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Lipomas of the Internal Auditory Canal and Cerebellopontine Angle

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Objectives: Lipomas of the internal auditory canal (IAC) and cerebellopontine angle (CPA) are exceedingly rare lesions. The purpose of this report was to describe our experience with lipomas of the IAC and CPA and perform a review of the literature.

Methods: We report 8 cases of lipomas involving the IAC and/or the CPA that were managed at Gruppo Otologico between April 1987 and October 2012.

Results: Four cases of entirely intracanalicular lipomas were radiologically misinterpreted as vestibular schwannomas and underwent tumor removal by a translabyrinthine approach. Two of these patients experienced postoperative facial nerve palsy. Lipomas were suspected in 4 patients on the basis of imaging findings and were managed conservatively. Of these 4 cases, 3 did not show any growth after an average period of 28 months, and 1 case demonstrated tumor growth on follow-up imaging.

Conclusions: Neuroimaging represents an extremely important tool for this diagnosis. Attempts to achieve complete resection may result in severe neurologic sequelae, especially in large lesions. Observation with repeated imaging in order to detect growth of the lesion is usually recommended. Debulking of the tumor, mainly aimed at brain stem and cranial nerve decompression, should be considered in cases of disabling and uncontrolled neurologic symptoms and signs.

Key Words: cerebellopontine angle, internal auditory canal, lipoma.

INTRODUCTION

Lipomas involving the internal auditory canal (IAC) and the cerebellopontine angle (CPA) are extremely rare lesions that account for approximately 0.14% of all CPA tumors.[1,2] There are no pathognomonic symptoms associated with these tumors, and their presentation usually mimics that of the more common vestibular schwannoma. The first case of a CPA lipoma was described by Klob[3] in 1859. Since that original report, fewer than 150 cases have been reported in the literature worldwide. Only a few studies have reported more than 4 patients,[1,4-8] and the majority of articles still present case reports. Because of the small number of patients in each of the series reported, it has been difficult to make recommendations for the management of IAC and/or CPA (IAC/CPA) lipomas. The aim of this study was to present our experience with 8 cases of IAC/CPA lipomas, highlighting the clinical and radiologic features, as well as the management and follow-up. We reviewed the literature regarding IAC/CPA lipomas and compiled all articles reporting 4 or more cases.

METHODS

We performed a retrospective analysis of all patients with IAC/CPA tumors seen at Gruppo Otologico between April 1987 and October 2012. Of 3,171 IAC/CPA tumors, 8 (0.25%) were lipomas and were the subjects of this study. The collected data were analyzed for age, sex, side of lesion, presenting symptoms, radiologic features, management, and follow-up. Hearing results were evaluated according to the new standardized format for reporting hearing outcomes in clinical trials.[9] Pure tone average was calculated as the mean of the thresholds at 500, 1,000, 2,000, and 3,000 Hz. The House-Brackmann grading system[10] was used to assess facial nerve function. We also conducted a literature search of Medline without language restriction for studies including the combined key terms "cerebellopontine angle" or "internal auditory canal" or "internal acoustic meatus" and "lipoma" or "rare tumor." This search was expanded by use of the "related articles" links in PubMed and references of the retrieved articles. Publications containing series of

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TABLE 1. RELEVANT CHARACTERISTICS OF PATIENTS WITH IAC/CPA LIPOMAS

Case	*Age (y)*	*Sex*	*Symptoms*	*Site*	*PTA (dB)*	*WRS (%)*	*Treatment*	*Follow-Up (mo)*	*Outcome*
1	43	M	HL, vertigo	IAC	55	40	TLA	12	Grade I FN function
2	39	M	HL, vertigo	IAC	50	50	TLA	13	Grade III FN function at discharge and grade II at 1 year after operation
3	44	M	HL, vertigo, tinnitus	IAC	65	20	TLA	12	Grade VI FN function at discharge and grade III at 1 year after operation
4	40	F	HL, vertigo	IAC	55	40	TLA	7	Grade I FN function
5	34	M	Vertigo	CPA (0.8 cm)	10	100	Observation	48	Growth of tumor; hearing is stable
6	45	F	HL	CPA (1 cm)	45	60	Observation	52	No tumor growth
7	43	M	Aural fullness, tinnitus	IAC, CPA (2.2 cm on right and 2.3 cm on left)	20 (bilaterally)	100 (bilaterally)	Observation	18	No tumor growth
8	71	F	HL	CPA (3.5 mm)	35	70	Observation	14	No tumor growth

IAC — internal auditory canal; CPA — cerebellopontine angle; IAC/CPA — IAC and/or CPA; PTA — pure tone average; WRS — word recognition score; HL — hearing loss; TLA — translabyrinthine approach; FN — facial nerve.

