DOI: 10.7860/JCDR/2025/76493.21487

Original Article

Physiology Section

Comparison of Auditory Brainstem Evoked Responses between Migraine Patients and Healthy Controls: A Cross-sectional Study

DANIYA KHALID[1], JYOTSNA SHUKLA[2], ABHISHEK SAINI[3], RAJNI FULWARIYA[4]

ABSTRACT

Introduction: Migraine is a complex neurological disorder associated with noise hypersensitivity most evident during attacks (ictal), however persistent during the post-attack (interictal) period as well. Previous studies on cortical auditory potentials have established abnormal sensory processing during the interictal phase but findings related to subcortical (brainstem) involvement remain inconclusive.

Aim: To compare Wave I and Wave V amplitude of Auditory Brainstem Evoked Responses (ABER) between migraineurs during the interictal phase and healthy controls at 40-, 50-, and 60-dB above Sensation Level (SL) for both ears.

Materials and Methods: The present study was a cross-sectional study conducted at the Department of Physiology, SMS Medical College, Jaipur, Rajasthan, India. Thirty-five diagnosed migraine patients during the interictal phase from the Neurology Outpatient Department (OPD) and 35 age and gender-matched healthy controls were enrolled. The Wave I and V amplitude (μV) were measured, at 40-, 50-, and 60-dB above SL bilaterally, in both groups. Statistical Package for Social Sciences (SPSS) software version 25 was used to analyse the results and an unpaired student's t-test was employed for comparison in a side-specific manner. The significance level was assigned at a p-value <0.05.

Results: The amplitudes of Wave V at 50-db SL (p-value: left ear=0.03, right ear=0.01) and 60-dB SL (p-value: left ear=0.005, right ear=0.013) were significantly higher among migraineurs during interictal phase than their matched controls. The amplitude difference for Wave I was statistically insignificant between the two groups at all recording intensities. At 40 dB SL, both Wave I (p-value: left ear=0.68, right ear=0.63) and Wave V (p-value: left ear=0.30, right ear=0.50) amplitudes did not exhibit any significant difference in results between migraine patients and healthy controls.

Conclusion: The subcortical auditory dysfunction is significant for brainstem nuclei beyond the cochlear nucleus (peripheral auditory neurons) at higher intensities, indicating a more noticeable central processing disorder. This could eventually lead to irreversible auditory pathway damage, ABER can be utilised as a tool for early detection and localisation of the insult, providing an opportunity for early intervention and use of prophylactic measures.

Keywords: Burden of disease, Central auditory processing disorder, Headaches, Migraine

INTRODUCTION

Migraine is the most common form of primary headache disorder (International Headache Society) affecting over one billion population worldwide [1,2]. It is a recurrent unilateral throbbing, pulsating headache of moderate to severe intensity, lasting for 4-72 hours with complete freedom from pain between the attacks [3], triggered by various external and internal factors. The Global Burden of Diseases (GBD) survey (2019) ranked it as the second most common cause of disability considerably paralysing sociopersonal, academic, and financial domains of life [4]. The disease exhibits a cyclical pattern, alternating between ictal and interictal phases. The headache phase is often associated with a vast multitude of symptoms, the most prominent being photophobia and phonophobia [5]. However, the detrimental effects extend beyond the attack phase as 70-90% of the patients continue to experience unpleasant sound perception such as noise aversion and hypersensitivity to sound, even during the asymptomatic phase of the disease due to abnormal recruitment of auditory neuronal networks [6].

During an attack, neurogenic inflammation triggers the release of cytokines, which damage auditory neurons. These inflammatory mediators accumulate, and the injury persists until the asymptomatic interictal phase. The underlying activation of the trigeminovascular system during the ictal phase, combined with the lack of interventions to limit noise exposure during the interictal period, increases the frequency of attacks. Recurrent attacks result in greater harm, ultimately leading to irreversible damage to the auditory pathway, along with sound discomfort experienced both during and between attack periods [6-8].

The auditory dysfunction assessed using ABER has a sensitivity and specificity of 70-90%. This objective, non-invasive test evaluates subcortical auditory circuits and is regarded as the most effective method for identifying functional defects in the central nervous system [9]. The mechanical sound energy converted into action potential by cochlear hair cells traverses through the auditory network and evokes a series of electrical responses while undergoing processing within each station [10]. They produce graphical waveforms with discrete peaks, each representing an averaged synchronised neural response evoked within the first 10 milliseconds of stimulus presentation [9].

