

**Improper aortic sinus-connected coronary artery: Ascertaining the course is essential (Comment on Ann Saudi Med 2010;1:81-3)**

**To the Editor:** In the January-February 2010 issue of this journal Yaymaci et al presented the case of 54-year-old man in whom the conventional coronary angiography performed because of exercise-induced angina and dyspnea revealed a left aortic sinus-connected single coronary artery (single ostium).<sup>1</sup> According to the authors both coronary arteries "followed their usual courses" and in the absence of hemodynamically significant atherosclerosis the patient was managed medically. My comments and opinion relate to the course of the right coronary artery (RCA) that has not been defined since its course at least proxi-

mally cannot be the usual one, as well as the management of the patient.

An improper aortic sinus-connected coronary artery can take several courses to reach its dependent territory; these include the prepulmonic, interarterial, intraseptal and retroaortic.<sup>2,3</sup> Accurate delineation of the course of ectopic coronary arteries is essential provided its pathophysiological, surgical and prognostic implications. The interarterial course, i.e., between the aortic root and pulmonary trunk, in particular, carries the greatest risk for adverse outcomes and specifically exercise-related sudden death in the young.<sup>2</sup> Other manifestations such as syncope, angina, myocardial infarction, pulmonary edema, dyspnea and palpitations are more prevalent among individuals aged 30 to 35 years and older; they may not be related to exercise, but the onset of hypertension, aortic regurgitation or rapid weight gain. The culprit mechanism has been elucidated by Angelini et al,<sup>3</sup> who showed with means of intravascular ultrasound that the proximal interarterial ectopic vessel is hypoplastic, laterally compressed and stenotic (30-70% at rest); it invariably runs within the aortic wall media. By increasing aortic wall strain, conditions entailing an increased stroke volume and/or aortic pressure may increase stenosis severity to a critical level thus leading to ischemia. Ischemia may be subclinical and lead to patchy myocardial necrosis and fibrosis, namely an arrhythmogenic substrate that may result in sudden death.<sup>4</sup>

The anomalous RCA presented by Yaymaci et al, in a right anterior oblique (RAO)/caudal view, assumes a rightward trajectory being oriented directly towards the right atrioventricular groove. Indeed, this angiographic picture favors the interarterial course.<sup>3,5</sup> During a 30°

RAO ventriculography or aortography such a RCA will be anterior to the aorta and may appear as a radiopaque "dot"; this sign was very likely revealed, but overlooked in the present case.<sup>5</sup> Conventional coronary angiography can sometimes depict the stenotic proximal ectopic vessel in projections orthogonal to the plane of compression, i.e. RAO/cranial or caudal for an interarterial left aortic-sinus connected RCA.<sup>3</sup> In the present case, if the RCA had been selectively injected, a proximal stenosis might have been revealed. In the absence of hemodynamically significant atherosclerosis, ischemia was likely due to an exercise-induced significant stenosis of the intussuscepted proximal ectopic RCA. The patient's history and admission electrocardiogram were unremarkable; hence, in the absence of a RCA-related acute coronary event, the dyskinetic inferior wall revealed was very likely due to cumulative stunning produced by repetitive sublethal ischemic episodes. Yet, the RCA was dominant hence the risk faced by this patient is high owing to the large myocardial area rendered ischemic each time precipitating conditions take place. Consequently, in my opinion this patient should have been managed surgically. In case of doubt regarding the course of improper aortic sinus-connected ectopic coronary arteries depicted during conventional coronary angiography, computed tomographic coronary angiography should be used. It can depict an interarterial ectopic vessel running through the aortopulmonary isthmus as well as its proximal stenotic segment.<sup>3,6</sup>

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DOI: 10.4103/0256-4947.81535

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*The corresponding author Yaymaci et al declined to respond.*

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