Panton-Valentine Leukocidin-Positive *Staphylococcus aureus* and Foreign Travel

We noted with interest the study by Rossney et al. (3) describing genotypic diversity among Panton-Valentine leukocidin-positive (PVL+) methicillin-resistant *Staphylococcus aureus* (MRSA) in Ireland, where they suggested foreign importation of the strains as one of the reasons for this diversity. In support of this, we have observed the last few years a number of patients with PVL-associated folliculitis and cutaneous abscesses, caused by both methicillin-sensitive *S. aureus* (MSSA) and MRSA, where the infection began during or shortly after foreign travel. This case series illustrates what is potentially a major route of transmission for strains of PVL+. *S. aureus* between countries and continents.

Patient 1, a 44-year-old male, developed multiple cutaneous abscesses and cellulitis a few days after returning from a trip to Jamaica. A PVL+ MRSA strain (sequence type 8 [ST8]) was isolated from the abscesses. Patient 2, the 44-year-old wife of patient 1, also traveled to Jamaica and developed recurrent folliculitis 5 weeks after her husband, with the same strain of MRSA isolated.

Patient 3, a 27-year-old male, had recurrent folliculitis for 7 months, the onset of which occurred while visiting Fiji. A PVL+ MSSA strain (ST30) was isolated from a lesion. The patient’s wife and a friend subsequently developed folliculitis but were not seen by us. Another friend of patient 3 was patient’s wife and a friend subsequently developed folliculitis during the same trip but was not seen by us.

Patient 4, a 25-year-old male, who presented almost 2 years after patient 3 with a cavitating pneumonia following the rupture of a perineal abscess. A PVL+ MSSA strain (ST30) was isolated from both abscess and blood cultures. This patient had no preceding foreign travel but may have acquired the infection from patient 3, as the isolates were genotypically identical.

Patient 5, a 21-year-old female, had frequent recurrent folliculitis for almost 3 years which began during travel to Africa (Tanzania, Zambia, and Malawi). A PVL+ MSSA strain (ST30) was isolated from a lesion.

Patient 6, a 35-year-old male, had recurrent cutaneous abscesses for 7 months following his return from worldwide travel (South America, Southeast Asia, and Australia). A PVL+ MSSA strain (ST22) was isolated from a groin swab.

Patient 7, a 24-year-old human immunodeficiency virus-positive female with a high CD4 count, >500, developed bilateral axillary abscesses a few days after returning from a trip to Zambia. A PVL+ MSSA strain (ST22) was isolated from the abscesses.

Patient 8, a 16-year-old male, developed recurrent folliculitis during a school trip to Fiji. A PVL+ MSSA strain (ST123) was isolated from a lesion.

Patient 9, a 43-year-old male, had recurrent folliculitis following travel to Thailand. A PVL+ MSSA strain (ST123) was isolated from a lesion.

Patient 10, a 16-year-old female, developed deep cutaneous abscesses on her back during travel to Bangladesh, with ongoing folliculitis following her return. A PVL+ MSSA strain (ST123) was isolated from the abscess. Her cousin also developed folliculitis during the same trip but was not seen by us, and no specimens were taken from her.

Patient 11, a 20-year-old female, had recurrent cutaneous abscesses, often requiring incision and drainage, for almost 2 years following travel to Tunisia. A PVL+ MSSA strain (ST1) was isolated from a labial abscess.

Patient 12, a 52-year-old female with type II diabetes, developed left lower leg cellulitis following folliculitis, which was noticed the day after arriving in the United Kingdom from Sudan, where she lived. A PVL+ MSSA strain (ST25) was isolated from the cellulitic area.

Patient 13, a 59-year-old male, developed widespread folliculitis while on holiday in Mallorca. He was admitted to the hospital on his return with olecranon bursitis and sepsis. A PVL+ MRSA strain (ST80) was isolated from the left olecranon bursa and from blood cultures. Patient 14, the 52-year-old partner of patient 13, developed a cutaneous abscess on her back after the same holiday, with an identical PVL+ MRSA strain (ST80) isolated from the lesion.

Genotyping was done by pulsed-field gel electrophoresis (PFGE), and multilocus ST results are inferred from the types of strains shown by PFGE to be closely related. PVL testing was done by PCR detection of the genes encoding the PVL toxin.

This case series lends support to the theory that international travel is contributing to the global spread of PVL-positive *S. aureus* (4). Especially, cases 1 to 4 describe scenarios where a PVL+ strain of *S. aureus* was potentially acquired abroad, with subsequent transmission of the infection on returning home. Although trips abroad are common nowadays, it is striking how many of the patients we have seen acquired their infections during or immediately after foreign travel. It is not clear why travelers to foreign countries may be at risk of acquiring PVL-associated staphylococcal infections. They may be exposed to new strains of *S. aureus* to which they may have little or no immunity. They may also be taking antimarial (e.g., doxycycline) or antibiotics with antibacterial efficacy. Previous antibiotic use is implicated in the acquisition of MRSA carriage (2) and has been associated with PVL staphylococcal infections (1). It is possible that other unidentified factors associated with foreign travel could predispose individuals to become colonized with new strains of *S. aureus* or even cause those who are already colonized with PVL-positive *S. aureus* to develop active infection. We propose that any study looking at the epidemiology of PVL-positive *S. aureus* should include foreign travel as a potential risk factor for the acquisition of PVL-positive *S. aureus* carriage and infection.

REFERENCES


3. Rossney, A. S., A. C. Shore, P. M. Morgan, M. M. Fitzgibbon, B. O’Connell, and D. C. Coleman. 2007. The emergence and importation of diverse genotypes of methicillin-resistant *Staphylococcus aureus* (MRSA) harboring the Panton-Valentine leukocidin gene (pvl) reveal that pvl is a poor marker for


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