


CASE REPORT

Open Access



# *Aerococcus urinae*, a rare cause of aortic root abscess: a case report

Chong Wei Tiong<sup>1\*</sup> , Caroline Bartolo<sup>2</sup>, Aaron Walton<sup>1,2</sup> and Eugene Athan<sup>2,3</sup>

## Abstract

**Background:** *Aerococcus urinae* is a bacterium of emerging clinical interest that most commonly causes urinary tract infections (UTI) but can also result in invasive infections. It is a catalase-negative, alpha-haemolytic gram-positive coccus that grows in clusters or tetrads and usually causes urinary tract infections. While rare, infective endocarditis must be considered when *A. urinae* is isolated in blood culture. The mortality rate of *A. urinae* infective endocarditis is similar to overall endocarditis mortality. We report a rare case of aortic root abscess caused by *A. urinae*.

**Case presentation:** An 82-year-old Caucasian man presented to hospital with behavioural change and severe malnutrition and was managed for psychotic depression. On day 34 of his inpatient stay, a febrile episode prompted blood cultures, which grew *Aerococcus urinae*. Investigations revealed a bicuspid aortic valve, aortic valve endocarditis and aortic root abscess. He also had prostatomegaly. He underwent aortic valve replacement, received 6 weeks of intravenous ceftriaxone and recovered.

**Conclusion:** Infective endocarditis should be considered in patients with persistent *Aerococcus urinae* bacteraemia. Accurate identification with mass spectrometry is recommended to avoid misidentification as staphylococcus, streptococcus or enterococcus, which is a possibility with conventional laboratory methods.

**Keywords:** Endocarditis, *Aerococcus*, Bacteraemia, Case report

## Background

*Aerococcus urinae* is a bacterium of emerging clinical interest that most commonly causes urinary tract infections (UTI) but can rarely result in invasive infections. It is a catalase-negative, alpha-haemolytic gram-positive coccus, which grows in clusters or tetrads [1, 2]. It accounts for an estimated 0.15–0.8% of all UTI cases [3, 4]. However, this is likely an underestimate because *A. urinae* has often been misclassified as *Staphylococcus*, *Streptococcus* or *Enterococcus*, due to similarities on microscopy and colony reaction to hemolysis [1, 5]. While rare, infective endocarditis must be considered when *A. urinae* is isolated in blood culture [1]. To the

best of our knowledge, this is the third reported case in the English literature and the first reported case in Australia of aortic root abscess caused by *Aerococcus urinae*.

## Case report

An 82-year-old Caucasian man was admitted to a regional tertiary hospital for low mood and behavioral change for 2 weeks, as well as reduced appetite, malnutrition and weight loss. His past medical history was unremarkable other than chronic lower urinary tract symptoms, and he was not taking any regular medications. He is a retired engineer who is independent with his activities of daily living. He is also the main caregiver for his daughter who has been diagnosed with brain cancer. His wife passed away a year ago. He does not have significant alcohol use and does not use recreational drugs. He has a distant smoking history, last smoked 50 years ago. On admission his blood pressure was 108/70 mmHg. His heart rate was

