A localized intimomedial defect resulted in aortic regurgitation

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Acute aortic regurgitation is usually caused by aortic dissection and infective endocarditis. We describe an unusual cause of acute aortic regurgitation caused by the dislocation of the commissure, along with the localized defect of the inner side of the aorta. Histologic study disclosed that the intimal tear extended deep into all layers of the media (an “intimomedial tear”) and caused acute aortic regurgitation through dislocation of the commissure.

Clinical Summary

A 41-year-old man with a diagnosis of annuloaortic ectasia and aortic insufficiency was referred to our hospital. He had acute onset of anterior neck and chest pain and was transferred to a local hospital before being transferred to ours. The results of coronary angiography were normal. An aortography showed a moderately dilated ascending aorta and grade 3/4 aortic regurgitation (Figure 1).

He was in New York Heart Association functional class II with grade III/VI diastolic murmur. Transesophageal echocardiography revealed a moderately dilated left ventricle with normal systolic function, no thickening of the aortic cusps, and prolapse of the right coronary cusp with severe aortic regurgitation. The aorta was dilated, measuring 25 mm at the aortic annulus, 49 mm at the sinus of Valsalva, and 40 mm at the sinotubular junction.

Surgical intervention was performed 2 months after the onset. The aortic root was spherically dilated, and the left side of the aorta was densely adherent to the pulmonary trunk as in chronic aortic dissection. After cardioplegic arrest, the aorta was open. No aortic dissection and detachment of the aortic commissure were present. The half-circumscribed sharp-edged defect at the inner side of the aortic wall extended between the right and left coronary ostium near the sinotubular junction, being 3 cm in length. The commissure between the right and left coronary cusps was dislocated from the sinotubular junction over a sector of approximately 1 cm (Figure 1). The tissue of the commissure itself was intact, and the right coronary cusp was redundant. A valve-sparing procedure (reimplantation technique) was performed with a self-made 26-mm Dracon polyester fabric Valsalva graft.

Figure 1. Inner aspect of the aorta with a lesion characterized by a sharply delineated shallow defect (asterisk). The aortic annulus was deformed by dislocation of the commissure between the right and left coronary cusps (arrow). RCA, Right coronary artery ostium.

Histopathology reports disclosed the presence of cystic medial necrosis in the ascending aorta. The defect at the inner side of the aorta extends throughout all layers of the media and reaches the adventitia. The outer part of the defect still contains elastin fibers of the original media. The luminal side of the defect is covered by fibrocellular proliferation (Figure 2). These findings suggest the sudden interruption of the preexistent structure caused by an abrupt laceration, as in aortic dissection.

The postoperative course was uneventful, and echocardiography revealed no evidence of aortic regurgitation. The patient is in good clinical condition 3 months after the operation.

Discussion

It is well known that Stanford type A aortic dissection occasionally induces aortic regurgitation. The mechanism of aortic regurgitation has been explained by showing that the aortic annulus is transformed by the markedly enlarged false lumen and the separation of the cusp commissure. In the present case aortic regurgitation and aortic cusp prolapse occurred as a consequence of dislocation of the commissure of the right and left coronary cusps to intimomedial tear.

In classical aortic dissection delamination of media follows primary intimal tear and extends up to the inner two thirds of media. It never reaches the adventitia.¹ This variant type of dissection has been called incomplete dissection,² limited dissection,³ class 3 dissection,⁴ or partial intimal tear “stretch mark.”⁵ Schlattmann and Becker² reported the localized defect in the inner side of

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Figure 2. Histologic section (elastica van Gieson stain) through the area that shows abrupt laceration (arrow) of all the layers of media. The outer part of the defect contains only the adventitia. The luminal side of the defect is covered by fibrocellular proliferation (asterisk).

the aorta in 9 patients with Marfan syndrome, coactation of the aorta, or chronic aortic dissection by autopsy. Histologic section revealed almost the same findings as in the present case. The mechanism of the occurrence of intimomedial tear could be associated with fragility of the aortic wall, such as cystic medial necrosis and Marfan syndrome. Svensson and colleagues^{3,4} also

reported this type of tear in 6.6% of patients with Marfan syndrome and suspected that it might propagate to become the classical type of aortic dissection.

In this case intimomedial tear occurred in the aorta facing the pulmonary trunk and did not result in aortic rupture or pseudoaneurysm formation but in aortic regurgitation.

Intimomedial tear of the aortic wall around the aortic commissure is a rare pathophysiologic cause of acute aortic regurgitation. The occurrence of intimomedial tear might be associated with fragility of the aortic wall, such as cystic medial necrosis. If acute aortic regurgitation with aortic cusp prolapse and intact ascending aortic wall is present in the patients with connective tissue disease, intimomedial tear of the aortic commissure should be taken into consideration.

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Novel left ventriculoplasty for nonischemic dilated cardiomyopathy with functional mitral regurgitation

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Functional mitral regurgitation (FMR) accompanying nonischemic dilated cardiomyopathy (DCM) has a poor prognosis, and its surgical management is still controversial. We report a novel surgical approach that consists of the repair of papillary muscle geometry and annuloplasty with

a semirigid ring and partial resection of the dilated left ventricle (LV). Mid-term postoperative results of our surgical approach included no recurrence of FMR, effective reduction of the LV volume and shape, and improvement of pulmonary artery pressure.

Clinical Summary

A 42-year-old man was admitted to our center for an easy fatigability on effort. On admission, echocardiogram demonstrated a dilated LV associated with severe mitral valve regurgitation. End-systolic volume index (ESVI) and end-diastolic volume index (EDVI) were 139.5 and 160.5 mL/m², respectively. Although severe regurgitation was present either at the mitral (3+) or tricuspid valve (3+), ejection fraction (EF) was decreased to 13 percentages, and pulmonary artery systolic pressure (PAP) was increased to 39 mm Hg. Preoperative gated single photon emission computed tomography quantification confirmed the diagnosis of DCM and showed the spherical shape of the dilated LV. Coronary angiography showed normal coronary findings.

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