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Case Report

A License to Diagnose: Idiopathic Intracranial Hypertension Unmasked During Driving-License Evaluation

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Abstract

Background: Idiopathic intracranial hypertension (IIH) classically presents with headache and visual disturbances; however, some patients remain minimally symptomatic and are identified only through incidental detection of optic disc edema. The prevalence of such presentations remains uncertain, and early recognition is essential to prevent visual morbidity.

Case Description: A 23-year-old woman without medical comorbidities underwent ophthalmological evaluation as part of a driving-license assessment. Fundus examination revealed bilateral papilledema, prompting neurological referral. On detailed questioning, she reported only occasional headaches over six months and intermittent right-sided tinnitus, without transient visual obscurations, diplopia, or visual blurring. Neurological examination was otherwise unremarkable. Magnetic resonance imaging demonstrated bilateral optic nerve tortuosity with flattening of the posterior globe on the left side, while magnetic resonance venography was normal. Cerebrospinal fluid analysis showed an opening pressure of 28 cm H_2O with normal cell counts, protein, and glucose levels. Based on modified Friedman diagnostic criteria, a diagnosis of definite IIH was established. Acetazolamide therapy was initiated, and follow-up ophthalmological assessment was planned.

Conclusion: This case highlights the importance of evaluating incidentally detected papilledema during routine visual screening. Driving-license examinations may occasionally uncover clinically silent or minimally symptomatic IIH, allowing timely intervention before irreversible visual complications occur.

Keywords: *Asymptomatic, Headache, Papilledema*

Key Messages

Visual prognosis remains same for both asymptomatic and symptomatic IIH, hence early detection prevent permanent blindness in asymptomatic cases.

Introduction

Idiopathic intracranial hypertension (IIH) is a disorder characterized by elevated intracranial pressure in the absence of an intracranial mass lesion, hydrocephalus, infection, or cerebral venous sinus thrombosis. The condition predominantly affects young women and has traditionally been associated with obesity, headache, transient visual obscurations (TVO) pulsatile tinnitus, diplopia, and papilledema.[1]

If symptoms left untreated, sustained elevation of intracranial pressure may result in progressive visual field loss and permanent visual impairment. Reported atypical manifestations include IIH without papilledema (IIHWOP), unilateral or highly asymmetric papilledema, trigeminal neuralgia, facial palsy, olfactory dysfunction, auditory and vestibular symptoms, seizures, radiculopathy, and spontaneous skull base CSF leaks. [2]

Although headache remains the most frequently reported symptom, the clinical spectrum of IIH is broader than commonly recognized. [1] Some patients present with very mild manifestations, whereas others are identified only after optic disc swelling is detected during routine eye examinations. [3, 4] The true prevalence of these minimally symptomatic or apparently asymptomatic presentations remains uncertain because existing evidence is derived largely from small case series and isolated reports. [4] In children symptoms are sparse when compared to adults. [5, 6] Furthermore, patients initially considered symptom-free may later acknowledge subtle symptoms when questioned in greater detail.

Incidental identification of papilledema provides an important opportunity for early diagnosis. Routine visual assessments conducted for employment, educational purposes, or driving-license certification may therefore reveal previously unrecognized neurological disease. We report a young woman whose evaluation for driving fitness led to the diagnosis of definite IIH.

Case Presentation

A 23-year-old woman with no known medical comorbidities presented for ophthalmological assessment as part of the requirements for obtaining a driving license. She had no prior history of neurological illness and was not taking any regular medications.

During the visual assessment, bilateral optic disc swelling was noted on funduscopic examination. In view of this finding, she was referred for neurological evaluation.

Detailed history-taking revealed occasional headaches occurring over the preceding six months. The headaches were mild, infrequent, and had not interfered with daily activities. She also described intermittent right-sided tinnitus. Importantly, she denied TVO, visual blurring, double vision, photophobia, colour desaturation, or episodes of visual loss. There were no symptoms suggestive of cranial neuropathy, radiculopathy, or raised intracranial pressure. She had no fever, weight loss, systemic illness, or constitutional symptoms.

