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Involvement of the visual pathway is not a risk factor of visual field deficits in patients with occipital arteriovenous malformations: an fMRI study

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Abstract

Background: Occipital arteriovenous malformations (AVMs) are still one of neurosurgery's most intriguing and challenging pathologies. In this study, we reviewed our series of patients with occipital AVMs admitted in Beijing Tiantan Hospital from June 2013 through January 2015 and attempted to evaluate the risk factors of visual field deficits (VFDs) in these patients at presentation.

Methods: Forty-two consecutive patients with occipital AVMs were included in our study. Patient parameters (age, sex, and history of hemorrhage) and AVM characteristics (size, side, venous drainage, Spetzler-Martin grade, and diffuseness) were collected. VFDs were quantified using an Octopus perimetry. Conventional MRI, blood oxygen level dependent fMRI (BOLD-fMRI) of the visual cortex, and diffusion tensor imaging (DTI) of the optic radiation were performed. The least distances from the AVM to the optic radiation (AVM-OR) and from the AVM to the visual cortex (AVM-VC) were measured. Univariate analyses were used to correlate initial VFDs with patient parameters, AVM characteristics, AVM-OR, and AVM-VC distances.

Results: VFDs were identified in 14 patients, among which 12 patients presented with a history of hemorrhage and 2 patients presented with nonhemorrhagic chronic headache. VFDs were more common ($P = 0.000003$) in patients with ruptured AVMs. VFD frequency was not associated with patient age, sex, and AVM characteristics (size, side, venous drainage, S-M grade, and diffuseness). Unlike other lesions involving the optic radiation and visual cortex, the frequency of VFDs in occipital AVMs did not correlate with the AVM-OR and AVM-VC distances ($P = 0.640$ and 0.638 , respectively).

Conclusions: A history of hemorrhage is an independent risk factor of VFDs in occipital AVMs. Most unruptured occipital AVMs may present with chronic headache and seizures other than VFDs. The distances from the AVMs to the optic radiation and the visual cortex are not associated with preexisting VFDs. Our results prompt us to probe into the plasticity of the visual pathway in patients with this congenital vascular anomaly.

Keywords: Arteriovenous malformations (AVMs), Visual field deficits (VFDs), fMRI, DTI

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Background

Optic radiation is a group of axons passing from the lateral geniculate nucleus (LGN) anteriorly over the roof of the lateral ventricle and then turning backwards to the visual cortex. Patients with occipital arteriovenous malformations (AVMs) involving the optic radiation and visual cortex tend to present with visual field disturbances and migraine-like headaches [4, 15, 25]. The risk factors of pre-existing visual field deficits (VFDs) of occipital AVMs have been discussed in previous studies [2, 5, 16, 22, 23]. A history of hemorrhage may be an independent risk factor of VFDs at presentation [2, 16]. Medial occipital AVMs may be associated with more preexisting VFDs [5]. VFDs might occur as a result of occipital lobe seizures [2, 16, 22]. For unruptured occipital AVMs, the etiology of preexisting VFDs may be related to ischemia and/or vascular steal in parenchyma adjacent to the arteriovenous malformation (AVM) [22, 23]. VFDs may also occur as a result of mass effect [2]. Surgical management of an occipital AVM may also cause new or worse VFDs.

Diffusion tensor imaging (DTI) tractography is currently the only noninvasive technique that enables in vivo visualization of the course and characterization of white matter fiber tracts. With DTI, study has demonstrated that the least distance from the lesion to the optic radiation was associated with preexisting VFDs [24], but their series predominantly included gliomas involving the optic radiation. Distinct from brain tumors such as gliomas and other congenital vascular lesions such as vein of Galen malformations and dural AVMs, brain AVMs are described as a congenitally abnormal complex of afferent arteries communicating with draining veins [11]. Risk factors of preexisting VFDs in occipital AVMs may differ from those caused by tumors and other pathologies. Till now, the literature provides few systematic accounts of the relationship between preexisting VFDs and the distance from the AVMs to the optic radiation or visual cortex. Is it possible that the preexisting VFD is associated with the distance from the AVMs to the optic radiation or visual cortex? What are the risk factors of preexisting VFDs in patients with occipital AVMs? In this study, we will attempt to answer these two questions and discuss our preliminary results.

Methods

Patient population

From June 2013 through January 2015, forty-two consecutive patients who were admitted for surgery in the Department of Neurosurgery in Beijing Tiantan Hospital, Capital Medical University, for occipital AVMs were included in this study (Table 1). Digital subtraction angiography (DSA) was performed in all patients. Records of patient parameters (age, sex, history of intracranial hemorrhage) and AVM characteristics (size, side,

Table 1 Demographics and characteristics of the 42 patients with occipital AVMs

Parameters	No. of patients
M/F (ratio)	29:13
Mean age in yrs (range)	26.0 (6–47)
Side (L/R)	21:21
AVM size	
<3 cm	11
≥3 cm, <6 cm	28
≥6 cm	3
Venous drainage	
Superficial	28
Deep	14
Diffuseness	
Compact	24
Diffuse	18
Spetzler-Martin grade	
I	2
II	19
III	14
IV	7
V	0
AVM-OR	
<5 mm	34
≥5 mm	8
AVM-VC	
<5 mm	17
≥5 mm	25
History	
Hemorrhage	15
Headache	16
Seizure	9
Dizziness	2
VF	
Grade 0	28
Grade 1	8
Grade 2	2
Grade 3	4
Grade 4	0

AVM-OR distance from AVM to optic radiation, AVM-VC distance from AVM to visual cortex, F female, L/R left/right, M male, No. number, VF visual field, yrs years

venous drainage, Spetzler-Martin grade, and diffuseness) were collected. This study was approved by the Institutional Review Board of Beijing Tiantan Hospital Affiliated to Capital Medical University, and informed written consent was obtained from all of the adult patients and from the parents of the pediatric patients.

