

Pediatric Idiopathic Intracranial Hypertension: Clinical Characteristics and the Impact of Obesity and Adolescence

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ABSTRACT

Pediatric idiopathic intracranial hypertension (IIH) is a rare but clinically important condition due to its heterogeneous presentation and risk of permanent visual loss. This study aimed to evaluate the clinical characteristics, neuroimaging findings, treatment approaches, and the influence of obesity and adolescence in pediatric IIH.

Pediatric patients aged 2–18 years with IIH followed at a single center were retrospectively reviewed. In accordance with the World Health Organization definition of adolescence, patients aged ≥ 10 years were classified as adolescents and those aged < 10 years as preadolescents.

Thirty-four patients were included, of whom 23 (67.6%) were female. The female-to-male ratio increased from 1.3:1 in the preadolescent group to 3:1 in the adolescent group. Obesity was significantly more common among adolescents than preadolescents (45.0% vs. 7.1%, $p=0.024$). Headache was the most frequent presenting symptom (79.4%), and papilledema was present in all patients. Neuroimaging findings suggestive of raised intracranial pressure were observed in 22 patients (64.7%). No significant differences were identified between groups regarding presenting symptoms, ophthalmologic findings, or neuroimaging characteristics (all $p>0.05$). All patients were managed conservatively. Median treatment duration was significantly longer in adolescents than in preadolescents (10 [6–12] vs. 6.5 [4–8] months, $p=0.001$).

Despite differences in obesity prevalence and treatment duration, the clinical and radiologic presentation of pediatric IIH remained largely consistent across age groups. These findings support maintaining a high index of suspicion for IIH in preadolescent children without obesity or other recognized risk factors.

Keywords: Idiopathic intracranial hypertension, Pediatrics, Obesity, Adolescence, Papilledema

Introduction

Pediatric idiopathic intracranial hypertension (IIH) is a rare neuro-ophthalmological condition of significant clinical importance due to its progressive course and risk of permanent visual loss (1). The reported annual incidence in children ranges from 0.5 to 0.9 per 100,000, with higher rates particularly observed among adolescent girls (2). IIH is a diagnosis of exclusion, characterized by increased cerebrospinal fluid (CSF) pressure in the absence of an underlying mass lesion, infection, hydrocephalus, or cerebral venous thrombosis (3–5). The clinical presentation most commonly includes headache and papilledema, and may be accompanied by reduced visual acuity, visual field defects, and diplopia secondary to sixth cranial nerve palsy (3,4,6).

While adult IIH typically exhibits a relatively homogeneous phenotype characterized by obesity

and female predominance, the clinical presentation in the pediatric population is markedly more heterogeneous (7). Although the effects of age, adolescent status, and obesity on symptom severity, diagnostic course, and visual prognosis have been reported, the clinical implications of phenotypic and risk profile differences between preadolescent and adolescent patients remain incompletely understood (3,5–7). In this context, evaluating pediatric IIH cases using data derived from routine clinical practice is important for improving understanding of age and adolescent related differences (7,8).

This study aimed to comprehensively evaluate demographic characteristics, clinical presentation, neuroimaging findings, treatment strategies, and the relationship with obesity in a rare cohort of pediatric IIH, with the goal of better understanding adolescent-related phenotypic

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variability based on a single-center experience at a tertiary referral center.

