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Case Report

Anaesthetic challenges in carotid body tumour resection: a case report and review of literature

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ABSTRACT

Carotid body tumour (CBT) is an extremely rare, non-chromaffin paraganglioma arising from chemoreceptor cells at the carotid bifurcation. Reported incidence is 1-2 per 100,000. Surgical excision of the tumour, the definitive treatment, poses several anaesthetic challenges and a high incidence of perioperative morbidity and mortality (20-40%). Very few cases have been reported so far. We report the anaesthetic management of a case of CBT excision with a review of recent literature on the same.

Keywords: Carotid body tumour, Hypotensive anaesthesia, Dexmedetomidine, Atrial fibrillation, Cranial nerve injury

INTRODUCTION

CBT is a rare, non-chromaffin paraganglioma arising from chemoreceptor cells at the carotid bifurcation. Reported incidence is 1-2 per 1,00,000.[1] Though slow-growing, painless and usually benign, they can invade or exert pressure on neighboring neurovascular tissues. Due to tendency for malignancy, surgical excision should be performed at the earliest. Surgical excision of the tumour poses several anaesthetic challenges and a high incidence of perioperative morbidity and mortality (20-40%).[2]

CASE REPORT

A 34year female patient weighing 39kg, presented with a soft, painless, gradually progressive swelling below the right pinna and giddiness since two years. She denied history of trauma, fever or cough. The lump was soft, non-pulsatile, non-collapsible with no bruit heard over it. Complaints like dysphagia, hoarseness, partial paralysis/numbness of tongue and vision changes were absent. General physical and systemic examination was unremarkable with heart rate (HR) 78/min and blood pressure (BP) 122/84mmHg. Airway assessment was normal. All routine haematological investigations including thyroid function tests were within normal limits. Indirect laryngoscopy (IDL) showed a bulge on the posterior pharyngeal wall on right side and mobile bilateral vocal cords. Ultrasonography (USG) neck showed a hyper-echoic lesion inferior to the right parotid gland with vascularity within and involvement of the right internal jugular vein (IJV). Computerised Tomography (CT) scan neck revealed a 6.7 x 5x 2.5cm, well-defined, strongly enhancing soft tissue lesion in the right carotid space extending from the bifurcation of common carotid artery (CCA) to the jugular fossa along the internal carotid artery (ICA) causing mild splaying, encasing right CCA and compressing and narrowing right IJV. Digital subtraction angiography (DSA) confirmed the diagnosis of CBT (hyper-vascular mass at carotid bifurcation with supply predominantly from ascending pharyngeal artery and posterior auricular artery). Left ICA, CCA and vertebral artery angiogram was normal.

Excision of right-sided CBT (Shamblin II) under general anaesthesia was planned. Preoperatively, adequate blood and intensive care unit (ICU) bed with ventilator was reserved. High risk consent was taken. Patient was premedicated with tab Alprazolam 0.25mg and tab ranitidine 150 mg night before surgery. In the operation theatre, standard ASA (American Society of Anaesthesiologists) monitoring was instituted, 16G intravenous (IV) cannula secured and Ringer's lactate infusion started. Radial artery cannulation was done for invasive arterial pressure monitoring and Cavafix secured in left cubital vein for central venous pressure monitoring. Conventional general anaesthesia with controlled ventilation was used. Intra-operatively, hypotension was induced with inj. Dexmedetomidine infusion (0.5mcg/kg/hr) and sevoflurane (1-2%) to maintain mean BP 80-90 mmHg and HR 60-70 beats/min. Mild hypothermia (31°C) was maintained. Sudden rise in BP at the time of tumor handling was managed by increasing sevoflurane concentration and IV boluses of inj. propofol. Immediately following tumor excision, BP fell to 80/50mmHg, which was managed by decreasing rate of dexmedetomidine infusion and sevoflurane concentration, fluid boluses and colloid infusion. Surgery lasted three hours. Intra-operative blood loss was 600 ml; 2units whole blood was transfused. At the end of the surgery, residual neuromuscular blockade was reversed with inj. Neostigmine (2.5mg) and inj. Glycopyrolate (0.5mg) IV and patient was extubated uneventfully. Post-extubation, deviation of the tongue to the right side was noticed. Inj. dexamethasone was started. Patient was shifted to ICU for observation.

Twelve hours postoperatively patient developed tachycardia (HR 180-188/min) & mild ECG changes. 2D-Echocardiography and cardiac screening were unremarkable. It was treated as sinus tachycardia. Tachycardia persisted after adequate hydration, analgesics, and removal of Cavafix. Repeat ECG revealed atrial fibrillation with a rapid ventricular rate. Inj. amiodarone 150 mg IV bolus followed by infusion was administered. HR settled after three hours. Post-operative day 2, patient had mild dysphagia and was unable to speak properly. Oropharyngeal candidiasis was diagnosed and treated with tab. fluconazole. Dysphagia persisted. IDL on day 4 revealed right vocal cord paresis for which speech and swallowing therapy were initiated and continued. Patient was shifted to ward and discharged on day 12. Histopathology confirmed paraganglioma.

