Over recent years, *Staphylococcus aureus* with reduced vancomycin susceptibility (SA-RVS) has become an emerging issue among hospitalised patients, and the first Australian case was reported from our institution.1 SA-RVS includes vancomycin-intermediate *S. aureus* (VISA; MIC to vancomycin of 8–16 µg/ml), vancomycin-resistant *S. aureus* (VRSA; MIC = 32 µg/ml) and heterogeneous VISA (hVISA). The latter is defined by the presence of sub-populations of MRSA, typically at a rate of one in every 10⁵ to 10⁶ cells, which express intermediate vancomycin resistance.1,2 The clinical significance of infections due to hVISA has been debated but there have been reports where vancomycin failure has occurred.2-10 A complicating factor is that the laboratory detection of hVISA is difficult. The gold standard for detection is population analysis profile (PAP), but the use of Etest (AB Biodisk) with both standard and heavy inocula appears to be a useful screening technique.11

Over a 12-month period at our institution, we performed a detailed analysis of all episodes of methicillin-resistant *S. aureus* (MRSA) bacteraemia to assess the frequency of hVISA infections amongst these and to identify whether there are clinical features that differentiate bacteraemias due to hVISA from those due to vancomycin-susceptible MRSA (VS-MRSA). This study has recently been published in full.12

There were 53 episodes of MRSA bacteraemia identified in the study period. Of these, 5 (9.4%) were found using PAP to have hVISA, whilst the remaining 48 (90.6%) had VS-MRSA. A PAP area under curve (AUC) result of ≥0.9 is required to meet the definition of hVISA,13 and the five hVISA isolates had PAP AUC results of 0.91, 1.14, 1.17, 1.37 and 1.43. By broth microdilution, the vancomycin MIC results of the hVISA isolates had PAP AUC results of ≤0.9 and had vancomycin MIC results of 0.5 - 2 µg/ml (median 1 µg/ml). Using Etest analyses, the hVISA isolates had MIC results of 4 µg/ml, whereas the VS-MRSA isolates had levels between 0.5 and 2 µg/ml (median 1.5 µg/ml).

“The presence of hVISA was associated with clinical evidence of vancomycin treatment failure”

Looking at the clinical features of the two groups, they were not significantly different in age, co-morbidities, history of prior MRSA infections, or prior antibiotic use. None of the hVISA patients had received vancomycin prior to the bacteraemic illness that was studied. However, there were significant differences between the two groups in the response to vancomycin. Patients with hVISA had a longer duration of fever (35 vs. 3 days; p<0.001), greater number of positive blood cultures (8 vs.2; p<0.001), longer time to clearance of bacteraemia (39 vs. 6.4 days; p=0.002) and longer in-hospital length of stay (107 vs. 37 days; p=0.006). Defining treatment failure as ongoing fever as well as bacteraemia at least one week after starting vancomycin therapy, the hVISA patients were more likely to fail (5/5 vs.1/48; p<0.001). Notably, more hVISA patients had low (<10 µg/ml) trough vancomycin levels during the first week of therapy than VS-MRSA patients (5/5 vs. 11/36; p=0.006), but despite subsequent dose adjustments to ensure appropriate vancomycin levels, patients with hVISA still clinically failed vancomycin therapy. Also, the hVISA patients had infections with a high bacterial load (HBL), defined as the presence of undrained MRSA collections, infected prosthetic material or echocardiographic evidence of endocarditis (5/5 vs. 10/48; p=0.001). The presence of infected prosthetic material was common to both groups. All five of the hVISA patients and 32/48 VS-MRSA patients had prosthetic material in situ and a comparable number could not be removed promptly (2/5 vs. 7/32; p=0.6). Four of the hVISA patients were changed to linezolid and all had prompt responses in terms of controlling their infections (even when prosthetic material was not removed). Mortality for the groups was similar.

