THE FIRST PHASE OF A MIGRAINE ATTACK ORIGINATE IN THE BRAINSTEM

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Migraine is a neurologic chronic disorder characterized by recurrent and reversible attacks of pain and associated symptoms.

Migraine has a complex pathophysiology and It involves integrated brain mechanisms among a number of central nervous system structures: cortex, brainstem, trigeminal system, meninges. It is generally recognized that migraine arises from a primary brain dysfunction that leads to activation and sensitization of the trigeminal system.

The brain of migraineurs is dysmodulated as a result of dysfunctional sensory processing within the brainstem. The dysmodulation would lead to enhanced sensory sensitivity, which could account for the symptoms of migraine. Dysfunction within the brainstem during migraine attack is supported by several reasons that we will discuss during the presentation.

The genetic factors can cause dysfunction of the brainsten structures. Some of the premonitory symptoms, part of the migraine attack, are originated in the structures of the brainstem and they happen also in migraine with aura.

Experimental evidence suggests that an episodic dysfunction in brainstem nuclei involved in the central control of nociception plays a significant role in migraine pathophysiology. Studies demonstrating altered excitation in the brainstem have led to the suggestion that abnormal brainstem activity may trigger migraine attacks (brainstem generator); alternatively, by contributing to the hyperexcitability in trigeminal pathways, these brainstem dysfunctions may lower the threshold for migraine attacks.

Dysfunctions of brainstem areas that are involved in the central control of pain, such as the periaqueductal gray (PAG), might release sensory inhibition in the brainstem, thus favoring central trigeminal hyperexcitability and contributing to the pronociceptive state in migraine.

Decreased descending inhibition from the PAG may favor initiation of a migraine attack and and alteration, an incresase of iron content , in the PAG has been showed in patients with chronic migraine

Other brainstem structures that may be involved in trigeminovascular activation and pain processing include are some nuclei located in this area as the magnus raphe nucleus and the Locus coeruleus.

We will show multiple studies with different imaging techniques (poitron emission tomography and functional MRI) showing consistently areas of brainstem activation during migraine, in areas hypothesized (dorsolateral pons, PAG, red nucleus..) to control the flow of nociceptive signals from the periphery.

There are also evidence, in the neurophysiologic studies (eg: nociception specific blink reflex), that the brainstem pathways are activated in the premonitory phase of migraine attacks

Migraine can start in close temporal relation with vascular or demyelinating lesions in the brainstem.

In summary, the role of brainstem dysfunctions is crucial in favoring central trigeminal hyperexcitability and in the start of the migraine attack.