Srce i krvni sudovi 2020; 39(3): 111-113

Case report

Hyperventilation test as a provocation test in catheterization laboratory

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Abstract We present a patient in whom hyperventilation test disclosed a culprit lesion on the coronary arteries in the Cath lab. A paient, 41-years of age, was treated at the Department of Invasive Cardiology in Medical center Zajecar due to the newly developed inferolateral STEMI. Coronary angiography revealed mild-to-intermiadate LAD stenosis, OM1 was medially occluded, OM2 branch ostial stenosis of 60%, and right coronary artery was minor, without significant angiographic changes. In the same session, primary percutaneous coronary intervention (PCI) was successfully performed on OM1 with Resolute Integrity 2.5x12 mm stent implanted. Due to repeated anginal pain exercise stress test was performed, which was evaluated as positive, and the stress echocardiographic test confirmed hypokinesia of the apical and medial segments of the inferior and lateral wall. A new coronary angiography showed similar stenoses up to 50% in medial LAD and ostial OM2 have been described, but a new 70% stenosis on first diagonal branch which was treated by PCI by another 2.5x16 mm CRE8 stent implantation. One month after discharge, the patient again complained of chest pain again on moderate exertion, occasionally occurring at rest, and resolving after nitroglycerin. Repeated coronary angiography disclosed the same angiographic findings as earlier without progression or in-stent restenosis, and the operator decided to perform a hyperventilation test for 2 minutes, as an additional test to assess the culprit lesion. Immediately following hyperventilation, the patient complained of chest pain with sweating, accompanied with ECG ST elevation in precordial leads. Control angiography demonstrated TIMI 0 flow at the site of the lesion on the medial segment of the LAD. In the same act, PCI was performed on LAD with implantation of CRE8 3x31 mm stent. The hyperventilation test is a simple diagnostic test that may be useful in some patients during coronary angiography to identify patophysiologic mechanism of myocardial ischemia and "culprit" lesion.

Kew words hyperventilation test, coronary vasospasm, angina pectoris

Background

Hyperventilation is a physiological process of forced, accelerated inhalation and exhalation of air, which leads to a decrease in the concentration and partial pressure of carbon dioxide in the blood[1]. Hyperventilation does not increase the concentration of oxygen in the blood and tissues, but increases the elimination of carbon dioxide, which results in the appearance of alkalosis. Alkalosis is manifested by a reduced concentration of hydrogen ions, which leads to more calcium entering the cell from the outside and, together with intracellular calcium, initiating contraction[2,3]. Increased intracellular calcium can cause vasospasm in sensitive epicardial coronary arteries[4]. It is necessary for the patient to breathe rapidly and deeply for 5 minutes, hyperventilate with a respiration rate of about 30 / minute. The test may be performed during invasive coronary angiography with monitoring of the patient's symptoms, ECG and angiographic documentation of coronary vasospasm. Changes in the positive test usually occur 1 to 5 minutes after the end of hyperventilation[5]. A positive provocative test implies the appearance of chest pain, ischemic changes on the ECG and spasm on the angiogram of the coronary arteries. Coronary artery vasospasm is defined as transient total or subtotal coronary artery occlusion (>90%)[6].

We present a case in whom hyperventilation test in Cath Lab disclosed coronary vasospasm at the site of intermediate coronary lesion.

Case presentation

Patient, 41-years old, previosly suffered STEMI of inferolateral localization which was treated at the Department of Invasive Cardiology, Medical center Zajecar. Coronary risk factors included hypertension, and smoking for 20 years. Coronary angiography demonstrated stenosis of 40% in the medial segment of the LAD, occlusion of OM1 branch medially, and intermedaite lesion

