

Anesthetic management of adrenalectomy: a case report

Abstract

Pheochromocytomas are catecholamine secreting tumours arising from the chromaffin cells of sympathoadrenal system. The patients usually present with hypertension, palpitation and sweating but may have varied presentation as well. Surgery is the definitive treatment of pheochromocytoma both to control the symptoms and more importantly to prevent further complications of hypertensive crisis, particularly the cerebrovascular and cardiovascular events. The laparoscopic adrenalectomy via transperitoneal or retroperitoneal approach is preferred due to advantages of laparoscopy over the open approach. The anesthetic management of such surgeries becomes challenging due to the potential for perioperative hemodynamic instability.

Keywords: pheochromocytoma, adrenalectomy, anesthesia

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Case report

Introduction

A 43years old female patient presented with complaints of headache, palpitation and sweating. Her laboratory investigation reports were in normal range. CT scan of abdomen confirmed the presence of a mass in right supra-renal area. She was provisionally diagnosed as a case of pheochromocytoma and then planned for adrenalectomy.

Preoperative period

History: She had history of episodic attacks of headache associated with palpitation, sweating, nausea and vomiting, lasting for few minutes to few hours in duration, since five years. She was then found to have hypertension and so was prescribed with Amlodipine 5mg and losartan 50mg once daily. She also gives history of lacunar infarction associated with weakness of right arm, three years ago. At that time clopidogrel 75mg, metoprolol 25mg and rosuvastatin 10mg were added to her daily medication. She had physically active lifestyle with metabolic equivalent (METS)>4.

Physical examination: on examination the patient was found to be conscious, well oriented and comfortable. Her weight was 65kg with body mass index(BMI) 32. Airway assessment revealed Mallampati grade II and appropriate neck mobility. Her pulse was 76/minute, blood pressure 110/70mmHg (supine), 100/70mmHg (standing) and SPO2 97% on room air. Rest of the examination was unremarkable.

Investigations: Complete Blood Count, random blood sugar, renal function test, liver function tests, coagulation profile were within normal range. 24hours urine collection sample measured urine Vanillylmandelic acid= 4.8 mg and metanephrine 94.5mcg/24hr. Electrocardiogram (ECG) recorded sinus rhythm and echocardiogram recorded mild mitral regurgitation with ejection fraction of 65%. CT scan of abdomen revealed 3cm x 3cm mass in right suprarenal area.

She was provisionally diagnosed as a case of pheochromocytoma and accordingly scheduled for open adrenalectomy of right side, under combined general and epidural anesthesia.

Preoperative medications

- Amlodipine 5mg, losartan 50mg, OD, since 5years
- clopidogrel 75mg, metoprolol 25mg, rosuvastatin 10mg, OD, since 3years
- Prazosin 1mg BD since 3weeks.

Clopidogrel was stopped 1week prior to operation and losartan was stopped 24hours before operation while continuing amlodipine, metoprolol and prazosin until morning of surgery. Lorazepam 2mg was added the night before.

Intraoperative period: Besides regular preparation, following drugs were made ready to use as needed: nitroprusside, nitroglycerin, phenylephrine and noradrenaline infusions. Esmolol, adrenaline, magnesium sulphate and labetalol were also placed within easy reach. American Society of Anesthesiologists(ASA) standard monitors (ECG, NIBP, SPO2) were connected and displayed HR 78/min, BP 124/82 mmHg and SPO2 98%. 16G IV canula was inserted on left hand and ringer's lactate infusion started. Midazolam 2mg and fentanyl 25 mcg injected IV followed by epidural catheter placement at T8-9 level and epidural test dose given. Right radial artery was cannulated with 20G canula for continuous intraarterial blood pressure monitoring. General anesthesia was induced with 120mg Propofol, 100mcg fentanyl, 60mg lignocaine and 50 mg rocuronium followed by smooth laryngoscopy and quick intubation with 7.5mm cuffed endotracheal tube. Triple lumen central venous catheter was used for ultrasound guided right internal jugular vein cannulation. One lumen was dedicated for continuous central venous pressure (CVP) monitoring and other two lumen for various medications as per need. 5ml of 1.5% lignocaine was injected epidural bolus, followed by epidural infusion (0.1% bupivacaine +2mcg/ml fentanyl) @4-5 ml/hr. Isoflurane was used as inhalation agent to maintain surgical anesthesia. At this time, GRBS was recorded 74mg%, so 500ml of DNS was infused. About 2 litre of crystalloid was infused before ligation of tumour. CVP was stable at 13-15mmHg and urine output around 1.5ml/kg throughout the operation which took 2hours 20minutes. The hemodynamic status was stable without need for any vasoactive drugs. At the end of the operation, patient was extubated and found to be alert, pain free and

obeying commands. ABG analysis was in normal range and GRBS 161mg%. The mass was sent for histopathology confirmation.

