Utility of Intracranial Pressure Monitoring for Diagnosis of Idiopathic Intracranial Hypertension in the Absence of Papilledema

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BACKGROUND: **Idiopathic intracranial hypertension (IIH) is characterized by headaches, visual obscurations, and papilledema, and the diagnosis involves lumbar puncture (LP) with an elevated opening pressure (OP) ≥20 cm H_20. When papilledema is absent, the diagnosis becomes less clear. Some physicians have argued that the absence of papilledema rules out IIH, whereas others maintain that elevated OP is sufficient for diagnosis.**

METHODS: **The authors performed a single-institution 4-year retrospective analysis of patients who underwent invasive intracranial pressure (ICP) monitoring for presumed IIH.**

RESULTS: **A total of 22 patients were reviewed, and 13 had classic symptoms of IIH, documented elevated OP, and absence of papilledema; 5/13 (38%) patients had proven intracranial hypertension as shown by invasive ICP monitoring, whereas 8/13 (62%) had normal ICP.**

CONCLUSIONS: **With the use of current diagnostic algorithms of clinical presentation and elevated OP, over half of patients without papilledema in our series would be falsely diagnosed with IIH, which could result in unnecessary medical and surgical intervention. Thus, elevated OP as determined by LP is insufficient to diagnose IIH. On the other hand, the absence of papilledema does not rule out intracranial hypertension.**

INTRODUCTION

Idiopathic intracranial hypertension (IIH) is characterized by elevated intracranial pressure (ICP) of unknown cause.[1-3] The symptoms include headaches that are typically exacerbated by lying down and by a Valsalva maneuver.[4] Other symptoms include visual disturbances, tinnitus, and dizziness.[5-8] Papilledema and visual field defects are hallmark signs of the disease.[3,9-12] Although the results of magnetic resonance imaging (MRI) is often normal, findings can include empty sella, flattening of the posterior globes, protrusion of the optic nerve head, optic nerve sheath distention, herniation of the cerebellar tonsils, cerebrospinal fluid (CSF) fistula, and transverse venous sinus stenosis.[13-15]

The diagnostic process for IIH is one of exclusion and relies on clinical symptoms and opening pressure (OP) measured by a lumbar puncture (LP).[1,2,4,13,16,17] In 2014, the IIH Treatment Trial proposed a diagnostic algorithm that used an OP of >20 cm H_2O; however, the criteria by Friedman et al.[14] 2013 and the modified Dandy criteria from 1985[18,19] both describe a higher threshold OP criterion of >25 cm H_2O.[1,11,13,20,21] Meanwhile, OP can be difficult to assess in anxious patients and in obese patients, who make up the classic demographic of people affected by IIH.[22] It can prove difficult to achieve access to the lumbar cistern, and inaccurate measurements are commonly due to incorrect positioning, partial needle obstruction, fluid loss, pharmacologic sedation, moment-to-moment fluctuations, and patient straining.[23] A Valsalva maneuver has been shown to increase OP from 14.6 cm H_2O to 32.3 cm H_2O.[24,25]

In many cases, the diagnosis of IIH is straightforward. However, in some cases the optic disc signs are difficult to interpret as

Key words
- Idiopathic intracranial hypertension
- Intracranial pressure
- Lumbar puncture
- Opening pressure
- Papilledema

Abbreviations and Acronyms
CSF: Cerebrospinal fluid
HVLP: High-volume lumbar puncture
ICP: Intracranial pressure
IIH: Idiopathic intracranial hypertension
LP: Lumbar puncture
OP: Opening pressure
MRI: Magnetic resonance imaging

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a result of congenital anomaly, gliosis, or pallor.[23] Meanwhile, visual field results are contingent on the patient's input, which may be unreliable.[23] In these circumstances, when papilledema is not clearly identified, intracranial ICP monitoring can prove useful in ruling in, or out, an IIH diagnosis. In a literature review, and to our knowledge, 4 articles have discussed the utility of ICP monitoring in the setting of presumed IIH, although none have looked specifically at patients without papilledema nor compared LP-obtained OP with ICP.[22,23,26,27] Moreover, diagnostic algorithms fail to account for patients without papilledema who experience classic IIH symptoms.

We present a series of patients with elevated OP in the absence of papilledema. We aim to corroborate past studies and emphasize a utility for ICP monitoring for selected cases of IIH. We argue that a diagnosis of IIH outside the setting of papilledema warrants more than an elevated OP on LP while also maintaining that absence of papilledema does not rule out intracranial hypertension.

