Case 8-2007: A 48-Year-Old Man with Chest Pain Followed by Cardiac Arrest

Gregory D. Lewis, M.D., Charles B. Holmes, M.D., M.P.H.,
Godtfred Holmvang, M.D., and Joan R. Butterton, M.D.

Presentation of Case

Dr. Steven J. Russell (Medicine): A 48-year-old man was admitted to this hospital in the early spring because of a 20-hour history of chest pain. The pain was substernal and was exacerbated when the patient was supine and inhaled deeply; it did not radiate and was not associated with shortness of breath, palpitations, nausea, or vomiting. The patient initially assumed that the pain was caused by heartburn, but it worsened overnight, and he visited his primary care physician in the morning. An electrocardiogram showed diffuse ST-segment elevation (2 to 4 mm) and PR-segment depression, and the patient was taken to the emergency department of this hospital.

He had hyperlipidemia but no history of angina, and he had previously been in good health, except for a 36-hour episode of nonbloody diarrhea 6 weeks earlier after he had eaten a chicken-salad sandwich purchased from a delicatessen. He worked in an office, was unmarried, did not smoke, and drank little alcohol. He had no recent history of travel or exposure to sick persons, pets, or other animals. He took no medications. He described the pain as 6 on a scale of 1 to 10, with higher numbers indicating worse pain.

On examination, the temperature was 37.1°C, the blood pressure 108/51 mm Hg, and the pulse 110 beats per minute. No cardiac murmur or pericardial rub was detected on auscultation, and the lungs were clear. The abdomen was nontender, without organomegaly. Repeated electrocardiographic examination showed the presence of diffuse ST-segment elevation and PR-segment depression (Fig. 1). Aspirin, a single intravenous dose of 2.5 mg of metoprolol, and sublingual nitroglycerin were administered, and the pain decreased to 3 on the scale of 1 to 10. Results of laboratory tests are shown in Table 1. A transthoracic echocardiogram showed no pericardial effusion, left ventricular hypertrophy, or left ventricular segmental wall-motion abnormalities. The ejection fraction was normal. Treatment with ibuprofen, at a dose of 800 mg every 8 hours, was initiated, and the patient was admitted to the medical floor for observation and cardiac monitoring. Shortly after the patient arrived on the floor, specimens of blood were obtained and sent for culture.

Twelve hours after presentation, the chest pain increased in severity. The electrocardiogram was unchanged. Results of laboratory tests performed at this time are...
shown in Table 1. Morphine, at a dose of 2 mg, was administered intravenously, and the pain resolved.

Approximately an hour later, in the early morning of the first hospital day, the cardiac monitor showed ventricular tachycardia at 260 beats per minute, and the patient was found pulseless and unresponsive. A biphasic shock at 150 J resulted in asystole for several seconds, followed by sinus bradycardia at 35 beats per minute without a pulse. Magnesium, at a dose of 2 g, and atropine, at a dose of 1 mg, were given intravenously, and sinus tachycardia developed, with palpable distal pulses and a systolic blood pressure of 160 mm Hg. He awoke almost immediately and was alert and oriented. A central venous catheter was placed; during the procedure he became agitated, and lorazepam was given for sedation. Shortly thereafter, his blood pressure fell to 60/40 mm Hg, and he again became unresponsive. Norepinephrine by intravenous infusion was initiated, and the trachea was intubated for airway protection; amiodarone, as a 150-mg bolus followed by infusion of 1 mg per minute, was administered intravenously, and he was transferred to the coronary care unit.

Two more specimens of blood were obtained for culture. The norepinephrine was quickly tapered, then discontinued, and the patient was extubated. He remained hemodynamically stable and alert, without chest pain; he had normal vital signs. Amiodarone was discontinued. Results of laboratory tests performed at this time are shown in Table 1. Later that day, cardiac catheterization showed left ventricular apical hypokinesis and normal coronary arteries. That evening, the temperature rose to 38.4°C, and two more samples of blood were obtained for culture. A nasal-swab specimen was negative for influenza A and influenza B, parainfluenza, respiratory syncytial virus, and adenovirus, and a culture of urine yielded no growth.

