Capnocytophaga canimorsus Sepsis Complicated by Myocardial Infarction in Two Patients with Normal Coronary Arteries

Hans-Ulrich Ehrbar, Jacques Gubler, Stefan Harbarth, and Bernhard Hirschel

We describe two patients who had acute myocardial infarctions during episodes of Capnocytophaga canimorsus sepsis. C. canimorsus is associated with severe infection in patients who are immunocompromised; one of these patients had undergone splenectomy for Hodgkin’s disease 11 years earlier, and the other consumed significant amounts of alcohol regularly. Both patients owned dogs that had licked them or produced minor skin wounds shortly before they became ill. Coronary angiographic findings were normal for both patients. The association of acute myocardial infarction and sepsis with a specific pathogen is unique. This finding suggests that endothelial damage and coronary thrombosis due to C. canimorsus sepsis is a possible mechanism of acute myocardial necrosis.

Capnocytophaga canimorsus is a fastidious gram-negative rod associated with severe systemic infections that occur mainly in immunocompromised patients (patients with cirrhosis and those who have undergone splenectomy) following exposure to dogs and cats [1]. Although the organism is susceptible in vitro to many commonly used antibiotics including penicillin, C. canimorsus sepsis is associated with a high mortality rate of ≈28% [1]. Although myocardial depression is a well-described phenomenon in patients with sepsis attributed to high levels of TNF [2], surprisingly few reports deal with myocardial infarction during or following episodes of septicemia [3, 4]. We describe two patients with C. canimorsus sepsis that mimicked acute myocardial infarction.

Case Reports

Patient 1. A 52-year-old male had been superficially bitten on the forearm by his poodle ~7 days before his illness. He had smoked 20–30 cigarettes and consumed ~100 g of alcohol daily for several years. The patient went into acute cardiac arrest 18 hours after experiencing an initial episode of typical retrosternal chest pain. Cardiopulmonary resuscitation and orotracheal intubation were performed at his home. When he arrived in the emergency department, he was found to have supraventricular tachycardia, which was electrically converted; a diagnosis of acute myocardial infarction was made on the basis of electrocardiographic tracings (ST elevations of 1.5 mV in leads I and aVL) and evidence of corresponding regional hypomotility on an echocardiogram. Thrombolysis with recombinant tissue-type plasminogen activator was started, and the patient was mechanically ventilated and received vasopressor therapy for 24 hours. At the time of admission, laboratory studies revealed thrombocytopenia (platelet count, 13,000/μL); a C-reactive protein level of 302 mg/L (normal level, 0–5 mg/L); and a serum creatinine-kinase level of 13,000 U/L (normal level, 10–190 U/L) with an MB-fraction of 406 U/L (normal value, < 27 U/L), both of which decreased thereafter.

On day 2, the patient was hemodynamically stable, and he was extubated; however, he developed a fever. Blood cultures were drawn, and intravenous therapy with amoxicillin/clavulanic acid (2.2 g every 8 hours) and metimicin (150 mg every 12 hours) was initiated. The blood cultures subsequently yielded C. canimorsus, which was identified by standard methods [5]. The patient made an uneventful recovery. Exercise tests performed on day 10 showed no signs of ischemia; findings on an echocardiogram were normal, and coronary angiography on day 14 showed normal vessels with no myocardial hypomotility.

Patient 2. A 53-year-old man with no history of chest pain or coronary artery disease was admitted to the hospital because of high fever, chest pain, and malaise of 1 day’s duration. He had had Hodgkin’s disease 11 years earlier and had apparently been cured with splenectomy, radiation therapy, and three cycles of chemotherapy. During the weeks before admission, his dog had licked a superficial wound on his right leg. On admission, the patient had a severe headache, intermittent retrosternal chest pain, and extensive purpuric lesions on the face and both arms and legs. His temperature was 40.2°C; respirations, 32/min; pulse, 140/min; and blood pressure, 90/50 mm Hg. The WBC count was 5,000/μL with a significant left shift. The platelet count was 15,000/μL. An arterial blood gas analysis performed while the patient was breathing 40% oxygen by mask showed a pH of 7.31, a PCO2 of 30 mm Hg, and a PO2 of 109 mm Hg. The serum lactate level was 5.98 mmol/L (normal level, <1.9 mmol/L).