Postoperative period: She was shifted to ICU for close monitoring. Postoperative analgesia was maintained with 1 gm paracetamol 6 hourly and continuous epidural infusion for 4days. The hemodynamic status was within acceptable range without need for any vasoactive drugs. She was shifted to surgical ward on 5th postoperative day. At that time her blood pressure was persistently recorded to be 140-150/90-95mmHg; so amlodipine 5 mg OD started and patient discharged from hospital on 9th postoperative day with followup advice.

Discussion

Pheochromocytomas are catecholamine secreting tumours arising from chromaffin cells of the sympathoadrenal system. 80-85% of them are located in adrenal medulla, more often on the right side. Extraadrenal tumours may be found anywhere along sympathetic ganglia, the most common site being the organ of Zuerkandl near aortic bifurcation.^{1,2} They account for about 0.2-0.6% of all cases of hypertension in adults.³

They secrete catecholamines, mostly norepinephrine (roughly norepinephrine: epinephrine=85:15), the inverse of normal adrenal secretion (epinephrine: norepinephrine=85:15) and dopamine. Classic signs and symptoms include paroxysmal or persistent hypertension associated with headache, sweating and palpitation; lasting from few minutes to few hours. Hemodynamic picture depends on the predominant catecholamine secreted i.e. with norepinephrine α adrenergic effect of systolic and diastolic hypertension associated with bradycardia while with epinephrine β effects of systolic hypertension and tachycardia.⁴ Indeed these patients are at risk of cerebrovascular and cardiovascular events. The clinical presentation of our patient was suggestive of pheochromocytoma.

Diagnosis is usually confirmed by biochemical and radiological investigations. 24hours urine collection sample shows rise in metanephrines and vanillylmandelic acid. As metanephrines are produced continuously within the tumour cells, measurements of plasma free metanephrines and urinary fractionated metanephrines are highly sensitive diagnostic tests. Some patients with paroxysmal hypertension may have normal catecholamine values between the attacks.^{4,5} CT, Magnetic Resonance Imaging (MRI) and Metaiodobenzyl guanidine (MIBG) are very useful for the localization of tumour. ECG and echocardiogram may reflect features of cardiac ischemia or cardiomyopathy. In our patient, although the CT scan suggested right adrenal mass; the biochemical tests were not in favour of pheochromocytoma.

Adequate preoperative preparation with α blockers is traditionally given the credit of reducing perioperative mortality from 45% to 0-3%.² These drugs also decrease the complications of hemodynamic instability particularly at the time of induction, intubation, surgical incision and tumour manipulation. Roizen et al.,⁷ proposed a set of criteria⁷ (now called the Roizen criteria) to objectively gauge the efficacy of adequate preoperative α blockade as:

- No in-hospital BP > 160/90mmHg for 24 hrs prior to surgery
- No orthostatic hypotension with BP < 80/45 mmHg
- No ST-T changes on ECG for a week prior to surgery
- No more than 5 premature ventricular contractions (PVCs)/minute

The objectives of preoperative preparation⁸ include

- Control of blood pressure
- Reversal of chronic intravascular volume depletion
- Control of arrhythmia and heart rate
- assessment & optimization of myocardial function
- reversal of glucose & electrolyte disturbances

Hypertension in these patients may be paroxysmal with baseline normal blood pressure; baseline elevated blood pressure with intermittent paroxysms or persistently high blood pressure. As the primary cause of hypertension is α stimulation by excessive catecholamines; the α receptor antagonists become the first choice to control blood pressure.⁹ α blocker controls blood pressure and also aids in expanding the highly contracted intravascular volume.¹⁰ Other antihypertensives can be used as adjuncts such as calcium channel blocker, β blocker, ACEI or angiotensin receptor blocker (ARB). Phenoxybenzamine; a nonselective, irreversible α blocker had been widely used, starting 10-14days prior to operation.^{1,4} α_2 blockade results in tachycardia while its irreversible nature and prolonged duration of action is responsible for undesirable significant hypotension following tumour ligation. Selective α_1 receptor antagonist (prazosin, doxazosin and terazosin) is preferred as α_2 activity remains intact, thus, avoiding severe tachycardia.¹¹ The reversible α_1 antagonism of relatively shorter duration is further beneficial in reducing the risk of hypotension following ligation. These are associated with better control of preoperative blood pressure, adequate restoration of intravascular volume and less chances of significant hypotension following tumour ligation. It has been suggested that α blockade for more than two weeks usually restores the blood volume. β blocker should never be used before α blockade as unopposed α activity may result in life threatening hypertensive crisis.¹² β blocker is indicated to counteract the tachycardia induced by nonselective α blocker, tachyarrhythmias or catecholamine induced cardiomyopathy. Cardioselective β_1 blocker is commonly prescribed. Labetalol (blocks both α and β receptors) may be used but never as an alternative to α blocker.¹³

Calcium channel blockers are useful adjunct to α blocker particularly in patients with episodic attacks. ACEI, ARB and magnesium sulphate have also proved to be useful. The Endocrine Society Clinical Practice Guidelines recommend a high salt and fluid intake to reverse the catecholamine induced intravascular volume contraction preoperatively and to prevent severe hypotension that may be encountered after tumour ligation intraoperatively.³

Ideally these patients are managed with a combination of epidural and general anesthesia. Besides the standard ASA monitors, invasive arterial blood pressure monitoring is done, to immediately diagnose and manage hemodynamic fluctuations. Multi-lumen central venous catheter is very useful as an access to vascular compartment. CVP is usually maintained around 12-15mmHg as excessive fluid administration may lead to pulmonary edema.

