Figure 1. Transthoracic echocardiogram (apical, 2-chamber view) showing mitral prosthesis (double arrow) and structure (attached on posterior papillary muscle) floating in the left ventricular cavity (arrow).

Figure 2. Transthoracic echocardiogram (apical, 4-chamber view) showing the torn remnant of the spared anterior leaflet still attached to the posterior papillary muscle (arrow).

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Aortic dissection without Marfan's syndrome in ankylosing spondylitis

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Mutations in the gene for fibrillin-1 (*FBNI*), which cause Marfan's syndrome, have been found not only in Marfan's syndrome but also in a range of connective tissue disorders collectively termed "fibrillinopathies," such as isolated ascending aortic aneurysm and dissection,^{1,2} isolated skeletal features, predominant skeletal manifestations without aortic dilatation, isolated ectopia lentis, and other variants. On the other hand, Simkin³ has hypothesized that the defective fibrillin of Marfan microfibrils and the inflammation-targeted fibrillin of ankylosing spondylitis may each lead to comparable structural phenotypes of failure. Two cases of coexistent ankylosing spondylitis and Marfan's syndrome without aortic dissection have been reported in the literature.^{4,5} To our knowledge, however, there have been no cases of aortic dissection in ankylosing spondylitis. Here we describe the first case of Stanford type A (DeBakey type I) acute aortic dissection without Marfan's syndrome in human leukocyte antigen (HLA)-B27-negative ankylos-

Figure 1. Computed tomography scanning disclosed Stanford type A (DeBakey type I) aortic dissection.

ing spondylitis, and the relationship of these 2 disorders is discussed.

Clinical Summary

A 65-year-old man with severe chest and back pains was referred to our department. He presented with no traits of Marfan's syndrome in physical examination. Computed tomography scanning disclosed Stanford type A (DeBakey type I) aortic dissection (Figure 1). Urgent replacement of the ascending aorta and the total aortic arch was successfully performed with the use of selective cerebral perfusion. The pathological examination of the aortic wall revealed neither aortitis nor cystic medial necrosis. Postoperatively, abdominal x-ray photography disclosed the characteristic deformation of the lumbar vertebrae, so-called "bamboo spine" (Figure 2). The patient was HLA-B27-negative but met the European Spondylarthropathy Study Group preliminary criteria for the classification of spondylarthropathy proposed by Dougados and associates⁶: inflammatory spinal pain, alternate buttock pain, and sacroiliitis. His postoperative course was uneventful, and he is doing well now 2 years and 4 months after the operation.

Discussion

According to Francke and associates,¹ a G-to-A transition at nucleotide 3379, predicting a Gly1127Ser substitution, has been revealed by single-strand conformation analysis of the entire fibrillin-1 (*FBNI*) cDNA of 10 kindred individuals in whom ascending aortic disease, ranging from mild aortic root enlargement to aneurysm and/or dissection, has been identified, and none of whom had classical Marfan's syndrome. Pulse-chase studies revealed normal fibrillin synthesis but reduced fibrillin deposition into the extracellular matrix in cultured fibroblasts from a Gly1127Ser carrier. Milewicz and collaborators² have also identified a missense mutation, D1155N in exon 27, in a 44-year-old man who did not have dolichostenomelia, arachnodactyly, myopia, or lens dislocation and who presented with 6- to 7-cm dilatation of his aortic root with an acute dissection of his ascending thoracic aorta. The mutation decreased the amount of fibrillin-1 deposited into the pericellular

Figure 2. Abdominal x-ray photography disclosed the characteristic deformation of the lumbar vertebrae, so-called "bamboo spine."

matrix of dermal fibroblasts from the affected individual. Results of these studies have supported the hypothesis that *FBNI* mutations cause thoracic aortic aneurysm or dissection in patients who do not have Marfan's syndrome. On the other hand, Simkin³ reported that most sites of inflammation in patients with ankylosing spondylitis involve fibrocartilage, and that fibrillin-1, a major component of the microfibrils in fibrocartilage, may be the target of this response in the aorta and the eye as well as in bones and joints. Thus, it has been hypothesized that spondylitic inflammation may include release and activation of latent transforming growth factor- β from binding sites on fibrillin-1.

