A previously healthy 15-year-old boy with a remote history of repaired coarctation of the aorta and ventricular septal defect developed 3 days of fatigue, diarrhea, and intermittent fevers. He was seen by his primary care physician and diagnosed with viral enteritis. Enteral fluids were encouraged to maintain adequate hydration. However, his clinical course deteriorated over the next 4 days with worsening fatigue, intermittent chest palpitations, and shortness of breath. On the morning of hospital admission (approximately 10 days after the first fever), he developed pink urine, difficulty walking, and slurred speech. He was rushed to the emergency department for evaluation. Further questioning of the patient’s mother revealed no additional pertinent medical or surgical history. The patient was taking no medications, and he had no known drug allergies.

On physical examination in the emergency department, the patient was moaning and ill-appearing. His...
vital signs were as follows: temperature 38.7°C, heart rate 111 beats per minute, respiratory rate 28 breaths per minute, blood pressure 92/64 mm Hg, and oxygen saturation 98% on room air. He had a 2/6 diastolic murmur at the left sternal border, diffusely warm extremities with bounding pulses, a left-sided facial droop, 2/5 strength in his left upper extremity, and 4/5 strength in his left lower extremity.

Initial laboratory study abnormalities included a white blood cell count of 13.2 × 10^9 cells/L, a Na⁺ of 123 mEq/L, and a Cl⁻ of 85 mEq/L. The remainder of the values from the patient’s initial complete blood count, basic metabolic panel, and coagulation studies were within normal limits. An electrocardiogram in the emergency department revealed sinus tachycardia without dysrhythmia.

The patient was admitted to the intensive care unit with presumed septic shock and concern for acute neurologic compromise. Aggressive fluid resuscitation was initiated and blood and urine cultures were obtained. The patient received broad-spectrum antibiotic coverage with intravenous vancomycin, ceftriaxone, clindamycin, and metronidazole. Soon after, inotropic support was initiated secondary to persistent hypotension.

Noncontrast computed tomography (CT) of the head and a trans-esophageal echocardiogram (TEE) were then obtained (Figures 1-2).

Given the large vegetations noted on TEE, the hypodensity on head CT scan (with the patient’s neurologic signs), the patient’s prior cardiac history, and his fever, a definitive diagnosis of infective endocarditis (IE) was made according to the Duke criteria (Table 1).

**Case Challenge**

**Figure 2.** Trans-esophageal echocardiography reveals two mobile vegetations, approximately 15 mm × 20 mm, growing from the leaflets of the patient’s bicuspid aortic valve. AA = ascending aorta; LV = left ventricle.

**TABLE 1.**

<table>
<thead>
<tr>
<th>Major Criteria</th>
<th>Minor Criteria</th>
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<tr>
<td>• Positive blood cultures for IE.</td>
<td>• Predisposing heart condition or history of intravenous drug use.</td>
</tr>
<tr>
<td>- Typical microorganisms for IE from two separate blood cultures.</td>
<td>• Fever, defined as temperature &gt; 38.0°C.</td>
</tr>
<tr>
<td>- Persistently positive blood cultures with a microorganism consistent with IE.</td>
<td>• Vascular phenomena (eg, major arterial emboli, mycotic aneurysm, Janeway lesions).</td>
</tr>
<tr>
<td>- Single positive blood culture for <em>Coxiella burnetii</em> or IgG antibody titer &gt; 1:800.</td>
<td>• Immunologic phenomena (eg, glomerulonephritis, Osler’s nodes, Roth spots).</td>
</tr>
<tr>
<td>• Evidence of endocardial involvement.</td>
<td>• Microbiologic evidence not meeting major criteria as noted above.</td>
</tr>
<tr>
<td>• Positive echocardiogram for IE.</td>
<td></td>
</tr>
<tr>
<td>• New valvular regurgitation.</td>
<td></td>
</tr>
</tbody>
</table>

*IE = infective endocarditis; IgG = immunoglobulin G.

Data from Li et alious*

*A definitive diagnosis of infective endocarditis is established if two major criteria, one major and three minor criteria, or five minor criteria are fulfilled. A diagnosis is “possible” if one major criterion and one minor criterion are fulfilled or if three minor criteria are fulfilled.

