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DEJERINE-ROUSSY SYNDROME: A COMPLEX NEUROLOGICAL SYNDROME

Neurology

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ABSTRACT

Dejerine-Roussy Syndrome also known as thalamic pain syndrome is the main symptom reported by the patients post stroke.However with lack of diagnosis and ineffectective management may lead to development of severe pain affecting the patient's quality of life. This article reviews theories regarding the post stroke pain syndrome as well as pain management. Further treatment modality studies regarding DRS requires further research and evidence.

KEYWORDS

central post stroke pain, thalamic pain, neuroplastic alternation, painful paresthesia, management of thalamic pain syndrome.

INTRODUCTION

DRS, often referred to as central post-stroke pain, is an uncommon central neuropathic pain that follows a ventroposterolateral thalamic infarction.

French neurologist Gustave Roussay (1874–1948) and Joseph Jules Dejerine (1849–1917) were the first to discover it, and they wrote about it in a 1906 paper titled "Le syndrome thalamique."(1)

The thalamus serves as a relay hub for the somatosensory pathway, but any injury that interferes with the spinothalamic tract—which includes the medulla, lower brain stem, capsular, and subcortical—can exacerbate DRS symptoms.

Etiology

- Due to the damage to the thalamus;
- Due to stroke which affects the ventroposterolateral thalamus which is responsible for the processing sensory information.
- Ischemic stroke i.e (blockage of blood flow) the thalamogeniculate artery, a branch of posterior cerebral artery, is often implicated.
- An intracerebral or subarachnoid hemorrhagic stroke, or bleeding in the brain, can harm the thalamus.(2)
- The stroke in the ventral posterior nucleus of the thalamus which includes the nerve fibres to the face,arm,trunk and legs- precedes the pain in the legs.
- Less commonly tumors,trauma,neurologic disorders cause thalamic pain which in turn leads to DRS.
- Most people who recently had a stroke often express this type of pain.

Epidemiology

After having a stroke, some people may start feeling pain. This can happen anywhere from a few days to several years after the stroke, but generally tends to start within the first six months. In some studies it has shown that patient's might even take up to ten years for the post stroke pain to develop. However, the prevalence of this pain following a stroke varies greatly: historical research have shown that between 11% and 85% of stroke patients have experienced changes in their perception of pain and sensations, while the prevalence of distinctive central post-stroke pain varies between 8% and 46%.(3)

A specific type of stroke, called the lateral medullary syndrome (or Wallenberg syndrome), sees a higher percentage of sufferers of central post-stroke pain, with a quarter of such cases occurring within six months.In actuality, this type of discomfort usually appears 4 weeks following the stroke, however it occasionally takes up to 24 weeks

It's important to note that measuring the exact number of people suffering from central post-stroke pain can be difficult. Other types of chronic pain, like shoulder pain, shoulder muscle stiffness, tension-type headaches, or joint pain (knees and hips), often occurring concurrently in stroke patients, can complicate the identification of the central post-stroke pain.

Ultimately, it's important to realize that core post-stroke discomfort can affect anyone.

It does not depend on factors like the patient's age, gender, or which side of their body was affected by the stroke.

Pathophysiology

The thalamus is a relay station for all sensory tracts in the brain which works to decode the sensory information and process it, which further goes to the somatosensory cortex where it is interpreted- stroke in this areas causes tactile, temperature,pressure, malfunction in the afferent pathway from the thalamus to the cortex. Nevertheless, a number of thalamic regions, including the ventrocaudal regions, the lateral posterior nuclei, the ventral posterior nuclei, the thalamic sensory tracts, the concurrently affected spinothalamic tract, and the pulvinar nucleus, have been identified as high-risk areas for the development of post-stroke thalamic pain.Many theories have emerged with this regard but all of these are poorly understood-

Another theory is The ectopic activity which is caused by a spontaneous firing of neurons in the processing of the somatosensory information- the patient feels pain as a result of imbalance in the sensory neural input and disrupted synaptic connection.

Another theory is of Neuroplastic alternation- it is activation of NMDA receptors by excitatory amino acids which causes neuroplastic alternation and hypersensitive states that happen during and after nerve damage to the somatosensory system. These disruptions can lead to a variety of painful sensations like allodynia and hyperalgesia.

The activation of NLRP3 inflammasome due to inflammatory responses following the event of stroke can cause decrease of descending fibres from the cortex to thalamus which induces the reduction of GABAergic release leads to increased excitability of ventral basal neurons in the thalamus, as on the other side NLRP3 inflammasome at the thalamus lesion strengthens response of microglia. Persistent inflammatory processes can induce GABAergic changes in thalamus reticular neurons to inhibit the functions of ventral basal interneurons.(4) These may also experience thalamic pain following a stroke.

