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Coronary Tortuosity: Normal Variant or Pathological Condition? A Case Report

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Highlights

Coronary tortuosity is a common coronary angiography finding. The aetiology and the clinical significant are not well defined, generally considered a normal variant. We showed a case of marked tortuosity of all coronary arteries associated with myocardial ischemia.

Keywords: Coronary tortuosity; distal hypoperfusion; myocardial ischemia.

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Coronary tortuosity (CT) is a finding commonly found at the angiographic evaluation[1]. This condition is defined by the presence of ≥3 bends (≥45° change in vessel direction) along the main trunk of at least one coronary artery, present both in systole and in diastole[2]. Up to date, the aetiology and the clinical significant are not well defined. Evidence suggests an association between CT, chronic pressure load and impaired left ventricular relaxation[2-4]. Moreover degeneration of elastin in the arterial wall plays an important role in development of arterial tortuosity[3]. Elastin is a fundamental component of arterial tunica media and is the basis of retraction forces. This latter normally opposes to the forces of traction and pressure that tend to lengthen vessels and this explains why tortuosity of arteries is a phenomenon age-dependent and related to pathological changes of the elastic material of vessels[3]. At this purpose arterial tortuosity syndrome is a rare autosomal recessive connective tissue disorder determining tortuosity and elongation of arteries, especially of aorta and coronary arteries[5]. Other conditions predisposing to tortuosity are atherosclerosis and hypertension. Tortuosity is more present in atherosclerotic arteries than in others. Mechanical forces related to the dynamics of blood flow in arteries can induce the development of atherosclerotic process[5-6]. Moreover experimental model in animal showed that enlargement, elongation, and tortuosity of arteries represent adaptive changes to high flow and high shear stress associated to smooth muscle cell and endothelial cell proliferation and migration[7]. CT is therefore one of the forms of artery remodelling induced by hypertension and can be recognized as an adaptive change of hypertension. Furthermore Yang Li et al. found an association between CT and female gender independently by the level of arterial hypertension[1]. Few cases in literature showed the presence of angina symptoms with abnormal exercise stress test in patients with CT without fixed atherosclerotic stenosis. We report a case of stress-induced myocardial ischemia in a middle age female with CT without coronary atherosclerosis.

A 53- year old women was referred to our Operative Unit for exertional (emotional and physical stress) angina disappearing at rest and after nitrates. She had undergone to an ECG exercise stress test, positive for chest pain and electrocardiographic changes (ST depression in left and inferior leads). Stress/rest myocardial scintigraphy showed a reversible defect in the proximal portion of the antero-lateral wall (figure 1). She had arterial hypertension and dyslipidemia under farmacological treatment, without other cardiovascular risk factors. Physical examination showed no abnormalities. Laboratory findings and echocardiography were normal. Coronary angiography showed marked tortuosity of all 3 coronary arteries (figure 2) without any fixed atherosclerotic lesions. The patient was discharged with beta blocker, cardioaspirin, antihypertensive therapy and statin.

In this patient CT determines flow alteration resulting in a reduction of coronary perfusion pressure distal to the folds of the coronary artery leading to myocardial ischemia. Flow alteration were enhanced by the high hart rate associated to emotional and physical stress with the shortening of the period of diastolic coronary perfusion. Panel A, C and E of figure 2 showed systolic frames of coronary arteries while B, D and F diastolic ones. We can observed changes of coronary conformation during the two phases of cardiac cycle, without a reduction in the degree of coronary folding during the diastolic phase. The increase of heart rate accentuated this phenomenon, by reducing the