4 or more cases of IAC/APC lipomas were selected for analysis.

RESULTS

Clinical Presentation. Eight patients with IAC/CPA lipomas were identified and analyzed. One patient reported previously is included in this study with longer follow-up.[11] Relevant information on the subjects is provided in Table 1. There was a sex disparity, with 5 men and 3 women. The mean age at the time of diagnosis was 44.8 years (range, 34 to 71 years). Four tumors (50%) were on the left side, and 3 (37.5%) were on the right side. One patient (12.5%) had bilateral CPA lipomas. The follow-ups, consisting of serial magnetic resonance imaging (MRI) scans, ranged from 7 to 52 months (mean, 36 months).

The most common symptoms at the time of presentation included hearing loss (75%), vertigo (62.5%), and tinnitus (25%). Four of the 8 patients had tumor limited to the IAC with no measurable extension into the CPA. Three patients (37.5%) had tumor located only in the CPA; 1 of these 3 patients had a CPA lipoma on the left side and an intracanalicular vestibular schwannoma on the opposite side (Fig 1). One patient (12.5%) had lipomas in both CPA cisterns that partially extended into the IACs.

Surgery. Four cases were radiologically misinterpreted as intracanalicular vestibular schwannomas. The MRI in these 4 patients revealed a purely intracanalicular mass with a high signal intensity on T1-weighted images and a hypointense signal intensity on T2-weighted images. A small amount of enhancement was present after gadolinium administration. Because these 4 patients had nonserviceable hearing, the translabyrinthine approach was used for complete tumor removal. At the time of operation, the tumor was thought to originate from the inferior vestibular nerve in 2 cases and from the cochlear nerve in the other 2 cases. A yellowish mass was found to be very adherent to the facial nerve in all of the cases, and particular difficulty was encountered in separating the tumor from the nerve. All cases were histologically proven to be lipomas. Histologic examination revealed a mass of mature fat cells interspersed between the nerve fibers and ganglion cells, with a minor component of fibrovascular tissue. Two patients experienced postoperative

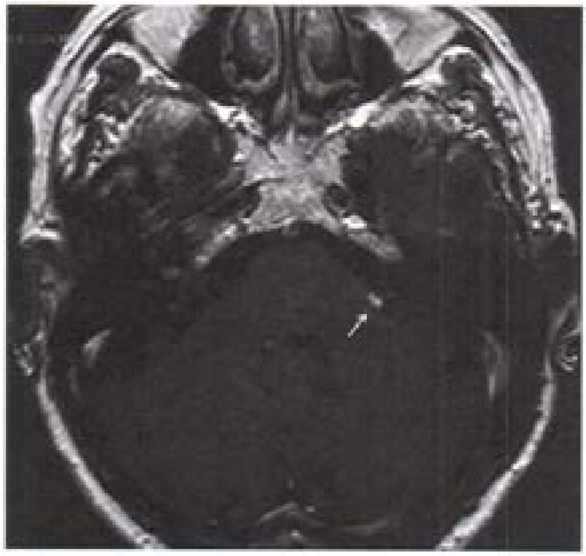

Fig 1. Contrast-enhanced axial T1-weighted magnetic resonance imaging (MRI) shows small cerebellopontine angle lipoma on left side (arrow). Note presence of intracanalicular vestibular schwannoma on opposite side.