On the second hospital day, the patient felt well. The temperature was 37.5°C; no pericardial rub was heard, and the remainder of the physical examination was normal. The cardiac monitor detected no ectopy or arrhythmias. Treatment was initiated with metoprolol, at a dose of 25 mg orally twice a day. On the third hospital day, an electrocardiogram showed nearly complete resolution of the ST-segment elevation. On the fourth hospital day, the temperature was 37.6°C. A diagnostic test result was received.

Differential Diagnosis

Dr. Gregory D. Lewis: This 48-year-old man presented with chest pain and diffuse ST-segment eleva-
tions on the electrocardiogram and required resuscitation from cardiac arrest within hours after admission to the hospital. I saw this patient on admission as the cardiology consultant, and I will discuss the evolution of the differential diagnosis of his cardiovascular problem.

Chest Pain with ST-Segment Elevation on Electrocardiogram

This patient presented with chest pain and ST-segment elevation, a combination that invokes a differential diagnosis that clinicians must recognize to initiate prompt, appropriate therapy (Table 2). The distribution and shape of the ST-segment elevations and the associated electrocardiographic findings provide important clues to the diagnosis. In myocardial infarction caused by occlusive thrombus, ST-segment elevations are typically convex in shape and occur in a localized anatomical distribution, whereas this patient had widespread ST-segment elevations across the precordial and limb leads, as well as upward concave ST segments and PR-segment depression (Fig. 1). This pattern is highly characteristic of pericarditis. Furthermore, the ratio of the ST-segment elevation (in millimeters) to T-wave amplitude (in millimeters) in excess of 0.24 in lead V₆ provides additional evidence in support of the diagnosis of pericarditis, since this finding has previously been shown to reliably distinguish pericarditis from other repolarization abnormalities. Finally, this patient had pleuritic chest pain that worsened in the supine position, a finding also consistent with pericarditis.

Myocarditis must be considered when pericar-

### Table 1. Results of Laboratory Tests.†

<table>
<thead>
<tr>
<th>Variable</th>
<th>On Admission</th>
<th>12 Hours after Admission</th>
<th>First Hospital Day</th>
<th>Reference Range (in adults)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (mmol/liter)</td>
<td>139</td>
<td>138</td>
<td>135–145</td>
<td></td>
</tr>
<tr>
<td>Potassium (mmol/liter)</td>
<td>3.7</td>
<td>4.3</td>
<td>3.4–4.8</td>
<td></td>
</tr>
<tr>
<td>Chloride (mmol/liter)</td>
<td>104</td>
<td>106</td>
<td>100–108</td>
<td></td>
</tr>
<tr>
<td>Bicarbonate (mmol/liter)</td>
<td>29.1</td>
<td>26.7</td>
<td>24–30</td>
<td></td>
</tr>
<tr>
<td>Urea nitrogen (mg/dl)</td>
<td>20</td>
<td>22</td>
<td>8–25</td>
<td></td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.3</td>
<td>1.3</td>
<td>0.6–1.5</td>
<td></td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>116</td>
<td>241</td>
<td>70–110</td>
<td></td>
</tr>
<tr>
<td>Magnesium (mmol/liter)</td>
<td>0.8</td>
<td>1.2</td>
<td>0.7–1.0</td>
<td></td>
</tr>
<tr>
<td>Creatine kinase (CK) (U/liter)</td>
<td>302</td>
<td>530</td>
<td>60–400</td>
<td></td>
</tr>
<tr>
<td>CK-MB (ng/ml)</td>
<td>Negative</td>
<td>24</td>
<td>44.6</td>
<td></td>
</tr>
<tr>
<td>CK-MB index (%)</td>
<td>7.9</td>
<td>8.4</td>
<td>0–3.5</td>
<td></td>
</tr>
<tr>
<td>Troponin I</td>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Troponin T (ng/ml)</td>
<td>0.77</td>
<td>1.35</td>
<td>0–0.09</td>
<td></td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>41.9</td>
<td>39.3</td>
<td>34.6</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>14.4</td>
<td>13.0</td>
<td>12.1</td>
<td></td>
</tr>
<tr>
<td>White-cell count (per mm³)</td>
<td>18,900</td>
<td>16,700</td>
<td>14,000</td>
<td></td>
</tr>
<tr>
<td>Neutrophils (%)</td>
<td>93</td>
<td>85</td>
<td>40–70</td>
<td></td>
</tr>
<tr>
<td>Lymphocytes (%)</td>
<td>5</td>
<td>13</td>
<td>22–44</td>
<td></td>
</tr>
<tr>
<td>Monocytes (%)</td>
<td>2</td>
<td>2</td>
<td>4–11</td>
<td></td>
</tr>
<tr>
<td>Eosinophils (%)</td>
<td>0</td>
<td>0</td>
<td>0–8</td>
<td></td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate (mm/hr)</td>
<td>86</td>
<td>68</td>
<td>0–17</td>
<td></td>
</tr>
</tbody>
</table>

