ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

www.surgicalneurologyint.com

Surgical Neurology International

Editor-in-Chief: Nancy E. Epstein, MD, Clinical Professor of Neurological Surgery, School of Medicine, State U. of NY at Stony Brook.

SNI: Neurovascular

Editor
Kazuhiro Hongo, MD
Shinshu University, Matsumoto, Japan

SNI
Open Access

Case Report

An isolated cavernous malformation of the sixth cranial nerve: A case report and review of literature

Mohammad Samadian[1,2#], Seyed Farzad Maroufi[2,3#], Mehrdad Hosseinzadeh Bakhtevari[4], Hamid Borghei-Razavi[5]

[1]Department of Neurosurgery, Shahid Beheshti University of Medical Sciences, [2]Skull Base Research Center, Loghman Hakim Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran, [3]Faculty of Medicine, Tehran University of Medical Sciences, [4]Department of Neurosurgery, Day General Hospital, Valiasr, Tehran, Iran, [5]Department of Neurosurgery, Neurological Institute, Cleveland Clinic-Taussig Cancer Center, Cleveland, Ohio, United States.

E-mail: Mohammad Samadian - mdsamadian@gmail.com; Seyed Farzad Maroufi - s.farzadmaroufi@gmail.com; *Mehrdad Hosseinzadeh Bakhtevari - mhbakhtevari@yahoo.com; Hamid Borghei-Razavi - borgheh2@ccf.org
[#]Both authors contributed equally.

***Corresponding author:**
Mehrdad Hosseinzadeh Bakhtevari,
Department of Neurosurgery,
Day General Hospital, Valiasr,
Tehran, Iran.

mhbakhtevari@yahoo.com

Received : 13 August 2021
Accepted : 07 October 2021
Published : 16 November 2021

DOI
10.25259/SNI_811_2021

Quick Response Code:

ABSTRACT

Background: Isolated cavernous malformation (CM) of the abducens nerve has not been reported in the literature. Herein, the authors address the clinical importance of these lesions and review the reported cases of CM from 2014 to 2020.

Case Description: A 21-year-old man presented with binocular diplopia and headache from 2 months before his admission. The neurological examination revealed right-sided abducens nerve palsy. The brain MRI revealed an extra-axial pontomedullary lesion suggestive of a CM. The lesion was surgically removed. During the operation, the abducens nerve was resected considering the lesion could not be separated from the nerve and an anastomosis was performed using an interposition nerve graft and fibrin glue. Pathological examination of the resected lesion revealed that it was originated from within the nerve. The patient's condition improved in postoperative follow-ups.

Conclusion: Surgical resection of the cranial nerves CMs is appropriate when progressive neurological deficits are present. If the lesion is originated from within the nerve, we suggest resection of the involved nerve and performing anastomosis. Novel MRI sequences might help surgeons to be prepared for such cases and fibrin glue can serve as an appropriate tool to perform anastomosis when end-to-end sutures are impossible to perform.

Keywords: Abducens nerve, Cavernous hemangioma, Cranial nerves, Surgical resection

INTRODUCTION

Cavernous malformations (CMs) are occult vascular lesions, defined as hyalinized capillary clusters, without an intervening muscle or neural tissue. These lesions account for 5–15% of all central nervous system vascular lesions[18,21,28,33,38,43] and may cause recurrent hemorrhage, with an annual incidence of 0.6–11% per patient-year.[3,24,26,35,38,41,43,57] It has been shown that genetic factors play a role in the occurrence of CMs, as nearly half of them are familial.[42] They are also equally common in males and females, but tend to occur earlier in men.[43,56]

The supratentorial region is the most common location for CMs followed by the posterior fossa in the pons and the cerebellum.[16,22,38,43,57] As a result of the hemorrhage, CMs commonly present with seizures, headaches, and neurological deficits. However, the symptoms may differ depending

on the lesion site. Intralesional hemorrhage, although rare, can cause growth of the lesion and produce mass effects on the surrounding structures. In terms of the diagnosis and evaluation of these vascular lesions, magnetic resonance imaging (MRI) is considered the gold standard imaging modality.[14,15,44] CMs appear as berry-like lesions with high- and low-intensity reticulated centers, encompassed by hypointense rims in MRI.[11]

Although uncommon, CMs with the involvement of different cranial nerves have been reported in the literature. In general, lesions on the abducens nerve are extremely rare. There is only one case of CM associated with the abducens nerve that originated from the Dorello's canal and encircled the nerve. Herein, we describe the first case of a pathologically proven isolated intraneural and intrafascicular CM of the cranial nerve.

