Case Report

Acute right heart syndrome: Rescue treatment with inhaled nitric oxide

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Abstract

Acute right heart syndrome is a common occurrence in intensive care units and is associated with a poor prognosis. There is lack of understanding of the involved pathophysiology, standard diagnostic protocols and treatment guidelines. Management goals include ensuring adequate right ventricle (RV) filling, maximizing RV contraction and reducing RV afterload. We describe a 39-year-old female with acute decompensated right heart failure secondary to multiple causes. She was managed with inhaled nitric oxide. Her condition improved, which was evident by a decrease in her pulmonary artery systolic pressure on serial echocardiography, decreased requirement of vasopressors and successful weaning from the ventilator.

Keywords: After load, heart failure, nitric oxide

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Introduction

We report the use of inhaled nitric oxide (iNO) as a rescue treatment in a patient with acute the right heart syndrome (ARHS) secondary to chronic pulmonary artery hypertension (PAH) decompensated by multifactorial causes.

Case Report

A 39-year-old female, diagnosed case of chronic kidney disease on maintenance hemodialysis (HD) for the past 9 years with severe PAH secondary to chronic pulmonary thromboembolism (PTE) presented with the complaints of progressive breathlessness and abdominal distension for the past 1 week. Her history was suggestive of Grade III dyspnea with orthopnea. There was no history of fever or cough; blood pressure [BP] 92/46 mmHg, pulse rate [PR] 96/min, respiratory rate [RR] 36/min). Her recent records showed recurrent hypotension during her last few HD sessions.

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Considering her non-tolerance to HD, it was planned to start her on peritoneal dialysis (PD). During the first cycle of PD, she developed severe hypotension and altered sensorium (after inflow phase). She was then shifted to intensive care unit (ICU) for further management.

At presentation to the ICU, she was drowsy, sluggishly responding to verbal commands. Her neck veins were distended. She had respiratory distress, with active accessory muscles of respiration. She had a loud P2, pan systolic murmur. Her abdomen was soft, mildly distended with evidence of free fluid and palpable liver (BP: 70/40 mmHg, PR: 102/min, RR: 30/min, SpO_2: 99% on high flow O_2 via non-rebreathing mask) Her labs were: Hemoglobin 12.3 g/dl, total leucocyte count 8600/μL, serum sodium 138 mEq/L, serum potassium 6 mEq/L, blood urea nitrogen 63 mg/dl, creatinine 6.0 mg/dl. Arterial blood gas (ABG) revealed hypoxia and hypercarbia with mixed acidosis (pH: 7.189, $PaCO_2$: 60.3 mmHg, pO_2: 52.7 mmHg, HCO_3 : 22.5 mmol/L, BE: 6.54 mmol/L, serum lactate 1.92 mmol/L). D-dimer and fibrin degradation product were high. An echocardiogram revealed markedly dilated right ventricle (RV)/right atrial/inferior vena cava (IVC); moderate tricuspid regurgitation (TR), severe pulmonary arterial hypertension (PAH) with (pulmonary artery systolic pressure [PASP] = 93 mmHg paradoxical septal

motion; decreased right ventricular (RV) function and reduced left ventricular (LV) compliance. Work-up for sepsis and acute myocardial ischemia was carried out and was ruled out. Considering her critically ill state, she was not subjected to any radiological investigation to rule out acute pulmonary embolus.

