CASE REPORT

Anesthetic management of a missed pheochromocytoma during exploratory laparotomy

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ABSTRACT

Pheochromocytomas are highly vascular and catecholamine producing tumours derived from sympathetic or parasympathetic nervous system, and are estimated to occur in 2-8 out of 1 million population per year; about 0.1% of all hypertensives harbour a pheochromocytoma. Patients usually present with signs and symptoms of sympathetic stimulation, e.g. tachycardia and hypertension etc. We present a rare presentation of pheochromocytoma; a patient with undiagnosed abdominal mass posted for exploratory laparotomy diagnosed to be pheochromocytoma only by histopathology postoperatively. This patient developed intraoperative hypertensive crisis and pulmonary oedema but was managed successfully with proper treatment.

Keywords: Pheochromocytoma; Catecholamines; Hypertension; Hypertensive crises

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INTRODUCTION

Pheochromocytomas are catecholamine producing tumours derived from sympathetic or parasympathetic nervous system.[1] A definitive diagnosis of a pheochromocytoma provides potentially a correctable cause of hypertension, and their removal can prevent hypertensive crises that can be lethal. Anesthetic considerations include proper preanesthetic check up, operative fluid management as well as management of hypertension and post clamping hypotension. The patients commonly present with fluctuating blood pressure, sweating and palpitations.[2] Uncontrolled catecholamine release can result in malignant hypertension, cerebrovascular accidents and myocardial infarction.[3] Thus, they present a great challenge to the anesthesiologist both in the operating room as well as in ICU. We present a rare presentation of pheochromocytoma; a patient with undiagnosed abdominal mass posted for exploratory laparotomy, developed intraoperative hypertensive crisis and pulmonary oedema, but was managed successfully with proper treatment. The diagnosis was confirmed as a pheochromocytoma on histopathologic examination postoperatively.

CASE REPORT

A thirty eight years old female patient, weighing 36 kg presented with complaints of dyspepsia and pain in epigastrium for the last four months. Patient noticed a swelling in her abdomen and gradually increasing dull aching pain for two weeks. She was diagnosed to be suffering from a lump in abdomen and was posted for exploratory laparotomy for excision of the lump. On general examination, she was a poor build and undernourished lady with pulse 80/min and blood pressure 110/70 mmHg. All other physical findings were within normal limits except for a palpable mass in her left hypochondrium.

In preoperative checkup USG abdomen showed a large heterogenous mass 12.8 x 8.9 cm, probably arising from the tail of pancreas. CT scan of abdomen and pelvis showed a large well defined solid mass measuring approximately 10.5 x 9.3 x 8.4 cm in retroperitoneum in relation to distal body and tail of pancreas and near upper pole of left kidney and medial surface of spleen,

suspected to be of neoplastic origin.

At the time of admission, her Hb was 4.9 gm, so two units of packed red blood cells and two units of whole blood were transfused during a period of 10 days to raise her Hb prior to surgery to 11 gm. Rest of the investigations were within normal limits. Chest x-ray PA view showed cardiomegaly with normal lung fields. ECG showed T-wave inversion in V4 and V5. 2-D echocardiography showed ejection fraction 60%, mild aortic regurgitation and diastolic dysfunction.

Anaesthetic management: Informed written consent was signed by the patient. In the operating rooms routine monitors were connected (SpO_2, NIBP and ECG) and IV infusion was started. Prior to surgery her pulse rate was 68/min, BP 124/82 mmHg with SpO_2 100%. She was premedicated with inj. glycopyrrolate 0.2 mg and inj. fentanyl 40 μg. Preoxygention was done with 100% O_2 for three min and anesthesia was induced with inj. propofol 100 mg, scoline 80 mg and she was intubated with 7.5 mm ID cuffed ETT. Anesthesia was maintained on isoflurane in O_2:N_2O (33:66) with inj. vecuronium bromide 0.08 mg/kg as a muscle relaxant. A double lumen central venous catheter was inserted and CVP was measured to be 8 cmH_2O. After opening the peritoneum, when the surgeon started exploring near the abdominal mass, the BP suddenly shot upto 200/120 mmHg and heart rate rose to 150/min. The depth of anesthesia was increased. As an acceptable reduction in BP and HR was not obtained, inj. labetalol 5 mg was administered IV. The BP dropped to 180/100 mmHg and HR reduced to 146/min. Labetalol 5 mg was repeated and nitroglycerine infusion was started at 0.5 μg/kg/min. Gradually, the dose of the infusion was increased to 1μg/kg/min.

Then suddenly, an increased resistance to manual ventilation was felt and pink frothy secretions were observed in ETT. Her SpO_2 dropped to 80%. On chest auscultation bilateral crepts were audible. These were the signs of pulmonary oedema, so we terminated all inhalational anesthetics, 100% O_2 was started and of inj. frusemide 40 mg was given IV; but the SpO_2 failed to rise above 76%. Inj. frusemide was repeated, PEEP of 5 cmH_2O was started and increased up to 10 cmH_2O. Five minutes later, the HR was 95/min, BP 145/96 mmHg and SpO_2 80%. Meanwhile, the surgeon explored the abdomen and found spleen, pancreas and the liver to be normal. At this time we all suspected the mass to be a pheochromocytoma, so the surgeon clamped the suprarenal vein following which there was a sudden fall in BP to 90/60 mmHg. Nitroglycerin infusion was stopped and dopamine infusion was started at 7 μg/kg/min. As the fall in BP continued, dobutamine infusion was also added at 5 μg/kg/min. Meanwhile, the mass was removed and was sent for histopathology. BP was still low (72/50 mmHg), so noradrenaline infusion was also started at 0.15 μg/kg/m. Intravenous fluids were given to improve the left ventricular filling. Total blood loss was estimated to be about one litre, so two units of packed cells were transfused. At the end of surgery the patient was still hemodynamically unstable so the patient was shifted to SICU on mechanical ventilation with full inotropic support. Total urine output was 400 ml.

