Right Ventricular Infarction and Refractory Hypoxemia Following Coronary Artery Bypass Graft Surgery

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Abstract

Right ventricular (RV) infarction occurs in 19% to 51% of patients with left ventricular inferior wall acute myocardial infarction.[1,2] The importance of RV infarction and its unique hemodynamic consequences were not well understood until the early 1970s. Among these consequences are hemodynamic disturbances, which may be mistaken for left ventricular dysfunction, pericardial tamponade, and pulmonary embolism. Another serious consequence associated with RV infarction is sudden right-to-left shunting through a previously unsuspected atrial septal defect (ASD) or patent foramen ovale (PFO). We herein describe a patient in whom postoperative RV infarction was associated with refractory hypoxemia through a PFO ***(Iranian Heart Journal 2009; 10 (3):50-52).***

Key words: coronary artery bypass graft ■ myocardial infarction ■ right ventricle ■ hypoxemia

Right-to-left shunting through a patent foremen is a well-described complication of right ventricular (RV) infarction, which can result in the development of severe hypoxemia.[1,2] Hypotension, increased jugular venous pressure, and clear lung fields are the other characteristics of RV infarction, and this condition is associated with considerable morbidity and mortality.[3] Right-to-left shunt should be suspected in any patient who develops acute cyanosis and hypoxemia that is not responsive to an administration of high flow oxygen.[4] Use of positive end-expiratory pressure (PEEP) ventilation in these patients even exacerbates hypoxemia with increasing RV and right atrial (RA) pressure as well as shunt flow through the PFO intra- and postoperatively.

Case report

A 69-year-old man was admitted to the surgical intensive care unit (ICU) after coronary artery bypass graft surgery (CABG). Preoperative angiography revealed diffuse 3-vessel coronary artery disease with an ejection fraction of about 45%.

He underwent surgery under cardiopulmonary bypass (CPB), and the grafts included the left internal mammary artery (LIMA) to the left anterior descending (LAD) and saphenous vein grafts on the second and third obtuse marginals (OMs) diagonal and also the right coronary artery (RCA) after a long endarterectomy.

Weaning from CPB was uneventful, and the patient was transferred to the ICU with stable hemodynamics. Hemodynamic variables and arterial blood gases were normal; however, three hours after he arrived at the ICU, he became centrally and peripherally cyanotic while still under full mechanical ventilation with an inspired oxygen fraction of 1 (FIO_2=1).

On arterial blood gas sampling, oxygen saturation (SPO_2) was 78% with PaO_2 of 47 mmHg.

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Electrocardiography (ECG) showed first A-V block with a heart rate of about 115-120/min. ST elevation was seen in II, III, and AVF leads and reciprocal ST depression in leads AVL and I, consistent with RCA infarction.
Right-sided leads, including V_4R, V_5R, and V_6R, also confirmed our diagnosis (Fig.1).

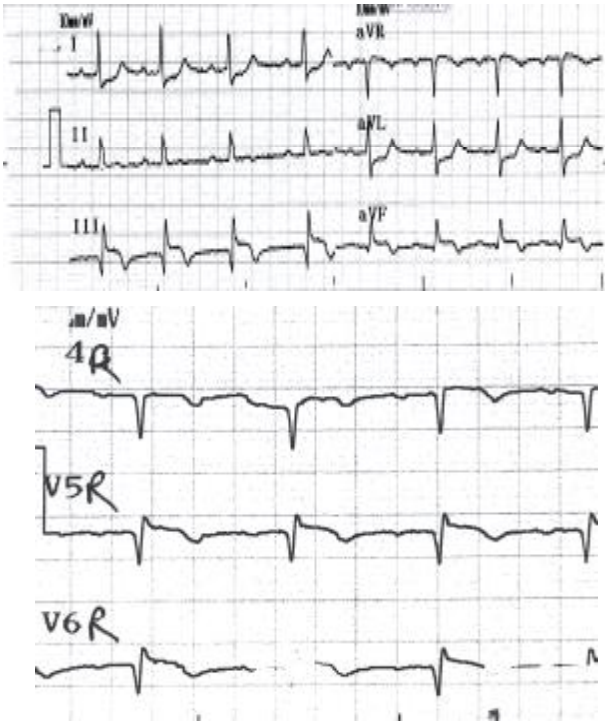

Fig. 1. Electrocardiography showing RV infarction

Blood pressure and central venous pressure (CVP) were 80/60 and 17 mmHg, respectively. After two hours, the patient showed further decrease in blood pressure and oxygen saturation. Chest X-ray was normal. Transthoracic echocardiography demonstrated severe hypokinesia of the inferoseptal, inferior, and inferoposterior walls of the left ventricle and dilation and diffuse hypokinesia of the RV. An IV injection of 20ml agitated saline through a CVP catheter revealed right-to-left shunting across the PFO (Fig. 2).
Treatment was started by increasing the CVP up to 18-20 mmHg, along with an infusion of noradrenalin, milrinone, dobutamine, and low-dose TNG.

During the subsequent 48 hours, RV function improved gradually, which allowed discontinuing noradrenalin and dobutamine.

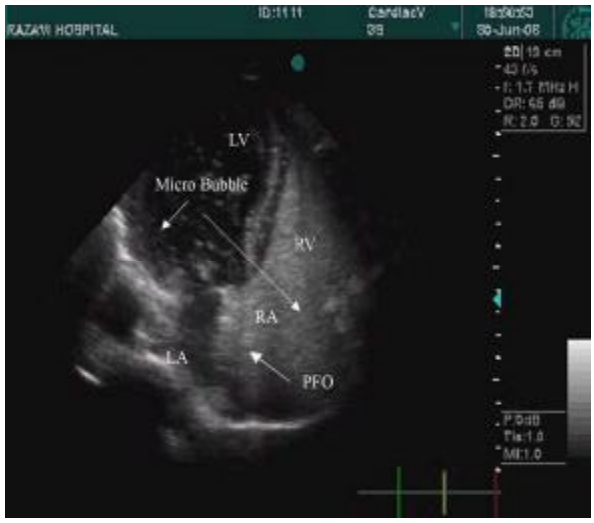

Fig. 2. Contrast echocardiography showing right-to-left shunt through the PFO

Oxygenation and clinical condition continued to improve after extubation (PaO_2 = 82mmHg, $SPO_2 \geq 94\%$ with FIO_2 = 0.4, $PaCO_2$ = 34mmHg).
The milrinone infusion was continued for the subsequent 72 hours. At the end of the fourth day, the patient's SPO_2 was 90 - 95% in room air. He was discharged to the ward after 1 week. The patient was referred to cardiology for further evaluation.

Discussion

Occlusion of the RCA proximal to the acute marginal branches, left circumflex artery (in a dominant left coronary system), and left anterior descending artery can result in the infarction of the anterior RV wall. RV infarction causes depressed RV contractility and compliance with increased RV end-diastolic and right atrial pressures. In patients with PFO, which is reported to occur in 27% of adult hearts, a right-to-left shunt may develop when the right atrial pressure exceeds

the left atrial pressure, and this shunt may produce severe hypoxemia. The development of cyanosis and refractory hypoxemia in patients with recent right ventricular infarction, particularly after endarterectomy, should alert the physician to the possibility of intracardiac shunting.[4]

Medical and surgical interventions include IV nitrates, sodium nitroprusside, inhaled NO, milrinone, beta-blockers, positive inotropes, alpha-1 agonists with midodrine, and surgical or catheter-deployed balloon occlusion of the PFO.[1,2,4,8,9]

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