Case of Myocardial Relapse of a T-Cell Lymphoma After Hematopoietic Stem Cell Transplantation Demonstrated by Cardiovascular Magnetic Resonance and Endomyocardial Biopsy

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A 23-year-old patient with recent history of hematopoietic stem cell transplantation as a result of a relapse of a cortical T-cell lymphoma presented with dyspnea and recurrent nightly tachycardia. The initial ECG showed ST-segment depression in leads I and II and over the anterior wall, as well as tachycardia (heart rate, 110 bpm; Figure 1). The troponin test was positive (1.780 μg/L; normal, <0.014 μg/L). Transthoracic echocardiography showed uniform thickening of the left ventricular wall with global hypokinesia and a moderate pericardial effusion without cardiac tamponade (Movie I in the online-only Data Supplement).

For further diagnostic workup, cardiac magnetic resonance imaging was performed with a clinical 1.5-T scanner (Achieva, Philips Medical Systems, Best, The Netherlands). Functional assessment showed a globally reduced left ventricular ejection fraction of 30% (Movie II in the online-only Data Supplement). Moreover, cine imaging confirmed the moderate pericardial effusion and the uniform thickening of the left ventricular wall seen on echocardiography and revealed bilateral pleural effusions (Figure 2). T2-weighted black-blood imaging performed for the detection of myocardial edema showed broad, inhomogeneous hyperintensity of the entire myocardium (Figure 3), correlating with highly abnormal T2 relaxation times up to 140 milliseconds in the T2-mapping sequence (Figure 4). To detect myocardial hyperemia, 1 of the features for the diagnosis of myocarditis according to the Lake Louise Consensus Criteria,1 myocardial gadolinium early enhancement was assessed during the first 2 minutes after 0.1 mmol/kg gadolinium-DOTA (Dotarem, Guerbet, Villepinte, France) contrast administration, as previously described.2 T1-weighted images showed an area of circular contrast enhancement that finally could be attributed to the patient's history, cardiac graft-versus-host disease,3 the effect of potential cardiotoxic drugs,4 and myocardial relapse of the underlying disease were also conceivable as potential diagnoses.

To rule out these differential diagnoses, endomyocardial biopsy was performed. In polymerase chain reaction, no virus genome could be identified. Histology (Figure 7) showed a dense lymphocytic infiltrate surrounding myocardial cells with considerably enlarged nuclei. In the immunohistochemical staining, the lymphocytes were completely positive for CD3 as a marker for T cells. After further molecular analysis, the lymphocytic T-cell receptors showed an oligoclonal rearrangement.

In the meantime, the patient experienced progressive, severe heart failure with several resuscitation events, and extracorporeal membrane oxygenation implantation. Finally, the patient died.

The case presented here shows an unusual type of myocardial contrast enhancement that finally could be attributed to a relapse of a T-cell lymphoma after hematopoietic stem cell transplantation. As outlined, several differential diagnoses should be considered in patients with history of hematopoietic stem cell transplantation presenting with new onset of cardiovascular symptoms, and the diagnosis has to be confirmed by endomyocardial biopsy.

Disclosures
None.

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References


Figure 1. The patient’s ECG at initial presentation: sinus rhythm; heart rate, 73 bpm; normal QRS axis; PQ, 160 milliseconds; broadened QRS complexes (120 milliseconds) with right bundle-branch (rsR’’) morphology in V1 and V2; and ST-segment depression with pronounced T-wave inversion in the left precordial leads. Prolonged corrected QT interval (Bazett)=510 milliseconds.
Figure 2. Four-chamber cine magnetic resonance imaging demonstrates a moderate pericardial effusion, bilateral pleural effusions, and uniform thickening of the left ventricular wall, which could be attributed to diffuse myocardial edema. In the apical section of the interventricular septum, an oval lesion with reduced signal intensity is shown (arrow).

Figure 3. T2-weighted black-blood imaging shows a diffuse hyperintensity of the entire myocardium, as well as an overall thickening of the left ventricular wall, consistent with myocardial edema.

Figure 4. The T2 mapping sequence demonstrates highly abnormal T2 relaxation times up to 140 milliseconds with predominantly yellow color-coded myocardium (green, relaxation times <60 milliseconds; yellow, relaxation times >60 milliseconds; orange/red, relaxation times >90 milliseconds) confirming myocardial edema.

Figure 5. Assessment of myocardial gadolinium early enhancement shows an area of circular contrast enhancement with central hypointensity in the apical lateral wall (arrow). In addition, diffuse enhancement of the entire myocardium during the early washout period is observed. The black bands covering the atria represent saturation bands with the aim of reducing intracavitary blood pool signal. CM indicates contrast medium.
Figure 6. A through C, Delayed enhancement imaging demonstrates multiple focal lesions with circular contrast enhancement in the midventricular inferoseptal and the apical anteroseptal and anterolateral left ventricular wall segments. D, In addition, multiple small areas of focal contrast enhancement can be observed, particularly in the septum and in the anterior/anterolateral left ventricular wall.

Figure 7. A and B, Histology showed a dense lymphocytic infiltrate surrounding myocardial cells with partially considerable enlarged cell nuclei (A, ×100 enlargement; B, ×200 enlargement). C, In the immunohistochemical staining, the lymphocytes were completely positive for CD3 as a marker for T cells (×100 enlargement). D, The lymphocytes were completely negative for CD20 as a marker for B cells (×100 enlargement).