CASE PRESENTATION

A 21-year-old man was admitted to our clinic with a sudden and progressive headache and binocular diplopia. His symptoms had appeared 2 months before his first visit to the clinic. He had also experienced a similar attack 6 months earlier, but he was in a good general condition. The neurological examination revealed right-sided abducens nerve palsy. A brain computed tomography (CT) scan was acquired, which showed a suspected lesion in the cerebellopontine angle. Brain CT angiography was also performed, which did not reveal any prominent findings. The brain MRI showed an extra-axial pontomedullary lesion with the classic appearance of a CM, that is, a reticulated salt-and-pepper core, surrounded by a halo rim [Figure 1]. With the diagnosis of brain stem CM with a history of multiple hemorrhages, progressive neurological deficit, and pial presentation of the CM, the patient was enlisted for the surgical resection of the lesion.

The patient underwent surgery using the suboccipital retrosigmoid approach with intraoperative neuromonitoring. The procedure was performed through general anesthesia as the patient laid in a lateral position, and his head was fixed in a three-pin Mayfield headrest. After opening the dura, a CM was detected, arising from the sixth cranial nerve, with no invasion to the brainstem or the surrounding tissues. Since the lesion could not be separated from the nerve and its integrity was unclear, we resected the CM by cutting the proximal and distal portions (before the Dorello's canal) of the sixth cranial nerve [Figure 2]. Intraoperative monitoring was used to detect changes in the function of the nervous structure. Motor and somatosensory evoked potentials were normal during the surgery. Direct nerve stimulation of the sixth nerve was also performed during the surgery. The response to stimulation was normal when stimulating distal to the lesion and weak proximal to the lesion.

The greater auricular nerve (>3 cm) was harvested as an interposition nerve graft. An anastomosis was performed using a fibrin glue sealant, as end-to-end suture was impossible. One day after surgery, the patient showed complete sixth nerve palsy. He was discharged on the 4th postoperative day, without any neurological deficit, except for the right abducens nerve palsy. In the 5-month follow-up visit, he showed complete recovery of the cranial deficit and diplopia. The histopathological examination of the lesion demonstrated typical characteristics of a CM [Figure 3]. To determine the origin of the lesion, immunohistochemistry staining for the S100 marker was carried out, and the results showed the intraneural origin of the lesion [Figure 3]. The follow-up imaging was normal after 2 years, and the patient was symptom free during this period.

DISCUSSION

Tumors associated with the peripheral nerve are rare entities. Depending on their origin, peripheral nerve tumors are classified into nerve sheath tumors and nonneural sheath tumors.[23] In general, schwannomas and neurofibromas are considered as the most common benign nerve sheath tumors.[48] Benign nonneural sheath tumors primarily consist of lipomas and vascular tumors. On the other hand, malignant peripheral nerve sheath tumors are much less frequent and may include metastasis. Cases of vascular lesions associated with the peripheral nervous system are rare and can be classified into three types: Intraneural extrafascicular malformations, intrafascicular encompassing type lesions, and lesions with both intraneural and extraneural components.[29]

The cranial nerves, as a part of the peripheral nervous system, may be affected by CMs. In 2014, Rotondo *et al*.[45] reviewed the literature on the reported cases of cranial nerves' CMs. To the best of our knowledge, we have gathered all the reported cases of CMs with cranial nerve involvement from 2014 to 2020 in [Table 1]. The demographic characteristics, clinical characteristics, treatments, and treatment outcomes are also summarized in [Table 1]. Based on our review, the optochiasmatic pathway is the most involved cranial nerve, with at least 75 reported cases, which commonly presented with visual loss and headache.[1, 4-6, 17, 25, 31, 32, 36, 46, 49, 51, 53, 54, 59] There are at least 10 reported cases of CMs in the VII/VIII nerve complex (excluding the internal auditory canal CMs), with the main complaint of hearing loss.[10, 30, 34, 46] In nine confirmed cases, the oculomotor nerve was involved, mainly with diplopia and ptosis as the prominent manifestations.[9, 39, 46] The CMs of the trigeminal nerve were also described in six cases, typically presenting with facial dysesthesia or hyperesthesia.[2, 20, 40, 46, 47] The trochlear nerve CMs have been only reported in five patients, with varying presentations.[27, 46, 55] Furthermore, only one case has been reported for the