These ABER responses in migraine patients become more intensity (sound) dependent over time due to underlying progressive damage to the cochlea as well as beyond the cochlea within the central auditory pathway [11,12].

Most previous studies have determined abnormal "cortical" sensory processing using visual-evoked or cortical auditory-evoked potentials [11,12]. However, abnormal subcortical processing of signals specifically during the interictal phase has been less studied with not much concrete pieces of evidence although subcortical involvement in migraine has been known for many years.

The present study aimed to utilise to compare Wave I and Wave V amplitude of ABER between migraineurs during the interictal phase and healthy controls at 40-, 50-, and 60-dB above SL for both ears.

MATERIALS AND METHODS

The present hospital-based cross-sectional study was carried out at the Physiology Department of SMS Medical College, Jaipur, Rajasthan, India from April 2023 to June 2024 after receiving the desired approval from the Research Review Board (RRB) and the Institutional Ethics Committee (Order no. 718/MC/EC/2023, 30/06/2023). The participants were recruited after obtaining the written informed consent. All the test procedure was explained before the study, as well as a detailed disease history was recorded.

Inclusion criteria: Cooperative and consenting migraine patients (diagnosed as per International Headache Society criteria) during the interictal phase and age-gender-matched healthy employees, 20-40 years of age were recruited from both genders [1].

Exclusion criteria: Migraine patients during ictal and immediate post-ictal phases, participants having a history of neurological, psychiatric, and acute or chronic medical illness, pregnant and lactating women, chronic alcoholic and smokers, patients having a history of otologic symptoms such as otalgia, vertigo, tinnitus were excluded from the study.

Sample size calculation: A total sample size of seventy subjects was calculated for the study purpose, using MedCalc 16.2 software at 95% confidence level (α error= 0.05) and 80% power.

The participants were divided into two groups. The case group had 35 migraine patients recruited from Neurology OPD during a headache-free period, while the control group comprised 35 age-sex matched healthy employees from SMS Medical College and Attached Hospitals, Jaipur, Rajasthan, India.

Study Procedure

Baseline anthropometric parameters such as height, weight, and Body Mass Index (BMI) were measured. BMI was calculated using the formula Weight (kg)/Height (m^2). All participants underwent subjective audiometric tests. Tuning Fork tests (512 Hz), specifically the Rinne test, Weber test, Absolute Bone Conduction (ABC) test, and Pure Tone Audiometry (PTA) were performed to identify any clinically evident hearing loss. ABER was recorded to evaluate the activity of auditory neurons involved in signal conduction up to the brainstem station [Table/Fig-1]. The assessed parameters included the amplitudes of Wave I and Wave V, which is the clinically most significant waves, measured at sound intensities 40-, 50-, and 60-dB SL consecutively [13].

[Table/Fig-1]: Auditory Brainstem Evoked Response (ABER) assessment.

The RMS EMG SALUS- 2C machine was used to conduct all the ABER recordings in the laboratory of the Physiology department. All participants were tested under uniform laboratory conditions while sitting in a relaxed position, having acclimatised to the experimental environment for 10 minutes. They were instructed to remove any jewellery; bluetooth devices and mobile phones that might interfere with the procedure. The skin of the forehead and both mastoid prominences were cleaned with alcohol wipes, followed by gentle rubbing with an abrasive paste to enhance electrical conductivity.

Candidates were advised against applying hair oil or gel to minimise interference during recordings with silver chloride cup electrodes. The electrodes were connected to the Pre-Amp and positioned on the scalp as follows: the reference electrode was placed at the high forehead (Fpz), the ground electrode over the forehead (glabella) (Fz), and the active electrodes over the mastoid processes (left mastoid- A1, right mastoid- A2). Impedance was consistently maintained below 5 kOhms for all electrodes [14].

The click stimuli of rarefaction polarity were delivered to the test ear using supra-aural headphones at 40, 50, and 60 dB above SL consecutively at the rate of 10/sec. The same process was repeated for the other ear. Each time the non-test ear was masked with a white noise (30dB below stimulus intensity) for better response [15]. Responses were live averaged for 1000 stimuli. The ipsilateral stimulation-derivation technique was used for recording and analysing amplitudes of Waves I and V in a side-specific manner [16].

