**INTRODUCTION**

Schwannomas are uncommon neurogenic benign nerve sheath tumours.\(^1\) Incidence is 40 - 50% in head and neck region. It comprises 20% of parapharyngeal space (PPS) tumours. PPS tumours are rare (0.5% of all head and neck tumours).\(^2\) Differential diagnosis should include salivary gland tumours, paragangliomas, neurofibromas, and metastatic lymph nodes. Most of them arise from vagus nerve and cervical sympathetic chain (CSC). Diagnosis is usually made by imaging techniques: contrast CT, magnetic resonance imaging (MRI), and magnetic resonance angiography (MRA). Fine needle aspiration cytology (FNAC) is useful diagnostic procedure but poor results are seen in neurogenic tumours. Rarely, a vascular CSC schwannoma at the level of carotid arteries bifurcation may mimic carotid body tumour (CBT) on imaging techniques, especially if they are vascular, causing splaying of internal and external carotid arteries. Clinically patient was asymptomatic except for a pulsatile swelling in neck for 5 years. The presented case resembled CBT clinically, on ultrasound and on imaging techniques causing splaying of carotid arteries. FNAC was inconclusive and was always hemorrhagic. During operation, it was found to be CSC schwannoma just posterior to carotid body. CSC was sacrificed and patient developed Horner syndrome postoperatively.

**CASE REPORT**

A 40-year female presented with 3 x 4 cm swelling on right side of neck for last 5 years. It appeared near upper border of thyroid cartilage, gradually increased in size laterally and became obvious near right angle of mandible and in submandibular space (Figure 1). There was no history of dysphagia, hoarseness, regurgitation through nose, sore throat, weight loss and earache. Swelling was pulsatile since beginning.

On examination, patient was thin lady, pulse was 64 per minute. Neck examination revealed palpable mass below right angle of mandible; swelling was pulsatile, mobile and moved laterally on deglutition. Right tonsil and posterior tonsil pillar were not displaced. Indirect laryngoscopy (IDL) was unremarkable. No neck node was palpable. Provisional diagnosis included enlarged lymph node of PPS, salivary gland tumours (deep lobe of parotid gland), CBT, and schwannoma.

FNAC was attempted thrice in different hospitals by different consultants but proved to be haemorrhagic. Ultrasound neck revealed well circumscribed mass in PPS measuring 3 x 3 x 2.7 cm situated at bifurcation of common carotid artery (CCA), causing splaying of internal and external carotid arteries. Colour and Power Doppler demonstrated significant flow signals within the mass. Diagnosis of CBT was made. MRA and MRI were advised.

MRA showed 3 x 3.4 x 2.7 cm mass in right PPS, splaying of internal and external carotid arteries without causing enhancement of these vessels. Internal jugular vein was compressed posteriorly (Figures 2 and 3). Patient failed...
to proceed for MRI, so CT with contrast was advised that revealed enhancing mass of 3.1 x 3.5 x 2.6 cm at carotid bifurcation occupying right PPS causing splaying of external and internal carotid arteries (Figure 4), suggestive of CBT.

Ultrasound abdomen was unremarkable. Urine was negative for vinyl mandalic acid (VMA). Final diagnosis of CBT and rarely of schwannoma was made. Excision was planned with or without vein graft. Vascular surgeon was involved. Operation was planned in open cardiac surgery operative room. Patient was informed about chances of stroke, neural deficit, hoarseness, dysphagia and Horner’s syndrome.

Vertical curved incision was given slightly posterior to the anterior border of sternocleidomastoid (SCM) behind the angle of mandible extending to middle 3rd of neck and curved medially to get better exposure of carotid arteries and to hide the scar. Right SCM was retracted, carotid sheath was opened, tumour was found to be arising from CSC just posterior to carotid body and displacing internal jugular vein laterally. Carotid arteries and vagus nerve were retracted, superior and inferior margins of encapsulated tumour were separated and tumour of size 3.5 x 4 x 2.8 cm was excised (Figures 5 and 6). Two lymph nodes in PPS were sent for frozen section and proved to be reactive. Drain was kept and removed after 24 hours. Patient developed Horner’s syndrome postoperatively. Bradycardia improved after operation and patient developed feeling of palpitation postoperatively but pulse was 84/minute, improved within 24 hours. Histopathology report revealed spindle cells with wavy nuclei, verocay bodies with edema, and vascular spaces consistent with the diagnosis of schwannoma.

**DISCUSSION**

Nerve sheath tumour can arise from any nerve except olfactory and optic that are deficient of these cells. Clinically, these tumours usually present with slow growing neck masses. Nerve deficit symptoms are rare. Nonspecific symptoms such as dysphagia, sore throat, and lump in throat may be present.1,2 Tumour expands medially to displace tonsil medially in oropharynx and posteriorly into retro-mandibular area.3

These tumours are radio-resistant; so complete surgical excision, with or without transection of nerve from which they grow, is mandatory. Sometimes, they can be enucleated from the nerve without nerve deficit. Surgical excision requires proper diagnosis for planning and counselling.2

Diagnosis is usually made by imaging techniques. Initial diagnosis can be made by contrast CT to measure size and extent of tumour.5 FNAC is useful diagnostic tool for PPS tumours, but poor results are shown in neurogenic tumours with accuracy of only 25%.

Ultrasound neck with Doppler technique is very helpful and noninvasive. Actual origin of tumour can be identified by assessing the displacement of carotid sheath contents and vascularity of tumour.5 MRI gives 95% accurate result and can even identify the nerve of origin.5 Angiography is usually used to assess PPS vascular tumour with preoperative embolization.6 In contrast to paraganglomas, there is no vascular flow in schwannoma. Rarely, hypervascular schwannomas are difficult to differentiate from paraganglomas, especially at carotid bifurcation.6 In this case, we depended on ultrasound with Doppler, CT with contrast and on MRA. CSC runs posterior and medial to carotid sheath, schwannoma of CSC displaces the carotid sheath contents common carotid artery (CCA), internal carotid
artery (ICA), jugular vein (JV) and vagus nerve anteriorly and laterally without any separation between CCA and JV or ECA and ICA, but the vagus nerve schwannoma, that is present between IJV and CCA, will cause separation of these vessels. Vagus nerve fibromas in carotid sheath run lateral to internal carotid artery (ICA) above the bifurcation, so cannot cause splaying of ICA and ECA. Splaying of carotids (ECA and ICA) near bifurcation, classically suggests a CBT. CSC schwannoma at the level of carotid bifurcation occasionally grows anteriorly between ICA and ECA, and can cause splaying of vessels near carotid bifurcation which is very difficult to differentiate with CBT, specially if they are hypervascular.

By observing the displacement of vessels on CT and MRI, nerve of origin for schwannoma can be predicted. Accurate preoperative diagnosis of nerve of origin is crucial for planning of nerve plasty. It also makes easier for the surgeon to evaluate postoperative nerve deficit, its rehabilitation; and also preoperative counselling of patient can be done.

In this case report as the tumour was from CSC just posterior to carotid body and was vascular in nature, so it was difficult to differentiate from CBT. It is a rare presentation, resembling with CBT, both clinically and on imaging.

REFERENCES