Fietta and Manganelli⁴ reported a 46-year-old man with coexistent ankylosing spondylitis and Marfan's syndrome without aortic dissection. The patient had the typical traits of Marfan's syndrome, along with the clinical and radiological findings of ankylosing spondylitis in which HLA typing was positive for the B27 antigen. Because of the rarity of such coexistence, at first they thought that it was accidental. The hypothesis suggested by Simkin,³ however, led them to reconsider this condition: both a genetically determined and an inflammation-derived fibrillin-1 defect might coexist in their patient.⁷

Although there have been 2 case reports of coexistent ankylosing spondylitis and Marfan's syndrome without aortic dissection in the literature,^{4,5} no cases of aortic dissection in ankylosing spondylitis have been found. In the present case, there is no evidence

that ankylosing spondylitis and aortic dissection without Marfan's syndrome are not coincidentally but causally related, because neither *FBNI* nor fibrillin-1 was examined. The extremely rare coexistence of these 2 disorders, however, may support the hypothesis proposed by Simkin.³

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Total magnetic vascular coupling for arterial revascularization

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Several new automatic proximal and distal anastomotic devices are in practical use. Their clinical performance, feasibility, and angiographic follow-up have been described.^{1,2} Most authors report on a single automatic anastomosis using a saphenous vein graft. Total arterial revascularization using beating heart techniques is suggested by many authors to be the method of choice for revascularization.³ However, to our knowledge, such an operation by means of automatic anastomotic devices has not been reported.

We describe the clinical and angiographic follow-up of a patient who underwent total arterial revascularization on the beating heart in which all anastomoses were performed with an automatic anastomotic magnetic coupling system, the Ventrica Magnetic Vascular Positioner (MVP) Distal Anastomosis System (Ventrica, Inc, Fremont, Calif).

Clinical Summary

A 69-year-old man with a 12-month history of angina (preoperative New York Heart Association III) underwent cardiac catheterization revealing 3 vessel disease with significant stenosis (>70%) of all 3 main coronary arteries. Total arterial revascularization was performed

using the right and left internal thoracic arteries (ITAs) and the left radial artery (RA). The surgical approach was through a median sternotomy. All target arteries were exposed using the Octopus 4 Tissue Stabilizer and Starfish Heart Positioner (Medtronic, Inc, Minneapolis, Minn). The patient was given an antiplatelet regimen of 150 mg of clopidogrel (Plavix; Bristol-Myers Squibb, New York, NY) and 100 mg of aspirin the day before surgery and received 300 IE/kg of heparin to maintain an activated coagulation time level of more than 350 seconds during the procedure.

All 4 anastomoses were performed with the Ventrica MVP Distal Anastomosis System. The system consists of 6 magnetic clips, with 3 clips forming a set. One magnetic clip set is positioned into the target artery, and the other is positioned into the bypass graft using a preloaded delivery system. These ports then form an anastomosis by magnetic coupling (Figure 1). The first anastomosis created was the left ITA to LAD. The RA was then coupled distally to the circumflex artery, and after shortening the RA, a T-graft anastomosis was also performed to the left ITA with the magnetic coupler. The right coronary artery required an endarterectomy before the fourth automatic anastomosis with the right ITA was performed. The 4 connections were each created in less than 90 seconds.

The patient's intraoperative and postoperative courses were uneventful. A control cardiac catheterization on postoperative day 6 showed all grafts and anastomosis patent (Figure 2). The patient was discharged on postoperative day 10. Ten weeks after surgery the patient is clinically doing well. He is pain free and was able to reach 125 W for 2 minutes while undergoing an exercise stress test.

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