THE bacterium Staphylococcus aureus is a successful and ubiquitous parasite of humans and other mammals. Large surveillance studies in diverse populations have shown that up to 30% of healthy individuals are colonised by S. aureus at any point in time, either transiently or permanently. Infection and disease do not occur in most people who are colonised with S. aureus, because of the efficacy of innate and adaptive host immune defences. However, given the opportunity, S. aureus will breach these defences and cause infection and disease. Staphylococcal infections are common in community-based medical practice. Many infections caused by S. aureus are minor and do not warrant antimicrobial therapy. However, S. aureus is a frequent cause of severe disease, ranging from deep-seated bone infection to life-threatening septicemia or toxic-shock syndrome.

A recent study estimated that there are up to 8000 cases of bloodstream infection caused by S. aureus in Australia every year, of which...
S aureus has overtaken viridans streptococci as the most common cause of infective endocarditis in all regions of the world, including Australia.

S aureus has been described in several populations. In addition to its ability to evade host immune defenses, S aureus has a remarkable “track record” for acquiring resistance to antimicrobial agents. Resistance to penicillin emerged soon after this antibiotic was first used for treating staphylococcal infection in 1941, and resistance to almost all other antimicrobials used to treat staphylococcal infection has been described, usually shortly after that agent was first used. Until recently, all S aureus infections acquired in the general community could be assumed to be susceptible to anti-staphylococcal antibiotics such as flucloxacillin, dicloxacillin and cephalaxin. However, in recent years new strains of antibiotic-resistant S aureus, known colloquially as community methicillin-resistant S aureus, or cMRSA, have emerged worldwide. These cMRSA strains have spread widely and rapidly and are an important cause of morbidity and mortality worldwide, including Australia. Of significant public health concern is the fact that some strains of cMRSA circulating in Australia have acquired potent virulence factors that make them more likely to cause invasive disease.

This review describes the epidemiology, clinical features, diagnosis and management of cMRSA infection.

**Epidemiology**

The semi-synthetic antibiotic methicillin was developed in the 1950s to combat the growing problem of penicillin resistance in S aureus. Just two years after its introduction, methicillin-resistant S aureus (MRSA) infection was first described in hospitalised patients and quickly became established in hospitals and health care facilities worldwide. For many years, MRSA was almost only found in hospitalised patients or in those in long-term care facilities such as nursing homes. In the 1970s and 1980s, small clusters of MRSA infection appeared in some urban communities (eg, IV drug users) in the US. In the late 1980s and early 1990s, cases of MRSA infection in patients with no history of contact with health care started appearing in the Kimberley region of WA. Shortly afterwards cases of so-called “community MRSA” (cMRSA) infections were reported from the east coast of Australia and the NT. These infections were caused by strains of MRSA that were not multi-resistant (see definitions in table 1) and were generally different to hospital MRSA strains. In the late 1990s and the early 21st century, cMRSA also rapidly emerged in many other parts of the world (eg, the US, Europe) and cases of severe and fatal invasive infection in previously healthy children and adolescents were reported.

The Australian Group on Antimicrobial Resistance (AGAR) performs regular surveillance on S aureus isolated in laboratories across Australia. Recent surveillance has shown that the proportion of S aureus identified in these laboratories that are cMRSA is increasing across Australia (figures 1 and 2). In the most recent survey performed in 2004, 15% of all S aureus isolated from patients in outpatient settings was cMRSA. Alarmingingly, a recent study of skin and soft tissue infections presenting to 11 major emergency departments in the US showed that cMRSA was the most common cause.

There are some characteristic strains, or “clones”, of MRSA currently circulating in Australia. In the main these tend to be restricted to certain geographical areas or populations. However, over time they may spread within and between countries.

**Risk factors**

High rates of infection or colonisation with cMRSA have been described in several populations in Australia, high rates of infection and carriage have been described in remote and rural communities and among expatriate Polynesians and among expatriate Polynesians and among expatriate Polynesians and among expatriate Polynesians and among expatriate Polynesians and among expatriate Polynesians. Similar infection rates are found in other indigenous populations, for example, the Inuit in the Arctic and the Natives in Australia. Outbreaks of cMRSA infection have been described in several settings. These include members of sporting teams (eg, rugby, American football), prisons, men who have sex with men, and day care centres. These outbreaks have generally been attributed to the introduction of cMRSA into a situation of overcrowding and poor hygiene. Molecular epidemiology studies of organisms isolated from individuals involved in these outbreaks have shown they have been caused by single strains of MRSA that have probably been transmitted from person to person. More recently, it has become clear that cMRSA is no longer restricted to these subgroups and is in fact widespread in many communities in Australia and worldwide.

