Sinus Node Dysfunction and Atrial Fibrillation Associated with Isolated Sinoatrial Node Artery Ectasia

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ABSTRACT

A 74 year-old female patient was admitted to emergency service with a complaints of dizziness and shortness of breath. Complete atrioventricular block was observed and patient was followed up two days with transient transvenous cardiac pacemaker. Afterthat, atrial fibrillation (AF) was progressed and then, coronary angiography was scheduled. Although all three main coronary arteries was free of the stenotic lesion and coronary ectasia, there was a local ectatic segment with prominent slow flow in the sinoatrial node artery that originated from proximal right coronary artery. Patient was treated with dual anti-agregants and enoxaparine in the hospitalization period. Bradiarrhtymias did not recur and AF was reverted to sinusal rhythm with medical therapy. Classical risk factors for AF progression was absent in the patient. We thought that, ectasia and slow flow in the sinoatrial node artery could trigger the bradiarrhtymia and AF by the mechanism of ischemic sinus node dysfunction.

Key words: *Coronary ectasia, sinoatrial node artery, atrial fibrillation*

İzole sinoatriyal nod arteri ektazisine bağlı sinüs nodu disfonksiyonu ve atriyal fibrillasyon

ÖZET

Yetmişdört yaşında kadın hasta baş dönmesi, nefes darlığı şikayetleriyle acil polikliniğe başvurdu. Atrioventriküler tam blok saptanan hasta iki gün geçici transvenöz kalp pili ile takip edildi. Sonrasında atriyal fibrillasyon gelişen hastaya, koroner anjiyografide uygulandı. Her üç ana koroner arterlerde kritik darlık ve koroner ektazi olmamasına karşın; proksimal sağ koroner arterden köken alan sinoatrial nod arterinde izole lokal ektazi ve ektazik segmentte belirgin yavaş akım izlendi. Medikal takip kararı alınan hastaya ikili antiagregan tedavi ve enoksaparin tedavisi uygulandı. Takipte bradiaritmi izlenmedi; atriyal fibrilasyon sinüs ritmine döndü. Hastada AF gelişimi için klasik etiyolojik faktörler yoktu. Sinoatriyal nod arterinde saptanan ektazi ve yavaş akımın, iskemik sinüs nod disfonksiyona yol açtığını ve buna bağlı olarak bradiaritmi ve AF geliştiğini düşünüyoruz.

Anahtar kelimeler: *Koroner ektazi, sinoatriyal nod arteri, atriyal fibrillasyon*

INTRODUCTION

Coronary ectasia (CE) was defined as a localized or diffuse non-obstructive lesion of the epicardial coronary arteries with a luminal dilation exceeding 1.5-fold the diameter of the normal adjacent arterial segment (1). The incidense of CE is reported up to 10 % of coronary angiography in different series (1).

CASE

A 74 year-old female patient was admitted to emergency service with a complaints of dizziness and shortness of breath. On medical background, patient's right coronary artery (RCA) was stented from middle segment one year ago and she was undermedication for hypertension and dyslipidemia. Complete atrioventricular block was

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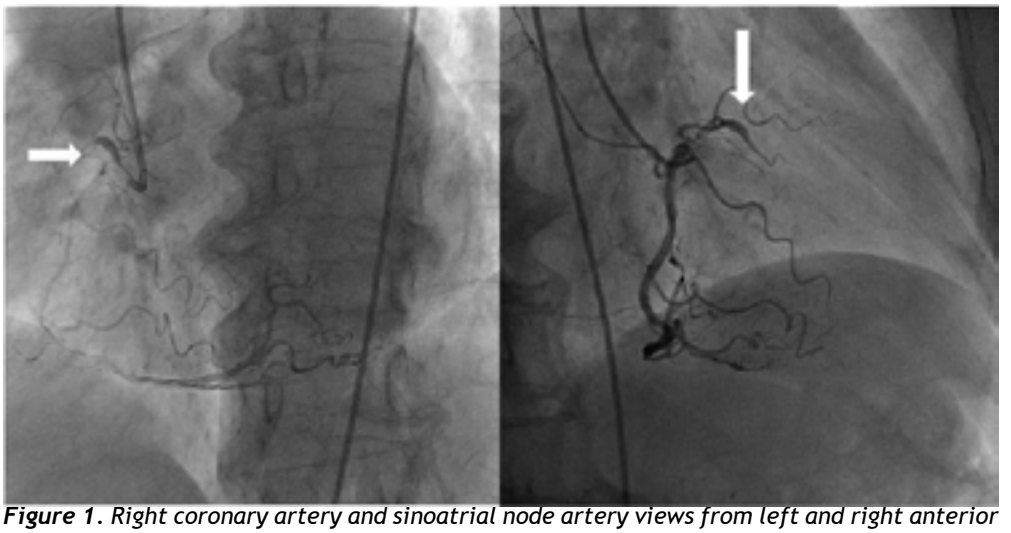

