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Sensitivity of orbital magnetic resonance imaging in acute demyelinating optic neuritis

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Abstract

Objective: to determine the sensitivity of orbital magnetic resonance imaging (MRI) in acute demyelinating optic neuritis (ON) in routine clinical practice, and the added value of a dedicated neuroradiology interpretation.

Design: Retrospective chart review

Participants: Patients with clinically proven ON evaluated between 2004 to 2014 in the University of Michigan Neuro-ophthalmology clinics. Inclusion criteria involved visual recovery and orbital MRI competed within 30 days of symptom onset and prior to corticosteroid treatment.

Methods: Demographics, clinical examination, and MRI report data (high T2 signal, gadolinium contrast enhancement) was abstracted for each eligible eye. Every MRI was re-interpreted by a neuroradiologist masked to the affected side. Descriptive statistics summarized patient and eye characteristics. Inter-rater agreement between the neuroradiologist and the radiology report for the radiographic diagnosis of ON was assessed with Cohens kappa statistic.

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Results: Of 92 patients who met all inclusion criteria, 70 (76.1%) were reported to have at least 1 MRI feature consistent with ON. After dedicated review by a neuroradiologist, 77 (83.7%) were determined to have a positive MRI for ON. Agreement between the neuroradiologist and MRI report was moderate (κ=0.63). Gadolinium enhancement was the most common feature in MRI positive ON (72 [78.3%] of neuroradiology reviewed MRIs; 66 [71.7%] of clinical MRI reports).

Conclusions: The sensitivity of MRI in ON was lower than previously reported and confirms the importance of making a clinical diagnosis of ON without relying on neuroimaging for confirmation. MRI interpretation by a skilled neuroradiologist increased sensitivity, underscoring the complexity of orbital MRI interpretation.

Keywords

optic neuritis; MRI; diagnosis; optic nerve

Introduction

Acute demyelinating optic neuritis (ON) affects over 20,000 people annually in the US, primarily between the ages of 20 and 40. ON is the presenting feature in 20% of multiple sclerosis (MS) patients and half of all MS patients experience ON during the course of their disease.[1, 2] Although MRI is not required for the diagnosis of ON, brain MRI is necessary to assess future MS risk and orbital imaging is often acquired at the same time. Therefore, orbital MRI is increasingly being relied on to confirm the diagnosis of ON, particularly when the clinical picture is uncertain.

The MRI radiographic features of ON include T1 weighted gadolinium enhancement, high T2 signal, or enlargement of the affected optic nerve.[3, 4] Animal models have shown that gadolinium contrast enhancement appears at 3 days, peaks at 10–14 days and persists for at least 30 days.[5] T2 signal alteration may appear later than enhancement.[5] Human studies show persistent contrast enhancement in ON for a median of 63 days and up to 113 days.[4] In acute demyelinating lesions in the brain, high dose steroids can suppress gadolinium contrast enhancement within 1 day of treatment.[6]

Prior studies have reported that orbital MRI is 88 −100% sensitive for detection of ON.[7–10] Our experience suggests that MRI-negative ON is more common in routine clinical practice than these studies indicate. This discrepancy may lead some less experienced clinicians to question a diagnosis of optic neuritis if an orbital MRI is reported as normal, leading to unnecessary investigations. A recent, small prospective study of MRI findings in a first episode of ON detected optic nerve lesions in only 80.6% of cases.[11] The aim of this study was to determine the sensitivity of orbital MRI in a larger group of patients with acute demyelinating ON, diagnosed using rigorous clinical criteria.

Methods

A retrospective analysis of the clinical records and neuroimaging of patients presenting to the neuro-ophthalmology service at the University of Michigan with ON was performed. All patients with ON evaluated in the University of Michigan neuro-ophthalmology clinics from

January 2004 to December 2014 were identified using the following ICD-9 codes: 377.3 (optic neuritis), 377.31 (optic papillitis), 377.32 (retrobulbar neuritis), 377.39 (other optic neuritis), 377.49 (other disorders of optic nerve), 377.9 (unspecified disorder of optic nerve). All instances of these diagnostic codes were reviewed by one author (LB) and discarded if the final assessment was anything other than ON. The diagnosis of ON was confirmed using criteria based on the Optic Neuritis Treatment Trial (ONTT).[8] Inclusion criteria were age over 18 years, acute unilateral visual symptoms with orbital MRI within 30 days of onset, relative afferent pupillary defect (unless there was a history of ON in the fellow eye) and visual field defect in the affected eye. Patients were excluded if they had a previous episode of ON in the affected eye or other systemic disease that might be the cause of ON. No patients with a prior diagnosis of MS were included because there were no instances where these patients underwent orbital imaging within 30 days of symptom onset.

