Circulation Cardiovascular Case Series

Chest Pain and Palpitations
Taking a Closer Look

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A 25-year-old man of Pakistani origin born in the United Kingdom was admitted with a 3-day history of chest tightness and palpitations. The chest tightness was central and heavy in nature with no radiation. Palpitations were regular, and both symptoms occurred in paroxysms of up to 10 seconds. He had never experienced these symptoms before. He had a medical history of well-controlled asthma on β-adrenergic agonist and steroid inhalers, and he was morbidly obese with a body mass index of 43 kg/m².

He smoked 2 cigarettes per day and denied consuming alcohol or illicit drugs. He lived with his parents and worked as the manager of an online retail company. His mother had suffered a previous transient ischemic attack; his father had a myocardial infarction in his 40s and was treated for pulmonary tuberculosis in 1985.

Dr Lefroy: This young patient presents with recent chest pain and palpitations. The differential diagnosis is wide at this stage; in an older patient, it would be most important to consider and exclude acute coronary syndrome. Although uncommon in younger patients, an acute coronary syndrome can occur in those with traditional risk factors such as smoking, obesity, hypertension, and diabetes mellitus. A number of inherited disorders also predispose to premature coronary artery disease such as familial hypercholesterolemia and factor V Leiden. Other rare causes include spontaneous coronary artery dissection and paradoxical embolism through a patent foramen ovale. It is also important to consider vasospasm from illicit drugs such as cocaine.

Myocarditis should also be considered as a differential for this patient. Such patients may present with heart failure, cardiac arrhythmias, or chest pain mimicking acute coronary syndrome. It is also possible that cardiac arrhythmia is the primary pathology with the symptom of chest tightness secondary to tachycardia.

Finally, it is important to consider gastric pathology such as gastroesophageal reflux or esophageal spasm in the differential diagnosis. Young patients may also suffer from anxiety-related chest symptoms and palpitations with no organic pathology, but this should be a diagnosis of exclusion.

On examination, heart sounds 1 and 2 were present with no added sounds. The apex beat was not displaced and jugular venous pressure was not elevated. The chest was clear to auscultation. His abdomen was soft and not tender with no organomegaly. His blood pressure was 113/72 mmHg, pulse was 93 bpm, oxygen saturation was 96% on air, and temperature was 36.5°C. His admission ECG is shown in Figure 1.

Dr Lefroy: His ECG is abnormal and demonstrates sinus rhythm, occasional premature atrial contraction/paroxysmal ventricular contraction, and ST-segment elevation with biphasic T wave in leads V2 and V3. This pattern has been called a Wellens pattern that associated with proximal left anterior descending artery stenosis. Multifocal ventricular ectopics are a worrying feature in the presentation, indicating ventricular hyperexcitability secondary to either myocardial ischemia or inflammation.

He underwent urgent coronary angiography, which showed normal coronary arteries with no stenosis or occlusive disease. His chest radiograph was reported as normal. Blood results showed a raised troponin I assay combined with a leukocytosis, chest pain, ECG changes, and normal coronary arteries on angiography strongly favor the diagnosis of myocarditis. There is no clinical or radiological evidence of heart failure. The differential diagnosis of coronary artery disease–negative troponin-positive syndrome is given in Table 1.

Myocarditis commonly presents with symptoms of fatigue, decreased exercise tolerance, and palpitations. Patients frequently report precordial chest pain, and ≈6% will present with pain mimicking acute myocardial infarction. The most common pathological correlate is lymphocytic myocarditis of idiopathic or viral origin.

