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Utility of coronal contrast-enhanced fat-suppressed FLAIR in the evaluation of optic neuropathy and atrophy

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ABSTRACT

Background and purpose: Evaluating chronic sequelae of optic neuritis, such as optic neuropathy with or without optic nerve atrophy, can be challenging on whole brain MRI. This study evaluated the utility of dedicated coronal contrast-enhanced fat-suppressed FLAIR (CE-FS-FLAIR) MR imaging to detect optic neuropathy and optic nerve atrophy.

Materials and methods: Over 4.5 years, a 3 mm coronal CE-FS-FLAIR sequence at 1.5T was added to the routine brain MRIs of 124 consecutive patients, 102 of whom had suspected or known demyelinating disease. Retrospective record reviews confirmed that 28 of these 102 had documented onset of optic neuritis >4 weeks prior to the brain MRI. These 28 were compared to the other 22 ("controls") of the 124 patients who lacked a history of demyelinating disease or visual symptoms. Using coronal CE-FS-FLAIR, two neuroradiologists separately graded each optic nerve (n = 50 patients, 100 total nerves) as either negative, equivocal, or positive for optic neuropathy or atrophy. The scoring was later repeated.

Results: The mean time from acute optic neuritis onset to MRI was 4.1 ± 4.6 years (range 34 days-17.4 years). Per individual nerve grading, the range of sensitivity, specificity, and accuracy of coronal CE-FS-FLAIR in detecting optic neuropathy was 71.4–77.1%, 93.8–95.4%, and 85.5–89.0%, respectively, with strong interobserver ($k = 0.667 - 0.678$, $p < 0.0001$), and intraobserver ($k = 0.706 - 0.763$, $p < 0.0001$) agreement. For optic atrophy, interobserver agreement was moderate ($k = 0.437 - 0.484$, $p < 0.0001$), while intraobserver agreement was moderate-strong ($k = 0.491 - 0.596$, $p < 0.0001$).

Conclusion: Coronal CE-FS-FLAIR is quite specific in detecting optic neuropathy years after the onset of acute optic neuritis, but is less useful in detecting optic nerve atrophy.

1. Introduction

Optic neuritis is an inflammatory condition commonly affecting patients with multiple sclerosis, in which inflammation and demyelination of the optic nerve causes clinical symptoms of acute vision loss, ophthalmalgia, and/or dyschromatopsia [1–3]. While the symptoms of optic neuritis can resolve quickly, the sequelae of subacute/chronic optic nerve changes – i.e. optic neuropathy either with or without nerve atrophy – may be detected clinically via dedicated testing once the acute symptoms have resolved [4].

MRI evaluation of the optic nerves can be difficult given the inherent difficulties of orbital imaging, such as motion artifact, small optic nerve size, surrounding osseous structures, and adjacent CSF or fat [5]. Various sequences with CSF or fat suppression and faster acquisition times have improved the detection of acute optic neuritis, such as contrast-enhanced (CE) fat-suppressed (FS)-T1WI, FS-FLAIR, FS-FSE T2WI, DIR, STIR, SPIR-FLAIR, and HASTE [6–10]. However, the diagnosis of its chronic sequelae can be more diagnostically challenging. As opposed to an enlarged, enhancing nerve in acute optic neuritis, the MRI findings in the subacute/chronic stages may be less apparent [11–16].

Hence, as both unenhanced and CE-FS-FLAIR have demonstrated utility in diagnosing acute optic neuritis and various CNS disorders, CE-FS-FLAIR may also be useful in detecting optic neuropathy or atrophy, particularly if the patient is a poor historian or the ophthalmologic findings are indeterminate [6,15–20]. Thus, the purpose of this study was to evaluate the utility of coronal CE-FS-FLAIR MR

Abbreviations: CE, Contrast-Enhanced; CE-T1WI, Contrast-Enhanced T1WI; FS, Fat-Suppressed; CE-FS-FLAIR, Contrast-Enhanced Fat-Suppressed FLAIR.

