CASE REPORT

Back pain in a previously healthy teenager

Felicity Fitzgerald,¹ Julia Howard,² Fiona Bailey,¹ Sorrush Soleimanian²¹Department of Paediatrics, North Middlesex University Hospital NHS Trust, London, UK²Department of Microbiology, North Middlesex University Hospital, London, UK**Correspondence to**Dr Felicity Fitzgerald,
Flic Fitzgerald@hotmail.com**SUMMARY**

We present the case of a 14-year-old previously healthy boy who presented to his general practitioner with back pain and fever after rugby training. He was initially treated for suspected discitis but during the course of his admission he rapidly deteriorated and developed severe necrotising pneumonia. He was intubated, ventilated and transferred to a paediatric intensive care unit. Panton-Valentine leukocidin *Staphylococcus aureus* was suspected and subsequently identified in blood cultures.

BACKGROUND

Panton-Valentine leukocidin (PVL) *Staphylococcus aureus* is associated with abscesses, musculoskeletal infections and life-threatening necrotising pneumonia. Necrotising pneumonia can evolve rapidly with a mortality rate of over 60% without prompt diagnosis and aggressive management.¹ Rates of PVL *S aureus* in the UK appear to be increasing.² This case highlights the need for a high degree of suspicion and vigilance when treating adolescents with possible infection with PVL-carrying *S aureus*.

CASE PRESENTATION

A 14-year-old previously healthy boy presented to his general practitioner with a 24-h history of fever and back pain after a warm up session of rugby training. He was referred to the paediatric team for assessment. Clinical examination revealed a temperature of 38.5°C, heart rate 107 bpm, respiratory rate 20/min, oxygen saturations 97% in air, capillary refill <2 s. He was well perfused with a normal cardiovascular and respiratory examination, with some pain on deep palpation of the lower abdomen. Examination of the back revealed slight erythema with tenderness over the sacrum and lower lumbar region and pain on straight leg flexion. He was admitted and on microbiological advice was started on intravenous flucloxacillin and clindamycin with stat dose of gentamicin for suspected discitis caused by PVL *S aureus*. His observations overnight were within normal limits. The next morning his oxygen saturations were found to be 56% in air. In view of his chest symptoms antibiotics were changed to ceftriaxone, clindamycin and clarithromycin. However, he continued to deteriorate over the course of the day with increasing respiratory distress with haemoptysis despite maximal oxygen therapy. Linezolid was added to his antibiotic regimen and intravenous immunoglobulin was given due to the assumption of necrotising pneumonia caused by PVL methicillin-sensitive *S aureus* (MSSA) or methicillin-resistant *S aureus* (MRSA). He was intubated, ventilated and transferred to a paediatric

intensive care unit. He was artificially ventilated for 5 days.

INVESTIGATIONS

Blood tests are documented in table 1. Initial blood tests revealed a mild leucopenia and thrombocytopenia with a raised C reactive protein. These deteriorated the following day with associated hyponatraemia and transaminitis. Serial blood gases (table 1) correlated poorly with the patient's clinical state. Chest radiographs showed bilateral interstitial infiltrates which developed into an image consistent with acute respiratory distress syndrome. Blood cultures showed Gram-positive cocci in clusters which were identified as *S aureus* sensitive to flucloxacillin and clindamycin. The isolate was sent to the Staphylococcal Reference Unit at the Health Protection Agency where it was confirmed that the isolate was carrying the PVL gene, using PCR. In view of his clinical status his MRI was delayed until after he was extubated and at that point an MRI of his spine had appearances consistent with a previous/resolving discitis with an epidural abscess at the level of L3–L4.

OUTCOME AND FOLLOW-UP

The patient was artificially ventilated for 5 days. He made a good recovery over time and was discharged from PICU after 7 days. Following his discharge he was treated as an outpatient with intravenous ceftriaxone via a percutaneously inserted intravenous catheter and oral clindamycin for 3 months. At the time of writing this case report he had made a complete recovery.

DISCUSSION

There are a number of features of this case which led to the early suspicion of invasive PVL *S aureus* disease. The patient was previously a healthy child with a history of contact sports along with symptoms and signs suggestive of discitis. In conjunction with the high fever, mild leucopenia and thrombocytopenia this led to prompt treatment through timely consultation with microbiology colleagues. *S aureus* strains producing PVL toxin make up less than 2% of *S aureus* samples submitted to the national reference laboratory but they have been associated with sudden death, overwhelming sepsis and fulminant necrotising pneumonia in previously well immunocompetent children and adolescents.^{1–3} Mortality rates of 56–63% have been observed, particularly associated with acute respiratory syndrome.¹ The onset of haemoptysis as reported in this case is a key symptom which should alert physicians to the possibility of invasive PVL *S aureus* disease, along with leucopenia and

