

involved intercostal nerves been tracked by high-resolution ultrasound at the onset of pain. Computed tomography and magnetic resonance imaging are considered superior tools to rule out intercostal neuralgia caused by intraabdominal pathologies because the rib cage has minimal influence on images obtained from both modalities. This case highlights the necessity of computed tomography for surveillance of intra-abdominal pathology and also underscores the importance of examining the whole innervated region before intercostal block, especially in patients with known etiology or history of malignancy.

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# LETTER TO THE EDITOR

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OXFORD

## Thalamic Amnesia Accompanying Disruption of Pain Memory: A Case of Right Anterior Thalamic Infarction and a Subsequent Vertebral Compression Fracture

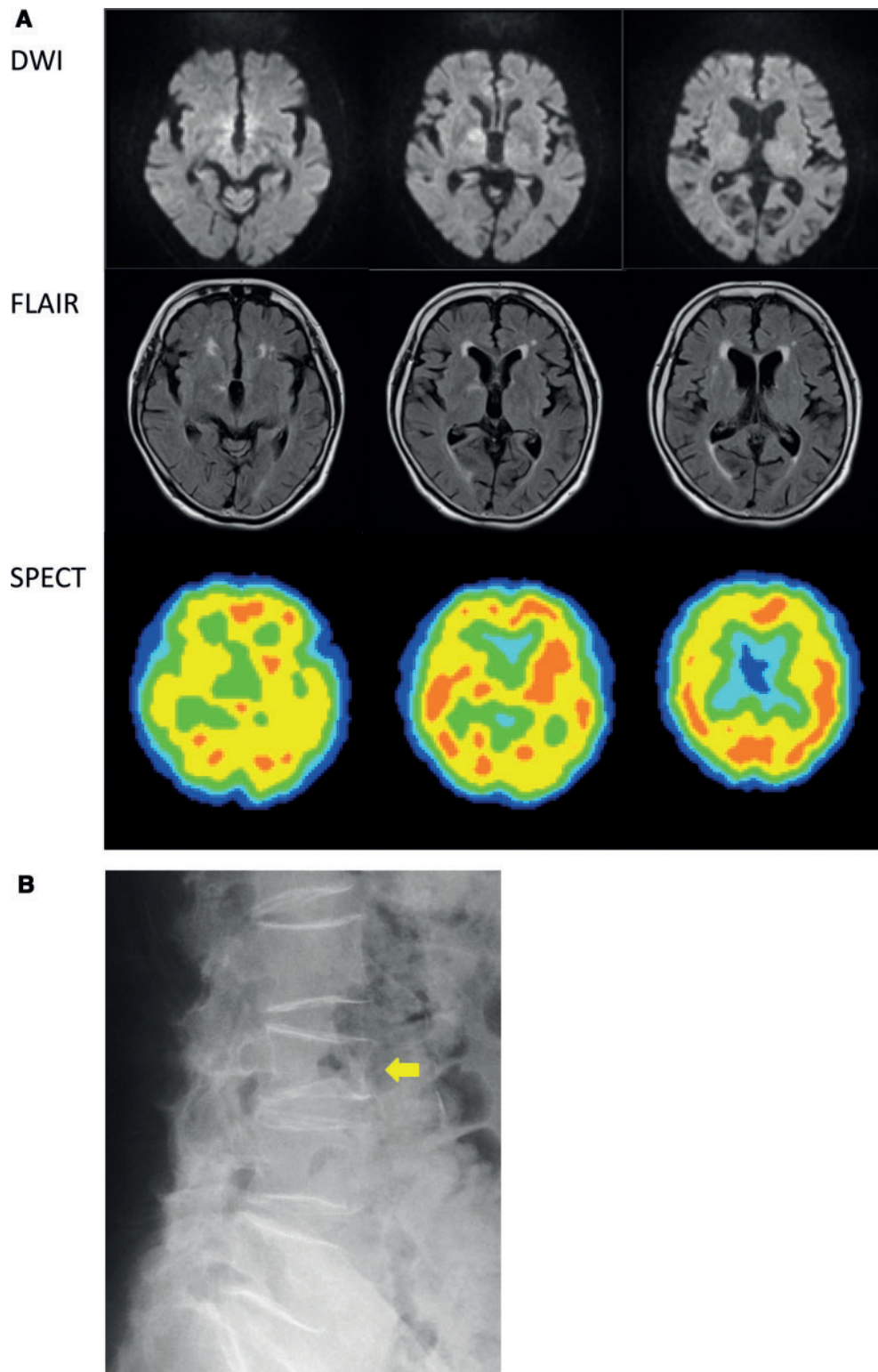
Dear Editor,

During the encoding of pain memory, the thalamus works as a hub between a variety of subcortical areas and the cerebral cortex. These functionally correlated brain areas, collectively called pain matrices, are activated during the establishment and processing of pain memory [1]. The thalamus is important in the discrimination and encoding of noxious stimuli [1–4]; however, the role of the thalamus in the persistence of pain memory remains poorly understood.

An 82-year-old, right-handed woman with 14 years of formal education was admitted to our hospital. She had been performing daily activities without assistance. She had a history of mild glucose intolerance and no prior history of neurological or musculoskeletal problems. At onset, she had a sudden feeling of dizziness and fell to the floor. Immediately thereafter, she complained of strong low back pain, and voluntary movements were severely restricted. At that time, she misidentified her 17-year-old grandchild. She repeatedly tried to stand up to walk without pain anticipation

in defiance of her families' advice not to forget her previous painful experiences, which resulted in recurrence of severe pain. Her memory was suspended for a period after the onset; however, she remained conscious through the entire event. She was taken to the hospital the same day and was diagnosed with a cerebral infarction. In the course of her conservative treatment, one day, she suddenly recognized her severe low back pain and her amnesia resolved while moving from the clinic bed to a wheelchair with assistance. She regained her memory functions, including pain memory, concomitantly. During her stay at the hospital, she did not receive any analgesics, including opioids or sedatives. She was referred to our hospital for rehabilitation 16 days after stroke onset.

Upon admission to our hospital, she was conscious and cooperative during examinations. No obvious abnormalities in movement or sensory abilities were observed. She did not manifest ataxia or coordination disturbances. Her low back pain was mostly alleviated, and she was able to walk without assistance. She was aware of being hospitalized because of illness. She



