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Case Report

Magnetic resonance imaging characteristics of glioblastoma of the optic pathway during adulthood ☆

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ABSTRACT

Glioma has been previously known as the most common adult brain tumor. Glioma of the optic pathway is predominated by low-grade neoplasms. High-grade glioma in adults is extremely rare. In this study, we present the case of a 46-year-old male patient who developed glioblastoma of the optic chiasm extending along the optic tract. This study aims to discuss several common differential diagnoses of nontumor diffuse lesions in the optic pathway and their clinical symptoms and magnetic resonance imaging findings, which could help navigate management.

Introduction

Optic glioma (OG) constitutes 0.6%-1.2% of all brain tumors [1]. The most common types behave as benign lesions in children aged <15 years (85%) and are closely related to neurofibroma type I (NFI) [2]. Meanwhile, the aggressive type is uncommon and typically occurs during adulthood. Since OG was first introduced in 1973 by Hoyt et al. [3], only 30 cases have been reported among 71 cases of optic pathway neoplasms [3,4,5–9].

The vast majority of OG has been found in middle-aged men with progressive visual loss within several weeks. In

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clinical practice, this entity is usually misdiagnosed as ischemic or inflammatory lesions of the optic tract owing to multiple overlapping features [5]. Nonspecific findings on ophthalmoscopy range from normal to papilledema and retinal vein occlusion. However, a mass extending into the eyeball is exceptional [2,4,5,7,8].

OG can arise from anywhere in the optic pathway, optic chiasm, and optic tract and even show multifocal patterns [5]. Although magnetic resonance imaging (MRI) yields multiple advantages, biopsy is still mandatory owing to nonspecific radiographic findings.

Herein, we describe the case of a 46-year-old man with a 6-week history of progressive visual loss. The initial clinical diagnosis was high-grade glioma of the optic pathway, and the pathological results confirmed glioblastoma.

Case report

A 46-year-old man complained to his ophthalmologist about gradual complete blindness in his left eye and partial blindness in his right eye. The patient was subsequently transferred to the hospital emergency department with initial suspicion of nonarteritic anterior ischemic optic neuropathy (NAION). On clinical examination, he denied any headache, intermittent jaw pain, or temporal region pain relating to eyeball movement. Reduction of reactivity to light and right eye discoloration were documented. There was no neuropathic sign in the other regions.

On brain MRI with intravenous contrast, the radiologist detected a mass in the optic chiasm and left optic nerve region, infiltrating the optic tract, optic radiation, and pituitary stalk (Fig. 1). Furthermore, the mean value within the solid part on the ADC map was 1.049×10^{-3} mm^3/s. Thus, MRI revealed high-grade glioma of the optic pathway, with a modified Dodge classification of grade 3. Biopsy was then indicated for the optic chiasm mass, which confirmed glioblastoma of the optic tract (Fig. 2).

The patient subsequently underwent tumor resection. Total resection was not performed owing to the tumor location, and the neurologic and clinical status of the patient rapidly worsened. Subtotal resection was then performed, and the residual part was found to be adhering to left cranial nerve II, which was unremovable. The postoperative MRI illustrated the tumor residual anterior to cranial nerve II (Fig. 3).

Discussion

OG is an uncommon neoplasm, representing 2% of all brain tumors [4]. The most common type of OG is pilocytic astrocytoma. In 2021, the WHO Classification of Tumors of the Central Nervous System classified this tumor as low-grade glioma (WHO grade 1), which is typically seen in children [4,10,11]. Meanwhile, high-grade OG is extremely rare and is usually found in adults. The vast majority of cases are anaplastic astrocytoma (WHO grade 3) and glioblastoma [1] (WHO grade 4) [4,10,11]. Our patient was aged 46 years, lower than the mean age of a typical patient with OG (61 years) [3,4–6] (Fig. 2).

