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Cerebrospinal Fluid Sleeve Distension Around the Oculomotor Nerve in Idiopathic Intracranial Hypertension: A Retrospective Case Series

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Abstract

Idiopathic intracranial hypertension (IIH) is a condition characterized by increased intracranial pressure without an underlying mass, hydrocephalus, or venous sinus thrombosis. The definitive diagnosis of IIH is based on clinical features; however, certain neuroimaging findings may suggest the condition. Beyond established diagnostic criteria, numerous other imaging abnormalities have been associated with IIH and may indicate raised intracranial pressure, including narrowed ventricles, tonsillar ectopia, and dilation of Meckel's cave. Thus, this study aimed to characterize abnormalities of the oculomotor nerve and its cistern in patients with IIH.

After reviewing all IIH cases over a three-year period, six female patients (mean age, 33 years; mean BMI, 36) who met the 2013 Friedman diagnostic criteria and demonstrated the imaging findings of interest were included in this retrospective case series. Coronal T2-weighted sequences were used to evaluate the oculomotor cistern and intraorbital division of the oculomotor nerve (cranial nerve III). Eleven of the 12 cisterns met the ⩾3.0 mm threshold (mean diameter, 3.8 mm bilaterally). T2 hyperintensity along the superior and inferior divisions of the right and left intraorbital oculomotor nerves was identified in two patients each. Overall, three patients demonstrated involvement of at least one division. However, none of the patients had evidence of third nerve palsy.

These observations describe oculomotor cisternal prominence and intraorbital oculomotor nerve T2 hyperintensity in a selected series of patients with IIH. The clinical significance of these findings and their relationship with IIH remain undetermined. Larger studies are needed to establish the utility of these findings and elucidate their underlying pathophysiology.

Categories: Neurology, Radiology, Neurosurgery
Keywords: cerebrospinal fluid, idiopathic intracranial hypertension (iih), magnetic resonance imaging, oculomotor cistern, oculomotor nerve

Introduction

Idiopathic intracranial hypertension (IIH) is defined by increased intracranial pressure in the absence of an identifiable underlying cause. The condition most commonly affects obese women of reproductive age, and its incidence is increasing in parallel with global obesity trends [1]. Walter Dandy established the original diagnostic criteria for IIH in 1937, characterizing the condition by elevated intracranial pressure, normal cerebrospinal fluid (CSF), and the absence of an identifiable brain tumor on ventriculography [2]. Typical clinical features of IIH include headaches, transient episodes of monocular or binocular vision loss, pulsatile tinnitus, diplopia, spontaneous CSF leaks, and papilledema [1].

Neuroimaging plays an integral role in the diagnostic workup of IIH, primarily to exclude intracranial pathologies that can cause elevated CSF pressure, such as space-occupying lesions, hydrocephalus, venous sinus thrombosis, and meningeal processes. The 2013 revised diagnostic criteria defined the neuroradiologic findings suggestive of IIH and distinguished between definitive and suggestive diagnoses [3]. Individual neuroimaging findings, such as an empty sella, are nonspecific and may occur in a substantial proportion of the general population. However, the presence of at least three of the four characteristic neuroimaging findings described by Friedman et al. may support a diagnosis of IIH in patients who do not meet the definitive diagnostic criteria, particularly in the absence of papilledema. Despite the established diagnostic criteria, misdiagnosis of IIH remains a concern, with up to 40% of individuals reportedly misdiagnosed [4].

Proposals have been made to update the diagnostic criteria for IIH, with particular emphasis on improving the accuracy of neuroradiologic findings [5]. A recently developed MRI-based score [6] has been shown to estimate the likelihood of papilledema in patients with IIH more accurately than existing neuroimaging

criteria [3]. Various additional MRI findings have been associated with IIH, including slit-like ventricles, inferior displacement of the cerebellar tonsils, and enlarged Meckel's caves [7]. The integration of these imaging features into current diagnostic criteria remains uncertain. Nonetheless, their utility warrants further investigation, given that MRI findings suggestive of IIH often precede invasive diagnostic testing via lumbar puncture.

