Case Report

Aortic Root Replacement in Case of Isolated Aortitis and Previous Coronary Artery Bypass

Efthymia Rouska[1], Nikolaos Charokopos[2], Efstathios D. Pagourelias[3], Panagiotis Artemiou[2], Antonios Ziakas[3], Nikolaos Papadopoulos[4]

[1]Second Cardiology Department, Evaggelismos General Hospital, Athens, [2]Cardiac Surgery Department, Euroclinic of Athens, [3]First Cardiology Department, AHEPA University Hospital, Medical School, Aristotle University of Thessaloniki, [4]Cardiac Surgery Department, Geniki Kliniki General Hospital, Thessaloniki, Greece

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The surgical management of aortic regurgitation in a patient with aortitis is potentially of high risk, especially if it is a reoperation. We present the case of a 59-year-old man for whom coronary artery bypass surgery was not feasible due to structural abnormalities of the aorta and hybrid management was applied. The histopathological examination showed aortitis. One year later, the same patient developed severe aortic regurgitation. In order to minimize the surgical risk of the reoperation we considered all the surgical options. The modified Bentall procedure still seems to be the gold standard in these cases.

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Address: Efthymia Rouska

30, Nikitara St. 152 35 Vrilissia, Attiki, Greece
e-mail: rouskamed@hotmail.com

Aortitis, meaning inflammation of the aortic wall, may be caused by several factors, infectious or non-infectious.[1] Isolated non-specific aortitis usually runs an asymptomatic course and is usually discovered incidentally during a pathological examination of aortic wall specimens after open-chest surgery.[2] In such patients, reoperation of the aorta, including aortic valve replacement, is extremely challenging.

Case presentation

A 59-year-old man was admitted to our department for re-evaluation of previously diagnosed aortic regurgitation. His main symptoms were progressively deteriorating dyspnea along with frequent episodes of angina pectoris. On his medical history there was hypertension, dyslipidemia and severe coronary artery disease (CAD). In particular, concerning his CAD history, the patient underwent coronary angiography for the first time in 2003, which revealed left anterior descending (LAD) disease and an ejection fraction of about 50%. The patient refused any intervention at that time. In 2006, due to worsening angina, he had a second angiography confirming 3-vessel disease. The patient finally consented to undergo cardiovascular surgery. During the operation, the ascending aorta was found to be thick, fibrous and extremely atherosclerotic. The patient underwent off-pump coronary artery bypass (OPCAB) with a left internal mammary artery (LIMA) graft to the LAD. During the same hospitalization, he was later referred to the catheterization laboratory where he underwent percutaneous transluminal coronary angioplasty and stenting to the circumflex (Cx) artery and the right coronary artery (RCA).

The histological examination of the aortic wall specimens, collected during the operation, was consistent with aortitis. Pathological evaluation revealed focal necrosis and scarring in combination with transmural and peri-aortic infiltrations with lymphocytes, plasma cells, granulocytes, histiocytes and Langham's giant

cells. A full immunological and biochemical laboratory examination showed elevation of both C-reactive protein and erythrocyte sedimentation rate without, however, any increase in other specific immunological markers. By means of immunohistochemistry, syphilitic, bacterial and fungal aortitis were ruled out. Biopsy of the temporal artery was also negative.

Magnetic resonance angiography (Figure 1) showed a thick, dilated ascending aorta of 4.6 cm diameter and a thick, dilated abdominal aorta with significant luminal stenoses on the lower third above the bifurcation. There were also 15-20% luminal stenoses of both common iliac arteries. Spiral computed tomography of the thorax revealed multiple atheromatous lesions across the aorta.

One year after the operation, the patient again presented with recurrent angina. The treadmill stress test was positive and an apical ischemic defect was shown by Th^{201} stress test. Coronary angiography showed an occluded LIMA graft, patent Cx and RCA arteries, a dilated ascending aorta and severe aortic regurgitation (AR) (3+/4+). Transthoracic and transesophageal echocardiography (Figure 2) showed a dilated left ventricle with mild concentric hypertrophy, apical akinesia and ejection fraction 40-45%. A thick and dilated ascending aorta (4.6 cm) and a dilated descending aorta (4.0 cm) with significant luminal stenoses were demonstrated. Severe AR with Perry index 80%, *vena contracta* 1 cm, and pressure halftime <200 ms with a reverse flow in the descending aorta, was also present. The right ventricular pressure was 45 mmHg.

The patient then again underwent surgery. Preoperatively, he received a 4-week treatment with glucocorticoids. He underwent a modified Bentall procedure, including aortic root replacement with a 23 mm composite graft fabricated with a prosthetic valve, along with direct anastomosis of the right coronary ostium to the composite graft and revascularization of the left coronary artery using a saphenous vein graft (SVG) (Modified Bentall procedure and concomitant coronary artery bypass grafting).

The patient had an uneventful postoperative course and was discharged home on postoperative day 11. Follow-up visits at 1 and 3 months after surgery were unremarkable.

