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MRI findings as markers of idiopathic intracranial hypertension

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Abstract

Purpose of review: Negative findings on neuroimaging are part of the diagnostic criteria for idiopathic intracranial hypertension (IIH), a syndrome characterized by increased intracranial pressure (ICP). Some positive neuroimaging findings are associated with increased ICP, but their role in diagnosis of IIH has not been established. We provide an overview of these findings and their relevance for diagnosis of raised intracranial pressure

Recent findings: MRI acquisition techniques have significantly improved in the last few decades leading to better characterization of the intracranial changes associated with IIH, including. empty sella turcica, optic nerve tortuosity, distension of the optic nerve sheath, posterior globe flattening, slit-like ventricles, and venous sinus stenosis. These may be MRI biomarkers of increased ICP. Prevalence difference between people with and without increased ICP, and reversibility of these MRI findings following treatment of increased ICP inform evaluation of their diagnostic potential.

Summary: MRI and MRV findings are important tools in the diagnosis of IIH. Empty sella turcica, optic nerve protrusion, distension of the optic nerve sheath, optic nerve tortuosity, posterior globe flattening, and transverse sinus stenosis have been found to be the most promising diagnostic markers for IIH, although absence of these findings does not rule out the diagnosis.

Keywords

Idiopathic intracranial hypertension; venous sinus stenosis; empty sella; optic nerve sheath tortuosity; globe flattening; slit like ventricles; intracranial pressure

Introduction:

First described by Quinke in 1893, idiopathic intracranial hypertension (IIH), which overlaps with pseudotumor cerebri, is characterized by an elevated intracranial pressure in the

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The authors report no commercial or proprietary interest in any product or concept discussed in this article.

absence of a radiographically evident mass lesion in the brain or other identifiable secondary cause [1,2].

IIH is diagnosed on the basis of high intracranial pressure, measured via lumbar puncture, and the absence of mass occupying lesion or venous thrombosis on neuro-imaging, normal cerebrospinal fluid, and no other secondary cause. Early criteria for IIH (The Modified Dandy Criteria) incorporated the use of a head computed tomography (CT) in the diagnosis of IIH [3]. Though the necessity of dedicated cerebrovenous imaging in people with suspected increased ICP of typical IIH age, gender and body habitus remains controversial, use of brain MRI and MRV has become common to exclude secondary causes in cases of suspected increased ICP, with CTV being used in people who can not have an MRI [4].

Beyond this diagnostic requirement for lack of neuroimaging findings of secondary causes of increased ICP, people with IIH often have positive neuroimaging findings, such as empty sella, thought to be caused by increased ICP. In 2002, these MRI changes were incorporated into updated diagnostic criteria for pseudotumor cerebri in the absence of papilledema [1]. However, interpreting the clinical implications for these findings remains challenging, particularly in people who are not suspected to have increased ICP (i.e. in whom the finding is incidental) and in people who have a history of increased ICP.

The purpose of this review is to give an overview of MRI findings that are associated with IIH, both subtle as well as pronounced, including an empty or partially empty sella turcica, optic nerve protrusion, distension of the optic nerve sheath (ONS), optic nerve tortuosity, slit-like ventricles, flattening of the posterior aspect of the optic globe, and transverse sinus stenosis. *The validity and diagnostic reliability, prevalence of these findings in people with* increased *ICP as compared to people with normal ICP, correlations between the extent of abnormal neuroimaging findings and the severity of clinical symptoms and the reversibility of these MRI findings following treatment of the* increased *intracranial pressure (ICP) are highlighted.*

Changes in the Pituitary Gland: Empty Sella

The term “empty sella” (Figure 1) refers to a neuroimaging finding in which the sella turcica, the skull base structure that houses the pituitary gland, is either partially or fully filled with cerebrospinal fluid (CSF). Primary empty sella (PES) is the occurrence of an empty sella without any known pathology of the pituitary gland, while secondary empty sella (SES) is that which occurs as a result of pituitary tumor, head trauma, surgery, radiotherapy, drug therapy, spontaneous regression, or rarely, Sheehan syndrome [5]. PES is less common than SES, and is associated with IIH. Traditionally, it was thought that the finding of PES in people with IIH was due to herniation of the subarachnoid space into the sella turcica from intracranial hypertension resulting compression of the pituitary gland. However more recently, it has been theorized that PES may in part be due to the bony enlargement of the sella turcica, caused by chronic increased ICP, resulting in a larger and therefore proportionally more “empty” sella turcica. This enlargement is partly due to the extremely thin floor of the bony sella turcica, which measures less than 1 mm and makes it malleable. On MRI, the dimensions of the sella turcica and pituitary gland are measured, and

an empty sella appears as containing a pituitary gland that has been flattened to some degree, accompanied by a CSF-intensity signal [6].

