ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 12 | December - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CASE REPORT: ANAESTHETIC MANAGEMENT OF AN OBESE PATIENT WITH SEVERE PULMONARY ARTERY HYPERTENSION POSTED FOR LOWER LIMB ORTHOPAEDIC INTERVENTION

Anaesthesiology

Dr. Tanmoy Biswas Peerless Hospital, Kolkata.
Dr. Sushmita Bhattacharya Peerless Hospital, Kolkata.
Dr. Janmejoy Sengupta Peerless Hospital, Kolkata.

ABSTRACT

Pulmonary artery Hypertension is a tricky disease which requires proper early diagnosis and proper optimisation before any elective procedure. It is defined as the condition where the Pulmonary capillary wedge pressure is greater than 20 mmHg at rest and 25 mm Hg after exercise. Due to the high pulmonary capillary wedge pressure the right ventricle is generally pressure intolerant which thereby results in right ventricular failure if not properly controlled.

KEYWORDS

INTRODUCTION

Pulmonary artery hypertension (PAH) is a specific form of high blood pressure that affects the arteries in the lungs and the right side of the heart. In PAH, the small arteries in the lungs, called pulmonary arteries, become narrowed or blocked, making it harder for blood to flow through them. This increased resistance causes the right side of the heart to work harder to pump blood, which can lead to right-sided heart failure over time.

Pulmonary artery Hypertension can be classified on the basis of etiological factors. It can be
1. Idiopathic
2. Cardiac Cause
3. Respiratory cause
4. Thromboembolic Cause
5. Systemic disorder

It manifests with cough, shortness of breath, swelling of limbs and recurrent history of lower respiratory tract infection.

Diagnosis can be done by
- Echocardiogram (to evaluate the heart function)
- Right heart catheterization (to measure the pressure in the pulmonary arteries)
- Blood tests and imaging, such as CT scans or MRI
- Lung function tests

Although there are several causes of Pulmonary artery hypertension, one unseen and unnoticed cause of pulmonary artery hypertension is obesity. Obese people generally have obstructive sleep apnoea which results in Pulmonary artery hypertension.

Here we will discuss a case report of an obese patient with BMI 34 with severe pulmonary artery hypertension posted for lower limb orthopaedic intervention and its perioperative management.

Case Report

A 62 years female patient, resident of Kolkata, with BMI 34, presented to the emergency department of Peerless Hospital and Bk Roy Research Centre with severe pain in the right lower limb on 20/10/2024. On examination the patient was tachypnoeic with room air saturation of 93%, hemodynamically patient was stable, x Ray of right limb showed distal femur fracture. She had a history of right sided Proximal Femoral Nailing on 2021, after which she was diagnosed with mild Pulmonary artery hypertension and was advised medical management and non invasive ventilation therapy overnight. She didn't comply with the therapy and didn't use CPAP or BiPAP therapy at all since 2022. She was also diagnosed with Multiple Myeloma in 2021 for which she received Pomalidomide. She had a history of visit to Kashmir 4 days ago where she developed shortness of breath and lost consciousness and fell on her hip. The patient was shifted to the ICU. All investigations were sent and showed the following reports.

Hb 10.5 gm%, PCV 34.2, TC 5010, PLATELET 1,78,000

CREATININE 0.96

ECG showed sinus rhythm with rate of 76

TSH 3.83
Na+ 135mEq/dl
K+ 4.5 mEq/dl
INR 1.33
NTproBNP 3550
Fibrinogen 705

Chest x ray showed bilateral hilar congestion

Echo2D showed Ejection Fraction of 67%

Severe pulmonary artery Hypertension with PCWP of 62 mmHg

Severe Tricuspid regurgitation

Severe Pulmonary Regurgitation

Right atrium and ventricle dilated with no regional wall motion abnormality

IVC was 2 cm with < 20 % collapsible

Arterial blood gas analysis showed pO2 of 45 mm Hg . rest all parameters were within normal limit.

The Patient was started on BiPaP therapy of IPAP 15 cm and EPAP 5 cm, Injection furosemide infusion. DVT prophylaxis was given using Injection Clexane 40 mg SC once daily. Fluid intake per day was 1.5 litre .Amlodipine was added on along with Telmisartan for the PAH. After proper optimisation, on 25/10/2024, repeat Echo screening was done along with repeat ABG analysis where all parameters were within normal limits except for the PCWP which was 62 mmHg. Patient was kept NPO 6 hrs solid diet and 2 hrs liquid diet. And shifted to OT on 26/10/2024. Plan of Anaesthesia was general anaesthesia with multimodal analgesia.