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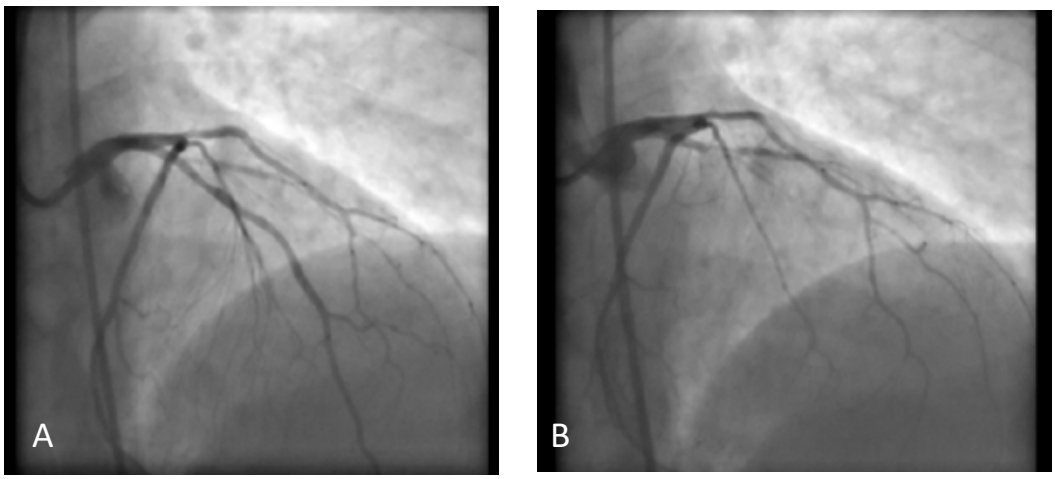

Figure 1. Coronary angiography of left coronary artery before (A) and after hyperventilation test (B) with complete vasospasm and obstruction of coronary flow downstream intermediate coronary lesion

of ostial OM2 branch. The right coronary artery was minor, and without significant angiographic changes. During the same session, primary percutaneous coronary intervention (PCI) was performed on OM1 with implantation of Resolute Integrity 2.5x12 mm stent. The echocardiographic examination showed mild dilatation of the left atrium 4.6 cm, with LV dimensions 5.06/3.68cm, and EF of 50% due to hypokinesia of the apical segment of the septum, and apical segment of the lateral wall. The patients was discharged with following therapy: clopidogrel 75mg, acetylsalicylic acid 100mg, ramipril 5mg, atorvastatin 40mg, bisoprolol 5mg. Due to repeated angiogenic pain, exercise stress test was performed, which was evaluated as positive, and subsequent stress echocardiographic confirmed hypokinesia of the apical and medial segments of the inferior and lateral wall. Repeated coronary angiography showed stenoses up to 50% on medial LAD and ostial OM2, with new 70% stenosis on diagonal branch which was treated with another2.5x16 mm CRE8 stent implantation. Lercanidipine 20mg was added to the therapy. After a month of discharge, the patient haa a pain in the middle chest again on faster walking on the flat surface, but also during resting conditions, dissapering after nitrates. New angiography disclosed similar stenosis in the medial segment of the LAD of 60% (Figure 1A) and the operator decided to perform hyperventilation test for 2 minutes, as an additional test to assess the culprit lesion. Immediately after the test, the patient complained of chest, sweating, accompanied with ECG ST elevation in the precordial leads. Angiography showed TIMI 0 flow at the site of the lesion on the medial segment of the LAD (Figure 1B). In the same session, PCI was performed on LAD by implantation of CRE8 3x31mm stent. The patient was discharged with the same medicatiosn including nitrates and calcium antagonists, and was free of chest pain in the follow-up period

Disscusion

Coronary artery vasospasm is an important mechanism of myocardial ischemia that may produce different clinical manifestation of coronary artery disease from myocardial ischemia, exertion-induced angina, variant angina, to acute coronary syndrome, including myocardial infarction or sudden cardiac death. Coronary artery endothelial dysfunction is thought to play a significant role in vasospasm including dominance of vasoconstrictor effects over vasodilatory factors. Basically, the production of vasoconstrictor substances at the local level, such as catecholamines, angiotensin II, thromboxane A2, serotonin, endothelin, histamine, vasopressin and leukotriene, is increased, with reduced production of vasodilators of nitric oxide and prostacyclin. Coronary artery spasm can occur at the site coronary stenosis that significantly narrows the lumen of the artery, but also in the segmental artery without visible fixed atherosclerotic narrowing. Prolonged spasm can lead to complete cessation of blood flow and to activation and aggregation of platelets with the development of coronary thrombosis and myocardial infarction[7]. A hyperventilation test as well as tests with ergonovine and acetylcholine can be used to provoke vasospasm[8]. Due to the low sensitivity of the hyperventilation test, additional intracoronary administration of acetylcholine or ergonovine is sometimes required[9-11]. If performed in Cath Lab, acetylcholine or ergonovine are safe diagnostic agents that mayt be applied directly to the left or right coronary artery. Drug-induced vasospasm in this way can be easily controlled by the use of intracoronary nitrates, but a small percentage of patients may develop ventricular tachycardia or fibrillation or bradyarrhythmia during a provocative test (3.2 and 2.7%). The incidence of these complications is similar to the incidence that occurs during spontaneous vasospasm episodes (7%). A provocative test can be considered positive if it causes: anginal symptoms, and ischemic changes on the ECG [8]. The development of chest pain after acetylcholine administration, in the absence of angiographically evident spasm, with or without accompanying ECG changes in the ST segment, may indicate microvascular spasm and is often observed in patients with microvascular angina pectoris[11]. In the work of Nakao and Ohgushia, the hyperventilation test proved to be highly specific for the diagnosis of coronary artery spasm[12]. Rassmusen showed that hyperventilation

