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CASE REPORT

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''More than skin deep'': Recurrent primary hand abscesses in a warehouse operative

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ABSTRACT

A 25-year-old male right-hand dominant warehouse operator presented with two hand infections within 12 weeks both requiring surgical drainage and antimicrobial therapy. Subsequent testing confirmed Panton-Valentine leukocidin-positive *Staphylococcus aureus* (PVL-SA). This case highlights the need for prompt multidisciplinary management of hand infections to consider, diagnose and manage atypical infections.

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KEYWORDS
Hand abscess; atypical infection; PVL-SA

Introduction

Prompt multidisciplinary management of hand infections is essential to prevent stiffness, loss of function and long-term disability. Although hand infections are relatively common in young patients particularly males engaged in sports, recurrent hand infections are not. *Staphylococcus aureus* is a commensal skin organism. Panton-Valentine leukocidin (PVL) is a toxin produced by some strains of *S. aureus* and usually implicated in skin and soft tissue infections. It can also cause invasive infections, in particular haemorrhagic pneumonia.

To date, there is only one previous case describing recurrent hand infections associated with Panton-Valentine leukocidin-positive *Staphylococcus aureus* (PVL-SA). This case highlights the need for multidisciplinary management of these patients as the diagnosis of PVL-SA may have been missed with potentially devastating consequences.

Case report

A 25-year-old right-hand dominant male warehouse operative presented to the Emergency Department (ED) with a three-day history of redness, pain and swelling in the right palm. He removed a wooden splinter from the palmar aspect of the metacarpophalangeal joint of his right middle finger 24 h before the symptom onset. In his past medical history, he had childhood-onset eczema and recalled always having "cracked hands" (Figure 1). He was not currently managing his eczema with medical therapy but described several flare-ups of his eczema since childhood.

He had no relevant family history, specifically no dermatological conditions. There was no recent history of skin and soft tissue infection in his household contacts. He was on noregular medications, had no allergies and smoked 10 cigarettes a day. On examination, his temperature was 37.5°C and he had tenderness, swelling and overlying erythema localised to his palm and extending to the proximal interphalangeal joint of his middle finger. Admission blood tests showed a serum white cell count of 10.71×10^9/L (reference range $3.5–10.5 \times 10^9$/L) and C-reactive protein of 14.1 mg/L (reference range 0–10 mg/L). Serum haemoglobin and electrolytes were within normal range. There was no radiological evidence of a foreign body or osteomyelitis. A right palmar abscess was suspected. At operation 10 mls of subcutaneous pus was drained and sent for microbiological analysis. Specimens cultured methicillin susceptible *S. aureus* (MSSA) for which he completed a 14-day course of intravenous flucloxacillin 1 g QDS. Following surgical drainage the wound was left open and dressed daily with bethidine wicks.

Twelve weeks later he represented to his general practitioner (GP) with a four-day history of increasing pain, redness and swelling over his dorsal left wrist. There was no history of trauma on this occasion. He was referred to the ED 48 h later when he failed to respond to oral flucloxacillin. On examination he had a 3×3 cm

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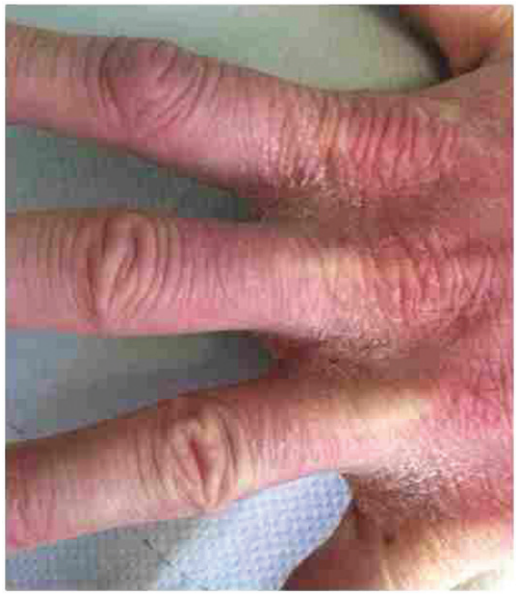

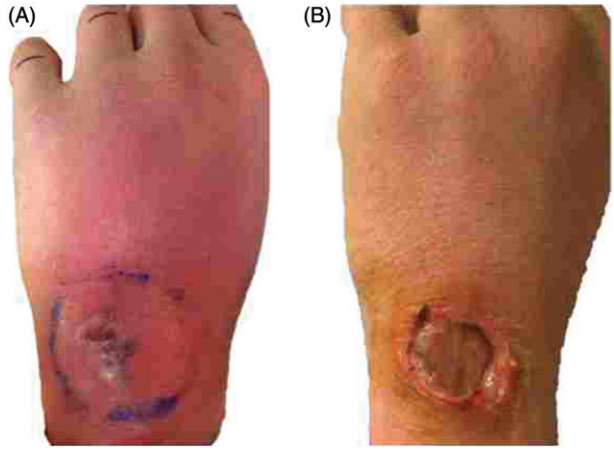

fluctuant swelling over his dorsal wrist with surrounding cellulitis (Figure 2A). Admission bloods showed serum white cell count of 13.86×10^9 g/L and C-reactive protein of 57.8 mg/L.