Imaging acquisition

A 3 T Siemens Tim Trio magnetic resonance imaging (MRI) scanner with a 12-channel head coil was used for scanning protocol. T1-weighted structural imaging, blood oxygen level dependent functional magnetic resonance imaging (BOLD-fMRI), time of flight magnetic resonance angiography (TOF-MRA), and DTI were collected for each patient. All imaging and image processing were performed by two technical assistants at the Medical Imaging Center, the 306th Hospital of PLA. All data analyzing procedures were done by two physicians at our institution.

T1-weighted three-dimensional magnetization-prepared rapid acquisition gradient echo sequence was measured with a echo time (TE) of 2.98 ms, a repetition time (TR) of 2300 ms, a matrix size of 256×256 , a field of view of 250×250 mm, a slice thickness of 1 mm, and a slab size of 17.6 cm.

For BOLD-fMRI, maps of neural activity within the visual cortex were generated in each patient. Visual task given was “black white checkerboard”. A “block” design was used in which there were 80 dynamics and every 10 dynamics of activity was alternated with 10 dynamics of rest, beginning with rest. The total time of the task was 240 s. The fMRI sequence was a fast-field echo single-shot echo planar imaging technique with an echo planar imaging factor of 79, a TR of 3000 ms, a TE of 30 ms, and a flip angle of 90. Contiguous slices, 6 mm thick, were taken, and 30 slices were obtained. The field of view was 210×210 mm, with a matrix size of 64 mm, resulting in an in-plane resolution of 3.3 mm. Voxel size was $3.3 \times 3.3 \times 4.0$ mm. For each slice, 80 dynamic time points were collected, providing a total imaging time of 4 min and 8 s, 30.1 s for each run.

Time of flight MRA (TOF-MRA) was obtained by the following parameters: TR, 22 ms; TE, 3.86 ms; matrix size, 384×256 ; field of view, 220×180 mm; bandwidth, 178 Hz per pixel; voxel size, $0.8 \times 0.6 \times 1.0$ mm; flip angle; 18. 30 slices were obtained, and the total imaging time was 3 min and 29 s.

For DTI, we applied a single-shot spin-echo diffusion-weighted echo planar imaging sequence (TE, 93 ms; TR, 6100 ms; matrix size, 128×128 ; field of view, 230×230 mm; bandwidth, 1396 Hz per pixel; voxel size, $1.8 \times 1.8 \times 3$ mm and diffusion-encoding gradients in 12 directions using b values of 0 and 1000 s/mm^2). We used 45 slices with no intersection gap. The slice thickness was 3 mm, and each slice was angulated by 40° anteriorly to cover the optic chiasm, the occipital lobe, the corpus callosum, and the upper two thirds of the temporal lobe. The time needed for image acquisition was 6 min and 38 s.

Data analysis

The MRI data were saved as DICOM format. The data were transferred to an off-line iPlan 3.0 workstation

(Brainlab, Feldkirchen, Germany) for analysis. Motion correction was done in raw BOLD-fMRI. The raw DTI images were corrected for eddy currents. The TOF-MRA, BOLD-fMRI, and DTI images then automatically integrated with T1 structural images. Using the general linear model (GLM), the BOLD data were analyzed according to the stimuli parameters of visual task. The 3D model of the visual area was reconstructed according to the activated volumes that were considered to consist of $P = 0.001$ activated areas. For fiber tracking of the optic radiation, the ipsilateral lateral geniculate was selected as the first seed volume. All the fibers that cross this volume of interest (VOI) were tracked. The second seed volume was the activated visual cortex. Fiber tracking was initiated with fractional anisotropy (FA) of 0.2. At last, the 3D anatomic structure of the AVMs was reconstructed by threshold adjustment of the TOF-MRA data.

Acquisition and labeling of important parameters

Through the iPlan 3.0 software, the maximum diameters of the AVMs were measured from the structural MRI on the axial, coronary, and sagittal directions. The distances of AVM from the optic radiation (AVM-OR) and visual cortex (AVM-VC) were measured respectively on the axial, coronary, and sagittal directions. The least distance was what this study required.

Visual field assessment

Visual fields testing of the central 30° were performed with an Octopus field analyzer (Haag Streit International, Koeniz, Switzerland) by an experienced ophthalmologist who was blinded to the results of the neuroimaging findings. As to Kupersmith et al. [16], VFDs were graded as normal VFs (grade 0), incomplete quadrantanopsia (grade 1), complete quadrantanopsia (grade 2), incomplete hemianopsia (grade 3), and complete hemianopsia (grade 4). Mild defects were defined as grade 1 and grade 2, while grade 3 and grade 4 were referred to as severe defects.

Statistical analysis

Statistical analyses were performed with the Statistical Package for the Social Sciences software (version 20.0; SPSS, Inc, Chicago, IL). Univariate analyses were used to assess the risk factors of preexisting VFDs. Statistical significance was established at a value of $P < 0.05$.

Results

Demographic data and clinical presentations

The baseline characteristics of the patients and AVMs are listed in Table 1. Besides VFDs, none of our patients presented with more than one symptom. Fifteen (36 %) patients presented with a history of hemorrhage, 9 (21 %) with seizures, 16 (38 %) with nonhemorrhagic chronic headaches, and 2 (5 %) with intermittent dizziness. In ten

patients with nonhemorrhagic headache, there was an absolute preference for the side ipsilateral to the AVM. The headache was described as throbbing in seven cases and associated with visual phenomena (blurred vision in hemifield or flickering) in six cases.