Materials and Methods

This single-center observational study retrospectively evaluated pediatric patients with IIH followed at the pediatric neurology clinic of Health Sciences University Van Training and Research Hospital between June 2023 and September 2025. Clinical and demographic data were obtained from patients' medical records and electronic databases. Patients aged 2–18 years who were diagnosed with IIH and had sufficient clinical, anthropometric, and diagnostic data available at the time of diagnosis were included. The diagnosis of pediatric IIH was established according to the revised diagnostic criteria for pseudotumor cerebri syndrome proposed by Friedman et al. (9). Diagnosis required the presence of papilledema, a normal neurological examination except for cranial nerve abnormalities, exclusion of secondary causes by neuroimaging, normal CSF composition, and elevated CSF opening pressure measured by lumbar puncture (≥ 28 cmH₂O in children, or ≥ 25 cmH₂O in non-obese, non-sedated children) (10). As all patients belonged to the pediatric age group, lumbar puncture was performed under sedation to ensure adequate cooperation and procedural safety. Therefore, a CSF opening pressure threshold of ≥ 28 cmH₂O was applied throughout the cohort. Patients with secondary causes of intracranial hypertension, including intracranial mass lesions, hydrocephalus, cerebral venous sinus thrombosis, infections, medication-related intracranial hypertension, and systemic diseases, were excluded. Because Tanner stage data were not consistently available in the retrospective records, adolescent status was defined using age-based criteria. In accordance with the World Health Organization (WHO) definition of adolescence, patients aged ≥ 10 years were classified as adolescents and those aged < 10 years as preadolescents (11,12). Anthropometric assessment was based on height and weight measurements recorded at the time of diagnosis. Body mass index (BMI) was calculated as body weight in kilograms divided by height in meters squared (kg/m²). In children younger than 5 years, obesity was defined according to WHO criteria as a weight-for-height Z-score $> +3$ SD (13). In children aged 5–19 years, obesity was defined according to WHO criteria as a BMI-for-age Z-score $> +2$ SD (14). Presenting symptoms,

including headache, blurred vision, diplopia, tinnitus, nausea, and vomiting, symptom duration, and neurological examination findings were recorded. Information regarding potential secondary causes and medication exposure, including tetracycline and vitamin A derivatives, was systematically obtained from the medical records. All patients underwent comprehensive ophthalmological evaluation, including assessment of visual acuity, papilledema severity according to the Frisén scale, and evaluation for associated cranial nerve involvement. Visual field examinations were performed using Humphrey automated perimetry in patients who were able to cooperate with the test, under the supervision of experienced ophthalmology personnel.

According to our institutional treatment protocol, acetazolamide was used as the first-line medical therapy and was initiated at a dose of 15–25 mg/kg/day, with dose adjustments made according to clinical response and tolerability (maximum dose: 3–4 g/day) (15). Topiramate was prescribed when clinically indicated and was initiated at 1.5–3 mg/kg/day in two divided doses, with gradual dose escalation when required (maximum dose: 200 mg/day) (1). Clinical and ophthalmological response was defined as resolution of papilledema, stabilization or improvement of visual field findings, and resolution of IIH-related symptoms. Recurrence was defined as the reappearance of papilledema and/or IIH-related symptoms during follow-up after cessation of treatment and initial clinical and ophthalmologic remission, necessitating reinitiation of medical treatment. This study was approved by the Medical Research Ethics Committee of Health Sciences University Van Training and Research Hospital (Approval No: 2025-09-35; Approval Date: November 7, 2025).

Statistical Analysis: Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as median (range), and categorical variables were presented as frequencies and percentages. Given the relatively small sample size, comparisons of continuous variables between preadolescent and adolescent patients were performed using the Mann–Whitney U test. Categorical variables were compared using Fisher's exact test. All statistical tests were two-sided, and a p-value < 0.05 was considered statistically significant. Exact p-values were reported to three decimal places. Given the retrospective nature of the study and the inclusion of all eligible patients diagnosed during the study

period, an a priori sample size calculation was not performed.

Results

A total of 34 patients were included in the study. The age at diagnosis ranged from 3.3 to 17.1 years. Fourteen patients were classified as preadolescents and 20 as adolescents. Female patients accounted for 67.6% of the cohort. The female-to-male ratio increased from 1.3:1 in the preadolescent group to 3:1 in the adolescent group, although the difference in sex distribution was not statistically significant ($p=0.458$). Obesity was significantly more common among adolescents than preadolescents (45.0% vs. 7.1%, $p=0.024$). Treatment duration was significantly longer in adolescents than in preadolescents (median 10 [6–12] vs. 6.5 [4–8] months, $p=0.001$) (Table 1).

