Severe Main Coronary Artery Disease in a Young Woman with Ankylosing Spondylitis

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Abstract

Patients with ankylosing spondylitis (AS) have an approximately two-fold increased death rate compared to the general population, which is predominately caused by increased cardiovascular risk. The prevalence rate for myocardial infarction is approximately 2-3 fold increased as compared with the general population. The inflammatory process appears to have an important role in causing this excess cardiovascular risk. In this paper, we present a case of severe coronary artery disease which could be demonstrated clearly by computer tomography in a 27-year-old woman who is being followed with AS.

Key words: ankylosing spondylitis, severe coronary artery disease, computed tomography

Introduction

Ankylosing spondylitis (AS) is a chronic inflammatory disease that primarily affects the axial skeleton and sacroiliac joints (1). Aortic root, aortic valve involvement and conduction defects are observed in 10% of asymptomatic patients (1). Coronary involvement is a relatively less common manifestation in patients with AS. In this study, a case of a 27-year-old woman with AS who presented with chest pain and whose coronary angiography revealed main coronary artery disease is presented and the challenges and solutions regarding visualization of coronary stenosis are discussed.

Case Report

A 27-year-old woman presented with a one-year history of a squeezing type of chest pain and back pain. The patient reported that her complaints had been aggravated in the last 2 months and she had pain with cold sweats during rest. The patient was being followed up with the diagnosis of AS for 4 years and had used indometazine for 12 months. She had no cardiovascular risk factors other than smoking. She was on no medication except for etodolac for 3 years, a nonsteroidal anti-inflammatory agent. Her Ankylosing Spondylitis Disease Activity Index (BASDAI) was 3.7. Ankylosing Spondylitis Functional Index was 2.5. On physical examination, blood pressure was 110/70 mmHg, and pulse was 88/min. There were no pathologic findings on cardiovascular or other system examinations. Electrocardiogram (ECG) revealed ST segment on electrocardiography (ST) depression in leads V3-6. Telecardiography was normal. Troponin I level was 0.004 ng/mL, and Creatine kinase myocardial band (CK-MB) was 0.04 ng/mL. The sedimentation rate was 8 mm and C-reactive protein (CRP) was 6.05 mg/L. Echocardiography revealed normal findings. The aortic root and aortic arch were normal and no aortic regurgitation was noted. Based on the patient’s findings, a coronary angiography was performed. Selective left coronary angiography revealed a severe stenosis extending from the left main coronary artery (LMCA) to the left anterior descending artery (LAD) and circumflex artery in the left cranial projection (Fig. 1). A simultaneous ECG revealed ST depression on chest derivations. There was no dampling or ventricularization in the pressure traces. The projection was repeated after the administration of 300 μg nitrate, with the suspicion of catheter-induced spasm. No improvement was observed in the stenosis in the same projection. Distal LMCA and ostial...
LAD could not be clearly visualized in other standard projections. The right coronary artery was normal on selective right coronary angiography. An aortography revealed aortic dilatation and regurgitation. Considering the inability to visualize stenosis clearly in other projections and the young age of the patient, we decided to perform multislice computed tomography. The stenosis documented on angiography was also confirmed by multislice computed tomography. The presence of stenosis was confirmed (Fig. 2, 3). The patient was recommended to undergo a surgery based on a joint decision by the cardiology and cardiovascular surgery departments. Upon consent of the patient, a bypass operation was performed [LIMA-LAD, Saphenous vein graft between aorta and circumflex artery (SVG-Cx), Saphenous vein graft (SVG)-diagonal]. Findings on follow-up examinations of the patient three months after the operation were normal. The patient was informed about the risk factors of cardiovascular disease.

Discussion

Ankylosing spondylitis is an autoimmune inflammatory disease with a prevalence ranging between 0.11% and 0.2% and an incidence of 7/100,000 (2). It is well known that patients with AS are at increased cardiovascular risk (3) and cardiac manifestations are found in 2-10% of patients with AS (4). Previous studies have suggested that other forms of cardiovascular diseases such as conduction defects, left ventricular dysfunction and aortic regurgitation are more common in patients with AS (5). Several studies have shown that patients with AS are at increased risk of myocardial infarction compared to the control group (6, 7). Similarly, the current case also presented with unstable angina pectoris. Our patient was found to have isolated LMCA disease, a severe form of coronary artery disease, which is quite rare in patients with AS.

Numerous studies have been conducted to explain the increased cardiovascular risk in patients with AS. It is known that the inflammatory process plays a role in almost all stages of atherosclerotic process from early atheroma formation to plaque instability and thrombus development (8). A previous study has demonstrated that endothelial functions that are known to play a key role in the early stages of atherogenesis are impaired using the flow dilatation technique (9). Similarly, it has been shown that coronary microvascular function is impaired in patients with AS, which is correlated with CRP and tumor necrosis factor (TNF-α) levels (10).

The actual role of Human Leukocyte Antigen (HLA)-B27 in triggering an inflammatory response causing disease is still not precisely known. There are some theories that can explain this issue. The oldest theory is that of molecular mimicry, in which an autoimmune response initially is mounted against a peptide from an infectious agent and is subsequently directed against HLA-B27 itself due to epitopic similarities. A second theory, that of the arthritogenic peptide, postulates that HLA molecules act as a peptide-binding molecule for infectious agents. The third theory suggests that the T-cell antigen is the true susceptibility factor,
and the last theory implicates an innate etiology unrelated to HLA. Finally, HLA-B27 may simply represent a marker locus, closely linked to the as yet unidentified true immune response gene responsible for the inflammatory response (11).

Several studies have reported a variety of factors increasing the risk of cardiovascular disease in patients with AS. In addition to the conventional risk factors such as hypertension, smoking, and increased body mass index, other risk factors include impaired lipid metabolism (7, 12), decreased physical activity (13), genetics (14), increased prevalence of metabolic syndrome and the use of nonsteroidal anti-inflammatory drugs (15). The case present had normal body mass index and had no hypertension. Our patient did not meet the criteria for the metabolic syndrome. The CRP was slightly higher and we did not entirely investigate inflammatory markers. However, we consider that not only the inflammatory process and use of nonsteroidal anti-inflammatory drugs but also smoking (2 packages of cigarettes a day) played a major role in the development of coronary artery disease in our patient leading to a sedentary life.

Catheter-induced spasm is common during coronary angiography. It may lead to confusion in diagnosis and errors in therapeutic strategies that can result in unnecessary revascularization (16). The routine administration of nitrate can be helpful in differentiating between spasm and true coronary artery disease (16). However, computed tomography is a valuable method used recently in the visualization of the coronary artery ostia which can resolve the diagnostic confusion (17). In this study, the critical stenosis on coronary angiography was not resolved with the administration of nitrates, which prompted us to consider it to be true coronary artery stenosis. However, because of the young age of the patient, we preferred to confirm our suspicion by computed tomography.

In conclusion, patients with AS may present with a variety of cardiovascular manifestations. These patients should also be evaluated for the presence of coronary artery disease. Regardless of age, patients should be evaluated by noninvasive testing, when necessary. It should be kept in mind that the inflammatory process plays a major role in the development of the disease. We consider that the development of atherosclerosis can be limited by both the elimination of conventional risk factors and effective control of inflammation through treatment beginning from the early stage in these patients.

The authors state that they have no Conflict of Interest (COI).

References