Further investigations were requested in the context of a recurrent infection in a healthy, young patient. These included an oral glucose tolerance test, a Mantoux test, serum HIV antibodies, immunoglobulin levels, complement levels, antibodies to atypical infections and a transesophageal echocardiogram.

Similar to his previous admission, he required incision and drainage of the abscess followed by daily bethidine wick dressings (Figure 2B). On this occasion pus was sent to the National Methicillin Resistant *S. aureus* (MRSA) Reference Laboratory and was reported positive for PVL-SA. The patient completed a 10-day course of intravenous flucloxacillin 1 g QDS with five days of clindamycin 600 mg qds for anti-toxin cover. A decolonization regimen of five days 4% chlorhexidine wash and intranasal mupirocin was prescribed once his wounds had fully healed. Dermatology advice to optimise his skin integrity was sought. He was advised not to share bedding or towels and the same decolonisation regimen was advised for his household contacts.

He was discharged following completion of antibiotic therapy and followed-up in the outpatient clinic (Figure 3). His wound healed by secondary intention. There have been no further abscesses at a 10-month follow-up period.

Discussion

PVL is a cytotoxin produced by 5% of *Staphylococci* (both MSSA and MRSA).[1] It primarily occurs sporadically in young healthy patients and as a consequence can be missed. PVL causes leukocyte destruction and tissue necrosis.[2] Although, predominately detected in isolates causing skin and soft tissue infections, PVL-SA strains can become invasive leading to necrotising fasciitis, purpura fulminans and necrotising pneumonia with a high mortality rate; up to 75% in necrotising pneumonia despite medical treatment.[3] PVL-SA infections are thought to be more highly transmissible than PVL-negative infections.[4] Healthcare workers, contact sports players and closed communities are considered as high-risk groups for transmission.[5]

PVL-SA infections have been described since the 1930s, but within the last decade their incidence in the community is rapidly increasing.[6] Although surveillance and improved case recognition may have contributed, this rapid increase is of growing international concern. In the United States, 36% of all *S. aureus* infections from 2004 to 2008 were PVL positive. MRSA isolates increased from 24% to 54% and MSSA isolates overall increased from 9% to 12%.[7] In Ireland, an increased prevalence of PVL-positive MRSA (from 0.2% to 8.8%) and a decrease in PVL-positive MSSA (from 20% to 2.5%) from 2004 to 2011 have been reported.[8] This contrasts to UK where PVL-positive MRSA infections are rare (0.8% of all isolates) but PVL-positive MSSA are more common (9.0% of all specimens).[9] MSSA isolates are not routinely referred to the reference laboratory for analysis in Ireland, which may partially explain these discrepancies.

To date, one case of recurrent PVL-SA hand abscesses has been reported in the literature. In 2013, Wearn et al. described a PVL-SA right palmar abscess in a healthy 24-year-old who developed a second abscess six weeks later in the contralateral palm.[10] Although PVL-positive MRSA was identified, the age, immune status and clinical

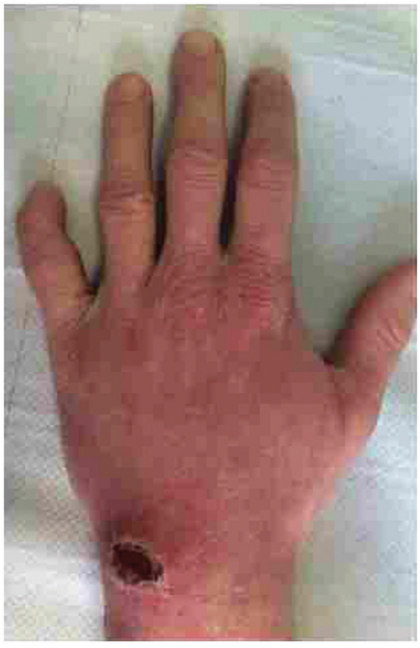

Figure 3. Wound left dorsal wrist at three weeks post-incision and drainage.

course were comparable to this case. Similar to Wearn et al., we consider our patient's second presentation to be a recurrence of PVL-SA. Following the initial infection he likely remained colonised with PVL-SA and his eczematous skin then facilitated further entry of PVL-SA with development of the second abscess. In addition to appropriate surgical and antibiotic management of this infection, management of his underlying skin condition, patient education regarding appropriate hygiene and decolonisation of the patient and his contacts facilitated prevention of a further recurrence.