**Diabetes mellitus and IV drug use**

Outbreaks of cMRSA infection and colonisation. Outbreaks of cMRSA infection have been described in IV drug users, but there is no evidence to suggest that the group is more likely to be infected or colonised with cMRSA compared with the background population.

**Superbug**

A term used commonly in the media, variously to describe resistant microorganisms, virulent microorganisms or organisms with both characteristics.

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**Table 1: Definitions**

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methicillin-resistant Staphylococcus aureus (MRSA)</td>
<td>An isolate of MRSA known to be readily transmissible from person to person. Examples of epidemic MRSA strains in Australia are the UK132 and UK166-MRSA strains and the community-acquired MRSA strains (Aus 2 and Aus 3 MRSA).</td>
</tr>
<tr>
<td>Non-methicillin-resistant S aureus (nmMRSA)</td>
<td>An MRSA strain that is resistant to beta-lactam antibiotics but susceptible to two or more non-beta-lactam antibiotics used to treat staphylococcal infection (eg, clindamycin, cotrimoxazole, doxycycline, rifampicin, fusidic acid, ciprofloxacin).</td>
</tr>
<tr>
<td>Community MRSA (cMRSA)</td>
<td>MRSA isolated from patients in the community in an individual with no history of recent contact with a hospital or long-term care facility.</td>
</tr>
<tr>
<td>Community-acquired MRSA infection</td>
<td>MRSA infection acquired in the general community in an individual with no history of recent contact with a hospital or long-term care facility.</td>
</tr>
<tr>
<td>Non-epidemic MRSA (nMRSA)</td>
<td>An isolate of MRSA not thought to be readily transmissible from person to person.</td>
</tr>
<tr>
<td>Non-multiple-resistant MRSA (nmpMRSA)</td>
<td>An isolate of MRSA that is resistant to two or more non-beta-lactam antibiotics, as above.</td>
</tr>
</tbody>
</table>

**Figure 1** MRSa in WA, 1983-2005.

**Figure 2** Community MRSA in Australia.
Resistance and virulence factors in cMRSA

The mec gene RESISTANCE to methicillin in staphylococci is conferred by the presence of the mec gene in the staphylococcal chromosome. This gene encodes for an altered penicillin-binding protein 2a (PBP2a), which is a component of the bacterial cell wall.

Alteration in configuration of the penicillin-binding proteins that beta-lactam antimicrobial agents (penicillins, cephalosporins and carbapenems) cannot bind to PBP2a, so the bacteria are not killed by these agents.

The mec gene is found as part of a larger genetic element known as staphylococcal cassette chromosome mec (SCCmec). This element is capable of mobilisation, and is therefore potentially transmissible from organism to organism, although this does not appear to be a particularly frequent event in practice.

Other genes that confer resistance to other antimicrobial agents can be found in the SCCmec element, or, alternatively, they may be inserted into other parts of the chromosome, or acquired independently on plasmids.

Panton-Valentine leukocidin

Panton-Valentine leukocidin (PVl) is an exotoxin that causes lysis of white blood cells and other cell lines, and is thought to be an important virulence determinant in 5 aureus. The genes coding for PVl are also found as a potentially mobile genetic element on the 5 aureus chromosome.

PVl was first described in the 19th century by researchers studying the effect of S aureus abscesses in animals. This toxin is not commonly present in community-acquired 5 aureus isolates. However, its presence has been closely associated with invasive 5 aureus infection, including cutaneous abscesses and necrotising pneumonia.

Certain strains of 5 aureus commonly isolated in Australia are bacteriologically similar to 5 aureus isolates from the United States and Europe, and are thus appropriately referred to as community MRSA (cMRSA).

Skin and soft tissue infection

SUPERFICIAL infection of the skin and supporting structures is the most common presentation of community MRSA infection.

Common types of cutaneous staphylococcal infection include impetigo, furuncles, carbuncles, furuncles, cellulitis and botryomycosis (a chronic granulomatous form of 5 aureus infection that results in nodule or ulcer formation, often in immunosuppressed hosts).

Symptoms and signs of cMRSA skin or soft tissue infection are similar to those caused by methicillin-susceptible 5 aureus (MSA) strains that produce PVl.

