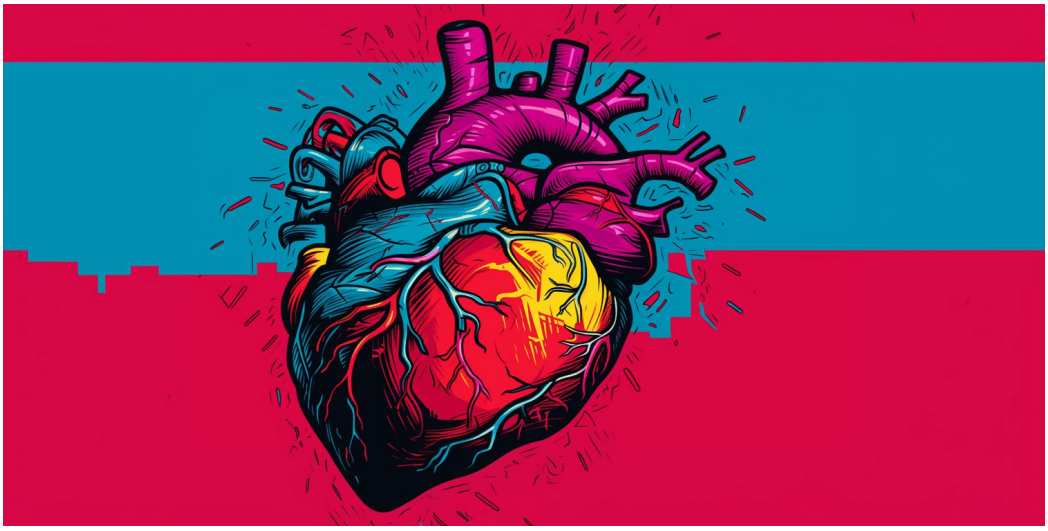

Aerococcus urinae Endocarditis: An Emerging Infectious Disease

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Abstract

Aerococcus urinae is an alpha-hemolytic, catalase-negative, Gram-positive coccus most frequently seen as a cause of urinary tract infections. It can, however, cause more severe diseases such as bacteremia, spondylodiscitis, peritonitis, and endocarditis. The first case of endocarditis was not reported until 1991. Since that time, cases of endocarditis have been reported increasingly in the literature. We report a 59-year-old man with *A. urinae* endocarditis of the aortic valve and review the literature since 1 Jan 2020. *A. urinae* is being reported more frequently and appears to be an emerging infectious disease problem. In our review of the 29 cases since 1 Jan 2020, we found that the aortic and mitral valves are most frequently involved, there is a 24% mortality rate, and those undergoing cardiac surgery have a significantly greater likelihood of survival than those who do not. Physicians should be aware of the potential of *A. urinae* isolated from blood cultures to cause life-threatening endocarditis.

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Introduction

The Genus *Aerococcus* was initially described in 1953[1] by Williams et al., and *Aerococcus urinae* (AU) was designated as a new species in 1992.[2] AU, previously *Aerococcus*-like organism, was first described as a cause of endocarditis in 1991 by Christensen and colleagues[3] and then again in 1995 by Christensen,[4] Kristensen and Nielsen,[5] and Skov and colleagues.[6] Its virulence in this clinical setting is enhanced by its potent biofilm-producing capacity.[7] Since 1995, there have been intermittent case reports and a few published series of AU endocarditis. Approximately 50 cases were published between 1991 and 2019.[8][9] Since 2020, there have been 28 published cases of AU endocarditis, and we herein report the 29th case.[7][10][11][12][13][14][15][16][17][18][19][20][21][22][23][24] With 28 cases having been published since January 1, 2020, we estimate that more than 80 cases will be published by December 31, 2029. Given this substantial increase in case reports of AU endocarditis since January 1, 2020, we would classify AU endocarditis as an emerging infectious disease.[25] We report a 59-year-old man with AU endocarditis, discuss the clinical implications, and review the literature since January 1, 2020.

Methods

We performed Google Scholar® and PubMed® searches using all combinations of the following terms: *Aerococcus urinae*, infective endocarditis, endocarditis, bacteremia. We then assembled the papers reporting patients with *Aerococcus urinae* endocarditis and searched the references of these articles for any further cases. Cases reported since 1 Jan 2020 are summarized in **Table 1.** Case distribution of AU endocarditis before 1 Jan 2020 was separated into 5-year periods (quinquennials), in which the number of cases per quinquennial is seen in **Figure 1.** The last column on the right of **Figure 1** reports only 4 years of accumulated data from the literature.

