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Cervical Vagal Schwannoma Review of all Reported Cases and Our Reports

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Abstract

Background: Extracranial cervical vagus nerve schwannomas are quite rare and we found in English literature only 133 cases. We provide an extensive revision of all reported cases we found in literature.

The gold standard for cervical vagal schwannomas is their complete surgical excision being often technically challenging in order to preserve the neural pathway of the vagus nerve and the recurrent laryngeal nerve.

Material and Methods: We operated two patients for cervical vagal schwannoma and reviewed all the reported cases.

Results: The patients underwent an en bloc excision via a transcervical approach under general anaesthesia and pathological and microscopic examination demonstrated the diagnosis of ancient schwannoma.

Conclusions: Differential diagnosis with other lesions of the parapharyngeal space is mandatory and radiological findings are often not specific.

Preoperative diagnosis could be challenging but the treatment of choice is complete surgical resection. Post-operative vocal cord palsy must be considered and clearly discussed with the patient.

Keywords: Cervical schwannoma; Vagal nerve schwannoma; Neck tumor

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Introduction

Schwannomas are benign, slow-growing encapsulated tumors originating from schwann cells of the nerve sheath and approximately between 25% and one third of them occur in the head and neck region[1-5].

Extracranial cervical vagus nerve involvement is quite rare and in English literature we found only 133 cases[1-82]. Cervical vagal schwannomas usually affect the third and fifth decade of life[2,4,16], presenting as a slow-growing, painless, palpable mass in the neck region; the symptoms are related to their huge volume with compression of the adjacent nervous and vascular structures.

CT findings are not specific and do not allow differential diagnosis with other cervical masses; MRI is the exam of choice sometimes in addiction to fine needle biopsy.

Complete surgical excision is the gold standard for cervical vagal schwannomas although often it could be technically challenging in order to preserve the neural pathway of the vagus nerve and the recurrent laryngeal nerve.

We operated two patients for cervical vagal schwannoma and reviewed all the reported cases.

Case Report

A 61-year-old male was referred to our Institute for the treatment of a palpable lesion in the right side of the neck. One month before admission, the patient had syncope during an atrial fibrillation but the cardiologic investigation excluded its cardiac origin. Further investigations, including brain and neck computed tomography (CT) and epiaortic ultrasound (US), demonstrated a

soft lesion (5 X 3 X 2, 4 cm) in the right side of the neck close to carotid and jugular vessels. Clinical examination revealed a soft and smooth mass in the right cervical region, medial to the sternocleidomastoid muscle (SCM). The neurological examination showed intact cranial nerves, no Horner's syndrome and a light hoarseness.

Suspecting a carotid body tumor, we performed an angiography and a balloon occlusion test demonstrating an extrinsic compression of the right internal carotid artery (ICA) and a vascular supply originating from the right occipital artery. (Figure 1)

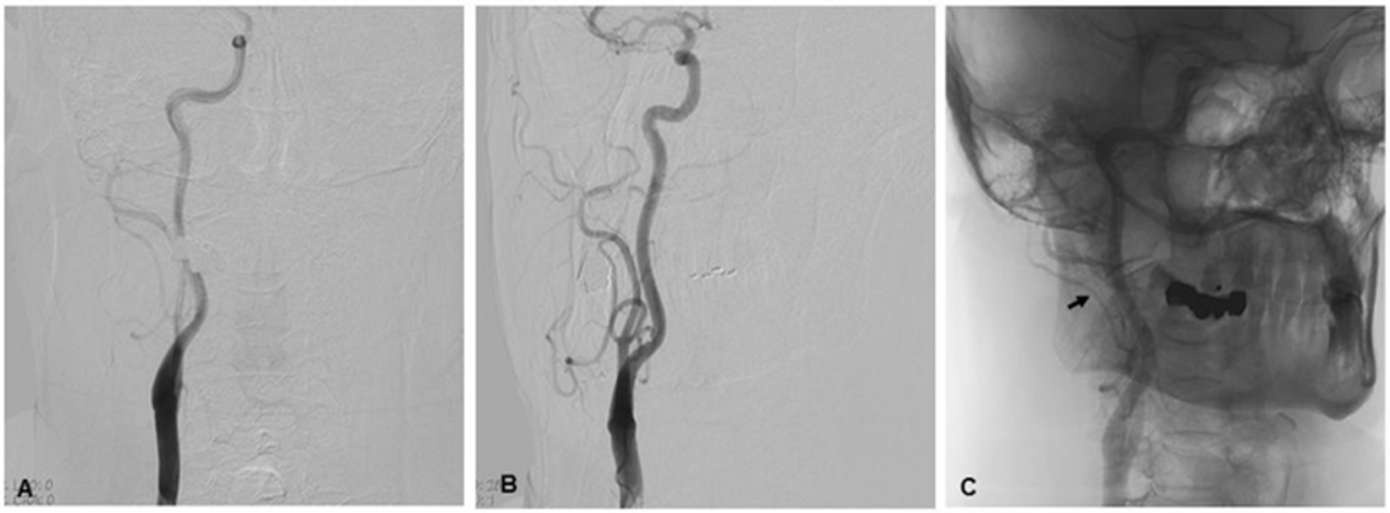


Figure 1: Angiography with balloon occlusion test clearly shows an extrinsic compression of the right internal carotid artery (ICA) which lies anteriorly and medially (A+B). Moreover the lesion seems to have a vascular supply originating from the right occipital artery (C).
(Arrows: feeding vessel of the lesion, a branch of the right occipital artery)