\*Correspondence: CHONG.TIONG@barwonhealth.org.au

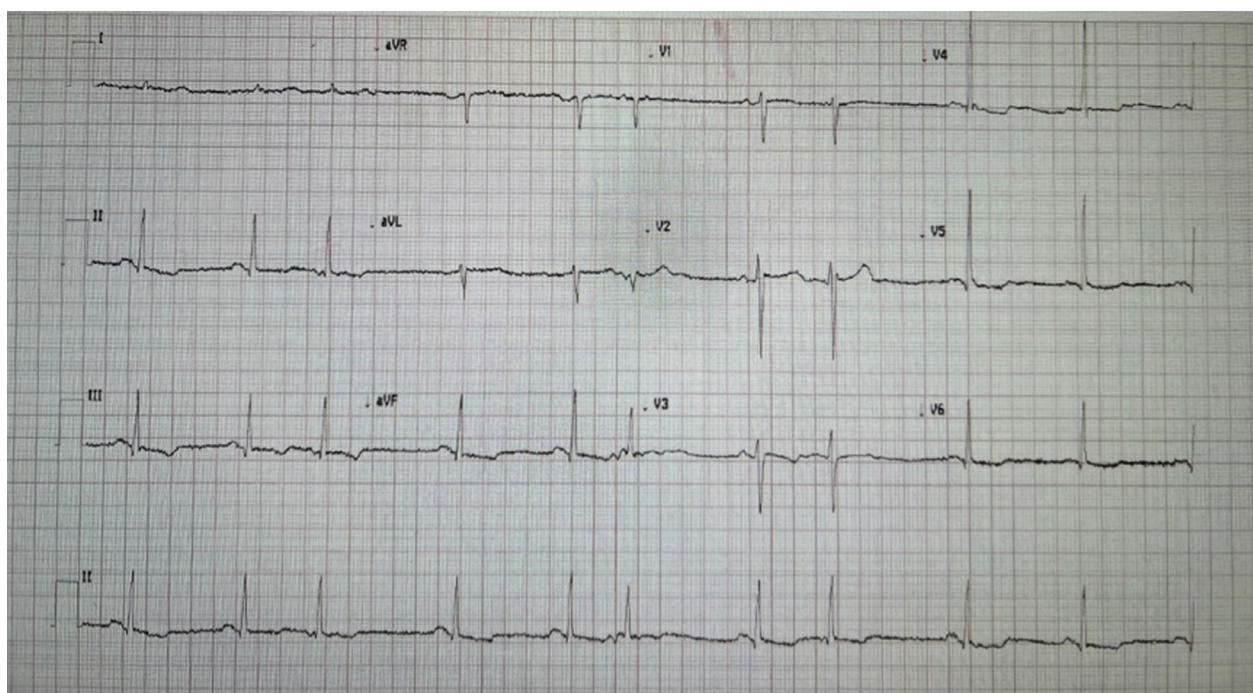
<sup>1</sup> Department of General Medicine, Barwon Health, Geelong, VIC, Australia  
Full list of author information is available at the end of the article



© The Author(s) 2022. **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>. The Creative Commons Public Domain Dedication waiver (<http://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article, unless otherwise stated in a credit line to the data.

70 beats per minute. His oxygen saturation was 98% on room air. His respiratory rate was 19 breaths per minute. He was afebrile at 36.3 °C. He was oriented to time and place, and performed serial sevens calculations without difficulty. Speech was slow and affect was flat. Cardiovascular examination was normal with dual heart sounds, no murmur and no clinical evidence of heart failure. Respiratory and abdominal examination was normal. Cranial nerve examination was unremarkable. Neurological examination of upper limbs and lower limbs was unremarkable, with normal tone, power, coordination, sensation and reflexes. A dental examination was not performed. Blood tests showed a normal white cell count (WCC)  $6.2 \times 10^9/\text{L}$  (reference range:  $4\text{--}11 \times 10^9/\text{L}$ ) and C-reactive protein (CRP)  $< 2.9 \text{ mg/L}$  ( $< 2.9 \text{ mg/L}$ ). Figure 1 shows his electrocardiogram at admission. Importantly, there was no atrioventricular (AV) block. Magnetic resonance imaging of his brain showed small old lacunar infarct in left lentiform nucleus, which would not explain his presentation. He was reviewed by the psychiatry team, who diagnosed severe psychotic depression and commenced the patient on citalopram and risperidone. On day 9 of his hospital stay, he developed acute urinary retention, requiring indwelling urinary catheter placement. A renal tract ultrasound revealed moderate prostatomegaly. He remained in hospital for management of his severe malnutrition and monitoring for re-feeding syndrome. He had two episodes of presumed

catheter associated urinary tract infection with growth of *Enterococcus faecalis* and *Escherichia coli*, and received courses of oral amoxicillin 500 mg TDS and oral trimethoprim 300 mg daily respectively. On day 34, a febrile episode prompted blood cultures. He had no localizing symptoms. On examination there were no heart murmurs, peripheral stigmata of infective endocarditis, or signs of heart failure. After 25 hours, both aerobic and anaerobic blood culture bottles flagged with gram-positive cocci in clusters resembling Staphylococci, which were eventually identified by mass spectrometry as *Aerococcus urinae*. Susceptibility testing was performed using Etest with breakpoints as described by Clinical and Laboratory Standards Institute (CLSI) criteria. The penicillin and ceftriaxone MICs were 0.064  $\mu\text{g/mL}$  and 0.50  $\mu\text{g/mL}$  respectively (both susceptible). Urine culture was sterile. A repeat WCC was  $5.5 \times 10^9/\text{L}$  ( $4\text{--}11 \times 10^9/\text{L}$ ) while CRP increased to 73  $\text{mg/L}$  ( $< 2.9 \text{ mg/L}$ ). See Table 1. Hepatitis B surface antigen, hepatitis C antibody, and Human Immunodeficiency Virus (HIV) antigen/antibody was not detected. *Treponema pallidum* Immunoglobulin G (Ig) was non-reactive. The patient was commenced on intravenous ceftriaxone 1 g twice a day. A summary of the antibiotics administered is shown in Fig. 2. He remained bacteraemic after 6 days so a transthoracic echocardiogram was performed which showed a bicuspid aortic valve and mobile densities on both mitral and aortic valves as well as aorto-mitral curtain thickening.



