Eur J Vasc Endovasc Surg **24**, 283–286 (2002)
doi:10.1053/ejvs.2002.1705, available online at http://www.idealibrary.com on IDEAL®

REVIEW ARTICLE

Methicillin-resistant *Staphylococcus aureus*: Vascular Surgeons Should Fight Back

J. J. Earnshaw

Gloucestershire Vascular Group, Gloucestershire Royal Hospital, U.K.

Methicillin-resistant Staphylococcus aureus *(MRSA) is now the commonest cause of serious vascular wound and graft infection in the U.K., and vascular departments in many other countries are similarly affected. There are no randomised trials that provide information about how to deal with this epidemic. There are, however, a number of clinical series that provide data that can be used to mount a logical and coherent response to the problem of preventing and managing MRSA infection. The risks and problems are different in every hospital and co-operation with local microbiologists is essential in creating individual protocols. Stratifying the risk to each patient is the first step; established antiseptic and surgical procedures are usually adequate as primary prevention. Studies into the role of targeted antibiotic therapy and isolation techniques are needed.*

Key Words: Vascular graft infection; Antibiotic prophylaxis; Methicillin-resistant Staphylococcus aureus.

Introduction

Vascular surgeons have adapted their practice to take into account the threat of wound and graft infection. Scientific studies have established the role of various prophylactic manoeuvres to minimise the risk of infection, yet graft infection still occurs at a rate of 3–5%.[1] In the last 8 years an epidemic of methicillin resistant *Staphylococcus aureus* (MRSA) infection has run rife through many hospitals and in many countries. It affects precisely the patients that vascular surgeons are called to treat: the elderly, the debilitated and those with ulcers, gangrene and chronic sepsis.

It is time for vascular surgeons to mount a coherent response in the battle against MRSA. Modern therapies rely on evidence from randomised clinical trials, but none exists. The only way to develop a strategy is to learn from the available studies and then add a large dose of common sense.

Please address all correspondence to: J. J. Earnshaw, Gloucestershire Vascular Group, Gloucestershire Royal Hospital, Great Western Road, Gloucester GL1 3NN, U.K.

The Problem with MRSA

A number of reviews have illustrated the effects of MRSA on vascular surgical practice.[2–5] In the United Kingdom (U.K.), there is evidence from the Public Health Laboratory Service that MRSA is causing an increasing number of infections,[6] peripheral vascular surgery and amputation are among the areas most affected (Fig. 1). Several large reviews from hospitals in the U.K. support this increase.[2–4] Most studies come from inner city teaching hospitals and it might be speculated that these high volume centres are the ones most at risk. The increasing incidence is paralleled by rising morbidity and mortality: patients with MRSA infection of a prosthetic infra-inguinal bypass graft have a high incidence of amputation,[3,4] MRSA aortic graft infection seems almost universally lethal,[3] and unusually, MRSA seems capable of causing primary infection of a native artery.[3] A multicentre survey done in 2000 by the Joint Vascular Research Group showed that MRSA was the current leading cause of vascular graft infection in the U.K. Patients with MRSA had a higher major amputation rate and prolonged hospital stay compared with other graft infections, however, mortality was not increased.[5]