In this case series, we present, for the first time, MRI abnormalities of the oculomotor nerve (cranial nerve III) in patients with definitive IIH. Increased intracranial CSF pressure may contribute to distension of the oculomotor cistern, analogous to the distension of the perioptic subarachnoid space observed in IIH. On this basis, we evaluated the oculomotor cistern and intraorbital course of the oculomotor nerve for potential pressure-related imaging abnormalities. These observations prompted us to characterize the appearance of the oculomotor cistern and intraorbital oculomotor nerve in a selected series of patients with established IIH.

Materials And Methods

This retrospective case series was conducted at Rush University Medical Center and included patients diagnosed with IIH who fulfilled the 2013 revised diagnostic criteria over a three-year study period. Of these patients, six demonstrated the imaging findings of interest and were included in the series. Clinical and imaging findings were retrospectively reviewed. Clinical variables included age, headache, pulsatile tinnitus, papilledema, diplopia, body mass index (BMI), CSF opening pressure, and CSF analysis. Given the small sample size and descriptive nature of the study, findings were summarized using descriptive statistics without statistical testing.

Current diagnostic neuroimaging criteria for IIH, including partially empty sella, posterior globe flattening, perioptic subarachnoid space distension with or without optic nerve tortuosity, and transverse sinus stenosis, were reviewed for each patient [3].

The oculomotor cistern and intraorbital segments of the oculomotor nerves (cranial nerve III) were evaluated for abnormalities by a single neuroradiologist with two years of experience. The imaging assessment was performed retrospectively, and the reader was not blinded to the patients' clinical information or established IIH diagnoses. All assessments of the oculomotor cistern and intraorbital oculomotor nerves were performed using coronal T2-weighted turbo spin-echo sequences (slice thickness, 4 mm).

The oculomotor cistern was defined as the region along the roof of the posterior portion of the cavernous sinus where cranial nerve III is at least 75% surrounded by dura. The diameter of the oculomotor cistern was measured at its posterior opening (oculomotor porus), corresponding to its widest diameter on coronal images. A diameter of ⩾3.0 mm was used as an exploratory threshold to characterize cisternal prominence [8,9]. For the intraorbital segment of the nerve, any definite T2 hyperintense signal similar to that of CSF along its superior and inferior divisions was considered an abnormal finding.

Results

All six patients were female and ranged in age from 15 to 51 years (mean, 33 years). All patients presented with headaches. Four patients experienced pulsatile tinnitus, while five had papilledema. One patient without papilledema had diplopia but otherwise fulfilled the diagnostic criteria for IIH. None of the patients had diplopia attributable to oculomotor nerve paresis. BMI ranged from 29 to 44 kg/m² (mean, 36 kg/m²). CSF opening pressure ranged from 260 to 700 mm H_2O (mean, 392 mm H_2O), and CSF analysis was normal in all patients.

The clinical features of the patients are summarized in Table *1*. All six patients demonstrated a partially empty sella. Posterior globe flattening was present in three patients, while perioptic subarachnoid space distension was observed in two patients. MR venography was available for four patients, three of whom demonstrated bilateral transverse sinus stenosis.

Patient	Age at diagnosis	Headache	Pulsatile tinnitus	Papilledema	Diplopia	BMI	CSF (mm of H_2O)
1	29	Y	N	Y	Y	43	420
2	41	Y	Y	Y	N	30	330
3	40	Y	Y	Y	N	44	260
4	15	Y	Y	Y	N	39	700
5	22	Y	N	Y	N	31	380
6	51	Y	Y	N	Y	29	260

TABLE 1: Clinical characteristics of patients with IIH

BMI: body mass index, Y: yes, N: no, IIH: idiopathic intracranial hypertension

The mean diameter of the oculomotor cistern was 3.8 mm on both the right (range, 2.8-5.1 mm) and left (range, 3.0-5.4 mm) sides (Figure *1*). The normal calculated transverse diameter of the oculomotor cistern is 2.2 ± 0.8 mm on the right and 2.1 ± 1.0 mm on the left [8]. Using a diameter of ⩾3.0 mm as a practical threshold for prominence, 11 of the 12 oculomotor cisterns met this criterion.

