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Ankylosing Spondylitis and Cardiac Abnormalities

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There are a number of systemic diseases with cardiovascular manifestations in which echocardiography plays a valuable role for surveillance and follow-up of anticipated cardiovascular abnormalities. Systemic autoimmune disorders are frequently associated to cardiac involvement such as pericarditis, myocarditis, valvular abnormalities or ischemic coronary disease.

Ankylosing spondylitis (AS) is also a chronic systemic inflammatory rheumatic disorder that primarily affects the axial joints and mainly involves young male. Extra-articular manifestations vary widely in terms of both frequency and severity. The most common extra-articular manifestations are uveitis, bowel disease, skin, lung and kidney involvement; less frequently cardiovascular involvement occurs.[1] Screening for extra-articular manifestations in patients with AS is important for appropriate management as the presence of extra-articular manifestations may be a consequence of uncontrolled systemic inflammation and may influence treatment decisions.[2] The prevalence of cardiac pathologies in patients with AS has been reported to be 10% to 30%.[3] It is related to a sclerosing inflammatory process that primarily involves the aortic root and the aortic valve, and the chronic inflammation may extend into the ventricular septum, atrioventricular node, proximal bundle of His and bundle branches or fascicles. Thus, various studies indicate a higher rate of conduction disturbances, valvular heart disease and ascending aortic involvement in patients with AS compared with the normal population.[4]

In this issue of the journal, Park et al.[5] reported the early valvular and aortic involvement in young male patients with AS using transesophageal echocardiography. A total of 70 AS patients were divided into AS group I (< 10 years of disease duration) and AS group II (≥ 10 years) depending on their disease duration after the first diagnosis. The thickness of aortic valve was increased in AS patients than in control (control vs. AS group I vs. AS group II, 1.2 ± 0.3 vs. 1.8 ± 0.7 vs. 2.1 ± 0.8 mm, $p < 0.01$). The thickness of mitral valve was also increased in AS patients (control vs. AS group I vs. AS group II, 0.9 ± 0.1 vs. 1.2 ± 0.3 vs. 1.2 ± 0.4 mm, $p < 0.01$). Aortic root diameter at the sinus of Valsalva was slightly increased in AS group II. Furthermore, aortic strain and distensibility were decreased and aortic stiffness beta index was increased in AS group II compared with control and AS group I. Mitral annulus early diastolic velocity (E') and systolic velocity (S') by tissue Doppler imaging were slightly decreased in AS group II. However there was no increased rate for valvular regurgitation (aortic and mitral valve) and for conduction disturbances.

There are some limitations in this study. As they described, the design of this study was observational and cross-sectional, and conducted in a single center. The clinical significance of the results is unclear in this design. Thus a large long-term prospective study is needed to elucidate the clinical significance of early changes in valve, aorta and myocardial function. There was no age-matched control group for AS group II. Mean ages of both control and AS group I were 27 years. However mean age of AS group II was 34 years. Thus it is unclear whether the slight differences in parameters of tissue Doppler and aortic stiffness were from difference of disease duration or from difference of age. Finally the effects of disease activities and drug therapy for AS on cardiac and aortic abnormalities were not fully evaluated in this study.

Despite the limitations, Park et al's study[5] give us useful informations about early subclinical changes of aorto-mitral valve and aortic stiffness in AS patients in the era of advanced management of AS with immunosuppressive and/or immunemodulating drugs.

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