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Case Report

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Aerococcus Urinae: A rare cause for multi-valve endocarditis

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Case Report

A 42-year-old male with a history of chronic nephrolithiasis presented to the hospital after neighbors found him unconscious at home. On initial evaluation, he was febrile (38.9) with blood pressure of 120/104mmHg. He was severely hypoxic with a sinus rate of 90 beats/minute. His examination revealed multiple abrasions, and he was minimally responsive with a holosystolic murmur at the apex. His electrocardiogram showed normal sinus rhythm with no acute ST-T wave changes. Laboratory data revealed a white cell count of 17.4/nl, platelet count of 109/nl, lactate level of 4.8 mmol/l and creatinine of 1.57mg/dl. Computed tomography of the head revealed multiple regions of acute infarction involving the right inferior cerebellum, left occipital lobe with mass effect on the left occipital horn. He underwent a transthoracic echocardiogram that showed vegetations on both the mitral and aortic valve. He underwent a trans-esophageal echocardiogram, which demonstrated multiple vegetations on the mitral (Figure 1), aortic (Figure 2) and pulmonic valves (Figure 3) with severe pulmonic, mitral and aortic insufficiency (Figure 4 & 5). Despite negative urine cultures, serial blood cultures grew Aerococcus urinae and he was given appropriate IV antibiotics. He was evaluated for possible valvular surgery and was deemed to be a poor surgical candidate. He later developed splenic and renal infarcts followed by significant hemodynamic instability requiring vasopressors and ultimately succumbed to his illness after he developed multi organ failure and disseminated intravascular coagulation.

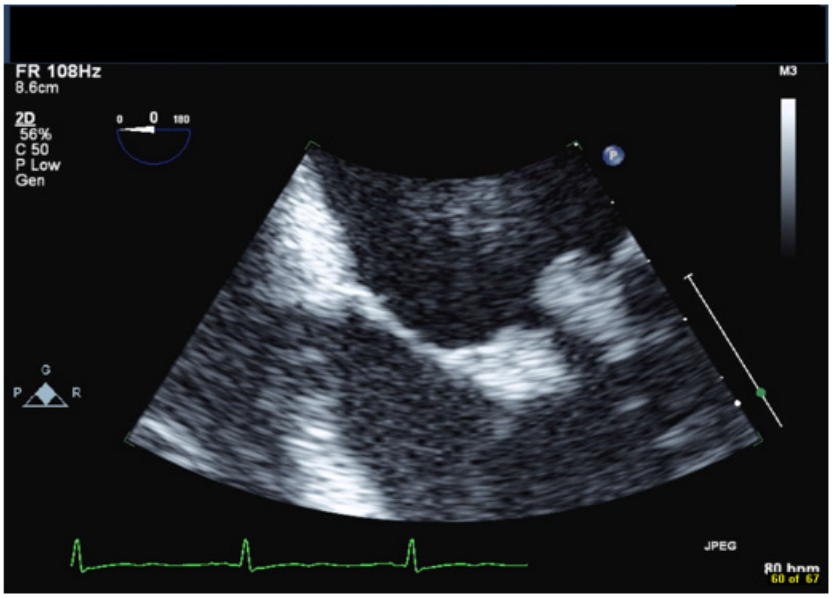

Figure 1: Mitral Veg.

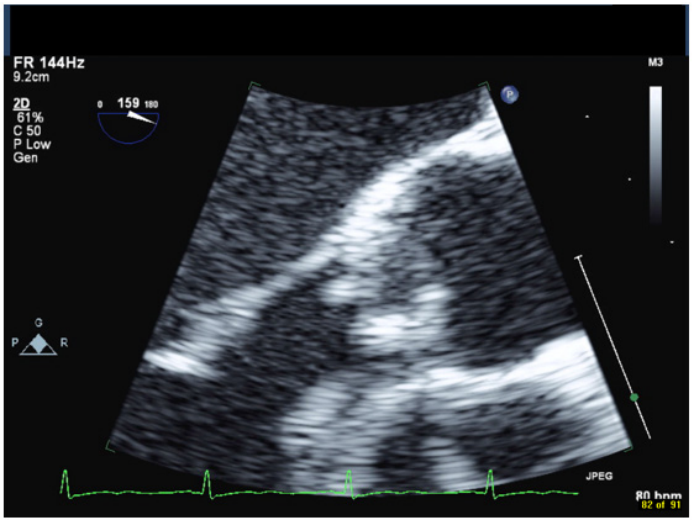

Figure 2: Aortic Veg.

