JOUR UMRANIYE PEDIATR
2024;4(3):108–113 doi: 10.14744/upd.2025.18199

ORIGINAL RESEARCH
Pediatric Emergency

Idiopathic intracranial hypertension in children: Etiological features and treatment modalities

Şeyma Sönmez Şahin,[1] Elif Yüksel Karatoprak,[2] Dilara Füsun İçağasıoğlu[3]

[1]Department of Pediatrics, Göztepe Prof. Dr. Süleyman Yalçın City Hospital, İstanbul, Türkiye
[2]Department of Pediatric Neurology, İstanbul Medeniyet University Faculty of Medicine, İstanbul, Türkiye
[3]Department of Pediatric Neurology, Medipol University Faculty of Medicine, İstanbul, Türkiye

ABSTRACT

Objective: Idiopathic intracranial hypertension (IIH) is characterized by increased intracranial pressure without mass lesions, structural abnormalities, or hydrocephalus. Its pathogenesis remains unclear, but inflammatory factors, natriuretic peptides, and aquaporins are believed to play a role. This study aimed to assess the etiology, clinical features, and treatment responses of pediatric patients with IIH.

Material and Methods: This retrospective study reviewed the records of 43 pediatric patients diagnosed with IIH between 2018 and 2023 at two hospitals. Diagnosis was based on modified Dandy criteria and pediatric pseudotumor cerebri criteria from Rangwala. Patients underwent neurological and ophthalmological evaluations, including optic disc assessment, MRI, and CSF analysis. Treatment was initiated with acetazolamide, with some patients receiving topiramate or steroids if necessary. Statistical analysis was performed using descriptive methods.

Results: The cohort consisted of 28 females (65%) and 15 males (35%), with a mean age of 11.6±3.4 years. The most common presenting symptoms were headache (56%) and blurred vision (28%). Optic disc edema was present in all patients, and visual field defects were observed in 16%. The average BMI was 26.3±7 kg/m², with 56% of patients having a BMI over 25 kg/m². The majority (53%) had no identifiable cause. Acetazolamide was used as the first-line treatment, with topiramate added for patients who did not respond. Papilledema resolved in 86% of patients within six months. Four patients (9%) required permanent shunt surgery.

Conclusion: IIH in children is more common in post-pubertal females and is often associated with obesity. Headache and visual disturbances are the most common symptoms. Acetazolamide was effective for most patients, with topiramate used for treatment-resistant cases. Surgery was required for patients with severe symptoms or inadequate response to medication. Early diagnosis and appropriate treatment are essential to prevent vision loss.

Keywords: Idiopathic intracranial hypertension; papilledema; pediatric; pseudotumor cerebri.

Cite this article as: Sönmez Şahin Ş, Yüksel Karatoprak E, İçağasıoğlu DF. Idiopathic intracranial hypertension in children: Etiological features and treatment modalities. Jour Umraniye Pediatr 2024;4(3):108–113.

Received (Başvuru): 18.11.2024 **Revised (Revizyon):** 06.01.2025 **Accepted (Kabul):** 07.01.2025 **Online (Online yayınlanma):** 26.02.2025

Correspondence (İletişim): Dr. Şeyma Sönmez Şahin. Göztepe Prof. Dr. Süleyman Yalçın Şehir Hastanesi, Çocuk Sağlığı ve Hastalıkları Kliniği, İstanbul, Türkiye.
Phone (Tel): +90 216 606 52 00 **e-mail (e-posta):** sonmezseyma@hotmail.com

İdyopatik intrakraniyal hipertansiyon tanılı pediatrik hastalarda etyolojik değerlendirme ve tedavi yaklaşımları

ÖZET

Amaç: İdyopatik intrakraniyal hipertansiyon (IIH), kitle lezyonları, yapısal anormallikler veya hidrosefali olmaksızın artmış intrakraniyal basınçla karakterizedir. Patogenezi tam olarak aydınlatılamamış olmakla birlikte, inflamatuar faktörler, natriüretik peptitler ve aquaporinlerin rol oynadığı düşünülmektedir. Bu çalışmanın amacı, IIH'li pediatrik hastaların etiyolojisini, klinik özelliklerini ve tedavi yanıtlarını değerlendirmektir.