MRI scan allowed our neuroradiologist to diagnose a cervical schwannoma demonstrating a well capsulated ovular mass (5X3X2, 6 cm) in the right retrostyloid parapharyngeal space which extended up to the carotid bifurcation without any infiltration of the surrounding structure. (Figure 2)

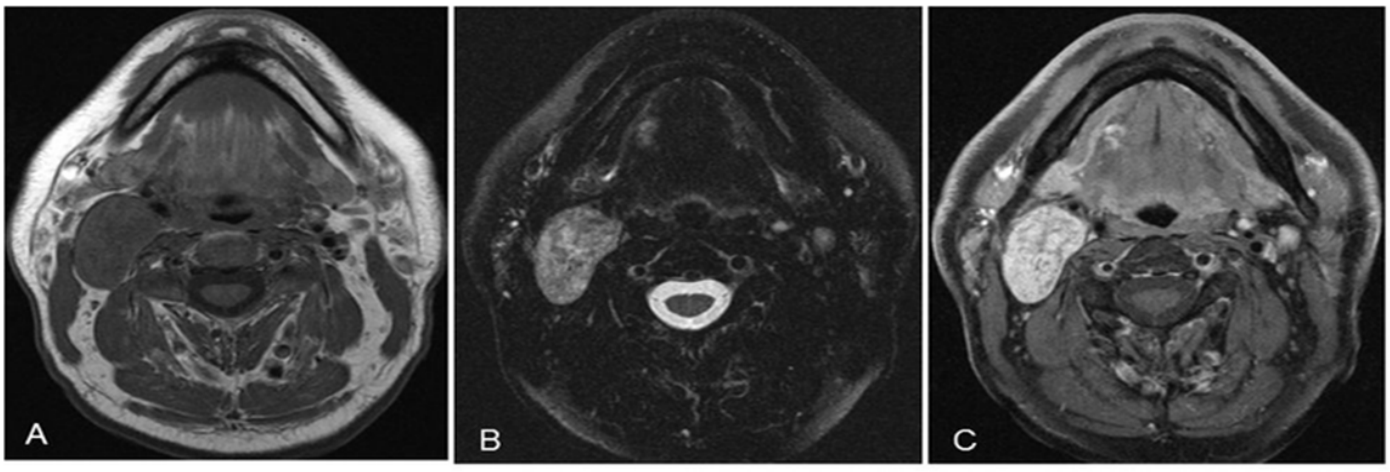


Figure 2: Head and neck MRI shows a capsulated ovalar mass in the right, retrostyloid, parapharyngeal space (A,B,C) with high and dishomogeneous signal intensity in post-contrastographic sequences (C); which extended up to the carotid bifurcation.

The lesion was hyperintense in T2WI, hypo-hysointense in T1WI and showed high and dishomogeneous signal intensity after contrast medium administration. The patient underwent an en bloc excision via a right transcervical approach under general anaesthesia; we drew a cervical incision along the medial border of the right sternocleidomastoid muscle and the dissection proceeded beneath the muscles. An ovoid-shape mass was observed between the internal carotid artery and the internal jugular vein that were easily dissected from it. (Figure 3)

