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Exploring the potential role of glucose metabolism in migraine: A cross-sectional study from a tertiary care hospital

Rumi Gayen[1], Subhendu Datta[2]

[1]Associate Professor, Department of Biochemistry, College of Medicine and Sagore Dutta Hospital, [2]Assistant Professor, Department of Psychiatry, Medical College, Kolkata, West Bengal, India

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ABSTRACT

Background: Migraine is a complex neurovascular disorder of the brain. Impaired glucose metabolism is suspected to have a potential role in the pathogenesis of migraine. **Aims and Objectives:** This study aimed to evaluate the association between fasting glucose, fasting insulin, and type 2 diabetes with migraine. **Materials and Methods**: A cross-sectional study was conducted involving 29 female patients diagnosed with migraine based on the international classification of headache disorders 3 criteria. Participants underwent physical examinations to assess obesity and metabolic syndrome. Serum insulin, homeostatic model assessment of insulin resistance (HOMA-IR), fasting blood glucose (FBG), and lipid profiles were measured. **Results:** The mean FBG (97.36 ± 31.7) and total cholesterol (162.96 ± 39.13), triglyceride, (112.5 ± 49.51), low-density lipoprotein (99.43–28.90), and high-density lipoprotein (48.75 ± 7.58) values were within normal limits. HOMA-IR was marginally elevated (mean 2.56 ± 1.69; 95% conflict interval: 1.9–3.22), indicating insulin resistance (IR). A statistically significant difference in FBG was observed between patients with and without IR (median FBG: 90; U = 29; z = −3.32; P = 0.001) but no significant differences were found in body mass index or lipid profile (P > 0.05). **Conclusion:** Impaired glucose metabolism was found to have an impact on migraine pathophysiology.

Key words: Migraine; Metabolic syndrome; Insulin resistance

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INTRODUCTION

Migraine is a chronic neurological disease often accompanied by throbbing headaches along with nausea, vomiting, photophobia, and phonophobia, aggravated by stress or activity and relieved by rest. It usually lasts for about 4–72 h[1] and may be associated with aura, though most of the headache attacks are without aura or premonitory symptoms. The lifetime prevalence of Migraine is 15–20%.[1] Migraine typically occurs during the most productive years of a person's life, between the ages of 25 and 50 years.[1] Lifetime and yearly prevalence of migraine in women is 33% and 18%, respectively, whereas it is 13% and 6% in men.[1]

The complexity of headaches is increased by environmental, metabolic, and psychological factors that have an impact on the economic and social functions of patients and their families.

Metabolic factors include impaired glucose homeostasis, insulin resistance (IR), dyslipidemia, reduced cerebrovascular reactivity, and abnormal brain metabolism.[2] The relationship between metabolic factors and migraine is inconsistent. Few studies support the view that metabolic factors and migraine have a close relationship while others oppose this view. Ali et al., reported that migraine patients often exhibit key features of metabolic syndrome, such as diabetes mellitus (DM), higher body mass index (BMI), and

Address for Correspondence:
Subhendu Datta, Assistant Professor, Department of Psychiatry, Medical College, Kolkata, West Bengal, India. **Mobile:** 9231970610.
E-mail: drsubhendu76@gmail.com

increased waist circumference (WC). Findings are the same in another study conducted by Ali et al., where there was a higher frequency of migraine in 154 non-insulin-dependent diabetic patients than in the control population. In contrast, there are 2 large population-based studies in which no connection was found between migraine and DM.[3]

Skipping meals or fasting for extended periods, leading to reduced blood glucose levels, may provoke or intensify migraine attacks. Fasting is one of the most well-known and frequently reported migraine triggers with a percentage range from 39% to 66%. In particular, some experts reported that small alterations in blood glucose might change pain receptors in the brain for some genetically predisposed individuals, which contributes to fasting headaches.[1]

Impaired glucose metabolism may have a role in the pathogenesis of migraine pain. Here we tried to find out the relationship between migraine and glucose-related traits like fasting glucose, fasting insulin, IR, and type 2 diabetes.

Although several studies have explored the potential links between migraine and metabolic dysfunction, including IR and type 2 DM, the findings remain inconsistent and inconclusive. While some research supports an association between migraine and impaired glucose metabolism, other large-scale population studies have failed to demonstrate any significant association. Moreover, limited studies have systematically evaluated specific biochemical markers such as fasting glucose, fasting insulin, and homeostatic model assessment of IR (HOMA-IR) in migraine patients, especially in tertiary care settings and within diverse ethnic populations. This gap highlights the need for focused investigations to clarify whether altered glucose metabolism contributes to migraine pathogenesis or serves merely as a coexisting condition.

