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ЛЕКАРСТВЕННО-ИНДУЦИРОВАННАЯ ГОЛОВНАЯ БОЛЬ (ОБЗОР ЛИТЕРАТУРЫ И РЕКОМЕНДАЦИИ ДЛЯ ПРАКТИКИ)

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Medication-Overuse Headache (Review of Literature and Recommendations for Practice)

Резюме

Лекарственно-индуцированная головная боль (ЛИГБ) является распространенной вторичной цефалгией, развивающейся у пациентов с частыми и хроническими головными болями при злоупотреблении анальгетическими препаратами и приводящей к значительному ухудшению качества жизни пациентов. Терапия ЛИГБ требует высокой степени комплаентности в отношениях врач-пациент и не всегда приводит к удовлетворительному результату. Поэтому важной задачей является профилактика излишне частого применения симптоматических средств для купирования головной боли. В обзоре представлены современные данные о ЛИГБ, ее лечении и профилактике.

Ключевые слова: *лекарственно-индуцированная головная боль; анальгетические препараты; лечение; профилактика*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Medication-overuse headache (MOH) is a highly prevalent secondary headache, developing in patients with frequent and chronic cephalalgias due to excessive use of "acute" medications for headache, and significantly affecting patients' quality of life. Treatment of MOH demands high compliance physician-patient, and the result is satisfactory not in all cases. For this reason, the prophylaxis of overuse of symptomatic medications for headaches is important. The review presents contemporary data on MOH, its treatment and prophylaxis.

Key words: *medication-overuse headache, analgesic medications, treatment, prophylaxis*

Conflict of interests

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fMRI — functional MRI, HA — headache, DIHA — drug-induced headache

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Introduction

The accessibility of medicinal products for symptomatic treatment of pain caused the spread of their uncontrolled use by patients with variously localised pains and aches. Considering the high incidence of headaches in the population (all types of headache: 47 %, migraines: up to 11–20 %, tension headache: approx. 42 % [1, 2]), their significant effects for patient's daily life as well as emotional and individual traits of persons with primary cephalalgia, the problem of drug-induced headache (DIHA) is gaining in significance. Frequent uncontrolled use of symptomatic agents for headache (HA) management causes more frequent cephalalgia episodes, reduced efficacy of symptomatic painkillers, and transformation of ocassional headache into chronic headache.

In the third edition of the International Classification of Headache Disorders [3], drug-induced headaches are described in Section 8.2.

Diagnostic criteria for DIHA [3]:

A. Chronic headache lasting for 15+ days per month in a patient with previous headaches

B. Regular abuse of one or several painkillers for symptomatic management of headaches for over three months

C. Headache cannot be explained by any other diagnosis from the International Classification.

Drug abuse means taking painkillers for 10 (triptanes, ergotamines, combined pain killers) or 15 (simple painkillers and NSAIDs) days per month, depending on teh drug.

DIHAs are classified according to abuse of certain painkillers and can develop in case of frequent use of any of them — ergotamines, triptanes, non-opioid analgesics, paracetamol (acetaminophen), nonsteroidal anti-inflammatory drugs (including aspirin), combined painkillers. Sub-section 8.2.6. of the Classification is of special interest; it is dedicated to abuse of several drug classes without abuse of each of them. What is meant here is a common clinical case when a patient alternates several medications.

Considering that DIHA develops in the patient with existing headache, the patient is diagnosed with both clinical diagnosis — primary cephalalgia and DIHA.

Epidemiology

The incidence of drug-induced headaches in the population is 1–7 % [4, 5], teh condition prevails in women with a ratio of 4:1 [6]. However, the incidence of DIHA in patients with headaches, especially with chronic migraines, is significantly higher [5].

According to international study results, DIHA is a global issue affecting daily life of approx. 60 million of patients, while social and economic implications of DIHA (disability, drug therapy, medical assistance) are three times higher than in migraines [7]. Of note, according to the World Health Organisation, migraines are among the top 10 conditions affecting patient's life.

