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Case Report

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Cavernous Angioma of the Cerebellopontine Angle

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Abstract

Cavernous angiomas within the cerebellopontine angle (CPA) and internal auditory canal (IAC) are uncommon vascular lesions. To date, fewer than 70 histologically proven cases are reported in the literature. The majority of these lesions are believed to arise from the dural vascular plexus. A small subset of cavernous angiomas originates from the vascular plexus of the vestibulocochlear nerve and, less commonly, the facial nerve. In this report, we present the case of a pathology-proven cavernous angioma likely arising from the cisternal segment of the facial nerve causing vestibulocochlear nerve dysfunction. We then review reports in the literature of cavernous angiomas within the CPA and IAC.

Keywords

Cavernous angioma; Cerebellopontine angle; facial nerve; Internal auditory canal; Cavernoma; Cavernous malformation

Case Report

Physical examination

A 48-year-old right-handed woman without significant past medical history was found to have a left CPA tumor during workup for progressive left-sided hearing loss and headaches. She denied facial weakness or sensory abnormalities. On physical examination, the patient had bilateral House-Brackmann (HB) grade I facial weakness. Audiogram was normal on the right ear, but on the left side it demonstrated moderate sensorineural hearing loss with poor speech discrimination (45 dB speech reception threshold and 40% speech discrimination score). CT of the temporal bone demonstrated a partially high-attenuation left CPA mass with suspicion of an intralesional calcification (Figure 1A). The mass had slightly expanded the IAC. Brain MRI (Figure 1B-E) showed a dumbbell-shaped left CPA angle mass extending into the IAC, measuring approximately 15 × 13 mm in maximal axial dimensions. The lesion was inhomogeneously isointense on T1-weighted MRI with a focal hyperintensity consistent with blood or calcification. On T2-weighted MR the lesion was inhomogeneously hyperintense with a central calcification. The lesion enhanced poorly with gadolinium No frank dural thickening nor was pathologic dural enhancement present.

While the imaging findings of a calcified mass with poor enhancement were somewhat unusual for vestibular schwannoma, the possibility remained and was overall more likely than a vestibular schwannoma mimic operation [1-4].

In the setting of an unfavorable audiogram for hearing preservation, a transtemporal translabyrinthine approach to the lesion was performed. After skeletonization of the IAC, the dura was incised and a variegated red-purple colored mulberry-appearing lesion was found within the canal extending into the cistern (Figure 2A). The majority of the lesion was on the lateral aspect of the facial nerve, between cranial nerves VII and VIII. No attachment to the dura or blood supply from the dura was identified. No subarachnoid blood, macroscopic hemosiderin deposition, or xanthosis was observed over or around the lesion. The superior vestibular nerve was identified and avulsed laterally. Bill's bar was identified, and the plane between the facial nerve and the superior vestibular nerve identified. The inferior vestibular nerve was avulsed laterally, and the cochlear nerve was sacrificed at the brainstem (Figure 2B). The 2 branches of the vestibular nerve and the cochlear nerve along with the lesion were then progressively dissected medially off of the facial nerve. The cerebellum and flocculus were identified and dissected from the lesion, which was in the cerebellopontine angle. The medial portion of the lesion was further dissected from the facial nerve, which actually bifurcated and was completely traversed by the lesion at the cisternal segment (Figure 2B). No arachnoid membrane between the angioma and the facial nerve was identified in this segment, and exfoliation of the lesion was not possible. After attempting gentle dissection of this part of the lesion, it was decided that the lesion was intrinsic to the nerve and could not be safely removed without undue risk to the facial nerve. The decision was made to amputate the lesion at this level and leave in place a small residual (<10%) within the central portion of the facial nerve (Figure 2C). Microscopic examination of the specimen showed a cluster of thin-walled, focally hyalinized blood vessels (Figure 2D) without intervening brain parenchyma. These abnormal vascular channels were further highlighted by reticulin and Movat pentachrome stains (not shown). These findings were consistent with a cavernous angioma.

