Neuroradiology (1996) 38: 769–773

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MRI of the optic nerve in benign intracranial hypertension

Received: 12 February 1996
Accepted: 2 March 1996

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Abstract We investigated the MRI appearance of the optic nerve and its cerebrospinal-fluid-containing sheath in 17 patients with benign intracranial hypertension (BIH) and 15 normal controls. Using phased-array local coils, 3-mm coronal T2-weighted fat-suppressed fast spin-echo images were obtained with an in-plane resolution of < 0.39 mm. The optic nerve and its sheath were clearly differentiated. An enlarged, elongated subarachnoid space around the optic nerve was demonstrated in patients with BIH. High-resolution MRI of the optic nerve offers additional information which may be of value for diagnosis and in planning and monitoring treatment.

Key words Magnetic resonance imaging · Benign intracranial hypertension · Papilloedema · Optic nerve

Introduction

In benign intracranial hypertension (BIH) there is raised intracranial pressure (ICP), with normal cerebrospinal fluid (CSF) constituents, and no evidence on brain imaging of ventricular obstruction or other causes of raised ICP [1, 2]. The incidence of BIH is 1 in 100,000 [3], it being most frequent in obese young women (19.3 per 100,000, aged 20–44 years). Permanent visual loss due to optic nerve damage occurs in about 10 % of patients [4]. Treatment to reduce ICP may be medical, usually with oral acetazolamide and weight loss [5], or surgical (optic nerve sheath decompression or lumboperitoneal shunting) [6]. Conventional MRI and CT are usually normal in BIH [7–9].

The optic nerve has been technically difficult to image because of its small size: it is 0.4–0.6 cm diameter within the orbit [10]. We used MRI to investigate the optic nerve and its CSF-containing sheath to see whether any abnormality could be identified in this area and how imaging might contribute to diagnosis and management of patients. We present our findings using two recent technical developments – multiarray local coils [11, 12] and the fast spin-echo (FSE) sequence [13, 14] – which make it possible to acquire high-resolution images of the optic nerve in an acceptable time. We used the current maximum resolution for clinical purpose, providing in-plane resolution < 0.39 mm on 3 mm contiguous slices, covering the optic nerve from the globe to the optic chiasm.

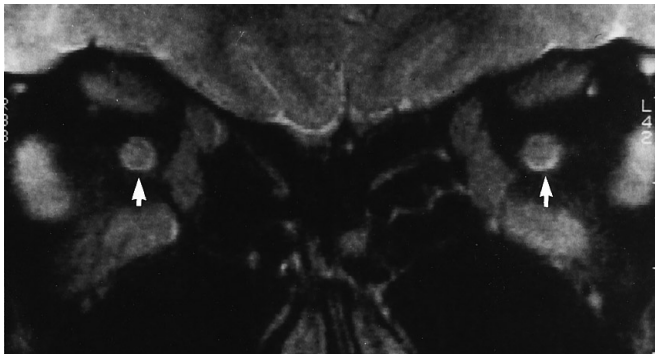


Fig. 1 Coronal T2-weighted fast spin-echo (FSE) fat-suppression image of the mid-orbit of a normal control subject. CSF signal can be identified surrounding the optic nerve *(arrows)*

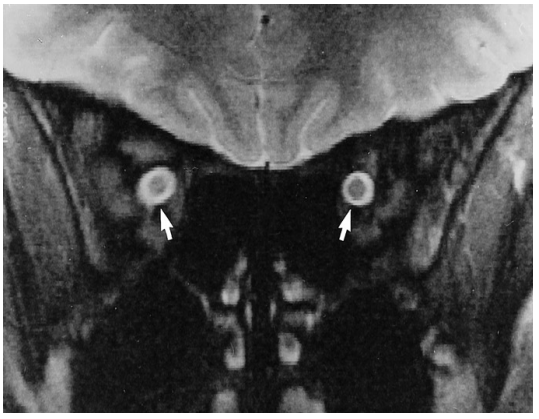


Fig. 2 Coronal T2-weighted FSE fat-suppression image of the posterior orbit in the immediate vicinity of the optic canal in a patient with benign intracranial hypertension. Expansion of the subarachnoid space, with a prominent ring of CSF signal around the optic nerve, can be seen bilaterally *(arrows)*. In normal control subjects the optic nerve sheath narrows from the globe towards the orbital apex and CSF surrounding the optic nerve is normally not seen this far posterior

