Staphylococcus Aureus - A Case Report

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ABSTRACT

Skin and soft tissue infections secondary to staphylococcus aureus are seen commonly in primary care settings. They are usually treated empirically based on history and clinical judgement with a high rate of success. With growing concerns about antibiotic resistance and emerging virulence factors relating to this common bacterium, it is pertinent to consider a more methodical approach to treating apparently common skin infections. The evolution of microbes and increasing use of antibiotics are creating therapeutic challenges for physicians worldwide. Skin swabs are a quick and easy tool accessible to many primary care physicians to gain insight into bacterial presence and antibiotic sensitivities to guide appropriate management. This reduces this risk of avoidable complications and possible sequelae of disease. Panton-Valentine Leukocidin is a staphylococcus aureus specific toxin which can be detected through skin swabs for culture. If left untreated it has the potential for devastating complications including invasive infections such as haemorrhagic pneumonia, necrotising fasciitis, osteomyelitis, purpura fulminans, even death.

KEYWORDS – staphylococcus, infection, skin, antibiotic
INTRODUCTION

Staphylococci are a common type of bacteria that live on the skin and mucous membranes of humans, with Staphylococcus aureus being the most important type of this bacteria in human disease (1)

It has an estimated incidence rate of 20 to 50 cases/100,000 population per year, with 10-30% of these patients dying as result of infection and infection-related complications (2). This confers a significant burden of disease, demanding appropriate recognition and treatment.

One of the key features determining optimal treatment for staphylococcal infections is understanding susceptibility to antibiotics dating back to the early use and discovery of penicillin. In 1929, Alexander Fleming published his crude findings of the discovery of penicillin in the British Journal of Experimental Pathology.
however it was to be refined over the following years. It was not until 1941 that the first patient with a suspected staphylococcal infection was treated successfully with penicillin (3). In the following years, methicillin, a semi-synthetic penicillin antibiotic ‘used principally in the treatment of severe, penicillin-resistant staphylococci infection’ was developed to combat the growing resistance to the original penicillin structure (4). However, by the 1960s, methicillin-resistance began to emerge, further complicating management options (5). This created the commonly recognized classification of methicillin-sensitive staphylococcus aureus (MSSA) and methicillin-resistant staphylococcus aureus (MRSA). In the following years, leading to the present day, variations of antibiotic resistance continue to grow, causing significant concern for the international community. The World Health Organisation (WHO) has published detailed factsheets on ‘Antimicrobial Resistance’ (AMR) and, more specifically, ‘Antibiotic Resistance’. They report that ‘antibiotic resistance is one of the biggest threats to global health, food security and development today…leading to higher medical costs, prolonged hospital stays and increased mortality’ (6). It is also reported that ‘resistance to first-line drugs to treat infections caused by staphylococcus aureus…is widespread (7).

There is continued research into possible virulence factors for staphylococcal infections to help guide future management. One such finding suggests the importance of the presence of Panton-Valentine Leukocidin (PVL).

PVL is a toxin produced by strains of staphylococcus aureus, which ‘can cause either neutrophil lysis or apoptosis and contributes to tissue necrosis’ (8), resulting in skin and soft tissue infections, but also more invasive infections such as haemorrhagic pneumonia, necrotising fasciitis, osteomyelitis and purpura fulminans (9). There is evidence to suggest complications from more invasive infections such as haemorrhagic pneumonia can result in death (10). Data suggests an estimated increase in the number of PVL positive cases, from 2% in 2005 to 20% in 2010, in hospital and community settings (11). It is seen in methicillin-resistant and sensitive strains of S. aureus, affecting subsequent management. This dictates the importance of skin swabs for culture – ensuring swabbing of affected skin and anterior nares (12), with resultant sensitivities guiding treatment. The severity of complications emphasise the importance of early detection followed by appropriate treatment.
Soft tissue and skin infections are commonly seen by all family practitioners in their daily work. A swift clinical diagnosis is usually made, with little need for follow up in most cases. However, there is the odd occasion where we see the same patient attending for the same problem, again and again with little response to our suggested treatment. This requires possible consideration of a wider differential with a more methodical approach to reach a definitive diagnosis, guiding subsequent management.

