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Exploring social cognition in migraine: a narrative review

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ABSTRACT

Background: Migraine is a neurological disorder that significantly impacts patients' quality of life, with a growing global burden. This condition is characterized by recurrent headaches with symptoms like nausea and photophobia, with limited analysis of its cognitive effects, especially in social cognition.

Methods: This narrative review investigates how migraine, especially in its chronic and medication-overuse forms, affects social cognition, focusing on domains such as Theory of Mind (ToM), emotion recognition, and empathy. Social cognition impairments in patients with migraine contribute to difficulties in maintaining relationships and effective communication, exacerbating the disorder's emotional and social burden.

Results: The reviewed literature shows that migraine is associated with a significant deficit in social cognition, particularly in chronic and medication-overuse headache (MOH). ToM deficits are frequently observed, as individuals with migraine struggle to attribute mental states to others, which impacts their ability to interpret social cues. Emotion recognition impairments, especially in recognizing subtle facial expressions, and difficulties with empathy are also reported, further complicating interpersonal interactions. Neuroimaging studies suggest that these cognitive deficits have a neural basis, with altered activation in areas involved in emotion processing. Alexithymia, often present in patients with migraine, is linked to these social cognitive difficulties, particularly affecting emotional awareness and empathy.

Conclusions: These findings highlight the need for specific assessment tools and interventions aimed at improving social cognition in these patients. Such interventions could help improve the emotional and social impact of the disorder and improve overall patient outcomes. Future research should explore these mechanisms further and develop targeted therapies to support patients' social and emotional well-being.

Key words: migraine, social cognition, Theory of Mind, emotion recognition, empathy.

Introduction

Migraine is a chronic neurological disorder characterized by headache attacks lasting 4-72 hours and typically accompanied by nausea, vomiting, photophobia, and phonophobia. (1) In approximately 25% of cases, these attacks are preceded or accompanied by transient focal neurological symptoms, collectively known as aura. (2) Affecting over one billion people worldwide, migraine ranks among the leading causes of disability and imposes a heavy economic burden through both direct medical costs and productivity losses. (3,4) Disability and productivity loss are markedly pronounced in chronic migraine (CM), particularly when complicated by the concomitant presence of medication overuse headache (MOH).

This multifaceted disorder not only imposes a physical and economic burden but is also frequently associated with conditions such as anxiety, depression, and alexithymia, which further exacerbate its impact on patients' quality of life, (5) and significantly impacts cognitive domains, including attention, memory, and executive function. (6-9) Among these, social cognition appears to be frequently affected, impacting the ability to maintain interpersonal relationships and engage in effective communication, and could be responsible for personal distress. (10-12)

Social cognition enables individuals to construct mental representations of relationships and use them flexibly to navigate social interactions. (13) This domain consists of three main dimensions. First, Theory of Mind (ToM), which consists of the ability to attribute beliefs, intentions, and desires to others in order to predict their behavior; this multifaceted psychological construct comprises two distinct components: the affective component, which enables the recognition and understanding of others' emotional states, and the cognitive component, which underlies the attribution and representation of others' mental states, including their beliefs, intentions, and thoughts. (14-16) The second aspect of social cognition is emotion recognition, defined as the capacity to identify emotions through facial and bodily expressions, which guides appropriate social responses. (17) Lastly, empathy consists of the ability to understand and vicariously share another person's emotional experience without confusing it with one's own. (18)

Growing evidence has highlighted a close association between social cognition deficits, particularly in ToM emotion recognition and empathy, and alexithymia, a personality construct characterized by difficulties in identifying and describing one's own emotions and by an externally oriented cognitive style. (19-25) This link may stem from the overlap of neural networks involved in self-related emotional awareness and in understanding others' affective states. (22) In this context, altered emotional self-awareness may disrupt the construction and flexible use of socio-emotional representations, thereby impairing empathic and mentalizing abilities. While the direct relationship between alexithymia and migraine remains debated, migraine sufferers often present elevated levels of depression and anxiety, which in turn strongly correlate with alexithymic traits. (26,27) Accordingly, even in the absence of primary alexithymia, migraine-associated emotional dysregulation – mediated by psychiatric comorbidities – may impair both intra- and interpersonal emotional processing, with potential downstream consequences for social cognition. Moreover, growing evidence suggests that alexithymia is linked to both the intensity and frequency of migraine attacks, indicating that it may represent a relevant psychological trait in patients with migraine. (26-31)

The robust association between alexithymia and social cognition deficits supports the hypothesis that migraine, particularly

in its chronic form, may contribute to impairments in social cognitive processes, thereby exacerbating its impact on daily functioning and social interactions. (23)

This narrative review aims to synthesize and critically analyze existing literature on the relationship between migraine and social cognition, with a specific focus on domains such as ToM, emotion recognition, and empathy. By examining recent findings, this review seeks to highlight potential cognitive and emotional mechanisms underlying these impairments, their clinical implications, and future research directions for improving assessment and intervention strategies.