On Day 1 of ICU admission, she was managed with high dose inotropes and vasopressors: Infusion nor-adrenaline (1 mcg/kg/min) and infusion dopamine (20 mcg/kg/min), empiric broad spectrum antibiotics, low molecular weight heparin and sustained low efficiency dialysis. By Day 2, her vasopressor requirement increased and infusion vasopressin (0.04 units/min) was added. In view of her poor sensorium, hemodynamic instability and acidosis she was electively intubated and ventilated. Her pre-intubation ABG was pH: 7.18, $PaCO_2$: 60 mmHg, PO_2: 66.6 mmHg, HCO_3: 21.9 mmol/L, BE: 7.1 mmol/L while post-intubation ABG was pH: 7.29, PaCO2: 36.7 mmHg, PO_2: 80.4 mmHg, HCO_3: 17.3 mmol/L, BE: 8.3 mmol/L. On Day 3, echocardiography revealed findings similar to Day 1 echocardiography (PASP 95 mmHg). In view of her refractory pulmonary hypertension iNO was started (Day 3) through nitric oxide blender (NOxBOX, Ben font Scientific Ltd. Kent, England) at 5 ppm and then increased to 10 ppm. Her hemodynamic parameters showed a progressively improving trend after initiation of iNO. By 12 h (ICU Day 4) of iNO, vasopressors were tapered down. PD was restarted on Day 5 with cumulative negative balance of 2 L (approx). Blood gasses on Day 5 revealed pH: 7.45, $PaCO_2$: 28.4 mmHg, PO_2: 136.2 mmHg, HCO_3: 20 mmol/L, BE: 5.3 mmol/L and lactates 1.61 mmol/L. Her follow-up echocardiography showed a significant reduction in PA pressures with PASP measuring 73 mmHg at 48 h and 63 mmHg at 72 h of iNO initiation [Figure 1]. Gradually, her vasopressor were weaned off completely and NO was withdrawn after 72 h (Day 6). She was extubated

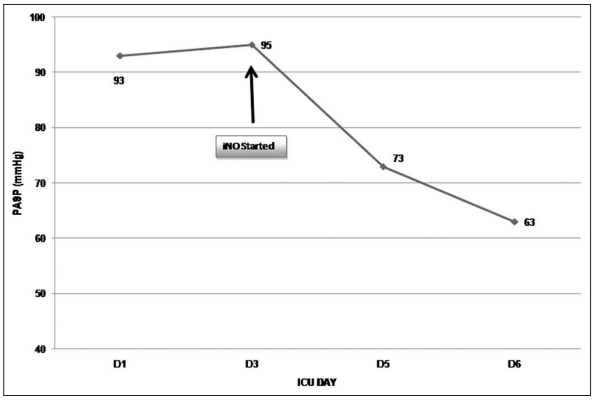

Figure 1: Pulmonary artery systolic pressure in mmHg on echocardiography before and after use of inhaled nitric oxide

the next day and subsequently was maintained on non-invasive ventilation with inspiratory positive airway pressure of 12 mmHg, expiratory positive airway pressure of 4 mmHg. She was shifted out of ICU on Day 10. Throughout the ICU stay the methemoglobin levels were < 1%, as measured by the co-oximetry test.

Discussion

ARHS is defined as a sudden deterioration in RV function that may result in systemic hypoperfusion.[1] This syndrome is secondary to an increase in pulmonary vascular resistance (PVR) and hence RV afterload usually precipitated by exacerbations of chronic lung disease with PAH[2] (e.g., hypoxic pulmonary vasoconstriction in chronic obstructive pulmonary disease), acute massive lung disease[3] (e.g., acute respiratory distress syndrome [ARDS] or pulmonary embolism), high positive end expiratory pressure[4] or acute RV infarction.[5]

Acute increase in RV afterload results in increased RV end-systolic volume and a decreased RV ejection fraction. Severe RV dysfunction may decrease LV end-diastolic volume, thus reducing LV stroke volume and cardiac output.

This paragraph enumerates all the possible multiple conditions that might have caused an imbalance and led to decompensation. She received LMWH in prophylactic dosage only. Because of her critical condition, she could not be subjected to CT Angiography. We postulate that a fresh episode of PTE resulted in an increase in RV afterload, which might have caused an imbalance in her then compensated hemodynamic state. Non-tolerance to her regular HD sessions resulted in fluid overload, pulmonary edema and metabolic acidosis. PD fluid inflow into the peritoneal cavity caused upward displacement of the diaphragm resulting in further respiratory compromise, ventilation/perfusion mismatch and hypoxia. These factors led to hypoxic pulmonary vasoconstriction and increase in PVR. Finally, the mechanical compression of the IVC by the peritoneal fluid caused a decrease in RV preload. All these factors working synergistically precipitated the ARHS.