STATISTICAL ANALYSIS

The data was tabulated and subjected to statistical analysis using "SPSS software" version 25. The outcomes were compared bilaterally between the case and control groups for Waves I and V amplitude parameters at 40-, 50-, and 60-dB SL by applying a two-tailed unpaired student's t-test. All quantitative data were expressed as Mean±SD. The results were considered statistically significant at a p-value <0.05.

RESULTS

There were 22 female and 13 male subjects in both case and control groups. The mean age of participants in the case and control groups was 31.6±6.27 and 31±6.11, respectively. No statistically significant differences were appreciated concerning height, weight, and BMI between both groups [Table/Fig-2].

Parameters	Case group (n=35)	Control group (n=35)	p-value#
Height (m)	1.65±0.06	1.64±0.08	0.35
Weight (Kg)	65.9±9.4	68.1±9.6	0.61
BMI (Kg/m^2)	24.22±3.1	25.24±2.8	0.21

[Table/Fig-2]: Baseline characteristics of migraine patients and control groups (Mean±SD).
p-value <0.05: S (significant); p-value >0.05: NS (non-significant); SD: Standard deviation; #Unpaired student t-test

No significant difference was recorded between the case and control groups for both left and right ears in terms of Wave I amplitudes at 40 (p-value=0.68 left, 0.63 right), 50 (p-value=0.13 left, 0.25 right) and 60 dB SL (p-value=0.26 left, 0.11 right) [Table/Fig-3].

ABER intensities	Side involved	Cases (n=35)	Controls (n=35)	p-value#
40 dB SL	Left ear	0.12±0.06	0.12±0.05	0.68
	Right ear	0.13±0.05	0.13±0.06	0.63
50 dB SL	Left ear	0.23±0.03	0.21±0.04	0.13
	Right ear	0.24±0.03	0.23±0.05	0.25
60 dB SL	Left ear	0.27±0.03	0.26±0.03	0.26
	Right ear	0.27±0.03	0.26±0.02	0.11

[Table/Fig-3]: Comparison (side specific) of Wave I amplitudes (μV) between migraine patients and healthy controls (Mean±SD).
p-value <0.05: S (significant); p-value >0.05: NS (non-significant); ABER: Auditory brainstem evoked responses; SD: Standard deviationl; #Unpaired student t-test

The amplitudes of Wave V were significantly higher among migraine patients binaurally than the matched healthy controls at 50 dB SL (p-value=0.03 left, 0.01 right) as well as 60 dB SL (p-value=0.005 left, 0.013 right) [Table/Fig-4].

ABER intensities	Side Involved	Cases (n=35)	Controls (n=35)	p-value
40 dB SL	Left ear	0.37±0.17	0.34±0.09	0.30
	Right ear	0.34±0.12	0.33±0.06	0.5
50 dB SL	Left ear	0.38±0.06	0.35±0.04	0.03
	Right ear	0.41±0.06	0.38±0.04	0.01
60 dB SL	Left ear	0.48±0.05	0.45±0.05	0.005
	Right ear	0.52±0.04	0.49±0.05	0.013

[Table/Fig-4]: Comparison (side specific) of Wave V amplitudes (μV) between Migraine patients and Healthy Controls (Mean±SD).
p-value <0.05: S (significant); p-value >0.05: NS (non-significant); ABER: Auditory brainstem evoked responses; SD: Standard deviation; #Unpaired Student t-test

DISCUSSION

Migraine is a complex neurological disorder that fundamentally alters how the brain processes sensory information. Individuals affected by migraines often experience heightened sensitivity to sound, a phenomenon driven by impaired inhibitory mechanisms in the cortex and the subsequent sensitisation of auditory neurons. Crucially, this abnormal noise perception can linger long after the attack has subsided due to neurovascular changes and dysfunction in vital subcortical structures, such as the brainstem and thalamus [17].

The statistically insignificant results for anthropometric parameters between the two groups suggest that confounding variables did not influence this study's outcomes. This aligns with the findings of Stockard JJ et al., that central auditory conduction is mostly independent of the subject's biophysical profile [18].

