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Anesthesia: Essays and Researches

Case Report

Anesthetic management of carotid body tumor excision: A case report and brief review

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Abstract

Carotid body tumor (CBT) is a rare tumor, which arises at bifurcation of carotid artery from chemoreceptor cells. These cells sense the partial pressure of oxygen and carbon dioxide from the blood. Hence, carotid body plays an important role in the control of ventilation during hypoxia, hypercapnia, and acidosis. The tumor arising from these cells is benign and has tendency to turn out malignant. This tumor is found in persons who live at high altitudes. Removal of tumor poses several anesthetic challenges and perioperative morbidity or mortality. We report successful anesthetic management of CBT excision.

Key words: Anesthesia, carotid body tumor, complications, excision

INTRODUCTION

The carotid body was first described by von Haller in 1743.[1] Carotid body tumor is a rare non-chromaffin paraganglioma arising from chemoreceptor cells found at carotid bifurcation but constitute the majority of head and neck paragangliomas.[2] These tumors are slow-growing tumor and can invade or exert pressure on neighboring neurovascular tissues.[3] These tumors can present at any age and seen in equal frequency in either sex.[4] The tumor is benign and has tendency to turn out to malignant, and the incidence is around 1-2 per 1,00,000.[5] Hence, surgical excision should be performed as early as possible to avoid the local spread and the possibility of eventual metastasis.[6] This article highlights the anesthetic management and the problems encountered during excision of CBT.

CBTs are painless tumors that grow very slowly and originate from neural crest cells that have migrated with ganglionic cells from the automatic nervous system. Digital subtraction angiography is regarded as the gold standard for the final diagnosis of CBTs.[7] Treatment of choice for CBT is surgical excision. Radiotherapy is being reserved for inoperable, recurrent, and/or bulky tumors.[8]

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CASE REPORT

A 40-year-old man presented swelling on the right side of neck since 2 years, which was insidious in onset, gradually progressive, and painless. There was no history of trauma, fever, or cough. On examination, nonpulsatile mass was seen on the right side of the neck, which was measuring about 3 × 4 cm, nontender, noncollapsible, and absence of bruit. On general physical examination, the patient

was 58 kg, 162 cm in height, pulse rate of 68/min, and blood pressure of 130/70 mm Hg. The airway assessment showed Mallampati grade II, thyromental distance of 6 cm, and adequate mouth opening. Cardiorespiratory per abdomen and central nervous system examination were within the normal limit.

Preoperative hemogram, Random blood sugar, electrocardiogram, electrolytes, renal function tests, and chest radiograph were normal. Special investigations such as Doppler study showed hypervascular lesion at the right carotid bifurcation suggestive of CBT. On magnetic resonance imaging, large well-defined ovoid intensely enhanced mass over right carotid bifurcation insinuating both internal and external carotid arteries and confirmed the findings of the Doppler study. The patient was diagnosed as having right CBT and planned for excision under general anesthesia.

The patient was premedicated with Tab. Diazepam 5 mg and Tab. Ranitidine 150 mg per oral night before surgery and on the day of surgery in the morning and was kept nil per oral for 6 h before surgery. A maintenance fluid Ringer lactate was given at the rate of 100 ml/h, throughout surgery through 18 G intravenous cannula. On arrival to the operating room, noninvasive blood pressure (BP) 128/74 mmHg, heart rate of 86/min, regular, respiratory rate of 14/min, and oxygen saturation of 98% were recorded. ECG and nasopharyngeal temperature monitoring were established, together with monitoring of invasive BP and central venous pressure (CVP). A left radial artery was cannulated with 20 G under local anesthesia for invasive BP monitoring.

The patient was premedicated with Inj. Midazolam 0.05 mg/kg and Inj. Fentanyl 2 mcg/kg intravenously. Preoxygenation was done for 3 min and induced with Inj. Thiopentone 5 mg/kg. Tracheal intubation was facilitated by Inj. Vecuronium 0.1 mg/kg. A cuffed Portex oral 8.5-mm endotracheal tube was passed. Anesthesia was maintained with oxygen and nitrous oxide (1:1), an end-tidal concentration of isoflurane of 0.8, and a minimum alveolar concentration of 1.1 with volume-controlled ventilation. Left subclavian vein was cannulated to monitor CVP. Inj. nitroglycerine was started, and the infusion rate was adjusted to keep mean blood pressure of 80-90 mm Hg. Since the operation theater temperature was kept at 21°C, there was a drift in patient temperature to 34-35°C, which was acceptable during the excision of tumor.