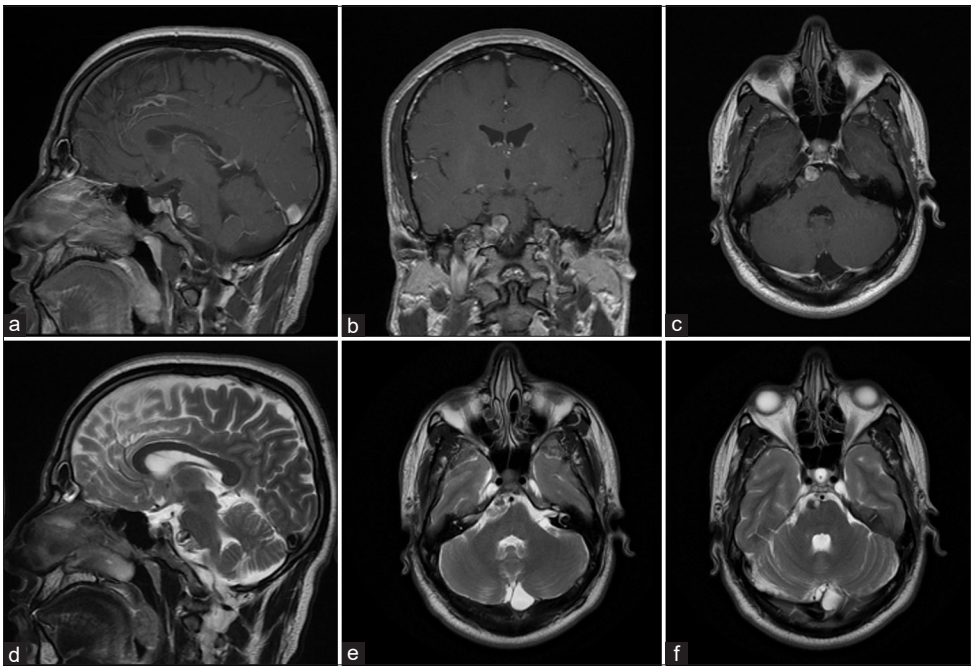

Figure 1: Preoperative MRI images. (a) Sagittal T1-weighted contrast-enhanced image; (b) coronal T1-weighted contrast-enhanced image; (c) axial T1-weighted contrast-enhanced image; (d) Sagittal FSE T2-weighted image; (e) axial T2-weighted sequence; and (f) axial T2-weighted sequence.

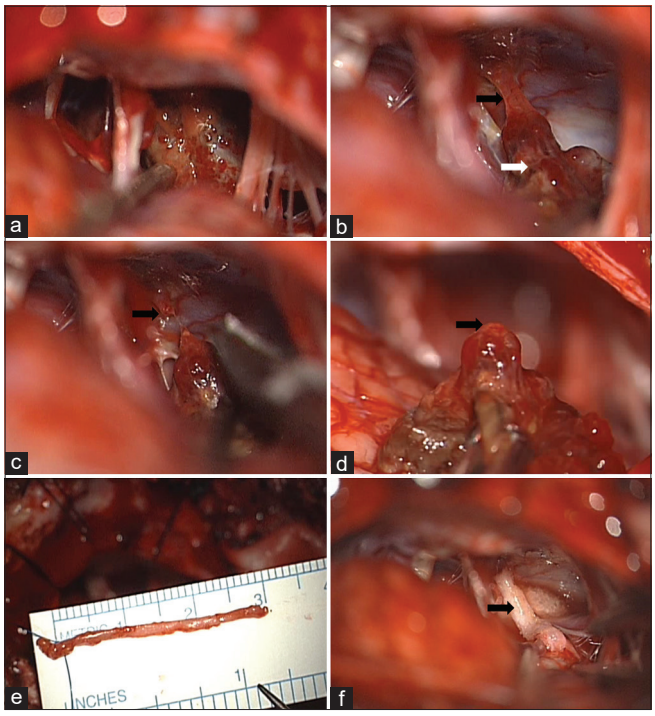

Figure 2: Intraoperative images. (a) A reddish pulsating lesion in the cerebellopontine angle; (b) view of the CM (white arrow) and the distal course of the abducens nerve entering the Dorello's canal (black arrow); (c) severed abducens nerve distal to the lesion (black arrow); (d) the resected CM with part of the abducens nerve (black arrow), passing through the lesion; (e) the prepared nerve graph (3 cm of the greater auricular nerve); (f) resection cavity after removal of the lesion, with the interposition nerve graft placed meticulously between the proximal and distal ends of the abducens nerve.

involvement of each hypoglossal, accessory, and abducens nerve.[13,37,50] Lesions originating from the cerebellopontine angle or the internal auditory canal are more common and may have clinical presentations of cranial nerve CMs, caused by the mass effect of the tumor.

To the best of our knowledge, this is the first case of a pathologically proven isolated intraneural and intrafascicular CM of the cranial nerve. Only one case of the abducens nerve involvement has been described in the literature.[37] The case was a 54-year-old woman with complaints of diplopia and headache and radiological findings, suggesting the hemorrhagic enlargement of the mass on the right cerebellopontine angle. It was reported that during the operation, a duplicated abducens nerve was observed, with a larger branch encircled with a lesion, originating from the Dorello's canal.

