CASE REPORT

Bali Journal of Anesthesiology (*BJOA*) 2019, Volume 3, Number 2: 143-145
E-ISSN: 2549-2276

Published by DiscoverSys

Anaesthesia management of carotid body tumour excision

Vijaya Kumara*

ABSTRACT

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

Keywords: anaesthesia, carotid body tumour, complications, excision
Cite This Article: Kumara, V. 2019. Anaesthesia management of carotid body tumour excision. *Bali Journal of Anesthesiology* 3(2): 143-145.
DOI:10.15562/bjoa.v3i2.186

Department of Anesthesiology
Kasturba Medical College, Manipal
India

INTRODUCTION

The carotid body was first described by von Haller in 1743.[1] CBT 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 are slow-growing tumours and can invade or exert pressure on neighbouring neurovascular tissues.[3] These tumours can present at any age and seen in equal frequency in either sex.[4] The tumour is benign and has a 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 anaesthetic management and the problems encountered during the excision of CBT.

CASE REPORT

A 42-year-old female weighing 55 kg presented with a history of swelling on the left side of the neck near the angle of the jaw for three years. The lump was slow-growing, painless, soft and non-pulsatile, of 68 cm in size. There were no other pathological findings. She was normotensive; her indirect laryngoscopy showed normal vocal cord movements, and there was no extension of tumour in the hypopharynx.

Computed tomography (CT) scan of the neck showed well-defined oval soft-tissue mass pushing the left-sided carotid vessels slightly medially and anteriorly. Magnetic resonance imaging (MRI) angiography showed a large well-defined soft-tissue lesion just above the left carotid artery bifurcation splaying internal and external carotid arteries and being fed by branches of these vessels. The patient was diagnosed as having left CBT and planned for excision under general anaesthesia. Her preoperative biochemical investigations, ECG and X-ray chest were within normal limits. Preoperatively vascular and neurosurgeon were consulted, cerebral protection therapy was discussed, and synthetic graft material was made available.

The patient was premedicated with pantoprazole 40 mg and metoclopramide 10 mg per oral the night before surgery and on the day of surgery in the morning and was kept nil per oral for six hours before surgery. A maintenance fluid RRinger 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) 110/70 mmHg, heart rate of 72/min, regular, respiratory rate of 16/min, and oxygen saturation of 100% were recorded. ECG and nasopharyngeal temperature monitoring were established, together with monitoring of invasive BP and central venous pressure (CVP). A right radial artery was cannulated with 20 G arterial cannula under local anaesthesia for invasive BP monitoring.

The patient was premedicated with midazolam 0.02 mg/kg and fentanyl 2 mcg/kg intravenously. Preoxygenation was done for three minutes and induced with propofol 2 mg/kg. Tracheal intubation was facilitated by vecuronium 0.1 mg/kg. A cuffed Portex oral 7-mm endotracheal tube was passed. Anaesthesia was maintained with oxygen and nitrous oxide, an end-tidal concentration of isoflurane of 0.8, and a minimum alveolar concentration of 1.2 with volume-controlled ventilation. The right internal jugular vein was cannulated to monitor

*Correspondence to:
Vijaya Kumara, Department of Anesthesiology, Kasturba Medical College, Manipal, India,
vijaya.kumara@manipal.edu

CVP. Tranexaemic acid 1 g IV given slowly. Since the operation theatre temperature was kept at 21°C, there was a drift in patient temperature to 34-35°C, which was acceptable during the excision of the tumor. Dexmedetomidine infusion was started at 0.7 microgram/kg/hour after bolus dose of 1 microgram/kg over 10 minutes. Throughout the procedure, the heart rate was maintained around 65/minute and blood pressure around 90/60.

The total surgical duration was 140 minutes, and the tumour was resected safely, and blood loss was around 700 ml. The vital parameters remained stable throughout the course of surgery, except for an episode of hypotension which was managed by phenylephrine 100 mcg. The intravenous fluid was titrated to maintain CVP of 10-12 mm Hg.

Surgery was uneventful, and the patient was extubated after reversal of residual neuromuscular blockade with neostigmine 2.5 mg and glycopyrrolate 0.4 mg. The patient was conscious, oriented, obeying verbal commands, and pain-free. Postoperatively, the patient was given Paracetamol 1000 mg IV 8 hourly and Tramadol 50 mg BD. The patient was shifted to ICU for further monitoring and was uneventful. The patient was discharged from ICU after 24 hrs.

DISCUSSION

The cells from which CBTs arise normally act as chemoreceptors detecting changes in tension of arterial oxygen. Classically these tumours may be displaced in the lateral plane, but cannot be mobilised in the vertical axis. History of uncontrolled or recently diagnosed hypertension, tachycardia, facial flushing, and excessive sweating suggests the possibility of catecholamine secreting tumour.[7] This warrants appropriate values of serum and urine catecholamine and breakdown products.

