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Solving the enigma of migraine

Episodic to Chronic Migraine: The Basis for the Transformation

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CM is currently defined as headache on ≥15 days/month for ≥3 months with ≥8 days/month fulfilling criteria for migraine and/or respond to a triptan or ergot. The estimated prevalence of CM worldwide is 1% to 3%, and every year, between 2.5 and 4.6% of people with episodic migraine experience progression to chronic migraine. Long-term follow up over 3 years indicates that only 26% experience remission of chronic migraine (defined as ＜15 headache days per month). Compared with EM, CM is associated with greater headache-related disability, reduced health-related quality of life, higher direct and indirect healthcare costs, and higher rates of medical and psychiatric comorbidities. The prevalence, disability, progression, and treatment needs associated with CM has created an urgent need for epidemiologic, clinical, and basic research to help better understand the clinical course of this disorder and to facilitate development of effective therapies.

A number of risk factors for the transformation from EM to CM have been identified, including acute headache medication overuse, high frequency of headache (10-14 days per month), obesity, snoring, allodynia, depression, anxiety, caffeine, head injury, stressful life events, and low education/socioeconomic status. Predictors of remission included a lower baseline headache fre-quency (15-19 versus 25-31 headache days/month), and the absence of allodynia.

Recent research suggests that CM is associated with functional and structural brain abnormalities. Activity-independent sensitization of central trigeminothalamic pathways is considered to be a possible cause of the development of chronic migraine. Such sensitization might occur during repeated migraine attacks through impaired descending inhibition and/or enhanced descending facilitation of trigeminal nociception. Enhanced cortical processing involving sensory, visual, auditory, and affective networks that exceed that seen in patients with episodic migraine or migraine-free controls, has also been demonstrated in individuals with chronic migraine. Whether this finding is due to intrinsically increased excitability or impaired intra-cortical inhibitory mechanisms is unclear.

The overuse of acute pain medications has plays a role in some patients with chronic migraine who overuse (＞10 days per month) acute headache pain medications. Experiments in animal models have revealed persistent pronociceptive adaptations following exposure to opioids and triptans, resulting in enhanced sensitivity to stimuli that trigger migraine in humans. These findings could provide insight into the adaptive changes that occur in patients who have chronic migraine associated with medication overuse and thus further elucidate the pathophysi-ology of chronic migraine.

※Author's disclosure of potential Conflicts of Interest (COI).

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