ORIGINAL RESEARCH PAPER

Anaesthesiology

KEY WORDS:
Intraoperatively diagnosed paraganglioma, catecholamine induced pulmonary oedema, difficult case.

INTRAOPERATIVELY DIAGNOSED CATECHOLAMINE SECRETING MASS: A CASE REPORT.

Puneet Verma*

MD, Anaesthesiologist. District Hospital, Mandi, Himachal Pradesh-175001.
*Corresponding Author

ABSTRACT

I hereby present a case of an asymptomatic patient whose catecholamine secreting tumor was diagnosed intraoperatively due to acute high increase in blood pressure on tumor manipulation, thought to be arising from pancreas. The fall in blood pressure was also present on removal on mass. The transient increase in catecholamines also led to pulmonary oedema which was managed accordingly. The fluctuations of blood pressure and heart rate in line with blood catecholamine levels along with other complications like arrhythmias and pulmonary oedema in such cases provide unique challenge to anesthesiologists.

INTRODUCTION

Pheochromocytomas and paragangliomas are catecholamine secreting tumours derived from the sympathetic or parasympathetic nervous system. The clinical presentation varies from an adrenal incidentaloma to a patient in hypertensive crisis with associated cardiovascular complications; the most challenging cases however are those diagnosed intraoperatively by release of catecholamines. The lack of preoperative preparation of the patient as well as the anaesthesia provider can set the stage for a clinical nightmare.

CASE REPORT

A 40 yr old, 55 kg female patient was scheduled for the removal of an intra-abdominal mass, suspected to be located in the tail of the pancreas as per the CT report. Routine preoperative examination was normal and her preoperative vitals were recorded as heart rate 96/min and blood pressure 130/80 mmHg. Her investigations like haemogram, renal functions, chest x ray and ECG were within normal limits. An informed consent was obtained and patient was premedicated with 0.5 mg Tablet Alprazolam on night before surgery.

The patient was administered combined spinal and epidural anaesthesia in the lateral position in T12L1 interspace. She was given 12.5 mg of hyperbaric bupivacaine intrathecally followed by immediate activation of epidural catheter with 10 ml of 2% lignocaine with adrenaline (1:200000), to achieve neuraxial blockade upto T6. The patient was given intravenous sedation with midazolam 1 mg and fentanyl 100µg and oxygen supplementation was done. A midline vertical incision upto T8 dematome was made and when tumour manipulation was done, the patient complained of headache and unpleasant feeling along with rapid rise in blood pressure (230/140 mmHg) and heart rate (180/min). This hypertensive crisis was managed with injection esmolol given in boluses of 30 mg (total of 150 mg needed) to reduce the blood pressure to 165/95 mmHg and heart rate around 120/min. Also injection loxicard 100mg intravenous was given as premature ventricular contractions started appearing.

The patient was immediately intubated using a cuffed orotracheal tube of size 7.5 mm under injection 150 mg of propofol and 6 mg of vecuronium to deepen the plane of anaesthesia and blunt airway reflexes to prevent further release of catecholamines. Anaesthesia was maintained with ventilation with oxygen and nitrous oxide in a ratio of 33% and 66% and isoflurane (1-2.5%) along with intermittent boluses of vecuronium.

Invasive monitoring was instituted using central venous pressure (CVP) and invasive blood pressure. Before this event, conventional monitoring was in place with ECG, non invasive blood pressure, pulse oximetry and end tidal CO2(after intubation). After resection of the mass the patient developed hypotension which was managed by CVP guided crystalloids and titrated phenylephrine infusion in view of persistent tachycardia. On desaturation, she was ventilated with 100% oxygen and auscultation of the chest revealed bilateral extensive crepitations along with appearance of pink frothy secretions in the endotracheal tube, suggesting a diagnosis of pulmonary oedema.

The patient was given 20 mg furosemide and 9 mg of morphine intravenously while continuing positive pressure ventilation with 100% O2, maintaining blood pressure with phenylephrine infusion. The patient was shifted to the ICU for further observation at the end of surgery and extubated 12 hours later. Her ABG report showed improvement from intraoperative pO2 of 68mmHg and pH of 7.23 to 102mmHg and 7.36 respectively, at time of extubation.

Microscopic examination of the mass revealed cells in Zellballen pattern with abundant granular amphophilic cytoplasm, thereby confirming clinical diagnosis.