Gereç ve Yöntemler: Bu retrospektif çalışmada, 2018 ile 2023 yılları arasında iki hastanede IIH tanısı konulan 43 pediatrik hastanın kayıtları incelendi. Tanı, modifiye Dandy kriterleri ve Rangwala'nın pediatrik psödotümör serebri kriterlerine göre konuldu. Hastalar nörolojik ve oftalmolojik değerlendirmelere tabi tutuldu, bu kapsamda optik disk değerlendirmesi, MRI ve BOS analizi yapıldı. Tüm hastalarda tedaviye asetazolamid ile başlandı, yan etki gelişen veya tedaviye yanıtsız olan hastaların tedavisine topiramat ve/veya steroid eklendi. İstatistiksel analizler tanımlayıcı yöntemler kullanılarak yapıldı.

Bulgular: Çalışmaya 28 kadın (%65) ve 15 erkek (%35) hasta dahil edildi ve ortalama yaşları 11,6±3,4 yıl olarak belirlendi. En sık görülen semptomlar baş ağrısı (%56) ve bulanık görme (%28) idi. Tüm hastalarda optik disk ödemi mevcut olup, %16'sında görme alanı kusurları gözlendi. Ortalama BMI 26,3±7 kg/m² saptandı ve hastaların %56'sının BMI'si 25 kg/m²'nin üzerindeydi. Hastaların %53'ünde etyolojik neden saptanamadı. Asetazolamid birinci basamak tedavi olarak kullanıldı ve yanıt vermeyen hastalara topiramat eklendi. Hastaların %86'sında papilödem altı ay içinde geriledi. Dört hasta (%9) kalıcı şant ameliyatına ihtiyaç duydu.

Tartışma: İdyopatik intrakraniyal hipertansiyon, çocuklarda görme kaybının önemli bir nedenidir. Baş ağrısı gibi semptomları hafifletmek ve görme kaybını önlemek için erken tanı ve tedavi büyük önem taşımaktadır. Ergenlik çağındaki kızlarda erkeklere kıyasla daha yüksek bir yaygınlık göstermektedir. Çok faktörlü etiyoloji göz önüne alındığında, kapsamlı bir tanısal değerlendirme esastır. Asetazolamid birincil tedavi yöntemi olmaya devam ederken, topiramatın eklenmesi cerrahi müdahale ihtiyacını azaltabilir. Genel olarak, IIH tıbbi tedaviye olumlu yanıt vermektedir; ancak, tedaviye dirençli vakalarda görme kaybını önlemek için zamanında cerrahi müdahale kritik öneme sahiptir.

Anahtar Kelimeler: Papil ödem; pediatrik; psödotümör serebri; idyopatik intrakraniyal hipertansiyon.

ORCID ID

ŞSŞ: 0000-0002-2648-3683; EYK: 0000-0003-2515-1764; DFİ: 0000-0002-7728-2611

[1]Göztepe Prof. Dr. Süleyman Yalçın Şehir Hastanesi, Çocuk Sağlığı Ve Hastalıkları Anabilim Dalı, İstanbul, Türkiye
[2]İstanbul Medeniyet Üniversitesi Tıp Fakültesi, Çocuk Nörolojisi Kliniği, İstanbul, Türkiye
[3]Medipol Üniversitesi Tıp Fakültesi, Çocuk Nörolojisi Kliniği, İstanbul, Türkiye

INTRODUCTION

Idiopathic intracranial hypertension (IIH) is characterized by symptoms and signs of elevated intracranial pressure in the absence of mass lesions, structural abnormalities, hydrocephalus, or meningeal enhancement on neuroimaging, along with elevated intracranial pressure and a normal cerebrospinal fluid (CSF) composition. Diagnosis typically relies on the modified Dandy criteria (1).

Common presenting symptoms include headache, blurred vision, diplopia, vomiting, dizziness, and pulsatile tinnitus (2). Notably, approximately 29% of children with IIH remain asymptomatic (3). The pathogenesis of IIH is not yet fully elucidated; however, inflammatory factors, natriuretic peptides, and aquaporins are believed to play a role (4).