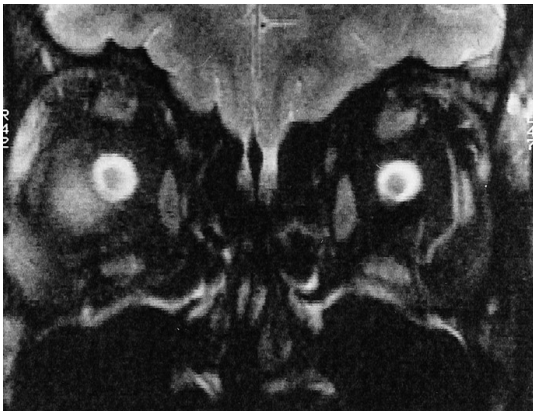


Fig. 3 Coronal T2-weighted FSE fat-suppression image of the anterior orbit in a patient with benign intracranial hypertension. Wide subarachnoid spaces with high signal from CSF are seen at the level of the optic nerve head

Patients and methods

We studied 17 patients (age 18–50 years, mean 32 years, 15 women, 2 men) with BIH, of whom one was studied before and after surgical optic nerve sheath decompression. At the time of the MRI examination all patients had headaches; 5 showed enlarged blind spots and papilloedema was present, bilateral in 15. In the surgically treated patient marked reduction of bilateral papilloedema was noted 2 days after optic nerve sheath decompression at the time of follow-up MRI. No patient had pinhole-corrected visual acuities worse than 6/12. The duration of the disease varied from 4 months to 5 years (mean 22 months). Patients had presented with symptoms frequent in BIH, including headaches, diplopia, transient obscurations, tinnitus and blurred vision. In all patients the diagnosis had been confirmed by raised CSF pressure at lumbar puncture, cranial imaging (CT or routine MRI) and additional diagnostic tests (e.g. angiography) when appropriate. All but one of the patients were being treated medically, and one underwent surgical optic nerve sheath decompression. Informed consent was obtained in writing from all subjects. The research protocol had prior approval of our Joint Medical Ethics Committee. MRI was also performed in a control group of 15 healthy individuals without more than one headache per month (age 20–41 years, 9 women, 6 men).

MRI was performed on a 1.5T unit. After axial T1-weighted localising gradient-echo images, the optic nerves were studied in the coronal plane using a mildly T2-weighted FSE sequence with frequency-selective fat suppression [15–17]. Signal was collected with a pair of 3″ circular coils (provided by General Electric Medical Systems) positioned symmetrically over the temples, such that optimum proximity to the optic nerves was obtained. FSE imaging parameters were: TR 3250/TEef 68 ms, echo-train length 16, 6 excitations, 24 cm rectangular field of view, matrix 512×512, 3-mm interleaved contiguous slices, measurement time 11 : 07 min.

All films were reviewed by an experienced neuroradiologist (I.F.M.). Structured reporting schemes were used assessing anatomical as well as potential pathological detail. The images of patients and normal controls were mixed and reported without knowledge of the clinical details.

Results

The entire length of both optic nerves was imaged in all studies. The intraorbital optic nerve was covered in 8–9 slices, the intracanalicular portion was seen on 2–3, and the intracranial optic nerve and chiasmal junction of the two nerves was covered by 3 slices.

Differentiation of the nerve from surrounding CSF within its dural sheath was possible, as there was strong contrast between the bright CSF and low optic nerve signal in all individuals (Fig. 1). In all cases the largest CSF space was seen in the sections adjacent to the globe. On anterior orbital slices bright CSF signal around the nerve was visible in all studies. The number of coronal slices on which CSF signal was identifiable was symmetrical in normal controls, with considerable variation between individuals, as CSF was visible on 2–

