

Coronary artery ectasia and atrial electrical and mechanical dysfunction

Koroner arter ektazisi ve atriyal elektriksel ve mekanik fonksiyon bozukluğu

Total atrial conduction time is measured as the time delay between the onset of the P-wave (preferably in lead II) of the surface electrocardiogram and the peak A'-wave on the tissue Doppler tracing of the left atrial (LA) lateral wall (PA-TDI duration). It is a good parameter together with P wave dispersion to assess LA electrical and hence mechanical functions (1). While, electrical remodeling can precede any structural abnormalities in the myocardium (2), those two parameters together could be of a good prognostic value in general cardiology settings.

PA-TDI duration shown to be independently predictive of new-onset atrial fibrillation (AF) and also AF in patients after acute myocardial infarction. Moreover, due to its trusted predictive value, The PA-TDI duration is considered a useful tool to identify patients with congenital heart disease at risk of developing atrial arrhythmia during follow-up (1, 3).

The correlations between isovolumic relaxation time, P wave duration and LA size and coronary ectasia may reflect, indirectly, the element of diastolic dysfunction in this cohort. It seems happening alongside with coronary artery ectasia (CAE).

CAE is the abnormal dilatation of coronary arteries such that the ectatic segment exceeds the diameter of the normal adjacent segments or the diameter of the patient's largest coronary vessel by 1.5 times. CAE may be diffuse or segmental and in approximately 20 of patients is believed to be congenital in origin (4).

The pathophysiology of CAE remains unclear and it is believed to be nonatherosclerotic in etiology (5). Kawasaki disease (KD) is another common cause of acquired heart disease in children, causing CAE. Kawasaki disease prevalence is overstated by its geographical distribution, however, the increasing attention given to the disease has had a huge implication on the increasing number of newly diagnosed KD in a nonfamiliar destination like the united states (6).

Patients with CAE may suffer from ischemic changes due to coronary slow flow and loss of media tone allowing ischemic manifestations to occur. That in term would again predict why CAE patients are more prone to ventricular arrhythmia and atrial arrhythmia consequently.

However, isolated, nonatherosclerotic CAE was not investigated thoroughly before and the mainstay always is the definite diagnosis of CAE patient with ought evidences of atherosclerosis. In few studies, the correlations between conventional cardio-vascular risk factor (like diabetes, hypertension, family history of ischemic heart disease and hypercholesterolemia) and CAE failed to prove that disease as a substrate to conventional atherosclerosis (7). Likewise, the histopathological analysis as well detailed a different nature of the disease. However, CAE remains mainly referred to as a failure of coronary walls and subsequently remodeling secondary to heavy atherosclerosis burden (8). Hence, the ideal definition of isolated CAE need to be more redefined by more specific targeted investigation. CAE could be classified as a nonatherosclerotic CAE if no obvious atherosclerosis is detected by quantitative angiogram.

Although the relationship between CAE and atherosclerosis remains only modest established, previous reports showed better prognosis in pure CAE patients when compared with both atherosclerotic obstructive CAE and non-obstructive coronary artery disease (CAD) (9). A result, which was contradicted and claimed that CAE is not a benign condition and it rather requires careful monitoring (10).

The direct relationship between CAE and the risk of coronary slow flow (CSF) phenomenon was described earlier as a marker for CAR pathogenesis. The CSF can be assessed by TIMI frame count method (TFC), an index of coronary flow velocity along the entire epicardial coronary artery. CAE is usually associated with a higher TFC (slower flow) (11), phenomena chronic ischemia that may create significant uncertainty about the indolent course of the disease (12). The other aspect of CSF that can predispose to thrombosis leading to acute thrombotic myocardial infarction during stress. This acute life-threatening condition has been reported previously (13). The possible arrhythmogenic ventricular complication is also documented.

Myocardial performance; where left ventricular (LV) function has been shown to be abnormal in CAE with raised myocardial performance index (MPI). That suggests a dyssynchronous function of the myocardium with CAE. Furthermore, MPI has

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been reported to be abnormally high in segments subtended by the ectatic coronary arteries (14). The relevance of these findings in the presence of other evidence supporting ischemia in CAE patients would be of great help. Similarly, the author in this paper, concluded that PA-TDI duration that is used to assess LA conduction time is prolonged. Which is also associated with prolonged P wave dispersions. In other word, there is clear evidence of atrial asynchronous happening as a consequence to CAE. This could be a potential prognostic factor to atrial arrhythmia.

Using terminology of CAE with non-obstructive CAD rather than isolated CAE might be more recommended. The word (isolated) does not identify accurately the nature nor the atherosclerotic burden of the ectatic segment.

Correlation of ectasia and inflammatory markers are not strongly appreciated and they mostly studied mixed ectasia against CAD. CAE is noticed to be associated with more elevated inflammatory markers but not huge power given to that notion (15).