Repetitive sound stimulation such as everyday noise exposure, excites fewer neurons in healthy individuals due to a phenomenon known as "habituation." This process leads to a decrease in response, which helps prevent neural over-stimulation. Habituation and sensitisation are opposing processes competing to determine the outcome of central sensory processing [19]. The cytokines released during migraine attacks damage the neuronal membrane of the auditory pathway. This lowers their threshold making individuals more susceptible to "Deficient Habituation," which is observed as a time-dependent increase in the evoked potential amplitude to a series of acoustic stimuli [20,21].

The studies by Schoenen J, Gawel M et al., and Judit A et al., documented this phenomenon of "Deficient Habituation" in individuals with migraines. Their studies demonstrated an increase in the amplitude of Visual Evoked Potentials (VEP) with repetitive light stimulation [20,22,23].

Main A et al., studied sound sensitivity to sound between migraineurs while headache-free and healthy people by exposing participants to increasing sound intensities until they complained of discomfort. There were significant differences between both the groups ($p<0.0005$), the auditory discomfort thresholds being lower in the migraineurs. This sound intolerance between attacks is a form of hyper-responsiveness that is a part of migraine pathophysiology [24].

Vingen JV et al., assessed sound-induced discomfort and pain thresholds using TDH39 transducers, 200 Hz square wave sound stimuli, monaural stimulation, and a "masking white noise" (SPLA of 56 dB) in the opposite ear, facilitating each ear to be evaluated separately. The discomfort was described as an unpleasant, bothersome, or disagreeable sensation. Migraineurs were found to be significantly more sensitive than headache-free controls even when tested outside attack ($p<0.0001$) [25].

Ashkenazi A et al., evaluated ictal and interictal phonophobia in Episodic Migraine (EM) using sound stimuli of pure tones at frequencies of 1000, 4000, and 8000 Hz. Sound Aversion Thresholds (SATs) were determined as the minimal sound intensity perceived as unpleasant or painful. Interictal mean SAT of migraineurs, averaged for the three frequencies, was found significantly lower than that of controls {90.4 (0.8) dB vs. 105.9 (1.1) dB, respectively, $p<0.0001$} [26].

These investigations by Main A et al., Vingen JV et al., and Ashkenazi A et al., additionally confirmed that individuals with migraines have a low sound discomfort threshold even during the interictal phase, suggesting auditory dysfunction.

The findings of the present study are in line with the results obtained by Wang W et al., and Kalita J et al., who demonstrated a significant rise in amplitude at 50- and 60-dB SL bilaterally [27,28] suggesting abnormal processing of auditory stimulus specifically at higher intensities among migraineurs during the interictal phase.

The difference in wave amplitude between both ears at 40 dB SL was not significant, but it became significant at higher stimulation levels of 50 and 60 dB SL. This finding indicates the intensity-dependent response of subcortical auditory pathway nuclei due to impaired habituation and sensitisation. These findings align with the results of studies by Judit A et al., and Ambrosini A et al., which showed that the maximum response difference between cases and controls occurred at higher stimulus intensities of 70- and 80-dB SL, rather than at lower intensities of 40- and 50-dB SL [23,29].

A significant increase in Wave V amplitude in our study at 50- and 60-dB SL among migraine patients bilaterally compared to controls is in concordance with the Yamada T et al., Gopal KV et al., and Vijayalakshmi TN et al., studies [30-32]. The study results, however, do not match with the findings of Benna P et al., and Sand T et al., who did not observe any differences in Wave V amplitude between cases during the interictal phase across all intensities and matched healthy controls [33,34].

Recurrence of attacks is fueled by dysfunctions in the descending pain modulatory pathway, leading to an increased sensitivity to stimuli, or auditory hyperexcitability, at all levels- even when there is no external sensory input during the periods between attacks. Understanding this complex interaction is essential for recognising the profound impact of migraines on daily life and the importance of effective management strategies.

The present study proposes a definitive involvement of auditory pathways among migraineurs. In patients experiencing frequent migraine attacks, these findings could indicate the potential for damage and otologic morbidities. The central auditory dysfunction was more evident than peripheral dysfunction. This was shown by the significantly larger Wave V peak amplitudes among migraine patients signifying sensitisation with higher neural recruitment that fires maximally at intensities of 50 and 60 dB SL.

Limitation(s)

The study employs a cross-sectional design, which is inadequate for establishing a direct causal association and necessitates a longitudinal follow-up investigation in the future. Additionally, participants are not categorised into episodic and chronic migraine groups as disease duration can influence the outcome of abnormal sensory processing.