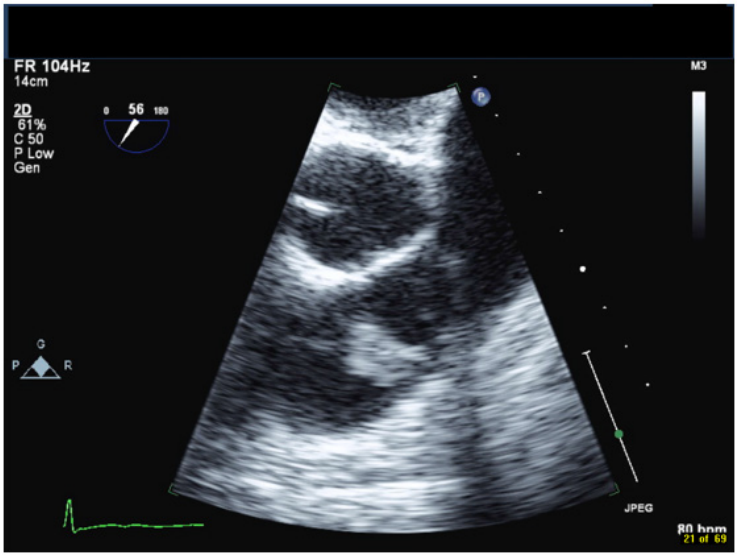

Figure 3: Pulm Veg.

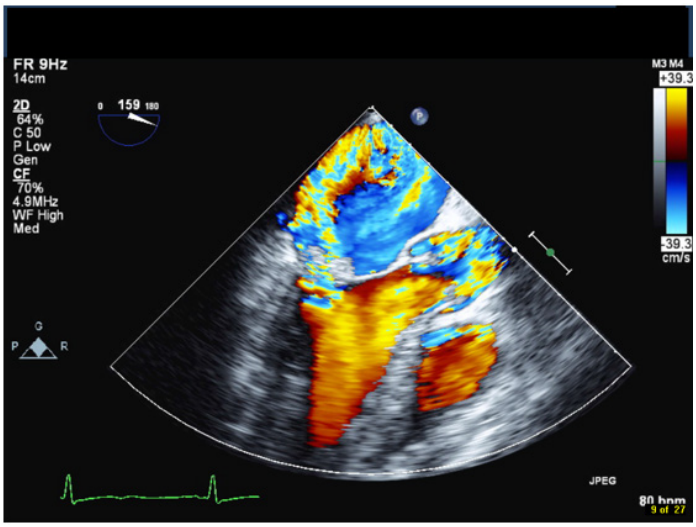

Figure 4: Mitral Reg.

