

in serum creatinine from pre- to post-contrast was calculated (%Scr). All EG high-risk patients were managed under the supervision of the renal team using the CIN protocol.

Results

During the six-month period, 21 patients were referred; 12 of these were from the early group and nine were from the late group. There was no significant difference in age (mean \pm standard deviation, EG: 70.6 ± 3.9 ; LG: 73.5 ± 2.2), number of diabetics (EG: 2 vs LG: 3; one person from each group was on insulin), types of contrast studies, renal function before contrast administration (EG: 223.0 ± 42.8 ; LG: 206.3 ± 29.1 mmol/l), or co-morbidities between the groups. The mean %Scr was significantly higher in the late group than the early group (LG: $+98.8 \pm 42.2$ vs EG: $+2.4 \pm 9.4$, $p=0.0098$).

Two patients from the late group required temporary haemodialysis, compared with none from the early group. Of the two patients who required dialysis, one had a percutaneous coronary intervention, the other a femoral angiogram. Both had recovered renal function sufficiently to be off haemodialysis 28 days after the contrast procedure. The volume of contrast used was significantly higher in the late group than the early group (LG: 241.6 ± 19.5 ; EG: 137.9 ± 17.3 , $p=0.0004$). The length of hospital stay (in days) was longer in the late group (LG: 12.0 ± 3.1 ; EG: 4.0 ± 0.2 , $p=0.004$). This was especially the case in the two individuals who required temporary haemodialysis.

Conclusion

With increasing interventional cardiology and radiology procedures being performed in high-risk individuals, the incidence of CIN may be expected to increase in the near future. This will have implications for workload, in the form of increased referrals to renal departments. It is, therefore, in our interest to educate ourselves and our colleagues in cardiology and radiology about this eminently preventable cause of AKI. In our experience, close and early liaison with our colleagues in other departments is crucial in reducing the risk of CIN.

The best way to prevent CIN is not clear. The interventions of oral or IV hydration and use of iso-osmolar contrast agents are supported by robust data in the literature. The issue of N-acetylcysteine is less clear, but our pragmatic decision has been to use it because it is safe, inexpensive and easy to administer. However, by far the most important intervention is achieving

good fluid balance before the contrast study. This will obviously have logistical implications in outpatient-based radiology investigations and day-case-based cardiology investigations.⁵⁻⁸

After a consultation period with the cardiology and radiology departments, an inpatient protocol to reduce CIN was developed and circulated to the relevant wards. The risk stratification used in the protocol helps the renal team to concentrate their supervision only on the high-risk group of patients, who are most likely to need renal replacement therapy. We propose our protocol will identify medium-to-high-risk patients, who may require inpatient admission before the investigation. The responsibility of identifying these patients is a contentious issue. We believe it should be a joint responsibility between the referring physician and the cardiologist or radiologist performing the contrast study. Identification of such patients will only occur if awareness of CIN exists among referring physicians.

Currently, awareness of CIN is poor, with 42% of referrals to the renal department being made after contrast study. The consequences of late referral are prolonged inpatient stay and increased risk of needing temporary haemodialysis. Both of these outcomes have a profound cost implication and also have significant morbidity associated with them. For the CIN protocol to be used effectively across hospitals, awareness needs to be increased dramatically and sustained ■

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Key points

- Contrast-induced nephropathy (CIN) is likely to become more common as more radio-contrast studies are performed.
- Early and close liaison across disciplines is important in reducing the incidence of CIN.



What I tell my patients about MRSA infection

Methicillin-resistant *Staphylococcus aureus* (MRSA) – also known as multidrug-resistant *Staphylococcus aureus* or a ‘superbug’ – is a bacteria that is responsible for several difficult-to-treat infections. It includes any strain of *Staphylococcus aureus* bacteria that is resistant to conventional antibiotics.

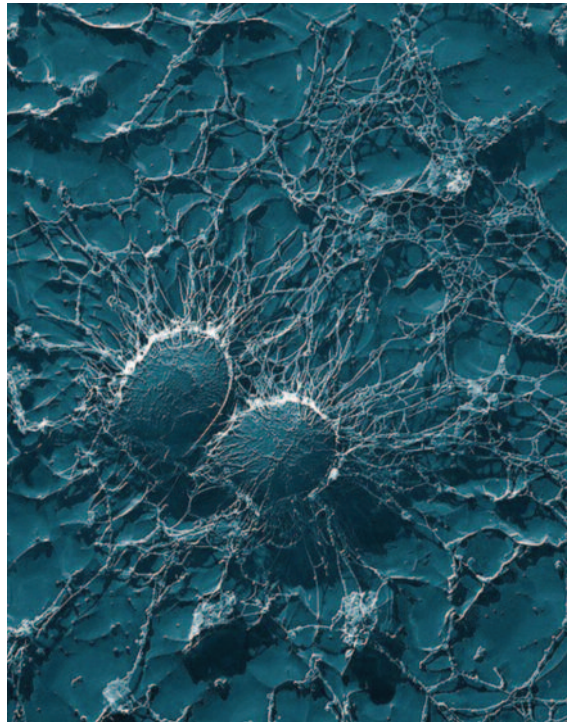
Healthy individuals may carry MRSA asymptomatically for a few weeks to many years. However, patients with weakened immune systems are at much greater risk of developing secondary infections – most commonly skin infection. More severe complications include pneumonia, and infection of the heart valves, bones or joints. MRSA is a particular problem in hospitals: the elderly, and patients with weakened immune systems, exposed wounds, diabetes and indwelling medical devices such as central venous catheters (CVCs) are most at risk. Patients with chronic kidney disease (CKD), those requiring renal replacement therapy, and kidney transplant recipients are therefore particularly prone to infective complications of MRSA, because this group includes those who are immunosuppressed, those with dialysis lines that are CVCs, and a significant number of patients with diabetes.

