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Please read this bit first

This CPD/ CEU exercise is designed to take approximately two hours as a small group exercise within your laboratory. The Thistle QA CPD No is: **MT00025.**

Please keep a register of those taking part in the exercise. When the exercise is completed, please ask using the above email address, and we will send you a sheet showing the correct responses to each question.

Each attendee should claim two CPD points for completing the questions correctly, by retaining a copy of the relevant Thistle QA Participation Certificate as proof of registration on a Thistle QA EQA.

Cycle 19 Organism 10.

The causative organism was Methicillin-Resistant *Staphylococcus aureus*

Staphylococci are Gram-positive spherical bacteria that occur in microscopic clusters resembling grapes. Bacteriological culture of the nose and skin of normal humans invariably yields staphylococci. In 1884, Rosenbach described the two pigmented colony types of staphylococci and proposed the appropriate nomenclature: *Staphylococcus aureus* (yellow) and *Staphylococcus albus* (white). The latter species is now named *Staphylococcus epidermidis. M*ore than 20 species of *Staphylococcus* are described in Bergey's Manual (2001) Taxonomically, the genus *Staphylococcus* is in the Bacterial family *Staphylococcaceae,* which includes three lesser known genera, *Gamella, Macrococcus* and *Salinicoccus.*

Pathogenesis of *S. aureus* infections

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 stream.
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**; (7) exotoxins that damage host tissues or otherwise provoke symptoms of disease (**SEA-G**, **TSST**, **ET** (8) inherent and acquired **resistance to antimicrobial agents**.

Resistance of Staphylococci to Antimicrobial Drugs

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, and vancomycin-resistant strains are increasingly-reported. 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 *mec*A gene, or that produce PBP2a (the *mec*A gene product), should be reported as oxacillin resistant.

A surrogate disk containing 30µg of cefoxitin 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.

Staphylococcal disease has been a perennial problem in the hospital environment since the beginning of the antibiotic era. During the 1950's and early1960's, staphylococcal infection was synonymous with nosocomial infection. Gram-negative bacilli (e.g. *E. coli* and *Pseudomonas aeruginosa*) have replaced the staphylococci as the most frequent causes of nosocomial infections, although the staphylococci have remained a problem, especially in surgical wounds.. *S aureus* responded to the introduction of antibiotics by the usual bacterial means to develop drug resistance: (1) mutation in chromosomal genes followed by selection of resistant strains and (2) acquisition of resistance genes as extrachromosomal plasmids, transducing particles, transposons, or other types of DNA inserts. *S. aureus* expresses its resistance to drugs and antibiotics through a variety of mechanisms.

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 ß-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.

References

1. Tenover, FC *et al*. 2001. Increasing resistance to vancomycin and other glycopeptides in *S. aureus. Emerging. Infect. Dis.* 7:327-332
2. Rohrer, MM *et al.* 2001. Improved methods for detection of methicillin-resistant *S. aureus. Eur. J. Clin. Microbiol. Infect. Dis.* 20:267-270.
3. Wenzel, RP. e*t al.* 1998. Methicillin-resistant *S. aureus* outbreak: a consensus panel's difinition and management guidelines. *Am. J. Infect. Control.* **26:**102-110.

CPD Questions.

1. How does *S. aureus* differ from the other staphylococci*?*

2. What is the mechanism of methicillin-resistance in *S. aureus*?

3. How would you test in the laboratory for methicillin-resistance?