Dejerine Roussy Syndrome: The Role Of Methadone In The Treatment Of A Central Pain Syndrome

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Citation

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Abstract

The Dejerine Roussy syndrome can occur in 2-6% of post-stroke patients. Pain is described as "burning" or "freezing," and is often accompanied by sensory disturbances, especially those of temperature. Pathophysiology appears to involve lesions of the spinothalamic pathyways with disinhibition and excitation of NMDA receptors in the thalamus. Treatment has traditionally been with TCA's. We are reporting a case of a 57 year old female with right hemibody pain, and MRI evidence for brain ischemia, whose pain relief failed with conventional therapy, but was obtained with methadone; an opioid that displays NMDA antagonism.

CASE REPORT

The patient was a 57-year-old female who had right hemibody pain for eight years. Her pain began after a cerebral angiogram, which was performed for neurologic (motor weakness) symptoms. She experienced a constant burning pain of variable intensity, associated with allodynia and increased diaphoresis on the affected side. The pain was intensified by activity and reduced by lying down. Severity was rated as 8 over 10 on the Visual Analog Scale (VAS). Her only other complaint was right lower extremity weakness, which was confirmed on physical examination.

An MRI of the head showed asymmetry in the size of the anterior cerebral arteries, right larger than left, a tortuous basilar artery compressing the lateral brain stem, and a right hypoplastic vertebral artery. It also demonstrated brain atrophy and a few areas consistent with chronic ischemia in the periventricular while matter. She was diagnosed with Dejerine Roussy syndrome by her neurologist.

Initial treatment consisted of Celecoxib, Paroxetine, Amitriptyline, Gabapentin and Oxycodone. When she first entered our pain management clinic she was using a fentanyl patch (Duragesic, 50mcg/hr), and taking sustained-release oxycodone (Oxycontin, 20mg bid) along with above-mentioned medications. She complained of inadequate pain relief, sedation, and constipation. Her pain level was 9/10 on VAS. Her opioid was changed to methadone starting at 10mg bid; this was gradually increased to 40mg bid.

Oxycontin was tapered off and fentanyl patch decreased to 25mcg/hr. Within few weeks following the changes in medication, the patient reported a decrease in pain intensity to VAS 4/10, without significant side effects.

DISCUSSION

This syndrome, originally described by Dejerine and Roussy in 1906, was ascribed to infarction in the thalamus ($_1$). It is also known as thalamic syndrome, or post-stroke syndrome. It has been associated with a variety of thalamic infarctions and hemorrhages. Its incidence is 2-6% in stroke patients, but can be as high as 25% after medullary infarct (Andersen et. al., 1995). The pain may be described as “burning” or “freezing,” and may be associated with mild motor disability. Pain may be exacerbated by sensory stimuli, body movement, or strong emotions. Various sensory abnormalities are commonly found in these patients, including disturbances of temperature discrimination. The area of sensory disturbance is usually larger than the area of the pain itself. Autonomic disturbance is common and the

limb may appear edematous and cold.

Several pathophysiologic mechanisms have been proposed. Pain could result from an “irritable focus” at the central site of injury. Or, as suggested more recently, pain could result from a lack of inhibition in the pain signaling system, i.e. the spinothalamic pathways . Another theory concludes that central pain is due to the disruption of thermosensory integration and the loss of cold inhibition resulting in the experience of burning pain ([2]). The lesion, somewhere along the spinothalamic projections, removes the suppressing activity exerted by the reticular thalamic nucleus, thereby releasing abnormal activity in other thalamic nuclei, which, in turn, leads to pain hypersensitivity.

The role of the ventroposterior nucleus of the thalamus (VPN) in central pain was analyzed in a series of article in the American Pain Society journal in 1992. Lenz showed that in primates the VPN receives nociceptive inputs ([3]). Other studies have shown the involvement of calcium channels and excitation of NMDA receptors in central pain. Wiesenfeld-Hallin has shown that pretreatment with the NMDA antagonist MK-801 prevents allodynia in rats with a lesion that strikes both white and gray matter. ([4])

Treatments with antidepressants, especially those adrenergically active (eg. amitriptyline), have been effective and should be started as soon as the diagnosis is suspected. Response to therapy usually occurs within four weeks and does require only low to moderate doses of TCA’s. ([5]) When response is poor the anti-convulsant carbamazepine (Tegretol), or Mexiletine may be useful. TENS may be used if the painful area has not lost touch and vibration sensibility. Surgical options are varied and have enjoyed some success. Use of opioids has been controversial, however, the rationale behind using methadone was its NMDA blocking property.

Although we do not know the exact mechanism whereby methadone relieves pain in the Dejerine Roussy syndrome, our patient has been consistently reporting pain relief since treatment was initiated.

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