Follow-up

Immediately postoperatively, the patient had minimal left facial weakness (HB grade II) and she was discharged home on postoperative day 3 in good condition on a short steroid taper. Two days after steroid discontinuation, she had rapid progression of left facial weakness to a HB grade IV, which was managed with high-dose oral steroids resulting in improvement of facial function. At 3-month follow-up visit, she was doing well with a HB grade II on the left side that remained unchanged for 2 years of follow-up. At the 2-year follow-up visit, MRI revealed a very small area of enhancing tissue in the internal auditory meatus consistent with residual lesion, and was unchanged from the postoperative MRI.

Discussion

Cavernous angiomas (cavernous malformations, cavernomas, cavernous hemangiomas) are well-circumscribed multilobulated lesions composed of a compact mass of thin-walled dilated capillaries without recognizable intervening neural parenchyma. These sinusoidal-type vessels have an endothelial lining and a variable

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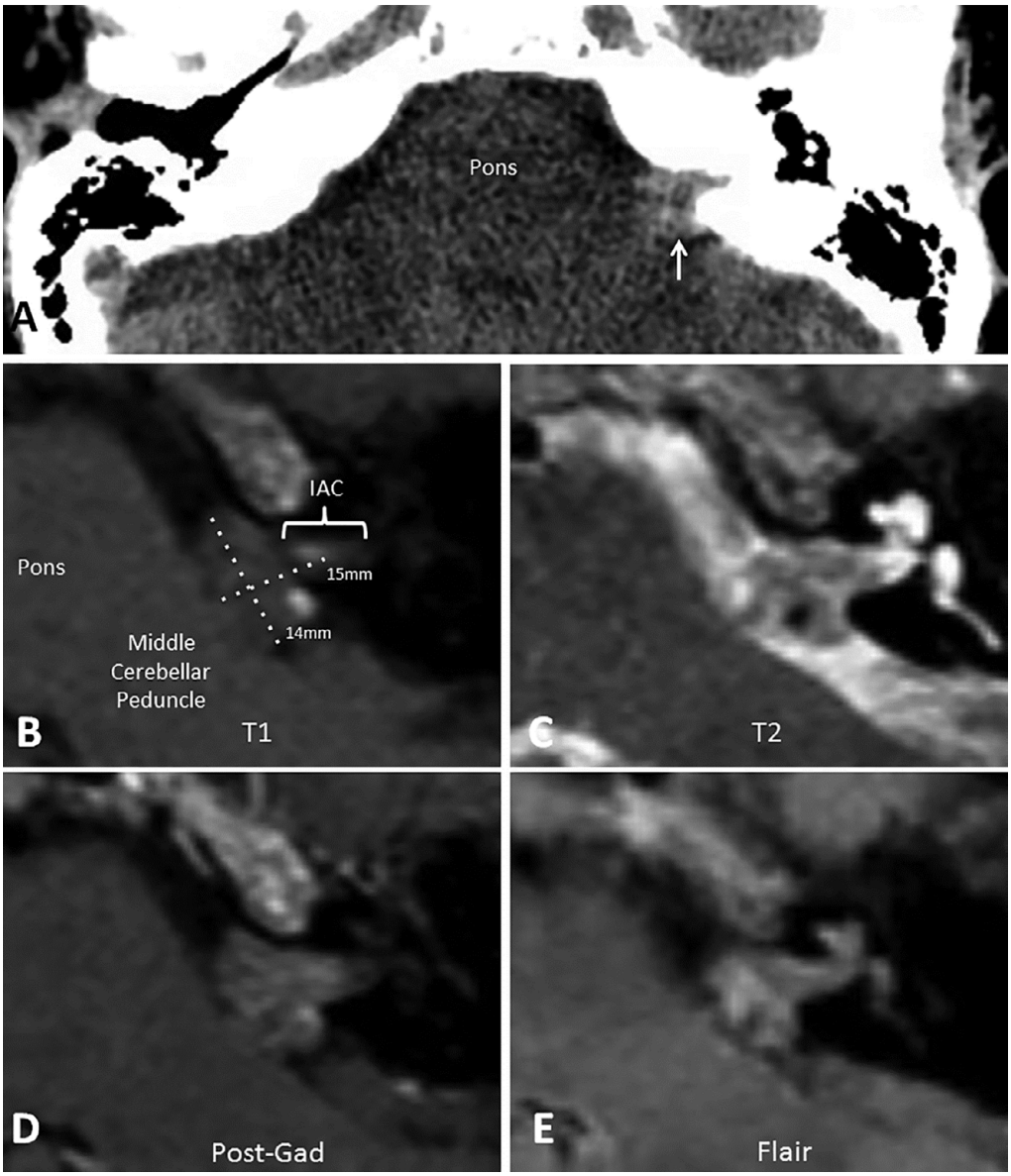

