



UNEVALUATED SEVERE PULMONARY ARTERIAL HYPERTENSION: MONITORED ANESTHESIA CARE FOR EMERGENCY CHRONIC SUBDURAL HYGROMA DRAINAGE USING SCALP BLOCK AND DEXMEDETOMIDINE - A CASE REPORT

Mahajan Shalvi

MD, DM, Attending Consultant Fortis Hospital Mohali, Punjab

Loganathan Sekar*

Senior Resident, Post Graduate Institute of Medical Education and Research, Chandigarh, India *Corresponding Author

ABSTRACT Irrespective of etiology pulmonary artery hypertension (PAH) is a complex medical condition. Non cardiac surgeries in patients with PAH have high perioperative morbidity and mortality. Before any elective surgical procedure in patients with PAH - anesthesia, cardiology, pulmonology and surgical teams assess and optimize patients. But during emergency surgical intervention preoperative optimization may not possible. Hence, anesthesiologist always play lead role in management of these cases during surgery using techniques to minimize further increase in pulmonary artery pressures. Here we report a successful management of emergency burr hole evacuation of chronic subdural hygroma in a young male with unevaluated severe pulmonary artery hypertension using scalp block along with dexmedetomidine infusion.

KEYWORDS : Pulmonary artery hypertension, Scalp block, Dexmedetomidine

INTRODUCTION:

Pulmonary artery hypertension (PAH) also known as pulmonary hypertension, is a rare, disease compounded with significant morbidity and mortality. PAH is an independent risk factor for perioperative complications including life threatening arrhythmias, myocardial infarction, right heart failure, even sudden cardiac death in elective non-cardiac surgeries. Hence it warrants pre-operative optimization.(1) During induction of anesthesia, it poses a significant challenge to the anesthesiologist complicated by hemodynamic compromise which manifests as pulmonary hypertensive crisis.(2) Here we present a case of chronic subdural hygroma with features of increased intracranial tension (ICP) diagnosed to have PAH in pre-anesthesia check-up. Emergency burr-hole drainage for the same was done under monitored anesthesia care (MAC) with scalp block-assisted intravenous dexmedetomidine infusion without complications.

CASE REPORT:

A 19 years old male weighing about 80 kg presented to the emergency neurosurgical outpatient department in the midnight with complaints of headache and vomiting for last 10 days. On examination except for mild proptosis of right eye, all were normal. Non-Contrast Computed Tomography (NCCT) head was done and he was found to have right frontal and fronto-tempero-parietal (FTP) hypodensity with midline shift which was suggestive of subdural hygroma. All routine investigations were done. Burr hole and drain of the hygroma was planned. Patient was shifted to pre-operative room for pre-anesthesia checkup (PAC). During PAC we found a significant history of decrease in the effort tolerance measured in terms of Metabolic Equivalents of Task (METS) which was less than 4. Patient and his family attributed this decrease in effort tolerance to his weight 80kg. His hemoglobin was 16.8g%. Blood urea nitrogen (BUN), serum creatinine, serum electrolytes, platelets, coagulogram were within normal range. Chest x-ray showed enlarged right atrium (RA), prominent aortic knuckle and suspected right ventricle (RV) forming the apex of the heart. Electrocardiogram (ECG) showed ST-segment depression with T wave inversion in leads II, III, aVf and V1-5 with a background heart rate of 91/min. The features suggestive of right ventricular hypertrophy (RVH), and right atrial abnormality (RAA) were also seen in ECG. However, the cardiovascular system by clinical examination was normal.

Emergency cardiology consultation and echocardiography (ECHO) was sought prior to the surgery. Echocardiography showed mild Tricuspid regurgitation (TR), RVH, enlarged right atrium (RA), right ventricle (RV). The left ventricle (LV) was D-shaped with visual ejection fraction- 55-60%. Right ventricular systolic pressure (RVSP) was right atrial pressure (RAP)+70, which was suggestive of severe PAH. The cause for the PAH was not evident in ECHO and patient was taken for emergency surgery under moderate to high risk. Further follow up for PAH was planned after the surgical procedure.