**Fig. 1** Electrocardiogram at admission

**Table 1:** Investigation results

	At admission	Day 34 of hospital admission	Normal range
Haemoglobin (g/L)	157	128	130–180
White cell count ( $\times 10^9/L$ )	6.2	6.9	4–11
Neutrophils ( $\times 10^9/L$ )	4.2	5.2	2–8
Lymphocytes ( $\times 10^9/L$ )	1.4	1.1	14
Monocytes ( $\times 10^9/L$ )	0.5	0.4	< 1.1
Eosinophils ( $\times 10^9/L$ )	< 0.1	< 0.1	< 0.6
Basophils ( $\times 10^9/L$ )	< 0.1	< 0.1	< 0.3
Platelets ( $\times 10^9/L$ )	227	257	150–450
Sodium (mmol/L)	139	132	135–145
Potassium (mmol/L)	4.2	4.5	3.5–5.2
Chloride (mmol/L)	103	94	95–110
Bicarbonate (mmol/L)	29	29	22–32
Anion gap (mmol/L)	11	14	9–19
Urea (mmol/L)	7.7	6.8	3.5–11
Creatinine ( $\mu M/L$ )	95	80	60–110
eGFR (mL/min)	64	79	> 59
Adjusted calcium (mmol/L)	2.16	2.12	2.15–2.55
Magnesium (mmol/L)	0.86	0.76	0.7–1.1
Phosphate (mmol/L)	1.14	0.81	0.75–1.5
Total protein (g/L)	65	51	60–80
Albumin (g/L)	38	23	35–50
Globulin (g/L)	27	28	23–39
ALP (U/L)	42	60	40–140
Bilirubin (micromole/L)	17	6	< 25
GGT (U/L)	18	49	< 51
AST (U/L)	19	24	< 41
ALT (U/L)	21	35	< 51
CRP (mg/L)	< 2.9	73.4	< 2.9
TSH (mIU/L)	1.98		0.50–5.00

*Hb* Haemoglobin, *WCC* White cell count, *eGFR* estimated glomerular filtration rate, *ALP* alkaline phosphatase, *GGT* gamma-glutamyl transferase, *AST* aspartate aminotransferase, *ALT* alanine transaminase, *CRP* C-Reactive protein, *TSH* Thyroid-stimulating hormone, *mmol/L* millimole per litre, *micromol/L* micromole per litre, *g/L* gram per litre, *L* Litre, *mg/L* milligram per litre, *U/L* Units per Litre, *ml/min* millilitre per minute, *mIU/L* milliunits per litre

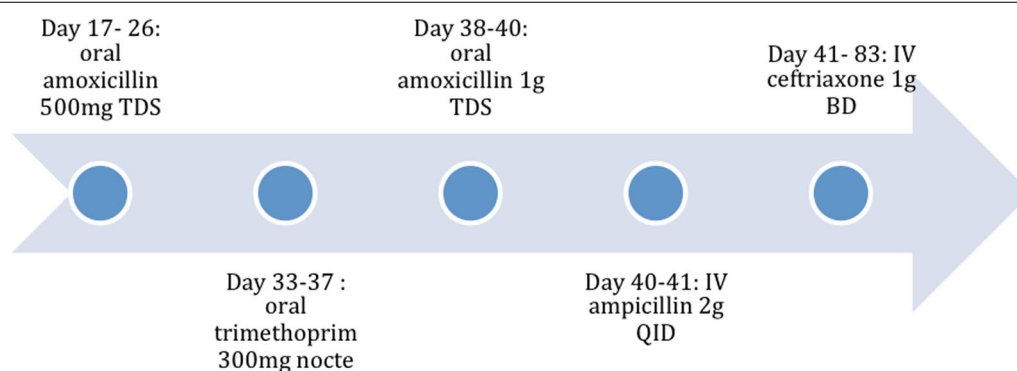
A transesophageal echocardiogram (TOE) subsequently showed a cavity (1.6 cm  $\times$  0.9 cm) at the aorto-mitral curtain, consistent with an abscess (Fig. 3). The patient proceeded to have an aortic valve replacement and patch repair of the left coronary sinus, which had been destroyed by the abscess. Operative specimens were sterile however the operation had been delayed due to the patient's initial reluctance to proceed with surgery and he had received antibiotics for over 2 weeks by the time surgery was performed. He received a total of 6 weeks of ceftriaxone, and was transferred to a subacute rehabilitation centre for re-conditioning.

