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Case Report

# The rare case of optic nerve cavernoma: A case report depicting the diagnostic challenge ☆

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ABSTRACT

The manuscript describes a case of Cavernous Malformation in the optic pathway which is extremely rare, accounting for less than 1% of central nervous system cavernomas. This case report highlights a patient initially diagnosed with a glioma, but subsequent MRI changes and extensive analysis ruled in favor of a hemorrhagic optic neuropathy caused by an optic nerve cavernoma. The patient experienced temporary vision loss but fully regained her vision within a week. Based on clinical, biochemical, and radiological findings, it was confirmed as a rare case of optic nerve cavernoma, and the patient was managed expectantly due to her complete recovery of vision. Follow-up imaging after 1 year indicated a stable lesion with evolving characteristics consistent with a cavernoma. This study provides an informative review of the condition and highlights the key radiologic features of this disease.



## Introduction

Cerebral cavernous malformations (CMs) are vascular lesions that contain a compact bundle of pathological capillary vessels without brain parenchyma between the vessels [1]. CMs are relatively common and affect approximately 1 in 200 individuals, accounting for 8%-15% of all intracranial vascular malformations, and optic pathway CMs represent less than 1% of these lesions [1,2].

For prechiasmatic and or chiasmal CMs, the clinical manifestation could be acute (with chiasmal apoplexy), subacute, or progressive loss of vision. Subacute and progressive vision loss patterns are observed in patients with suprasellar tumors and optic nerve gliomas [3,4]. Whereas, CMs of the optic nerve (ON) and chiasm usually present with chiasmal apoplexy that is, sudden onset blurred vision, headache, nausea, and retro-orbital pain. Clinically and radiologically, acute presentation of ON CMs may also mimic hemorrhagic ON neuropathy of infective, inflammatory, or autoimmune etiology [5]. However, when discovered incidentally, ON CMs may mimic other focal mass lesions, especially ON gliomas [4,6]. Thus, it is important to consider ON CMs in the differential diagnosis of these optic pathway mass lesions, despite their rarity.

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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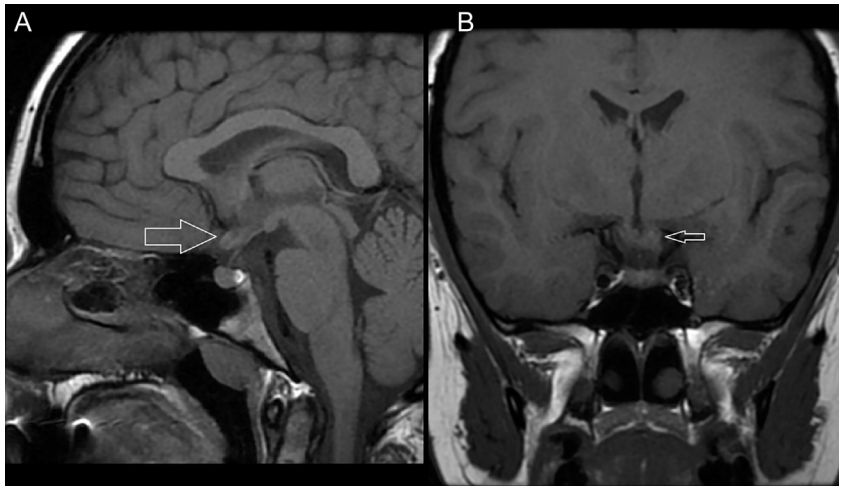


**Fig. 1 – T1WI sagittal view (A – left) and corresponding coronal view (B – right) demonstrating chiasmal thickening.**

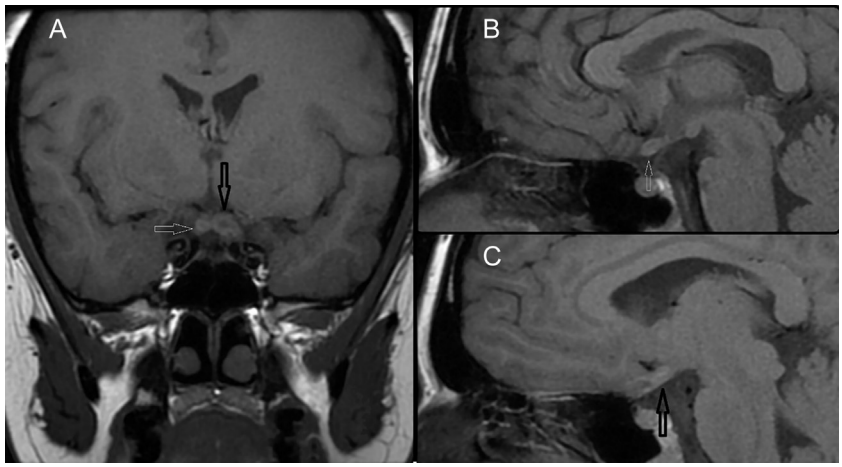


**Fig. 2 – T1WI at the level of prechiasmatic optic nerves. (A – left ) visualizes the thickened left prechiasmatic optic nerve (black arrow) in comparison to right prechiasmatic optic nerve (white arrow) in coronal plane. (B – bottom right) demonstrates this thickening in sagittal plane when compared to the normal contralateral prechiasmatic optic nerve (C – top right).**

## Case report

A 21-year-old female presented with acute headache and visual field defect. Although, she had recurring episodes of headache and was diagnosed with migraine previously, this was the first time she noticed visual changes. She denied any other past medical or surgical history, specifically personal or family history of Neurofibromatosis. Patient was otherwise a fit and well individual working in retail and completing part-time studies.

