Snakebite and a heart murmur

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Figure 1. Admission electrocardiogram. See text for explication.

A 59-year-old commercial crawfisherman came to the emergency department complaining of a swollen, red, painful right hand and forearm and subjective fever. The hand was bitten by a snake 4 days earlier when the man was fishing. His only pertinent past history was that 3 years previously he had been sent for an echocardiogram when he saw a physician for fever and fatigue. The patient did not know the results of that test.

Findings on the current physical exam were a temperature of 101.8°F, an erythematous right hand with edema to the midforearm, and a harsh grade 3/6 systolic murmur best heard at the left sternal edge with radiation to both carotid arteries. An electrocardiogram was recorded (Figure 1), and the chest

radiographs were read as “no evidence of acute pleuropulmonary disease” (Figure 2).

The electrocardiogram shows sinus rhythm with a minimally prolonged (0.21 sec) P-R interval. The large negative P-wave deflection in lead V₁ indicates left atrial enlargement. Huge QRS

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Figure 2. Chest radiographs. Left: The posteroanterior film demonstrates a dilated ascending aorta (a) and a prominent left ventricle (L). Right: The lateral film shows the left ventricle (open arrow) extending well behind the inferior vena cava (solid arrow). The complete circle of calcium (surrounded by dark lines) represents calcific deposits within the aortic valve and indicates severe stenosis.

complexes meet most of the commonly used voltage criteria for left ventricular enlargement (where 1-mm deflection = 0.1 mv) (1): Sokolow and Lyon (2):

- a) $SV_1 + RV_5$ or $V_6 > 35$ mm or
- b) RV_5 or $RV_6 > 26$ mm or
- c) $RaVL > 10$ mm

Gubner, Ungerleider, and Rodstein (3, 4):

- a) $R_I + S_{III} > 25$ mm or
- b) $R_I > 15$ mm

McPhie (5):

- $R + S > 45$ mm (the largest R in any precordial lead plus the largest S in any precordial lead)

Lewis index (6):

- $(R_I + S_{III}) - (R_{III} + S_I) \geq 17$ mm

However, the electrocardiogram does not meet the now widely used and usually more sensitive Cornell criteria ($RaVL + SV_3 > 28$ mm in men or > 20 mm in women) (7).

In addition, the electrocardiogram easily fulfills the more complex point score of Romhilt and Estes for left ventricular enlargement (8):

- QRS voltage (3 points)
 - Largest R or S in limb leads ≥ 20 mm or
 - SV_1 or $SV_2 \geq 30$ mm or
 - RV_5 or $RV_6 \geq 30$ mm
- Left atrial enlargement (3 points)
 - Terminal negativity of $PV_1 \geq 1$ mm and duration ≥ 0.04 sec
- Left axis deviation (2 points)
 - $QRS \leq -30^\circ$ in frontal plane
- ST-T vector shifted opposite to the mean QRS vector as seen in Figure 1
 - 3 points in the absence of digitalis
 - 1 point in the presence of digitalis
- QRS duration ≥ 0.9 sec (1 point)
- Intrinsicoid deflection in V_5 or $V_6 \geq 0.05$ sec (1 point)
- Total possible points = 13
 - 4 points = probable left ventricular enlargement
 - 5 points = definite left ventricular enlargement

Our patient's score was 11 points, meeting all criteria except left axis deviation.

Although left ventricular enlargement of this magnitude may be seen with many conditions, in the vast majority of patients it is due to severe systemic arterial hypertension, advanced aortic valvular disease, or hypertrophic cardiomyopathy. The loud systolic murmur in this patient suggests valvular aortic stenosis or hypertrophic cardiomyopathy with obstruction. The chest radiographs (Figure 2) settle the question in favor of valvular aortic stenosis. The posteroanterior film shows post-stenotic dilatation of the aorta and left ventricular prominence. On the lateral radiograph, not only is left ventricular enlargement confirmed by the significant protrusion of the

left ventricle behind the inferior vena cava, but a full circle of calcific deposits is seen in the region of the aortic valve.

In a study of 148 patients with aortic valvular disease, the extent of valvular calcific deposits noted fluoroscopically or on lateral chest radiograph correlated positively with the peak systolic pressure gradient across the aortic valve in those with predominant stenosis (9). In addition, all 55 patients with grade 3 calcific deposits, as noted in Figure 2, had severe disease: stenosis in 29, stenosis and regurgitation in 25, and pure regurgitation in 1. The extent of aortic valvular calcification did not correlate with the degree of aortic regurgitation. Aortic valvular calcium is seen on the posteroanterior radiograph only rarely because in that projection the valve overlies the more heavily calcified spine.

Aortic stenosis presenting in adulthood usually is due to immobilization of the aortic valvular cusps by calcific deposits with little or no commissural fusion. A recent study of operatively excised valves found that weight, which in the heavier valves is primarily due to calcium, provides objective evidence of stenosis (10). In adults >70 years of age the valve is usually tricuspid, whereas in those younger than 70 it is more often congenitally bicuspid (11, 12). Presumably the inherently abnormal flow pattern across bicuspid valves (13) and defective microfibrils within the valvular leaflets (14) lead to earlier degeneration and dystrophic calcification than in tricuspid valves. Unicommissural, unicuspid valves usually are intrinsically stenotic and are responsible for most cases of aortic stenosis presenting in infancy or early childhood but may also present for the first time in adulthood (11, 12). Atherosclerotic plaques in the cusps themselves appear to play a role in the genesis of aortic valvular sclerosis and eventually stenosis, and evidence is accumulating that reducing atherosclerotic risk factors delays not only arterial disease but the onset and progression of aortic valvular disease (14, 15).

This patient's snakebite and resulting cellulitis, which responded promptly to antibiotics, had nothing to do with his cardiac murmur. As is often the case, a patient presenting with one medical problem has signs of an unrelated but potentially more serious condition. Echocardiography confirmed severe aortic valvular stenosis with a peak instantaneous pressure gradient of >100 mm Hg, a mean systolic gradient of 69 mm Hg, an aortic

valvular area of 0.6 cm², marked concentric left ventricular hypertrophy, a small left ventricular cavity, and normal left ventricular systolic function. Cardiac catheterization revealed normal right heart pressures, and coronary arteriography showed luminal irregularities without significant narrowing. At operation a heavily calcified congenitally bicuspid aortic valve was found. This morphology was predicted with considerable confidence preoperatively from the patient's age (<65 years) and the presence of aortic stenosis, a dilated aortic root and ascending aorta, and normal systemic arterial pressure (11, 12, 16).

Because the patient does sheet-metal work when crawfish are not in season, he did not wish to take warfarin sodium. Accordingly, his valve was replaced with a 25-mm Carpentier-Edwards Perimount bovine pericardial prosthesis rather than with a mechanical one. He had an uneventful course and was discharged on the fifth postoperative day.

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