1078–5884/02/040283 + 04 $35.00/0

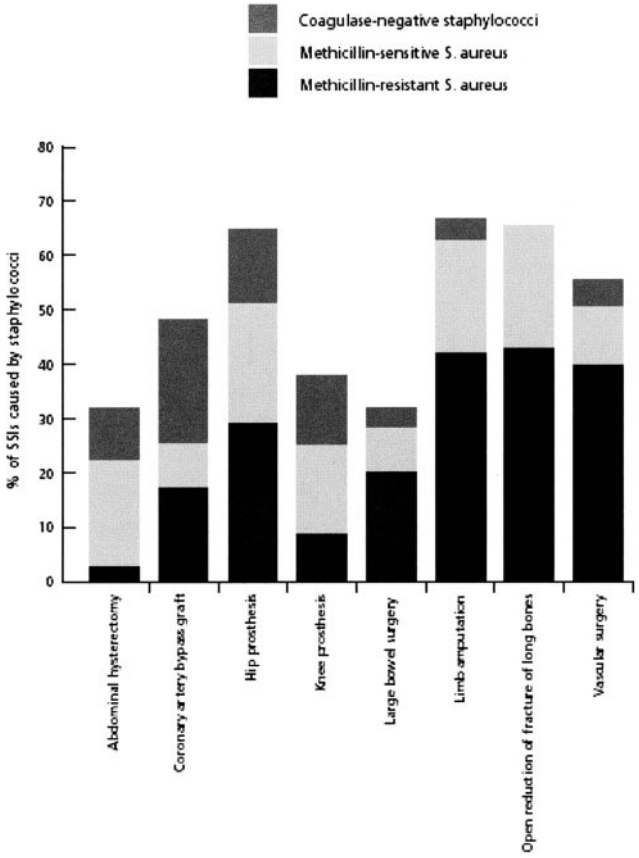

Fig. 1. Data from surveillance of surgical site infection in English hospitals 1997–9 demonstrating the high incidence of *Staphylococcus*, and in particular, MRSA infection in patients undergoing vascular or amputation surgery. Available online – www.phls.org.uk/publications/NINSS.htm

Much of the presented evidence derives from the U.K. but review of articles from Medline suggests that there is an increasing worldwide epidemic with this virulent organism.

Preventing MRSA infection in vascular patients requires adaptation of all the techniques vascular surgeons have used over the years to combat postoperative sepsis. The measures employed have to account for the varying risks of infection among the different vascular patients, and also the prevalence of MRSA in each hospital. Patients vary in risk, ranging from those who are not colonised with MRSA having a low risk procedure (elective aortic aneurysm repair), through those with known sepsis and possible, but undocumented MRSA colonisation or infection (patient admitted with gangrene from a nursing home, or who has previously been in hospital), to a patient with established MRSA infection who needs a vascular surgical procedure. Dealing with each of these categories requires variation on the same procedures.

Preoperative Measures

In a hospital with a low prevalence of MRSA, little needs to be done other than normal prophylaxis against infection. In a high prevalence hospital it makes sense to separate low risk patients preoperatively from those known to have, or be at risk of MRSA colonisation. Patients having elective aortic surgery or bypass for claudication should be admitted on the day of operation and into clean wards/bays. In a high prevalence hospital it is logical to keep patients at home as long as possible up until the day of surgery so that they cannot become colonised, particularly if they have an open ulcer or gangrene.

Patients with ischaemic rest pain, gangrene or ulcers are likely to have skin colonisation with bacteria (possibly including MRSA). The identification of MRSA colonisation/infection is important and many surgeons screen moderate or high risk patients either before, or as soon as they are admitted to hospital; patients admitted as an emergency out of hours must not be allowed to escape the system. Although it has not been possible in a randomised trial to show that reducing the bacterial load with bathing reduces the rate of wound or graft infection,[7] many surgeons employ preoperative antiseptic baths to try and cleanse the skin (chlorhexidine or triclosan are appropriate).

Patients with established MRSA infection are at high risk of infective complications after vascular reconstruction. There may be time to attempt to eradicate the infection before surgery but this is rarely possible in patients with established skin necrosis. Judicious debridement, use of systemic antibiotics for cellulitis or topical antibacterials (mupirocin) for infected open ulcers may reduce the bacterial load. Early reports suggest that debridement with larval therapy may be a way to eradicate MRSA.[8] Finally it may be appropriate to review the indication for vascular reconstruction; occasionally it may be possible to do a long subintimal angioplasty instead of a femorodistal bypass. Whilst vascular surgeons rarely like to admit defeat when a reconstruction is technically possible, in a patient with gangrene whose only hope is a high risk, distal prosthetic bypass, the risks of the reconstruction may outweigh the disadvantages of primary amputation. In this circumstance, MRSA may shift the balance of therapy against vascular reconstruction. In the modern era,

the burden of these complex decisions should be shared with the patients and their relatives.