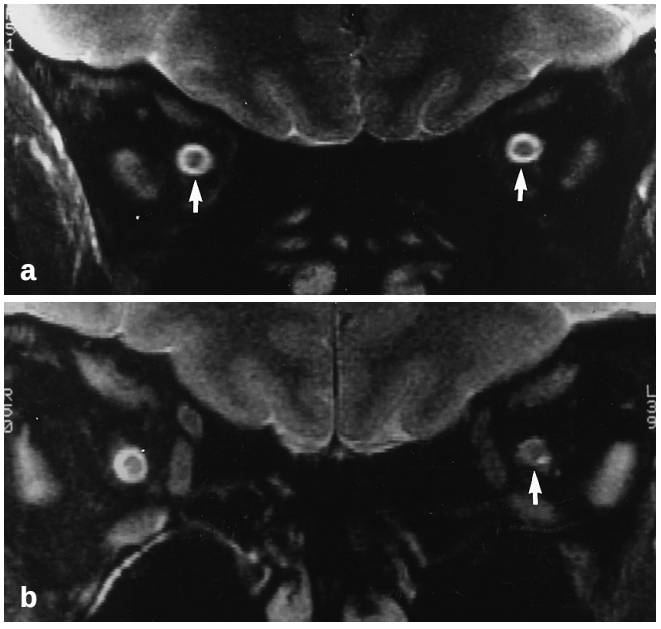


Fig. 4 Coronal T2-weighted FSE fat-suppression images of the posterior orbit in a patient with benign intracranial hypertension **a** before, **b** after surgical optic nerve sheath decompression on the left. Expansion of the subarachnoid space with a prominent ring of CSF signal around the optic nerve can be seen bilaterally *(arrows)* preoperatively. Two days postoperatively the subarachnoid space on the left *(arrow)* has collapsed and no CSF signal is identified around the optic nerve. No definite change of CSF can be seen on the right

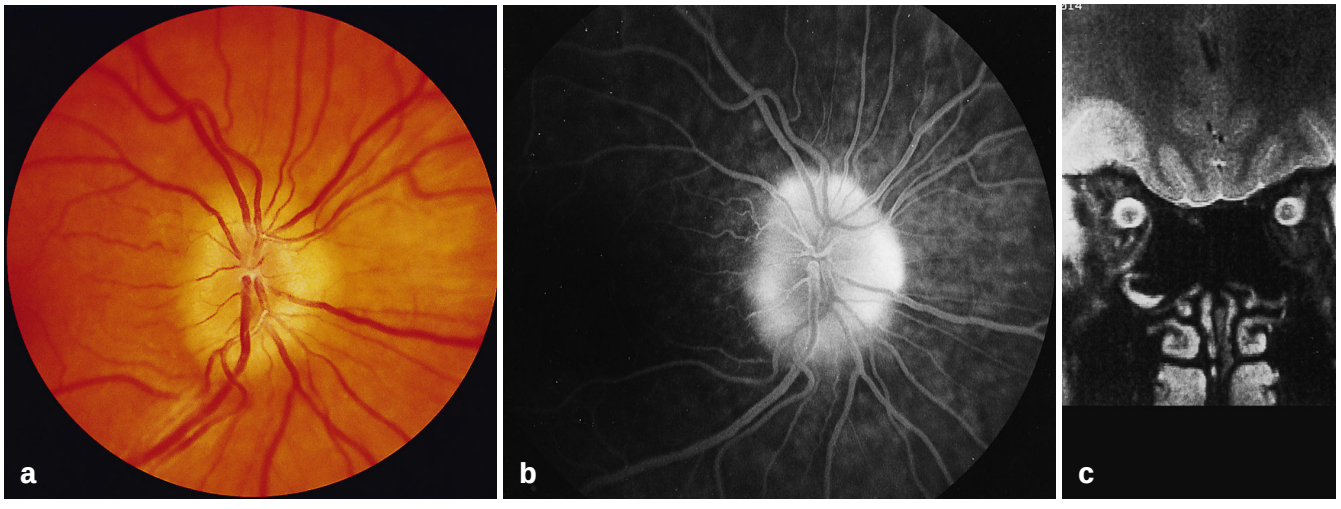
5 slices in the orbit. The width of the sheath narrowed in all individuals from the globe progressively towards the orbital apex, resulting in absent CSF signal in the most posterior orbital portion and the intracanalicular portion of the optic nerve. The optic canal was identified on 2–3 slices in all studies.

The ring of CSF surrounding the optic nerve was seen on more slices (8–11) in patients than in normal controls (2–5) and the sheath also appeared distended. CSF signal was always seen throughout the orbital images (Figs. 2, 3) and in six cases on the first intracanalicular slice. The optic canal was identified surrounding the optic nerve on 2–3 slices, which was not different from the length of the optic canal in normal controls. No intrinsic signal abnormality of the optic nerves was demonstrated and no compressing or deforming effect was visible.

In the patient who underwent decompression of his left optic nerve sheath there was a marked difference between the pre- (Fig. 4a) and postoperative MRI (Fig. 4b). The distention of the sheath demonstrated on preoperative images was no longer present on follow-up. Although the left optic nerve-sheath complex showed a reduction in the extent of CSF signal the right side was unchanged, showing CSF signal on 10 slices on both preoperative and postoperative studies.