At the early stage, the clinical symptoms of OG can be nonspecific and overlap with those of several other ischemic or inflammatory diseases. The early signs tend to be skeptical and partially respond to corticosteroid therapy [5]. However, OG can progress to a series of symptoms, including visual loss, eye discoloration, retinal vein occlusion, exophthalmos, and neurological signs relating to the tumor location in the optic pathway [2,5,12]. These aggressive signs suggest a diffuse infiltrating lesion, which should be evaluated on MRI. The mean time to total visual loss has been reported to be within 5-6 weeks [3]. Differential diagnoses encompass NAION, which usually occurs in patients aged >50 years and presents with sudden visual loss either ipsilaterally or bilaterally [13,14]. Another candidate is optic nerve inflammation occurring on one side in patients aged <50 years and rapidly responding to corticosteroid therapy [15,16]. Retinal vein occlusion and temporal arteritis should also be considered as differential diagnoses owing to some overlapping features [1].

Most OGs arise from the optic chiasm, cranial nerve II, or less frequent sites, including the hypothalamic region, temporal lobe, and basal ganglion system [11,17]. Assessment of the tumor location is based on clinical symptoms encompassing ipsilateral or bilateral onset and ophthalmic signs. A cranial nerve mass typically presents with ipsilateral visual loss and abnormal findings on ophthalmoscopy (optic disc swelling and retinal vein occlusion). On the contrary, an optic chiasm mass highlights bilateral visual loss, normal ophthalmoscopy findings, and infiltration of the suprasellar region, hypothalamic region, and third ventricle [18]. The infiltration of the basal ganglion nucleus is consistent with hyperphagia at the later stage. Optic tract lesions could lead to contralateral hemianopsia [11].

The MRI features are indistinguishable between general optic pathway lesions and OGs. Mass-like lesions in the optic chiasm; infiltration of the optic nerves, optic tract, and pituitary stalk; and vivid enhancement on postcontrast images are all characteristic findings. In our medical literature search, classic OGs tend to infiltrate along the optic nerve to the optic chiasm, optic tract, and optic radiation in some cases [2,4,7,8]. This diffuse behavior is valuable in evaluating OG using the modified Dodge classification. Stage 1a includes ipsilateral cranial nerve infiltration; 1b, bilateral cranial nerve infiltration; 1c, intracranial segment infiltration; 2, optic chiasm infiltration; 2a, central optic chiasm infiltration; 2b, ipsilateral optic chiasm infiltration; 3, optic tract infiltration; and 4, posterior tract infiltration, including the hypothalamic and meningeal regions and relating to NFI. Eyeball infiltration has yet to be reported, which might be attributed to the mechanical barrier in the lamina cribrosa and the local biological barrier [5]. MRI signals are usually hypointense on T1-weighted images and reveal vivid enhancement on sequences [5,19]. OG predominates by the irregular blurry border, large mass effect, peritumoral vasogenic edema, and intratumoral hemorrhage [11].

According to Warinthorn et al., 1.119×10^{-3} mm^2/s is the cutoff ADC that differentiates between low- and high-grade gliomas, with a sensitivity of 90% and a specificity of 88.9%. Our patient demonstrated a barely higher value (1.049×10^{-3}