Our patient presented with recurrent attacks of progressive diplopia, which showed a similar pattern to other cases of cranial nerve CMs, with acute or progressive neurological deficits, associated with the involved nerve. The recurrence of attacks is probably due to hemorrhage, as reported in the latter case. Although some reported cases of cranial nerve CMs associated with nerve deficits resolved without any intervention,[8,9] since patients usually undergo surgery due to its symptomatic nature and neurological deficit, it is unclear whether the patient's condition improves without any intervention or not.

MRI imaging is crucial for the evaluation of CMs, especially if hemorrhage has occurred previously. In T2-weighted gradient-echo images, lesions with mixed

Table 1: Reported case of cranial nerve CMs from 2014 to 2019.

Authors	Sex and Age	Cranial nerve	Manifestations	MRI findings	Treatment	Outcomes
Algoet *et al*., 2019[6]	M 38	II N	Progressive visual loss	Oval-shaped T2 hyperintense zone	GTR	Improved
DelPino *et al*., 2019[17]	M 42	OC, II N	Intermittent visual disturbances, Intermittent headaches	Oval-shaped lesion with intrinsic T1 hyperintensity	STR	Improved
Ajhoun *et al*., 2019[4]	F 38	OC	Acute visual loss, headaches	Heterogeneous oval lesion and a surrounding rim appeared as hypointense in the T2 and hyper intense in the T1 sequence	OB	Stable
Scavo *et al*., 2018[47]	F 62	V N	Trigeminal neuralgia, gait ataxia, and hypoesthesia	Cystic lesion with a small solid portion that appeared hyper-iso-intense on T1 and hyper-hypo-intense on T2	TR	Improved
Kim *et al*., 2018[25]	F 42	II N, OC	Acute visual disturbance, headache	Ovoid T2 hypointense and T1 isointense lesion	TR	Stable
	M 16	OC	Progressive visual disturbance, headache	Heterogeneous hemorrhagic mass	STR	Improved
	M 17	OC	Acute visual disturbance	Poorly enhanced lesion	STR	Improved (recurred)
Pease *et al*., 2018[40]	F 80	V N	Trigeminal neuralgia	Classic mulberry appearance with an associated developmental venous anomaly	GKRS	Worsened
Meng *et al*., 2017[36]	M 42	OC	Acute visual loss, headache	Isointense T1 and hypointense T2 lesion without enhancement	GTR	Improved
Abou-Al-Shaar *et al*., 2016[1]	F 33	OC	Progressive visual loss, headache	Large heterogeneous hyperintense, hemorrhagic cystic structure on T1	GTR	Improved
Maiodna *et al*., 2016[30]	F 52	VII N (auditory canal)	Headaches, peripheral facial weakness, slurring of speech and dizziness	Lesion with punctate foci of hyperintensity on T1 and hypointensity on T2	GTR+VII,VII N resection+ Anastomosis	Improved but loss of hearing persisted
Trentadue *et al*., 2016[51]	M 49	OC	Asymptomatic	lobulated lesion with smooth, thin, and hypointense borders, an inhomogeneously hyperintense core on T2	FU	Stable
Marnat *et al*., 2015[32]	M 43	OC	Acute visual loss	Heterogeneous lesion on T1 and T2 with a surrounding hyposignal in T2*	FU	Improved
Venkataramana *et al*., 2016[53]	M 27	OC	Subacute visual disturbances, headaches	Lobulated mixed signal intensity lesion	TR	Improved
Zoia *et al*., 2019[59]	F 53	OC	Progressive visual loss, headache	NA	GTR	Improved
Alafaci *et al*., 2015[5]	F 48	OC	Progressive visual loss	Hypointense lesion on T2-weighted images	GTR	Improved
Obaid *et al*., 2014[39]	M 71	III N	Acute binocular diplopia, headache, ptosis	Oval T1 hyperintense and T2 heterogeneous lesion	GTR	Stable
Adachi *et al*., 2014[2]	M 62	V N	Trigeminal neuralgia and hypoesthesia	Lesion appeared hyperintense on T1; heterogeneous, high intense, and isointense with a hemosiderin rim on T2	GTR	Improved but hypoesthesia remained

(Contd...)

