Virtually All Complications of Active Infective Endocarditis Occurring in a Single Patient

William C. Roberts, MD[a,*], Divya Kapoor, MD[b], and Michael L. Main, MD[b]

Described herein is a 49-year-old black man with advanced polycystic renal disease, on hemodialysis for 6 years, who during his last 12 days of life had his vegetations on the aortic valve extend to the mitral and tricuspid valves, through the aortic wall to produce diffuse pericarditis, to the atrioventricular node to produce complete heart block, and embolize to cerebral arteries producing multiple brain infarcts, to a branch on the left circumflex coronary artery producing acute myocardial infarction, and to mesenteric arteries producing bowel infarction. **(Am J Cardiol 2020;137:127−129)**

Vegetations dangling on one or more aortic valve cusps (active infective endocarditis) are life threatening because they may extend to adjacent tissues (ring abscess) and/or embolize to arteries causing infarction. It is unusual for aortic valve vegetations to extend to 2 other cardiac valves or to embolize to multiple arteries or to cause septic pericarditis. Such was the case in the patient to be described herein.

Case Description

A 49-year-old black man with known advanced polycystic renal disease on hemodialysis (for 6 years), diabetes mellitus, systemic hypertension, and obstructive sleep apnea (without obesity—body weight 176 lbs.) had been in his usual health until he developed epigastric, back, and vague chest pain 3 days before hospitalization. Examination in the emergency room disclosed a grade 2/6 systolic murmur along the lower left sternal border. The patient indicated that his chest pain worsened when lying flat. The electrocardiogram was consistent with posterior wall acute myocardial infarction (Figure 1). His troponin level was 1.7 rising to 76 mg/L the next day. From the emergency room he was taken to the cardiac catheterization laboratory where coronary angiogram showed normal right (nondominant), left main, left anterior descending, and left circumflex coronary arteries, and a totally occluded third marginal coronary artery, which could not be opened. The following pressures in mmHg were recorded: pulmonary artery 47/24; pulmonary arterial wedge a 28, v 37, mean 24; aorta 82/54. Blood drawn for culture later disclosed methicillin-sensitive Staphylococcus aureus.

Despite appropriate antibiotic therapy his condition progressively worsened. Because of continued abdominal pain, abdominal imaging was performed which revealed bowel necrosis. Laparoscopic bowel resection was performed the day after admission and a 5 cm portion of necrotic ileum was resected. He became progressively more confused and magnetic resonance imaging of the brain showed several cerebral infarcts. During his 9 days in the hospital he progressed from sinus tachycardia with prolonged PR interval to second degree heart block (Wenckebach), to complete heart block (pacemaker inserted), and finally to fatal ventricular fibrillation.

At necropsy, the pericardial surfaces were diffusely covered by fibrin deposits (fibrinous "pericarditis") (Figure 2). The heart weighed 720g (normal < 350g). The aortic valve was 3 cuspid. Vegetation was present on each of the 3 cusps and calcific deposits were present in the left cusp. The vegetations extended through the wall of aorta to the space between the walls of the aorta and left atrium. The aortic vegetations extended on to the anterior mitral leaflet and into the calcium of the mitral annulus (Figure 3). The infection at the aortic and left atrial wall extended into the atrial septum to destroy the atrioventricular node and infect the adjacent tricuspid valve leaflet. All epicardial coronary arteries were wide open except for the third obtuse marginal which was totally occluded by a septic embolus. A large acute myocardial infarct involved the posterior left ventricular free wall and the posteromedial papillary muscle (Figure 3). Septic emboli were found in several small intracerebral arteries and in several mesenteric arteries.

Discussion

Of the various cardiac valves affected by infective endocarditis, the aortic by far is the most common and it also is associated with the most complications.[1] Chronic renal disease, as in the present patient is a major risk factor for infective endocarditis.[2] These complications include destruction of the aortic valve cusps causing aortic regurgitation, extension of the cuspal infection to adjacent tissues (ring abscess), including the anterior mitral leaflet and its chordae tendinea producing mitral regurgitation; to the atrioventricular node or bundle or both producing heart block; to the atrial septum (or membranous ventricular septum) to infect the tricuspid valve; through the aortic wall to produce pericardial disease, and to embolize to multiple systemic arteries. The patient described herein had all of these complications. The least common is pericardial disease.