The incidence of IIH is reported to be 0.63 per 100,000 children (5). It occurs equally among boys and girls under the age of 12, but its prevalence increases in females post-puberty (6). Despite its relatively low frequency, accurate diagnosis and appropriate treatment are crucial to preventing permanent vision loss (7).

Treatment modalities include weight loss, pharmacotherapy, and surgical interventions (6). Acetazolamide and topiramate are frequently utilized in medical management. In cases unresponsive to medical treatment, surgical options such as ventriculoperitoneal shunt, lumboperitoneal shunt, or optic nerve sheath fenestration may be considered (8). Remission is defined as the absence of headache and improvement in optic nerve function during a six-month treatment-free interval (9).

This study aims to elucidate the etiology and clinical characteristics of pediatric patients diagnosed with idiopathic intracranial hypertension and to assess their treatment responses.

MATERIAL AND METHODS

This retrospective study examined the records of patients diagnosed with idiopathic intracranial hypertension (IIH) who were followed for at least one year at the child neurology outpatient clinics of Göztepe Prof. Dr. Süleyman Yalçın City Hospital and Bezmialem Foundation University Hospital. A total of 43 patients were included. The diagnosis of pediatric IIH was made using the modified Dandy criteria and Pediatric Pseudotumor Cerebri diagnostic criteria adapted from Rangwala (1, 10) (Table 1).

Table 1. Modified Dandy criteria adapted by Friedman

Modified Dandy criteria for Pseudotumor Cerebri
1. Signs and symptoms of increased intracranial pressure
2. No localizing findings on neurological examination
3. Normal MRI/CT without central venous sinus thrombosis
4. Cerebrospinal fluid(CSF) pressure is 250 cm H_2O and above with normal CSF composition [≥250 mm CSF in adults and ≥280 mm CSF in children (250 mm CSF if the child is not sedated and obese)]
5. No identifiable cause of intracranial hypertension, including medications

MRI: Magnetic resonance imaging; CT: Computed Tomography; CSF: Cerebrospinal fluid.

Patient files were reviewed for medical history, physical examination findings, laboratory investigations, radiological assessments, and treatments. All patients underwent neuro-ophthalmological evaluations. The severity of optic disc edema was assessed using the Frisen scale, where stage 1 represents the mildest form and stage 5 the most severe (11). Visual acuity, visual field examination, optical coherence tomography, color vision tests, and fundus photography were conducted based on patient compliance.

Laboratory tests, including complete blood count, liver and kidney function tests, electrolyte levels, thyroid function tests, erythrocyte sedimentation rate, vitamin D and B12 levels, parathormone, and cortisol levels, were performed to rule out underlying conditions. Lumbar puncture was conducted in the lateral decubitus position to measure opening CSF pressure, and CSF analysis was performed for microbiological and biochemical parameters.

Childhood obesity is defined as a body mass index (BMI) at or above the 95th percentile for age and sex in children (12). Brain MRI and MR venography were performed on all patients, with those diagnosed with sinus thrombosis excluded from the study. Female patients underwent ovarian ultrasound to assess for polycystic ovary syndrome (PCOS), and patients were screened for sleep apnea syndrome and medication use.

Patients were monitored weekly for the first month, monthly for three months, and every three months thereafter. Acetazolamide and/or topiramate were utilized as first-line treatment options.

This study was approved by the Medipol University Ethics Committee (2024-675) and was conducted in accordance with the Declaration of Helsinki Principles.

Statistical Analysis

Statistical analyses were performed using the NCSS (Number Cruncher Statistical System) 2007 Statistical Software (Utah, USA). Descriptive statistical methods (mean, standard deviation, median, interquartile range) were applied to evaluate the data.

RESULTS

Among the 43 patients included in the study, 28 (65%) were female and 15 (35%) were male. The age range was 4 to 17 years, with a mean age of 11.6±3.4 years. The ratio of girls to boys aged 12 and under was 13:8, whereas for those over 12 years of age, it was 16:6. Clinical features are detailed in Table 2.