Based on the current background and gaps in the literature, we hypothesized that individuals with migraine exhibit significantly higher levels of fasting insulin, fasting glucose, and IR (as measured by HOMA-IR). Furthermore, we proposed that there may be a higher risk of type 2 DM among individuals suffering from migraine.

Aims and objectives

To explore the relationship between migraine and glucose metabolism with a focus on insulin resistance and other metabolic indicators in adult female patients.

MATERIALS AND METHODS

It was a cross-sectional study carried out at the outpatient department (OPD) of psychiatry and department of Biochemistry of the College of Medicine and Sagore Dutta Hospital, a tertiary care center in West Bengal, India.

A total of 29 female patients diagnosed with migraine were enrolled in the study. Patients were recruited from the psychiatry OPD. The diagnosis was made by the psychiatrist according to the international classification of headache disorders (ICHD-3) criteria for migraine**.** The sample was limited due to the short recruitment period and the availability of eligible participants at our tertiary care center. All participants were female, consistent with the known higher prevalence of migraine among women. This homogeneity allowed for focused analysis but may limit the generalisability of the findings to male populations.

Inclusion criteria

Participants aged between 15 and 50 years, without any known endocrine dysfunction and not currently on medications that could affect metabolic parameters, were included in the study.

Exclusion criteria

Patients with known endocrine disorders (such as DM or thyroid dysfunction), or those on medications affecting lipid metabolism or body weight (e.g., statins, fibrates, corticosteroids, oral contraceptive pills, sodium valproate), were excluded. Pregnant individuals were also excluded. The purpose of the study was explained to the patients and written informed consent was obtained thereafter. The institutional ethics committee approved the study (Vide no; CMSDH/IEC/230/03-2021 dated March 27, 2021).

Procedures

After collecting sociodemographic data patients were evaluated based on the presence or absence of aura, precipitating, aggravating, or relieving factors, and duration of headache. The severity of migraine was assessed based on the patient's report.

Measures/anthropometric measurements

In this study height and WC in centimeters, and weight in kilograms had been taken as anthropometric data of the participants. WC was measured using a plastic tape midway between the last rib and the iliac crest at the end of normal expiration. BMI was determined by the standard formula of calculating the weight (in kilograms) divided by height squared (in meters) (kg/m^2).

We used a BMI of 23–24.99 as overweight and a BMI of more than 25 as obese for our current study.[4]

Biochemical analysis

Blood samples were collected for blood glucose, triglyceride (TG), total cholesterol (TC), high-density lipoprotein-

cholesterol (HDL-C) and low-density lipoprotein-cholesterol (LDL-C), Fasting insulin, thyroid stimulating hormone (TSH), free thyroxine (FT4) and analyzed in the Department of Biochemistry at College of Medicine and Sagore Dutta Hospital. The (HOMA-IR) was determined by: Fasting insulin (μU/mL)×fasting glucose (mmoL/L)/22.5. HOMA-IR value >2.5 indicates a state of IR.

The National Cholesterol Education Program Adult Treatment Panel III criteria necessitate at least three of the following to define metabolic syndrome: Fasting blood glucose (FBG) ≥100 mg/dL, blood pressure ≥130/80 mmHg, a WC of >90 cm for men and >80 cm for women, HDL-C <40 mg/dL for men and <50 mg/dL for women, and TGs >150 mg/dL.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using appropriate statistical techniques. Descriptive statistics included means, standard deviations, and frequency distributions (percentages). The normality of continuous variables was assessed using the Shapiro-Wilk test. For non-parametric data, the Mann-Whitney U test was used. P<0.05 was considered statistically significant.

RESULTS AND ANALYSIS

Demographic profile of participants (vide Table 1)

The study was conducted on a total of 29 female patients diagnosed with migraine. The age of participants ranged from 18 to 50 years, with a mean age of 32.4±9.04 years. All participants belonged to a low socioeconomic background, with an average monthly family income of approximately Rs. 7,000. In terms of marital status, a majority of the participants (80%) were married. Regarding educational attainment, 86% of the subjects had an education level below the 10th standard.

