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Cavernous angioma of the VIIIth cranial nerve

A case report

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Abstract

We report a case of a 24-year-old woman affected by a cavernous angioma of the right VIIIth cranial nerve associated with a venous angioma. The malformation was diagnosed by MRI, performed in relation to an acute onset of right anacusia. The case report is indicative that, even if unusual, an acute onset of an cerebellopontine angle syndrome can be subsequent to a bleeding cavernous angioma. This occurrence must be kept in mind in the differential diagnosis of the cerebellopontine angle tumors.

Keywords: acute anacusia, cavernous angioma, cerebellopontine angle VIII cranial nerve, subarachnoid hemorrhage.

1 Introduction

Cavernous angiomas are vascular malformations which not infrequently involve the central nervous system, and account for between 8 % and 15 % of CNS vascular malformations [6, 9]; in 75–85 % of cases they have a subcortical hemispheric localization [4, 17]. Cavernous angioma of a cranial nerve is exceptional [14, 19]. The authors describe a case of cavernous angioma of the VIIIth cranial nerve.

2 Case report

This 24-year-old woman was referred to us because 2 months previously she had presented with acute onset of right anacusia, together with nausea and vomiting, that regressed the following day.

Audiovestibular testing demonstrated right anacusia and vestibular impairment on the right. MRI detected a lesion at the level of the right cerebellopontine angle that was hyperintense on T1-weighted images and hyper-hypointense on T2 with a hypointense rim (Figure 1). The lesion was visible after i.v. administration of gadolinium and there was also a vascular image whose appearance was suggestive of a dilated draining vein (Figure 2). Digitalized angiography showed the presence of a venous angioma of the right cerebellopontine angle draining into the homolateral tranverse sinus (Figure 3).

On admission the patient presented right anacusia and right lateropulsion at the Romberg test. Since the clinical and radiological evidence indicated a cavernous angioma which had caused hemorrhage, associated with a venous angioma of the righ cerebellopontine angle, the patient was submitted to surgery. Via a right suboccipital retrosigmoidal craniectomy, the cerebellopontine angle was exposed. Once the cistern of the cerebellopontine angle had been opened, a reddish-brown lesion of approximately 8 mm in diameter and displayng signs of the previous bleed was identified at the exit point of the VIII cranial nerve. Once the lesion had been removed (Figure 4), it was evident that it was extra-axial and that the previous bleed had partially interrupted the nerve radicules. There was also a large draining vein that was spared. From the histological point of view the lesion was diagnosed as a cavernous angioma (Figure 5). Postoperatively, the patient presented a mild deficit of the VII cranial nerve that regressed within 10 days. The right anacusia persisted.

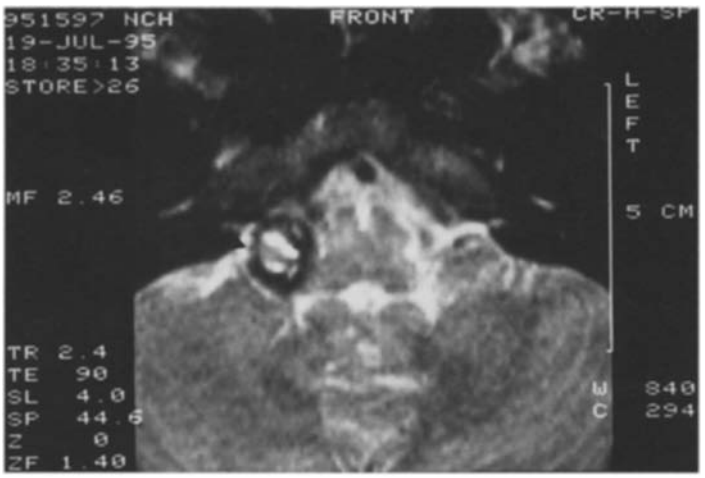

Figure 1. MRI (T2 weighted images) showing an hyper-ipointense cerebellopontine angle lesion with hypointense rim. Arrowhead indicates right VIIIth cranial nerve.

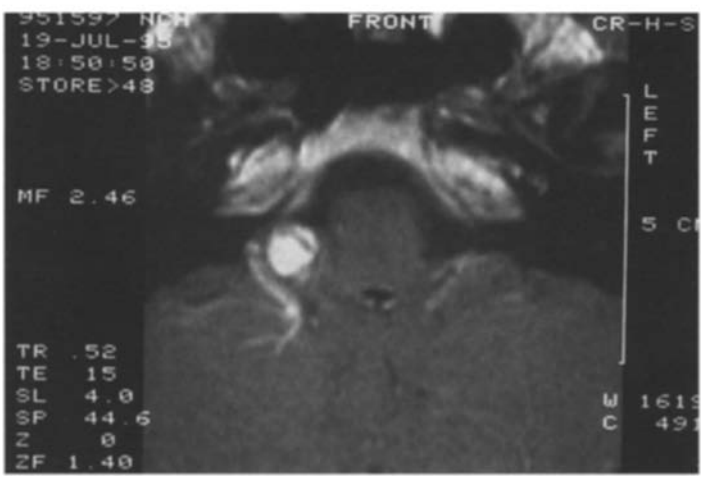

Figure 2. MRI (T1 weighted images after i.v. injection of gadolinium) showing the lesion with a vascular image suggestive of a vein draining from a venous angioma.