Table 1: *(Continued).*

Authors	Sex and Age	Cranial nerve	Manifestations	MRI findings	Treatment	Outcomes
Mano *et al.*, 2014[31]	F 20	II N, OC	Progressive visual loss	Enhanced lesion with hemorrhagic changes	GTR	Stable
Voznyak *et al.*, 2020[54]	M 26	II N, OC	Progressive visual loss, headaches, blood pressure	T1 hyperintense lesion	GTR	Improved
Kraschl *et al.*, 2014[27]	M 70	IV N	Progressive torsional diplopia	A small contrast enhancing lesion	GTR+IV N resection+ anastomosis	Stable
Terterov *et al.*, 2016[50]	F 46	XI N	Headaches, nausea, dizziness, gait instability	T1 isointense and T2 hyperintense mass with enhanced heterogeneously following gadolinium injection	GTR	Improved
Wang *et al.*, 2020[55]	F 49	IV N	Progressive diplopia	Hypointense lesion on T1, mixed signal intense in T2, with heterogeneous enhancement on postgadolinium T1	GTR+IV N resection	Stable
Mastronardi *et al.*, 2016[34]	F 21	VII, VIII	Dizziness, hearing loss	T1 and T2 isointense lesion, with contrast enhancement	STR	Improved
Frossard et al., 2016[20]	F 56	V N	Trigeminal neuralgia, facial anesthesia, temporalis muscle atrophy	Lesion with hemosiderin deposition	STR	NA
Blizzard *et al.*, 2018[9]	M 3 m	III N	Ptosis, decreased extraocular motility, left-sided mydriasis	T1 hyperintense and T2 hypointense zone with postgadolinium enhancement	FU	Improved
Bonfort *et al.*, 2015[10]	M 45	VIII N	Hearing loss, episodic tinnitus	NA	GTR	Improved
Tan *et al.*, 2015[49]	F 60	II N	Acute visual loss, headache	T1 isointense and hyperintense, and T2 isointense and hypointense lesion with minimal contrast enhancement	STR	Stable

F: Female, M: Male, N: Nerve, OC: Optochiasmatic pathway, GTR: Gross total resection, STR: Subtotal resection, GKRS: Gamma Knife radiosurgery, FU: Follow-up, TR: Total resection, NA: Not available

intensity and reticulated cores, surrounded by hypointense rims (corresponding to hemosiderin deposition), suggest CMs. If intact CMs occur, there may not be any prominent feature characteristic of CMs, except for a vague enhancement on gadolinium-based contrast-enhanced MRI. In our case, MRI showed a CM with a previous hemorrhage located at the pontomedullary angle, but we could not determine the origin of the lesion. An important issue that has not been resolved yet is whether CMs develop within the nerve or originate from outside, encompassing the nerve. Although T1- and T2-weighted images are frequently used for the diagnosis of CMs, they cannot provide adequate information about these lesions. The constructive interference in steady-state (CISS) and fast imaging employing steady-state acquisition (FIESTA) sequences is proven to be useful in revealing the lesion boundaries and their relations to the surrounding tissues.[58] Furthermore, reliable assessment of cranial nerves is feasible by this method.[7,12] It can be safely stated that the CISS/FIESTA sequence should be considered as a tool for evaluating the CM origin and nerve continuity and planning a surgical approach.

Full functional recovery after the surgical repair of the transected abducens nerve has been previously reported.[46] In our patient, considering the location of the lesion, we could not repair the abducens nerve using sutures. We used fibrin glue, which has been proven as a safe and effective nerve glue, to repair the transected abducens nerve. Its superiority over sutures has been demonstrated in patients for whom nerve repair using sutures is not possible.[19,52] In our case, no postoperative adverse effect was reported, and repair was successful, as his condition improved in the later follow-ups.