Methods

This narrative review followed a non-systematic search to identify, screen, and synthesize relevant literature on the relationship between migraine and social cognition. An initial literature search was carried out in November 2024. Articles were retrieved from databases such as PubMed, Google Scholar, and PsycInfo, selected for their characteristics and relevance for the purposes of this study. The search included the keywords "migraine", "social cognition", "Theory of Mind", "emotion recognition", and "empathy". The final search was performed in January 2025. We selected studies published between 2015 and 2025.

The inclusion criteria were as follows: peer-reviewed articles published in English; studies focusing on social cognition in adult patients with migraine; articles assessing components of social cognition, such as ToM, emotion recognition, or emotional regulation; and studies that have used psychometrically validated instruments or *ad hoc* constructed tasks to assess aspects of social cognition.

Exclusion criteria included: studies addressing pediatric populations; articles focusing on cognitive aspects unrelated to social cognition; case reports or studies without a robust methodological design; articles not peer-reviewed (*i.e.*, grey literature) or under review at the time the search was conducted; and qualitative studies.

After screening for duplicates and relevance, we included 8 studies that met all criteria. These studies are summarized in **Table 1**.

Results

The reviewed studies provide comprehensive insights into the relationship between migraine and social cognition, highlighting consistent impairments across various domains. In the following sections, we will provide a detailed examination of the domain altered in migraine.

Theory of Mind deficits in migraine. ToM is the most extensively investigated aspect of social cognition in migraine patients. Across different studies, patients with migraine consistently

Table 1. Summary of included studies.

Authors and year	Study design	Sample size	Assessment tools	Key findings
Romozzi *et al.*, 2022	Cross-sectional study	61 (EM: 21; MOH: 22; HC: 18)	ThOMAS, RMET	MOH patients showed worse performance across all ThOMAS scales and RMET, higher alexithymia, and anxiety than EM and HC. EM showed intermediate performance.
Bouteloup *et al.*, 2021	Case-control study	50 (CM: 25; HC: 25)	miniSEA (FER, FP test)	Impairments in ToM and emotion recognition in CM patients.
Bottiroli *et al.*, 2023	Case-control study	212 (CM+MOH: 71; EM: 61; HC: 80)	FP test, SS Task, RMET, Tromsø Social Intelligence Scale	CM+MOH patients showed impairments in socio-cognitive abilities, higher alexithymia and autistic traits, and lower social support than HC. They performed worse in RMET compared to both EM and HC.
Bottiroli *et al.*, 2024	Case-control study	72 (CM+MOH: 30; EM: 42)	MASC, RMET	CM+MOH patients had lower accuracy in mental state attribution, with greater impairments in affective ToM and a higher tendency for hypo-mentalization errors compared to EM.
Ataoglu *et al.*, 2024	Case-control study	60 (EM: 23; CM: 19; 30; HC: 30)	RMET	Migraine patients showed significantly lower performance on RMET.
Raimo *et al.*, 2022	Case-control study	80 (CM: 40; HC: 40)	ATT, TMPS, RMET, EAT,	CM patients showed significant impairments in both affective and cognitive ToM and exhibited executive and memory deficits. ToM performance was significantly correlated with migraine frequency and intensity.
Gómez-Beldarrain *et al.*, 2011	Case-control study	81 (CM+MOH: 27; EM: 27; HC: 27)	FP test, RMET, EQ	No significant differences in EQ and RMET at baseline. Lower EQ scores associated with worse clinical outcome at 1-year follow-up. CM+MOH showed significantly lower performance on the FP test compared to EM and HC.
Taxer *et al.*, 2024	Cross-sectional study	70 (CM/high-frequency: 45; HC: 25)	FER test	No overall group difference, but reduced accuracy for sadness/disgust in CM.

