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Original Article

Novel Magnetic Resonance Imaging Findings in Children With Intracranial Hypertension

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ABSTRACT

BACKGROUND: Increased intracranial hypertension is defined as elevated intracranial pressure with absence of hydrocephalus, vascular or structural abnormalities, and normal cerebrospinal fluid content. Magnetic resonance imaging abnormalities of the optic nerve and sheath that have been described in adults include increased nerve tortuosity, flattening in posterior aspect of globe, intraocular protrusion of the optic nerve, and enlarged optic nerve sheath. **PURPOSE:** We evaluated accuracy of those proposed markers on magnetic resonance imaging in children with increased intracranial hypertension that are described in adults. **MATERIALS AND METHODS:** Eleven patients between 3 and 15 years of age with intracranial hypertension were selected for re-evaluation of magnetic resonance imaging that had been previously described as normal to determine the presence of: (1) increased tortuosity and elongation of the optic nerve; (2) enlargement of the optic nerve sheath on axial and coronal T2 so called by us "target sign" and postcontrast T1 sequences; (3) flattening in posterior aspect of the globe; and (4) intraocular protrusion of the optic nerve head. **RESULTS:** Of the 11 patients, tortuosity of the optic nerve (10/11, 90.9%) and enlarged optic nerve sheath—target sign (7/11, 63.6%)—were the most common findings. Flattening in the posterior aspect of globe (5/11, 45.5%) and intraocular protrusion (3/11, 27.3%) were also detected as a novel magnetic resonance imaging findings. **CONCLUSION:** Magnetic resonance imaging findings of the optic nerve and sheath include valuable signs of intracranial hypertension not only in adults but also in children. This is the first detailed analysis of the magnetic resonance imaging findings in children with increased intracranial hypertension.

Keywords: intracranial hypertension, MRI findings, children, increased intracranial pressure

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Introduction

Idiopathic intracranial hypertension (IIH) is defined by increased intracranial pressure with no clear etiology and the absence of hydrocephalus, vascular, or other structural lesion and normal cerebrospinal fluid (CSF) content.[1-12] Common complaints are headache, blurred vision, and vomiting.[1-16] The most important complication is permanent visual disturbance in approximately 10% of the patients.[1-16] Although magnetic resonance imaging (MRI) may be unremarkable in terms of gross pathology, some studies point to subtle radiological markers including compression of the optic globe, particularly in the posterior aspect, intraocular protrusion and enlargement of optic nerve sheath, and increased tortuosity in optic nerve that may be important observation in the early diagnosis of IIH.[14-28] These findings have been studied in adult populations, but there are few studies in children with IIH.[7,20,26-31]

Our aim was to investigate these MRI markers and their possible contribution to the diagnosis of IIH in the pediatric age group.

Material and Methods

Eleven patients aged between 3 and 15 years old (mean age 10.45 years) with IIH were selected retrospectively from Department of

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Pediatric Neurology, Gazi University School of Medicine, between January 2012 and December 2013. The diagnosis of IIH was based on the Modified Dandy Criteria: (1) signs and symptoms of increased intracranial pressure; (2) absence of localizing findings on neurological examination; (3) increased CSF opening pressure with normal CSF ingredients; (4) no evidence of hydrocephalus, mass, structural, or vascular lesions on imaging; and (5) no other causes of increased intracerebral pressure identified.[1,9] To confirm the diagnostic criteria, medical records of the patients were evaluated in terms of detailed history, physical and neurological examination, laboratory screening, and MRI findings as follows.

