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Original Article

Gut microbiota–derived trimethylamine N-oxide (TMAO) and its association with neuroinflammation in migraine

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Background and objectives: Migraine is a common primary headache disorder associated with neuroinflammation, with evidence implicating gut microbiota and metabolites in neurological mechanisms pathways. Since trimethylamine-N-oxide (TMAO), derived from dietary precursors by gut microbial metabolism, is associated with cardiovascular and neurodegenerative conditions, and its role in migraines is unclear, we aimed to evaluate whether serum TMAO level and related metabolites in migraine patients can be biomarkers.

Methods: A total of 106 participants were enrolled, consisting of healthy controls (n=46; mean age ± SD=39±8 yr old) and migraine patients (n=60). The migraine cohort was divided into migraine with aura (n=18; mean age±SD=33±11 yr old) and migraine without aura (n=42; mean age±SD=37±10 yr old), according to the International Classification of Headache Disorders, 3rd edition (ICHD-3). Blood samples were centrifuged, stored at –80°C, and analysed for TMAO, carnitine, betaine, and choline levels using liquid chromatography–mass spectrometry (LC-MS/MS).

Results: There were no significant differences between the groups for age, smoking, or body mass index (BMI). Serum TMAO levels were significantly higher in patients with migraine with aura [2.75 (1.98–3.91) ng/mL] and migraine without aura [2.85 (2.08–5.53) ng/mL] compared to controls [1.40 (1.00–3.01) ng/mL] (P<0.001). Serum choline, betaine, and carnitine levels were significantly higher in the migraine groups (all $P < 0.01$).

Interpretation and conclusions: These findings indicate elevated TMAO levels in patients with migraine, suggesting a possible involvement of TMAO in migraine-associated neuroinflammatory processes. Targeted studies and cohort-based investigations are needed to clarify its role in migraine and evaluate its potential as a biomarker.

Keywords Gut–brain axis; Inflammatory pathways; Microbiota-derived metabolites; Migraine; Neuroinflammation; Trimethylamine-N-oxide

Migraine is a common neurological disorder that causes recurrent headache attacks. The pathophysiology of migraine involves intricate interactions between neurons, glial cells, the vascular system, and inflammatory mediators.[1] In recent years, attention has been directed towards identifying novel biomarkers that may provide insights into migraine mechanisms and facilitate objective diagnosis. One potential biomarker is trimethylamine-N-oxide (TMAO), a small molecular amine oxide synthesised through the hepatic oxidation of trimethylamine (TMA) *via* flavin-containing monooxygenases (FMO), primarily FMO1 and FMO3.[2-5] TMAO is primarily derived from dietary precursors metabolised by gut microbiota, including phosphatidylcholine/choline, carnitine, betaine, dimethylglycine, and ergothioneine. Phosphatidylcholine-rich food products, including eggs, liver, dairy, meat, and fish, are considered the main dietary sources of choline and, consequently, TMAO.[6] Its levels are affected by a range of factors, including age, sex, gut microbiota composition, diet, renal function, and hepatic flavin monooxygenase activity.

Recent findings suggest that TMAO plays a significant role in neurodegenerative diseases by modulating neuroinflammation and disrupting

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endothelial function. Elevated TMAO levels have been linked to increased pro-inflammatory cytokine release, blood-brain barrier dysfunction, and impaired nitric oxide signalling, all of which contribute to disease progression.[7,8] TMAO also activates key inflammatory pathways, including NLRP3 inflammasomes, NF-κB, and MAPK/JNK signalling, further exacerbating neuroinflammation.[9] These findings highlight the critical involvement of TMAO in neurodegenerative disease mechanisms. This study aims to evaluate the potential relationship between circulating TMAO levels and neuroinflammatory processes in individuals diagnosed with migraine

Methods

This observational study was conducted at the Neurology Outpatient Clinic, Lokman Hekim Akay Hospital after obtaining ethical approval from the University Scientific Research Ethics Committee. Written informed consent was obtained from all participants prior to enrollment, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

Study design and participants: This study employed an observational cross-sectional design and was carried out at the Neurology Outpatient Clinic, Lokman Hekim Akay Hospital. Venous blood samples were collected from patients diagnosed with migraine in the interictal phase during routine examinations and treatments at the Neurology Outpatient Clinic between December 2023 and December 2024.

