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Occipital *versus* Non-Occipital Migraine Pain: Clinical Characteristics, Differential Diagnosis, Treatment Response, Anatomical, and Pathophysiological Perspectives

Dor Occipital *versus* Dor Não Occipital na Enxaqueca: Características Clínicas, Diagnóstico Diferencial, Resposta ao Tratamento e Perspetivas Anatómicas e Fisiopatológicas

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Abstract

Introduction: In this study, we aimed to compare clinical characteristics and treatment responses in patients with migraine with occipital pain and those with non-occipital pain. We hypothesized that the area of pain could influence clinical features and treatment responses.

Methods: We conducted a retrospective review of patients diagnosed with episodic or chronic migraine who attended a Neurology (Headache) outpatient clinic between January 2022 and December 2024. Patients were divided into two groups: Group 1 (People with migraine with occipital pain) and Group 2 (People with migraine with non-occipital pain). Data were collected on demographic characteristics, clinical features, and treatment responses.

Results: A total of 100 patients were enrolled, with 50 included in Group 1 [39 patients (78%) with episodic migraine; 40 (80%) females], and 50 patients included in Group 2 [40 patients (80%) with episodic migraine; 43 (86%) females]. No significant difference was found in gender distribution (p=0.603), age of migraine onset (p=0.904), or time until diagnosis (p=0.205). Group 1 had more frequent bilateral pain (50% *vs* 38%, p=0.003) and a higher mean of migraine days per month (11 *vs* 6, p=0.004). Similar proportions of patients started oral preventive treatment (70% *vs* 80%, p=0.248). In Group 1, the most prescribed drug was amitriptyline, while in Group 2, it was topiramate. Group 1 had higher treatment failure rates than Group 2 (70% *vs* 31%, p<0.001). After adjusting for monthly migraine frequency, occipital pain remained independently associated with a poorer response to preventive treatment (adjusted OR = 0.33; 95% CI: 0.14–0.77; p=0.01).

Conclusion: Patients with occipital migraine experience more bilateral pain, migraine frequency, and higher treatment failure rates. These findings suggest the need for tailored treatment strategies based on migraine pain localization.

Resumo

Introdução: Este trabalho tem como objetivo comparar as características clínicas e as respostas ao tratamento em doentes com enxaqueca com dor de localização occipital versus não occipital, tendo por hipótese que a área da dor pode influenciar as características dos doentes bem como a resposta ao tratamento.

Métodos: Foi realizada uma análise retrospetiva de doentes com diagnóstico de

enxaqueca episódica ou crónica, seguidos em consulta externa de Neurologia (Cefaleias) entre janeiro de 2022 e dezembro de 2024. Os doentes foram divididos em 2 grupos: Grupo 1 (enxaqueca com dor occipital) e Grupo 2 (enxaqueca com dor não occipital). Foram recolhidos dados demográficos, clínicos e relativos à resposta ao tratamento.

Resultados: Foram incluídos um total de 100 doentes, sendo que 50 foram incluídas no Grupo 1 [39 doentes (78%) com enxaqueca episódica; 40 (80%) do sexo feminino] e 50 foram incluídas no Grupo 2 [40 doentes (80%); 43 (86%) do sexo feminino]. Não se verificaram diferenças estatisticamente significativas na distribuição por género (p=0,603), idade de início da enxaqueca (p=0,904) ou tempo até ao diagnóstico (p=0,205). O Grupo 1 apresentou com maior frequência dor bilateral (50% *vs* 38%, p=0,003) e uma média mais elevada de dias com enxaqueca por mês (11 *vs* 6, p=0,004). Proporções semelhantes de doentes iniciaram tratamento com preventivo oral (70% *vs* 80%, p=0,248). A amitriptilina foi o fármaco mais usado no Grupo 1, enquanto que o topiramato foi o preventivo mais utilizado no Grupo 2 A taxa de insucesso terapêutico foi superior no Grupo 1 (70% *vs* 31%, p<0,001). Após ajustamento para a frequência mensal de enxaquecas, a dor occipital manteve-se associada de forma independente a uma menor resposta ao tratamento preventivo (OR ajustado = 0,33; IC 95%: 0,14–0,77; p=0,01).