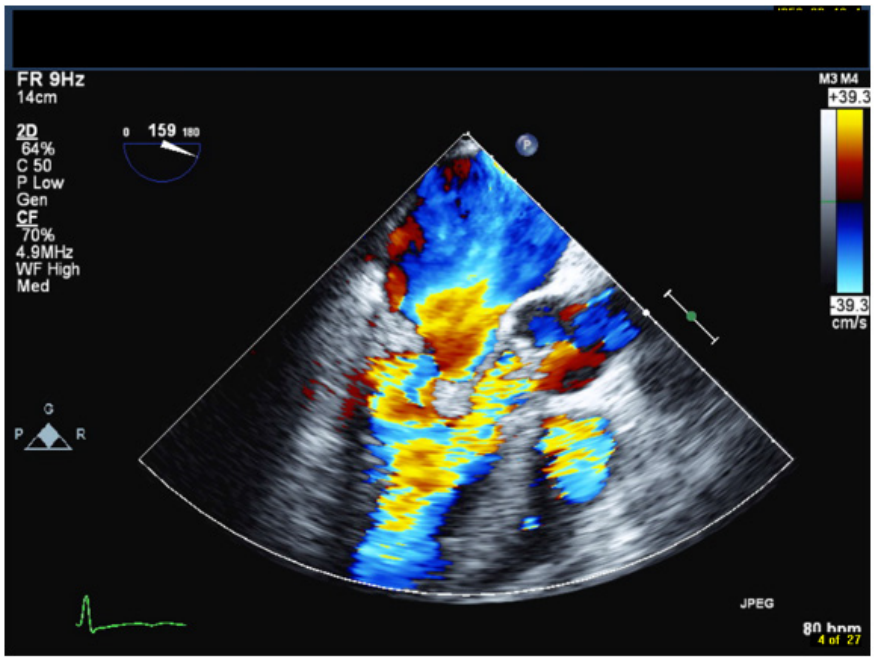

Figure 5: Aortic Reg.

Discussion

A.urinae was first identified in 1991 as a causative organism for endocarditis [1]. Since then, a total of twenty-three cases (including our patient) of *A. urinae* endocarditis have been reported in the literature to date (Table 1). Aerococci are gram-positive cocci that morphologically resemble staphylococci but have biochemical and growth characteristics of streptococci and enterococci [9]. *A. urinae* has preponderance in patients with underlying urinary tract pathologies and are reported to be found in 0.3 to 0.8% of urine samples. The incidence of *A. urinae* bloodstream infection is estimated to be 3 per 1,000,000 per year, as reported by a hospital which serves a 500,000 population. The organism is considered to be of low pathogenicity and may not always need to be treated. Sometimes this organism may initially be dismissed as a contaminant in clinical cultures from non-sterile sites. However, underdiagnoses of this organism can lead to disastrous outcomes [13].

Table 1: List of all of the cases of Aerococcus endocarditis published since 1992.

Age	Sex	Underlying Urological problems	Predisposing valvular disease	Valves involved	Initial Symptoms	Blood cultures +ve or –ve	Complications	Survived	Ref
81	M	Prostate cancer	-	-	-	+ve	MI and embolic complications	No	[1]
73	M	BPH/TURP	-	Aortic valve	Fever	+ve	Hemiplegia	No	[2]
78	M	Nephrolithiasis	-	-	-	+ve	None	Yes	[2]
55	F	-	-	Mitral and aortic valve	Fever and back pain	+ve	Cardiac complications	No	[2]
78	M	-	-	-	-	+ve	MI, Renal failure	No	[3]
89	M	TURP/suprapubic catheter	Degenerative mitral valve disease	-	-	+ve	Unknown	No	[4]
81	M	-	Aortic stenosis	-		+ve	MI	No	[5]
43	M	-	-	-	-	+ve	Septic embolization, MI	No	[6]
48	M	-	-	-	-	+ve	Hemisyndrome	Yes	[7]
79	F	-	Aortic insufficiency	-	-	+ve	CVA	Yes	[7]
75	M	BPH/Suprapubic catheter	-	Aortic valve	Fever	-ve Blood cultures/+ve PCR of aortic valve	Septic embolization	Yes	[8]