* To convert values for urea nitrogen to millimoles per liter, multiply by 0.357. To convert values for creatinine to micromoles per liter, multiply by 88.4. To convert values for glucose to millimoles per liter, multiply by 0.05551. To convert values for magnesium to milliequivalents per liter, multiply by 2. CK-MB denotes the MB isoform of creatine kinase.

† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at the Massachusetts General Hospital are for adults who are not pregnant and who do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.
Righ ventricular involvement

Typically confined to a single coronary vascular territory

Right coronary artery: Lead II>III, aVF

Left circumflex artery: Leads I, aVL, V6

Left anterior descending artery: Leads V1, V6

Acute pericarditis

Diffuse involvement of precordial and limb leads, associated ST-segment depression in aVRR

PR-segment depression, diffuse concave ST segments, and ST:T ratio >0.24 in lead V6

Clinical triad of chest pain, pericardial friction rub, and diffuse ST-segment elevations

Myocarditis

May mimic either myocardial infarction or pericarditis

May be associated with ventricular or atrial arrhythmias, heart block, or both

Clinical features vary, from asymptomatic abnormalities on ECG to fulminant heart failure and cardiogenic shock

Pulmonary embolism

Acute right ventricular overload may produce a pattern mimicking right ventricular infarction

Sinus tachycardia, incomplete or complete right bundle-branch block, S1Q3T3 pattern

Often hypoxemia with elevated alveolar–arterial oxygen gradient, and acquired or inherited hypercoagulable state

Type A aortic dissection involving a coronary ostium or the pericardium

Right coronary artery ostium involvement is more common than left main coronary involvement

If hemopericardium is present, low voltage with tachycardia

Abrupt onset of pain, widened mediastinum (63%), pulse differential

Apical ballooning syndrome

Anteroapical distribution is most common

Often associated with prolonged QT interval and deep T-wave inversions

Precipitated by profound emotional or physical stress, typically in women

Ventricular contusion

Right ventricular involvement

Follows blunt trauma; right ventricular involvement most common because of anterior location of the right ventricle