Conclusão: Os doentes com enxaqueca de localização occipital apresentam maior frequência de dor bilateral, maior frequência mensal de enxaquecas e taxas mais elevadas de insucesso terapêutico. Estes dados sugerem a necessidade de estratégias terapêuticas adaptadas à localização da dor da enxaqueca.

Introduction

Occipital headaches are frequently observed in both secondary and primary headache disorders, including migraine.[1] Migraine is a primary headache typically described by pulsating pain in the anterior regions, such as retro-orbital or frontotemporal areas, with approximately 60% of cases being unilateral.[2,3] However, it is not uncommon for individuals to report attacks with predominantly posterior pain, particularly in the occipital region, which raises the possibility of other etiologies, such as cervicogenic headache or occipital neuralgia.[4] Notably, previous studies have reported that 39.8% of migraine attacks involve the occipital area.[5] The occipital region, encompassing the great occipital nerve (GON) and lesser occipital nerve (LON), plays a key role in the pathophysiology of occipital pain in migraine.[6] Treatment often focuses on non-oral approaches, including nerve blocks or peripheral occipital nerve decompression surgery.[1,6] However, there is limited data on the clinical features of individuals with migraine experiencing occipital pain and even less on their response to preventive oral treatments.

In this study, we aimed to investigate whether there are differences in clinical characteristics and treatment responses between patients with migraine with occipital pain and those with non-occipital pain migraine. We hypothesized that the area of pain could influence clinical features and treatment responses.

Methods

This retrospective observation cohort study was exempted from our hospital's Institutional Review Board approval, and patient-informed consent was not required. This study was conducted and reported by the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines for observational research.[7]

We systemically reviewed the clinical records of consecutive patients diagnosed with episodic or chronic migraine, meeting the diagnostic criteria of the International Classification of Headache Disorders 3rd edition. The study spanned from January 2022 to December 2024, focusing on patients attending a neurology (headache) outpatient clinic at a tertiary center. Exclusion criteria included patients primarily presenting with other headache types.

The cohort was categorized into two groups: patients with migraine with occipital pain (Group 1) and patients with migraine with non-occipital pain (Group 2). Patients included in Group 1 were those who reported exclusive pain in the occipital region at the onset of attacks and persistence of this same location (with or without irradiation to other areas) for at least the last 5 years. Patients included in Group 2 were those who reported exclusive pain in non-occipital regions and persistence of this same location for at least the last 5 years. Data concerning demographic characteristics (sex, age at onset of migraine, and time until diagnosis), clinical features (lateralization accompanying symptoms, presence of aura, duration of each attack, and headache frequency), other headache comorbidities, and information about acute and preventive treatment were extracted from the electronic records. Treatment responses were categorized as complete (100% reduction in migraine frequency), partial (≥50% reduction), or poor (<50% reduction). For analysis purposes, patients were classified as responders (complete or ≥50% response) or non-responders (<50% response). There were no missing data in the present study.

Statistical analyses

This study constitutes a primary analysis (a priori) based on information from a retrospective cohort. No statistical power calculation was conducted before the study.

Categorical variables are presented as frequencies and percentages, and continuous variables as medians and interquartile ranges. Normal distribution was assessed using the Shapiro-Wilk test. Group comparisons utilized the Mann-Whitney U-test for continuous variables and chi-squared for categorical data. All *p* values were two-tailed, with statistical significance defined as $p < 0.05$. To evaluate whether the lower response rate observed in the occipital group could be explained by the higher baseline migraine frequency, we performed a binary logistic regression model adjusted for the number of monthly migraine days. Results are reported as adjusted odds ratios (OR) with 95% confidence intervals (CI).

The statistical analysis was done using IBM Statistical Package for the Social Sciences (SPSS), version 29.0. The data supporting the study's findings are available from the corresponding author upon reasonable request.

Results

The demographic details, headache characteristics, and treatment responses of the patients included in the study are summarized in **Table 1**.