3 Discussion

Reports of cavernous angiomas of the cranial nerves are very few and far between. They have been described as affecting the IInd [10, 13], IIIrd [14, 21], and Vth [7] cranial nerves. As far as we know, 18 cases of cavernoma effecting the VII–VIII complex have been reported, the majority of which were localized in the internal accoustic meatus (Table I). In just 4 cases, including the present case, the cavernous angioma affected the VIIIth cranial nerve and in only 1 case (ours) was it localized at the level of the cerebellopontine angle.

The first case of cavernous angioma of the VIII cranial nerve was described by Sundaresan in 1976 [19] in the context of a series of vascular malformations of the internal acoustic meatus: besides a case of AVM of the right VIIIth cranial nerve and a case of cavernoma of the left intermediate nerve, he describes the case of a 23-year-old man with acute onset of hypoacusia and deficit of the left VIIth cranial nerve, harboring a cavernoma of the left VIIIth cranial nerve.

The second case, described by Matias-Guiu in 1990 [14], was that of a 24-year-old woman who presented progressive hearing loss in the right ear and positive Romberg test, due to a cavernous angioma originating from the intrameatal portion of the cochlear nerve. Recently, Bricolo et al. reported the case of a 59-year-old man with a 4-year history of left hypoacusia, caused by the intrameatal portion of a cavernous angioma of the VIIIth cranial nerve. There-

Table I. Details of the 18 reported cases of cavernoma affecting the VII–VIII complex

No.	Reference	year	Sex	Age	Cranial nerve	Side	Clinical presentation	History m.	Symptoms	Audiogram	CT scan
1	Sundaresan	1976	M	23	VIII	L	PNHL	132	PNHL, Defic.VII	–	Cisternography: left ponto-cerebellar angle tumor
2	Sundaresan	1976	M	50	VII	L	PNHL	24	PNHL, Defic. VII	–	Cisternography: intracanalicular tumor
3	Mangham	1981	M	32	VII	R	Def. VII CN	11	Def. VII CN; VII spasm	–	–
4	Mangham	1981	F	54	VII	R	Tinnitus	12	Tinnitus; def. VII	–	–
5	Pappas	1989	M	26	VII	L	Tinnitus	48	Tinnitus; PNHL	SRT 40 dB; PB 48 %	HRCT; air contrast (+)
6	Pappas	1989	M	29	VII	–	PNHL	12	PNHL; Fullness	SRT 30 dB; PB 96 % ABR prolonged ITL-V	HRCT: 7 mm tumor ext. into CPA
7	Pappas	1989	F	31	VII	R	Tinnitus	1,5	Tinnitus; PNHL; Unsteady; defic. VII	PTA 72 dB; PB 0 %	–
8	Pappas	1989	M	39	VII	–	PNHL	2	PNHL; Unsteady	SRT 40 dB; PB 24 %; ABR no response	–
9	Pappas	1989	M	44	VII	R	Tinnitus	60	Tinnitus; PNHL; Unsteady; Fullness	Deaf	–
10	Pappas	1989	M	56	VII	R	Tinnitus	–	Tinnitus; PNHL; Unsteady; Fullness	SRT 70 dB; PB 12 %	HRCT: Normal; HRCT-air cisternography (+)
11	Pappas	1989	M	66	VII	–	HL	Acute	Unsteady; Fullness; Defic. VII	Deaf	HRCT: Flared IAC; HRCT-air cisternography (+)
12	Matias-Guiu	1990	F	24	VIII	R	PNHL	–	PNHL; Tinnitus	–	CT scan (+) with bone erosion
13	Bordi	1991	–	29	VII	L	PNHL	36	PNHL	PTA 60 dB (500–2000 Hrz)	HRCT: Flared IAC; HRCT-air cisterno-graphy (+)
14	Jacobson	1991	F	41	VII	R	Unsteady	1	Unsteady	ABR prolonged I–III	–
15	Saleh	1993	M	44	VII	R	PNHL	7	PNHL; Tinnitus; Defic. VII	PTA 70 dB high tones; PB 45 %; ABR increa-sed V wave latency	–
16	Babu	1994	M	36	VII	R	PNHL	12	PNHL; Unsteady; Vertigo; Nausea	PB 20 %	–
17	Bricolo	1996	M	51	VII	L	PNHL	48	PNHL; Tinnitus; Vertigo; Unsteady	PTA 70 dB middle & high tones; ABR increased V wave latency	HRCT: Flared IAC; small intrameatal calcification
18	Ferrante	1996	F	24	VII	R	HL	Acute	Deafness in right ear	Deaf in right ear	–