What is *Staphylococcus aureus*?

Staphylococcus aureus is a bacterium, often just called ‘*S aureus*’ or ‘staph’. It is commonly found on the skin and in the nostrils of healthy people, being present in up to one in three of the population. These people are called ‘carriers’ and, in healthy carriers, *S aureus* is usually harmless. However, *S aureus* can sometimes invade the skin to cause infection, particularly if there is a skin break or an open wound, which then allows bacteria a portal of entry.

Skin infections

Most *S aureus* infections are skin infections, including: boils (infections of the hair root); abscesses (collections of pus under the skin); styes (infections of the eyelid glands); carbuncles (pus-filled lumps under the skin); cellulitis



Staphylococcus aureus seen with an electron microscope – 50,000x magnification

(infection in the deep layer of the skin and the underlying layer of tissue); and impetigo (a highly contagious blistering skin infection).

Bloodstream infections

In severe cases, *S aureus* can enter the bloodstream and cause more serious infections by travelling to, and infecting, internal body parts. These infections include: septicaemia (blood poisoning); septic shock (infection of the blood with low blood pressure and organ failure); septic arthritis (severe joint infection); osteomyelitis (bone marrow infection); abscesses deep within the body; meningitis; pneumonia; endocarditis (infection of the heart valves); and discitis (infection of the discs within the spine).

These are all life-, organ- or limb-threatening infections that are more likely to occur in people who are already unwell or debilitated, or who have a poor immune system.

How is MRSA different?

Various subtypes of *S aureus* exist, and MRSA infections are due to a subtype that is resistant to

Philip Masson BA
MRCP Specialist
Registrar, Renal
Medicine
Wendy Metcalfe
MD FRCP Consultant
in Renal Medicine,
Department of
Renal Medicine,
Royal Infirmary of
Edinburgh



the antibiotic methicillin (a type of penicillin) and also to many other types of antibiotics. In common with any other strain of *S aureus*, some healthy people are carriers of MRSA and others develop the same spectrum of infections.

MRSA has become much more common since the 1980s, and is now the cause of over 40% of *S aureus* bloodstream infections. MRSA strains are no more aggressive or infectious than other strains of *S aureus*. However, infections are harder to treat because many antibiotics do not work. Therefore, infections with MRSA can become more severe than infections with other strains if they are not recognised early, and are initially treated with ineffective antibiotics.

Does everyone with MRSA become unwell?

Not everybody with MRSA will develop illness. People can be carriers of MRSA for long periods of time without any symptoms of infection. However, carrying the bug is a risk factor for going on to develop problems, especially in the presence of critical illness, surgical wounds or a dampened down immune system.

MRSA colonisation (carrier of MRSA)

Many people carry MRSA without any symptoms, the most common places for colonisation being the armpits, nostrils, skin, throat and urine. These can act as reservoirs from which MRSA infections can later develop in your own body or spread to other people. A study in Scotland found that 7.5% of people being admitted to hospital carried MRSA.

Carriers of MRSA are often offered eradication therapy before planned hospital admissions. The aim of this is both to prevent future problematic infections in the carriers themselves and to stop the spread of MRSA to others.

Normally, this treatment is in the form of an ointment called mupirocin. This is applied inside the nostrils. Additionally, depending on where the bacteria have been found on your body, antiseptic washes have been shown to be effective in reducing the number of MRSA bugs on the skin. Regular changing of bed sheets is needed, too, during such eradication efforts, to avoid re-infection with MRSA from contaminated bedclothes. It is still uncertain from clinical trials as to which treatment is the most effective.

MRSA infection

MRSA most commonly affects people in hospital, especially long-term inpatients.

Patients who are critically unwell in intensive care or high-dependency units are most at risk. Those who have open wounds or sores, as well as those with urinary catheters, drips and dialysis access are also particularly prone to MRSA.

MRSA can also cause infections in people in the community, but much less commonly than in hospitalised people.

How is MRSA spread?

Direct skin-to-skin contact is the most effective route for MRSA to spread. Shared bedding, towels and clothes, and contact with discarded dressings that have been used by someone who has MRSA are other potential routes. Common measures to reduce transmission include:

- Hand washing and use of alcohol hand rubs
- Wearing gloves if in direct physical contact with a person with MRSA
- Avoiding sharing towels, facecloths, bedding and so forth with people who have MRSA
- Covering open wounds with waterproof adherent dressings.