METHODS

Patient Inclusion

The patients were referred to the neurosurgery team for IIH evaluation. Their symptoms commonly included headaches and visual disturbances, and OP on lumbar puncture were elevated. Funduscopic examinations with pupils dilated were performed by our institution's neuro-ophthalmologists for evidence of papilledema. **Table 1** gives details of the patients' characteristics and demographics. The patients were indicated for invasive ICP monitoring when they had met the following criteria: 1) documented elevated OP on lumbar puncture and a diagnosis of IIH, 2) absence of papilledema, 3) persistent severe headache, and 4) multiple medications trial failure. All patients had received various combinations of treatments for presumed IIH, including high-volume lumbar puncture (HVLP), acetazolamide, migraine treatment, or sinus stenting. However, none of the patients had received HVLP in the weeks before admission for ICP monitoring. Each patient was admitted to the neuroscience intensive care unit, and a frontal ICP monitor was placed with use of the Codman Cranial Access Kit (DePuy Synthes, Raynham, Massachusetts, USA) and Integra Camino intraparenchymal monitor (Integra, Plainsboro, New Jersey, USA). The transducer was positioned at the Kocher point and passed approximately 5 to 10 mm within the cranial cavity. An appropriate waveform was verified. ICPs were monitored for an average of 2 days (range, 1–4 days), and the patients were allowed to mobilize and position themselves to comfort. Pressure was considered elevated if >20 mmHg was sustained for more than 5 minutes and was unrelated to activity. Symptoms were repeatedly assessed during ICP monitoring. All patients reported persistent headaches, unchanged from baseline.

A retrospective review (September 2012 to August 2016) of patients who underwent ICP monitoring for workup of IIH without papilledema was undertaken. The study was approved by the Oregon Health & Science University Institutional Review Board, with a waiver of patients' consent. Twenty-two patients were identified, 17 of whom had documented results of LP OP, 14 of which were elevated (1 OP >20 cm H_2O, 13 OPs ≥25 cm H_2O).

Table 1. Patient Demographics

Characteristic	Number of Patients (%), n = 13
Mean age (years)	32
Mean body mass index (kg/m^2)	39.5
Female sex (n)	12
Comorbidities (n)	
Migraines	5
Sleep apnea	7
Pulmonary disease (COPD, asthma)	2
Diagnosed psychiatric disorder*	11
Thyroid disorder	3
Papilledema (n)	0
Intact neurological examination results (n)†	13
Prior treatment (n)	
Acetazolamide	6
Topiramate	6
Diuretic	2
Serial high-volume lumbar punctures	8
Optic nerve sheath fenestration	1
Venous sinus stent	2
Permanent CSF diversion‡	0

Values are listed as the number of patients (%) unless otherwise indicated.
COPD, chronic obstructive pulmonary disease; CSF, cerebrospinal fluid.
*Includes depression, anxiety, obsessive compulsive disorder, posttraumatic stress disorder, attention deficit hyperactivity disorder.
†Does not include subjective complaints of visual field impairment or sensation loss.
‡Includes ventriculoperitoneal shunt and/or lumboperitoneal shunt in place at the time of intracranial pressure monitoring; 2 patients had shunts that were explanted because of infection well before continuous intracranial pressure monitoring was done.

Neuro-ophthalmology findings were reviewed, and 13/14 patients with elevated OPs had absence of papilledema. The patients in this subgroup (elevated OP, no papilledema; n = 13) were assessed in this study. Each patient's age, body mass index, medical comorbidities, clinical presentation, imaging findings, neuro-ophthalmology findings, previous OPs, and continuously monitored intraparenchymal ICPs were reviewed.

Statistical Analysis

Statistics were performed by use of a conventional χ^2 test with Yates correction for small sample size. To correct for the same patients within the study, a McNemar test was additionally performed.

RESULTS

Twenty-two patients had undergone continuous ICP monitoring for workup of presumed IIH in the time period reviewed. There were no complications. Thirteen of these patients had

Table 2. Clinical Features of 13 Patients with Elevated Opening Pressure and Absence of Papilledema