Our criteria differed from the ONTT in a few key ways. Patients were not required to present within 8 days of symptom onset, could be included if they had been previously treated with corticosteroids for ON in the fellow eye and if they were over the age of 46. Because our study was not judging response to therapy, the short window of presentation was not deemed necessary. All patients underwent MRI brain and orbits with and without contrast within 30 days of symptom onset without corticosteroid treatment prior to MRI, and demonstrated visual recovery on follow up visits, which is a clinical hallmark of most patients with acute demyelinating ON.[12] A period of 30 days was selected based on animal and human studies of optic nerve enhancement duration.[4,5]

Visual recovery was assessed at the visit closest to 3 months after symptom onset. Using criteria adapted from the Ischemic Optic Neuropathy Decompression Trial, visual recovery was defined as two or more lines of improvement in Snellen visual acuity, or more than 2dB of improvement in mean deviation on 24–2 Humphrey visual fields if presenting vision was 20/30 or worse.[13] In patients with presenting visual acuity of 20/25 or better, recovery was defined as improvement of one or more lines of Snellen visual acuity or more than 2dB in mean deviation on 24–2 Humphrey visual fields.

All orbital MRIs performed at the University of Michigan used a 1.5 or 3.0 Tesla magnet with a 3mm or 2.5mm slice thickness respectively. Orbital pre-contrast sequences included coronal T2, coronal and axial T1 with and without fat saturation. Orbital post-contrast sequences included coronal T1 with fat saturation, and axial T2 with and without fat saturation. Any MRI performed outside of the University of Michigan was included in the study only if comparable orbital sequences were included. Data was abstracted from each MRI radiology report. Because the MRIs and reports were generated as part of the patient's clinical care, the radiologists that generated the reports were not masked to the clinical information. However, each MRI was additionally reviewed by a single, fellowship trained neuroradiologist (MP) who was masked to the side of the clinical symptoms. The neuroradiologist reviewer also assessed MRI quality. The orbital MRI was considered "positive" for ON if the optic nerve on the affected side demonstrated high T2 signal within the nerve or post-contrast enhancement on T1 weighted sequences.

Descriptive statistics of the sample were used to summarize patient and eye characteristics, including means and standard deviations (SD) or medians and interquartile ranges (IQR) for continuous measures, and frequencies and percentages for categorical measures. Snellen visual acuities were converted to logMAR for the purpose of analysis, but converted back to Snellen for reporting.[14] Patients with light perception or no light perception vision were not included in average estimates of visual acuity since visual acuity cannot be accurately estimated. The percentage of patients with an abnormal MRI was calculated overall and for each MRI finding for both the clinical reports and the masked neuroradiology reviewer. Differences by MRI result were assessed using bivariate logistic regression for continuous and categorical variables. Inter-rater agreement between the neuroradiologist (MP) and the radiology report for the radiographic diagnosis of ON was assessed with Cohens kappa statistic. Kappa values of 0–0.33 were considered weak agreement, 0.34–0.66 as moderate agreement, and 0.67–1.00 as strong agreement. Statistical analysis was conducted using the statistical program Stata v. 13 (College Station, TX).

Ethics approval was obtained for this study from the local Institutional Review Board. The study was deemed to be HIPAA-compliant and adhered to the tenets of the Declaration of Helsinki.