One study found that the cross-sectional area of the sella was 38% greater in people with IIH than those without with only slight reduction in size of pituitary gland, and Patterson et al found that an MRI-measured pituitary-to-sella turcica ratio of <0.5 increased the likelihood of increased ICP [7,8]. Agid et al found empty sella to be present in 26.7% of people with IIH and 5.4% of controls [9]. Partial empty sella has been found to be a highly specific finding of IIH (95.3%, $p < 0.0001$), but the absence of this finding does not rule out the diagnosis [10]. The finding of partial empty sella in people with IIH has been reported to be reversible, and this was proposed to depend on the chronicity of the increased intracranial pressure as well as the structural integrity of the sella turcica at baseline [11].

Changes in the Optic Nerve: Protrusion, Tortuosity, & Sheath Distension

Orbital findings that have been associated with IIH include optic nerve protrusion, optic nerve tortuosity, and optic nerve sheath (ONS) distension. Optic nerve protrusion (Figure 2) is the MRI representation of papilledema, the swelling of the optic nerve head due to increased CSF pressure in the optic nerve sheath and correlates with optic nerve edema seen on OCT [12]. Optic nerve protrusion may not display a difference in intensity on routine MRI, but with contrast this intraocular protrusion appears as focal hyperintensity at the optic nerve head [13]. Agid et al found optic nerve protrusion to be present in 3.3% of people with IIH and 0% of controls [9]. Reversibility of this finding with treatment of IIH is unknown since the association between papilledema grade and optic nerve hyperintensity is not well-established [14]. Recently however, quantitative analysis of MRI using 2D mapping demonstrated that optic nerve protrusion is a clinically relevant marker of papilledema risk in IIH, due to an association of the extent of optic nerve protrusion with papilledema grade ($R = 0.74$, $P = .01$) [15].

Tortuosity of the optic nerve (Figure 3) occurs due the fixation of the nerve at proximal and distal points and increased CSF pressure in the optic nerve sheath and appears “kinked”. Detection of tortuosity depends on the MRI slice thickness and orientation, with horizontal tortuosity being less common but more specific for increased ICP than vertical tortuosity. Vertical tortuosity is often accompanied by a “smear sign,” where the middle of the optic nerve is covered by a “smear” of orbital fat [13]. Agid et al found optic nerve tortuosity to be present in 40% of people with IIH and 8.9% of controls [13]. Caglayan et al found that horizontal tortuosity is suggestive of IIH for diagnosis due to the difference in frequency in pre-treatment and post-treatment IIH groups, but is not a valuable marker of prior IIH based on the similar frequency of horizontal tortuosity in post-treatment IIH groups versus controls (controls: 90% normal, 10% horizontal tortuosity; pre-treatment: IIH 20% normal, 80% horizontal tortuosity; post-treatment: 80% normal, 20% horizontal tortuosity) [16].

Distension of the ONS (Figure 4) presumably reflects high CSF pressure in the optic nerve sheath and can be seen on imaging as a widened ring of CSF surrounding an optic nerve [17]. Definitions for distention of the ONS vary, but a CSF ring > 2 mm is commonly used [18]. Agid et al found this marker to be present in 66.7% of people with IIH and 17.9% of controls [9]. Caglayan et al found that ONS thickness improved following treatment but

remained higher than controls (controls: 5.13 ± 0.41; pre-treatment: 7.08 ± 0.75; post-treatment: 6.53 ± 0.76), and proposed that the ONS may return to its original diameter over time given its elasticity [16].

Changes in the Globe: Posterior Globe Flattening

The normal convexity of the posterior globe is maintained by the equilibrium between the intracranial and intraocular pressure. Posterior globe flattening (Figure 5) refers to the straightening out of the curvature of the posterior sclera in the region where the sclera attaches to the optic nerve [9]. It is thought to occur as a result of elevated CSF pressure that is transmitted from the subarachnoid space, through the ONS, and onto the posterior globe [17]. This mechanical response likely contributes to chorioretinal folds and acquired hyperopia, sometimes seen in people with increased ICP [13]. Agid et al found globe flattening to be present in 43.3% of people with IIH and 0% of controls [9]. The presence and degree of posterior globe flattening can be subjective, as it is determined by individual raters and can be influenced by orientation of the image. Use of a 2D distance map was found by Alperin et al to improve the reliability of this finding by providing a quantitative method of comparison, and was supported by statistically significant differences between control and IIH groups (0.93 ± 0.020 versus 0.91 ± 0.022 (P = .003)) [15]. This marker was found by Caglayan et al to improve with treatment, with MRI findings in subjects with IIH being 10% normal, 50% with flattening of the sclera, and 40% with a concave globe pre-treatment, and improving to 50% normal and 50% flattening of the sclera post-treatment of IIH [16].

Changes in Ventricular Size: Slit-like Lateral Ventricles

Obstructive hydrocephalus with enlarged lateral ventricles is a secondary cause of increased ICP. In contrast, narrowing or collapse of the walls of the lateral ventricles, referred to as slit-like ventricles (Figure 6), have been described in association with IIH. Slit-like ventricles were first noted as a sign of IIH in early studies using CT, but this finding has not been corroborated on MRI and subsequent studies have shown ventricle size to be normal in people with IIH and with a range of variability. Agid et al found this marker to be present in 3.3% of people with IIH and 0% of controls. Today, slit-like ventricles are thought of as a historical finding, given their uncommon occurrence [9].

