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PATIENTS SATISFACTION WITH MIGRAINE MANAGEMENT BY FAMILY PHYSICIANS IN SAUDI ARABIA JEDDAH: A CROSS-SECTIONAL STUDY

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The most common neurological symptom is a headache, which may be a sign of something dangerous like a brain tumor or something relatively harmless like a migraine or tension headache. Although migraine is more common and more debilitating, most of population suffer from it. Since most of population are continuously under pressure, it was important to assess the prevalence of migraine headaches among this population. Lack of research concentrating on migraine headache among population encouraged this study.

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INTRODUCTION

Problem statement

A dark room, soft bed, and cold towel are all things that may help alleviate the symptoms of migraine, a form of headache illness that causes recurrent episodes. Some of the most typical triggers of an anxiety attack include: stress; changes in sleep, weather, or habit; mild dehydration; specific meals; coffee; smoking; rapid exposure to light or strong scents; and prescription use [1]. A migraine is a pounding headache on one side of the brain that may also cause nausea, vomiting, sensitivity to light and sound, and a loss of appetite [2]. People under the age of 35 are disproportionately affected, and the illness tends to deteriorate with time [3]. According to the Global Burden of Disease 2000 (GBD), migraine is the third most prevalent cause of disability-adjusted life years lost. The World Health Organization ranked migraine as the sixth largest cause of migraine in 2013 [4]. Migraine headaches have been theorized to be brought on by temporary alterations brought on by the release of chemical compounds in the brain, which irritate the blood vessels and lead to swelling, inflammation, and the activation of pain signals [5].

Background

Migraine pain affects over 28 million people [6]. While only about a third of people with migraines ever see a doctor [7-8], that proportion has increased dramatically in recent years, with the number of migraine-related doctor visits rising from 2.3 million in 1990 to over 5 million in 1998 and still rising [9]. According to recent research, over 65% of people with migraine have seen a doctor at least once, and 40% of people with migraine have seen a doctor five or more times. More than 72% of all medical visits are to primary care doctors, most of them families [10].

Studies show that fewer than 30% of those who suffer from migraines are very happy with the therapy they are receiving [11-12]. Only 21% of migraine patients in a survey of 15 primary care clinics reported being pleased or very satisfied with the care they were receiving [13]. Patients enrolled in clinical trials8 or referred to specialized headache clinics make up the majority of the patient population in studies claiming poor patient satisfaction with primary care therapy of migraine [14]. It is possible that the percentage of patients who were unsatisfied with their primary care provider's migraine management might be higher across these categories. Since there was a dearth of information in the literature about the experiences of unselected primary care patients who had sought medical attention for migraines, we decided to ask about patients' satisfaction with their care in a survey of those who had seen their family doctor about the condition. Our primary objective is to compare the experiences of individuals who were happy with their migraine treatment to those who were not.

Aim and Objectives

Aim

To assess patients' satisfaction with migraine management by family physicians.

Specific objectives

1- To evaluate the level of satisfaction and quality of life with migraine management led by family physician.
2- To determine the statistical relationship between participants' characteristics and migraine management by family physicians.

Literature Review

According to the World Health Organization, migraine is the most debilitating neurological illness and the sixth most disabling ailment in the world [15-16]. Regrettably, it's standing is rising with time [17-18]. If both episodic and chronic migraine are included, it has a worldwide frequency of 15-18% [19] and imposes a significant economic burden on countries like the United States ($19.6) and the European Union (€27 billion) per year. Despite a recent research highlighted approximately £6 billion in service consumption and missed employment due to migraine in the United Kingdom alone, the real socioeconomic cost of migraine is likely far greater at present [20]. The prevalence of migraine is higher in women than in men, and it has a major negative impact on quality of life [21], especially during the most productive years of life. Attacks of severe, unilateral head pain coupled with heightened sensitivity to touch, sound, and light define this condition [21]. The premonitory period, which may last up to 48 hours, includes symptoms including fatigue, irritation, diminished focus, and yawning. The postdrome is the period of time after an assault in which the victim continues to feel ill, often fatigued. About a third of migraine sufferers also have neurological impairments during attacks; these deficiencies, which may manifest as cortical disturbances, are referred to as migraine aura [22-23]. Migraine is a diverse brain illness that may have varying symptoms and linger for days at a time. The four stages of a migraine are the prologue, aura, headache, and postdrome. Even though migraines have distinct phases, those stages sometimes overlap, making the linear ordering seem both appealing and deceiving. Although we shall attempt a stage-by-stage dissection of the pathophysiology of the symptomatology, it is crucial to note that certain

symptoms, such as fatigue or difficulties focusing, may be present at all stages. Only the presence or absence of head discomfort may be used to reliably identify a person [24].

