CASE REPORT

Unusual Manifestations after a Case of Carotid Body Tumour Excision: A Case Report

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Abstract:

Carotid body tumours are rare tumours arising from chemoreceptor cells at the bifurcation of carotid artery. They are highly vascular and mostly benign but with potential to turn into malignancy. Even though tumours are nearly always non functional, catecholamine-producing tumours do exist and can produce paroxysmal hypertension. As surgical removal is the commonest mode of treatment, anaesthetic management poses several challenges. Here we report a case of carotid body tumour excision with an eventful perioperative course.

Keywords: Anaesthesia, Carotid body tumour, Hypertension

Introduction:

Carotid body tumours are rare paragangliomas, which during surgical excision can pose unique perioperative challenges to anaesthesiologist [1]. Here we present a case with an eventful perioperative course. A 35 year old female with swelling on the right side of the neck since 6 years, diagnosed as a carotid body tumour was posted for surgical excision. During last 6 years, she had undergone various local therapies, including anti tubercular treatment for a brief period.

On admission she had history of loss of weight and appetite and difficulty in swallowing solid foods. She also complained of epigastric burning, nausea and vomiting. She was poorly nourished, weighed 52 kg, but general physical and systemic examination were unremarkable. A soft, mobile, pulsatile swelling of size 4cm × 4cm was located in the right carotid triangle. Ultrasonography (USG) revealed a carotid body tumour and diagnosis was confirmed by Magnetic Resonance Imaging (MRI) of the neck and Fine Needle Aspiration Cytology (FNAC). Urinary Vanillyl Mandelic Acid (VMA) levels were raised. Routine haematological investigations were within normal range. Electrocardiogram and echocardiography reports were normal. As blood pressure was within normal range, preoperative alpha blockers were not started.

On the night before and morning of surgery patient received oral alprazolam and ranitidine tablets. Inside operation theatre, routine hemodynamic monitoring was instituted and it included non invasive blood pressure, electrocardiogram and haemoglobin saturation. Anti aspiration and anti emetic prophylaxis was given. She was pre medicated intravenously (i. v) on operation table with injections glycopyrrolate 0.2 mg, clonidine 150 mcg, fentanyl 100 mcg, midazolam 2 mg. General anaesthesia was induced with i.v thiopentone 250 mg and endotracheal intubation was achieved with i.v succinyl choline 75mg. Maintenance of anaesthesia was done with oxygen and nitrous oxide in 70:30 and titrated propofol infusion.

Left subclavian vein was cannulated for central venous pressure monitoring. For supplementation of analgesia superficial cervical plexus block was given with 12 ml of 0.5% bupivacaine. Tranexamic acid 1gm was given as slow i. v infusion. Injection thiopentone 500 mg was given

i.v as two boluses for neuroprotective effect. Soon after surgery began, blood pressures were high viz in the range of 180-200 mm of Hg (systolic) and 100-140 mm of hg (diastolic), which was controlled by judicious use of nitroglycerin intravenous infusion and intravenous boluses of injection labetalol.

Blood loss was approximately 800 to 1000 ml and total intraoperative fluids transfused were 2000 ml. At the end of surgery, patient was reversed and shifted to the Intensive Care Unit (ICU) with endotracheal tube *in situ* and oxygenation with T piece. Nitroglycerin infusion was continued as the systemic blood pressure was in the higher range. Patient was extubated within an hour as she had good ventilatory efforts. But three hours after extubation, patient had an episode of desaturation and was not maintaining saturation on face mask oxygen, hence was reintubated and put on assist control mode of ventilation. As she had bilateral crepitations, we made a clinical diagnosis of pulmonary edema, and gave i.v bolus of injection lasix 60 mg, i.v morphine 4 mg. The oxygen saturation normalised, but blood pressure continued to be high. Next morning, she was extubated after she fulfilled all extubation criteria. After seeking cardiologist opinion, oral antihypertensives enalapril and amlodepine were started in an attempt to wean off intravenous nitroglycerin infusion. Patient was discharged from ICU after pressures were stabilised with oral antihypertensives after two days. Histopathology confirmed the diagnosis of paraganglioma and the urinary VMA levels normalised. An USG abdomen, ten days post surgery revealed a left suprarenal mass 4.3 × 4 cms, for which an endocrinologist opinion was sought. But patient did not comply and was lost to follow up.