Discussion

The pathological term "aortitis" refers to inflammation of the aortic wall. The classification used for the syndrome is either etiological, based on the underly-

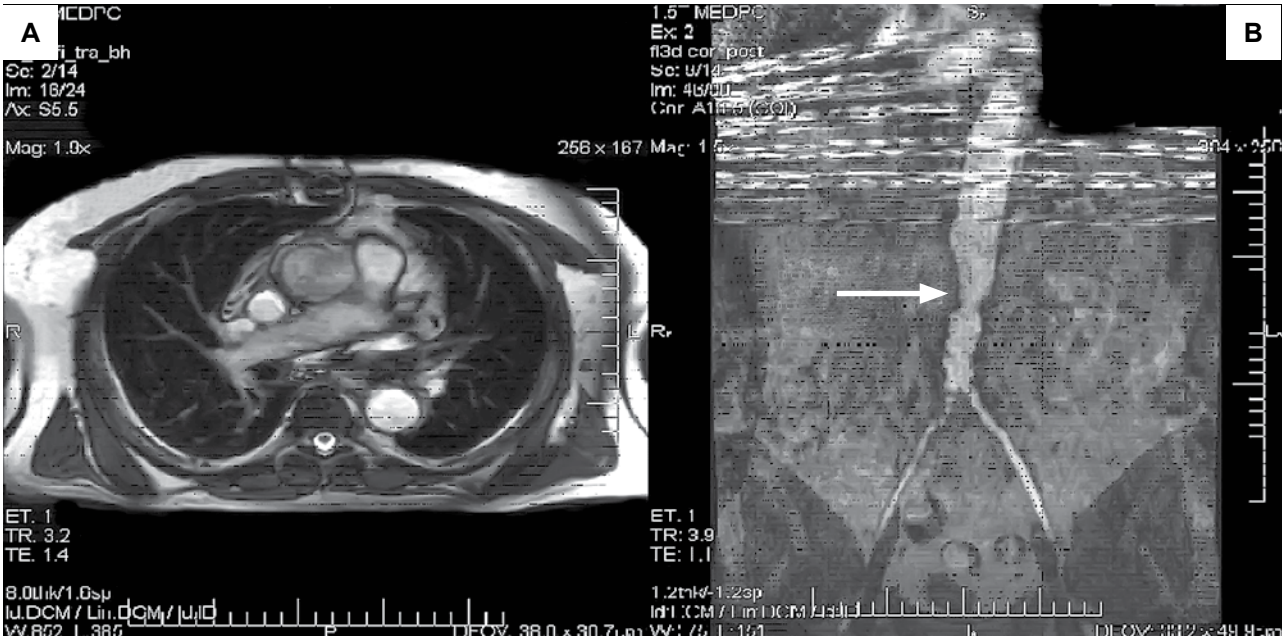

Figure 1. Magnetic resonance angiography enhanced with gadolinium contrast. A) Dilatation of the ascending aorta (4.6 cm) with circumferential thickening of the aortic wall. Increased signal intensities are noted within the wall, representing inflammatory process. B) There is irregular thickening of the aortic wall involving the lower third of the abdominal aorta (arrow) and the common iliac arteries, with severe stenosis of their lumen. Increased signal intensities are noted within the wall, probably representing inflammatory process.

ing cause (infectious and non-infectious [inflammatory] aortitis) or pathological, according to the histological findings (necrotizing and non-necrotizing).[1] The pathological differential diagnosis of aortitis is rather vague, due to the overlapping, non-discrete histological pictures of the various types of the disease.[1]

In a large clinical series from Cleveland clinic, involving 1204 histopathological specimens from patients who underwent surgery of the aorta over 20 years, aortitis was found in only 4.3% of them, with the largest portion, over 70%, being isolated non-specific aortitis.[2] Autoimmune mechanisms and genetic predisposition seem to be mainly involved in the pathogenesis of the syndrome.

The management of patients with aortitis depends mainly on the cause of the disease. The goal is to control the inflammation/infection and treat any concomitant complications. Particularly in inflammatory aortitis of surgical patients, it is essential to start glucocorticoid treatment immediately for the control of the inflammation. Surgical patients who receive preoperative glucocorticoids have less postoperative complications than patients who do not.[1] In cases where the glucocorticoid therapy is ineffective, various immunosuppressive agents can be used.[1,3]

In patients with severe AR and a dilated ascending aorta, as in our case, it is advised to perform aortic root replacement with a composite graft (modified Bentall procedure) in order to avoid the risk of complications after surgery, such as valve detachment after single aortic valve replacement (AVR) or anastomotic pseudoaneurysm as a result of fragility of the aortic wall or annular tissue, and the presence of active inflammation.[4] The use of biological prosthetic valves should be discouraged in cases of aortitis, since the immune response to the bioprosthesis may lead to rapid degeneration and calcification of the valve, resulting in high rates of reoperation.[5,6] Furthermore, transapical and transfemoral AVR are not recommended for the management of such patients, given the risk of prosthetic valve detachment, since biological prosthetic valves are so far the only commercially available valves for the above procedures.[5,6] Additionally, in our patient the severe stenosis of both iliac arteries was a contraindication for any percutaneous transfemoral intervention.