1. History and clinical features: Recent history of infection and medication comprising antibiotics, vitamins, hormones including oral contraceptives, and the presence of headache or additional complaints such as visual disturbances, nausea, and vomiting were investigated. Physical and neurological examination including abducens/facial nerve palsy and funduscopic examination for papilledema, if available, Snellen visual acuity, and Goldman visual field perimetry was evaluated. Body mass index was also noted for each patient. A higher percentile of 95% for each age was defined as obesity.
2. Laboratory results: Cerebrospinal fluid and blood testing was performed to exclude secondary reasons for IIH: Lumbar puncture was performed to evaluate the CSF profile and to measure opening pressure that was accepted as increased levels at >180 mm H_2O for children younger than 8 years of age and >250 mm H_2O for those older than 8 years.[4-6,8] All patients underwent some additional laboratory testing including complete blood count, liver and renal functions, electrolytes, sedimentation rate, serum ferritin, iron and iron-binding levels, thyroid function, parathyroid hormone, adrenocorticotropic hormone, cortisol, anti-nuclear antibody, anti—double-stranded DNA, and vitamins A and D levels.
3. MRI slices that were previously reported as normal in terms of congenital malformations, hydrocephalus, and mass, structural lesions were re-evaluated by T.H.; K.A., who is most experienced and educated physician in MRI interpretation in the group; and A.S. independently in terms of proposed MRI findings. The axial and coronal T2 and postcontrast T1 sequences were investigated for the presence of proposed subtle signs in optic nerve and sheath for each patient: (1) flattening at posterior aspect of the globe are described as disturbed normal convexity and also straightening of the normal outwards of the globe in the area of attachment to optic nerve, (2) intraocular protrusion of optic nerve: beyond the flattening as described previously, there is a concave protrusion towards to the globe; and (3) enlargement of optic nerve sheath on T2 axial serial—we newly proposed a “target sign” on coronal T2 sequences. The measurements of optic nerve sheath were performed from 5 mm posterior to the globe.[18,20,32-34] If the mean optic nerve sheath diameter was greater than 5 mm in diameter, it was considered as abnormal and meaningful for IIH,[19,20,22,25,30,31] (4) increased tortuosity in optic nerve can also be described as vertical tortuosity of the optic nerve which are observed as “redundant” or “S-shaped” appearance and (5) a narrowed ventricle so called “slit-like ventricles,” empty sella, and transverse sinus abnormalities were evaluated.[17-31]

Approval for this study was granted by Gazi University School of Medicine Ethical Committee (N:2014-534). Statistical Package for Social Science, version 16.0, SPSS Inc, Chicago, IL, was used for statistical analysis. Descriptive analyses were performed and data were presented as mean ± standard deviation. Correlation coefficient values were calculated for comparing CSF opening pressure and MRI findings.

Results

Eleven patients were found to fit the criteria for re-evaluation for subtle MRI findings of IIH. Five of the patients were female (45.5%) and 6 (55.5%) were male. Symptom duration was highly variable, ranging from 2 days to 1 year before admission to our department.

The most common complaints were headache (100%) and ophthalmologic symptoms (81.8%). Nausea and vomiting occurred in five (45.5%) patients. Four (36.4%) patients had diplopia and one had an abducens palsy. Eight (72.8%) patients had a problem in visual acuity, and one of these had a significant visual field defect. Bilateral papilledema was detected in all patients (11/11, 100%).

A total of eight patients (72.7%) was older than 8 years and the remaining three (27.3%) were younger than 8 years of age. The CSF pressure was measured as a mean of 190 mm H_2O (ranged 180-200 mm H_2O) in those younger than 8 years old and 465 mm H_2O (ranged 250-790 mm H_2O) in those older than 8 years old.

For etiology, basic hematologic, and biochemical results, hormone levels including thyroid functions, parathyroid hormone, adrenocorticotropic hormone, cortisol, and vitamins were within normal limits. The most important factor was obesity with increased body mass index (5/11, 45.5%). As to secondary IIH, one patient had Behcet disease with congenital hypoplasia of the transverse and sigmoid sinus and jugular vein; one patient had thrombosis in superior sagittal sinus with a factor V Leiden mutation. Detailed demographics and clinical signs are presented in Table 1.

MRI scans of all patients were within normal limits in terms of congenital, vascular, mass, structural lesions, and hydrocephalus in idiopathic IIH. One patient with Behcet disease had hypoplasia in transverse and sigmoid sinus; another one with factor V Leiden mutation had thrombosis in the anterior third of the superior sagittal sinus in the secondary IIH group (Fig. 1).

