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Vagal Schwannoma Hiding Behind a Lipoma: Rare Clinical Case Report

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Abstract: Cervical vagal schwannomas are rare, slow-growing, benign peripheral nerve sheath tumors originating from the vagus nerve, representing a diagnostic challenge in head and neck surgery, particularly when coexisting with other cervical lesions. We report a rare presentation of a 47-year-old female presenting with an 8-year history of a right neck swelling. Clinical examination revealed an isolated, painless lesion along the sternocleidomastoid muscle, accompanied by a concurrent 8 x 3.5 cm lipoma overlying the swelling. Contrast-enhanced magnetic resonance imaging (CE-MRI) confirmed a well-encapsulated post styloid parapharyngeal mass characteristically displacing the internal carotid artery anteromedially and the internal jugular vein laterally. The patient successfully underwent transcervical complete excision of lesion with identification and preservation of critical surrounding neurovascular structures. Management. Preoperative magnetic resonance imaging remains the gold standard for diagnosis and anatomical localization, preventing hazardous biopsy procedures. Cervical vagal schwannomas coexisting with other superficial neck masses are exceptionally rare. A combination of clinical assessment, high-resolution CE-MRI, and meticulous nerve-sparing microsurgical technique allows the successful surgical outcomes and preservation of cranial nerve function.

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I. INTRODUCTION

Schwannomas, historically classified as neurilemmomas or neurinomas, represent benign, slow-growing, encapsulated neoplasms originating from Schwann cells of the peripheral, cranial, or autonomic nervous systems [2,9,11]. Within the head and neck region, the parapharyngeal space represents an important anatomical site of origin for neurogenic tumors, particularly those arising from the lower cranial nerves [2,3,13]. Among these, the vagus nerve (cranial nerve X) is a significant source of cervical neurogenic tumors, accounting for approximately 20% of cervical neurogenic neoplasms [2,3]. Owing to their characteristically indolent growth, with reported rates of approximately 2.7–3.0 mm annually, these tumors frequently remain clinically asymptomatic until they attain considerable dimensions. The typical presentation occurs during the third to fifth decades of life, with no marked sex predilection [2,3,11]. The coexistence of a vagal schwannoma with another cervical mass, such as a lipoma, may further complicate clinical assessment, obscure the underlying diagnosis, and pose an additional diagnostic challenge.

II. CASE PRESENTATION

47year old female presented with right neck swelling for 8 years, not associated with pain, difficulty swallowing, difficulty in breathing, prolonged fever or any other swelling in head and neck (Picture -1). On clinical examination swelling was located in right neck just anterior to posterior border to SCM muscle, soft on palpation, mobile in all four directions, compressible with well-defined margins. On palpation another isolated mass was felt, located deep to or along the anterior and medial border of the sternocleidomastoid (SCM) muscle, which was non-tender, freely mobile along a horizontal (transverse) plane perpendicular to the axis of the carotid vessels, but restricted along the vertical (longitudinal) axis. On manual compression, or sharp rotation of the neck triggers an involuntary, paroxysmal dry cough. Further clinical examination revealed no compromise to the adjacent lower cranial nerves (CN IX, XI, XII) or the cervical sympathetic chain, as there was no complains of hoarseness of voice, dysphagia, Horner's syndrome, or shoulder drop.

