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Case Report

Bilateral cerebral oxygen saturation monitoring in carotid body tumour excision under general anaesthesia: a case report

Jagriti Singh*, Gayatri Karandikar, Kaustubh Zirpe, Balasaheb Govardhane

Department of Anaesthesiology, Dr. D. Y. Patil Medical College, Nerul, Navi Mumbai, Maharashtra, India

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***Correspondence:**
Dr. Jagriti Singh,
E-mail: jagritisingh2060@gmail.com

ABSTRACT

Carotid Body tumour is an uncommon non-chromaffin paraganglioma arising from chemoreceptor cells at the external and internal carotid artery bifurcation. The reported incidence is 1-2 cases per 1,00,000 population. Surgical excision is extremely difficult and possess challenges to both surgeon and the anaesthetist. We report here a case of CBT excision with the use of cerebral oximetry to reduce the chances of intra-operative cerebral ischemia.

Keywords: Carotid body tumour, Cerebral blood flow, Hypotensive anaesthesia

INTRODUCTION

Carotid body tumour is an extremely rare tumour with a reported incidence of 1-2 cases per 1,00,000 population.[1] It is an extra-adrenal paraganglioma arising at the bifurcation of internal and external carotid arteries. Usually, it represents a benign tumour but has a high tendency of local invasion and malignancy. Thus, surgical resection at the earliest is preferred. Due to its hypervascular nature and relation to the surrounding anatomical structures, the excision of carotid body tumour possess several challenges for anaesthetists. Anaesthesia challenges faced by anaesthetists during the surgery are haemodynamic variations and maintenance of cerebral perfusion pressure to prevent cerebral ischemia. In this case report, we used a Masimo cerebral oximeter to monitor cerebral perfusion for carotid body tumour excision surgery.

CASE REPORT

A 51-year-old male presented with a history of fall in the washroom at home after having an episode of giddiness. Fall was not associated with a history of head trauma, loss of consciousness or seizure. Patients also complained of repeated episodes of giddiness for 1 year. On examination, the patient had a right para symphysis fracture of the mandible for which he was planned to be operated on by an oral maxillofacial surgeon.

On further examination patient had a soft, painless, gradually progressing swelling approximately measuring 3×3 cm in the left submandibular region. The swelling was mobile, non-collapsible, with no pressure symptoms. Pulsations were felt on deep palpation and bruit was heard on auscultation. Computerized tomography (CT) Neck angiogram revealed a well-circumscribed lobulated heterogenous soft tissue round lesion measuring 3.2×3.1×4.3 cm at C4-C6 level involving left carotid space. The lesion was anteromedially abutting the left submandibular gland, posteromedially abutting the left common carotid artery compressing the left internal jugular vein. All routine investigations were within normal limits.

Excision of left-sided CBT was planned under general anaesthesia. The patient was premedicated with a tablet of alprazolam 0.5 mg orally the night before surgery. After taking informed high-risk consent and reservation of adequate blood and Intensive care unit bed, patient was taken to the operation theatre and standard ASA (American society of anaesthesiology) monitors 3 lead

echocardiography, oxygen saturation pulse oximetry, non-invasive blood pressure (NIBP) was attached. A large bore 16 G IV cannula was instituted, and a ringer lactate drip was started at 80 ml/hour. Before anaesthesia induction, an adult adhesive O3 sensor of Masimo Cerebral oximeter was attached over each frontotemporal area and baseline regional oxygen saturation (rSO_2) readings were measured. The readings were 65% and 73% for the left and right sides respectively.