Headache was the most common presenting complaint, reported by 24 patients (56%), followed by blurred vision in 12 patients (28%), vomiting in 6 patients (14%), and diplopia in 6 patients (14%). Fourteen patients presented with multiple complaints. Neurological examinations revealed sixth cranial nerve palsy in 14% of patients, while papilledema was noted in all patients. Visual field defects were identified in 7 patients, with one patient exhibiting decreased visual acuity and defects in all quadrants of the visual field. Normal visual evoked potential tests were recorded for this patient, whose symptoms resolved following IIH treatment.

The average BMI of the patients was 26.3±7 kg/m² (range: 15–53 kg/m²), with 24 patients (56%) having a BMI over 25 kg/m² and 8 patients (19%) having a BMI of 30 kg/m² or higher.

Papilledema staging for the right eye was as follows: stage 1 (n=4), stage 2 (n=16), stage 3 (n=16), stage 4 (n=7). For the left eye: stage 1 (n=6), stage 2 (n=15), stage 3 (n=15), stage 4 (n=7).

CSF examination results were normal for biochemical and microbiological parameters in all patients. The average opening pressure was measured at 405±153 mmH_2O (range: 250–760 mmH_2O). Brain MRI findings are summarized in Table 2.

Etiological assessments are outlined in Table 3, with no identifiable cause found in 23 patients (53%).

Acetazolamide treatment was initiated for all patients at a dosage of 15–25 mg/kg/day. Two patients who could not tolerate acetazolamide were switched to topiramate, while in 7 patients with insufficient symptom improvement, topiramate was added to the regimen. Steroids were incorporated into the treatment of one patient.

Within the first six months, papilledema completely regressed in 86% of patients. Temporary lumbar drainage was performed four times in two patients with CSF pressures of 730 and 750 mmH_2O, respectively. Despite these interventions and ongoing medical treatment, four patients (9%) ultimately required a permanent shunt. Permanent visual field loss was noted in two patients despite undergoing ventriculoperitoneal shunt placement. No recurrences were observed during the 12-month follow-up period.

One patient reported changes in processing speed and reaction time during follow-up. Complications following lumbar puncture occurred in one patient, who developed intracranial bleeding due to thrombosis, despite having normal MRI venography and no family history of coagulopathy.

Table 2. Clinical characteristics [n=43(%100)]

Characteristics	n (%)
Median age (years)	11.6±3.4
Female/male ratio	28/15
Presenting symptoms	
Asymptomatic	9 (21)
Headache	24 (56)
Blurred vision	12 (28)
Double vision	6 (14)
Vomiting	6 (14)
Dizziness	1 (2)
Back pain	1 (2)
Initial findings	
Papilledema	43 (100)
Cranial nerve VI palsy	6 (14)
Visual field defect	7 (16)
Papilledema Stage	
Right eye	
Stage 1	4 (9)
Stage 2	16 (37)
Stage 3	16 (37)
Stage 4	7 (16)
Left eye	
Stage 1	6 (14)
Stage 2	15 (35)
Stage 3	15 (35)
Stage 4	7 (16)
MRI/MRV findings	
Optic nerve sheat distention	13 (30)
Posteroscleral flattening	4 (9)
Partial empty sella	1 (2)
Transvers sinüs compressing	9 (20)
Median opening pressure (mm H_2O)	405±153 (250–760)
Median BMI (kg/m^2)	26.3±7 (15–53)

MRI: Magnetic resonance imaging; MRV: Magnetic Resonance Venography; BMI: Body mass index.

Table 3. Etiological causes (n=43)

Etiology	n (%)
Obesity	17 (40)
Iron deficiency	4 (9)
Vitamin D deficiency	1 (2)
Polycystic ovary syndrome	1 (2)
No identifiable cause	23 (53)

DISCUSSION

Idiopathic intracranial hypertension is marked by signs and symptoms indicative of elevated intracranial pressure without space-occupying lesions (13). It is often associated with obesity and puberty in children. Previous studies report that 60% of IIH patients are over 10 years of age (14). In our study, the distribution of patients aged 12 and under compared to those over 12 was nearly equal (22/21). While previous research indicated average ages of 10.79 years Per et al. (15) and 10 years Hacıfazlıoğlu et al. (16), our cohort had a mean age of 11.6 years.