**Figure 1** (A) Magnetic resonance imaging (MRI) and single-photon emission computed tomography (SPECT) scans that were performed upon admission. The MRI scan revealed a cerebral infarction in the right anteromedial thalamus. The SPECT scan showed an area of low perfusion in the right thalamus as well as in the right frontal and temporal lobe. (B) X-ray film; lateral view of the lumbar spine. A fresh L3 vertebral compression fracture was detected (indicated by a yellow arrow). DWI = diffusion-weighted imaging; FLAIR = fluid-attenuated inversion recovery.

consistently answered our questions, except for questions regarding the period of amnesia. On further inquiry, the patient described things after the return of her memory mostly was compatible with what her daughter said. Then, it was judged that the duration of her memory deficits accompanying disturbance of pain memory was about 10 days from the onset, around the end of a series of her conservative treatment. Due to the experienced anterograde amnesia, she exhibited disorientation with regard to the date. Although there was no loss of intelligence, neuropsychological tests revealed slight memory dysfunction. She scored 26/30 in the Mini-Mental State Examination, with declining performance in orientation, attention and calculation, and reproduction. Results of the Auditory Verbal Learning Test indicated a decrease in immediate recall (2, 3, 3, 7, 8/15) and delayed recall (0/15). On the Wechsler Memory Scale-Revised, her verbal memory was 81, visual memory was 80, general memory was 79, concentration/attention was 89, and delayed recall was 79. Her Wechsler Adult Intelligence Verbal IQ was 101, Performance IQ was 94, and Full-Scale IQ was 98. An MRI performed 18 days after onset revealed a cerebral infarction, which involved the anteromedial aspect of the right thalamus (Figure 1A). Single-photon emission computed tomography (SPECT) (N-isopropyl-p-(iodine-123)-iodoamphetamine) that was performed on the same day revealed an area of low perfusion in the right thalamus, which involved the right frontal lobe (Figure 1A). Her fresh L3 vertebral compression fracture was diagnosed by x-ray radiography (Figure 1B). Rehabilitation was performed using a lumbar corset. She had no resting pain, nor visual cue, for awareness of the low back pain. Therefore, we repetitively induced her to put on her lumbar corset when she got out of bed. In her memory rehabilitation, we performed conventional rehabilitative intervention as to lost memory function, such as reconstructing autobiographical memory or learning methods, especially emphasizing encoding. We also provided compensatory techniques using external memory aids such as a memory note, a timetable, and a timer. The fracture fused without pseudoarthrosis or delayed union. She regained her former abilities to perform her daily activities independently, but she did not regain her lost memory accompanying the painful experiences during her three-month hospitalization period.