Table 1. Differential Diagnosis of ST-Segment Elevations on the Electrocardiogram (ECG) in Patients Presenting with Chest Pain.*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Distribution of ST-Segment Elevations</th>
<th>Associated ECG Features</th>
<th>Associated Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial infarction with occlusive thrombus</td>
<td>Typically confined to a single coronary vascular territory</td>
<td>Convex ST segment, abnormal Q waves</td>
<td>Elevations in troponin I or T arising within 6–8 hr after the onset of chest pain</td>
</tr>
<tr>
<td>Acute pericarditis</td>
<td>Diffuse involvement of precordial and limb leads, associated ST-segment depression in aVRR</td>
<td>PR-segment depression, diffuse concave ST segments, and ST:T ratio &gt;0.24 in lead V6</td>
<td>Clinical triad of chest pain, pericardial friction rub, and diffuse ST-segment elevations</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>May mimic either myocardial infarction or pericarditis</td>
<td>May be associated with ventricular or atrial arrhythmias, heart block, or both</td>
<td>Clinical features vary, from asymptomatic abnormalities on ECG to fulminant heart failure and cardiogenic shock</td>
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<td>Pulmonary embolism</td>
<td>Acute right ventricular overload may produce a pattern mimicking right ventricular infarction</td>
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<tr>
<td>Apical ballooning syndrome</td>
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<td>Often associated with prolonged QT interval and deep T-wave inversions</td>
<td>Precipitated by profound emotional or physical stress, typically in women</td>
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<tr>
<td>Ventricular contusion</td>
<td>Right ventricular involvement</td>
<td>Follows blunt trauma; right ventricular involvement most common because of anterior location of the right ventricle</td>
<td></td>
</tr>
</tbody>
</table>

* ST:T denotes the ratio of the ST-segment elevation to T-wave amplitude, S1Q3T3 an S wave in lead I and a Q wave and an inverted T wave in lead III, and lead III>II greater ST-segment elevation in lead III than in lead II.
lowed by an episode of sustained ventricular tachycardia. These events made us consider diagnoses other than pericarditis. Structural heart disease associated with coronary atherosclerosis, cardiomyopathy, or valvular heart disease is present in more than 90% of adults who have sudden cardiac death caused by ventricular arrhythmias.\textsuperscript{11,12} However, the results of coronary angiography were normal, and the echocardiogram showed a preserved left ventricular ejection fraction without evidence of hypertrophy or valvular heart disease.

In the absence of structural heart disease, we have to consider myocarditis, right ventricular dysplasia, and primary electrical disorders such as the Brugada syndrome,\textsuperscript{13} long QT-interval syndromes, and preexcitation.\textsuperscript{14} A primary electrical disorder is unlikely in this case, because the electrocardiographic findings were not characteristic of the Brugada syndrome,\textsuperscript{13} the QT interval remained normal, and there was no evidence of supraventricular tachycardia, which typically precedes ventricular tachycardia in preexcitation.

**MYOCARDITIS**

In a patient with a life-threatening ventricular arrhythmia in the absence of structural heart disease, myocarditis is a likely diagnosis.\textsuperscript{15,16} In infectious myocarditis, either a direct effect of the infectious agent or the resultant immune response can cause repolarization abnormalities in affected myocytes as well as arrhythmogenic foci in the form of microabscesses or microaneurysms.\textsuperscript{17,18} Twenty-four hours after admission, this patient had evidence of systemic inflammation, including persistent leukocytosis, an elevated erythrocyte sedimentation rate, and fever. Thus, myocarditis is the most likely explanation of this patient’s arrhythmia.

**DIAGNOSTIC TESTING FOR MYOCARDITIS**

Endomyocardial biopsy has limited sensitivity for the diagnosis of myocarditis, probably because of the focal and transient nature of inflammatory infiltrate.\textsuperscript{19-21} Furthermore, the procedure confers a 0.1% risk of death and a 0.25% risk of cardiac perforation.\textsuperscript{21} Therefore, endomyocardial biopsy is generally reserved for patients with rapidly progressive heart failure or arrhythmias in which a histologic diagnosis (i.e., eosinophilic or giant-cell myocarditis) is needed before the initiation of immunosuppressive therapy; a biopsy was not performed in this patient.\textsuperscript{22}

Several noninvasive methods have been used to study patients with suspected myocarditis. The echocardiographic examination may be normal in patients with acute myocarditis, as it was in this patient. Recently, magnetic resonance imaging (MRI) has emerged as the noninvasive imaging method of choice for detecting myocardial inflammation and necrosis. In biopsy-proved lymphocytic myocarditis, MRI has a sensitivity and specificity greater than 90%.\textsuperscript{23}