Following routine general anaesthetic induction with inj. fentanyl 2 mcg/kg, inj. propofol 2 mg/kg, and inj. atracurium 0.5 mg/kg and patient was intubated with north pole endotracheal tube size 7.5 mm ID and was put on controlled mechanical ventilation. The right subclavian vein was monitored to measure central venous pressure (CVP). Intra-operatively CVP was maintained between 8-10 mmHg. Right radial artery catheterization was done. Hypotensive anaesthesia was induced using inj. dexmedetomidine 0.5 mcg/kg/hour and sevoflurane 1-2% to maintain mean arterial pressure between 55-65 mmHg. Intraoperative hypotensive incidences were managed using bolus doses of 5 mg ephedrine and crystalloid fluid boluses. The sudden rise in blood pressure was managed using inj. propofol boluses, sevoflurane 1.5-2% and inj. esmolol 5 mg boluses. One episode of intraoperative bradycardia was managed using inj. atropine 0.6 mg IV. Intraoperative cerebral monitoring was achieved by using cerebral pulse oximetry with maximum drop in regional oxygen saturation (rSO_2) of 65% and 61% for left and right side respectively. Post-excision of the tumour sudden fall in blood pressure to 76/40 mmHG was observed. This was managed by titrating Sevoflurane and inj. dexmedetomidine infusion. The surgery for the excision of CBT lasted for 4 hours following which the oral maxillofacial surgeon operated on the mandible and fixation was done using mini plates and screws.

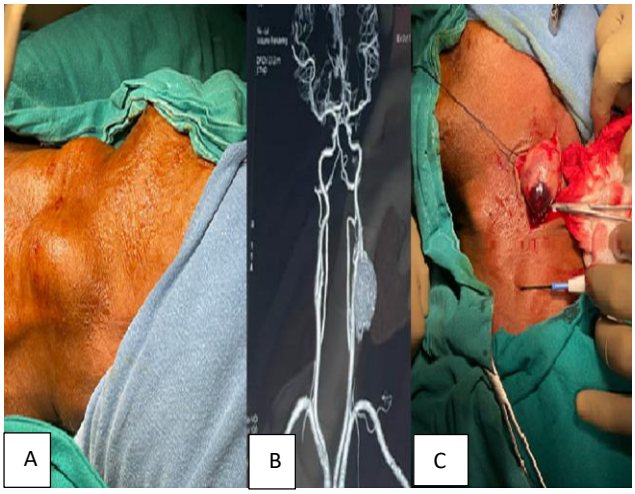

Figure 1 (A-C): Carotid body tumour swelling in the left submandibular region, CT angiogram report showing carotid body lesion and hypervascular carotid body tumour excision.

Figure 2: Baseline cerebral pulse oximeter values before anaesthesia induction.

Figure 3: Maximum drop in cerebral pulse oximeter value during surgery.

Intraoperative total blood loss was 900 ml and 1 pint of packed red blood cells was transfused intraoperatively. At the end of the surgery, the patient was reversed with inj. glycopyrrolate 8 mcg/kg and inj. neostigmine 0.05 mg/kg and the patient was extubated on the operation theatre table. For postoperative observation patient was shifted to the intensive care unit. Postoperatively, the patient was awake, oriented and without any sensory and motor deficit. Postoperative pain was managed using inj. paracetamol 1 gm IV TDS and inj. tramadol 50 mg IV SOS. Arterial blood gas analysis, complete blood count, and chest x-ray were all within normal limits. The patient was shifted to the ward the coming morning and discharged on day 10 post-surgery.

DISCUSSION

Carotid body tumours are paragangliomas also known as chemodectomas which are very rare. These paragangliomas arise from chemoreceptor cells. Von Haller in 1743 described carotid body tumour for the first

time. These tumours are usually benign although less than 10% of paragangliomas are malignant. Its incidence is 1-2 in 1,00,000 patients.[1,2] It is more frequent in women than men and can occur at any age but frequently appears in the fourth and fifth decade.[3]

The carotid body is a highly specialized organ 2-6 mm in size which is usually situated in the adventitia of common carotid artery bifurcation that is between ICA and ECA. These are vascular lesions, and their feeding vessels run primarily from the external carotid artery. It has two types of cells glomus type I and II which are similar to neurons. The carotid body through complex neural pathways controls cardiovascular and respiratory systems and also maintains blood temperature.[4,5] Reflex adjustment of respiration during hypoxia, hypercapnia, and acidosis according to the arterial blood gas values is the major function of the carotid body.