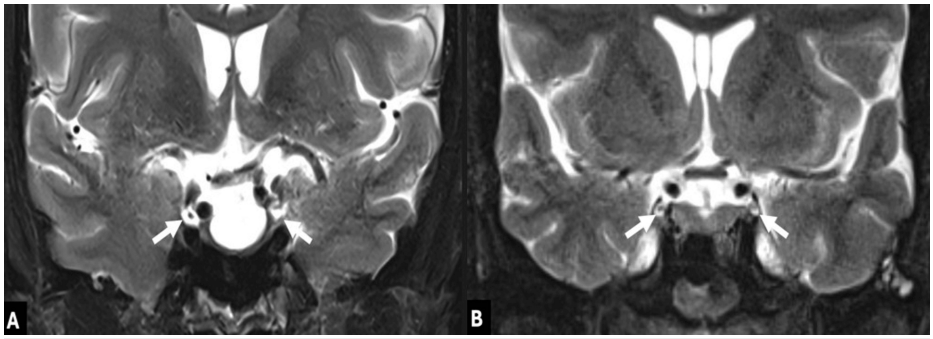


FIGURE 1: Prominent (A) and normal (B) oculomotor cisterns

Coronal T2-weighted image of a 40-year-old female with morbid obesity (BMI, 44.2), headache, visual disturbances, pulsatile tinnitus, and elevated CSF opening pressure, demonstrating bilateral oculomotor cistern distension (arrows in A). For comparison, a 39-year-old female who underwent MRI to evaluate a suprahyoid carotid space lesion demonstrates normal oculomotor cisterns (arrows in B).

BMI: body mass index, CSF: cerebrospinal fluid, MRI: magnetic resonance imaging

T2 hyperintensity along the superior and inferior divisions of the right intraorbital oculomotor nerve was identified in two patients each. On the left side, T2 hyperintensity along the superior and inferior divisions was also observed in two patients each (Figure *2*). The neuroimaging features are summarized in Table *2*.

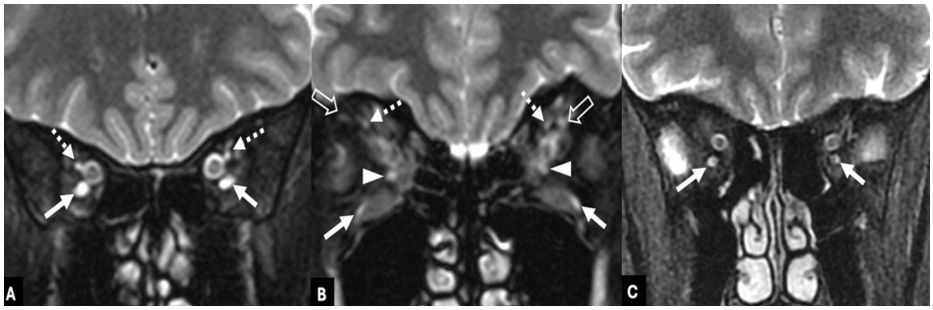


FIGURE 2: Abnormal signal intensities of the superior and inferior intraorbital oculomotor nerves

Coronal T2-weighted images of a 29-year-old female (A, B) with class III obesity (BMI, 43.05), presenting with headache, visual disturbances, papilledema on fundoscopy, and elevated CSF opening pressure (420 mm H_2O). T2 hyperintensity is observed along the superior (dashed white arrows in A) and inferior (solid white arrows in A) divisions of the bilateral oculomotor nerves. A more anterior section again demonstrates T2 hyperintensity along the bilateral superior divisions (dashed white arrows in B), with the superior ophthalmic veins located lateral to the nerves (hollow arrows). The inferior divisions further subdivide into branches that innervate the medial rectus (arrowheads) and inferior oblique (solid white arrows in B), both of which demonstrate persistent T2 hyperintensity.

A coronal T2-weighted image of another patient (C), a 22-year-old female with class I obesity (BMI, 30.87), headache, visual disturbances, papilledema, and elevated CSF opening pressure (380 mm H_2O), demonstrates T2 hyperintensity along the inferior divisions of the bilateral oculomotor nerves (solid arrows in C).