The risks related to the anesthesia were discussed with the

neurosurgical team and explained to the patient relatives. We planned scalp block-assisted with intravenous dexmedetomidine infusion for the procedure, avoiding tracheal intubation and positive pressure ventilation. Electrocardiogram (ECG), non-invasive blood pressure (NIBP), pulse oximetry (SPO2) and temperature probe were attached. An intravenous access was secured. An arterial line was placed in right radial artery under ultrasound guidance after two failed attempts. During arterial cannulation we ensured that there was no/minimal pain by giving lignocaine local anesthesia prior to cannulation. Base line NIBP was 148/80 mmHg, pulse rate- 101/min. Pulse oximetry recorded 97% oxygen saturation at room air. Oxygen supplementation was given via venturi mask and end tidal carbon dioxide (EtCO2) was monitored. Before induction he was pre-medicated with injection midazolam 1mg and ondansetron 8 mg intravenously. Patient was induced with dexmedetomidine at 1mcg/kg over 10 minutes and reduced to 0.5mcg/kg/hour with injection phenylephrine (100:1) bolus as a rescue drug. Right side scalp block was given using bupivacaine 0.5% plain total of 14 ml. There was no movement and tachycardia during incision and throughout the procedure. The lowest NIBP recorded was 126/62 mmHg and the heart rate were 74/min during the surgery and phenylephrine bolus was not used. There was no episode of oxygen desaturation. Temperature was maintained throughout procedure. Burr hole evacuation was successful without any complications.

DISCUSSION:

PAH is defined as abnormal elevation of mean pulmonary artery pressure (mPAP) above 25mmHg with normal pulmonary capillary wedge pressure (PCWP) of less than 15mmHg.(3) Right heart catheterization is required for confirming the diagnosis of PAH along with transesophageal echocardiography (TEE) for evaluating underlying cause. Etiology of the disease is diverse includes cardiac, pulmonary and/or intrinsic vascular diseases. Irrespective of the etiology, PAH indicates advanced stage of the disease.(4) PAH itself and anesthesia administration for the patients with PAH have a high cardiovascular risk for morbidity and mortality in the peri-operative period. In our case we do not know the etiology for the PAH. The patient was operated on the emergency basis since NCCT head showed midline shift with the associated lesion and he had signs of elevated ICP. Hence cardiac catheterization and further workup for PAH was postponed. It poses a greater challenge to even experienced anesthesiologists at middle of the night without proper preoperative optimization to conduct such case.

Our ultimate aim during conduct of procedure was to minimize the increase in PAP so as to prevent pulmonary hypertensive crisis. Pulmonary hypertensive crisis can be triggered by hypoxia, hypercarbia, hypothermia, acidosis, pain and stress.(2) Maintenance of systemic vascular resistance (SVR) was carried out by maintaining systemic blood pressure and mean arterial pressure (MAP) thus avoiding the development of acute right ventricular failure leading to decrease in cardiac output (CO) and cardiac arrest. Sudden rise in PAP during tracheal intubation even in anesthetized patients may

precipitate oxygen desaturation and acute right ventricular failure.(5) Intravenous inducing agents, inhaled anesthetics significantly decreases systemic vascular resistance and are harmful.(2,5) Thus regional anesthesia technique involving scalp block was preferred in our case.

PAH is associated with significant hemodynamic compromise during anesthesia and surgical stimuli hence arterial cannulation was done. There were two failed attempts during arterial cannulation since skin was thick enough to shear the canula tip. Collagen vascular diseases like systemic sclerosis presents with PAH at later stage in which skin thickening is a major sign and that should be considered in the differential diagnosis in this case.(6) Fluid management following induction of anesthesia is a challenge because the determination of volume status from hemodynamic parameters such as systolic pressure variation and pulse pressure variation is not reliable in patients with PAH and with application positive pressure ventilation⁽⁷⁾ Transesophageal echocardiography (TEE) for monitoring RV function is helpful with limitation of user experience and availability. Nerve block technique holds best role in these situations whenever feasible. Intravenous sedatives and analgesics integrated with block techniques are good option in this respect.