CONCLUSION(S)

The study reveals that migraine patients experience atypical sensory processing of auditory stimuli, a condition that persists even during symptom-free periods. This suggests that damage to the central auditory pathway in the brainstem is a significant contributing factor to these functional impairments. This can be objectively deciphered using non-invasive tests like ABER as they highlight noticeable differences in auditory processing elucidated in the form of abnormal wave amplitudes. By utilising these tests for early detection and pinpointing the source of the damage, we can create crucial opportunities for timely intervention and the adoption of proactive treatment strategies.

REFERENCES

[1] Headache Classification Committee of the International Headache Society (IHS). The international classification of headache disorders, (beta version). Cephalalgia. 2013;33(9):629-808.

[2] Ashina M, Katsarava Z, Do TP, Buse DC, Pozo-Rosich P, Özge A, et al. Migraine: Epidemiology and systems of care. The Lancet. 2021;397(10283):1485-95.
[3] Pescador Ruschel MA, De Jesus O. Migraine headache. 2023 Aug 23. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan–. PMID: 32809622.
[4] Vos T, Lim SS, Abbafati C, Abbas KM, Abbasi M, Abbasifard M, et al. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: A systematic analysis for the Global Burden of Disease Study 2019. The Lancet. 2020;396(10258):1204-22.
[5] Goadsby PJ, Holland PR, Martins-Oliveira M, Hoffmann J, Schankin C, Akerman S. Pathophysiology of migraine: A disorder of sensory processing. Physiological Reviews. 2017;97(2):553-622.
[6] de Tommaso M, Ambrosini A, Brighina F, Coppola G, Perrotta A, Pierelli F, et al. Altered processing of sensory stimuli in patients with migraine. Nat Rev Neurol. 2014;10(3):144-55.
[7] Burch RC, Buse DC, Lipton RB. Migraine: Epidemiology, burden, and comorbidity. Neurologic Clinics. 2019;37(4):631-49.
[8] Noseda R, Burstein R. Migraine pathophysiology: Anatomy of the trigeminovascular pathway and associated neurological symptoms, cortical spreading depression, sensitization, and modulation of pain. Pain. 2013;154:S44-53.
[9] Balasubramanian T. Brain stem evoked response audiometry A review. Online Journal of Otolaryngology. 2015;5(1):1.
[10] Nuwer MR, Aminoff M, Goodin D, Matsuoka S, Mauguière F, Starr A, et al. IFCN recommended standards for brain-stem auditory evoked potentials. Report of an IFCN committee. International Federation of Clinical Neurophysiology. Electroencephalogr Clin Neurophysiol. 1994;91(1):12-17.
[11] Dash AK, Panda N, Khandelwal G, Lal V, Mann SS. Migraine and audiovestibular dysfunction: Is there a correlation? Am J Otolaryngol. 2008;29(5):295-99.
[12] Lipkin AF, Jenkins HA, Coker NJ. Migraine and sudden sensorineural hearing loss. Arch Otolaryngol Head Neck Surg. 1987;113(3):325-26.
[13] Chiappa KH, Ropper AH. Evoked potentials in clinical medicine (first of two parts). N Engl J Med. 1982;306(19):1140-50. Doi: 10.1056/NEJM198205133061904. PMID: 7040957.
[14] Wikipedia contributors. 10–20 system (EEG). Wikipedia, The Free Encyclopedia. January 29, 2024,00:50UTC. Available at: https://en.wikipedia.org/w/index.php?title=10%E2%80%9320_system_(EEG)&oldid=1200194996. Accessed July 9, 2024.
[15] Jewett DL, Williston JS. Auditory-evoked far fields averaged from the scalp of humans. Brain. 1971;94(4):681-96.
[16] Ray B, Raman S, Sen S, Sharma M, Ghosh KC, Saha AM. A study of brainstem auditory evoked responses in normal human subjects and normal variations as a function of stimulus and subject characteristics. Int J Res Med Sci. 2016;4(9):4042-49.
[17] Moulton EA, Burstein R, Tully S, Hargreaves R, Becerra L, Borsook D. Interictal dysfunction of a brainstem descending modulatory center in migraine patients. PloS One. 2008;3(11):e3799.