DISCUSSION

The carotid body, first described by von Haller in 1743,[3] is a highly specialized chemoreceptor organ situated at the bifurcation of CCA, which detects changes in arterial oxygen tension.[4] It is stimulated by hypoxia, hypercapnoea and acidosis and in response, controls the BP, HR and respiration by increasing the sympathetic flow.[5] Chronic hypoxemia-sustained/intermittent stimulates hyperplasia of the gland.[6]

It receives blood supply predominantly from ascending pharyngeal artery (branch of ECA) and is innervated by glossopharyngeal and vagus nerves.[3,7]

CBTs are rare neoplasms (~0.5% of all body tumours).[8] Though slow-growing and benign, they can invade/exert pressure on neighbouring neurovascular tissues.[9] 5-7% may be malignant.[10]

History of uncontrolled/recently diagnosed hypertension, tachycardia, flushing and excessive sweating suggests a catecholamine-secreting CBT.[11-13] We did not do preoperative catecholamine studies as our patient had no such symptoms and was non-affording.

Biopsy, in a suspected case of CBT may be catastrophic and hence, is contraindicated.[14] Diagnosis is made by USG, CT scan and MRI, with carotid angiography being the gold standard.[15]

Surgery is the definitive treatment for CBT. Radiotherapy is reserved for elderly and those in poor general condition.[16-18]

Shamblin's tumor classification is based on the size of the tumour and difficulty of surgical resection.[8] Shamblin II/III tumours require more extensive surgery and are more commonly associated with vascular injuries and cranial nerve injuries.[19]

Administration of alpha-blockers preoperatively and beta-blockers intra-operatively can prevent/control the dangerous surges in BP occurring during induction of anaesthesia and surgical manipulation during excision of functional paragangliomas.

Risk of considerable rapid blood loss during surgical resection[20] necessitates the use of invasive monitoring, hypotensive anaesthesia and availability of minimum four units of cross-matched blood.

Moderate cooling of the patient (31°C) is recommended to prevent cerebral hypoxia.[21] Inj. Thiopentone (3-5 mg/kg/hr) infusion can be used to decreases the cerebral metabolic oxygen demand,[20] if carotid artery has to be clamped.

Intraoperatively, carotid sinus stimulation causes reflex bradycardia which usually responds to IV atropine. Alternatively, infiltration with inj. 2% lignocaine at surgical site may be useful.[6] Our patient, however did not have any bradycardia.

Involvement of cranial nerves (IX, X, XI) by tumor invasion preoperatively, nerve injury intra-operatively, or tissue oedema causing nerve palsy postoperatively may predispose to airway obstruction or aspiration.[22]

Gastroparesis may also increase risk of aspiration necessitating intermittent nasogastric suctioning in the postoperative period.

Figure 1: Swelling under the right pinna.

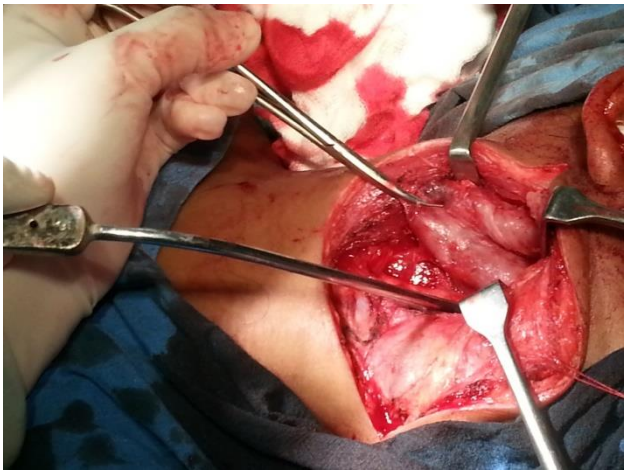

Figure 2: Dissection during resection of a vascular tumour at the bifurcation of common carotid artery.

Figure 3: Removal of a homogeneous rubbery, tumour with smooth contour.

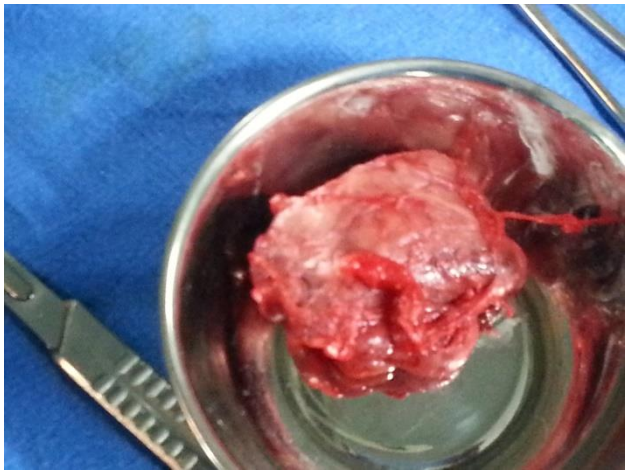

Figure 4: Carotid body tumor specimen after excision.

Frequent observation for stridor and wheezing following extubation and prophylactic ventilatory support is indicated due to the dynamic nature of oedema around cranial nerves postoperatively.[20]

Other postoperative neurological complications like hemiplegia, recurrent laryngeal nerve palsy, Horner's syndrome and hypoglossal nerve palsy may be unavoidable in difficult surgery.[6]

CONCLUSION

Utmost vigilance by the anesthesiologist is essential during CBT excision. A detailed history, specific investigations, proper optimization of the patient, invasive monitoring, hypotensive anesthesia and a high index of suspicion for possible complications with prompt management result in successful outcome.

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