Notes:
- **Table 1.** Summary of Modified Duke Criteria for Diagnosis of Infective Endocarditis*
- **Case Challenge:**
- **Figure 2.** Trans-esophageal echocardiography reveals two mobile vegetations, approximately 15 mm × 20 mm, growing from the leaflets of the patient’s bicuspid aortic valve. AA = ascending aorta; LV = left ventricle.
HOSPITAL COURSE
After acute resuscitation efforts, the patient underwent surgical replacement of the diseased aortic valve with a homograft; intraoperatively, an aortic valve annular abscess was discovered and drained. Three days postoperatively, two blood cultures grew out multiple species of *Staphylococcus epidermidis*. Pathologic analysis of the aortic valve vegetations (Figure 3) with genotyping confirmed the bacterial agent.

The patient was treated with 2 weeks of parenteral gentamicin and 6 weeks of parenteral vancomycin and ampicillin/sulbactam. He required several months of physical and occupational therapy. Six months postoperatively, the patient was functioning at a near-normal baseline with only mild left–upper extremity residual weakness. He was prescribed 5 mg of enalapril by mouth twice daily for mechanical valve–related aortic regurgitation.

DISCUSSION
In healthy children, IE is a rare diagnosis. A longitudinal study of children in Arkansas between 1990 and 2002 reported only approximately 0.6 cases of IE per 100,000 patients per year. Conversely, the incidence among children with congenital heart disease (CHD) is orders of magnitude greater, perhaps as high as 150 cases per 100,000 children. Abnormal cardiac anatomy impairs cardiac function and disrupts blood flow, which allows bacteria to colonize damaged valves or infect sterile thrombotic clots. Echocardiography revealed a bicuspid aortic valve. This anatomic variant, found in 30% to 40% of patients with coarctation of the aorta, is a well-known risk factor for IE.

Of all complications of IE, cardiac complications are most common, but the rate of neurologic sequelae is not negligible. A single-center, 17-year chart review in the United States reported that 6% (7 of 115) of cases of IE were complicated by stroke, close to the 11% reported among Japanese children with IE at 66 institutions between 1997 and 2001. Patients who suffer neurologic sequelae classically present with the triad of neurologic signs (eg, weakness, dysarthria), fever, and new-onset murmur. Morbidity and mortality are high. Of those children who develop neurologic sequelae, up to 60% may suffer permanent deficits. In-hospital mortality is approximately 5% for patients without pre-existing heart disease, but it approaches 50% for children with severe cyanotic disease.

The diagnosis of IE may be clinical or pathologic, according to the Duke criteria listed in Table 1. On the day of presentation, this patient fulfilled one major criterion (oscillating intracardiac valvular mass on TEE) and three minor criteria (predisposing cardiac lesion, temperature > 38°C, and evidence of arterial embolism). Of note, as stated in the most recent revisions of the Duke criteria, the role of radiologic imaging in the early diagnosis of patients can be...
crucial. In this case, without the TEE results, a major criterion would not have been fulfilled until several days after presentation, when multiple blood cultures returned positive for *S. epidermidis*.

When an infectious organism is identified, *Staphylococci* and *Streptococci* are the usual agents. However, the mechanism of infection is rarely known. Interestingly, in this case, an interval history from the patient’s mother revealed that the patient’s ex-girlfriend bit into his right arm 1 week prior to the onset of his symptoms. A review of the pertinent literature found that up to 40% of salivary and 60% of gingival plaque samples may contain multiple isolates of *S. epidermidis*. Thus, it is presumed that the patient’s ex-girlfriend inoculated his bloodstream with her oral flora, which attached to his bicuspid aortic valve, developed into IE, and precipitated an embolic stroke. Yet, although bites — human or otherwise — comprise an estimated 1% of all emergency department visits, IE remains an extremely rare complication. IE resulting from bites has been described mainly in case reports, and a thorough literature review found only one other documented case in which a human bite was the source of IE further complicated by neurologic sequelae.

Due to the risk of IE in patients with CHD, the American Heart Association publishes guidelines for the prescription of prophylactic antibiotics prior to dental and minor surgical procedures, which are well-recognized mechanisms for bacterial inoculation of the blood. The most recent revisions were published in 2007 and endorsed by the American Academy of Pediatrics.

**CONCLUSIONS**

Children with CHD are at increased risk of IE. Therefore, extremely vigilant follow-up is necessary to avoid significantly innocent clinical presentations. In the evaluation of IE by the Duke criteria, radiologic findings — namely CT and TEE — may be extremely useful for a timely diagnosis. This case, to our knowledge, is only the second documented report of IE complicated by neurologic sequelae that is postulated to be due to a human bite.

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**REFERENCES**