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Whole-Brain Contrast-Enhanced 3D TSE T1WI Should Be Preferred Over Orbits Contrast-Enhanced 2D Coronal T1WI for the Detection of Optic Nerve Enhancement in Patients with Acute Vision Loss

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LETTER TO THE EDITOR

We were interested to read the article by Prillard *et al*. on a retrospective study comparing contrast-enhanced 3D TSE-T1WI with contrast-enhanced coronal 2D T1WI of the orbits for the detection of optic nerve enhancement in patients with acute vision loss but without obvious ophthalmologic involvement [Prillard, D. *et al*., 2024]. It was found that there was a strong agreement between 3D TSE-T1WI and 2D coronal T1WI with regard to the detection of optic nerve enhancement [Prillard, D. *et al*., 2024]. In patients with canalicular involvement, 3D TSE-T1WI had a higher sensitivity for the detection of optic nerve enhancement than 2D coronal T1WI [Prillard, D. *et al*., 2024]. The study is convincing, but several points need to be discussed.

First, optic nerve enhancement may be due not only to immunologic diseases such as multiple sclerosis, neuromyelitis optica or MOG-associated diseases, but also to anti-CRMP-5 optic neuropathy, autoimmune GFAP astrocytopathy, neurosarcoidosis, acute disseminated encephalomyelitis, chronic recurrent inflammatory optic neuropathy, systemic lupus erythematosus, Sjögren's syndrome and Behcet's disease. In addition, optic neuritis with enlargement of the optic nerve can also be caused by an infectious optic neuritis (e.g. neuroborreliosis, toxoplasmosis or an infection with HIV, varicella zoster virus, herpes simplex virus), a hereditary disease (e.g. Leber's hereditary optic neuropathy), glioma, meningioma, Erdheim-Chester disease, Krabbe disease, metachromatic leukodystrophy, pseudotumor cerebri, posterior ischemic optic neuropathy, anterior ischemic optic neuropathy, toxins or radiation therapy. As the authors state in the introduction that it is important to correctly diagnose optic neuritis, it is important to rule out all these different causes of optic nerve contrast enhancement. We should know how many of the included patients had optic nerve enhancement due to causes other than multiple sclerosis or neuromyelitis optica.

The second point is that acute vision loss may be due not only to optic nerve involvement, but to involvement of any segment of the optic pathway. It is therefore recommended to first perform an MRI of the entire brain with contrast medium and not just a focused MRI of the eye socket.

Thirdly, acute visual loss may not only be due to damage to the optic nerve or the intra-parenchymal visual pathways, but also to insufficient blood supply to these structures. Therefore, it is imperative to rule out ischemia in patients with acute vision loss. This can generally be due to macroangiopathy, microangiopathy, cardioembolism, coagulopathy or hypercellularity of the blood cells [Adams, H. P. *et al*., 1993]. Therefore, in patients with acute visual loss, it is imperative to rule out all of these causes, e.g. carotid stenosis, carotid artery dissection, carotid aneurysm, vasculitis, seizures, heart disease or hematologic disease. Even in patients with a cardioembolism, the ophthalmologist can detect a normal funduscopy and optical coherence tomography.

In conclusion, it can be said that this interesting study has limitations that relativize the results and their interpretation. Removing these limitations could strengthen the conclusions and reinforce the message of the study. Acute vision loss should not only be attributed to optic neuritis, optic neuritis is not only due to immunologic disease, and vascular causes should be considered in patients with acute vision loss. To assess the entire visual pathways in patients with acute visual loss, whole-brain contrast-enhanced 3D TSE T1WI should be preferred over orbits contrast-enhanced 2D coronal T1WI.

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