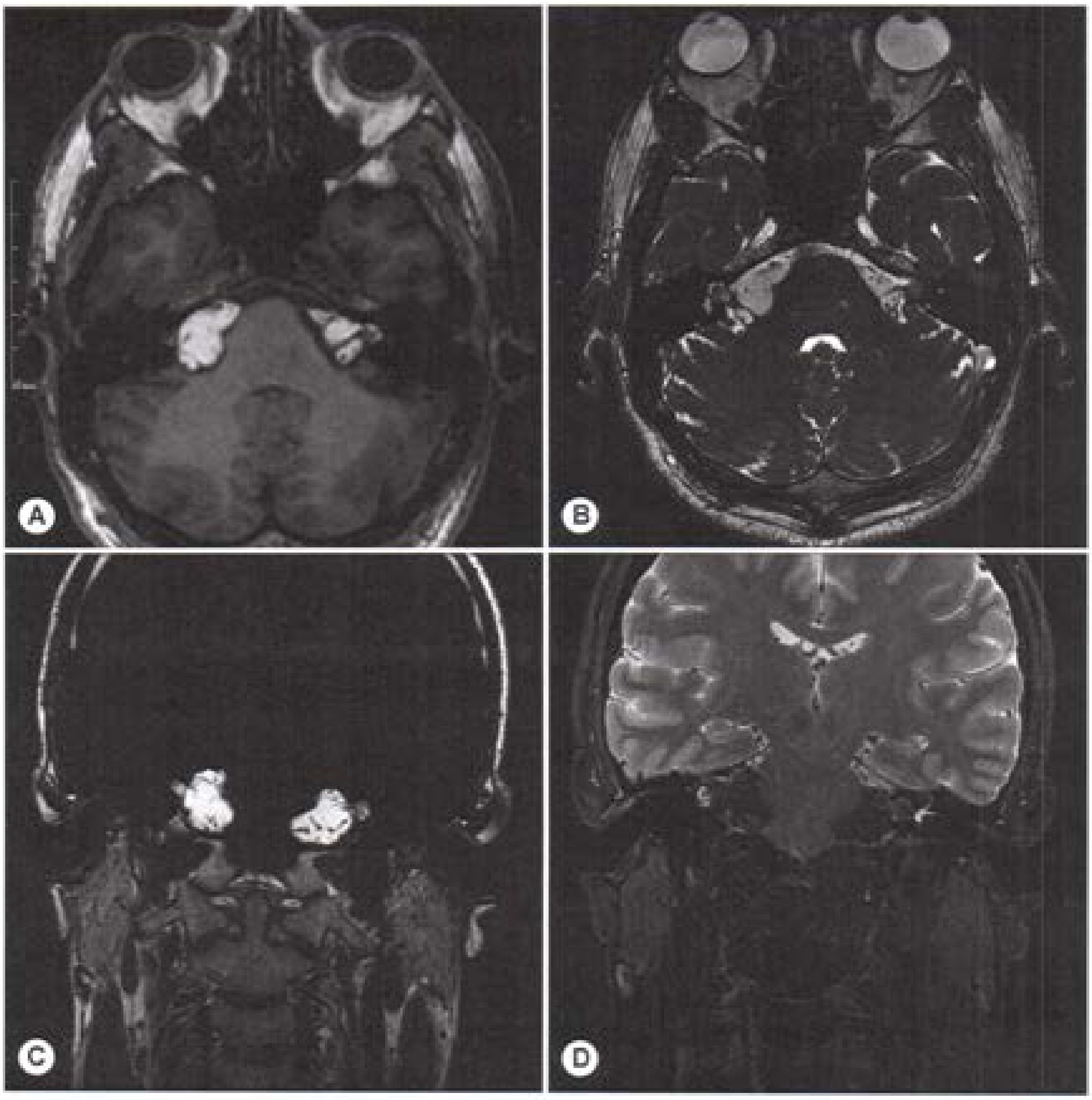

Fig 2. A) Axial T1-weighted MRI shows bilateral hyperintense cerebellopontine angle lesions. **B)** Axial T2-weighted 3-dimensional FIESTA (fast imaging employing steady-state acquisition) shows symmetric cerebellopontine angle lesions with areas of lower intensity consistent with encased vascular and nervous structures. **C)** Coronal IDEAL (iterative decomposition of water and fat with echo asymmetry and least squares estimation) imaging with water suppression emphasizes hyperintense signal of cerebellopontine angle lesions, reflecting their high fat tissue content. **D)** Coronal IDEAL fat-saturated T2-weighted imaging shows signal loss.

facial nerve palsy. Perhaps because of the limited size of the tumors, no other complications were encountered. To date, no recurrences or residual tumors have been observed at neuroradiological follow-up visits.

