Cardiac lymphoma causing coronary compression, pericardial effusion, and atrioventricular block

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A 75-year-old male patient underwent stenting of a mid-right coronary artery (RCA) lesion because of exertional chest pain. The procedure was complicated by acute stent-thrombosis 3 days later managed by thrombectomy and repeat stenting with favourable results. However, 3 weeks later, the patient was re-admitted with symptoms of progressive dyspnea. An echocardiographic examination showed an additional structure attached to the right ventricular wall and inhomogeneous thickening of the interatrial septum, together with a significant amount of pericardial fluid causing echocardiographic signs of tamponade (Panel A). These findings were confirmed by subsequent cardiac magnetic resonance (MR) (Panel B) and computed tomography (CT) (Panels C and D), revealing a multifocal mass extending from the right atrioventricular groove towards the adjacent wall of the right ventricle and atrium with secondary encasement of the RCA and its stent. A second large, contiguous mass reached from the posterior ascending aorta, the roof, and posterior part of the interatrial septum towards the posterior wall of the left atrium, invading its lumen. The mass appeared iso-intense on T1-w TSE images and inhomogeneously iso- to hyper-intense on T2-w TSE images. During hospitalization, transient episodes of complete atrioventricular block were observed necessitating insertion of a temporary pacing lead. Owing to haemodynamic deterioration, a pericardial window was created while at the same time obtaining biopsies from the right ventricular mass which proved highly friable, easily bleeding during manipulation. Anatomo-pathology led to diagnosis of a large B-cell non-Hodgkin lymphoma (Panels E – G) for which chemotherapy was initiated consisting of vincristine/cyclofosfamid/medrol and rituximab. Before discharge, the patient had a dual-chamber permanent pacemaker fitted. After two cycles of chemotherapy, the patient reported a marked symptomatic ameloriation and repeat CT demonstrated a significant reduction in tumor size (Panel H) and no pericardial effusion. Magnetic resonance follow-up could unfortunately not be performed since a non-MR compatible pacemaker was implanted.

Panel A. Transoesophageal echocardiographic demonstration of an added structure attached to the right ventricular wall (arrow), inhomogeneous thickening of the interatrial septum (arrowhead), and pericardial fluid (asterisk).

Panel B. Cardiac magnetic resonance confirming tumour (arrows) extending from the right atrioventricular groove towards the right ventricular wall and interatrial septum. Asterisk indicates pericardial fluid.

Panels C and D. Cardiac computed tomography demonstrating the multifocal mass (arrowheads) encircling the heart from the right ventricular apex to the posterior wall of the left atrium, with invasion of the cardiac lumen and secondary encasement of the right coronary artery and stent (arrow).

Panel E. Low-power magnification (×40) of haematoxylin and eosin stain of myocardial biopsy, diffusely infiltrated by a lymphocytic process.

Panel F. At higher power magnification (×400), the infiltrate consists of large atypical cells with vesicular nuclei and often a nucleolus. Mitotic figures (arrow) and apoptotic bodies (arrowhead) are frequent.

Panel G. The lymphoid elements express CD20 at their membrane surface, pointing out they are of B-cell origin.

Panel H. Follow-up computed tomography after two cycles of chemotherapy, showing a significant reduction in tumour size. Arrow indicates permanent pacemaker lead.