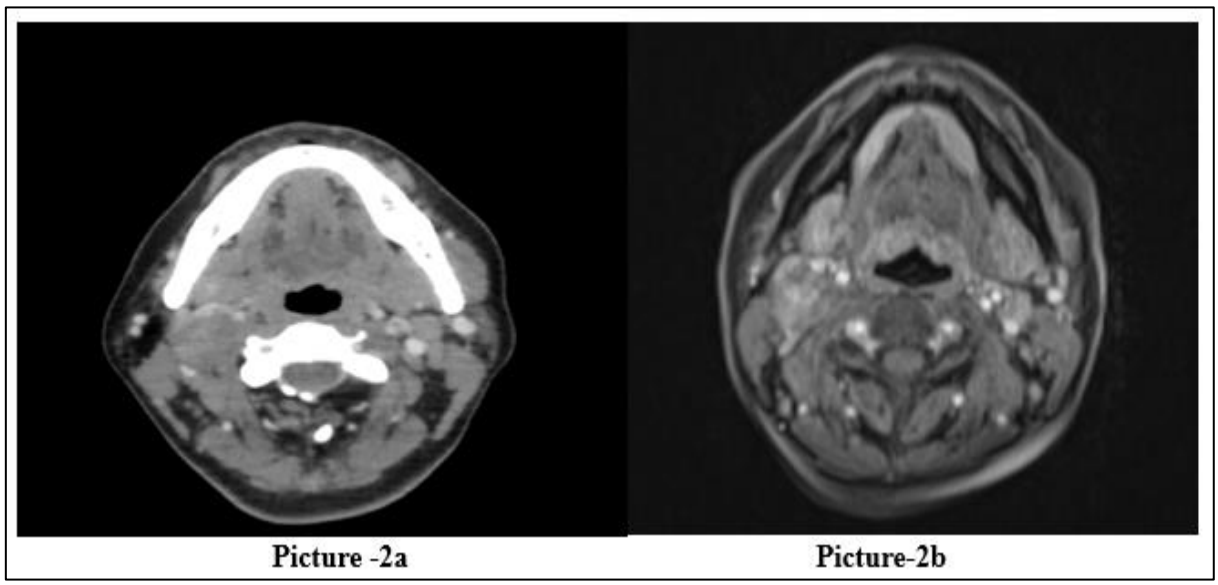
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FNAC in this case was skipped, while widely utilized for general neck masses, FNAC yields low diagnostic specificity (~20%) for schwannomas, frequently shows inconclusive spindle cell findings. Open incisional biopsies or core needle biopsies are strictly contraindicated due to the catastrophic risks of severe haemorrhage, perineural scarring, and life-threatening vagal stimulation (profound bradycardia, hypotension, or cardiac arrest).



Picture 1 Showing the Clinical Appearance of the Patient with Right Sided Neck Swelling

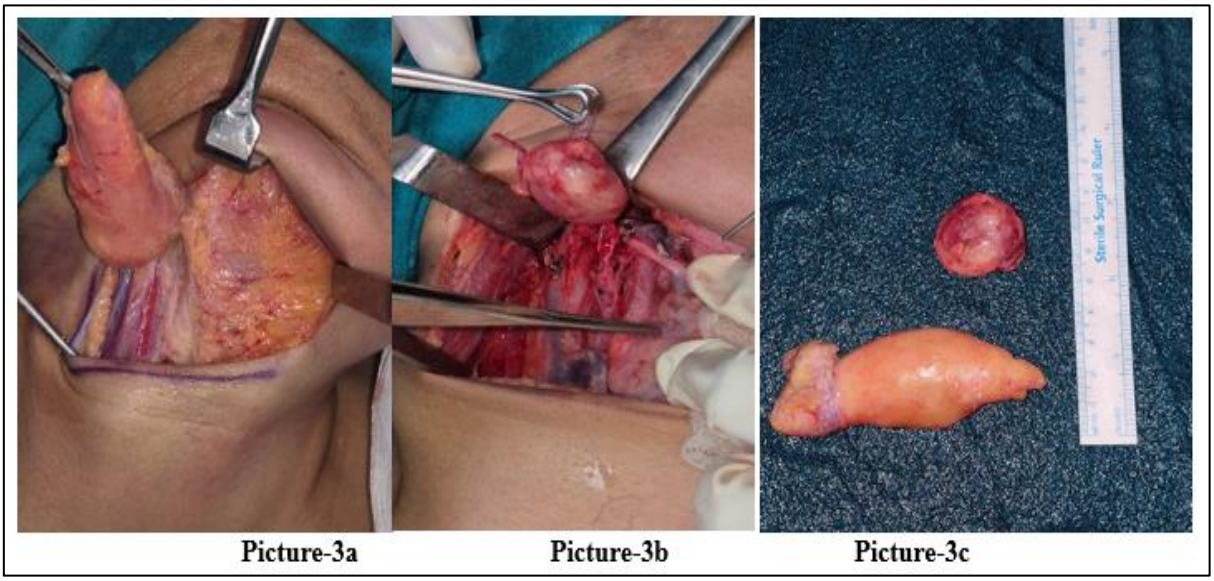
CE-MRI (Picture-2) was performed in this case Showed a well-encapsulated hypointense to isointense lesion on T1(Picture 2a) and marked, heterogeneous hyperintense lesion on T2(picture-2b), displacing the internal carotid artery (ICA) anteromedially and the internal jugular vein (IJV) laterally, reflecting myxoid degeneration and cystic spaces (Antoni B regions) and highlighting the classic MR target sign (hyperintense periphery with a hypointense central fibrous core).