[18] Stockard JJ, Stockard JE, Sharbrough FW. Nonpathologic factors influencing brainstem auditory evoked potentials. Am J EEG Technol. 1978;18(4):177-209.
[19] Groves PM, Thompson RF. A dual-process theory of habituation: Neural mechanisms. In. Physiological substrates. 1973 Jan 1 (pp. 175-205). Academic Press.
[20] Schoenen J. Deficient habituation of evoked cortical potentials in migraine: A link between brain biology, behavior and trigeminovascular activation? Biomed Pharmacother. 1996;50(2):71-78.
[21] Coppola G, Pierelli F, Schoenen J. Habituation and migraine. Neurobiol Learn Mem. 2009;92(2):249-59.
[22] Gawel M, Connolly JF, Rose FC. Migraine patients exhibit abnormalities in the visual evoked potential. Headache: The Journal of Head and Face Pain. 1983;23(2):49-52.
[23] Judit A, Sandor PS, Schoenen J. Habituation of visual and intensity dependence of auditory evoked cortical potentials tends to normalize just before and during the migraine attack. Cephalalgia. 2000;20(8):714-19.
[24] Main A, Dowson A, Gross M. Photophobia and phonophobia in migraineurs between attacks. Headache. 1997;37(8):492-95.
[25] Vingen JV, Pareja JA, Støren O, White LR, Stovner LJ. Phonophobia in migraine. Cephalalgia. 1998;18(5):243-49.
[26] Ashkenazi A, Mushtaq A, Yang I, Oshinsky ML. Ictal and interictal phonophobia in migraine- a quantitative controlled study. Cephalalgia. 2009;29(10):1042-48.
[27] Wang W, Timsit-Berthier M, Schoenen J. Intensity dependence of auditory evoked potentials is pronounced in migraine: An indication of cortical potentiation and low serotonergic neurotransmission? Neurology. 1996;46(5):1404.
[28] Kalita J, Misra UK, Bansal R. Phonophobia and brainstem excitability in migraine. Eur J Neurosci. 2021;53(6):1988-97.
[29] Ambrosini A, Rossi P, De Pasqua V, Pierelli F, Schoenen J. Lack of habituation causes high-intensity dependence of auditory evoked cortical potentials in migraine. Brain. 2003;126(9):2009-15.
[30] Yamada T, Dickins QS, Arensdorf K, Corbett J, Kimura J. Basilar migraine: Polarity-dependent alteration of brainstem auditory evoked potential. Neurology. 1986;36(9):1256.
[31] Gopal KV, Allport JM, Baldridge MR. Auditory behavioral and evoked potential measures in migraineurs during attack-free period. Audiological Medicine. 2007;5(3):160-68.
[32] Vijayalakshmi TN, Jayaraman RM, Bhanu, Anandan H. Evaluation of brainstem auditory evoked potentials in migraine individuals. International Journal of Contemporary Medical Research. 2016;3(9):2522-25.
[33] Benna P, Bianco C, Costa P, Piazza D, Bergamasco B. Visual evoked potentials and brainstem auditory evoked potentials in migraine and transient ischemic attacks. Cephalalgia. 1985;5(2_suppl):53-58.
[34] Sand T, Vingen JV. Visual, long-latency auditory and brainstem auditory evoked potentials in migraine: Relation to pattern size, stimulus intensity, sound and light discomfort thresholds and pre-attack state. Cephalalgia. 2000;20(9):804-20.

PARTICULARS OF CONTRIBUTORS:
1. Postgraduate Student, Department of Physiology, SMS Medical College, Jaipur, Rajasthan, India.
2. Professor, Department of Physiology, SMS Medical College, Jaipur, Rajasthan, India.
3. Associate Professor, Department of Physiology, SMS Medical College, Jaipur, Rajasthan, India.
4. DM Student, Department of Neurology, SMS Medical College, Jaipur, Rajasthan, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Abhishek Saini,
F-8, Major Shaitan Singh Colony, Shastri Nagar, Jaipur, Rajasthan, India.
E-mail: drabhi16@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Oct 25, 2024
- Manual Googling: Apr 12, 2025
- iThenticate Software: Apr 15, 2025 (10%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Oct 24, 2024**
Date of Peer Review: **Jan 31, 2025**
Date of Acceptance: **Apr 17, 2025**
Date of Publishing: **Sep 01, 2025**