

Acute respiratory failure in right ventricular myocardial infarction and role of Nitric oxide

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Citation

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Abstract

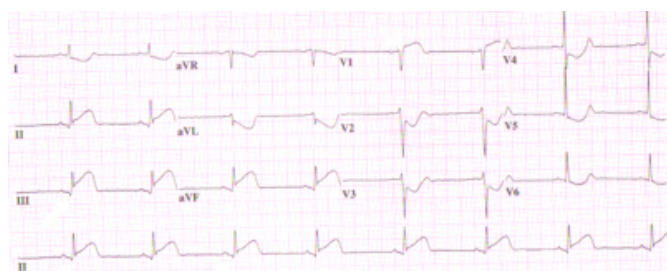
Respiratory failure in right ventricular infarction can occur due to right to left shunt often at inter atrial level. We discuss a patient of inferior wall myocardial infarction with right ventricular extension who developed hypoxia and acute respiratory failure refractory to high concentrations of oxygen. She required mechanical ventilation. Echocardiography with bubble contrast showed a right to left shunt at interatrial level. She was treated with nitric oxide enriched oxygen therapy which improved her pulmonary arterial and right heart pressures and her hypoxia improved. She was weaned off the ventilator and successfully extubated.

CASE HISTORY

A 60 year old woman with a medical history of hypertension, hyperlipidemia and diabetes mellitus presented with a chief complaint of chest pain. Chest pain was retrosternal in location, graded 6/10 subjectively in intensity and was associated with breathlessness and perspiration. . Her electrocardiogram (EKG) revealed ST elevation in leads II, III and aVF with reciprocal changes in the anterior leads (Fig1).

Figure 1

Figure 1: Ekg showing ST elevation in inferior leads and reciprocal changes in anterior leads.



She was immediately taken to cardiac catheterization laboratory for percutaneous coronary intervention. On her way to cardiac catheterization laboratory she became hypotensive and hypoxic and her oxygen saturations dropped to 70's. She failed to improve with supplemental oxygen and subsequently required intubation. Multiple boluses of intravenous saline were given and subsequently she was started on pressors while in catheterization

laboratory. Her repeat EKG showed extension of infarction to right ventricle. Her cardiac catheterization revealed totally occluded right coronary artery proximally and nonobstructive lesions in mid left anterior descending and left circumflex. She also had a fresh clot in her proximal right coronary artery which was aspirated and a stent was placed. After stenting she persisted to be dependent on high concentrations of oxygen although her need for pressors was decreasing. An echocardiogram showed high right atrial pressures (20 mm HG). In view of persistent hypoxia (saturation in high 80's to low 90's) despite being on 100% oxygen a right to left shunt was suspected. She got a contrast echo (bubble study) which revealed right to left shunting at interatrial level. Transesophageal echocardiography revealed a patent foramen ovale (Fig 2). Patient was placed on Nitric oxide (NO) therapy and her oxygen requirement decreased. Her echocardiogram showed improvement in right sided pressure and she was subsequently weaned off the ventilator after 3 days of NO therapy.

Figure 2

Figure 2: TEE showing patent foramen ovale



DISCUSSION

Isolated right ventricular myocardial infarction (RVMI) is rare and is usually seen in association with inferior wall myocardial infarction. The incidence of right ventricular infarction in such cases ranges from 10-50% (1). Right ventricular infarction is usually associated with significant hemodynamic abnormalities. Right ventricle is considered a low-pressure volume pump; its contractility is highly dependent on diastolic pressure. Diastolic dysfunction seen during the right ventricular infarction results in increased diastolic pressure and decreased systolic pressures. Concomitant left ventricular dysfunction may increase right ventricular after load substantially resulting in further decline in right ventricular output. Right ventricular diastolic pressures and the right atrial pressure increases. Increased right atrial pressure causes reversal of flow from right to left in patients with patent foramen ovale leading to hypoxia that is resistant to conventional therapy including supplemental oxygen. (2). Afterload reduction therapy for the failing RV with a selective pulmonary vasodilator might be expected to lead to improved cardiac performance without producing systemic vasodilation and hypotension (3,4,5). NO diffuses to smooth muscle cells in lung arterioles and activates soluble guanylate cyclase to generate cyclic 3, 5-monophosphate (5, 6). This intracellular messenger leads to a reduction in the intracellular concentration of calcium and inhibition of myosin light chain kinase, producing smooth muscle cell relaxation and vasodilation (7). The vasodilator effects of inhaled NO are restricted to the pulmonary circulation, as NO are readily bound to hemoglobin in circulating erythrocytes and inactivated (8). NO has been shown to

decrease pulmonary vascular tone in adults and children with pulmonary hypertension of diverse etiology without causing systemic vasodilation, including patients with congenital heart disease (9), primary pulmonary hypertension (1), and pulmonary hypertension secondary to a variety of etiologies (2). Inhaled NO has also been observed to decrease RV afterload in a porcine model of RVMI (10, 11).

Ignacio Inglessis et. al (12) in their study on patients with RVMI showed that NO was associated with decrease in mean right atrial pressure by $12 \pm 3\%$, mean pulmonary arterial pressure by $13 \pm 2\%$, and pulmonary vascular resistance by $36 \pm 8\%$ (all $p < 0.05$). Nitric oxide inhalation increased the cardiac index by $24 \pm 11\%$ and the stroke volume index by $23 \pm 12\%$ ($p < 0.05$). However no change in systemic blood pressure or peripheral vascular resistance was noted. Breathing NO decreased shunt flow by $56 \pm 5\%$ ($p < 0.05$) and was associated with markedly improved systemic oxygen saturation.

Our patient was able to wean off from the ventilator following an improvement in systemic oxygenation. This is a unique case where right ventricular infarction lead to acute respiratory failure and failing to wean from mechanical ventilator.

CONCLUSION

Respiratory failure from left to right shunt is a rare complication of RVMI which may at times lead to persistent dependence on a mechanical ventilator. NO therapy by improving right ventricular hemodynamics decrease right to left shunt in these patients and might help in subsequent weaning from mechanical ventilator.

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