No.	Reference	year	Sex	Age	RMN	Angiography	Approach	Surgery	Follow-up
1	Sundaresan	1976	M	23	–	Vert.: indirect signs	Suboccipital retromastoid	Total resection	Excellent
2	Sundaresan	1976	M	50	–	Vert.: negative	Suboccipital retromastoid	Total resection	Excellent
3	Mangham	1981	M	32	–	–	Middle fossa & trans-mastoid	5 mm autograft	Good
4	Mangham	1981	F	54	–	–	Middle fossa & trans-mastoid	Resection of VIIth CN with anastomosis	Poor
5	Pappas	1989	M	26	–	–	Trans-labyrintine	–	Excellent (no time)
6	Pappas	1989	M	29	–	–	Trans-labyrintine	Resection of VIIth CN with direct anastomosis	Excellent (no time)
7	Pappas	1989	F	31	1 cm; 1/2 in CPA hyperintense in T1 and T2	–	Trans-labyrintine	–	Excellent (no time)
8	Pappas	1989	M	39	hyperintense in T1 and T2	–	Trans-labyrintine	7-mm tumor	Excellent (no time)
9	Pappas	1989	M	44	6 mm hyperintense in T1 and T2	–	Trans-labyrintine	–	Excellent (no time)
10	Pappas	1989	M	56	Thickness of VIIth VIIIth CN; hyperint. in T1 and T2	–	Trans-labyrintine	5-mm tumor	Excellent (no time)
11	Pappas	1989	M	66	–	–	Trans-labyrintine	8-mm tumor	Excellent (no time)
12	Matias-Guiu	1990	F	24	–	–	–	–	Poor (4 years)
13	Bordi	1991	–	29	–	Hypoplasia L vertebral artery	Lateral PCF	VIIh t CN resection: delayed anastomosis	Excellent (no time)
14	Jacobson	1991	F	41	8 × 4 mm in CPA	–	Middle fossa	8 × 4 mm tumor	Excellent (6 months)
15	Saleh	1993	M	44	Hyperintense in R IAC with Gd-DTPA; 0.7 cm	–	Trans-labyrintine	7 mm	Good
16	Babu	1994	M	36	Hyperintense in R IAC with Gd-DTPA;	–	Lateral PCF	4 × 6 × 4 mm	Excellent (4 months)
17	Bricolo	1996	M	51	Isointense in L IAC with Gd-DTPA; 0.8 cm	–	Suboccipital retromastoid.	Total resection	Excellent
18	Ferrante	1996	F	24	1 cm; Hyperintense in T1; Hyper-ipo in T2 with Hypo-intense ring	Venous angioma	Suboccipital retromastoid.	Total resection	Excellent

ABR = auditory brain response; ITL-V = wave V interaural latency difference; PTA = Pure tone audiogram; SRT = speech reception threshold; PB = discrimination score; PNHL = progressive neural hearing loss; HL = hearing loss; HRCT = high-resolution computed tomography; IAC = internal auditory canal; CPA = cerebello-pontine angle

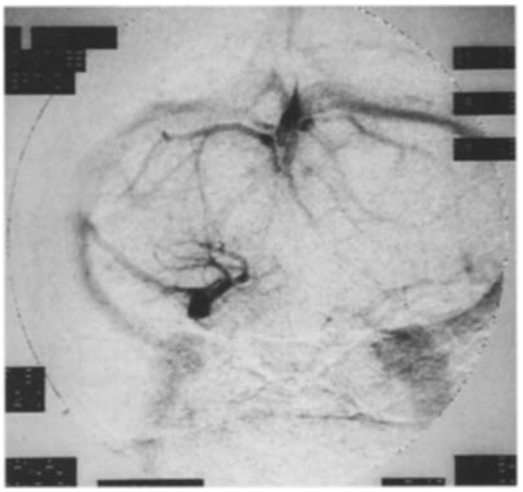

Figure 3. Digitalized angiography showing the presence of a dilated vein of the right cerebellopontine angle draining into the homolateral transverse sinus.