EM, episodic migraine; CM, chronic migraine; HC, healthy controls; MOH, medication-overuse headache; ThOMAS, Theory of Mind Assessment Scale; ToM, Theory of Mind; RMET, Reading the Mind in the Eyes Test; mini-SEA, mini-Social Cognition and Emotional Assessment; FER test, facial emotion recognition test; FP test, faux pas test; SS task, Strange Stories task; MASC, Movie for the Assessment of Social Cognition; ATT, Advanced Test of ToM; TMPS, Theory of Mind Picture Sequencing Task; EAT, Emotion Attribution Task; EQ, Empathy Quotient.

demonstrate difficulties in recognizing social *faux pas*, interpreting mental states, and accurately inferring intentions and emotions. In a case-control study, Bouteloup *et al.* evaluated 23 severe patients with migraine at a tertiary headache center using the mini-Social Cognition and Emotional Assessment (mini-SEA), which includes a Facial Emotion Recognition (FER) test and a modified faux pas (mFP) test. (10) Although FER performance was comparable to that of controls, 74% of patients scored below the normative range on the mFP test, indicating difficulties in recognizing social *faux pas* and inferring intentions and feelings. Notably, higher levels of alexithymia correlated with lower mFP scores, suggesting that impaired emotional awareness contributes to ToM deficits. These findings point to the potential benefit of cognitive rehabilitation or psychoeducational interventions targeting mentalization abilities to improve social and emotional functioning in individuals with migraine. Romozzi *et al.* further extended their investigation to CM with MOH. The study enrolled 21 episodic migraine (EM) patients, 22 MOH patients, and 18 healthy control (HC) participants. ToM was assessed using the Theory of Mind Assessment Scale (ThOMAS) and the Reading the Mind in the Eyes Test (RMET), alongside measures of alexithymia, anxiety, depression, and dissociation. The findings revealed that MOH patients demonstrated significant impairments in all ToM domains compared to HC and EM patients, especially in recognizing and reasoning about mental and emotional states. Additionally, MOH patients exhibited higher levels of alexithymia and anxiety. Interestingly, no significant differences in depressive or dissociative symptoms were found across groups, though mild to moderate depression was noted in MOH. (11) A more recent study assessed the same group of patients with a broader array of social cognition tasks, including the Strange Stories task (SS), the RMET, the FP test, and the Tromsø Social Intelligence Scale. (31,32) Their results confirmed significant impairments in mental state inference, particularly in affective ToM, while general social intelligence remained preserved. Patients with CM with MOH exhibited higher levels of alexithymic and autistic traits than EM patients and HC. Despite frequent interactions with family members, they reported lower perceived social support, highlighting a disconnect between social engagement and subjective emotional connection, which underscores patients' social difficulties. A key limitation of traditional ToM assessments is their reliance on static, decontextualized tasks, which may not fully capture real-world social cognition. To address this, Bottiroli *et al.* employed the Movie for the Assessment of Social Cognition (MASC), a video-based tool designed to simulate naturalistic social interactions. (32) The authors found that CM+MOH patients were particularly more impaired in the affective than in the cognitive ToM dimension, struggling to infer emotions from dynamic social cues. Notably, patients with migraine exhibited a higher frequency of hypo-mentalization errors, indicating a tendency to under-attribute mental states rather than over-interpret them. This aligns with prior findings on alexithymia, which is reported in nearly 70% of patients with CM or CM+MOH, and suggests that a reduced sensitivity to social cues may hinder emotional engagement. (33) The progression from static to ecologically valid ToM assessments underscores the complexity of social cognition impairments in migraine. Additionally, the MASC findings highlight specific difficulties in processing dynamic, affective cues, with potential consequences for social functioning, emotional well-being, and even adherence to treatment. Future research should explore whether targeted cognitive interventions or psychotherapy could mitigate these deficits, potentially improving both quality of life and migraine management. The association between ToM impairments and broader cognitive dysfunctions in migraine has also been explored. Raimo *et al.* investigated whether these deficits were linked to executive, visuospatial, and memory dysfunction. (12) They enrolled 40 CM patients and 40 HC matched for age, education, and sex. Participants underwent a clinical assessment, a neuropsychological battery, and specific ToM tasks. Cognitive ToM was assessed using the Theory of Mind Picture Sequencing Task (TMPS) and the Advanced Test of ToM (ATT), which measure the ability to infer beliefs, motivations, and intentions. Affective ToM was evaluated with the RMET and the Emotion Attribution Task (EAT), which assesses emotion recognition based on facial expressions and storytelling. In addition, a comprehensive neuropsychological battery assessed executive functioning, memory, and visuospatial abilities. Results showed that CM patients performed significantly worse on cognitive ToM tasks, particularly those requiring second- and third-order false belief reasoning, and also exhibited deficits in executive function, long-term memory, and cognitive flexibility, linking these broader cognitive impairments to their ToM deficits. Interestingly, while cognitive ToM abilities were compromised, affective ToM appeared less altered, as RMET scores did not significantly differ between CM and HC. However, CM patients showed reduced accuracy in recognizing specific emotions, particularly disgust and embarrassment, suggesting selective impairments in processing social and emotional cues.