Invasive infection

All forms of invasive staphylococcal infection can be caused by 5 aureus. This includes deep-seated abscesses, pyomyositis, pneumonia, septic arthritis and osteomyelitis, necrotising fasciitis (eg, kidney, liver, lung or brain), baccharaemia and infective endocarditis.

Tissues cannot be overstated in the management of cMRSA.

General measures

TREATMENT of cMRSA infections is significantly different from that of other staphylococcal infections. The basic principles of treatment are:

- Drain pus and debride infected tissue.
- If there are signs and symptoms of systemic infection or extensive local infection, prescribe systemic antimicrobial therapy.
- Review the patient regularly to ensure clinical response.

The importance of draining pus and debriding infected tissue cannot be overstated in staphylococcal infection, particularly that caused by cMRSA. M. aureus antimicrobial agents penetrate poorly or not at all into the centre of abscesses, and dead or denatised tissue is not perfused.

Therefore, surgical management is primary in post-forming infection; antimicrobial therapy plays an adjunctive role only. In fact, in many cases of mild-moderate cMRSA infection, antimicrobial therapy may not be necessary if the infection is minor and has been dealt with surgically.

Clinical features of MRSA infection

Necrotising pneumonia

Necrotising pneumonia is caused by an organism that is sensitive to beta-lactam antibiotics such as flucloxacillin, dicloxacillin, and clindamycin.

Pneumonia is typically caused by the presence of the mec gene in the staphylococcal chromosome. This gene encodes for an altered penicillin-binding protein 2a (PBP2a), which is a component of the bacterial cell wall.

Tetracyclines act to inhibit bacterial ribosome synthesis. They have bacteriostatic activity against 5 aureus. Clindamycin is active against most strains of 5 aureus, has excellent bioavailability after oral dosing, and is widely distributed in most body fluids and tissues (including bone and abscesses).

Accordingly, the following recommendations regarding antimicrobial therapy are based on data from laboratory studies, animal models, non-controlled clinical trials, or have been extrapolated from clinical trials conducted in hospital-acquired 5 aureus infection.

Antimicrobial agents commonly used to treat patients with mild-moderate cMRSA infections are listed below and summarised in table 2.

Lincosamides (clindamycin, lincomycin)

The lincosamides are a group of antibiotics that can be used to treat infections caused by 5 aureus. They have good tissue penetration, and most cMRSA isolates are susceptible to these agents.

Tetracyclines (tetracycline, doxycycline, minocycline)

Tetracyclines act to inhibit bacterial ribosome synthesis. They are bacteriostatic activity against 5 aureus. Clindamycin is active against most strains of 5 aureus, has excellent bioavailability after oral dosing, and is widely distributed in most body fluids and tissues (including bone and abscesses).

Antimicrobial therapy

Unfortunately there are minimal published data on the clinical efficacy of any antimicrobial agent for the treatment of cMRSA infection. At the time of writing, there are no published randomised controlled trials comparing one antimicrobial (or combination of antimicrobials) to another for the treatment of cMRSA infection. How-ever, there is some evidence that certain combinations of antimicrobial agents are effective therapy in mild-moderate cMRSA infection.

However, several adverse events are associated with 5 aureus infection. Nausea, diarrhoea and rash are the most common, but more serious reactions such as Stevens-Johnson syndrome, neutro-penia, thrombocytopenia and nephro- and hepatotoxicity may also occur. Serious reactions are more likely to occur in the elderly.

Tetracyclines (tetracycline, doxycycline, minocycline)

Tetracyclines are well absorbed orally, have good tissue penetration, and are effective against staphylococcal infection.

Clindamycin

Clindamycin is typically used in the treatment of cMRSA. It is effective against most strains of 5 aureus, has good tissue penetration, and is widely distributed in most body fluids and tissues (including bone and abscesses).

Figure 3: Isolate of cMRSA that is susceptible to clindamycin (DA2) and erythromycin (E15).

Figure 4: Isolate of cMRSA that is susceptible to clindamycin (DA2) and erythromycin (E15).

Figure 5: Isolate of cMRSA that is susceptible to clindamycin (DA2) and erythromycin (E15).

Figure 6: Isolate of cMRSA that is susceptible to clindamycin (DA2) and erythromycin (E15).

Figure 7: Isolate of cMRSA that is susceptible to clindamycin (DA2) and erythromycin (E15).
One recently published US retrospective case series of 24 patients with MRSA infections reviewed the outcomes of patients treated with oral minocycline or doxycycline. Of the 100mg twice daily, 50% of patients (79%) had completed 10 days of oral treatment caused by cMRSA. The overall clinical success rate was 83%, and three of the four clinical failures were in patients with osteomyelitis or septic arthritis. There was one case of superinfection from staphylococci after minocycline after five days, and another due to vancomycin-resistant enterococcus after 10 days.