Migraine auras are temporary neurological impairments experienced by around one-third of migraineurs [25]. According to the Third International Classification of Headache Disorders (ICHD-3), a migraine with aura consists of one or more transient, fully reversible neurological deficits, at least one of which must have a unilateral localization, that develop over 5 minutes or more and each deficit lasts between 5 and 60 minutes. A comprehensive prospective diary research found that 26% of patients have at least one of three auras that lasts for more than an hour [26]. Given that it would be a huge waste of resources to conduct an analysis of aura lasting more than an hour, this highlights the polythetic issue. Perhaps the cutoff of 5%, or 4 hours [27], is appropriate since many auras are longer than that. Over 90% of cases include some kind of visual aura, which may manifest as either a positive (fortification spectra) or negative (scotoma) phenomenon, but other sensory, motor, speech, brain stem, and retinal aura symptoms are also possible. In some cases, aura symptoms begin during the headache phase and continue through all three stages. Recent research have shown that the overlap of the aura and headache phases is typical, as opposed to being the exception to the rule, disproving the conventional wisdom that these phases occur in sequential sequence [28].

Motor aura symptoms, such as those seen in hemiplegic migraine, tend to be more persistent, perhaps persisting for as long as 72 hours in some patients. The impairments are often present with the migraine headache in these instances of migraine aura. Positive occurrences in hemiplegic aura are very rare; if there was always an early depolarization, then one would expect jerks before the weakening set in. Since this is the exception rather than the norm, clinical phenomenology mandates an open attitude toward the underlying pathophysiology of migraines with auras [29].

Some researchers suggest that a pathophysiological brain mechanism underpinning the clinical occurrence of migraine aura is a transitory wave of neuronal depolarization of the cortex, known as the cortical spreading depression (CSD). Although the electrophysiological correlate of a CSD during a migraine aura has not been demonstrated in humans, there is a correlation between the neurophysiological characteristics of a CSD, its retinotopic propagation on the visual cortex, and the characteristics and dynamics of the visual deficits that suggests CSD as its pathophysiological correlate [30]. Imaging studies' indirect findings provide more credence to this theory [31]. Although the possibility of a direct relationship between CSD and the onset of headache is still hotly contested and addressed in further depth below, it seems unlikely that CSD is involved in the onset of the full migraine spectrum given our present knowledge of the disorder [31].

The most recent version of the ICHD-3 defines migraine as bouts of headache lasting 4-72 h that are accompanied by nausea, photophobia, phonophobia, or both. Unilateral, pulsing, moderate-to-severe, and exacerbated by physical activity are some of the key features of this kind of headache, albeit two of them are sufficient to meet the diagnostic criteria alone. Chronic migraine, defined as occurring on 15 or more days per month, is now more clearly distinguished from episodic migraine in ICHD-3 than in prior editions, expanding on the appendix definition [32]. As it is, it's not apparent whether the difference is useful, or if the 15-day cutoff is even reasonable. So far, the differentiation hasn't provided any surprising physiological discoveries.

The ICHD-3beta lexicon does not even include a definition for the postdrome phase, despite its importance [33]. The few studies that have examined the last stages of a migraine attack have shown that its symptoms are similar to those seen in the warning stage [34-35]. Postdrome effects include fatigue, impaired concentration, and neck stiffness, according to a prospective, systematic electronic diary research. It is not known whether these symptoms begin in the premonitory phase, carry over into the headache phase, and terminate in the postdrome phase, or if they may begin at any time throughout the headache phase or even after the headache phase has concluded. Some migraine sufferers attribute the conclusion of their headache to the medicine that finally eliminated their postdrome symptoms, suggesting that these symptoms only manifest or return after the headache phase has gone. Postdrome symptoms are noticed most often in the placebo group after pain relief, according to a meta-analysis of a clinical study program [36].