Demographics

A total of 100 patients were enrolled, with 50 categorized as having migraine with occipital pain [Group 1, 39 patients (78%) with episodic migraine], and 50 as having migraine with non-occipital pain [Group 2, 40 patients (80%) with episodic migraine]. In group 1, only 10 patients (16.7%) reported a modification in the pain pattern within the first 5 years since the onset of their migraine attacks, indicating that the location of occipital pain was persistent over time. All patients underwent head CT or MRI (performed at the time of referral), all of which were normal. Patients with concomitant neck tenderness/pain before or during migraine attacks underwent cervical CT or MRI, without any relevant findings.

Group 1 comprised 40 (80%) females, while Group 2 comprised 43 (86%) females, showing no statistically significant difference in gender distribution between the groups ($p=0.0603$). The median (IQR) age of migraine onset was 20 years (17.5) in the occipital pain group and 14.5 years (10) in the non-occipital group, but this difference was not statistically significant ($p=0.904$); the median time (IQR) until the diagnosis was 10 years (23) in Group 1 and 17 years (19) in Group 2 ($p=0.205$).

Clinical features and comorbidities

Patients in Group 1 presented more frequently with bilateral pain (50% *vs* 38%, $p=0.003$). The frequency of accompanying symptoms, including aura, was similar between the two groups, as was the duration of each attack (**Table 1**). However, Group 1 had a higher average of migraine days per month (11 days *vs* 6 days, $p=0.004$). The prevalence of other headache disorders was low in both groups (12% *vs* 10%, $p= 0.749$). There were no statistical differences regarding medication-overuse headaches in both groups (14% *vs* 22%, $p=0.298$). In Group 1, 20 patients (40%) reported neck pain or tenderness before occipital headaches, while 10 patients (20%) reported neck pain or tenderness during occipital migraine attacks. In Group 2, 18 patients (36%) reported neck pain or tenderness before occipital headaches, while 8 patients (16%) reported neck pain or tenderness during occipital migraine attacks. There

were no differences between the two groups regarding the prevalence of neck pain/tenderness (60% *vs* 52%, *p*=0.204). None of the patients in either group reported neck pain as a migraine trigger or postdromal symptom. During the observation, no patient in either group exhibited tenderness in the neck, paracervical, or occipital areas, a positive occipital Tinel's sign, or restriction to the neck movements, except for two patients who also had cervicogenic headache (one in each group).

Treatment and follow-up

A similar proportion of patients in both groups started oral preventive treatment (70% *vs* 80%, *p*=0.248). In Group 1, the most prescribed drug was amitriptyline (n=14, 10-50 mg/day), followed by topiramate (n=9, 50-100 mg/day), propranolol (n=8, 40-120 mg/day), sodium valproate (n=2, 500-100 mg/day), and finally others (n=2, venlafaxine 150mg/day; flunarizine 10 mg/day). In Group 2, the most prescribed prophylactic treatment was topiramate (n= 16, 500-100 mg/day), followed by propranolol (n=12, 40-120 mg/day), amitriptyline (n= 14, 10-50 mg/day), sodium valproate (n=2, 500-100 mg/day), and finally others (n=1, venlafaxine 150 mg/day). Throughout the follow-up period, patients in Group 1 had higher treatment failure rates than those in Group 2 (70% *vs* 31%, *p*<0.001). A logistic regression model was performed to evaluate whether the lower response rate observed in the occipital group could be explained by the higher baseline migraine frequency. After adjusting for the number of monthly migraine days, occipital pain remained significantly associated with a poorer response to preventive treatment (adjusted OR = 0.33; 95% CI: 0.14–0.77; *p*= 0.01).

Discussion

Migraines are not usually classified according to the location of the headache, which contributes to the limited amount of literature specifically addressing occipital migraine pain.[1] In this retrospective study, we compared the demographic, clinical, and treatment characteristics of patients with occipital pain to those with non-occipital pain in an outpatient setting.

Both groups exhibited a similar age of headache onset, similar proportions of episodic and chronic migraine, and largely equivalent frequencies of associated symptoms. However, bilateral pain was significantly more frequent in the occipital group, consistent with prior studies reporting bilateral involvement in approximately one-third of occipital migraine attacks.[8] The involvement of the greater and lesser occipital nerves may explain this pattern of bilateral pain.[9]

Aura frequency was similar in both groups. Although Wattiez *et. al.*[10] reported a greater prevalence of occipital pain among patients with vestibular migraine, in our sample, only two patients reported vestibular symptoms, both with occipital pain attacks. Given this small number, no firm conclusions can be drawn. In our cohort, the proportion of patients with neck pain/tenderness was substantially similar in both groups.