Changes in the Cerebral Venous Sinuses: Transverse Venous Sinus Stenosis

Recently, venous stenosis observed on magnetic resonance venography (MRV) has gained attention as a cause of IIH as well as a result of increased ICP. A three-dimensional gadolinium- enhanced MRV is considered more sensitive than conventional MRV for detecting areas of subtle cerebral venous stenosis [19].

The cerebral sinuses are the venous channels located between the layers of the dura mater, and stenosis of the transverse sinuses in particular have been associated with IIH. The transverse sinuses of people with IIH can be particularly susceptible to stenosis as ICP increases, due to absent or diminished bony grooves adjacent to the structures, which increases the likelihood of narrowing [17]. In some patients congenital or acquired narrowing of the venous sinuses contributes to IIH by increasing cerebral venous pressure.

Whether transverse sinus stenosis is a primary or secondary finding (or both) of IIH is still under debate, but the reversibility of stenosis with treatment of the increased ICP supports the hypothesis that it is a secondary finding in some patients [20]. Stenting of transverse sinus stenosis in people with IIH has been shown to be a safe and effective treatment supporting its role in causality [21].

Bilateral transverse sinus stenosis (Figure 7) greater than 50% has been found to be an extremely sensitive imaging marker of IIH [20]. Multiple studies have found that severe bilateral transverse sinus stenosis is present on MRV in almost 100% of people with IIH, depending on the definition of transverse sinus stenosis [18].

Clinical Correlations:

While the presence of one or more neuroimaging findings increases the likelihood of IIH, it remains unknown whether the extent of abnormal neuroimaging findings is associated with the severity of clinical symptoms [10,14]. For example, empty sella and optic nerve distention are useful markers for papilledema, the swelling of the optic disc secondary to increased ICP and that is seen in people with IIH, but papilledema grade does not perfectly align with visual field loss or intracranial pressure [22].

In regards to a combination of neuroimaging signs, Maralani et al. reported an increased odds of IIH diagnosis, and hence an improved sensitivity, when looking at any single sign or the combination of partial empty sella turcica, posterior globe flattening, and a combined transverse sinus stenosis score (CSS) <4 [10]. Similarly, Mallery et al found that the presence of any combination of 3 out of 4 signs including reduced pituitary gland height, posterior globe flattening, distended optic nerve sheath, and transverse sinus stenosis had a sensitivity of 64% and a specificity of nearly 100% for increased ICP and is suggestive of IIH without papilledema [23]. Lim et al. additionally reported a sensitivity of 43% and a specificity of 95% for IIH when the presence of three or more cross-sectional MRI signs were seen in a pediatric cohort [24]. It should also be noted that absence of MRI findings does not rule out IIH.

Accuracy of MRI findings:

Each aforementioned neuroimaging finding differs in its sensitivity and specificity when reported individually and when found in combination with other signs. None of the findings discussed have both high sensitivity and specificity when isolated. Studies vary in their reports of these statistics, which may be due to differences in definitions, methods, or emphasis on certain findings, and these reports are summarized in Table 1. It is important to remember that the positive or negative predictive value, which is most relevant for diagnosis, will depend on the prevalence in the population being studied.

Conclusion:

Several MRI based findings including empty sella turcica, optic nerve protrusion, distension of the optic nerve sheath, optic nerve tortuosity, posterior globe flattening and venous sinus stenosis have been found to be associated with IIH, and the specificity/sensitivity improve with combination of markers. Future studies are needed to establish measurement

definitions, normal ranges, and to define how these can be used in the diagnosis and monitoring of IIH.

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Key points:

- MRI and more recently, MRV have been established as important tools in the diagnosis of IIH with regards to excluding secondary causes.
- Empty sella turcica, optic nerve protrusion, vertical tortuosity, optic nerve sheath distension, posterior globe flattening, and transverse sinus stenosis can be caused by high ICP and have high sensitivity and specificity for diagnosis of IIH.
- The presence of multiple imaging markers increases the likelihood of IIH diagnosis, although absence of these findings does not rule it out.

