Intramural hematoma and thoracic aortic aneurysm with syphilis

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S yphilitic aortitis is usually associated with aortic aneurysm, aortic regurgitation, and coronary ostial stenosis.¹ However, aortic dissection and intramural hematoma (IMH) due to syphilitic aortitis have rarely been reported. We present a case of ascending aortic IMH and aortic arch aneurysm with syphilis; the patient underwent total arch replacement.

Clinical Summary

A 76-year old woman was admitted because of continuous low-grade pyrexia. The chest computed tomographic scan (CT) revealed ascending aortic IMH (55 mm) and aneurysm (65 mm) of the aortic arch (Figure 1). Results of the treponema pallidum hemagglutination test was positive; however, the result of the serologic test for syphilis was negative, which revealed past syphilitic infection. Total arch replacement with selective antegrade cerebral perfusion and deep hypothermic circulatory arrest in the lower body was performed. The severe adhesion with the pericardial cavity and thickened aortic wall suggested existence of an old inflammatory event. In the ascending aorta, there was IMH without an intimal tear. The culture of aortic wall and hematoma was negative; however, the pathology of the ascending aorta revealed diffuse infiltration of lymphocyte and plasma cells in the adventitia (Figure 2). The postoperative course was uneventful.

Discussion

Cardiovascular syphilis mainly occurs in the third stage of syphilis and is likely to appear in the ascending aorta and aortic arch. Syphilitic aortitis is followed by aortic aneurysm (40%), rupture (14%), aortic valve regurgitation (29%), and coronary ostial stenosis (26%).¹ However, aortic dissection due to syphilitic aortitis has rarely been reported to date, probably because of scar tissue formation in the aortic wall. Histologically, both the intima and adventitia are cicatrized and thickened after inflammation of syphilitic aortitis.² We could find only 2 cases of syphilitic aortic dissection^{3,4} and syphilitic IMH.⁵ IMH is a type of aortic dissection and is regarded as hematoma with neither intimal flap nor direct blood communication between false and true lumen.

In this case, IMH was diagnosed by preoperative CT and operative findings. The pathologic study of the ascending and aortic arch revealed inflammatory cellular infiltration of lymphocyte and plasma cells in adventitia that is characteristics of syphilitic aortitis. We thought the IMH was

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Figure 1. The chest CT revealed ascending aortic IMH (55 mm) (left) and aortic arch aneurysm (65 mm) (right).

Figure 2. The pathology of the ascending aorta revealed inflammatory cellular infiltration of lymph corpuscle and plasma cells in adventitia.

caused by the rupture of vasa vasorum of fragile media after syphilitic aortitis and by cicatrized intima and adventitia.

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Purse-string deformity of the right coronary artery: A pitfall in de Vega tricuspid annuloplasty

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Injury to the right coronary artery (RCA) is a rare complication of tricuspid annuloplasty. Because of its course in the right atrioventricular groove, the RCA is vulnerable to direct mechanical injury.¹ However, we present a case in which a de Vega annuloplasty caused excessive deformation of the RCA and resulted in an acute coronary syndrome.

Clinical Summary

A 64-year-old woman was referred for mitral and tricuspid annuloplasty. She had undergone mechanical aortic valve replacement

because of acute endocarditis 14 years before. Subsequently she had been followed for mitral insufficiency and presented recently with dyspnea. There was severe mitral insufficiency caused by systolic restrictive motion of the posterior mitral leaflet (Carpentier 3b) with a dilated left ventricle and severely impaired systolic function. A concomitant tricuspid insufficiency was found with an annulus diameter of 42 mm (25 mm/m²), a dilated right ventricle, and good systolic function. There was no coronary artery disease.

Restrictive mitral annuloplasty was performed with placement of a 30-mm ring (Edwards Lifesciences, Irvine, Calif). A de Vega tricuspid annuloplasty was performed. A control transesophageal echocardiogram showed mild residual mitral insufficiency and no tricuspid insufficiency or stenosis (annulus diameter 22 mm, 13 mm/m²). The course of the operation was uneventful until closure of the chest when ST elevations appeared in the inferior leads. A transesophageal echocardiogram showed a worsening of the wall motion abnormalities of the posterior and inferior left ventricular wall. Reinspection showed no exterior compression of the RCA, and the patient was subsequently presented for coronary angiography.

In contrast with the preoperative angiography, there was marked kinking of the proximal RCA followed by an occlusion of the third segment (Figure 1). This site showed sharp angulation that was not visible on the preoperative angiogram and was sug-

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