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Olgu Sunumu

Sekundum Atriyal Septal Defektli Bir Hastada İzole Sağ Ventrikül Miyokard Enfarktüsü: Olgu Sunumu

Isolated Right Ventricular Myocardial İnfarction in A Patient With Secundum Atrial Septal Defect: A Rare Case

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ÖZET

Sağ ventrikül miyokart infarktüsü, ani olarak gelişen sağ ventrikül sistolik işlev bozukluğu ve sağ ventrikül genişlemesinin yol açtığı hemodinamik bozukluklara neden olan acil bir durumdur. İnferiyor duvar miyokart infarktüslerinin yaklaşık %25 ile %50'sine, sağ ventrikül miyokart infarktüsü de eşlik etmektedir. Atriyal septal defekt ise; atriyumlar arasında şant oluşturan, sağ atriyum ve sağ ventrikül genişlemesine neden olabilen doğuştan bir anomalidir. Daha önceden atriyal septal defekti olan bir hastada sağ ventrikül miyokart infarktüsü nadiren bildirilmiştir. Ancak atriyal septal defekti olan bir hastada izole sağ ventrikül miyokart infarktüsü ise hiç bildirilmemiştir. Bu yazıda; 56 yaşında olan, izole sağ ventrikül miyokart infarktüsü ile başvuran ve daha önce tanı konulmamış sekundum atriyal septal defekti bulunan bir hastayı sunuyoruz.

Anahtar Kelimeler: Atriyal septal defekt, sağ ventrikül miyokart infarktüsü, sağ koroner arter

ABSTRACT

Right ventricular myocardial infarction is a serious emergency that causes hemodynamic disturbances as a result of sudden right ventricular systolic dysfunction and dilatation. 25% to 50% of inferior wall myocardial infarction cases are accompanied by right ventricular myocardial infarction. Atrial septal defect is a congenital anomaly that causes a shunt between the atria and can cause dilatation of the right atrium and ventricle. Right ventricular myocardial infarction has been rarely reported in a patient with atrial septal defect previously. In addition, isolated right ventricular myocardial infarction was not reported in a patient with atrial septal defect. In this article, we present a 56-year-old patient with a previously undiagnosed secundum atrial septal defect presenting with isolated right ventricular myocardial infarction.

Keywords: Atrial septal defect, right ventricular myocardial infarction, right coronary artery

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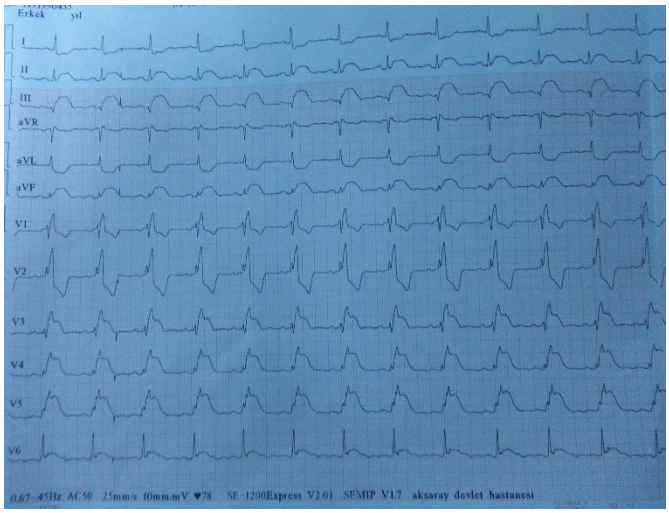
INTRODUCTION

25% to 50% of inferior wall myocardial infarction cases are accompanied by right ventricular myocardial infarction (RVMI) (1). The clinical presentations of RVMI vary widely and range from hemodynamically stable to cardiogenic shock, depending on the degree of right ventricle (RV) ischemia. Atrial septal defect (ASD) is a persistent communication between the atria and causes a shunt between systemic and pulmonary circulation. According to ASD size, it causes volume overload of the pulmonary circulation and the RV. In this article, we present an isolated RVMI in a patient with ASD.

CASE REPORT

A 56-year-old male patient with no known disease was admitted to the emergency room with a chest pain complaint that began 1 hour ago. There were ST elevations in II, III, aVF, and V3-4-5-6 leads, ST depression in I, aVL, and right bundle branch block morphology on the electrocardio¬gram (ECG) taken at the time of admission (Figure 1).