Inclusion/ Exclusion criteria: Participants were consecutively recruited from the Neurology Outpatient Clinic during the study period. The migraine group included adults (≥18 yr) diagnosed with migraine according to the International Classification of Headache Disorders, 3rd edition (ICHD-3), who voluntarily agreed to participate and provided written informed consent. The control group comprised healthy volunteers aged ≥18 yr with no history of migraine or other primary headache disorders. Controls were age- and sex-matched to the patient group and provided written informed consent before enrollment. To minimise potential confounding factors, the same exclusion criteria were applied to both groups. Individuals with a history of ischemic stroke, atherosclerosis, atrial fibrillation, hypertension, myocardial infarction, heart failure, chronic kidney disease, diabetes mellitus, metabolic syndrome, cancer, other neurological disorders, psychiatric disorders, or gastrointestinal diseases were excluded. Participants who had used antibiotics or L-carnitine within the previous three months, as well as pregnant or breastfeeding women, were also excluded.

Demographic variables, including education level, sex, age, body mass index (BMI), and smoking status, were obtained through face-to-face interviews using a standardised questionnaire. Information regarding comorbidities (*e.g.*, hypertension, diabetes mellitus, thyroid disease, rheumatoid arthritis, systemic lupus erythematosus, heart disease, infection, and other inflammatory diseases) and migraine-related clinical characteristics (migraine duration, migraine subtype, analgesic use, presence of photophobia or phonophobia) were collected based on both patient self-reports and electronic medical records to ensure accuracy and completeness of the data.

Blood sample collection and processing: Following at least 8 h of overnight fasting, venous blood samples were collected into vacuum tubes containing gel separators (Vacutainer SST, Becton Dickinson, USA). Blood samples were allowed to clot at room temperature for 30 min, then centrifuged at 1300 ×g for 10 min. Serum fractions were separated and transferred to sterile Eppendorf tubes. They were stored at -80 °C until biochemical analyses were carried out.

Analysis of serum TMAO levels: Serum TMAO concentrations were quantified using a slightly modified version of the method described by Li *et al*[10] with liquid chromatography–tandem mass spectrometry (LC-MS/MS). For sample preparation, 50 µL of serum, 10 µL of trimethylamine-d$_9$ N-oxide (internal standard solution, 500 ng/mL), and 200 µL of acetonitrile (ACN) were mixed. The obtained mixture was vortexed at room temperature for 10 min and centrifuged at 4 °C at 2000 xg for 5 min. Subsequently, 50 µL was taken from the supernatant and transferred to a new microtube, diluted with 50 µL of 30% ACN, and 5 µL was injected into the LC-MS/MS system for analysis. Chromatographic separation was achieved using a Thermo Dionex Ultimate 3000 HPLC system coupled to a TSQ Quantum Access Max triple quadrupole mass spectrometer (Thermo Fisher Scientific, USA). Data were acquired using positive electrospray ionisation mode with multiple reaction monitoring for TMAO and the internal standard. Quantification was based on calibration curves prepared from standards. The results were given in µmol/L.

Biochemical and haematological analyses: Fasting venous blood samples were collected from all participants after an overnight fast. Routine biochemical parameters, including fasting blood glucose (FBG),

Table. Comparison of demographic characteristics between control and migraine groups

Characteristics	Control (n=46)	Migraine with Aura (n=18)	Migraine without Aura (n=42)	*P* value
Age (yr, median [IQR])	41 (36-43)	30 (24-43)	36 (28-45)	0.097
Gender, Female (%)	40 (87)	15 (83)	37 (88)	0.619
BMI (Body mass index)	25.6 (22.4-28.4)	23.6 (21.4-27.2)	25.3 (21.7-27.8)	0.629
Pre-university graduate/University graduate and above	15/31	5/13	13/29	0.932
Smoking (No/Yes)	42/4	14/4	37/5	0.305

urea, creatinine, estimated glomerular filtration rate (e-GFR), aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), lipid profile, ferritin, C-reactive protein (CRP), thyroid-stimulating hormone (TSH), vitamin B12, vitamin D, and folic acid, were analysed using standard laboratory methods. Complete blood count parameters were measured.

Statistical analysis: Statistical analyses were performed using IBM SPSS Statistics® 27.0 (IBM Corp., NY, USA) and Microsoft Excel® 2010. The normality of data distribution was assessed using the Shapiro–Wilk test.