Table 1. Clinical characteristics and treatment outcomes in patients with occipital versus non-occipital migraine pain.

Variables		Occipital Pain (n = 50)	Non-Occipital Pain (n = 50)	*p*-value
Demographic	Age of onset of migraine, years, median (IQR)	20 (17.5)	14.5 (10)	*p*= 0.904
	Time to diagnosis, years, median (IQR)	10 (23)	17(19)	*p*= 0.205
	Sex (F/M; %)	80/20	86/14	*p*= 0.603
Clinical features	Lateralization (% bilateral)	50	38	*p*= **0.003**
	Sonophobia and/or photophobia (%)	80	70	*p*= 0.088
	Nausea and/or vomiting (%)	82	72	*p*= 0.743
	Aura (%)	34	32	*p*= 0.832
	Duration of migraine episodes <24h, 24-48h, 48-72h, ≥72h (%)	44/18/24/14	40/18/34/8	*p*= 0. 534
	Neck pain/ tenderness (%)	60	52	*p*= 0.204
	Migraine attacks/Month (mean), days	11	6	*p*= **0.004**
Treatment	Medication-overuse (%)	14	22	*p*= 0,298
	Preventive requirement (%)	70	80	*p*= 0.248
	Response to preventives (%)*	50	69	*p*< **0.001**

This table summarizes the demographic and clinical features, as well as treatment outcomes, of patients with occipital and non-occipital migraine. Patients with occipital pain reported a significantly higher frequency of bilateral pain (50% *vs* 38%, *p*=0.003), a greater monthly migraine burden (mean of 11 *vs* 6 days, *p*=0.004), and lower response rates to preventive treatment (69% *vs* 50% responders, *p*<0.001).

F – female; M – male; IQR – interquartile range; SD – standard deviation. *Response to preventive treatment was defined as ≥50% reduction in monthly migraine frequency.

Another finding in our cohort was the shorter median time to diagnosis in the occipital pain group compared to the non-occipital group (10 *vs* 17 years, $p=0.205$). Although not statistically significant, somewhat unexpected, as occipital pain is often perceived as atypical and could theoretically delay diagnosis. A plausible explanation is that occipital pain may prompt earlier referral to a neurologist due to suspicion of secondary causes, thereby facilitating an earlier diagnosis.

Despite a higher proportion of episodic migraine in the overall sample, patients with occipital pain reported nearly double the number of monthly migraine days compared to the non-occipital group. This finding is in line with studies suggesting an association between occipital pain and chronic migraine.[5] Additionally, in studies involving occipital nerve stimulation for chronic migraine, patients with occipital pain experience a high frequency of migraine days, further reinforcing this link.[11]

Differentiating occipital pain in patients with migraine, ON, and CGH can be challenging due to significant phenotypic overlap.[4] Neck pain can be present in all conditions, appearing as a prodromal symptom, during the attack, or as a postdromal symptom in migraine. Symptoms such as tinnitus, dizziness, and nausea or vomiting may be features of both ON and CGH, though they are less prominent than in migraine. While migraine pain tends to be unilateral, shifting, ON, and CGH are often side-locked. ON presents with severe paroxysmal pain lasting seconds to minutes, often accompanied by a dull ache between attacks. Dysesthesia or allodynia in the occipital nerve area and tenderness over nerve branches are key clinical clues. Tinel's sign and the "pillow sign" (pain with neck extension or rotation) may also indicate ON. In contrast, CGH arises from cervical spine or soft tissue disorders. It is linked to neck dysfunction, with headaches often triggered or worsened during head or neck movements. Continuous occipital pain without dysesthesia or allodynia should raise suspicion for possible referral of pain from the cervical structures.[4] Spontaneous intracranial hypotension (SIH) can present with a headache mimicking or having migraine or as a comorbidity (e.g., cervicogenic trigger to migraine).[12] Key findings supporting a diagnosis of SIH include orthostatic headache, which worsens when upright and improves when lying down; it often occurs after activities that increase spinal pressure (e.g., heavy lifting, coughing, sneezing). Other findings include low-CSF-pressure symptoms (such as diplopia or tinnitus) and suggestive MRI findings (pachymeningeal enhancement, brain sagging, engorged venous structures, subdural fluid collections, or an enlarged pituitary gland).[12,13]