An ophthalmological assessment of her presentation revealed a junctional scotoma on visual field testing and the patient was urgently referred for an MRI to rule out pituitary apoplexy and to a Neurosurgeon for an opinion.

Imaging was obtained on a 3T MRI. Multiplanar and multisequence MRI of the brain and orbits was performed. Following sequences and thickness slices were utilized:

- 2 mm T1 fat-saturated turbo spin echo, with and without contrast
- 2 mm T2 fat-saturated turbo spin echo
- 1.5 mm T2 SPACE FLAIR
- 0.5 mm T2 3D fast spin echo (SPACE and CUBE)
- 1.5 mm T1 fat-saturated turbo spin echo with contrast in sagittal oblique plane

MRI revealed isointense thickening of the optic chiasm on T1WI with a normal pituitary gland (Fig. 1). This thickening extended into the left prechiasmatic optic nerve (Fig. 2). This thickening also exhibited mild heterogenous contrast enhancement on T1WI with contrast (T1+C) (Figs. 3 and 4). T2WI showed central hyperintensity and no hemosiderin deposition to suggest prior bleed (Fig. 5). FLAIR imaging showed a very subtle increase in signal intensity of this lesion (Fig. 6). There were no other cavernomas identified within the brain. She was initially diagnosed with ON glioma and subsequently

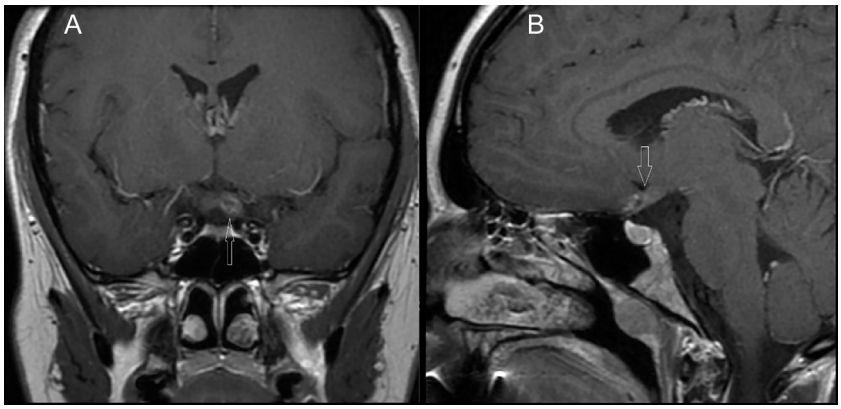


**Fig. 3 – Coronal (A – left) and corresponding sagittal (B – right) views of T1+C imaging demonstrating heterogenous contrast enhancement of the thickened left prechiasmatic optic nerve.**

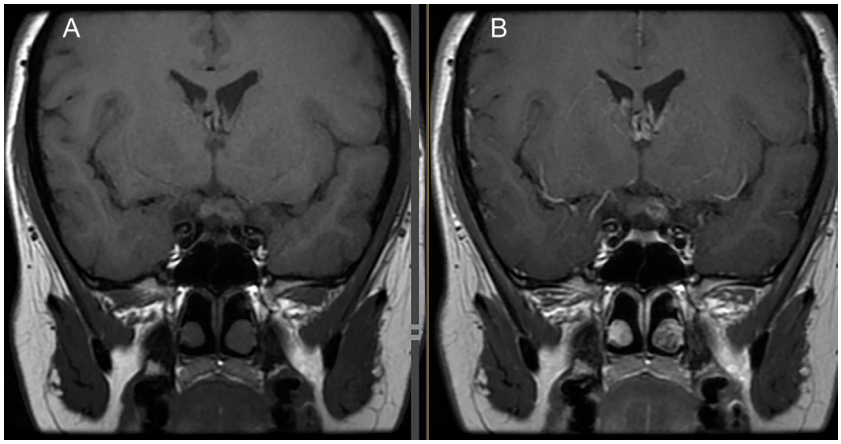


**Fig. 4 – T1+C (B - right) when compared to T1 (A - left) shows contrast enhancement of the left prechiasmatic optic nerve.**