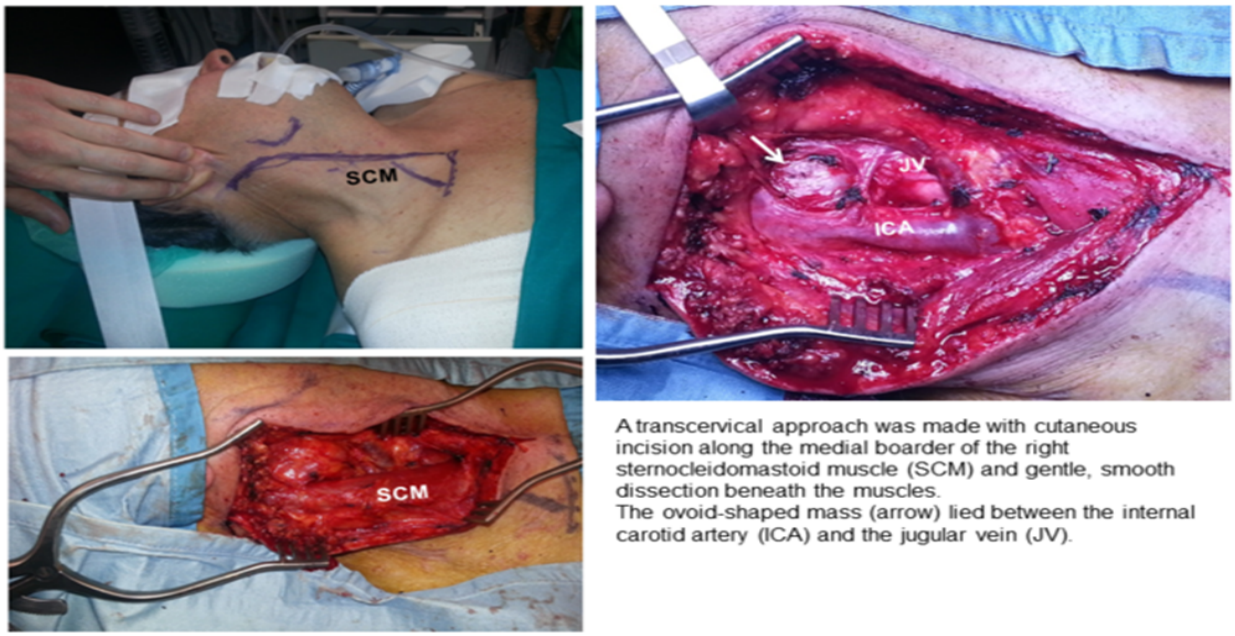


Figure 3: A transcervical approach was made with cutaneous incision along the medial border of the right sternocleidomastoid muscle (SCM) and gentle, smooth dissection beneath the muscles (A+B). The ovoid-shaped mass (arrow) lied between the internal carotid artery (ICA) and the jugular vein (JV) (C).

The superior pole of the tumor appeared to be in continuity with the vagus nerve (Figure 4) and a little twig was sacrificed while we kept the integrity of its main trunk.

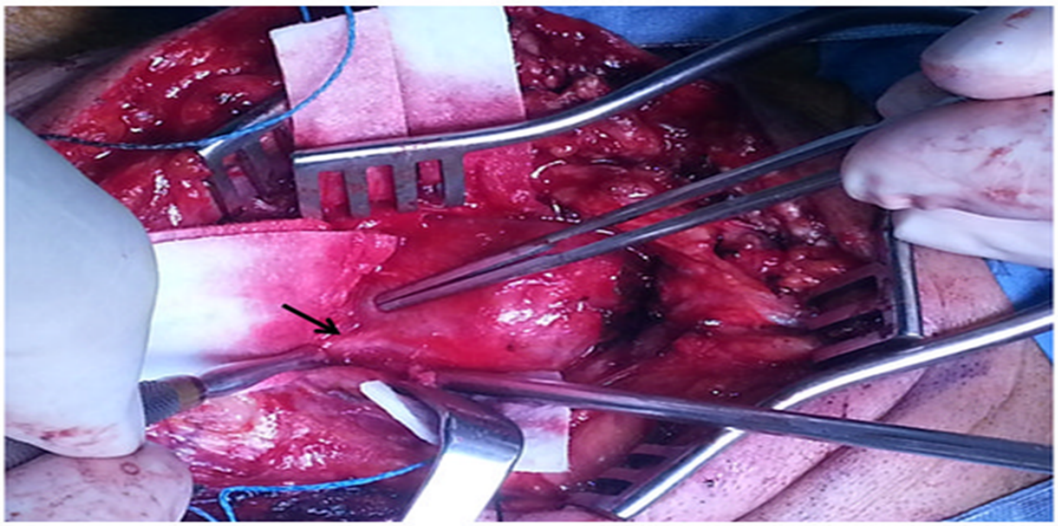


Figure 4: The mass was easily dissected from the internal carotid artery and from the jugular vein. In the upper pole, as well as in the inferior one, was detectable a twig in continuity with the vagus nerve (arrow) that had to be sacrified. Main nerve trunk integrity was preserved.

The mass was completely resected en bloc (Figure 5) with a continuous monitoring of ECG not reporting disturbance. Post-operatively the patient complained of moderate hoarseness due to vocal cord paralysis and was followed by our otolaryngologist; at six months follow-up he clinically improved for the compensation of the contralateral vocal cord and refused further surgical options. Pathological examination confirmed the diagnosis of benign schwannoma of the vagus nerve demonstrating small fascicles of spindle cells S100+ without atypia and necrosis or mitosis were not observed. (Figure 6)