Two weeks after completion of antibiotic course, he had persistently fluctuating cognitive state, hence had further blood cultures which had no growth as well as a lumbar puncture, which was unremarkable. Cerebrospinal fluid (CSF) white cell count was  $2 \times 10^6/L$ , erythrocyte count was  $380 \times 10^6/L$ . No organisms were seen and there was no growth on CSF culture. CSF protein was 0.26 g/L (0.15–0.45 g/L), while CSF glucose was 3.4 mmol/L (2.8–4.2 mmol/L). A repeat transthoracic echocardiogram 6 weeks after the surgery showed no evidence of ongoing endocarditis. On review 3 months later, repeat WCC and CRP were  $5.1 \times 10^9/L$  and < 2.9 mg/L respectively. He reported feeling well at review 7 months later, without any dyspnea, dizziness or palpitations. He was advised to have annual echocardiogram for surveillance of valve function.

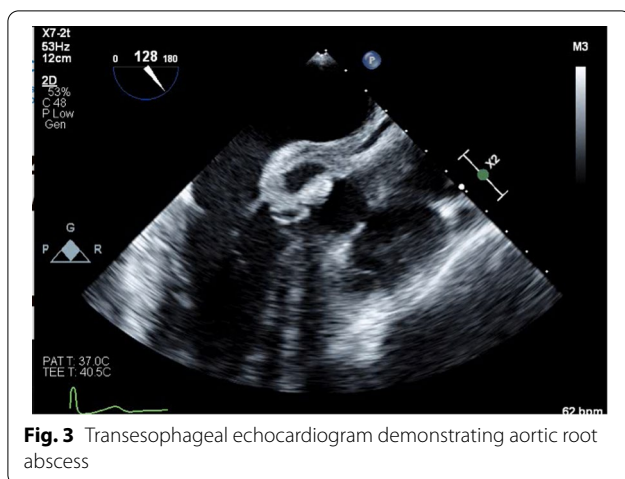
## Discussion

To the best of our knowledge, this is the third reported case in the English literature and the first reported case in Australia of aortic root abscess caused by *Aerococcus urinae*.

Although infective endocarditis is a rare manifestation of *Aerococcus urinae*, a high index of suspicion of deep-seated infection is required if a patient is bacteraemic, even in the absence of clinical findings such as a new heart murmur, clinical signs of heart failure, or

**Fig. 2** Summary of antibiotics given during this hospital admission





**Fig. 3** Transesophageal echocardiogram demonstrating aortic root abscess

other peripheral stigmata of infective endocarditis, as described in this case. Due to the high clinical index of suspicion given the prolonged bacteraemia, with appropriate investigations, the diagnosis was established leading to appropriate therapy.

*Aerococcus urinae* belongs to the group of organisms called *Aerococcus-like organisms* (ALO). Along with *Aerococcus viridans* and *Aerococcus sanguinicola*, *Aerococcus urinae* is increasingly being recognized as a causative pathogen in many human infections [5]. *A. urinae* most commonly causes urinary tract infections, however invasive infections such as endocarditis can occur. There are also occasional reports of spondylodiscitis and spontaneous bacterial peritonitis caused by *A. urinae* [6, 7]. Risk factors for *A. urinae* infection are male gender, age greater than 65 years, and underlying urinary tract abnormalities [8]. The virulence mechanisms displayed by *A. urinae* are the formation of biofilm on plastic surfaces and the aggregation of human platelets, which can result in the formation of heart valve vegetations [1].

