

Case Report

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Vagal schwannoma mimicking carotid body tumour treated by intracapsular excision: a case report

**Khushbu Solanki^{1*}, Michael Prakasam¹, Ameya Bihani², Shubhanshi Kangloo²,
Pratiksha Pawar², Sachin Thakur¹**

¹Department of Oral and Maxillofacial Surgery, Modern Dental College and Research Centre, Indore, Madhya Pradesh, India

²Department of Head and Neck Oncology, Shalby Hospital, Indore, Madhya Pradesh, India

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***Correspondence:**

Dr. Khushbu Solanki,

E-mail: rahul2018vivaan@gmail.com

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ABSTRACT

Vagal schwannomas are rare benign neurogenic tumours of the head and neck that may closely mimic carotid body tumours (CBTs) on clinical and radiological evaluation. Differentiating the two entities is essential to optimize surgical planning, preserve neural function, and prevent unnecessary morbidity. We present the case of a 23-year-old female with a slowly enlarging, non-pulsatile left neck swelling in the carotid triangle. Clinical examination and magnetic resonance imaging (MRI) suggested a carotid space lesion displacing the common carotid artery anteromedially and internal jugular vein posterolaterally. Preoperative biochemical screening for catecholamine excess was negative. The lesion was excised via a transcervical approach using intracapsular enucleation, with careful preservation of vagal nerve fibres. Intraoperative findings confirmed an encapsulated spindle-shaped tumour arising from the vagus nerve. Histopathology revealed Antoni A and Antoni B areas with Verocay bodies, strongly positive for S-100 protein, consistent with schwannoma. Postoperative recovery was uneventful, with preserved vocal cord mobility and no features of Horner's syndrome. At 12-month follow-up, the patient remained asymptomatic with no evidence of recurrence. This case highlights the diagnostic challenge of differentiating vagal schwannomas from CBTs. MRI vascular displacement patterns-anteromedial displacement of the carotid artery and posterolateral displacement of the IJV-are key imaging clues. Intracapsular excision offers the advantage of neural preservation with low recurrence risk when combined with meticulous dissection and long-term follow-up.

Keywords: Vagal schwannoma, Carotid body tumour, Intracapsular enucleation, Neck mass, Nerve-sparing surgery

INTRODUCTION

Schwannoma is a rare, benign, and encapsulated tumour arising from the nerve sheath. It originates from Schwann cells and typically exhibits a slow-growing behaviour.¹ First identified in 1908 by the pathologist José Verocay from Prague, this tumour can develop along cranial, peripheral, or autonomic nerves-excluding the olfactory and optic nerves, which lack Schwann cells.^{2,3} Approximately 25-45% of schwannomas are located in the extracranial head and neck region, with a predilection for the lateral aspect of the neck.^{4,5}

Representing less than 0.5% of all head and neck tumours, Head and Neck Paragangliomas are rare tumours. Mostly arises in the bifurcation of the carotid artery (i.e. CBTs, splaying internal carotid artery and external carotid artery). Vagal paragangliomas originate from the autonomic component of the vagus nerve (cranial nerve X).

Tumours located within the petrous portion of the temporal bone arise from either the tympanic glomus or the jugular glomus. When the exact origin cannot be

distinguished, they are collectively termed jugulotympanic paragangliomas.⁶

The anatomical location of these tumours significantly influences the presenting signs and symptoms. Most of these neoplasms are non-functional, meaning they do not secrete biologically active substances. However, in approximately 1-3% of head and neck paragangliomas, catecholamine overproduction can occur, potentially leading to tachycardia, hypertension, and episodes of excessive sweating.⁶

CBTs arise from paraganglia located in the covering of the carotid artery in neck. These are usually clinically silent before presenting as a painless, slowly enlarging mass in the lateral neck.⁷⁻⁹ The initial symptoms presented could be a pulsating mass in the neck. These show a finding known as a positive Fontaine's sign that is CBTs can be moved horizontally rather than vertically.⁷ Dysfunction of the vagal nerve may be induced by large CBTs and, less frequently, of cranial nerves IX, XI, and XII.

Vagal nerve schwannomas do not show a gender predilection and are most frequently diagnosed in individuals during their third to fifth decades of life.¹⁰ Although malignant transformation is rare, it has been documented in the literature.¹¹ Preoperative evaluation typically includes ultrasonography (US), computed tomography (CT), MRI, and fine-needle aspiration cytology (FNAC).¹² These tumours are often asymptomatic, but in some cases, patients may experience hoarseness of voice due to vocal cord paralysis. A paroxysmal cough triggered by palpation of the mass is a highly suggestive clinical sign indicating a vagal nerve origin.¹³

While complete surgical excision remains the primary treatment modality, the diagnosis and management of vagal schwannomas present significant clinical challenges. This is due to the non-specific nature of patient history and examination, as well as the persistent risk of vagal nerve injury following surgical removal.¹⁴

Surgical excision involving division of the nerve of origin (NOO) often results in permanent morbidity due to loss of nerve function. While nerve-sparing techniques such as intracapsular enucleation aim to preserve the nerve, they do not always ensure functional integrity postoperatively.