Patient	Age/Sex	BMI	Symptoms	Neurologic Examination Results*	Ophthalmology Findings	MRI/Angiogram Findings	Headache Treatment Before ICP Monitoring	LP Opening Pressure (cm H_2O)	ICP Range (mm Hg)
1	27/F	29	Headache, visual disturbances	Intact	No papilledema; "some field cut"	7mm pineal cyst; partially empty sella; right transverse sinus stenosis (25mm Hg drop at transverse-sigmoid junction)	Acetazolamide, nortriptyline, paroxetine, opioids	25	1036
2	31/F	36	Headache, bilateral monocular diplopia	Intact	No papilledema; "some retinal dysfunction"	Normal	Spironolactone (for heart)	28	5—25
3	32/F	53	Headache, pulsatile tinnitus	Intact	No papilledema	Normal	Serial HVLPs, acetazolamide, topiramate, lasix, oxycodone	15—33	1—28
4	36/F	41	Headache	Intact	No papilledema	Normal	Topiramate, imitrex, fioricet	23—39	3—22
5	16/F	39	Headache, emesis, visual disturbances	Intact	No papilledema	Normal	Serial HVLPs	>40	6—42
6	57/M	36	Headache, tinnitus, dizziness, phonophobia, blurred vision	Intact	No papilledema	Normal	Serial HVLPs, topiramate, venlafaxine	>25	3—10
7	38/F	46	Headache, photophobia, phonophobia, blurred vision, emesis, pulsatile tinnitus	Intact	No papilledema	Normal	Serial HVLPs, acetazolamide, fluoxetine	30—36	1—12
8	24/F	40	Headache	Intact	No papilledema	Normal	Serial HVLPs, acetazolamide, weight loss	31—36	2—14
9	32/F	44	Headache, pulsatile tinnitus, blurred vision	Intact	No papilledema	Transverse sinus stent patent; no pressure gradient at transverse—sigmoid junction	Serial HVLPs, acetazolamide, transverse sinus stent, VPS (previously explanted because of infection)	24—45	0—18
10	15/F	26	Headache, visual disturbances, blurred vision, emesis	Intact	No papilledema	Normal; no venous restriction with provocative measures	Serial HVLPs, topiramate	20s—30s	4—27 (elevated only upon head rotation)
11	24/F	33	Headache, visual disturbances	Intact	No papilledema; bilateral optic neuropathy and pallor	Left sigmoid sinus stent patent	Left sigmoid sinus stent	26	—1 to14
12	34/F	23	Headache, pulsatile tinnitus, dizziness	Intact	No papilledema	Normal	Serial HVLPs, acetazolamide, topiramate	20—25	2—12
13	45/F	40	Headache, neck pain	Intact	No papilledema; optic disc atrophy	Normal	LPS & VPS (explanted), Chiari decompression, bilateral ONSFs, topiramate	17—22	1—13

BMI, body mass index; MRI, magnetic resonance imaging; ICP, intracranial pressure; LP, lumbar puncture; HVLP, high-volume lumbar puncture; LPS, lumboperitoneal shunt; VPS, ventriculoperitoneal shunt; ONSF, optic nerve sheath fenestration.
*Does not include subjective complaints of visual field impairment or sensation loss.

Table 3. Outcomes in 5 Patients with Elevated Opening Pressure, Absence of Papilledema, and Elevated ICPs by Intracranial Monitoring

Patient	Age/Sex	BMI	Symptoms	Ophthalmology Findings	MRI/Angiogram Findings	LP Opening Pressure (cm H_2O)	ICP Range (mm Hg)	Treatment	Outcome	BMI at Last Follow-Up
1	27/F	29	Headache, visual disturbances	No papilledema, "some field cut"	7-mm pineal cyst, partially empty sella, right transverse sinus stenosis (25 mm Hg drop at transverse–sigmoid junction)	25	10–36	Right transverse sinus stent, VPS	Persistent symptoms	23
2	31/F	36	Headache, bilateral monocular diplopia	No papilledema, "some retinal dysfunction"	Normal	28	5–25	VPS	Persistent symptoms	40
3	32/F	53	Headache, pulsatile tinnitus	No papilledema	Normal	15–33	1–28	VPS	Significant improvement	48
4	36/F	41	Headache	No papilledema	Normal	23–39	3–22	VPS	Mild improvement	40
5	16/F	39	Headache, emesis, visual disturbances	No papilledema	Normal	>40	6–42	VPS	Resolution of symptoms	41

ICP, intracranial pressure; BMI, body mass index; MRI, magnetic resonance imaging; LP, lumbar puncture; VPS, ventriculoperitoneal shunt.

documented elevated OPs and absence of papilledema by ophthalmologic examination; thus, they were included in a full analysis. All 13 patients (12 female) had intact results of on neurologic examination. The mean age was 32 years (range, 15–57 years), and the mean body mass index was 39.5 (range, 23–53) (Table 1). Common symptoms included headache, visual disturbances, and decreased visual acuity at the time of presentation for continuous ICP monitoring. Imaging consistently included MRI and occasionally cerebral angiography. All but 1 MRI revealed normal findings without evidence of increased ICP. This patient, who later received a diagnosis of true intracranial hypertension, had a 7-mm pineal cyst, partially empty sella, and right transverse sinus stenosis with 25 mm Hg drop at the transverse–sigmoid junction during cerebral angiography.