Although a marked increase in levels of cardiac troponin I or T can signal the presence of myocarditis, the elevations are not uniformly present and have not been shown to parallel the histologic severity of inflammation.\textsuperscript{24,25} Markers of immunoreactivity such as cytokines and antibodies to myosin, cardiac mitochondria, and sarclemma have limited value because of their lack of specificity.\textsuperscript{26}

A cardiac MRI was performed to determine whether this patient had findings consistent with viral myocarditis, evidence of a purulent pericardial-fluid collection, or evidence of other conditions associated with ventricular tachycardia, such as right ventricular dysplasia. Dr. Holmvang, may we see the imaging studies?

**Dr. Gøtfred Holmvang:** The cardiac MRI study addressed two questions: was there a structural abnormality such as right ventricular dysplasia that could cause the ventricular arrhythmias, and was there evidence of myocarditis? Right ventricular dysplasia is characterized by the presence of changes in adipose and fibrous tissues within the right ventricular myocardium and by wall-motion abnormalities of the right ventricle. Neither of these was found. The myocardial inflammation in myocarditis is associated with abnormally increased enhancement of the signal intensity of the myocardium early after the intravenous administration of gadolinium, as compared with the enhancement of the signal intensity of skeletal muscle; this enhancement is calculated as a ratio of the left ventricular signal to the thoracic skeletal muscle signature. In this case, cardiac MRI showed a small pericardial effusion, and the ratio of enhancement of the left ventricle to skeletal muscle was 3.8, which exceeded the normal threshold of 3.5\textsuperscript{27} (Fig. 2). These findings favor a diagnosis of myopericarditis. Delayed-enhancement imaging and an assessment of the ratio of myocardial to skeletal muscle on $T_1$-weighted images, which are now a routine part of the MRI
evaluation for myocarditis, were not available when this patient underwent evaluation.

**CAUSES OF MYOCARDITIS**

*Dr. Lewis:* Myocarditis can be caused by infectious agents, autoimmune disorders, toxins, and hypersensitivity reactions. This patient did not have a history of autoimmune disease and had not been exposed to cardiac toxins or other agents implicated in hypersensitivity reactions. Thus, an infectious cause seemed most likely, and infectious disease consultation was requested.

*Dr. Charles B. Holmes:* Estimates of the incidence of various infectious causes of myocarditis are variable because of the high proportion of cases that are asymptomatic and those for which a cause is never identified (Table 3). Viral infection is the most common cause of myocarditis in developed countries, and the most frequently identified virus is the enterovirus coxsackievirus B. The majority of enteviral infections occur in the late summer and fall, and an infection in the early spring, such as that in this case, makes this diagnosis less likely. Respiratory viruses including influenza, adenovirus, and respiratory syncytial virus are more common in the spring than are enterviral infections, although this patient did not have antecedent or concomitant symptoms to implicate these viruses in the diagnosis.

In cases of viral myocarditis, infection typically occurs 2 to 3 weeks before the onset of myocarditis. Thus, this patient’s self-limited diarrheal episode 6 weeks before the current admission could have been the first infection with a viral cause of myopericarditis. However, a general viral culture of stool obtained from the patient on the second hospital day was negative, and a nasalswab specimen obtained on admission was negative for influenza, parainfluenza, adenovirus, and respiratory syncytial virus antigens. Infections with human immunodeficiency virus (HIV) and hepatitis C virus have also been reported as causes of myocarditis, although this patient had no risk factors for their acquisition. 28,29

Fungal myocarditis is exceedingly rare and can usually be attributed to a specific geographic exposure (*Coccidioides immitis* and other fungi endemic to the area) or to suppression of cell-mediated immunity (aspergillus species and candida species). Parasitic infection with *Trypanosoma cruzi* (Chagas’ disease) or *Plasmodium falciparum* can cause myocarditis. However, infection with these pathogens requires exposure in areas in which the pathogen is endemic, outside the United States, and this patient had no history of such travel.