Figure 1: A) Axial non-contrast CT section through the mass showing some subtle high attenuation (arrow) with possibly a central calcification. Axial (B) T1-weighted and (C) T2-weighted MRI sequences showing a 15 x 14 mm extra-axial lesion in left CPA extending into the internal acoustic canal (IAC). On T1 (Fig 1B) there is an eccentric hyperintensity consistent with blood or calcification. On T2 (Fig 1C) there is a central hypointensity probably calcification. The lesion enhances slightly after gadolinium administration (D; Post-Gad) and is hyperintense on (E) FLAIR sequence compared with brainstem parenchyma.

layer of fibrous adventitia without elastin, and they can undergo thrombosis, cystic changes, or hemorrhage.

Cavernous angiomas should be considered in the differential diagnosis of CPA/IAC lesions. A history of acute onset of deficits localizing at the CPA/IAC, with partial improvement followed by acute recurrence of symptoms, and early facial weakness with small intracanalicular tumor, would all suggest cavernous angioma. On CT cavernomas may show some high attenuation in the mass, especially if the cavernoma is calcified or has hemorrhaged recently. There is mostly some enhancement after contrast [46]. Depending on the size of the cavernoma, there may be bone erosion or expansion of the IAC. On MRI, these lesions are typically inhomogeneous with mixed signal on T1- and T2-weighted sequences, but often show some pre-contrast T1 shortening (high signal) and low signal on T2-weighted images due to blood degradation product in different stages of evolution [47]. T2*-weighted (magnetic susceptibility) images usually show very low signal due to chronic hemorrhages in the lesions [48]. Cavernomas usually enhance on intravenous contrast. On MRI too, cavernomas usually show some enhancement after intravenous contrast. Angiography is usually not helpful, as there is little to no angiographic blush in cavernomas due to the large venous channels with very slow flow [49].

Central nervous system cavernous angiomas affect 0.4% to 0.9% of the population, and the vast majorities are intra-axial lesions. The