These data suggest that doxycycline or minocycline may be reasonable oral options for the treatment of mild-to-moderate cMRSA infection.

Rifampicin is a powerful inhibitor of rifamycin, clarithromycin, azithromycin, and fusidic acid. Like the lincosamides, these agents inhibit bacterial protein synthesis by binding to the 30S subunit of the 70S ribosome. They are bacteriostatic against S. aureus. They are generally inactive against multiresistant MRSA isolates but are active against 65-75% of cMRSA.

Resistance to rifampicin often confers resistance to the newer macrolides and azalides, as well as reducing activity of other classes of antimicrobials, including the lincosamides and streptogramins. Erythromycin and clarithromycin are powerful inhibitors of cytochrome P450 (CYP450) enzymes, particularly the CYP3A4 and CYP2D6 isoenzymes, and can thereby increase the plasma drug levels of various agents, such as warfarin, which can become hyperpotassium, leading to serious adverse drug reactions.

The newer agents, azithromycin and clarithromycin, are less potent CYP450 inhibitors and are less susceptible to serious interactions.

Rifampicin

Rifampicin is a potent anti-staphylococcal agent, with high oral bioavailability, with extensive penetration into tissue (including bone and lung), body fluids, and cell membranes. It is not PBS approved for the treatment of staphylococcal infections, and is difficult to obtain outside public hospitals.

Because of the rapid emergence of resistance when used as monotherapy, rifampicin must be used in combination with another antimicrobial. It is usually combined with either fusidic acid, tetracycline or a quinolone. Rifampicin is a powerful enzyme inhibitor and is therefore used in combination with other drugs to reduce the risk of drug resistance occurring.

Rifampicin is also used as an oral route to treat Gram-positive infections, including MRSA isolates, and is only available through the special access scheme (SAS) for unlicensed products. Rifampicin has been used as an oral alternative in MRSA infection when other agents cannot. Clinicians are advised not to use it for reasons of resistance, allergy or intolerance.

Topical antimicrobial agents (eg, mupirocin, fusidic acid, clindamycin)

In most cases of mild superficial staphylococcal infection in children and infants exposed during pregnancy, premature sun exposure or neonatal infection, is expensive and is generally inactive against MRSA. However, the development of resistance when used for reasons of resistance, allergy or intolerance.

Future treatment options

Moxifloxacin has excellent oral bioavailability and is therefore used in combination with other antimicrobials to treat Gram-positive infections, including MRSA isolates, and is only available through the special access scheme (SAS) for unlicensed products.

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Future treatment options

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How to treat - community MRSA infection

Infection-control implications of cMRSA

S aureus

Methicillin-resistant S aureus

Mec

S aureus

Methicillin-susceptible S aureus

PVl

Clindamycin S Rifampicin S

Microscopy

Many white cells seen

Many Gram-positive cocci seen

Culture

Heavy growth of methicillin-resistant Staphylococcus aureus (MRSA)

Suscceptibilities

Difloxacin Ciprofloxacin S

Cefalexin R Doxycycline S

Erythromycin S Fusidic acid S

Clindamycin R Rifampicin S

R. = resistant; S. = susceptible

Figure 7: Possible origin of community MRSA

LITTLE is known about the transmissibility of cMRSA strains in the community setting. However, several outbreaks of cMRSA infection have been described in hospitals (where the patient came from the community). This suggests that transmission of cMRSA can occur from person to person within health-care facilities. However, patients with moderate-severe infection are at higher risk of morbidity or mortality should they receive inappropriate empirical therapy for cMRSA infection.

A wariness of the local epidemiology of cMRSA is important in determining which antimicrobial should be used. A standard empirical therapy for 5 aureus infection. Information regarding the incidence and prevalence of cMRSA and advice regarding appropriate empirical and definitive therapy should be available from a clinical microbiologist in your region. In regions or populations where cMRSA is known to be prevalent, consider initiating antimicrobial therapy that is active against both methillin-susceptible and methillin-resistant 5 aureus, such as clindamycin, cotrimoxazole or doxycycline.

The choice of agent will be determined by patient factors and prescribe preference: at present there are no comparative data which suggest which particular agents should be used as first-line therapy, although this data may become available in the future.

