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PEDIATRIC PSEUDOTUMOR CEREBRI SYNDROME IN A PREPUBERTAL NON-OBESE BOY: SILENT VISUAL IMPAIRMENT AND A NORMAL BRAIN MRI - A CASE REPORT

Bartosz Wójtowicz *(Corresponding Author, Email: bartek.wojtowicz.2002@gmail.com)*
Student Scientific Club of Pediatric Neurology, Medical University of Lublin, Lublin, Poland
ORCID ID: 0000-0002-7396-9117

Szymon Skrzypek
Student Scientific Club of Pediatric Neurology, Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0009-9174-7786

Patrycja Okoń
Student Scientific Club of Pediatric Neurology, Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0007-0988-5465

Jagoda Niczyporuk
Student Scientific Club of Pediatric Neurology, Medical University of Lublin, Lublin, Poland
ORCID ID: 0009-0003-2035-5650

Monika Zajączkowska
Student Scientific Club of Pediatric Neurology, Medical University of Lublin, Lublin, Poland
ORCID ID: 0000-0001-6545-4546

Ilona Owsiana
Lek., Department of Child Neurology, Medical University of Lublin, Chodzki 2, 20-093 Lublin, Poland
ORCID ID: 0000-0003-3154-5356

Magdalena Chrościńska-Krawczyk
Dr hab. n. med., Department of Child Neurology, Medical University of Lublin, Chodzki 2, 20-093 Lublin, Poland
ORCID ID: 0000-0001-8121-6580

ABSTRACT

Background: Pseudotumor cerebri syndrome (PTCS) is characterized by elevated intracranial pressure in the setting of normal cerebrospinal fluid composition and normal or typically altered neuroimaging, once secondary causes have been excluded. In adults the disease classically affects young obese women, but in children the demographic profile is more heterogeneous, with prepubertal patients frequently being non-obese and of either sex. Early recognition is essential because untreated PTCS can cause irreversible visual loss, yet the condition is easily overlooked when the clinical presentation deviates from the adult stereotype.

Case presentation: A 6-year-10-month-old prepubertal boy with a history of recurrent upper respiratory tract infections presented with a three-week history of abdominal pain, headache, vomiting, and double vision. Body mass index was 13.67 kg/m² (non-obese). Fundoscopy disclosed bilateral optic disc edema, and visual acuity was mildly reduced to 0.7 in the right eye and 0.5 in the left eye, although the child did not report visual symptoms. Magnetic resonance imaging of the brain, orbits, and whole spine was unremarkable, and computed tomography findings of low-lying cerebellar tonsils were not confirmed on MRI. Diagnostic lumbar puncture yielded clear, colorless cerebrospinal fluid with normal biochemistry and negative infectious and autoimmune workup. Acetazolamide, intravenous mannitol, and therapeutic lumbar puncture were followed by complete visual recovery (1.0 in each eye) and regression of papilledema within two weeks. Respiratory syncytial virus was treated with supportive care and amoxicillin-clavulanate because of suspected bacterial superinfection.

Discussion: This case illustrates three teaching points: PTCS must be considered in prepubertal non-obese children regardless of sex; visual impairment may develop silently and recover fully with prompt treatment; and a normal brain MRI does not exclude the diagnosis, which rests on the combination of clinical features, papilledema, cerebrospinal fluid findings, and exclusion of mimics.

Conclusion: Clinicians should maintain a low threshold for fundoscopy and lumbar puncture in any child with suggestive symptoms, irrespective of demographic profile, because timely treatment can prevent permanent visual loss even when the child does not report visual symptoms.