Categorical variables were evaluated with Pearson's chi-square test. Since most variables did not show a normal distribution, non-parametric tests were applied. Comparisons among groups were conducted using the Kruskal–Wallis test. When a significant difference was detected, post-hoc pairwise comparisons were performed using the Bonferroni correction to adjust for multiple comparisons. The Bonferroni correction was applied to post-hoc pairwise comparisons following the Kruskal–Wallis test to adjust for multiple testing among the study groups (control, migraine with aura, and migraine without aura). Data are presented as mean ± standard deviation (SD) or median (interquartile range, IQR), as appropriate. $P<0.05$ was considered statistically significant.

Results

A total of 106 individuals were included, comprising a healthy control group (n=46; 40 females) and a migraine group (n=60; 52 females). The migraine group was further divided into two subgroups according to the ICHD-3 diagnostic criteria: migraine with aura (n=18) and migraine without aura (n=42).

The control, migraine with aura, and migraine without aura groups demonstrated comparable demographic profiles (**Table**).

Serum levels of TMAO, choline, betaine, and carnitine were significantly elevated in both migraines with aura (n=18) and migraine without aura (n=42) groups compared to controls (n=46) (all $P<0.001$) **(Supplementary Table; Figure)**. No significant differences were observed among groups in fasting blood glucose (FBG), urea, hemoglobin, vitamin B12, vitamin D, folic acid, LDL cholesterol, HDL cholesterol, triglycerides, aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), creatinine, estimated glomerular filtration rate (e-GFR), thyroid-stimulating hormone (TSH), C-reactive protein (CRP), or ferritin levels ($P>0.05$). White blood cell (WBC) counts were significantly higher in the migraine without aura group compared to controls ($P=0.015$), and monocyte counts were also elevated in the migraine without aura group relative to controls ($P=0.037$).

There were no significant differences between migraine with aura and migraine without aura groups in terms of age of migraine onset, disease duration, monthly attack frequency, and pain intensity (VAS score) ($P>0.05$). However, prodromal symptoms were significantly more common in migraine with aura group ($P=0.006$). No significant differences were found between the groups in terms of family history, nausea, vomiting, photophobia, phonophobia, menstrual relationship, vestibular migraine status, use of prophylactic treatment, analgesic overuse, or presence of brain MRI lesions ($P> 0.05$).

Significant positive correlations were found between TMAO, choline, and betaine levels (TMAO-choline: r=0.503, $P<0.001$; TMAO-betaine: r=0.353, $P=0.018$). Choline and betaine (r=0.437, $P<0.001$) and carnitine (r=0.393, $P<0.001$) levels also showed positive correlations.

This multinomial logistic regression analysis examined the associations between estimated glomerular filtration rate (e-GFR), metabolites, and migraine subtypes compared to healthy controls, while adjusting for age and monocyte levels. For migraine with aura, younger age remained a significant risk factor, with each additional yr associated with an 11.7%