Duration of therapy

Unfortunately, few clinical studies have directly addressed the issue of duration of therapy for most infections caused by 5 aureus. Therefore any recommendations are based largely on treatment guidelines and/or data from non-randomised non-controlled studies.

At present, there is no evidence that cMRSA infections are more ‘resistant’ to appropriate antimicrobial therapy, or require longer duration of therapy to achieve cure. Therefore duration of therapy for most moderate-critical 5 aureus infections is the same as that for infection caused by methillin-susceptible 5 aureus. For example, carbuncules caused by cMRSA should be treated for a minimum of 5 days in addition to adequate drainage, and mild-to-moderate cellulitis should be treated for a minimum of 7-10 days. In all cases the patient should be reviewed regularly and the total duration of treatment determined by clinical response. If the patient deteriorates, despite apparently appropriate therapy, they should be considered for IV antimicrobial therapy in addition to surgical intervention, if indicated.

All patients with bacteremia (bloodstream infection) due to 5 aureus (including cMRSA) should undergo careful clinical examination and imaging (including echocardiography) to elucidate the cause of the bacteremia and to exclude endocarditis and metastatic infection.

Because of the significant risk of subclinical endocarditis and metastatic infection being present at the time of diagnosis, all patients with bacteremia due to cMRSA should require a minimum of 14 days of IV antimicrobial therapy. If the above complications are present, prolonged IV therapy (2-6 weeks) is commonly required, often followed by prolonged oral therapy.

Further reading

Australian Society for Antimicrobials: www.asanic.net.au

Australian Group on Antimicrobial Resistance: www.antimicrobial-resistance.com

For patients


Website of the Australian Department of Health: www.health.gov.au

Further reading

Online resources

For Australian doctors and nurses: www.cdc.gov/ncidod/dhqp

Antimicrobial Agents and Resistance: www.tufts.edu/med/apua/methicillin.htm

Antimicrobial Resistance: www.cdc.gov/ncidod/factsheets/Antimicrobials.htm

Antimicrobials:

www.fda.gov/cdrh/mdl/0203_07_16/2007_5.05_PM.htm

www.cdc.gov/ncidod/ftp_023_030__FEB23_07

www.cdc.gov/ncidod/ftp_023_030__FEB23_07

For Australian doctors: www.tufts.edu/med/apua/Antimicrobials.htm

Antimicrobial Agents and Resistance: www.cdc.gov/ncidod/dhqp

Antimicrobial Resistance: www.antimicrobial-resistance.com

For patients


Website of the Australian Department of Health: www.health.gov.au

Figure 6: Possible origin of PVI: community MRSA

Australia Doctor 23 February 2007

www.australiandoctor.com.au
### How to treat – community MRSA infection

**GP’s contribution**

**Case study**

Mr B, 75, has long-term type 2 diabetes and morbid obesity. His diabetes is poorly controlled, largely due to non-compliance. He has a persistent lower leg ulcer that has been present for months despite regular debridement and dressings both at home and in the surgery. Often his ulcer oozes serous fluid, and swabs of this consistently grow *S. aureus*. He has at various times been treated with topical Bactroban, with limited success.

**Questions for the authors**

1. **Which TWO statements about infection caused by *S. aureus* are correct?**
   - a) It is the most common cause of infectious endocarditis in Australia.
   - b) It commonly affects the skin and soft tissue.
   - c) It is a common cause of community-acquired pneumonia.
   - d) It is a cause of septic arthritis in children.

2. **Which TWO statements about isolating children with MRSA are correct?**
   - a) It is done when staff start caring for patients with MRSA.
   - b) It is done when patients with MRSA are hospitalised.
   - c) It is done when patients with MRSA are isolated in hospitals, but these are not generally not recommended in the community setting.
   - d) It is done when patients with MRSA are discharged from hospitals, but these are not generally not recommended in the community setting.

3. **Which TWO statements about treatment for MRSA infections are correct?**
   - a) Dicloxacillin should be used as monotherapy in empirical treatment.
   - b) Ceftriaxone is effective in treating MRSA infections.
   - c) Ciprofloxacin should not be used as monotherapy in treating MRSA infections.
   - d) Gentamicin is a suitable alternative to ceftriaxone.

4. **Which TWO statements about the treatment of MRSA are true?**
   - a) Treatment should be started before results of culture and sensitivity are available.
   - b) Treatment should be continued for at least 2 weeks.
   - c) Treatment should be continued for at least 3 weeks.
   - d) Treatment should be stopped once the infection is cured.