Table 1: Presents the detailed sociodemographic characteristics of the study population

Characteristics	Migraine patients (n=29) (%)
Age (mean±standard deviation)	32.4±9.05
Education	
Below 10th standard	86
Above 10th standard	14
Marital status	
Married	80
Unmarried	20
Employment status	
Employed	40
House wife	40
Unemployed	20
Family income (average per month)	Rs. 7000
Family history of headache	
Yes	14
No	86
Family history of metabolic syndrome	Nil

Clinical variables in migraine patients (vide Table 2)

The majority of patients reported recurrent headaches. A weekly frequency (1 episode/week) was noted in 17.2% of the participants, whereas 3.4% reported experiencing 5 episodes/month. Sleep duration among patients ranged from 4 to 9 h, with 37.9% reported 9 h of sleep and 10.3% reported only 4 h. A notable 55.2% of patients reported unilateral headaches. The distribution of headache location included: Whole head: 24.1%, Frontal region: 20.7%, Left temporal: 13.8%, Right temporal: 13.8%, Occipital: 10.3%, Vertex: 10.3%, Parieto-temporal: 6.9%. The majority of patients (89.7%) did not report any history of aura. However, most patients experienced identifiable triggering factors and premonitory symptoms before the onset of migraine. Among the associated symptoms, nausea was reported in 69% of patients, while vomiting occurred in 41%. Photophobia was present in 58.8%, and phonophobia in 31%. A positive family history of migraine was present in 80% of the patients. Based on self-reported severity, 87% of the participants described their migraine attacks as severe.

The duration of individual headache episodes varied: 1 h: 10.3%, 1 day: 41.1% (most common), Up to 3 days: 27.6%.

Laboratory variables in migraine patients (vide Table 3)

Laboratory assessments were conducted after 12 h of overnight fasting. Elevated FBG (>125 mg/dL) was observed in 10.3% of patients, indicating potential undiagnosed diabetes. Impaired fasting glucose (100–125 mg/dL) was noted in 13.8% of patients. The majority (72.4%) had normal FBG levels (<100 mg/dL). TC was elevated (>200 mg/dL) in 10.3% of the participants. TG were elevated (>150 mg/dL) in 13.8%. Notably, none of the patients had elevated LDL-C (>100 mg/dL). The mean values of all lipid parameters remained within reference limits (Table 4). The mean HOMA-IR value was 2.56±1.69 (95% conflict interval: 1.9–3.22), which indicates marginally elevated IR in the group. Blood pressure remained within the normal range for all participants, with a mean value of 120/80 mmHg±7.8. Overall, while the mean fasting glucose and lipid values were within normal ranges, a subset of patients exhibited biochemical indicators of impaired glucose metabolism and dyslipidemia. Marginal elevation in IR, as evidenced by HOMA-IR, suggests a possible link between metabolic dysfunction and migraine pathophysiology.

Table 2: Clinical variables in migraine patients

Characteristics	Migraine patients (n=29) (%)
Duration of sleep (hours)	
4 h	10.3
5 h	17.2
6 h	13.8
7 h	3.4
8 h	17.3
9 h	37.9
Frequency of headache	
1episode/3 months	7
1episode/6 months	10.3
1 episode/month	31
1 episode/week	17.2
2 episodes/month	13.8
2 episodes/week	6.9
3 episodes/month	10.3
5 episodes/month	3.4
Duration of headache (hours)	
1 h	10.3
2 h	13.8
12 h	3.4
24 h	41.4
48 h	3.4
72 h	27.6
Laterality of headache	
Yes	55.2
No	44.6
Location of headache	
Frontal	20.7
left temporal	13.8
occipital	10.3
parieto temporal	6.9
Right temporal	13.8
Vertex	10.3
Whole head	24.1
Triggering factor of headache	
Exposure to sunlight	24.1
Loud noise	3.4
Stress	27.6
Tension	10.3
No	34.5
Premonitory symptoms	
Yes	44.8
No	55.2
Presence of aura	
Yes	10.2
No	89.7
Presence of nausea	
Yes	69
No	31
Presence of vomiting	
Yes	41.4
No	58.6
Presence of photophobia	
Yes	58.8
No	41.2
Presence of phonophobia	
Yes	31
No	69
Intensity of headache	
Severe	87
Moderate	13
Presence of vertigo	
Yes	0
No	100
Relieving factor	
Medicine	96.6
Sleep	3.4

Comparison of migraine patients with and without IR (vide Table 5)