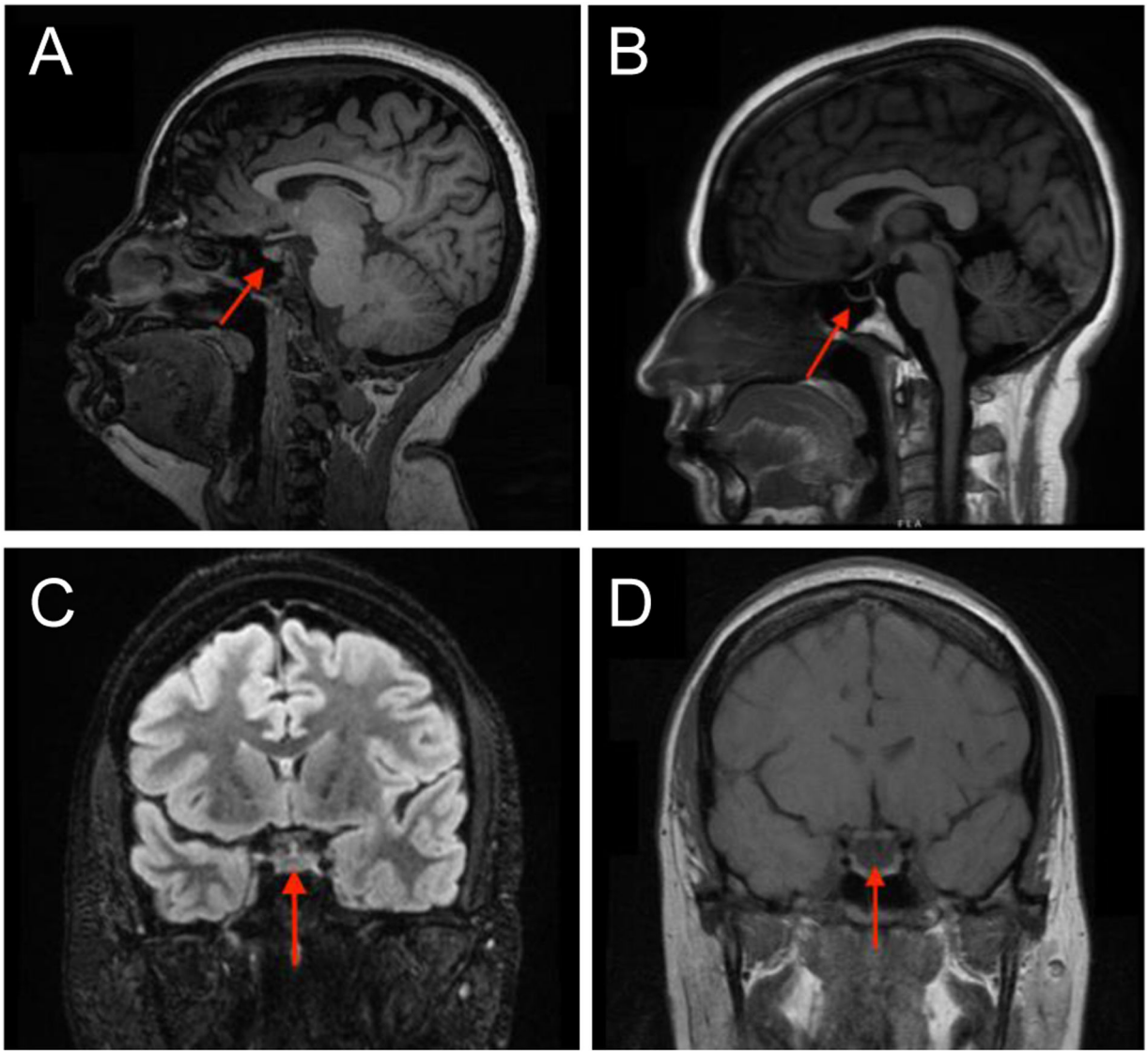

Figure 1:

A. Mid-sagittal T1 noncontrast magnetic resonance imaging (MRI) demonstrating a normal sella turcica (arrow). B. Mid-sagittal T1-weighted MRI demonstrating a partially empty sella turcica (arrow). C. Coronal T2-weighted MRI shows a normal sella turcica (arrow). D. Coronal fluid-attenuated inversion recovery (FLAIR) imaging shows a partially empty sella turcica (arrow).