In contrast, metacognitive abilities, understood as the capacity to monitor, regulate, and reflect upon one's own cognitive and emotional states – including emotional self-awareness and the recognition of emotions in both oneself and others – remain unclear in individuals with migraine. (34) A longitudinal study by Ataoglu *et al.*, comparing CM, EM, and HC, found no significant differences in metacognition, suggesting that while ToM impairments worsen with migraine chronicity, metacognitive abilities may remain preserved. (35) Given the well-documented link between cognitive function and stress in migraine, researchers have also investigated how stress influences ToM performance. Fernandes *et al.* explored this connection by administering the FP test before and after an induced stressor. (36) Their findings showed that patients with migraine, particularly those with chronic forms, exhibited worsened ToM performance under stress, highlighting their vulnerability to stress-related cognitive impairments. These findings have important clinical implications and suggest that interventions enhancing stress resilience and emotional regulation may mitigate stress-induced ToM impairments.

Emotion recognition in migraine. Emotion recognition is a fundamental component of social cognition, enabling individuals to accurately interpret others' emotional expressions and respond appropriately in interpersonal contexts. In individuals with migraine, difficulties in recognizing emotional cues, particularly subtle or ambiguous ones, have been hypothesized to contribute to impaired social functioning and emotional regulation. (24,35) Although research directly targeting emotion recognition in migraine is scarce, a few behavioral studies have attempted to assess this function. The most relevant example is the study by Bouteloup *et al.*, which used the FER subtest of the Mini-SEA to compare patients with CM and HC. (10) While CM patients exhibited significantly worse performance on the FP test, no significant group differences were found on the FER subtest. Although a trend toward lower FER scores was noted in the CM group, the absence of statistical significance suggests that deficits in emotion recognition may not be a consistent feature across all migraine populations or may depend on specific stimulus characteristics, such as the static nature of the emotional expressions used. A more recent study explored FER performance in individuals with high-frequency or CM using a multimodal assessment approach. (37) Although overall accuracy on the FER task did not significantly differ between patients and healthy controls, descriptive trends indicated reduced accuracy in identifying specific emotions, particularly sadness and disgust, in the migraine group. Moreover, within

this group, higher alexithymia scores (measured *via* the Toronto Alexithymia Scale [TAS]-20) were negatively correlated with recognition accuracy for basic emotions such as fear, sadness, and anger. These findings suggest that subtle impairments in emotional decoding may be present in some patients with migraine and could be linked to reduced emotional awareness and central sensitization mechanisms, frequently observed in chronic pain conditions.

Complementary evidence from neuroimaging studies has provided insight into how migraine may alter the neural processing of emotional stimuli. For instance, Szabó *et al.*, using functional MRI, found that individuals with migraine exhibited increased activation in the right middle frontal gyrus and somatosensory cortex when viewing fearful facial expressions, despite not being instructed to evaluate the emotions (implicit task). (38) Likewise, Ren *et al.* demonstrated, *via* magnetoencephalography (MEG), that individuals with migraine showed enhanced effective connectivity from the dorsolateral prefrontal cortex to the superior temporal gyrus during exposure to negative affective images. (39) While these studies do not measure explicit emotion recognition, they highlight altered reactivity to emotional stimuli at the neural level, particularly in response to threat-related cues.

Together, these findings suggest a dissociation between neural reactivity and behavioral performance: patients may exhibit heightened sensitivity to emotionally salient stimuli without consistent impairments in tasks requiring explicit recognition. Future research should aim to clarify whether and how such affective hyper-responsivity contributes to functional deficits in social cognition. Integrating ecologically valid emotion recognition tasks with neurophysiological measures may help identify subgroups of patients who could benefit from targeted interventions, such as emotion regulation training or social skills programs.

Empathy and social adaptability in migraine. Although empathy has received limited direct investigation in migraine populations, Gómez-Beldarrain *et al.* offered an early attempt to explore this domain within a broader neuropsychological assessment of orbitofrontal functioning. (40) In their study, the authors administered the Empathy Quotient (EQ), a self-report scale measuring both affective and cognitive aspects of empathy, along with other social cognition tasks, to patients with CM and MOH, EM, and HC.

Interestingly, no statistically significant differences were observed in EQ scores between the three groups, suggesting that baseline empathic self-perception may not be markedly affected in CM compared to EM or controls. However, a secondary analysis revealed that patients who showed a poor clinical outcome at one-year follow-up (*i.e.*, persistent overuse) had slightly lower baseline EQ scores compared to those with a favorable prognosis. Although its clinical relevance remains uncertain, this difference was statistically significant.