TABLE 1.
Demographics and Clinical and Laboratory Findings of the Patients

	Number (Range or %)
Age	10.45 (3-15)
<8 years old	3 (27.3%)
>8 years old	8 (72.8%)
Sex	
Female	5 (45.5%)
Male	6 (55.5%)
Symptom duration before admission	2 days to 1 year
Headache	11 (100%)
Nausea ± vomiting	5 (45.5%)
Ophthalmologic symptoms: findings	9 (81.8%)
Diplopia	4 (36.4%)
Disturbed visual acuity	8 (72.8%)
Abducens nerve palsy	1 (9%)
Papilledema on funduscopic examination	11 (100%)
CSF pressure	
<8 years old	190 mm H_2O (180-200)
>8 years old	465 mm H_2O (250-790)
Etiology	
Obesity	5 (45.5%)
Venous sinus thrombosis (factor V Leiden mutation)	1 (9%)
Hypoplasia of venous sinus (Behcet disease)	1 (9%)
Treatment	
Acetazolamide	11 (100%)
Acetazolamide and topiramate	1 (9%)
Ventriculoperitoneal shunt	1 (9%)

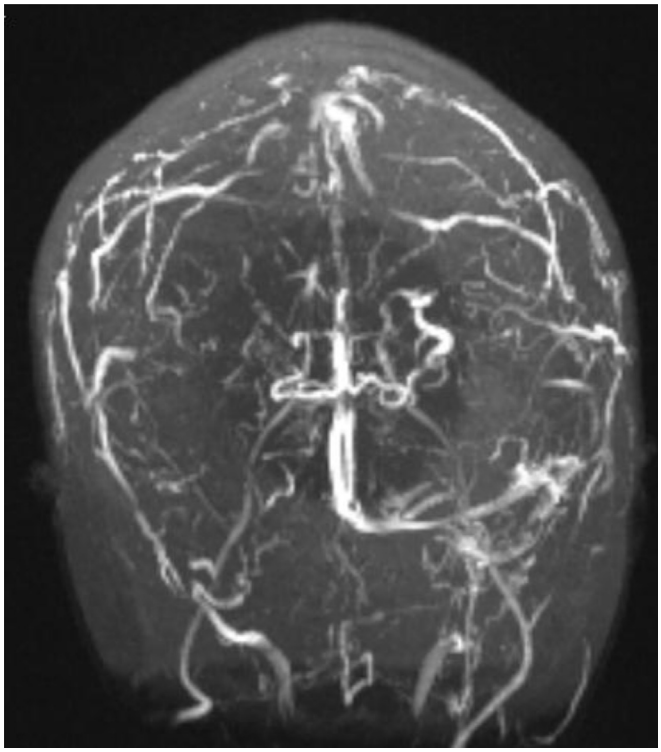


FIGURE 1.
Magnetic resonance venography demonstrates thrombosis in the anterior superior sagittal sinus.

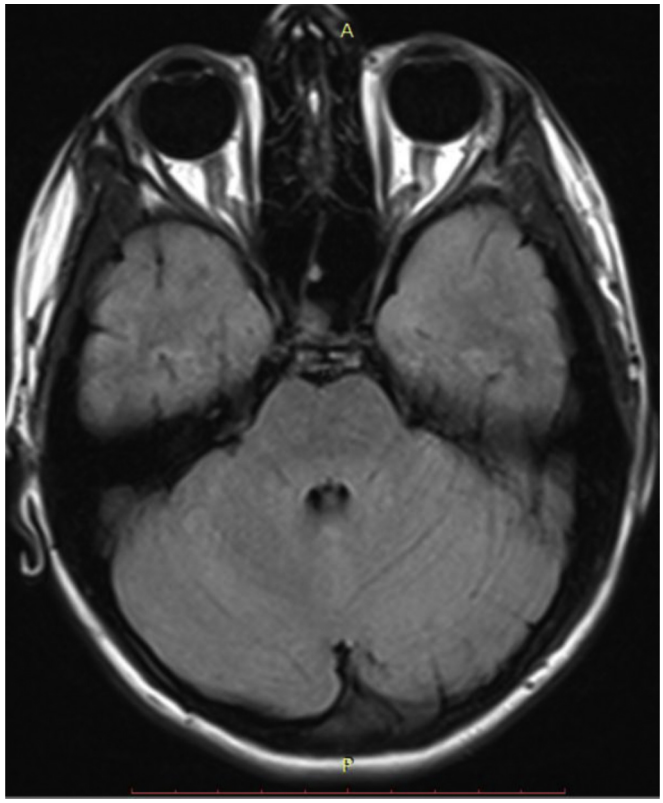


FIGURE 2.
Axial T1-weighted, fluid-attenuated inversion recovery image shows increased tortuosity.