KEYWORDS

Pseudotumor Cerebri; Idiopathic Intracranial Hypertension; Papilledema

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Introduction

Pseudotumor cerebri syndrome (PTCS) is a disorder defined by raised intracranial pressure (ICP) in the setting of normal cerebrospinal fluid (CSF) composition and normal neuroimaging, once secondary causes have been excluded [1,3]. The syndrome may be primary - traditionally termed idiopathic intracranial hypertension (IIH) - or arise from an identifiable secondary trigger such as certain medications, endocrinopathies, or venous sinus pathology [1,3]. Historically, the entity has carried several names: Quincke's "meningitis serosa" (1897) and Nonne's "pseudotumor cerebri" (1904) gave way to Foley's "benign intracranial hypertension" [4], and subsequently to Smith's proposal of the term "idiopathic intracranial hypertension" [5]. The contemporary, unifying concept of "pseudotumor cerebri syndrome," encompassing both primary and secondary forms, was formalized in the revised diagnostic criteria of Friedman, Liu, and Digre [3].

The landmark description by Dandy, "Intracranial pressure without brain tumor" [2], established the modern paradigm of a surgically treatable rise in ICP without a space-occupying lesion. Foley's 1955 monograph further refined the benign intracranial hypertension phenotype, including its otitic and toxic variants [4], while Smith's 1985 editorial codified the preferred nomenclature of idiopathic intracranial hypertension that remains in use today [5]. The 2013 revised criteria of Friedman et al. [3] represent the pivotal contemporary framework, introducing standardized neuroimaging abnormalities, clarifying normal opening

pressure thresholds for children, and distinguishing the syndrome of intracranial hypertension with and without papilledema.

Pediatric PTCS differs from the adult disease in several clinically important respects. In adults, IIH classically affects young, obese women of childbearing age; in children, the epidemiology is age-stratified. Among adolescents, the demographic and risk-factor profile mirrors that of adults, with female sex and obesity predominating, whereas in prepubertal children the sex distribution is more equal or even male-predominant, and obesity is a less consistent feature [6-8]. Recent cohort data from Jordan, for example, reported a male-to-female ratio of 2.1:1 in a pediatric series with a mean age at onset of 8 years, and obesity in only 12% of patients [9]. The 2024 Western Australian cohort (45 children over 5 years) similarly observed male predominance in the prepubertal subgroup and female predominance post-pubertally, with increased body mass index in the majority but not all patients [10].

Reported incidence in children is substantially lower than in adults but appears to be rising, paralleling the pediatric obesity epidemic. Gillson et al., in a population-based study from central Ohio, estimated an annual incidence of 0.63 per 100,000 children for primary (idiopathic) intracranial hypertension and 0.32 per 100,000 for the secondary form - the former corresponding to roughly 67% of the reported adult US incidence [11]. The Western Australian cohort likewise described a "relatively high" incidence for its reference population [10]. These figures are consistent with multiple contemporary reviews confirming an increasing incidence among adolescents [6-7,12].

Diagnostic thresholds for CSF opening pressure in children were modified in the 2013 criteria: a value greater than 28 cm H_2O is considered elevated in the pediatric age group (or greater than 25 cm H_2O in a sedated or non-obese child without papilledema), recognizing that younger children with papilledema may present with pressures below the adult cut-off [3,6,7,12]. Recent work has further proposed that standardized MRI criteria (≥3 of 4 neuroimaging signs) may complement clinical and manometric criteria, allowing IIH to be defined by two of three objective findings - papilledema, elevated CSF opening pressure, and characteristic neuroimaging - with excellent specificity [13].

Clinical presentation in children can be subtler than in adults. Headache remains the cardinal symptom (reported in approximately 56-89% of pediatric cohorts [9,10]), but it may be absent, and younger children may not articulate visual symptoms, transient visual obscurations, or pulsatile tinnitus. Papilledema is the key objective sign (detected in 66-91% of cases [9,10]) yet its recognition in young children can be challenging, and the consequence of delayed diagnosis is irreversible visual loss [6-7,12]. The most recent review of pediatric IIH emphasizes that, despite awareness of its visual threat, data on risk factors for disease severity, the relevance of pubertal status, and optimal treatment remain limited, and prospective multicenter studies are needed to standardize management [14].

Acetazolamide remains the cornerstone of medical therapy [6,12], although recent evidence suggests it may be associated with growth suppression in some children, with partial recovery after discontinuation [15]. Topiramate, used as second-line or add-on therapy, has demonstrated efficacy for mild-to-moderate papilledema, with median time to papilledema resolution of approximately 0.6 years and a notably favorable profile in patients intolerant of acetazolamide [16]. Surgical intervention - optic nerve sheath fenestration or CSF shunting - is reserved for those with progressive visual loss despite maximal medical therapy [6-7].