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Reprints or correspondence: Dr. Jacques G. H. Gubler, Medizinische Klinik, Stadtspital Triemli, CH - 8063 Zürich, Switzerland.
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Further laboratory investigations revealed diffuse intravascular coagulation (DIC), with a partial thromboplastin time of 179 seconds (normal time, <32 seconds) and a prothrombin time of 35% of normal. The fibrinogen level was 1.2 g/L (normal level, >2.0 g/L), and fibrin split products were present. The serum creatinine level was 152 μmol/L; the creatinine kinase level was 481 U/L (normal level, 5–270 U/L) and subsequently rose to 1,539 U/L, with a significant MB-fraction (maximal value, 122 U/L; normal value, <30 U/L).

The patient was treated empirically with intravenous cefazidime (2 g every 6 hours), gentamicin (120 mg every 12 hours), and metronidazole (500 mg every 8 hours). His hypotension persisted, requiring treatment with systemic epinephrine and dopamine. Fresh plasma, thrombocytes, and antithrombin III were given because of the severe DIC. Twelve hours after admission, electrocardiographic tracings revealed an evolving lateral myocardial infarction. Blood for cultures that was drawn on admission yielded long, thin, gram-negative bacteria that were subsequently identified as *C. canimorsus*. Treatment with intravenous clindamycin (900 mg every 8 hours) was continued for 2 weeks. He recovered fully.

On day 18, exercise tests revealed no signs of ischemia, findings on an echocardiogram were normal, and coronary angiography showed normal coronary arteries with mild myocardial hypomotility of the lateral left ventricle. The ejection fraction was 60%. The patient was asymptomatic when he was discharged after 3 weeks of hospitalization.

**Discussion**

Both patients presented with typical clinical and laboratory signs of acute myocardial infarction. In the first patient, the cardiac event preceded the manifestations of sepsis, while in the second patient, myocardial infarction evolved during sepsis. The high levels of cardiac enzymes, together with the rapid resolution of cardiac dysfunction and the documented regional hypomotility observed in patient 1, are best explained by temporary coronary occlusion and not by diffuse septic myocardial necrosis. Significant atherosclerotic disease was excluded on the basis of the normal coronary angiographic findings.

Although chronic infections with *Chlamydia pneumoniae* or *Helicobacter pylori* have been linked epidemiologically to chronic atheromatous disease of the coronary arteries [6], our search of the literature revealed only two recent reports concerning sepsis as a cause of acute myocardial infarction [3, 4]. Guest and co-workers [4] described 209 critically ill patients and found evidence of myocardial injury in 32, five of whom had sepsis [4]. *C. canimorsus* was not the offending agent in any of these patients (A. S. Jaffe, personal communication). To our surprise, the second report also concerned a case of sepsis due to *C. canimorsus* [3]. The patient, a 24-year-old kennel operator, was evaluated for chest pain without signs of infection. Electrocardiographic and enzymatic changes over time were suggestive of acute myocardial infarction. An angiogram showed an irregular filling defect, suggesting coronary thrombosis. The patient was discharged after 11 days and was readmitted to the hospital the next day when cultures of blood obtained on day 6 yielded *C. canimorsus*. At this time, the patient was febrile. He received a course of therapy with penicillin (12 million U/d) for 4 weeks. Findings on a repeated coronary angiogram obtained 2 months later were normal.

Infection with *C. canimorsus* has a propensity to cause severe systemic and local inflammatory responses, including DIC and gangrene [1]. Infection due to this organism is also believed to cause mononeuropathy of the musculocutaneous nerve by occlusion of the vasa nervorum and infarction of the nerve [7]. There are other reports of infarction affecting multiple organs [1, 8]. The association of myocardial infarction with *C. canimorsus* infection in three patients with normal coronary arteries is remarkable. None of these patients had evidence of endocarditis, which could have caused embolic occlusion of the coronary arteries.

We hypothesize that besides a systemic inflammatory response, *C. canimorsus* infection may induce localized endothelial damage in the coronary arteries, leading to thrombosis and subsequent myocardial infarction. We conclude that when patients at risk for *C. canimorsus* infection (i.e., those who have cirrhosis or who have undergone splenectomy) have an otherwise unexplained myocardial infarction and signs of sepsis, *C. canimorsus* bacteremia should be considered as a possible cause.

**References**