The migraine patients were categorized into two groups based on the HOMA-IR cut-off value of 2.5, which distinguishes those with IR from those without. According to the revised consensus BMI classification for Asian Indians:[4] 27.6% of patients were obese (BMI ≥25), and 24.1% were overweight (BMI 23–24.99). The mean BMI of the study population was 23.95±4.28. However, statistical

Table 3: Laboratory characteristics of migraine patients

Variable	Mean±standard deviation	Conflict interval
FBG (mg/dL)	97.36±31.7	85.05–109.68
T-CHO (mg/dL)	162.96±39.13	147.79–178.14
TG (mg/dL)	112.5±49.51	93.30–131.70
LDL (mg/dL)	99.43–28.90	88.22–110.64
HDL (mg/dL)	48.75±7.58	45.81–51.79
TSH (μIU/mL)	3.18±2.08	2.37–3.98
FT4 (μIU/mL)	103±0.22	0.94–1.12
Fasting insulin (μIU/mL)	10.31±3.88	8.81–11.82
HbA1c %	5.76±1.13	5.1–5.62
HOMA-IR	2.56±1.69	1.9–3.22

FBG: Fasting blood glucose, HDL-C: High density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, T-CHO: Total cholesterol, TG: Triglycerides, TSH: Thyroid stimulating hormone, FT_4: Free tetra-iodothyronine, HOMA-IR: Homeostatic model assessment of insulin resistance

Table 4: Reference interval of blood biochemistry

Parameter	Reference interval
FBG	70–100 mg/dL
T-CHO	<200 mg/dL
TG	<150 mg/dL
LDL-C	<100 mg/dL
HDL-C	40–59 mg/dL
HOMA-IR	≤2.5

HOMA-IR: Homeostatic model assessment of insulin resistance, HDL-C: High-density lipoprotein-cholesterol, LDL-C: Low-density lipoprotein-cholesterol, TG: Triglyceride, FBG: Fasting blood glucose, T-CHO: Total cholesterol

Table 5: Characteristics of migraine headache with or without insulin resistance

Variables	Median	Mann–Whitney u	z	P
BMI	23.4	75	−1.3	0.19
WC	86	86.5	−0.80	0.41
FBS	90	29	−3.32	0.001
T- CHO	157	72	−1.44	0.15
LDL-C	92	72.5	−1.41	0.15
HDL-C	47	98.5	−0.28	0.77
TG	94.5	77	−0.94	0.34
TSH	2.5	101	−0.17	0.86
FT4	1	98	−0.31	0.75
Sleep	8	93.5	−0.52	0.6

BMI: Body mass index, WC: Waist circumference, FBG: Fasting blood glucose, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, T-CHO: Total cholesterol, TG: Triglycerides, TSH: Thyroid stimulating hormone, FT_4: Free tetraiodothyronine, HOMA-IR: Homeostatic model assessment of insulin resistance

analysis using the Mann-Whitney U-test showed no significant difference in BMI between the IR and non-IR groups (median BMI: 23.4, U=75, z=−1.3, ρ=0.19). When comparing other metabolic and endocrine parameters-including WC, FBG, TC, LDL-C, HDL-C, TG, TSH, and free thyroxine (FT4)-the median values remained within normal reference ranges (Table 5). None of these variables showed a statistically significant difference between the two groups. However, FBG was the only parameter that demonstrated a statistically significant difference between patients with and without IR (Median FBG: 90 mg/dL. Mann-Whitney U=29, z=−3.32, ρ=0.001) this suggests a notable association between elevated fasting glucose and IR among migraine patients, even when other metabolic indicators remain normal.

DISCUSSION

In our study, female migraine patients had a mean age of 32.34±9.05 years and a mean BMI of 23.95±4.25. These findings were consistent with Amiri et al., review article which reported migraine to be most prevalent in females below the age of 45 years, aligning with our cohort's demographic distribution.[5] The observed BMI skews toward the upper limit, suggesting a trend toward overweight that may be clinically relevant in the context of metabolic health.