Table 1. Historical Risk Factors for *Aerococcus urinae* Endocarditis

Risk Factor*	Number Patients	Percentage
Urinary Tract	13	45.0%
• **Urinary Tract Infection**	(7)	(24.0%)
• **Urinary Retention**	(2)	(7.0%)
• **Urinary Stricture**	(1)	(3.5%)
• **Bladder Stones**	(1)	(3.5%)
• **Hydroureter**	(1)	(3.5%)
• **Recent Robotic Prostatectomy**	(1)	(3.5%)
Underlying Cardiac Abnormalities	6	20.7%
• **Cardiac Pacemaker**	(2)	(6.9%)
• **Prior Endocarditis**	(2)	(6.9%)
• **Aortic Stenosis**	(1)	(3.45%)
• **Bioprosthetic Mitral Valve**	(1)	(3.45%)
Injection Drug Use	2	6.9%
End-Stage Renal Disease on Hemodialysis	1	3.45%
Immunosuppressive Therapy	1	3.45%
Septic Arthritis, Knee	1	3.45%
None	2	6.9%
No Data Available	8	27.6%

Some patients had more than one risk factor

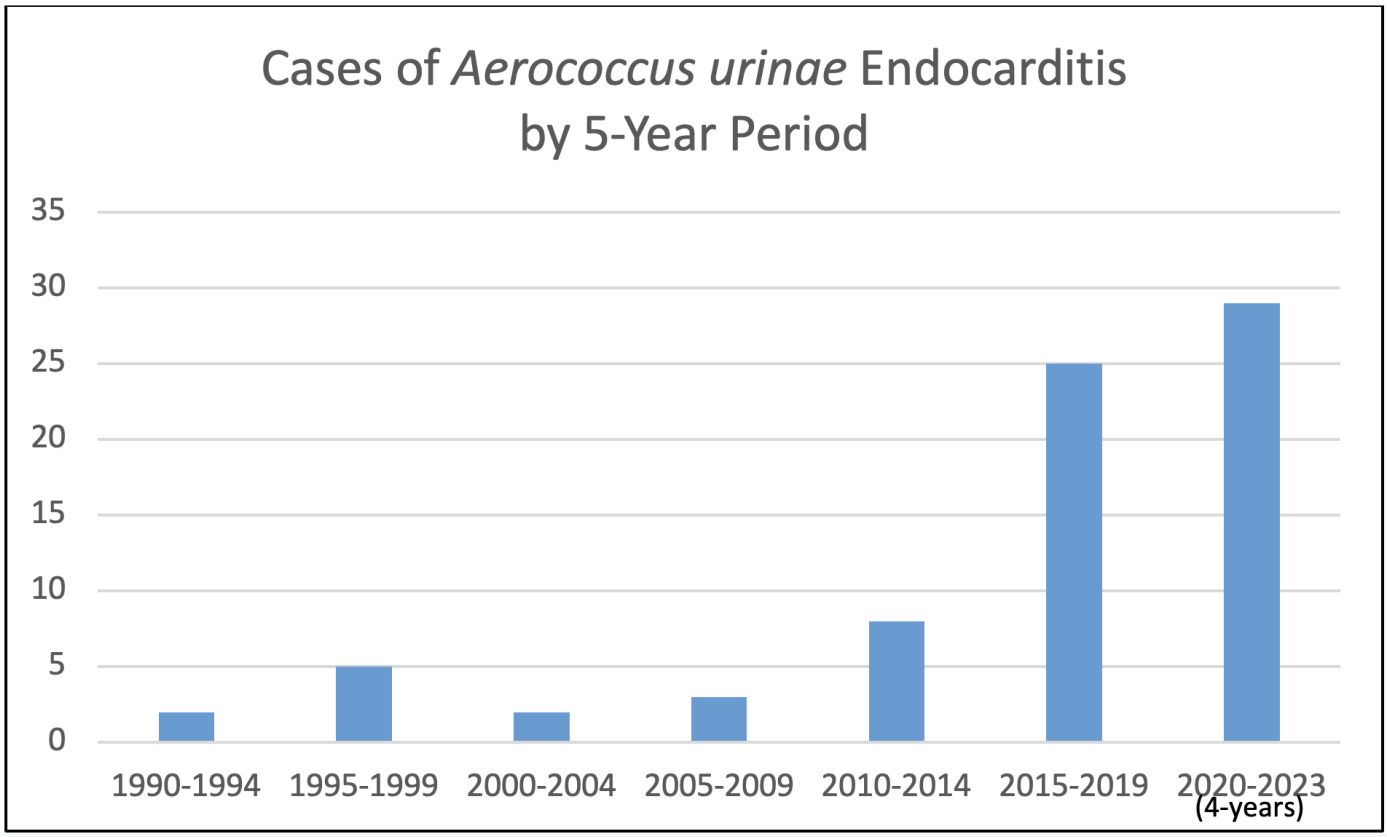

Figure 1. Reported Cases of *Aerococcus urinae* Endocarditis By 5-Year Periods (Except 2020-2023)

