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Opinion Article

Idiopathic Intracranial Hypertension and Vision: Understanding Papilledema and its Ocular Consequences

Dragan Petrovic*

Department of Neuro-Ophthalmology, University of Belgrade, Belgrade, Serbia

DESCRIPTION

Idiopathic Intracranial Hypertension (IIH), also known as pseudotumor cerebri, is a neurological disorder characterized by elevated intracranial pressure in the absence of an identifiable cause such as a brain tumor or venous thrombosis. It predominantly affects women of childbearing age with a higher body mass index, and its clinical manifestations often include headache, pulsatile tinnitus, transient visual obscurations, and papilledema. Among these, papilledema is the most concerning feature, as it directly threatens visual function and can lead to irreversible vision loss if not promptly recognized and managed.

Papilledema, defined as optic disc swelling due to increased intracranial pressure, is a hallmark of IIH. Bilateral disc edema typically presents with blurred disc margins, elevation of the optic nerve head, and obscuration of retinal vessels. The mechanism involves transmission of cerebrospinal fluid pressure through the optic nerve sheath, resulting in axoplasmic flow stasis and subsequent swelling. Fundoscopic examination remains the primary method for detecting papilledema, although newer imaging modalities have enhanced diagnostic accuracy and monitoring capabilities.

Optical Coherence Tomography (OCT) provides high-resolution cross-sectional imaging of the optic nerve head and peripapillary Retinal Nerve Fiber Layer (RNFL), allowing quantitative assessment of disc swelling. In acute papilledema, the RNFL thickness is markedly elevated, whereas chronic cases may show thinning due to optic atrophy. Serial OCT scans are invaluable in tracking disease progression and response to treatment. Additionally, visual field testing using automated perimetry helps detect early functional deficits, often manifesting as enlarged blind spots or peripheral constriction.

While papilledema is often asymptomatic in early stages, prolonged or severe swelling can lead to progressive visual field loss, decreased color vision, and central acuity decline. In some patients, optic nerve damage occurs despite minimal subjective symptoms, highlighting the importance of routine ophthalmologic evaluation in all suspected cases of IIH. Visual function may deteriorate silently over time, particularly in individuals with poor treatment adherence or recurrent disease flares.

The pathophysiology of IIH remains poorly understood, though several hypotheses implicate impaired cerebrospinal fluid absorption, hormonal factors, and metabolic disturbances. Obesity, especially in women of reproductive age, is the strongest risk factor, and weight loss is consistently shown to improve outcomes. Other contributing factors include use of certain medications such as tetracyclines and retinoids, sleep apnea, and polycystic ovary syndrome. Genetic predisposition may also play a role in select populations.

Management of IIH focuses on reducing intracranial pressure, alleviating symptoms, and preserving vision. First-line treatment typically involves weight reduction through diet and lifestyle modifications, with even modest weight loss resulting in measurable improvement in symptoms and papilledema. Pharmacologic therapy includes acetazolamide, a carbonic anhydrase inhibitor that reduces cerebrospinal fluid production. Topiramate, another agent with carbonic anhydrase inhibition and appetite-suppressing properties, is also used, though its cognitive side effects require monitoring.

In cases of acute or progressive vision loss, more aggressive interventions may be necessary. Therapeutic lumbar punctures provide temporary relief by directly reducing cerebrospinal fluid pressure, and can be repeated in selected cases. Surgical options such as optic nerve sheath fenestration and cerebrospinal fluid shunting (lumboperitoneal or ventriculoperitoneal) are considered for refractory cases with vision-threatening papilledema. Each procedure carries inherent risks and requires careful selection and post-operative follow-up.

Advances in neuroimaging have enhanced the diagnosis of IIH and exclusion of secondary causes. Magnetic Resonance Imaging (MRI) may reveal signs suggestive of elevated intracranial pressure, including an empty sella, posterior globe flattening, distension of the optic nerve sheath, and transverse sinus stenosis. Magnetic Resonance Venography (MRV) is essential to rule out cerebral venous sinus thrombosis, a condition that mimics IIH and requires anticoagulation.

Correspondence to: Dragan Petrovic, Department of Neuro-Ophthalmology, University of Belgrade, Belgrade, Serbia, E-mail: d.petrovic.no@ubg-rs.edu

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Monitoring of IIH extends beyond ophthalmic parameters to include symptom tracking and assessment of quality of life. Headache, the most common presenting complaint, often persists despite resolution of papilledema and may require a multidisciplinary approach involving neurology and pain management specialists. Psychological support and counseling are crucial for patients dealing with chronic illness, body image issues related to weight management, and fear of vision loss.