At the time of ICP monitoring, 11/13 (85%) patients had been treated medically with carbonic anhydrase inhibitors, diuretics, or both. Two patients had prior stenting of the transverse sinus (with patent stents confirmed on imaging). Two other patients had permanent CSF diversion; however, their shunts were explanted secondary to infection before ICP monitoring. Elevated OPs were confirmed after all treatment modalities, but none of the patients underwent HVLP in the weeks before ICP monitoring.

The OPs ranged from 22 to 45 cm H_2O. One patient's highest OP was 22 cm H_2O, and all the others were $\geq$25 cm H_2O. Intracranial pressure monitoring demonstrated elevated ICP in 5/13 (38%) patients, and the ICPs were within normal limits in 8/13 (62%) of patients (Table 2). A conventional χ^2 test with Yates correction revealed a P value of 0.0029, and the McNemar correction provided a 1-tail P value of 0.03682, well within statistical significance.

All 5 patients with elevated ICPs were treated with ventriculoperitoneal shunting. One patient received a right transverse sinus stent 1 year before permanent CSF diversion. Two of these patients had significant improvement in their symptoms, 1 had mild improvement, and the symptoms in 2 were unchanged (Table 3). Those who had normal ICPs were not followed up at length by the neurosurgery team; thus, outcome data are unavailable.

Of interest, 5 patients from the original cohort of 22 patients had ventriculoperitoneal or lumboperitoneal shunts in place at the time of ICP monitoring (Table 4). None had documented elevated OPs since shunt placement, and thus they were excluded from the full analysis. However, ICPs were monitored, and no patients in this shunted group (n = 5) had elevations in ICP.

DISCUSSION

Idiopathic intracranial hypertension affects approximately 1 to 2 per 100,000 people and is nearly 20 times more likely to affect obese women of childbearing age.[1-4,8,15,28-33] Proposed causes include increased venous sinus pressure, decreased CSF absorption, increased CSF secretion, and increased blood volume with cerebral edema.[1,4] Inflammatory markers may also be involved, as may elevated retinol levels.[1,15,32]

Treatment goals include alleviating symptoms while preserving vision.[1,4,15,34] Options include weight reduction, medication management with acetazolamide, drainage of CSF through HVLP, optic nerve sheath fenestration, stent angioplasty of sinus stenosis, and permanent CSF diversion such as lumboperitoneal and ventriculoperitoneal shunts.[1,8,13,35,36] There is growing information that bariatric surgery can be an effective treatment in obese women.[37-39]

The universally accepted criteria for the diagnosis of IIH have included signs and symptoms of increased ICP, including papilledema, and OP on LP $\geq$25 cm H_2O.[1,4,8,11,13,15,21,40-42] When papilledema is absent, the diagnosis of IIH becomes less clear. In

Table 4. Subgroup of Patients Previously Treated with Permanent CSF Diversion Before Intracranial Pressure Monitoring

Patient	Age/Sex	BMI	Symptoms	Neurologic Examination Results*	Ophthalmology Findings	MRI/Angiogram Findings	Headache Treatment Before ICP Monitoring	LP Opening Pressure (cm H_2O)	ICP Range (mm Hg)
1	28/F	28	Headache, visual disturbances	Intact	No papilledema	Normal	VPS, ONSF	15	1–15
2	18/F	41	Headache, nausea, blurred vision	Intact	No papilledema	Normal	Serial HVLPs, VPS, acetazolamide	25–40 (measured before VPS)	−8 to 14
3	32/F	40	Headache, transient vision loss, syncope	Intact	No papilledema	Posterior fossa arachnoid cyst, status post fenestration	VPS	N/A	2–14
4	33/F	46	Headache, syncope	Bilateral exotropia	No papilledema	Decreased supracerebellar cyst, status post fenestration	VPS, supracerebellar cyst fenestration	19	3–12
5	60/F	38	Headache, pulsatile tinnitus, photophobia	Right cranial nerve VI palsy	No papilledema	Slit ventricles; thickening of dura	Bilateral VPS	"Normal"	0–16

CSF, cerebrospinal fluid; BMI, body mass index; MRI, magnetic resonance imaging; ICP, intracranial pressure; VPS, ventriculoperitoneal shunt; OPNS, optic nerve sheath fenestration; HVLP, high-volume lumbar puncture.
*Does not include subjective complaints of visual field impairment or sensation loss.