The AVMs ranged in diameter from 13.5 to 70.0 mm (mean 39.3 mm). The size was <3 cm in 11 cases and ≥3 cm in 31 (including ≥6 cm in 3, Table 1). The primary feeding arteries to the AVM were through the cortical branches of the posterior cerebral artery, but all AVMs ≥3 cm in diameter had multiple feeding arteries involving the anterior circulation. Of the 42 cases in this study, 14 had deep venous drainage or both deep and superficial venous drainage while the other 28 had only superficial venous drainage. AVM configuration (compact vs. diffuse) was defined according to previous study [17].

Evaluation of preexisting VFDs

As listed in Table 1, 14 patients presented with varied degree of homonymous VFDs at evaluation. No one in our series had complete hemianopsia (grade 4) at presentation. All patient data were entered into a statistical spreadsheet, and univariate analyses were performed to correlate patient parameters (age, sex, and clinical presentation), AVM characteristics (size, side, venous drainage, S-M grade, diffuseness, the least AVM-OR and AVM-VC distances) with preexisting VFDs (Table 2). Twelve of the 15 patients who bled had preexisting VFDs (grade 1 in 7, grade 2 in 1, grade 3 in 4). VFDs were identified in only 2 of the 27 patients with unruptured AVMs (one with grade 1 and the other with grade 2) and both 2 patients had chronic headaches. Patients with a history of hemorrhage were more likely to experience initial VFDs ($P = 0.000003$). VFD may be more likely to occur in patients that had AVMs with deep venous drainage. However, that difference did not reach statistical significance ($P = 0.165$).

As to the relationship between AVMs and the optic radiation or the visual cortex, the incidence of VFDs was not associated with the least AVM-OR distances ($P = 0.640$) and AVM-VC distances ($P = 0.238$) (Table 2). Asymmetry of the optic radiation or the activated visual cortex occurred in 38 patients (Figs. 1, 2, and 3). All 14 patients with VFDs had asymmetrical optic radiation and visual cortex (Fig. 1). In patients with VFDs, the optic radiation fibers on the lesion side were significantly thinner than those on the contralateral side (Fig. 1). For the seven patients with both the least AVM-OR distance and AVM-VC distance were zero (Figs. 1, 2, 3, and 4), preexisting VFD was observed in only two patients who had a history of hemorrhage (Fig. 1). For the other five patients with unruptured AVMs, the VF was normal (Figs. 3 and 5). Of the 11 cases that only the AVM-OR distance was zero, 4

Table 2 VF evaluation in the 42 patients with occipital AVMs

Parameters	Normal VFs	VFDs	P value
Gender (n)			0.729
M	20	9	
F	8	5	
Age (ys)	26.6 ± 9.9	24.8 ± 14.3	0.674
Side (n)			0.744
Left	13	8	
Right	15	6	
Size (mm)	39.8 ± 12.8	38.6 ± 13.4	0.787
Size			1.000
<3 cm (n)	7	4	
≥3 cm (n)	21	10	
Venous drainage (n)			0.165
Superficial	21	7	
Deep	7	7	
Diffuseness (n)			0.742
Compact	15	9	
Diffuse	13	5	
S-M grade	2.7 ± 0.8	2.5 ± 0.9	0.515
AVM-OR (mm)	2.9 ± 3.9	2.4 ± 2.7	0.640
AVM-OR			1.000
<5 mm (n)	23	11	
≥5 mm (n)	5	3	
AVM-VC (mm)	7.8 ± 9.4	11.3 ± 7.2	0.238
AVM-VC			0.102
<5 mm (n)	14	3	
≥5 mm (n)	14	11	
History of hemorrhage			0.000003
Ruptured	3	12	
Unruptured	25	2	
Chronic headache			0.042
Yes	14	2	
No	14	12	
Seizure			0.017
Yes	9	0	
No	19	14	

AVM-OR distance from AVM to optic radiation, AVM-VC distance from AVM to visual cortex, F female, M male, VFDs visual field deficits, VFs visual fields, ys years

cases who bled presented with VFDs. The visual field was normal in the remaining seven nonhemorrhagic cases. In those four cases with only the AVM-VC distance was zero, none had a history of hemorrhage and all had a normal VF. Overall preexisting VFDs occurred in six patients that had a history of hemorrhage and zero distance from the optic radiation or visual cortex or both. For those three