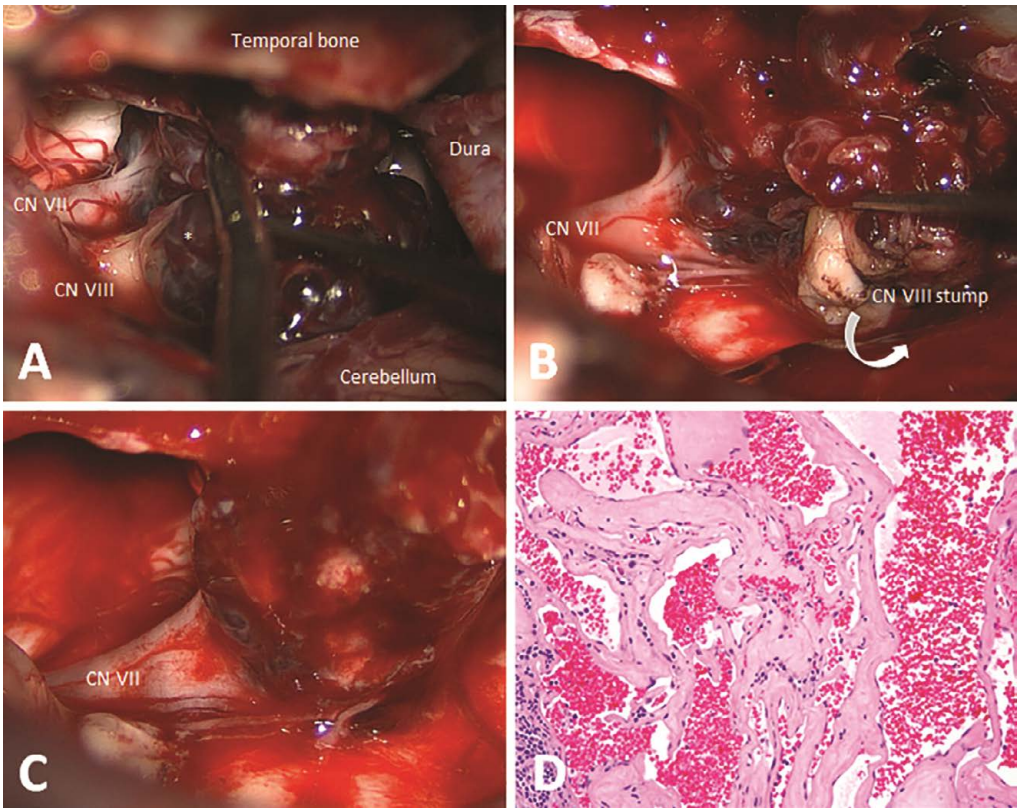

Figure 2: Intraoperative images of left CPA via transtemporal translabyrinthine approach. Exposure of CPA cistern and IAC revealed (A) a mulberry-like lesion attached to (B) the vestibulocochlear nerve (VIII) and piercing the cisternal segment of the facial nerve (VII). (B) Lateral avulsion of vestibulocochlear nerve exposed the deeper portion of the lesion that completely traversed the facial nerve separating its fascicles. (C) The lesion was amputated and a small residual was left in the facial nerve. (D) Histological examination (hematoxylin & eosin staining; 200x magnification) revealed a cavernous angioma with tightly packed collection of thin-walled, hyalinized blood vessels.

rare intracranial extra-axial cavernous angiomas are generally located in the sinuses, optic apparatus, and Meckel's cave, but they can also occur sporadically within the CPA and IAC and pose a true challenge for surgical resection (Table 1). A total of 66 histologically proven cases of cavernous angioma at the CPA/IAC have been reported since its first description by Dale in 1968 [1]. According to a recent paper from Adachi's group, cavernous angiomas of the CPA can be classified into IAC-type or cisternal-type, depending on site of origin [2]. IAC-type cavernous angiomas are more frequent (about 7:1), develop from the vascular plexus of the dura or of the cranial nerves, and are associated with a better rate of postoperative facial function improvement. Cisternal-type angiomas are thought to develop from the vascular plexus of the vestibulocochlear nerves and are extremely uncommon lesions, with less than a dozen cases reported in the literature to date. This type is associated with worse facial function after surgery, likely due to the absent arachnoid membrane between tumor and nerve and the degree of adhesion between the tumor and the nerve. Our patient's lesion occupied the CPA and extended into the ICA, which was nearly completely filled but was not attached to nor had blood supply from the dura. The lesion was adherent to the vestibulocochlear complex and pierced the facial nerve, which was splayed into multiple fascicles surrounding the lesion. It is unclear why the patient had preserved facial function with hearing loss considering the operative findings described above, but a similar case was reported in the literature. One of the possibilities is that the cavernous angioma was a congenital lesion originating from the vascular plexus of the cisternal segment of the facial nerve, and over time, grew enough to compress the vestibulocochlear nerve causing progressive hearing loss. The congenital formation of cavernous angiomas has been strongly supported in the past, but a growing body of evidence sustains a *de novo* formation of these lesions. A recent study regarding the natural history of intra-axial cavernous angiomas demonstrated that imaging prevalence of these vascular lesions increases with advancing age during childhood and early adulthood. Another possibility would be *de novo* formation of the cavernous angioma from the vascular plexus of the facial nerve that grew over time, spreading the fascicles of the nerve and eventually causing compressive neuropathy on the more delicate vestibulocochlear complex. Finally, another possibility would be congenital or *de novo* vascular malformation from the vascular plexus of the vestibulocochlear nerve causing intrinsic nerve dysfunction and hearing loss, which over time asymptomatically eroded throughout the facial nerve fascicles.