Although bacterial causes of myocarditis account for a small proportion of infectious cases, many bacterial pathogens have been implicated, including *Staphylococcus aureus*, *salmonella* species, *campylobacter* species, *Mycoplasma pneumoniae*, *Chlamydia psittaci*, and *Borrelia burgdorferi*, among others. 30 Bacterial infections typically occur clos-
er to the onset of myocarditis than do viral infections. Thus, the diarrheal episode occurring 6 weeks before this admission would not be a likely source of inoculation with a bacterial pathogen. The diagnostic test result received on the fourth hospital day was a report from the microbiology laboratory of the growth of thin, curved gram-negative rods in each of the three aerobic blood-culture specimens obtained on the day of admission.

Dr. Lewis: The presence of myonecrosis with high-grade bacteremia invokes three possible diagnoses. The first is sepsis without direct myocardial infection, which may promote myocardial-cell injury. However, most patients who have septic shock and myonecrosis have transient global depression in left ventricular function, whereas in this patient, myonecrosis occurred in the presence of normal blood pressure and preserved global left ventricular function. Whereas in this patient, myonecrosis occurred in the presence of normal blood pressure and preserved global left ventricular function. Bacteremia with elevated levels of troponin also raises the possibility of endocarditis with myocardial invasion, but this diagnosis is not supported by the pattern of epicardial injury on the electrocardiogram, the lack of antecedent constitutional symptoms, and the normal echocardiogram. The presence of bacteremia and biochemical evidence of myonecrosis makes pyogenic bacterial myocarditis the most likely diagnosis.

Dr. Butterton will describe the additional testing to characterize the organism.

**DR. GREGORY D. LEWIS’S DIAGNOSIS**

Bacterial myopericarditis.

**PATHOLOGICAL DISCUSSION**

Dr. Joan R. Butterton: Thin, curved, gram-negative rods grew from seven consecutive cultures of blood drawn over 5 days. This organism was notable for its fastidious growth. The morphologic features of the organism, which were clearly visualized in the primary cultures only by the use of acridine orange staining, were suggestive of a member of the group of campylobacter and helicobacter species (Fig. 3). A preliminary identification of this organism as a campylobacter species was made biochemically; however, poor growth of the isolate made biochemical testing unreliable. Further identification of the bacterial isolate was attempted by nucleic acid amplification of the *Campylobacter lpxA* gene, which suggested that the isolate was not *C. jejuni* or *C. lari*. Next, the organism was sent to the microbiology laboratory at the Texas Children’s Hospital, where the isolate was successfully subcultured. Purified chromosomal DNA was used for polymerase-chain-reaction (PCR)
amplification of the 16S rRNA gene, and on the basis of 16S ribosomal DNA sequencing, the organism was identified as *Helicobacter cinaedi*.

Dr. Holmes: *H. cinaedi*, previously known as *C. cinaedi*, is typically reported as a pathogen in immunocompromised patients, especially patients infected with HIV. Transmission has been linked to fecal–oral spread, well water, and hamsters. *H. cinaedi* has been implicated in a wide range of clinical illnesses, including proctocolitis, gastroenteritis, bacteremia, soft-tissue infections, septic arthritis, and meningitis. The only cases suggestive of cardiac involvement among those reported to the Centers for Disease Control and Prevention between 1982 and 1990 occurred in two patients with arrhythmias, not otherwise specified, and in one patient with an accumulation of pericardial fluid. To our knowledge, this is the first reported case of myopericarditis caused by *H. cinaedi*.

**DISCUSSION OF MANAGEMENT**

Dr. Holmes: We initially treated this patient with gentamicin and levofloxacin, guided by reports of in vitro testing and of successfully treated cases. Although fluoroquinolones have been used successfully in numerous cases of *H. cinaedi* infection, there are reports of relapse with resistant strains. Preliminary disk-diffusion sensitivity testing suggested that this patient’s isolate was resistant to gentamicin, and gentamicin and levofloxacin were discontinued in favor of intravenous meropenem, which had previously been reported to be successful in the treatment of this pathogen. The patient completed a 3-week course of meropenem without further symptoms.