Further investigation is indicated in this presentation. Cardiac biomarkers are nonspecific and are elevated in only...
a minority of individuals. The ECG has poor sensitivity and specificity, with the most common abnormalities being non-specific ST/T-wave changes. Certain features (left bundle-branch block, abnormal cardiac axis, prolonged QTc interval >440 milliseconds, and frequent ventricular ectopy) are associated with a poorer prognosis. Echocardiography is readily available and allows the assessment of left ventricular function and the presence or absence of pericardial effusion and the exclusion of other diagnoses such as Takotsubo cardiomyopathy (stress) or hypertrophic cardiomyopathy. There are no specific features of myocarditis on transthoracic echocardiography; patterns mimicking dilated, ischemic, and hypertrophic cardiomyopathy have all been described. Cardiac magnetic resonance imaging (MRI) is an emerging valuable tool in the diagnosis of myocarditis, and an argument could be made for instead proceeding directly to this modality. A combination of T2-weighted images with early and late gadolinium enhancement is currently recommended. Cardiac MRI can lead to a diagnosis in 65% to 90% of individuals with coronary artery disease–negative troponin-positive chest pain and is able to discriminate between myocarditis (50%–60%), infarction (11%–12%), and Takotsubo cardiomyopathy (3.4%–14%). However, it is limited by cost and availability and is precluded in those with implanted cardiac devices or, as in this case, morbid obesity.

Viral infections are the most common identifiable cause of myocarditis, with viral genome detectable on endomyocardial biopsy in up to 67%, of cases. Classically, adenovirus and enterovirus (particularly Coxsackie B) have been implicated, but more recent evidence suggests a more prominent role for parvovirus B19 and human herpes virus 6,7,10 In one series, only 4% of patients with positive viral serology had that same viral genome identified on endomyocardial biopsy, giving a specificity of 9% and a sensitivity of 77%. However, routine serological testing is often performed and may help to confirm the diagnosis of viral myocarditis and to avoid the need for invasive procedures such as endomyocardial biopsy.

These patients normally respond to standard therapy for heart failure in the form of medications (angiotensin-converting enzyme inhibitor/β-blocker) and mechanical or pharmacological circulatory support as needed, particularly in fulminant presentations.

**Handheld transthoracic echocardiography was performed and documented normal left ventricular function. Short runs of supraventricular tachycardia were captured on telemetry. The diagnosis of myopericarditis was made, and the patient was started on a β-blocker, observed for 24 hours, and discharged home for outpatient follow up.**

**Dr Lefroy:** Patients with myocarditis present on a spectrum from subclinical disease to sudden death. This patient is not in cardiac failure, has normal left ventricular function, and has no sustained ventricular tachyarrhythmia on continuous monitoring. The patient’s initial ECG shows sinus rhythm, with frequent multifocal ventricular ectopics and 2-mm ST-segment elevation with T-wave inversion in leads V2 and V3.
ECG monitoring. Importantly, there is no evidence of pericardial effusion, which would be suggestive of an underlying inflammatory process and may predict increased risk of pericardial tamponade. The most likely pathological correlate is lymphocytic myocarditis, the treatment of which is supportive with standard heart failure medications if left ventricular dysfunction is present. Poor prognostic features include advanced New York Heart Association functional class, positive immunohistology, and lack of β-blocker therapy but not left ventricular ejection fraction. The presence of late gadolinium enhancement on cardiac MRI may be an independent predictor of cardiac and all-cause mortality. The disease is usually self-limiting, and although the mortality is high (20% at 1 year), there is no unequivocal evidence of benefit for the routine use of immunosuppression in these patients. We do believe, however, that treatment with high doses of corticosteroids should be considered in cases of biopsy-proven fulminating myocarditis in which there is rapid decline of ventricular function.

He presented again 23 days later with a recurrence of palpitations and newly developed shortness of breath on exertion. He was admitted from the ECG department with a broad QRS complex tachycardia (Figure 2) and a blood pressure maintained at 96/77 mm Hg. He was cardioverted chemically with amiodarone into sinus rhythm with frequent ventricular ectopic beats and was started on regular amiodarone and β-blocker. Troponin I was elevated at 0.116 µg/L (<0.03 µg/L). A formal departmental echocardiogram was performed that showed globally impaired left ventricular function with an estimated ejection fraction of 15% to 20% (Movie I in the online-only Data Supplement).

