ORIGINAL RESEARCH

Evaluation of MRI findings in Migraine patients

[1]Dr. Pooja, [2]Dr. Pragya Soni, [3]Dr. Yashveer Arya, [4]Dr. Paresh Kumar Sukhani, [5]Dr. Hemant Kumar Mishra

[1,2,3]PG resident Second year, [4]Professor, [5]Professor and Head, Department of Radiodiagnosis, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

Corresponding Author
Dr. Paresh Kumar Sukhani
Professor, Department of Radiodiagnosis, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India

Received: 08 September, 2023 Accepted: 13 October, 2023

ABSTRACT

Background: To evaluate MRI findings in migraine patients. **Materials & Methods:** A total of 50 subjects were included in the analysis. Conventional procedures, including a clinical assessment and the collection of pertinent medical histories, were conducted. **Results:** The majority of the patients fell within the age range of their forties and fifties. Approximately 26% of the patient group consisted of individuals aged between 31 and 40, and 10% of the patients were over 50 years old. **Conclusion:** Migraine patients may, at times, exhibit irregular MRI results that could offer an explanation for their headache symptoms.

Keywords: Migraine, MRI.

INTRODUCTION

Acute or chronic headache is one of the common presenting complaints in patients attending the out patient's department or emergency care of any hospital or general practitioner however, only about 10% of patients with recurrent headache have secondary cause. [1] It has been contended that most of the patients suffering from primary headache can be managed with primary care and no need of neuroimaging in most of the cases. [2] Headache is a common symptom with a wide variety of potential causes. More than 70% of the U.S. population are estimated to experience headaches, [3,4] with the vast majority of headaches being caused by benign primary headache disorders and not significant pathological conditions. [3,5] Migraine is a severe, disabling brain condition that ranks 6th most disabling disorder globally according to the World Health Organization (WHO). [6,7] Migraine is the most frequent neurological disorder in adults, affecting up to 12% of the general population. [8] The annual costs of migraine – including lost productivity – are more than $19.6B in the U.S. and €27B in Europe, making it a significant public health issue.

Headache cause understandable concern not only on the part of the patient but also health care professional. [9] Few serious brain pathology presents with secondary headache for example a brain tumour or space occupying lesion (SOL) is a secondary cause of headache and CT/MRI is best tool for the diagnosis and treatment follow up which is essential for optimal management of secondary headache however brain tumours accounts less than 0.1% in the lifetime prevalence as a cause of headache. [10] So many times it is so difficult to discriminate between primary and secondary type of headache and it presents diagnostic dilemma before clinician. Since by definition, primary headache does not require any neuroimaging because no such underlying disease process exists which can be picked up by neuroimaging. Primary headache, which include migraine, tension headache and cluster headache are benign in nature. These types of headache are usually recurrent in nature and have no underlying organic disease in their root. Secondary headache is always caused by underlying organic diseases ranging from extra cranial benign condition such as sinusitis or mastoiditis to life threatening intracranial pathology like sub arachnoid haemorrhage or brain tumours. In general, clinical practice, it is well accepted that the so called red flags of headache needs search for secondary headache. Red flag signs and symptoms include: Early morning headache, new persistent and unexplained headache in a patient aged over 50-year-old, headache associated with changes in posture or vomiting, change in personality, cognition or conscious level, headache with seizure, new onset neurological deficit, headache precipitated by coughing, sneezing or exercise, associated with visual

disturbance (papilloedema) or jaw claudication (giant cell arteritis), immunosuppressed or history of malignancy, atypical aura, headache with sign and symptoms of glaucoma, headache associated with signs of systemic illness e.g. neck rigidity, rash, fever and headache subsequent to head injury. [11]