Early identification of *A. urinae* in cultures can be challenging due to the following reasons. Firstly, *A. urinae* is alpha-haemolytic, producing semi-transparent colonies, hence can be misidentified as *Streptococcus* on culture. Additionally, on Gram stain, *Aerococcus* and *Staphylococcus* share similar appearance, as Gram-positive cocci in clusters. However, unlike staphylococci, *Aerococcus* is catalase negative. Due to these challenges, there are often in delays in accurate diagnosis. However, due to improved diagnostic techniques identification of *A. urinae* has improved [5, 9]. *A. urinae* can reliably be identified with 16s rRNA-gene sequence (the gold standard) and mass spectrometry, but less so with VITEK 2 [9]. In addition, routine urine cultures may not isolate *A. urinae*, because *A. urinae* optimally requires prolonged incubation in 5% carbon dioxide in

anaerobic conditions to be isolated in urine culture [6]. In a review by Senneby *et al.* [1], out of 42 patients who had *A. urinae* bacteraemia, only 6 patients had urine culture that were positive for *A. urinae*. 15 others had sterile urine, while the rest had urine culture that grew other organisms. In a large number of patients, *A. urinae* was not isolated from the urine samples despite having obvious UTI symptoms [1].

*Aerococcus urinae* displays in-vitro susceptibilities to penicillin, amoxicillin, cefotaxime, ceftriaxone, doxycycline, linezolid, and vancomycin. There is variable resistance to clindamycin, fluoroquinolones, and erythromycin. *A. urinae* is resistant to sulphamethoxazole, while sensitivities to trimethoprim and trimethoprim-sulphamethoxazole are poorly defined and variable depending on methodology [1, 10, 11]. Hence, clinicians need to be aware that the commonly used antibiotics for empirical treatment of urinary tract infections when isolate on culture is pending may be ineffective for treatment of *Aerococcus urinae* infections. In a study of 30 *A. urinae* isolates, 97% of isolates had a ceftriaxone MIC  $\leq 0.5$   $\mu\text{g/mL}$ . 100% had penicillin MIC  $\leq 0.06$   $\mu\text{g/mL}$  and vancomycin MIC  $\leq 1$   $\mu\text{g/mL}$ . High erythromycin and levofloxacin MICs were identified in 17% and 33% of the *A. urinae* isolates respectively [12]. Two older studies have showed a synergistic killing effect when there is combination therapy of penicillin and an aminoglycoside. A recent larger study identified this synergy in only 7 of 15 isolates [13]. Both the Clinical and Laboratory Standards Institute (CLSI) and the European Committee on Antimicrobial Susceptibility Testing (EUCAST) have published recommended clinical breakpoint MICs for *A. urinae* [14, 15].

The incidence of infective endocarditis among patients with *A. urinae* bacteraemia is unclear. A population-based study reports that of 16 patients with *A. urinae* bacteraemia, 3 patients had infective endocarditis [1]. In a review of 46 cases of *A. urinae* infective endocarditis, mortality was reported as 28.2%, similar to overall endocarditis mortality reported. Of patients who underwent surgery, 80% survived. On review of the patients who had a good outcome, 80% of them received antibiotic therapy for 4–6 weeks. While a majority of individuals with *A. urinae* infective endocarditis had urologic abnormalities, underlying valvular conditions were less frequent [8].

Duration of antibiotics for *A. urinae* infective endocarditis is currently guided by expert opinion. However, the outcomes of previous cases in the literature would suggest the need for further research regarding extended intravenous antibiotic therapy for uncomplicated bacteraemia. Two case reports described patients who failed therapy after a short course ( $\leq 10$  days) of intravenous antibiotics [2, 16].

## Conclusion

There are a few learning points from this case. Firstly, it is important to consider infective endocarditis in patients with *A. urinae* bacteraemia especially when there is persistent bacteraemia. Second, laboratory diagnosis may initially misidentify *Aerococcus* spp. as *Staphylococcus*, *Streptococcus* or *Enterococcus* from microscopy and culture therefore use of diagnostic modalities such as mass spectrometry for confirmation of identification is recommended as it will allow for more targeted antimicrobial therapy until susceptibility results are available.

## Acknowledgements

We would like to thank Dr. Michael Lim for his help in obtaining the echocardiogram image.

## Author contributions

CT, CB—writing and editing of manuscript. AW—Treating physician. CB, AW, EA—Supervision. All authors read and approved the final manuscript.

## Funding

None to declare.

## Availability of data and materials

All data generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

## Declarations

## Ethics approval and consent to participate

Not applicable.

## Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

## Competing interests

The authors declare that they have no conflicts of interest.

## Author details

<sup>1</sup>Department of General Medicine, Barwon Health, Geelong, VIC, Australia.