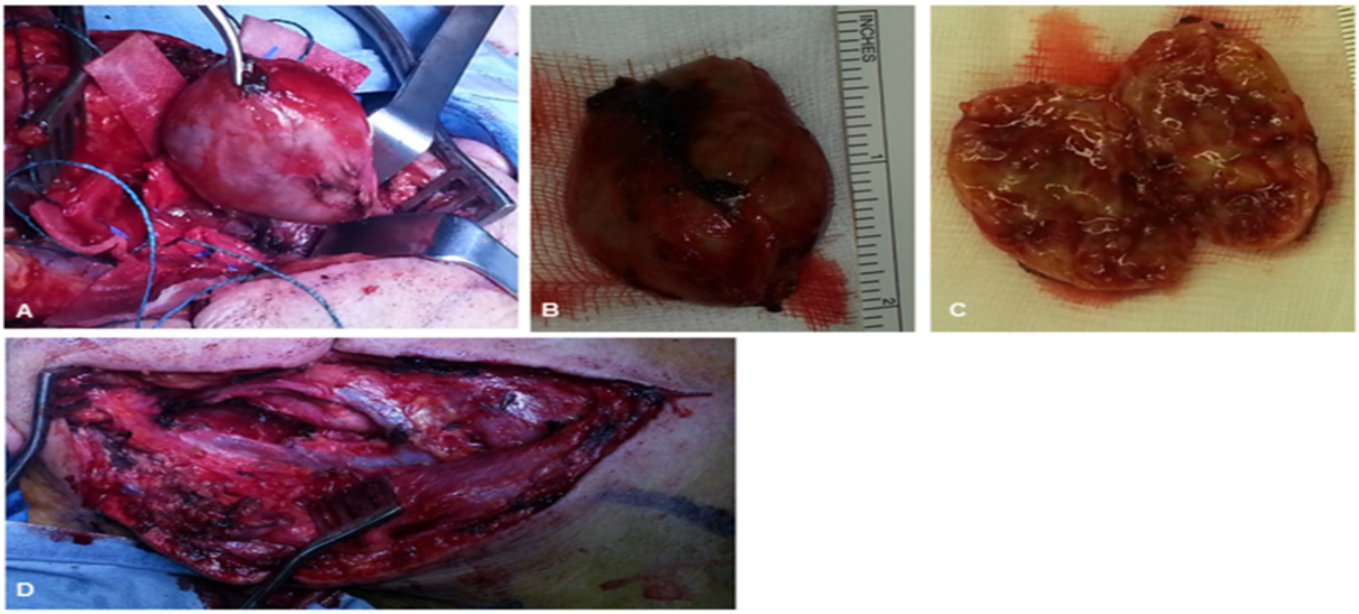
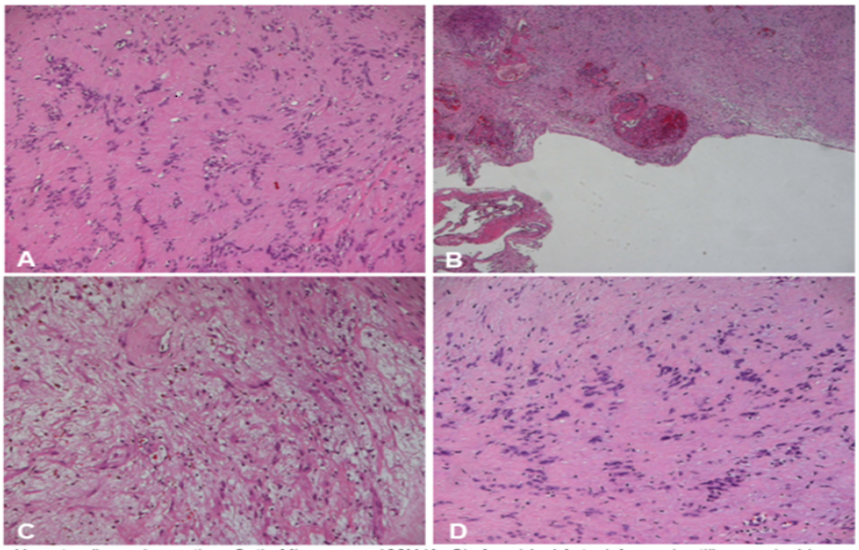


Figure 5: After coagulation and recision of the upper and inferior poles, the mass was finally totally removed en bloc (A,B,C). Immediate post-operative local inspection showed no pathological bleeding of the major vascular trunks (D). The lesion was send to pathological examination for histological characterization.



Hematoxylin-eosin section, Optic Microscope 100X (A+C). A residual Antoni A area is still recognisable, with residual Verocay bodies (A). Antoni B area with stromal rarefaction and myxoid aspects (C). Hematoxylin-eosin section, Optic Microscope 25X (B) 100 X (D). Tissue shows cystic degeneration, stromal hemorrhage and vascular thrombosis (B). Few Verocay bodies are still recognizable, immersed in a jaline paucicellular collagenized stroma (D).

Figure 6: Hematoxylin-eosin section,, Optic Microscope 100X (A+C). A residual Antoni A area is still recognisable with residual Verocay bodies (A). Antoni B area with stromal rarefaction and myxoid aspects (C). Hematoxylin-eosin section, Optic Microscope 25X (B) 100X (D). Tissue shows cystic degeneration, stromal hemorrhage and vascular thrombosis (B). Few Verocay bodies are still recognizable, immersed in a jaline paucicellular collagenized stroma (D).

A 31-year-old male patient presented with a progressive swelling in the right submandibular region since several months. Examination of the neck showed no visible mass but at palpation an oval, non-tender and firm lesion was appreciated in the right submandibular region. Pain or paroxysmal cough was not elicited by palpation and pressure. The neurological examination showed intact cranial nerves and no Horner's syndrome. Ultrasound (US) of the epiaortic trunks showed a hypervascularized tumor (4, 6 X 3, 5 cm) which displaced not compressing the right carotid artery. MRI confirmed the presence of the oval, right parapharyngeal lesion lying between the displaced internal jugular vein and the carotid artery and showing a dishomogeneous enhancement due to its central hypervascularized component.

The lesion showed a dishomogeneous hyperintense signal in T2WI and hypointensity in T1WI, with mild to moderate dishomogeneous hyperintesity after gadolinium administration. Under general anaesthesia the patient underwent the excision of the mass through a transcervical approach. Dissection was made in the subplatysmal plane and the carotid trunk was exposed. The mass was well-capsulated clearly arising from the right vagus nerve. The upper pole of the lesion was adjacent to the skull base while the inferior pole extended to the carotid bifurcation.An en bloc excision was successfully performed. Post-operatively the patient

showed a moderate hoarseness still present at six months follow up due to the vocal cord paralysis but him refuse surgical treatments due to the well acceptable and tolerable discomfort. Pathological and microscopic examination demonstrated the diagnosis of ancient schwannoma. (Figure 7)