For the revascularization of the LAD an SVG was preferred. The occlusion of the LIMA graft to the LAD is a complication of the inflammatory disease of the aorta and its branches. In order to avoid a similar condition in the future, the use of the right internal mammary artery as a graft was not preferred. In our patient the coronary artery disease was thought to be atherosclerotic in origin, although isolated lesions in coronary arteries could rarely be associated with non-specific aortitis.[7]

In conclusion, our patient was diagnosed as suffering from "non-specific isolated aortitis", since any

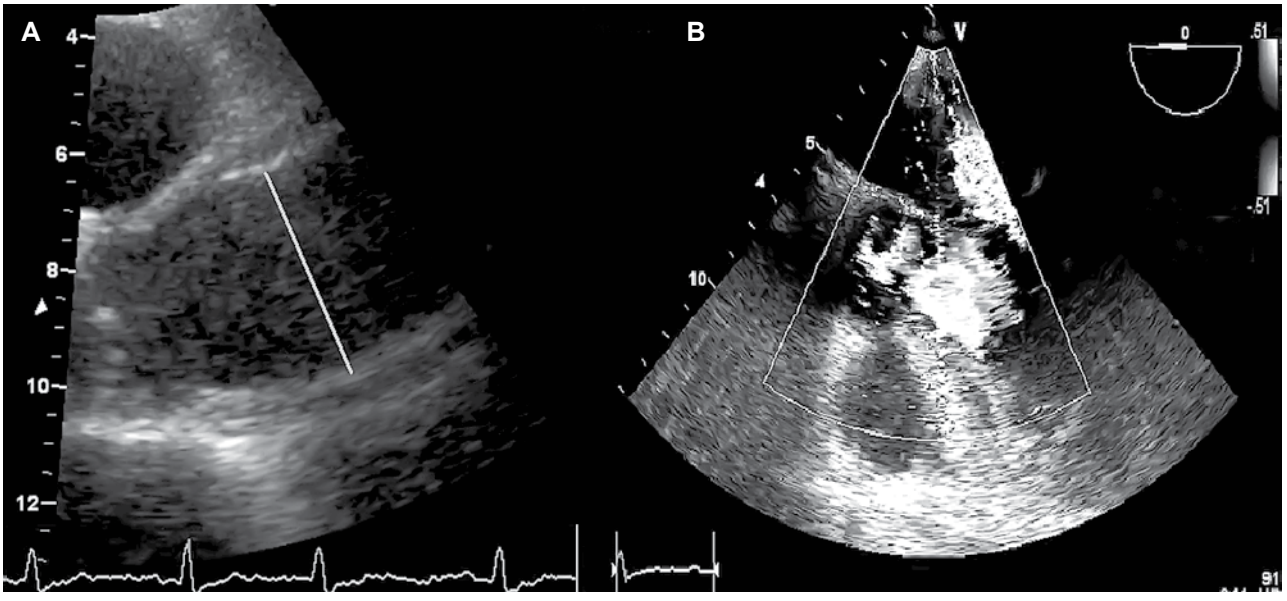

Figure 2. A) Transthoracic echocardiographic image showing the ascending aorta dilated at 4.6 cm (gray line), with circumferential thickening of the aortic wall indicative of aortitis. B) Transesophageal image showing severe aortic regurgitation.

infectious cause was excluded and the criteria set by the American College of Rheumatology for the diagnosis of giant cell arteritis and Takayasu were not met.[1] A modified Bentall procedure and concomitant coronary artery bypass surgery using an SVG to the LAD were the solution of choice for our patient.

References

1. Gornik HL, Creager MA. Aortitis. Circulation. 2008; 117: 3039-3051.
2. Rojo-Leyva F, Ratliff NB, Cosgrove DM 3rd, Hoffman GS. Study of 52 patients with idiopathic aortitis from a cohort of 1,204 surgical cases. Arthritis Rheum. 2000; 43: 901-907.
3. Valsakumar AK, Valappil UC, Jorapur V, Garg N, Nityanand S, Sinha N. Role of immunosuppressive therapy on clinical, immunological, and angiographic outcome in active Takayasu's arteritis. J Rheumatol. 2003; 30: 1793-1798.
4. Matsuura K, Ogino H, Kobayashi J, et al. Surgical treatment of aortic regurgitation due to Takayasu arteritis: long-term morbidity and mortality. Circulation. 2005; 112: 3707-3712.
5. Human P, Zilla P. Characterization of the immune response to valve bioprostheses and its role in primary tissue failure. Ann Thorac Surg. 2001; 71: S385-388.
6. Murashita T, Yoshimoto K, Sugiki H, Yasuda K. Bilateral coronary ostial patch angioplasty with autologous pericardium in Takayasu arteritis: a case requiring replacement of the aortic valve and ascending aorta. Eur J Cardiothorac Surg. 2004; 26: 866-868.
7. Hountis P, Dedeilias P, Vourlakou C, Bolos K. Isolated bilateral coronary artery ostial stenosis in aortitis syndrome. Hellenic J Cardiol. 2010; 51: 472-474.