Etiology

DIHA usually develops as a result of progression of frequent occasional or chronic headaches. The main cause of DIHA is chronic migraine leading to frequent use of symptomatic painkillers, including triptanes and combined painkillers; however, DIHA can be caused by other variants of frequent and chronic cephalalgia (tension headache, headache associated with vascular diseases, etc.).

In 2020, Salhofer-Polanyi S, et al. published results of a 32-year retrospective analysis of drugs abused by 787 patients of a specialised headache center, who had DIHA. In 2004–2015, patients usually used ordinary painkillers (54.4 %) and simple painkillers (33.5 %), triptanes (31.9 %) and ergotamines (8.2 %) were less common. At the same time, 53 % of patients were diagnosed with abuse of two or more drug classes [8]. The risk of DIHA from a new class of antimigraine agents — ditans (not registered in Russia) has also been studied [9].

It is worth mentioning that the structure of this list of drugs has gradually changed: ergotamines are now used less frequently; there are more and more patients with abuse of triptanes (also in Russia, resulting from teh availability of cheaper generics) or of several classes of painkillers.

NSAIDs and paracetamol are associated with a lower risk of DIHA, whereas triptanes easily cause DIHA. However, headaches caused by abuse of triptanes react to therapy better and have a shorter withdrawal headache period [8, 10, 11].

DIHA pathogenesis is still not clear. It is assumed that a certain role is played by genetics, in particular by polymorphism of dopaminergic system genes (DRD4, DRD2, COMT, SLC6A3) and genes associated with addictive state mechanisms [12]. However, the number of study subjects and the quality of evidence did not allow making clear conclusion from the metaanalysis [12].

It is believed that frequent and excessive use of medications is facilitated by emotional and personal factors, e.g. [13]:

1. Desire to reduce pain and have normal daily activities
2. Fear/anxiety because of pain
3. Presence of withdrawal headache

4. Comorbid depression and anxiety
5. Presence/susceptibility to addictive states.

As a result, patients start taking painkillers not only at onset of headache, but also in advance, believing it to be for prevention purposes.

DIHA pathophysiology is not clear. Probably, painkillers provide a basis for DIHA by interacting with neurotransmitter systems [5]. It is assumed that excessive use of painkillers for headaches causes dysregulation of descending nociception inhibiting control [14,15], impaired functional links in the brain with formation of abnormal neuronal networks and changes in nociception information processing [16], lower pain threshold, and increased expression of 5HT2a serotonin receptors in the cortex and trigeminal ganglion [11].

A major part in development of chronic pain syndromes is played by central sensitisation, i.e., a long-term (however reversible) increase in neuron irritation in the central nociceptive structures as a result of repeated nociceptive stimulation. The mechanisms of this phenomenon include imbalance between excitatory and inhibitory neurotransmitters, neuronal and synaptic plasticity, and impaired glia–neuron connections [15]. Central sensitisation syndromes can also include chronic cephalalgia (chronic migraine, chronic tension headache, and DIHA) [17]. A clinical marker of central sensitisation is dermal allodynia (dermal pain from non-pain stimuli), observed in 50–80 % of patients with migraine and it is a predictor of condition perpetuation [11].

Since not all patients with abuse of painkillers in accordance with the International Classification develop DIHA, a primary task for further studies is development of a marker (either a biochemical or neuroimaging), that could help in identification of patients with true or potential DIHA [18]. Morphometric studies demonstrated that patients with DIHA (vs. healthy subjects) have a larger amount of cerebrum in ventral striatum (an area participating in behavioural activities of reward and addiction) [19] and a smaller amount in frontoorbital area (an area of the mesocortical-limbic system also involved in addictive behaviour) [20]. Functional MRI (fMRI) showed higher neuronal activity in thalamus [21].In magnetic resonance spectroscopy, Niddam et al. observed several markers (N-acetyl aspartate, myo inositol) in patients with DIHA and concluded that they could contribute to disease development [22].

Unfortunately, currently neither structural nor biochemical markers have clinical application.

