Variant Angina in the Setting of Food-Borne Botulism

To the Editor—A 41-year-old female presented with acute onset of dizziness, dysphagia, diplopia, dysarthria, and sensation of cotton balls in her mouth. The patient had no prior cardiac history or risk factors for coronary artery disease including smoking. Thirty-six hours prior to presentation, she consumed a can of soup that had been left open on the shelf for several days. Her symptoms rapidly progressed, and she developed severe proximal muscle weakness and respiratory failure requiring intubation and ventilation support. The results of an extensive neurological evaluation, including lumbar puncture, magnetic resonance imaging, magnetic resonance angiography, tensilon test, nerve conduction studies, and electromyogram, were unremarkable. A clinical diagnosis of botulism was made, and the patient was treated with equine serum trivalent antitoxin. Stool specimens and the soup tested positive for *Clostridium botulinum* toxin A. Normal hemoglobin and haptoglobin levels ruled out significant intravascular hemolysis. During her hospitalization, episodes of striking ST segment depression in the inferior and ST segment elevation in the anterolateral leads were noted (Figure 1). These episodes responded to intravenous nitroglycerin, and a nitroglycerin patch and an oral calcium channel blocker prevented further episodes. Serial cardiac enzymes showed no evidence of myocardial necrosis, and 2-dimensional echocardiograms revealed normal ventricular function without regional wall motion abnormalities. Ventilation support and cardiac medications were discontinued 6 weeks after presentation with no further electrocardiogram (EKG) changes noted and only mild proximal lower limb weakness at discharge.

Botulism is an acute paralytic illness produced by potent neurotoxins with types A, B, and E most often implicated. Autonomic dysfunction of both the sympathetic and parasympathetic systems is common in botulism [1] and may predispose patients to arrhythmias and sudden cardiac death. Minor EKG abnormalities have been described including conduction disturbances, arrhythmias and minor ST segment depression, and T-wave changes [2, 3]. In our patient, striking ST segment depression and elevation occurred, which resolved rapidly with nitrates and a calcium channel blocker supporting the diagnosis of coronary artery spasm (variant angina). Although the precise mechanisms remain elusive, endothelial cell dysfunction and heightened sensitivity to vasoconstrictors are postulated to play an important role in the pathogenesis. Recently, a hemolytic toxin (botulinolysin) has been identified and characterized from *C. botulinum*, which produces coronary vasoconstriction [4] experimentally by inhibiting endothelial-dependent dilatation [5]. Because the patient was not on any agents known to precipitate coronary spasm, we hypothesize that the toxin(s) induced focal coronary spasm.

Although rare, it may be prudent to monitor patients with acute botulism for coronary spasm because this may play a

Figure 1. A, Electrocardiogram (EKG) 5 days after intubation showing 2-millimeter ST segment depression and deep T-wave inversion in the inferior leads and nonspecific ST-T wave changes V₁–V₆. B, EKG performed 3 days later demonstrated marked ST segment elevation in the anterolateral leads and ST depression in inferior leads. C, After intravenous nitroglycerin and diltiazem, ST elevation resolved with residual biphasic inferior T waves.
role in sudden cardiac death, which occurs in ~6%–10% of patients with this disorder. Furthermore, because post-marketing literature reports arrhythmias, myocardial infarction, and fatal outcomes after cosmetic use of the toxin, particularly in patients with pre-existing conditions, we suggest that patients with known coronary artery disease may be at increased risk for vasospasm. Because botulism-like toxins are produced by a number of bacteria, we speculate that subclinical infections or consumption of preformed toxins may play a role in the pathophysiology of variant angina in patients with inherent endothelial dysfunction.

Note

Potential conflicts of interest. All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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References