CAE cohort who defined as a nonobstructive CAD, with 30% stenosis or less, should not identified as isolated (or nonatherosclerotic) CAE. It is clearly known heavy atherosclerotic burden can simulate CAE with apparently minimal quantitative angiographic changes (positive remodeling of vessels) (8). Again, that is another reason to count against using the word isolated CAE as it might involve CAD up to 30-40% coronaries atherosclerosis in that cohort. The methods are clearly written.

Abstract is clearly indicating that study is the first in assessing LA dysfunction associated with CAE. Also, pointing at the significance of possible atrial arrhythmia, decrease cardiac output and hence heart failure.

Finally, this study Öztürk et al. (16) presents a new idea in explaining the pathophysiology of atrial tachyarrhythmia in CAE patient. That would potentiate the trend of using oral anticoagulants as a treatment option over antiplatelets (17). It will serve both guarding against the risk of stroke with AF and also thrombotic acute coronary syndrome in CAE patients.

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References

- den Uijl DW, Gawrysiak M, Tops LF, Trines SA, Zeppenfeld K, Schlij MJ, et al. Prognostic value of total atrial conduction time estimated with tissue Doppler imaging to predict the recurrence of atrial fibrillation after radiofrequency catheter ablation. *Europace* 2011; 13: 1533-40. [\[CrossRef\]](#)
- Kuznetsova T, Herbots L, López B, Jin Y, Richart T, Thijs L, et al. Prevalence of left ventricular diastolic dysfunction in a general population. *Circ Heart Fail* 2009; 2: 105-12. [\[CrossRef\]](#)
- van der Hulst AE, Roest AA, Holman ER, Vliegen HW, Hazekamp MG, Bax JJ, et al. Relation of prolonged tissue Doppler imaging-derived atrial conduction time to atrial arrhythmia in adult patients with congenital heart disease. *Am J Cardiol* 2012; 109: 1792-6. [\[CrossRef\]](#)
- Syed M, Lesch M. Coronary artery aneurysm: a review. *Prog Cardiovasc Dis* 1997;40:77-84. [\[CrossRef\]](#)
- Antonios AP, Chatzizisisb YS, Giannoglou GD. Pathogenetic mechanisms of coronary ectasia. *Int J Cardiol* 2008; 130: e 335-43.
- Newburger JW, Burns JC. Kawasaki disease. *Vasc Med* 1999; 4: 187-202. [\[CrossRef\]](#)
- Boles U, Zhao Y, David S, Eriksson P, Henein MY. Pure coronary ectasia differs from atherosclerosis: Morphological and risk factors analysis. *Int J Cardiol* 2012; 155: 321-3. [\[CrossRef\]](#)
- Glagov S, Weisenberg E, Zarins CK, Stankunavicius R, Koletts GJ. Compensatory enlargement of human atherosclerotic coronary arteries. *N Engl J Med* 1987; 316: 1371-5. [\[CrossRef\]](#)
- Demopoulos VP, Olympios CD, Fakiolas CN, Pissimissis EG, Economides NM, Adamopoulou E, et al. The natural history of aneurysmal coronary artery disease. *Heart* 1997; 78: 136-41.
- Endoh S, Andoh H, Sonoyama K, Furuse Y, Ohtahara A, Kasahara T. Clinical features of coronary artery ectasia. *J Cardiol* 2004; 43: 45-52.
- Papadakis M, Manginas A, Cotileas P, Demopoulos V, Voudris V, Pavlides G, et al. Documentation of slow coronary flow by the TIMI frame count in patients with coronary ectasia. *Am J Cardiol* 2001; 88: 1030-2. [\[CrossRef\]](#)
- Tatlı E, Yıldırım T, Aktöz M. Does coronary slow flow phenomenon lead to myocardial ischemia? *Int J Cardiol* 2009; 131:e101-2. [\[CrossRef\]](#)
- Çelik T, İyisoay A, Kursaklıoğlu H, Yüksel C, Turhan H, Işık E. ST elevation during treadmill exercise test in a young patient with slow coronary flow: a case report and review of literature. *Int J Cardiol* 2006; 112: e1-4. [\[CrossRef\]](#)
- Ceyhan K, Koç F, Özdemir K, Çelik A, Altunbaş F, Karayakalı M, et al. Coronary ectasia is associated with impaired left ventricular myocardial performance in patients without significant coronary artery stenosis. *Med Princ Pract* 2012; 21: 139-44. [\[CrossRef\]](#)
- Li JJ, Nie SP, Qian XW, Zeng HS, Zhang CY. Chronic inflammatory status in patients with coronary artery ectasia. *Cytokine* 2009; 46: 61-4. [\[CrossRef\]](#)
- Öztürk S, Özyaşar M, Ayhan S, Özlü F, Erdem A, Alçelik A, et al. Coronary artery ectasia is associated with atrial electrical and mechanical dysfunction: an observational study. *Anadolu Kardiyol Derg* 2012;12:00.00.
- Endoh S, Andoh H, Sonoyama K, Furuse Y, Ohtahara A, Kasahara T. Clinical features of coronary artery ectasia. *J Cardiol* 2004; 43: 45-52.