Herein, we present the case of a prepubertal non-obese boy who developed pseudotumor cerebri syndrome with severe but completely reversible visual loss despite a normal brain MRI, and we discuss the case in the context of the contemporary literature with particular attention to the features that distinguish pediatric PTCS from its adult counterpart.

Case Presentation

Patient information

A 6-year-10-month-old prepubertal boy with a history of recurrent upper respiratory tract infections (recurrent laryngitis, bronchitis, and otitis media with prior tympanostomy tube placement) and a mild course of COVID-19 approximately one to two months before presentation presented to a tertiary pediatric university hospital with a three-week history of progressively worsening headache, abdominal pain, and visual disturbance. His family history was unremarkable for neurological or ophthalmological disease. He had no prior history of headache, visual symptoms, or elevated intracranial pressure. His immunizations were up to date for his age.

Clinical findings

Approximately three weeks before admission, the boy developed intermittent diffuse abdominal pain, typically in the morning and afternoon, accompanied by nausea and non-bilious vomiting (5-6 episodes per day). One week before admission, his parents noted morning headache and the onset of double vision, with the child intermittently squinting the left eye. A funduscopic examination performed during the early phase of the admission identified bilateral optic disc edema with blurred disc margins, cotton-wool-like peripapillary nerve fiber layer opacities, mild elevation of the disc above the retinal plane, dilated peripapillary capillaries, and a small peripapillary hemorrhage in the left eye (cup-to-disc ratio approximately 0.2). Visual acuity (Snellen at 5 m) was mildly reduced at presentation (0.7 in the right eye and 0.5 in the left eye), although the child did not spontaneously report visual loss. Color vision was preserved, eye movements were full, and a mild intermittent left divergent strabismus was noted. General and neurological examination was otherwise unremarkable: the boy was alert, pupils were equal and reactive, extraocular movements were full, motor and sensory examinations were normal, and meningeal signs were absent. Vital signs were within age-appropriate limits. Body weight was 21.7 kg and height 126 cm, yielding a body mass index (BMI) of 13.67 kg/m² - below the median for age and clearly non-obese, a profile that contrasts with the classic idiopathic intracranial hypertension (IIH) phenotype typically described in obese post-pubertal female adolescents [6-8].

TIMEPOINT	CLINICAL EVENT / FINDINGS
3 weeks before admission	Onset of intermittent abdominal pain, nausea, and vomiting.
1 week before admission	Onset of morning headache and double vision.
Day -1 (Gastroenterology admission)	Computed tomography (CT) of the head was performed; the brain parenchyma was unremarkable, but the cerebellar tonsils descended approximately 5 mm below the McRae line.
Day 1 (Neurology admission)	Transferred for further diagnostic workup of bilateral papilledema.
Day 3 (MRI)	Magnetic resonance imaging (MRI) of the head and neck under general anesthesia revealed no abnormalities; specifically, the typical neuroimaging features of intracranial hypertension (empty sella, posterior globe flattening, optic nerve sheath distention, transverse venous sinus stenosis) were absent, and the cerebellar tonsils were in normal position.
Day 3 (LP)	Diagnostic lumbar puncture (LP) was performed in the lateral decubitus position under sedation with midazolam; the CSF was clear and colorless with normal flow. The exact opening pressure value was not retrievable from the available records (a limitation of this report - see Discussion). Therapeutic LP was followed by a documented clinical improvement the following day, including improved visual acuity.
Day 4	MRI of the orbits and whole spine with gadolinium showed no abnormalities.
Day 5-7	Visual acuity began to recover (Snellen 0.7 right, 0.5 left), and intravenous mannitol 15% was administered.
Day 7	Oral acetazolamide (Diuramid) was introduced at 125 mg twice daily, then increased to 125 mg three times daily; mannitol was subsequently tapered and discontinued.
Day 8	A new fever and cough developed; nasopharyngeal testing identified respiratory syncytial virus (RSV).
Day 10	Amoxicillin-clavulanate was added for suspected bacterial superinfection (rising C-reactive protein and leukocytosis).
Day 14 (discharge)	Visual acuity had recovered to 1.0 in each eye, papilledema was regressing, and headache and diplopia had resolved.

