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Phaeochromocytoma- A Case Report of Anaesthetic Management of An Atypical Presentation with Postoperative Polyuria

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1. Abstract

Phaeochromocytoma is a rare case to present for surgical excision and the key in decreasing the morbidity and mortality associated with this disease is surgical excision of the tumour with preoperative adequate medical management and optimisation. We describe the management of a case of phaeochromocytoma who presented with atypical symptoms, catecholamine induced cardiomyopathy who was evaluated and optimised preoperatively and taken up for open left adrenalectomy under epidural with general anaesthesia along with central venous access and invasive blood pressure monitoring; and in whom there was postoperative polyuria. We hereby describe the challenges encountered with management of this case and discuss the measures taken to mitigate and manage the same.

2. Introduction

Successful management of Phaeochromocytoma requires careful preoperative optimisation, meticulous preparation for intraoperative events and impeccable hemodynamic monitoring and management.

3. Case Report

50 year old male with hypertension and diabetes mellitus, admitted by the physician with decreased urine output, easy fatigability and recurrent syncope. On admission he was found to have labile hypertension manifested by profound postural hypotension and hyperglycaemia. At the time of admission his vitals were as follows: pulse rate- 104 beats/min, Blood pressure- 210/120 mm Hg in supine position and 84/40 mm Hg in sitting position with syncope on standing. Patient weighed 45 kg and had a BMI of 16.1 kg/m2. He was catheterised and an adequate urine output was observed with normal renal parameters. Glycemic control was achieved with regular insulin. The fluctuating blood pressure remained the same despite adequate fluid supplementation and pharmacological agents. The contrast enhanced computed tomography revealed a left adrenal mass. Oncosurgeon opinion was sought and an Endocrinologist was involved. Further work up discovered significantly elevated urinary VMA (28943 ng/mL) and VMA/Creatinine ratio. A contrast MRI scan demonstrated a left adrenal tumour of 4.5 x 2.7 x 3.6 cm. The patient was started on oral alpha adrenergic blocker PRAZOSIN and after achieving adequate alpha blockade, on beta receptor antagonist- PROPRANOLOL. These medications brought a symptomatic improvement and decreased the degree of variability in the blood pressure. The cardiac status of the patient was evaluated with an electrocardiogram that showed T inversions in leads II and V6; and an echocardiogram which revealed severe concentric left ventricular hypertrophy, left atrial dilation, global LV hypokinesia with an ejection fraction of 40%. During the course of the first 20 days of hospital admission urine output was normal, glycemic control was achieved with regular insulin and symptoms improved. He was then posted for exploratory laparotomy. At the time of pre anaesthetic evaluation, the patient had been on alpha and beta blockade for 15 days, had achieved adequate glycemic control and had persistent postural hypotension. At the first PAE, serum calcium, thyroid function test and HRCT Thorax were advised to rule out associated MEN syndromes and extra adrenal tumours respectively and both ruled out. The patient was further optimised for another 7 days by adjusting the alpha blocker dosage, encouraging an oral salt intake of 5-10 g per day and water intake of 2-3 L and familiarising the patient with the OT and OT staff to ensure a calm patient on the day of surgery. Prior to surgery his vitals were as follows: pulse rate- 77 beats/min, Blood pressure- 110/60 mm Hg in supine position and 70/40 mm Hg in sitting position. Despite significant symptomatic improvement with medical management, all of the