Figure 1. Admission electrocardiographyesim



Blood pressure was 160/90 mm Hg, heart rate 74/min, and oxygen saturation was 92%. Routine blood tests were normal. Portable echocardiography showed RV free-wall hypokinesis, RV dilatation, and normal left ventricle (LV) wall motion. After that, thorax computed tomography (CT) was taken to eliminate pulmonary embolism and was viewed normally. Later, the patient underwent coronary angiography. The non-dominant right coronary artery (RCA) was observed to be obstructed. Percutaneous coronary intervention (PCI) was performed on the RCA (Figure 2). After PCI, the patient's ECG findings improved and there was no chest pain (Figure 3). The patient was evaluated as isolated RVMI. Nevertheless, there were no signs of RVMI. However, another underlying disease was investigated due to clinical incompatibility. Secundum ASD was observed in detailed echocardiography (Figure 4). The patient was discharged healthily after 2 days of follow-up. In control echocardiography after one month, RV dilatation persisted, but RV wall movements were normal. Hereby, percutaneous closure of the ASD was decided.

Figure 2. Coronary angiography; right coronary artery obstruction and non-dominant right coronary artery after succesful percutaneous coronary intervention electrocardiographyesim

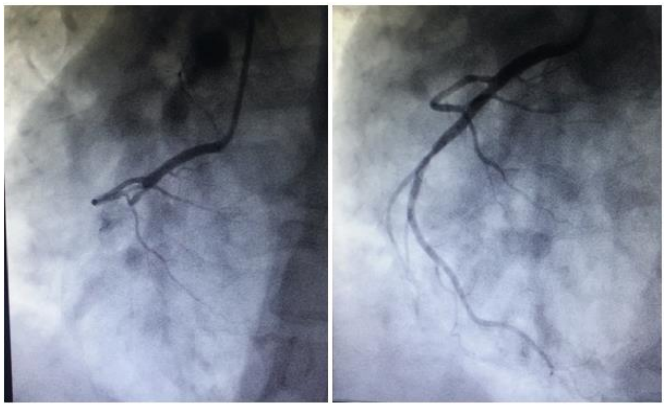


Figure 3. Electrocardiography after succesful percutaneous coronary intervention

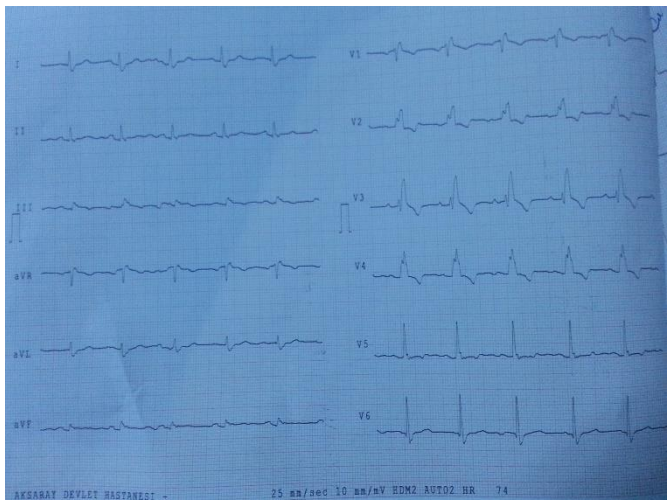
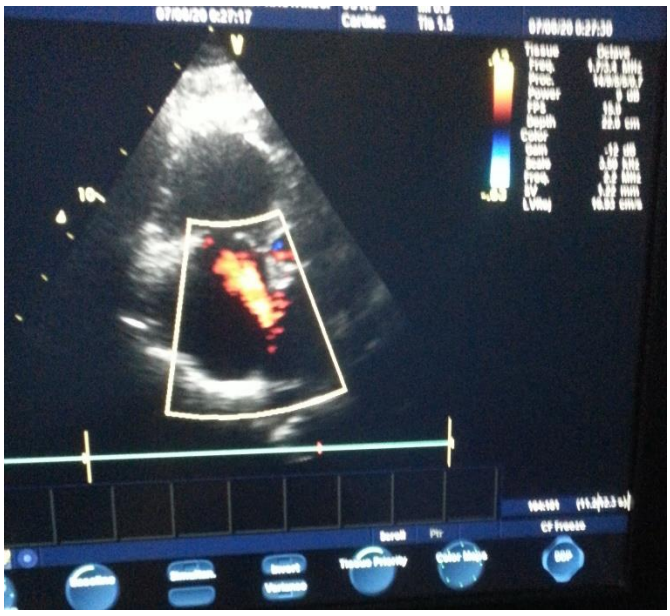


Figure 4. Secundum atrial septal defect by transthoracic echocardiography window



DISCUSSION

The primary effect of RVMI is to cause impaired RV contractility. This leads to reduced blood flow to the lungs and then to the LV. Its clinical manifestations are elevated RV pressures, and decreased LV preload (2). For this reason, RVMI is usually characterized by hypotension, increased venous pressure, and cardiogenic shock (3).

Our patient was hemodynamically stable. Blood pressure and heart rate were normal. There were no signs of RVMI. Persistent moderate or large ASDs cause left-to-right shunts, resulting in right atrial and RV volume overload. These unrepaired shunts can eventually lead to pulmonary arterial hypertension, increased pulmonary vascular resistance, and ultimately RV hypertrophy and dilatation. In our patient, chronic RV dilatation due to ASD may have prevented the reduction of preload LV due to its preconditioning effect. In short, since acute events observed in RVMI developed on a chronic basis due to ASD in our patient, signs of RVMI may not have been observed in our patient.