MRSA infections in hospital can be kept to a minimum by using such hygiene measures, as well as isolating patients with MRSA into single or 'cohort' (group) rooms.

How is MRSA diagnosed and treated?

Carriers of MRSA are generally confirmed to be so by having a swab taken from the nose or skin.

If MRSA infection is suspected, a blood sample, urine specimen, body fluid or swab of a wound can be sent to the laboratory as guided by their clinical signs and symptoms. If *S aureus* is detected, further tests will determine which antibiotics will kill the bacteria.

Although *S aureus* is usually treated with antibiotics, many MRSA infections can only be treated with antibiotics that need to be given intravenously (through the vein). The most commonly used antibiotics include:

- Vancomycin – this requires close monitoring to ensure sufficient levels in the bloodstream to effectively fight infection
- Rifampicin – common side-effects include causing bodily secretions (tears and urine) to turn orange/pink and disturbance of normal liver function
- Linezolid – this is expensive, but is effective when taken orally; it can affect the bone marrow and platelet function
- Doxycycline.

Depending on where in the body the infection is, treatment can vary from days to several weeks



in duration. Common treatment durations include two weeks for CVC-related MRSA blood stream infection and at least six weeks for endocarditis and discitis.

There is not a universal screening programme in the UK. However, MRSA testing is currently recommended for people at high risk of colonisation; for example, patients being transferred from one hospital to another or re-admitted to the same hospital. Patients in renal, vascular surgery, dermatology and care of the elderly wards have been shown to have higher than average rates of colonisation. Screening is also recommended in people with a high risk of infection. Examples are those admitted to intensive care units, orthopaedic and vascular surgery wards, where they are at high risk of having invasive monitoring devices, or those with open wounds. These are all prone to becoming infected with MRSA.

MRSA screening for most people going into hospital in England and Wales (other than in an emergency) was introduced at the end of March 2009. Those excluded from screening are patients having a day-case operation on eyes or teeth, having an endoscopy as a day case, or having minor skin procedures. Children and pregnant women are also excluded from screening (unless the woman is having a planned caesarean section).

What are the implications?

Patients with CKD

CKD is the gradual and irreversible loss of kidney function, classified into five stages, with deterioration to stage 5 CKD being known as established renal failure (ERF). CKD patients, even before reaching ERF, have a two or three times greater risk of contracting skin and lung infections compared with the general population. They are also at increased risk of complications from these infections. If lung infection is caused by MRSA, the risk of death is 1.5–2 times greater than in patients without CKD. Patients with CKD are also more difficult to manage, because many of the drugs used to treat MRSA are cleared by the kidney and may, therefore, require dose modifications.

Haemodialysis patients

The majority of patients who reach ERF and who start renal replacement therapy have haemodialysis (HD). This form of dialysis requires access to the circulating blood volume to allow toxin removal by the 'artificial kidney'.

Vascular access

Veins are accessed in one of three main ways:

- Arterio-venous fistula (AVF) creation, in which an artery is joined to a vein, the high blood pressure in the artery causing the vein to become thickened and suitable for repeated insertion of dialysis needles
- Arterio-venous graft (AVG) creation, in which a man-made tube of refined plastic or Gore-Tex® is plumbed into the circulation, and acts as a puncture site for dialysis needle insertion
- CVC insertion, in which a plastic tube is passed into one of the large veins in the neck or groin. This allows the passage of blood directly from the blood vessels into the kidney machine. The plastic tubes, called 'lines', can either be 'tunnelled' under the skin, or non-tunnelled. Tunnelled lines are used in situations where dialysis is likely to be required for longer than in the acute settings, in which non-tunnelled lines are used. Both are regarded as a 'last resort' option of necessity and not one of choice.

Between four and eight per cent of all episodes of MSRA blood infections in the UK occur in patients who are on haemodialysis. This represents an approximately 100-fold increased risk relative to the general population. This risk directly relates to the type of vascular access being used; those with indwelling CVCs are up to eight times more prone to MRSA sepsis than patients with native AVFs or AVGs. Recent worldwide studies of multiple dialysis centres suggest that patients with a CVC are at a 41% higher risk of an infection-related death than dialysis patients using an AVF, this risk being even higher among diabetics. Patients with CVCs are particularly prone to the complication of infection entering the bloodstream and causing more serious infections by travelling to and infecting distant internal body parts.

The UK Renal Association sets guidelines relating to all aspects of caring for kidney patients. With regards to HD and reducing the risk of line-related blood infections, it recommends that in any dialysis unit, more than 85% of HD patients should be receiving dialysis through an AVF. The guidelines also state that among patients newly starting HD, 65% should do so through an AVF. This presents a challenge for renal doctors, with reasons cited for failing to meet these ambitious targets including late referrals to renal services from primary care or other specialties; delay in access to formation; lack of sufficient time for an AVF to develop and be useable; and increasing rates of vascular