Observation. Lipomas were suspected in 4 patients (cases 5, 6, 7, and 8) on the basis of imaging findings, and observation was undertaken with repeated MRI at 6-month and then 12-month intervals. The MRI findings revealed all lesions as hyperintense on both T1- and T2-weighted images. Gadolinium administration yielded no enhancement. The facial and vestibulocochlear nerves, together with tortuous vascular structures and fibroconnective septa, were identified on T2-weighted sequences as areas of lower intensity coursing through the masses. In 1 of the patients (case 7) we also acknowledged the benefits of an FSE (fast spin echo) IDEAL (iterative decomposition of water and fat with echo asymmetry and least squares estimation) technique that provides optimal fat suppression with a minimal

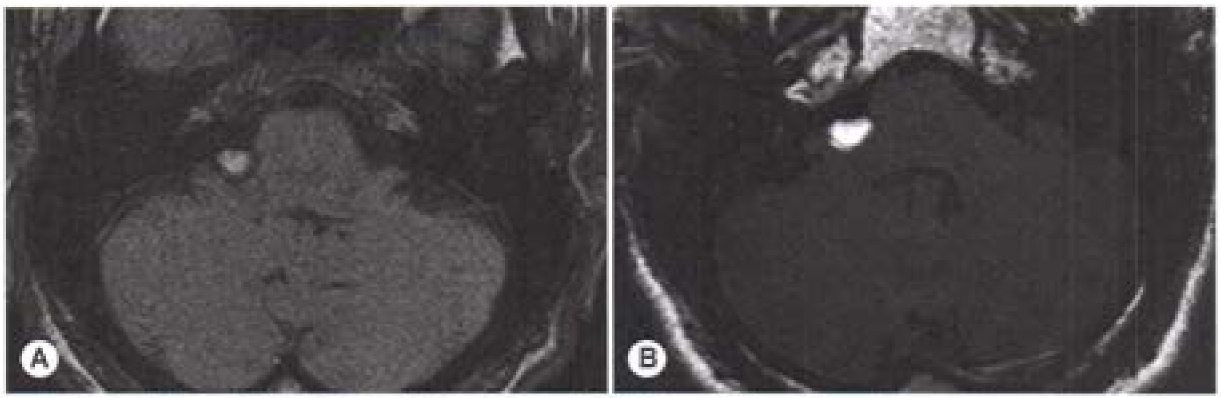

Fig 3. A) Axial T1-weighted MRI from 2007 demonstrates right-sided cerebellopontine angle lipoma (8.6 mm in largest diameter). **B)** Axial T1-weighted MRI of same patient in 2011 reveals growth of tumor (10.4 mm in largest diameter).

increase in scan time as compared with conventional fat-saturated FSE sequences. The FSE-IDEAL sequence of the CPA lesions demonstrated bright hyperintensity on water-saturated T2-weighted images, and signal loss on fat-saturated T2-weighted images (Fig 2). After an average interval of 28 months, MRI revealed no growth of the tumor in 3 patients (cases 6, 7, and 8). Repeat MRI has shown growth of the tumor in 1 patient (case 5; Fig 3). None of these patients reported worsening of hearing during the course of observation.