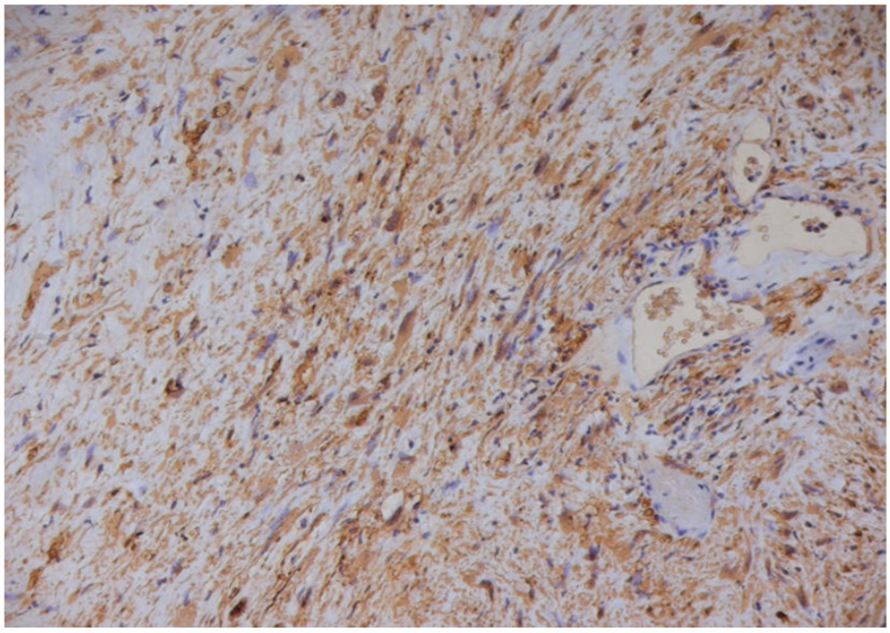


Figure 7: S-100 protein immunostaining, 100X magnification. Stromal spindle cells are strongly S-100 protein positive.

Discussion

Schwannomas are benign nerve sheath tumors arising from schwann cells which normally surround the nerves. They may originate from any peripheral, cranial or autonomic nerve except the olfactory and the optic nerves. Approximately ranging between 25% and one third of the cases, schwannomas occur in the head and neck region and are more frequently found in the eighth cranial nerve followed by the lower cranial nerves (IX, XI, XII) and the sympathetic chains[1-5].

The vagus nerve involvement is quite rare and a number ranging between 70, 95 and 100 cases have been described in the literature but actually we extensively revised all the available English papers and found 133 cases[1-76](Table 1).

Table 1: Extensive revision of the reported cases

Authors & Year	sex	Age (yrs)	Size (cm)	volum	surgery	End-to-end anastomosis	outcome	Sd.horner post-op	side
Sekiguchi et al., 1926	M	39	5,5 x 4,5	58,33	NA	NA	vocal cord paralysis		R
Serafini et al., 1931	F	33	7,5 x 6	141,42	en bloc	no	vocal cord paralysis		L
Sinakevic et al., 1934	M	NA	NA	NA	NA	NA	vocal cord paralysis		R
Cutler et al., 1936	M	38	10,5 x 7	269,49	NA	no	uneventful		L
	M	69	6 x 4	50,28	NA	no	vocal cord paralysis		L
Davidovic et al., 1938	M	22	NA	NA	NA	NA	NA	NA	L
Heinlein et al., 1941	F	61	NA	NA	intracapsular enucleation	no	vocal cord paralysis		L
Ehrlich et al., 1943	F	39	1,5 x 1,5	1,76	NA	NA	vocal cord paralysis		R
Stich et al., 1943	M	28	NA	NA	en bloc	NA	vocal cord paralysis		L
Turchik et al., 1946	F	27	7 x 4,5	74,24	en bloc	no	vocal cord paralysis		R
Garandel et al., 1946	F	NA	NA	NA	en bloc	no	vocal cord paralysis	x	R
Murley et al., 1948	F	19	3,5 x 2	7,33	en bloc	no	vocal cord paralysis		R
Slaughter et al., 1949	M	53	5 x 3	23,57	en bloc	no	uneventful	x	L
	F	60	7 x 3	32,99	en bloc	no	uneventful		R
	F	65	NA	NA	intracapsular enucleation	no	vocal cord paralysis		L
	F	65	NA	NA	intracapsular enucleation	NA	vocal cord paralysis		L

