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Original Research Paper

Anaesthesiology

UNDETECTED PHEOCHROMOCYTOMA

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KEYWORDS :

INTRODUCTION

Pheochromocytoma is a rare catecholamine secreting tumour characterised by excessive production of catecholamines (epinephrine,norepinephrine and dopamine).The incidence of pheochromocytoma is 0.8 per 100000 years

Anaesthesia and surgery in unsuspected cases have high mortality.Pheochromocytoma represents very significant challenges to the anaesthesiologist especially when undiagnosed.

In this case, the patient presented with an undiagnosed pheochromocytoma, which led to a life-threatening hypertensive crisis and myocardial ischemic events after the induction of anesthesia. This case serves to emphasize the importance of being vigilant and prepared for such unexpected occurrences.

Case Report

A 36 year old male weighing 70 kg who is a known case of Neurofibromatosis type 1 was posted for right foot neurofibromas excision. The patient had no history of hypertension, diabetes, headaches, diaphoresis, visual disturbances, chest pain or any other comorbidities

Blood pressure was 120/80 mmHg Routine blood and urine investigations ,liver and ECG and ECHO were normal

On the preoperative night oral pantoprazole 40mg and anxit 0.5 mg were advised.On the day of surgery inj pantoprazole 40 mg IV and maintenance fluid were infused.General anesthesia was planned for the patient.

Standard monitors were applied as per department protocols and the blood pressure, heart rate and peripheral capillary oxygen saturation (SpO2) measurements were 108/70 mmHg, 80 bpm and 97% respectively on arrival in the operating room

General Anaesthesia was induced with Ing propofol 100 mg and cisatracurium 8 mg and fentanyl 100 mcg and maintained with oxygen ,N2O and sevoflurane with IPPV Patient was hemodynamically stable during intubation and no events were recorded . 20 minutes following intubation ,consecutive readings of hypotensive episodes were recorded following which 6 mg of Mephantamine was administered in view of hypotension (80/50).There appeared sudden tachycardia120 and hypertension 250/150.

This was followed by ECG changes ,ventricular bigeminy with varying pulse beat.immediately plane of anaesthesia was deepened with propofol , Injection Lidocaine 80 mg administered intravenously .Ing Esmolol was given to control the Blood pressure .Immediately help was sought from other collegue anaesthesiologist and suspicion of undiagnosed catecholamine secreting tumour was made. Immediate placement of arterial Invasive catheter and invasive blood pressure monitoring was done .The crisis lasted for 15 -20 mins.Surgery was aborted and after hypertension was stabilized around 160/90 patient was extubated after administering neostigmine 2.5 mg and glycopyrolate 0.5 mg.

The patient was shifted to Surgical ICU and Vitals were monitored . cardiac and endocrine reference were given I/v/o suspicion of pheochromocytoma ..Repeat ECG showed ST depression in V5-V6 .Immediate post op ECHO was done showing Basal inferno posterior hypokinesia with EF 50,with grade 1 MR,MVP.USG abdomen was done showing well defined heterogenously hyperechoic lesion in Right suprarenal region measuring 5*4.7 S/O Pheochromocytoma . Troponin I was found to be elevated 535.4

Plasma free metanephrine , plasma free nor metanephrine ,24 hr urine VMA was send.

Patient was started on T Prazosin 5 mg OD. Plasma free metanephrine was 950 pg/ml. Plasma nor metanephrine 600pg/mladrenal gland, Urine VMA was found to be 18.38mg/24hrs. CECT showed enchansing well defined lesion in right adrenal gland measuring4.3*4.8cms and another similar small lesion present medial to above lesion suggestive of pheochromocytoma

Five weeks later the patient returned to the hospital for elective surgery for excision of the tumor. This time induction of anesthesia was uneventful as a consequence of adequate alpha-blockade.The surgery was uneventful.

The postoperative course was unremarkable. The patient was discharged 3 days later. The final pathologic report confirmed the presence of a malignant pheochromocytoma.

DISCUSSION

A prevalence of PHEO in NF1 of 0.1–5.7%, which increased to 20–50% if hypertension was present.In fact, persistent hypertension is seen in about 60% of patients with NF1 and PHEO.

Management of the intraoperative pheochromocytoma crisis consists of elimination of the triggering factors, as well as the administration of short-acting vasodilators such as the alpha-adrenergic blocker phentolamine or sodium nitroprusside infusion.

Clinical suspicion remains the single most important factor in the identification of pheochromocytoma. Adequate preoperative diagnosis of pheochromocytoma is based on both imaging and biochemical tests.Diagnostic laboratory tests have traditionally included 24 hour urinary metanephrine and vanillylmandelic acid levels. In addition, a clonidine suppression test will rule out any other cause for an increase of catecholamines. Catecholamine secretion from a pheochromocytoma is independent of neurogenic control and therefore will not be suppressed by clonidine. Plasma-free metanephrine levels have proven to provide the best test for excluding or confirming pheochromocytoma, and should be the test of first choice for diagnosis of the tumor.

The treatment consists in adrenalectomy. Preoperative preparation includes alpha-adrenergic blockade initiated 7–14 days before. Beta-blockers can be used if reflux tachycardia results from treatment with alpha-blockade. Liberalization of salt consumption and fluid intake are advised to prevent post-operative hypotension. Blood pressure, heart rate and plasma glucose levels should be monitored. Normal plasma or urinary metanephrines 2–4 weeks after surgery document therapeutic success.21,28,33

CONCLUSION

Patients with NF1 have a genetic susceptibility to the development of PHEO and a high level of suspicion for PHEO is needed in symptomatic and hypertensive individuals with NF1. Biochemical Screening for PHEO by checking plasma or urinary metanephrines should be performed in all patients with NF1 who are hypertensive. Furthermore, systematic screening for PHEO might be considered in all patients with NF1, irrespective of their blood pressure, given the atypical presentation. Early identifications of PHEO could reduce the mortality and morbidity associated with PHEO crisis in patients with NF1; patients with NF1 can develop life threatening complications if PHEO diagnosis is missed.

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