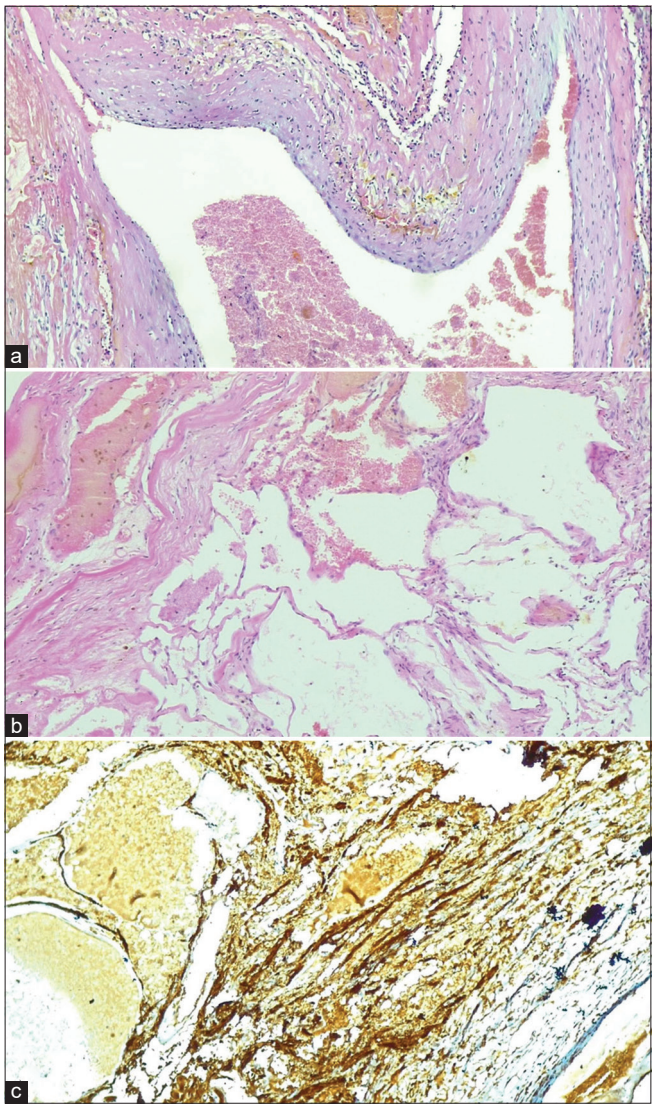

Figure 3: Histopathological image of the lesion. (a) Blood-filled medium-sized vessels (H&E staining; ×100); (b) large cavernous space (H&E staining; ×100); and (c) black stained elongated nuclei of nerve fibers (S100 protein staining, ×100).

CONCLUSION

CMs of the cranial nerve, although rare, pose a significant threat to the nerve functions. Except for our patient, only one other case of CM affecting the abducens nerve was reported earlier. Due to the limited number of cases and scarce evidence, no definite recommendation is available on the indications of surgery and treatment. However, it seems essential to treat patients with progressive neurological deficits, and physicians should consider novel MRI sequences (e.g., CISS/FIESTA), as the traditional sequences do not provide enough information regarding the nature and location of the lesions.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Abou-Al-Shaar H, Bahatheq A, Takroni R, Al-Thubaiti I. Optic chiasmal cavernous angioma: A rare suprasellar vascular malformation. Surg Neurol Int 2016;7 Suppl 18:S523-6.
2. Adachi K, Hasegawa M, Hayashi T, Nagahisa S, Hirose Y. A review of cavernous malformations with trigeminal neuralgia. Clin Neurol Neurosurg 2014;125:151-4.
3. Aiba T, Koike T, Takeda N, Tanaka R. Intracranial cavernous malformations and skin angiomas associated with middle fossa arachnoid cyst: A report of three cases. Surg Neurol 1995;43:31-3; discussion 34.
4. Ajhoun Y, Ismail A, Nisrin L, Yasmine CR, Yassine M, Reda K, *et al*. Unusual etiology of bilateral acute visual impairment: Optochiasmatic cavernoma haemorrhage. Am J Emerg Med 2019;37:564.e561-4.
5. Alafaci C, Grasso G, Granata F, Cutugno M, Marino D, Salpietro FM, *et al*. Cavernous malformation of the optic chiasm: An uncommon location. Surg Neurol Int 2015;6:60.
6. Algoet M, Van Dyck-Lippens PJ, Casselman J, Sirimsi S, Fletcher CD, Van Den Berghe I, *et al*. Intracanal optic nerve cavernous hemangioma: A case report and review of the literature. World Neurosurg 2019;126:428-33.
7. Amemiya S, Aoki S, Ohtomo K. Cranial nerve assessment in cavernous sinus tumors with contrast-enhanced 3D fast-imaging employing steady-state acquisition MR imaging. Neuroradiology 2009;51:467-70.
8. Arrué P, Thorn-Kany M, Vally P, Lacroix F, Delisle MB, Lagarrigue J, *et al*. Cavernous hemangioma of the intracranial optic pathways: CT and MRI. J Comput Assist Tomogr 1999;23:357-61.
9. Blizzard ST, Collins ME, Miller NR. Acute transient oculomotor nerve palsy from presumed cavernous angioma in an infant. Neuroophthalmology 2018;42:229-32.
10. Bonfort G, Veillon F, Debry C, Kehrli P, Chibbaro S. VIIIth nerve cavernous hemangioma mimicking a stage 1 acoustic schwannoma. Neurochirurgie 2015;61:352-5.
11. Borges A, Casselman J. Imaging the cranial nerves: Part II: Primary and secondary neoplastic conditions and neurovascular conflicts. Eur Radiol 2007;17:2332-44.
12. Casselman JW, Kuhweide R, Deimling M, Ampe W, Dehaene I, Meeus L. Constructive interference in steady state-3DFT MR imaging of the inner ear and cerebellopontine angle. AJNR Am J Neuroradiol 1993;14:47-57.