5. **Which TWO statements about the treatment of MRSA are true?**
   - a) Treatment should be started before results of culture and sensitivity are available.
   - b) Treatment should be continued for at least 3 months.
   - c) Treatment should be continued for at least 4 months.
   - d) Treatment should be stopped once the infection is cured.

6. **Which THREE other measures would you advise for Rebecca?**
   - a) Screen for diabetes.
   - b) Refer to an immunologist for investigation of possible immunodeficiency.
   - c) Wash all bedclothes in hot water twice a week.
   - d) Test her partner for cMRSA colonisation as well.

7. **Which TWO statements about antibiotics used to treat cMRSA are correct?**
   - a) Mupirocin is effective in treating MRSA infections.
   - b) Clindamycin is effective in treating MRSA infections.
   - c) Ciprofloxacin is effective in treating MRSA infections.
   - d) Gentamicin is effective in treating MRSA infections.

8. **What is the correct antibiotic for cMRSA infection?**
   - a) Tetracycline.
   - b) Rifampicin.
   - c) Clindamycin.
   - d) Ciprofloxacin.

9. **Which statement is true about cMRSA infection?**
   - a) cMRSA infections are more common in children than in adults.
   - b) cMRSA infections are more common in adults than in children.
   - c) cMRSA infections are more common in men than in women.
   - d) cMRSA infections are more common in women than in men.

10. **Which TWO statements about cMRSA infections are correct?**
    - a) cMRSA infections are more common in patients with diabetes.
    - b) cMRSA infections are more common in patients with obesity.
    - c) cMRSA infections are more common in patients with heart disease.
    - d) cMRSA infections are more common in patients with lung disease.

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**INSTRUCTIONS**

Complete this quiz to earn 2 CPD points and/or 1 PDP point by marking the correct answer(s).

**How to Treat Quiz**

Community MRSA infection — 23 February 2007

**How to Treat Community MRSA infection**

**Case study**

MR B, 75, has long-term type 2 diabetes and morbid obesity. His diabetes is poorly controlled, largely due to non-compliance. He has a persistent lower leg ulcer that has been present for months despite regular debridement and dressings both at home and in the surgery. Often his ulcer oozes serous fluid, and swabs of this consistently grow *S. aureus*. He has at various times been treated with topical Bactroban, with limited success.

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1. **Which TWO statements about infection caused by *S. aureus* are correct?**
   - a) It is the most common cause of infectious endocarditis in Australia.
   - b) It commonly affects the skin and soft tissue.
   - c) It is a common cause of community-acquired pneumonia.
   - d) It is a cause of septic arthritis in children.

2. **Which TWO statements about MRSA are correct?**
   - a) MRSA is a cause of recurrent infections.
   - b) MRSA is a cause of community-acquired pneumonia.
   - c) MRSA is a cause of recurrent infections.
   - d) MRSA is a cause of hospital-acquired pneumonia.

3. **Which TWO statements about the treatment of MRSA are true?**
   - a) Treatment should be started before results of culture and sensitivity are available.
   - b) Treatment should be continued for at least 3 months.
   - c) Treatment should be continued for at least 4 months.
   - d) Treatment should be stopped once the infection is cured.

4. **Which TWO statements about the treatment of MRSA are true?**
   - a) Treatment should be started before results of culture and sensitivity are available.
   - b) Treatment should be continued for at least 4 months.
   - c) Treatment should be continued for at least 5 months.
   - d) Treatment should be stopped once the infection is cured.

5. **Which TWO statements about the treatment of MRSA are true?**
   - a) Treatment should be started before results of culture and sensitivity are available.
   - b) Treatment should be continued for at least 5 months.
   - c) Treatment should be continued for at least 6 months.
   - d) Treatment should be stopped once the infection is cured.

6. **Which THREE other measures would you advise for Rebecca?**
   - a) Screen for diabetes.
   - b) Refer to an immunologist for investigation of possible immunodeficiency.
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**How to Treat**

**Editor:** Dr Marcela Cox

**Co-editor:** Julian McAllan

**Quiz:** Dr Marcela Cox

**NEXT WEEK**

Squamous cell carcinoma is the second most common skin cancer behind basal cell carcinoma. Increase your chances of picking them up by picking up next week’s How to Treat on SCC and its precursors. The author is Dr Suresh Chandra, consultant dermatologist specialising in Mohs’ micrographic surgery and cosmetic dermatology. He is in private practice in Bentleigh, Victoria; VMO, Monash Medical Centre, Clayton, Victoria; and director, Victorian Mohs Micrographic Surgery and Cancer Centre, Bentleigh.