Dr Lefroy: Although a form of pre-excited tachycardia could conceivably cause this broad QRS complex tachycardia, the arrhythmia was considered most likely to be of ventricular origin because of the evidence of left ventricular impairment and the absence of pre-excitation during sinus rhythm in previous ECG recordings. There is subtle evidence of A-V dissociation with the suggestion of p waves superimposed on ST segments, defining this rhythm as ventricular tachycardia (VT).

This patient has developed sustained VT causing impaired left ventricular function. One must now consider rarer causes of myocarditis such as sarcoidosis, granulomatosis with polyangiitis, giant-cell myocarditis (GCM), hypersensitivity myocarditis, or cardiac lymphoma. A joint statement by the American Heart Association, American College of Cardiology, and European Society of Cardiology advocates endomyocardial biopsy in patients with symptom onset of between 2 weeks and 3 months’ duration associated with a dilated left ventricle, ventricular arrhythmias, heart block, or failure to respond to usual care within 1 to 2 weeks. Indications for endomyocardial biopsy are listed in Table 2.

Invasive cardiac electrophysiology testing was normal, with no inducible VT or accessory pathway demonstrated.

Dr Lefroy: An electrophysiological study was performed to confirm that the diagnosis of VT was correct and to rule out other causes of broad QRS complex tachycardia, especially pre-excited tachycardia such as antidromic atrioventricular reentrant tachycardia. The latter was unlikely in view of the absence of pre-excitation on the sinus rhythm ECG, but it was nonetheless considered important to exclude, particularly in view of the diagnostic uncertainty that confronted us at the time.

Six endomyocardial biopsies were taken. They were reported as showing separation of myocytes by an inflammatory infiltrate composed of lymphocytes, histiocytes, and an occasional multinucleate giant cell. There were no well-formed granulomata or necrosis (Figure 3).

Dr Sheppard: This is most consistent with a diagnosis of GCM. Myocarditis is often idiopathic or results from viral infection giving a lymphocytic myocardial infiltrate on histological examination. Of the rare causes, cardiac sarcoidosis and GCM have a similar histological appearance with giant cells present in both. The presence of granulomata and fibrosis favor sarcoidosis, whereas myocyte necrosis and eosinophilia in the absence of well-formed granulomata are more consistent with GCM.

Further investigation with cardiac MRI was desired but could not be accomplished because of the patient’s body habitus. An implantable cardioverter-defibrillator (ICD) was inserted. He was started on immunosuppressive therapy in the form of high-dose steroids (1 g methylprednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenously daily for 3 days and then 80 mg prednisolone intravenous...
The presenting features of GCM are not dissimilar to those of other forms of myocarditis. Lack of response to usual supportive care should prompt the clinician to consider GCM as a differential. Ventricular arrhythmias (29%) and high-grade heart block (15%) are found more often in patients with GCM or cardiac sarcoidosis than in those with lymphocytic myocarditis. Endomyocardial biopsy has a high sensitivity (85%) because of the pattern of diffuse involvement of the endocardium in most cases. Approximately 20% will therefore require a second procedure for diagnosis, and cardiac MRI can be useful in selecting an area to biopsy for a greater diagnostic yield. Although possessing high sensitivity and specificity in the diagnosis of myocarditis, cardiac MRI is unable to identify specific pathologies such as GCM. When there is a high index of suspicion but the right ventricular biopsy is normal, then a left ventricular biopsy should be considered, but the significant thromboembolic risk of this procedure should be taken into account.

Untreated GCM is associated with a poor survival rate; therefore, early diagnosis of GCM is vital because outcome is dramatically improved with appropriate immunosuppression. Trial data for GCM are limited to small observational studies and case series. A retrospective observational study in 1997 of 63 patients gathered internationally in the Giant Cell Myocarditis Treatment Registry found significantly increased survival associated with immunosuppression with prednisolone plus another agent such as cyclosporine or azathioprine (12.3 months) compared with conservative therapy or steroids alone (3.0 months). The rate of heart transplantation or death was 89%. A prospective trial comparing steroids with GCM reported in 2008 having enrolled 11 patients. As a result of issues with patients declining randomization, 7 were treated with steroids and cyclosporine for 12 months and 4 also received muromonab-CD3 for 10 days. Mortality at 1 year was lower at 9%, with 18% undergoing cardiac transplantation.