Literature Review. We identified only 6 articles (Table 2) reporting series of 4 or more well-documented cases.[1,5-8,12] There were 38 patients and a male predominance (male-to-female ratio of 3 to 1). The age of initial presentation ranged between 7 months and 74 years (mean, 42.6 years; SD, 16.4 years). Eighteen lesions (47.4%) were purely intracanalicular, and 20 (52.6%) were extrameatal. One case was diagnosed at autopsy. Clinically, the symptoms caused by CPA and intracanalicular lipomas replicated those of the much more common vestibular schwannomas, with hearing loss being the most common presenting symptom (65.8%), along with tinnitus (44.7%), vertigo (23.7%), headache (21%), facial weakness (5.2%), and dysphagia (2.6%). Three patients (7.9%) were asymptomatic. Sixteen cases (42.1%; 10 intracanalicular and 6 CPA) were treated surgically. The translabyrinthine approach was used in 6 patients for removal of intracanalicular tumors. Hearing preservation tumor removal was attempted via a retrosigmoid approach in 9 patients (6 CPA, 3 intracanalicular) and via a middle cranial fossa approach in 1 patient (intracanalicular). Serviceable hearing was preserved in only 1 case (11%). Total removal of the tumor was accomplished in 9 cases (90%) of intracanalicular lipomas and in only 1 case (16.6%) of a CPA lipoma. Two (20%) of the 10 surgically treated patients with intracanalicular lipomas had postoperative facial palsy. Three (50%) of the 6 surgically treated patients with CPA lipomas complained of postoperative facial nerve palsy. Other complications of surgery included hemiparesis (2 patients), infarctions in areas supplied by the anterior inferior cerebellar artery (1 patient), severe dizziness (1 patient), and significant intraoperative bleeding (1 patient). Twenty-one patients were treated conservatively. Of these, 7 were lost to follow-up. In the remaining 14 cases managed conservatively, no evidence of tumor growth was recorded within a period of observation ranging from 1 to 18 years.

DISCUSSION

Intracranial lipomas are rare lesions that account for 0.1% to 1.5% of all intracranial tumors.[1] Approximately 45% of all intracranial lipomas occur in the interhemispheric fissure. Additional intracranial locations, in decreasing order of frequency, are as follows: quadrigeminal superior cerebellar cisterns (25%), suprasellar interpeduncular cisterns (15%), and CPA (10%).[2] Nowadays, intracranial lipomas should be considered as neither hamartomas nor true neoplasms, but rather, congenital malformations resulting from abnormal persistence and lipomatous differentiation of "meninx primitiva" (a mesenchymal derivative of neural crest) during the development of the subarachnoid cisterns. This hypothesis can also explain the typical location of lipomas in the subarachnoid spaces and the fact that lipomas encompass rather than displace vascular and nervous structures.[2]

Lipomas may be incidentally discovered on brain imaging, as they often remain asymptomatic. Nonetheless, their size may increase in parallel either to the physiologic increase of adipose tissue in pubertal age or to the overall increase of body fat tissue in adults. Therefore, intracranial lipomas located in

TABLE 2. COMPARATIVE REVIEW OF MAJOR IAC/CPA LIPOMA SERIES

Study	Age (y)	Sex	Side	Major Symptoms	Location	Management	Outcome
Christensen et al[12]	61	M	R	None	CPA	Autopsy finding	
	41	F	L	HL, headache, tinnitus, dysphagia	CPA	RS (partial removal)	Loss of hearing; hemiparesis; trigeminal and facial paralysis
	55	F	L	HL, vertigo, tinnitus, facial weakness	CPA	RS (total removal)	Loss of hearing; facial paralysis
	28	M	L	Headache	CPA	RS (partial removal)	Loss of hearing
Saunders et al[5]	54	M	R	HL, tinnitus	IAC	TL (total removal)	
	50	M	L	HL, tinnitus	IAC	TL (total removal)	
	48	M	L	HL	IAC	TL (total removal)	Postoperative grade IV FN function, which recovered to grade II after 1 year
	69	F	R	HL	IAC	TL (total removal)	
	35	M	L	HL, tinnitus	IAC	TL (total removal)	
Greinwald and Lassen[6]	31	M	L	HL, tinnitus	IAC	RS (total removal)	Loss of hearing; postoperative grade IV FN function, which recovered to grade I after 3 mo
	19	M	L	HL, tinnitus	IAC	MFA (biopsy)	No growth at 2 years
	36	M	R	HL, tinnitus	IAC	Observation	No growth at 2 years
	19	M	L	HL, vertigo	IAC	Observation	No growth at 2 years
Bigelow et al[7]	46	M	L	HL, tinnitus	IAC	TLA (total removal)	
	16	M	L	HL, headache, vertigo	CPA	RS (only biopsy)	Significant intraoperative bleeding; loss of hearing
	55	F	L	HL, vertigo	IAC	RS (total removal)	Loss of hearing; severe dizziness
	48	M	L	HL	IAC	RS (total removal)	Hearing preserved
	48	M	L	Headache	IAC	Observation	No follow-up
	(7 mo)	M	R	None	CPA	Observation	No growth at 2 years
	32	M	L	Headache	IAC	Observation	No follow-up
	61	M	R	Vertigo	CPA	Observation	No growth at 2 years
	32	F	L	HL	CPA	Observation	No follow-up
	14	F	Bilateral	Headache	CPA	Observation	No growth at 2 years
	57	F	L	Vertigo, tinnitus	CPA	Observation	No follow-up
	36	F	R	Headache, seizures	CPA	Observation	No follow-up
	63	M	L	None	CPA	Observation	No growth at 2 years
	74	M	R	HL, vertigo	CPA	Observation	No growth at 3 years
	33	M	L	HL	IAC	Observation	No growth at 10 years
	47	F	L	HL, tinnitus	CPA	Observation	No growth at 2 years
	41	M	R	Vertigo	IAC	Observation	No growth at 1 year
Tankéré et al[1]	29	M	L	HL, tinnitus	CPA	Observation	No follow-up
	57	M	L	Tinnitus	CPA	Observation	No growth at 11 years
	56	M	R	HL, facial weakness	IAC	Observation	No follow-up
	56	M	L	HL, tinnitus	CPA	Observation	No growth at 18 years
Rodriguez Prado et al[8]	39	M	R	HL, tinnitus	CPA	RS (partial removal)	Hemiparesis; infarction in areas supplied by AICA; loss of hearing; grade II FN function
	30	M	L	Tinnitus, headache	CPA	RS (partial removal)	Loss of hearing
	45	F	R	HL	CPA	Observation	No growth at 2 years
	60	F	R	HL, tinnitus, vertigo	IAC	Observation	No growth at 1 year