***Figure 1**. Right coronary artery and sinoatrial node artery views from left and right anterior oblique projections. Segmenter ectasia in the proximal sinoatrial node artery and slow flow characterized by the contrast persistance and delayed wash-out seen in the left side of the figure (white arrow indicate sinoatrial node artery course and ectatic segment).*

observed in the initial evaluation and patient was followed up two days with transient transvenous cardiac pacemaker. Afterthat, atrial fibrillation (AF) was progressed and then, coronary angiography was scheduled. Although all three main coronary artery was free of the critical stenosis and coronary ectasia, there was a local ectatic segment with prominent slow flow in the proximal segment of sinoatrial node artery (SNA) that originated from proximal RCA (Figure 1). Stent placed in the middle part of RCA was also patent. Patient was treated with dual anti-agregants and enoxaparine in the hospitalization period. Bradiarrhtymias did not recur and AF was reverted to sinusal rhytm with medical therapy. Classical risk factors for AF development was absent in the patient. Echocardiography showed normal left ventricular and valvular function. We thought that, ectasia and slow flow in the SNA could trigger bradiarrythmia and AF by the mechanism of ischemic sinus node dysfunction.

DISCUSSION

Coronary ectasia most frequently seen in the RCA. Isolated ectasia of coronary side branch without major coronary vessels involvement is exteremely rare. Coronary ectasia usually cause slow coronary blood flow, microemboli or thrombosis in the coronary vessels which can trigger the ischemic coronary events (1). Thus, CE in the SNA may also lead to transient ischemia and it would result with arrythmia. Although there was not clear data about SNA ectasia and dysfunction, several reported case proposed that, SNA dysfunction may trigger arrythmias. During percuteneous intervention of RCA, any iatrogenic disturbance in the SNA blood flow may lead to severe bradiarrythmias and hemodynamic instability (2). Sinoatrial node artery also supply the right atrium with side branches (3). Disturbance in the this flow may also yield to progression of AF. Sinus node dysfunction may also seen after mitral valve surgery and AF ablation as a complication. Such complication usually lead to formation of junctional bradiarrytmias (4). It was also reported that sinus node dysfuntion could be one of the proposed mechanism of postoperative AF occurance (5). Although isolated coronary side branch ectasia is unusual clinical picture, we thought that hypertension, dyslipidemia and atherosclerosis with prominent endotelial dysfunction may lead to formation of ectasia in the SNA which can induce intermittant sinus node dysfunction. We managed this patient medically with clopidogrel 1*75 mg, aspirin 1*100 mg, atorvastatin 1*80 mg, telmisartan 1*80 mg and amlodipin 1*10 mg. First month control ambulatory rhythm holter

monitoring was free of the bradiarrytmias and there was marked decreament in the number and duration of the paroxismal AF attacks.

In conclusion, isolated SNA ectasia is a rare clinical situation. It can cause sinus node dysfunction and bradiarrythmias and also AF may be progressed as a result of blood flow disturbances in the SNA. Patients with bradiarrythmias and paroxismal lone AF, SNA ectasia should be thought in the differential diagnosis of etiologic factors.

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