We believe this study has some important implications for clinicians. We found the presence of hVISA to be associated with clinical evidence of vancomycin treatment failure. In particular, persistent fever and bacteraemia for ≥7 days after commencing vancomycin was significantly associated with its presence. Also, the association

---

Also in this issue

- Treatment of hVISA infections ................................................................. 2
- The QHPSS antibiogram: a practical system for passive susceptibility surveillance? ................................................................. 4
- AGAR update .......................................................................................... 6
- Picture quiz answer: an unusual isolate of MRSA ............................................ 8
between HBL infections and presence of hVISA emphasises the important role of surgical debulking of these infections (including removal of infected prosthetic material) to optimise antibiotic efficacy. Furthermore, in the presence of HBL infections, the low trough serum vancomycin concentrations observed in patients with hVISA patients during the first week of therapy could logically result in either induction of any pre-existing vancomycin resistance or potentially lead to the selection of new vancomycin-resistant strains. The presence of reduced susceptibility to vancomycin among these strains appears to be important, as vancomycin failed even when dose adjustments were made to ensure appropriate vancomycin concentrations. Finally, we found the currently recommended laboratory screening methods for hVISA to be insensitive, when compared to results obtained by PAP testing. An MIC to vancomycin of >4 μg/ml using Etest with a standard inoculum would have differentiated between hVISA and VS-MRSA with reasonable accuracy in our patients.

Limitations of our study include its relatively small size and retrospective design, such that observed associations may not absolutely confer clinical predictive value. A prospective study is planned so that we can control for the potential confounding between the presence of infected prosthetic material, treatment failure with vancomycin, the number of blood cultures obtained and the rate of identified bacteraemia.

In the meantime, we believe clinicians should consider the presence of HBL infections and persistent fever and bacteraemia ≥7 days after commencing vancomycin therapy as potential markers of hVISA infection. Given the current difficulties in identifying a reliable screening test for hVISA and the time-consuming nature of PAP analyses, such clinical markers may assist laboratories in targeting diagnostic efforts and clinicians in reassessing treatment options for this important emerging pathogen. For a complete description of the study, readers are directed to the original paper.

“Vancomycin therapy failed in the hVISA group even when doses were adjusted to ensure appropriate vancomycin concentrations”

References

Treatment of heterogenous vancomycin - intermediate Staphylococcus aureus (hVISA) infections

B Howden, P Ward, P Charles, ML Grayson
Depts of Infectious Diseases and Microbiology, Austin Health, Vic

Although infections caused by heterogenous vancomycin-intermediate Staphylococcus aureus (hVISA) have been reported from a number of countries, including Australia, the optimal therapy is unknown. We reviewed the clinical features, therapy and outcome of patients with serious infections due to hVISA in Australia and New Zealand. The results of this study have recently been published. Initially the clinical significance of hVISA strains was debated, but there is increasing data associating hVISA infections with glycopeptide treatment failures, and higher in-hospital patient mortality than patients infected with vancomycin-susceptible MRSA. Serious clinical infections with S. aureus with reduced vancomycin susceptibility, both vancomycin intermediate S. aureus (VISA) and hVISA, have now been reported in many countries, yet treatment efficacy has not been systematically assessed. Our study reviewed the treatment and outcomes of 25 patients. All patients with a sterile site isolate confirmed as hVISA by population analysis profile testing and calculation of the area under the curve (AUC) of test strains and Mu3 control was performed as previously described. The ratio of AUC of the test isolate to that of Mu3 was calculated (PAP AUC ratio). hVISA was defined as an isolate of S. aureus with a PAP AUC ratio of ≥ 0.9.

Twenty-five clinical treatment questionnaires were completed. There were 16 males and 9 females. The median age was 65 years. All 25 patients had received glycopeptide therapy at some time prior to the diagnosis of hVISA. The source of the hVISA infection was typical of hospital-acquired infections (e.g. surgical wound infections, central intravenous line infections). The types of infections (summarised in table 1) included 17 patients with bacteraemia, 6 patients with osteomyelitis and/or septic arthritis without bacteremia, and 2 patients with post-surgical empyema. Prolonged bacteremia whilst receiving vancomycin therapy was common among patients with endocarditis and those
with osteomyelitis and/or septic arthritis (table 2). Nineteen of 25 patients (76%) failed glycopeptide therapy.

