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MR of Optic Papilla Protrusion in Patients with High Intracranial Pressure

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PURPOSE: To evaluate the signal characteristics of the optic papilla (optic nerve head) on routine cranial MR images in patients with clinical evidence of optic papilla elevation caused by high intracranial pressure, and to compare these findings with findings in healthy adult volunteers. **METHODS:** We reviewed retrospectively the MR imaging examinations of 15 patients who were referred with objectively decreased visual acuity and funduscopic findings of optic papilla elevation. T1-weighted and T2-weighted axial MR images were obtained by using conventional spin-echo acquisitions on 1.5-T MR imagers. In addition, the MR imaging studies in 10 healthy adult volunteers without visual impairment were reviewed as controls. **RESULTS:** In 10 (67%) of the 15 patients, visual elevation of the optic papilla was shown by MR imaging. In all 15 patients, the MR signal intensity of the optic papilla was hypointense relative to the vitreous of the globe on T2-weighted images. In the healthy volunteer group, the optic papillae were all similarly hypointense relative to the vitreous of the globe on T2-weighted images; however, these optic papillae were flat. **CONCLUSION:** Clinical examination and MR imaging may show elevation of the optic papilla in patients with high intracranial pressure. When chronic, optic papilla elevation has been shown to correlate well with severe loss of vision. Actual edema of the optic papilla seems to play little role in the physical elevation observed clinically in the chronic stages of this pathologic process.

Index terms: Hypertension; Nerves, magnetic resonance; Nerves, optic [III]

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In patients with so-called papilledema, funduscopy shows elevation of the optic papilla as a protrusion of the optic nerve head into the posterior aspect of the globe. It was previously postulated that intracellular fluid accumulation (ie, true edema) within the optic papilla contributed in large part to this protrusion. However, if intercellular or intracellular edema is a contributing factor in the subacute or chronic stage of this process, then there might be evidence of optic head edema (ie, hyperintensity) on T2-weighted magnetic resonance (MR) images.

The purpose of this study was to evaluate the signal characteristics of the optic papilla on cranial MR images of patients with clinical evidence of elevation of the optic papilla.

Materials and Methods

We reviewed retrospectively the routine cranial images of 15 patients who were referred for MR imaging and who had subjectively and objectively decreased visual acuity and objective funduscopic findings of optic papilla elevation. The mean age of the patients was 26.4 years (age range, 10 to 60 years); 4 patients were male and 11 were female. The images were acquired on 1.5-T MR units. Axial images (section thickness, 5 mm; intersection gap, 1.5 mm) were obtained with T1 weighting (500/20/2 [repetition time/echo time/excitations]) and T2 weighting (2400/30-80/1); conventional spin-echo acquisitions, a 256 × 256 matrix, and a 24-cm field of view were used. To avoid chemical shift artifact in the region of the optic papilla, we applied frequency encoding in the anteroposterior axis of the cranium. The configuration and MR signal intensity of the optic papillae were evaluated for evidence

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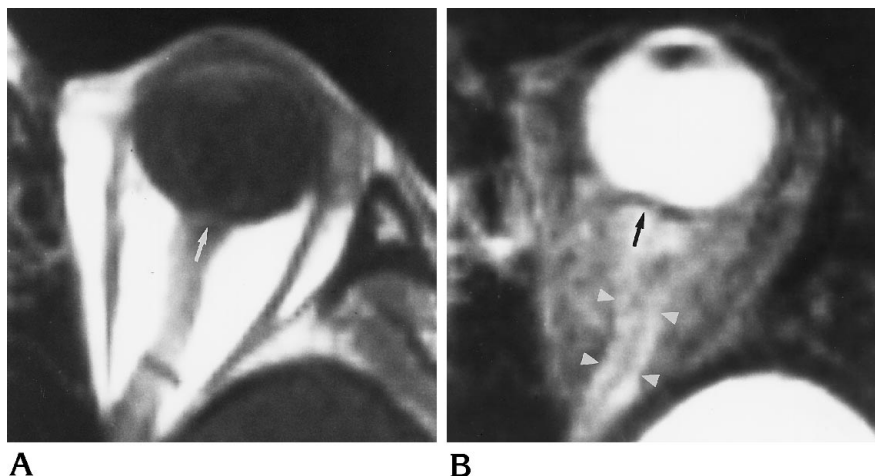
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Fig 1. MR images of optic papilla protrusion caused by cranial hypertension in a patient with subependymoma, arachnoid cyst, and hydrocephalus.

A, Axial T1-weighted (500/20/2) conventional spin-echo image through the left orbit shows ballooning of the optic papilla (*arrow*), causing it to protrude into the posterior aspect of the globe.