13. Chow M, Addas B, Sangalang V, Holness R. Cavernous malformation of the hypoglossal nerve: Case report and review of the literature. Can J Neurol Sci 2002;29:191-4.
14. Cooper AD, Campeau NG, Meissner I. Susceptibility-weighted imaging in familial cerebral cavernous malformations. Neurology 2008;71:382.
15. de Champfleur NM, Langlois C, Ankenbrandt WJ, Le Bars E, Leroy MA, Duffau H, *et al*. Magnetic resonance imaging evaluation of cerebral cavernous malformations with susceptibility-weighted imaging. Neurosurgery 2011;68:641-7; discussion 647-8.
16. Del Curling O Jr., Kelly DL Jr., Elster AD, Craven TE. An analysis of the natural history of cavernous angiomas. J Neurosurg 1991;75:702-8.
17. DelPino B, Durden J, Deshaies EM. Postoperative management of anterior visual pathway cavernoma, a unique perspective: Case report. Cureus 2019;11:e3819.
18. Di Rocco C, Iannelli A, Tamburrini G. Cavernous angiomas of the brain stem in children. Pediatr Neurosur 1997;27:92-9.
19. Egloff DV, Narakas A. Nerve anastomoses with human fibrin. Preliminary clinical report (56 cases). Ann Chirurg Main 1983;2:101-15.
20. Frossard JT, Domingues F, Neves P, Canhedo N, de Souza JM. Cavernous malformation in the trigeminal distribution: A case report of aggressive presentation and management. World Neurosurg 2016;86:514.e519-22.
21. Giombini S, Morello G. Cavernous angiomas of the brain. Account of fourteen personal cases and review of the literature. Acta Neurochirurg 1978;40:61-82.
22. Kashiwagi S, van Loveren HR, Tew JM Jr, Wiot JG, Weil SM, Lukin RA. Diagnosis and treatment of vascular brain-stem malformations. J Neurosurg 1990;72:27-34.
23. Kim DH, Murovic JA, Tiel RL, Moes G, Kline DG. A series of 146 peripheral non-neural sheath nerve tumors: 30-year experience at Louisiana State University Health Sciences Center. J Neurosurg 2005;102:256-66.
24. Kim DS, Park YG, Choi JU, Chung SS, Lee KC. An analysis of the natural history of cavernous malformations. Surg Neurol 1997;48:9-17; discussion 17-8.
25. Kim HS, Phi JH, Kim JE, Lee JY, Kim SK, Wang KC, *et al*. Cavernous malformations at optic apparatus: Three cases. J Cerebrovasc Endovasc Neurosurg 2018;20:176-80.
26. Kondziolka D, Lunsford LD, Kestle JR. The natural history of cerebral cavernous malformations. J Neurosurg 1995;83:820-4.
27. Kraschl J, Spendel MC, Kiefer A, Kau T, Grossauer S, Vince GH. Cavernous haemangioma of the trochlear nerve: Case report and review of the literature. Clin Neurol Neurosurg 2014;125:65-8.
28. Lonjon M, Roche JL, George B, Mourier KL, Paquis P, Lot G, *et al*. Intracranial cavernoma. 30 cases. Pres Med (Paris, France 1983) 1993;22:990-4.
29. Louis DS, Fortin PT. Perineural hemangiomas of the upper extremity: Report of four cases. J Hand Surg 1992;17:308-11.
30. Maiodna E, Ahmad FU, Morcos JJ. Cavernous malformation of the seventh cranial nerve: Case report and review of literature. World Neurosurg 2016;91:676.e613-21.
31. Mano Y, Kumabe T, Saito R, Watanabe M, Tominaga T. Cavernous malformation of the optic pathway mimicking optic glioma: A case report. Childs Nerv Syst 2014;30:1753-8.
32. Marnat G, Gimbert E, Berge J, Rougier MB, Molinier S, Dousset V. Chiasmatic cavernoma haemorrhage: To treat or not to treat? Concerning a clinical case. Neurochirurgie 2015;61:343-6.
33. Martin NA. Pathology and grading of intracranial vascular malformations. In: Awad I, Barrow D, editors. Intracranial Vascular Malformations. Park Ridge, IL: American Association of Neurological Surgeons; 1990.
34. Mastronardi L, Carpineta E, Cacciotti G, Di Scipio E, Roperto R. Cavernous hemangioma of the internal auditory canal encasing the VII and VIII cranial nerve complex: Case report and review of the literature. Neurosurg Rev 2016;39:349-54.
35. Mathiesen T, Edner G, Kihlström L. Deep and brainstem cavernomas: A consecutive 8-year series. J Neurosurg 2003;99:31-7.
36. Meng X, Feng X, Wan J. Endoscopic endonasal transsphenoidal approach for the removal of optochiasmatic cavernoma: Case report and literature review. World Neurosurg 2017;106:1053.e1011-4.
37. Moon KS, Jung S, Lee KH, Lee MC. Cavernous hemangioma of the abducens nerve: Clinical implication of duplicated variants: Case report. Neurosurgery 2011;69:E756-60; discussion E760.
38. Moriarity JL, Wetzel M, Clatterbuck RE, Javedan S, Sheppard JM, Hoenig-Rigamonti K, *et al*. The natural history of cavernous malformations: A prospective study of 68 patients. Neurosurgery 1999;44:1166-71; discussion 1172-63.
39. Obaid S, Li S, Denis D, Weil AG, Bojanowski MW. Resection of an oculomotor nerve cavernous angioma. Surg Neurol Int 2014;5 Suppl 4:S203-7.
40. Pease M, Withrow J, Ozpinar A, Lunsford LD. Gamma knife radiosurgery for trigeminal neuralgia caused by a cavernous malformation: Case report and literature review. Stereotact Funct Neurosurg 2018;96:412-5.
41. Randall WP, Paul WD, Robert FS, Michael TL, Jonathan JB, Patrick TD, *et al*. Cavernous malformations of the brainstem: Experience with 100 patients. J Neurosurg 1999;90:50-8.
42. Rigamonti D, Spetzler RF. The association of venous and cavernous malformations. Report of four cases and discussion of the pathophysiological, diagnostic, and therapeutic implications. Acta Neurochirurg 1988;92:100-5.
43. Robinson JR, Awad IA, Little JR. Natural history of the cavernous angioma. J Neurosurg 1991;75:709-14.
44. Robinson RJ, Bhuta S. Susceptibility-weighted imaging of the brain: Current utility and potential applications. J Neuroimag 2011;21:e189-204.
45. Rotondo M, Natale M, D'Avanzo R, Pascale M, Scuotto A. Cavernous malformations isolated from cranial nerves: Unexpected diagnosis? Clin Neurol Neurosurg 2014;126:162-8.
46. Sawamura Y, Ikeda J, Miyamachi K, Abe H. Full functional recovery after surgical repair of transected abducens nerve: Case report. Neurosurgery 1997;40:605-7; discussion 607-8.
47. Scavo CG, Roperto R, Cacciotti G, Mastronardi L. Cystic progression of a cavernous malformation at the level of the trigeminal root entry zone presenting with sudden onset of trigeminal neuralgia. J Craniofac Surg 2018;29:e728-30.
48. Schröder J. Tumors of the peripheral nervous system. In: Schröder J, editor. Pathology of Peripheral Nerves: An Atlas