RS — retrosigmoid approach; MFA — middle fossa approach; AICA — anterior inferior cerebellar artery.

TABLE 3. MRI FEATURES OF LIPOMAS AS COMPARED WITH OTHER, MORE COMMON LESIONS OF IAC/CPA

Lesion	*T1-Weighted MRI*	*T2-Weighted MRI*	*MRI With Gd*	*Fat Suppr.*
Lipoma	Hyperintense	Isointense, hypointense, or hyperintense	No enhancement	No signal
Schwannoma	Isointense or hypointense	Isointense or hyperintense	Marked or moderate enhancement	Unchanged
Meningioma	Isointense or hypointense	Isointense or slightly hyperintense	Marked enhancement; "dural tail" sign	Unchanged
Arachnoid cyst	Isointense	Hyperintense	No enhancement	Unchanged
Cholesterol granuloma	Hyperintense	Hyperintense		
Epidermoid	Isointense or hypointense	Isointense or hyperintense	No enhancement	Unchanged
Hemangioma	Hyperintense	Hyperintense	Uniform enhancement	Unchanged

MRI — magnetic resonance imaging; Gd — gadolinium enhancement; Suppr. — suppression.

critical areas may generate symptoms due to compression of adjacent brain structures. Lipomas of the CPA may become progressively symptomatic, as they usually tend to encase and incorporate blood vessels and cranial nerves. Clinically, IAC/CPA lipomas mimic other, more common lesions of the IAC and CPA, such as schwannomas and meningiomas. The most common presenting symptom in our patients was hearing loss, followed by vertigo and tinnitus. These findings are consistent with those reported in the literature.[1,4-8,12]

The most important radiologic differential diagnosis of these tumors includes vestibular schwannomas, meningiomas, arachnoid cysts, cholesterol granulomas, hemangiomas, and epidermoid tumors.[1,11] Table 3 shows the MRI features of lipomas as compared with other, more common IAC/CPA lesions. On high-resolution computed tomography, a CPA lipoma usually presents as a low-density mass without enhancement after administration of contrast medium.[4] On MRI, lipomas are hyperintense on T1-weighted images. The T2-weighted images may have a variable signal intensity (isointense, hypointense, or hyperintense) and a missing signal on fat suppression sequences. Lipomas do not usually enhance with gadolinium.[1,6,11] A missing signal on fat suppression sequences confirms the diagnosis of lipoma. Lower-intensity areas inside the mass may be found and are usually consistent with neurovascular structures. The majority of lipomas topographically originate from the area of the root entry zone of the 7th and 8th nerve complex, whereas schwannomas usually arise from the lateral IAC and extend medially. Cerebellopontine angle meningiomas originate from the posterior surface of the petrous bone anterior or posterior to the IAC; they are large and sessile, and have a broad base of attachment along the posterior petrous surface. These characteristic features make the diagnosis of CPA lipomas relatively simple. A purely intracanalicular lipoma may represent a diagnostic dilemma.[5,6,13] Enhancement of CPA angle lipomas has never been reported in the literature. On the contrary, enhancement of a purely intracanalicular lipoma is not an unusual finding.[5,6] In fact, the majority of surgically treated intracanalicular lipomas, including those of our series, were suspected before operation to be vestibular schwannomas. Therefore, fat suppression MRI is the imaging study of choice to distinguish intracanalicular lipomas from other intracanalicular lesions.

