Utility of cardiac magnetic resonance in recurrent myocarditis

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We report a 26-year-old man who presented to the emergency department four times within a 4-year period with recurrent myocarditis. His presentations were characterized by chest pain, elevated troponin I, and normal coronary angiography. Endomyocardial biopsy showed nonspecific inflammatory process. Laboratory workup including viral screening and autoimmune markers were negative. Cardiac magnetic resonance imaging showed evidence of recurrent myocarditis with progressive appearance of new areas of myocardial delayed enhancement seen in each admission.

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Introduction

Myocarditis is inflammation of the heart muscle that may be due to infectious or noninfectious causes [1]. The diagnosis of myocarditis can be challenging but often requires endomyocardial biopsy (EMB), which is highly encouraged to be performed in all cases suspected to have myocarditis by the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases [1]. Cardiac magnetic resonance (CMR) has an important role in the diagnosis by providing noninvasive myocardial tissue characterization. In the following case report, we present clinical features, cardiac enzymes level, and CMR findings in a patient with recurrent myocarditis.

Case report

A 26-year-old man, a known patient of diabetes mellitus type-1, presented to the emergency department in March 2009 with sudden onset of retrosternal chest pain of 3 hours’ duration. The pain was partially relieved by sublingual nitrate. Other than being a heavy smoker, he had no significant past medical history. On examination, he was conscious, afebrile, and not in distress. His blood pressure was 120/60 mmHg and heart rate was 106 beats/min. Physical examination including cardiovascular examination was unremarkable. Urgent electrocardiogram in the emergency room showed sinus tachycardia without ST segment changes (Fig. 1). Blood investigations showed elevated cardiac markers. Other investigations were normal including complete blood cell
Table 1. Screening for connective tissue disease and viruses were done. Some were repeated more than once.

| Virology                                      |                  |
|-----------------------------------------------|------------------|
| Coxsackie virus antibodies (A9, B1, B2, B3, B4, B5, B6) | Negative         |
| HAV IgM                                       | Negative         |
| HAV IgG                                       | Positive         |
| AntiHbc                                       | Negative         |
| AntiHbs                                       | 1.9              |
| HBsAg                                         | Negative         |
| EBV-IgG                                        | 390.00           |
| EBV-IgM                                        | 10.00            |
| EBNA-IGG                                        | 60.70            |
| EBV-EA                                        | 5.40             |
| CMV IgM                                        | Negative         |
| CMV-IgG                                        | 147.30           |
| VZV-IgG                                        | 1168.00          |
| VZV-IgM                                        | 0.10             |
| HSV 1/2 IgM                                    | Negative         |
| HSV 2-IgG                                      | 0.92             |
| HSV 1-IgG                                      | 45.30            |
| Hepatitis C Antibody                           | Negative         |
| ASOT                                          | <200             |

| Connective tissue disease                     |                  |
| ANA                                           | 0.50             |
| Anti-dsDNA                                     | 6.05             |
| CRP                                           | <3.5             |

| Toxicology/others                             |                  |
| Amphetamine screen                            | Negative         |
| Barbiturates screen                            | Negative         |
| Benzodiazepine screen                          | Negative         |
| Cocaine screen                                 | Negative         |
| Opiate screen                                  | Negative         |

(continued on next page)
Table 1 (continued)

| Cannabis screen | Homocysteine | Negative |
|-----------------|-------------|----------|
| ANA = antinuclear antibody; Anti-dsDNA = anti-double strand DNA; AntiHbc = hepatitis B core antibody; AntiHbs = hepatitis B surface antibody; ASOT = Antistreptolysin O titer; CMV = cytomegalovirus; CRP = C-reactive protein; EBNA = Epstein–Barr nuclear antigen; EBV = Epstein–Barr virus; EBV-EA = Epstein–Barr early antigen; HAV = hepatitis A virus; HBsAg = hepatitis B surface antigen; HSV 1 = herpes simplex virus 1; HSV 2 = herpes simplex virus 2; HSV1/2 = herpes simplex virus 1/2; Ig = immunoglobulin; VZV = varicella zoster virus. |

Figure 2. Coronary angiography showed normal coronary arteries.