Migraine's stages, including the inter-attack period, outlined above provide a therapeutically relevant framework for evaluating findings from brain imaging studies. Since it is generally accepted that migraine is a hereditary disorder, and since electrophysiological investigations [37] provide enough evidence for this hypothesis, we will briefly review these research as context before moving on to the imaging results. All of these approaches have some degree of difficulty

with the central problem of trait and state. Because there is no external confirmation that nothing has begun or that the assault has ceased, functional interictal investigations have to assume that an attack is occurring. A possible benefit of triggered trials is that they are more likely to have participants with similar migraine episode onset times. Another universal problem is how to account for false-negative history in controls, which is present in all physiological investigations of migraine and any study that uses controls. Women in North America had a 43% lifetime risk of developing episodic migraine [38]. It is difficult to be convinced that a control does not hold migraine biology when one considers the existence of probable migraine, chronic migraine, and the hotly contested overlap with tension-type headache. Finding "vanilla" controls is the hardest part of the process, according to the authors, who have found this to be true across all of their studies. All of these different methods have the same restrictions.

Important insights on migraine have been uncovered via the use of neurophysiological techniques on people who suffer from the disorder. In comparison to magnetic resonance imaging (MRI), these methods are better at temporal than spatial discriminating and provide more possibilities for repetition. What has become abundantly obvious from research spanning the visual, somatosensory, auditory, and nociceptive domains is the presence of activation that consistently deviates from controls. According to one popular interpretation of the available evidence, thalamocortical dysrhythmia plays a crucial role in the pathogenesis of migraine [39]. The intensity dependence of auditory evoked potentials, for instance, is amplified in migraine sufferers between episodes, a phenomenon that has been known for some time [37]. Surprisingly, this returns to normal in the hours and days preceding an assault [38]. Triptans, agonists of the serotonin 5-HT1B/1D receptor, may have an effect on this metric (see subsection IX) since serotonin plays a role in its production and regulation [39]. Migraineurs have an interical habituation deficit as evaluated by the nociceptive blink reflex, and potentiation of the passive "oddball" auditory event-related potential supports this idea [40]. As a result, it's been hypothesized that those who suffer from migraines have a brain that over-responds, as opposed to being hyperexcitable [40].

Research on underlying structures Migraine sufferers' brains have been shown to look different from those of healthy people in many studies. Cross-sectional examinations of structures are the norm, and their interpretation requires keeping in mind the trigeminal system's role in the processing of pain. Reduced grey matter has been found using voxel-based morphometry in regions of the brain involved in pain processing, including the anterior cingulate cortex, amygdala, insula, operculum, and frontotemporal and precentral gyri. It's intriguing that the prevalence of migraines was linked to a loss of grey matter in the anterior cingulate cortex [41]. On the other hand, compared to individuals with low attack frequency, migraineurs with high attack frequency showed increased grey matter volume in the caudate nuclei on both sides of their brains [42].

Also, when comparing patients to controls, we found thicker representations of the head and face in the somatosensory cortex [43]. Diffusion tensor imaging was utilized to assess white matter integrity by the same team. Migraineurs showed decreased fractional anisotropy in the thalamocortical tract, the ventral trigeminothalamic tract, and the ventrolateral periaqueductal gray (PAG) compared to controls [44]. Diffusion tensor imaging was used to assess brain tissue damage in a separate investigation including 16 patients and 15 healthy controls. The grey matter showed only modest alterations in diffusivity, whereas the white matter and volume of the brain were normal and were shared evenly across the groups [45]. Among a larger sample of 22 patients and 20 controls, those with migraines accompanied by an aura had a shorter T1 relaxation period in the thalamus than those with migraines without an aura or healthy controls [46]. Volume reduction in the central nuclear complex, anterior nucleus, and lateral dorsal nucleus, and decreased striatal volume were seen in patients compared with controls in a pooled analysis of 3T scans from 131 migraine sufferers collected via an international partnership [47].

Collectively, the findings show that changes in the structure of pain-processing regions including the anterior cingulate cortex and the trigeminal somatosensory system are correlated with the propensity to have migraine episodes. The approach used makes it difficult to deduce whether these alterations are incidental to the pathophysiology of migraines or the result of chronic migraine bouts. Researchers used diffusion tensor MRI to demonstrate that between episodes, those with migraine without aura had greater fractional anisotropy and lower mean diffusivity in their thalami than controls did. Migraine sufferers often experience this shift while having an episode [47].

