Fig. 1 Resistance genes detected in third generation cephalosporin nonsusceptible (R, black bars) and susceptible (S, grey bars) *E. coli* isolates from urinary specimens.

fibrosis.¹⁰ Fosfomycin offers an additional option but gastrointestinal side effects are common and the development of resistance is commonly observed.¹¹

Pivmecillinam has a low prevalence of gastrointestinal side effects, can be safely used in renal impairment and has a high barrier to resistance.^{6,12} Australian laboratories should consider testing for mecillinam susceptibility in *E. coli* isolates from patients with recurrent, problematic multidrug resistant urinary tract infections.

Conflicts of interest and sources of funding: The authors state that there are no conflicts of interest to disclose.

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DOI: <https://doi.org/10.1016/j.pathol.2020.10.016>

Discrepancy between VITEK2 and Etest aminoglycoside susceptibility testing for multidrug-resistant *Acinetobacter baumannii*

Sir,

Acinetobacter baumannii is an important Gram-negative organism responsible for various nosocomial infections and is frequently multidrug-resistant. We report a case of carbapenem-resistant *Acinetobacter baumannii* infection with discrepant aminoglycoside susceptibility testing results. Whole genome sequencing identified a 16S methylase gene mediating aminoglycoside resistance and OXA-23 carbapenemase.

A 24-year-old previously well male was transferred to an Australian tertiary hospital from Thailand following a motorbike accident one month prior in Vietnam. He sustained multiple intracranial, maxillofacial and orthopaedic injuries requiring bilateral decompressive craniectomies, tracheostomy placement and prolonged intensive care admission. His orthopaedic injuries included compound left femoral, patella and tibial fractures, requiring open reduction and fixation,

surgical debridement, and vacuum-assisted closure (VAC) dressings. Antimicrobial therapy on arrival included levofloxacin, ceftolozane-tazobactam and tigecycline, for treatment of a line-related *Pseudomonas aeruginosa* bacteraemia, a polymicrobial ventilator-acquired pneumonia, and a carbapenem-resistant *Acinetobacter baumannii* (CRAB) infection of the left knee surgical wound. These antimicrobials were ceased given the bacteraemia and pneumonia had received adequate therapy, and surgical exploration of the left knee was arranged.

Intraoperative appearance revealed grossly unhealthy tissue and bone, consistent with active infection. Microscopy identified Gram-negative bacilli and Gram-positive bacilli on operative tissue specimens; *Acinetobacter baumannii* complex and *Corynebacterium striatum* were cultured, and intravenous amikacin and vancomycin commenced empirically.

Matrix-assisted laser desorption ionisation-time of flight (MALDI-TOF) mass spectrometry (VITEK MS, bioMérieux) identified the first isolate as *Acinetobacter baumannii* with 99.9% certainty. The VITEK2 instrument (bioMérieux) and AST-N246 card reported susceptibility of the *Acinetobacter* to tobramycin and amikacin, but resistance to all other antimicrobials (Table 1). Our patient proceeded to six successive washout-debridements, including removal of patella and extensor apparatus, with plan for eventual arthrodesis. While operative specimens became culture-negative for *Corynebacterium* after 4 days of vancomycin, *Acinetobacter* continued to grow from operative specimens. A confirmatory Etest (bioMérieux) on Mueller Hinton agar (Pathology Queensland) for amikacin MIC was performed to guide therapeutic drug monitoring (TDM). Etest revealed amikacin and tobramycin MICs of >256 mg/L, indicating resistance, with susceptibility only to colistin. An Etest on the initial operative specimens prior to amikacin therapy revealed that the isolate had always been aminoglycoside non-susceptible.