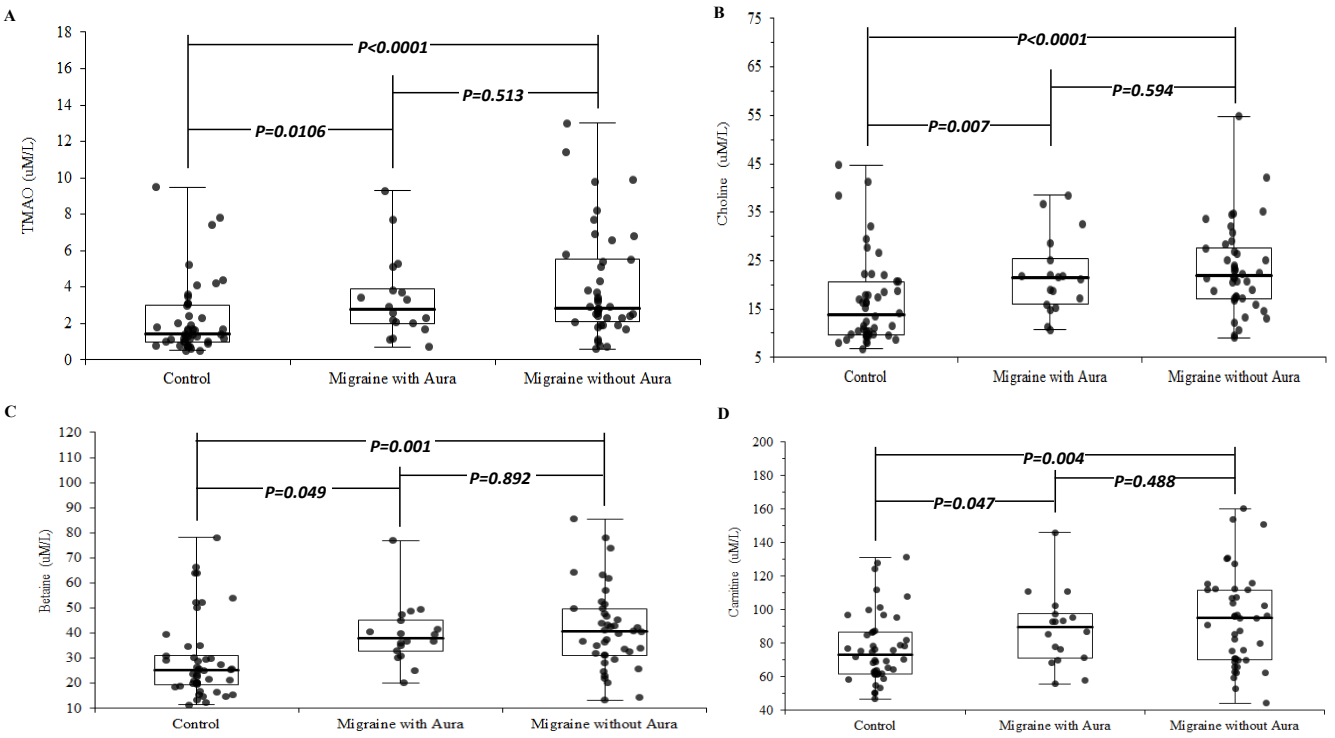


Figure. Serum TMAO (**A**) choline, (**B**) betaine, (**C**) and carnitine (**D**) concentrations in the control, migraine with aura, and migraine without aura groups. Data are shown as median (interquartile range). Statistical differences were evaluated using the Kruskal–Wallis test with post-hoc pairwise analysis. P^*<0.05 compared to control.

reduction in odds (OR=0.883, 95% CI: 0.819–0.953, *P*=0.001). Interestingly, e-GFR showed no significant association (*P*=0.397), while betaine showed a positive but non-significant association (OR=1.034, 95% CI: 0.989-1.081, P=0.144). Carnitine emerged as a significant predictor, with each 1 µM/L increase associated with 4.1% higher odds (OR=1.041, 95% CI: 1.008–1.075, *P*=0.013), and TMAO again showed the strongest association with a 59.5% increased odds per unit increase (OR=1.595, 95% CI: 1.128–2.255, *P*=0.008). Choline and monocyte levels were not significantly associated with migraine with aura. In the migraine without aura group, age showed a weaker but still significant protective effect (OR=0.932, 95% CI: 0.873–0.996, *P*=0.036). Betaine reached significance in this subtype (OR=1.039, 95% CI: 1.001–1.078, *P*=0.045), as did carnitine (OR=1.045, 95% CI: 1.016–1.074, *P*=0.002) and TMAO, which demonstrated the most robust association with 59.3% increased odds (OR=1.593, 95% CI: 1.172–2.164, *P*=0.003). Choline and monocyte levels remained non-significant predictors.

Discussion

In this study, serum TMAO, choline, betaine, and carnitine levels were observed to be significantly higher in both migraine with aura and migraine without aura patients compared to the control group; this supports the presence of a metabolic component in migraine pathophysiology. Lack of significant differences in TMAO-related metabolites between migraine with aura and migraine without aura subgroups suggests that these metabolic pathways may exert similar pathophysiological effects across different migraine phenotypes.

Elevated TMAO levels have previously been reported to be associated with vascular dysfunction, atherosclerosis, and cerebrovascular diseases, increasing the risk of stroke, and triggering oxidative stress by impairing endothelial function.[11,12] High circulating TMAO levels were shown to impair nitric oxide signalling and inhibit nitric oxide–mediated vasodilation, leading to endothelial dysfunction.[13] It was also reported that through reduction of claudin expression, TMAO disrupted the blood–brain barrier, contributing to cerebral small vessel dysfunction after stroke.[14] Upon crossing the blood-brain barrier, TMAO was suggested to contribute to neuroinflammation and demyelination, in turn affecting central nervous system functions.[15,16] These studies indicate that elevated TMAO levels in migraine patients may contribute to pathogenesis through both peripheral vascular

mechanisms and direct effects on the central nervous system.