Importantly, alterations in the right thalamus correlated with time elapsed since the previous

incident [48]. Based on these findings, researchers used voxel-based morphometry with T1-heighted 3T MRI to demonstrate that patients suffering from migraine without aura have decreased grey matter density in the right inferior parietal lobule, right temporal inferior gyrus, right superior temporal gyrus, and left temporal pole during interictal periods, but normal densities during ictal phases [49]. Together, the findings point to plastic alterations associated with migraine attacks as a possible mechanism for the development of the illness.

Analyses of function Brain imaging may be supplemented by functional investigations. Both the resting state (the genuine interictal state) and the reactive state (the onset of an attack) of the migrainous brain are the focus of many studies [50].

Assessing regional brain metabolism with 18F-FDG PET is the gold standard for comparing resting functional differences across groups. This method detects functional differences in migraineurs without relying on predetermined brain regions, as in seed-based "resting state" analysis, or on particular external stimuli, as in BOLD-fMRI. Therefore, 18F-FDG PET investigations are considered to be very relevant to the biology of the illness, despite obvious drawbacks like as radiation exposure and limited spatial and temporal resolution. Brain regions involved in central pain processing, such as the insula, anterior and posterior cingulate cortex, premotor and frontal cortex on the left, and primary somatosensory cortex on the left, were shown to be hypometabolized in migraine sufferers. This may indicate that the brain is predisposed to developing migraine episodes during the interictal stage, indicating a malfunction in central pain processing. Interestingly, no hotspots of increased metabolism were found [51].

The blood flow is altered because of the stimulation. Migraine is characterized by a variety of symptoms other than headache, and photophobia is a prominent one. They often complain that the light is too strong (photophobia) or that it hurts their eyes or heads (phototic allodynia). Migraine sufferers have a lower light tolerance, even between attacks. Similarly, seven interictal migraineurs using H215O-PET showed more visual cortex (cuneus and lingual gyrus) activation in response to varying light intensities than did a group of healthy controls. Interestingly, similar activation occurred in control patients when trigeminal pain was applied. This suggests that during migraine episodes, the trigeminal system becomes activated, which may lead to a stimulation of the retino-geniculate-cortical pathway of visual processing and/or a malfunction of visual association regions leading to photophobia [52].

METHODS:

Study design and settings

A descriptive, correlational cross-sectional design will be employed for this study. Since this study aims to assess the patients satisfaction with migraine management by family physician at a single point of measurement, this is the most appropriate design. This enables the researcher to measure the effect and the outcome at a single point of time. This study design gives reliable results with short time and less effort. The study will be conducted at primary health care centers in Jeddah, from this list: [King Faisal Specialist Hospital, King Abdulaziz Hospital, King Fahad General Hospital – Jeddah, Al Faisaliyyah Medical Center, Al Sulimania Medical Center, Taiba Al Jonoobiyah Medical Center, Al Rayan Medical Center, Al Bawadi -1 Medical Center, Life Care Medical Center, Al Hamraa Medical Center, Alfaysaleya PHC Center, University District Medical Center, Al Safa 2 Medical Center, Old Airport Medical Center, Naseem Jeddah Medical Center, Chronic Care Specialized Medical Hospital, International Medical Center Hospital, Al Tasamuh Polyclinic 2]. The participants will be selected during the period from December 2022 to March 2023

Population, Sampling and Sample size

Study participants will be selected on two steps, stratified random sampling at the level of primary health care centers, to determine the required sample size from each center, then non-probability convenient sampling technique to collect the sample size from each center.

Sample size will be calculated according to the total number of migraine patients at primary health care center with a confidence level of 95% and marginal error of 5%. To calculate the sample size, we used this statistics: Total number of population in Jeddah 2022 is 4,781,000 [53], the percentage of adults [25-54 years] is 51.86% [54], and the prevalence of migraine in Jeddah was estimated to be 37.2% [55]. Given the previous numbers, we used Epi-info software to calculate the required sample size which will be 385.

Data collection

Data will be collected using a questionnaire filled through a self-administered approach.

Instruments

Study instruments consists of two domains. First is sociodemographic characteristics of participants and

satisfaction about management by family physician. Second is migraine disability assessment test (MIDAS) [56].

Statistical analysis

Data obtained from questionnaire were entered and analyzed using SPSS program version 23 computer software. Sociodemographic data are presented using descriptive statistics as means, median, percentages and standard deviation. Independent T test and one-way Anova are used to show statistical significance among participants characteristics. Chi square test is used to show relationship between categorical variables.