Factors associated with DIHA development

It is still not clear why DIHA develops in some patients with primary cephalalgia and does not affect others. Viana M, et al. [23] reported results of an examination of 318 patients with long-lasting (over 10 years) migraines. DIHA was observed in a half of them (162 patients); mean migraine duration was similar both with and without DIHA. Factors associated with DIHA development were reported both as highly expected (low physical activity, history of depression, insomnia, brain injury) and unexpected, e.g., marital status (DIHA was more frequent in married and divorced persons than in single subjects) and younger age at migraine onset. In this study, we have not found any correlation between DIHA and patient sex, body weight index, coffee and alcohol consumption or smoking [23]. Also, another risk factor is lower level of education and chronic gastrointestinal conditions.

The most significant risk factor for DIHA is the frequency of primary headache episodes [24]. Therefore, there is still a point of issue: Is the frequent use of painkillers a cause or a consequence of headache perpetuation [18, 23]? However, it has been proven that an efficient therapy for DIHA is withdrawal of contributing painkillers; therefore, their role in condition pathogenesis has been verified.

At the same time, discussions of the role of depression and anxiety in development of chronic cephalalgia, particularly of DIHA [13, 25], have been observed in literature. According to COMOESTAS study results, high levels of anxiety were found in 57.7 % and depression — in 40 % out of 492 patients with DIHA [26]; however, no association was observed between anxiety/depression and number of days of headache duration. According to Park H-K et al. [27], depression was diagnosed in 83 % out of 229 patients with DIHA, anxiety — in 62 %.

DIHA is a biobehavioural disorder facilitated by personal traits and behavioural patterns of patients [28, 29]. It was noted that up to 50 % of patients with DIHA demonstrate addictive behavior, reduced or lost control of the use of painkillers [5], i.e., they take painkillers as a ritual [11].

Therapeutic strategy in DIHA

For DIHA management, it is essential to create trust relationship between the doctor and the patient and to clearly explain to the patient the mechanism of abnormal association between drug intake and increased frequency of headaches.

During an information talk, it is recommended to tell the patient about the following aspects [13]:

1. The role of excessive use of symptomatic painkillers in higher frequency and severity of pain and in potential reduction in the efficacy of other therapies.

2. Possible DIHA development even if painkillers are taken 2–3 times a week
3. Discussion of withdrawal headache, i.e., temporary deterioration of patient's condition when contributing medications are discontinued
4. Information on the maximum frequency of symptomatic painkillers and inadmissibility of the use in anticipation of headache.

The objective of DIHA treatment is reduction of the frequency of headaches, reduction in the use of painkillers, and improved therapeutic response to preventive and symptomatic therapy. Treatment includes discontinuation (single-step or gradual) of contributing drugs, a course of preventive therapy, and arrest of withdrawal symptoms.

Drugs can be discontinued in a single step or their frequency can be gradually reduced. Both strategies have advantages and disadvantages. A single-step withdrawal is more efficient and usually causes marked withdrawal symptoms resulting in reduced compliance. Gradual withdrawal is easier for a patient, however, it bears a risk of voluntary increase in the frequency of drugs and return to the previous abuse pattern. Nielsen M, et al. [30] evaluated results of drug discontinuation (complete withdrawal or reduced frequency, follow-up duration: 6–12 months) in 72 patients with DIHA and concluded that complete withdrawal from symptomatic painkillers is most efficient. At the same time, researchers demonstrated that the mean efficiency of the painkiller discontinuation program is still low, just approx. 30 % [31]. Therefore, the search for the ways to increase efficiency has continued.