[a]Baylor Scott & White Heart Institute, Baylor University Medical Center, Dallas, Texas; and [b]Saint Luke's Mid American Heart Institute, Kansas City, Missouri. Manuscript received July 7, 2020; revised manuscript received and accepted September 23, 2020.

*Corresponding author.

E-mail address: William.Roberts1@BSWHealth.org (W.C. Roberts).

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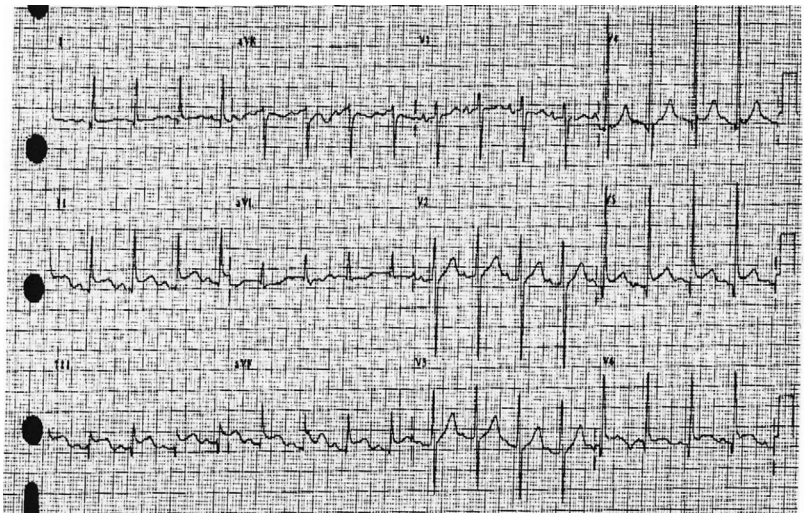

Figure 1. Electrocardiogram shortly after admission with findings of acute myocardial infarction.

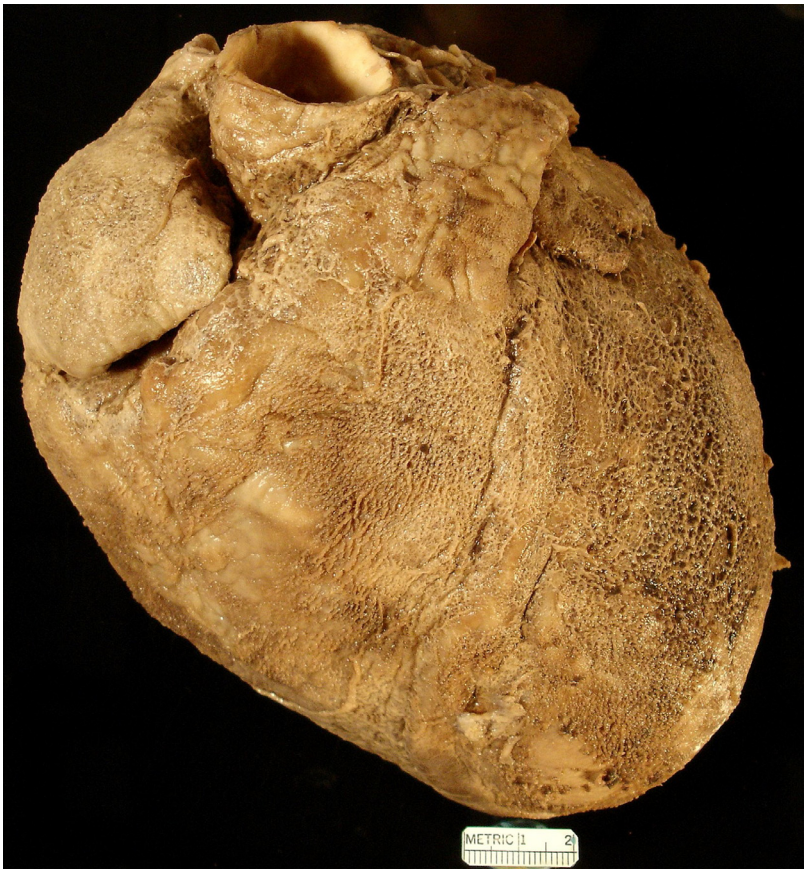

Figure 2. Heart showing acute diffuse pericarditis.