Ethical consideration

An approved permission will be gained from KAU to collect quantitative data from migraine patients. After explanation of study objectives, participants will be asked to volunteer to participate at our study. In addition, verbal informed consent will be gained from participants before asking questions.

Data collection sheet (English Version)

Age		Gender	Male	Female
Number of years with migraine				
Medical treatment of migraine by family physician				
Very satisfied	Satisfied	Unsatisfied	Very unsatisfied	
Drugs used for migraine				

Analgesics	Combination drugs	Narcotics
Current triptan use	Yes	No
Discontinued triptan	Yes	No
Never used triptan	Yes	No
Experience of >1 year with triptan	Yes	No

Migraine Disability Assessment Test (MIDAS)

1	On how many days in the last 3 months did you miss work or school because of your headaches?	
2	How many days in the last 3 months was your productivity at work or school reduced by half or more because of your headaches? (Do not include days you counted in question 1 where you missed work or school.)	
3	On how many days in the last 3 months did you not do household work (such as housework, home repairs and maintenance, shopping, caring for children and relatives) because of your headaches?	
4	How many days in the last 3 months was your productivity in household work reduced by half of more because of your headaches? (Do not include days you counted in question 3 where you did not do household work.)	
5	On how many days in the last 3 months did you miss family, social or leisure activities because of your headaches?	
	Total (1-5)	
A	On how many days in the last 3 months did you have a headache? (If a headache lasted more than 1 day, count each day.)	
B	On a scale of 0 - 10, on average how painful were these headaches? (where 0=no pain at all, and 10= pain as bad as it can be.)	

Data collection sheet (Arabic Version)

العمر (بالسنوات)		الجنس	ذكر	أنثى
عدد السنوات منذ التشخيص بصداع الشقيقة				
ما هو مدى رضاك عن العلاج الدوائي لصداع الشقيقة من قبل طبيب الأسرة				
راضي بشدة	راضي	غير راضي	غير راضي بشدة	
العلاج المستخدم لعلاج صداع الشقيقة				
المسكنات	الأدوية المخدرة	خليط من العلاجات		
استخدام حالي للتربتان (نوع دواء علاج الشقيقة)		نعم	لا	
التوقف عن استخدام التربتان		نعم	لا	
عدم استخدام التربتان مطلقا		نعم	لا	
استخدام التربتان لأكثر من سنة		نعم	لا	

تقييم الإعاقة بسبب صداع الشقيقة (MIDAS)

1	في كم يوم في الأشهر الثلاثة الماضية تغيبت عن العمل أو المدرسة بسبب صداعك؟	
2	كم عدد الأيام في الأشهر الثلاثة الماضية التي انخفضت فيها إنتاجيتك في العمل أو المدرسة بمقدار النصف أو أكثر بسبب الصداع الذي تعاني منه؟ (لا تقم بتضمين الأيام التي قمت بحسابها في السؤال 1 حيث فاتتك العمل أو المدرسة.)	
3	في كم يومًا في الأشهر الثلاثة الماضية لم تقم بالأعمال المنزلية (مثل الأعمال المنزلية، وإصلاح وصيانة المنزل، والتسوق، ورعاية الأطفال والأقارب) بسبب صداعك؟	
4	كم عدد الأيام في الأشهر الثلاثة الماضية التي انخفضت فيها إنتاجيتك في العمل المنزلي بمقدار النصف أو أكثر بسبب الصداع الذي تعاني منه؟ (لا تقم بتضمين الأيام التي تحسبها في السؤال 3 حيث لم تقم بالأعمال المنزلية.)	
5	في كم يومًا في الأشهر الثلاثة الماضية فقدت الأنشطة العائلية أو الاجتماعية أو الترفيهية بسبب صداعك؟	
	المجموع لأول 5 اسئلة	
أ	ما هو عدد الأيام التي شعرت فيها بالصداع خلال الأشهر الثلاثة الماضية؟ (إذا استمر الصداع لأكثر من يوم واحد، فاحسب كل يوم.)	
ب	ما هو عدد الأيام التي شعرت فيها بالصداع خلال الأشهر الثلاثة الماضية؟ (إذا استمر الصداع لأكثر من يوم واحد، فاحسب كل يوم.) على مقياس من 0 إلى 10، في المتوسط، ما مدى ألم هذا الصداع؟ (حيث 0 = لا يوجد ألم على الإطلاق، و10 = ألم بقدر ما يمكن أن يكون.)	

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