Literature Review

We found 10 reports in the literature of significant facial nerve involvement due to the vascular malformation. Nine of these lesions were confined in the IAC, and only 1 lesion was mainly located in the CPA compartment, similar to our patient. The predilection of facial nerve cavernous angiomas to the IAC was previously described by Eby et al., who reported a series of lesions along the facial nerve, and

Table 1: Summary of the literature reporting cavernous angiomas within the cerebellopontine angle and internal auditory canal.*

Case No.	Author & Year	Patient Age (yrs), Sex	Lesion Location	Main Symptoms	Surgical Approach	Intraoperative Finding	Postoperative Results
1	Sundaresan et al., 1976 [5]	23, M	IAC	HL, FW	Retrosigmoid	Lesion attached to CN VIII	Improvement of FW
2		50, M	IAC	HL, FW	Retrosigmoid	Lesion attached to intermediate nerve	Improvement of FW; HL remained
3	Brackmann et al., 1979 [6]	NA	IAC	NA	NA	NA	NA
4		NA	IAC	NA	NA	NA	NA
5	Mangham et al., 1981 [7]	29, M	IAC	HL	Translabyrinthine	CN VII involved, facial-facial anastomosis	Total HL, facial paralysis
6		44, F	IAC	HL	Translabyrinthine	CN VII involved, tumor left around intact facial nerve	Total HL, no FW
7	Iplikcioglu et al., 1986 [8]	30, F	CPA	HL, FW, HA, PE	Retrosigmoid	Cystic tumor attached to CN VII & VIII and pons; cyst filled with xanthochromic fluid, solid nodule	Stable FW
8	Pappas et al., 1989 [9]	26, M	IAC	HL, TN	Translabyrinthine	Lesion adherent to CN VII	Delayed total FW, recovering
9		31, F	IAC & CPA	HL, TN, FW	Translabyrinthine	Lesion adherent to CN VII	FW, improved
10		29, M	IAC & CPA	HL	Translabyrinthine	Facial-facial anastomosis	FW, improved
11		39, M	IAC	HL	Translabyrinthine	NA	Normal FNF
12		56, M	IAC	HL, TN	Translabyrinthine	Lesion adherent to CN VII	Delayed post-op FW, improved
13		44, M	IAC	HL, TN	Translabyrinthine	Lesion adherent to CN VII	Delayed post-op FW, improved
14		66, F	IAC	HL, FW	Translabyrinthine	NA	Normal FNF
15	Madden & Sirimanna, 1990 [10]	36, F	IAC	HL, FW, HFS	Translabyrinthine	Facial-facial anastomosis	1 yr post-op, no improvement in FW
16	Matias-Guiu et al., 1990 [11]	24, F	IAC	HL, TN	NA	Lesion attached to cochlear nerve	Deaf on affected side
17	Bordi et al., 1991 [12]	29, M	IAC	HL	Retrosigmoid	Lesion completely displacing & sectioning CN VII	Facial nerve reconstruction 2 wks post-op, deaf
18	Cremers et al., 1991 [13]	39, M	IAC	HL, TN, FW	Transotic	Facial nerve anatomically intact	Considerable FW
19	Jacobson et al., 1991 [14]	41, F	IAC	Unsteadiness	Middle fossa	Lesion compressing facial nerve	Preservation of hearing, full facial nerve recovery
20	Atlas et al., 1992 [15]	38, F	IAC	HL	Translabyrinthine	NA	NA
21	Eby et al., 1992 [3]	42, F	IAC	HL, HFS, FW	Transotic	Lesion compressing CN VII, CN VIII destroyed	Facial nerve preserved
22		53, M	IAC	HL, HFS, FW, vertigo	Translabyrinthine	8-mm long tumor sharply dissected from facial nerve	Facial nerve preserved
23		41, M	IAC	HL, HFS, FW	Transotic	Severe facial nerve compression (turned into thin transparent fibrous layer)	Facial nerve reconstruction with sural nerve graft (HB grade III in long term)
24	Fujino et al., 1993 [16]	58, M	IAC	HL, TN, vertigo	Retrosigmoid	Lesion adherent to facial & cochlear nerve	Auditory deficit & mild FW
25	Saleh et al., 1993 [17]	44, M	IAC	HL, FW	Translabyrinthine	Lesion surrounding CN VII	FW
26	Babu et al., 1994 [18]	36, M	IAC	HL, vertigo	Retrosigmoid	Lesion originating in CN VII	FNF normal, HL improved
27	Dufour et al., 1994 [19]	NA, M	IAC	HL, HFS	NA	NA	NA
28		NA, M	IAC	HL	NA	NA	NA
29		NA, M	IAC	HL, HFS	NA	NA	NA
30		NA, M	IAC	HL	NA	NA	NA
31		NA, M	IAC	HL, HFS	NA	NA	NA
32	Bricolo et al., 1995 [20]	51, M	IAC	HL, TN, vertigo	Retrosigmoid	Lesion adherent to CN VII	Improvement to asymptomatic
33	Fukuda et al., 1995 [21]	34, M	IAC	Vertigo, HA, HL	Middle fossa	Lesion near CN VII	Normal FNF, HL same
34	Brunori et al., 1996 [22]	60, M	CPA	Facial numbness, TN, HL, vertigo	Retrosigmoid	30-mm cystic tumor attached to CN VII & VIII and pons	Death
35	Greiner-Perth et al., 1997 [23]	32, M	IAC	DZ, TN, HL	Retrosigmoid	Lesion intimately connected to CN VII & VIII	Neurological deficits subsided