The female gender is a recognized risk factor for IIH in adults; however, in children, the distribution varies based on age and risk factors (17). It has been documented that IIH occurs at similar rates in boys and girls pre-puberty, but shows a higher prevalence in females post-puberty (18). A meta-analysis of childhood-onset IIH reported equal prevalence among prepubertal children, with increased occurrence in girls aged over 11 years (19). Our findings corroborate this trend, with a greater proportion of females in both age categories. The ratio of girls to boys aged 12 and under was 1.6, increasing to 2.7 for those over 12 years of age, which aligns with existing literature (19–21).

In our study, headache was the predominant complaint, affecting 56% of patients, consistent with other research (2, 16, 22). Visual impairment, reported by 28% of patients, aligns with findings from Moavero et al. (2). While older children often present with headache, younger patients may exhibit symptoms such as lethargy and drowsiness (23). Among our cohort, five patients aged 6 years and under were observed; three were asymptomatic, while two presented with blurred vision. The prevalence of asymptomatic patients in the literature ranges from 10% to 31%, with our study reporting 16% (3, 15, 24).

Papilledema was present in all patients (100%), with sixth cranial nerve palsy noted as the second most common sign (14%). Serin et al. (22) reported similar findings, where papilledema was the most frequently observed neurological sign, followed by sixth cranial nerve paralysis. In the study by Per et al. (15), papilledema was recorded in all patients, while sixth cranial nerve paralysis occurred in 19%.

Conditions associated with IIH include various medications, systemic diseases, and metabolic/endocrine disorders. Relevant medications include tetracyclines, oral contraceptives, hypervitaminosis A, nalidixic acid, and corticosteroid use and withdrawal. Systemic conditions linked to IIH encompass iron deficiency anemia, leukemia, and Behçet's disease, while metabolic/endocrine disorders include hypothyroidism, hyperthyroidism, adrenal insufficiency, and obesity (25). Our study identified obesity, iron deficiency, vitamin D deficiency, and PCOS as associated conditions.

Agraz et al. (26) reported an obesity rate of 50%, significantly higher than the national average in the United States. In Türkiye, the Ministry of Health reported a childhood obesity prevalence

of 9.9%. In our study, the obesity prevalence was notably higher at 40%. In Tovia et al.'s (20) study, 48% of patients had a BMI over 25 kg/m², while PCOS was observed in 3.3% of patients. In our cohort, iron deficiency was the second most common etiology (9%), with one patient diagnosed with PCOS (2%) and another with vitamin D deficiency (2%). No medications that could induce IIH were identified in the patient population.

MRI findings in IIH may include optic nerve head protrusion, optic nerve tortuosity, posteroscleral flattening, increased perioptic CSF, and transverse sinus stenosis (27). Hirfanoglu et al. (28) reported optic nerve tortuosity and enlargement as the most prevalent MRI findings. In our study, optic nerve sheath enlargement was the most common finding, observed in 13 patients (30%), followed by transverse sinus stenosis in 9 patients (20%). Transverse sinus stenosis has demonstrated high sensitivity (74%) and specificity (100%) for IIH (29). Hartmann et al. (27) compared MRI findings across prepubertal, adolescent, and adult patients, noting significantly lower occurrences of posteroscleral flattening and transverse sinus stenosis in prepubertal children. Although puberty was not classified in our analysis, we found increased MRI and MR venography findings in patients over 12 years of age. Specifically, optic nerve sheath prominence was seen in 3 (14%) of the 22 patients aged 12 and under, compared to 10 (48%) of the 21 patients aged over 12.