Headache was the most common presenting symptom, occurring in 27 patients (79.4%), followed by visual symptoms in 14 patients (41.2%). Papilledema was present in all patients, whereas sixth cranial nerve palsy was observed in five patients (14.7%) and visual field defects in 14 patients (41.2%). Neuroimaging findings suggestive of raised intracranial pressure were identified in 22 patients (64.7%). Venous sinus stenosis was the most frequent neuroimaging abnormality (50.0%), followed by optic nerve sheath enlargement (32.4%) and partial empty sella (26.5%). No significant differences were identified between preadolescent and adolescent patients regarding presenting symptoms, ophthalmologic findings, or neuroimaging characteristics (all $p>0.05$) (Table 2).

All patients were managed conservatively. Acetazolamide monotherapy was the most commonly prescribed treatment (70.6%), followed by topiramate monotherapy (17.6%) and combination therapy (11.8%). Adverse effects related to acetazolamide were reported in 41.2% of patients and included paresthesia (23.5%), fatigue (17.6%), gastrointestinal symptoms (5.9%), nocturnal polyuria/enuresis (5.9%), and low-pressure headache (2.9%).

One patient receiving topiramate reported attention-related difficulties. No adverse effects related to acetazolamide or topiramate, including nephrolithiasis, electrolyte abnormalities, or elevated liver enzymes, were observed during follow-up. During follow-up, recurrence occurred in 5 patients (14.7%); four of these patients were obese, and one was overweight. Post-lumbar puncture headache was observed in five patients

(14.7%). No patient required surgical intervention, including ventriculoperitoneal shunting or venous sinus stenting.

Discussion

This single-center study highlights the heterogeneous clinical spectrum of pediatric idiopathic intracranial hypertension and indicates that obesity is more commonly associated with the disease, particularly in the adolescent period.

Female predominance in pediatric IIH became more evident in adolescents in this study, with the female-to-male ratio increasing from 1.3:1 in preadolescent patients to 3:1 in adolescents. This observation aligns with findings from a large pediatric meta-analysis by Sheldon et al. (7), which demonstrated a pubertal shift in sex distribution, with a markedly higher prevalence of idiopathic intracranial hypertension among adolescent females. Aylward et al. (16) reported a female-to-male ratio of 6:1 in the adolescents. In our study, sex distribution in pediatric IIH varied according to adolescent status, with a marked female predominance emerging during adolescence. The increasing sex disparity observed during adolescence suggests that preadolescent pediatric IIH may represent a clinical and etiopathogenetic phenotype distinct from adolescent and adult forms of the disease. In the context of existing literature, our findings suggest that changes in gonadal and growth hormone levels may contribute to the emergence of female predominance during adolescence.

Previous studies have reported conflicting findings regarding the association between pediatric IIH and obesity across age groups. While Tibussek et al. (10) observed obesity predominantly in adolescents, Bhalla et al. (17) demonstrated an association across the pediatric age spectrum. In a large multicenter study, Sheldon et al. (7) further identified a significant relationship between age at diagnosis and BMI standard deviation score. Similarly, obesity was significantly more common among adolescents than preadolescents in our cohort, supporting the concept that excess body weight becomes a more prominent risk factor during adolescence. In contrast, the low prevalence of obesity among younger children suggests that obesity-independent mechanisms may contribute more substantially to disease development in this age group. These findings support the multifactorial nature of pediatric IIH and indicate age-related variation in its risk-factor profile.