Therefore, achieving an accurate preoperative diagnosis-ideally including clear identification of the NOO-is essential for effective management. This helps in both surgical planning and patient counselling, given the significant risk of nerve palsy associated with the procedure.¹⁵

In this case report we highlight neck swelling resembling Carotid Body Tumour and its treatment by intracapsular excision to prevent complications.

CASE REPORT

A 23-year-old female reported to our department with a slowly progressive, asymptomatic, non-pulsatile swelling over the left side of neck. Examination revealed a diffuse swelling in the left side of the neck in carotid triangle. Cranial nerve examination was normal. On US images, it appears as a round or elliptical cross-section with a clear border tumour. CT shows it as a well-covered, well-defined mass which is usually of higher attenuation than muscle on contrast-enhanced images.

MRI evaluation typically shows well-circumscribed mass lying between the internal jugular vein and the carotid artery, isointense or hypointense signal on T1-weighted images and hyperintense signal on T2-weighted images are seen. It showed a well-defined space occupying lesion in the left carotid space displacing left common carotid artery and carotid artery bifurcation anteromedially and internal jugular vein posterolaterally suggestive of paraganglioma (Figure 1).

All required preoperative investigations were completed. A 24-hour urinary vanillylmandelic acid (VMA) assay was performed to evaluate catecholamine secretion, and the result was within normal limits. The procedure was carried out under general anaesthesia. A radial arterial line was established for continuous blood pressure monitoring, and a central venous catheter was inserted via the right internal jugular vein to allow for rapid fluid resuscitation and inotropic support if needed during intraoperative emergencies.

A transcervical skin incision was marked along a natural cervical skin crease. After infiltration with 2% lignocaine with adrenaline for local vasoconstriction and anaesthesia, the skin was incised (Figure 2). Subplatysmal flaps were elevated to expose the underlying structures. The sternocleidomastoid muscle was gently retracted, allowing access to the carotid sheath, where the tumour was visualized (Figure 3).

Upon dissection of carotid sheath, IJV was posterolaterally displaced while carotid artery anteromedially. IJV was dissected and retracted laterally and common carotid artery retracted medially isolating schwannoma. Cervical sympathetic chain was identified and preserved.

On further dissection, spindle shaped schwannoma was seen encapsulated within the nerve sheath of vagus nerve (Figure 4 and 5). Intracapsular enucleation of tumour was carefully done preserving remaining nerve fibres (Figure 6). Recovery of patient was uneventful and was discharged on 4th postoperative day. The presumed paraganglioma was sent for histopathological examination which revealed a schwannoma (Figure 7).

The function of the vagus nerve and the sympathetic nerve was evaluated preoperatively and postoperatively by examining vocal cord mobility with laryngoscope and symptoms of Horner's syndrome.

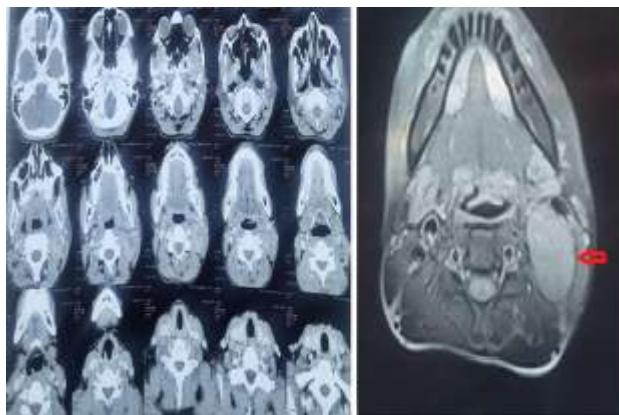


Figure 1: MRI imaging of a vagal schwannoma (red arrow) on the left cervical side.



Figure 2: Incision line marking from mastoid to hyoid region left side of neck.



Figure 3: Exposure of carotid sheath with tumour.



Figure 4: Intraoperative photograph of the vagal schwannoma being exposed.

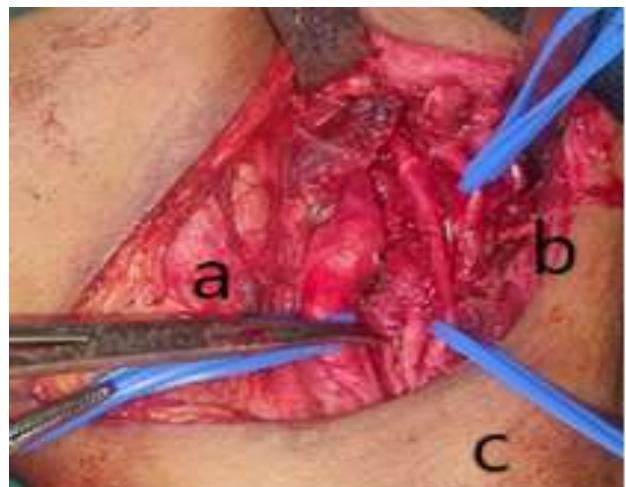


Figure 5: (a) Common carotid artery (b) internal jugular vein and (c) cervical sympathetic chain.