There are further anecdotal data from case reports of clinical improvement after immunosuppression with combinations of steroid, cyclosporine, azathioprine, muromonab-CD3, and rabbit antithymocyte globulin. Heart transplantation is an effective therapy, with a 71% 5-year survival; however, GCM recurs in ≈20% to 25% of patients.

The decision to use an ICD and the timing of ICD placement in such cases are challenging. We considered that this patient who had recurrent VT and deteriorating left ventricular function was at high risk of sudden arrhythmic death. However, with a potentially treatable cause having been identified, we were hopeful that there would be a good chance that left ventricular function would improve over time and that the risk of sudden death would decrease. Were that to be the case, the implantation of an ICD could be regarded as a bridge to recovery in the same way that an ICD may be implanted in some patients as a bridge to cardiac transplantation. The patient could therefore reasonably be discharged home with an ICD rather than having to stay hospitalized for an indeterminate period awaiting potential recovery.

VT ablation was considered but not undertaken during the early phase of the presentation because there was evidence of ongoing active myocardial inflammation, and there are no data to suggest that VT ablation improves the outcome in these circumstances. Most operators would reserve VT ablation for use in cases in which there is evidence of a fixed scar-related reentrant circuit usually related to previous myocardial infarction, where high success rate can be achieved. Even in these cases, there is no conclusive evidence that VT ablation improves patient survival rates; therefore, VT ablation can be considered to offer symptomatic relief only.

Dr Lefroy: GCM is a rare and frequently fatal disorder affecting young adults. It is a pathological diagnosis characterized by inflammatory cell infiltrate with myocyte necrosis and the presence of multinucleated giant cells that was made exclusively on autopsy until the advent of endomyocardial biopsy in 1987.

Table 2. Indications for Endomyocardial Biopsy

<table>
<thead>
<tr>
<th>Clinical Scenario</th>
<th>Class of Recommendation</th>
<th>Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>New-onset heart failure &lt;2-wk duration associated with a normal or dilated left ventricle and hemodynamic compromise</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>New-onset heart failure of 2-wk to 3-mo duration with a dilated left ventricle and new ventricular arrhythmias, second- or third-degree heart block, or failure to respond to usual care within 1–2 wk</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Heart failure of &gt;3-mo duration with a dilated left ventricle and new ventricular arrhythmias, second- or third-degree heart block, or failure to respond to usual care within 1–2 wk</td>
<td>lla</td>
<td>C</td>
</tr>
<tr>
<td>Heart failure with DCM with suspected allergic reaction and/or eosinophilia</td>
<td>lla</td>
<td>C</td>
</tr>
<tr>
<td>Heart failure associated with suspected anthracycline toxicity</td>
<td>lla</td>
<td>C</td>
</tr>
<tr>
<td>Heart failure associated with suspected restrictive cardiomyopathy</td>
<td>lla</td>
<td>C</td>
</tr>
<tr>
<td>Suspected cardiac tumours</td>
<td>lla</td>
<td>C</td>
</tr>
<tr>
<td>Unexplained cardiomyopathy in children</td>
<td>lla</td>
<td>C</td>
</tr>
<tr>
<td>New-onset heart failure of 2-wk to 3-mo duration with a dilated left ventricle without new ventricular arrhythmias, second- or third-degree heart block that responds to usual care within 1–2 wk</td>
<td>llb</td>
<td>B</td>
</tr>
<tr>
<td>Heart failure of &gt;3-mo duration with a dilated left ventricle without new ventricular arrhythmias, second- or third-degree heart block that responds to usual care within 1–2 wk</td>
<td>llb</td>
<td>C</td>
</tr>
<tr>
<td>Heart failure with unexplained HCM</td>
<td>llb</td>
<td>C</td>
</tr>
<tr>
<td>Suspected ARVD/C</td>
<td>llb</td>
<td>C</td>
</tr>
<tr>
<td>Unexplained ventricular arrhythmias</td>
<td>llb</td>
<td>C</td>
</tr>
<tr>
<td>Unexplained atrial fibrillation</td>
<td>III</td>
<td>C</td>
</tr>
</tbody>
</table>