Total surgical duration was 2 h and tumor was resected safely [Figure 1], and blood loss was around 300 ml, which was within allowable blood loss in this patient. The vital parameters remained stable throughout the course of surgery, except for two episodes of bradycardia which was observed during tumor excision. Bradycardia reverted to normal once the tumor handling was stopped, and the surgeon was requested to infiltrate 2% Inj Lignocaine locally. The intravenous fluid was titrated to maintain CVP of 10-12 mm Hg.

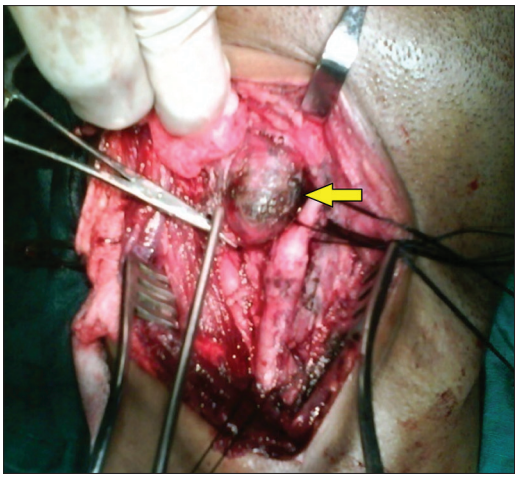

Figure 1: Arrow showing carotid body tumor

Because the surgery was uneventful, the patient was extubated after residual neuromuscular blockade was reversed with IV Neostigmine 2.5 mg and Glycopyrolate 0.4 mg and once the extubation criteria were met. The patient was conscious, oriented, obeying oral commands, and pain free. Postoperatively, the patient received Inj. Diclofinac 75 mg IV infusion twice a day and Inj. Tramadol 100 mg as and when the visual analogue score was equal to or more than 5. The patient was shifted to high dependency unit (HDU) for further monitoring and was uneventful. The patient was discharged from HDU after 48 h.

DISCUSSION

Carotid body plays an important role in the control of ventilation during hypoxia, hypercapnia, and acidosis as it senses partial pressure of oxygen and carbon dioxide from the blood.[9] CBT or chemodectomas are rare tumors, which are considered to be non-chromaffin paragangliomas arising from chemoreceptor cells present at the bifurcation of carotid artery.[10] The CBTs are benign with an incidence of 1-2 per 1,00,000 population.[5] About 5-7% of tumors are malignant. CBTs are most commonly noted in fifth or sixth decades and are slow growing painless lump in the neck.[11] The patient may present with symptoms of dysphagia, choking, or hoarseness depending upon the cranial nerve involvement as there is a close anatomical relationship with cranial nerves X-XII.[12] Diagnosis of the tumor is made by ultrasound, CT, or MRI, but digital subtraction angiography being the gold standard for diagnosis.[7] The differential diagnosis of CBT is medullary thyroid carcinoma and neuroendocrine carcinoma, middle ear adenoma, meningioma, and schwannoma.[13] Occasionally, these tumor secrete catecholamines and serotonin and may be associated with phaeochromocytomas, necessitating preoperative catecholamine studies in such cases. Because our patients did not have symptoms suggestive of

inappropriate catecholamine secretion, we did not do such tests due to cost factor.

