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A case of exercise-induced sinus node deceleration without evident coronary artery disease

Ciddi koroner lezyonu olmayan hastada egzersizin tetiklediği bir sinüs nod deselerasyonu vakası

Sinus node deceleration (SND) has been described as an initial increase and subsequent decrease in heart rate with exercise while having higher work load. Exercise-induced SND was firstly reported by Miller and Gibbons (1). Takeuchi et al. (2) reported that it occurs about 1% of patient during exercise stress testing and 8% of patient during dobutamine stress testing. We faced to just only one case between 2000-2006 years. The patient was 47 years old and he has a history of stenting to the left anterior descending artery before 3 years ago. A treadmill exercise stress test was performed at the routine control. During the test, the heart rate steadily increased up to 130beats/min and then immediately decreased up to 25beats/min with complaining of dizziness and near syncope. Electrocardiography was revealed sinus bradycardia with rare ventricular extra systoles and no ischemic ST-T changes (Fig. 1).

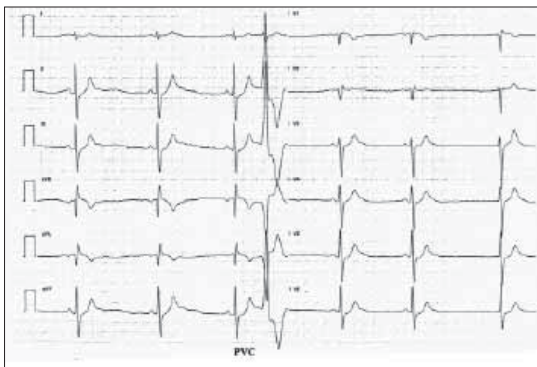


Figure 1. The sinus node deceleration with ventricular premature contraction is seen on electrocardiogram recorded during exercise treadmill test. There are no ST-T changes indicating probable ischemia in any leads

In literature, Gündüz et al. reported a case of SND with two-vessel disease including severe ostial right coronary artery stenosis (RCA) (3). Although first reports discussed that SND might be a marker of significant RCA lesion, later reports demonstrated that SND could be also seen without significant coronary lesion (2, 4, 5). The sensitivity of dobutamine induced SND was 7% for significant RCA lesion, specificity was 92% and the overall diagnostic accuracy was 65%, these results according to them showed that SND is a relatively common during dobutamine stress echocardiography but its importance as a marker of significant RCA lesion was limited due to the high incidence of false positive results (2). Hopfenspirger et al. (5) reported that SND was observed in 10 of 58 patients (17%) during dobutamine perfusion scintigraphy, all of these patients had an inferior perfusion defect and 9 of 10 patients had reversible perfusion defect which was notable for myocardial ischemia, however their study group was relatively small and there was no coronary angiography available. Attenhofer et al. (4) also reported that SND induced by dobutamine was observed in 14 of 181 patients undergoing both coronary angiography and dobutamine echocardiography, they detected that significant coronary artery disease was present only in eight patients and they found no increased prevalence in patient with SND for RCA lesion.

The mechanisms of SND during exercise are thought to be mainly due to increased provocation of the Bezold-Jarisch reflex caused by inferior ischemia or direct sinus node ischemia or coronary angiography or intrinsic sinus node dysfunction. It is also manifested by vigorous myocardial contraction with the drugs (dobutamine, isoproterenol) or exercise. Activation of this reflex leads to both an increase in parasympathetic activity and also a decrease in sympathetic activity resulting in bradycardia, vasodilatation and hypotension. Because the patient was asymptomatic and there was not any available proof of ischemia in the exercise test, we thought that the most probable mechanism of the SND is the Bezold-Jarisch reflex. For this reason, we preferred multislice computed tomography (CT) as a non-invasive imaging method. Multislice CT revealed that the RCA was completely normal and the stent was patent (Fig. 2, 3). Any cardiovascular event was not seen during two years follow-up.