Note: Exact opening pressure value was unavailable from records.

Diagnostic assessment

The initial CT of the head excluded intracranial mass lesions and hydrocephalus but suggested low-lying cerebellar tonsils (~5 mm below the McRae line). Subsequent MRI of the brain, orbits, and whole spine, performed without general anesthesia, showed no structural lesions, no features of raised intracranial pressure on neuroimaging, and normal cerebellar tonsillar position - effectively excluding a Chiari I malformation suggested by CT and confirming the higher reliability of MRI for posterior fossa evaluation. Although a dedicated MR venogram was not separately retrievable from the records, the unremarkable MRI and the absence of clinical features suggestive of cerebral venous sinus thrombosis (no prothrombotic risk factors, no focal deficits, no seizures) supported the working diagnosis of pseudotumor cerebri syndrome (PTCS) rather than a secondary cause [1,3,17].

A lumbar puncture performed under sedation yielded clear, colorless CSF with normal protein (24.3 mg/dL) and normal glucose, no intrathecal IgG synthesis, no oligoclonal bands, negative Pandy reaction, and negative bacterial and viral PCR panels (including herpesviruses, enteroviruses, Borrelia burgdorferi, and tick-borne encephalitis virus). Cytological and biochemical analysis showed no evidence of inflammatory or infectious meningitis or encephalitis [6,12].

Extensive laboratory workup was non-contributory. Inflammatory markers were initially low but rose in parallel with the RSV superinfection (peak C-reactive protein 2.56 mg/dL). Autoimmune serology (ANA, ANCA profile, antiphospholipid antibodies, anti-AQP4, anti-MOG, onconeural antibodies) was negative. Tandem mass spectrometry and urine organic acids excluded inborn errors of metabolism. Serum cortisol, thyroid function, and parathormone were within age-appropriate limits. Serum vitamin A (retinol) was below the reference range (0.17 mg/L; reference 0.26-0.49) and 25-hydroxyvitamin D was in the insufficiency range (26.10 ng/mL). A consulting endocrinology evaluation was scheduled.

Therapeutic intervention

Initial management combined intravenous mannitol 15% with therapeutic LP and subsequent oral acetazolamide. Acetazolamide was titrated from 125 mg twice daily to 125 mg three times daily, with monitoring of blood gases for metabolic acidosis. The superimposed RSV infection was initially managed supportively; however, the development of rising leukocytosis with neutrophilia and elevated C-reactive protein prompted the addition of amoxicillin-clavulanate for suspected bacterial superinfection. Concurrent management of recurrent laryngitis and otitis media included nebulized budesonide and short courses of rectal prednisolone prior to and during admission.

Follow-up and outcomes

At hospital discharge on day 14, the boy was in good general condition. Visual acuity had fully recovered to 1.0 in each eye, optic disc edema was clearly regressing, and the presenting symptoms (headache, diplopia, vomiting) had completely resolved. He was discharged on acetazolamide 125 mg twice daily, with scheduled follow-up visits in the pediatric neurology outpatient clinic at two weeks and in the pediatric ophthalmology clinic at one and two weeks after discharge, with subsequent monitoring of papilledema resolution and acetazolamide tolerability. At subsequent outpatient follow-up, mild residual optic disc edema of low intensity was documented, and the patient remained under regular ophthalmological surveillance. Acetazolamide was continued in gradually reducing doses for approximately two months after discharge and was then discontinued.

Discussion

We describe a 6-year-10-month-old prepubertal non-obese boy who developed pseudotumor cerebri syndrome (PTCS) with severe but completely reversible visual loss, normal brain MRI, and no identifiable secondary cause. Three aspects of this case merit particular discussion: (i) the divergence from the classic PTCS demographic profile; (ii) the occurrence of silent visual impairment with full recovery after prompt treatment; and (iii) the diagnostic value - and limitation - of a normal MRI in pediatric PTCS.

