An 18-year-old man with no previous cardiac history presented to his local hospital complaining of severe chest pain. One day before admission, he was hit in the chest by a ball during a basketball game. He did not notice any symptoms and was able to finish the game; however, the next day, he developed severe central chest pain. Cardiac troponins were elevated at 79 µg/L (normal range, 0 to 1.2 µg/L). He subsequently underwent cardiac catheterization, which showed normal coronary anatomy with no significant stenoses. Subsequently, he had another episode of chest pain that was associated with diffuse ST elevation involving the anterior and lateral leads. The ST elevation persisted for >90 minutes before subsiding spontaneously. On transfer, the patient was specifically questioned about recreational drug use. He denied any use of cocaine, although he did admit to using marijuana 1 week before admission. On examination, he was pain free and hemodynamically stable. An echocardiogram showed minimal persistent ST segment elevation in the anterior leads (Figure 1). A cardiac magnetic resonance imaging (MRI) scan (Magnetom TRIO, Siemens, Germany; field strength 3 T) was performed to assess morphology of myocardial injury. This scan demonstrated normal left ventricular size with mild left ventricular systolic dysfunction (Movie I in the online-only Data Supplement). Left ventricular ejection fraction was calculated at 42% using QMass® MR version 7.0.28 (Leiden, The Netherlands). There was severe hypokinesis of the basal to midanterolateral, inferolateral, and inferior walls and hypokinesis of the midanterolateral and apical segments. On delayed enhancement imaging, there was diffuse patchy midmyocardial and epicardial late gadolinium enhancement involving these segments (Figure 2A and B). The subendocardium was spared. T2-weighted imaging revealed high-intensity signal in these areas, suggesting the presence of myocardial edema (Figure 3). On rest perfusion, a perfusion defect was seen, which involved the basal to midlateral walls. Overall, the findings were strongly suggestive of myocarditis. Subsequently, his parvovirus B19 antibody levels were noted to be elevated at 5.5 index value (normal range, 0 to 0.89 index value), confirming the diagnosis of parvovirus B19-induced myocarditis. The patient was commenced on angiotensin-converting enzyme inhibitors and discharged. He was reviewed in our clinic after 10 weeks and reported feeling significantly better with no specific ongoing symptoms. An echocardiogram showed improvement in left ventricular function with improvement in contractility of the lateral wall (Movie II in the online-only Data Supplement).

Discussion
This case highlights the utility of cardiac magnetic resonance (CMR) in making a confirmatory diagnosis of acute myocarditis in a patient presenting with symptoms, signs, and an echocardiogram suggestive of an acute myocardial infarction in the absence of angiographically significant coronary artery disease.

Approximately 10% of patients who present with symptoms suggestive of coronary ischemia and elevated troponins are found to have normal or only minimally diseased coronary arteries on angiography. In most cases, acute myocardial infarction secondary to coronary vasospasm or spontaneously reperfused coronary occlusion is suggested, but no definitive diagnosis is sought.

Myocarditis represents infectious, autoimmune, or toxic inflammation of the heart. The spectrum of clinical presentation is wide, ranging from absence of any symptoms to fulminant cardiogenic shock and sudden death. Myocarditis can also cause severe vasospasm and hence mimic acute myocardial infarction with acute onset of chest pain, echocardiogram abnormalities, and elevated cardiac enzymes. This pattern of severe vasospasm mimicking acute myocardial infarction is most commonly seen in patients with biopsy-proven parvovirus B19-induced myocarditis. Increases in oxidative stress, reduced bioavailability of vasodilator nitric oxide availability, and ensuing endothelial dysfunction have been implicated as possible mechanisms for induction of vasospasm.

The clinical diagnosis of myocarditis can be challenging because of the often nonspecific pattern of clinical presenta-
tion and lack of universally accepted and standardized diagnostic criteria. Postmortem findings identify myocarditis as a cause of sudden cardiac death in 20% of men younger than age 40, and yet this diagnosis is made far less frequently in day-to-day clinical practice. Traditionally, the diagnosis has been made by application of the so-called Dallas criteria, which required identification of inflammatory cellular infiltrate with or without myocardial necrosis on conventionally stained myocardial tissue biopsy specimens. A histological diagnosis was found to be prone to sampling error, considerable interobserver variability, and low sensitivity, however, and the Dallas criteria are now considered to be inadequate to reliably make the diagnosis.4,5 Immunohistologic techniques are more sensitive but still require an endomyocardial biopsy, which is highly invasive and not without risk.

More recently, cardiovascular magnetic resonance with the adjunctive use of gadolinium has shown great promise as a means of diagnosing myocarditis without the requirement of a biopsy.6 Studies by Mahrholdt et al have demonstrated the presence of infiltrates characterized by areas of enhancement (late gadolinium enhancement) on approximately 10 minutes of gadolinium administration appear to correlate well with regions of histologically proven myocarditis.7 These infiltrates characteristically are located in the midwall and tend to spare the subendocardium, which differentiates them from areas of infarction. Mahrholdt’s group has also demonstrated that clinical presentation, pattern of delayed enhancement on CMR, and clinical course are related to the type of virus present in the myocardium.3 Parvovirus B19-induced infection tends to present commonly as severe acute recurring
chest pain and is characteristically associated with delayed enhancement in the lateral wall. In general, patients have a benign course with significant or complete recovery. In contrast, patients with human herpesvirus 6 infection tend to present with subacute, new onset of heart failure. These patients tend to have delayed enhancement in the midwall of the interventricular septum and tend to have an overall worse prognosis with further deterioration in left ventricular function. Although CMR using different sequences, T1 and T2 weighting, and late gadolinium enhancement is specific for diagnosis of myocarditis, sensitivity is still limited because current MRI techniques only provide a qualitative visual assessment of myocardial fibrosis. Myocarditis often proceeds from a focal to a global pattern, and only 30% to 40% of patients present with focal myocardial injury that is visible as infiltrates of late gadolinium enhancement on the CMR. Much current research is being focused on the development of techniques that may allow quantitative assessment of diffuse or global myocardial fibrosis. It is likely that when these techniques become available, sensitivity of CMR for making this diagnosis will be improved significantly.

Disclosures

None.

References

Parvovirus B19-Induced Myocarditis Mimicking Acute Myocardial Infarction: Clarification of Diagnosis by Cardiac Magnetic Resonance Imaging
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