The primary objective of IIH treatment is to prevent vision loss and alleviate symptoms by reducing CSF pressure. Both medical and surgical methods are employed in the management of this condition. Acetazolamide is typically the first-line treatment, functioning by inhibiting carbonic anhydrase to reduce CSF production (30). In our study, all patients (100%) initiated treatment with acetazolamide. Common side effects include metallic taste, paresthesia, transient anorexia, and metabolic acidosis (31). Two patients discontinued acetazolamide due to paresthesia and were transitioned to topiramate. Of the remaining patients, acetazolamide treatment continued in 41 (95%), with 34 (79%) demonstrating clinical improvement. Similar results were reported by Tovia et al. (20), with 76% of patients benefiting from acetazolamide, while Per et al. (15) found this rate to be 38%. Celebisoy et al. (32) noted that the effectiveness of topiramate was equivalent to acetazolamide. In our study, topiramate was added to the treatment regimen of 7 patients who did not respond to acetazolamide. Topiramate was employed in 9 patients (21%) and proved effective in 4. Tovia et al. (20) indicated that patients unresponsive to acetazolamide underwent surgical intervention without utilizing topiramate. In contrast, Per et al. (15) reported improvement with topiramate in 7 of 10 patients unresponsive to repeated lumbar drainage.

Although steroids were previously employed as a first-line treatment for IIH, they are now reserved for fulminant cases due to their side effects (29). One study indicated no clinical response when steroids were administered as the first treatment for two patients (15). In our cohort, steroids were added to the regimen of one patient who did not respond to acetazolamide, leading to clinical improvement.

Research in adults has shown that a weight loss of 6% of total body weight can lead to regression of papilledema (33). All patients with a BMI exceeding 25 were evaluated by a dietitian, with clinical improvement observed in 5 patients (12%) following medication and weight loss interventions.

Ventriculoperitoneal shunts were placed in four patients unresponsive to treatment. Recent studies suggest that temporary lumbar drainage can serve as a bridge to avoid permanent shunting while waiting for medical treatment to take effect in cases of acute-onset, rapidly progressive IIH (29). In our study, two patients with BMIs over 30 underwent temporary lumbar drainage; however, shunt placement was delayed due to family reluctance. Consequently, permanent visual field loss was noted in these two patients. Two patients who received direct shunt placement following treatment failure benefited from the procedure.

All patients were followed for a duration of 12 months, during which no recurrences were noted. Hilely et al. (9) reported recurrence rates of 23% in prepubertal patients and 50% in adolescents, emphasizing that prolonged treatment duration correlates with decreased recurrence.

One patient exhibited intracranial bleeding secondary to venous thrombosis following lumbar puncture, which was evaluated due to persistent headache. This highlights the necessity for caution regarding complications associated with persistent headaches following lumbar puncture. Venous hypotension that develops post-lumbar puncture may lead to venous dilation and thrombosis (34).

CONCLUSION

Idiopathic intracranial hypertension represents a significant cause of vision loss in children, underscoring the importance of early diagnosis and treatment in preserving visual function and alleviating symptoms such as headache. The condition exhibits a higher prevalence in adolescent females compared to males. Given the multifactorial etiology, a comprehensive diagnostic evaluation is warranted. While acetazolamide remains the primary treatment modality, the incorporation of topiramate can mitigate the need for surgical intervention. Overall, IIH demonstrates a favorable response to medical management; however, timely surgical intervention is crucial to prevent vision loss in treatment-resistant cases.

Ethics Committee Approval: The Medipol University Clinical Research Ethics Committee granted approval for this study (date: 04.07.2024, number: 675).

Authorship Contributions: Concept – ŞSŞ, EYK; Design – ŞSŞ, DFİ; Supervision – EYK, DFİ; Fundings – ŞSŞ; Data collection and/or Processing – ŞSŞ; Analysis and/or Interpretation –ŞSŞ, EYK; Literature Search – ŞSŞ; Writing – ŞSŞ, EYK; Critical Review – EYK, DFİ.

Conflict of Interest: No conflict of interest was declared by the authors.

Use of AI for Writing Assistance: Not declared.

Informed Consent: Written informed consent was obtained from the families of the patients who participated in this study.

Financial Disclosure: The authors declared that this study has received no financial support.