Active antibiotic therapy was used in 21 of the 25 patients, while four patients received either minimal or no therapy directed at their infection. In the 21 actively treated patients, a number of antibiotic combinations were used, but treatment with linezolid was most common (n=18), either alone (n=9) or sequentially with other agents, especially rifampicin and fusidic acid (n=8). One patient was treated with fusidic acid and chloramphenicol after initial control with linezolid, because of resistance to rifampicin and all other available agents. Notably, one patient with osteomyelitis and septic arthritis of the 1st metatarsophalangeal joint was treated (and cured) with 11 weeks of vancomycin therapy. Of the 21 actively treated patients, 16 (76%) had an effective response to therapy. Seven patients (33%) had died at follow up. The duration of linezolid treatment varied (range 7-78 days), but was generally prolonged. Side effects whilst receiving linezolid occurred in eight patients and included thrombocytopenia (n=5), abnormal liver function tests (n=1), nausea (n=1), and taste disturbance (n=1). Surgery was an important component of therapy in 13 patients in the actively treated group. The surgery usually involved significant debulking of infection (eg. vegetectomy, debridement and removal of infected prosthetic devices, drainage of multiple liver abscesses), and, in some cases, repeated surgery was required.

In summary, we found that serious infections due to hVISA in Australasia were generally identified in patients in whom glycopeptide treatment had failed. Nevertheless, the majority of these patients could be effectively treated with either antibiotics alone, or in combination with surgical debulking of the infected site. Linezolid was most commonly used, particularly for initial therapy, followed in some patients by a switch to combination rifampicin plus fusidic acid. Overall, adverse reactions were uncommon. The mechanisms of vancomycin resistance in S. aureus are not known, although a thickened cell wall is a common feature and this appears to be induced by vancomycin. Consistent with this observation, most of the patients in the study had previous MRSA infection and glycopeptide exposure. This has implications in terms of the emphasis placed on surgical drainage and debulking of high bacterial load infections in such patients, and possibly on vancomycin dosing. Our study suggested that hVISA is not just a laboratory phenomenon, but that such strains are associated with serious infections which respond to carefully targeted non-glycopeptide therapy. Complete details of this study are available in a recent publication.1

Acknowledgements
The hVISA study group included; T. Korman, Southern Health; E. Grabsch, P. Johnson, B. Mayall, Austin Health; A. Fuller, P. du Cros, Alfred Hospital; S. Roberts, A. Morris, Auckland District Health Board; J. Robson, Sullivan Nicolaides Pathology; K. Read, North Shore Hospital; J. Hurley, Ballarat Health Services; N. Bak, Western Hospital.

References

“Most patients were effectively treated with either antibiotics alone, or in combination with surgical debulking of the infected site”

“Serious hVISA infections may respond to carefully targeted non-glycopeptide therapy”

Table 1. Summary of infection types and outcomes

<table>
<thead>
<tr>
<th>Treatment category</th>
<th>Infection Type</th>
<th>Patients (n)</th>
<th>Glycopeptide failure (n)</th>
<th>Effective therapy (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actively treated</td>
<td>Endocarditis</td>
<td>8</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Bacteraemia</td>
<td>6</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Bone / joint infection</td>
<td>5</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Empyema</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Bacteraemia</td>
<td>3</td>
<td>2</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>Prosthetic joint infection</td>
<td>1</td>
<td>0</td>
<td>N/A</td>
</tr>
<tr>
<td>Not treated</td>
<td>Bacteraemia</td>
<td>3</td>
<td>2</td>
<td>N/A</td>
</tr>
<tr>
<td></td>
<td>Prosthetic joint infection</td>
<td>1</td>
<td>0</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Table 2. Duration of bacteraemia while receiving vancomycin in patients with endocarditis and bone and joint infections

<table>
<thead>
<tr>
<th>Disease</th>
<th>Patients (n)</th>
<th>Median days of bacteraemia (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocarditis</td>
<td>8</td>
<td>13 (7-32)</td>
</tr>
<tr>
<td>Osteomyelitis and septic arthritis</td>
<td>4</td>
<td>9.5 (5-33)</td>
</tr>
</tbody>
</table>
It is widely recognised that passive surveillance of antimicrobial susceptibility is a useful tool in the fight against the spread of antimicrobial resistance. While many individual laboratories are able to produce local cumulative susceptibility summaries, it has not been possible so far to maintain a national system of passive surveillance. Passive surveillance systems have many potential uses. They have the potential to document in a timely fashion trends of increasing or decreasing resistance to particular antimicrobials in individual organisms or a range of organisms. It can be argued that they are an essential element in monitoring the effectiveness of programs designed to reduce the prevalence of antimicrobial resistance.