The pathophysiology of occipital pain in migraine is complex and involves several theories, including vascular, neurogenic, and muscular mechanisms. A transient central mediated by the trigeminocervical complex (TCC) and its peripheral nociceptors is considered the most likely mechanism initiating migraine headache.[14,15] Activation of nociceptors from the dura mater and cranial vessels is an important source of pain in primary headaches, including migraine.[16]

It has been proposed that occipital headaches arise from neurons with the TCC, where the anatomical and functional overlap of the cervical nerves and trigeminal afferents occurs at the level of the caudal trigeminal nucleus (CTN). Recent research suggests that this complex plays a key role in modulating nociception during migraines, cluster headaches, and other headache disorders. Several studies have demonstrated that pain involving the periorbital and/or frontal regions can coexist with occipital or neck pain in patients with migraine and other headache disorders via TCC. These observations support the idea that the cervical nerves could play a role in head pain that occurs in the frontal region. The upper cervical nerves (C2-C3 nerves) can modulate trigeminal nociceptive signalling based on the convergence of cervical and trigeminal afferent pathways in the trigeminal nucleus caudalis.[17]

Roseda R *et al*[1] study highlights new findings of occipital headaches, revealing that the cerebellum's overlying posterior dura receives innervation from cervicovascular neurons located in the C2 dorsal root ganglion. These neurons, with axons traversing both intracranial and extracranial pathways, connect to the posterior dura. When central cervicovascular neurons associated with the posterior dura undergo sensitization, they may result in increased responsiveness to stimulation of the neck muscles. These findings imply that the source of occipital and frontal migraines may differ, with occipital migraine attacks potentially being more associated with cerebellar abnormalities than non-occipital migraines.[1]

We hypothesize that occipital pain is a marker of more severe or chronic disease. Cervical and occipital pain are frequently reported in patients with chronic migraine and might reflect a process of central sensiti-

zation involving the TCC.[1,14-16] In this context, patients with more migraine days per month and occipital pain could represent a more refractory or chronic pain subgroup, as also supported by studies on occipital nerve stimulation in chronic migraine.[11] Also, Noseda et al revealed that cervicovascular neurons in the C2 dorsal root ganglion innervate the posterior dura, and their sensitization could increase neck-related pain responsiveness, possibly contributing to the chronicity of occipital migraine.[1] These mechanisms warrant further investigation and suggest that occipital pain might be more than a mere topographical variant; it could be a marker of disease severity and chronic central involvement, and this mechanism could explain the higher burden and poorer treatment response in patients with occipital migraine. Also, we hypothesize that in individuals with migraine with occipital pain, there might be an early activation of the occipital area leading to headache, possibly due to intrinsic pathophysiological factors favoring the preferential location of pain in the occipital region. This could explain why only 10 patients (16.7%) in our cohort reported a modification in pain pattern since the onset of migraine. Previous studies suggest that non-oral pharmacological approaches targeting the greater and lesser occipital nerves may be more beneficial for occipital headaches than non-occipital headaches due to their direct action on the involved nerves.[1] Occipital nerve block is effective in both acute and preventive treatment of migraine.[18] In our cohort, fewer than one-third of patients with occipital headaches responded to the oral group. These findings suggest that oral drugs may be less useful for occipital migraines.

To address the potential confounding effect of migraine severity, we conducted a logistic regression analysis adjusted for monthly migraine frequency. The analysis confirmed that patients with occipital migraine were significantly less likely to respond to oral preventive treatment, independently of disease burden (adjusted OR = 0.33; 95% CI: 0.14–0.77; $p = 0.01$). This suggests that the poorer outcomes observed in this group may reflect intrinsic differences beyond baseline frequency.

