

## Educational case report

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# A case report of a thalamic stroke associated with sudden disappearance of severe chronic low back pain

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### Abstract

**Background:** Chronic pain conditions are associated with neuroplasticity within the central nervous system. In most patients the maladaptive consequence of neuroplasticity supports prolonged course of chronic pain, despite the absence of a commensurate etiology. From a pain neuromatrix perspective it can involve three different circuits within the central nervous system; the classical sensory pathway, the limbic system pathway, and the associative pathways involving the parietal cortical connections. Although this can be conceptualized as a fluid system composed of several interacting networks, it can be broadly separated into a nociceptive specific network of spino-thalamic neurons and second order neurons beyond thalamus that are not nociceptor specific. Thalamus acts as an important relay station that conveys nociceptive signaling to higher centres. Neuroplastic changes can potentially involve any parts within this neuromatrix. It is very uncommon to observe the sudden disappearance of such a chronic pain condition.

**Methods and results:** In this case report, the author describes the clinical course of a patient with severe chronic low back pain (CLBP), whose pain suddenly disappeared after a stroke involving his left thalamus. Although extremely rare, existing case reports of such disappearance of pain with a secondary stroke in patients suffering from central post stroke pain (CPSP) are reviewed. The author further postulates hypotheses that could potentially explain this phenomenon based on the existing knowledge.

**Conclusions and implications:** Although extremely rare and unpredictable, a thalamic stroke involving areas that are involved in chronic pain signaling can potentially lead to disappearance of an existing chronic pain condition. This is the first case report of such sudden disappearance of CLBP with well established nociceptive pathology supported by clinical and imaging findings. This unique case report could potentially generate ideas for future research and clinical treatment in the field of neuromodulation and brain stimulation.

**Keywords:** neuromodulation; neuroplasticity; thalamic pain; post-stroke pain; thalamic stroke; deep brain stimulation.

## 1 Introduction

Pain is transmitted from peripheral nociceptors to higher cortical areas, where it is perceived, localized and emotionally experienced. The various neural networks of the body-pain neuromatrix sub serve the sensory-discriminative, affective-motivational, and evaluative-cognitive dimensions of pain experience [1, 2]. Within this network, thalamus acts as an important relay centre to process peripheral signals before they are relayed to higher cortical areas [3]. Thalamus is also prominently involved in patients who suffer from central post stroke pain (CPSP) syndrome [4], and is an important target for deep brain stimulation for chronic pain [5, 6]. Through this report, the author highlight a case of sudden disappearance of chronic low back pain (CLBP) in a patient who had left thalamic stroke and review the relevant literature.

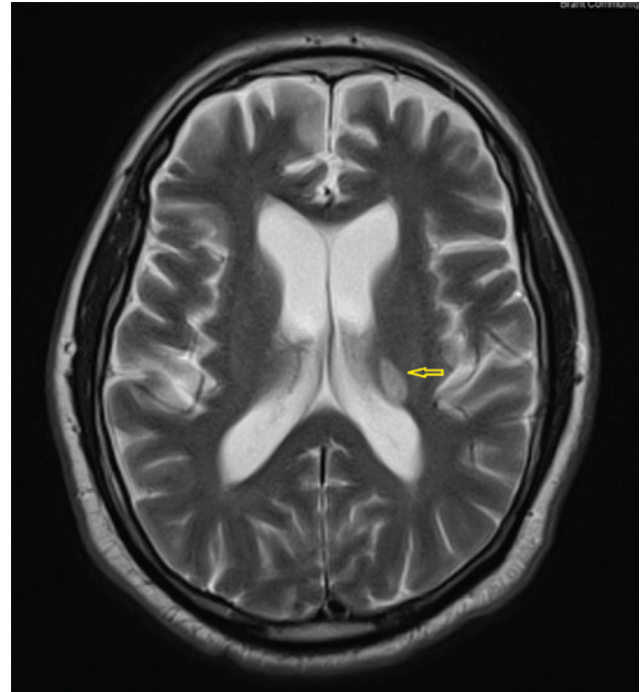
## 2 Case report

A 66 year old gentleman with a 6 year history of CLBP was referred to our pain clinic for management in November