Silent infarct lesions, also known as white matter hyperintensities (WMH), are referred to as obvious lesions with MRI infarction features and are not accompanied by clinical symptoms or other stroke-related signs. [12] Although their pathogenesis is probably multifactorial, the clinical importance of these lesions is yet to be understood. These hyperintense lesions are commonly considered to be ischemic, which is logical given their association with vascular risk factors. [13,14] While seemingly transient ischemic events in posterior blood circulation of the brain are responsible for such lesions, [15,16] other factors have also been put forth regarding the pathophysiology of these abnormalities. These include glutamatergic excitotoxicity, hyperlipidemia, hypertension, smoking, immunogenic demyelination of white matter, mitochondrial dysfunction, endothelial dysfunction, attack-related oligemia and focal hypoperfusion, and vasoactive drugs like triptans or ergots. [17] Hence, this study was done to evaluate MRI findings in migraine patients.

MATERIALS & METHODS

A total of 50 subjects were included in the analysis. Conventional procedures, including a clinical assessment and the collection of pertinent medical histories, were conducted. The patients underwent MRI scans while positioned supine, with images taken using a head coil. The MRI data was subsequently generated and analyzed using the SPSS software, following a predefined template.

RESULTS

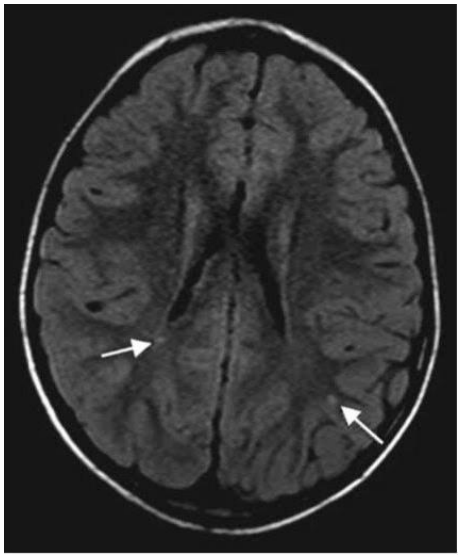
The majority of the patients fell within the age range of their forties and fifties. Approximately 26% of the patient group consisted of individuals aged between 31 and 40, and 10% of the patients were over 50 years old. Notably, the MRIs of 15 patients, which accounts for 30% of the total, revealed significant results. Specifically, T2 FLAIR imaging displayed hyperintensity in the subcortical white matter, aligning with the observations made in traditional T2 imaging.

Table 1: Age-wise distribution of patients

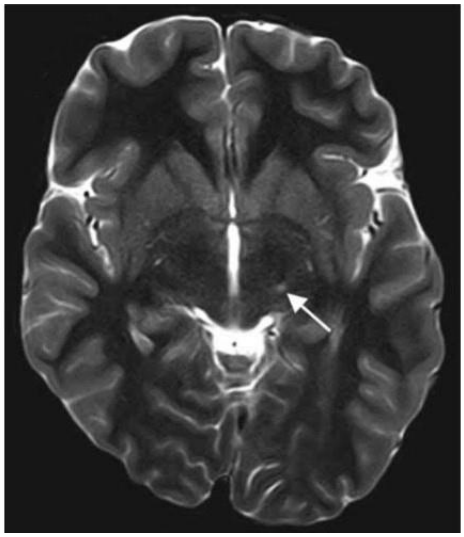
Age group	Number of patients	Percentage of patients
Less than 20	5	10
20 to 30	8	16
31 to 40	13	26
41 to 50	19	38
More than 50	5	10
Total	50	100

Table 2: Distribution of patients with Migraine on the basis of MRI findings

Parameter	Presence of significant MRI findings		Absence of significant MRI findings	
	Number of patients	Percentage of patients	Number of patients	Percentage of patients
Patients with Migraine	15	30	35	70



FLAIR HYPERINTENSITIES IN SUBCORTICAL AND DEEP WHITE MATTER.



