A fifty-four year-old male patient presented to Sultan Qaboos University Hospital, Oman, with recurrent atypical chest pain. He had no significant electrocardiogram changes and serial serum troponin levels were normal. He did not suffer from hypertension or diabetes mellitus. He underwent multidetector computed tomography (MDCT) coronary angiography [Figure 1] to rule out obstructive coronary artery disease. The patient had had similar symptoms 2 years previously, for which he had conventional coronary angiography [Figure 2]. Both examinations showed diffuse ectasia of the right coronary artery (RCA), left main, left anterior descending artery and circumflex artery. Mild narrowing of the distal RCA was shown in both examinations.

Coronary artery ectasia (CAE) is characterised by segmental or diffuse dilatation of the coronary artery to more than 1.5 times the diameter of the adjacent segment of the same artery or different arteries. Markis proposed a classification of CAE based on the extent of ectatic involvement. In decreasing order of severity, diffuse ectasia of two or three vessels was classified as Type I; diffuse disease in one vessel and localised disease in another vessel as Type II; diffuse ectasia of one vessel only as Type III, and localized segmental ectasia as Type IV. More than half of CAE cases are due to coronary atherosclerosis, but occasionally they are related to other pathological entities. All three coronary vessels can be affected by CAE, but in 75% of

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**Figure 1a**: Three-dimensional volume-rendered 64-multidetector CT image of the proximal segments of the left and right coronary arteries which are ectatic.

**Figure 1b**: Curved multiplanar reformation of the right coronary artery (RCA) showing diffuse ectasia. There is a non-calcified eccentric plaque (white arrow) in the distal segment of the RCA causing mild stenosis.
patients an isolated artery is ectatic. In patients with concomitant coronary artery disease, the proximal and mid-segments of the right coronary artery are the most frequently involved, followed by the left anterior descending artery and the circumflex artery.

The specific causative mechanisms of abnormal dilatation of the lumen in CAE are essentially unknown. Elevated C-reactive protein levels may suggest an inflammatory process as a cause. Coronary angiography remains the gold standard for the assessment of CAE. Intravascular ultrasound is an excellent tool to assess luminal size and characterise arterial wall changes; it differentiates correctly a true from false aneurysm caused by plaque rupture. Recently, MDCT has allowed non-invasive diagnosis of CAE. The examination is performed in an outpatient setting and requires minimal preparation which may include slowing the heart rate by using beta blockers. The examination is performed in a single breath-hold lasting 10-12 seconds. It has gained wide acceptance among cardiologists as a non-invasive modality in ruling out coronary artery disease in low to intermediate risk patients presenting with chest pain. Leschka et al. found the incidence of CAE was 3% among 677 patients who underwent cardiac CT using dual source MDCT.

In the majority of cases, CAE accompanies atherosclerotic coronary disease. The clinical presentation and the long-term cardiac complications are mostly associated with the severity of the co-existing coronary lesion. CAE appears to be associated with traditional cardiovascular risk factors such as hypertension, smoking and lipidaemia, but not with diabetes mellitus. The co-existence of CAE with obstructive coronary lesions in the great majority of patients and the isolated coronary ectasias have led to the generalised administration of aspirin in all patients with CAE. Current literature suggests that ectatic coronary arteries, even without the presence of coronary stenosis, are subject to thrombus formation, vasospasm, and spontaneous dissection. Regarding management and therapy, there are several opinions. Some studies, which are based on significant flow disturbances within the ectatic segments, suggest anticoagulation should be the main therapy. Invasive management, with or without bare or covered stent positioning, has been recommended with caution, as the effects still have to be proven.

References