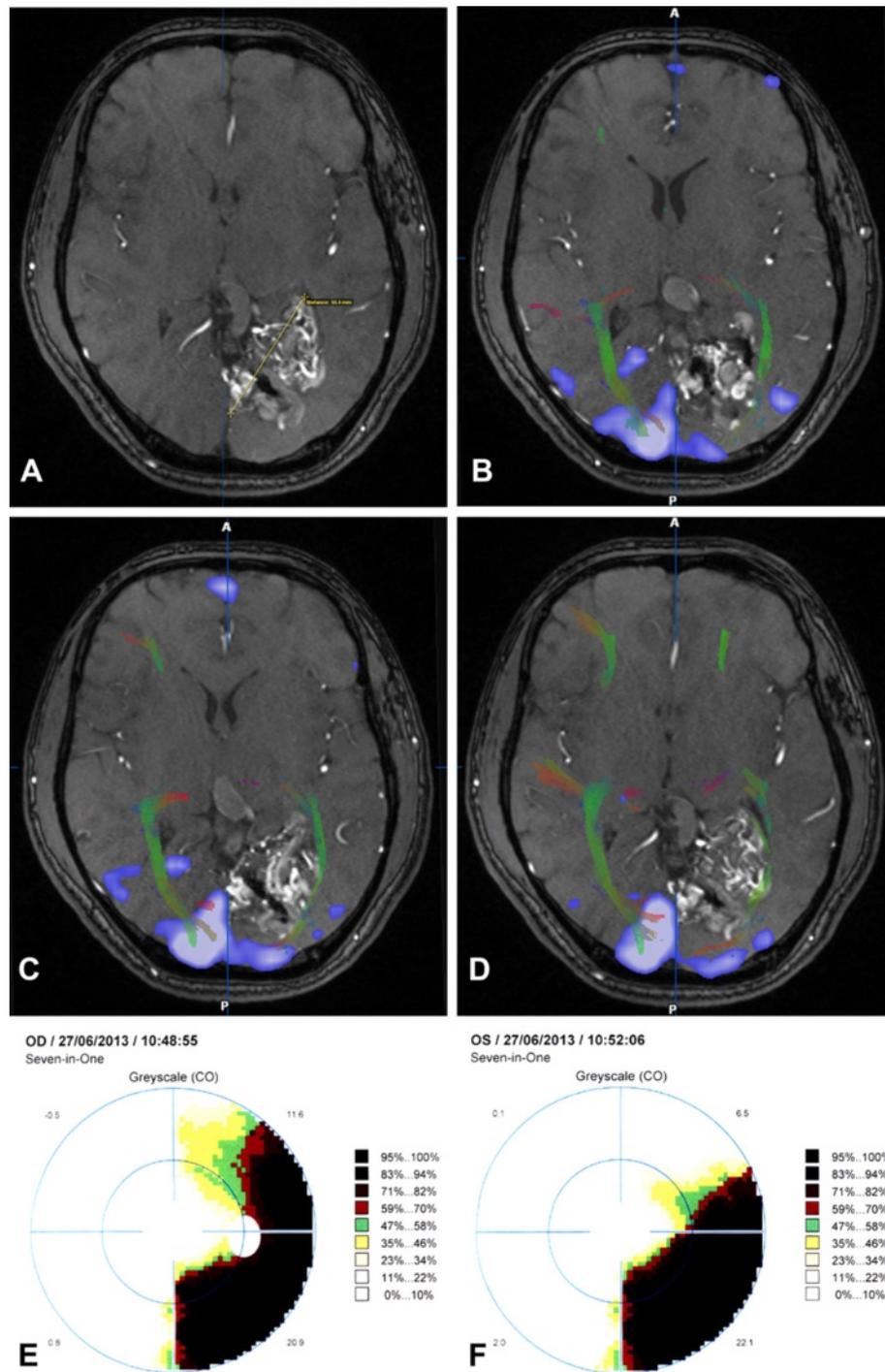


Fig. 1 Case 3. A 42-year-old male with a history of hemorrhage. **a** Enhanced structural axial T1-weighted image showing a left occipital AVM with a maximum diameter of 55.4 mm (yellow line). **b–d** Based on enhanced structural axial T1-weighted image, fiber tracking showing the asymmetry of optic radiation (green) and visual cortex (blue). Both the AVM-OR and AVM-VC distances were zero. The optic radiation ipsilateral to the AVM is much thinner than that of the contralateral. **e–f** Visual fields testing showing a homonymous partial hemianopsia

patients who bled but maintained a normal VF, the least AVM-OR distances were 5.9, 12.3, and 12.5 mm, respectively. The least AVM-VC distances were 5.3, 20.0, and 25.6 mm, respectively. For those two patients

presenting initial VFDs without a history of hemorrhage, the least AVM-OR distances were 2.6 and 3.0 mm, respectively. The least AVM-VC distances were 4.1 and 8.2 mm, respectively.

