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Osteomyelitis

A case report

Background
Methicillin resistant *Staphylococcus aureus* (MRSA) infection has classically been associated with institutional health care settings such as hospitals and nursing homes.

Objective
This article presents a case of community acquired MRSA infection resulting in severe osteomyelitis of the humerus, followed by a brief discussion and literature review.

Discussion
Over the past few years, more community acquired cases of MRSA have occurred. Methicillin resistant *S. aureus* usually infects skin and soft tissue. Occasionally, a life threatening infection occurs involving the blood, lungs, heart and bone.

Case study

An Aboriginal male, 16 years of age, presented with a history of left shoulder pain for 1 month. The pain followed a kick during a Taekwondo class and was followed by mild fever, especially at night, weight loss and malaise.

Clinical examination revealed gross limitation of left shoulder movement with disuse atrophy of the shoulder and forearm muscles on the left side. There was no tenderness, erythema or swelling of the shoulder joint. The patient was admitted for further evaluation.

Investigations revealed:

- haemoglobin: 103 g/L
- white cell count: 9.4 cells/mm^3
- blood film: normocytic normochromic red cells
- erythrocyte sedimentation rate (ESR): 98 mm/hour
- C-reactive protein: 69 mg/L
- shoulder X-ray: diffuse osteolysis of the upper half of the humeral shaft extending in to the head with inferior subluxation of the humeral head
- computerised tomography (CT): an extensive osteolytic lesion involving the upper humeral shaft with extensive regional adenopathy and nonspecific swelling of the soft tissues around the bone (*Figure 1, 2*).

Figure 1. CT scan of left shoulder showing the extensive lytic lesion

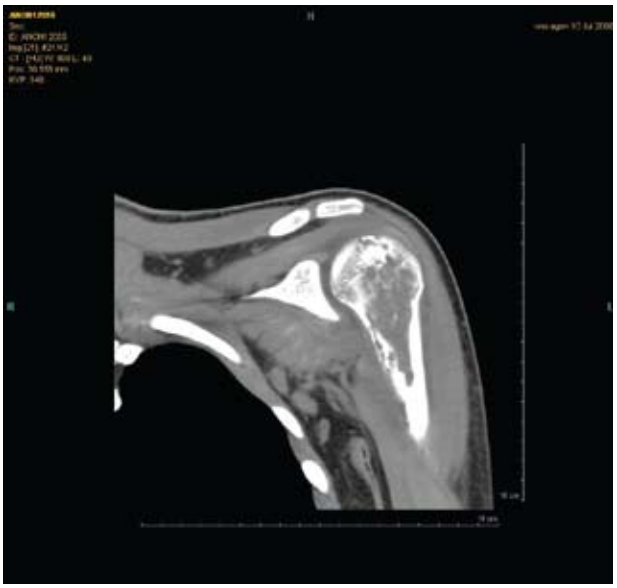
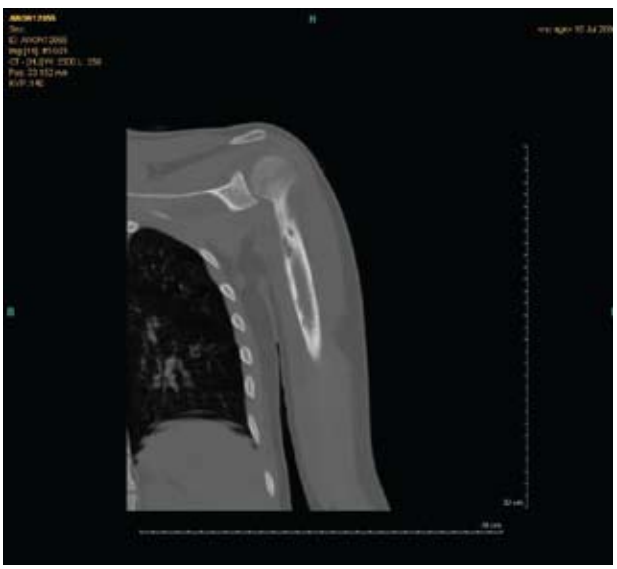


Figure 2. CT scan of left shoulder showing the lytic lesion involving the shaft and head of humerus



The differential diagnosis at this stage included osteomyelitis and bone tumour (Ewing sarcoma or osteosarcoma). A bone biopsy revealed mature bone with extensive acute inflammation on a background of granulation tissue. Micro-abscesses were seen and fragments of necrotic bone were noted within the areas of inflammation. Gram staining of the sections revealed Gram positive cocci. There was no evidence of malignancy in any sections and the features were consistent with acute and chronic osteomyelitis.

Culture of the biopsied specimen grew non-multiresistant methicillin resistant *Staphylococcus aureus* (MRSA), which was resistant to penicillin, methicillin/flucloxacillin and cephalosporins, but sensitive to cotrimaxozole, tetracycline, vancomycin, fusidic acid, erythromycin and rifampicin.

The patient was started on rifampicin 300 mg twice per day and fusidic acid 500 mg three times per day. After a week he was discharged with a final diagnosis of osteomyelitis of the left humerus caused by community acquired non-multiresistant MRSA. He steadily improved over the next month with improvement in joint movements and weight gain. He did not report any side effects from the treatment. At 4 weeks his inflammatory markers and haemaglobin had returned to normal. Treatment was continued for a total of 6 weeks.