When the axial and coronal T2 and postcontrast T1 sequences were reexamined if the presence of proposed subtle markers, increased tortuosity in the optic nerve was found as the most (10/11, 90.9%) common finding (Fig. 2). Seven of 11 (63.6%) patients had optic nerve sheath enlargement that we named a target sign on T2-coronal slices by our group (Fig. 3A,B). Those patients had also significantly increased value of optic nerve sheath diameter (6.7 ± 0.74) than standard value on T2-axial images (Fig. 4A). Posterior globe flattening of sclera was detected in 5/11 (45.5%) patients (Fig. 4B). Three of 11 (27.3%) patients had intraocular protrusion of the optic nerve (Fig. 5). No patient had slit-like ventricle or empty sella (Table 2).

There were no correlations between the CSF opening pressure and flattening of the posterior globe (r: 0.462; p: 0.153), protrusion toward to the globe (r: 0.387; p: 0.239), and target sign (r: 0.06; p: 0.861). However, these results may be caused by small number of the patients. There were significantly increased CSF opening pressure in the patients with posterior globe flattening (5/11) and intraocular protrusion of optic nerve (3/11) (mean 53.3 mm H_2O and 54.8 mm H_2O, respectively).

The patients were treated with acetazolamide for 2-6 months' duration. One patient used both topiramate and acetazolamide and then underwent ventriculoperitoneal shunting because of significant vision loss.

Discussion

Intracranial hypertension was first defined by Quincke in 1897 as the presence of elevated intracranial pressure in the absence of hydrocephalus or other structural lesions.[1-14] The disease is relatively rare in children and more often seen in postpubertal ages.[1-14] As confirmation of previous studies,[2,15-17,23,26] we found that the most important risk factor was obesity (45.5%); headache (100%), decreased visual acuity, or visual field defects (30%), and papilledema (100%) were the most frequent symptoms and signs in our cohort.

Normal MRI findings are required for exclusion of secondary reasons of increased intracranial pressure, including hydrocephalus, vascular lesions, or other structural lesions.[17-28] Some specific signs of increased intracranial pressure such as empty sella, slit-like ventricles, and transverse sinus narrowing can be infrequently detected in idiopathic-primary intracranial hypertension.[17-28] Beyond those classical findings, some additional markers have been proposed in adults with IIH who have distension of the perioptic subarachnoid space confining an enlarged optic nerve space on the T2 axial and coronal slices, elongation and tortuosity of the optic nerve, optic nerve head protrusion, and flattened posterior globes of sclera on MRI.[17-28] The specificity, sensitivity, and importance of those signs vary in the literature for adults.[17,18,21,22,27] Limited and controversial studies, mostly case reports, have been reported in children about the signs of IIH.[20,26-28] Our results suggest that these relatively subtle MRI findings can provide useful supporting evidence for diagnosis of IIH in almost 56.8% of pediatric patients.

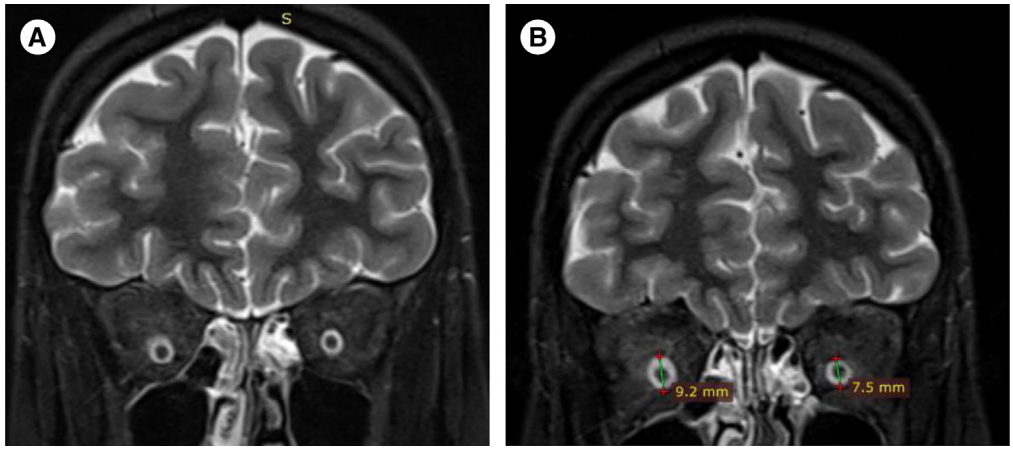


FIGURE 3.
(A) Coronal T2-weighted image shows a target sign. (B) Coronal T2-weighted image shows a "target sign" with measured optic nerve sheath diameter. (The color version of this figure is available in the online edition.)