Roizen's criteria [1] could not be satisfied. Preoperatively, the patient and his attenders were counselled regarding the illness, associated perioperative risks, advantage of tumour resection, anaesthetic, surgical and postoperative management; and the associated morbidity and mortality. On the day before surgery, anxiolytics in the form of oral ALPRAZOLAM 0.5mg was given at night. Fasting guidelines were advised. On the day of surgery, a complete blood count with hematocrit, fasting blood sugar and ECG were done and 1 unit of compatible blood was made available. Careful preparation of OT was undertaken and in view of the expected labile haemodynamic, antihypertensive medications including- sodium nitroprusside, nitro-glycerine, esmolol and diltiazem infusions; as well as vasopressors including phenylephrine for bolus doses, noradrenaline, epinephrine and vasopressin infusions were prepared. The patient was taken inside OT and ASA standard monitors were connected and baseline readings taken. A baseline GRBS was done. Two 18 G IV cannula were secured one in each upper limb, Inj. MIDAZOLAM 1 mg IV and Inj. HYDROCORTISONE 100 mg IV were given. Intravenous Ringer Lactate was started and a bolus infused. O2 was administered via a simple face mask at 5 L/min. A 20 G arterial line was secured in the left radial artery and invasive intra-arterial blood pressure monitoring started. The baseline arterial blood gas analysis was normal. A central venous catheter was secured in the right subclavian vein. Following this, the patient was turned to the left lateral position and an epidural catheter was secured in T10-T11 space by loss of resistance to air technique and test dose injection Lignocaine 2% with Adrenaline, 3 mL given which was negative. Due to adequate anxiolysis, during the above invasive procedures no sympathetic response was noted on the monitors. Supine position was resumed and general anaesthesia was induced after preoxygenation and premedication with Inj. ONDANSETRON 4 mg IV and Inj. FENTANYL 100 mcg IV. Induction agent used was Inj. ETOMIDATE 16 mg IV in view of the cardiac status. Inj. Lignocaine 60 mg IV was used to attenuate stressor response. Muscle relaxant Inj. VECURONIUM 4.5 mg IV was given and intubated under direct laryngoscopy (Cormack-Lehane Grade 1) with 8.0 mm ET tube and tube secured. Maintenance of anaesthesia was done with ISOFLURANE 1.0% and 50-50 mixture of nitrous oxide and oxygen. Epidural top-up of 4 mL 0.5% Bupivacaine was given. There was no sympathetic response to laryngoscopy or skin incision. At the time of surgical manipulation and tumour handling, due to catecholamine surge blood pressure was severely elevated up to 280/130 mm Hg, for which Inj.

SODIUM NITROPRUSSIDE was started at a dose of 0.5 mcg/kg/min and administered for 1 minute. Once the blood pressure dropped to 180/120 mm Hg with a heart rate of 140 bpm the infusion was stopped and a bolus dose of Inj. ESMOLOL 30 mg was given, by which time the surgeon had ligated the left adrenal vein. 5 minutes post tumour vein ligation blood pressure showed a precipitous fall to 60/40 mm Hg, for which a bolus of Inj. Phenylephrine 100 mcg IV was given and Inj. Noradrenaline in a dose of 0.2 mcg/kg/min and titrated according to the blood pressure. A total of 2500 mL of fluid was administered intraoperatively. Adequate urine output of 1100 mL was present during the procedure and normoglycemia was maintained. Total duration of the surgery was 45 minutes. After completion of the surgery, the patient was kept on table for 1 hour and monitored. After stable intra-arterial blood pressure was noted with minimal vasopressor support along with return of spontaneous respiratory efforts, neuromuscular blockade was reversed with Inj. NEOSTIGMINE 2.25 mg and Inj. GLYCOPYRROLATE 0.45 mg IV. Extubating was uneventful. After a bolus dose of 6 mL 0.0625% Inj. BUPIVACAINE, epidural infusion was started at 6 mL/hr with the same strength solution. Patient was shifted to the surgical ICU with invasive blood pressure, urine output and other standard ASA monitoring. Postoperatively, blood pressure remained stable with Inj. Noradrenaline infusion at a dose of 0.2 mcg/kg/min and down titrated according to the blood pressure. Intravenous fluid administration was continued and analgesia maintained with multimodal analgesia. 1 pint of packed red cells was transfused. GRBS was monitored every hour. On POD 0 at 12 AM there was a spike in blood pressure to 210/120 mm Hg with CVP being 0.5 cm of H2O resolved on administration of fluid bolus of 1L. Urine output monitoring demonstrated a high output state (500-700 mL/hr), which was replaced on an hourly basis with crystalloid infusions. The first 24 hour urine output was 12 L. Nephrologist opinion was sought and urinary specific gravity, urine sodium excretion and urine osmolality was done which were normal along with advice for 6th hourly monitoring of renal function tests and serum electrolyte levels. Serum electrolyte levels done showed low serum potassium in the background of normal serum sodium and chloride levels, for which intravenous correction was done and oral potassium and magnesium supplementation started. Invasive blood pressure monitoring was done for 48 hours. Inj. NORADRENALINE was continued for 36 hours and stopped after blood pressure stabilised to 100/60 mm Hg. Persistent polyuria was noted with daily 24 hour urine output in excess of 7-10 L. Hourly output was replaced with crystalloids including 0.9% normal saline and 0.45% normal saline in conjunction with oral intake. Although the cause for polyuria is most commonly idiopathic, other causes for polyuria could include hypoadrenalism, high BNP levels, SIADH and renal tubular acidosis. Steroid therapy was continued in the form of Inj. HYDROCORTISONE and tapered over 6 days and then converted to oral PREDNISOLONE 10 mg per day. Patient remained in the surgical ICU till postoperative Day 5 after which he was shifted to the ward. Potassium correction was required daily postoperatively for a duration of 8 days and polyuria decreased by postoperative Day 10, also oral steroid tapered and stopped. Patient was discharged on post op Day 12 with oral potassium and magnesium supplements. Patient was followed up on an outpatient basis 2 weeks after discharge and all clinical and biochemical parameters were found to be normal.