Concerns have been raised about the quality of data generated by passive surveillance compared with that of active or targeted surveillance. Issues include diversity of susceptibility testing methods, lack of uniformity in identification of microorganisms and differing regimes of quality assurance. Strategies for dealing with these limitations need to be included in the design of an effective system. However, comparison of passive and active surveillance of resistance in Escherichia coli in the United Kingdom supports the utility of passive surveillance as a relatively inexpensive and reliable means of monitoring trends.1 Passive surveillance data have been reported more extensively in the United States. For example, an examination of trends in resistance of urinary isolates of E. coli has been used to validate therapeutic recommendations2 and aggregated hospital antibiogram data has been used to monitor regional trends in resistance to penicillin and macrolides in Streptococcus pneumoniae3.

In Australia, the development of a practical and sustainable system for passive surveillance of antimicrobial susceptibility has been problematic. The National Antimicrobial Resistance Surveillance Program (NARSP) of the early to mid 1990s proved impossible to maintain due to the laborious and time-consuming nature of data acquisition. Establishment of an Australian arm of The Surveillance Network® (TSN®) in the late 1990s promised to provide the timely passive surveillance recommended in the JETACAR Report.4,5 Unfortunately, this commercial venture was not viable in Australia and no longer operates here.

“We believe this system has the potential to fulfill the recommendation for national passive surveillance contained in the JETACAR report”

Clinical demand for local antibiograms and a commitment to monitoring regional and supra-regional trends in antimicrobial susceptibility have led Queensland Health Pathology and Scientific Services (QHPSS) to develop its own state-wide system of passive surveillance. Susceptibility data are downloaded from the QHPSS laboratory information system (LIS), imported into a relational database (Microsoft® SQL Server 2000®) and screened for duplicates (Figure 6). Results are presented with a 3-tiered-architecture web application developed using Microsoft® Dot Net™ technologies. Web-browser access is provided via the Queensland Health intranet or secure internet connection (Figure 2). Data are obtained from 24 Queensland Health Pathology Service (QHPS) laboratories throughout the state.

Ad hoc inquiries can be made by specifying a number of parameters including period (month or year), specimen type, organism name, antibiotic, region, health care facility, testing laboratory, clinical unit, ward of collection and inpatient or outpatient status. Figures 3 to 6 show examples of ad hoc inquiries to illustrate a number of these features. It is also possible when specifying specimen type to distinguish between clinical isolates and infection control screening isolates (Figure 6).

The use of a robust industry standard relational database gives the system the capacity for enlargement to handle data input from other laboratory information systems. The web application will allow remote inquiry access with appropriate levels of security and confidentiality. Therefore, we believe this system has the potential to fulfill the recommendation for national passive surveillance contained in the JETACAR report. A submission has been made to the Department of Health and Aged Care for funding of this national initiative.

References
Figure 1. Exclusion criteria for duplicate isolates

- Same patient identifiers (name, record number, date of birth)
- Same organism name
- Same susceptibility pattern
- Isolation within 5 days (rolling algorithm) of previous isolate with same criteria
- Same specimen type included for specimen specific data sets

Figure 2. Antibiogram system architecture

Figure 3. *Streptococcus pneumoniae* antibiogram for all QHPS laboratories and all antibiotics tested, Jan - June, 2003

Figure 4. Susceptibility of blood culture isolates to vancomycin in a metropolitan teaching hospital laboratory, Jan - June 2003

Figure 5. Antibiogram of all MRSA isolated from a metropolitan teaching hospital, Jan - June 2003

Figure 6. Antibiogram of infection control screening isolates of MRSA from a metropolitan teaching hospital, Jan - June 2003

