Sensorial Dysfunctions in Migraine: Translational Aspect

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Migraine is the most prevalent brain disorder and the first cause of disability under age of 50. Accompanying sensorial symptoms suggesting hyperresponsivity in the somatosensory, visual, auditory, and olfactory systems are distinguishing features of migraine attacks and multisensory stimuli may worsen the headache severity and disability. Our understanding regarding the neurobiology of multisensory disturbances in migraine is inadequate.

Ability of discriminating the exact entry of consecutive somatosensory stimuli is crucial and impaired somatosensory temporal discrimination for the suggested central sensory processing dysfunction during migraine without aura attacks. Recent study using short afferent inhibition (SAI) paradigm and transcranial magnetic stimulation revealed sensorimotor integration problem within migraine attack. SAI results demonstrated an impaired sensorimotor integrity and reduced cortico-cortical inhibition between somatosensory and motor cortices in migraine without aura attacks. Also, visual temporal discrimination thresholds are prolonged both ictally and interictally in patients suffering from migraine without aura attacks. Integration of somatosensory and visual stimuli was also shown in migraine patients.

Manifestation of sensory symptoms related to distant cortical areas such as visual, and sensorimotor cortices may suggest a dysfunction of cortico-cortical connections or an interconnection through a thalamic hub. Sensorial disruptions in more than one domain accompanying migraine headache can be attributed to multisensory integration dysfunction of the higher order thalamocortical network. Involvement of TRN would contribute sensory hypersensitivity in multiple sensory modalities, lateral inhibition and sensory discrimination problems associated with migraine headache. Also, Sensory augmentation can be induced by stimulating CGRP expressing neurons in the thalamus or cerebellum. In conclusion sensory symptoms are conventionally justified by dysfunctions confined to the cerebral cortex, but a perspective through the complex interplay of thalamocortical network, involvement of cerebellum and CGRP would provide a better picture, more pertinent to the central sensory processing associated with a migraine attack.

Key words: Migraine, sensory augmentation, somatosensory temporal discrimination, Thalamic reticular nucleus, higher order sensory nuclei.