The typical duration of headache episodes (1 h–3 days) and associated symptoms such as nausea (69%), vomiting (41%), photophobia (58.5%), and phonophobia (41.5%) corroborate the symptom profile with previous literature.[6,7] Munjal et al., highlighted comparable symptom burdens within the American population, supporting the cross-cultural consistency in migraine presentation.[8] Similarly, our findings related to sleep duration (mean: 7.14±1.88 h) appear normal, yet sleep quality, rather than quantity, may play a more critical role in migraine pathophysiology, as suggested by Xie et al.[9]

Notably, 89.7% of our patients had migraine without aura, consistent with global trends showing this subtype as the most prevalent.[10] The symptom profile-unilateral (55.2%), pulsatile, moderate to severe in intensity, aggravated by physical activity, and associated with nausea and sensory sensitivities-matches the ICHD-3 criteria for migraine without aura.[11] We observed the pain location, most commonly generalized (41.1%) or frontal (20.7%), echoes findings from Leslie Kelman's large-scale study, which observed migraine pain distribution along the trigeminal nerve and fronto temporal areas.[12]

Regarding metabolic parameters, our study found a mean HOMA-IR of 2.56±1.69, exceeding the threshold of 2.5 for IR. This aligned with several previous reports indicating increased IR in both episodic and chronic migraine populations.[1] However, Sacco et al., and two large population-based studies did not find such an association, indicating that this relationship may be context-dependent and influenced by ethnicity, diet, and environmental factors.[3]

Our finding of 10.3% diabetes prevalence among migraineurs aligned with Ali et al., who reported increased rates of diabetes and elevated BMI in migraine patients.[3] However, this contrasts with studies that found no such link or suggested a protective effect of diabetes against migraine.[5] This inconsistency could stem from differences in study design, diagnostic criteria, and population characteristics.

With respect to dyslipidemia, the majority of our lipid profile values remained within normal ranges, although 10.3% and 13.8% had elevated TC and TGs, respectively. This partial dyslipidemic trend has been echoed in some studies,[13,14] while others found no significant differences in lipid profiles between migraine patients and controls.[15] The lack of a strong association in our sample may be due to the relatively young age and controlled co-morbid conditions in the study population.

Interestingly, only FBG showed a statistically significant difference between migraine patients with and without IR (Mann-Whitney U=29; z=−3.32; P=0.001), suggesting glucose dysregulation as a more sensitive indicator in this population. While lipid abnormalities were not significantly different, the presence of central obesity (mean WC: 87.8 cm) and overweight status (BMI: 23.95) in conjunction with elevated HOMA-IR hints at early-stage metabolic syndrome, even in the absence of overt hyperlipidemia.

The biological plausibility linking migraine to IR is supported by evidence of shared pathophysiological pathways. IR was associated with endothelial dysfunction, impaired cerebral glucose uptake, and mitochondrial abnormalities-all of which may contribute to altered cortical excitability and neurovascular dysregulation seen in migraine.[2] Furthermore, IR may elevate circulating free fatty acids and inflammatory cytokines, potentially sensitizing trigeminal nociceptive pathways and lowering the threshold for migraine attacks.[6]

Fasting and hypoglycemia are also known migraine triggers. Even minor fluctuations in glucose levels may activate hypothalamic and brainstem nuclei, areas implicated in migraine initiation. This could explain why a substantial proportion of patients with normal average fasting glucose

still experience migraine exacerbations during fasting states.[16]

Our findings suggest that migraine may serve as a clinical indicator for underlying metabolic dysfunction, particularly IR. Early identification of metabolic abnormalities in migraine patients could allow for preventive strategies, including dietary regulation, weight management, and insulin-sensitizing therapies. Such interventions may not only reduce migraine frequency but also mitigate long-term risks of cardiovascular and metabolic diseases.

Routine screening for metabolic syndrome components in migraineurs-especially in tertiary care settings-may improve outcomes and promote more holistic care. Clinicians should consider evaluating HOMA-IR, WC, and hemoglobin A1C even in non-obese, non-diabetic migraine patients, as subtle metabolic changes may precede overt disease.

Limitations of the study

Several limitations should be acknowledged. First, the cross-sectional design restricts our ability to infer causality between migraine and IR. Longitudinal studies are needed to determine whether metabolic dysfunction precedes the onset of migraine or vice versa. Second, the hospital-based sample may not be representative of the general population and may have introduced selection bias, as patients seeking tertiary care may have more severe disease or concurrent conditions. Third, potential confounders such as physical activity, diet, hormonal fluctuations, and genetic predisposition were not fully controlled for, which could influence both migraine expression and metabolic parameters.

Furthermore, the sample size was modest, and the study population was entirely female, limiting generalisability to male patients.

CONCLUSION

This study adds to the growing body of evidence suggesting a link between migraine and impaired glucose metabolism. Our findings support the need for metabolic screening in migraine patients, especially for IR and abdominal obesity. While FBG and HOMA-IR emerged as potentially useful indicators, further large-scale, longitudinal, and mechanistic studies are needed to clarify causality, evaluate intervention effects, and define guidelines for integrated migraine-metabolic syndrome management.

