Mastoid T2-Hyperintensity in Patients with Migraine-Related Aural Fullness

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Introduction

- Migraine headaches are a type of unilateral, pulsating headache that has been linked to both vascular and neurologic etiologies.[1-7]
- Migraine headaches are predominantly induced by a spreading cortical depression which leads to the activation of the trigeminal-vascular system. This activation leads to the vasodilation of the intracranial vasculature supplied by the branches of the affected trigeminal-vascular system.
- The mastoid region is supplied by the mastoid artery; which branches from the same vasculature associated with migraine headaches.
- Aural fullness is the sensation of ear fullness or clogging that can cause significant discomfort. Aural fullness/pressure that is not due to ETD or third window syndrome is often due to a migraine etiology. [5,6]
- Migraine can present with a variety of vestibular and cochlear symptoms, including aural fullness, sudden hearing loss, tinnitus, otalgia, hyperacusis, dizziness, imbalance, and vertigo.[5-7]
- Hyperintensity in T2-weighted MRI signals the presence of water in an area. Thus, vascular vasodilation would cause T2-hyperintensity.
- The purpose of this investigation was to determine whether aural fullness in patients with migraine headaches correlates to mastoid T2 hyperintensity on magnetic resonance imaging (MRI).

Methods

- A retrospective review was conducted for patients who presented to a tertiary-care neurotology clinic with migraine-related aural fullness.
- Eligible patients were seen between 2018 and 2020 and satisfied the most recent diagnostic criteria.
- ETD was ruled out with previous myringotomy not causing relief or normal TM exam and type A tympanogram on at least 2 occasions while symptomatic.
- Patients' headache status was determined by the presence of ICHD-3-defined migraine headaches, non-migraine headaches, and no headaches.
- T2-weighted MRI sequences were reviewed by two neurotologists blinded to patients' headache status for T2 hyperintensities in the mastoid.
- Results were compared between patients of different headache status.
- Chi-squared testing was conducted in R version 4.3.0, with $p<0.05$ marking significance.

Results

- In total, 70 patients were included with a mean age of 58±15 years comprised of 49 (70%) females.
- Fifteen (21%) patients fulfilled ICHD-3 criteria for migraine headaches, 29 (41%) had headaches that did not meet the criteria, and 26 (37%) did not report headaches.
- In total, 6 (8%) patients had fluid signal in the mastoid.
- Among patients with migraine headache, 4 of 15 (27%) had T2 hyperintensity in the mastoid **(Figure 1)**.
- Conversely, of the 26 patients without migraine headache, only 2 (8%) had T2 hyperintensity on imaging.
- Patients with migraine headache had greater frequencies of T2 hyperintensities compared to patients with headaches not fulfilling ICDH-3 criteria ($p=0.004$).

Conclusions

- Patients experiencing aural fullness with migraine headaches were more likely to present with mastoid fluid compared to non-migraineurs.
- Migraine-related aural fullness is likely caused by tensor tympani sustained activity in addition to hypersensitivity of the neural input from the TM.
- The T2 hyperintensity in the mastoid is likely due to increased vascular permeability or neurogenic inflammation, rather than Eustachian tube dysfunction in patients with this condition.

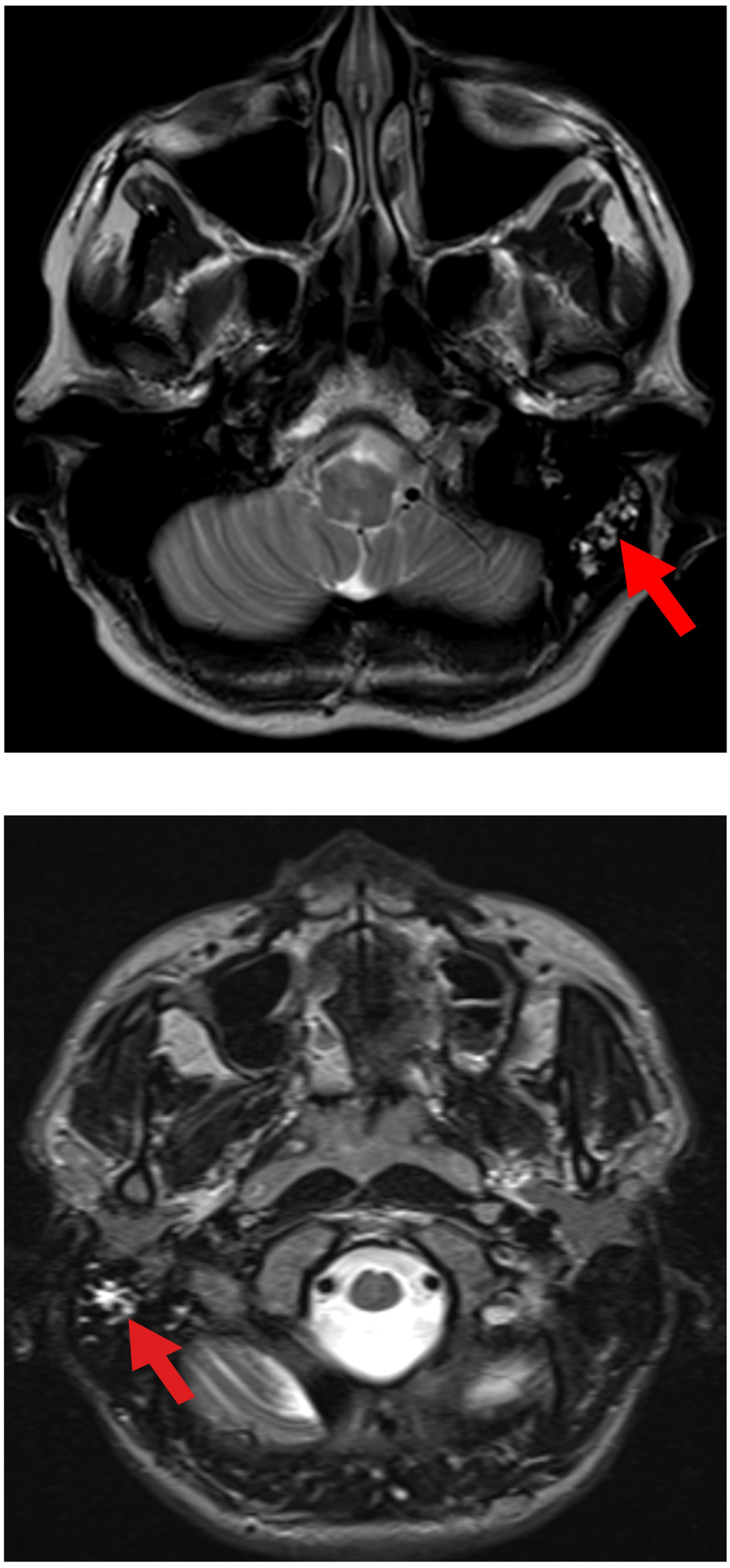

Figure 1. MRI of patients meeting ICHD-3 criteria for migraine. Hyperintensity was noted upon examination of the mastoid region (red arrow) on the same side as the patient's aural fullness and headaches.

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