Intraoperative hemodynamic instability presents as hypertension during various phases of surgery before tumour ligation and as hypotension following the ligation. Excessive rise in blood pressure is seen with norepinephrine release while epinephrine is responsible for significant tachyarrhythmia and hypertension.⁸ Acute hypertension must be treated with nitroprusside or nitroglycerin while esmolol with

a vasodilator is effective in a setting of tachycardia and hypertension. Our patient did not have any significant hemodynamic turbulence, which may be because of non secreting nature of the mass. It has been suggested that the patient must receive about 2-3litre of crystalloid before tumour ligation as sudden withdrawal of catecholamines may precipitate hypotension and hypoglycemia. Dopamine and norepinephrine infusion are usually required to manage hypotension. Vasopressin is useful in refractory hypotension as its pressor effect doesn't depend on adrenergic receptors. 1-2mg/kg methylene blue may also be considered in refractory hypotension, which acts by inhibiting cGMP. Blood sugar must be checked frequently and managed accordingly, atleast for next 24hours. Most of the patients can be extubated at the end of surgery and need close monitoring in SICU during immediate postoperative period. Our patient had received selective α blockade from 3weeks prior to operation together with β blocker and amlodipine. Intravascular volume was well maintained intraoperatively with cvp 12-15mmHg and urine output nearly 1.5ml/kg/hour.

Conclusion

Surgery is the definitive treatment for pheochromocytoma as most of the patients become symptom free. More important is the fact that it prevents further complications of catecholamine excess. Adequate preoperative patient preparation with α blockade and intravascular volume restoration, meticulous preparation of various vasoactive drugs, close monitoring, communication with surgical team, anticipation and immediate management of hemodynamic fluctuations are vital to minimize perioperative morbidity and mortality. In spite of suggestive history, our patient had normal biochemical investigations and did not have significant perioperative hemodynamic instability. Whether secretory or not; all patients of adrenal mass should be considered potentially dangerous and thus planned accordingly. The final diagnosis is obviously confirmed by histopathology.

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None

Conflict of interest

Author declares there is no conflict of interest towards this manuscript

References

1. Michael F, Roizen, Lee A Fleisher. Anesthetic Implications of concurrent diseases. Chapter 35, Miller's Anesthesia. Ronald D Miller, editor, 7th ed. New York: Churchill Livingstone, Elsevier; 2010. p. 1084–1085.
2. Bravo EL. Pheochromocytoma. Hines, Marschall, editors. Stoelting's Anesthesia and Co-existing Diseases. 5th ed, Churchill Livingstone, An Imprint of Elsevier; Philadelphia, 978-1-4160-3998-3.
3. Lenders JW, Duh QY, Eisenhofer G, et al. Pheochromocytoma and paraganglioma. An Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab.* 2014;99(6):1915–1942.
4. Jeffrey J. Schwartz, Shamsuddin Akhtar, et al. Rosenbaum. Endocrine Function, chapter 49 Clinical Anesthesia. Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC, editors. 7th ed. Philadelphia: Wolters Kluwer, Lippincott Williams & Wilkins; 2013. p. 1339–1342.
5. Woodrum DT, Khetarpal S. Anesthetic management of Pheochromocytoma. *World J Endocrine Surg.* 2010;2(3):111–117
6. Lenders J, Pacak K, Walther MM, et al. Biochemical diagnosis of pheochromocytomas. *JAMA.* 2002;287(11):1427–1434.
7. Roizen MF, Horrigan RW, Koike M, et al. A prospective randomized trial of four anesthetic techniques for resection of pheochromocytoma. *Anesthesiology.* 1982;57:A43.
8. Connor D Boumphrey S. Perioperative care of pheochromocytoma. *BJA Education.* 2016;16(5):153–158.
9. Ramachandran R, Rewari V. Current perioperative management of pheochromocytomas. *Indian J Urol.* 2017;33(1):19–25.
10. Ramakrishna H. Pheochromocytoma resection: current concepts in anesthetic management. *J Anaesthesiol Clin Pharmacol.* 2015;31:317–323.
11. Agarwal R, Mishra SK, Bhatia E, et al. Prospective study to compare perioperative hemodynamic alterations following preparation for pheochromocytoma surgery by phenoxybenzamine or prazosin. *World J Surg.* 2014;38(3):716–723.
12. Sibal L, Jovanovic A, Agarwal SC, et al. Pheochromocytoma presenting as acute crisis after beta blockade therapy. *Clin Endocrinol.* 2006;65(2):186–190.
13. Pacak K. Preoperative management of the pheochromocytoma patient. *J Clin Endocrinol Metab.* 2007;92(11):4069–4079.