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Coronary Artery Ectasia and Aneurysm

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Abstract

Coronary artery aneurysms and ectasia are often incidentally diagnosed and may cause arrhythmia, ischemia, rupture, thromboembolism, and heart failure; in adults are caused by atherosclerosis, in pediatrics Kawasaki disease and Takayasu arteritis are main factors. Others are connective tissue disorders, infections, vasculitis, and genetics. Authors comment on recent literature data.

Keywords: aneurysm; coronary artery; ectasia

Dear** Journal of Clinical Medicine of Kazakhstan **Editors,

The long-term outcomes of revascularization in patients with multivessel coronary artery disease and comorbidity have been a major concern, as exposed by the review of Madiyeva MI and colleagues, published in this Journal [1]. The objective of this letter is additional comment on issues of coronary aneurysm (CAA) and ectasia (CAE) [2–10].

CAA may be congenital or acquired, fusiform or saccular, single or multiple, 1.5 times larger than adjacent normal artery segments [2, 9]; CAEs occur in up to 8% of atherosclerotic patients, being found by coronary angiography in up to 5.3% of cases [3, 9]. Atherosclerosis precedes up to 50% of these changes in adults, while Kawasaki disease Takayasu arteritis, and Ehlers-Danlos syndrome are main causes in pediatrics [2, 6]. Additional etiologies include systemic connective tissue diseases, infections, vasculitis, congenital anomalies, genetic factors, and idiopathic [9]. Complications are spasm, thrombosis, embolization, rupture, and compression [9]. Targeted treatments avoid the CAA and improve outcomes [2]. Matrix metalloproteinases, inflammatory cytokines, and growth factors may promote arterial wall remodeling with weakening, favoring the CAAs; growth factors act in angiogenesis and vascular remodeling [2].

A 54-year-old diabetic man with hypertension and dyslipidemia had chest pain and coronary angiography showed right CAE the left anterior descending artery [3]. He became asymptomatic using heparin, aspirin, clopidogrel, angiotensin-converting enzyme inhibitor, beta blocker, statin, and Rivaroxaban, for two years of follow-up. [3]. A 50-year-old hypertense male had severe aortic regurgitation and paroxysmal nocturnal dyspnea; the angiography study showed multiple coronary segments; he was successfully managed by the aortic valve replacement [3]. A 67-year-old diabetic man with dyspnea on exertion had angiography images of the right, circumflex, and left anterior descending coronaries with large diameters; he was treated by aspirin, beta blocker, statin, Dapagliflozin, and Rivaroxaban [3]. A 51-year-old hypertense man had typical angina and the angiography showed CAEs in the left anterior descending and proximal circumflex; he became asymptomatic using aspirin, beta blocker, statin, and Rivaroxaban [3]. A retrospective study compared 260 CAE patients with 419 controls, the average ages: were 59.9 years, and t 38.3% of CAE patients had higher levels of RC than controls; there was association between the levels of RC and elevated risk of CAE [4]. A retrospective evaluation among 16600

patients who had coronary angiography showed isolated CAE in 1.7%; and left anterior descending artery was more affected (52%) [5]. A 79-year-old hypertensive and dyslipidemic man with atrial fibrillation was diagnosed with right CAA, the lesion was at the proximal right coronary, with a maximum diameter of 9 mm and an extension of near 18 mm [6]. A retrospective comparison between primary and secondary outcomes and predictors of mortality of ad-hoc versus planned percutaneous coronary intervention (PCI) in CAE, included 3,179 CAEs (ad-hoc PCIs: 1,286 and planned PCIs: 1,893) [7]. An adolescent with Ehlers-Danlos syndrome had electrocardiographic and biomarker patterns of infarction; CAEs were found in the left coronary and the anterior descending branch, the left circumflex branch, and the right coronary were occluded [8].

Reporting rare entities may reduce the underdiagnosed and misdiagnosed cases.

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