BMI: body mass index, CSF: cerebrospinal fluid

Patient	Partial empty sella†	Flattened posterior globe†	POSD-ONT†	Transverse sinus stenosis†	Right oculomotor nerve			Left oculomotor nerve		
					Cistern	Superior division	Inferior division	Cistern	Superior division	Inferior division
1	Y	Y	Y	NA	Y	Y	Y	Y	Y	Y
2	Y	N	N	NA	N	N	N	Y	N	N
3	Y	N	N	N	Y	N	N	Y	N	N
4	Y	N	N	Y	Y	N	N	Y	N	N
5	Y	Y	N	Y	Y	N	Y	Y	N	Y
6	Y	Y	Y	Y	Y	Y	N	Y	Y	N

TABLE 2: Neuroradiologic features of patients with IIH

† Current neuroimaging criteria suggestive of IIH diagnosis.

NA: not available (MR venogram was not obtained), IIH: idiopathic intracranial hypertension, POSD-ONT: perioptic subarachnoid space distension with or without optic nerve tortuosity, Y: yes, N: no

Discussion

We have described six cases of IIH characterized by prominent oculomotor cisterns and T2 hyperintensity along the intraorbital segments of the oculomotor nerves on MRI.

The oculomotor nerve originates from the midbrain nuclei and courses along the medial aspects of the cerebral peduncles within the interpeduncular cistern [8]. Cranial nerve III has five segments: the nuclear segment, intra-axial midbrain segment, cisternal segment, cavernous segment, and intraorbital segment. After entering the orbit through the superior orbital fissure, it divides into superior and inferior divisions (Figure *3*) [10].

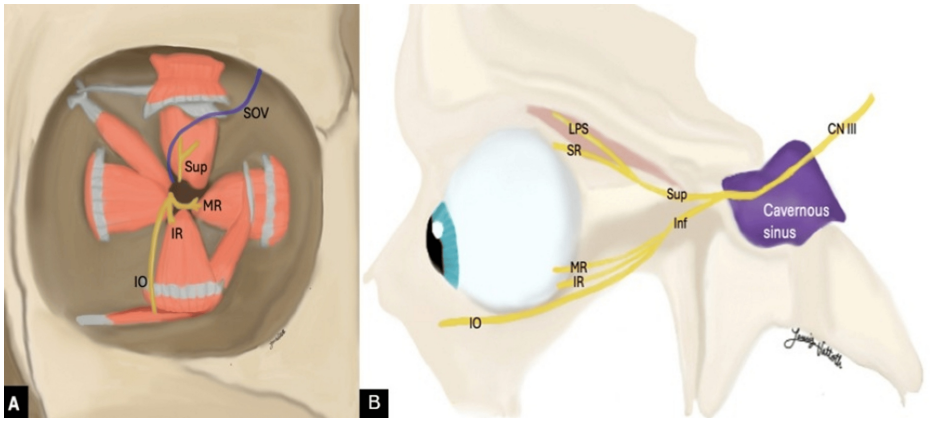


FIGURE 3: Anatomy of the oculomotor nerve and its intraorbital divisions

Diagrammatic representation of the oculomotor nerve anatomy in frontal (A) and lateral (B) views. The oculomotor nerve (cranial nerve III) enters the orbit through the superior orbital fissure and divides into Sup and Inf divisions. The superior division innervates the SR and LPS muscles. The inferior division gives rise to branches that innervate the MR, IR, and IO muscles.

On imaging, T2 hyperintensity along the superior division of cranial nerve III may be confused with the SOV, as both structures traverse the orbit between the optic nerve and superior rectus muscle and enter through the superior orbital fissure. However, the nerve follows a linear trajectory, whereas the vein follows a tortuous mediolateral course as it travels posteriorly toward the fissure.

Sup: superior, Inf: inferior, SR: superior rectus, LPS: levator palpebrae superioris, MR: medial rectus, IR: inferior rectus, IO: inferior oblique, SOV: superior ophthalmic vein

Illustration created using Procreate Pocket (Savage Interactive Pty Ltd, Tasmania, Australia). Image credit: Aysa L. Vattoth.