These findings do not provide strong evidence for trait-level empathic deficits in CM. Still, they raise important questions about the role of socio-affective dimensions in disease progression and treatment response. Future studies should aim to clarify whether reduced empathy, particularly in its affective or interpersonal components, may contribute to maladaptive coping styles or decreased adherence to behavioral interventions in CM.

Discussion

This review summarizes mounting evidence that migraine is accompanied by deficits across key social-cognitive domains, including ToM, emotion recognition, and empathy. These deficits, while varying in intensity and detection depending on the assessment tools and patient populations, have significant implications for the social and emotional functioning of affected individuals.

The findings on ToM deficits are among the most consistent across studies. Patients with CM and MOH tend to perform worse in tasks requiring the inference of mental states and affective intentions, as shown in studies using both traditional tasks (*e.g.*, FP test, Strange Stories) and dynamic, ecologically valid measures (*e.g.*, MASC). (21,32) Recently, a study confirmed ToM deficits also in patients with migraine not complicated by medication overuse, using the Yoni test to assess both affective and cognitive components of ToM. (41) The severity of ToM impairment appears to correlate with alexithymia, autistic traits, and the tendency to hypo-mentalize errors, further supporting the role of emotional awareness in social inference processes. (22) Furthermore, the role of alexithymia in modulating social cognition impairments in migraine warrants further investigation. As highlighted in several studies, higher levels of alexithymia, especially difficulties in identifying and describing feelings, were associated with poorer performance in ToM and emotion recognition tasks. This suggests that impaired emotional self-awareness may interfere with the construction of internal representations of others' mental states, contributing to social cognitive dysfunction. Given its high prevalence in chronic and medication-overuse migraine populations, alexithymia could represent not only a comorbid psychological trait but also a potential target for therapeutic intervention aimed at improving emotional processing and social functioning.

Findings regarding emotion recognition were more limited. Only two studies directly assessed this domain using FER tasks, and their results were mixed. Bouteloup *et al.* found no statistically significant group differences on the FER subtest of the Mini-SEA, although a trend toward lower scores in the CM group was noted. (10) Taxer *et al.* similarly reported no significant group-level deficits in FER performance. Still, they identified reduced accuracy for specific emotions, such as sadness and disgust, in patients with chronic or high-frequency migraine. (42) Moreover, in this study, higher alexithymia scores were negatively associated with FER performance for fear, anger, and sadness, suggesting that deficits in emotional awareness may modulate emotion recognition abilities. While these behavioral data are inconclusive, they point to subtle socio-affective processing vulnerabilities in some patients with migraine.

Empathy, a less explored domain, was assessed in a single study through the EQ. No baseline group differences emerged, but lower empathy scores were linked to poorer prognosis at follow-up. Preliminary, these findings suggest a potential contribution of empathic disposition in modulating disease progression or response to treatments.

The etiopathogenesis of altered social cognition in migraine likely involves a complex interplay between neurobiological, psychological, and environmental factors. Although the precise mechanisms linking migraine to social cognition impairments remain to be fully elucidated, several hypotheses have been proposed, drawing on findings from neuroimaging, cognitive neuroscience, and clinical research. A growing body of evidence suggests that migraine is associated with alterations in large-scale brain networks, particularly the default mode network (DMN), the salience network (SN), and the central executive network (CEN). These networks are critically involved in functions such as self-referential thinking, attentional control, emotional regulation, and mentalizing, all of which are essential components of social cognition. (43) In particular, dysfunction of the DMN – which includes the medial prefrontal cortex, posterior cingulate cortex, temporo-parietal junction, precuneus, and lateral temporal cortex – may impair ToM processes by disrupting the brain's ability to simulate others' mental states and shift between internal and external perspectives. Altered connectivity within this network has been observed in migraine patients even during

interictal periods, suggesting persistent neurobiological vulnerability. (44,45)

Moreover, abnormalities in the limbic system, especially the amygdala, have been reported in response to emotional stimuli, such as facial expressions. Studies using functional MRI, such as the one by Szabò *et al.*, have shown heightened activity in limbic and prefrontal areas in response to fearful faces, pointing to emotional hypersensitivity and inefficient emotional regulation in migraine. (38) These alterations may lead to heightened vigilance or misinterpretation of social signals, thereby affecting emotion recognition and interpersonal functioning. From a cognitive standpoint, researchers such as Raimo *et al.* have emphasized the role of executive dysfunctions, including deficits in planning, inhibition, and cognitive flexibility. (12) These impairments, often linked to prefrontal cortex dysfunction, may underlie the reduced performance in ToM tasks observed in CM, particularly in tasks requiring complex perspective-taking or abstract reasoning. Finally, psychological comorbidities frequently associated with migraine, such as alexithymia, anxiety, and depression, may further compromise social cognitive processes by reducing emotional insight and responsiveness. (21)

Moreover, converging evidence supports a partial functional overlap between pain processing and social cognitive systems, with consistent involvement of the anterior insula (AI) and anterior cingulate cortex (ACC). Activation patterns in pain-related areas of these regions have been repeatedly demonstrated in neuroimaging studies both during the direct experience of pain and the observation of others' pain. (46,47) Statistical conjunction analyses and large-scale meta-analyses (48,49) further confirm this co-activation, implicating a core affective-motivational system consistently engaged across first-person and vicarious pain contexts.