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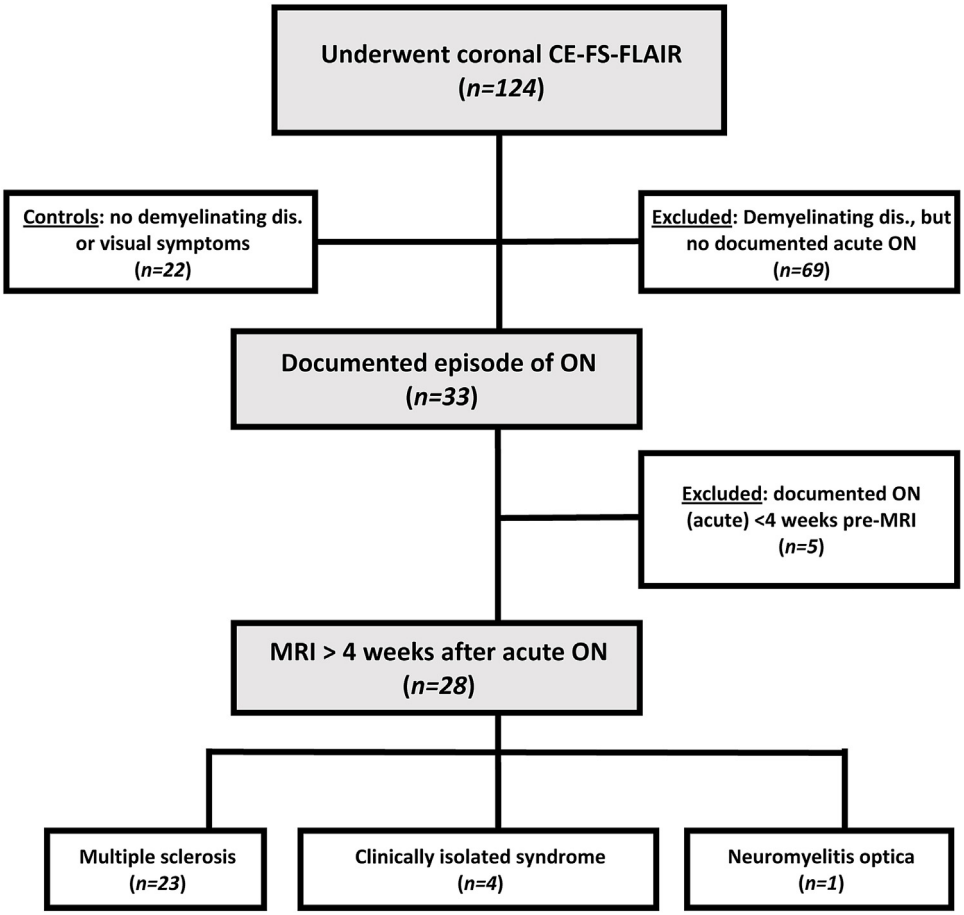

Fig. 1. Cohort and control selection criteria. CE-FS-FLAIR = contrast-enhanced fat-suppressed-FLAIR; dis. = disease; ON = optic neuritis.

images in detecting both optic neuropathy and optic nerve atrophy as chronic sequelae of acute optic neuritis.

2. Materials and methods

2.1. Patient cohort and controls

After internal review board approval for this retrospective study, an electronic health record review was performed on 124 consecutive patients who underwent coronal CE-FS-FLAIR imaging on two 1.5T MRI scanners over a 4.5-year period at a single institution (Fig. 1). Of these 124 patients, 102 were obtained for known or suspected demyelinating disease. Inclusion criteria for the patient cohort was solely confirmation of a prior episode of optic neuritis > 4 weeks prior to the MRI, as confirmed by a neurologist or ophthalmologist on dedicated neuro-ophthalmic evaluation. Of the 102 patients, 33 patients met the inclusion criteria. Exclusion criteria from this 33 patient cohort were: 1) an additional episode of optic neuritis that occurred within four weeks of the MRI ($n = 5$), or 2) the CE-FS-FLAIR MR images were too compromised by motion or artifact for review ($n = 0$). Ultimately, 28 patients were included as the "cohort". Another 22 patients with various vague symptoms (e.g. headache, paresthesias, etc.) had a whole brain MRI series to exclude demyelinating disease. These patients had no history of optic neuritis or demyelinating disease, and their routine brain MRI had no findings to suggest optic nerve or orbital pathology, demyelinating disease, or other cerebral pathology. Thus, this group was included as "controls".

2.2. Image Acquisition/Technique

Over a 4.5-year period, 3 mm thickness postcontrast coronal CE-FS-FLAIR (spectral fat saturation) images of the entire brain were added to a routine brain MRI protocol used to evaluate for demyelinating disease. Notably, CE-T1WI of the entire brain was part of the protocol in both the patient cohort and controls, but neither coronal thin-section (3 mm) T1WI or CE-FS-T1WI was obtained of the orbits, as the clinical concern expressed was not specifically for optic evaluation.

All examinations were performed on two 1.5T field strength MRI scanners (*Siemens 1.5T Symphony and Siemens 1.5T Avanto, Siemens Medical Solutions USA, Inc., Malvern, PA, USA)*. The sequence parameters were: 6500–9000/105–110/2000–2100/1–2/15–23 (TR/TE/TI/NEX/echo train length). A weight-based dose of gadolinium-based contrast (gadobutrol) was administered (*Gadovist, Bayer Healthcare, Whippany, NJ, USA*) at 0.1 mmol/kg concentration, with a maximum dose of 10 ml. The CE-FS-FLAIR sequence was acquired between 5 and 7 min following the contrast bolus, and the acquisition time was 4–5 min.

2.3. Image review

Prior to image review, two staff neuroradiologists (AMM, JBR) agreed that optic neuropathy would be diagnosed as optic nerve hyperintensity relative to the intraorbital musculature. The cerebral hemispheres were covered in order to avoid bias from visualizing any cerebral abnormalities. Optic nerve atrophy would be subjectively assessed as a decreased caliber of the optic nerve.