Genomic DNA was extracted using QIAamp DNA mini-kits (Qiagen, Australia) and paired-end DNA libraries prepared using Nextera XT kits (Illumina, Australia). Whole genome sequencing was performed using Illumina NextSeq (150 bp paired-end reads), with sample analysis via the Queensland Genomics customised pipeline (<https://gitlab.beatsonlab.com/binf/SnapperRocks/wikis/home>). Resistance gene detection was performed by screening the draft genome assembly against the NCBI Bacterial Antimicrobial Resistance Reference Gene Database¹ using Abricate 0.9.8 (<https://github.com/tseemann/abicate>). Only genes with a minimum of 90% nucleotide sequence identity and 90% sequence coverage were reported. Whole genome sequencing analysis identified a 16S methylase gene (*armA*) implicated in pan-aminoglycoside resistance, in addition to an OXA-23 carbapenemase. Given sufficient treatment of the *Corynebacterium* and multidrug resistance of the *Acinetobacter* isolate, all antimicrobials were ceased. Fortunately, there was no aminoglycoside-related nephrotoxicity, although ototoxicity assessments were constrained by the patient's dislocated left ear ossicles and ophthalmoplegia. Colistin was not pursued given toxicity concerns with combination therapy and limited evidence for use as monotherapy.

Limb-salvaging therapies were pursued with Prontosan antiseptic washes through the VAC apparatus, and further surgical washouts. Favourable intraoperative appearances with healthy granulation tissue suggested possible

Table 1 Comparison of VITEK2 and Etest susceptibility testing results

Antibiotic	VITEK2		Etest	
	MIC mg/L	Interpretation ^a	MIC mg/L	Interpretation ^a
Ticarcillin-clavulanic acid	≥128	IE		
Piperacillin-tazobactam	≥128	IE		
Cefazolin	≥64	IE		
Ceftazidime	≥64	IE		
Ceftriaxone	≥64	IE		
Cefepime	≥64	IE		
Ceftazidime-avibactam			64	Not calibrated
Ceftolozane-tazobactam			64	Not calibrated
Meropenem	≥16 (≤4)	R	>32	R
Gentamicin	≥16 (≤4)	R		
Amikacin	4 (≤8)	S	>256	R
Tobramycin	4 (≤4)	S	>256	R
Ciprofloxacin	≥4 (0.001)	R		
Norfloxacin	≥16	IE		
Trimethoprim	≥16	IE		
Co-trimoxazole	≥320 (≤2)	R		
Tigecycline			8	Not calibrated

Note: Colistin MIC 0.25 mg/L by Micronaut method (susceptible).

EUCAST breakpoints listed in parentheses after measured MIC where available.

IE, insufficient evidence for use of listed antimicrobial, including presumed resistance; R, resistance to listed antimicrobial; S, susceptibility to listed antimicrobial.

^a Interpretation based on EUCAST Clinical Breakpoint Tables v.10.0.

Acinetobacter wound colonisation rather than invasive infection. Our patient proceeded to surgical closure with a medial gastrocnemius flap 18 days following antimicrobial cessation. After 2 months in rehabilitation, our patient was evaluated for placement of a stabilising screw to facilitate weight-bearing. Nuclear medicine bone scan and magnetic resonance imaging scan revealed a partially united proximal left tibia fracture and features consistent with osteomyelitis of the anterior left tibial metadiaphysis. After extensive multidisciplinary discussions, our patient proceeded to above-knee amputation seven months after initial injury. He is currently being fitted for a prosthesis and continues to progress with physical and cognitive rehabilitation with no ongoing infective complications.

Acinetobacter baumannii is a non-motile Gram-negative bacillus in its growth phase, and adopts a coccobacillary form in its stationary phase.² It is strictly aerobic, catalase-positive and is differentiated from other *Moraxellaceae* by being oxidase-negative. It grows readily on routine media, and forms colourless, mucoid, non-haemolytic colonies on blood agar.^{2,3} There are at least 26 species of *Acinetobacter*. The grouping *Acinetobacter calcoaceticus*-*Acinetobacter baumannii* complex (ACB) is used when *A. calcoaceticus*, *A. baumannii*, *Acinetobacter pittii* and *Acinetobacter nosocomialis* are not differentiated by the VITEK2 instrument.^{4,5} *Acinetobacter baumannii* utilises a unique combination of pathogenic factors but relies heavily on biofilm formation.⁴ Biofilm-associated protein (BAP) and BAP-like proteins ensure biofilm maintenance and facilitate adherence to host cells, whilst fimbriae aid attachment to host tissues and fomites. Outer-membrane protein A (OmpA) contributes to biofilm integrity and binds Factor H, an alternative

complement pathway inhibitor. Some strains also possess a K1 polysaccharide capsule, which hinders phagocytosis and prevents complement activation. Instead of transferrin or lactoferrin, *A. baumannii* utilises siderophores to acquire iron.²