Paul et al., 1949	M	31	NA	NA	NA	NA	vocal cord paralysis	NA	R
Kohler et al., 1949	F	38	NA	NA	en bloc	no	vocal cord paralysis		NA
Boles et al., 1952	M	26	6 x 4	50,28	en bloc	end-to-end anastomosis	vocal cord paralysis	x	R
Boisseau et al., 1955	F	35	10 x 4	83,8	en bloc	no	vocal cord paralysis		L
	M	29	NA	NA	en bloc	no	vocal cord paralysis		R
	M	29	5	65,47	en bloc	no	vocal cord paralysis		R
Parnes et al, 1955	F	51	7 x 4	58,66	en bloc	no	vocal cord paralysis		R
Conley et al., 1955	F	11	8 x 6	150,85	en bloc	end-to-end anastomosis	vocal cord paralysis		R
Altany et al., 1956	M	39	3 x 3	14,14	en bloc	no	vocal cord paralysis	x	R
Gore et al, 1956	M	36	4,5 x 3	21,21	en bloc	no	vocal cord paralysis		R
	F	36	4 x 3	18,85	en bloc	no	vocal cord paralysis		L
	M	34	6,5 x 5	85,11	en bloc	no	vocal cord paralysis	x	R
	F	55	6 x 3	28,28	en bloc	no	vocal cord paralysis	x	L
Harrison et al., 1956	F	82	8 x 8	268,18	NA	no	NA		L
Penido et al., 1957	M	52	6 x 3	28,28	en bloc	no	vocal cord paralysis		R
	M	58	NA	NA	NA	NA	NA	NA	R
	M	59	NA	NA	NA	NA	NA	NA	L
Haas et al., 1958	M	26	NA	NA	NA	NA	NA	NA	R
	F	33	NA	NA	NA	NA	vocal cord paralysis		L
	M	34	NA	NA	NA	NA	NA	NA	R
Mitchell et al., 1958	F	51	NA	NA	NA	NA	NA	NA	R
Zippel et al., 1960	F	36	NA	NA	en bloc	no	vocal cord paralysis	x	L
Horwich et al., 1962	F	34	10x5	130,95	en bloc	no	uneventful		R
Bales et al., 1962	M	26	6,4 x 4	53,63	en bloc	end-to-end anastomosis	vocal cord paralysis	x	R
Pang et al.,	F	39	6,5 x 2,8	26,69	NA	no	vocal cord paralysis		R
Horwich et al., 1962	M	47	6 x 5	78,56	en bloc	no	uneventful		R
Leichtling et al., 1963	M	51	4,5 x 2	9,42	en bloc	no	vocal cord paralysis		R
	M	48	6,5 x 4	54,47	en bloc	no	vocal cord paralysis		R
Putney et al., 1964	M	71	4 x 4	33,52	en bloc	no	vocal cord paralysis		L
Wayoff et al., 1964	F	59	NA	NA	en bloc	no	vocal cord paralysis		R
Vogl et al., 1966	M	59	0,5 x 0,5	0,06	NA	NA	vocal cord paralysis		L
Tanaka et al., 1967	F	36	NA	NA	NA	NA	vocal cord paralysis		R
	F	50	NA	NA	NA	NA	vocal cord paralysis		L
	M	56	NA	NA	NA	NA	vocal cord paralysis		R
Rosenfield et al., 1968	F	14	3 x 2	6,28	en bloc	end-to-end anastomosis	vocal cord paralysis		L
	F	32	3x2	6,28	en bloc	end-to-end anastomosis	uneventful		R
	M	64	5x4	41,9	NA	no	vocal cord paralysis		R
	F	45	4x3	18,85	NA	no	vocal cord paralysis		R
Holland et al., 1968	F	53	5x2	10,47	en bloc	no	uneventful		R
Bestir et al., 1969	F	14	6x4	50,28	NA	NA	vocal cord paralysis		L
Das gupta et al., 1969	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
Katz et al., 1971	F	35	5,5x3	25,92	en bloc	no	vocal cord paralysis		L
Reddick et al., 1973	F	24	2x1,5	2,35	intracapsular enucleation	no	uneventful		L
	M	35	2x2.5	6,54	intracapsular enucleation	no	vocal cord paralysis		L
Andrè et al., 1975	M	64	4x3,5	25,66	en bloc	no	vocal cord paralysis		L

	F	52	5 x 5	65,47	en bloc	no	vocal cord paralysis		L
	M	38	6 x 5	78,56	en bloc	no	vocal cord paralysis		L
Mair et al., 1976	M	71	3,5 x 3	16,49	en bloc	no	uneventful		R
Mukherjee et al., 1979	M	50	10 x 10	523,8	intracapsular enucleation	no	uneventful		R
Pesavento et al., 1979	F	12	NA	NA	en bloc	no	vocal cord paralysis		L
Schultze et al., 1982	M	73	5 x 4	41,9	en bloc	no	vocal cord paralysis		R
Chang et al., 1984	M	32	10 x 8	335,23	en bloc	no	uneventful		R
Gupta et al., 1984	F	40	6 x 4	50,28	en bloc	no	vocal cord paralysis		R
St pierre et al., 1985	F	56	3 x 2	6,28	en bloc	no	uneventful		L
	F	61	4 x 3	18,85	en bloc	no	uneventful		NA
Wood et al., 1986	F	62	5 x 3	23,57	en bloc	no	uneventful		L
Green et al., 1988	M	20	1 x 1	0,52	en bloc	no	vocal cord paralysis		R
	F	36	4 x 3	18,85	en bloc	no	vocal cord paralysis		R
Hallett et al., 1988	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
	NA	NA	NA	NA	NA	NA	NA	NA	NA
Hussain et al., 1989	M	65	6 x 3	28,28	intracapsular enucleation	no	vocal cord paralysis		L
	F	63	4,6 x 3,5	29,51	en bloc	no	uneventful		L
Bradley et al., 1989	F	34	6 x 4	50,28	en bloc	no	vocal cord paralysis		R
Peetermans et al., 1991	F	24	10 x 3	47,14	en bloc	no	NA		L
Park et al., 1991	F	49	10 x 5	130,95	intracapsular enucleation	no	uneventful		L
	M	29	5 x 2	10,47	intracapsular enucleation	no	vocal cord paralysis		L
	F	25	8 x 4	67,04	en bloc	no	uneventful		L
	F	50	3 x 3	14,14	intracapsular enucleation	no	vocal cord paralysis		L
Walker et al., 1991	M	38	4 x 4	33,52	en bloc	no	uneventful		L
Galli et al., 1992	M	19	5,7 x 1,7	8,62	en bloc	no	deficit		R
Nouls et al., 1993	M	54	5 x 4	41,9	en bloc	no	vocal cord paralysis		L
Yumoto et al., 1996	M	33	5 x 3	23,57	en bloc	no	vocal cord paralysis		L
Furukawa et al., 1996	F	11	NA	NA	NA	NA	NA	NA	NA
	M	36	NA	NA	NA	NA	NA	NA	NA
	F	42	NA	NA	NA	NA	NA	NA	NA
	M	50	NA	NA	NA	NA	NA	NA	NA
	F	53	NA	NA	NA	NA	NA	NA	NA
Gilmer et al., 2000	F	64	2 x 1	1,04	en bloc	no	uneventful		R
	F	50	4 x 4	33,52	intracapsular enucleation	no	vocal cord paralysis		R
	F	55	5 x 2	10,47	en bloc	no	vocal cord paralysis		L
	F	38	5 x 4	41,9	en bloc	no	vocal cord paralysis		R