In two patients who presented with headaches suggestive of raised ICP, clinical assessment of was inconclusive because of a mild degree of disc swelling. In the first patient, there were no discernible optic disc cups (Fig. 5a), the retinal vascular pattern was anomalous, and the late phase of the fluorescein angiogram showed relatively little leakage (Fig. 5b), so that congenital pseudopapilloedema was considered. In the second the limited degree of disc swelling possible reflected optic atrophy secondary to previous inflammatory optic nerve disease. In both patients CSF pressure was ele-

Fig. 5 **a** Fundus photograph, **b** late phase of fluorescein angiogram of right eye of a 26-year-old woman presenting headaches. The mild of optic disc swelling, the absence of an optic disc cup, and the anomalous pattern of retinal vessels suggested pseudo-papilloedema. However, CSF pressure was elevated at 30 cm, and the optic nerve sheaths were distended **c**. The optic nerves show normal intrinsic signal intensity similar to that of cerebral white matter ▼



vated and optic nerve sheath distention was present on MRI (Fig. 5c).

Discussion

Using high-resolution MRI we have demonstrated features of the optic nerve and its CSF-containing sheath in normal controls and in patients with BIH which cannot be reliably identified with lower resolution MRI. Our main findings were:

1. There is a variation in size and extent of the CSF space surrounding the optic nerve in normal controls. The optic nerve sheath narrows towards the optic canal and no CSF can be identified on the slices to or through the optic canal. The widest CSF space is just behind the globe.
2. In patients with BIH an enlarged CSF space can be identified, most prominent behind the globe and extending as far back as the optic canal.
3. In our group of patients with BIH who did not have severe deterioration of vision, the optic nerve showed no intrinsic signal abnormality.
4. There was no difference in the length of the optic canal between normal controls and patients with BIH.

Occasionally clinical assessment of the optic discs is hampered by the presence of congenital anomalies, which may suggest pseudopapilloedema, or by pre-existing optic atrophy, which may limit the development of disc swelling (Fig. 5). In such cases the demonstration of optic nerve sheath distension on MRI may indicate that there is likely to be raised intracranial pressure [18].

Once the diagnosis of BIH is established the appearance of the discs and in particular the severity of papilloedema is commonly used as a measure of disease severity and response to therapy [19]. However, the degree of papilloedema does not predict the severity of symptoms [2]. This could be related to the normal variation of the width of the posterior part of the optic nerve sheath we demonstrated in normal controls. Increased CSF pressure might produce different disc abnormalities depending on the normal size of the optic nerve sheath.

The optic nerve head is assumed by most workers to be the vulnerable site responsible for most visual symptoms in BIH [2, 19]. Our observation that the CSF space surrounding the nerve is widest just behind the globe would be compatible with this argument; visual obscurations induced by postural changes could be explained by transiently increased CSF pressure in the area of the nerve head.

FSE sequences are highly sensitive, showing intrinsic high signal in both the inflammatory-demyelinating lesion in optic neuritis and in the late stages of anterior ischaemic optic neuropathy [20], conditions normally differentiated from BIH on clinical grounds. Although in patients with more severe visual loss one might see intrinsic signal abnormality of the nerve, we did not find this. The absence of intrinsic signal change in the optic nerve might provide useful information in atypical presentations of BIH. Even small intraorbital optic nerve sheath meningiomas [21], which may produce optic disc swelling as well as visual blurring [22], can be identified using high anatomical resolution.

Given our finding that the optic nerve sheath is narrowest in the optic canal the question arises whether anatomical variations in this area might make individuals more or less susceptible to the development of symptoms and signs from increased ICP, e.g. could a long narrow canal prevent transmission of CSF pressure? This seems unlikely as we found no significant difference between normal controls and patients in this area.

Medical treatment of benign hypertension is largely empirical and surgical intervention, such as decompression of the optic nerve sheath, has been advocated in patients with progressive visual field loss, as it can provide relief of visual symptoms [9, 23, 24]. High-resolution MRI can show that the sheaths are dilated and may relatively safely be incised. In patients with symmetrical visual compromise it may indicate which sheath is wider and should be incised. Postoperatively, the change in the width of the sheath can be shown, as we have demonstrated (Fig. 4). MRI may therefore also serve to follow patients not only over the short term but also over longer periods to evaluate responses to medical or surgical treatment strategies. Open-bore interventional MRI units have recently been introduced to aid microsurgical interventions. Although to our knowledge no such procedure has yet been performed, the safety and success rate of optic nerve sheath decompression might be increased using intraoperative MRI information.