In our cohort, although a similar proportion of patients in both groups initiated oral preventive treatment, the specific agents prescribed differed. Amitriptyline was more commonly used in the occipital pain group, whereas topiramate was the predominant choice in the non-occipital group. This asymmetry in pharmacological strategy may have influenced outcomes, as these drugs have distinct mechanisms of action, efficacy profiles, and tolerability. Notably, topiramate and amitriptyline, while both considered first-line treatments for migraine prevention, differ in their mechanisms of action and tolerability profiles.[19] Notably, both are first-line agents in migraine prevention, yet previous meta-analyses and clinical guidelines suggest that treatment response may vary across subgroups based on migraine phenotype, including pain localization.[20] Furthermore, treatment response in migraine is multifactorial and may also be affected by other variables such as psychiatric comorbidities, pain phenotype, and duration of disease, factors that we could not fully adjust for due to the sample size. Further studies with larger samples and stratified treatment analysis are warranted.

Notably, none of the patients in our cohort received non-oral interventions such as occipital nerve blocks, limiting our ability to compare the efficacy of alternative preventive strategies. Future real-world studies comparing oral and non-oral approaches are needed to clarify whether treatment response truly differs according to pain location.

To our knowledge, this is the first Portuguese study in a tertiary hospital detailing the clinical findings of patients with occipital pain and comparing them with patients with non-occipital pain observed in a headache outpatient setting. Our study's limitations include its hospital-based and retrospective nature, small sample size, and potential information bias. An additional limitation lies in the classification of treatment response using a simplified scale [complete, partial (≥50%), or poor response). Although this trichotomous classification is widely used in real-world studies and aligns with several clinical trial standards and international guideline recommendations, it does not capture more stratified outcomes. Future prospective studies with structured follow-up may allow a more nuanced and comprehensive assessment of treatment response.

In conclusion, within our cohort, there was an overlap between clinical characteristics and treatment between the 2 groups, except for the bilaterality of pain, number of monthly migraines, and the response to oral preventive drugs, with occipital pain in people with migraine revealing the worst preventive outcomes. These findings provide valuable insights into the demographic and clinical characteristics of occipital migraine patients, emphasizing the need for further research to understand potential

differences in treatment responses of oral and non-oral treatments in this specific migraine subtype. Additionally, larger cohort studies are essential to understand if real differences in treatment responses exist between occipital and non-occipital people with migraine. ■

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Contributorship Statement / Declaração de Contribuição

Conceito e desenho do estudo: Gonçalo Cabral, Rita Pelejão. Aquisição de dados: Gonçalo Cabral, Carolina Gonçalves, Miguel Serôdio. Análise e interpretação dos dados: Gonçalo Cabral, Carolina Gonçalves, Miguel Serôdio. Elaboração do manuscrito: Gonçalo Cabral, Carolina Gonçalves, Miguel Serôdio. Revisão do conteúdo intelectual: Gonçalo Cabral, Rita Pelejão. Aprovação final do manuscrito concluído: Gonçalo Cabral, Carolina Gonçalves, Miguel Serôdio, Rita Pelejão.

Study concept and design: Gonçalo Cabral, Rita Pelejão. Acquisition of data: Gonçalo Cabral, Carolina Gonçalves, Miguel Serôdio. Analysis and interpretation of data: Gonçalo Cabral, Carolina Gonçalves, Miguel Serôdio. Drafting of the manuscript: Gonçalo Cabral, Carolina Gonçalves, Miguel Serôdio. Revising it for intellectual content: Gonçalo Cabral, Rita Pelejão. Final approval of the completed manuscript: Gonçalo Cabral, Carolina Gonçalves, Miguel Serôdio, Rita Pelejão.

Responsabilidades Éticas

Conflitos de Interesse: Os autores declaram a inexistência de conflitos de interesse na realização do presente trabalho.

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Proteção de Pessoas e Animais: Os autores declaram que os procedimentos seguidos estavam de acordo com os regulamentos estabelecidos pela Comissão de Ética responsável e de acordo com a Declaração de Helsínquia revista em 2024 e da Associação Médica Mundial.

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