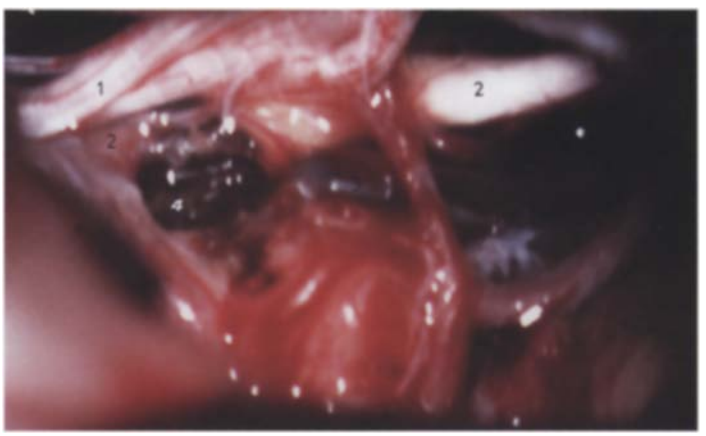

Figure 4. Surgical field. 1: IX-X cranial nerves; 2: VIIth-VIIIth cranial nerves; 3: large draining vein; 4: microsurgical aspect at the end of the excision: the lesion was extra-axial and the old bleeding provoked a partial interruption of the VIIIth cranial nerve rootlets.

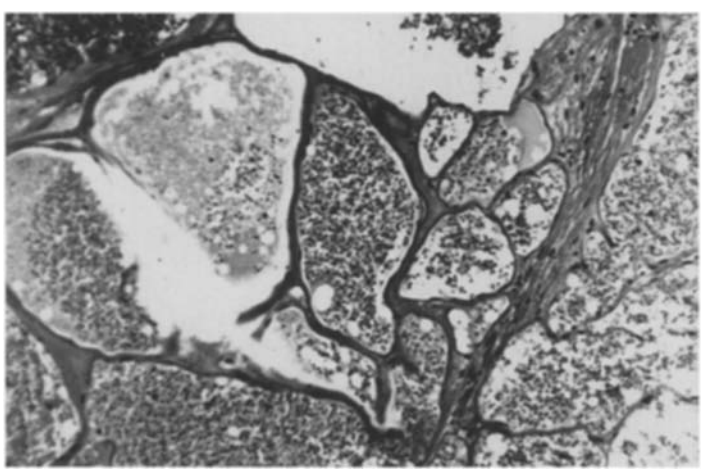

Figure 5. Histology of the lesion. The specimen consists of large, thin-walled pathological vessels separated by connective fibrous tissue. Final diagnoses: cavernous angioma. Hematoxylin and eosin, × 200.

fore, clinical onset was acute in 2 of the 4 cases, because the lesion had caused bleeding. In these cases, the remains of the hemorrhage were visible, although in Sundaresan's case it is likely that the cystic portion of the cavernoma, which contained 2 cc, of coagulated blood, was caused by a hemorrhage subsequent to the one reponsible for clinical onset 11 years prior to surgical treatment.

In the case described by Matias-Guiu, the cavernoma affected the cochlear nerve. Sunderesan does not mention from which portion of the VIIIth cranial nerve the cavernoma originated. In Bricolo's case the cavernoma adhered to both the VIIth and VIIIth cranial nerves, compressing the latter against the lower wall of the internal acoustic meatus. In our case the cavernoma affected the right VIIIth cranial nerve in the area of its entry point into the pons.

CT-scan was performed in 2 cases (Matias-Guiu, Bricolo) and both of them visualized a widening of the internal acoustic meatus. In Matias-Guiu's case, not documented by MRI, this finding suggested an acoustic neuroma, while in Bricolo's case the presence of a calcification within the dilatated internal acoustic meatus, which at operation was found to be a small osteoma, ruled out a preoperative diagnosis of VIIIth cranial nerve neuroma.

MRI was performed in the present case and in the one described by Bricolo. In the latter, the lesion was isointense on T1-weighted images with enlargement of the intracanalicular portion of the VII-VIII complex, and displayed significant enhancement after i.v. administration of gadolinium. In our case, the image was similar to the one described for supratentorial cavernous angiomas with the typical hyper-hypointense appearance on T2-weighted images and a hypointense rim.

In our case, the cavernous angioma was associated with a venous angioma. Between 8 % and 23 % of "occult" vascular malformations [7, 16, 20] present this association and some authors [6, 20] believe that an etiopathogenetic connection exists between these lesions. In cases where this association exists, the acute clinical onset may be attributed to bleeding of the cavernoma. Surgical treatment in these cases consists of removal of the cavernoma alone, because of the risk of cerebral edema secondary to removal of venous angioma [16, 20].

The case reported here demonstrates that, in exceptional cases, the underlying cause of an acute onset of anacusia and vertigo may be bleeding of a vascular malformation and that this eventuality should be considered in the differential diagnoses of tumors of the cerebellopontine angle with an acute onset.

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