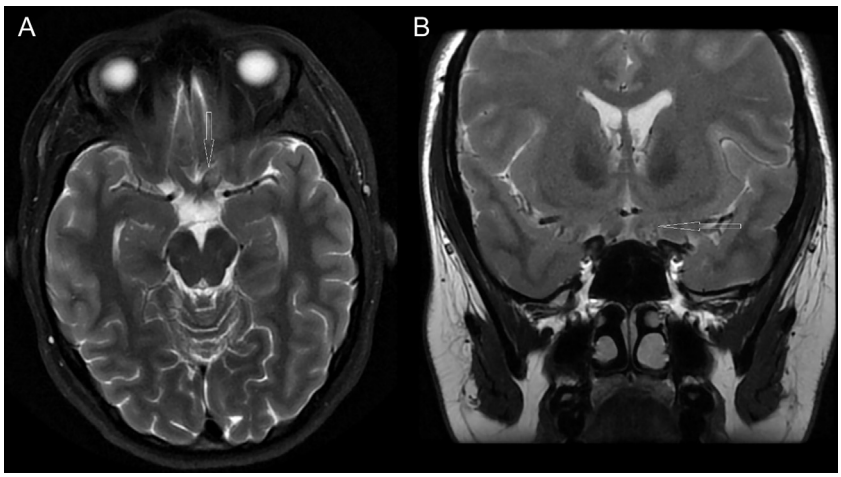


**Fig. 5 – T2WI axial view (A – left) and coronal view (B – right) showing expanded prechiasmatic optic nerve with central hyperintensity and no hemosiderin deposit.**

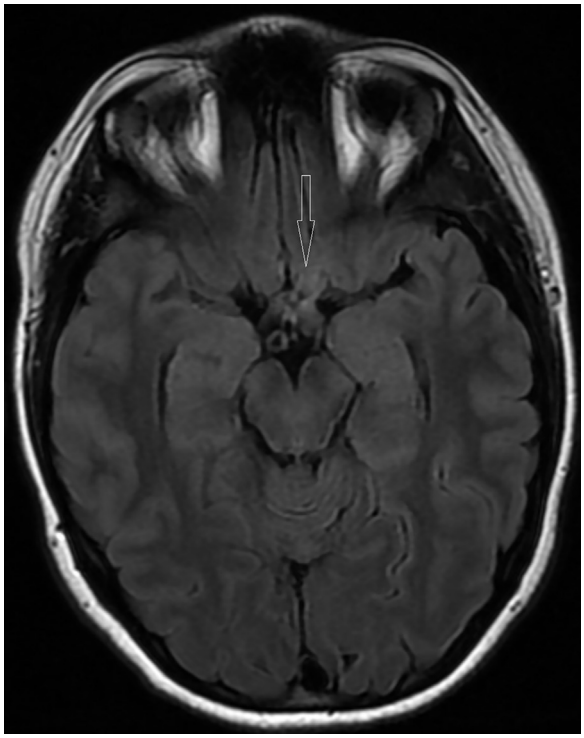

**Fig. 6 – FLAIR sequence axial view showing subtle high signal intensity of the thickened prechiasmatic optic nerve lesion.**

proceeded with an MRI of the whole neural axis to look for neurofibromas given the strong association of optic nerve glioma and NF-1. No neurofibromas were identified during this screening. Her vision improved over the next 48 hours and recovered completely within a week of onset. The plan was for expectant management and repeat imaging in 2 months' time to establish whether this was a benign or an aggressive lesion.

A month later the patient had another episode of headache and vision changes but of increased severity. A repeat MRI demonstrated a hemorrhagic lesion involving the optic chiasm as well as the prechiasmatic and ipsilateral postchiasmatic segments. T1WI hyperintensity and blooming on SWI imaging favored an acute hemorrhagic event (Fig. 7). Upon comparing T1 with and without contrast, ongoing contrast enhancement was visualized in the prechiasmatic optic nerve with acute hemorrhage extending posteriorly into the optic chiasm and along the visual pathway (Fig. 8). There was minimal extension of bleed or blood products across the chiasmal midline. T2WI elicited expansion of the prechiasmatic optic nerve and postchiasmatic segment in the context of acute blood product (Figs. 9A–C). These optic pathway changes also showed a new high signal on FLAIR sequence (Fig. 10).

Given the rarity of ON CMs, it was prudent to rule out other causes of hemorrhagic optic neuropathy. The patient was tested for anti-MOG, anti-NMO, viral (Zoster, Varicella, Adeno) neuropathies, SLE, and neurosarcoidosis. All tests were negative. Her blood and CSF biochemistry (immunophenotyping FLOW cytometry, oligoclonal bands, anti-Aquaporin 4 antibodies, ACE, viral PCRs, protein, and glucose) were all unremarkable.

The case was discussed in a Neuroradiology multidisciplinary team meeting which included multiple Neuroradiologists and Neurosurgeons. She was then given the diagnosis of ON CM as a diagnosis of exclusion.

Her symptoms yet again improved over the next 48 hours and she made an almost full recovery within 1 week. Hence, it was deemed highly risky to pursue a histopathological diagnosis. After discussion with the patient and uncertainty around the diagnosis, the team reached the conclusion of expectant management for this patient's particular case. Plan was to continue with 6 monthly clinical follow-ups and yearly radiological surveillance with consideration of surgical resection if cavernoma increased in size and spread towards the chiasm or caused significant vision loss including any visual field compromise.

Patient's vision remained stable during clinical follow-ups. Annual imaging surveillance was performed showing radiological stability of the lesion. High resolution CUBE sequence was utilized which showed a heterogenous "berry-like" appearance of this lesion with possible tiny fluid-fluid levels indicating prior hemorrhage (Fig. 11). T1WI hyperintensity in the previous imaging had now almost completely resolved and the degree of nerve expansion had also reduced (Fig. 12). There was still residual minor contrast enhancement but much decreased compared to previous (Figs. 13–15). These findings were best in keeping with a low-flow vascular lesion such as a cavernoma that had previously hemorrhaged but now remained stable.