Results

We reviewed the charts of 410 total patients. Of the 210 patients diagnosed clinically with ON at the University of Michigan during the study period, 92 met all inclusion criteria. Thirty patients were excluded because the MRI occurred more than 30 days after symptom onset, 26 patients had an MRI of the brain only, 29 had MRI reports but no images available for review, 11 were seen in consultation only with no follow up, and 24 patients had inadequate visual recovery to meet our criteria. Of the included patients, 67 (72.8%) were women and most were white (n=77, 83.7%). Optic disc edema was present in 29 (31.5%) patients and 66 (71.7%) reported pain with eye movement. The median age at diagnosis was 38.7 years (IQR: 31.8–50.0 years) and a median of 11.5 days (IQR: 7.0–19.0 days) elapsed between the onset of symptoms and the MRI. At the time of diagnosis, the mean best corrected visual acuity was 0.960 logMAR (SD 0.949, range −0.125–3.00; Snellen equivalent 20/182, range 20/15-HM). Four subjects had light perception vision and 1 had no light perception vision. The mean Humphrey visual field mean deviation was −14.05dB (SD 8.46; range −33.2- −0.41dB; 31 missing). The follow up visit occurred at a median of 65.0 days after symptom onset (IQR 35.0–152 days). The mean best corrected visual acuity at the follow-up visit was 0.148 logMAR (SD 0.319, range −0.125–2.00; Snellen equivalent 20/28, range 20/15-count fingers) and mean Humphrey visual field mean deviation was −4.63dB (SD 4.83; range −19.48– 2.27dB; 21 missing). Twenty-three patients (25.0%) were diagnosed with multiple sclerosis during the follow-up period.

Four MRIs were degraded by some motion artifact per the neuroradiologist’s assessment, but only 1 was thought to be of poor quality. In 3 cases, there was discordance between the neuroradiologist’s masked interpretation and the clinically affected nerve. In 2 of those cases, the neuroradiologist noted T2 changes which were consistent with the patient’s history of ON affecting the fellow eye previously. In the remaining case, the neuroradiologist

was uncertain if a finding of optic nerve enhancement on the appropriate side was secondary to artifact. After accounting for this discordance, 77 (83.7%) demonstrated at least one MRI feature consistent with ON upon review by a neuroradiologist versus 70 (76.1%) based on the MRI report. Agreement between the neuroradiologist and clinical MRI report was moderate (κ=0.63) and the neuroradiologist had generated 4 of the original clinical MRI reports. Patient characteristics by MRI result are described in Table 1. In patients presenting with pain on eye movement, the odds were significantly higher of having a positive MRI (OR 3.75 [95%CI 1.19–11.75]; P=0.023). Gadolinium contrast enhancement was the most common feature in MRI positive ON, seen in 72 (78.3%) of neuroradiology reviewed MRIs and 66 (71.7%) of clinical MRI reports. T2 hyperintensity was seen in 68 (73.9%) of neuroradiology reviewed MRIs and noted in 44 (47.7%) of clinical MRI reports. Both features were found concurrently in 64 (69.6%) of neuroradiology reviewed MRIs and 40 (43.5%) clinical MRI reports. No imaging features suggestive of ON were identified in 15 (16.3%) cases. The clinical characteristics of patients with a negative MRI are listed in Table 2.

There were 27 patients older than age 46 at the time of symptom onset. Among the older group, 24 (88.9%) had a positive MRI when reviewed by a neuroradiologist versus 21 (77.8%) with positive findings noted in the clinical MRI report. On review by the neuroradiologist, all 24 positive MRIs demonstrated enhancement, 21 (77.8%) had enhancement and T2 hyperintensity, none had T2 hyperintensity alone. Of the 3 patients with normal MRI, the first was a 49 year old man without optic nerve edema, but pain on eye movement with an initial VA 20/50, which improved to 20/30 (no initial visual field). The second was a 50 year old woman with painless scotoma secondary to an enlarged blind spot. She was noted to have optic disc edema, but no vascular risk factors or crowding of her fellow optic nerve. Her visual acuity was 20/20 at presentation and improved to 20/15 at her follow-up visit with resolution of her scotoma. The third patient was a 54 year old woman without any vascular risk factors who developed painful vision loss in her right eye with a swollen optic disc. Her initial VA was 20/200 and improved to 20/80.

Discussion

This study investigated the sensitivity of a standard clinical orbital MRI protocol to detect acute ON and found a sensitivity of 76.1% on the radiology report. The sensitivity was higher (83.7%) when the MRI was reviewed by a single dedicated board certified neuroradiologist. We identified gadolinium contrast enhancement as the most common feature (78.3%), but T2 changes were also frequently found and simply not described in clinical MRI reports. These findings underscore the complexity of reviewing orbital MRI and the importance of having a neuroradiologist interpret images when the diagnosis may be uncertain.