The elevated choline, betaine, and carnitine levels found in migraineurs in this study are also noteworthy. In addition to their primary role in TMAO production, these metabolites also play independent roles important for nervous system functions. Choline is a critical substrate for acetylcholine synthesis, methylation reactions, and synaptic plasticity.[17] Betaine and carnitine, on the other hand, are associated with energy metabolism and mitochondrial function[18] Therefore, increases in these metabolites may be related to energy metabolism disorders and neuronal hyperexcitability that underlie migraine. Similar to increasing TMAO levels, which are shown to be associated with neuroinflammation, oxidative stress, and endothelial dysfunction influencing the development and severity of migraine,[13] elevated levels of choline, betaine, and carnitine were shown to be consistent with the disruptions in methylation pathways, one-carbon metabolism, and energy metabolism, potentially contributing to neuronal excitability and vascular tone.[19] The parallel elevation of these metabolites in both migraine groups suggests that metabolic derangement may be a common feature of migraine, regardless of the presence of aura.

In this study, no statistically significant differences were found between the migraine with aura and migraine without aura groups in terms of migraine age of onset, disease duration, monthly attack frequency, and pain intensity (VAS score). This suggests that both subtypes share similar characteristics in terms of clinical course and disease burden. Although prodromal symptoms were more frequent in the migraine with aura group, the groups did not differ significantly in nausea, vomiting, photophobia, phonophobia, family history, menstrual association, vestibular migraine, prophylactic treatment use, analgesic overuse, or brain MRI lesions. These results suggest that migraine subtypes largely overlap in clinical features, but those prodromal symptoms may be a distinguishing feature in migraine with aura.

This study has some limitations. The number of patients in migraine with aura subtype was relatively limited due to its lower prevalence among migraine patients. This resulted in a small sample size for the migraine with aura subgroup and is considered a methodological limitation that may have affected the statistical power of the subgroup analyses. Due to the relatively low prevalence of migraine with aura subtype in patients with migraine, the sample size for this group was limited. This should be considered a methodological limitation that may affect the statistical power of subgroup comparisons.

A significant limitation of this study is the lack of assessment of dietary habits. Serum TMAO levels can be affected by nutritional factors, particularly consumption of red meat, eggs, and fish. However, because the study involved a clinically specific patient group, standardised nutritional data specific to each group could not be obtained.

One of the main limitations of this study is the inability to measure specific pro-inflammatory cytokines (TNF-α, IL-1β, IL-6, and YKL-40) that directly reflect neuroinflammation. These analyses could not be performed due to the limited sample size and biological material. Although CRP was assessed and no significant difference was found between the groups, the inability to measure cytokine levels limits a more detailed interpretation of the possible mechanistic relationships between TMAO and neuroinflammation.

Our findings suggest that TMAO-related vascular and immune processes may mediate a state of low-grade inflammation in migraine. Multinomial logistic regression analyses adjusted for age and monocyte levels demonstrated that gut microbiota–derived metabolites, particularly TMAO and carnitine, were independently associated with migraine in both subtypes. The consistently strong association of TMAO supports its potential role in migraine pathophysiology, while the significance of carnitine suggests a link with energy metabolism and mitochondrial processes. Collectively, these findings highlight TMAO and carnitine as potential biomarkers involved in shared pathophysiological mechanisms across migraine phenotypes. Future studies integrating metabolic, vascular, and haematological parameters are warranted, and therapeutic strategies targeting gut microbiota-derived metabolites or inflammatory pathways may hold promise for migraine prevention and treatment.

Declaration: Authors declare that a preliminary version of this work was presented as a poster at the 49th FEBS Congress, Istanbul, Türkiye, from July 5-9, 2025, and was published as a conference abstract in *FEBS Open Bio. 2025;15(2), p. 462* (*doi:* https://doi.org/10.1002/2211-5463.70071).

Author contributions: YA, EE, ZÖ, CCS, MAS, DY: Conceptualisation, design, methodology, manuscript writing, supervision; YA, ZÖ: Data acquisition; YA, CCS, MAS: Statistical analysis; YA, DY: Interpretation; YA, MAS: Intellectual content;

शोध-संदेश

माइग्रेन के मरीजों में TMAO (trimethylamine N-oxide - आंत के बैक्टीरिया द्वारा बनने वाला एक रसायन) का स्तर अधिक पाया गया, जिससे संकेत मिलता है कि आंत (गट) के बैक्टीरिया और उनसे बनने वाले पदार्थ माइग्रेन में भूमिका निभा सकते हैं। भविष्य के बड़े अध्ययनों से यह पता चल सकेगा कि TMAO माइग्रेन की पहचान और उपचार में कितना उपयोगी हो सकता है।

YA, ZÖ: Resources; EE: Sample collection, data acquisition; MAS: Analysis, visualisation. All authors have read and approved the final printed version of the manuscript.

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