Table 1: Demographic and Baseline Clinical Characteristics of The Study Population

	Preadolescent (n = 14)	Adolescent (n = 20)	p value
Age, years, median (range)	6 (3–9)	15 (11–17)	
Sex			
Female, n (%)	8 (57.1)	15 (75.0)	0.458 ^a
Male, n (%)	6 (42.9)	5 (25.0)	
Treatment duration, months, median (range)	6.5 (4–8)	10 (6–12)	0.001 ^b
Obesity, n (%)	1 (7.1)	9 (45.0)	0.024 ^a

^a Fisher's exact test. ^b Mann–Whitney U test

Table 2: Clinical and Neuroimaging Characteristics of Preadolescent and Adolescent Patients With Pediatric Idiopathic Intracranial Hypertension

	Preadolescent (n=14)	Adolescent (n=20)	p value
Presenting symptoms, n (%)			
Headache	9 (64.3)	18 (90.0)	0.097
Any visual symptoms*	4 (28.6)	10 (50.0)	0.296
Vomiting	2 (14.3)	0	0.162
Dizziness	0	2 (10.0)	0.501
Ophthalmologic findings, n (%)			
Papilledema	14 (100)	20 (100)	-
Cranial nerve VI palsy	2 (14.3)	3 (15.0)	1.000
Visual field defect	4 (28.6)	10 (50.0)	0.296
Neuroimaging findings, n (%)			
Any sign of raised intracranial pressure	8 (57.1)	14 (70.0)	0.487
Venous sinus stenosis	5 (35.7)	12 (60.0)	0.186
Optic nerve sheath enlargement	4 (28.6)	7 (35.0)	1.000
Partial empty sella	2 (14.3)	7 (35.0)	0.250
Normal MRI/MRV	6 (42.9)	6 (30.0)	0.491

MRI: Magnetic resonance imaging; MRV: Magnetic resonance Venography

*Blurred vision and/or diplopia

Headache was the most frequent presenting symptom in our cohort, affecting 79.4% of patients, consistent with previous reports identifying headache as the predominant manifestation of pediatric IIH (5,18). Visual symptoms were present in 41.2% of patients, highlighting the clinical relevance of visual involvement in this population (5,19). Less frequent symptoms included vomiting and dizziness, reflecting the heterogeneous clinical presentation of pediatric IIH (20).

Papilledema was present in all patients in our cohort, underscoring its pivotal role in the

diagnosis of pediatric IIH. This finding is consistent with current diagnostic criteria, in which papilledema remains the principal clinical sign guiding diagnosis (9). Sixth cranial nerve palsy was observed in 14.7% of patients, a frequency within the range reported in previous pediatric series (21). These findings suggest that, although papilledema is the hallmark feature of pediatric IIH, cranial nerve VI involvement is variable and its absence does not exclude the diagnosis.

In our cohort, asymptomatic papilledema was identified during routine ophthalmologic

examination in three patients, leading to the diagnosis of IIH. Previous studies have similarly shown that asymptomatic presentation is not uncommon in younger children with IIH. In a cohort of 45 pediatric IIH patients, 31.1% were asymptomatic at diagnosis; compared with symptomatic patients, asymptomatic children were significantly younger and more frequently male (71.4% vs. 38.7%), with a lower prevalence of obesity (14.3% vs. 48.4%). The authors further suggested that asymptomatic IIH may represent either a milder clinical phenotype or a presymptomatic stage preceding the development of typical manifestations. (22). Notably, all three patients with incidentally detected papilledema in our cohort were male, younger than 10 years of age, and of normal weight. Although the small sample size precludes definitive conclusions, these observations suggest that younger, non-obese boys may present with a less overt clinical picture and emphasize the importance of careful funduscopic examination for the early recognition of IIH in children with subtle or absent symptoms.

In our cohort, all patients underwent brain magnetic resonance imaging (MRI) and MR venography, with neuroimaging findings suggestive of raised intracranial pressure identified in 22 patients (64.7%). Although the primary role of neuroimaging in pediatric IIH is to exclude secondary causes, imaging features related to elevated intracranial pressure may provide supportive diagnostic information (9). Importantly, neither the overall frequency nor the distribution of neuroimaging abnormalities differed significantly between preadolescent and adolescent patients, suggesting that radiologic manifestations of pediatric IIH remain largely consistent across age groups. Among the 22 patients with abnormal neuroimaging findings, venous sinus stenosis was the most frequent abnormality (77.3%), followed by optic nerve sheath enlargement (50.0%) and partial empty sella (40.9%), consistent with previous studies evaluating MRI findings associated with papilledema (23). None of the patients required venous sinus stenting during follow-up, and all were successfully managed with conservative treatment. The high prevalence of venous sinus stenosis suggests a close relationship between impaired venous outflow and intracranial hypertension in pediatric IIH. However, whether venous sinus stenosis represents a cause or a consequence of elevated intracranial pressure remains unclear, and further prospective studies