Etik Kurul Onayı: Medipol Üniversitesi Klinik Araştırmalar Etik Kurulu'ndan bu çalışma için onay alınmıştır (tarih: 04.07.2024, sayı: 675)

Yazarlık Katkıları: Fikir – ŞSŞ, EYK; Tasarım – ŞSŞ, DFİ; Denetleme – EYK, DFİ; Kaynaklar – ŞSŞ; Veri Toplanması ve/veya İşlemesi – ŞSŞ; Analiz ve/veya Yorum – ŞSŞ, EYK; Literatür Taraması – ŞSŞ; Yazıyı Yazan – ŞSŞ, EYK; Eleştirel İnceleme – EYK, DFİ.

Çıkar Çatışması: Yazarlar çıkar çatışması bildirmemişlerdir.

Yazma Yardımı için Yapay Zeka Kullanımı: Beyan edilmedi.

Hasta Onamı: Yazılı hasta onamı bu çalışmaya katılan hastaların ailelerinden alınmıştır.

Mali Destek: Yazarlar bu çalışma için mali destek almadıklarını beyan etmişlerdir.

REFERENCES

1. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology 2013;81:1159–65.
2. Moavero R, Sforza G, Papetti L, Battan B, Tarantino S, Vigevano F, et al. Clinical features of pediatric idiopathic intracranial hypertension and applicability of new ICHD-3 criteria. Front Neurol 2018;9:819.
3. Bassan H, Berkner L, Stolovitch C, Kesler A. Asymptomatic idiopathic intracranial hypertension in children. Acta Neurol Scand 2008;118:251–5.
4. Baykan B, Ekizoğlu E, Altıokka Uzun G. An update on the pathophysiology of idiopathic intracranial hypertension alias pseudotumor cerebri. Agri 2015;27:63–72.
5. Gillson N, Jones C, Reem RE, Rogers DL, Zumberge N, Aylward SC. Incidence and demographics of pediatric intracranial hypertension. Pediatr Neurol 2017;73:42–7.
6. Barmherzig R, Szperka CL. Pseudotumor cerebri syndrome in children. Curr Pain Headache Rep 2019;23:58.
7. Ball AK, Clarke CE. Idiopathic intracranial hypertension. Lancet Neurol 2006;5:433–42.
8. Akçakaya NH, Akçakaya MO, Sencer A, Yapıcı Z. Idiopathic intracranial hypertension: Diagnosis and therapeutic approach. Turk J Neurol 2017;23:43–50.
9. Hilely A, Hecht I, Goldenberg-Cohen N, Leiba H. Long-term follow-up of pseudotumor cerebri syndrome in prepubertal children, adolescents, and adults. Pediatr Neurol 2019;101:57–63.
10. Rangwala LM, Liu GT. Pediatric idiopathic intracranial hypertension. Surv Ophthalmol 2007;52:597–617.
11. Frisén L. Swelling of the optic nerve head: A staging scheme. J Neurol Neurosurg Psychiatry 1982;45:13–8.
12. Neyzi O, Günöz H, Furman A, Bundak R, Gökçay G, Darendeliler F, et al. Türk çocuklarında vücut ağırlığı, boy uzunluğu, baş çevresi ve vücut kitle indeksi referans değerleri. Çocuk Sağ Hast Derg [Article in Turkish] 2008;51:1–14.
13. Vitaliti G, Pavone P, Matin N, Tabatabaie O, Cocuzza S, Vecchio M, et al. Therapeutic approaches to pediatric pseudotumor cerebri: New insights from literature data. Int J Immunopathol Pharmacol 2017;30:94–7.
14. Babikian P, Corbett J, Bell W. Idiopathic intracranial hypertension in children: The Iowa experience. J Child Neurol 1994;9:144–9.
15. Per H, Canpolat M, Gümüş H, Poyrazoğlu HG, Yıkılmaz A, Karaküçük S, et al. Clinical spectrum of the pseudotumor cerebri in children: Etiological, clinical features, treatment and prognosis. Brain Dev 2013;35:561–8.
16. Hacifazlioglu Eldes N, Yilmaz Y. Pseudotumour cerebri in children: Etiological, clinical features and treatment modalities. Eur J Paediatr Neurol 2012;16:349–55.