Long-term memory is classified as the following: declarative (explicit) and nondeclarative (implicit) memory. Explicit memory divides into two categories, episodic memory and semantic memory. In these, previous experiences or concepts are consciously recalled and are interpretable verbally. On the other hand, implicit memory, which includes procedural memory, operant conditioning, and associative learning, is comprised of memories, which are difficult to verbalize because a series of obtained actions such as skills work unconsciously.

The initial perception and cognition of pain, which results in a defensive withdrawal reflex, produces an

increase in unpleasant emotional and psychological factors, including fear, caution, anxiety, sadness, and anger [5]. Emotion and memory are closely related, and affective cognition plays a role in the establishment and processing of pain memory. Heightened pain intensity enhances the pain memory encoding and recall [6,8,9]. When processing pain memory, several brain components (collectively referred to as pain matrices) functionally collaborate, including the primary and secondary somatosensory cortex, medial prefrontal cortex (mPFC), amygdala, insular cortex, hippocampus, anterior cingulate cortex, and thalamus [1,5]. Patients experiencing pain due to lumbago normally restrict voluntary movement in fear of pain recurrence [6]; in addition, low back pain can lead to further activation of these pain matrices [7]. The somatosensory pain system includes thalamic nuclei that receive sensory signals through spinothalamic pathways and transport them to associated primary and secondary cortical areas [2]. The thalamus plays a central role by activating reciprocal thalamic projections in this pain-related neural network. The ventral posterolateral (VPL) nuclei of the thalamus are associated with discrimination for nociceptive stimuli through the reciprocal pathways to these somatosensory cortices [2,3]. Dorsomedial (DM) nuclei of the thalamus are involved in pain-related working memory through the interconnections with mPFC [4]. mPFC–basolateral amygdala (BLA) transmission contributes to fear memory encoding [8]. The BLA then regulates hippocampal activity, where the pain memory is encoded for storage by changing the strength of neural connections [9].

The anterior nuclei of the thalamus are not commonly implicated in the establishment or maintenance of pain memory. Previous reports describe patients with anterior thalamic lesions that exhibit amnesia [10,11]. The anterior nuclei of the thalamus are functionally connected to the hippocampus through the mammillothalamic tract. This circuit is part of the Papez circuit, which is involved in the establishment and maintenance of long-term memory and is important for episodic memory [2].

In our case, a pain signal caused by nociceptive stimuli of an L3 vertebral compression fracture ascended bilaterally through the spinothalamic tract to cortical areas via the ipsilateral thalamus. The patient complained of severe low back pain immediately after the fall. Thus, functions of pain cognition and pain discrimination were intact. However, she could not remember her previous painful experience in the absence of pain-related visual stimuli (unusual status that provides pain association, such as contusion or bandages on extremities or trunk) [7]. She frequently attempted to stand up and walk without hesitation [12]. One of the possible explanations for recovery of amnesia is that, after the acute period, the residual function of affected The anterior nuclei of the thalamus compensates after alleviation of edematous change around the lesion or that the thalamus in the dominant hemisphere is covered for memory impairment. It is likely

that the anterior nuclei of the thalamus contribute to pain memory processing, which is induced by acute painful stimuli, in addition to the VPL and DM nuclei of the thalamus, normally regarded as integral to pain processing [2–4]. A limitation of our report is that the resolution does not clearly distinguish the localization of The anterior nuclei of the thalamus.

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