INTRODUCTION

We report the anaesthetic management of a patient undergoing removal of a previously non-diagnosed phaeochromocytoma. Major haemodynamic changes were recorded because the phaeochromocytoma was not preoperatively suspected. However, adequate preoperative assessment and clinical vigilance allowed a safe and satisfactory outcome.

The aim of presenting this case is to highlight the rarity of the multiple coexisting pathologies, clinical challenges faced due to presence of high-risk comorbidities and highly eventful intraoperative period in spite of the tumour being biochemically negative for phaeochromocytoma.

CASE REPORT

A 39-year-old male, a case of von Hippel Lindau (VHL) syndrome and follow-up case of renal cell carcinoma (right) post radical nephrectomy and right adrenalectomy 2 weeks ago, was planned for left radical adrenalectomy for left adrenal mass and radiofrequency ablation (RFA) of left renal tumour. A preoperative assessment revealed the patient to be a known hypertensive, diabetic (on insulin) with a post coronary artery bypass graft (CABG) status (2005) on β-blocker (Tab. Metoprolol). Aspirin was stopped 5 days ago. Clinical history revealed episodes of headache, dizziness on change to erect posture, palpitations, recent episode of retrosternal chest pain with radiation to epigastrium and functional capacity of 2-3 metabolic equivalents. Clinical examination revealed an afebrile, averagely built and nourished patient with blood pressure (BP) of 130-170/80-90 mm Hg and pulse rate of 70-78/min. Airway assessment showed Mallampati airway class II, adequate mouth opening, thyromental distance >3 finger breadth and normal range of neck movements. Systemic examination did not reveal any significant findings. Investigations were within normal limits except capillary blood glucose (CBG) which ranged between 178 and 266 mg/dl. Cardiological evaluation showed ischaemic changes in electrocardiogram (ECG)
for inferior and basal regions, dobutamine stress echocardiography (DSE) positive for provokable ischaemia in anterior, mid and basal lateral walls, echocardiography revealing akinetic inferior wall in mid and basal region and mid posterior wall with partial thickness scar, left ventricular ejection fraction (LVEF)=50% and mild mitral regurgitation with grade I diastolic dysfunction. Urinary catecholamine levels, including values of 24 hours collection were within normal limits (metanephrines-58 ng/ml, and 284.2 μg/day, epinephrine-1.86 pg/ml and norepinephrine-48.3μg/day). Histopathological examination of previously excised adrenal tumour was suggestive of adrenocortical carcinoma. Glycaemic control was achieved with insulin as per sliding scale. Patient was accepted under ASA class III with high-risk consent. Two units of packed red blood cells (PRBC) were kept ready.

On the morning of surgery, patient was shifted to OT and connected to monitors – noninvasive blood pressure (NIBP), SpO₂ and ECG. Initial recordings displayed BP=220/100 mm Hg, heart rate (HR)=82/min and SpO₂=100%. Midazolam 2.0 mg IV was administered. Before induction, a thoracic epidural catheter was placed, followed by a 20-G arterial line over left radial artery and a 7-Fr triple-lumen central line over right internal jugular vein. A 7-Fr Swan Ganz pulmonary artery (PA) catheter was inserted under local anaesthesia, keeping in mind the findings of cardiological evaluation. PA catheter showed value of 26/13 mm Hg. Post-intubation, patient was mechanically ventilated in volume control mode with end-tidal carbon dioxide (ETCO₂) and minimum alveolar concentration (MAC) monitoring. No haemodynamic disturbances were observed during or after induction/intubation. Patient was catheterised. Insulin infusion was started for glycaemic control. Dexmedetomidine infusion was added to supplement depth of anaesthesia. A baseline arterial blood gas (ABG) and CBG were done post-induction. DJ stenting (left) was done in lithotomy position.