In addition to withdrawal from contributing drugs, patients are also recommended to undergo a course of preventive therapy with proven efficiency. A list of drugs depends on the primary headache: topiramate, beta-blockers, amitriptyline in migraines; antidepressant drugs (primarily cyclic ones) in tension headaches [18,32]. According to the international multicenter analysis of DIHA register, in real life, most common are antiepilepsy drugs (25 %), beta-blockers (13 %), tricyclic antidepressants (12 %), botulinum toxin A (9 %), monoclonal anti-CGRP antibodies (7 %), calcium-channel blockers (6 %), angiotensin II receptor blockers (2 %), and selective serotonin reuptake inhibitors (1 %) [27]. Medications with the highest level of evidence in DIHA are topiramate and onabotulinumtoxin A [18], as well as monoclonal anti-CGRP antibodies (fremanezumab, frenumab), which are new target drugs for the treatment of episodic and chronic migraine, including with DIHA [33]. Of note, neurometabolic drugs commonly used in Russia are not indicated in DIHA or primary cephalalgia.

Specific time limits for preventive therapy are to be discussed. It can start on the first day following painkiller discontinuation or at a later date [18, 32]. Delayed therapy initiation may be due to the fact that later approximately a half of patients do not need it [34]. Patient management and support in their efforts to overcome medication abuse are essential.

It is also recommended to arrange for detoxication therapeutic programs [32], that include, in addition to discontinuation or limitation of painkillers, a course of preventive therapy, corticosteroids (prednisolone), and fluid resuscitation [34-36]. Such programs can be organised either in outpatient or inpatient settings. A short-term inpatient treatment is especially indicated for patients with depression and anxiety and chronic stress situations [8]. During first days after symptomatic drug discontinuation, naprosine, acetaminophen, promethazine, metoclopramide can be prescribed to relieve withdrawal symptoms. There are reports of long-term efficacy of detoxication programs when patients are followed up for 6 months [26] and 5 years [37].

As far as non-drug non-invasive methods of chronic headache management are concerned, most interesting are repetitive transcranial magnetic stimulation [38], feedback methods (computer-aided biocontrol and mobile apps for behavior therapy) [39] and transcutaneous trifacial electrical stimulation — Cefaly [40].

Prognosis

Therapeutic measures in DIHA do not always ensure long-term success. Prospective studies demonstrate that 20–30–50 % of patients continue abusing medications [5, 23, 41]; there is also a 25–45 % probability of DIHA recurrence in years after successful discontinuation of contributing painkillers [18,42]. Usually recurrences develop within a year after withdrawal; in case of a recurrence, a majority of patients continue abusing the same medications they used before their diagnosis [43]. Another attempt to discontinue medications usually causes a negative reaction from patients. This is another proof of the need in long-term and regular follow-up of patients after therapy [44]. Moreover, it is essential to identify and correct comorbid anxiety, psychological dependence, pain catastrophizing [11].

Prognostically favourable factors are successful discontinuation of contributing drugs and absence of chronic headache by the end of the first year after withdrawal [41, 43].

DIHA prevention consists of identification of a drug dependence risk group and face-to-face work with the patient including educational measures [5]. It is believed that a majority of DIHA cases can be avoided with the help of timely preventive measures. Patients should be advised to minimise symptomatic medications for

headache. The national DIHA prevention program initiated in Denmark includes not only face-to-face work with patients, but also development of awareness on admissible use of painkillers through mass media, online resources, pharmacies, and primary healthcare professionals [7]. In Russia, the TV slogan "headache cannot be tolerated" should be declared incorrect, and patients should receive appropriate information. Another important prevention method is timely prescription of a course of headache prevention with proven efficacy. Studies show that, before going to a specialised center, just 21–38 % of patients undergo preventive therapy for cephalalgia [8,27]; lack of preventive therapy eventually results in chronic headaches.

Conclusion and practical recommendations

Drug-induced headache is a common secondary cephalalgia leading to a poorer quality of patients' life. DIHA therapy requires good compliance and not always gives satisfactory results. Therefore, it is essential to prevent excessively frequent use of symptomatic agents to manage headache. The most important part of this work is making the community aware of unfavourable consequences of frequent use of painkillers. Patients should be aware that, in accordance with international standards, the highest frequency of symptomatic medications for headaches is 2 days per week and that not all types of headaches are indications for painkillers.

The objective of this publication is to inform GPs of drug-induced headaches.

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