## Discussion

### *Cavernous malformation*

CMs are well-circumscribed, benign vascular hamartomas consisting of irregular thick, and thin-walled sinusoidal vascular channels. These are located within the brain but lack intervening neural parenchyma, large feeding arteries, or large draining veins [7]. These are low-flow vascular malformations and are angiographically occult and account for up to 15% of all intracranial vascular malformations [7,8]. Histologically, the lesions consist of a single layer of endothelial cells and lack structural elements found in mature vasculature, including smooth muscle and elastin [6]. Macroscopically, the lesions appear reddish purple. They are often multilobulated and may be encapsulated by variable layers of fibrous adventitia, giving them their characteristic mulberry-like appearance. They may hemorrhage, calcify, or thrombose [7].

CMs can be either familial or sporadic [9]. The familial form usually manifests with multiple lesions in the setting of a family history of neurological disease. In the sporadic form, patients rarely have more than two lesions, and family history is typically absent. Mutations in three genes: CCM1, CCM2, and CCM3 have been associated with familial disease, accounting for 96% of all mutations [1,9].

Being angiographically occult, MRI is the modality of choice to demonstrate the characteristic "popcorn" or "berry"

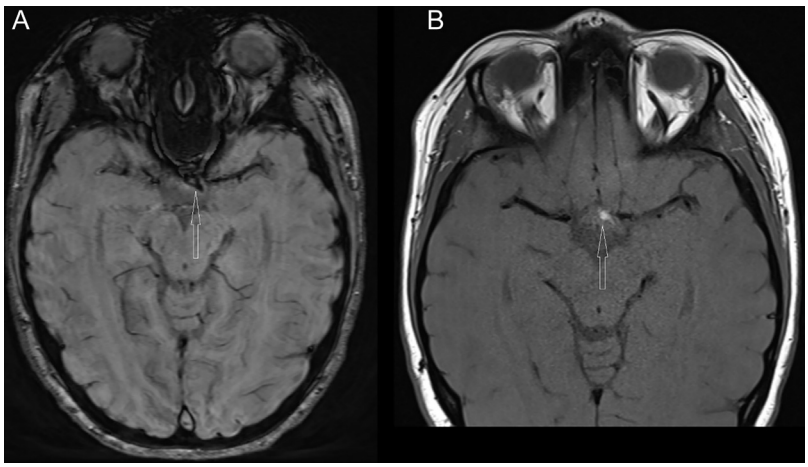


**Fig. 7 – SWI (A – left) demonstrating blooming artefact which corresponded to focal hyperintensity at the region of interest on T1WI (B – right). This was a result of chiasmal apoplexy from the cavernoma and represented acute hemorrhage.**

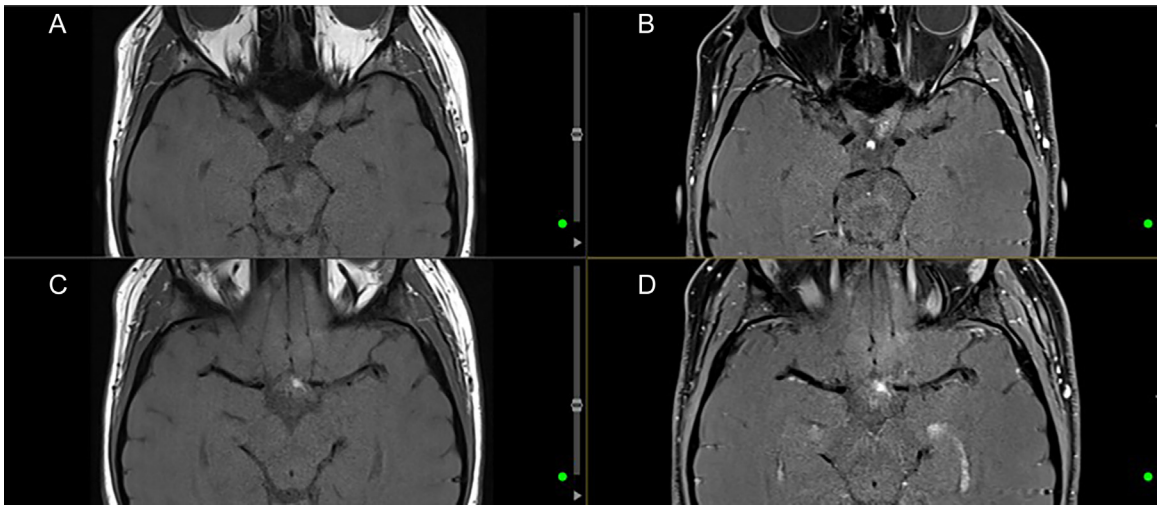


**Fig. 8 – T1 (A - top left, C - bottom left) and T1+C (B - top right, D - bottom right) comparisons to delineate contrast enhancement and hemorrhage.**