The ECG findings suggestive of RVMI include ST-segment elevation in leads II, III, and aVF with reciprocal ST-segment depression in the lateral leads (4). ST-segment elevation in lead V4R $\geq$ 1.0 mm is diagnostic of RVMI (5). Isolated RVMI occurs in fewer than 3% of all patients with myocardial infarction (6). Isolated RVMI has been described as acute occlusion of a non-dominant RCA or occlusion of the RV marginal branch during the PCI of the mid-RCA (7,8).

In our patient, there was ST-segment elevation in the anterior and inferior leads, ST-segment depression in I, aVL and there was also a right bundle branch block. Right-sided ECG was not performed because the patient did not have RVMI findings. However, our patient had a non-dominant RCA, normal LV wall movements, hypokinesia in the RV, and RV movements returned to normal after one month, suggesting isolated RVMI.

In some cases of pulmonary embolism, ST-segment elevations mimicking myocardial infarction are observed (9). Additionally, RV dilatation, acute signs of right heart failure, and RV ischemia can be seen.

Our patient had RV dilation, right bundle branch block, and LV wall movements were normal. Therefore, thorax CT was performed to rule out pulmonary embolism.

A rare complication of RVMI is that a pre-existing ASD causes an acute right-to-left shunt. A right-to-left shunt should be suspected in patients who develop acute cyanosis and hypoxemia unresponsive to oxygen administration (10). RVMI may increase right ventricular pressures and cause shunt reversal and hypoxia in patients with pulmonary hypertension due to ASD.

Our patient did not have the right-to-left shunt and hypoxic findings were not observed. The left-to-right shunt was present and oxygen saturation was normal. The patient was likely admitted without an increase in RV pressures, possibly due to early admission. Because our patient applied at our hospital in the first hour of chest pain. If our patient had been admitted to the hospital lately, RV pressures would gradually increase and the left-to-right shunt could have turned to right-to-left shunt.

As a result, RV enlargement due to ASD complicates the diagnosis of isolated RVMI and may affect its hemodynamic effects. The coexistence of RVMI and ASD has been reported very rarely, but isolated RVMI has not been previously reported in a patient with ASD. So, we presented a rare case of the coexistence of ASD and isolated RVMI in this article.

REFERENCES

1. Kakouros N, Cokkinos DV. Right ventricular myocardial infarction: pathophysiology, diagnosis, and management. Postgrad Med J 2010 Dec;86(1022):719-28. doi: 10.1136/pgmj.2010.103887
2. Jeffers JL; Boyd KL; Parks LJ. Right Ventricular Myocardial Infarction. Treasure Island (FL): StatPearls Publishing; 2021 Jan.
3. Cohn JN, Guiha NH, Broder MI, Limas CJ. Right ventricular infarction. Clinical and hemodynamic features. Am J Cardiol 1974 Feb;33(2):209-14. doi: 10.1016/0002-9149(74)90276-8.
4. Nagam MR, Vinson DR, Levis JT. ECG Diagnosis: Right Ventricular Myocardial Infarction. Perm J 2017;21:16-105. doi: 10.7812/TPP/16-105
5. Somers MP, Brady WJ, Bateman DC, Mattu A, Perron AD. Additional electrocardiographic leads in the ED chest pain patient: right ventricular and posterior leads. Am J Emerg Med 2003 Nov;21(7):563-73. doi: 10.1016/j.ajem.2003.08.008.
6. Kinch JW, Ryan TJ. Right ventricular infarction. N Engl J Med 1994;330(17):1211-7. doi: 10.1056/NEJM199404283301707
7. Gregory SA, Desai AS, Fifer MA. Images in cardiovascular medicine. Isolated right ventricular infarction resulting from occlusion of a nondominant right coronary artery. Circulation 2004; 110 (20): e500-1. doi: 10.1161/01.CIR.0000147608.81302.D3
8. Hilliard AA, Nkomo VT, Mathew V, Prasad A. Isolated right ventricular infarction--an uncommon cause of acute anterior ST segment elevation. Int J Cardiol 2009;132(2):e51-3. doi: 10.1016/j.ijcard.2007.08.017
9. Goslar T, Podbregar M. Acute ECG ST-segment elevation mimicking myocardial infarction in a patient with pulmonary embolism. Cardiovascular Ultrasound 2010, 8:50. doi: 10.1186/1476-7120-8-50
10. Bassi S, Amersey R, Andrews R. Right ventricular infarction complicated by right to left shunting through an atrial septal defect: successful treatment with an Amplatzer septal occluder. Heart 2005;91(4):e28. doi: 10.1136/hrt.2004.052100