Picture 2 a-CEMRI T1 Series of Hypo Intense to Isointense Lesion b-T2 Series of Hyper Intense Lesion Displacing Internal Carotid Artery and Internal Jugular Vein.

Complete surgical excision remains the definitive treatment for symptomatic or progressively enlarging cervical vagal schwannomas. The surgical approach depends heavily on tumour size, cranial extension toward the skull base, and parapharyngeal involvement. A transverse cervical skin crease incision placed two fingerbreadths below the mandible provides excellent exposure of the carotid triangle, superior mediastinum, and parapharyngeal space. 8 x 3.5 cm lipoma identified over the superior surface of right SCM and excised, preserving EJV and transverse cervical nerve (Picture-3a and 3c). Carotid sheath opened to identify the vagal nerve and its schwannoma. 3 x 3 cm smooth white lesion identified extending superiorly from posterior border of digastric and inferiorly till carotid bifurcation. intra-capsular excision of the vagal schwannoma done preserving the nerve in length (Picture 3b and 3c).

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Picture 3a Exposed Superficial Lipoma 3b- Exposed Schwannoma within Carotid Triangle with Preservation of Nerve 3c-Excised Specimen of Lipoma (Right) and Schwannoma (Left)

Post operatively patient was stable without any neurological deficit and was discharged on 4th post operative day.

III. DISCUSSION

A central surgical dilemma in the management of vagal schwannomas is the choice between nerve-sparing intracapsular enucleation and complete en-block tumour resection with sacrifice of the parent nerve [2,3,14]. The reported total postoperative vagal morbidity rate is approximately 67% following intracapsular enucleation, compared with 100% following complete tumor resection with nerve division [1,10,12]. Because schwannomas are true encapsulated neoplasms that typically grow eccentrically, displacing native nerve fascicles toward the periphery, meticulous sharp and blunt dissection under microscopic or loupe magnification may facilitate tumor removal while preserving functional neural fascicles [9,11,14]. Intracapsular enucleation involves opening the epineurial covering along the tumor's longitudinal axis and carefully dissecting the tumor from the displaced nerve fascicles, thereby maintaining anatomical nerve continuity whenever feasible [2,3,14]. In cases of extensive nerve involvement, complete tumor encasement, or suspected malignant transformation, segmental resection of the vagus nerve may become necessary. When nerve sacrifice is unavoidable, primary microscopic end-to-end neurorrhaphy, provided a tension-free repair, or interposition cable grafting using the greater auricular or sural nerve may be considered to facilitate neural regeneration and functional recovery [2,14]. Preservation of the native nerve and its functional continuity should remain the preferred surgical objective whenever oncologically and anatomically feasible [2,3,14].

