A 36-year-old man who had undergone splenectomy several years earlier as a result of trauma was admitted with malaise and scattered hemorrhagic lesions on his nose (Figure, A). Two days earlier, his dog had bitten him. The next day, he developed chills, watery diarrhea, and vomiting. On admission, the patient complained of chest discomfort. On physical examination, his core temperature was 37.8°C, his blood pressure was 80/60 mm Hg, his heart rate was 90 beats/min, and his respiration was 10 breaths/min, with normal cardiopulmonary auscultation. At the site of the dog bite, a discrete lesion was visible, without any sign of infection (Figure, B). The surface ECG showed ST elevation in the inferolateral leads (Figure, C). Laboratory examination revealed a white blood cell count of 32 400 cells/μL, a C-reactive protein level of 549 mg/dL, a creatinine level of 268 μmol/L, disseminated intravascular coagulation with thrombocytopenia of 31 000 cells/μL, and D-dimers of 10.2 mg/L. Creatine kinase levels rose from 788 IU/L to 3029 IU/L after 6 hours, and they peaked at 3306 IU/L after 12 hours. The troponin-I concentration rose from 1.2 μg/L to 108 μg/L after 6 hours and declined to 38 μg/L on the second hospital day.

Diagnostic procedures included a thorough examination of the peripheral blood smear (Pappenheim stain). Polymorphonuclear leukocytes showed intracytoplasmic rods, which were Gram-negative (Figure, D). A presumptive diagnosis of *Capnocytophaga canimorsus* sepsis complicated by acute myocardial infarction was made on the basis of clinical presentation, ECG tracing, and laboratory results. Treatment with meropenem, heparin (10 000 IU per day), and isotonic fluid administration was begun. Coronary angiography was performed the next day, and it demonstrated normal vessels without signs of atherosclerosis. Transthoracic echocardiography showed normal valves, diffuse hypokinesia, and a decreased ejection fraction of 35%. The patient recovered rapidly and was dismissed in good condition after 2 weeks of antibiotic treatment. Blood cultures taken at admission yielded *Capnocytophaga canimorsus* (formerly designated DF-2 by the Centers for Disease Control).1

When acute myocardial infarction occurs in patients with bacteremia, it usually results from hypotension or from endocarditis with septic emboli to the coronary arteries. In our patient, however, and in 3 others with *Capnocytophaga canimorsus* bacteremia,2,3 acute myocardial infarction developed in the absence of both hypotension and endocarditis.

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**References**

A. Hemorrhagic lesions on the nose made the patient seek medical attention. B. Easily overlooked bite wound had no signs of local infection. C. ECG demonstrated transmural ischemia in the inferolateral leads. D. Polymorphonuclear leukocyte contains intracytoplasmic rods (arrow).
Myocardial Infarction After Dog Bite
Christoph Scharf and Urs Widmer

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