CASE REPORT / OLGU SUNUMU

Vertigo and Presyncope: Clinical Presentation of Sinus Dysfunction

Vertigo ve Presenkop : Sinüs Disfonksiyonunun Klinik Görünümü

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ABSTRACT

Aim: Aim of this case presentation is to emphasize on the ECG recording of patients with history of drowsiness or presyncope.

Case: 87-year-old male was admitted to the emergency department (ED) with complaints of dizziness, light-headedness, and drowsiness. He had a history of coronary artery disease and he was being treated as per vertebrobasilar insufficiency because of symptoms. Atrioventricular nodal reentrant tachycardia (AVNRT) and right bundle branch block with bifascicular block was documented in the electrocardiography (ECG) records obtained in our ED. Following the treatment with diltiazem infusion, sinoatrial arrest-pause was captured during the monitorization in the ED. He was handed over to cardiology department for electrophysiologic (EP) study afterwards.

Discussion: Sinoatrial (SA) node dysfunction results from any pathology in impulse generation by pacemaker cells in the node or in conduction perinodal transmission cells. The etiologic factors leading to sinus node dysfunction are classified into two pathologies; first is the intrinsic pathology of the SA node, and second group is external causes that affect SA node. The most common intrinsic cause is the age-related fibrosis of the natural pacemaker cardiac tissue in the SA node. If the pause is longer than 3 seconds, the patient is a candidate for ablation therapy which depends on the result of the non-fluoroscopic electroanatomic mapping (CARTO) ablation technique.

Conclusion: Neurological symptoms such as drowsiness and presyncope may often manifest as a result of underlying cardiac pathology. ECG is a very useful tool for these patients if the records are taken at appropriate time.

Keywords: Drowsiness, presyncope, sinoatrial pause, sinoatrial node dysfunction, CARTO ablation

ÖZ

Amaç: Bu vaka sunumunun amacı, öyküsünde baş dönmesi veya presenkop olan hastalarda EKG kaydının önemine vurgu yapmaktır.

Olgu: 87 yaşında erkek hasta acil servise bayılma hissi, sersemlik ve baş dönmesi şikayetleriyle başvurdu. Hastada koroner arter hastalığı öyküsü vardı ve semptomları nedeniyle vertebrobaziler yetmezlik tedavisi almakta idi. Elektrokardiyografi (EKG) kayıtlarında acil servisimizde atriyoventriküler nodal reentran taşikardi ve bifasiküler blok ile birlikte sağ dal bloğu tespit edildi. Acil serviste diltiazem ile tedavi edilirken sinoatriyal pause gelişti. Daha sonra elektrofizyolojik çalışma ve yakın izlem için kardiyoloji bölümüne devredildi.

Tartışma: Sinoatriyal (SA) düğümde disfonksiyon, düğümdeki impuls üreten pacemaker hücrelerde veya perinodal iletide görevli hücrelerdeki patoloji sonucunda ortaya çıkar. Sinus düğümü disfonksiyonuna yola açan etyolojik faktörler iki gruba ayrılır. İlki SA düğümün intrinsik patolojisi, ikinci grup SA düğümü etkileyen ekstrinsik patolojilerdir. En sık intrinsik neden yaşa bağlı olarak SA düğümdeki doğal pacemaker kardiyak dokuda gelişen fibrozisdir. Eğer sinus pause 3 saniyeden uzun sürüyor ise hasta floroskopi kullanmadan uygulanan elektroanatomik haritalama sistemi (CARTO) ablasyon tekniği sonucuna bağlı olarak ablasyon tedavisine adaydır.

Sonuç: Hastalarda baş dönmesi, presenkop gibi nörolojik semptomlar altta yatan kardiyak patoloji sonucunda ortaya çıkabilir. Eğer kayıtlar uygun zamanda alınırsa EKG bu hastalar için çok faydalı bir araçtır.