If a neck mass is thought to be a CBT based on history and physical examination, biopsy should not be attempted as it can turn out to be catastrophic.[8] Diagnosis can be suggested by CT scan, and more convincing diagnosis can be made by MRI angiography. Temporary balloon occlusion of the carotid artery to assess the adequacy of collateral circulation across the circle of Willis can be done at the time of carotid angiography; however, we did not use this. We used a shunt to bypass the cross-clamping.[9]

More extensive surgery is often required for shambling II and III type tumour.[10] Blood loss during resection and reconstruction may be considerable, and dissection is often difficult. To reduce intraoperative blood loss, we used hypotensive anaesthesia with nitroglycerine (NTG) and clonidine infusion. This was stopped when there was accidental carotid injury requiring rapid transfusion with grafting of the vessel. It demands to keep graft material ready with a minimum of four units of blood cross-matched. Measures for cerebral protection, monitoring for adequacy of cerebral blood flow with EEG and somatosensory-evoked potential, measurement of blood flow using transcranial Doppler must be considered.[11] Mild hypothermia (34-35°C) has been shown to have a protective effect.[12] A single dose of thiopentone causes the cerebral metabolic rate of oxygen ($CMRO_2$) suppression for ten minutes, and infusion of 3-5 mg/kg/h have shown neuroprotection.

There are other points to consider in anaesthetic management. Involvement of cranial nerves may predispose to airway obstruction or aspiration which may occur in one of the several ways: Tumour invasion preoperatively, nerve injury intraoperatively, or tissue oedema causing nerve palsy postoperatively. Ninth, tenth and twelfth cranial nerve dysfunction can predispose to airway compromise either by aspiration or obstruction.[13] Postoperatively, the dynamic nature of oedema around cranial nerves mandates prophylactic ventilator support with frequent observation for stridor and wheezing after extubation.[14]

CONCLUSION

CBT excision requires utmost vigilance by the anesthesiologist. The diagnosis of CBT should be made on the basis of patient's clinical history, physical examination and MRI. Biopsy to confirm the diagnosis might turn out to be catastrophic. Intraoperative techniques to reduce blood loss and arrhythmias must be used, and cerebral protection therapy needs to be considered in the event of brain ischemia. The surgical expertise must be available for neurovascular preservation to ensure safe, yet complete removal of neoplasm. Postoperative care must be taken to check cranial nerve involvement and institute prophylactic ventilation to ensure a safe outcome.

ACKNOWLEDGEMENT

Written informed consent was taken from the patient for publication as a case report.

REFERENCES

1. Milewski C. Morphology and clinical aspects of paragangliomas in the area of head-neck. *HNO*. 1993; 41:526–31.
2. Davidovic LB, Djukic VB, Vasic DM, Sindjelic RP, Duvnjak SN. Diagnosis and treatment of carotid body paraganglioma: 21 years of experience at a clinical center of Serbia. *World J Surg Oncol*. 2005; 3:10.
3. Van den Berg R. Imaging and management of head and neck paragangliomas. *Eur Radiol*. 2005;15:1310–8.

4. Pacheco-Ojeda L. Malignant carotid body tumors: Report of three cases. *Ann Otol Rhinol Laryngol*. 2001;110:36–40.
5. Sevilla Garcia MA, Llorente Pendas JL, Rodrigo Tapia JP, *et al*. Head and neck paragangliomas: Revision of 89 cases in 73 patients. *Acta Otorrinolaringol Esp*. 2007;58:94–100.
6. Luna-Ortiz K, Rascon-Ortiz M, Villavicencio-Valencia V,Granados-Garcia M, Herrera-Gomez A. Carotid body tumors: Review of a 20-year experience. *Oral Oncol*. 2005;41:56–61.
7. Clarke AD, Matheson H, Boddie HG. Removal of catecholamine secreting chemodectoma management. *Anaesthesia*. 1976;31:1225-30.
8. Rosa M, Sahoo S. Bilateral carotid body tumour: The role of fine needle aspiration biopsy in the preoperative diagnosis. *Diagn Cytopathol*. 2008;36:178-80.
9. Little VR, Reilly LM, Ramos TK. Preoperative embolization of carotid body tumours: When is it appropriate? *Ann Vasc Surg*. 1996;10:464-8.
10. Arya S, Rao V, Juvekar S, Dcruz AK. Carotid body tumors: Objective criteria to predict the Shamblin group on MR imaging. *Am J Neuroradiol*. 2008;29:1349-54.
11. Gonzalez M. Vascular disease in Stoelting's anaesthesia and co-existing disease. 5th ed. Philadelphia: Churchill Livingstone; 2008. p. 154.
12. Tamai H, Kuribayashi T, Sawamura S, Sumida T, Chinzei M, Hanaoka K. Perioperative treatment for carotid endarterectomy with induced mild hypothermia: A case report. *Masui*. 2002;31:1132-6.
13. McConkey PP, Kien ND. Cerebral protection with thiopentone during combined carotid endarterectomy and clipping of intracranial aneurysm. *Anaesth Intensive Care*. 2002;30: 219-22.
14. Wee DT, Goh CH. Current concepts in the management of carotid body tumors. *Med J Malaysia*. 2010;65:268-71.

This work is licensed under a Creative Commons Attribution