General physical examination was unremarkable. Neurological examination showed no focal deficits. Visual acuity was preserved, and ocular motility was normal. Fundus examination confirmed bilateral papilledema [Figure 1].

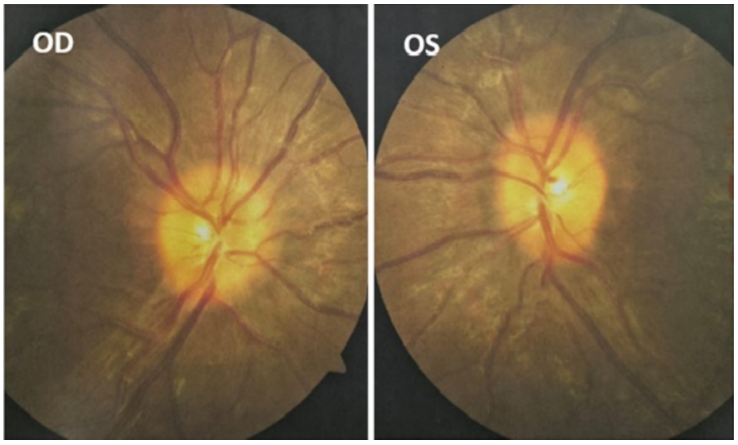

Figure 1. Showing bilateral grade 3 papilledema Frisen grading scale.

Given the presence of optic disc edema, neuroimaging was undertaken to exclude secondary causes of intracranial hypertension. Magnetic resonance imaging of the brain demonstrated bilateral tortuosity of the optic nerves along with flattening of the posterior globe on the left side [Figure 2]. These findings are recognized radiological markers associated with elevated intracranial pressure. Magnetic resonance venography showed normal venous sinuses without evidence of thrombosis or stenosis.

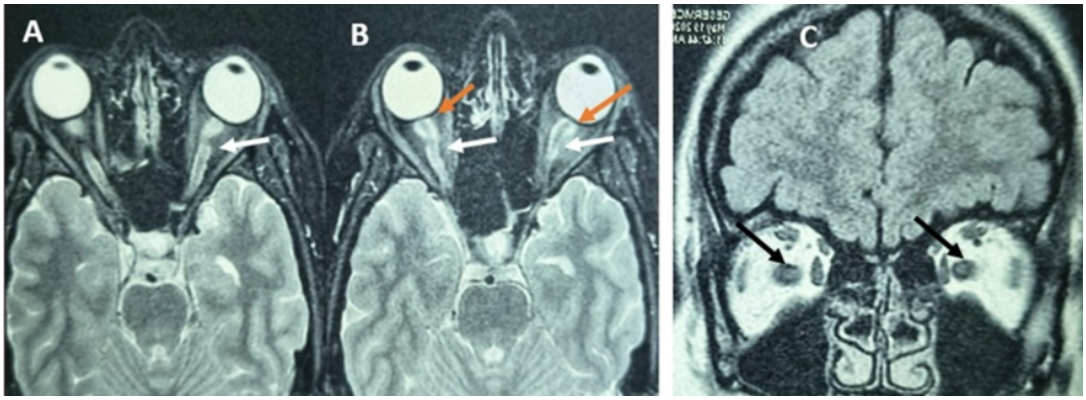

Figure 2. Showing optic nerve tortuosity (white arrow A, B), flattening of posterior globe of orbit (orange arrow in B), widening of optic nerve sheath (back arrow pointing in C figure)

Subsequent lumbar puncture revealed an opening cerebrospinal fluid pressure of 28 cm H_2O. Cerebrospinal fluid analysis demonstrated normal cell counts, protein concentration, and glucose levels. No evidence of infection, inflammation, or neoplastic involvement was identified.

Based on the clinical findings, characteristic imaging features, elevated opening pressure, and normal cerebrospinal fluid composition, the patient fulfilled the criteria for definite idiopathic intracranial hypertension. [7]

She was started on acetazolamide 250 mg twice daily and advised regarding weight monitoring and the need for regular ophthalmological surveillance. Follow-up with repeat fundus photography was scheduled after one month to assess treatment response and monitor for any progression of optic nerve changes.