| 3/23/2009 Admission | 11/26/2011 Admission | 3/17/2012 Admission | 7/9/2012 Admission | 8/19/2013 Follow up |
|---------------------|---------------------|---------------------|-------------------|---------------------|
| Basal short axis view |                     |                     |                   |                     |
| Mid short axis view  |                     |                     |                   |                     |
| Apical 2-chamber view |                   |                     |                   |                     |
| Peak Troponin, (µg/L) | 19.2               | 58.7               | 41.9              | 143                 | -                   |
| CMRLVEF, (%)         | 48                  | 51                  | 48                | 48                  | 46                  |
| CMR LVEDVi, (mL/m²) | 76                  | 91                  | 85                | 81                  | 84                  |
| CMR LVESVi, (mL/m²) | 39                  | 45                  | 44                | 42                  | 45                  |
| CMR LVSV, (mL)       | 62                  | 71                  | 69                | 67                  | 75                  |

Figure 3. The cardiac magnetic resonance (CMR) findings during each episode. Column 2 shows short- and long-axis images of CMR delayed enhancement during the first admission. Column 3 shows short- and long-axis images of CMR delayed enhancement during the second admission in November 2011 where the patient presented with chest pain and elevated troponin I (peak was 58.7 µg/L). Column 4 shows short- and long-axis images of CMR delayed enhancement during the third admission. He presented again with chest pain and elevated cardiac markers (troponin I peaked at 41.9 µg/L). Column 5 shows short- and long-axis images of CMR delayed enhancement during the fourth admission. Column 6 shows short- and long-axis images of CMR delayed enhancement of the patient during subsequent visit in cardiology outpatient clinic. LVEF = left ventricular ejection fraction; LVEDVi = indexed left ventricular end diastolic volume; LVESVi = indexed left ventricular end systolic volume; LVSV = left ventricular stroke volume.
count, renal function test, liver profile, and toxicology screening (Table 1). Echocardiography showed no regional wall motion abnormalities with an ejection fraction of 50–55%. He was initially treated as acute coronary syndrome using dual antiplatelet treatment, β-blocker, and statin. While in the hospital, he remained stable with improvement of his chest pain. A subsequent cardiac catheterization showed normal coronary artery arteries (Fig. 2). CMR later, confirmed the diagnosis of myocarditis with mild left ventricular dysfunction (Fig. 3, Column 2).

Subsequently, in November 2011, March 2012, and July 2012, the patient presented with similar symptoms. His cardiac markers were also elevated. Viral screening and markers of connective tissue diseases were all negative (Table 1). The patient was treated with aspirin, colchicine, intravenous immunoglobulin, and prednisolone. On each admission, CMR showed appearance of new areas of myocardial delayed enhancement (MDE) in addition to the old unresolved ones (Fig. 3) consistent with recurrent myocarditis. Nevertheless, he remained stable with complete resolution of his symptoms and was discharged in a stable condition. The most recent assessment was in December 2015. The patient was active and has no cardiac symptoms, but, Holter monitor showed multiple episodes of asymptomatic nonsustained ventricular tachycardia (Fig. 4). He was treated with angiotensin converting enzyme inhibitors and β-blocker and he maintained stable with no further attacks of myocarditis.

Discussion

Recurrent myocarditis has been rarely reported in adults. Our case may be the first to show serial and progressive increase of MDE documented on multiple occasions on CMR late gadolinium enhancement. CMR greatly helped initially in confirming the diagnosis and in the subsequent presentations with recurrence of symptoms to guide treatment. A recent European Society of Cardiology position statement considers performing CMR as reasonable in stable patient prior to EMB [1]. In patients with recurrent myocarditis, guidelines are lacking with regards to the role of CMR. Our case demonstrates that CMR could show multiple additional myocardial injuries with each episode of myocarditis. The challenge was to determine the etiology and EMB may be the test of choice. Current American College of Cardiology/ American Heart Association guidelines for the treatment of heart failure [2] describe EMB as a class-IIb recommendation. Biopsy is generally reserved for patients with rapidly progressive cardiomyopathy refractory to conventional therapeutic management or an unexplained cardiomyopathy that is associated with progressive
conduction system disease or life-threatening ventricular arrhythmias. Sampling error remains a significant limitation to the diagnostic accuracy of the EMB [3]. In our case, the presence of MDE in ventricular septum near right ventricular aspect made it more likely to be diagnostic. Endomyocardial biopsy was performed in another hospital and reported as nonspecific inflammatory lymphocytic reaction. Unfortunately, the specimen is not available to us for more detailed analysis. The incidence of ventricular arrhythmias in patients with recovered myocarditis is unknown. Recent prospective postmortem data have implicated myocarditis in sudden cardiac death of young adults at rates of 8.6–12% [4]. There is no direct link between sudden death and ventricular arrhythmias, but it underscores the importance of screening and monitoring these patients for the presence and proper treatment of ventricular arrhythmias.

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