Acinetobacter species are common environmental organisms, found in water, soil, foodstuffs, animals and healthy human subjects.^{2,3} Given their virulence factors, *Acinetobacter* species can survive on inanimate objects, including medical equipment and in hospital bedspaces. Responsible for various clinical syndromes, *A. baumannii* frequently causes ventilator-acquired pneumonia and hospital-acquired pneumonia in the context of prolonged admissions.^{2,4} Community-acquired pneumonia can occur in tropical regions and is associated with rapid progression and severe illness.^{6,7} Nosocomial bloodstream infections commonly occur secondary to vascular-access catheters or respiratory tract spread, with mortality rates up to 44% in ICU populations.⁴ Important to our case, *A. baumannii* can cause cellulitis, necrotising fasciitis or osteoarticular infections, which may be acquired post-surgically or from traumatic inoculation.^{8,9} Rarer clinical syndromes include urinary tract infections, endocarditis, meningitis and ocular infections.^{4,6}

Despite its relatively low virulence, the propensity of *A. baumannii* to develop antibiotic resistance is profound. Isolates resistant to all known antimicrobials have been identified.^{2,4} Alterations to cell-wall channels, efflux pump overexpression and small size and quantity of porins reduce antimicrobial concentrations and drug-binding.^{9,10} Chromosomally-encoded AmpC β -lactamases exist in all *Acinetobacter* species, although clinical resistance only occurs if higher β -lactamase production is facilitated by insertion of an IS*Aba1* promoter sequence next to the *ampC* gene.^{3,4} Many isolates possess Class D β -lactamases, or oxacillinases (OXAs), which can have carbapenemase activity. *Acinetobacter baumannii* may also acquire metallo- β -lactamases, conferring resistance to carbapenems and other β -lactams drugs. Carbapenem-resistant isolates pose an increasing threat worldwide, with some series reporting alarming resistance rates, including Singapore (50%), Pakistan (62%), Chile (70%), India (85%) and Korea (92%).^{2,9} Fluoroquinolone resistance has been reported, with high-level resistance typically associated with mutations to quinolone resistance-determining regions, including DNA gyrase A subunit (*gyrA*) and topoisomerase IV subunit (*parC*). Most relevant to this case, *A. baumannii* can develop resistance to aminoglycosides. Various aminoglycoside modifying enzymes (AMEs) have been identified across multiple genes and alterations to RNA binding sites may also confer aminoglycoside resistance.^{2,10}

In our patient's case, routine susceptibility testing with the VITEK2 instrument reported the *A. baumannii* isolate as tobramycin and amikacin susceptible. Given ongoing culture of *A. baumannii* from operative tissue specimens, treatment with amikacin was pursued. An Etest was performed to determine the amikacin MIC of the isolate and guide TDM. The most significant finding of this case is the conflicting amikacin susceptibility on VITEK2, and amikacin resistance on Etest. This likely signifies a very major error (VME) by the VITEK2 instrument, falsely identifying a resistant isolate as susceptible to a given antimicrobial. This is not a new phenomenon. A 2010 series identified VITEK2 misreporting of susceptibility to aminoglycosides in *Acinetobacter*

baumannii-calcoaceticus.¹¹ This paper reviewed 107 isolates between 2006 and 2008 using broth microdilution (BMD) as the gold standard for MIC testing. Globally, the Etest performed well, with false susceptibility reporting for an isolate resistant to gentamicin, tobramycin, and amikacin in only 0%, 2.8% and 0.9% of cases, respectively. VITEK2 falsely reported susceptibility to gentamicin, tobramycin, and amikacin in 0%, 13.1% and 36.4% of cases, respectively. This high VME rate was most pronounced for the VITEK2 instrument specifically for amikacin MIC testing and did not correspond to any specific isolate genotype or AME. The same VITEK2 instrument was used at our facility, although a new VITEK2 card has been implemented since 2010. It is important to address the possibility of sampling from different *Acinetobacter* subpopulations, which in our case could account for our VITEK2 result if a more susceptible colony had been sampled. Nevertheless, the potential for this susceptibility reporting error remains today with the current VITEK2 card and may pose a clinical risk to patients.