of Structural and Molecular Pathological Changes. Berlin, Heidelberg: Springer-Verlag Berlin Heidelberg; 2001.
49. Tan T, Tee JW, Trost N, McKelvie P, Wang YY. Anterior visual pathway cavernous malformations. J Clin Neurosci 2015;22:258-67.
50. Terterov S, McLaughlin N, Vinters H, Martin NA. Angiographically occult vascular malformation of the intracranial accessory nerve: Case report. J Neurosurg 2016;125:167-72.
51. Trentadue M, Mucelli RP, Piovan E, Pizzini FB. Incidental optochiasmatic cavernoma: Case report of an unusual finding on 3 Tesla MRI. Neuroradiol J 2016;29:289-94.
52. Tse R, Ko JH. Nerve glue for upper extremity reconstruction. Hand Clin 2012;28:529-40.
53. Venkataramana NK, Rao SA, Arun LN, Krishna C. Cavernous malformation of the optic chiasm: Neuro-endoscopic removal. Asian J Neurosurg 2016;11:68-9.
54. Voznyak O, Lytvynenko A, Maydannyk O, Kalenska O, Hryniv N. Cavernous hemangioma of the chiasm and left optic nerve. Cureus 2020;12:e8068.
55. Wang P, Ning W, Qu Y, Zhang H. Cavernous hemangioma of the trochlear nerve in asian population: Case report and review of the literature. Neurol India 2020;68:1203-6.
56. Yamasaki T, Handa H, Yamashita J, Paine JT, Tashiro Y, Uno A, *et al*. Intracranial and orbital cavernous angiomas. A review of 30 cases. J Neurosurg 1986;64:197-208.
57. Zabramski JM, Wascher TM, Spetzler RF, Johnson B, Golfinos J, Drayer BP, *et al*. The natural history of familial cavernous malformations: results of an ongoing study. J Neurosurg 1994;80:422-32.
58. Zausinger S, Yousry I, Brueckmann H, Schmid-Elsaesser R, Tonn JC. Cavernous malformations of the brainstem: Three-dimensional-constructive interference in steady-state magnetic resonance imaging for improvement of surgical approach and clinical results. Neurosurgery 2006;58:322-30; discussion 322-30.
59. Zoia C, Bongetta D, Dorelli G, Luzzi S, Maestro MD, Galzio RJ. Transnasal endoscopic removal of a retrochiasmatic cavernoma: A case report and review of literature. Surg Neurol Int 2019;10:76.

How to cite this article: Samadian M, Maroufi SF, Bakhtevari MH, Borghei-Razavi H. An isolated cavernous malformation of the sixth cranial nerve: A case report and review of literature. Surg Neurol Int 2021;12:563.