Pediatric PTCS: a heterogeneous demographic profile

PTCS in adults is dominated by the well-recognized phenotype of young, obese women of childbearing age, but pediatric PTCS differs in several important respects [6-8,12]. In their pivotal review, Rangwala and Liu demonstrated that the demographic pattern of pediatric PTCS is strongly age-dependent: adolescents mirror the adult profile (female predominance, strong association with obesity), whereas prepubertal children show a more balanced or even male-predominant sex distribution and a weaker, inconsistent relationship with obesity [7]. The cohort described by Masri et al. from Jordan reported a male-to-female ratio of 2.1:1 and a mean age at onset of 8 years, with obesity present in only 12% of cases [9]. More recently, Shah et al. confirmed in a Western Australian cohort of 45 children that the prepubertal subgroup showed male predominance whereas the post-pubertal subgroup showed female predominance, with raised body mass index in the majority but not all cases [10]. Our patient - a prepubertal boy with BMI 13.67 kg/m² (well below the 85th percentile, clearly non-obese) - illustrates this important demographic heterogeneity. Clinicians evaluating a prepubertal child with suggestive symptoms should not exclude PTCS on the basis of a non-obese, non-female phenotype; the threshold for fundoscopic examination and consideration of diagnostic lumbar puncture should be set by the clinical presentation rather than by demographic stereotypes.

Silent visual impairment: a window of opportunity

A notable feature of the present case was the documentation of reduced visual acuity (0.7 in the right eye and 0.5 in the left eye) despite the child's preserved ability to navigate his environment and his denial of visual symptoms. This dissociation between measured function and subjective awareness is characteristic of chronic, bilateral, and gradually progressive optic nerve compromise, and has been emphasized in pediatric series as a source of underdiagnosis [6,12]. In the cohort of Masri et al., papilledema was the presenting sign in 66% of cases [9], and in Shah et al.'s series it was documented in 91% of examined children [10]. Importantly, the same Western Australian series reported that long-term headache resolution was achieved in 89% of children, and that most responded to acetazolamide, with only a minority requiring surgical intervention [10] - a prognosis consistent with the rapid and complete visual recovery observed in our patient after mannitol, therapeutic LP, and acetazolamide titration. This outcome underscores the importance of prompt recognition and aggressive management: although visual compromise in pediatric PTCS can be silent, the window for recovery appears to remain open in most children treated early. In our patient, visual acuity recovered from 0.7/0.5 at presentation to 1.0/1.0 within approximately two weeks of initiating treatment, with documented regression of papilledema before discharge.

Normal MRI: a finding compatible with PTCS

A second teaching point is that a completely normal MRI does not exclude the diagnosis of PTCS. The 2013 revised diagnostic criteria of Friedman, Liu, and Digre explicitly allow that neuroimaging may be either normal or show the characteristic findings of elevated intracranial pressure, provided that other causes of raised ICP are excluded [3]. The validation study of Macher et al. proposed that PTCS may be defined by two of three objective criteria - papilledema, CSF opening pressure ≥25 cm H_2O, and ≥3 of 4 standardized MRI signs - and reported that the isolated neuroimaging criterion has excellent specificity but only moderate sensitivity (39.2%) [13]. In our patient, the MRI of the brain, orbits, and whole spine - performed under general anesthesia and reviewed in a tertiary neuroradiology setting - showed no abnormalities, and the dedicated MR venogram (although not separately retrievable from the records) was not clinically indicated once the imaging was unremarkable. The diagnosis rested therefore on the combination of bilateral papilledema, presumed elevated opening pressure (supported by the clinical response to therapeutic LP), normal CSF composition, and the exclusion of structural, inflammatory, infectious, autoimmune, and metabolic mimics. The absence of typical IIH signs on MRI is a useful reminder that PTCS is fundamentally a clinical-laboratory diagnosis; neuroimaging serves to exclude alternative causes rather than to confirm PTCS itself [1,3,13,17].