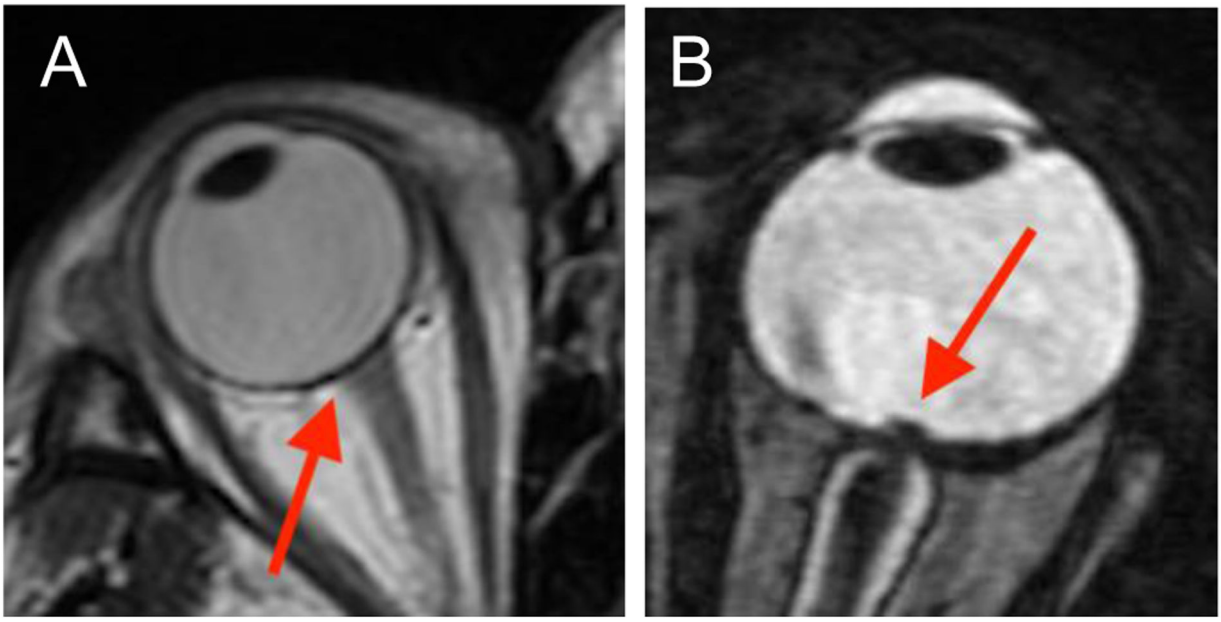

Figure 2:
A. Axial T1-weighted MRI shows normal appearance of the optic nerve head. B. Axial T2-weighted MRI orbit demonstrates a swollen optic nerve head (arrow).

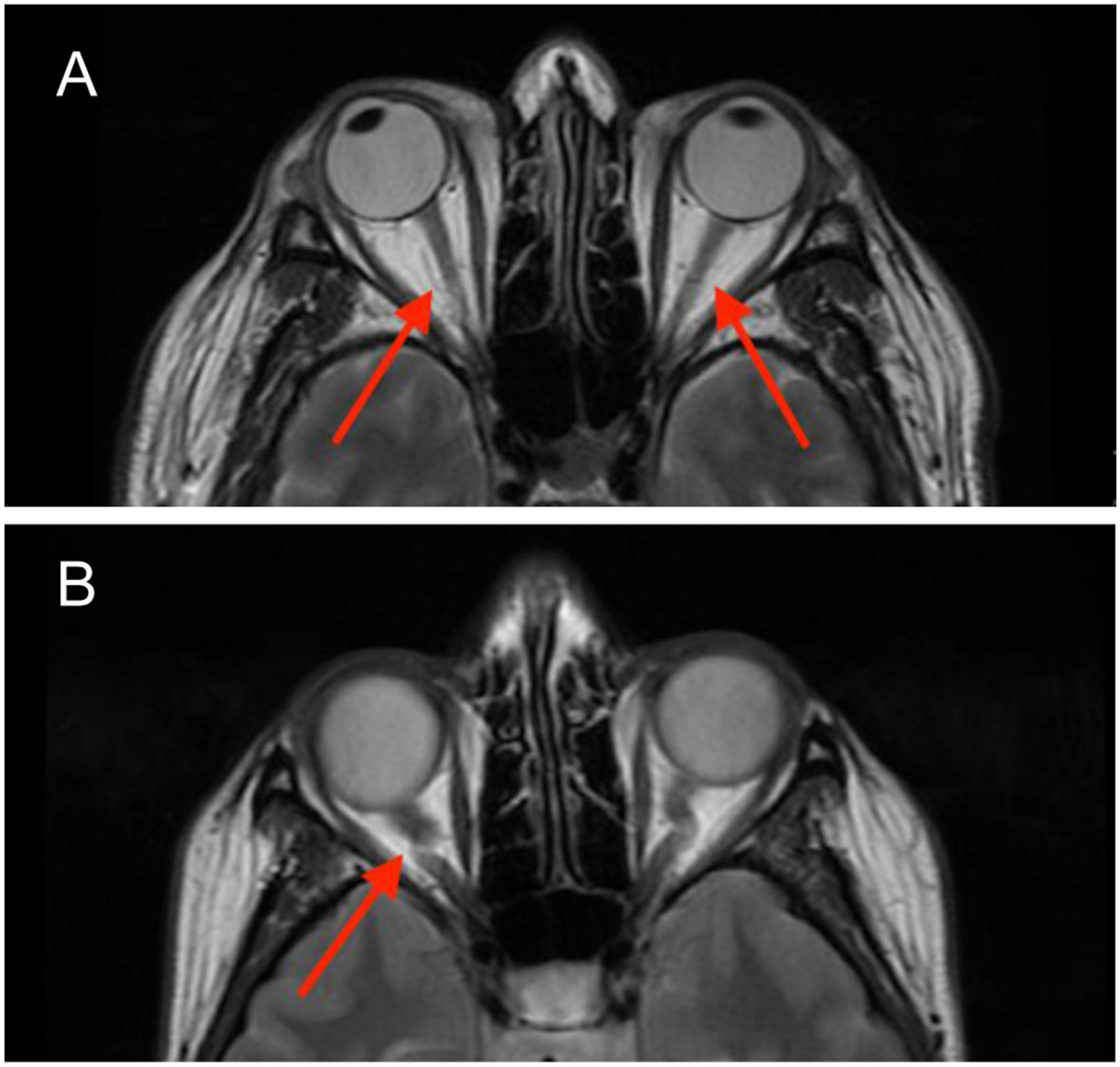

Figure 3:
A. Axial T1-weighted MRI demonstrates normal appearance of the optic nerves (arrows). B. Axial T1-weighted MRI demonstrates tortuosity of the optic nerves and presence of a "smear sign" on the right optic nerve (arrow).