Anahtar Kelimeler: Baş dönmesi, presenkop, sinoatriyal pause, sinoatriyal düğüm disfonksiyonu, CARTO ablasyon

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Introduction

In the United States, 20% of patients over the age of 60 who complained of dizziness had severe dizziness that interfered with their regular activities. Because symptoms are typically ambiguous and the differential diagnosis is broad, determining the cause of dizziness can be difficult. Tests based on the patient's medical history and physical examination might help narrow down the list of possible diagnoses. However, the underlying reason of this common symptom might range from psychological issues to life-threatening central nervous system disease (1).

Case Report

87 year old male was admitted to the ED with complaints of dizziness, light-headedness, and drowsiness. He was unable to stand up and prone to fall down if left unattended. He was suffering from headache started 10 days ago. He denied accompanying nausea, vomiting, or tinnitus. He had mild chest pain without palpitation. In his medical history he had prior coronary artery disease and had a percutaneous transluminal coronary angioplasty (PTCA) about 5 years ago. Before that intervention, he had suffered from dizziness and light-headedness; which had resolved in time. He had been using drugs for vertigo and vertebrobasilar insufficiency according to his medical prescription records.

At the time of the presentation to ED, his ECG was recorded as soon as he was presented. His vital signs were; blood pressure: 101/71 mm/Hg, heart rate: 160/min, irregular, fever: 36.4°C, O_2 saturation: 94%, Glasgow Coma Scale score: 15. His physical examination was completed and the only pathological sign was rapid heart rate at the oscultation. The remaining cardiovascular, respiratory, gastrointestinal, neurological, and peripheral examination showed no pathology. There was no specific pathological sign at the neurological examination. Neither nystagmus, nor ataxia, nor hearing loss was noted. The cranial CT was normal without any acute anomaly.

ECG (Figure 1) recording showed a rapid regular supraventricular rhythm with about 160 beats/minute. AVNRT was documented as well as right bundle branch block with bifascicular block. After the patient was given diltiazem 0.25 mg/kg intravenously (IV) in order to stabilize the rhythm, the patient had sinoatrial arrest-pause at the ECG which was documented only on the monitor (Figure 2). During that sinoatrial arrest-pause period, the patient felt dizzy again. Afterwards, the patient's rhythm strip revealed bradycardia-tachycardia syndrome, and AVNRT off-and-on (Figure 3). The patient was already consulted to the cardiology department for planning the work-up. Echocardiography (ECHO) was performed under emergency conditions by the cardiologist. ECHO was reported no kinetic disorder in the myocardial structures, no pericardial effusion, no regurgitation of the aortic and mitral valve, and, no anomaly in the systolic function of the left ventricle. Amiodarone (1mg/kg) infusion was started in the ED and the patient was admitted to the Coronary Intensive Care Unit for close monitoring, and further therapeutic intervention. On admission, he was catheterized in the right femoral vein with two 6 F catheters and in right ventricle and coronary sinus with two 6 F EP catheters for EP study. EP study revealed no abnormality in terms of SA and atrioventricular (AV) node functions and associated aberrant conduction. Following 24 hours of monitorization without any need for further medical intervention, he was discharged after starting the drug regimen amiodarone 200 mg twice a day, amlodipine 5 mg once a day, acetyl salicylic acid 100 mg once a day, orally.

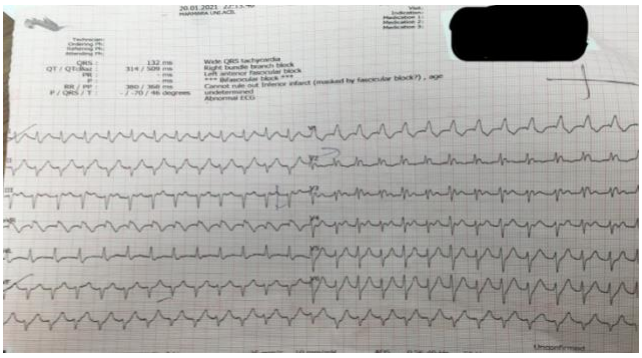

Figure 1. Initial ECG on admission

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Discussion

SA node dysfunction results from any pathology in impulse generation by pacemaker cells in the node or in conduction perinodal transmission cells. The transient abnormality of