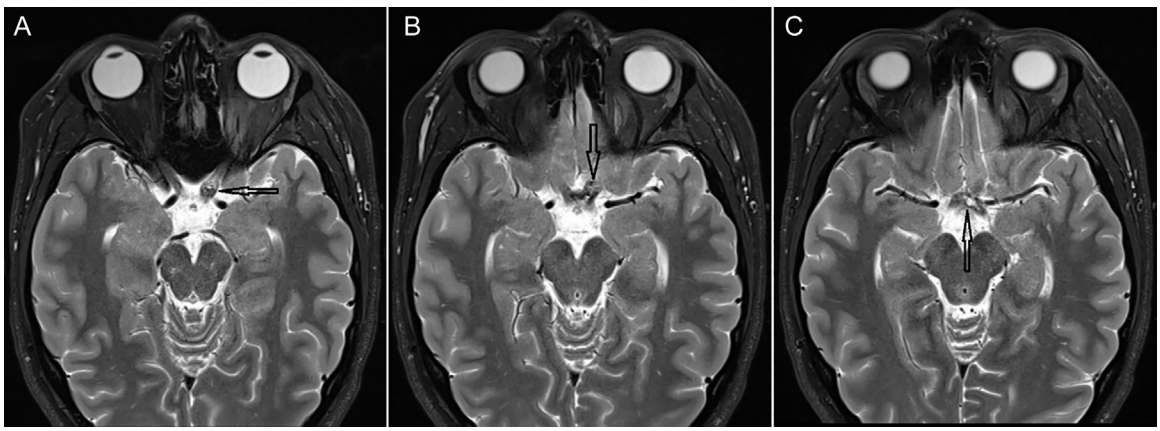


**Fig. 9 – (A–C, left to right): Axial views of T2WI demonstrating expansion of the prechiasmatic optic nerve (A), chiasm (B) and postchiasmatic segment (C) in the context of acute blood product.**

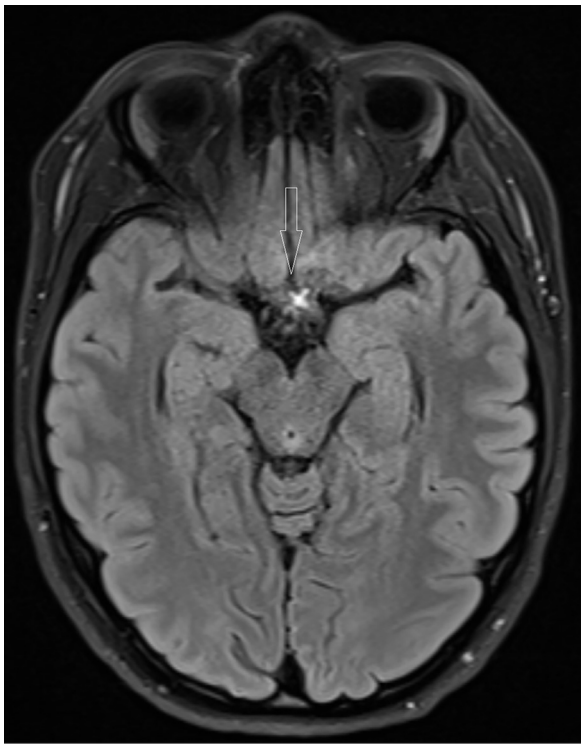

**Fig. 10 – Axial FLAIR image at the level of chiasm demonstrating new hyperintense signal due to acute hemorrhagic event.**

appearance of CM. T2 sequence is quite effective in demonstrating hypointense hemosiderin rim, varying signal intensity of the age-dependent blood product, and fluid-fluid levels. CMs demonstrate prominent blooming on SWI. In the event of an active hemorrhage, T1 hyperintensity can be seen with possible surrounding edema on FLAIR sequence. CMs generally do not enhance contrast imaging [9].

The distribution of CMs pertains to following the volume of neural axis, such that 80% are supratentorial, 15% are infratentorial, and 3%-5% in spinal cord [5]. Cranial nerve CMs constitutes less than 1%, with optic chiasm being the most frequently affected and prechiasmatic optic nerve involvement being an extremely rare entity [2,5,10].

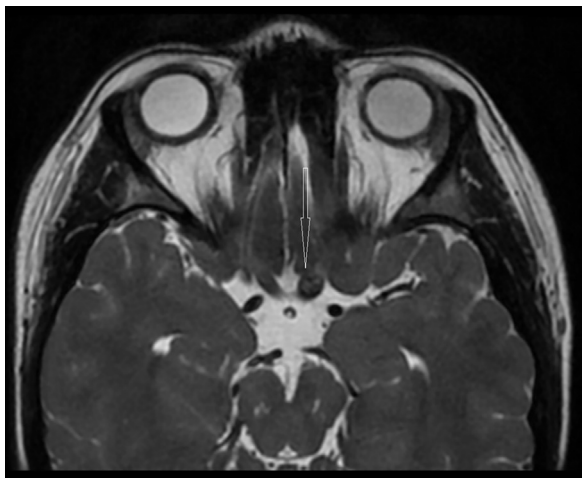

**Fig. 11 – High resolution CUBE sequence demonstrating heterogenous "berry-like" appearance of this lesion.**

The most common clinical manifestation of prechiasmatic ON lesion is loss of vision (98%), followed by headache and or retro-orbital pain (60%). Vision loss can be acute (58%), subacute (15%), or progressive (26%) [11]. The onset and progression of vision loss may help delineate CMs from gliomas or other optic neuropathies. CMs usually become symptomatic acutely when they hemorrhage, whereas, optic gliomas often present with slow unilateral loss of vision. Nevertheless, malignant gliomas of the ON may also present with sudden loss of vision in 70%-84% of this cohort [4]