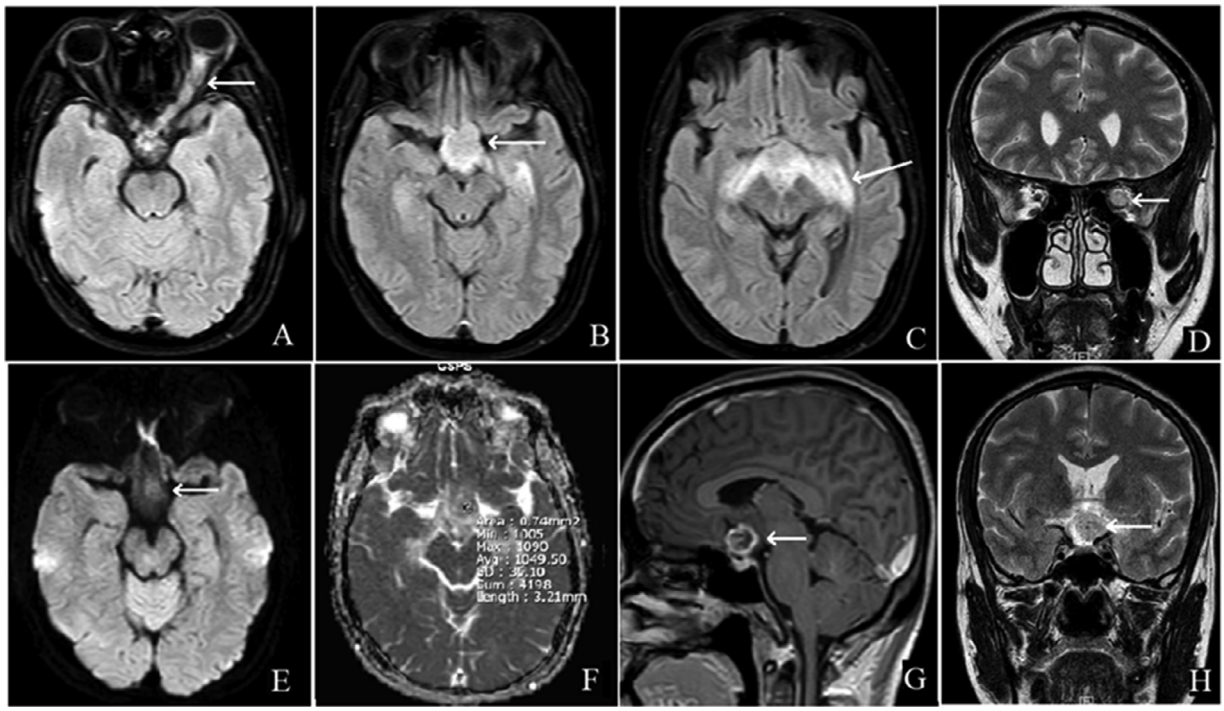

Fig. 1 – Gadolinium-based magnetic resonance images show an optic chiasm mass extending along the optic pathway. (A-C) Fluid-attenuated inversion recovery images on the axial plane demonstrate a diffuse lesion in the left optic nerve, infiltrating the optic chiasm, pituitary region, optic tract, and optic radiation bilaterally (white arrow). (D and H) T2-weighted images on the coronal plane demonstrate a mass in the optic chiasm (white arrow in [H]) and left cranial nerve II (white arrow in [D]), which has a hyper-signal intensity compared with the contralateral side. (E and F) Diffusion images demonstrate an ADC of 1.049 x 10^{-3} mm^3/s. (G) Gadolinium-based T1-weighted image on the coronal plane demonstrates a heterogeneous enhancement in the optic chiasm.

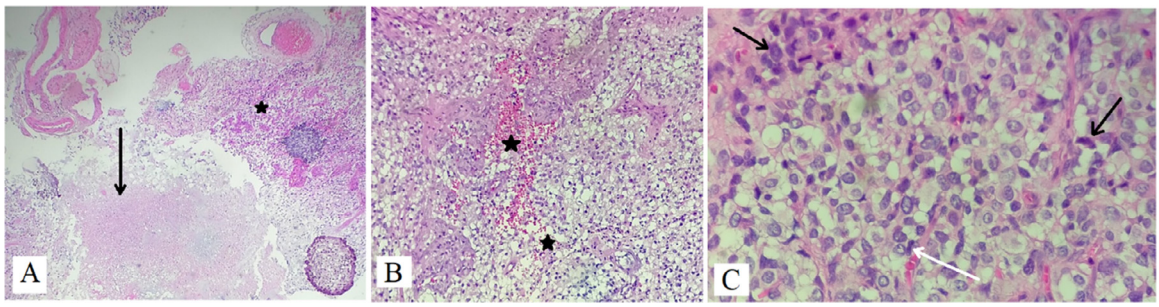

Fig. 2 – Pathological structure of optic pathway glioblastoma. (A) Microscopic image of the tumor composed of hyper-cellular regions (star) arranged in a perpendicular fashion surrounding the necrotic areas (black arrow) (H & E, x 10 magnification). (B) Multiple microvascular proliferations (stars) located in both hyper-cellular and infiltrating regions (H & E, x 40 magnification). (C) Tumor composed of multiple poorly differentiated, uniform, small cells resembling the structure of small-cell glioblastoma (H & E, x 200 magnification).