As a result, non-invasive approaches such as CT angiography could be preferred instead of invasive coronary angiography if there is not available high suspicion of significant coronary lesion. This approach may substantially remove this suspicion from both clinician and patient.



Figure 2. A computed tomography angiography image shows the normal right coronary anatomy without any evident lesion along



Figure 3. A computed tomography angiography image shows the patent stent in left anterior descending artery

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Intraoperative measurement of Qp/Qs ratio may be helpful in determining the strategy for sinus venosus type ASD

Intraoperatif Qp/Qs oranının hesaplanması sinüs venosus tipi ASD'nin kapatılması stratejisinde yardımcı olabilir

Nearly all sinus venosus type atrial septal defect (ASD) cases are associated with partial anomalous pulmonary venous connection (PAPVC). Location of orifice and size of PAPVC are important factors in determining

surgical treatment method. The most commonly used method in surgical repair is ASD closure by using a single patch and leaving the PAPVC orifice in the left atrium. After this treatment an occlusion in vena cava superior may be observed which may require cavoplasty with a second patch (2-4). In this technique particularly if PAPVC drains into vena cava superior at a very high level, two corridors should be formed by placing a patch on vena cava superior through a long posterolateral caval incision. This may lead to systemic pulmonary venous obstructions and supraventricular arrhythmias in early or late postoperative days (2). Furthermore, it was reported that the frequency of supraventricular arrhythmias were increased in patients with low diameter SVC which was expanded before two corridor formation. If PAPVC is too large to be left in the right circulatory system, it can be treated by Warden technique, in which supraventricular arrhythmias are observed less frequently. In two different series, sinus rhythms are reported to be maintained and no pulmonary vein obstruction is observed in patients operated by Warden technique (5).

Our case, is an 18 years old women. Sinus venosus type ASD was determined by transthoracic and transesophageal echocardiographies (TEE) and the shunt ratio Qp/Qs was 3:1. Sinus venosus type ASD was also confirmed by cardiac catheterization and the calculated Qp/Qs ratio was 3:2. Pulmonary artery pressure was 50 mmHg. The interdisciplinary consultation decision was surgical closure of the ASD.

Operative technique

Median sternotomy was performed. Surrounding tissues around superior vena cava were dissected up to azygos vein in order to examine presence of partial anomalous pulmonary venous connection and high PAPVC small-medium size was observed. Qp (O₂ content of pulmonary vein-O₂ content of pulmonary artery/oxygen consumption)/Qs (O₂ content aorta-mixed venous O₂ content/ O₂ consumption) rate was found as 2.9. Fick principle was used to detect Qp/Qs rate, intraoperatively (1). Qp/Qs ratio was 2.9 before cardiopulmonary bypass (CPB). This values were obtained by echocardiography (3.1), and cardiac catheterization (3.2). Then superior vena cava cannula was placed into the innominate vein and after bicaval cannulation CPB was initiated. Sinus venosus type ASD was observed. One small-medium sized pulmonary vein draining to vena cava superior closer to innominate vein was observed. This small-medium sized PAPVC was considered to be left in the right circulation system and ASD was closed with pericardial patch. Cardiopulmonary bypass was terminated. Qp/Qs ratio was calculated with switching back to the respiratory measures and FiO₂ values prior to surgery by obtaining blood gas samples from appropriate sites, Postoperative Qp/Qs ratio was calculated as 1.1. The patient was discharged on the 4 th postoperative day without any complications.

In conclusion, PAPVC with small-medium size can be left in right circulation system (6). In our case, the encountered small medium size PAPVC was left in the right circulation system. Preoperative Qp/Qs ratios (TEE;3.1, cardiac catheterization:3.2) and intraoperative Qp/Qs ratio (2.9) before the closure of ASD were similar. Intraoperative Qp/Qs ratio after ASD closure (1.1) and postoperative Qp/Qs ratio on TEE (1.2) were low. So the PAPVC was considered to be left in the right circulation.

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