Operative Measures

Whilst the use of autologous material for reconstruction is not a total protection against sepsis, clearly the important principle is that prosthetic material should seldom be employed in patients known, or suspected to have MRSA infection. Whereas this is not a new message for patients undergoing femorodistal bypass surgery, for other reconstructions greater ingenuity may be required; deep femoral vein is an alternative in patients who need an aortoiliac reconstruction. When conventional reconstruction is employed, standard surgical procedures such as oblique skin incisions in the groin and skin bridges in the thigh have been shown to reduce the rate of infection.[9] The development of antibiotic impregnated vascular grafts heralded much promise but it has not been possible to confirm their potential in randomised studies, and there is some evidence that rifampicin, the most commonly employed antibiotic, is relatively ineffective against MRSA.[10]

Most vascular graft infection is caused by implantation of bacteria at the time of vascular reconstruction. Therefore, prevention of contamination at operation is important. Whilst none of the available measures has basis in scientific trials, many surgeons employ occlusive wound drapes, antiseptic or antibiotic wound lavage, or implantation of antibiotic coated vehicles such as gentamicin collagen swabs or gentamicin beads. At least none of these measures has proved harmful.[11] One other way to reduce late recolonisation is to remove all infected or necrotic tissue at the end of the reconstruction operation.

Antibiotic prophylaxis is the one measure clearly shown to reduce the risk of wound, though not graft infection.[1,9] None of the available randomised trials studied MRSA infection specifically. Antibiotics effective against MRSA include teicoplanin, vancomycin and linezolid. Vancomycin is cheapest but the most toxic. Teicoplanin is safer but more expensive. Linezolid has the advantage that it is equally well absorbed orally as parenterally, however, the cost of the tablets is the same as the parenteral drug. All patients having a vascular reconstruction should receive antibiotic prophylaxis; those at risk of, or with established MRSA infection should have an antibiotic effective against MRSA included. Two studies in vascular surgical patients used teicoplanin, though neither trial was sufficiently powered to show a statistically significant benefit.[12,13] Each vascular unit should create its own protocol for antibiotic prophylaxis. This process should include local knowledge of the types and prevalence of MRSA infection and is facilitated by advice from a microbiologist. Whereas targeted prophylaxis is not indicated in low prevalence vascular units, it can be argued that patients with established MRSA infection should have a longer course of perioperative antibiotics than the standard one to three doses. New trials of antibiotic prophylaxis in high prevalence vascular units would be very helpful.

Postoperative Measures

It is unlikely that much can be achieved by varying postoperative care. Low risk patients should avoid contact with MRSA patients soon after surgery. A recent study documented that patients continue to pick up MRSA during their postoperative stay in ITU and back on the ward.[14] Dressings probably do not affect the outcome for a wound, though in high risk patients covering the wounds with an antibacterial dressing might help. Any wound infection should be treated promptly with help from a microbiologist. The lessons learned from the open clinical studies are that bold remedial surgery is less likely to be effective in a patient with deep MRSA wound or graft infection.[2–4] Revision therapy should include excision of all infected and necrotic material with recourse directly to major limb amputation, if necessary.

The Role of Isolation

Some hospitals practice isolation of patients with MRSA. This can only be done if facilities are available and if the burden of patients in a particular hospital is not too great; MRSA is endemic in many hospitals in the U.K. Isolation can reduce the opportunity for transmission of MRSA and was effective at St Mary's Hospital in reducing the rate of graft infection.[4] Transmission of MRSA can be minimised by isolating patients known to be colonised or infected with MRSA, but the availability of isolation rooms (usually side rooms off the main ward – few hospitals have an isolation unit) has long ago been outstripped by the number of colonised patients.

Nosocomial acquisition can be reduced by strict attention to handwashing and the appropriate use of other isolation nursing techniques such as wearing an apron, correct handling of linen and dressings. The

strictest measures, which may include the screening of staff for nasal or skin carriage can be applied in high risk areas of the hospital, which may include the vascular unit.[15] An alternative might be to create MRSA-free areas where patients having clean surgery may be housed.