<sup>2</sup>Department of Infectious Diseases, Barwon Health, Geelong, VIC, Australia.

<sup>3</sup>Deakin University, Melbourne, VIC, Australia.

Received: 24 January 2022 Accepted: 10 August 2022

Published online: 17 November 2022

## References

1. Senneby E, Petersson A, Rasmussen M. Clinical and microbiological features of bacteraemia with *Aerococcus urinae*. *Clin Microbiol Infect*. 2012;18(6):546–50.
2. De Jong M, Soetekouw R, Kate R, Veenendaal D. *Aerococcus urinae*: severe and fatal bloodstream infections and endocarditis. *J Clin Microbiol*. 2010;48:3445–7.
3. Sierra-Hoffman M, Watkins K, Jinadatha C, Fader R, Carpenter JL. Clinical significance of *Aerococcus urinae*: a retrospective review. *Diagn Microbiol Infect Dis*. 2005;53:289–92.
4. Schuur PM, Kasteren ME, Sabbe L, Vos MC, Janssens MM, Buiting AG. Urinary tract infections with *Aerococcus urinae* in the south of The Netherlands. *Eur J Clin Microbiol Infect Dis*. 1997;16:871–5.
5. Sahu K, Lal A, Mishra A, Abraham G. *Aerococcus*-related infections and their significance: a 9-year retrospective study. *J Microsc Ultrastruct*. 2021;9(1):18–25.
6. Colakoglu S, Turunc T, Taskoparan M, Aliskan H, Kizilkilic E, Demiroglu YZ, et al. Three cases of serious infection caused by *Aerococcus urinae*: a patient with spontaneous bacterial peritonitis and two patients with bacteremia. *Infection*. 2008;36(3):288–90.
7. Astudillo L, Sailer L, Porte L, Lefevre JC, Massip P, Arlet-Suau E. Spondylodiscitis due to *Aerococcus urinae*: a first report. *Scand J Infect Dis*. 2003;35:890–1.
8. Rodriguez F, Lasarcina A, Grijalva F. Mitral valve endocarditis with perforation from a urinary source: an unusual case and literature review. *Case Rep Cardiol*. 2019. <https://doi.org/10.1155/2019/5496851>.
9. Rasmussen M. *Aerococci* and *aerococcal* infections. *J Infect*. 2013;66(6):467–74.
10. Humphries RM, Hindler JA. In vitro antimicrobial susceptibility of *Aerococcus urinae*. *J Clin Microbiol*. 2014;52(6):2177–80.
11. Rasmussen M. *Aerococcus*: an increasingly acknowledged human pathogen. *Clin Microbiol Infect*. 2016;22:22–7.
12. Shelton-Dodge K, Vetter EA, Kohner PC, Nyre LM, Patel R. Clinical significance and antimicrobial susceptibilities of *Aerococcus sanguinicola* and *Aerococcus urinae*. *Diagn Microbiol Infect Dis*. 2011;70(4):448–51.
13. Carkaci D, Nielsen XC, Fuursted K, Skov R, Skovgaard O, Trallero EP, et al. *Aerococcus urinae* and *Aerococcus sanguinicola*: susceptibility testing of 120 isolates to six antimicrobial agents using disk diffusion (EUCAST), Etest and broth microdilution techniques. *Open Microbiol J*. 2017;11:160–6.
14. Clinical and Laboratory Standards Institute. Methods for antimicrobial dilution and disk susceptibility testing of infrequently isolated or fastidious bacteria. 3rd ed. Wayne: Clinical and Laboratory Standards Institute; 2016. p. M45-Ed3.
15. The European Committee on Antimicrobial Susceptibility Testing. Breakpoint tables for interpretation of MICs and zone diameters 2020.10.0 [www.eucast.org](http://www.eucast.org).
16. Samuelsson S, Kennergren C, Rasmussen M. Case report: a case of pacemaker endocarditis caused by *Aerococcus urinae*. *Case Rep Infect Dis*. 2018. <https://doi.org/10.1155/2018/9128560>.

## Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Ready to submit your research? Choose BMC and benefit from:

- fast, convenient online submission
- thorough peer review by experienced researchers in your field
- rapid publication on acceptance
- support for research data, including large and complex data types
- gold Open Access which fosters wider collaboration and increased citations
- maximum visibility for your research: over 100M website views per year

At BMC, research is always in progress.

Learn more [biomedcentral.com/submissions](https://biomedcentral.com/submissions)

