Letters to the Editor

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- Include no more than 500 words of text, three authors, and five references
- Type with double-spacing
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Spondyloarthropathies and aortic dissection

To the Editor:

I have read with great interest the 2 descriptions of Takagi and colleagues^{1,2} about aortic dissection without Marfan syndrome in ankylosing spondylitis recently published in the *Journal*. I would like to make some rheumatologic comments and ask some questions about them.

Ankylosing spondylitis is a prototype disease of the so-called spondyloarthropathies, which includes other diseases, such as undifferentiated spondyloarthropathy, psoriatic arthritis, and ankylosing spondylitis related to inflammatory bowel disease. Ankylosing spondylitis as a definite entity is easy to diagnose, although at the beginning of the disease, the symptoms and complementary explorations cannot be so clear. It is known that many undifferentiated spondyloarthropathies will progress to ankylosing spondylitis, and the diseases of the spondyloarthropathy group present symptoms and radiologic alterations similar to those of ankylosing spondylitis.

The criteria used by Takagi and colleagues are those of the European Spondyloarthropathy Study Group³ because his patient² clearly presents with the criteria of inflammatory spinal pain and alternate buttock pain and the possible presence of sacroiliitis. These symptoms are enough to classify the patient in the undifferentiated spondyloarthropathy group. The abdominal radiography, as we can see in his study,² does not show an evident sacroiliitis, but some spondyloarthropathy-like radiographic alterations (bamboo spine) appear. There was no association with HLA B27, and the same occurs in 5% of the patients affected by ankylosing spondylitis,⁴ presenting in 20% to 40% of patients with psoriatic arthritis, 80% to 90% of patients with the reactive disease, 33% to 75% of patients with ankylosing spondylitis related to inflammatory bowel disease, and 40% to 80% of patients with undifferentiated spondyloarthropathy. The abdom-

inal radiograph presented in the first patient¹ shows clearly an ankylosis in both sacroiliac joints, and although this patient was not associated with HLA B27, the diagnosis of ankylosing spondylitis is evident.

I would like to ask the authors whether other complementary explorations were practiced (computed tomography, magnetic resonance imaging, and radionuclide techniques) to make the diagnosis of sacroiliitis because a nonconclusive radiograph is presented. HLA B27 negativity opens the possibility of some other entity in the spondyloarthropathy group, such as psoriatic arthritis, reactive arthritis, ankylosing spondylitis related to inflammatory bowel disease, or undifferentiated spondyloarthropathy, which present with less HLA B27. This type of disease might also present a less radiologic alteration in sacroiliac articulations and an asymmetry in the spondyloarthropathies' typical syndesmophytes.⁵

I do not cast doubt on the ankylosing spondylitis diagnosis in any patient presented by Takagi and colleagues, although the apparently normal sacroiliac joints in the second patient² and the absence of HLA B27 oblige us to reject other spondyloarthropathy types. I would be grateful to the authors if they could discuss whether they performed some complementary explorations to look for the sacroiliitis and whether other entities, such as psoriatic arthritis, reactive arthritis, ankylosing spondylitis related to inflammatory bowel disease, or undifferentiated spondyloarthropathy were rejected. That possibility would have an enormous clinical interest because there is not aortic dissection without Marfan syndrome descriptions in other spondyloarthropathy types without being an ankylosing spondylitis.

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Reply to the Editor:

This letter is to acknowledge the rheumatologic comments and queries by Juna regarding our recently published case reports^{1,2} of aortic dissection without Marfan syndrome in ankylosing spondylitis. In regard to the second case,² Juna asked us whether other complementary explorations, such as computed tomography scanning, magnetic resonance imaging, or radionuclide imaging, were practiced to obtain the diagnosis of sacroiliitis. We practiced computed tomography scanning (Figure 1), not magnetic resonance imaging and radionuclide imaging, which demonstrated relatively

apparent sacroiliitis, not typical ankylosis in the sacroiliac joints, despite no evident sacroiliitis on the abdominal radiography in the figure of the second case report. To be accurate, however, the diagnosis of the 2 cases might not be ankylosing spondylitis itself but spondyloarthropathies, which consist of ankylosing spondylitis, psoriatic arthritis, reactive arthritis, inflammatory bowel disease-related arthritis, and undifferentiated spondyloarthropathy, because the patients met only the European Spondyloarthropathy Study Group criteria³ for the classification of spondyloarthropathy. In both cases, the diagnosis of psoriatic arthritis, reactive arthritis, or inflammatory bowel disease-related arthritis may be denied because of the absence of psoriasis, genitourinary or gastrointestinal infection, or inflammatory bowel disease. Undifferentiated spondyloarthropathy is one of the probable diagnoses as mentioned by Juna, because criteria might not be fulfilled for any specific spondyloarthropathy in the second patient. Even though the second case is not ankylosing spondylitis but undifferentiated spondyloarthropathy, there have been no cases of aortic dissection in undifferentiated spondyloarthropathy in the literature except for our case.

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Figure 1. Computed tomography of the second case.

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Implantable cardioverter-defibrillator after left ventricular reconstruction?

To the Editor:

We read with great interest the article by O'Neill and coworkers,¹ which addresses an important question: Is implantable cardioverter-defibrillator (ICD) implantation indicated after left ventricular reconstruction (LVR)?

The authors present their large experience of LVR as a nontransplant surgical strategy for patients with heart failure, with a focus on postoperative malignant arrhythmias. Primary end points were all-cause mortality and appropriate ICD therapies, and median follow-up was 381 days. In addition to the LVR, a small proportion of patients (13%) received a specific antiarrhythmic surgical procedure consisting of cryoablation, about half (46%) underwent a mitral valve procedure, and most patients (88%) were revascularized. The main findings were that patients remain at high risk of ventricular arrhythmias after LVR and that the arrhythmias occur early postoperatively, in two thirds of the cases within 90 days. The authors recommend early ICD implantation or electrophysiology (EP)-guided ICD therapy before hospital discharge after LVR.

We have 2 questions regarding the study by O'Neill and coworkers¹: (1) How many patients had clinical arrhythmias before surgical intervention? (2) Were EP studies conducted before surgical intervention in any of the patients?

The answers to these questions are important to assess the effect of the procedure per se on the incidence of postoperative arrhythmias. There is some theoretic or indirect evidence that LVR promotes electrical stability in the heart by different mechanisms.²