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REFERENCES

1. Islam MR and Nyholt DR. Glucose-related traits and risk of migraine-a potential mechanism and treatment consideration. Genes (Basel). 2022;13(5):730.
https://doi.org/10.3390/genes13050730
2. Del Moro L, Rota E, Pirovano E and Rainero I. Migraine, brain glucose metabolism and the "neuroenergetic" hypothesis: A scoping review. J Pain. 2022;23(8):1294-1317.
https://doi.org/10.1016/j.jpain.2022.02.006
3. Ali M, Hussein M, Magdy R, Khamis A, Al-Azayem SA, Othman AM, et al. The potential impact of insulin resistance and metabolic syndrome on migraine headache characteristics. BMC Neurol. 2022;22(1):422.
https://doi.org/10.1186/s12883-022-02966-x
4. Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, et al. Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. J Assoc Physicians India. 2009;57:163-170.
5. Amiri P, Kazeminasab S, Nejadghaderi SA, Mohammadinasab R, Pourfathi H, Araj-Khodaei M, et al. Migraine: A review on its history, global epidemiology, risk factors, and comorbidities. Front Neurol. 2021;12:800605.
https://doi.org/10.3389/fneur.2021.800605
6. Rivera-Mancilla E, Al-Hassany L, Villalón CM and MaassenVanDenBrink A. Metabolic aspects of migraine: Association with obesity and diabetes mellitus. Front Neurol. 2021;12:686398.
https://doi.org/10.3389/fneur.2021.686398
7. Maniyar FH, Sprenger T, Schankin C and Goadsby PJ. The origin of nausea in migraine-A PET study. J Headache Pain. 2014;15(1):84.
https://doi.org/10.1186/1129-2377-15-84
8. Munjal S, Singh P, Reed ML, Fanning K, Schwedt TJ, Dodick DW, et al. Most bothersome symptom in persons with migraine: Results from the migraine in America symptoms and treatment (MAST) study. Headache J Head Face Pain. 2020;60(2):416-429.
https://doi.org/10.1111/head.13708
9. Xie YJ, Lin M, Wong YT, Yan L, Zhang D and Gao Y. Migraine attacks and relevant trigger factors in undergraduate nursing students in Hong Kong: A cross-sectional study. J Pain Res. 2022;15:701-713.
https://doi.org/10.2147/JPR.S337465
10. Lucas C. Migraine with aura. Rev Neurol (Paris). 2021;177(7):779-784.
https://doi.org/10.1016/j.neurol.2021.07.010
11. Pescador Ruschel MA and De Jesus O. Migraine Headache. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2025. Available from: http://www.ncbi.nlm.nih.gov/books/nbk560787 [Last accessed on 2025 Jun 11].
12. Kelman L. Migraine pain location: A tertiary care study of 1283 migraineurs. Headache J Head Face Pain. 2005;45(8):1038-1047.

https://doi.org/10.1111/j.1526-4610.2005.05185.x

13. Giri A, Acharya S, Kumar S and Bakre A. Study of clinical profile and association of migraine with dyslipidemia. J Pharm Res Int. 2021;33:1-7.
    https://doi.org/10.9734/jpri/2021/v33i64B35321
14. Uygur-Kucukseymen E and Akca G. Serum lipid profile in migraine and its association with clinical characteristics. Neurol Res. 2023;45(1):57-61.
    https://doi.org/10.1080/01616412.2022.2119723
15. Ge W, Gao L, Zhang Y, Wu K, Chen N and He L. Association between serum lipid levels and severe headache or migraine in representative American population: A cross-sectional study. Curr Neurovasc Res. 2021;18(3):333-342.
    https://doi.org/10.2174/1567202618666210923145635
16. Grech O, Sassani M, Terwindt G, Lavery GG, Mollan SP and Sinclair AJ. Alterations in metabolic flux in migraine and the translational relevance. J Headache Pain. 2022;23(1):127.
    https://doi.org/10.1186/s10194-022-01494-w

Authors' Contributions:
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Work attributed to:
College of Medicine and Sagore Dutta Hospital, Kamarhati, Kolkata, West Bengal, India.

Orcid ID:
Rumi Gayen - https://orcid.org/0009-0002-1787-2318
Subhendu Datta - https://orcid.org/0009-0004-8246-7743

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