Taken together, these findings suggest that social cognition impairments in migraine likely arise from a complex interplay between structural and functional brain abnormalities, cognitive deficits, emotional dysregulation, and chronic stress, rather than from a single isolated mechanism. Building on these findings, social cognition assessment could be fruitfully integrated into the routine clinical management of patients with CM and CM+MOH. Brief validated tools, such as the RMET or FP test, may help clinicians identify individuals with social cognition impairment. Patients presenting high alexithymia, low perceived social support, or comorbid anxiety and depression appear to be most vulnerable to socio-cognitive dysfunction and may benefit from targeted interventions. From a therapeutic standpoint, promising strategies include mentalization-based therapy, emotion regulation interventions, and ToM training programs, all of which have shown efficacy in populations with similar socio-affective deficits (*e.g.*, autism spectrum, borderline personality disorder). Furthermore, interventions such as biofeedback, stress management, and mindfulness-based approaches may help reduce stress-induced declines in social cognition, especially in individuals whose ToM performance worsens under cognitive or emotional load. (50-52)

Integrating these assessments and treatments into multidisciplinary headache care could contribute not only to symptom relief but also to improved emotional well-being, treatment adherence, and interpersonal functioning. These considerations open valuable directions for applied research, including the development of standardized screening algorithms for social cognition in migraine and clinical trials testing socio-cognitive rehabilitation protocols. Despite these findings, several methodological limitations must be acknowledged. Sample sizes of the analyzed articles remain relatively small, particularly in studies involving neuroimaging. There is also considerable variability in the tools used to assess social cognition, which may account for some of the discrepancies in results. Most importantly, very few studies have explored the clinical implications of these deficits or proposed structured interventions. The existing literature provides compelling evidence that migraine, especially in its chronic and medication-overuse forms, is associated with disruptions in social cognition, including ToM, emotion recognition, and affective empathy. These impairments may be both behaviorally observable and neurally mediated, contributing to the social and psychological difficulties often reported by migraine patients.

Future research should prioritize the development and validation of ecologically valid assessment tools that better capture real-life social processing, such as dynamic video-based tasks. Longitudinal studies are also needed to understand whether socio-cognitive impairments precede migraine chronicity or result from it and whether they fluctuate with disease activity. From a clinical perspective, the inclusion of social cognition screening in the assessment of individuals with migraine may help identify those at risk of poorer psychosocial outcomes and risk for chronification. Targeted interventions – such as mentalization-based therapy, social cognition training programs, or emotion recognition training – could be developed to support patients in improving their interpersonal functioning. Additionally, stress regulation strategies, such as biofeedback, mindfulness, or cognitive-behavioral therapy, may help attenuate the negative impact of social cognition impairments, especially in patients who show worsening performance under stress.

Conclusions

Social cognition represents a promising and underexplored domain in migraine research. Understanding and addressing its alterations may offer new opportunities to improve patients' social functioning, emotional well-being, and overall quality of life. Treating patients with migraine by targeting social cognition, alongside other cognitive domains such as executive functions and comorbidities like depression and alexithymia, could lead to a more comprehensive management approach. This holistic treatment strategy has the potential to enhance patients' overall quality of life by not only addressing headache symptoms but also improving emotional regulation, social interactions, and cognitive resilience.

References

1. Olesen J. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia 2018;38:1-211.
2. Evers S, Tassorelli C. Migraine with aura. Handb Clin Neurol 2023;198:169-86.
3. Linde M, Gustavsson A, Stovner LJ, Steiner TJ, Barré J, Katsarava Z, et al. The cost of headache disorders in Europe: the Eurolight project. Eur J Neurol 2012;19:703-11.
4. Gil-Gouveia R, Oliveira AG, Martins IP. The impact of cognitive symptoms on migraine attack-related disability. Cephalalgia 2016;36:422-30.
5. Lantéri-Minet M, Radat F, Chautard MH, Lucas C. Anxiety and depression associated with migraine: Influence on migraine subjects' disability and quality of life, and acute migraine management. Pain 2005;118:319-26.
6. Latysheva N, Filatova E, Osipova D, Danilov AB. Cognitive impairment in chronic migraine: a cross-sectional study in a clinic-based sample. Arq Neuropsiquiatr 2020;78:133-8.
7. Lee SH, Lee Y, Song M, Lee JJ, Sohn JH. Differences in frontal lobe dysfunction in patients with episodic and chronic migraine. J Clin Med 2021;10:2779.