Increased tortuosity of the optic nerve is a common feature of IIH on MRI.[17-24] Fixation of the proximal and distal points of the optic nerve despite gradually increasing intracerebral pressure causes increased tortuosity of the optic nerve in IIH.[17,18,24,25] The choice of appropriate imaging in terms of thickness and plane is important for detection of optic nerve tortuosity in both horizontal and vertical planes.[17,18,24,25] Increased tortuosity can be named as redundant and S-shaped on sagittal slices[19,25] or "smear sign" on T1-weighted images.[24] Horizontal tortuosity is seen less frequent, but may be an important indicator of increased cerebral pressure.[17,18,24] We observed that 90.9% of our patients had optic nerve tortuosity on axial images, a higher proportion than in adults with a range of 35%-63% in the literature.[19,22,25] There were no studies for children regarding for this finding.

Enlargement of optic nerve sheath

The optic nerve sheath is relatively flexible and thus reacts to changes as enlargement in presence of increased pressure.[30,31] Optic nerve sheath diameter is decreased in cerebral hypovolemia and increased in individuals with IIH.[17,18] Enlargement of the optic nerve sheath may be seen as compressed shape on T2-coronal images, with expanded cerebrospinal fluid signal intensity surrounding the optic nerve in axial series in IIH.[17,18] Increased cerebral pressure and optic nerve sheath diameter are strongly associated with IIH in adults.[20,21,30,31] Mean optic nerve sheath diameter is significantly higher than in healthy subjects (5.52 mm) in both adults (7.54 mm) and children (4.69 mm) with IIH.[20] Hansen et al. proposed that a >5-mm diameter of optic nerve sheath should be considered

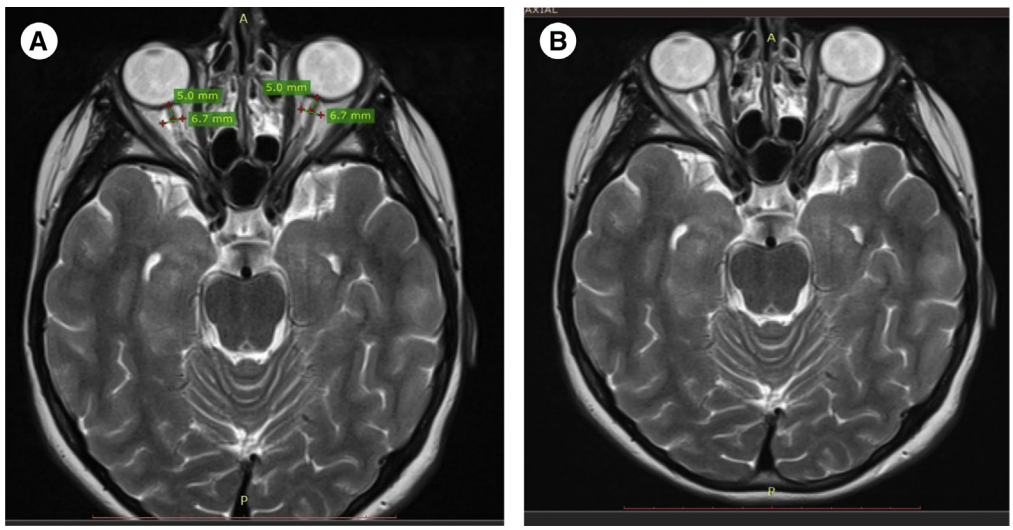


FIGURE 4.
(A) Axial, T2-weighted demonstrates flattening of optic nerve sheath and optic nerve sheath distension with measured of optic nerve sheath diameter. (B) Axial, T2-weighted demonstrates flattening of optic nerve sheath and optic nerve sheath distension. (The color version of this figure is available in the online edition.)