From a histopathological perspective, schwannomas are distinct from neurofibromas because they are encapsulated neoplasms composed predominantly of differentiated Schwann cells, with native axons displaced toward the periphery rather than distributed throughout the tumor [9,11]. Grossly, schwannomas typically appear as smooth, well-circumscribed, yellowish-white or pinkish-gray ovoid masses. Microscopically, conventional schwannomas exhibit a characteristic biphasic architecture comprising Antoni A and Antoni B areas [9,11]. Antoni A areas are hypercellular and consist of elongated spindle cells with wavy or twisted, rod-shaped nuclei arranged in compact interwoven fascicles. Nuclear palisading around eosinophilic fibrillary processes produces the characteristic Verocay bodies, a classical histological feature of schwannoma [9,11]. In contrast, Antoni B areas are hypocellular, myxoid, and loosely arranged, with a reticular architecture that may demonstrate microcystic changes, edema, xanthomatous histiocytes, hyalinized blood vessels, and hemorrhage [9,11].

Secondary degenerative changes, including hyalinization, calcification, cystic degeneration, and hemorrhage, may occur in longstanding lesions, producing the so-called ancient schwannoma appearance. These changes can occasionally mimic malignancy on imaging and histopathological examination [9,11,13]. Immunohistochemically, conventional schwannomas characteristically demonstrate diffuse and strong positivity for S-100 protein and SOX10, supporting Schwann cell differentiation. Neurofilament staining generally highlights axons displaced toward the tumor periphery rather than showing widespread intratumoral axonal distribution [9,11].

Although the overwhelming majority of cervical vagal schwannomas are benign, malignant peripheral nerve sheath tumors (MPNSTs) represent an important differential diagnostic consideration, particularly in patients with neurofibromatosis type 1 (NF1) [1,5,8,10,12]. MPNSTs are aggressive soft-tissue sarcomas accounting for approximately 5–10% of soft-tissue sarcomas [5,8,10]. Approximately half of MPNSTs are associated with NF1, frequently arising through malignant transformation of pre-existing plexiform neurofibromas, whereas others occur sporadically or arise de

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novo [1,5,8,10,12]. Malignant transformation of a conventional solitary schwannoma is exceptionally uncommon and should not be considered the usual pathway of MPNST development [1,5,8,12]. Histopathologically, MPNSTs are characterized by increased cellularity, marked nuclear pleomorphism, anaplasia, brisk mitotic activity, and geographic tumor necrosis, sometimes demonstrating a characteristic perivascular or pseudopalisading arrangement of tumor cells [1,5,8,12]. Heterologous divergent differentiation may occasionally occur, including rhabdomyosarcomatous differentiation in malignant Triton tumors and, less commonly, osteosarcomatous or chondrosarcomatous differentiation [1,6,8].

According to established clinicopathological studies, important adverse prognostic factors in MPNST include coexistent NF1, large tumor size (particularly greater than 5 cm), and incomplete surgical excision or positive resection margins [1,5,10,12]. The reported overall 5-year and 10-year survival rates, as well as the specific survival differences between NF1-associated and sporadic MPNSTs, vary across published series and require verification against the original studies before inclusion [1,10,12]. Complete radical en bloc surgical excision with histologically negative margins remains the cornerstone of treatment for localized MPNST, while radiotherapy and chemotherapy may be considered as adjunctive treatments according to tumor grade, resection margins, and disease extent [1,5,8,10,12].

IV. CONCLUSION

Cervical vagal schwannoma is a rare, generally slow-growing neurogenic neoplasm requiring rigorous diagnostic precision and refined microsurgical technique [2,6,8,11,12]. Its coexistence with another neck mass, such as a lipoma, may further obscure the clinical presentation and represents an unusual diagnostic scenario. Preoperative fine-needle aspiration (FNA) and open biopsies should generally be avoided because of their limited diagnostic yield and the potential risk of complications, particularly in highly vascular or anatomically sensitive lesions [2,3,14]. Contrast-enhanced magnetic resonance imaging (MRI) is the preferred imaging modality, providing accurate localization of the tumor within the retrostyloid parapharyngeal space and helping establish its vagal origin through characteristic displacement of the carotid artery and internal jugular vein [2,3,4,13,14]. Through meticulous transcervical dissection, judicious use of intraoperative nerve monitoring, and nerve-sparing intracapsular enucleation whenever anatomically feasible, head and neck surgeons can achieve complete tumor excision while minimizing postoperative cranial nerve dysfunction and preserving functional outcomes [2,3,14].

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