Dexmedetomidine is a sedative-anxiolytic and holds amnestic property.(8) It has potent analgesic property as well reduces requirements of opioids.(9) All these properties of novel drug accounted to its highly selective alpha-2 agonistic activity in brain and spinal cord. On a dose dependent manner, it decreases sympathetic outflow from the central nervous system with side effects which are clinically beneficial. Though it was initially approved for intubated patients in intensive care unit for sedation, respiratory depression is not seen with this drug.(10) The amnestic property is comparable with benzodiazepines but it does not produce confusion at emergence.(11, 12) Its sympatholytic properties can be reversed with atipamezole.(13) Hence it holds the whole property of being a total intravenous anesthetic agent with safer profile in PAH along with regional and general anesthesia techniques.(14)

To conclude, perioperative management of patients with PAH revolves around the triad of detailed preoperative evaluation and optimization by multidisciplinary team, cautious intraoperative management to prevent and treat pulmonary hypertensive crisis and intensive care in postoperative period. Initial preoperative component is missing in emergency noncardiac surgeries. Hence regional anesthesia with scalp block in neurosurgical emergencies with dexmedetomidine infusion provides an alternative anesthesia technique with minimum hemodynamic disturbances.

REFERENCES:

1. Minai OA, Yared JP, Kaw R, Subramaniam K, Hill NS. Perioperative risk and management in patients with pulmonary hypertension. *Chest*. 2013;144(1):329-40.
2. Schisler T, M Marquez J, Hilmi I, Subramaniam K. Pulmonary Hypertensive Crisis on Induction of Anesthesia: Recommendations for Safe Practice 2016.
3. Galiè N, Humbert M, Vachiery J-L, Gibbs S, Lang I, Torbicki A, et al. 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Heart and Lung Transplantation (ISHLT). 2015;46(4):903-75.
4. Montani D, Günther S, Dorfmueller P, Perros F, Girerd B, Garcia G, et al. Pulmonary arterial hypertension. *Orphanet journal of rare diseases*. 2013;8:97.
5. Fischer LG, Van Aken H, Burkle H. Management of pulmonary hypertension: physiological and pharmacological considerations for anesthesiologists. *Anesthesia and analgesia*. 2003;96(6):1603-16.
6. Khanna D, Denton CP. Evidence-based management of rapidly progressing systemic sclerosis. *Best practice & research Clinical rheumatology*. 2010;24(3):387-400.
7. Shinohara H, Hirota K, Sato M, Kakuyama M, Fukuda K. Monitored anesthesia care with dexmedetomidine of a patient with severe pulmonary arterial hypertension for inguinal hernioplasty. *Journal of anesthesia*. 2010;24(4):611-3.
8. Rozet I. Anesthesia for functional neurosurgery: the role of dexmedetomidine. *Current opinion in anaesthesiology*. 2008;21(5):537-43.
9. Gurbet A, Basagan-Mogol E, Turker G, Ugun F, Kaya FN, Ozean B. Intraoperative infusion of dexmedetomidine reduces perioperative analgesic requirements. *Canadian journal of anaesthesia*. 2006;53(7):646-52.
10. Venn RM, Hell J, Grounds RM. Respiratory effects of dexmedetomidine in the surgical patient requiring intensive care. *Critical care (London, England)*. 2000;4(5):302-8.
11. Lepouse C, Lautner CA, Liu L, Gomis P, Leon A. Emergence delirium in adults in the post-anaesthesia care unit. *British journal of anaesthesia*. 2006;96(6):747-53.
12. Hauber JA, Davis PJ, Bendel LP, Martyn SV, McCarthy DL, Evans MC, et al. Dexmedetomidine as a Rapid Bolus for Treatment and Prophylactic Prevention of Emergence Agitation in Anesthetized Children. *Anesthesia and analgesia*. 2015;121(5):1308-15.
13. Scheinin H, Aantaa R, Anttila M, Hakola P, Helminen A, Karhuvaara S. Reversal of the sedative and sympatholytic effects of dexmedetomidine with a specific alpha2-adrenoceptor antagonist atipamezole: a pharmacodynamic and kinetic study in healthy volunteers. *Anesthesiology*. 1998;89(3):574-84.
14. Ramsay MA, Luteran DL. Dexmedetomidine as a total intravenous anesthetic agent. *Anesthesiology*. 2004;101(3):787-90.