T2 HYPERINTENSE LESION IN LEFT THALAMUS

DISCUSSION

While standard anatomic imaging appears to be of limited diagnostic value in migraine, recent studies have suggested significant cortical thinning may occur within regions within the pain matrix. Additionally, patients with migraine appear to be at higher risk for T2 hyperintense lesions, suggesting ischemic or degenerative processes may be involved. Early voxel based morphometry (VBM) studies focusing on gray matter thickness and density did not observe significant differences in cortical density in patients with migraine. [18] However, subsequent larger studies have noted significant reductions in gray matter density in cortical areas involved in pain processing, as well as an increase in gray matter density within the PAG in patients with visible T2 lesions. [19,20] Interestingly, in patients with migraine with visual aura, studies have identified thicker visual cortex, presumably due to more frequent activation in these areas. Hence, this study was done to evaluate MRI findings in migraine patients. [21]

In the present study, the majority of the patients fell within the age range of their forties and fifties. Approximately 26% of the patient group consisted of individuals aged between 31 and 40, and 10% of the patients were over 50 years old. A study by Negm M et al, sixty-five patients with migraine aged from 18 to 50 years were included. We excluded smokers and patients with hypertension, cardiac disease, diabetes mellitus, endocrine dysfunction, oncological and hematological diseases, infectious diseases, demyelinating disorders, and Alzheimer disease. Brain MRI and laboratory investigation was done for all patients. White matter hyperintensities were significant more frequent in migraine with aura than those without aura. According to MIGSEV scale, white matter hyperintensities were highly significantly more frequent in grade III severity than grades II and I. The number of white matter hyperintensities increases significantly with increase intensity of pain during attack. The number of white matter hyperintensities increases significantly with increase intensity of nausea, disability, tolerability during attack and age. Resistance to treatment also shows statistically significant difference in increase number of WMHs. White matter hyperintensities are present in 43.1% of migraine patients. Age, presence of aura, nausea, disability during attack, resistance to treatment, and severity of headache and duration of migraine are considered a risk factor for development of white matter hyperintensities. [22]

In the present study, notably, the MRIs of 15 patients, which accounts for 30% of the total, revealed significant results. Specifically, T2 FLAIR imaging displayed hyperintensity in the subcortical white matter, aligning with the observations made in traditional T2 imaging. Another strudy by Toghae M et al, ninety patients with migraine headache (70 without aura and 20 with aura) were enrolled and interviewed. Information on their headache (severity, frequency, and mean disease duration) and other related data was obtained by completing a clinical checklist. Subsequently, brain magnetic resonance imaging (MRI) was performed and each patient was then evaluated for hyperintense lesions. Of the 90 patients, 29 (32%) had silent hyperintense lesions on their MRI. The mean age of the patients with hyperintense foci was 41 years while those with no lesions was 33 years ($p<0.010$). Supratentorial hyperintense lesions represented the majority of lesions in the patients (n=46, 63%). Moreover, 56.3% of the lesions (n=41) were located within the right hemisphere. Cardiovascular risk factors such as smoking, serum cholesterol, oral contraceptive pills use, and body mass index (BMI) were not significantly different in these two groups ($p>0.050$). The lesions were found significantly more frequently in the patients who experienced chronic migraine ($p=0.032$). A study adds weight to the theory that disease duration has a key role in the formation of hyperintense brain lesions. Certain cardiovascular risk factors such as sex, smoking, serum cholesterol, and