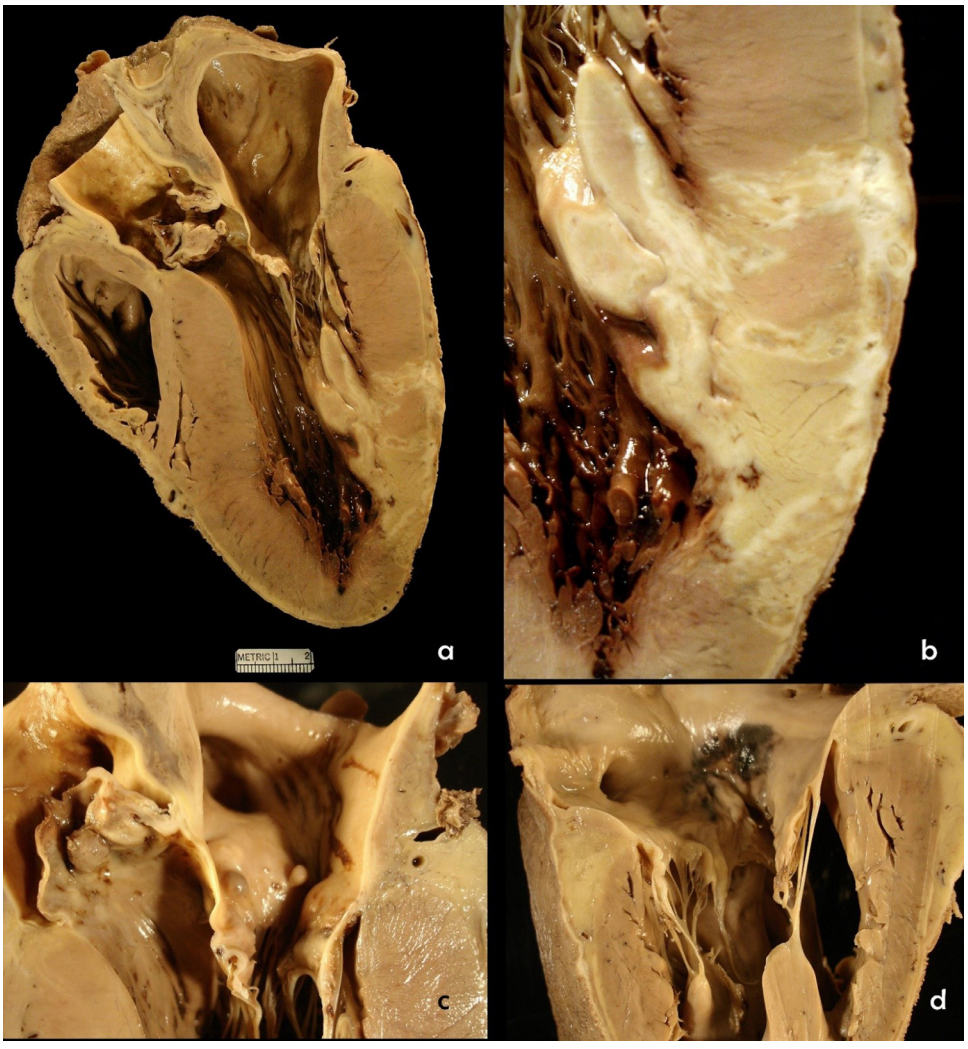

Figure 3. Views of the heart. (*a*) left parasagittal view showing the infection involving the aortic and mitral valves and the posterior wall acute infarct, a close-up of which is shown in (*b*). (*c*). A close-up view of the aortic and mitral valves showing vegetations on each. Ao = aorta; LA = left atrium (*d*). Right parasagittal view showing vegetation in the area of the atrioventricular node with extension into the septal tricuspid valve leaflet.

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