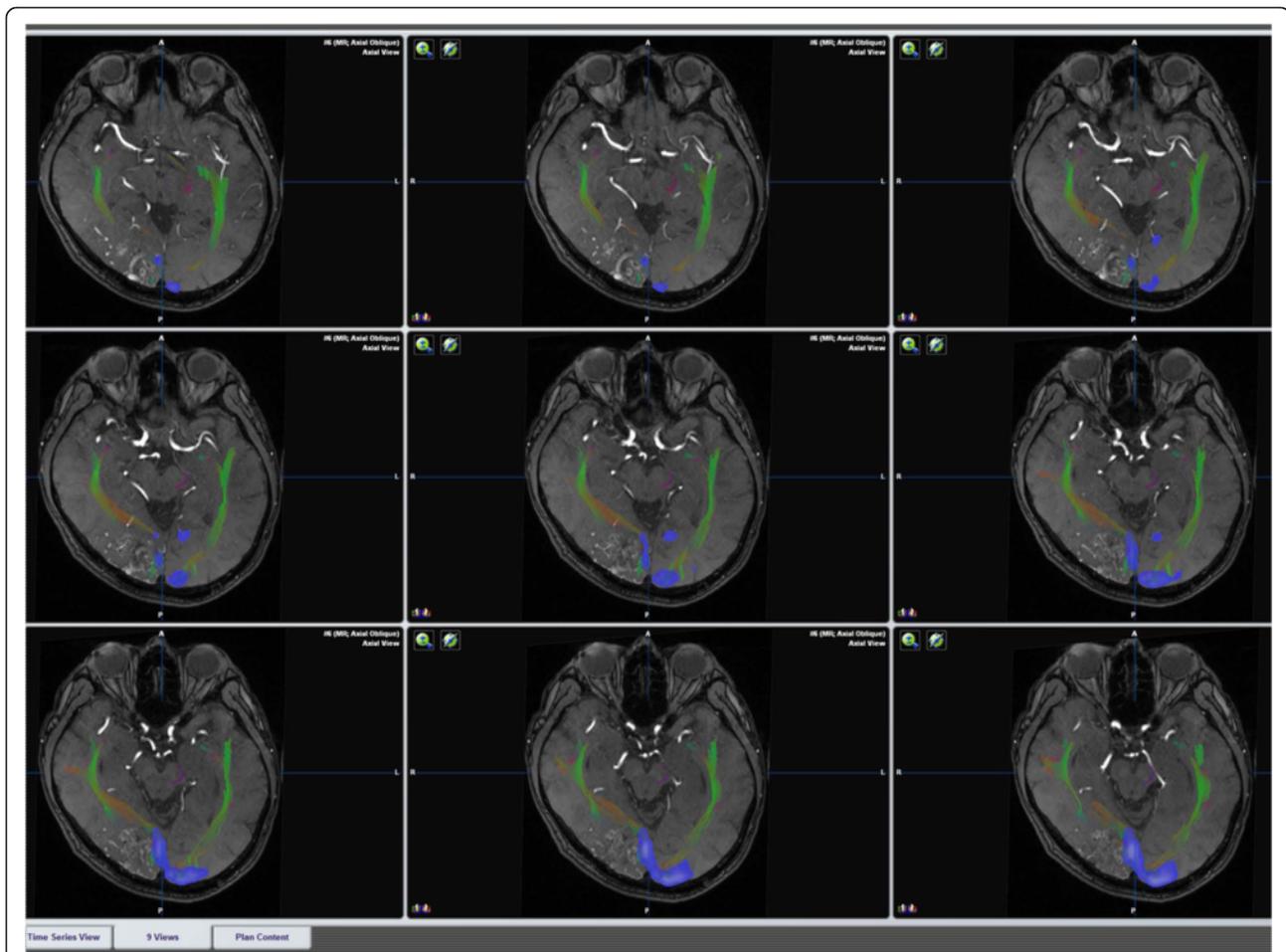


Fig. 2 Case 38. A 36-year-old male with a history of sustained epilepsy. Enhanced structural axial T1-weighted image showing a right occipital AVM. Based on enhanced structural axial T1-weighted image, fiber tracking showing the asymmetry of optic radiation (*green*) and visual cortex (*blue*). Both the AVM-OR and AVM-VC distances were zero

Discussion

Occipital AVMs are still one of neurosurgery's intriguing and challenging pathologies because of their proximity to the visual cortex and optic radiations. The prevalence of VFDs at presentation has been reported by several studies. Pollock et al. reported 7 (21 %) of 34 patients with postgeniculate visual pathway AVMs had VFDs before stereotactic radiotherapy [19]. Anderson et al. [1] reported that 13 (50 %) of 26 patients with occipital AVMs presented with VFDs. Martin and Wilson [18] reported that 9 (56 %) of 16 patients had preexisting VFDs before surgical excision. Bartolomei et al. [2] reported a frequency of preexisting VFDs in 10 (43 %) of 23 patients. Similar prevalence occurred in our series and 14 (33 %) of the 42 patients presented with VFDs.

Risk factors of preexisting VFDs

Studies reported that contralateral homonymous VFDs occurred in 67–90 % of patients with ruptured occipital AVMs [2, 16, 18, 25]. But in a series of 42 patients who

received curative treatment of occipital AVMs, 22 (52.4 %) of the patients presented with VFDs and only 11 (50.0 %) of these patients had a history of hemorrhage [23]. In our series of 42 patients, 15 (35.7 %) had a history of hemorrhage and 12 (80 %) patients had preexisting VFDs. Our results showed that a history of hemorrhage was an independent risk factor of preexisting VFDs in occipital AVMs. Three patients with a history of hemorrhage did not present with VFDs at evaluation. One reason might be that the AVM-OR or the AVM-VC distance was far away enough to avoid VFDs after hemorrhage. The other reason might be that VFDs secondary to hemorrhage had spontaneously recovered to normal VFs over time [16]. For the two patients who presented with VFDs but had nonhemorrhagic chronic headache, the etiology may be associated with ischemia or vascular steal in parenchyma adjacent to the AVM [23].

Studies have shown that the frequency of VFDs varied with the locations of the AVMs. Dehdashti and colleagues found that patients with medial occipital AVMs were more