36	Kim et al., 1997 [24]	32, M	CPA	Facial numbness, HL	Retrosigmoid	Lesion attached to pons	Facial paresis cleared
37	Kohan et al., 1997 [25]	NA	IAC	HL, TN, FW	Translabyrinthine	Subtotal tumor resection	No CN deficit
38	Omojola et al., 1997 [26]	45, M	IAC	HL, TN, HFS, FW	Translabyrinthine	Facial nerve separated from tumor	FW
39	Roche et al., 1997 [27]	34, F	IAC	HL	Translabyrinthine	Facial nerve anterosuperior to tumor	FW
40		62, F	IAC & CPA	HL, TN	Translabyrinthine	Lesion adherent to CN VII	FW, hypoglossal–facial anastomosis
41	Ferrante et al., 1998 [28]	24, F	CPA	HL	Retrosigmoid	Lesion originating from proximal CN VIII	Transient post-op facial weakness; persistent HL
42	Sasaki et al., 1999 [29]	39, F	IAC	TN, HL	Retrosigmoid	Lesion compressing CN VIII & fanned CN VII; 70% tumor removal	Hearing preserved, FW resolved
43	Gjuric et al., 2000 [30]	43, F	IAC	TN, aural fullness, HA, HL	Middle fossa	Tumor arising from inferior vestibular nerve	Hearing & FNF preserved
44	Sepehrnia et al., 2000 [31]	53, M	IAC	HL, TN, FW	Retrosigmoid	Lesion adherent to CN VII	Mild FW, HL increased
45	Shaida et al., 2000 [32]	30, F	IAC	HL, FW, TN	Translabyrinthine	Lesion compressing CN VII	FW improved
46	Alobid et al., 2002 [33]	61, M	IAC	TN, HL, FW	Retrosigmoid	Lesion compressing CN VII	FNF unchanged
47	Beskonakli et al., 2002 [34]	19, M	CPA	DZ, facial numbness, TN, HL	Retrosigmoid	CN VII & VIII compressed and deformed by mass	Facial hypesthesia
48		25, M	CPA	Facial numbness and HL	Retrosigmoid	Lesion adherent to CN VII & VIII	
49	Aquilina et al., 2004 [35]	29, F	IAC & multiple infratentorial locations	HL & FW	Translabyrinthine	Lesion adherent to CN VII & VIII	FW unchanged
50	Barrera et al., 2004 [36]	21, M	IAC	HL, TN	Translabyrinthine	Lesion adherent to superior vestibular nerve	Uneventful
51	Samii et al., 2006 [37]	23, F	IAC & CPA	SNHL, FW	Retrosigmoid	Preexistent intrameatal hemorrhage	Functional deafness and profound facial weakness pre-op, improved post-op, and resolved at FU
52		28, M	IAC	SNHL, TN	Retrosigmoid	NA	Functional deafness, transient profound post-op facial weakness, persistent moderate FW at FU
53		29, M	IAC & CPA	SNHL, TN	Retrosigmoid	NA	Functional deafness, transient moderate to profound post-op facial weakness, persistent moderate FW at FU
54		40, M	IAC	SNHL, HFS	Retrosigmoid	NA	Functional deafness, immediate post-op cessation of HFS
55		42, M	IAC	SNHL, TN	Retrosigmoid	NA	Functional deafness
56		53, M	IAC	SNHL, FW	Retrosigmoid	Lesion unable to be resected off CN VII; required nerve sacrifice w/sural nerve graft	Functional deafness; required facial nerve reconstruction with immediate complete facial palsy; partial recovery and persistent moderate FW at FU
57		53, F	IAC	SNHL, TN, HFS	Retrosigmoid	Lesion unable to be resected off CN VII; required nerve sacrifice w/sural nerve graft	Functional deafness, required facial nerve reconstruction with immediate complete facial palsy, with partial recovery and persistent moderate FW in FU
58	Adachi et al., 2008 [2]	39,F	CPA	Recurrent episodes of vertigo & HL	Retrosigmoid	Lesion adherent to vestibular nerve, not in contact with cochlear or facial nerve	Deafness
59	Alcantara et al., 2006 [38]	51, F	IAC	TN, HL, DZ	Translabyrinthine & retrosigmoid	Translab procedure aborted due to intense intraop bleeding when opening dura. Tumor resected by suboccipital approach 48 hrs later.	NA
60	Cotton et al., 2006 [39]	45, F	CPA	TN, facial weakness	NA	NA	NA

