CLINICAL VIGNETTE

Severe coronary artery disease secondary to graft-versus-host disease after bone marrow transplantation in a 24-year-old woman

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A 24-year-old woman with a history of aplastic anemia treated with an autologous bone marrow transplantation (BMT) at the age of 4 and acute myeloid leukemia treated with allogeneic BMT at the age of 9 with a graft derived from an unrelated donor (8/10 human leukocyte antigen-matched and ABO-mismatched). The preparative regimen included 14.3-Gy total-body irradiation and standard chemotherapy. On day 1 after BMT, the patient developed graft-versus-host disease (GVHD) and responded to corticosteroids. At the age of 17, she underwent bilateral total hip arthroplasty due to aseptic necrosis of both femoral heads following high-dose steroid treatment.

The patient was admitted to our Department of Cardiology for urgent evaluation due to suspicion of myocarditis. On presentation, she complained of fatigue and slight limitation in physical activity manifested by moderate exertion. Her medical treatment included methylprednisolone for GVHD (2 mg per day and 4 mg per day every 3 days), escitalopram (7.5 mg), and hormone replacement therapy with estrogen and progesterone. Electrocardiography revealed sinus rhythm of 86 bpm, pathologic Q waves in leads II, III, and aVF, as well as QS complexes in V_1–V_3. Moreover, akinesis of the apex and apical segments of the left ventricle with ejection fraction of 30% was found. Biochemical findings showed hypercholesterolemia (total cholesterol, 8.77 mmol/l; low-density lipoprotein cholesterol, 5.30 mmol/l; triglycerides, 2.64 mmol/l), while the troponin level was within reference range. Cardiac magnetic resonance revealed subendocardial scar in the apex and apical segments (>75% of wall thickness) of the left ventricle and in the medial segments of the interventricular septum and inferior wall (25%–75% of wall thickness) typical of ischemia with severely reduced ejection fraction (29%); no signs of myocarditis were found (FIGURE 1A). Furthermore, coronary angiography was performed and it showed advanced coronary artery disease (CAD): chronic total occlusion of the left anterior descending artery (LAD) with poor collateral flow, chronic total occlusion of the right coronary artery with good collateral flow, and critical stenosis of the left circumflex artery (LCx) (FIGURE 1B). Due to the lack of a distal LAD target for bypass grafting, the patient underwent percutaneous coronary intervention (PCI) in the LCx with successful drug-eluting stent implantation (FIGURE 1C). Also, PCI in the totally occluded right coronary artery was planned as the next stage of revascularization. Optical coherence tomography of the treated LCx revealed multiple atherosclerotic lesions (FIGURE 1D–1F). Significant coronary artery stenosis in young women has been reported previously.[1] However, CAD rarely occurs as a late complication of GVHD after BMT. Thus, the pathogenesis and morphology of coronary lesions are barely known. Nevertheless, there are many potential risk factors after BMT, which are related to premature CAD, such as total-body irradiation, chemotherapy, corticosteroid treatment, local inflammation in GVHD. In addition, an elevated low-density lipoprotein cholesterol concentration and premature CAD indicate that the familial hypercholesterolemia

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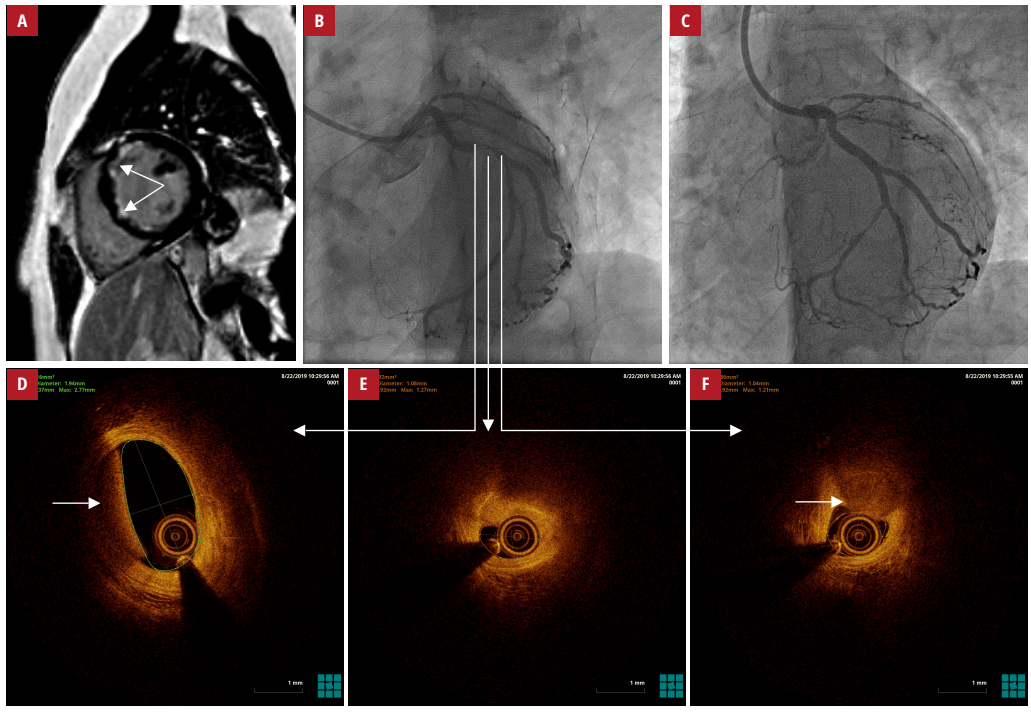

FIGURE 1 **A** – cardiac magnetic resonance (short axis view) with late gadolinium enhancement: subendocardial scar in the medial segments of the intraventricular septum and anterior wall (arrows); **B** – angiography (caudal left anterior oblique view) before percutaneous coronary intervention: critical stenosis of the proximal segment in the left circumflex artery; **C** – angiography (caudal left anterior oblique view) after percutaneous coronary intervention in the left circumflex artery; **D** – optical coherence tomography (OCT): a thrombus attached to the arterial wall (arrow); **E** – OCT: fibroatheroma with an organized fibrotic thrombus; **F** – OCT: a thin-capped lipid-rich plaque (arrow)

cannot be definitely excluded. However, the non-typical features of atherosclerotic plaque, such as increased cellular proliferation of the intima with advanced fibrosis detected on optical coherence tomography, suggest that chronic inflammation played a crucial role in the formation of lesions in our patient (FIGURE 1D–1F). To the best of our knowledge, 7 cases of CAD associated with GVHD after BMT were reported in the literature.[2-5] Among them, 2 patients underwent PCI with stent implantation and both survived.[2,3]

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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