tetracycline (Fisher's exact test).



Table 2: The prevalence of mecA and pvl genes in S.aureus isolated from clinical samples taken by community-based physicians.

	(Р*		
	City A	City B	City C	
Ν	255	151	162	
Mec A	15 (5.7%)	13 (8.6%)	19 (11.7%)	0.106
Pvl	19 (7.2%)	1 (0.6%)	6 (3.7%)	0.007

Table 3: Antimicrobial resistance and pvl carriage rates (%) in meticillin sensitive S.aureus from nasal swabs or clinical isolates.

	Nasal Isolates	Clinical Isolates	Р
Ν	541	531	-
Erythromycin	2.6	10.2	<0.0001
Mupirocin	0.0	0.8	0.06
Fusidic Acid	7.2	14.1	0.0002
Clindamycin	0.0	0.8	0.06
Ciprofloxacin	0.7	5.6	<0.0001
Tetracycline	1.5	1.5	0.81
Pvl carriage	1.0	4.5	0.0006

Conclusions

Community-acquired infection with PVL producing SA or MRSA is uncommon in SW England but occurs with a strong geographic association. In contrast, no geographic associations were seen in nasal carriage of these organisms. Rates of resistance in SA from nasal screening were lower than in SA from community-acquired infections. We conclude that community-based nasal screening poorly predicts *pvl* carriage and antimicrobial resistance in community-acquired infections. We advise caution in the use of population screening to make estimates of the prevalence of antimicrobial resistance or pvl carriage in clinical infection.

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