

Please read this bit first

The HPCSA and the Med Tech Society have confirmed that this clinical case study, plus your routine review of your EQA reports from Thistle QA, should be documented as a “Journal Club” activity. This means that you must record those attending for CEU purposes. Thistle will **not** issue a certificate to cover these activities, nor send out “correct” answers to the CEU questions at the end of this case study.

The Thistle QA CEU No is: **MT00025.**

Each attendee should claim **THREE** CEU points for completing this Quality Control Journal Club exercise, and retain a copy of the relevant Thistle QA Participation Certificate as proof of registration on a Thistle QA EQA.

Cycle 21 Organism 10:

Methicillin-Resistant Staphylococcus aureus (MRSA)

Staphylococcus aureus are well documented as human pathogens. As a nosocomial pathogen, *S. aureus* has been a major cause of morbidity and mortality. *Staphylococcus aureus* causes a variety of suppurative (pus-forming) infections and toxinoses in humans. It causes superficial skin lesions such as boils, styes and furunculosis; more serious infections such as pneumonia, mastitis, phlebitis, meningitis, and urinary tract infections; and deep-seated infections, such as osteomyelitis and endocarditis. *S. aureus* is a major cause of hospital acquired (nosocomial) infection of surgical wounds and infections associated with indwelling medical devices. *S. aureus* causes food poisoning by releasing enterotoxins into food, and toxic shock syndrome by release of superantigens into the blood stream¹.

S. aureus expresses many potential virulence factors: (1) surface proteins that promote colonization of host tissues; (2) invasins that promote bacterial spread in tissues (leukocidin, kinases, hyaluronidase); (3) surface factors that inhibit phagocytic engulfment (capsule, Protein A); (4) biochemical properties that enhance their survival in phagocytes (carotenoids, catalase production); (5) immunological disguises (Protein A, coagulase, clotting factor); and (6) membrane-damaging toxins that lyse eukaryotic cell membranes (hemolysins, leukotoxin, leukocidin^{1,2}.

S A N A S



PROFICIENCY TESTING SANAS Accredited to ISO Guide 43 / ILAC G13

Hospital strains of *S. aureus* are usually resistant to a variety of different antibiotics. A few strains are resistant to all clinically useful antibiotics except Vancomycin. The term MRSA refers to Methicillin resistant *Staphylococcus aureus*. Methicillin resistance is widespread and most methicillin-resistant strains are also multiply resistant. Isolates that carry the *mecA* gene, or that produce PBP2a (the *mecA* gene product), should be reported as oxacillin resistant. A surrogate disk containing 30µg of ceftiofuran has been recommended to replace the oxacillin disk for testing for MRSA. A plasmid associated with vancomycin resistance has been detected in *Enterococcus faecalis*, which can be transferred to *S. aureus* in the laboratory, and it is speculated that this transfer may occur naturally (e.g. in the gastrointestinal tract). In addition, *S. aureus* exhibits resistance to antiseptics and disinfectants, such as quaternary ammonium compounds, which may aid its survival in the hospital environment.

Hospital acquired infection is often caused by antibiotic resistant strains (MRSA) and can only be treated with vancomycin or an alternative. Until recently, infections acquired outside hospitals have been treated with penicillinase-resistant β-lactams. However, many of the community acquired (CA) Staphylococcal infections are now methicillin resistant. Particularly in Georgia, Texas, and California, the prevalence of CA-MRSA is widespread. Over 60% of abscess isolates from the emergency department of an Austin, Texas hospital yielded MRSA. These organisms are uniformly resistant to penicillins and cephalosporins. The glycopeptide vancomycin has been regarded as the drug of choice for the treatment of infections caused by MRSA. Other antimicrobials that have been used for the treatment of MRSA strains, include linezolid, quinupristin/dalfopristin, daptomycin, semisynthetic glycopeptides and glycycline^{3,4}.

References

1. Lowry FD. 1998. *Staphylococcus aureus* infections. *N Eng J Med*. 339: 520-531.
2. Bannerman. *Staphylococcus, Micrococcus, and Other Catalase-Positive Cocci That Grow Aerobically*. In: Murray PR, et al. Eds. Manual of Clinical Microbiology. 8th ed. Washington DC: ASM Press; 2003: 384-404.
3. Rohrer, MM et al. 2001. Improved methods for detection of methicillin-resistant *S. aureus*. *Eur. J. Clin. Microbiol. Infect. Dis*. 20:267-270.
4. Wenzel, RP. et al. 1998. Methicillin-resistant *S. aureus* outbreak: a consensus panel's definition and management guidelines. *Am. J. Infect. Control*. 26:102-110.

Questions

1. How does *S. aureus* differ from the other staphylococci?
2. What is the mechanism of methicillin-resistance in *S. aureus*?
5. How would you test in the laboratory for methicillin-resistance?