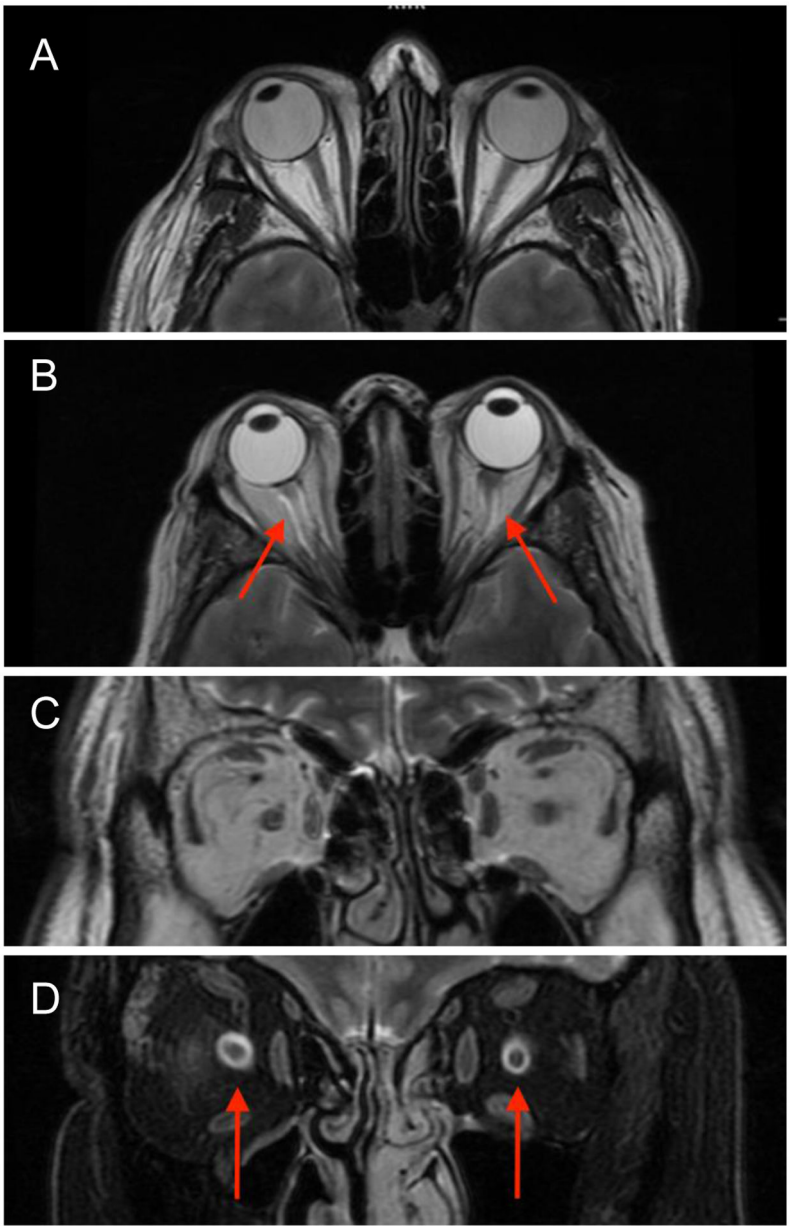

Figure 4:

A. Axial T1-weighted MRI shows normal appearance of the optic nerve sheath. B. Axial T1-weighted MRI demonstrates dilation of optic nerve sheath (arrows). C. Coronal T2-weighted MRI shows normal appearance of the optic nerve sheath. D. Coronal T2-weighted MRI demonstrates an enlarged CSF ring surrounding the optic nerve (arrows).

