Thalamic pain syndrome

Thalamic pain syndrome is associated with damage to the ventral posterior nucleus of the thalamus.

This syndrome can be caused by stroke, multiple sclerosis, tumors, epilepsy, brain or spinal cord trauma, or Parkinson's disease. The character of the pain associated with this syndrome differs widely among individuals partly because of the variety of potential causes. Central pain syndrome may affect a large portion of the body or may be more restricted to specific areas, such as hands or feet. The extent of pain is usually related to the cause of the CNS injury or damage. Pain is typically constant, may be moderate to severe in intensity, and is often made worse by touch, movement, emotions, and temperature changes, usually cold temperatures. Individuals experience one or more types of pain sensations, the most prominent being burning. Mingled with the burning may be sensations of “pins and needles;” pressing, lacerating, or aching pain; and brief, intolerable bursts of sharp pain similar to the pain caused by a dental probe on an exposed nerve. Individuals may have numbness in the areas affected by the pain. The burning and loss of touch sensations are usually most severe on the distant parts of the body, such as the feet or hands. Central pain syndrome often begins shortly after the causative injury or damage, but may be delayed by months or even years, especially if it is related to post-stroke pain.

Based on experimental and clinical evidence of central pain produced by hyperactivity of deafferented neurones and associated with irritative foci at thalamic or cortical levels, stereotactic low-dose (10 Gy) radiosurgery was performed in 3 patients with central pain syndromes. SEEG findings and results of stereotactic radiosurgery on painful conditions were presented and mechanisms of action discussed by Barcia-Salorio et al [1)].

Dejerine Roussy syndrome or thalamic pain syndrome is a condition developed after a thalamic stroke, a stroke causing damage to the thalamus.

Ischemic strokes and hemorrhagic strokes can cause lesioning in the thalamus. The lesions, usually present in one hemisphere of the brain, most often cause an initial lack of sensation and tingling in the opposite side of the body. Weeks to months later, numbness can develop into severe and chronic pain that is not proportional to an environmental stimulus, called dysaesthesia or allodynia.

As initial stroke symptoms, numbness and tingling, dissipate, an imbalance in sensation causes these later syndromes, characterizing Dejerine–Roussy syndrome. Although some treatments exist, they are often expensive, chemically based, invasive, and only treat patients for some time before they need more treatment, called “refractory treatment.”

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Research into its underlying mechanisms and treatment options could benefit from a valid animal model. Nine different animal models have been published, but there are relatively few reports on successful reproductions of these models and so far only little advances in the understanding or the management have been made relying on these models. In general, the construct validity (similarity in underlying mechanisms) of these animal models is relatively high, although this cannot be evaluated into depth because of lack of understanding the mechanisms through which thalamic stroke can lead

to thalamic pain syndrome. The face validity (symptom similarity) is relatively low, mainly because pain in these models is tested almost exclusively through evoked mechanical/thermal hypersensitivity assessed by reflexive measures and given the conflicting results with similar tests in patients with thalamic pain syndrome. The predictive validity (similarity in treatment efficacy) has not been evaluated in most models and incorporates difficulties that are specific to thalamic pain syndrome.

De Vloo et al., compare the different models regarding these types of validity and discuss the robustness, reproducibility, and problems regarding the design and reporting of the articles establishing these models. They conclude with various proposals on how to improve the validity and reproducibility of thalamic pain syndrome animal models. Until further improvements are achieved, prudence is called for in interpreting results obtained through these models [2].

1)
Barcia-Salorio JL, Roldan P, Lopez-Gomez L. Radiosurgery of central pain. Acta Neurochir Suppl (Wien). 1987;39:159-62. PubMed PMID: 3314376.
2)
De Vloo P, Morlion B, van Loon J, Nuttin B. Animal models for central poststroke pain: a critical comprehensive review. Pain. 2017 Jan;158(1):17-29. PubMed PMID: 27992392.

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