The sensitivity of gadolinium contrast enhancement of the affected optic nerve was substantially lower in our series (78.3%) than previously reported. In the largest retrospective series, 87 of 93 patients (94%; 6 months of follow-up) with acute ON who had an orbital MRI (1.5 Tesla) performed within 20 days of vision loss demonstrated gadolinium contrast enhancement of the affected optic nerve when reviewed by a neuroradiologist.[7]

Patients were excluded from the study if their visual acuity did not return to 20/20, formal visual fields showed a mean deviation less than −2.00dB, and color vision was abnormal by pseudoisochomatic plate testing. In contrast, our study required improvement of visual acuity or visual fields as expected with ON, but did not follow stringent criteria that required recovery of nearly all visual function.

Additionally, we included patients over the age of 46. It is possible that the reduced sensitivity in our series may be secondary to the inclusion of older patients with a diagnosis of non-arteritic ischemic optic neuropathy (NAION) rather than ON. However, the 3 patients over 46 years old in our study who did not have gadolinium enhancement of the affected optic nerve, were unlikely to have NAION based on their clinical presentation and visual recovery. Even if these patients were incorrectly misdiagnosed as ON, the sensitivity of gadolinium contrast enhanced MRI would only increase from 78.3% to 80.9% when interpreted by a fellowship-trained neuroradiologist. Conversely, NAION demonstrated enhancement on orbital MRI in 5 out of 32 patients (15.6%) in a retrospective study by Rizzo et al. [9] Due to our rigorous clinical criteria, we feel it is unlikely that patients with NAION were misclassified as ON. Accidental inclusion of patient with NAION and optic nerve enhancement would have artifactually raised the sensitivity in this analysis.

In 2 smaller retrospective studies, MRI was also found to be more sensitive. These studies showed that 31 of 32 (97%) and 30 of 34 (88%) orbital MRI studies showed contrast enhancement of the clinically affected optic nerve when reviewed by a neuroradiologist.[9, 15] Unlike our study which used ONTT-based prespecified inclusion criteria, neither study provided the criteria for the ON diagnosis. Although the diagnosis was made by experienced neuro-ophthalmologists, it is possible that MRI negative patients were excluded from the study because the diagnosis of ON was brought into question by the normal imaging. A prospective study of 33 patients with their first episode of ON detected a lesion on fat suppressed fast spin echo orbital MRI in all 33 patients, and gadolinium contrast enhancement in 27 out of 28 patients (96%) with triple-dose gadolinium fat-saturated T1 weighted spin echo.[8] As the authors acknowledge, the use of triple-dose gadolinium is not recommended in routine care and is often cost prohibitive. Additionally, there are several studies that have demonstrated that gadolinium is deposited in brain and the long term implications are unclear.[16–18]

In contrast to these earlier findings, our study is in excellent agreement with a recent prospective study of MRI findings in first episode optic neuritis that detected lesions of the optic nerve in 80.6% of 31 patients.[11] Soelberg et al used 1.5 Tesla orbital imaging including 3D fluid-attenuated inversion recovery (FLAIR), 2D FLAIR or 2D short tau inversion recovery (STIR) within 55 days of symptom onset. The similarity between our findings suggests that prospective imaging with uniform protocol, or inclusion of STIR sequences would be unlikely to increase sensitivity.

Our study also provides new information regarding the sensitivity of MR in patients over 46 with clinical ON. In older patients with acute vision loss and optic disc edema, the diagnosis of NAION can be difficult to differentiate from ON. In this subgroup, the sensitivity was nearly 90% and all demonstrated gadolinium contrast enhancement of the clinically affected

optic nerve. When combined with clinical information, MRI could be a useful tool for distinguishing NAION from ON in older patients, but should be interpreted with caution when the diagnosis is uncertain.

Dedicated review by a neuroradiologist increased the sensitivity of orbital MRI from 76.1% to 83.7%. Scans performed outside of the University of Michigan may have been interpreted by general radiologists rather than neuroradiologists, leading to a lower detection rate. All but one MRI requisition included a clinical history of visual symptoms or eye pain, 55 of which specifically mentioned “optic neuritis” or “retrobulbar neuritis, indicating that in 91 of 92 cases, the radiologist was aware of a vision-related diagnosis.