8. Mongini F, Keller R, Deregibus A, Barbalonga E, Mongini T. Frontal lobe dysfunction in patients with chronic migraine: a clinical–neuropsychological study. Psychiatry Res 2005;133: 101-6.
9. Schmitz N, Arkink EB, Mulder M, Rubia K, Admiraal-Behloul F, Schoonmann GG, et al. Frontal lobe structure and executive function in migraine patients. Neurosci Lett 2008;440:92-6.
10. Bouteloup M, Belot RA, Noiret N, Sylvestre G, Bertoux M, Magnin E, et al. Social and emotional cognition in patients with severe migraine consulting in a tertiary headache center: a preliminary study. Rev Neurol (Paris) 2021;177:995-1000.
11. Romozzi M, Di Tella S, Rollo E, Quintieri P, Silveri MC, Vollono C, et al. Theory of mind in migraine and medication-overuse headache: a cross-sectional study. Front Neurol 2022;13: 968111.
12. Raimo S, d'Onofrio F, Gaita M, Costanzo A, Santangelo G. Neuropsychological correlates of theory of mind in chronic migraine. Neuropsychology 2022;36:753-63.
13. Adolphs R. The neurobiology of social cognition. Curr Opin Neurobiol 2001;11:231-9.
14. Premack D, Woodruff G. Does the chimpanzee have a theory of mind? Behav Brain Sci 1978;1:515-26.
15. Frith C, Frith U. Theory of mind. Curr Biol 2005;15:R644-5.
16. Enrici I, Bara BG, Adenzato M. Theory of mind, pragmatics and the brain: converging evidence for the role of intention processing as a core feature of human communication. Pragmat Cogn 2020;26:5-38.
17. Stolier RM, Freeman JB. The neuroscience of social vision. Neuroimaging Pers Soc Cogn Charact 2016;139-57.
18. Decety J, Lamm C. Human Empathy Through the Lens of Social Neuroscience. Sci World J 2006;61146-63.
19. Taylor GJ, Bagby RM, Parker JDA, Grotstein J. Disorders of affect regulation: alexithymia in medical and psychiatric illness. Cambridge: Cambridge University Press, 1997.
20. Brewer R, Collins F, Cook R, Bird G. Atypical trait inferences from facial cues in alexithymia. Emotion 2015;15:637-43.
21. Di Tella M, Enrici I, Castelli L, Colonna F, Fusaro E, Ghiggia A, et al. Alexithymia, not fibromyalgia, predicts the attribution of pain to anger-related facial expressions. J Affect Disord 2018;227:272-9.
22. Di Tella M, Benfante A, Castelli L, Adenzato M, Ardito RB. On the relationship between alexithymia and social cognition: a systematic review. Clin Neuropsychiatry 2024;21: 236-65.
23. Lane RD, Hsu CH, Locke DEC, Ritenbaugh C, Stonnington CM. Role of theory of mind in emotional awareness and alexithymia: implications for conceptualization and measurement. Conscious Cogn 2015;33:398-405.
24. Subic-Wrana C, Beutel ME, Knebel A, Lane RD. Theory of mind and emotional awareness deficits in patients with somatoform disorders. Psychosom Med 2010;72:404-11.
25. Grynberg D, Luminet O, Corneille O, Grèzes J, Berthoz S. Alexithymia in the interpersonal domain: a general deficit of empathy? Pers Individ Dif 2010;49:845-50.
26. Yalınay Dikmen P, Onur Aysevener E, Kosak S, Ilgaz Aydınlar E, Sağduyu Kocaman A. Relationship between MIDAS, depression, anxiety and alexithymia in migraine patients. Acta Neurol Belg 2020;120:837-44.
27. Balaban H, Semiz M, Şentürk İA, Kavakçı Ö, Çınar Z, Dikici A, et al. Migraine prevalence, alexithymia, and post-traumatic stress disorder among medical students in Turkey. J Headache Pain 2012;13:459-67.
28. Muftuoglu MN, Herken H, Demirci H, Virit O, Neyal A. Alexithymic features in migraine patients. Eur Arch Psychiatry Clin Neurosci 2004;254:182-6.
29. Galli F, Caputi M, Sances G, Vegni E, Bottiroli S, Nappi G, et al. Alexithymia in chronic and episodic migraine: a comparative study. J Ment Health 2017;26:192-6.
30. Rota E, Cavagnetto E, Immovilli P, Morelli N, Salari P, Battaggia A. Alexithymia increases the headache pain index in women with migraine: preliminary results. J Clin Med 2025;14:1629.