BMI, do not affect the presence or absence of such lesions, suggesting that the relationship between migraine and these lesions may be directly due to the effects of migraine itself. [23] Palm-Meinders IH et al, determine whether women or men with migraine (with and without aura) have a higher incidence of brain lesions 9 years after initial MRI, whether migraine frequency was associated with progression of brain lesions, and whether progression of brain lesions was associated with cognitive decline. In a follow-up of the 2000 Cerebral Abnormalities in Migraine, an Epidemiological Risk Analysis cohort, a prospective populationbased observational study of Dutch participants with migraine and an age- and sexmatched control group, 203 of the 295 baseline participants in the migraine group and 83 of 140 in the control group underwent MRI scan in 2009 to identify progression of MRI-measured brain lesions. Comparisons were adjusted for age, sex, hypertension, diabetes, and educational level. The participants in the migraine group were a mean 57 years (range, 43–72 years), and 71% were women. Those in the control group were a mean 55 years (range, 44–71 years), and 69% were women. Progression of MRI-measured cerebral deep white matter hyperintensities, infratentorial hyperintensities, and posterior circulation territory infarctlike lesions. Change in cognition was also measured. Of the 145 women in the migraine group, 112 (77%) vs 33 of 55 women (60%) in the control group had progression of deep white matter hyperintensities (adjusted odds ratio [OR], 2.1; 95%CI, 1.0–4.1; P=.04). There were no significant associations of migraine with progression of infratentorial hyperintensities: 21 participants (15%) in themigraine group and 1 of 57 participants (2%) in the control group showed progression (adjusted OR, 7.7; 95% CI, 1.0–59.5; P=.05) or new posterior circulation territory infarctlike lesions: 10 of 203 participants (5%) in the migraine group but none of 83 in the control group (P=.07). There was no association of number or frequency of migraine headaches with progression of lesions. There was no significant association of high vs nonhigh deep white matter hyperintensity load with change in cognitive scores (3.7 in the migraine group vs 1.4 in the control group; 95% CI, 4.4 to 0.2; adjusted P=.07). In a community-based cohort followed up after 9 years, women with migraine had a higher incidence of deep white matter hyperintensities but did not have significantly higher progression of other MRI-measured brain changes. There was no association of migraine with progression of any MRI-measured brain lesions in men. [24] Diffusion MR changes have also been observed in patients with migraine. In particular, studies have shown higher apparent diffusion coefficient (ADC), or mean diffusivity (MD), and lower fractional anisotropy (FA) in the frontal lobe along with the genu, splenium, and body of the corpus callosum, consistent with microstructural alterations along these pathways. [25] In migraine patients with aura, a reduced FA along the thalamocortical tract and reduced FA along ventral trigeminothalamic tract have been observed, whereas a reduced FA in the ventrolateral PAG has been observed in migraine patients without aura. [26] Additionally, diffusion MR has revealed enhanced connectivity between temporal pole and entorhinal cortex, as well as high connectivity between frontal lobe regions with reduced FA and regions within the pain network (orbitofrontal cortex, insula, thalamus, and dorsal midbrain/pons). [25] Interestingly, a lower ADC has been observed in migraine patients with T2 hyperintense lesions and transient diffusion changes in the thalamus (increased FA and lower MD) have been observed during migraine without aura, which were normalized after attack. [27] Together, these observations suggest dynamic changes in water mobility may occur during the various stages of attacks.

CONCLUSION

Migraine patients may, at times, exhibit irregular MRI results that could offer an explanation for their headache symptoms.

REFERENCES

1. Peters KS. Secondary headache and head pain emergencies. Primary Care: Clinics in Office Practice. 2004;31(2):381–93.
2. Frishberg BM, et al. Evidence-based guidelines in the primary care setting: neuroimaging in patients with non-acute headache. Available from: URL: http://www.aan.com/public/practice guidelines (2000).
3. Medina LS, D'Souza B & Vasconcellos E Adults and children with headache: evidence-based diagnostic evaluation. Neuroimaging Clin N Am 13, 225–235 (2003).
4. Evans RW Diagnostic testing for the evaluation of headaches. Neurol Clin 14, 1–26 (1996).
5. Jordan JE & Expert Panel on Neurologic, I. Headache. AJNR Am J Neuroradiol 28, 1824–1826 (2007).
6. Goadsby PJ, Lipton RB & Ferrari MD Migraine--current understanding and treatment. N Engl J Med 346, 257–270,
7. Global Burden of Disease Study, C. Global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet 386, 743–800,
8. Lance JW Current concepts of migraine pathogenesis. Neurology 43, S11–15 (1993).
9. Bartleson JD. When and how to investigate the patient with headache. SeminNeurol. 2006;26(2):163–70.
10. Goadsby PJ. To scan or not to scan in headache. Editorial. BMJ. 2004;329:469–70.
11. Clinch CR. Evaluation of acute headaches in adults. Am Fam Physician. 2001;63(4):685–92.
12. Masuda J, Nabika T, Notsu Y. Silent stroke: pathogenesis, genetic factors and clinical implications as a risk factor. Curr Opin Neurol 2001. Feb;14(1):77-82.
13. Dufouil C, de Kersaint-Gilly A, Besançon V, Levy C, Auffray E, Brunnereau L, et al.. Longitudinal study of