### *Optic nerve glioma*

Optic pathway gliomas are usually seen in the setting of NF-1 and majority are histologically consistent with pilocytic astrocytomas [12]. Most widely accepted classification of optic pathway gliomas is the Dodge classification which divides these tumors into 3 groups based on anatomy: [13].

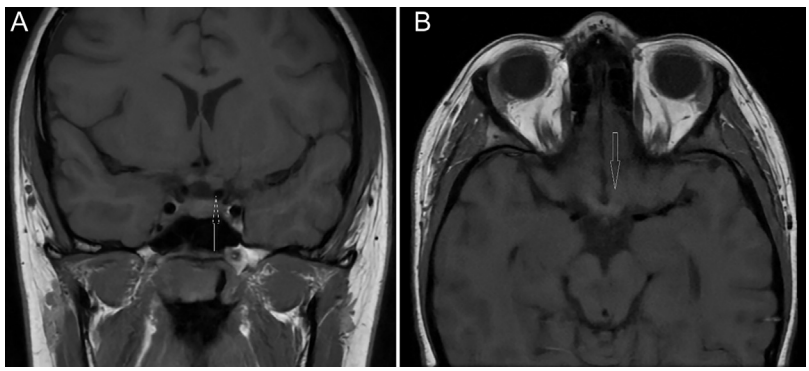


**Fig. 12 – Coronal (A – left) and axial (B – right) views of T1WI sequence one year post hemorrhage demonstrating complete resolution of blood products and decreased expansion of the left prechiasmatic optic nerve.**

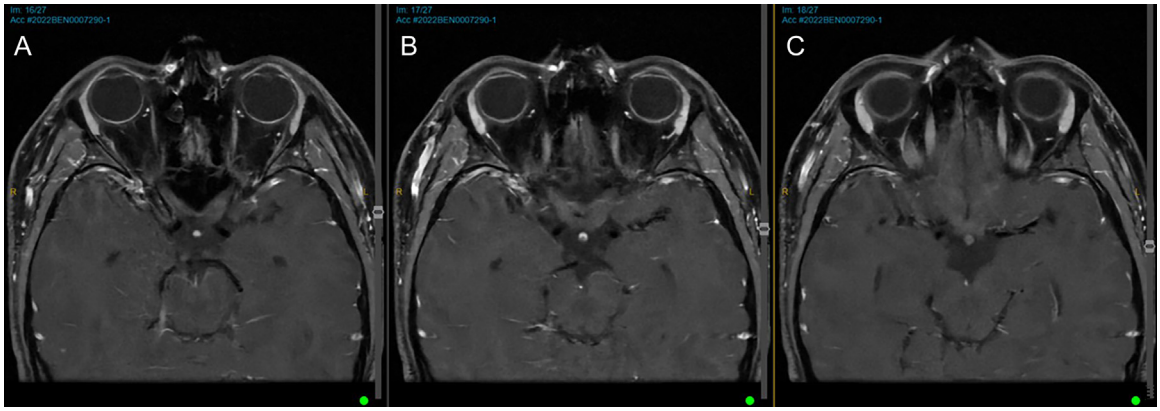


**Fig. 13 – One-year posthemorrhage T1+C sequence demonstrating minimal to no contrast enhancement along the left prechiasmatic optic nerve towards the chiasm (A-C, left to right).**

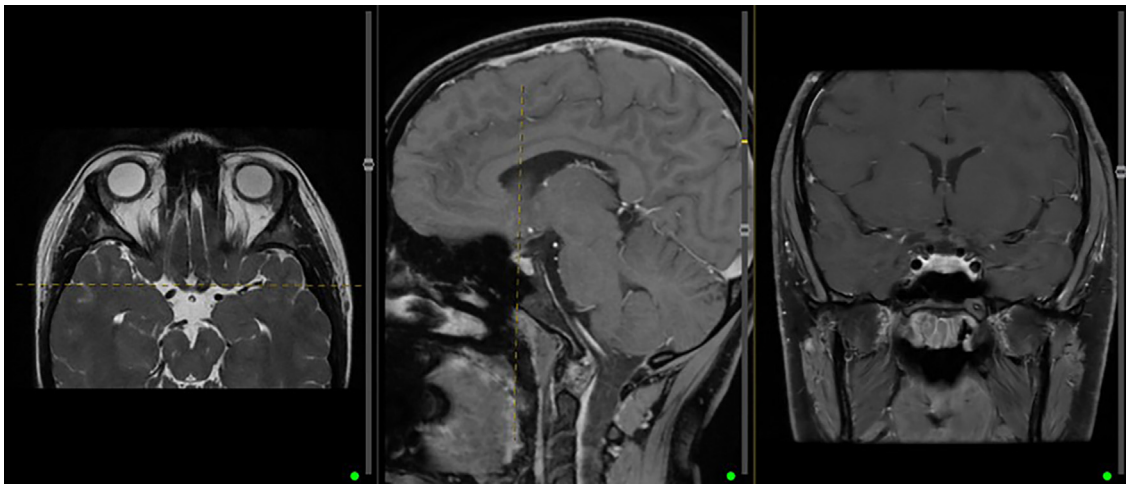

**Fig. 14 – One-year posthemorrhage – minimal to no contrast enhancement demonstrated on anatomically aligned different corresponding views at the level of the lesion (line of reference [left]).**