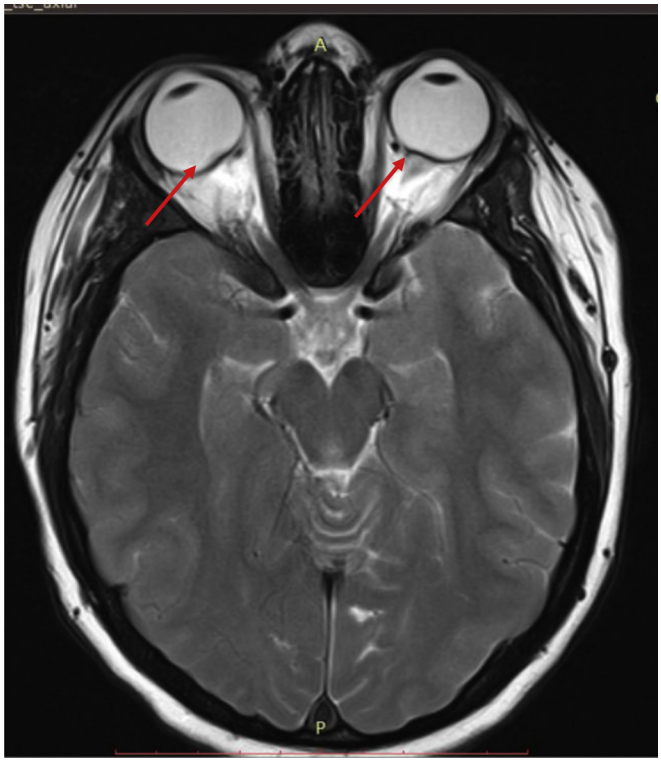


FIGURE 5.
Axial, T2-weighted demonstrates intraocular protrusion of optic nerve with hypointensity (red arrows). (The color version of this figure is available in the online edition.)

abnormal.[30,31] In adults, 51%-73% of the patients have an increased optic nerve sheath diameter.[19,22,25] As supported by those findings, we found a significantly increased diameter of the optic nerve sheath (6.7 ± 0.74) in 63.6% of our patients. Furthermore, we observed a "target sign" in these same patients. The target sign, as we propose here, is a new description for optic nerve sheath enlargement and it can be easily observed on T2-coronal sequences. The enlargement of the optic nerve sheath can resolve in patients who have undergone successful fenestration of the optic nerve sheath.[26,29] One patient in the current study recovered visual function after treatment with a ventriculoperitoneal shunt.

Flattening of posterior globe in the sclera

Even if this sign is often subtle, it might be detected in both MRI and computed tomography with an experienced and careful examination.[17-19,22,24-26,28] Intraocular and intracranial pressure are closely correlated and can be explained by extension of subarachnoid space to the posterior globe through the optic sheath.[17,18,26] Agid et al. demonstrated that compression of the posterior globe is a single most diagnostic sign associated with IIH in adults (sensitivity 43.5%, specificity 100%).[25] Similarly, Maralany et al. noted that flattening of posterior globe is a prevalent finding (53.4%) in IIH.[19] Furthermore, some studies show that flattening of posterior globe can be predicted for presence of papilledema.[17-19,22,24,25,28] We found that flattening of the posterior globe was the second-most prevalent marker and occurred with a similar ratio (45.5%) as in adults. Furthermore, those children had also considerably increased intracranial cerebral pressure and papilledema, similar to the previous adult series as mentioned previously.

TABLE 2.
Magnetic Resonance Imaging Findings in Patients

MRI Findings	Total (11, %)
Tortuosity of optic nerve	10 (90.9%)
Enlarged optic nerve sheath	7 (63.6%)
Flattening in posterior globe	5 (45.5%)
Intraocular protrusion of optic nerve head	3 (27.3%)
Venous sinus thrombosis	1 (9%)
Structural abnormalities in venous sinus	1 (9%)

Intraocular protrusion of optic nerve is a sign associated with IIH that may occur through a mechanism similar to posterior globe compression.[17] MRI reveals occasionally intraocular protrusion of the optic papilla and can be visualized in case of contrast-enhanced MRI. Focal hyperintensity adjacent to protrusion are caused by disruption of blood flow in the optic prelaminar capillaries is the most apparent MRI finding.[18] In T2-weighted imaging, the optic papilla may appear hypointense relative to the vitreous because of the predominant presence of myelinated axons.[18] This sign is often absent in IIH patients without visual symptoms.[17,18,27,28] Enhancement of the optic nerve head was demonstrated in 50%-67%[24,35] of patients, with a different range of 3.3%-63% of intraocular protrusion of the optic nerve in adult patients with IIH.[19,22,25] We found this sign in 27.3% of our patients, one of whom had serious loss in visual acuity with protrusion of the optic nerve head. There are no studies of these findings in the pediatric age group aside from a few case reports.[26-28]