91	M	Urinary cathter, BPH, Renal cancer	-	Aortic valve	Fever	+ve	-	Yes	[9]
96	M	Urinary catheter	-	Mitral valve	Dysuria	+ve	-	Yes	[9]
86	M	Prostate cancer, Urethral stricture	-	Aortic valve	Fever, Dysuria	+ve	Septic embolization	Yes	[9]
62	M	BPH, Urolithiasis	-	Mitral and aortic valves	-	+ve	Cardiogenic shock	Yes	[10]
69	M	-	-	Aortic valve	-	-ve Blood culture/+ve PCR aortic valve	-	Yes	[11]
68	M	BPH	-	-	-	-	-	Yes	[12]
81	M	Yes	-	Mitral valve	Fever	+ve	Unknown	Unknown	[13]
78	M	Suprapubic catheter	-	Aortic valve	Sepsis	+ve	Unknown	Unknown	[13]
87	M	Yes	-	Mitral valve	Dyspnea	+ve	Unknown	No	[13]
77	M	BPH	-	Aortic valve	Found down at home	+ve	Sepsis/ Noncardiogenic pulmonary edema	No	[14]
80	M	-	-	Aortic valve	Fever	+ve	Septic emboli	Yes	[15]
42	M	Nephrolithiasis	-	Aortic, mitral and tricuspid valves	Found down at home	+ve	Septic emboli/ DIC	No	Present case

The clinical presentation for the infection can range from simple urinary tract infections, balanitis, cellulitis or spondylodiscitis [17], to life threatening illnesses such as septicemia [1], endocarditis, [10-16] or peritonitis [19]. Initial presenting symptoms can vary from fever, dysuria, sepsis, dyspnea, alter mental status to back pain in various case reports [18]. Advanced age, male sex and underlying urologic conditions, such as prostatic hyperplasia or chronic nephrolithiasis are risk factors for infection [18]. However, it has been described in young patients without known chronic medical conditions [6,7]. Apart from the above-mentioned risk factors, preexisting valvular dysfunction and systemic comorbidities such as ischemic heart disease, diabetes mellitus and malignancy have been proposed as risk factors for *A. urinae* endocarditis [4-7]. The most commonly involved valve is aortic followed by mitral; rarely the right sided valves are involved as seen in our case [18].

A urinae is a gram-positive, microaerophilic, catalase-negative, alpha-hemolytic coccus, growing predominately in tetrads and clusters. In contrast to streptococci, it is capable of growing in 6.5% NaCl. Its growth on 5% sheep-blood agar depends on incubation in carbon dioxide. Therefore, growth on urine-dipslide agar is not reliable and, as in our patient, blood cultures may be positive in the setting of negative urine cultures. Though correct identification of *A. urinae* with biochemical methods is possible, sequencing of the 16S rRNA gene remains the confirmatory test for identification. Although generally indistinguishable from endocarditis caused by other bacterial agents, one distinguishing feature is the relatively high incidence of systemic embolization (55% vs 20-40% for other microorganisms). It is believed that both platelet activation and biofilm formation participate in the pathogenesis of endocarditis caused by *A. urinae* [20]. While it is a rare cause for endocarditis it has an associated high mortality rate due to a significant incidence of embolic events. Complications include cardiogenic shock, septic emboli, renal failure, stroke and disseminated intravascular coagulation. Two frequently used antibiotics for urinary tract infections, cotrimoxazole and quinolones, have no or only moderate activity against *A. urinae* which is sensitive to B lactam and aminoglycoside antibiotics. Due to the rareness of *A. urinae* endocarditis, controlled studies are not possible and therefore antibiotic treatment is not standardized. In vitro susceptibilities of 56 isolates showed little inter-isolate variability with low minimal inhibitory concentrations (MIC) for penicillin, amoxicillin, piperacillin, cefepime, vancomycin and rifampicin, but variable MIC for ceftriaxone. Antibiotic therapy for at least 6 weeks is effective in patients with endocarditis who survive the 1st week of therapy.

Conclusion

In conclusion, urologic conditions predispose for infections with *A. urinae*. Generally considered to be of low pathogenicity, this microorganism may cause severe bloodstream infections, including endocarditis. The identification of *A. urinae* can be challenging. Late diagnosis of *A. urinae* endocarditis can be devastating; therefore physicians and microbiologists should consider endocarditis in patients when it is isolated from the blood.

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