Discussion:

Carotid body paragangliomas are rare non chromaffin tumors arising from chemoreceptor cells of the carotid body and has ability to secrete catecholamines [1]. Exact cause of carotid body tumours is not known, but their increased incidence in people residing in high altitudes and Chronic Obstructive Pulmonary Disease (COPD) suggest a role of chronic hypoxia [2]. They are typically slow growing non functional tumours with increased incidence in females [2]. Even though asymptomatic in the early stages, eventually most of the patients develop symptoms such as neck pain, asymmetry, enlarging mass, hoarseness (due to laryngeal or vagal nerve involvement), dysphagia (due to glossopharyngeal and hypoglossal nerve involvement) and syncope (due to carotid sinus or internal carotid artery compression). Cranial nerves may be involved in up to 20% of cases. If the tumours are secreting catecholamines patient may have labile blood pressure, headaches, cardiac arrhythmias, weight loss and unusual flushing or sweating [3]. These features occur when catecholamines are produced at 4 to 5 times the normal. Diagnosis is usually by USG, CT scan, MRI angiography and serum urinary levels of catecholamine metabolites [4]. Our patient had a marginal increase in the blood pressure and increased urinary VMA levels. Even though FNAC was done in our patient for diagnosis, it is not recommended as routine investigation in carotid body tumour as it can lead to fatal bleeding. The treatment trend for such tumours is increasingly surgical, partly because of improved preoperative imaging and better results post surgery; radiotherapy does remain an option in few cases.

Anaesthesia in such patients can be a formidable challenge and attention has to be given to proper pre operative preparation with alpha blockers, control of intraoperative hypertensive episodes (due to intubation or due to surgical handling of tumours) with appropriate antihypertensives and adequate depth of anaesthesia, blood loss

replacement and cerebral oxygenation monitoring particularly if cross clamping of artery is planned [5]. Post operative complications like hypotension (due to fall in level of catecholamines) and neurological insults (hemiplegia, hypoglossal or recurrent laryngeal nerve palsy, Horner's syndrome etc) have been previously documented [1].

The intra operative hemodynamic variations in our patient could be because of surgical handling of tumour. But it was effectively managed with pre emptive clonidine, nitroglycerin, labetalol boluses and deepening the plane of anaesthesia with propofol infusion. In our case tumour was adherent to carotid vessel (shamblin type 2) [6], there was no invasion into the artery and the tumour was planned to be resected without clamping of artery. We did not use any brain oxygenation monitoring, but this is recommended to be used if available, nevertheless we did give intravenous thiopentone as a brain protective strategy.

Cranial nerve involvement could have been contributed to desaturation postoperatively in our case as our patient did complain of dysphagia to solids in the preoperative period. Cranial nerve deficits can worsen post operatively due to surgical injury or edema. Post operative aspiration and airway obstruction risk is high in such patients (up to 20%).

A normal postoperative course in carotid body tumour excision is profound hypotension requiring vasopressors due to sudden fall in catecholamines following surgical removal of tumour [1]. However, our case did not manifest like this. In fact the blood pressure remained high with an episode of postoperative pulmonary edema. After USG finding of adrenal tumour postoperatively, retrospectively we could explain the high pressures.

Conclusion:

Hypertensive complications can occur during carotid body tumour excision and adrenal tumours can co exist with carotid body tumours and one should look out for these in the pre anaesthesia check up. Anaesthetic management of such tumour excision requires good pre operative blood pressure optimisation and being prepared for unusual blood pressure changes both in the intra operative and post operative period.

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