The anesthetic techniques that are practiced for CBT excision are continuous cervical plexus block and general anesthesia. The advantages of regional technique over general anesthesia are easy to learn, lower conversion rate to general anaesthesia, continuous neurological assessment, better hemodynamic stability, a decrease in incidence of stroke and cardiovascular adverse events, faster recovery, and reduced hospital stay.[12] Whatever the technique of anesthesia used, the main aim is to maintain optimal cerebral perfusion and better operating conditions for the surgeon. Because our patient did refuse a regional technique and surgeon preferred general anesthesia, we did select general anesthesia for this case. It is important to monitor the cerebral blood flow (CBF) intraoperatively. Many invasive and noninvasive monitors are available, namely, transcranial Doppler that helps to diagnose cerebral vasospasm and cerebral vascular response to carbon dioxide. Jugular venous oximetry to obtain three parameters that are helpful in assessing the adequacy of CBF, namely, jugular venous oxygen saturation ($SjVO_2$), cerebral arteriovenous oxygen difference - the difference between arterial and jugular venous oxygen content ($A\text{-}VDO_2$)-and cerebral oxygen extraction - the difference between SaO_2 and $SjVO_2$ (CEO_2). The cerebral ischemia can be suspected at the earliest stage by a low $SjVO_2$, a high (A-V) DO_2, or a high CEO_2 indicating increased extraction of oxygen. Near infrared spectrometry helps in quantification of CBF, cerebral blood volume, regional cerebral oxygen saturation, and cerebral metabolism. The technique is based on the principle of absorption of near-infrared light by chromophores in the body such as oxyhemoglobin (OHb), deoxyhemoglobin (dOHb), and cytochrome aa3.[14]

Considering different wavelengths, it is possible to simultaneously and continuously measure the changes in OHb, dOHb, and cytochrome aa3 in brain from where regional CBF, cerebral blood volume, cerebral oxygen saturation, and cerebral metabolism are measured.

In vivo optical spectroscopy successfully used as a surrogate for cerebral perfusion monitoring in a patient undergoing excision of a recurrent CBT with prior contralateral CBT excision.[15] With the use of electroencephalogram (EEG), ischemia and hypoxia are characterized by any change in EEG and may represent cerebral ischemia. Slowing and flattening of EEG progressing to isoelectricity are the characteristic changes. Other monitoring system that may contribute is somatosensory evoked potential. Such monitoring will help timely intervention to reduce or to prevent patient morbidity and/or mortality. Because we did not have such specialized equipment, we did not monitor.

Hemodynamic instability intraoperatively like bradycardia and hypotension is commonly seen. In this case, there were two episodes of reflex bradycardia that got reverted to normal after stopping tumor handling. Sometimes Inj. Atropine is required to revert it back to normal heart rate or Inj. Lignocaine is infiltrated locally to avoid bradycardia. Most common cause of hypotension is because of massive and rapid blood loss. This can be treated with colloids and blood replacement depending upon the amount of loss. CVP was monitored in our patient and maintained at 8-10 mm Hg. The blood loss throughout the procedure was 300 ml and was allowable in this patient.

In procedure where the carotid artery is clamped, it is necessary to protect brain pharmacologically by using barbiturates, infusing non-dextrose containing fluids to prevent exacerbation of ischemic cerebral damage.[16] Anesthetic drugs do have a significant effect on cerebral blood vessels, namely, a single dose of Inj. Thiopentone causes cerebral metabolic rate of oxygen suppression for 10 min and an infusion of 3-5 mg/kg/h has shown neuroprotection,[17] and volatile anesthetic agents cause reduction in the tension of cerebral vascular smooth muscle resulting in vasodilatation thereby increasing in CBF. Depending upon the concentration of the inhalational agent, there will be reduction in neuronal function and metabolic demands. Temperature management is also important as the cerebral metabolic rate is reduced by 7% for each 1°C decrease in body temperature. The hyperventilation should be avoided in this case as it causes vasoconstriction of cerebral blood vessels and thereby decreased delivery of oxygen. Postoperative complications noted are stroke, permanent and transient cranial nerve palsy or paresis, profound hypotension, Horner's syndrome, and sometimes, respiratory depression.[18,19] Hence, monitoring in HDU postoperatively for these complications is important.

CONCLUSION

A high vigilance is necessary for an anesthesiologist during the excision of CBT. Cerebral protection, hypotension, and management of arrhythmias are challenging. A high index of suspicion should be made for possible complications that may occur during the perioperative period. The successful management depends upon detailed history, specific investigation, invasive monitoring, optimization, and managing postoperative complications.

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