

Pulmonary hypertensive crisis and its efficient management. A Case report and literature review

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Abstract

Perioperative anaesthetic management of patients with pulmonary hypertensive crisis is complex and challenging because of limited window period and treatment options. This case report describes the successful management of a patient with severe valvular heart disease superimposed by severe pulmonary hypertension, who developed pulmonary hypertensive crisis soon after induction of general anaesthesia for cardiac surgery. Pulmonary hypertensive crisis is not a usual phenomenon in these patients, but if it occurs it can be fatal. Anticipatory care of the patient to prevent crisis is the mainstay of treatment, otherwise it adds additional morbidity to such patients. Fortunately our patient survived the crisis without causing any additional morbidity during his hospital stay.

Keywords: Pulmonary hypertensive crisis, General anaesthesia, Perioperative management, Severe pulmonary hypertension.

Introduction

The haemodynamic definition of Pulmonary hypertension is when resting mean pulmonary artery pressure is ≥ 25 mmHg, Pulmonary Capillary wedge Pressure/Left Atrial Pressure <15 mmHg and Pulmonary vascular resistance of ≥ 3 wood units, as measured by right heart catheterization.¹ Pulmonary hypertension is severe and supra systemic when mean pulmonary artery pressure is > 50 mmHg and > 60 mmHg respectively. Patients with severe pulmonary hypertension have higher mortality about 4%-24% during surgical procedures.²

Rationale for writing this case report is dissipating knowledge and awareness about a rare and fatal event in patients with severe pulmonary artery hypertension.

Case History

A 45 years old male, snuff user, banker by profession,

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known case of valvular heart disease, presented in the cardiothoracic clinic of Aga Khan University Hospital, on 18 February 2013 with worsening exertion dyspnoea, orthopnoea, syncope and palpitation since 1 year. He was referred from another city for valve replacement surgery at our hospital. On preoperative assessment, he was a young, alert and normal built man. His functional class was III (Marked limitation in less than ordinary activity) according to New York Heart Association (NYHA) classification.³ On Auscultation, there was mid diastolic murmur and decreased air entry over the right lower zone. Airway assessment revealed Mallampati of class I. Twelve lead Electrocardiography showed sinus rhythm, right ventricular hypertrophy and T- wave inversion in anterior leads. Echocardiography revealed Ejection fraction of 55%, severely dilated left atrium, severe pulmonary hypertension, severe stenosis of Mitral valve with area of 0.7cm², and moderate stenosis of Aortic valve with area of 1.2cm². Chest x-ray showed right sided pleural effusion and other investigations were within normal limits.

Patient was pre-medicated with oral midazolam 7.5mg with oxygen supplementation. After application of standard anaesthesia monitoring, left radial artery was cannulated under local anaesthesia for invasive blood pressure monitoring. Anaesthesia was induced with intravenous injection of 2mg midazolam, 2ug/kg fentanyl, 0.2mg/kg etomidate and 0.9mg/kg rocuronium. Endotracheal intubation was performed and controlled mode ventilation initiated. Anaesthesia was maintained with 50%oxygen with air and isoflurane. A triple lumen 15cm Central venous catheter was inserted in right internal jugular vein with ultrasound guidance and pulmonary artery catheter was floated through the swanz sheath at the same place. Immediate pulmonary pressures were high as expected, with systolic, diastolic and mean pressures of 92/49mmHg and 63mmHg respectively, low normal Systemic vascular resistance of 799 dynes/cm⁵ and high cardiac output of 7.3L/min. Patient was positioned supine with both legs slightly elevated for surgical procedure and preparation for surgery was started. It

was immediately noticed that the Pulmonary pressures was elevated to 155/55mmHg, much higher than the systemic pressures. This was managed as pulmonary hypertension crisis.

The possible triggering factor was not figured out exactly but it was thought that, light phase of anaesthesia during positioning for the procedure probably led to the crisis. Immediately 100% oxygen was administered, epinephrine infusion 0.2ug/kg/min and dobutamine infusion 5ug/kg/min were started simultaneously for cardiac support. Glycerine trinitrate infusion 1ug/kg/min-2ug/kg/min was started to achieve pulmonary vasodilation by increasing cyclic Guanosine Monophosphate in the constricted pulmonary vasculature.^{4,5} Nebulized through endotracheal tube continuously with Alprostadil (prostaglandin E1) at a rate of 5ml/hour, a potent inhaled pulmonary vasodilator. Pulmonary pressures settled down gradually to 90/50mmHg and surgery was started after successful management of the crisis.

Surgery went uneventful with pump time of 180 minutes and cross clamp time of 150 minutes. Diseased mitral and aortic valves were replaced with tissue valves. The patient was weaned off from the pump with high inotropic support and shifted to Cardiac Intensive Care Unit. He was extubated on 2nd postoperative day when inotropic support was minimal and was started on sildenafil (phosphodiesterase inhibitor) 25mg given through nasogastric tube. Pulmonary pressures were continuously monitored, with trends remaining low 48/32, 50/25, 46/18mmHg throughout his stay in CICU. Patient was discharged after complete recovery and cardiac rehabilitation. He remained well on the follow up visit in the clinic and was advised to travel back to his city in a month. He continued his job in the bank and is now living his life with improved quality.

Patient was contacted through phone for his wellbeing after a year and was requested for written informed consent for publishing his case report in a journal for the reason of dissipating the knowledge among health care providers for which he agreed upon and documents were exchanged through courier.

Discussion

Pulmonary hypertension crisis is a life threatening condition in which series of events culminate in biventricular failure and death. It is characterized by an acute rise in pulmonary pressures to the point

where it exceeds systemic pressures due to rapid increase in pulmonary vascular resistance, causing pressure overload of right ventricle, decrease pulmonary blood flow, hypoxia, and decrease cardiac output due to leftward displacement of the inter ventricular septum with impending cardiac failure.

Perioperative Pulmonary hypertension is associated with worse outcome and mortality rate may exceed 50%.⁶ Anaesthesia related risk factors for perioperative pulmonary hypertensive crisis are hypercarbia, hypoxia, painful stimuli, acidosis and airway instrumentation, which leads to rapid increase in pulmonary vascular resistance.⁷ The best way to prevent such a predictable event is to be prepared for it ahead of time. Anaesthetic management goals are to provide adequate anaesthesia and analgesia, vigilant invasive haemodynamic monitoring, avoidance of stimuli causing pulmonary vasoconstriction and maintenance of cardiovascular stability. Confirmation of pulmonary hypertensive crisis if occurred, is via invasive haemodynamic monitoring through pulmonary artery catheter, parameters such as rising right atrial and pulmonary pressures with decreasing cardiac output are the hallmark.

Perioperative pulmonary hypertensive crisis mandates immediate management to decrease pulmonary vascular resistance by pulmonary vasodilators either intravenous or inhalational, inotropic support and identification of the cause. Supportive measures with 100% oxygen, hyperventilation to decrease CO₂, and treating acid base disturbance are also proven to prompt pulmonary vasodilation.

Conclusion

Patient with severe pulmonary artery hypertension are at high risk for pulmonary hypertensive crisis during surgical procedures, therefore immediate recognition and prompt treatment is the mainstay of successful management.

Disclaimer: This case was seen on 18th Feb 2013 and was presented as interesting case in the weekly departmental meeting of department of Anaesthesiology at Aga Khan University Hospital Karachi on 18th March 2013.

Conflict of Interest: None.

Funding Disclosure: None.

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