2004 AstraZeneca ASA ICAAC Travel Award

AstraZeneca have once again kindly agreed to fund the AstraZeneca ASA ICAAC Travel Award in 2004. This Award will be for attendance at ICAAC (Interscience Conference on Antimicrobial Agents and Chemotherapy) in Washington DC in November 2004. The Award consists of return economy airfare, accommodation and conference registration to attend ICAAC. ASA members who wish to apply for the award are invited to submit their ICAAC abstracts to the ASA secretary, Dr Wendy Munckhof at wendy_munckhof@health.qld.gov.au. Members are reminded that the electronic abstract submission deadline for ICAAC is May 7th, 2004. We thank AstraZeneca for their ongoing support.
The Australian Group for Antimicrobial Resistance (AGAR) recently received 12 months funding from the federal government to maintain its activities for the financial year commencing 1 July 2003. To facilitate this funding, AGAR, although remaining a separate identity, now falls under the auspices of ASA and the Chairperson of AGAR (A/Prof Peter Collignon) has become a co-opted member of the ASA Committee. Consequently, the funding agreement for AGAR’s activities was made between the Commonwealth of Australia (represented by the Department of Health and Ageing) and the Australian Society for Antimicrobials. This agreement was supported by the AGAR Executive and the ASA Committee and was signed on behalf of ASA by the Secretary A/Prof Keryn Christiansen. All financial matters relating to the agreement are administered by the Treasurer of ASA Mr Geoffrey Coombs.

This funding will allow AGAR to:
• Employ a 0.5FTE Scientist (Ms Julie Pearson, Royal Perth Hospital) to analysis and prepare data from the surveillance programmes conducted by AGAR
• Hold two committee meetings including a half day scientific meeting
• Reimburse participating laboratories for consumables used in performing the Staphylococcus aureus and Gram Negative Bacteria Surveys
• Perform epidemiological typing of MRSA

Background
AGAR was formed in 1985 as an initiative of Lilly Industries Pty Ltd and was originally known as the “Staphylococcus Awareness Program”. The group consists of a mix of clinical and scientific members. Although antimicrobial resistance in S aureus has been the major research interest of AGAR, other surveys conducted have included:
• Haemophilus influenzae
• E coli, Klebsiella and Enterobacter species
• Streptococcus pneumoniae
• Enterococcus species

Committee members
The Committee consists of 42 participants from 25 institutions including:
• ACT
  The Canberra Hospital
• New South Wales
  South Western Area Pathology Service
  Royal North Shore Hospital
  Royal Prince Alfred Hospital
  Westmead Hospital
  Concord Repatriation General Hospital
  Prince of Wales Hospital
  Nepean Hospital
  Douglas Henley Moir Pathology
• Northern Pathology
  Royal Darwin Hospital
• Queensland
  QHPS, Princess Alexandra Hospital
  Royal Brisbane Hospital
  Sullivan Nicolaides Pathology
• South Australia
  Institute of Medical and Veterinary Science
  Flinders Medical Centre
  Gribbles Pathology
• Tasmania
  Royal Hobart Hospital
• Victoria
  Alfred Hospital
  Royal Childrens and Womens Hospital
  St Vincent’s Hospital
  Gribbles Pathology
• Western Australia
  PathCentre
  Fremantle Hospital
  Royal Perth Hospital
  Saint John of God Pathology , WA

AGAR executive committee
The Group’s activities and planning of antimicrobial surveys and annual scientific meetings are formalised by an Executive elected by the AGAR Committee. The current Executive consists of:
• Chairperson
  Peter Collignon
• Deputy Chairperson
  Graeme Nimmo
• Secretary/Treasurer
  Geoffrey Coombs
• Minute Secretary (non elected position)
  Julie Pearson
• Principal Scientific Convenor
  Iain Gosbell
• Scientific Convenors
  Keryn Christiansen
  Tom Gottlieb
  Clare Franklin
• Executive Members
  John Turnidge
  Jan Bell

2003/2004 surveys
Antimicrobial surveillance programmes planned for 2003/2004 include:
• S. aureus (commencing 01/11/03)
• E. coli, Klebsiella and Enterobacter species (commencing 01/01/04)

Web page
The AGAR webpage was originally established by Gary Lum. With support from Astra Zeneca, the webpage (www.antimicrobial-resistance.com) is currently undergoing reconstruction. It is anticipated that when completed data from previous surveys will be available.

Scientific meeting
The next AGAR scientific meeting will be held at the Novotel, Brighton Beach Sydney on Thursday 1330 – 1700, 22nd April 2004. Non AGAR members are invited to attend, however a registration fee of $10 per delegate is required to cover costs. For information regarding the agenda, please contact Iain Gosbell (i.gosbell@unsw.edu.au). To assist with the preparation of the meeting including catering requirements, non AGAR members are requested to contact Geoffrey Coombs (geoffrey.coombs@health.wa.gov.au) before 15th April 2004.
A 77 year old man presented to his local GP with symptoms of a urinary tract infection. He had a history of recurrent urinary tract infections following the diagnosis of bladder cancer 2 years earlier.