Case Report

A 59-year-old man with no significant past medical history presented to the emergency department of another hospital complaining of fever, chills, generalized weakness, diaphoresis, and vague chest pain. Physical examination revealed a blood pressure of 126/84 mmHg, a heart rate of 106 beats/minute, a respiratory rate of 20 breaths/minute, and a temperature of 100.3ºF, as well as an SPO2 of 95% on ambient air. The cardiac exam revealed a diastolic murmur along the left parasternal border but no jugular venous distention and no lower extremity edema. The skin exam revealed a splinter hemorrhage on his left third fingernail. The pulmonary exam showed expiratory wheezing bilaterally. The remainder of the exam was normal. The white blood cell count was = 11,100/µL, and the hemoglobin was = 11.8 g/dL. Electrolytes were normal. The INR was = 1.8. A SARS-CoV-2 nasopharyngeal swab was negative. AST = 77 U/L, and ALT = 55 U/L. On the third hospital day, *Aerococcus urinae* was isolated from 3 of 3 blood cultures. The chest x-ray and CTA of the chest were normal. A SPECT cardiac perfusion study showed no scintigraphic evidence of inducible ischemia. The transthoracic echocardiogram (TTE) showed moderate to severe aortic regurgitation with no visible vegetations. The patient was treated with parenteral ceftriaxone for 3 days with rapid improvement of his fever and weakness, and he was therefore transitioned to parenteral vancomycin on day 4 after the identification of the organism. He was not felt to have endocarditis and was changed to oral amoxicillin on hospital day 5 and discharged home with another 9 days of amoxicillin prescribed. Five days after discharge from the other institution, he presented to our emergency department with worsening dyspnea, fatigue, and chest pain. Vital signs showed a normal temperature, a heart rate of 100

beats/minute, a respiratory rate of 18 breaths/minute, and a blood pressure of 118/59 mmHg. He had an oxygen saturation of 95% on room air. The ECG showed only tachycardia, but the chest x-ray and CTA of the chest revealed a pulmonary edema pattern with bilateral pleural effusions. At the time of his initial presentation to our facility, he complained of fever, chills, weakness, diaphoresis, and chest pain. His physical exam showed a blood pressure = 126/84 mmHg, a heart rate = 106/min, a temperature = 100.3ºF, a respiratory rate = = 20/min, and an SPO2 = 95% on ambient air. The complete blood count and basic metabolic panel were normal, troponin was normal (twice), but the patient had an elevated NT-proBNP of 2486 pg/mL. The examination revealed conversational dyspnea, elevated jugular venous pressure, and a diastolic parasternal heart murmur. A diagnosis of acute decompensated heart failure was made. He was started on intravenous furosemide 40 mg and moved to the coronary critical care unit. A TTE showed severe aortic insufficiency with prolapse of the non-coronary cusp, moderate mitral and tricuspid regurgitation, and a normal but hyperdynamic left ventricular ejection fraction (LVEF) with no definitive valvular vegetations. He underwent cardiac catheterization, which showed non-obstructive coronary artery disease and severe aortic insufficiency. Given his severe acute aortic insufficiency and cardiac decompensation, cardiothoracic surgery was consulted, and the patient was determined to require urgent aortic valve replacement. A preoperative transesophageal echocardiogram (TEE) revealed a tri-leaflet aortic valve with a large and highly mobile long vegetation, flow reversal in the aorta consistent with severe 4+ aortic insufficiency. At surgery, his aortic valve showed significant leaflet destruction, with vegetations present and perforations at the base between the right and left coronary cusps. The patient received a 25 mm Carpenter-Edwards Magna Ease valve. Postoperatively, he was transferred back to the coronary care unit and underwent rapid recovery, and he was discharged home after 4 days with a PICC line and plans for 6 weeks of outpatient parenteral antibiotic therapy with ceftriaxone. He completed his course of ceftriaxone and quickly returned to normal pre-hospital functionality.