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2012. His comorbidities included obesity, hypertension, type 2 diabetes mellitus, obstructive sleep apnea, hypothyroidism, and mild depression. Before being seen in our clinic, he was being managed with hydromorphone contin 12 mg (equivalent to 40 mg of morphine)-three times a day, along with hydromorph 2 mg (equivalent to 10 mg of morphine) breakthrough tablets-taken as 3–4 times a day, in addition to pregabalin 150 mg twice a day. He was referred to our clinic for consideration of spinal interventions as his CLBP was moderate to severe in intensity, despite his medical management. His clinical examination did not reveal any red flags of neurological pathology. Imaging findings suggested age related multi-level degeneration of disc and facet elements, with significant arthropathy at lower three lumbar levels, along with moderate foraminal narrowing. Based on his clinical and imaging findings, he was offered radiofrequency (RF) ablation of medial branches of the affected facet joints, after a positive diagnostic medial branch blockade. As the RF intervention was successful in controlling his spinal pain for an average duration of 6–8 months, he continued with repeat RF procedures with predictably good success, without the need for other interventions or increases to his medication. Over the course of 4 years (2012–2015) he underwent seven RF sittings, and was due for a repeat of the RF procedure during February 2016. However, having missed his procedural appointment, the patient and his wife visited our clinic during April 2016 to inform us of the following surprising developments. As per their report, during the month of January 2016, patient was observed to exhibit odd cognitive and behavioral changes by his wife. He demonstrated forgetfulness, did not remember how to put on his positive pressure mask for sleep apnea, and was searching for his deceased brother at home. He was taken to the hospital, considering a diagnosis of early dementia or vasculitis. However, further workup, including a CT scan revealed that the patient had suffered an acute left thalamic infarct. His MRI confirmed the location of infarct to be posterior medial aspect of the left thalamus (Figure 1). Because of the nonavailability of a 3D MRI, a more precise location of the thalamic nucleus was not possible.

Over the course of the next 1–2 weeks he was evaluated at the stroke prevention clinic. It was observed that the patient recovered completely in his cognitive and behavioral functions, as well as in his memory. Also, fortunately enough, his CLBP completely disappeared without the need to continue with any of his pain medications, including daily opioids, which he had been on for more than 10 years. The following findings were



**Figure 1:** T1 weighted MRI image demonstrating stroke in the left thalamus.

observed during the neurological examination in March 2016 (2 months after his thalamic stroke). His pupils were equal and reactive to light. Visual fields were intact to confrontation; however, he was not able to count fingers in the upper quadrants in both eyes, but was able to appreciate movement in these areas. Extraocular movements were full with no nystagmus. Facial sensation to light touch and movements were normal. Hearing was grossly normal. There was symmetrical palate elevation, normal trapezius strength and tongue was midline. Muscle bulk, tone and power were normal in all four extremities. Sensation was intact to light touch and vibration sense, observed in all dermatomes. Deep tendon reflexes were 2+ throughout. Plantar response was flexor bilaterally. Coordination was normal on finger-to-nose testing and rapid alternating movements. There was no bradykinesia. There were no extrapyramidal features. Gait was normal. Tandem gait was slightly unsteady, but within normal limits. Romberg was negative. Similar neurological findings were observed during his examination in April 2016, as well as in his last follow up visit in September 2017 (more than 18 months after his stroke). With the sudden disappearance of CLBP, patient has not felt the need for repeating his RF procedure. He continues to be with minimal pain even 18 months after the stroke without any need for opioid or other medications or spine interventions.

## 3 Discussion

This is a unique case report of a thalamic stroke resulting in the disappearance of CLBP, with well established nociceptive pathology supported by clinical and imaging findings. To the author's knowledge this is the only case, besides the description by Head and Holmes of a case in which a phantom limb pain disappeared after an infarct of the opposite parietal lobe [7], of a peripheral source of pain that has been positively affected as a result of stroke. A few case reports of such occurrence in CPSP have been described.