61	Albanese et al., 2009 [40]	48, M	CPA	Gait instability, loss in tone of voice	Retrosigmoid	Origin of cavernoma seemed to be CN IX–X bundle	Improvement of gait disturbances and hoarseness
62	Sasani et al., 2010 [41]	16, F	CPA	HAs	Retrosigmoid	CN VII & VIII not involved; tumor adherent to brainstem; intratumoral hematoma	Resolution of symptoms
63	Otani et al., 2012 [42]	74, F	CPA	HL, vertigo, TN	NA	Tumor was small, easy to dissect from facial nerve	Normal facial function, complete HL
64	Huang et al., 2011 [43]	50, M	CPA	Vertigo, HL	Retrosigmoid	Lesion adherent to brain stem & cerebellar hemisphere, trigeminal nerve, and facial & acoustic nerves	Improvement of presenting symptoms
65	Wu et al., 2012 [44]	36, M	3 × 3.5 cm lesion adherent to petrous dura with a thin tail extending into IAC	Profound facial weakness, hypesthesia, HL	Retrosigmoid	Flattened but intact CN VII-VIII complex, non-adherent	Facial numbness disappeared and facial paresis improved significantly to HB grade III
66	Ghanta et al., 2013 [45]	50, M	CPA	Dysarthria, dysphagia, unsteady gait	Retrosigmoid	NA	Improvement of symptoms at 3 mos

*Cavernomas at other sites, including temporal bone, middle ear, or geniculate ganglion, were excluded.

Abbreviations: CN = Cranial Nerve; CPA = Cerebellopontine Angle; DZ = Dizziness; FNF = Facial Nerve Function; FU = Follow Up; FW = Facial Weakness; HA = Headache; HB = House–Brackmann; HFS = Hemifacial Spasm; HL = Hearing Loss; IAC = Internal Auditory Canal; NA = Not Available; PE = Papilledema; SNHL = Sensorineural Hearing Loss; TN = Tinnitus

found the most common locations to be the geniculate region, the distal IAC, and the mastoid segment at the departure of the chorda tympani nerve [3]. They concluded that the location of these lesions corresponded to the vascular plexus along the facial nerve described by Maffei et al., which are located in the meatal fundus, geniculate ganglion, and mastoid segment at the takeoff of the chorda tympani [4]. Our case is the second report in the literature of a cavernous angioma arising from the cisternal segment of the vascular plexus of the facial nerve.