Discussion

This case illustrates how routine screening performed for non-medical administrative purposes can unexpectedly uncover an important neurological diagnosis. The patient had only mild, nonspecific symptoms that had not prompted medical consultation. Detection of optic disc swelling during driving-license evaluation ultimately led to investigations confirming IIH.

The frequency of truly asymptomatic IIH remains difficult to determine. Many published studies define incidental papilledema as bilateral optic disc edema discovered during routine eye examinations, often in optometry or ophthalmology settings, followed by confirmation of IIH. However, careful clinical questioning frequently reveals subtle symptoms that patients had not previously considered significant. Consequently, distinguishing genuinely symptom-free individuals from those with overlooked manifestations can be challenging. A study by Mark Thaller et al [8] found that completely asymptomatic IIH was observed in 10.49% cases, and 35.27% cases had incidentally found papilledema in a prospective IIH cohort of 343 people. They also found that visual prognosis was same for asymptomatic and symptomatic cases. Asymptomatic IIH was also reported in post female to male gender transition where a routine optician visit found to have bilateral papilledema. [9] Another case of asymptomatic IIH was diagnosed after post nasal swab for covid test where patient developed spontaneous CSF rhinorrhoea and imaging showed features of IIH with skull base defect secondary to chronic IIH that caused CSF leaks. [10]

Our patient represents a minimally symptomatic presentation rather than a completely asymptomatic one. Her intermittent headaches and occasional tinnitus were mild and did not raise concern before ophthalmological assessment. The absence of visual symptoms is particularly noteworthy because visual complaints are often responsible for referral in patients with raised intracranial pressure.

The diagnosis of IIH requires exclusion of secondary causes of intracranial hypertension. Neuroimaging plays a critical role in this process. Several MRI features support the diagnosis, including optic nerve tortuosity, posterior globe flattening, enlarged perioptic subarachnoid spaces, and partially empty sella. Although none of these findings are individually diagnostic, their presence increases confidence in the diagnosis when combined with elevated cerebrospinal fluid pressure and compatible clinical findings.

The lumbar puncture findings in this patient were consistent with IIH. An opening pressure of 28 cm H_2O exceeded the accepted upper limit of normal, while routine cerebrospinal fluid parameters remained within normal ranges. Together with the absence of structural lesions or venous sinus thrombosis, these findings satisfied contemporary diagnostic criteria.

An important lesson from this case is that the severity of symptoms does not necessarily correlate with the degree of optic nerve involvement. Some individuals may develop significant papilledema despite minimal clinical complaints. Because visual field defects can evolve gradually, patients may remain unaware of progressive visual dysfunction until substantial damage has occurred. Early diagnosis therefore provides an opportunity to initiate therapy before irreversible visual loss develops.

Driving-license screening is not generally considered a neurological case-finding tool. Nevertheless, visual assessments performed during certification frequently involve examination of ocular structures and may reveal abnormalities requiring further investigation. Such encounters highlight the broader value of routine health evaluations and the importance of maintaining a high index of suspicion when optic disc edema is identified.

Management strategies for IIH focus on preserving vision and reducing intracranial pressure. Acetazolamide remains the first-line pharmacological therapy and has been shown to improve papilledema and visual outcomes in many patients. Close follow-up with serial ophthalmological examinations remains essential, particularly during the early stages of treatment.

Conclusion

Idiopathic intracranial hypertension may occasionally present with minimal symptoms and come to medical attention only after incidental discovery of papilledema. This case demonstrates how a routine driving-license evaluation led to the diagnosis of definite IIH in a young woman who had not sought medical care despite several months of mild symptoms. Recognition of optic disc swelling during screening examinations should prompt timely neurological assessment and appropriate investigations. Early identification and treatment are crucial to prevent visual deterioration and optimize long-term outcomes.

Conflicts of Interest

The authors declare no conflicts of interest.

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