Transverse sinus narrowing, "empty sella," and slit-like ventricles are reliable markers that identify increased cerebral pressure based on extraocular findings.[17,27] We observed this in only one patient with hypoplasia in the transverse and sigmoid sinus with Behcet disease. Transverse sinus narrowing occurs as a result of elevated increased cerebral pressure and can be observed in magnetic resonance venography or axial and sagittal MRI.[17-19,22,25]

In summary, subtle MRI markers such as increased tortuosity of the optic nerve, optic nerve sheath enlargement, our newly proposed "target sign" on coronal T2 sequences, optic nerve head protrusion, and flattened posterior globes of the sclera were significantly associated with IIH in children. These findings can occur with both primary and secondary intracranial hypertension. Furthermore, they can be also used as an indicator of the response to the therapy. The weakness of our study was its cross-sectional design, so we were unable to compare patients with healthy controls or to assess the reversal of MRI findings. Reversal of these signs with treatment may be demonstrated with future MRI findings. Future studies will be needed, but our study helps open a new line for future studies in children with IHH.

Conclusion

Novel MRI findings in IIH include increased tortuosity of the optic nerves, increased diameter of optic nerve sheath that we refer to as the "target sign" that can be seen on T2-coronal slices, flattening of posterior globe of sclera, and protrusion of the optic disc. These signs can be useful for the early diagnosis of IIH in not only adults but also in children. On the other hand, increased intracranial pressure can occur without papilledema. Therefore, these MRI findings can be useful as a warning and noninvasive marker for early stages or in the absence of papilledema in IIH. Furthermore, the application of those findings can facilitate the detection of IIH by encouraging the completion of a lumbar puncture, an invasive procedure and can support clinical management and potential surgical intervention in early stages of papilledema.

This is the first cross-sectional study to investigate the subtle changes on MRI without gross pathology among children with IIH. Future studies are needed to better understand the correlation between clinical signs and CSF opening pressure to the proposed MRI findings by comparing large pediatric cohort and control groups to enable early diagnosis and follow-up IIH.