In SICU, patient was put on pressure control mode with FiO_2 0.7, PEEP 5 cmH_2O, pressure support above PEEP 15 cm H_2O and RR 15/min. Inotropic support was continued. ABG's report showed respiratory acidosis with pH 6.9, PCO_2 88, PO_2 65 and HCO_3 18. Chest x-ray was ordered, which depicted perihilar infiltrates and an increase in pulmonary vasculature suggestive of pulmonary oedema. Within five hours of ventilation and inotropic support, BP gradually rose from 90/60 mmHg to 110/70 mmHg with fluctuations, SpO_2 also gradually improved from 92% to 94% to 100%. Her chest was then clear. Repeat ABG's were also within normal limits. Overnight, mechanical ventilation was continued with high inotropic support. On second post-op day, her HR was 110/min, BP 106/74 mmHg, and the chest was clear without any additional sounds. So we decided to wean him off ventilator and taper off the inotropes. When the patient was fully awake and followed verbal commands, ventilator setting was changed from pressure control mode to continuous positive pressure mode. After one hour, a trial with T-piece delivering O_2 at 4 lit/min was given. The patient remained comfortable and tolerated the trial well, so was extubated with thorough suctioning. Nebulisation with salbutamol was given and O_2 was administered at 2 lit/min via venturi mask. Histopathology report was received after two days which confirmed the diagnosis of benign pheochromocytoma. The patient was observed in SICU for four more days and was shifted to the ward on sixth postop day.

DISCUSSION

Pheochromocytomas are catecholamine producing tumours which originate in adrenal medulla or in chromaffin tissue along with vertebral sympathetic chain from pelvis to base of the skull. Sympathetic ganglia in the wall of the urinary bladder may be a site for pheochromocytoma[1]. These tumours occur sporadically and are inherited as features of multiple endocrine neoplasia type 2 or several other pheochromocytoma associated syndromes. It is estimated to occur in 2-8 out of 1 million persons per year and is present in about 0.1% of all hypertensives. Generally patients present with a widely fluctuating blood pressure, sweating and palpitations[2]. Preoperative management consist of control of blood pressure and restoration of intravascular volume.[4] Generally the

control of blood pressure is done with α-adrenergic blockers over a period of 10-14 days. The drug of choice is phenoxybenzamine. βeta adrenergic blockers can be added after α-blockers because β-blockers can induce a paradoxical increase in blood pressure in absence of α-blockade, they should be administered only after effective α-blockade.

Our patient did not show any signs and symptoms suggestive of pheochromocytoma preoperatively, as there was no complaint of headache, palpitation or hypertension. Despite long term increase in catecholamine levels, some patients do not appear to produce hemodynamic response characteristic of acute administration. A desensitisation of cerebrovascular system or increased down-regulation of adrenergic receptors may explain this finding. The sensitivity of smooth muscle cells is decreased secondary to decrease in the number of receptors or alteration in receptor-effector coupling. The hypertensive crisis does, however, mimic the response to acute catecholamine administration. Blood vessels of these patients require extremely high concentration of catecholamines produce hypertension.[4]

We were unaware preoperatively about the diagnosis and it was only postoperatively diagnosed on histopathology of the removed mass. Hypertensive crisis which occurred at the time of handling of the tumour was well-managed by increasing the depth of anesthesia, supplementation with opioids, inj labetalol which is combined α_1 and β_1 adrenoceptor antagonist. It lowers the blood pressure by blocking the α adrenoceptors in arterioles and thus reduces the peripheral resistance and concurrent β-blockade protects the heart from reflex sympathetic drive normally induced by peripheral vasodilatation.[5] Nitroglycerin is a potent vasodilator so helps to reduce the blood pressure. Other drugs of choice in this situation are inj nitroprusside, esmolol, nicardipine, phentolamine, phenoxybenzamine and propranolol etc.[6]

In our patient acute pulmonary oedema/congestion developed due to release of catecholamines in circulation during handling of the tumour, so there was an increase in heart rate and systemic vascular resistance which led to an increase in blood pressure and therefore, afterload. An acute increase in left ventricular end diastolic pressure causes an acute increase in left atrial pressure and back pressure, that leads to increased pulmonary capillary pressure.[7] In order to reduce the after load, inj. frusemide, which is a rapidly acting loop diuretic and a venodilator, is the drug of choice. The PEEP helps to improve the oxygenation and reduce the pulmonary congestion. Clamping the suprarenal vein causes the sudden withdrawal of the circulating catecholamines thus leading to a sudden fall in the blood pressure, which has to be treated with IV fluids, correction of blood loss and inotropes i.e dopamine, dobutamine and noradrenaline. The cardiac involvement may manifest itself as cardiomyopathy, ischemic heart disease or cardiac failure.[8] Postoperatively these patients require intensive care monitoring with inotropic support as they are highly vulnerable to hypertensive, hypotensive, or hypoglycemic episodes.[1]

CONCLUSION

The patients of pheochromocytoma pose a great challenge to the anesthesiologists in the operating room as well as in ICU. A high degree of suspicion, close cooperation between the surgical team and the anesthesiologist and readily available pharmacotherapeutic agents are essential for a successful outcome in these patients.

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