Histologic analysis of lipomas typically reveals the presence of strands of connective tissue and mature adipose cells incorporating nerve fascicles and vessels.[5,7,12] The tendency of adipocytes to infiltrate and separate nerve fibers is unique to IAC/CPA lipomas, as opposed to other intracranial lipomas.[12] This feature makes surgical removal technically difficult.[4]

Treatment of IAC/CPA lipomas presents several drawbacks, especially if the lesion is large. The possible surgical alternatives for IAC lipomas include the retrosigmoid approach, the middle fossa approach, and the translabyrinthine approach, depending on the presence of serviceable preoperative hearing and the tumor location within the IAC. Analysis of the literature showed that the hearing could be preserved in only 18% of the patients who underwent hearing preservation surgery. Postoperative loss of hearing occurs even in very small intracanalicular lipomas because of the gross and microscopic involvement of the cochlear nerve that is usually present with these tumors.[5,14,15] Interestingly, 2 of our patients had postoperative facial nerve palsy, a rare complication in intracanalicular vestibular schwannoma surgery. The data from the literature showed that 29.4% of the surgically treated patients complained of postoperative facial nerve palsy. This outcome can be explained by the technical difficulty of separating the tumor from the facial nerve. The majority of authors remark on the strong adhesions of the tumor to the nerve.[5,6,14,15] It has also been reported that these tumors do not have a capsule, and consequently, the delineation of the tumor from the facial nerve is more difficult.[5]

In tumors extending into the CPA, attempts to achieve complete resection may result in severe neurologic sequelae because of the tendency of the lipoma to adhere to the brain stem and neurovascular structures. The hypervascularization of these tumors, their tight adhesion to the brain stem, and their frequent encroachment on neurovascular structures are some of the criteria that make the resection of these lesions particularly challenging. In our opinion, the risks of surgery should be well balanced against its potential benefits in such slow-growing lesions. Debulking of the tumor, mainly aimed at brain stem and cranial nerve decompression, should be considered in cases of disabling and uncontrolled neurologic symptoms and signs such as vertigo, trigeminal neuralgia, facial weakness, or hemifacial spasm.[1,4] Conservative management in the form of serial MRI is recommended by the majority of authors.[1,4,8] In fact, analysis of the literature demonstrated no evidence of tumor growth within a period of observation ranging from 12 months to 18 years. In our series, growth was demonstrated in 1 patient after 4 years of follow-up. In the series reported by Bigelow et al,[7] a partially resected lipoma had 15% growth by volume in an 8-year period. Since the natural history of these lesions is still not known, indefinite follow-up for these patients will be required. We recommend that repeat imaging should be done at 6 months and then annually.

CONCLUSIONS

Because of the lack of specific symptoms and the limited diagnostic findings, preoperative diagnosis of an IAC/CPA lipoma, especially if it is purely intracanalicular, still represents a challenge. A missing signal on fat suppression sequences constitutes an important differentiating sign between intracanalicular lipomas and the more common lesions of the IAC. The ability to distinguish between an intracanalicular or CPA lipoma and the more common vestibular schwannoma is important for several reasons: 1) preservation of hearing and facial function is less likely with a lipoma than it is with a vestibular schwannoma, even in purely intracanalicular lesions; 2) surgical resection of lipomas involving the CPA may lead to serious consequences due to the marked tendency of these lesions to adhere to the nerves, vascular structures, and surrounding tissue; and 3) observation in the form of serial MRI seems to be the management of choice for such slow-growing lesions.

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