Cavernous angiomas in the CPA/IAC generally manifest with sensorineural hearing loss, tinnitus, facial weakness, and hemifacial spasm. Other less common presentations include compression of the cerebellum with ataxia and dysmetria, obstruction of fourth ventricle with resulting hydrocephalus, and very rarely, rupture with clinically significant hematoma or subarachnoid hemorrhage. These symptoms can occur in a slow progressive fashion secondary to long-term compression, or can have an acute presentation followed by stable deficits, partial improvement, or step-wise progression, likely secondary to hemorrhagic events. It is important to note that only 3% of patients harboring intrameatal vestibular schwannomas have facial weakness, and therefore, it has been suggested that onset of facial weakness in patients with unilateral sensorineural hearing loss and a small intrameatal lesion is highly indicative of a cavernous angioma in the ICA. Our patient presented with hearing loss and normal facial function making the differential diagnosis of vestibular schwannoma more difficult. This combination of symptoms would favor the theory that proposes the vascular plexus surrounding the Scarpa ganglion as the place of origin for ICA cavernous angiomas, thus explaining why sensorineural hearing loss is most often the first symptom. However, this was not supported by our operatory findings where the facial nerve was intrinsically involved with the vascular malformation. In addition, surgically confirmed intracanalicular cavernous malformations in patients with normal audiometric and auditory brainstem response findings are present in the literature, and cases where cavernous angiomas arise in the apparent origin of the vestibulocochlear nerve at the pons have been reported.

Residuals or post-surgical remnants of intra-axial nervous system cavernous angiomas carry a high risk of re-bleeding (approximately 40%). To our knowledge, no data is available for cavernous angiomas related to cranial nerves, but complete surgical removal of cavernous angiomas within the CPA/IAC while avoiding complication from bleeding or collateral damage to the nerves has been considered the goal of treatment. In our literature review, 24% of cavernous angiomas at the IAC/CPA were significantly attached to the facial nerve (16 of 66 cases), and 18% were distorted and spread into fascicles (12 of 65 cases). In one case the facial nerve was sectioned by the lesion. From the available data, 33% of the patients (22 cases) presented with preoperative facial weakness that improved in 50% of patients (11 cases), remained stable overtime in 32% of patients (7 cases), and got worse in 14% of patients (3 cases). The risk of developing new postoperative facial weakness was 31% (13 of 41 patients that had normal facial function upon presentation), with potential to occur in a delayed fashion at least in 7% of the cases, and generally recovered if the anatomical continuity of the nerve was preserved. The exact etiology of the delayed facial function decline (HB grade II to IV) experienced by our patient 2 weeks after surgery is unclear, but it was chronologically related to steroid discontinuation and significantly improved shortly after restarting this medication. Another possibility would be intratumor hemorrhage with transitory worsening of her facial function. Per our literature review, 77% of patients with new postoperative facial weakness were found to have robust adhesions of cavernous angiomas to the facial nerves (10 of 13 cases) during surgery, which required excessive nerve manipulation during lesion exfoliation and electrocauterization of feeding vessels, with resulting collateral vascular plexus and nerve damage. In order to achieve gross total resection, 6 patients required nerve sacrifice with reconstruction, leading to HB grade III in all cases at follow-up. In our case, the intimate relationship between the angiomas and facial nerve would have required nerve sacrifice and intrameatal reconstruction to achieve gross total resection, which is associated with transitory complete facial palsy with partial improvement in the long term to HB grade III in 79% of patients and HB grade IV in 29% of patients. Given the intact preoperative facial function of

our patient, we decided to leave a small residual lesion at the facial nerve with the goal of achieving a more cosmetically favorable facial function, and at 18 months after surgery the patient continues to have normal facial function with a small unchanged residual lesion on MRI. As stated above, total excision of these benign lesions should be the goal of treatment when possible, but in those patients with good preoperative facial function found to have a non-resectable portion of the lesion over the facial nerve, we consider debulking with facial nerve preservation followed by observation is an option to consider, given the slow-growing nature of this entity.

Conclusions

In summary, cavernous angiomas of the CPA and IAC are rare lesions that should be considered in the differential diagnosis of vestibular schwannoma. The majority of these lesions originate from the dura, and can be adherent to surrounding cranial nerves. In rare instances they may rise directly from the vascular plexus of the vestibulocochlear and facial nerves. Facial weakness is a common postsurgical complication, even in patients with normal preoperative facial function and with an anatomically preserved facial nerve during surgery. Weakness generally improves if the nerve was not severely damaged or sacrificed. In the exceedingly uncommon situation of a patient with normal facial nerve function with a vascular lesion that cannot be resected from the facial nerve, nerve sacrifice with reconstruction can be attempted to achieve gross total resection. A more conservative option is to leave a small residual to preserve long-term cosmetically acceptable facial function.

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