are needed to better define its role in disease pathophysiology and management (24). Normal MRI/MRV findings were observed in 35.3% of patients, indicating that the absence of neuroimaging abnormalities does not exclude pediatric IIH and highlighting the continued importance of clinical and ophthalmologic assessment in establishing the diagnosis.

Acetazolamide-related adverse effects were observed in 41.2% of patients in this study, including paresthesia, fatigue, gastrointestinal symptoms, nocturnal polyuria/enuresis, and low-pressure headache. This indicates that carbonic anhydrase inhibitor-related side effects may be clinically relevant in pediatric patients (1). Consistent with previous pediatric IIH studies, these adverse effects were dose-dependent, reversible, and rarely required treatment discontinuation (15). The occurrence of attention and concentration difficulties in one patient receiving topiramate highlights the need for careful monitoring of potential neuropsychiatric adverse effects in children (25). Overall, these findings underscore the importance of treatment tolerability and close surveillance of adverse effects, particularly during long-term therapy, in pediatric IIH.

Marked improvement in headache after treatment initiation in most symptomatic patients (81.5%) indicates that IIH can achieve favorable outcomes with early diagnosis and appropriate medical therapy (18). Post-lumbar puncture headache related to intracranial hypotension was the only procedure-related complication observed (15%), with no additional adverse events. This rate is comparable to those reported in prospective studies (17.5–32.2%), supporting the overall safety and transient nature of lumbar puncture-related complications in pediatric IIH (26–28).

In our cohort, recurrence occurred in 5 patients (14.7%) during follow-up, which is within the range of 5%–36% reported in previous pediatric IIH studies (29,30). All recurrent cases occurred in adolescent patients with excess body weight, including four patients with obesity and one who was overweight. Previous studies have identified obesity, adolescent status, weight gain during follow-up, poor treatment adherence, premature discontinuation of acetazolamide therapy, and severe or prolonged papilledema at diagnosis as potential risk factors for recurrence in pediatric IIH (6,31,32). Our findings are consistent with these observations and further support the potential contribution of excess body weight to recurrence risk, particularly in adolescent patients.

Previous studies have shown that a 6–10% reduction in body weight may lead to improvement in papilledema and IHH-related symptoms, while weight gain during follow-up has been associated with an increased risk of recurrence (29,33). In light of the established association between excess body weight and both disease occurrence and recurrence, weight management should be considered an integral component of long-term therapeutic strategies in patients with IHH.

This study has several limitations. First, its retrospective and single-center design may limit the generalizability of the findings. Second, patients were classified according to the World Health Organization age-based definition of adolescence rather than Tanner staging. Nevertheless, this approach is consistent with that used in several previous pediatric IHH studies and allowed standardized comparison between age groups. Despite these limitations, our findings contribute to the understanding of age-related differences in pediatric IHH by demonstrating variation in obesity prevalence, sex distribution, and treatment duration despite broadly similar clinical, ophthalmologic, and neuroimaging characteristics.

In conclusion, adolescents with pediatric IHH had a higher prevalence of obesity, a more pronounced female predominance, and longer treatment duration than preadolescent children. In contrast, the clinical presentation, ophthalmologic findings, and neuroimaging characteristics were largely similar between preadolescent and adolescent patients. Preadolescent patients may be non-obese and asymptomatic, with papilledema detected only during routine ophthalmologic examination. Therefore, IHH should be considered even in preadolescent children without obesity or other typical risk factors, and careful neuro-ophthalmologic evaluation should be performed.

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