Whole genome sequencing conducted in this case revealed a 16S methylase gene (*armA*), a genotype known to mediate pan-aminoglycoside resistance by rRNA target site alteration, and interestingly typically coexisting with OXA-type carbapenemases.¹⁰ We note that the updated European Committee on Antimicrobial Susceptibility Testing (EUCAST) guidelines currently do not recommend aminoglycoside monotherapy.¹² Therefore, we suggest that for all *Acinetobacter* infections requiring aminoglycoside therapy, MICs be routinely determined at treatment outset, either by BMD, Etest or other method. This will identify resistant isolates early and expedite alternative treatment choices. Furthermore, identification of MICs will encourage TDM and minimise aminoglycoside-related adverse effects when treating sensitive isolates. We would encourage the use of molecular testing to characterise aminoglycoside resistance mechanisms if available.

Acknowledgements: Special thanks to Dr Brian Forde from the School of Chemistry and Molecular Biosciences at the University of Queensland, for his contribution to whole genome sequencing for this case.

Conflicts of interest and sources of funding: This article received no financial support or funding. The authors state that there are no conflicts of interest to disclose.

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DOI: <https://doi.org/10.1016/j.pathol.2020.10.021>

Absence of *mecC* methicillin-resistant *Staphylococcus aureus* in a tertiary hospital in Singapore

Sir,
Methicillin-resistant *Staphylococcus aureus* (MRSA) are important causes of community acquired and hospital acquired infections. Contact precautions are required to prevent spread from colonised patients to non-colonised patients. Screening for and detection of colonisation is key for infection control purposes. The most common genetic basis for methicillin resistance is the acquisition of the *mecA* gene coding an altered penicillin-binding protein (PBP)-2A. Divergent β -lactam resistance conferring *mecA* homologues such as *mecB*, *mecC*, and *mecD* are also known.¹

MRSA screening in our hospital is performed using nasal, axilla, and groin swabs inoculated on MRSA Select II agar (Bio-Rad, USA). Although discrepancies between cefoxitin and oxacillin results are more apparent in MRSA harbouring *mecC* (cefoxitin resistant, oxacillin susceptible), cefoxitin remains highly reliable for phenotypic detection.² To investigate the molecular epidemiology of MRSA in Singapore, polymerase chain reaction (PCR) screening for *mec* genes was performed as previously described for *mecA*,³ *mecC*⁴ and *mecB*⁵ on all included isolates. Between April 2018 and June 2020, a total of 2902 screening isolates from 2482 patients were included. All isolates were confirmed as *S. aureus* with Bruker MALDI Biotyper (Bruker, USA) and were *mecA*-positive (2902/2902; 100%). *mecC* and *mecB* were not detected, confirming *mecA* as the dominant mechanism of methicillin-resistance in MRSA in our hospital. A previous

meta-analysis indicates $\approx 0.004\%$ (95% CI 0.002–0.007) *mecC* prevalence in humans and in animals it is higher at $\approx 0.098\%$ (95% CI 0.033–0.174).⁶ We did not detect any *mecC* in our clinical setting.

mecC-positive isolates have been found to belong to two predominant lineages, CC130 and ST425.⁷ Although *mecC* MRSA is found from humans and animals, both CC130 and ST425 lineages are adapted to animal hosts suggesting a zoonotic link with acquisition of *mecC* MRSA.⁷ In Singapore, for geographical reasons, agriculture and farming are not major economic activities. The absence of *mecC* in our population reflects the limited direct contact with livestock and ruminants. Reports of *mecC* in Asia have so far been restricted to single case reports from animals.^{8,9} One isolate from a domestic cat has also been described in Australia.¹⁰

In summary, *mecC* remains undetected in Singapore, with scant reports in neighbouring countries. PCR-based commercial assays such as Xpert MRSA NxG (Cepheid, USA), BD MAX StaphSR Assay Kit (Becton Dickinson, USA) and artus MRSA/SA QS-RGQ Kit (Qiagen, Germany) have incorporated *mecC* detection but this is unlikely to improve the yield of detection in our local population.

Conflicts of interest and sources of funding: The authors state that there are no conflicts of interest to disclose.

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DOI: <https://doi.org/10.1016/j.pathol.2020.11.006>