ArVD/C indicates arrhythmogenic right ventricular dysplasia cardiomyopathy; DCM, dilated cardiomyopathy; and HCM, hypertrophic cardiomyopathy. Adapted from “The Role of Endomyocardial Biopsy in the Management of Cardiovascular Disease: A Scientific Statement From the American Heart Association, the American College of Cardiology, and the European Society of Cardiology” by Cooper et al.10

Orally once daily and rabbit antithymocyte globulin (150 mg over 6 hours, 3 doses over 3 consecutive days).
A computed tomography scan of the chest was performed, revealing a subcarinal lymph node mass measuring 53×26 mm and an enlarged right paratracheal lymph node. Computed tomography of the abdomen and pelvis showed a 75×82-mm lymph node mass in epigastrium near the porta hepatis with no calcification or cystic change. A fluorodeoxyglucose positron emission tomography computed tomography was also obtained, showing abnormal nodal activity in the right paratracheal and subcarinal regions with no increased uptake in the mass at the porta hepatis.

**Dr Lefroy:** Computed tomography imaging was sought because GCM is associated with inflammatory disorders in ≥20% of cases and has been associated with both hematologic and solid-organ malignancy, including thymoma.20 There is, however, no evidence to support the use of routine cross-sectional imaging in patients with GCM. No abnormalities were reported on the initial chest radiograph. At this stage, the main differential diagnoses in this patient were felt to be lymphoproliferative disease and tuberculosis.

Specialist opinions from the hematology and respiratory teams were sought. Further blood tests revealed a positive cytoplasmic antineutrophil cytoplasmic antibody with a proteinase 3 titer of 118. Mantoux testing was positive with 16-mm induration after 48 hours, and tuberculosis interferon-γ release assay was positive. Negative results included antinuclear antibody, rheumatoid factor, hepatitis B and C, and HIV. The following tests were normal: C3/4, serum protein electrophoresis, immunoglobulins, hepatitis B and C, and HIV. The following tests were normal: C3/4, serum protein electrophoresis, immunoglobulins, hepatitis B and C, and HIV. The following tests were normal: C3/4, serum protein electrophoresis, immunoglobulins, hepatitis B and C, and HIV.

The clinical suspicion of tuberculosis infection is high, suggested by positive Mantoux and interferon-γ release assay tests, granulomata on histology, and positive family history. It should be noted that both Mantoux and interferon-γ release assay tests are used in the diagnosis of latent but not active tuberculosis and that positive testing simply represents previous exposure. A diagnosis of active tuberculosis requires microscopy, culture of respiratory secretions or histology, and culture of tissue for suspected nonrespiratory tuberculosis. Prolonged culture is required for 3 to 8 weeks (sensitivity, 80%; specificity, 98%) and is necessary for identification and drug susceptibility testing. Newer tuberculosis nucleic acid amplification tests use a polymerase chain reaction technique to give a result within 24 to 48 hours (sensitivity, 92%; specificity, 98%). The presence of granulomata on biopsy is highly suggestive of active tuberculosis in this clinical context despite negative Ziehl Neelson staining and polymerase chain reaction, and he was started on quadruple antituberculous therapy accordingly.

He was readmitted to hospital on day 80 after the initial presentation with further episodes of palpitations. Interrogation of his ICD showed 6 episodes of VT requiring electric cardioversion. A repeat echocardiogram now showed normal left ventricular systolic function with a normal cavity size (Movie II in the online-only Data Supplement). Resting ECG demonstrated resolution of anterior ST-segment change, with persisting T-wave inversion anteriorly (Figure 4).