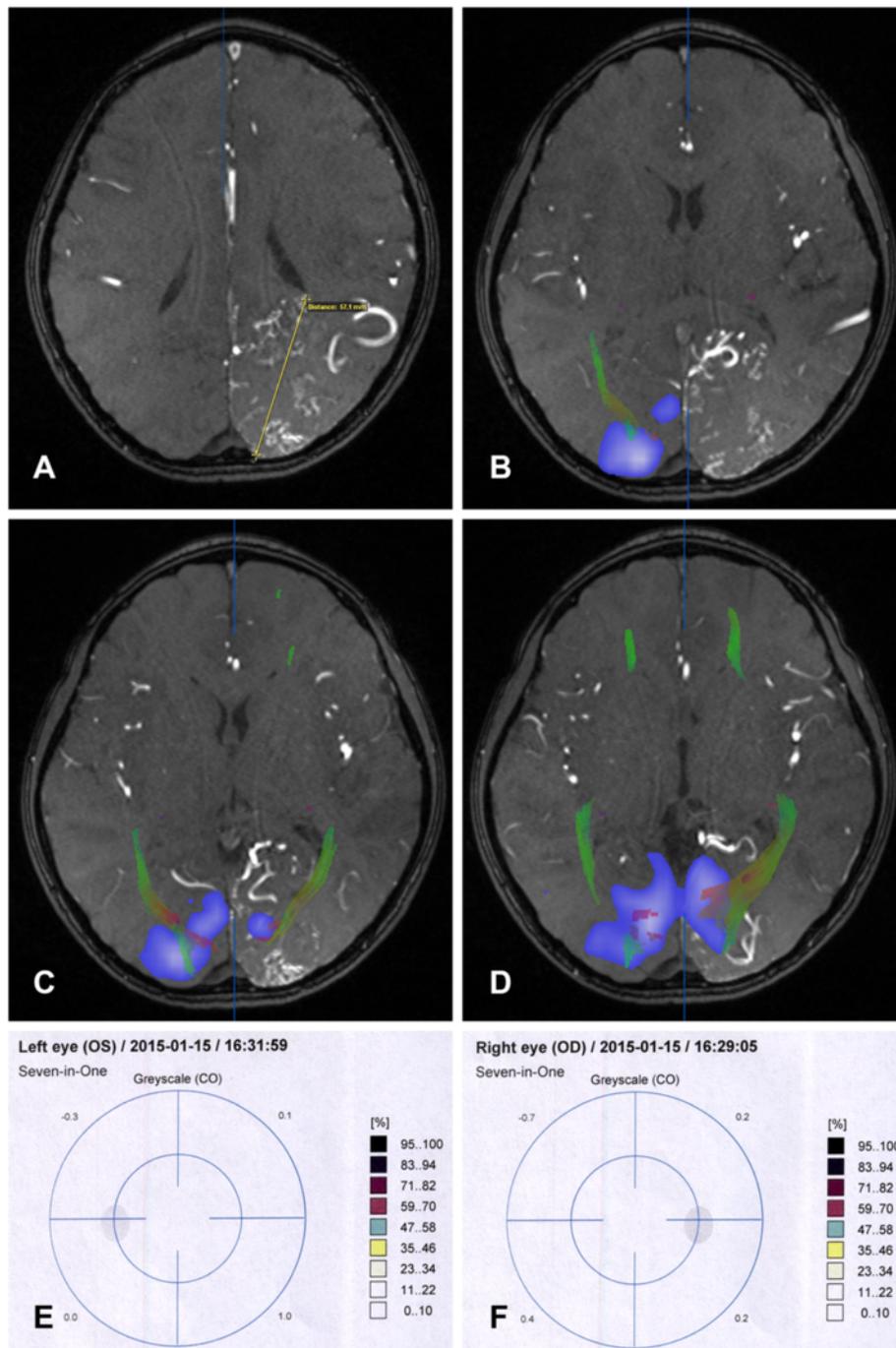


Fig. 3 Case 42. A 10-year-old male without a history of hemorrhage. **a** Enhanced structural axial T1-weighted image showing a left occipital diffuse AVM with a maximum diameter of 57.1 mm (yellow line). **b–d** Based on enhanced structural axial T1-weighted image, fiber tracking showing the asymmetry of optic radiation (green), and visual cortex (blue). Both the AVM-OR and AVM-VC distances were zero. **e–f** Visual fields testing showing normal visual fields

likely to experience a preexisting VFD [5]. Kupersmith et al. [16] reported that VFD occurred in 75 % of the patients with medial occipital AVMs, 50 % of those with lateral and 43.5 % of those with anterior occipital AVMs. But no location was associated with a significantly higher

frequency of visual defects. In our study, we did not classify the location of AVMs as medial, lateral, anterior, or the whole occipital lobe. Instead, we used the DTI tractography and BOLD-MRI to evaluate the relationship between the AVMs and the optic radiation or the visual

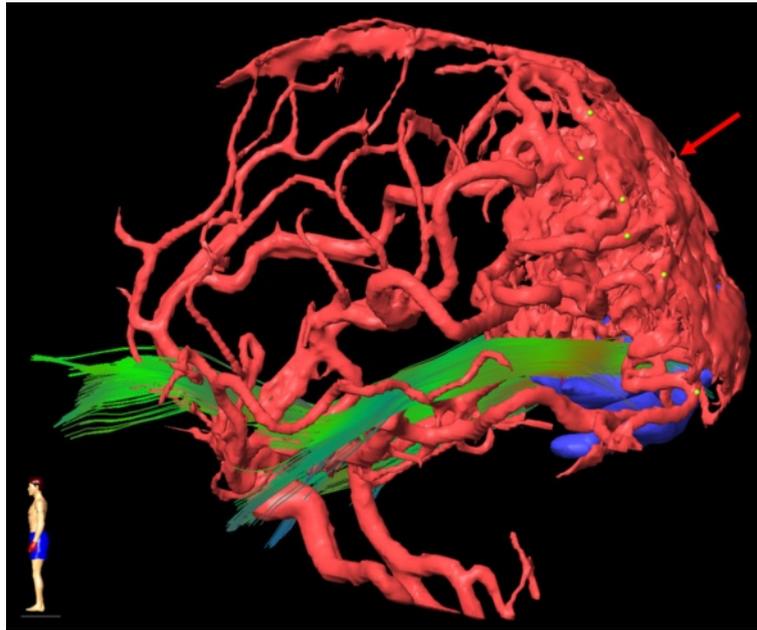


Fig. 4 Case 42. A 3D reconstruction of BOLD-MRI, DTI, and TOF-MRA showing the relationship of the AVM (red arrow), optic radiation (green), and visual cortex (blue)

cortex [24]. Our study was the first to use DTI and BOLD-MRI to evaluate the relationship between preexisting VFDs and occipital AVM locations. With optic fiber tracking, we found that the VFD frequency was not associated with the distance from the AVM to the optic radiation or the visual cortex. Eleven (32.4 %) of the 34 patients with AVM-OR distance <5 mm and 3 (37.5 %) of the 8 patients with that distance \geq 5 mm experienced preexisting VFDs; VFDs were identified in 17.6 % (3/17) of the patients with AVM-VC distance <5 mm and 44 % (11/25) of patients with that distance \geq 5 mm. The differences did not reach statistical significance. This result is not

consistent with the previous studies that predominantly dealt with gliomas involving the optic radiation. Previous study has found that 55.6 % (15/27) of the patients in whom the distance from the lesion to the optic radiation was <5 mm had various degrees of preoperative VFDs, and all nine patients in whom the distance from the lesion to the optic radiation was >5 mm had preoperative normal VFs [24]. In our study with AVMs, the fiber tract adjacent to the lesion can often be displaced. While in the study predominately dealing with gliomas, the gliomas tended to infiltrate or destruct the white matter diffusely. The different results from brain tumors and AVMs are probably

