An isolated right ventricular myocardial infarction and severe tricuspid regurgitation due to occlusion of a non-dominant right coronary artery: role of delayed revascularization

Dominant olmayan sağ koroner arterin tıkanması sonucu gelişen izole sağ ventrikül miyokart infarktüsü ve ciddi triküspit yetersizliği: Geç revaskülarizasyonun tedavideki yeri

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Introduction

Isolated right ventricular myocardial infarction (IRVMI) results from atherothrombotic disease of acute marginal arteries or a non-dominant right coronary artery (RCA) and normal coronary arteries in the presence of right ventricle (RV) hypertrophy (1-4). Although it is rarely manifests, an IRVMI might have dramatically consequences such as dilatation of RV, severe tricuspid regurgitation, pulmonary embolization, severe bradycardia, RV free wall rupture, cardiogenic shock, ventricular tachyarrhythmia and sudden death.

In this paper, we discussed the role of delayed percutaneous coronary intervention (PCI) in the treatment of a complicated IRVMI due to occlusion of a non-dominant RCA.

Case Report

A 69-year-old man with a 40-year-history of diabetes mellitus and hypertension has suffered recurrent (>10 times) and progressive chest pain lasting 20 min. to two hours within last two months, radiating to the inter-scapular region or throat. When he had weakness, fatigue, dizziness and pre-syncope, he had eventually admitted to the emergency room. After 16 hours, he was referred to our hospital for cardiac catheterization. Some of his parameters were as followings; BP, 110/50 mm Hg; heart rate, 50 bpm; respiratory rate, 26/min; body temperature, 36.6 oC; urea, 210 mg/dL; creatinine, 3.02 mg/dL; sodium, 130 mmol/L; potassium, 4.5 mmol/L, Troponin I, 7.29 ng/ml; creatine kinase-MB, 68 U/L. His electrocardiography (ECG) showed ≥ 1 mm ST elevation in D2, D3 and aVF; nearly 2 mm ST elevation in V1, V2 and V3 (Fig. 1); and 0.5 mm ST elevation in V4R. Echocardiographic examination revealed normal left ventricular function, dilation of the right heart chambers, decreased RV systolic function, severe tricuspid regurgitation (TR). These findings indicated an IRVMI (Fig. 2).

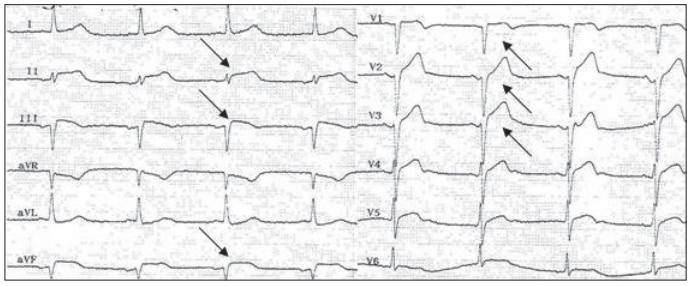

Figure 1. The electrocardiography view of ST segment elevations in precordial leads V1 to V3 (up arrows) and extremity leads D2, D3 and aVF (down arrows)

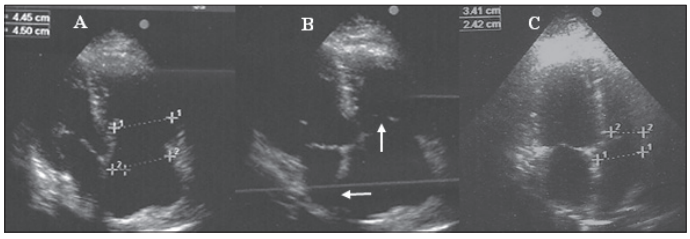

Figure 2. Echocardiographic views of the right heart chambers during diastole (A) and systole (B). Right atrium and ventricle are dilated (A and B). While mitral valve is closed during systole, tricuspid valve is hanging above the tricuspid annulus (B) with a limited coaptation (vertical arrow) resulting in severe tricuspid regurgitation. Interatrial septum is also shifting to the left during systole (horizontal arrow) due to severe tricuspid regurgitation and increased intracavitary pressure. Echocardiography view three weeks after percutaneous coronary intervention: Mild tricuspid regurgitation, normal right heart diameters and interatrial septum (C)

Rhythm monitorization showed sinus node dysfunction presenting with short attacks of bradycardia and sinus arrest followed with sinus tachycardia. Atrial fibrillation attacks, which returned spontaneously to sinus rhythm, were also observed.

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Coronary intervention was delayed because of poor renal function. When renal function improved greatly on the 5th day (urea: 96 U/L and creatinin:0.99 mg/dL), coronary angiography and stent implantation were performed to maintain blood flow of the non-dominant RCA (Fig. 3).

A week later, severe tricuspid regurgitation was persisting although the diameters of right heart chambers were slightly reduced. After three weeks, normal diameter and function of right heart chambers and mild TR were detected (Figure 2-C). We declined an interventional approach to left circumflex OM-2 branch because myocardial perfusion imaging showed only small ischemic area on its territory (Fig. 4).