MRI and MR angiography (MRA) are often requested in the investigation of patients with BIH. The technique we have used can be added without too much time penalty (approx. 15 min) to a conventional MRI and MRA examination. It offers additional information which may be of value both in diagnosis and in planning and monitoring treatment.

Acknowledgements The NMR Research Group is funded by a generous grant from the Multiple Sclerosis Society of Great Britain and Northern Ireland, and supported by the Brain Research Trust and the Medical Research Council. We are grateful for the provision of fast spin-echo and phased-array local coils by General Electric, Milwaukee, USA.

References

1. Wall M (1991) Idiopathic intracranial hypertension. Neurol Clin 9: 73–95
2. Wall M, George D (1991) Idiopathic intracranial hypertension. Brain 114: 155–180
3. Durcan FJ, Corbett JJ, Wall M (1988) The incidence of pseudotumor cerebri: population studies in Iowa and Lousiana. Arch Neurol 45: 875–877
4. Corbett JJ, Savino PJ, Thompson HS, et al (1982) Visual loss in pseudotumor cerebri: follow-up of 57 patients from 5 to 41 years and a profile of 14 patients with permanent severe visual loss. Arch Neurol 39: 461–474
5. Corbett JJ, Thompson HS (1989) The rational management of idiopathic intracranial hypertension. Arch Neurol 106: 1049–1051
6. Horton JC, Seiff SR, Pitts LH, et al (1992) Decompression of the optic nerve sheath for vision-threatening papilledema caused by dural sinus occlusion. Neurosurgery 31: 203–211
7. Moser FG, Hilal SK, Abrams G, et al (1988) MR imaging of pseudotumor cerebri. AJR 150: 903–909
8. Silbergleit R, Junck L, Gebarski SS, et al (1989) Idiopathic intracranial hypertension: MR imaging. Neuroradiology 170: 207–209
9. Weisberg LA (1985) Computed tomography in benign intracranial hypertension. Neurology 35: 1075–1078
10. Kupfer C, Chumbley L, Downer J (1967) Quantitative histology of optic nerve, optic tract and lateral geniculate ganglionucleus of man. J Anat 101: 393–401
11. Hayes CE, Roemer PB (1990) Noise correlations in data simultaneously acquired from multiple surface coil arrays. Magn Reson Med 16: 181–191
12. Roemer PB, Edelstein WA, Hayes CE, et al (1990) The NMR phased array. Magn Reson Med 16: 192–225
13. Constable RT, Anderson AW, Zhong J, et al (1992) Factors influencing contrast in fast spin-echo MR Imaging. Magn Reson Imaging 10: 497–511
14. Hennig J, Naureth A, Friedberg H (1986) RARE imaging: a fast imaging method for clinical MR. Magn Reson Med 3: 823–833
15. Simon J, Szumowski J, Tottermann S, et al (1988) Fat suppression MRI of the orbit. AJNR 9: 961–968
16. Miller DH, MacManus DG, Bartlett PA, et al (1993) Detection of optic nerve lesions in optic neuritis using frequency selective fat saturation sequences. Neuroradiology 35: 156–158
17. Gass A, Barker GJ, Moseley IF, et al (1994) High resolution MRI of optic neuropathies. Neurology 44: A267
18. Marcelis J, Silberstein SD (1991) Idiopathic intracranial hypertension without papilledema. Arch Neurol 48: 392–399
19. Orcutt JC, Page NG, Sanders MD (1984) Factors affecting visual loss in benign intracranial hypertension. Ophthalmology 91: 1303–1312
20. Miller DH, Newton MR, Poel JC van der, et al (1988) Magnetic resonance imaging of the optic nerve in optic neuritis. Neurology 38: 175–179
21. Lindblom B, Truwit C, Hoyt WF (1992) Optic nerve meningioma: definition of intraorbital, intracanalicular, and intracranial components with magnetic resonance imaging. Ophthalmology 99: 560–566
22. McDonald WI (1982) The symptomatology of tumors of the anterior visual pathways. Can J Neurol Sci 9: 381–390
23. Kelman SE, Heaps R, Wolf A, et al (1992) Optic nerve decompression surgery improves visual function in patients with pseudotumor cerebri. Neurosurgery 30: 391–395
24. Corbett JJ, Nerad JA, Tse DT, Anderson RL (1988) Results of optic nerve sheath fenestration for pseudotumor cerebri. The lateral orbitotomy approach. Arch Ophthalmol 106: 1391–1397