Infections with methicillin resistant *Staphylococcus aureus* (MRSA) have occurred since the 1960s, but were mainly limited to hospital and health care settings. Sporadic reports of community acquired MRSA (CAMRSA) infections occurred between 1980 and 1999. There has been an increase in the number of reported community acquired cases since 1999.[1]

Most CAMRSA infections occur in skin and soft tissue and in specific population groups such as children, prisoners, athletes, Indigenous Australians, and homosexual men.[2–4] Occasionally, a life threatening infection occurs involving the blood, lungs, heart and bone. In the United States, a series of fatal cases in children were caused by a clone of MRSA known as 'USA 400'.[5] A recent USA study[6] identified a second clone, known as 'USA 300', as a predominant CAMRSA in Georgia.

Epidemiology

Community acquired MRSA was first described in Australia during the 1980s. The first detailed description was in 1989 in patients from remote Aboriginal communities in Western Australia.[7] More reports of CAMRSA emerged after the Northern Territory government made it compulsory to report MRSA infections in 1991. Between 1991 and 1995, infections caused by CAMRSA outnumbered those acquired from institutional health care settings. In the top end of the Northern Territory, Aboriginal people were 13 times more likely than non-Aboriginal people to be infected with CAMRSA.[4]

Molecular analysis of Northern Territory isolates found them to be genetically different from Western Australia strains.[8] In Queensland, two additional clones were identified which were found to infect both Aboriginal and non-Aboriginal populations. In the mid to late 1990s, an epidemic of CAMRSA infections was reported in Auckland, New Zealand. At this time, 10 isolates from Australia, New Zealand and Samoa were found to be genetically identical; it was thought that the infection arrived in Australia via New Zealand during the 1980s and 1990s.[4]

Community acquired versus hospital associated MRSA

- Almost all CAMRSA clones contain genes encoding for Panton-Valentine leukocidin which is a cytotoxin that causes leukocyte destruction and tissue necrosis. By contrast, hospital acquired MRSA classically lacks the Panton-Valentine leukocidin toxin
- The genes encoding for methicillin resistance in CAMRSA reside on the staphylococcal chromosome cassette mec (SCCmec) type 4 allele.[9] Hospital acquired MRSA is associated with other SCC mec alleles
- Nasal colonisation has not been found to consistently precede CAMRSA infections, whereas it is a common precedent to methicillin sensitive *S. aureus* (MSSA) infections[10] and a feature of hospital acquired MRSA. Recent studies have shown that gastric colonisation and household pets may serve as reservoirs for these organisms.[11] In a study by Munckhof et al,[12] nasal carriage of CAMRSA was found to be only 0.3%
- Hospital acquired MRSA are usually resistant to multiple antibiotics, while CAMRSA has been found to be susceptible to a range of antibiotics such as tetracyclines and cotrimaxozole.

Infections

The clinical spectrum of infections caused by CAMRSA has been wide ranging from minor skin and soft tissue infections to life threatening pneumonias, septicaemia, osteomyelitis, and bacterial endocarditis. Skin infections caused by these organisms are often misdiagnosed as spider bites, and appropriate antibiotics are not initiated. A recent study by Mark et al[6] revealed that 63% of skin and soft tissue infections caused by MRSA were community acquired.

Treatment

Treatment of infections caused by CAMRSA remains challenging. Many are resistant to oral cephalosporins and antistaphylococcal penicillins, which are most commonly used to treat skin and soft tissue infections. However, the majority of infections are susceptible to tetracyclines, cotrimaxozole, rifampicin, fusidic acid and vancomycin.

In Australia, strains vary in their resistance to erythromycin. However, inducible erythromycin resistant isolates are phenotypically susceptible to lincosamide antibiotics, so clindamycin is an attractive option. Unfortunately there are concerns that this organism can mutate from inducible erythromycin resistant/clindamycin susceptible phenotype to the constitutive erythromycin/clindamycin resistant phenotype.[13]

Mild infections often require incision and drainage alone, however in areas with high prevalence of CAMRSA, serious skin and soft tissue

infections warrant therapy with agents such as vancomycin that are active against MRSA. Minor infections can be treated with oral co-trimaxozole. Additional medications include linezolid (which may have a role in antagonising the Panton-Valentine leukocidin toxin in addition to its antibiotic properties) and intravenous immunoglobulin (which may have antibodies against the toxin).

Clinicians should have a low threshold for obtaining specimens for culture when patients present with skin and soft tissue infections. This is important so that infections caused by Streptococci and MSSA can be treated with other antibiotics preventing the inappropriate use of glycopeptides.[14]

Summary of important points

- Community acquired MRSA infections are becoming increasingly prevalent.
- CAMRSA commonly causes minor skin and soft tissue infections. Occasionally, it can be responsible for significant morbidity and mortality.
- Nasal colonisation has not been found consistently to precede the infection with CAMRSA.
- Clinicians should have a low threshold for obtaining specimens for culture in the setting of skin and soft tissue infections particularly when the patient does not respond rapidly to drainage and standard antimicrobial therapy.
- The majority of CAMRSA are susceptible to tetracyclines, cotrimaxozole, rifampicin, fusidic acid and vancomycin.
- Strains vary in their resistance to erythromycin. Erythromycin resistant isolates may respond to clindamycin, but there is a concern that these clones can mutate to a erythromycin/clindamycin resistant strain.
- In the presence of more serious and invasive infections, immediate therapy with vancomycin or linezolid should be instituted without delay until treatment can be modified by culture results.

Conflict of interest: none declared.

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