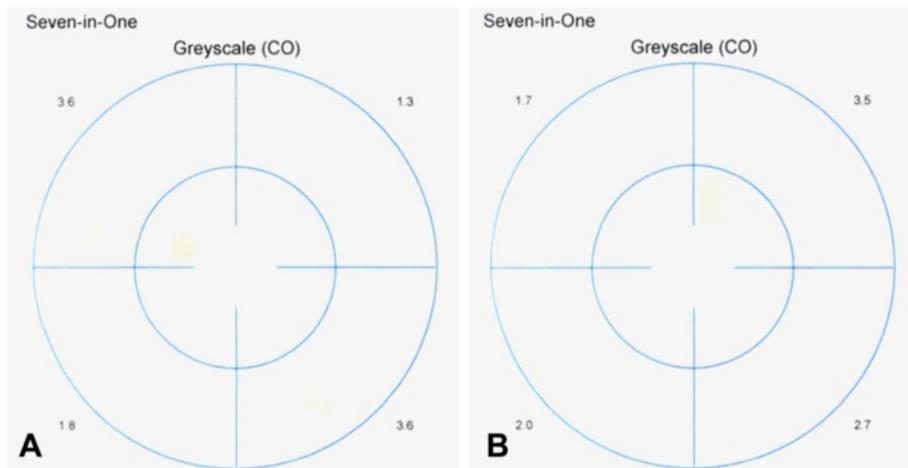


Fig. 5 Case 38. a, b Visual field testing showing normal visual fields

due to the pathologic nature of these two diseases. We would speculate that from early gestation, the trajectories of the optic radiations in the affected hemisphere may deviate from their normal course, bypass the AVMs, and reach their final target in the occipital lobe. A similar process can be observed in the visual system of patients with unilateral peri-ventricular brain damage early in the third trimester of gestation [9]. Our speculation is also consistent with animal studies [13, 20, 21] and postmortem findings in human fetuses [10] which showed that developing geniculostriate axons after mid-gestation “wait” in the subplate for weeks before entering the cortical plate.

Coexistence of occipital AVMs and optic pathway

In our series, 22 patients presented with a zero distance of the AVM-OR or AVM-VC or both. Of these, six patients had a history of hemorrhage and presented with preexisting VFDs. The other 16 patients with unruptured AVMs had normal VFs. We can conclude that if the AVM is intermixed with the optic radiation or the visual cortex or both, a subsequent hemorrhage will definitely cause secondary VFDs. We can also conclude that as a congenital disease, many AVMs might have coexisted with the optic radiation and visual cortex from very early phases of development and the visual pathway may modify continuously by virtue of brain plasticity to maintain normal VFs. This can also be observed from the findings of Guzzeta et al. One patient in their study showed normal VFs despite a large lesion of the left peri-ventricular white matter involving most of the tissue where optic radiations would normally sit [9]. Most occipital AVMs may present with normal VFs unless a hemorrhage occurs. For patients with unruptured occipital AVMs, the probability of VFD that is caused by ischemia or vascular steal is very small. Asymmetry of the activated cortex and optic radiation fibers occurred in some of our patients, especially in those with zero AVM-OR or AVM-VC distance, but that did not necessarily represent preexisting VFDs. In those with both unruptured AVMs and normal VFs, the asymmetry may also elucidate the plasticity of visual pathway. There might be some type of cortical or subcortical reorganization and development of new connections to other areas of the brain. In all, the brain plasticity obeys the brain capacity to diminish the effects of the neuronal damage at an early age, being of genetic origin or produced by an injury [1, 3, 6–8, 12, 14].

Limitations of our study

We have evaluated 42 cases of occipital AVMs with the focus on preexisting VFDs. We did not describe in detail other symptoms associated with occipital AVMs. There are some consistency and discrepancies between our results and previous findings. The reason may be that the

sample is relatively smaller. In the future, we will recruit more patients with occipital AVMs to evaluate the underlying mechanism of preexisting VFDs.

Conclusions

Occipital AVMs with a history of hemorrhage present with a much higher frequency of VFDs than unruptured ones. Most unruptured occipital AVMs may present with chronic headache and seizures other than VFDs. Preexisting VFDs are not associated with the distances from the AVMs to the optic radiation or the visual cortex.

Abbreviations

AVM: arteriovenous malformation; AVM-OR: distance from AVM to the optic radiation; AVMs: arteriovenous malformations; AVM-VC: distance from AVM to the visual cortex; BOLD-fMRI: blood oxygen level dependent functional magnetic resonance imaging; DTI: diffusion tensor imaging; fMRI: functional magnetic resonance imaging; MRA: magnetic resonance angiography; MRI: magnetic resonance imaging; TOF-MRA: time of flight magnetic resonance angiography; VFDs: visual field deficits; VFs: Visual fields; VOI: volume of interest.

Competing interests

All authors certify that we have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or nonfinancial interest (such as personal or professional relationships, affiliations, knowledge, or beliefs) in the subject matter or materials discussed in this manuscript.

Authors' contributions

Author contributions to the study and manuscript preparation include the following. XT, JW, FL, and ZJ carried out the fMRI studies, participated in the fMRI data acquisition, and drafted the manuscript. XT, JW, FL, and YC participated in the design of the study and performed the statistical analysis. SW, YC, and YZ conceived of the study, participated in its design and coordination, and helped to draft the manuscript. All authors read and approved the final manuscript.