leading to an arterial PH of about 7.6 has basically the same potency as the 0.4mg ergometrine test, but the hyperventilation test appears to be safer[13].

With this case, we demonstrate how simple hyperventilation test can be used in Cath Lab to reveal patophysiologic mechanism of chest pain and myocardial ischemia. The question remains open on the value of stenting in the patient where vasospasm was superimposed on intermediate coronary lesion. We decided to perform PCI of this lesion on the basis of documentation of large myocardial ischemia with clinically mixed and unstable presentation, and slow but evident angiographic progression of coronary lesion, but the justification of this approach needs to be confimed in the future follow-up of the patient.

Conclusion

The hyperventilation test is a simple diagnostic test that may be important in some patients during coronary angiography to identify vasospasm and the culprit lesion on the coronary arteries.

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Sažetak

Hiperventilacioni test kao provokacioni test u angio sali

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Predstavljamo pacijenta sa kome smo u angio Sali radili hiperventilacioni test radi otkrivanja razloga bola u grudima i ishemije miokarda. Pacijent star 41 godinu je prethodno lečen zbog STEMI inferolateralne lokalizacije. Na koronarografiji nađena je stenoza od 40% u medijalnom segmentu LAD, OM1 grana medijalno okludirana, OM2 grana ostijalno sužena 60%. Desna koronarna arterija je bila minorna, bez značajnih angiografskih promena. U istom aktu urađena je primarna perkutana koronarna intervencija (PCI) na OM1 sa plasiranim stentom Resolute Integriti 2.5x12 mm. Zbog ponovljenih angionoznih bolova urađen je test opterećenja koji je ocenjen kao pozitivan, što je potvrđeno na stres ehokardiografskom testu razvojem hipokinezije apikalnih i medijalnih segmenata inferiornog i lateralnog zida leve komore. Urađena je ponovna koronarografija sa nalazom slične stenoze do 50% na medijalnom LAD i na ostijalnom OM2 i novom stenozom na dijagonalnoj grani oko 70%. Procenjeno je da je moguća "culprit" lezija na DG1 i urađena je PCI sa implantaciojom stenta CRE8 2,5x16 mm. Nakon mesec dana od otpusta pacijentkinja ponovo ima bolove u sredogrudju na brže hodanje po ravnom, tegobe se povremeno javljaju i u miru, prolaze na 1-2 lingvalete nitroglicerina, zbog čega je urađena ponovna koronaografija. Opisana je ista stenoza u medijalnom segmentu LAD i odlučeno je da se uradi hiperventilacioni test u trajanju od 2 minuta, kao dodatni test za procenu miokardne ishemije i "culprit" lezije. Neposredno nakon testa bolesnica oseća bol u grudima praćen preznojavanjem. Na monitoru EKG viđena je ST elevacije u prekordijalnim odvodima. Učinjena je ponovna angiografija na kojoj se sada vidi TIMI 0 protok na mestu lezije na medijalnom segmentu LAD. U istom aktu urađena je PCI na LAD plasiranjem stenta CRE8 3x31 mm. Hiperventilacioni test je jednostavan dijagnostički test koji može kod nekih pacijenata u toku koronarografije da bude od značaja u identifikaciji miokardne ishemije i "culprit" lezije na koronarnim arterijama.

Ključne reči: hiperventilacioni test, koronarni vazospazam, angina pectoris