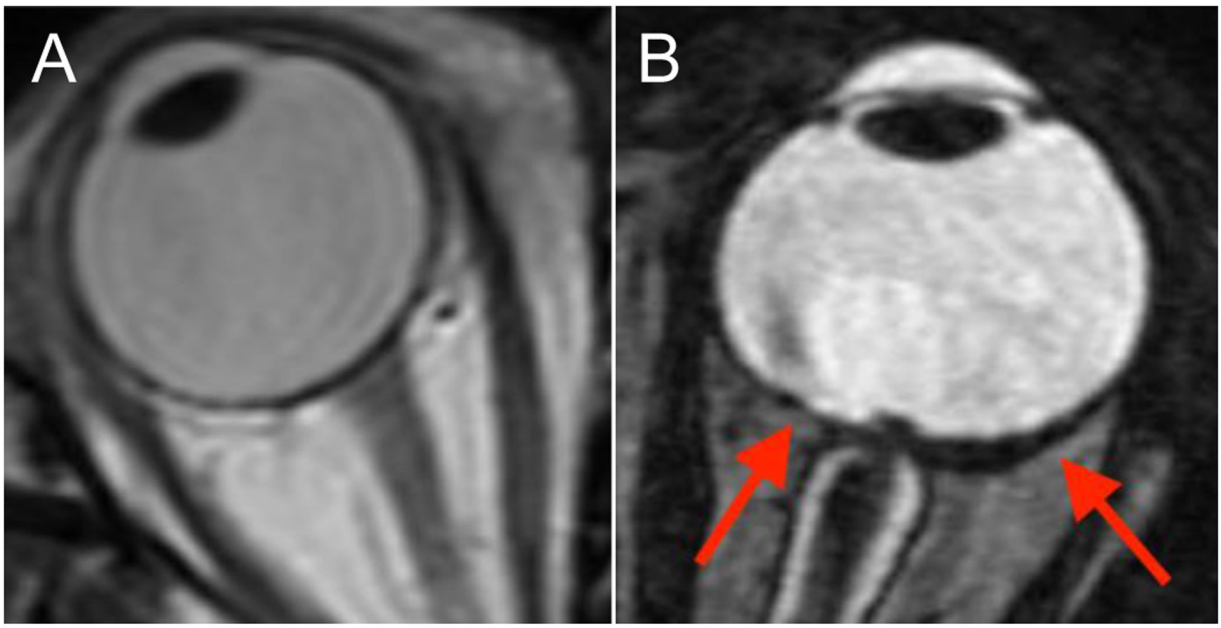

Figure 5:
A. Axial T1-weighted imaging demonstrates normal curvature of the globe. B. Posterior globe flattening is demonstrated on axial T2-weighted imaging (arrows).

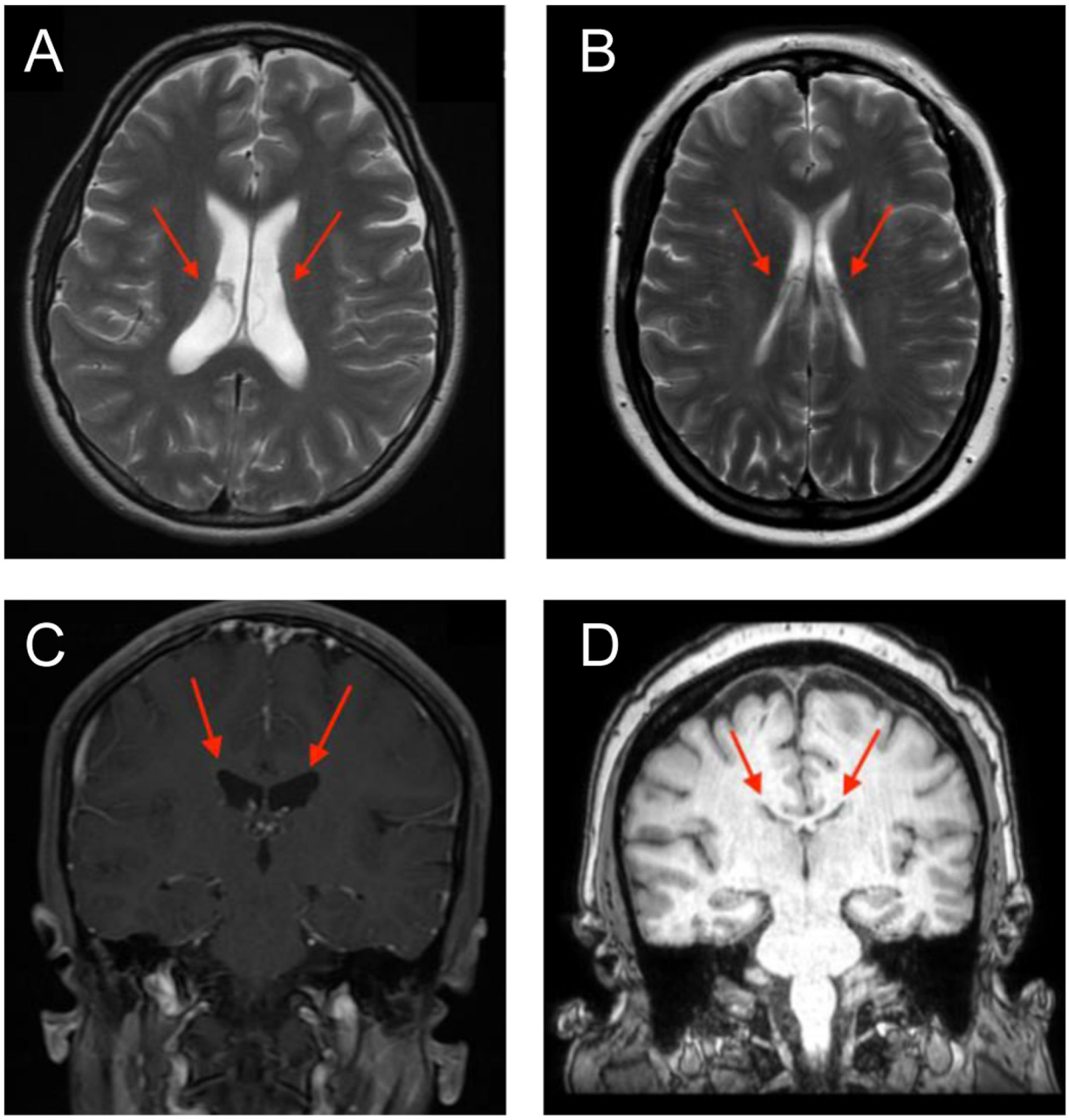

Figure 6:

A. Axial T2-weighted MRI shows normal ventricular size (arrows). B. Axial T2-weighted MRI shows slit-like ventricles (arrows). C. Coronal T2-weighted MRI shows normal ventricular size (arrows). D. Coronal T1-weighted MRI demonstrates the appearance of slit-like ventricles (arrows).