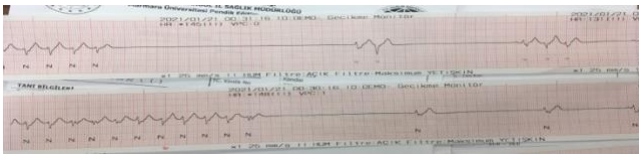

Figure 2. ECG strip following IV diltiazem administration

SA pacemaker is caused by an underlying disease or may occur in healthy individuals. Sinus pause association of atrial fibrillation is a sinus node dysfunction and it may be named as tachycardia- bradycardia syndrome (1). Medical interventions during tachycardia may result with prolonged pauses. If the pause is longer than 3 seconds, the patient is a candidate for ablation therapy which depends on the result of the CARTO ablation technique (2, 3).

Sinus pause often allows escape beats or rhythms to occur following the pause period. Longer episodes of sinus arrest can produce symptoms of dizziness, syncope, and even sudden cardiac death. The etiologic factors leading to sinus node dysfunction are classified into two pathologies; first is the intrinsic pathology of the SA node, and second group is external causes that affect SA node. The most common intrinsic cause is the age-related fibrosis of the natural

pacemaker cardiac tissue in the SA node. In addition; sinus node dysfunction may result from lasting arrhythmias, metabolic or congenital disorders, and surgery (4). The underlying cause sometimes is a mutation which offends the functioning of the ion channels; and it results with sick sinus syndrome. In animal models; it has been shown that long lasting heart failure with or without atrial tachyarrhythmias results in cellular remodeling of the sinus node. The systemic diseases such as collagen vascular diseases, sarcoidosis, amyloidosis, or even metastatic cancer result in the infiltration of SA node. After cardiac surgery for valve replacement, for correction of a structural heart disease or for heart transplantation, there might be secondary damages to SA node or the sinus nodal artery; this repair in turn may lead to SA node dysfunction. On the other hand, infections such as bacterial endocarditis, or viral myocarditis can often result in atrioventricular conduction problems rather than sinus node dysfunction. Since the SA node is located within the atrial wall, SA node is well protected against ischemic injury by atherosclerosis of the arteries feeding node; although SA node dysfunction is unusual, there are a few cases reported (5).

There are several external causes that can affect the normal function of the SA node. These conditions are majorly defined as the abnormally increased vagal tone with vasovagal syncope, or disfunction in the autonomic nervous system. The most common metabolic pathologies leading to SA pause are hypothermia, myxedema coma in hypothyroidism, hyper- or hypokalemia, hypocalcemia, and most significantly hypoxia of the patient due to any cause. Chronic obstructive pulmonary disease (COPD) and sleep apnea often may cause bradycardia with sinus pause because of the deep and profound hypoxia during episodes, especially during sleep (4, 6). Central nervous system lesions with increased intracranial pressure may induce Cushing's reflex which in turn may cause bradycardia with sinus pause. There are several pharmacologic and toxic agents which effects the SA node; these are commonly digoxin, class I to IV antiarrhythmic drugs, mad honey, herbal medications, lithium and other toxins with sympatholytic activity. The examples for toxic substances which create hypoxic conditions at the cellular level which lead to sinus pause are carbon monoxide poisoning or inhalation of chemical warfare gases (6).

CARTO ablation is an invasive technique used to treat patients with different arrhythmias. The mainstay of the technique is the mapping of the conduction system in the heart. CARTO stands for Cartographic information system. Conventional fluoroscopic catheter mapping has limited spatial resolution and has high risk for radiation exposure due to the fluoroscopy. The CARTO system is much safer, three dimensional and more precise in detection of the anomaly (7).

Conclusion

In the emergency departments, ECG monitoring with frequent ECG records of the patients with both cardiac history and neurological symptoms is a must. The fact that cardiac and neurological pathologies often accompany each other in elder patients should be kept in mind.

Figure 3. QR code for the video : On the monitor, bradycardia-tachycardia syndrome, and AVNRT off-and-on

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