Fujino et al., 2000	M	26	5 x 5	65,47	intracapsular enucleation	no	vocal cord paralysis		L
	M	63	NA	NA	intracapsular enucleation	no	uneventful		R
Saydam et al., 2000	F	57	9 x 8	301,7	en bloc	no	vocal cord paralysis		R
Leu et al., 2002	F	NA	NA	NA	NA	NA	NA		NA
	M	NA	NA	NA	NA	NA	NA		NA
Heasley et al., 2003	M	63	3 x 3	14,14	NA	NA	NA		R
Cunningham et al., 2003	M	45	4,5 x4	37,71	en bloc	no	vocal cord paralysis		R
Ford et al., 2003	M	38	5 x 4	41,9	en bloc	no	vocal cord paralysis		L
Mevio et al., 2003	M	22	7 x 4	58,66	intracapsular enucleation	no	uneventful		L
Singh et al., 2006	M	14	4 x 3	18,85	en bloc	no	vocal cord paralysis		R
Shetty et al., 2006	M	42	5 x 3	23,57	NA	NA	NA	NA	R
Nardello et al., 2007	M	63	3 x 2	6,28	en bloc	end-to-end anastomosis	vocal cord paralysis		R
Nakano et al., 2008	F	59	NA	NA	intracapsular enucleation	no	uneventful		L
Chiofalo et al., 2009	M	33	3 x 3	14,14	en bloc	end-to-end anastomosis	vocal cord paralysis		R
Schupp et al., 2009	M	45	6 x 3	28,28	intracapsular enucleation	no	vocal cord paralysis		R
Bilancia et al., 2011	M	44	2,5 x 1,7	3,78	intracapsular enucleation	no	vocal cord paralysis		R
Bergmark et al., 2011	F	50	3 x 3	14,14	en bloc	no	vocal cord paralysis		R
Imperatori et al., 2011	F	67	3 x 3	14,14	en bloc	no	uneventful		L
Sreevatsa et al., 2011	F	58	5 x 4	41,9	en bloc	no	uneventful		R
	F	22	6 x 4	50,28	en bloc	no	vocal cord paralysis		L
	F	13	7 x 3	32,99	en bloc	no	uneventful		R
Bhandary et al., 2011	F	50	6 x 4	50,28	NA	no	uneventful		L
Chai et al., 2012	M	32	7 x 6	131,99	en bloc	no	vocal cord paralysis		R
D'andrea et al., 2013	M	31	4 x 3	18,85	en bloc	no	vocal cord paralysis		R

Ganglion areas seem the regions of origin of vagal schwannomas1 being the superior ganglion prone to produce intracranial tumors while the inferior ganglion causes the extracranial cervical ones. Cervical vagal schwannomas are slow growing, encapsulated tumors usually occurring in patients between 30 and 50 years of age[2,4,16,23] and not demonstrating a sex-related predisposition[4,16,23,77].

Our extensive revision of the reported cases confirms such data showing a median age of 42,7 y/o, a male/female ratio of 1/1 and a moderate predominance of the right side at the neck (47% right, 36% left, 17% not available) (Table 1). These tumors often reach large sizes demonstrating a volume up to 10 cc in 11,27% of cases, up to 50 cc in 32,33%, up to 100 cc in 15,78% and larger than 100 cc, up to about 500 cc, in 6,76% (Table 1); in 40.6% of the reported cases these lesions have a diameter larger than 5 cm. (Table 1).

They are usually asymptomatic and painless palpable mass in the neck but large schwannomas can produce dysphagic, dysphonic or dyspnoic signs as a result of the compression of the adjacent neck structures[1,2,16,63,77].When symptoms are present, hoarseness is the most common[16,66,77]. Palpation could produce a paroxysmal cough representing a specific sign of vagal schwannoma[15,16,23,24,27].