Management is based on treatment of the underlying cause of increased RV afterload. Supportive therapies are based on reducing PVR, which include improving oxygenation, avoiding respiratory acidosis, correction of metabolic acidosis, avoiding over inflation of the lung alveoli etc., [Figure 2]. Though theoretically promising, substantial improvements in cardiovascular function has not been uniformly demonstrated with the use

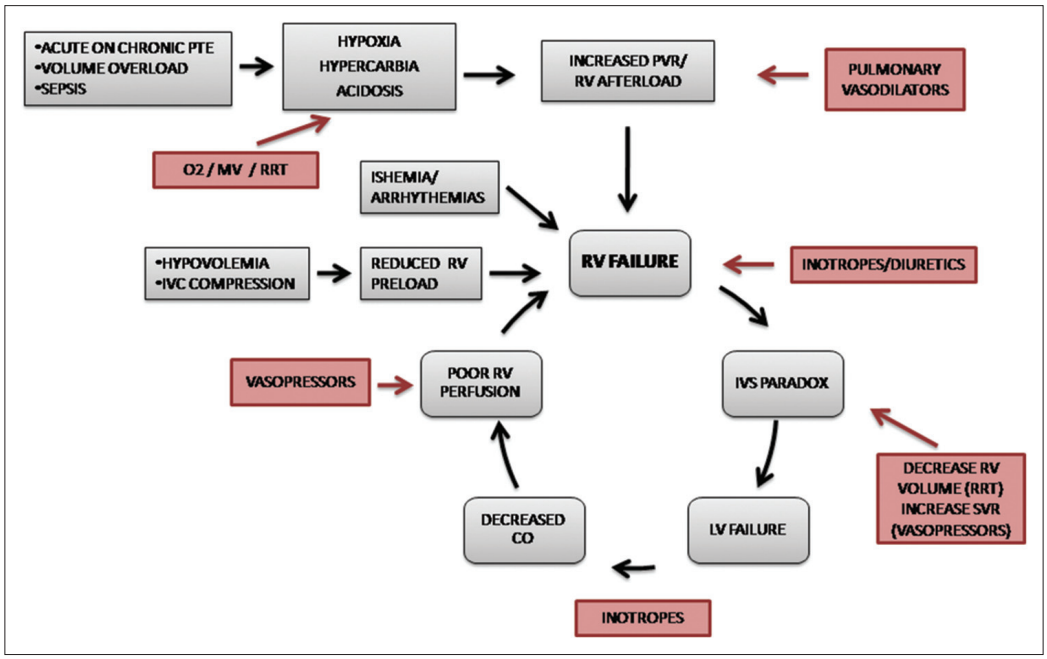

Figure 2: Mechanisms of the right ventricle dysfunction in critically Ill patients with therapeutic interventions aimed at improving RV function

of vasoactive drugs when administered systemically because of systemic hypotension to an undesirable degree.[6] iNO preferentially vasodilates the pulmonary vasculature with its effects limited to ventilated areas of the lung. iNO attenuates hypoxic pulmonary vasoconstriction, decrease pulmonary artery pressures and improve oxygenation in several pulmonary diseases, including persistent pulmonary hypertension in neonates,[7] ARDS,[8] primary pulmonary hypertension,[9] pre-operative and post-operative cardiac surgery.[10]

We report the use of iNO as a rescue treatment in patient with ARHS. Our patient demonstrated a substantial improvement in hemodynamic parameters characterized by decreased vasopressor requirement, decrease in PASP and successful weaning. This is likely of clinical significance in terms of acute management of her hypoperfused state.

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