4. Discussion

Phaeochromocytoma is a challenging tumour to manage for the perioperative physician. The key to perioperative medical optimisation is alpha blockade to manage the effects produced by the elevated levels of circulating norepinephrine and epinephrine produced by this tumour, and has been so for the past 60 years [2]. Our patient did not present with the classical symptom triad commonly associated with this tumour and had decreased urine output, easy fatigability, recurrent syncope and postural hypotension along with constant hypertension and hyperglycaemia requiring pharmacotherapy, thereby living up to its name of the 'great mimic [3]. Familial associated syndromes and extra adrenal tumours were ruled out with clinical history, serum calcium levels and HRCT Thorax [4]. As is protocol, cardiac evaluation must be done in these patients to evaluate them for possible catecholamine induced cardiomyopathy [5]. In our case successful alpha blockade and consequently beta blockade was achieved with the use of oral PRAZOSIN and PROPRANOLOL both of which have proven efficacy in such cases [6] and their use has decreased mortality from 50% to <6% [7]. Since haemodynamic stability is expected in these cases[8], appropriate monitoring is required apart from the standard ASA monitors[9] including invasive blood pressure and central venous pressure monitoring [10]. Choice of drug for induction in our case with poor cardiac reserve was ETOMIDATE its ability to confer haemodynamic stability [11]. VECURONIUM is the most widely used and preferred muscle relaxant as it has no autonomic effects and does not result in histamine release [12]. Attenuation of response to laryngoscopy with various agents is crucial to prevent a hypertensive crisis [13]. Although the gold standard for this size of tumour is the laparoscopic surgical approach, in this case an open approach was undertaken [14]. From when the patient is taken into the theatre there are multiple causes (positioning, intubation, skin incision) for catecholamine surges that are associated with regular surgeries as well, which are transient and respond quickly to therapy [15]. However, tumour manipulation results in a far more dramatic pressor response due to the circulating catecholamine as opposed to the stored form released from nerve endings [16]. In order to manage these crises SODIUM NITROPRUSSIDE infusion and ESMOLOL bolus were used in our case. To offset the profound hypotension resulting after tumour vein ligation, the patient was given 1.5 litres of fluids before this critical step and managed with vasopressor support [17]. Most cases with hypotension respond to norepinephrine, epinephrine or dopamine, however, some may be refractory to these agents in which case vasopressin has been found useful [18,19]. Intraoperative blood glucose monitoring is required to ensure normoglycemia in these patients. Postoperatively hypotension may persist as variably described in 20-70% of cases dependent on the agent used for preoperative alpha blockade as well as the intraoperative hypotensive agent used [20]. Monitoring with an invasive blood pressure monitor is valuable for at least 24 hours postoperatively. Sudden depletion of catecholamine levels due to tumour removal may lead to rebound hyperinsulinemia which can result in profound hypoglycemia [21]. Therefore, hourly postoperative monitoring of blood glucose levels is mandatory. Careful attention to fluid administration and electrolyte levels is also imperative. In our case the patient developed severe polyuria after resection of the tumour (500-700 mL/hr), the mechanism of which is not fully understood [22]. Only a handful of cases have been reported worldwide. There are a myriad mechanisms that could potentially be the cause of this diuresis including mineralocorticoid deficiency secondary to hypoadrenalism [23], elevated plasma atrial natriuretic peptide and brain natriuretic peptide levels or renal tubular acidosis. In our case the polyuria may be attributed to a concentrating defect of the kidneys possibly due to decreased mineralocorticoids as evidenced by improvement with steroid supplementation. There was postoperative hypotension for 48 hours which was managed with minimum vasopressor support. Blood sugars were monitored on an hourly basis and were consistently within normal limits. Postoperative polyuria after resection of pheochromocytoma is a rare clinical situation, which needs to be kept in mind and studied further. We conclude that perioperative management of pheochromocytoma is a thrilling roller coaster ride for any anaesthesiologist demanding a rise to the title of 'perioperative physician.'

5. Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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