References

1. Smith JL. Whence pseudotumor cerebri? *J Clin Neuroophthalmol*. 1985;5:55-56.
2. Lessell S. Pediatric pseudotumor cerebri (idiopathic intracranial hypertension). *Surv Ophthalmol*. 1992;37:155-166.
3. Kesler A, Fattal-Valevski A. Idiopathic intracranial hypertension in the pediatric population. *J Child Neurol*. 2002;17:745-748.
4. Rangwala LM, Liu GT. Pediatric idiopathic intracranial hypertension. *Surv Ophthalmol*. 2007;52:597-617.
5. Standridge SM. Idiopathic intracranial hypertension in children: a review and algorithm. *Pediatr Neurol*. 2010;43:377-390.
6. Avery RA, Licht DJ, Shah SS, et al. CSF opening pressure in children with optic nerve head edema. *Neurology*. 2011;761:658-661.
7. Phillips PH. Pediatric pseudotumor cerebri. *Int Ophthalmol Clin*. 2012;52:51-59.
8. Aylward SC. Pediatric idiopathic intracranial hypertension: a need for clarification. *Pediatr Neurol*. 2013;49:303-304.
9. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. *Neurology*. 2013;81:1159-1165.
10. Galgano MA, Deshaies EM. An update on the management of pseudotumor cerebri. *Clin Neurol Neurosurg*. 2013;115:252-259.
11. Wakerley B, Tan M, Ting E. Idiopathic intracranial hypertension. *Cephalalgia*. 2015;35:248-261.
12. Kosmorsky GS. Idiopathic intracranial hypertension: pseudotumor cerebri. *Headache*. 2014;54:389-393.
13. Babiker MO, Prasad M, MacLeod S, Chow G, Whitehouse WP. Fifteen-minute consultation: the child with idiopathic intracranial hypertension. *Arch Dis Child Educ Pract Ed*. 2014;99:166-172.
14. McGeeney BE, Friedman DI. Pseudotumor cerebri pathophysiology. *Headache*. 2014;54:445-458.
15. Per H, Canpolat M, Gümüş H, et al. Clinical spectrum of the pseudotumor cerebri in children: etiological, clinical features, treatment and prognosis. *Brain Dev*. 2013;35:561-568.
16. Değerliyurt A, Teber S, Karakaya G, et al. Pseudotumor cerebri/idiopathic intracranial hypertension in children: an experience of a tertiary care hospital. *Brain Dev*. 2014;36:690-699.
17. Degnan AJ, Levy LM. Pseudotumor cerebri: brief review of clinical syndrome and imaging findings. *AJNR Am J Neuroradiol*. 2011;32:1986-1993.
18. Passi N, Degnan AJ, Levy LM. MR imaging of papilledema and visual pathways:effects of increased intracranial pressure and pathophysiologic mechanisms. *AJNR Am J Neuroradiol*. 2013;34:919-924.
19. Maralani PJ, Hassanlou M, Torres C, et al. Accuracy of brain imaging in the diagnosis of idiopathic intracranial hypertension. *Clin Radiol*. 2012;67:656-663.
20. Shofty B, Ben-Sira L, Constantini S, Freedman S, Kesler A. Optic nerve sheath diameter on MR imaging: establishment of norms and comparison of pediatric patients with idiopathic intracranial hypertension with healthy controls. *AJNR Am J Neuroradiol*. 2012;33:366-369.
21. Padhye LV, Van Stavern GP, Sharma A, Viets R, Huecker JB, Gordon MO. Association between visual parameters and neuroimaging features of idiopathic intracranial hypertension. *J Neurol Sci*. 2013;332:80-85.
22. Saindane AM, Bruce BB, Riggeal BD, Newman NJ, Biousse V. Association of MRI findings and visual outcome in idiopathic intracranial hypertension. *Am J Roentgenol*. 2013;201:412-418.
23. Roy AG, Vinayan KP, Kumar A. Idiopathic intracranial hypertension in pediatric population: case series from India. *Neurol India*. 2013;61:488-490.
24. Brodsky MC, Vaphiades M. Magnetic resonance imaging in pseudotumor cerebri. *Ophthalmology*. 1998;105:1686-1693.
25. Agid R, Farb RI, Willinsky RA, Mikulis DJ, Tomlinson G. Idiopathic intracranial hypertension: the validity of cross-sectional neuroimaging signs. *Neuroradiology*. 2006;48:521-527.
26. Soler D, Cox T, Bullock P, Calver DM, Robinson RO. Diagnosis and management of benign intracranial hypertension. *Arch Dis Child*. 1998;78:89-94.
27. Bansal L, Niccoli L, Bailey A, Zuccoli G. Imaging the eye in idiopathic intracranial hypertension. *Headache*. 2014;54:1046-1047.
28. Mandelstam S, Moon A. MRI of optic disc edema in childhood idiopathic intracranial hypertension. *Pediatr Radiol*. 2004;34:362.
29. Sallomi D, Hibbert TJ, Sanders MD, et al. The MRI appearance of the optic nerve sheath following fenestration for benign intracranial hypertension. *Eur Radiol*. 1998;8:1193-1196.
30. Seitz J, Held P, Strotzer M, et al. Magnetic resonance imaging in patients diagnosed with papilledema: a comparison of 6 different high-resolution T1- and T2-weighted 3-dimensional and 2-dimensional sequences. *J Neuroimaging*. 2002;12:164-171.
31. Hansen HC, Helmke K. Validation of the optic nerve sheath response to changing cerebrospinal fluid pressure: ultrasound findings during intrathecal infusion tests. *J Neurosurg*. 1997;87:34-40.
32. Le A, Hoehn ME, Smith ME, et al. Bedside sonographic measurement of optic nerve sheath diameter as a predictor of increased intracranial pressure in children. *Ann Emerg Med*. 2009;53:785-791.
33. Beare NA, Kampondeni S, Glover SJ, et al. Detection of raised intracranial pressure by ultrasound measurement of optic nerve sheath diameter in African children. *Trop Med Int Health*. 2008;13:1400-1404.
34. Soldatos T, Chatzimichail K, Papathanasiou M, et al. Optic nerve sonography: a new window for the non-invasive evaluation of intracranial pressure in brain injury. *Emerg Med J*. 2009;26:630-634.
35. Jinkins JR, Athale S, Xiong L, et al. MR of optic papilla protrusion in patients with high intracranial pressure. *AJNR Am J Neuroradiol*. 1996;17:665-668.