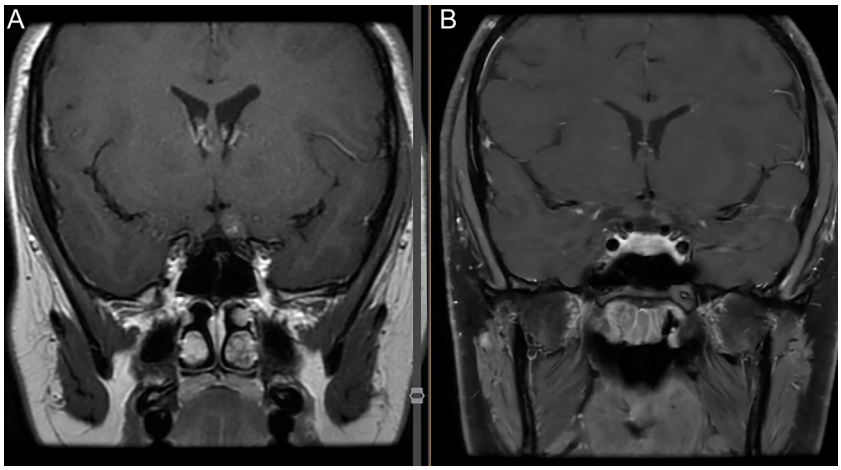


**Fig. 15 – Coronal T1+C sequence 1 year posthemorrhage (B - right) of the intracranial optic nerve cavernoma demonstrating decreased contrast enhancement in comparison to prehemorrhage image (A - left).**

- Stage 1: optic nerves only
- Stage 2: chiasmal involvement (with or without optic nerve involvement)
- Stage 3: hypothalamic involvement and/or other adjacent structures

In cases involving the optic nerve in isolation that is, Dodge stage 1, these are referred to as ON glioma. These exhibit varying degrees of changes and enhancement patterns, hence, the appearance may be smooth, fusiform, eccentric or lobulated. These tumors can be quiescent with minimal progression over years or demonstrate more aggressive features with extension along the optic pathways [12].

In ON gliomas, MRI shows tubular or fusiform enlargement/thickening of the ON with possible kinking or buckling [14]. Larger lesions typically have both solid and cystic components. ON gliomas are usually isointense to cortex and hypointense to white matter and orbital fat on T1; they are isointense to hyperintense relative to white matter and the cortex on T2 [14]. ON gliomas may only minimally enhance as they are low grade astrocytoma, however, in the presence of intense enhancement or other malignant features may represent an aggressive form of the lesion [15].

### *Optic nerve sheath meningioma*

Another strong diagnostic consideration for focal mass lesions of the ON is Optic Nerve Sheath Meningioma (ONSM). ONSMs originate from the arachnoid cap cells of the optic nerve sheath and are intradural tumors. They are typically smooth with lobulated contour and circumferentially encase the optic nerve. They can gradually compress the optic nerve and may exhibit anterior or posterior extension over time [14].

The diagnosis of ONSM can be confirmed with MRI on gadolinium contrast-enhanced fat-suppressed sequences. They appear isointense to grey matter on both T1 and T2 imaging. They show vivid homogenous contrast enhancement which appears as “tram-track” sign on axial images due to the inner nonenhancing optic nerve. In coronal images, this is usually visualized as a “doughnut” or “non-enhancing dot” sign [16,17]. On imaging, ONSMs can be visualized as tubular (62%), globular (23%), fusiform (11%), or focal enlargement of the optic nerve (4%). ONSMs can calcify but rarely hemorrhage or undergo malignant transformation [16,17].

### *Diagnostic considerations*

Focal mass lesions of the ON are a radiological diagnostic challenge. Radiographically CMs are best detected on MRI and are characterized by mixed-signal intensities on T1WI and T2WI with heterogenous features. Interestingly, these features are also shared amongst ONSMs and ON gliomas. Despite significant similarities there are a few distinguishing radiological diagnostic considerations which may help delineate these lesions.

On T2WI, a hypointense peripheral ring which represents hemosiderin deposits (hemoglobin degradation products) is usually present in 60% of ON CMs [11]. This is rather unique to CMs as ON glioma and ONSM are highly unlikely to hemorrhage. ON glioma does not calcify or hemorrhage, however, there have been case studies and reports of both such rare phenomena [15,18]. Hence, despite intralesional hemorrhage on MRI favoring CM, the diagnosis of ON glioma can not entirely be eliminated. Another important consideration is intralesional calcification, these are usually depicted on SWI as blooming artifact and should be analyzed in tandem with T1WI and T2WI to ensure the blooming artifact does not represent a form of hemorrhage. If calcification is present, ONSM should be the primary suspicion [16,17]. In that regard, even a CT scan can be utilized in order to check for the presence of any calcification.