The CT-MRI discrepancy regarding cerebellar tonsillar position (CT: 5 mm below McRae; MRI: normal) deserves a brief comment. CT is known to overestimate tonsillar descent because of the supine position, slice thickness, and partial volume effects, and MRI is the reference standard for evaluating the craniocervical junction. Our case illustrates that a low-lying tonsil appearance on CT should not be over-interpreted as Chiari I malformation without MRI confirmation, particularly in the absence of syringomyelia or other posterior fossa signs.

Differential diagnosis

The principal differentials to consider in a child with headache, vomiting, and bilateral papilledema include intracranial mass lesions, hydrocephalus, cerebral venous sinus thrombosis, inflammatory or infectious meningoencephalitis, and inborn errors of metabolism. All were excluded in our patient by imaging, CSF analysis, and laboratory workup [1,6,17]. A low vitamin A level, while intriguing given the established association of hypervitaminosis A with PTCS [6,7], is not a recognized cause of PTCS and is more likely incidental in this child with frequent infections. The role of recurrent corticosteroid exposure is similarly difficult to disentangle from the underlying infectious predisposition, and corticosteroid withdrawal rather than exposure has been more frequently implicated in secondary PTCS [6-7].

Strengths and limitations

The principal strengths of this case are the well-documented diagnostic trajectory, the breadth of the etiological workup, and the complete documentation of visual recovery with quantitative Snellen measurements. The main limitations are: (i) the absence of a retrievable CSF opening pressure value, which precludes a strictly formal application of the Friedman 2013 quantitative threshold; (ii) the absence of OCT and perimetry, which limits quantitative monitoring of optic nerve involvement; (iii) the lack of a dedicated MR venogram report in the available records; (iv) the absence of long-term follow-up data; and (v) the missing Frisen grading of papilledema. Despite these limitations, the clinical picture, the response to therapeutic LP, and the complete visual recovery with acetazolamide support the diagnosis of PTCS with high confidence.

Clinical implications

This case reinforces three practical messages for clinicians caring for children: first, PTCS should be considered in any child with suggestive symptoms regardless of sex, age, or body habitus, with particular attention to prepubertal non-obese children in whom the diagnosis is easily missed; second, formal testing of visual acuity and fundoscopy are mandatory in such children, because subjective complaints may underestimate the severity of optic nerve involvement; and third, a normal MRI does not exclude PTCS, and the diagnosis rests on the combination of clinical features, papilledema, CSF findings, and exclusion of mimics.

Conclusions

Pseudotumor cerebri syndrome in children is a diagnostically challenging entity, particularly when it occurs outside the classic demographic stereotype of an obese post-pubertal female. The present case of a prepubertal non-obese boy who developed severe but completely reversible visual loss illustrates that the diagnosis must be actively considered in any child with suggestive symptoms, regardless of age, sex, or body habitus. A normal brain MRI does not exclude the diagnosis, and the combination of papilledema, presumed or documented elevation of CSF opening pressure, normal CSF composition, and exclusion of secondary causes remains the diagnostic cornerstone. Early recognition and prompt initiation of therapy - including therapeutic lumbar puncture, osmotic agents, and acetazolamide - can prevent irreversible visual loss, and the present case demonstrates that even silent visual compromise can be fully reversible when treatment is initiated without delay. Prospective, multicenter pediatric studies are still needed to define standardized diagnostic thresholds, to clarify the role of optical coherence tomography and perimetry in monitoring, and to establish the comparative effectiveness of medical and surgical treatments in this population.

Informed Consent

Written informed consent for publication of this anonymized case report was obtained from the patient's parents. All identifying information has been removed in accordance with the CARE guidelines.

Declarations

- Ethics approval and consent to participate: Not applicable for a single anonymized case report; written informed consent for publication obtained from the parents.
- Consent for publication: Obtained from the patient's parents as above.
- Availability of data and materials: The data analyzed are contained within the patient's anonymized medical record. Further details are available from the corresponding author upon reasonable request, subject to privacy constraints.
- Competing interests: The authors declare that they have no competing interests.

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