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An Overview of Migraine Prophylaxis

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Editorial

Migraine is a common main headache, with a global frequency of 15% in the general population after one year. Migraine pathophysiology is complicated and poorly understood. The pathogenesis of migraine with aura has been linked to cortical spreading depolarization (CSD) and aberrant brain stem activity. The nociceptive sensory fibres transmitting signals from intracranial and extra cranial blood vessels, as well as other cranial structures such as the dura mater, skin, muscles, and periosteum, are thought to be the source of pain in migraine. During migraine attacks, peripheral and cerebral sensitization of trigeminovascular nociceptive pathways may develop. In the premonitory and headache phases of a migraine episode, cognitive symptoms are common, and they may last into the postdrome. Outside of migraine attacks, some migraineurs experience cognitive problems. Treatments for acute attacks are not usually successful in alleviating cognitive symptoms. Migraine attack-related disability is also exacerbated by cognitive dysfunction, particularly executive function impairment. Indeed, cognitive symptoms came in second to pain in terms of intensity and attack-related disability, making them an important target in migraine treatment [1-3].

Neurobiology of migraine

Pain sensitivity is mostly restricted to the meningeal blood vessels within the skull, which are extensively innervated by nociceptive sensory afferent fibres of the trigeminal nerve's ophthalmic division. The stimulation of these afferents is thought to play a role in the development of migraine headaches. The impulses are subsequently transported rostrally to thalamic nuclei and the ventrolateral area of the caudal periaqueductal grey region, which are implicated in pain perception (PAG). The PAG is involved in craniovascular pain not only via ascending projections to the thalamus, but also via descending regulation (mostly inhibitory) of nociceptive afferent information. When the trigeminovascular afferents are activated, vasoactive neuropeptides such as calcitonin gene–related peptide (CGRP) and substance P are released from their peripheral nerve ends. The mechanism that causes the trigeminovascular system to activate is an important concern in migraine neurobiology. The activation of nociceptive afferent fibres of trigeminal ganglion (TG) neurons innervating the blood vessels in the meninges, as well as second-order dorsal horn neurons in the trigeminal nucleus caudalis (TNC) and neurons in structures involved in the processing and perception of pain, such as the thalamus, the caudal periaqueductal grey region (PAG), and the cortex, leads (blue pathway). The PAG is involved in craniovascular pain through both ascending and descending regulation (mostly inhibitory) of nociceptive afferent information via projections to serotonergic neurons in the magnus raphae nucleus (MRN) [2,3].

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Efficacy of gabapentin

Gabapentin contains both antinociceptive (analgesic) and anticonvulsant properties. Gabapentin has been used to treat neuropathic pain problems such as diabetic neuropathy, post herpetic neuralgia, and trigeminal neuralgia in recent years. Gabapentin has been demonstrated to interact with the $\alpha 2\sigma$ subunit of Ca^{2+} channels (modulating calcium ion channel current) and to raise the concentration and possibly the rate of g-aminobutyric acid (GABA) synthesis in the brain, despite the fact that its mechanism of action is unknown. For the placebo and gabapentin treatment groups, the migraine headache rate during the second 4 weeks of the SP2 is given for patients keeping a steady dose of 2400 mg/day gabapentin. The major efficacy measure showed a statistically significant difference between treatment groups at the end of the SP2. Gabapentin was found to be beneficial as a preventive drug in reducing the frequency of headaches in migraine sufferers in this controlled clinical experiment [3,4].

Topiramate as a migraine preventative

Topiramate was first licenced for the treatment of people with partial-onset seizures. Topiramate is now licenced as an epilepsy monotherapy. Topiramate's activities on excitatory neurotransmitter receptors and voltage-gated ion channels suggest that this medicine could be useful in reducing the frequency of migraine attacks. Topiramate's clinical experience, as stated above, indicates that, if effective, the medicine could offer some advantages over current migraine prophylaxis drugs. The primary effectiveness outcome, change in average monthly migraine frequency (based on migraine periods), was studied with a linear model that included baseline value as a covariate and analysis centre and therapy as variables. Topiramate is a new migraine preventative treatment option. The topiramate 100 mg/d and propranolol groups had similar changes in average monthly migraine frequency and other secondary efficacy characteristics from baseline to core double-blind phase, as measured by confidence intervals. Both medications, however, have different adverse event and contraindication profiles, and either drug could be taken if the other is not tolerated or contraindicated.

Atypical antipsychotic drugs

Olanzapine is a new atypical antipsychotic medication that is a thienobenzodiazepine. It's an antagonist for the 5-HT2A/2B/2C, D1-4 DA, M1-5 muscarinic, and a2-adrenergic receptors. It is less likely than traditional antipsychotics like haloperidol to cause tardive dyskinesia and acute extrapyramidal responses (particularly dystonias). Patients with refractory headache, particularly those who have failed to react to conventional preventive medications, may benefit from olanzapine, according to a retrospective research. In patients with mania, bipolar illness, or psychotic depression as concomitant disorders of migraine, olanzapine should be evaluated. Its ability to prevent migraines could be attributed to its selective activity on the 5-HT2A/2B/2C and D1-4 DA receptors. In fact, serotonergic dysfunction and DA hypersensitivity are hallmarks of migraine [4,5].

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Conflict of Interest

The authors reported no potential conflict of interest.

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