The patient was then transported on C-circuit to the Department of Radiology for computed tomography (CT)-guided RFA. Patient was placed in prone position in the CT gantry. Ventilation was managed with ventilator using oxygen only. Anaesthesia was managed with increased infusion rates of dexmedetomidine and periodic boluses of propofol, fentanyl and rocuronium. At the end of the 3-h procedure, patient was transported back to the operation theatre and radical adrenalectomy was started in the right lateral decubitus position. Blood loss was estimated to be approximately 1500 ml. PA wedge pressure (PAWP) was found to be 3-4 mm Hg, while central venous pressure (CVP) was 2-6 cm H₂O. Fluid resuscitation was carried out with crystalloids and colloids, the total intraoperative volumes being 3500 ml and 2000 ml, respectively. Two units of PRBC were also transfused intraoperatively. During tumour handling, massive fluctuations in haemodynamics were noted with BP shooting up to 208/110 mmHg and HR upto 125/min. SpO₂ and EtCO₂ were within normal limits. Immediately sodium nitroprusside (SNP) infusion was started and vitals were restored. Following tumour removal, blood pressure dropped drastically to 70/32 mm Hg, which prompted us to start an infusion of noradrenaline and adrenaline. SNP infusion was discontinued. Hydrocortisone 200 mg IV was administered. Sodium bicarbonate was administered as serial ABGs showed progressive metabolic acidosis. Urine output was 2700 ml intraoperatively.

Analgesia was maintained with boluses of IV fentanyl, and IV tramadol. Approximately 30 min before closure, IV ondansetron was administered. Post-extubation, patient was comfortable with stable haemodynamics and satisfactory visual analogue scale (VAS) score. Patient was shifted to surgical intensive care unit (ICU) for postoperative monitoring and evaluation. Postoperative tenure was uneventful and patient was discharged on 27 March 2012. Postoperatively, histopathological evaluation of the adrenal tumour revealed it to be a phaeochromocytoma and a renal cell carcinoma (RCC) from the biopsy of renal tumour prior to the RFA. The slide of previously excised adrenal tumour was re-evaluated and found to be a phaeochromocytoma too.

**DISCUSSION**

Phaeochromocytoma is a catecholamine secreting tumour originating in the adrenal medulla or in chromaffin tissues along the paravertebral sympathetic chain. The incidence of phaeochromocytoma is reported to be 1.55-2.1 per million population per year.¹ The perioperative mortality rate for elective resection of phaeochromocytoma is 0%-3%, and in an undiagnosed or ill-prepared patient, this can be as high as 50%.² In the hypertensive population, about 0.1% have phaeochromocytoma. Approximately 10% of the tumours are bilateral, 10% are extra-adrenal and 10% are malignant. In children, 70% are bilateral, the
majority being benign and extra-adrenal.[3] The greatest frequency occurs in the fourth and fifth decades of life, with slightly higher female preponderance (60%). Incidence of phaeochromocytoma in VHL syndrome has been reported between 10% and 20%. Phaeochromocytoma releases large amounts of catecholamines (CCA) (adrenaline, noradrenaline and dopamine), and various peptides and ectopic hormones (enkephalins, somatostatin, calcitonin, oxytocin, vasopressin, insulin and adrenocorticotrophic hormones).[4] Phaeochromocytoma whether it is secreting or not does not change anaesthetic care as it may become intraoperatively secreting. Even clinically silent phaeochromocytomas can be lethal.[5]

The problem differs if the tumour is not preoperatively diagnosed, as it happened here – haemodynamic changes are not expected and intraoperative care is more difficult leading to worsening of prognosis. The patient is prepared for surgery by appropriate alpha blockade over a period of 10-14 days. Tachycardia and arrhythmias are controlled by carefully introducing beta adrenergic blockers. Patient with adrenal masses suspected to be phaeochromocytoma represents the greatest challenge for the anaesthetist in the perioperative management. However, manipulation of the tumour during open surgery, with its haemodynamic responses, may be inevitable but can usually be of short duration.[6] Although this patient's tumour behaved as intraoperative phaeochromocytoma on excision, the preoperative biochemical tests and hormonal results did not give any clue that this patient could have secreting mass. In spite of that, the intraoperative plan took into account the high possibility of occurrence of hypertensive crisis and other events. For these reasons, patient had the arterial and central line in place with emergency drugs ready. A PA catheter is not recommended anymore in recent studies. However, a PA catheter was introduced to specifically differentiate a cardiac event or hypovolaemia from catecholamine release due to tumour handling, in a scenario of haemodynamic instability, as a transoesophageal echocardiography (TOE) monitoring was not possible in prone position of patient.

**CONCLUSION**

An unbiased sound clinical judgement should always get precedence as histopathological and biochemical assessment may at times be misleading, as it happened in this situation. Due to early and proactive care, we could ensure intraoperative safety of this patient.

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