PVL-SA testing is not routinely available in routine diagnostic laboratories. Clinical suspicion of PVL production is key and if suspected, advice should be sought from the clinical microbiological service regarding patient management and referral of positive specimens to a reference laboratory for PVL testing. The recommended management of PVL-SA infection includes treatment of infection (abscess drainage and antibiotic therapy), decolonisation of the index patient, increased individual (covering wounds and hand hygiene) and environmental hygiene and appropriate management of close contacts. The main strategy to prevent reinfection with or transmission of PVL-SA is stringent hygiene combined with decolonisation treatment.[11] In this case guidance from the Centers for Disease Control and Prevention was carefully considered. It refers to the "five Cs" of risk factors for PVL-related infection: contaminated items, close contact, crowding, cleanliness, and cuts and compromised skin integrity.[5] Particular emphasis was placed on the management of our patient's eczema to optimise skin integrity. Health Protection Agency (HPA) guidelines recommend that all patients and their close contacts undergo decolonisation (chlorhexidine wash 4% with intranasal mupirocin), although internationally there is no consensus on this approach. This relates to individual countries' testing policies for the PVL toxin. In the UK where cases are considered rare and severe, testing is recommended as it is an aggressive approach to treatment including decolonisation. Despite these published guidelines, risk factors and recommendations to prevent recurrence have not yet been proven in the literature.

Conclusion

Recurrent primary hand abscesses in the young, healthy patient are uncommon and should raise suspicion of an underlying condition, atypical infection or PVL-SA. Prompt multidisciplinary management of these infections is needed to prevent stiffness, loss of function and long-term disability.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

References

[1] Lina G, Piémont Y, Godail-Gamot F, et al. Involvement of Panton-Valentine leukocidin-producing *Staphylococcus aureus* in primary skin infections and pneumonia. Clin Infect Dis. 1999;29:1128–1132.

[2] Genestier AL, Michalete MC, Prévoset G, et al. *Staphylococcus aureus* Panton-Valentine leukocidin directly targets mitochondria and induces Bax-independent apoptosis of human neutrophils. J Clin Invest. 2005;115:3117–127.

[3] Gillet Y, Issartel B, Vanhems P, et al. Association between *Staphylococcus aureus* strains carrying gene for Panton-Valentine leukocidin and highly lethal necrotising pneumonia in young immunocompetent patients. Lancet. 2002;359:753–759.

[4] Health Protection Agency. Guidance on the diagnosis and management of PVL-associated *Staphylococcus aur*eus infections (PVL-SA) in the UK 2008. Available from: http://webarchive.nationalarchives.gov.uk/20140714084352/http://hpa.org.uk/webc/hpawebfile/hpaweb_c/1218699411960 (Last accessed November 29 2015).

[5] Diagnosis and management of PVL-Staphlococcus aureus infection quick reference guide for primary care. Available from: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/391168/PVL_guidance_in_primary_care_quick_reference_guide.pdf. (Last accessed November 29 2015)

[6] Panton PN, Valentine FCO. Staphylococcal toxin. Lancet. 1932;1:506–508.
[7] Brown ML, O Hara FP, Close NM, et al. Prevalence and sequence variation of Panton-Valentine leukocidin in methicillin-resistant and methicillin-susceptible *Staphylococcus aureus* strains in the United States. J Clin Microbiol. 2012;50:86–90.
[8] Shore AC, Tecklenborg SC, Brennan GI, et al. Panton-Valentine leukocidin-positive *Staphylococcus aureus* in Ireland from 2002 to 2011: 21 clones, frequent importation of clones, temporal shifts of predominant methicillin-resistant *S. aureus* clones, and increasing multiresistance. J Clin Microbiol. 2014;52:859–70.
[9] Shallcross LJ, Williams K, Hopkins S, et al. Panton-Valentine leukocidin associated staphylococcal disease: a cross-sectional study at a London hospital, England. Clin Microbiol Infect. 2010;16:1644–1648.
[10] Wearn CM, Breuning EE, Chester DL. Recurrent primary abscesses in the hand: consider Panton-Valentine leukocidin-positive *Staphylococcus aureus*. J Hand Surg Eur Vol. 2015;40:314–315.
[11] Shallcross LJ, Mbeledogu CN, Hayward AC. Should we screen and decolonise contacts of patients with Panton Valentine leukocidin associated *Staphylococcus aureus* infection? BMJ. 2011;343:d5479.