### 3.1 Comparative literature

Soria and Fine described a report of disappearance of thalamic pain after parietal cortical stroke and attributed this to possible disruption of the thalamo-parietal radiations [8], while Hirato et al. [9] considered the possibility of secondary cortical damage in a patient who had accidental putaminal lesion leading to pain disappearance. It is interesting that the laterality of the subsequent stroke lesion may not be crucial for the pain relieving effect, as shown by the Daniele et al. [10] and Helmchen et al. [11], both cases reporting resolution of thalamic pain following a second stroke in the contralateral hemisphere. They postulated that the bilateral connections of the spinothalamic tract could partially explain this phenomenon, and insisted that CPSP resulted from a bilateral disorder of functional plasticity. Even in the present patient, bilateral pain was affected, despite the left sided lesion. There is ample evidence to support that the pain processing during noxious stimulation in normal subjects involves bilaterally distributed networks [12].

### 3.2 Role of thalamus and thalamo-cortical projections in pain processing

It is well known that thalamus plays an integral part, and acts as the major relay station between the spinothalamic tracts (STT)-carrying the pain signals, and the cortical structures [2]. Further, the thalamus is also intricately involved in processing pain memory [13]. The nociceptive inputs can be transmitted directly through STT, or indirectly through the spinoreticular, spinomesencephalic, or even dorsal column pathways to the thalamus [2]. Although the pain pathways beyond the thalamus have been hard to decipher [14], a nociceptive-specific matrix comprising the cortical regions receiving direct STT input has been described,

which includes the posterior operculum and insular areas [2]. Following activation of this specific network, studies of functional neuroimaging have consistently indicated that there are many cortical areas that contribute to the pain experience. However, none of them are specific to pain [2]. The cortical areas commonly activated by these thalamo-cortical projections include the areas of somato-sensory cortex, the insula, anterior cingulate cortex (ACC), the prefrontal and posterior parietal areas.

### 3.3 Potential hypotheses to explain the disappearance of pain

In the present patient, disruption within the thalamo-cortical pathways could have led to selective loss of afferent nociceptive signaling, leading to modulations in the thalamo-parietal connections, and thereby abolishment of pain. Another hypothesis that can be considered is based on the specificity of nociceptive signaling, as reported by Craig et al. [15]. Although not widely accepted, Craig et al. reported that nociceptive signals from lamina I are specifically projected via the posterior part of the ventral medial thalamic nucleus (VMpo) and the ventral caudal part of the medial dorsal nucleus (MDvc) [14, 15]. The present patient had a stroke that involved the posterior medial aspect of the left thalamus, which encompasses the above areas. However, he did not develop central thalamic pain or contralateral loss of cold sensation, which would be expected with Craig's hypothesis. It is interesting to note that this thalamic stroke did not result in CPSP, which can happen in 11%–55% of patients, and is more common after lateral medullary infarction or lesions in the ventroposterior part of the thalamus [4]. It is suggested that thalamic pain develops when there is impairment of spinothalamic transmission with involvement of the anterior pulvinar nucleus [16]. From a therapeutic perspective, Rasche et al. [6] noted that the best long-term results after deep brain stimulation were observed in patients with chronic low-back and leg pain, compared to central pain syndromes such as due to spinal cord injury and poststroke pain.

## 4 Conclusion and implications

While the consequence of a stroke is indeed poor on health, it is intriguing that it carries the potential to eliminate a daily source of pain, anguish, and disability. It is important that the potential for such neuroplasticity is

taken into account in future research and neuromodulatory treatments, including deep brain stimulation.

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**Author's statements**

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**Ethical approval:** Not applicable.

**Author's contributions:** The author has been the patient's pain physician and attests to the veracity of this report. All relevant literature search and writing of this report was performed by the author.

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