31. Bottiroli S, Rosi A, Sances G, Allena M, De Icco R, Lecce S, et al. Social cognition in chronic migraine with medication overuse: a cross-sectional study on different aspects of mentalization and social relationships. J Headache Pain 2023;24:1-12.
32. Bottiroli S, Rosi A, Lecce S, Sances G, Allena M, De Icco R, et al. Theory of mind in chronic migraine with medication overuse assessed with the MASC. Sci Rep 2024;14:1-8.
33. Galli F, Tanzilli A, Simonelli A, Tassorelli C, Sances G, Parolin M, et al. Personality and personality disorders in medication-overuse headache: a controlled study by SWAP-200. Pain Res Manag 2019;2019:1874078.
34. Natalucci G, Faedda N, Quinzi A, Fegatelli DA, Fazi M, Verdecchia P, et al. Metacognition and theory of mind in children with migraine and children with internalizing disorders. Neurol Sci 2019;40:187-9.
35. Erkoc Ataoglu E, Toptan T, Vuralli D, Bozdag Y, Bolay H. Theory of mind and metacognition in migraine patients. Ann Med Res 2024;31:249-53.
36. Fernandes C, Dapkute A, Watson E, Kazaishvili I, Chądzyński P, Varanda S, et al. Migraine and cognitive dysfunction: a narrative review. J Headache Pain 2024;25:1-14.
37. Demers LA, Koven NS. The Relation of Alexithymic traits to affective theory of mind. Am J Psychol 2015;128:31-42.
38. Szabó E, Galambos A, Kocsel N, Édes AE, Pap D, Zsombók T, et al. Association between migraine frequency and neural response to emotional faces: an fMRI study. Neuroimage Clin 2019;22:101790.
39. Ren J, Yao Q, Tian M, Li F, Chen Y, Chen Q, et al. Altered effective connectivity in migraine patients during emotional stimuli: a multi-frequency magnetoencephalography study. J Headache Pain 2022;23:6.
40. Gómez-Beldarrain M, Carrasco M, Bilbao A, García-Moncó JC. Orbitofrontal dysfunction predicts poor prognosis in chronic migraine with medication overuse. J Headache Pain 2011;12:459-66.
41. Romozzi M, Di Tella S, Anzuino I, Vollono C, Calabresi P, Silveri MC. Affective and cognitive Theory of Mind in migraine assessed through the Yoni test. Arch Psychol Neurol Psychiatry 2025;1:47-59.
42. Taxer B, Piekartz H, Lauth W, Christova M, Leis S. Exploring facial somatosensory distortion in chronic migraine: the role of laterality and emotion recognition - a cross-sectional Study. Appl Sci 2024;14:8102.
43. Li W, Mai X, Liu C. The default mode network and social understanding of others: what do brain connectivity studies tell us. Front Hum Neurosci 2014;8:52017.
44. Schwedt TJ, Chiang CC, Chong CD, Dodick DW. Functional MRI of migraine. Lancet Neurol 2015;14:81-91.
45. Tessitore A, Russo A, Giordano A, Conte F, Corbo D, De Stefano M, et al. Disrupted default mode network connectivity in migraine without aura. J Headache Pain 2013;14:89.
46. Botvinick M, Jha AP, Bylsma LM, Fabian SA, Solomon PE, Prkachin KM. Viewing facial expressions of pain engages cortical areas involved in the direct experience of pain. Neuroimage 2005;25:312-19.
47. Morrison I, Lloyd D, di Pellegrino G, Roberts N. Vicarious responses to pain in anterior cingulate cortex: is empathy a multisensory issue? Cogn Affect Behav Neurosci 2004;4: 270-8.
48. Lamm C, Decety J, Singer T. Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. Neuroimage 2011;54:2492-502.
49. Singer T, Seymour B, O'Doherty J, Kaube H, Dolan RJ, Frith CD.

Empathy for pain involves the affective but not sensory components of pain. Science 2004;303:1157-62.
50. Ashina M, Katsarava Z, Do TP, Buse DC, Pozo-Rosich P, Özge A, et al. Migraine: epidemiology and systems of care. Lancet 2021;397:1505-18.
51. Perez-Munoz A, Buse DC, Andrasik F. Behavioral Intervention for Migraine. Neurol Clin 2019;37:789-813.
52. Sullivan A, Cousins S, Ridsdale L. Psychosocial Intervention for migraine: A systematic review. J Neurol 2016;263:2369-77.

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