B, Axial T2-weighted (2400/80/1) conventional spin-echo image through the left orbit shows hyperintense cerebrospinal fluid in the optic nerve sheath (*arrowheads*) associated with hypointense optic papilla (*arrow*), which protrudes into the posterior aspect of globe.



of abnormalities; specifically, the optic papilla of each patient was analyzed to determine whether it was flat or inwardly concave and whether its signal on T2-weighted images was isointense, hypointense, or hyperintense relative to the vitreous of the globe. Because this was a retrospective study of routine cranial images, no special high-resolution, surface-coil, or fat-suppression techniques were used in the imaging of the orbits.

As a means of control, we reviewed the MR examinations in 10 adult volunteers who had neither visual complaints nor other known intracranial abnormalities. The imaging parameters were the same as those used in the imaging of the patients, and the same analysis of the optic papilla was carried out.

Results

The optic papillae in the volunteer group were universally flat, and the signal of the optic papillae in all volunteers was hypointense relative to the vitreous of the globe on T2-weighted, conventional spin-echo MR images. The remainder of the cranial MR examination showed no abnormalities in the volunteer group.

In all 15 (100%) of the patients, the signals of the optic papillae on T2-weighted, conventional spin-echo images were hypointense relative to the vitreous of the globe (Figs 1B and 2C). Visible protrusion of the optic papilla into the

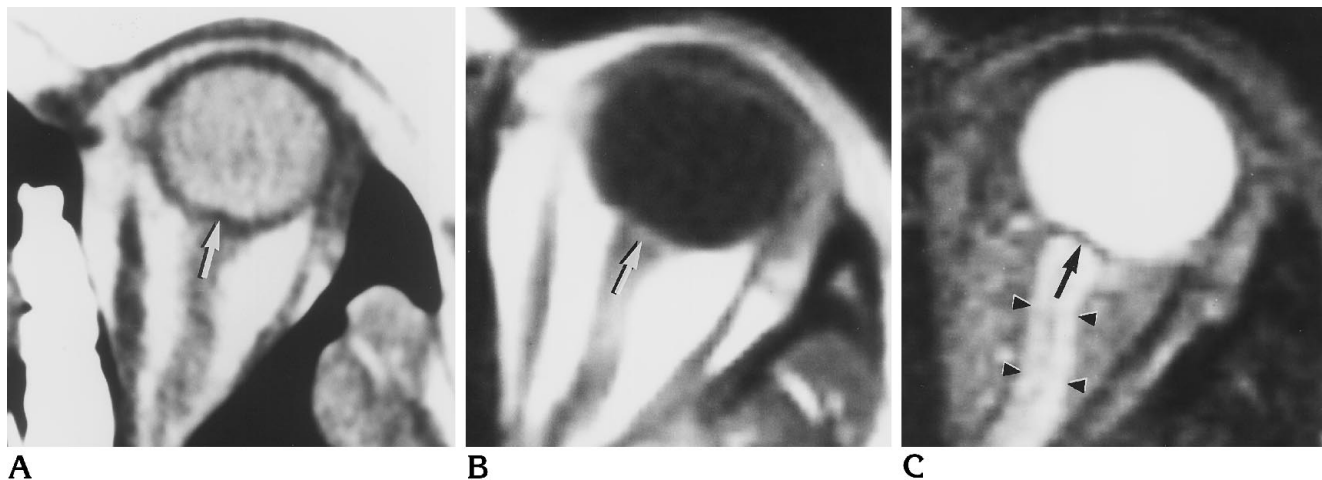


Fig 2. Optic papilla protrusion caused by cranial hypertension in patient with right transverse sinus thrombosis with associated venous hemorrhage.

A, Axial CT scan through left orbit, magnified and filmed on video-reverse (window/level, 400/50), shows protrusion of optic papilla (*arrow*) into the posterior aspect of the globe.

B, Axial T1-weighted (500/20/2) conventional spin-echo MR image through the left orbit shows protrusion of the optic papilla (*arrow*) into the posterior aspect of the globe.

C, Axial T2-weighted (2400/80/1) conventional spin-echo MR image through the left orbit shows hyperintense cerebrospinal fluid in optic nerve sheath (*arrowheads*). The hypointense optic nerve head (*arrow*) protrudes into the posterior aspect of the globe.

posterior aspect of the globe was seen in 10 (66.7%) of the patients (Figs 1 and 2). On the basis of clinical criteria and imaging findings, the patients had final diagnoses of various pathologic processes or states, including cerebral venous sinus thrombosis ($n = 1$), neoplasia ($n = 3$), and primary pseudotumor cerebri ($n = 11$). Repeat imaging evaluation after conservative or surgical therapy was not a part of this study.

