

USE OF VASOPRESSIN TO TREAT CATECHOLAMINE RESISTANT REFRACTORY HYPOTENSION AFTER RESECTION OF MASSIVE PHEOCHROMOCYTOMA: A CASE REPORT

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Abstract

We present a case of massive pheochromocytoma planned for surgical resection. The case was taken up for surgery after preoperative catecholamine blockade and intravascular volume resuscitation. The major challenge was profound hypotension after tumour resection which was refractory to catecholamine therapy but responded successfully to the application of vasopressin therapy

Introduction

Anaesthetic management of pheochromocytoma has always been a subject of interest. Acute variations in serum catecholamine levels may present as hypertensive or hypotensive crises, depending on tumour type and stage of the procedure. Major advances in perioperative management include preoperative catecholamine blockade and adequate preoperative volume resuscitation.

Case Report

A 44-yr-old, 45kg, female presented with a history of headache for past 9 yrs, palpitation for 12 days, occasional facial flushing & heat intolerance. She was a known case of left adrenal pheochromocytoma.

On examination her heart rate was 104 beats/min, blood pressure was 180/110 mmHg and notably patient was sweating in the air conditioned room. Her present medications were amlodipine, atenolol,

nicorandil and her comorbidities were Diabetes (on oral hypoglycemics for 1yr).

Investigations revealed: Hb 6 g/dl, PCV 17.5%, S. Creatinine 3.7 mg/dl, S. Electrolytes: Na⁺ 126meq/l, K⁺ 6meq/l, Plasma free norepinephrine 8176.9pgm/ml, (range 95-446 pgm/ml). Plasma free epinephrine 4227.10pgm/ml (range: 0-67pgm/ml), Dopamine 142.1pgm (normal < 87pgm).

CT scan abdomen revealed 10 cm × 8cm left adrenal mass. CXR suggested cardiomegaly. ECG suggested left axis deviation and an old lateral wall infarction, but Echocardiography performed showed normal left ventricular function with marked concentric left ventricular hypertrophy.

Preoperative optimisation was done with volume expansion, oral phenoxybenzamine 10mg TDS, and subsequently, 20mg TDS, 20mg 6hrly & 30mg 6hrly over 8 days. Tab. metoprolol was started with 100mg/day and increased upto 200mg/day. Prazosin and clonidine were added. A blood pressure of 130/80(supine) and 120/80(standing) mmHg, (postural drop 10mmHg) was achieved. Patient was accepted for surgery on day 9. Hemoglobin was increased to 12.4 g/dl by blood transfusions.

Patient was premedicated with Tab. Diazepam 5mg & Tab. Ranitidine 150mg (night before and on the morning of surgery) and Tab. Ondansetron 4mg (morning of surgery). In the operating room following monitoring was instituted: Noninvasive (5 lead ECG, pulse oximetry, NIBP, ETCO₂, Temperature, PNS) and Invasive (IBP, CVP). Next, the patient was induced with propofol 1 mg/kg, fentanyl 3 µg/kg, vecuronium 0.1 mg/kg. Hemodynamic response to tracheal intubation were attenuated with beta blockers and Inj. Xylocard (maximum blood pressure, 160/84 mmHg; maximum heart rate, 100 beats/min). Anaesthesia was maintained with isoflurane in nitrous oxide: oxygen and titrated fentanyl.

During tumour resection, blood pressure could not be lowered to <200/100 mmHg despite sodium nitroprusside and nitroglycerin infusions. Intravascular volume was maintained with crystalloid infusion (CVP 15-18mmHg). Immediately after tumour removal, profound hypotension developed. The blood pressure was 60mmHg systolic. All vasodilators were terminated just prior to excision of tumour in anticipation of hypotension. Aggressive intravascular volume expansion initiated along with inj.ephedrine boluses (upto 24mg), Dopamine infusion (20 µg/kg/min), Norepinephrine (infusion of 1.5µg/kg/min). BP remained at 60/20 mm Hg for next 20 min. Epinephrine (bolus of 1mg; infusion of 20 µg/min) was given followed by Dobutamine infusion (10 µg/kg/min)-

BP rose to max of 80/40 mm Hg and remained at that level next 20 mts. Next, Inj. phenylephrine 6ml was given but BP remained same. Then, Vasopressin infusion was started at the rate of 2units/hr and increased upto 5units/hr Blood pressure started showing an upward trend (100-110 mmHg /50-60mmHg in 30 min). Her extremities were warm and urine output greater than 3 ml/kg/h. Blood loss in the surgery was minimal around 150ml. Elective Post operative ventilation planned. Vasopressor infusions were gradually withdrawn over next 48 h in the post operative period. Extubation performed after 48 hrs.

Discussion

Persistent hypotension due to alpha and beta blockade after surgical resection of neuroendocrine tumours is documented. Patients are preoperatively treated for 1-2 weeks with phenoxybenzamine as they are chronically vasoconstricted. Doxazosin may be a superior alternative to phenoxybenzamine because it is a specific alpha₁-blocker with a shorter half-life of about 12 h¹. For the raised BP patient was given Beta blockers (atenolol), Amlodipine and Nicorandil. Even Clonidine was started preop as BP was not controlled. These additional antihypertensives used preoperatively could have contributed to this refractoriness. Associated significant blood loss can also attribute to such a situation. No significant blood loss was seen in this case. Total blood loss was only 150ml.

Chronic increase of circulating norepinephrine leads to down-regulation of neurohypophyseal vasopressin synthesis². This case has a long standing history with level of catecholamines in the blood of almost 20 times normal. A possible explanation for vasopressin deficiency in our case may be the excessive circulating norepinephrine levels that are known to inhibit vasopressin release².

Few other reports suggest that vasopressin can be safely and effectively used to treat postadrenalectomy catecholamine-resistant hypotension in patients with pheochromocytoma³⁻⁵. In this case catecholamine replacement (most of the vasopressors and inotropes available in the pharmacopeia) could not restore vascular tone and ultimately vasopressin was used to reverse catecholamine-resistant intractable hypotension. Angiotensin is probably the only other agent that may have helped but is not readily available.

Conclusion

This case emphasizes on the use of vasopressin for treatment of refractory hypotension after resection of symptomatic pheochromocytoma.

References

1. Prys-Roberts C, Farndon JR: Efficacy and safety of doxazosin for perioperative management of patients with pheochromocytoma. *World J Surg* 2002; 26:1037-42
2. Day TA, Randle JC, Renaud LP: Opposing alpha and beta-adrenergic mechanisms mediate dose-dependent actions of norepinephrine on supraoptic vasopressin neurons in vivo. *Brain Res* 1985; 358:171-9.
3. Tan SG, Koay CK, Chan ST: The use of vasopressin to treat catecholamine-resistant hypotension after phaeochromocytoma removal. *Anaesth Intensive Care* 2002; 30:477-80
4. Augoustides JG, Abrams M, Berkowitz D, Fraker D: Vasopressin for hemodynamic rescue in catecholamine resistant vasoplegic shock after resection of massive pheochromocytoma. *Anesthesiology* 2004; 101:1022-4
5. Deutsch E, Tobias JD: Vasopressin to treat hypotension after pheochromocytoma resection in an eleven-year-old boy. *J Cardiothorac Vasc Anesth* 2006; 20:394-6