Cranial nerve III is surrounded by a cone-shaped oculomotor cistern that acts as an arachnoid cuff and communicates with the basal cisterns. This sleeve-like cistern has dural and arachnoid walls that extend from the oculomotor porus, covering the oculomotor nerve for a variable distance and terminating inferior or anterior to the tip of the anterior clinoid process [9].

The diameter and length of the oculomotor cistern have been previously measured and described in the literature, primarily to delineate the microsurgical anatomy and facilitate the management of pathologies in this region [8,9]. The normal calculated transverse diameter of the oculomotor cistern is 2.2 ± 0.8 mm on the right and 2.1 ± 1.0 mm on the left [8]. A diameter of ⩾3.0 mm was observed in 11 of the 12 oculomotor cisterns in this series. We noted that this measurement approximately corresponds to the width required to accommodate a second nerve of the same diameter within the cistern, offering a pragmatic criterion for evaluation in a high-volume setting. The enlargement of the oculomotor cisterns in our patients with IIH may be attributable to increased intracranial pressure within the subarachnoid spaces, reflecting the same pathophysiological mechanism responsible for distension of the optic nerve sheaths [11].

The underlying etiology of T2 hyperintensity along the intraorbital oculomotor nerve remains unclear. We believe this finding may represent distension of the oculomotor cistern along the nerve beyond its normally described anatomical boundaries, likely as a consequence of increased intracranial CSF pressure. Although no patients in our series had oculomotor nerve deficits, IIH has been associated with the development of third nerve palsy [12-14].

The superior ophthalmic vein can be mistaken for T2 hyperintensity along the superior division of the oculomotor nerve because both structures enter the orbit through the superior orbital fissure and travel between the superior rectus muscle and optic nerve [15]. However, they can be distinguished based on their course and morphology (Figure *4*). The superior division of the oculomotor nerve follows a straight trajectory, terminating at the superior rectus and levator palpebrae superioris muscles, which it innervates [10]. In contrast, the superior ophthalmic vein can be traced anteriorly to the superior orbital rim, where it is formed by the confluence of the angular and supraorbital veins [15]. It follows a tortuous course, running from medial to lateral as it travels posteriorly toward the superior orbital fissure [16].



FIGURE 4: Normal anatomical course of the superior ophthalmic vein

Axial T2-weighted image (A) of a 58-year-old male with an unremarkable study, demonstrating the normal tortuous course of the bilateral superior ophthalmic veins (solid arrows in A). Sequential axial T2-weighted images (B and C) of a 29-year-old female with IIH (same patient as in Figure 2), showing the straight course of the superior divisions of the bilateral oculomotor nerves (dashed arrows in B), with the superior ophthalmic veins located laterally (solid arrows in B). The left superior ophthalmic vein (solid arrow in C) deviates laterally as it courses posteriorly from the superior orbital rim.

IIH: idiopathic intracranial hypertension

This study has several limitations, including its retrospective design, small sample size, and single-center setting. The absence of a control group limits the assessment of the specificity of these imaging findings for IIH. Additionally, the findings were evaluated descriptively without formal statistical analysis. The use of conventional T2-weighted imaging represents a further limitation, as it does not allow definitive characterization of the observed intraorbital oculomotor nerve hyperintensity. Dedicated high-resolution cranial nerve imaging and appropriate sequence optimization are necessary to distinguish perineural CSF from intraneural signal abnormalities.

Conclusions

IIH is a diagnosis of exclusion characterized by a diverse and often complex clinical presentation. Several imaging features on brain MRI and MR venography may help strengthen clinical suspicion. In addition to oculomotor cistern distension, T2 hyperintensity along the intraorbital course of the oculomotor nerve may represent an intraorbital extension of the oculomotor cistern or edema along the nerve bundles. Although the diagnostic significance of these findings remains undetermined, awareness of these appearances may be useful when evaluating the oculomotor nerve and its surrounding cistern on MRI. These observations are exploratory and require further validation in larger prospective studies.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have

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