Discussion

Aerococci are Gram-positive cocci that grow in clusters and have a colony appearance on blood agar similar to that of viridans streptococci.[2] They can be biochemically differentiated from staphylococci by the catalase test. Staphylococci are catalase-positive. Biochemical techniques such as leucine aminopeptidase, which is positive only for AU, can also be used to differentiate it from *Staphylococcus*.[26] The gold standard for identifying this species remains hippurate hydrolysis testing or sequencing of the 16 SrRNA gene.[27] More recently, MALDI-TOF (Matrix-associated laser desorption ionization time-of-flight mass spectrometry) has been used to identify this organism from clinical samples.[28] AU is one of the described species of aerococci and was originally designated as an "*Aerococcus*-like organism."[29] AU is a Gram-positive, microaerophilic, catalase-negative, alpha-hemolytic coccus which grows predominantly in tetrads and clusters. AU has been identified as an uncommon cause of UTI (0.15% to 0.54%)[30] [6] and as an occasional cause of bloodstream infections.[4] AU infective endocarditis is rare but is being reported more frequently.

The Centers for Disease Control and Prevention defines an emerging infection as one whose incidence has increased recently or is threatening to increase in the near future.[31] Aerococcal infections in general, and aerococcal endocarditis in particular, may have been underreported in the past due to lack of confirmatory identification, difficulties in growing the

organism, and misidentification as another organism. More recently, however, MALDI-TOF identification of pathogens has made it easier to identify and less likely to be misidentified, and clinicians are now aware of the importance of AU as an endocardial pathogen.[28] This increased number of reported cases of AU endocarditis may be the result of better technology (organism isolation and identification) and the advent of numerous on-line journals that accept case reports for publication, making it easier to publish such cases.

Aerococcus urinae isolates are usually susceptible to penicillin G and ampicillin as well as ceftriaxone, cefotaxime, meropenem, and vancomycin.[18][32] In vitro synergism of a beta-lactam and an aminoglycoside has been documented[33] suggesting that combination therapy may be successful in severe infections caused by this organism.[34] In our review of the 29 cases reported since 2020 (including this case), 18 patients (62%) were men, 8 patients (28%) were women, and data were not available for 3 patients (10%). Patient ages ranged from 43 years to 92 years with a mean of 69 years and a median of 70 years. Risk factors for *A. urinae* endocarditis are delineated in **Table 2.** 15 patients had aortic valve endocarditis (14 native, 1 prosthetic), 8 had mitral valve endocarditis (7 native, 1 prosthetic), 3 had aortic and mitral valve endocarditis (all native valves), 1 had native pulmonary valve endocarditis, 1 had native tricuspid valve endocarditis, and 1 had pacemaker endocarditis. Antimicrobial therapy consisted of ceftriaxone alone (7 patients) or with another agent (4 patients), ampicillin alone (2 patients) or with gentamicin (2 patients), penicillin G alone (3 patients) or with gentamicin (3 patients); and vancomycin alone (4 patients) or with another agent (2 patients). 16 patients received 6 weeks of antimicrobial therapy; 7 patients received 4 weeks of therapy; 3 received 2 weeks of therapy; and 2 received 1-2 weeks of treatment. No duration of antimicrobial therapy was available for one patient. Cardiac surgical intervention was undertaken in 11 of 29 patients (37.9%). 22 patients (76%) survived initial hospital admission, while 7 patients (24%) died. Replacement surgery was performed in all 11 patients who underwent emergency valvular replacement surgery, and all 11 survived. All 11 patients who underwent emergency valvular replacement surgery survived. Cardiac surgery was statistically significantly associated with improved survival (p = 0.02, Fisher's exact test).

Table 2. *Aerococcus urinae* Infective Endocarditis Since 2020:Review of the Literature for an Emerging Infection

Case No.	Year	1st Author	Ref. No.	Age	Sex	Risk Issue	Valve(s)	Cardiac Surgery	Primary Antiobiotic	Antibiotic Duration	Outcome
1	2020	John		65	M	ND	Pulmonary	Yes	Penicillin G	6 weeks	Lived
1a	2020	Ravji		77	M	Bladder stones; prior endocarditis and bioprosthetic mitral valve	Mitral – Prosthetic	No	Penicilin G + Gentamicin	6 weeks	Lived
2	2020	Varughese		43	M	Prior endocarditis	Aortic – Prosthetic	No	Ceftriaxone + Vancomycin	ND	Lived
3	2020	Sahu		81	ND	UTI + Pacemaker	Pacemaker	No	Vancomycin	10 days	Died
4	2020	Sahu		80	ND	UTI + Injection drug use	Mitral	No	Vancomycin	2 weeks	Lived
5	2020	Sahu		72	ND	None	Aortic	No	Ceftriaxone	6 weeks	Lived
6	2020	Rosoborough		92	M	Pacemaker	Mitral	No	Penicillin G	4 weeks	Lived
7	2020	Ludwahni		55	M	Urinary tract infection	Aortic	No	Vancomycin	6 weeks	Died
		Martin-							Ceftriaxone +	6 weeks +	