17. Balcer LJ, Liu GT, Forman S, Pun K, Volpe NJ, Galetta SL, et al. Idiopathic intracranial hypertension: Relation of age and obesity in children. Neurology 1999;52:870–2.
18. Değerliyurt A, Teber S, Karakaya G, Güven A, Şeker ED, Arhan EP, et al. Pseudotumor cerebri/idiopathic intracranial hypertension in children: An experience of a tertiary care hospital. Brain Dev 2014;36:690–9.
19. Genizi J, Lahat E, Zelnik N, Mahajnah M, Ravid S, Shahar E. Childhood-onset idiopathic intracranial hypertension: Relation of sex and obesity. Pediatr Neurol 2007;36:247–9.
20. Tovia E, Reif S, Oren A, Mitelpunkt A, Fattal-Valevski A. Treatment response in pediatric patients with pseudotumor cerebri syndrome. J Neuroophthalmol 2017;37:393–97.
21. Bektaş G, Yıldız EP, Aksu Uzunhan T, Aydınlı N, Çalışkan M, Özmen M. Primary and secondary pseudotumor cerebri syndrome in childhood: Symptoms and imaging findings. İst Tıp Fak Derg 2017;80:146–52.
22. Serin H, Şimşek E, Kanmaz S, Biler E, Üretmen Ö, Yılmaz S, et al. Çocuklarda intrakraniyal hipertansiyon: Etiyolojik, klinik özellikler, tedavi ve prognoz. Turkiye Klinikleri Pediatri Derg [Article in Turkish] 2018;27:89–95.
23. Matthews YY. Drugs used in childhood idiopathic or benign intracranial hypertension. Arch Dis Child Educ Pract Ed 2008;93:19–25.
24. Kesler A, Fattal-Valevski A. Idiopathic intracranial hypertension in the pediatric population. J Child Neurol 2002;17:745–8.
25. Wainwright MS. Disorders of intracranial pressure. In: Swaiman K, Schor N, Ferriero D, Ashwal S, eds. Pediatric Neurology: Principles and Practice. Elsevier; 2011.
26. Agraz D, Morgan LA, Fouzdar Jain S, Suh DW. Clinical features of pediatric idiopathic intracranial hypertension. Clin Ophthalmol 2019;13:881–6.
27. Hartmann AJ, Soares BP, Bruce BB, Saindane AM, Newman NJ, Biousse V, et al. Imaging features of idiopathic intracranial hypertension in children. J Child Neurol 2017;32:120–6.
28. Hirfanoglu T, Aydin K, Serdaroglu A, Havali C. Novel magnetic resonance imaging findings in children with intracranial hypertension. Pediatr Neurol 2015;53:151–6.
29. Varma H, Aylward SC. Pediatric intracranial hypertension: A review of presenting symptoms, quality of life, and secondary causes. Ann Child Neurol Soc 2024;2:15–26.
30. Distelmaier F, Sengler U, Messing-Juenger M, Assmann B, Mayatepek E, Rosenbaum T. Pseudotumor cerebri as an important differential diagnosis of papilledema in children. Brain Dev 2006;28:190–5.
31. Ten Hove MW, Friedman DI, Patel AD, Irrcher I, Wall M, McDermott MP; NORDIC Idiopathic Intracranial Hypertension Study Group. Safety and tolerability of acetazolamide in the idiopathic intracranial hypertension treatment trial. J Neuroophthalmol 2016;36:13–9.
32. Celebisoy N, Gökçay F, Sirin H, Akyürekli O. Treatment of idiopathic intracranial hypertension: Topiramate vs acetazolamide, an open-label study. Acta Neurol Scand 2007;116:322–7.
33. Johnson LN, Krohel GB, Madsen RW, March GA Jr. The role of weight loss and acetazolamide in the treatment of idiopathic intracranial hypertension (pseudotumor cerebri). Ophthalmology 1998;105:2313–7.
34. Miglis MG, Levine DN. Intracranial venous thrombosis after placement of a lumbar drain. Neurocrit Care 2010;12:83–7.