Dr. Lewis: Since bacterial myocarditis is substantially less common than viral myocarditis, guidelines for management have been extrapolated from experience with viral myocarditis. Admission to the hospital for cardiac telemetry during an episode of active infection is important for early detection of arrhythmias or conduction defects, and in this patient, it was lifesaving. The patient did not have heart failure, but when heart failure is present, it is treated with diuretics, angiotensin-converting–enzyme inhibitors, and beta-blockers. Beta-blockers are particularly important if sustained ventricular tachycardia occurs, as it did in this patient. Avoiding sustained physical

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**Figure 3. Isolation and Characterization of Helicobacter cinaedi.**

Staining of the culture of blood obtained from this patient with acridine orange (Panel A) revealed long, thin organisms with a corkscrew appearance. Gram staining of the organism after growth on blood agar revealed the bacteria (Panel B, crystal violet). The phylogenetic dendrogram (Panel C) was based on 16S ribosomal DNA (rDNA) sequencing data. Variable regions within 16S rDNA, specifically region V1, were used for direct sequence comparisons. The isolate was identified as *H. cinaedi*. MGH and MGH 2 are distinct chromosomal DNA preparations of the same isolate.
exertion is recommended, in light of studies in animal models in which exercise increased viral replication and shortened survival and the fact that myocarditis is the third leading cause of sudden death from cardiac causes in young athletes.

This patient was kept on bed rest for the initial 7 days of his hospitalization and continued to receive beta-blockers throughout his hospitalization and on discharge. We recommended that after discharge he receive beta-blockers indefinitely.

Antiarrhythmic drugs and the implantation of a cardioverter–defibrillator have not been studied in controlled trials in myocarditis complicated by ventricular tachycardia. Because acute myocarditis often represents a transient condition from which recovery is common, the implantation of a permanent cardioverter–defibrillator should not be routine. However, for patients in whom chronic heart failure develops, treatment should mirror that for heart failure with dilated cardiomyopathy, in which implantable cardioverter–defibrillators have recently been shown to confer a survival advantage.47 This patient had complete resolution of his symptoms without further ectopy.

Dr. Holmvang: MRI repeated 6 months after his acute illness showed an enhancement ratio of left ventricular to skeletal muscle of 2.8, which is within the normal range.

Dr. Nancy Lee Harris (Pathology): What is the recent follow-up?

Dr. Lewis: The patient is doing well; his exercise restriction has been lifted, and he runs 8 km regularly, 3 years after the illness. Long-term treatment with beta-blockers was recommended, but the patient has not continued this therapy.

Dr. Hasan Bazari (Nephrology): Do you think the chicken salad was the source of his infection? Could this organism have established an infection and later become disseminated, we cannot rule out this possibility in this case. I think it is more likely that the source of the infection was a more recent exposure not known to or disclosed by the patient.

Dr. Harris: Dr. Butterton, how expensive was the identification of this organism, and how important is it to have an exact identification in deciding how to treat the patient?

Dr. Butterton: In comparison with the cardiology evaluation, the cost of the microbiology tests was trivial. The PCR amplification and 16S rDNA studies were performed by research laboratories without cost to this hospital and are not commercially available. We were able to use available susceptibility data to help guide therapy without knowing the exact identification of the organism. However, a definitive identification of this organism is important in understanding the natural history of this pathogen and will help to improve our empiric choices of therapy when faced with similar cases in the future.

ANATOMICAL DIAGNOSIS

*Helicobacter cinaedi* myopericarditis.

Dr. Holmes reports being an employee of Gilead Sciences and having equity ownership and stock options in the company; and Dr. Butterton, being an employee of Merck & Co., Inc., and having stock options in the company. No other potential conflict of interest relevant to this article was reported.

REFERENCES


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LANTERN SLIDES UPDATED: COMPLETE POWERPOINT SLIDE SETS FROM THE CLINICOPATHOLOGICAL CONFERENCES

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