All monitors were connected, Central venous access to right internal jugular vein was done, invasive blood pressure monitoring was done through right radial artery. After connecting the monitors, at first USG and nerve stimulator guided femoral and sciatic nerve block was given using 0.5% ropivacaine 30 ml in each nerve. Ponit of care lung ultrasound and TEE was done where still IVC showed 2 cm with <50 % collapsibility and presence of B lines in LUS.

Preoxygenation was done for 3 minutes and was anticipated difficult intubation due to OSA and obesity. Induction was done using Inj. Etomidate 6 mg, Injection succinylcholine 100 mg and Injection fentanyl 100 mcg and airway was secured with ETT 7 with help of bougie and laryngoscopic surge was prevented by keeping time < 10 seconds. Ventilator setting given was TV 450 ML, RR 14/MIN, I:E 1:2

and PEEP of 5 cm H2O. Maintenance was done using Sevoflurane 1 MAC, oxygen and air. After proper positing and securing all accesses and airway when incision was given, tourniquet was inflated at pressure of 300 mm Hg. The patient was hemodynamically stable for 30 minutes after which all parameters started to get deranged. Invasive blood pressure decreased from MAP 75 to 40 mmHg, Heart rate increased from 86/ min to 120/min and pulse oximeter reading decreased from 99% to 89%.

Fluid bolus of 250 ml colloid was given, PEEP was decreased to 0 and patient was started on inj. Noradrenaline(8/50) 5 ml/hr. Fluid was given checking IVC and pulse oxiform waveform analysis. Goal directed fluid therapy was given using colloid and crystalloid. But still there was improvement in hemodynamics. Then the patient was started on Inj Vasopressin 2 U/hr and both the inotropes were titrated accordingly. A stress dose of Inj. Methylprednisolone 1 gm and Inj aminophylline 125 mg was given. All inhalation anaesthetics were stopped and patient was only on oxygen an air and to prevent awareness, inj midazolam 4 mg was given. The procedure continued for 3 hours. Total blood loss was 1000ml and total fluid given was 500 ml colloid and 1000 ml crystalloid. After tourniquet deflation there was again decrease in mean arterial blood pressure. After closure the patient was shifted. During shifting, the patient was on inj noradrenaline , injection vasopressin and heart rate was 140/min and blood pressure was 90/50 in the ICU.

In The ICU, bedside TTE and lung USG was done which showed B lines and IVC 2cm and <20 % collapsible, moderate pericardial effusion, adequate left and right ventricular contractility. Patient was then started on Injection furosemide and postoperatively mechanical ventilation was continued. Goal directed fluid therapy was given. For postoperative analgesia, the femoral and sciatic block was adequate enough to control the pain till 22 hrs. The patient was under invasive ventilation at TV 500 ml, RR 16/min and Fio2 40 % with PEEP of 8 cm H2O. Slowly by recording the arterial blood gas analysis and the saturation PEEP was decreased to 5 cm and the patient was allowed to wean at Pressure Support mode 10 cm and CPAP 5cm. After 2 hrs of spontaneous breathing trial when all hemodynamic and respiratory parameters were within normal limits and very low dose of ionotropic support was required, the patient was extubated on postoperative day 2 and continued on BiPAP 15/5 overnight. Finally after tolerating all interventions, the patient was shifted to HDU on POD 3 and then to ward on POD 4 , after which the patient was discharged.

DISCUSSION

Pulmonary artery hypertension is a detrimental disease which if goes un noticed and inadequately treated can lead to complications. Obesity itself can exaggerate the PAH. And thus before selecting any patient with PAH for anaesthesia, the patient must be properly optimised. Fluid therapy also plays an important role in the patients of PAH.

In patients with PAH, the right ventricle is already compromised, thus increasing the fluid load by giving liberal fluid strategy can result in pulmonary oedema while restrictive fluid strategy can decrease the right ventricular flow. Thus goal directed fluid therapy is the ideal process of fluid therapy. Depending on the procedure the anaesthesiologist should plan the choice of anaesthesia and also the agent to be used. Generally during induction excessive hypotension is to be avoided and also laryngoscopic surge can be avoided using lignocaine.