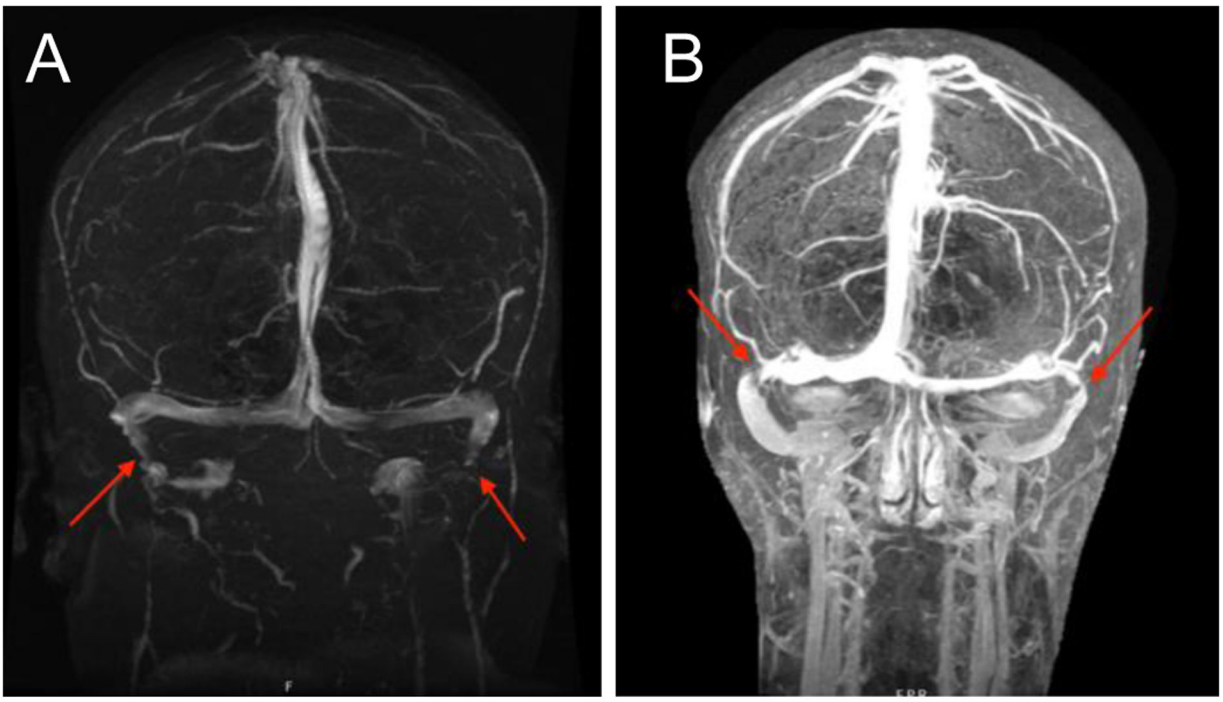

Figure 7:

A. Posterior view of magnetic resonance venography (MRV), Coronal T1 without contrast depicts normal venous structure (arrows). B. MRV with contrast demonstrates bilateral transverse sinus stenosis (arrows).

Table 1:

Imaging Findings in IIH

MRI finding	References	Sensitivity	Specificity
Empty sella	Bidot et al, 2015	80% *	83% *
	Agid et al, 2006	26.7%	94.6%
	Brodsky and Vaphiades, 1998 [25]	70%	95%
Partially empty sella	Maralani et al, 2012	65.1%	95.3%
	Agid et al, 2006	53.3%	75%
Optic nerve tortuosity	Bidot et al, 2015	43% *	90% *
	Maralani et al, 2012	34.9%	86%
	Agid et al, 2006	40%	91.1%
	Brodsky and Vaphiades, 1998	40%	95%
Distended optic nerve sheath	Bidot et al, 2015	58% *	89% *
	Maralani et al, 2012	48.8%	88.4%
	Agid et al, 2006	66.7%	82.1%
	Brodsky and Vaphiades, 1998	45%	95%
Optic nerve head protrusion	Maralani et al, 2012	37.2%	100%
	Agid et al, 2006	3.3%	100%
	Brodsky and Vaphiades, 1998	30%	95%
Posterior globe flattening	Maralani et al, 2012	53.5%	100%
	Agid et al, 2006	43.3%	100%
	Brodsky and Vaphiades, 1998	80%	95%
Slit-like ventricles	Maralani et al, 2012	39.5%	79.1%
	Agid et al, 2006	3.3%	100%
Transverse sinus stenosis	Morris et al.	93%	-
Combined transverse sinus stenosis score (CSS) <4	Maralani et al, 2012	62.8%	100%

*: pooled data from multiple studies