Signs And Symptoms

DRS or post stroke thalamic pain often impacts areas of the body affected by stroke, and may also present with hemiplegia.

Head and facial pain within six months of a stroke, even if there is no other reason, is a good indicator of the onset of post-stroke thalamic pain.

Pain can be described as sharp,stabbing like with burning sensation(paresthesia). It could vary from mild to moderate.

Vibration and proprioception sensation is intact.

- **Pain Could Be Divided Into 3 Categories-**

1st- Constant pain with burning, aching, prickly, freezing and squeezing sensation.
2nd- Spontaneous and intermittent - the pain is sharp-shooting like,intensity fluctuates and lasts from seconds to minutes. It comes and goes throughout the day with periods of relief lasting for a few hours.
3rd- Pain is provoked by touch or pressure and leads to increased sensitivity to pain and touch which causes Hyperalgesia, hyperesthesia

or allodynia.(3)

Diagnosis
The patient's history and physical examination are used to make the diagnosis, and these include:

Full Neurological Finding- proprioceptive sensations, cranial nerves, balance and speech should also be assessed.

Sensory Examination- thermal and pinprick sensations partial or complete impairment could be seen in the patient with post stroke thalamic pain while proprioception and vibration sensation might be intact.

Imaging- CT/MRI without contrast can be used to confirm the history of stroke, it's location, visualize infarcted areas, volume of lesions if any present, and to rule out any other underlying causes.

Neurophysiological Tests-
Electroencephalogram (EEG) identifies changes in the brain's electrical activity which can be useful in assessing stroke.

Somatosensory evoked potential(SEP)- to assess brain's response to sensory stimuli and can help in predicting stroke outcomes,monitoring for ischemia. (5)

Treatment
Pharmacological Treatment
1st line-
Only amitriptyline significantly reduced pain as compared to a placebo in a study of placebo- controlled trials using oral amitriptyline and carbamazepine for post-stroke thalamic pain.

Imipramine and amitriptyline lessen pain and mechanical and thermal sensitivities.

Selective serotonin reuptake inhibitor (SSRI)- Fluvoxamaine had significant pain relief in visual analogue scale after 4 weeks of treatment and the pain reduction was only in patients who got treated within one year.

Fluoxetine, another SSRI, prevented mechanical hypersensitivity and pain, but it had no effect on pain associated with heat.

Serotonin norepinephrine reuptake inhibitor (SNRI)- Duloxetine has also been known to significantly reduce post stroke thalamic pain intensity- but the adverse effects of withdrawing this medication were nausea,agitation,and somnolence.(6)

2nd line - Anticonvulsant
Lamotrigine- most effective anti-convulsant for the treatment of post stroke thalamic pain as it decreases pain with much effectiveness and and it is safe,well tolerated by the patient.Could be withdrawn due to skin rashes.

Gabapentin- can be used in management of post stroke thalamic pain in the patients with thalamic stroke.(7)

3rd line- Opioid and opioid antagonists, mexiletine,clonidine and beta blockers can be recommended.

IV infusions of lidocaine often together with propofol/ ketamine, steroid, naloxone also can be used in few patients with acute severe pain of post stroke thalamic pain. Although these can only give temporary pain relief as it has potential side effects and cannot be used as a long term therapy.

Non-pharmacological Treatment
- Transcutaneous electrical nerve stimulation(TENS)- uses a device to deliver mild electrical pulses through the skin to relieve pain but it has mixed effects in different individuals.(8)
- Transcranial magnetic stimulation(TMS)-uses magnetic fields to stimulate nerve cells in the brain to relieve pain as it has shown modest improvement in patients following treatment of 4 weeks.
- Motor cortex stimulation-neurosurgical method in which the brain's motor cortex is stimulated with electrodes to treat refractory or chronic pain, effectiveness is shown in 1/3rd of patients with facial pain.
- Deep brain stimulation- neurosurgical method in which electrodes are implanted and cause electrical stimulation. It has shown effectiveness in 50-70% patient with post stroke thalamic pain.(9)
- Spinal cord stimulation- surgical procedure which uses low-level electrical impulses to relieve pain by interrupting pain signals between the spinal cord and the brain, effective in 50% patients following the treatment of 28 months.(10)
- In order to deactivate the source of the patient's pain, thalamotomy, a last resort, involves destroying or ablating the portion of the thalamus or region involved in relaying sensory and motor signals.
- Allodynia symptoms are alleviated with thalamotomy and mesencephalic tractotomy. The best candidates for this surgery are those who have paroxysmal shooting pain.

Prognosis
It is typically poor and is upto 5% as patients experience moderate to severe pain following cerebral infarction.There is limited management options with efficiencies mixed and variable.

After an infarction patients are much likely to present either with allodynia or dysesthesia.

Early identification of pain following an event of stroke and initial to therapy can give more favourable outcomes.

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