On gadolinium contrast-enhanced MR imaging, CMs show minimal (usually heterogenous pattern if any) or no enhancement in comparison to the significant avid enhancement of ON gliomas and ONSMs. This may aid in reaching a likely diagnosis of CM safely but the diagnostic challenge remains if the focal mass lesion enhances. Despite strong overlapping imaging characteristics of ONSMs and ON gliomas, there are a few radiological diagnostic clues that could help differentiate them. ONSM usually shows homogenous contrast enhancement which may be similar for some cases of ON glioma, however, an ON glioma will not have the classic “tram-track” sign which corresponds to the enhancing outer ON sheath tumor encircling the inner non-enhancing ON [16,17].

In very small cavernomas, as depicted in this case report, hemorrhagic optic neuritis was also considered. The thickening of prechiasmatic ON, chiasm, and optic tract can be present with neoplasms and inflammation [11,17]. In this particular case, slight FLAIR hyperintensity (perilesional edema) in the absence of contrast enhancement of the thickened ON favored some form of underlying neuritis rather than malignant ON glioma or ONSM [19]. Infective, inflammatory and autoimmune demyelinating causes were ruled out clinically and biochemically. For this particular case, these included Varicella Zoster, HSV-1, HSV-2, CMV, Enterovirus, Multiple Sclerosis, Neuro-myelitis Optica, Lymphoma and Neurosarcoidosis. Given such overlapping characteristics on MRI, it is important that clinical and biochemical surrogates are used to delineate certain diagnoses, however, it is of utmost importance to not dismiss the possibility of a rare lesion such as CM of the ON.

### *Treatment options*

There are 4 recognized options for managing CMs: expectant management, medical management, surgical resection and stereotactic radiosurgery [1]. Expectant management consists of regular interval radiological follow-up of lesions. The interval period may start from 2 months and increase over time to as long as 2 years, given the lesion establishes its stability. MRI remains the best modality for visualizing CMs for ongoing surveillance. CMs do not originate from or invade neural parenchymal tissue, however, may slowly grow to cause mass effect on the surrounding tissue [1]. If the patient is symptomatic and depicts radiological lesion expansion, mass effect or hemorrhage, surgical intervention might be necessary.

Previous retrospective studies and multiple case series’ have reported high rates of visual preservation and improvement postcomplete microsurgical resection of these lesions [4,20–22]. Many surgeons favor complete resection due to intraoperative findings of a well circumscribed mass that does not intervene with the white matter tract of the ON [23]. Complete resection is recommended because any resid-

ual carries significant likelihood for recurrence and rehemorrhage [22–24]. This renders the CMs in eloquent brain regions (optic nerve included) very high risk [1,18,22]. Although there is sufficient evidence in favor of surgical resection, the timing of the surgery is controversial. Some authors have illustrated that even though favorable surgical outcomes are as high as 70% in restoring normal vision, the optimal results were mostly associated with patients who presented with milder symptoms and without longstanding damage to optic apparatus [21]. Furthermore, reoperations have been regarded as dangerous and highly risky for the patients due to adhesions and unintentional ON injury [24]. Biopsy of ON lesions is also not recommended due to high risk of hemorrhage and visual deterioration [24].

Stereotactic radiosurgery remains a controversial alternative to surgery for nonsurgical candidates or patients who have lesions in surgically inaccessible areas [1,20,22–25]. Current evidence suggests radiosurgery can help manage intentional residual (due to eloquent regions) when used as an adjunct to microsurgical resection [26]. However, current evidence lacks to establish stereotactic radiosurgery or gamma-knife radiosurgery treatment superiority over surgical resection alone [20–26]. Although there is a literature gap in this domain, current studies do not recommend radiosurgery alone for ON CM due to high risks of radiation-induced toxicity to the optic visual pathway and the likely need for histological confirmation preradiosurgery (due to difficult radiological diagnosis) [24].

Medical management for CMs is only supportive and includes analgesia for headaches and antiepileptics for seizure control, but these may not be applicable to ON CMs [1].

## Conclusion

This case report serves to outline the radiological diagnostic challenge revolving around prechiasmatic optic nerve focal mass lesions. In the context of such confined and overlapping symptomatology, a correct radiological diagnosis of these lesions might be the only factor that determines the clinical implications for a patient. Although, in our case report there was hemorrhage and optic nerve thickening during the course of the disease, which enabled further biochemical investigation, this might not always be the case. In contemporary encouragement of noninvasive practice the clinical implications and management decision is vastly dependent on radiological diagnosis, hence, it is important to keep a wide but limited differential for intracranial optic nerve focal mass lesions and not dismiss the possibility of an optic nerve cavernoma despite its rare incidence.

## Patient consent

The author of this case report declares that written informed consent to publish this case report was obtained from the patient.

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