Our study has important limitations. To reduce the likelihood of including patients with a diagnosis other than ON, we excluded eyes that did not experience visual recovery and almost certainly excluded some patients with ON, considering that 10% of ONTT patients did not recover vision. Follow-up visit time was only 2 months on average, so it is possible that some patients were excluded because they did not show adequate improvement early but may have improved if more follow-up data was available. Lastly, there are potential technical limitations with our study since MRIs were performed at numerous centers and by different machines. Even those performed at the University of Michigan were done on different brands of MRI scanners and of varied magnet strength (1.5T and 3T). However, this more closely reflects actual clinical practice and makes our results more applicable to real patient care. In this large series examining the sensitivity of orbital MRI in detecting changes consistent with clinically proven ON, 16.3% of patients had a normal optic nerve on MRI. This is higher than most prior reports and confirms the importance of making a clinical diagnosis of ON without relying on neuroimaging for confirmation. In our study, dedicated review by a skilled neuroradiologist identified an additional 7 patients (7.6%) with radiographic features of ON when compared to the report alone. However, even with the increased sensitivity of dedicated neuroradiology interpretation, nearly 1 in 5 patients with ON still had normal orbital imaging. The primary utility of MRI in ON remains detection of cerebral white matter lesions to aid in prognostication of future MS risk. It is important for clinicians to be aware that a normal MRI does not contradict a clinical diagnosis of ON.

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Table 1:

Clinical characteristics of patients with optic neuritis by orbital MRI result

Characteristic	MRI Positive[a] (n=77)	MRI Negative (n=15)	OR (95% CI); P value
Female n (%)	56 (72.7%)	11 (73.3%)	0.97 (0.28–3.38; 0.96
Age median (IQR)	38.0 (31.9–50.3)	39.7 (26.5–46.261)	1.03 (0.98–1.08); 0.25
Caucasian n (%)	62 (80.5%)	15 (100%)	
Affected eye			1.25 (0.41–3.85); 0.70
Right n (%)	35 (45.5%)	6 (40.0%)	
Presence of pain	59 (76.6%)	7 (46.7%)	3.75 (1.19–11.75); 0.023
Presenting visual function			
Mean visual acuity (SD)	1.011 (0.981)	0.606 (0.601)	1.78 (0.74–4.31); 0.20
Visual field mean deviation (SD)	−14.2 (8.09)	−13.5 (10.4)	0.99 (0.92–1.07); 0.81
Presence of disc edema	23 (29.9%)	6 (40.0%)	0.64 (0.20–2.00); 0.44
Days to MRI mean (SD)	13.0 (8.2)	11.8 (7.8)	1.02 (0.95–1.09); 0.59
Visual function at final follow-up visit			
Mean visual acuity (SD)	0.153 (0.334)	0.117 (0.209)	1.52 (0.16–14.90); 0.72
Visual field mean deviation (SD)	−4.99 (5.11)	−2.97 (2.64)	0.87 (0.71–1.07); 0.18

[a]Orbital MRI was considered “positive” if the optic nerve on the affected side demonstrated high T2 signal within the nerve or post-contrast enhancement on T1 weighted sequences.

Table 2:

Clinical characteristics of 15 patients with optic neuritis and negative orbital MRI

Age	Sex	Pain	Edema	Initial VA	Final VA	Initial MD	Final MD	Multiple Sclerosis
18	F	no	no	20/30	20/25	−16.63	−7.19	No
21	F	no	no	20/50	20/15		−2.72	No
25	M	yes	no	20/200	20/20			Yes
26	F	yes	no	20/200	20/20		−0.39	No
26	F	yes	no	LP	20/40	−26.16	−7.16	No
27	F	no	no	CF	20/20	−33.2		No
38	F	no	yes	20/20	20/15	−2.25	−1.6	No
39	F	no	yes	20/100	20/20	−13.75	−3.23	No
41	F	no	yes	20/100	20/30	−20.78	−4.11	No
42	M	yes	no	20/100	20/40	−9.58	−5.44	Yes
45	M	yes	no	20/20	20/15	−6.2	−0.68	No
46	F	no	yes	20/20	20/20	−3.64	−1.3	No
49	M	yes	no	20/50	20/30		−1.52	No
50	F	no	yes	20/20	20/15	−0.41	0.95	No
54	F	yes	yes	20/200	20/80	−15.6		No