Culture results became available from the mediastinal lymph node fine-needle aspirate. They confirmed fully sensitive mycobacterium tuberculosis, and he was stepped down to dual antituberculous therapy with rifampicin and isoniazid.

A further 6 shocks from the ICD led to another admission 166 days after the initial admission. Device interrogation showed recurrent episodes of VT successfully treated by antitachycardia pacing (shown in Figure 4) and a further 6 episodes of VT terminated by electric cardioversion.
His antiarrhythmic drug therapy was adjusted with mexiletine changed to flecainide. A repeat endomyocardial biopsy was performed. It revealed no active inflammation but evidence of myocardial fibrosis (Figure 5). A repeat computed tomography of the thorax/abdomen/pelvis was performed, showing a reduction in the size of the mediastinal lymph node masses but very little reduction in the mass at the porta hepatitis.

Dr Lefroy: His ongoing ventricular arrhythmias are secondary to reentrant circuits associated with myocardial scarring from the now resolved inflammatory process. His left ventricular function has significantly improved with treatment and is now normal.

Flecainide was chosen for its high degree of antiarrhythmic effectiveness, lack of tendency to QT prolongation and torsade-de-points, ease of administration both orally and intravenously, short half-life (eg, compared with amiodarone), and low incidence of extracardiac side effects. The cautions engendered by the findings of the Cardiac Arrhythmia Suppression Trial (CAST) were not felt to be directly applicable in this case, given that CAST included only patients with ischemic heart disease and the main concern was an increase in the risk of sudden, presumed arrhythmic, death from which our patient was considered to be protected by the presence of an ICD. CAST predated the widespread use of ICDs, and the findings of CAST do not preclude the use of class I drug treatment to suppress recurrent symptomatic VT in patients who are protected from sudden arrhythmic death by an ICD.

Tuberculosis causing myocarditis is rare. There is cardiac involvement in ≈1% of tuberculosis cases, but it affects primarily the pericardium. Myocardial involvement was first reported in 1664, but it is described by only a handful of case reports in the literature and is usually a postmortem diagnosis. As with GCM, tuberculosis myocarditis is being recognized more frequently antemortem with the availability of percutaneous endomyocardial biopsy, but it is still extremely rare. Three routes of cardiac spread are proposed: direct infection from the pericardium, hematogenous seeding, and lymphatic spread. Three patterns have similarly been described: miliary,
diffuse infiltrative, and a nodular type with central caseation. A set of criteria for the histological diagnosis of tuberculosis myocarditis (not covered in the Dallas Criteria) has been proposed, but there is currently no consensus.21 Tuberculosis polymerase chain reaction testing was not performed on the initial endomyocardial biopsy samples in this patient.

Response to antituberculosis therapy is suggested by the reduction in the size of the mediastinal lymph nodes on repeat cross-sectional imaging and by normalization of left ventricular function. It is unclear why the appearance of the mass at cross-sectional imaging and by normalization of left ventricular function remained unchanged.

Summary
This case highlights the importance of considering a wide differential diagnosis in a young patient with chest pain and an abnormal ECG. Rarer causes of myocarditis such as GCM should be sought in patients who develop ventricular arrhythmias or high-grade heart block because the treatment is different and dramatically influences outcome. Our patient is the first reported case of GCM and a concurrent diagnosis of tuberculosis. It is most likely that the histological appearance of GCM was due to the presence of mycobacterial infection within the myocardium, and we believe that effective antituberculosis therapy has led to resolution of the GCM without the need for continued long-term immunosuppression.

Disclosures
None.

References

KEY WORDS: arrhythmia, cardiac defibrillator, implantable electrophysiology, heart failure, myocarditis, tachycardia, tuberculosis, ventricular
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Movie legend

Movie 1. Transthoracic echocardiogram: apical 4 chamber view shows globally severely impaired left ventricular ejection fraction.

Movie 2. The repeat transthoracic echocardiogram post treatment shows complete resolution of left ventricular impairment with a normal ejection fraction.