These rare neuroendocrine neoplasms are often invasive, slow growing, mobile, non-tender and sometimes even go unnoticed in individuals. These tumours are pulsatile as they are present in the vicinity of the carotid artery. Medial enlargement can produce, effects on cranial nerves IX, X, and XII, which in turn result in clinical signs such as dysphagia, odynophagia and hoarseness. Sympathetic chain involvement can cause miosis, anhydrosis and ptosis. Most of the tumours are non-functional; only 1-3% secrete mediators like histamine, serotonin, norepinephrine, and epinephrine. Catecholamine production can give rise to blushing, palpitations, and fluctuating hypertension.

Shamblin classified these tumours into three categories on gross examination. In our case tumour belonged to Shamblin class II.

Carotid body tumours can be diagnosed by using different imaging modalities like USG, MRI, CT angiography, etc. MRI shows a typical 'Salt and pepper' appearance where salt represents low signal flow voids and pepper represents high signal foci haemorrhage.[6] CT angiography shows hypervascular mass, tumour blush, draining veins and enlarged feeding arteries.[7] Indium-111 octreotide used in nuclear imaging studies is also useful in diagnosing paragangliomas. Biochemical screening for plasma metanephrines and urine catecholamines can give the diagnosis of paragangliomas.

Surgical excision is the mainstay treatment for carotid body tumours but it is challenging for both anesthesiologists and surgeons as this tumour lies in the vicinity of major nerves and vessels.[8,9]

The surgery can be done under general anaesthesia as well as cervical plexus block. We preferred general anaesthesia in our case because the patient had to undergo two procedures; carotid body tumour excision and mandible fracture repair with open reduction internal fixation with mini plates and screws. Cervical plexus block provides better hemodynamic stability, and continuous neuromonitoring and avoids postoperative cardiac complications. Invasive arterial blood pressure monitoring is necessary for beat-to-beat monitoring as while handling the tumour there are chances of fluctuations in blood pressure. Preoperative use of alpha-blockers can prevent hypertension intraoperatively. Sudden hypertension can be managed by propofol boluses, titrating inhalational anaesthetic doses and using infusions of drugs like nitroglycerine, sodium nitroprusside, and beta blockers while hypotensive episodes are usually managed by small boluses of phenylephrine or ephedrine. In the case of bradycardia, a bolus of atropine can be given.

A central venous line should be established for CVP monitoring and fluid management. CVP should be maintained between 8-10 cmHg. Intraoperative adequate fluid administration should be done using crystalloids like RL, NS and colloids. Adequate reservation of blood and blood products as well as the cross match should be kept ready. Urine output should be maintained up to 0.5-1 ml/kg/hr.

Intraoperative neurological monitoring in a patient under general anaesthesia is very important to detect early neurological insults which are potentially reversible. Early interventions done to maintain cerebral oxygen saturation have shown successful outcomes in 80-90% of the cases.

Other neuromonitoring modalities like electroencephalogram, sensory evoked potentials, AEP, transcranial doppler for flow velocities, stump pressure, and jugular venous oxygenation for perfusion can also be used.[7]

Accidental arterial puncture or sudden bleeding from the tumour can cause hypotension and sudden hemodynamic changes cannot be allowed. Hence vigilant blood loss monitoring is necessary.

Cerebral perfusion should be maintained at all times as due to intraoperative clamping of CCA chances of cerebral ischaemia are high. Cerebral oximetry uses the method of light transmission and absorption to measure the ratio of oxygenated to deoxygenated haemoglobin in the cerebral tissue. cerebral oximeters use only near-infrared spectroscopy (NIRS) without plethysmography.[10,11]

Cerebral protective strategies should be used. Hypocapnia and hypercapnia should be avoided as it will cause a steal phenomenon and worsens ischaemia. Hypothermia is also considered to be cerebral protective. Thiopentone infusion decreases the cerebral metabolic rate of oxygen which provides neuroprotection. Volatile anaesthetics reduce cerebral vascular smooth muscle tension and in turn increase cerebral blood flow.

The patient can have post-operative hemiplegia, Horner's syndrome, airway obstruction, recurrent laryngeal nerve palsy, hypoglossal nerve palsy due to injury to cranial nerves IX, X, XII or due to postoperative oedema around the nerves.

CONCLUSION

A simple continuous non-invasive cerebral perfusion monitoring by cerebral oximetry for maintaining cerebral perfusion pressure may improve the patient outcome in carotid body tumour excision surgery.

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