blood pressure and white matter hyperintensities: the EVA MRI Cohort. Neurology 2001. Apr;56(7):921-926.
14. Jongen C, van der Grond J, Kappelle LJ, Biessels GJ, Viergever MA, Pluim JP, Utrecht Diabetic Encephalopathy Study Group . Automated measurement of brain and white matter lesion volume in type 2 diabetes mellitus. Diabetologia 2007. Jul;50(7):1509-1516.
15. Kruit MC, Launer LJ, Ferrari MD, van Buchem MA. Infarcts in the posterior circulation territory in migraine. The population-based MRI CAMERA study. Brain 2005. Sep;128(Pt 9):2068-2077. 10.1093/brain/awh542
16. Scher AI, Gudmundsson LS, Sigurdsson S, Ghambaryan A, Aspelund T, Eiriksdottir G, et al.. Migraine headache in middle age and late-life brain infarcts. JAMA 2009. Jun;301(24):2563-2570.
17. Kruit MC, van Buchem MA, Hofman PA, Bakkers JT, Terwindt GM, Ferrari MD, et al.. Migraine as a risk factor for subclinical brain lesions. JAMA 2004. Jan;291(4):427-434.
18. Matharu MS, Good CD, May A, Bahra A & Goadsby PJ No change in the structure of the brain in migraine: a voxel-based morphometric study. Eur J Neurol 10, 53–57 (2003).
19. Rocca MA et al. Brain gray matter changes in migraine patients with T2-visible lesions: a 3-T MRI study. Stroke 37, 1765–1770,
20. Valfre W, Rainero I, Bergui M & Pinessi L Voxel-based morphometry reveals gray matter abnormalities in migraine. Headache 48, 109–117,
21. Gaist D et al. Migraine with visual aura associated with thicker visual cortex. Brain, doi: 10.1093/brain/awx382 (2018).
22. Negm M, Housseini AM, Abdelfatah M, Asran A. Relation between migraine pattern and white matter hyperintensities in brain magnetic resonance imaging. Egypt J Neurol Psychiatr Neurosurg. 2018;54(1):24.
23. Toghae M, Rahimian E, Abdollahi M, Shoar S, Naderan M. The Prevalence of Magnetic Resonance Imaging Hyperintensity in Migraine Patients and Its Association with Migraine Headache Characteristics and Cardiovascular Risk Factors. Oman Med J. 2015 May;30(3):203-7.
24. Palm-Meinders IH, Koppen H, Terwindt GM, Launer LJ, Konishi J, Moonen JM, Bakkers JT, Hofman PA, van Lew B, Middelkoop HA, van Buchem MA, Ferrari MD, Kruit MC. Structural brain changes in migraine. JAMA. 2012 Nov 14;308(18):1889-97.
25. Szabo N et al. White matter microstructural alterations in migraine: a diffusion-weighted MRI study. Pain 153, 651–656,
26. DaSilva AF et al. Interictal alterations of the trigeminal somatosensory pathway and periaqueductal gray matter in migraine. Neuroreport 18, 301–305,
27. Aradi M et al. Quantitative MRI studies of chronic brain white matter hyperintensities in migraine patients. Headache 53, 752–763,