DISCUSSION

The actual prevalence of pheochromocytoma is about 0.2%[1] but the diagnosis is made in only about half of these patients[2]. The prevalence of incidental catecholamine secreting tumours (pheochromocytoma and paraganglioma) on operating table, is unknown but are known to have a mortality rate of upto 40%.[3] On the other hand the perioperative mortality for elective pheochromocytoma surgery approaches near 0% due to better preparation.[4]

Our patient was asymptomatic preoperatively and only the development of an intraoperative hypertensive crisis, due to manipulation of the tumour aroused a suspicion. The management of such hypertensive crisis (defined as SBP>180 and/or DBP>120)[5], as in previously diagnosed cases, comprises the use of beta blockers and/or nitrates with limited use of alpha blockers.[6] The short acting drug esmolol is a widely accepted drug in such a situation which consists of hypertension and tachycardia; also as hypotension following the removal of the mass, can be compounded by longer acting drugs. In our case, the excessive tachycardia (HR around 180/min) precludes the use of nitrates leaving beta blockers as the acceptable option.

Another challenging aspect in such cases is the catecholamine induced pulmonary oedema, as occurred in our patient. This occurs due to increased pulmonary vascular resistance[7], leading to increased left ventricular diastolic pressures and pulmonary congestion. It further impairs cardiac contractility thus worsening the pathology. The excessive adrenaline is also known to cause toxic lung injury which could be an alternative mechanism.[8]

The intraoperative management of the developing pulmonary

oedema includes positive pressure ventilation with 100% O2, propped up position, use of diuretics and morphine. Current evidence is in favour of use of nitrates as they reduce both preload and afterload and simultaneously do not increase myocardial oxygen demand.[9]

Morphine acts on preload as well as afterload and is preferred in patients who are planned for elective ventilation and ICU observation. Our patient was electively ventilated for 12 hours as she was already intubated for the surgery, and gas exchange was impaired as seen in the ABG report.

Patients with intraoperatively diagnosed catecholamine secreting tumour are at increased risk of postoperative hypotension and need for inotropic support due to a lack of preoperative optimisation and less likelihood of a curative. Drugs like dopamine, phenylephrine and norepinephrine may be required.[6]

The need to maintain euvolemia can only be overemphasised so as to strike a fine balance between the extremes of pulmonary oedema and hypotension.

Thus, constant vigilance and rapid corrections of sudden variations, with quick thinking and aggressive management, can lead to favourable outcome in these potentially dangerous cases, which despite advancements in diagnostic aids do occur in operation theatres.

REFERENCES

1. Neumann Hartmut P.H. Pheochromocytoma . In: Longo Dan L, Fauci AS, Kasper DL. Harrison's Principles of Internal Medicine, 18th edn. New York: McGraw Hill Companies Inc; 2011. P. 2962-7.
2. Anderson GH, Blakeman N, Streetan DH. The effect of age on prevalence of secondary forms of hypertension in 4429 consecutively referred patients. J Hypertens 1994; 12: 609-15.
3. Sutton MG, Sheps SG, Lie JT. Prevalence of clinically unsuspected pheochromocytoma. Review of a 50-year autopsy series. Mayo Clin Proc 1981; 56: 354-60.
4. Van Braeckel P, Carlier S, Steelant PJ, Weyne L, Vanfleteren L. Perioperative management of pheochromocytoma. Acta Anaesthesiol Belg 2009; 60: 55-66.
5. Fontes ML, Varon J. Perioperative hypertensive crisis: newer concepts. Int Anaesthesiol Clin 2012; 50: 40-58.
6. Roizen Michael F, Fleisher Lee A. Anaesthetic implications of concurrent diseases. In: Miller Ronald D, Eriksson Laes I, Fleisher Lee A. Miller's Anaesthesia, 7th edn, P 1084-85.
7. Ersoz N, Finestone SC. Adrenaline-Induced Pulmonary Oedema and its treatment. A report of two cases. Survey of Anaesthesiology 1972; 16(3): 216.
8. Wang NS, Huang SN, Sheldon H, Thurlbeck MB. Ultrastructural Changes of Clara and type II alveolar cells in adrenaline-induced pulmonary edema in mice. Am J of Path 1971; 62(2): 237-45.
9. Beltrame JF, Zeitz CJ, Unger SA. Nitrate therapy is an alternative to furosemide/morphine therapy in the management of acute cardiogenic pulmonary edema. J Card Fail 1998; 4: 271–9.