1972, Lipton and Michelson[43] were the first to report IIH without papilledema, describing a single case study of a woman with bilateral cranial nerve VI palsy, OP >55 cm H_2O, and improvement of symptoms after CSF drainage. In 1991, Marcelis and Silbertstein[44] published an article identifying 10 patients without papilledema and OPs >20 cm H_2O, exemplifying that IIH without papilledema was a clinically important headache syndrome. In 1998, Wang et al.[45] performed a case–control study of 25 consecutive patients without papilledema who had OP >20 cm H_2O on 2 occasions. However, more recently, Friedman et al.[14] reported that papilledema is the "hallmark" of IIH, and Chan[3] added that "the diagnosis of IIH without papilledema can be made only if a unilateral or bilateral sixth nerve palsy is present." In our study, all 13 patients had elevated OPs and absence of papilledema, and none had cranial nerve palsy. Continuous ICP monitoring demonstrated that 5/13 (38%) patients in this cohort had true intracranial hypertension. Meanwhile, the remaining 8/13 (62%) patients with elevated OPs had normal ICPs when continuously monitored. The implications of this data are 2-fold: 1) absence of papilledema does not rule out intracranial hypertension, and 2) an elevated OP by LP is insufficient to diagnosis IIH, as outlined in most accepted diagnostic algorithms.

In 2013, Friedman et al.[14] incorporated MRI findings into the diagnostic criteria of IIH. This included flattening of the posterior pole of the eyes, dilatation and tortuosity of the optic nerve sheaths, empty sella turcica, and stenosis of 1 or both transverse sinuses.[8,15] In our patient cohort, only 1/5 (20%) patients with true intracranial hypertension had suggestive findings on MRI, which included a partially empty sella and right transverse sinus stenosis. This suggests that a lack of imaging findings does not rule out IIH, and further workup is indicated.

To our knowledge, 4 articles discuss the utility of ICP monitoring in the setting of presumed IIH. In 2010, Toma et al.[22] used intraparenchymal probes in 20 patients who did not fulfill the diagnostic criteria for IIH or presented with persistent symptoms after shunt insertion. The authors found that about half of patients had normal ICPs, whereas an equal number had true intracranial hypertension. Horcajadas Almansa et al.[26] performed ICP monitoring on 10 patients with incomplete criteria for IIH and found ICPs <25 cm H_2O in 7. In 2011, Warden et al.[23] studied 8 patients with elevated OP, and 2/8 (25%) patients had elevated ICPs on continuous monitoring. No complications were reported. In 2017, Xu et al.[27] reviewed 30 patients who underwent ICP monitoring for workup of IIH, 17 of whom had recent LPs. All 17 had elevated OPs, while only 2 had elevated ICP during intracranial monitoring. These authors demonstrate the value of intraparenchymal ICP monitoring for a diagnosis of IIH, and the data we present here corroborate the value of intraparenchymal ICP monitoring. Our results are unique in that there is a specific focus on a cohort of patients with classic symptoms of intracranial hypertension, elevated OP on LP, but absence of papilledema.

Current diagnostic algorithms do not address the kind of cases referenced above. Carver and Drew[4] in 2017 published an IIH diagnosis update; their criteria included headache, nausea, vomiting, transient visual changes, papilledema, and absence of neurologic symptoms with the exception of a sixth nerve palsy. The authors added that OP ≥25 cm H_2O and no cytologic or chemical abnormalities "is diagnostic." If we were to apply current diagnostic algorithms of clinical presentation and elevated OP to our patient cohort, over half the patients without papilledema would receive false diagnoses of IIH, which would result in unnecessary medical and surgical intervention. Therefore, we propose a modified algorithm for workup of IIH, which includes a recommendation for continuous ICP monitoring in the absence of papilledema.

Study Limitations

This study aimed to explore the utility of intraparenchymal ICP monitoring to diagnose IIH in patients without papilledema who had elevated OP by LP. The study was limited in that there was a relatively small number of patients; for rare scenarios, such as elevated OP in the absence of papilledema, large sample sizes would be needed. The study was also limited by potential selection bias; as such, the results may not be representative of the general population. Last, the use of electronic records introduces a potential bias from limitations inherent to variables such as data collection and abstraction.

CONCLUSIONS

Continuous intraparenchymal ICP monitoring is an effective and safe diagnostic instrument that should be used in the setting of presumed IIH without papilledema. The OP by LP was falsely elevated in 62% of patients in our cohort, suggesting that OP is insufficient to diagnose IIH in the absence of papilledema. On the other hand, an absence of papilledema does not rule out intracranial hypertension, inasmuch as 38% of our cohort had true elevated ICP outside the setting of papilledema. Thus, we propose a modified algorithm for IIH determination, including a recommendation for continuous ICP monitoring in the absence of papilledema.

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