This case looks at the topics we have touched upon and the importance of a step-wise approach.

CASE REPORT

MH is a 30-year-old male, usually fit and well, with no past medical history and no known drug allergies. He has a known family history of type 2 diabetes mellitus and hypertension. He is a non-smoker, teetotal and leads an active lifestyle when not at his desk-based job. He is an infrequently presenting patient.

He presents with a 10-day history of recurrent small lesions in his nostrils with generalized irritation and is treated successfully with nasal mupirocin. The following week he presents with a small painful pustular lesion in his right groin, with no systemic symptoms of note – no fever, cough or coryza. He is treated for folliculitis with topical fusidic acid.

A week later, the pustule has reduced in size but is still present, however a new one appears on his abdomen, lateral to the umbilicus. It increases in size from approximately 3mm to 1cm within 5 days. Though not actively discharging, it appears purulent with surrounding erythema. He remains systemically well. He is given a 1-week course of oral flucloxacillin and told that this is likely a skin infection and sent home.

Within 3 days, he is back with a boil on his upper lip – increasing in size and painful to touch, with one developing on his neck. Given the recurrence of similar lesions, he is told this may be secondary to bed bugs and is given hygiene measures to follow with another course of flucloxacillin and topical hydrocortisone cream. He is also advised to attend for blood tests, including HbA1c. He returns the following week with no improvement in his symptoms despite conservative measures as advised and is told his fasting glucose and HbA1c are normal.
Within the week, he develops a new lesion on his right shoulder – this time increasing in size quicker than the previous lesions with some purulent discharge. He is advised another course of flucloxacillin for suspected cellulitis, but this time for 14 days. He does not start given the futility of his previous treatments and increasing frustration at recurrence of symptoms and repeated trips to the doctor. Within 24 hours he develops a fever and begins to feel unwell. The lesion on his shoulder doubles in size, restricting movement of his shoulder due to pain. He attends A&E and is admitted under orthopaedics for surgical debridement of abscess and antibiotics.

Upon admission, the lesion is swabbed and demarcated to monitor for deterioration, he is started on broad spectrum antibiotics, intravenous fluids and he is listed for surgery the following morning. Prior to surgery, the results of his swab return, positive for PVL positive MRSA. He is started on IV clindamycin and linezolid and monitored for 24hrs, postponing surgical intervention. With improvement in his clinical condition and the shoulder abscess, it is agreed that he will continue IV and oral treatment with twice daily monitoring. After 48hrs, it is agreed that surgical intervention is no longer necessary, and he is discharged on 1-week oral treatment with decolonisation protocol (chlorhexidine wash and nasal mupirocin) after completing 1 week as an in-patient. Full recovery was made with no sequelae of disease at 1- and 3-month follow-up.

CONCLUSIONS

Perhaps most importantly, this case highlights the need for continuity of care and good history taking. This patient was seen by a different doctor at each presentation and managed acutely at each stage with repetition of futile treatment adding to patient distress. This can easily be avoided with the family medicine model of care.

The recurrence was hardly noted until a full history was taken on attendance to A&E. Further history-taking detailed recent travel with accommodation in an overcrowded hostel with shared bathroom facilities, both of which are are commonly cited risk factors for developing PVL (13). Identifying these risk factors may lead to earlier consideration of potential differentials and instigate appropriate investigative techniques to establish the diagnosis.
The importance of using skin swabs for microbiological analysis and appropriate treatment according to sensitivities is another key factor in optimising treatment to prevent subsequent morbidity and potential associated mortality.

Lastly, repetitive use of antibiotics, especially when there is no clear benefit following empirical use, should be avoided and challenged where possible. The use of evidence-based medicine to justify therapeutic decisions should be applied to ensure patient recovery and avoid antibiotic overuse.

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