mm^2/s). The diffuse extension, heterogeneous enhancement, and absence of a clear restriction part should be distinguished from those of other tumoral and nontumoral diffuse lesions [10,11,20]. Optic nerve sheath meningioma commonly arises surrounding cranial nerve II, with tram track signs of meningioma [21]. Sellar and suprasellar neoplasms typically occur at the optic chiasm but show no optic nerve enhancement. Nontumoral lesions encompass infectious and noninfectious inflammation of cranial nerve II, including multiple sclerosis (ipsilateral and short segment), neuromyelitis optica, and anti-MOG encephalomyelitis (bilateral, long segment, and spinal involvement) [21]. This group is self-limiting and generally responds to corticoid therapy and recovers within a few weeks [21].

Cranial nerve II lesions can be classified according to the anatomic lesion. One group comprises nerve involvement and

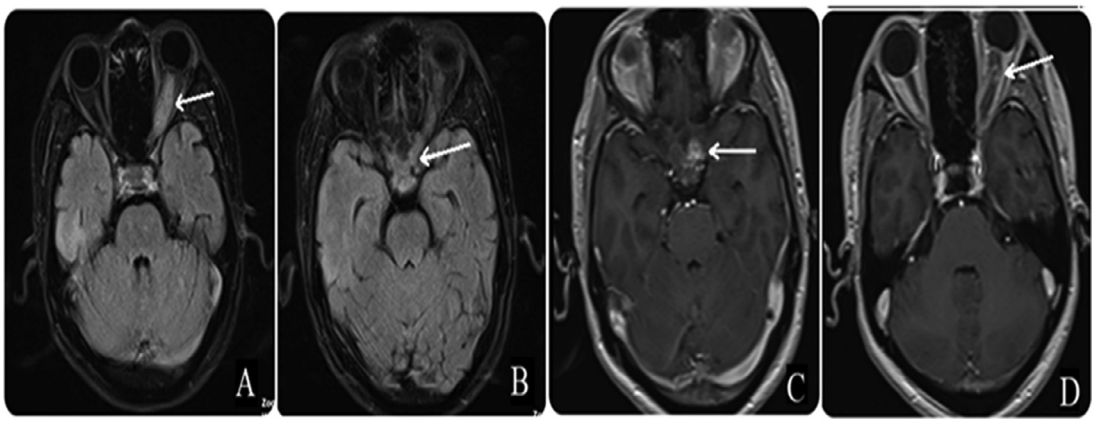

Fig. 3 – Magnetic resonance images 1 month after operation.

nerve sheath preservation (NMO and NAION). The other group includes meningioma and pseudotumoral lesions. Both nerve and nerve sheath involvements include high- and low-grade gliomas, secondary perineuronal inflammation due to infection, granulomatous inflammation, lymphoma, and metastasis [17].

Biopsy is still mandatory when OG is suspected, especially in cases when nonspecific inflammation is confirmed the first time, but the patient status progressively worsens [5]. If available, the standard strategy encompasses tumor resection, radiotherapy, and chemotherapy (temozolomide) [5]. Generally, glioblastomas of the optic nerve have a poor prognosis. The advent of temozolomide has further improved the mean survival time from 2 to 3 weeks to 2 years (10%-26%) [4,22]. Although the development of gene mutation detection has revolutionized management, an efficient treatment option yielding a good prognosis remains unavailable [1,23]. However, advancements in radiologic diagnosis in a timely fashion can be promising [5].

Conclusion

Glioblastoma of the optic nerve is an uncommon neoplasm, which is rapidly progressive and has a poor prognosis. Early diagnosis is challenging owing to subtle clinical symptoms and radiographic findings. Glioblastoma should be the last diagnostic option after ruling out all other differential diagnoses of optic pathway lesions.

Authors' contributions

Le Thanh Dung and Nguyen Duy Hung contributed equally to this article as first authorship. Le Thanh Dung and Nguyen Duy Hung: Case file retrieval and case summary preparation. Nguyen Duy Hung and Nguyen Minh Duc: preparation of manuscript and editing. All authors read and approved the final manuscript.

Availability of data and materials

Data and materials used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

Our institution does not require ethical approval for reporting individual cases or case series. Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

Patient consent

Informed consent for patient information to be published in this article was obtained.

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