Urine microscopy: WBC 220 x 10^6/L, RBC > 1000 x 10^6/L, epithelial cells = < 10 x 10^6/L. *Klebsiella pneumoniae* was isolated from his urine, although the organism would not grow on horse blood agar but grew well on CLED (Cystine Lactose Electrolyte Deficient) agar (Oxoid).

**Figure 1** shows Mueller Hinton agar with growth of the organism at 24 hrs up to and around the trimethoprim 5ug disc (4 o’clock), then clockwise cefalothin 30 ug, amoxicillin/clavulanic acid 30 ug, ampicillin 10 ug, nitrofurantoin 300 ug and norfloxacin 10ug.

**Figure 2** shows Mueller-Hinton agar with growth of the organism at 24 hrs around the edge of sulfamethoxazole and trimethoprim disc 25 ug (4 o’clock), then clockwise cefazidime 30ug, timentin 85ug, ceftriaxone 30ug, tobramycin 10ug and gentamicin 10ug.

- Why does this *Klebsiella pneumoniae* grow on CLED agar but not on Horse blood agar?
- Why is the organism trimethoprim-dependent? Is this a common laboratory finding?
- How should susceptibility testing be reported for this organism?

Please email your responses to the ASA Newsletter Editor Ronan Murray at ronan.murray@health.wa.gov.au. Answers will be published in the next issue, and correct responses will be acknowledged.

*Picture quiz provided by Drs. Jeannie Botes and Jenny Robson of Sullivan Nicolaides Pathology, Brisbane.*
In July 2003 an elderly man presented to hospital with a purulent discharge from a suprapubic catheter site. This was swabbed for culture.

The swab was inoculated onto horse blood agar and MacConkey plates. After 24 hours incubation at 35°C, there was a pure growth of *S aureus* and Group G *Streptococcus*. Figure 1 shows a disc susceptibility test for *S aureus* (penicillin 10 ug disc on the left, tetracycline 10 ug disc on the right). Figure 2 shows an oxacillin E test for *S aureus*.

**Comment on the disc sensitivity patterns in Figure 1 and the E test result in Figure 2.**

The original *S aureus* isolate was NCCLS disc 10ug penicillin sensitive and 1ug oxacillin resistant.

**What needs to be done to sort out this finding?**

Nitrocefin beta-lactamase testing was negative and *mec A* (Oxoid latex test) was negative. Induction of beta-lactamase using a 0.5mg/L methicillin plate at 35°C showed the consistent emergence of 2 colony types, small and large.

The results of the tests on the original isolate and subsequent subcultures are tabulated below (Table 1). Comparative testing with ampicillin and amoxicillin / clavulanic acid did not confirm hyper-beta-lactamase production.

The antibiotic sensitivities of the original *S aureus* isolate using the NCCLS method were: Penicillin S, Tetracycline R, Erythromycin S, Fusidic acid R, Rifampicin S, Gentamicin S, Oxacillin R

In conclusion, this organism is a heterogenous (h)MRSA strain. The organism has been stored as no similar isolates have been seen previously in this laboratory.

**Table 1. Results of tests on the original isolate and subsequent subcultures**

<table>
<thead>
<tr>
<th>Test</th>
<th>Original isolate (large/mix)</th>
<th>Small colony form</th>
<th>Large colony form</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillinase (nitrocefin) prior to induction with low dose methicillin</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Penicillinase post induction with low dose methicillin (0.5mg/L)</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Zone of inhibition to penicillin 10ug disc (NCCLS)</td>
<td>Large &gt;22mm</td>
<td>Large &gt;22mm</td>
<td>Large &gt;22mm</td>
</tr>
<tr>
<td>Zone of inhibition to oxacillin 1ug disc (NCCLS)</td>
<td>&lt;5mm</td>
<td>&lt;5mm</td>
<td>&lt;5mm</td>
</tr>
<tr>
<td><em>Mec A</em></td>
<td>Negative</td>
<td>Positive</td>
<td>Negative</td>
</tr>
</tbody>
</table>

*Picture quiz provided by Dr Joan L Faoagali and Jan Bodman, Royal Brisbane Hospital. Photographs by Cassy Faux.*