Parameters to be monitored carefully in patients with PAH are:
Mean arterial blood pressure (65-75mm Hg)
Heart rate (70-100)
Rhythm (sinus rhythm)
Saturation

Not only hemodynamics, adequate analgesia also plays a vital role as decreasing the pain can decrease the increase in systemic vascular resistance and prevent variable changes in PA pressure. In this case we have used femoral and sciatic nerve block as the preferred mode of analgesia technique which decreased the requirement of opioids. Regional anaesthesia like spinal or epidural can be given but there are high chances of hypotension in these patients to which the right ventricle becomes intolerant.

The changes of tourniquet inflation and deflation are also to be noticed by the anaesthesiologist. Due to multiple changes due to tourniquet, proper analgesia and inodilators can be used to prevent the rise of PA pressure. Thus the main goal in the perioperative period is

1. to maintain hemodynamics
2. adequate analgesia
3. adequate hydration
4. preventing blood loss
5. perioperative lung ultrasound and echocardiography to guide fluid therapy
6. maintain the rhythm and contractility of the heart
7. maintaining oxygenation.
8. Avoiding excessive opioids due to risk of hypoventilation which can increase PA pressure

CONCLUSION

PAH is one of the deadliest diseases involving both cardiac and respiratory system. Anaesthetic management not only includes maintaining all parameters within normal limits but also preventing rise in PA pressure. The anaesthesiologist must be vigilant in diagnosing these kinds of situations in the pre operative period and be successful in optimising pre operatively. Adequate point of care lung ultrasound and echocardiography plays a vital role in preventing any untoward complications. Proper monitoring of hemodynamic during induction and laryngoscopy should be done because the choice of the inducing agent and also the time of laryngoscopy plays an important role in affecting hemodynamics.

In our case we specifically used etomidate to prevent significant changes and avoided use of nitrous oxide during maintenance. The main goal during the maintenance time is to prevent severe change in hemodynamics due to high risk of right ventricular failure. Fluid therapy should be given based on the IVC diameter and central venous pressure. Opioid free anaesthesia should be given. This includes the use of peripheral nerve block techniques. In our case we used femoral and sciatic nerve block technique. This decreased the acute rise of systemic vascular resistance and also resulted in decreased opioid requirement in the post operative time period. Inotrope should be started to maintain hemodynamics as liberal fluid strategy results in pulmonary oedema.

The anaesthesiologist must be vigilant in maintaining the hemodynmaics of the patient. Proper weaning of the patient is also required to prevent pulmonary atelectasis. Thus multiple factors are to be kept in mind while managing a patient of PAH.

REFERENCES

[1] Subramaniam K, Yared JP. Management of pulmonary hypertension in the operating room. Semin Cardiothorac Vasc Anesth. 2007 Jun;11(2):119-36
[2] ilkington SA, Taboada D, Martinez G. Pulmonary hypertension and its management in patients undergoing non-cardiac surgery. Anaesthesia. 2015 Jan;70(1):56-70.
[3] Pritts CD, Pearl RG. Anesthesia for patients with pulmonary hypertension. Curr Opin Anaesthesiol. 2010 Jun;23(3):411-6.
[4] McGlothlin D, Ivascu N, Heerdt PM. Anesthesia and pulmonary hypertension. Prog Cardiovasc Dis. 2012 Sep-Oct;55(2):199-217.
[5] Green JB, Hart B, Cornett EM, Kaye AD, Salehi A, Fox CJ. Pulmonary Vasodilators and Anesthesia Considerations. Anesthesiol Clin. 2017 Jun;35(2):221-232.
[6] Minai OA, Yared JP, Kaw R, Subramaniam K, Hill NS. Perioperative risk and management inpatients with pulmonary hypertension. Chest. 2013 Jul;144(1):329-340.
[7] Kim D, George MP. Pulmonary Hypertension. Med Clin North Am. 2019 May;103(3):413-423.
[8] Ashes C, Roscoe A. Transesophageal echocardiography in thoracic anesthesia: pulmonary hypertension and right ventricular function. Curr Opin Anaesthesiol. 2015 Feb;28(1):38-44.