Myocardial Infarction After Dog Bite

To the Editor:

We do not agree with the diagnosis of myocardial infarction in the case of a 36-year-old man 2 days after a dog bite. For several reasons, we believe that the patient had myopericarditis rather than myocardial infarction.

First, the patient had symptoms, signs, and laboratory findings consistent with sepsis, and blood cultures yielded Capnocytophaga canimorsus. Myocardial infarction occurs occasionally in patients with bacteremia, and it usually results from sepsis-associated hypotension or from endocarditis with septic embolism to the coronary arteries. However, viral and bacterial infections are more commonly the cause of pericarditis and myocarditis. ECG patterns of acute myopericarditis are known to resemble those seen with acute myocardial infarction. The presented ECG demonstrated ST elevations in both anterior and inferior leads in a manner consistent with the pattern found in acute myopericarditis.

Second, the absence of regional wall motion abnormalities and the documentation of diffuse hypokinesia with left ventricular systolic dysfunction are typically seen in myopericarditis. Abnormal regional wall motion is nearly universally present in acute myocardial infarction. Presentation with chest discomfort and laboratory examinations with elevated levels of creatine kinase and raised troponin-I concentrations are consistent with myopericarditis as well as with myocardial infarction.

Third, coronary angiography demonstrating normal vessels without signs of atherosclerosis is also consistent with the diagnosis of myopericarditis. This finding makes acute myocardial infarction less likely but does not definitely rule out previous coronary artery occlusion.

In summary, we think that the diagnosis of acute myopericarditis is more likely than acute myocardial infarction in the reported case.

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To the Editor:

Scharf and Widmer describe a case of dog bite resulting in sepsis, implicating the Gram-negative bacteria Capnocytophaga canimorsus, with accompanying chest discomfort, ST-segment elevation, and a cardiac enzyme rise. They conclude that myocardial infarction occurred as a complication of bacteremia “in the absence of both hypotension and endocarditis.” They did not consider another possibility that should have entered into the differential diagnosis given the following elements of the clinical presentation: (1) although the presenting ECG might at first glance suggest transmural ischemia and an acute inferolateral myocardial infarction, it is uncharacteristic to find ST elevation in lead I and an upsloping ST segment in V4 and V5, rather than reciprocal ST depression given this degree of ST elevation in inferolateral leads; (2) echocardiography showed diffuse left ventricular hypokinesia and not the typical regional akinesia expected with an inferolateral infarction; and (3) in the absence of embolic endocarditis or severe sustained hypotension, there is no plausible pathogenic link (and the authors suggest none) between bacteremia/disseminated intravascular coagulation and the acute occlusion of an epicardial coronary artery leading to myocardial infarction, and the latter is rarely, if ever, described as a complication of the former. The far more likely diagnosis is toxic-infectious myocarditis, which can notoriously mimic acute myocardial infarction. Acute myocarditis would better account for the atypical ECG features and the diffuse left ventricular hypokinesia in the context of a severe systemic clinical presentation.

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To the Editor:

I read with interest the report by Scharf et al. of a 36-year-old man with an acute myocardial infarction presenting 2 days after a dog bite that was associated with leucocytosis and elevated C-reactive protein (CRP). Although I agree with the authors that infection and bacteremia may precipitate an acute myocardial infarction, I disagree with their statement that “it usually results from hypotension or from endocarditis with septic emboli to the coronary arteries.” None of my patients with pharyngitis and consequent acute myocardial infarction had hypotension or evidence of endocarditis, and those catheterized had normal or minor coronary artery disease.

Inflammation, both local and systemic, plays a role in plaque vulnerability. Inflammation at the systemic level leads to elevated CRP and amyloid A levels and to activation of monocytes and adhesion molecules, all of which have been associated with acute coronary syndromes. Thus, the inflammatory response to the dog bite and the elevated CRP and leucocytosis, among other factors, led to endothelial dysfunction and changes in circulating clotting factors such as fibrinogen, which led to an increased clotting tendency and thrombotic coronary occlusion. Because no intravascular ultrasound was done, a minor atheroma instability or small plaque rupture associated with the inflammatory response cannot be excluded.

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In conclusion, Capnocytophaga canimorsus sepsis can rarely present as a distinct clinical pattern with disseminated intravascular coagulation and widespread capillary thrombosis, similar to Waterhouse-Friderichsen syndrome or thrombotic thrombocytopenic purpura, which can lead to extensive gangrene and a mortality >30%. Clinical diagnosis before time-consuming microbiological isolation is mandatory to insure early antibiotic treatment. The old Roman mosaic in the “House of the Tragic Poet” in the ruins of Pompeii with the engraved Latin comment cave canem (beware of dog) and our images should be kept in mind when taking care of patients with signs of acute myocardial infarction after animal bites.

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