Discussion

The meningeal covering of the brain normally continues into the orbit to surround the optic nerve. These perioptic meninges, or the optic nerve sheath, continue all the way to the globe. For this reason, pressure changes in the intracranial space may be transmitted to the optic papilla via the subarachnoid space accompanying the optic nerve sheath.

The term *papilledema* has been widely interpreted as a swollen optic papilla caused by generalized edematous expansion (1). While this phenomenon may occur (eg, optic neuritis), actual physical elevation (protrusion) of the optic papilla into the globe in the absence of edema is encountered more frequently. This type of optic papilla protrusion is usually seen in patients with chronically high intracranial pressure. Most commonly, this raised intracranial pressure is caused by pathologic states such as intracranial neoplasia, idiopathic or benign intracranial hypertension (pseudotumor cerebri), cranial trauma, intracranial inflammation, and some forms of hydrocephalus (1, 2). In this circumstance, direct transmission of the elevated intracranial pressure results in a ballooning of the optic papilla, causing it to protrude physically into the posterior aspect of the globe (3, 4). Moreover, a variable amount of associated intracellular or intercellular fluid may accumulate (ie, true edema) within the optic papilla because of one or more factors, including the cellular edema that accompanies tissue ischemia (5, 6), transarachnoid transudation of fluid, or extravascular fluid extravasation (5, 7). However, in patients with chronically high intracranial pressure, the major factor in disk elevation is the physical protrusion of the optic papilla (8).

If the pressure exerted on the optic papilla is sufficiently severe or chronic, varying degrees of permanent degeneration of the optic nerve may be seen in the optic papilla in response to

this mechanical injury. In a study of patients with pseudotumor cerebri, Gibby et al (9) showed that the majority of the patients for whom computed tomography (CT) showed a reversal (ie, protrusion) of the optic papilla had severe vision loss. Hayreh (10) suggested that vision loss occurs in chronic papilla elevation from perilaminar ischemia caused by the mechanically induced optic papilla protrusion. Thus, timely diagnosis is important because papilla protrusion may presage progressive, irreversible blindness (11, 12).

In 67% of patients in the present series, MR imaging showed a reversal of the spatial orientation of the optic papilla. Funduscopy had shown disk elevation to some degree in all these patients, so the clinical examination was more sensitive than the MR imaging studies. Moreover, these MR results are inferior to the published results of studies that used thin-section CT for detection of protrusion of optic papilla. This difference can be explained by the lower resolution of routine cranial MR imaging. The use of high-resolution, surface-coil MR techniques should improve the sensitivity of MR imaging in the detection of optic papilla protrusion.

In a typical case from the present study, T2-weighted spin-echo MR images showed the signal of the optic papilla to be hypointense relative to the vitreous of the globe in both volunteers and in patients (Figs 1B and 2C). These results suggest that true edema of the optic papilla was not a major component of the process of chronic optic papilla elevation shown by funduscopy in these patients. However, these findings do not preclude an element of true cellular or interstitial edema during the acute phase of high intracranial pressure (13). Further work needs to be done concerning the acute stages of optic papilla protrusion.

Therefore, on the basis of MR imaging data, we believe that an increased amount of fluid within the optic papilla in chronic papilla elevation is not high enough to cause prolongation of the T2-weighted MR signal. The precise reason for shortening of the T2-weighted MR signal in the optic papilla in both normal and abnormal cases is not understood and requires further in vitro study. Nevertheless, because the optic papilla is largely composed of myelinated axons and because white matter is relatively hypointense on T2-weighted images, some degree of low signal intensity is to be expected.

Because the frequency-encoded axis was in the anteroposterior direction, chemical shift artifact did not affect our results. At no point did orbital fat have an interface with the optic papilla along this axis. However, if the optic globe moved during MR imaging, the hyperintense signal from the vitreous of the globe could interfere with the signal intensity of the relatively small optic nerve and optic papilla on T2-weighted images.

In summary, in patients with high intracranial pressure, MR images may reveal physical protrusion of the optic papilla into the vitreous of the globe caused by the transmission of the increased CSF pressure along the subarachnoid space surrounding the optic nerve. Because the optic papilla was shown adequately in all cases and because it was universally hypointense, we think that actual edema (ie, MR signal hyperintensity on T2-weighted images of the optic papilla) was not very important in this process in our group of patients.

Because optic papilla protrusion is associated with severe and often permanent visual loss, it should be considered as a possibility in all patients with clinically suspected cranial hypertension. In the future, high-resolution MR imaging of the optic globe may clarify the difference between true swelling of the optic papilla caused by intrinsic disease (eg, optic neuritis) and disk elevation resulting from physical protrusion (as seen in cases of cranial hypertension).

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