Opinion is divided on whether attempts should be made to eradicate MRSA from clinical areas. Some North European countries have kept the infection under control with elimination and infection control policies.[16]

Conclusions

There are a number of simple measures that vascular surgeons can employ that might reduce the risk of MRSA infection. Just how much to do depends on the prevalence of MRSA in the individual unit. The first imperative is that vascular surgeons should take stock of the situation in their hospitals and agree a local response. This will require close collaboration with the hospital's microbiologists. Together, written protocols should be produced for dealing with each category of patient (low risk/high risk/known MRSA), including antibiotic prophylaxis. Junior staff should be trained to grade patients into risk categories on admission, just as they do for risk of deep vein thrombosis, as part of a care plan. Perhaps using this common sense approach, the MRSA epidemic in vascular patients could be controlled without waiting for the results of scientific studies.

Acknowledgements

I would like to thank Margaret Logan, one of our consultant microbiologists in Gloucester, and Dr Roger Bayston, consultant microbiologist at the City Hospital, Nottingham, U.K., for their help and advice in the preparation of this manuscript.

References

1 Bunt TJ. Vascular graft infections: an update. *Cardiovasc Surg* 2001; **9**: 225–233.
2 Murphy GJ, Pararajasingham R, Nasim A, Dennis MJS, Sayers RD. Methicillin resistant *Staphylococcus* aureus infection in vascular surgery patients. *Ann R Coll Surg Engl* 2001; **83**: 158–163.
3 Nasim A, Thompson MM, Naylor AR, Bell PRF, London NJM. The impact of MRSA on vascular surgery. *Eur J Vasc Endovasc Surg* 2001; **22**: 211–214.
4 Chalmers RTA, Wolfe JHN, Cheshire NJW *et al*. Improved management of infrainguinal bypass graft infection with methicillin-resistant staphylococcus aureus. *Br J Surg* 1999; **86**: 1433–1436.
5 Naylor AR, Hayes PD, Darke S on behalf of the joint Vascular Research Group. A prospective audit of complex wound and graft infections in Great Britain and Ireland: the emergence of MRSA. *Eur J Vasc Endovasc Surg* 2001; **21**: 289–294.
6 Communicable Disease Reports (available online) www.phls.co.uk/facts/bacteraemia/stap.htm.
7 Earnshaw JJ, Berridge DC, Slack RCB, Makin GS, Hopkinson BR. Do preoperative chlorhexidine baths reduce the risk of infection after vascular reconstruction? *Eur J Vasc Surg* 1989; **3**: 323–326.
8 Thomas S, Andrews AM, Hay NP, Bourgoise S. The antimicrobial activity of maggot secretions: results of a preliminary study. *J Tissue Viability* 1999; **9**: 127–132.
9 Ratliffe DA. New initiatives in the prevention and treatment of graft infection. In: Earnshaw JJ, Murie JA, eds. *The Evidence for Vascular Surgery*. TFM Publishing Ltd, Cheltenham 1999; **27**: 173–182.
10 Earnshaw JJ. The current role of rifampicin-impregnated grafts: pragmatism versus science. *Eur J Vasc Endovasc Surg* 2000; **20**: 409–412.
11 Benearts PJ, Ridler BM, Vercaeren P, Thompson JF, Campbell WB. Gentamicin beads in vascular surgery: long term results of implantation. *Cardiovasc Surg* 1999; **7**: 447–450.
12 Marroni M, Cao P, Florio M *et al*. Prospective randomised double blind trial comparing teicoplanin and cephazolin as antibiotic prophylaxis in prosthetic vascular surgery. *Eur J Clin Microbiol Inf Dis* 1999; **18**: 175–178.
13 Kester RC, Antrum R, Thornton CA, Ramsden CH, Harding I. A comparison of teicoplanin versus cephradine plus metronidazole in the prophylaxis of postoperative infection in vascular surgery. *J Hosp Inf* 1999; **41**: 233–244.
14 Scriven JM, Silva P, Swann RA *et al*. Acquisition of methicillin resistant *Staphylococcus aureus* in vascular patients. *Br J Surg* 2002; **89**: A510.
15 Working Party Report. Revised guidelines for the control of methicillin resistant *Staphylococcus aureus* infection in hospitals. *J Hosp Inf* 1998; **39**: 253–290.
16 van Belkum A, Verbrugh H. 40 years of methicillin resistant *Staphylococcus aureus*. *BMJ* 2001; **323**: 644–645.

Accepted 13 May 2002