Discussion

Chest pain is the main symptom of IRVMIs (5). Syncope, hypotension, hypothermia or left hand paresis are also among symptoms/signs of IRVMIs. We documented chest pain radiating to interscapular area and/or throat, sweating, weakness, dizziness, pre-syncope and prerenal azotemia in our case. Bed-side echocardiography was also decisive in our patient (6). Chronic obstructive pulmonary disease (COPD) usually contributes to the development of an IRVMI. However, COPD was not shown in our case.

Right ventricle has a more favorable oxygen supply-demand ratio than the left ventricle, a difference that is attributed to a lower oxygen requirement resulting from its smaller muscle mass, as well as improved oxygen delivery due to the biphasic nature of coronary blood flow during both systole and diastole. In addition, RV receives more extensive collateral flow from the rich left-to-right collateral system (7). As a result of these protective mechanisms, IRVMIs are rarely manifest and generally need a contributory factor such as COPD and/or RV hypertrophy. In addition, GISSI-3 (The Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardio-3) Echo substudy investigators suggested that RV can sustain a long period of ischemia and demonstrate excellent recovery of contractile function after a reperfusion therapy. Compensation of the RV is also demonstrated in two patients in whom limited functional RV myocardium is remained (8, 9).

A successful reperfusion is important to treat an IRVMI, which has higher morbidity and mortality (10). Primary PCI is more successful than thrombolytic because of osteal occlusion and small caliber of culprit lesion and systemic hypoperfusion in IRVMIs. On the other hand, a delayed PCI is usually performed and more successful than medical therapy in patients with cardiogenic shock due to a large left ventricular infarction. Because our patient had unstable cardiac rhythm, presyncope, prerenal azotemia, poor RV systolic function, dilatation of right heart chambers and severe TR due to an IRVMI, we performed a successful (very) delayed PCI.

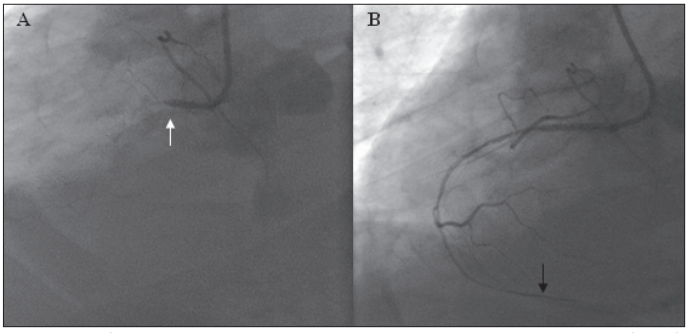

Figure 3. A) Coronary angiography view of right coronary artery (RCA) before percutaneous coronary intervention (PCI) B) Coronary angiography view of non-dominant RCA after a balloon angioplasty with 1.5x20 mm diameter. The black arrow shows the tip of the 0.14 mm guide wire. The geometry of RCA was changed according to remodeling of the enlarged right heart chambers

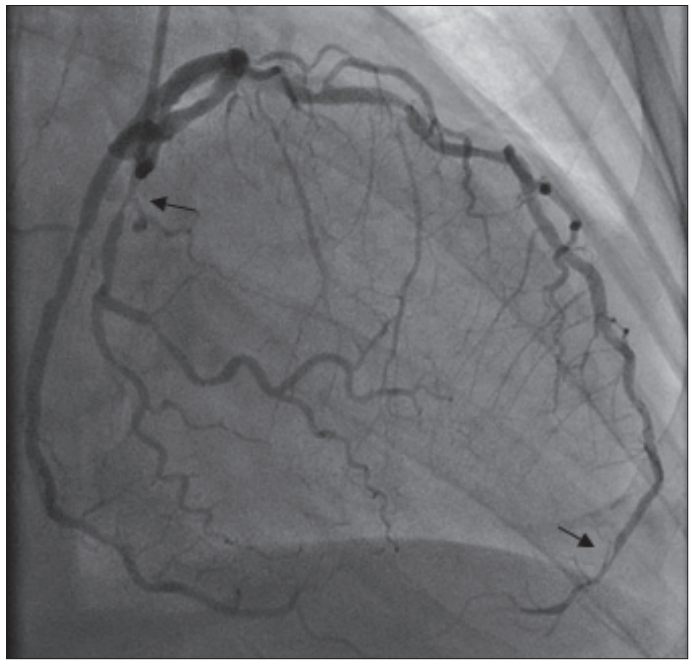

Figure 4. Right cranial oblique view of left coronary artery system: critical stenosis of the second obtuse marginal branch of the left circumflex artery and the distal segment of left anterior descending artery (arrows)

Conclusion

We suggested that complete recovery and/or compensation of RV dysfunction, reverse remodeling of right heart chambers and regression of severe TR could be achieved and/or accelerated with the help of a delayed PCI in selected patients with an IRVMI.

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