Editorial

Learning to live with MRSA

Methicillin-resistant Staphylococcus aureus (MRSA) emerged in the 1960s, soon after methicillin was introduced for clinical use. Apart from occasional incidence or outbreak reports, it was not given much attention by healthcare staff until the late 1970s or 1980s, when several outbreaks were reported in various hospitals in Australia, and later in the UK and elsewhere. As one strain of a particular phage type was shown to spread very quickly in several hospitals in UK, this was called ‘epidemic’ strain or EMRSA. Soon, different strains appeared with similar abilities to spread, and therefore EMRSA in the UK began to be numbered, EMRSA-1 being the earliest epidemic strain of the 1980s. The number has now gone up to EMRSA-16. There are many other strains which do not belong to an EMRSA type or are not phage-typable. The spread of MRSA alarmed infection control practitioners worldwide, and various national guidelines appeared. Strict isolation of patients, preferably in an isolation unit, appropriate topical or systemic treatment, patient and staff screening, environmental sampling and disinfection, marking patients’ case notes and notification of transfer of positive patients, screening of new patients from hospitals in other areas, or even sampling patients on discharge—all have been suggested and practised, often at considerable expense and with the disruption of normal activities.

Were the measures successful or practicable?

Soon after the first UK guidelines appeared, strong critical views against them were expressed in a leading article. No other dissenting voices were heard for a few years. Meanwhile, newer strains continued to evolve and more and more hospitals or areas were now seeing MRSA. A continued increase in the number of MRSA cases has been observed worldwide, including the UK and US. In the author’s institution, MRSA constituted 1–2% of all Staphylococcus aureus up to 1994; 3% in 1995, and by the end of 1996, 9% (unpublished data). Initial reported successes at clearing MRSA after various treatments or control measures are now being neutralised by the findings that some strains, eg, EMRSA-1, disappear naturally, and that screening tests are not very reliable. One may have negative results for three consecutive weeks and positive results on the 4th or 5th weeks’ samples, or even after one year. Some individuals may be transient carriers, while others may carry the organism in unusual body sites (and not in the nose). This means that one has to continue sampling from multiple body sites for weeks or even months to be sure of reliable negative results, resulting in huge numbers of samples examined per patient in some cases. In our hospital, one patient needing repeated hospital admissions had nearly 200 samples examined for MRSA within a period of 18 months.

Apart from costs and inconvenience, many hospitals do not have adequate isolation facilities. Isolation of MRSA cases brings additional problems, unlike those with other organisms. For MRSA, there is no clear-cut end point of isolation as patients may continue carrying the organism for weeks, months, or even years. As many of them are elderly long-stay patients, the isolation may continue indefinitely. Harmful effects of prolonged isolation, eg, psychological problems and reduced level of healthcare, are such that sometimes isolation has to be discontinued (personal communications from infection control teams in various districts and personal observations over 10 years).

Thus, more voices are now heard again against the ‘search and destroy’ policy recommended by various guidelines and adopted by many infection control practitioners. The previously held view that hospitals are the source of MRSA must now change. The author has seen several cases from general practice who were not associated with any hospital. A recent study in nursing homes in a large city in the UK showed a high prevalence of MRSA. Thus, selective screening on admission may be impractical or illogical if the organism is already widely spread in the community, although there may be some merit in focussing attention on certain groups, depending on local risk assessment, eg, intensive care, transplant and cardiovascular units, as proposed in the draft revised UK guidelines (report of combined BSAC/HIS/ICNA MRSA working party, due for publication in 1998).

The first UK national guidelines of 1986 were revised in 1996. In 1996, modified guidelines, applicable to the community, were produced by the same group. As stated above, a further revision of the main guidelines is now expected to be published in 1998. This provides a further indication that the early measures were either not successful or not practicable.

Could the measures be justified?

A study of the many reported outbreaks demonstrates that, in the vast majority of cases, MRSA were just colonising or causing minor superficial infections. There have been some reports of severe infections or bacteraemia from MRSA, but no-one has convincingly demonstrated that MRSA were more virulent or pathogenic than other strains of S aureus (methicillin-sensitive or MSSA). The only reason for taking extra measures to control MRSA (as opposed to MSSA) is that they are more resistant to many common antibiotics. On this basis, one should take similar measures against Pseudomonas spp, coagulase-negative staphylococcus, and enterococcus groups of organisms which are similarly resistant to common antibiotics. Reports of the antibiotic resistance of MRSA were also somewhat exaggerated. Statements like ‘vancomycin is the only effective antibiotic against MRSA’ should now be challenged.

There is very little evidence, if any, from controlled comparative clinical trials to support that statement. In the author’s own experience, out of several hundred MRSA strains seen in his area over last 10 years, there was not a single strain which was not sensitive to at least five different classes of antibiotics available in the UK, and whenever treatment was necessary, antibiotics other than vancomycin were used. In fact, there are many published reports of successful use of other antibiotics against MRSA, eg, fusidic acid, rifampicin, minocycline, co-trimoxazole, cline-damycin, newer quinolones, aminoglycosides, teicoplanin, etc, while others, such as trimethoprim, nitrofurantoin, novobiocin, chloramphenicol, fosfomycin, etc, may be used in certain circumstances. Newer drugs in development, eg, glycyclines and quinupristin/dalfopristin, are also known to have good anti-MRSA activity. So there are several antibiotics available to treat MRSA infections. The situation does not really call for desperate measures.
Some authors believe that costs of not controlling MRSA are higher than those of control. The opposite argument may be equally valid, if not stronger. As serious infections do not occur in the vast majority of cases, and as there are several effective antibiotics, it might be cheaper and easier to treat clinical infections when they arise than trying routinely to identify and treat all colonised cases (contact tracing, isolation for indefinite periods, treating simple carriers, having extensive pre-admission screening programmes, and obtaining samples from staff and environment whenever two or more cases occur, etc.). It might be justified to introduce special measures in selected areas or circumstances, based on local risk assessment, but this will be no different from general principles of infection control for any organism.

Conclusion

It seems that the special measures introduced to control or eradicate MRSA have not succeeded. Considering the costs and difficulties with these measures, and the facts that MRSA has not proven to be any more pathogenic than other S. aureus strains and that a reasonable choice of antibiotics is available to treat MRSA infections, there is no justification to continue with the ‘search and destroy’ policy. It is time that we learnt to live with MRSA in the way we have been living with many other pathogens. MRSA should not be viewed any differently from other S. aureus strains in terms of treatment or infection control, ie,

positive action should be taken when there is evidence or strong suspicion of clinical problems. Routine measures to eradicate or ‘search and destroy’ whenever an MRSA is cultured, in the absence of clinical problems, waste valuable resources and time, and may even reduce the quality of healthcare.

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Key points

- there are many different strains of MRSA, with different antibiotic sensitivities and different pathogenic potential
- in the vast majority of cases, they have proved to be harmless commensals or caused minor superficial infections only
- various antibiotics are available to treat when clinical infections arise
- special measures to eradicate or control the spread of MRSA are disruptive, costly, not always successful and largely unnecessary
- for control of infections, basic principles should be similar to those for other S. aureus

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