8	2020	Guerra		61	M	Urinary retention	Aortic	Yes	Gentamicin	2 weeks	Lived
9	2020	Yaban		67	M	Urinary tract infection	Mitral	Yes	Ampicillin + Gentamicin	6 weeks + 2 weeks	Lived
10	2020	Yaban		86	F	Urinary tract infection	Aortic	Yes	Ampicillin + Gentamicin	6 weeks + 2 weeks	Lived
11	2021	Ahmed		58	M	Urinary tract infection	Aortic	Yes	Vancomycin + Nafcillin	6 weeks + 6 weeks	Lived
11	2021	Feghaly		79	M	ESRD on hemodialysis	Aortic	No	Vancomycin	6 weeks	Died
13	2021	Khan		86	F	Urinary tract infection + known aortic stenosis	Aortic	No	Ceftriaxone + Gentamicin	6 weeks + 2 weeks	Died
14	2021	Sulaman		67	M	Urethral stricture	Aortic	No	Ceftriaxone	4 weeks	Lived
15	2021	Tai		56	F	ND	Aortic + Mitral	Yes	Ceftriaxone + Vancomycin	2 weeks + 4 weeks	Lived
16	2021	Tai		54	M	ND	Mitral	Yes	Ampicillin	6 weeks	Lived
17	2021	Tai		79	F	ND	Mitral	No	Ceftriaxone	4 weeks	Lived
18	2021	Tai		72	F	ND	Aortic	No	Ampicillin	4 weeks	Lived
19	2021	Tai		46	M	ND	Aortic + Mitral	No	Ceftriaxone	2 weeks	Died
20	2021	Tai		80	M	ND	Mitral	Yes	Ceftriaxone + Gentamicin	6 weeks + 2 weeks	Lived
21	2021	Tai		70	M	ND	Mitral	Yes	Penicillin + Gentamicin	4 weeks + 4 weeks	Lived
22	2022	Akinboboye		48	M	Injection drug use	Aortic	No	Vancomycin, Ceftriaxone, Daptomycin	2 weeks	Died
23	2022	Banerjee		61	F	Immunosuppressive therapy	Tricuspid	No	Penicillin G	6 weeks	Lived
24	2022	Tiong		82	M	Urinary retention	Aortic + Mitral	Yes	Ceftriaxone	6 weeks	Lived
25	2022	Al-Asad		75	M	Robotic prostatectomy	Aortic	No	Penicillin G + Gentamicin	1 week + 1 week	Died
26	2023	Yee		80	F	Hydroureter	Aortic	No	Ceftriaxone	4 weeks	Lived
27	2023	Endo		65	F	Septic arthritis of knee	Aortic	No	Ampicillin + Ceftriaxone	6 weeks + 2 weeks	Lived
28	2023	This patient		59	M	None	Aortic	Yes	Ceftriaxone	6 weeks	Lived

M = Male; F = Female; ND = No data available.

Conclusion

Aerococcus urinae endocarditis is an emerging infectious disease.[31] It is often seen as a complication of underlying urinary tract anatomic or functional disorders. Aortic and mitral valves are the most common anatomic sites of infection. All recently reported patients received an appropriate antimicrobial agent intravenously, but a few patients received less than optimal lengths of antimicrobial therapy. The disease has a mortality rate of 24%. Those undergoing cardiac surgical intervention (valve replacement) were significantly more likely to survive compared to those who did not. When *Aerococcus urinae* is isolated from blood cultures, a search for underlying urinary and cardiac anatomic abnormalities

should be initiated, and the diagnosis of infective endocarditis should be considered significant.

Statements and Declarations

Author Contributions to Manuscript

- Japheth Okpebholo, DO: Writing of manuscript
- Joseph P. Myers, MD: Writing of manuscript; Editing of manuscript

Conflict of Interest

The authors hereby declare that they have no conflicts of interest and no sources of funding related to this manuscript.

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