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References

- Anderson V, Spencer-Smith M, Leventer R, Coleman L, Anderson P, Williams J, et al. Childhood brain insult: can age at insult help us predict outcome? *Brain*. 2009;132:45–56.
- Bartolomei J, Wecht DA, Chaloupka J, Fayad P, Awad IA. Occipital lobe vascular malformations: prevalence of visual field deficits and prognosis after therapeutic intervention. *Neurosurg*. 1998;43:415–21.

3. Berker EA, Berker AH, Smith A. Translation of Broca's 1865 report. Localization of speech in the third left frontal convolution. *Arch Neurol*. 1986;43:1065–72.
4. Bruyn GW. Intracranial arteriovenous malformation and migraine. *Cephalalgia*. 1984;4:191–207.
5. Dehdashti AR, Thines L, Willinsky RA, terBrugge KG, Schwartz ML, Tymianski M, et al. Multidisciplinary care of occipital arteriovenous malformations: effect on nonhemorrhagic headache, vision, and outcome in a series of 135 patients. *J Neurosurg*. 2010;113:742–8.
6. Gall C, Prilloff S, Sabel BA. Recovery of function and plasticity after lesions of the central visual pathway. *J Neurol Neurosurg Ps*. 2010;11:18–30.
7. Gallegos-Duarte M, Moguel-Ancheita S, Mendiola-Santibañez JD, Morales-Tlalpan V, Saldaña C. Plasticity of the visual pathway and neuroimaging. In: Eröndü OF, editor. *Medical Imaging in Clinical Practice*. Rijeka, Croatia - EUROPEAN UNION: InTech; 2013. p 309–326.
8. Guzzetta A. Plasticity of the visual system after congenital brain damage: a few weeks can matter. *Dev Med Child Neurol*. 2010;52:699.
9. Guzzetta A, D'Acunto G, Rose S, Tinelli F, Boyd R, Cioni G. Plasticity of the visual system after early brain damage. *Dev Med Child Neurol*. 2010;52(14):891–900.
10. Hevner RF. Development of connections in the human visual system during fetal mid-gestation: a Dil-tracing study. *J Neuropathol Exp Neurol*. 2000;59:385–92.
11. Joint Writing Group of the Technology Assessment Committee American Society of Interventional and Therapeutic Neuroradiology, Joint Section on Cerebrovascular Neurosurgery a Section of the American Association of Neurological Surgeons and Congress of Neurological Surgeons, Section of Stroke and the Section of Interventional Neurology of the American Academy of Neurology, Atkinson RP, Awad IA, Batjer HH, Dowd CF, Furlan A, et al. Reporting terminology for brain arteriovenous malformation clinical and radiographic features for use in clinical trials. *Stroke*. 2001;32:1430–42.
12. Kennard M, Fulton JF. Age and reorganization of central nervous system. *Mt Sinai J Med*. 1942;9:594–606.
13. Kostovic I, Rakic P. Developmental history of the transient subplate zone in the visual and somatosensory cortex of the macaque monkey and human brain. *J Comp Neurol*. 1990;297:441–70.
14. Krageloh-Mann I, Horber V. The role of magnetic resonance imaging in elucidating the pathogenesis of cerebral palsy: a systematic review. *Dev Med Child Neurol*. 2007;49:144–51.
15. Kupersmith MJ. *Neurovascular neuro-ophthalmology*. Heidelberg: Springer; 1993. p. 307–24. pp 443–444.
16. Kupersmith MJ, Vargas ME, Yashar A, Madrid M, Nelson K, Seton A, et al. Occipital arteriovenous malformations: visual disturbances and presentation. *Neurology*. 1996;46:953–7.
17. Lawton MT, Kim H, McCulloch CE, Mikhak B, Young WL. A supplementary grading scale for selecting patients with brain arteriovenous malformations for surgery. *Neurosurg*. 2010;66:702–13.
18. Martin NA, Wilson CB. Medial occipital arteriovenous malformations: surgical treatment. *J Neurosurg*. 1982;56:798–802.
19. Pollock BE, Lunsford LD, Kondziolka D, Bissonette DJ, Flickinger JC. Stereotactic radiosurgery for postgeniculate visual pathway arteriovenous malformation. *J Neurosurg*. 1996;84:437–41.
20. Rakic P. Prenatal genesis of connections subserving ocular dominance in the rhesus monkey. *Nature*. 1976;261:467–71.
21. Rakic P. Prenatal development of the visual system in rhesus monkey. *Philos Trans R Soc Lond B Biol Sci*. 1977;278:245–60.
22. Schlosser MJ, McCarthy G, Fulbright RK, Gore JC, Awad IA. Cerebral vascular malformations adjacent to sensorimotor and visual cortex: functional magnetic resonance imaging studies before and after therapeutic intervention. *Stroke*. 1997;28:1130–7.
23. Sinclair J, Marks MP, Levy RP, Adler JR, Chang SD, Lopez JR, et al. Visual field preservation after curative multi-modality treatment of occipital lobe arteriovenous malformations. *Neurosurg*. 2005;57:655–67.
24. Sun GC, Chen XL, Zhao Y, Wang F, Hou BK, Wang YB, et al. Intraoperative high-field magnetic resonance imaging combined with fiber tract neuronavigation-guided resection of cerebral lesions involving optic radiation. *Neurosurg*. 2011;69:1070–84.
25. Troost BT, Newton TH. Occipital lobe arteriovenous malformations: clinical and radiologic features in 26 cases with comments on differentiation from migraine. *Arch Ophthalmol*. 1975;93:250–6.

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