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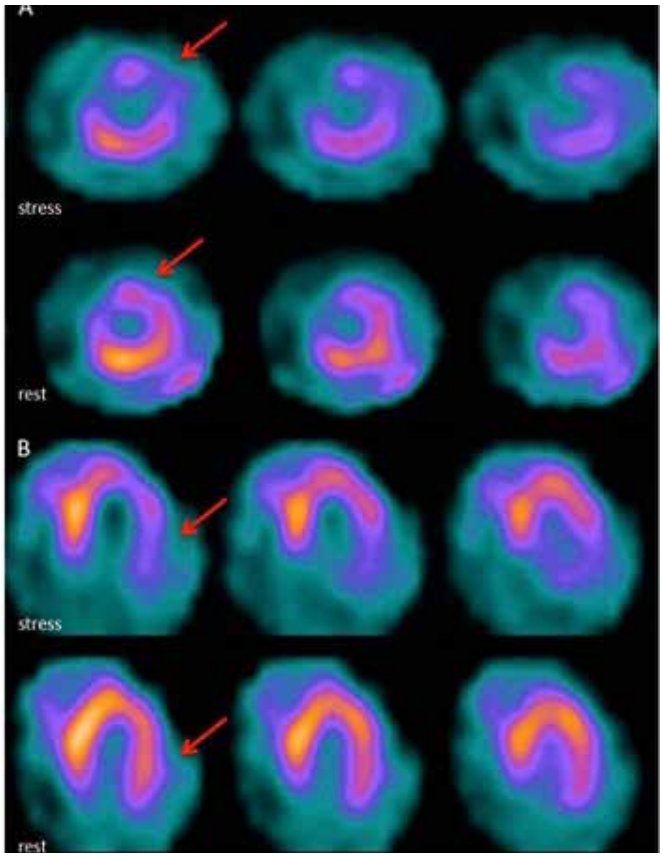

Figure 1.
Myocardial rest/stress scintigraphy:
A. Short axis views showing on the top, stress-induced hypoperfusion of the proximal portion of the antero-lateral wall (red arrow), which disappears at rest, see the second line (reversible defect).
B. Horizontal long axis views showing, on the top, stress-induced hypoperfusion of the proximal portion of the antero-lateral wall (red arrow), which disappears at rest, see the second line (reversible defect).

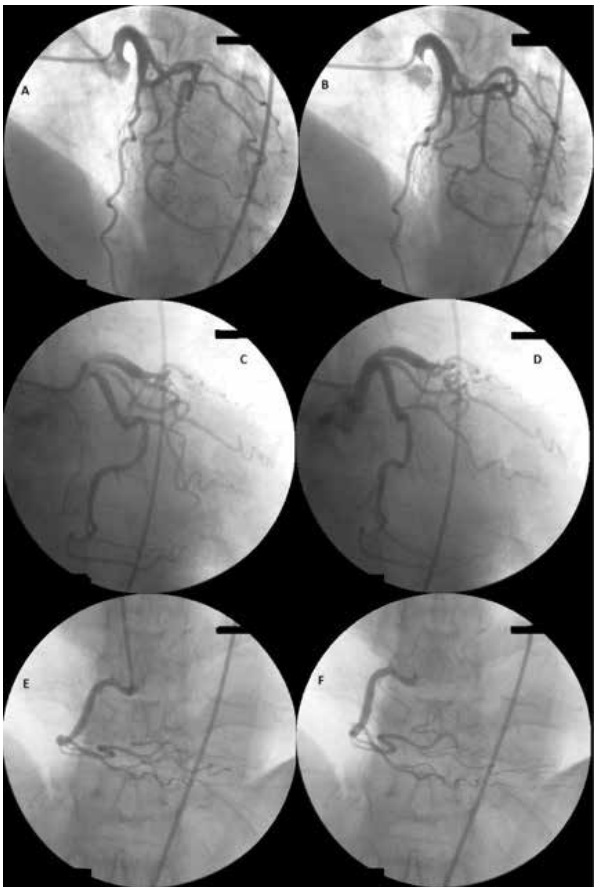

Figure 2.
Panels A and B: left anterior oblique views with cranial angulation showing left main trunk with very tortuous left descending and circumflex arteries in systole and diastole, respectively.

Panels C and D: right anterior oblique views with caudal angulation showing left descending and circumflex arteries with marked tortuosity in systole and diastole, respectively. Finally panels E and F show right coronary artery in right anterior oblique views with caudal angulation: we observe marked tortuosity of all segments of the artery in both systolic and diastolic frames (E and F respectively).

diastolic phase and the subsequent coronary deployment. The flow alterations are therefore emphasized by the increase in heart rate associated with emotional and physical stress, thus determining distal hypoperfusion and subendocardial ischemia.

This case shows that, in some circumstances, i.e. marked tortuosity extended to the three coronary districts, CT could represent a pathological condition rather than a normal variant. Patients should therefore be treated with proper medical therapy and followed over time. A single case is not enough to affirm this concept, other experiences are needed.

Declarations of Interest

The authors declare no conflicts of interest

Acknowledgements

The authors state that they abide by the statement of ethical publishing of the International Cardiovascular Forum Journal[8].

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