To cite: Fitzgerald F, Howard J, Bailey F, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-200139

Table 1 Serial blood test results

	Venous (on presentation)	Capillary (at 20 h)	Venous (24 h)	Arterial (26 h)	Capillary (28 h)
Haemoglobin (g/dL)	13.3				
White cell count ($\times 10^9/L$)	3.3		2.9		
Platelets ($\times 10^9/L$)	91		79		
C reactive protein (mg/L)	176		207		
Sodium (mmol/L)	129		130		
Aspartate transaminase (u/L)	165		229		
pH		7.39	7.34	7.41	7.36
pO ₂ (kPa)		8.56	4.29	10.2	5.4
pCO ₂ (kPa)		5.44	6.53	5.21	6.32
HCO ₃ (mmol/L)		24.7	26.2		26
Lac (mmol/L)		2.4	2.2	1.8	2.8

thrombocytopenia again seen in this case. Those with necrotising pneumonia often report a prior 'flu-like' illness.³

PVL is more commonly associated with skin and soft tissue infections than invasive disease. Factors that suggest PVL-associated *S aureus* include recurrent abscesses or furunculosis as well as necrotising skin or soft-tissue infection.³ PVL is a necrotising, pore forming cytotoxin that targets human leucocytes. In 2010 there were 2227 cases of PVL-producing *S aureus* (PVL-SA) identified in the UK, down from a peak of 2557 in 2009, although it is not clear if this decrease is due to less frequent centralised testing and more local identification.² The initial focus may be a bone, joint or skin infection: the presence of PVL toxin is significantly associated with systemic sepsis compared with PVL-negative *S aureus* bone and joint infections.⁴

In the USA, the Centre for Disease Control and Prevention (CDC) has drawn up guidance referring to risk factors for PVL-related infection as '5 C's': contaminated items, close contact, crowding, cleanliness, cuts and other compromised skin integrity.³ There should be high suspicion of PVL-SA infection in cases of recurrent furunculosis or abscesses within a household or social group, or in the case as here of an invasive pneumonia in a previously healthy child or adolescent. Haemoptysis and a marked inflammatory response (raised C reactive protein) with associated leucopenia should also raise suspicion.

In terms of antimicrobial therapy PVL-producing strains are often MRSA rather than MSSA.² Therefore, it is prudent to use antibiotics effective against MRSA strains, although this is dependent on the background prevalence of MRSA in the community. Additionally, antibiotics such as linezolid and clindamycin have been shown to decrease production of PVL in vitro.⁵ Long duration antibiotic therapy is usually needed, especially if infection is complicated by seeding to other foci: osteomyelitis may require repeated surgical debridement.

In terms of public health implications, cases of PVL *S aureus* should be notified to the local Health Protection Unit for risk assessment and consideration of screening and/or decolonisation of close contacts.³

Learning points

- ▶ Panton-Valentine leukocidin (PVL) *Staphylococcus aureus* necrotising pneumonia is rapidly evolving and frequently fatal.
- ▶ Awareness of PVL *S aureus* pneumonia needs to be raised among adult physicians and paediatricians in district general hospital settings.
- ▶ Appropriate antimicrobial therapy and prompt transfer to an intensive care setting is vital.
- ▶ Consideration of screening and decolonisation of contacts should be undertaken in conjunction with the local health protection unit.

Acknowledgements The authors are grateful for additional advice and support from Dr Lesley Alsford.

Contributors The paper was conceived by FF. FF, FB and JH were directly involved in patient care under the supervision of SS. The paper was drafted by FF, JH and FB with contributions by SS. All authors read and approved the final draft of the paper.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Gillet Y, Vanhems P, Lina G, et al. Factors predicting mortality in necrotizing community-acquired pneumonia caused by *Staphylococcus aureus* containing Panton-Valentine leukocidin. *Clin Infect Dis* 2007;45:315–21.
- 2 HPA. *PVL–Staphylococcus aureus* infections: an update. London: HP Agency, 2011.
- 3 HPA. *Guidance on the diagnosis and management of PVL-associated Staphylococcus aureus* (PVL-SA) in England. 2nd edn. HP Agency, 2008.
- 4 Dohin B, Gillet Y, Kohler R, et al. Pediatric bone and joint infections caused by Panton-Valentine leukocidin-positive *Staphylococcus aureus*. *Pediatr Infect Dis J* 2007;26:1042–8.
- 5 Dumitrescu O, Boisset S, Badiou C, et al. Effect of antibiotics on *Staphylococcus aureus* producing Panton-Valentine leukocidin. *Antimicrob Agents Chemother* 2007;51:1515–19.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow