MacCallum Plaque Causes Acute Myocardial Infarction in a Young Man

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A 27-year-old man died suddenly of acute extensive myocardial infarction.

The patient was healthy with no known systemic diseases. In October 2013, he visited our cardiovascular outpatient department because of progressive shortness of breath. Physical examination showed grade III systolic murmur at the apex. A transesophageal echocardiogram revealed diffuse thickening of the mitral valve with severe mitral regurgitation and nodular lesions at the posterior wall of the left atrium and posterior mitral leaflet (Movie I in the online-only Data Supplement). Surgical intervention with mechanical mitral valve replacement and resection of intra-atrial tumors was uneventful. The pathology of the intracardiac tumor showed papillary fibroelastoma, and the mitral valve showed fibrosis and myxomatous degeneration. After surgery, the patient was free of symptoms. Unfortunately, chest tightness was noted 3 months later. An ECG showed diffuse T-wave inversion over the anterior leads. The patient experienced a sudden collapse with limb twitching during thallium scan examination. His pulse returned several minutes later after cardiopulmonary resuscitation, and an ECG showed ST-segment elevation over the inferior leads. Emergent coronary catheterization revealed severe stenosis over both ostia of the left main and right coronary arteries (Figure 1). During catheterization, ventricular tachycardia/fibrillation occurred, and defibrillation/cardiopulmonary resuscitation was immediately applied again. Coronary stents were placed over the ostia of left main and right coronary arteries under the support of extracorporeal membrane oxygenation and intraventricular balloon pump. After the procedure, an extremely elevated troponin I level was noted (troponin I >1000 ng/mL). An echocardiogram revealed general akinesia of both the left and right ventricles, and his left ventricular ejection fraction was ≈10%. The patient died.

A postmortem autopsy was done. Gross photographs of the left ventricle and aortic root are shown in Figures 2 and 3. The left ventricle showed severe myocardial infarction with reperfusion injury, and the residual tendinous cords showed thickening and fusion. The aortic root revealed extraordinarily thickened map-like lesions above the aortic valve leaflets. Microscopic findings of left atrial endocardium and thickened aortic intima showed diffuse fibrotic change (Figure 4A and 4B). Mixed inflammatory cell infiltrate with focal areas of interstitial edema and neovascularization in the area adjacent to the fibrosis was also noted (Figure 5). All of these findings suggested that the thickened aortic intima was MacCallum plaque, one of the characteristic features of rheumatic heart disease. Although we cannot evaluate the mitral valve after surgery, remarkable subendocardial fibrosis in the left atrium and residual tendinous cords and the preoperative finding of the echocardiogram support the final diagnosis of rheumatic heart disease with extensive MacCallum plaque formation in aortic root, which resulted in coronary ostium narrowing and extensive myocardial infarction.

MacCallum plaque is thought to be caused by regurgitant jets of blood flow secondary to valve deformity and usually on the endocardium of the left atrium in rheumatic heart disease. To the best of our knowledge, our case is the first case reporting that extensive MacCallum plaque formation over aortic root results in acute myocardial infarction. Here, we report this case with the rare but dreadful complication of rheumatic heart disease.

Disclosures

None.

References

Figure 1. Both ostia of the left main and right coronary arteries showed nearly total occlusion by coronary angiography.

Figure 2. Gross photograph of the left ventricle showed transmural hyperemia over the interventricular septum and subendocardial area. The mitral valve was replaced by a mechanical valve, and thickening and fusion of residual chordae tendineae (arrow) are shown.

Figure 3. Thickening and fibrosis of the cusps of the aortic valve and intima of the aortic root. The map-like MacCallum plaque (arrow) extended to the coronary artery ostia, which were dilated by coronary stents.

Figure 4. Masson trichrome stain of the thickened area in the left atrium (A) and aortic intima (B), which revealed diffuse fibrosis (A, ×20; B, ×40).

Figure 5. The area adjacent to the fibrosis showed mixed inflammatory cell infiltrate, with focal areas of interstitial edema and neovascularization (×200).
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