The mass is usually fusiform, ovoidal or spherical, firm in consistency and can be moved horizontally but not vertically[66,78]. Several tumors, producing neck swelling, must be considered in the differential diagnosis including carotid body tumor, paraganglioma, salivary gland tumors, branchial cleft cyst, lipomas, malignant lymphoma, metastatic cervical lymphadenopathy and thyroid mass[1,2,16,60,77,78].

CT findings are usually non specific while ultrasound (US) and MRI represent the gold standard for the differential diagnosis with other cervical masses; the latter providing also an evaluation of the relationships of the tumor with the surrounding structures.MRI findings are quite typical and also useful in providing a pre-operative distinction between schwannoma of the vagus nerve and schwannoma of the sympathetic chain. MRI suggests the origin of the tumor relating to vascular displacement; usually vagal schwannoma displaces the internal jugular vein laterally and the carotid artery medially as we noted (Figure 1), while schwannoma of the sympathetic chain displaces both together without their separation[25].

Typically, schwannoma shows a hypo/isointense signal on T1-weighted images and hyper-intense signal on T2-weighted images[60,63,66] and has a marked enhancement after contrast medium administration but presenting an inhomogeneous pattern in contrast with the homogeneous enhancement of the hypervascular glomus tumor[25,60].

Hypervascularity associated to carotid bifurcation splaying usually indicates a carotid body tumor while its absence suggests a sympathetic schwannoma[60]; the mass of carotid body tumor is usually painless but pulsatile[60]. Glomus tumor are well defined mass with intense enhancement after contrast medium administration with a rapid initial contrast accumulation that schwannomas do not show[63]. The usual "salt and pepper" pattern of paragangliomas, due to the areas of signal void, are not demonstrated for schwannomas[63]. Also, pleomorphic adenomas could be better detected by MRI rather than CT[63] showing a hypo/isointense signal on T1 weighted images and a hyper-intense signal on T2 weighted images. Such differential diagnosis and the correlated surgical approach dramatically affect the postoperative integrity of facial nerve.

However, preoperative diagnosis of vagal schwannoma still remains challenging and the utility of pre-operative fine needle biopsy is also controversial being often inconclusive and not recommended by some Authors moreover because surgery remains the treatment of choice[1,2,16,77]. Encapsulated schwannomas are completely benign and rarely undergo malignant transformation, therefore total intra or extracapsular excision should be the treatment of choice1. The primary aim is the dissection of the tumors from the vagus nerve with its preservation[1,16,37] considering that the tumor usually occupies an eccentric position in the nerve, thus individual fibers of the vagus nerve are splayed over its surface within the capsule but not in the mass itself. Some adherent and not detachable fascicles may be excised without sacrificing the main nerve trunk[74]. If vagus nerve trunk preservation is technically demanding and if the lesion is less than 2 cm in length, microsurgical end-to-end nerve anastomosis can also be a valid surgical option[16,23,32]. Otherwise for lesions more than 2 cm in length, free nerve grafting using a sensory nerve should be considered.

However, surgeon must keep in mind that such procedures often produce a permanent vocal cord palsy16 as such occurrence in six of seven reported end to end anastomosis demonstrated (Table 1). Complete resection of the tumor and its capsule is mandatory to prevent recurrence[1,16] and en bloc excision represents the best strategy to achieve it. Nakano et al[4] state that the intracapsular enucleation, as Fujino et al[24] described in 2000, should actually represent the standard surgical strategy but between 2000 and 2013 29 cases have been operated on (Table 1), in 5 cases the Authors did not report the kind of resection while among the remaining 21 patients 17 received an en bloc excision not confirming such statement. In fact the recovery of neural function appears incomplete also in intracapsular enucleation[49,77].

If tumor recurrence is remote, recovery of neural function is frequently incomplete and often results in definitive vocal cord paralysis so that postoperative vocal cord paralysis is the most frequent complication in 56, 4% of the reported cases (Table 1); we found an uneventful postoperative course only in 19, 6% of the patients but we must register a 24% of cases in which this data was not available. The reported incidence of pre-operative vocal cord palsy is about 12%[16,23] but hoarseness is almost always present following surgery. An incidence of 85% of post-operative vocal cord palsy has been reported[1,16,23,67]. Such complication is dramatically high but not up to 85% as these Authors[1,16,23,67] stated occurring totally in 56,4% of the reported cases (Table 1).

Since that, pre-operative assessment of the vocal cord mobility and patient clear information are recommended even if several conservative and surgical treatments are available to manage and improve such deficit. Resection of vagal schwannoma could furthermore cause a postoperative Horner's syndrome[32,60,79]. Some Authors[2,4,43,80] advocate the use of intraoperative neurophysiologic monitoring of the recurrent laryngeal nerve function using an electromyographic (EMG) endotracheal tube and direct endoscopic observation of the vocal cord.

Conclusion

Differential diagnosis including carotid body tumor, paraganglioma, salivary gland tumors, branchial cleft cyst, lipomas, malignant lymphoma, metastatic cervical lymphadenopathy and thyroid mass must be considered. However preoperative diagnosis of vagal schwannoma still remains challenging and the utility of pre-operative fine needle biopsy is also controversial because the complete surgical excision is the gold standard for cervical vagal schwannomas. Post-operative vocal cord palsy must be considered and clearly discussed with the patient because surgery could be technically challenging in order to preserve the neural pathway of the vagus nerve and the recurrent laryngeal nerve.

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