Figure 6: Macroscopic examination of vagal schwannoma.

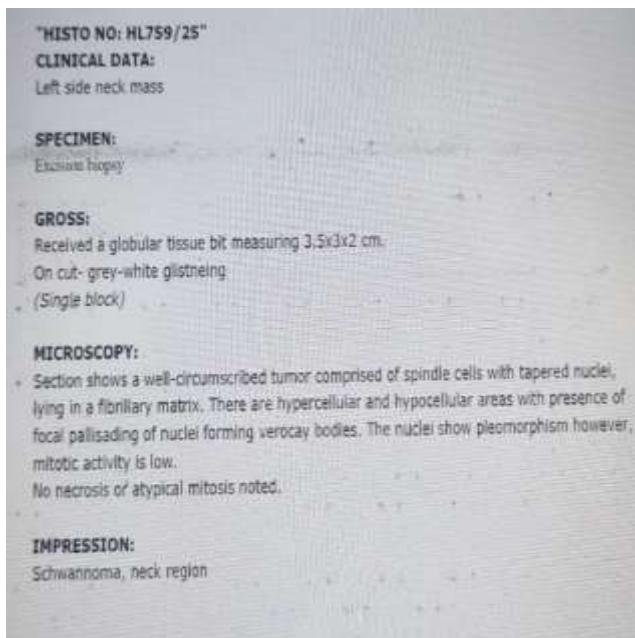


Figure 7: Histopathology report of tumour conforming it to be schwannoma.

DISCUSSION

Vagal schwannomas account for a small subset of extracranial schwannomas, which themselves represent approximately 25-45% of all head and neck schwannomas.¹⁶ Their slow growth, deep location, and non-specific presentation often make preoperative diagnosis challenging, with a significant proportion being misdiagnosed as CBTs.¹⁷

Differentiating vagal schwannomas from CBTs is clinically important because surgical risks, vascular involvement, and nerve preservation strategies differ between the two. CBTs typically splay the internal and external carotid arteries due to their origin at the carotid bifurcation, whereas vagal schwannomas usually displace the common and internal carotid arteries anteromedially and the internal jugular vein posterolaterally. Both lesions may appear as well-defined masses on MRI, but CBTs often display the “salt-and-pepper” appearance on T1- and T2-weighted images due to flow voids from hypervascularity, a feature typically absent in schwannomas.¹⁸

In our patient, the displacement pattern and lack of prominent flow voids suggested a vagal schwannoma; however, the preoperative impression favoured a paraganglioma due to proximity to the carotid bifurcation. This diagnostic overlap underscores the value of high-resolution Doppler ultrasonography and diffusion-weighted MRI, which have been shown to improve preoperative discrimination between vascular and neurogenic lesions.¹⁹ Surgical management remains the treatment of choice for symptomatic or enlarging lesions. While traditional resection often involves

sacrificing the NOO, leading to permanent vocal cord paralysis, intracapsular enucleation offers a nerve-sparing alternative in benign schwannomas. Recent series report postoperative vocal cord palsy rates as low as 10-20% with intracapsular techniques, compared to over 50% with complete nerve resection.²⁰

Nevertheless, incomplete removal carries a small recurrence risk (~5-10%), mandating long-term follow-up. Intraoperative nerve monitoring-either intermittent or continuous-has been shown to reduce postoperative neuropraxia and was employed in some recent studies, although it was not used in our case. Histopathology is the gold standard for diagnosis, demonstrating Antoni A (cellular) and Antoni B (hypocellular) areas, Verocay bodies, and S-100 positivity, as in our case. These features help confirm schwannoma and rule out other spindle cell neoplasms. Postoperative outcomes are generally excellent in benign lesions, especially when nerve preservation is achieved. Our patient retained normal vocal cord mobility and swallowing function, consistent with the favourable outcomes reported in similar nerve-sparing series.²¹

Vagal schwannomas should be considered in the differential diagnosis of carotid space tumours. Careful analysis of vascular displacement patterns on MRI, combined with targeted biochemical screening and surgical planning, allows for both accurate diagnosis and optimal functional outcomes. Intracapsular excision remains a valuable approach for benign lesions, provided that meticulous dissection and long-term surveillance are ensured.¹⁵

CONCLUSION

These neurogenic, pharyngeal tumours often present with minimal or no symptoms, making it challenging to distinguish them from other cervical contemporaries. One useful diagnostic clue is the pattern of carotid bifurcation displacement, which varies between tumour types. When there are no contraindications, surgical removal remains the standard treatment for both CBTs and vagal schwannomas. Establishing a correct diagnosis before surgery is crucial for planning the appropriate approach and informing patients about potential postoperative risks. In cases where the diagnosis is uncertain, a structured diagnostic workup-including ultrasound, MRI, and magnetic resonance angiography-should be undertaken. Key imaging features such as vascular displacement and the uniform contrast enhancement seen in CBTs support accurate identification. On the other hand, the absence of prominent hypervascularity on MRA may suggest the less common scenario of a vagal schwannoma imitating a CBT.

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