

Lesson of the month

MRI demonstration of an anterolateral plaque at C1: a note on some sensory changes including analgesia

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Abstract

A patient developed a partial right-sided Brown-Séquard syndrome at C1. Impairment of left-sided spinothalamic function was associated with abolition of pre-existing left- (but not right-) sided low back pain and sciatica, and prevented the postoperative pain expected after surgery for a benign left-sided breast lump. Later, slight left-sided alteration of light touch appreciation, then allodynia and spontaneous burning pain developed. MRI scanning revealed an isolated abnormality in the right anterolateral quadrant of the spinal cord at C1, consistent with a plaque of demyelination. The site of this abnormality, demonstrated during life and non-invasively, accounted for the partial Brown-Séquard syndrome, the analgesic effects of interrupting spinothalamic pathways, and perhaps the delayed-onset central pain phenomena.

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For over a century it has been known that tracts which subserve spinothalamic functions including pain ascend in the anterolateral quadrant of the spinal cord. In humans confirmation of this localisation has come from four lines of evidence: the contralateral loss of pain produced by diseases affecting an anterolateral quadrant¹; the pain-relieving effects of open surgical² and percutaneous³ anterolateral cordotomy; the rare case of pain that remains after section of the spinal cord that has spared just one anterolateral quadrant,⁴ and the contralateral pain that results from stimulation of the anterolateral quadrant in awake subjects.⁵

The sensitivity of modern MRI now allows visualisation of small areas of focal disease hitherto not possible. The present case demonstrates an area characteristic of a plaque of demyelination in the right anterolateral quadrant of the spinal cord at C1, and for the first time confirms non-invasively the function of this area in transmission of spinothalamic functions and modulation of pain.

Case report

This 46 year old woman presented with sudden inability to appreciate the temperature of bathwater with the left foot. Within hours the whole of the left side of her body became similarly involved, though the face was spared. There was some painful sensitivity over the left shoulder, and the left hand felt slightly numb. Examination 24 hours after the onset of her symptoms showed complete absence of pinprick appreciation on the left below and including the C2 dermatome; temperature appreciation of both hot and cold was considerably impaired. Light touch appreciation was intact, apart from some slight tingling that was induced over the left shoulder. Deep pain from pressure on the fingers and Achilles tendon was appreciated less on the left. Postural and vibration sense were entirely normal. The cranial nerves including palatal and trigeminal sensation and the corneal responses were also normal. Her reflexes were normal, except the right plantar response was less firmly flexor compared with the left.

For many years she had experienced chronic low back pain with diffuse spread of the pain down the back of both thighs, attributed to degenerative spine disease. Three weeks before her neurological symptoms began, a left breast lump had been detected, and 72 hours after her sensory symptoms had begun the mass was uneventfully biopsied under general anaesthetic. The mass proved to be a benign fibroadenoma.

When reviewed two weeks after surgery, the loss of temperature appreciation had persisted, but she had begun to drag the right leg and the right hand had become weak and clumsy. There was no sphincter dysfunction. Examination showed the cranial nerves were still normal, she had persistent left-sided loss of pinprick and temperature appreciation below C2, and in the same area light touch felt "different"—slightly thicker and less sensitive compared with the right; deep pain was still impaired but just appreciated. Postural and vibration sense remained perfectly preserved in all four limbs. There was pyramidal weakness in the right arm and leg with a very clumsy right hand and slowed finger movements. She had exaggerated right-sided

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Figure 1 MRI scan showing altered signal (black arrow-heads) in the right anterolateral quadrant of the spinal cord (open arrow) lying within spinal fluid at C1 level. A) axial image from a volume acquisition, gradient recalled acquisition in steady state pulse sequence, flip angle 8°, TR 100/TE 20. Slice thickness 1.5mm. (GE Vectra 0.5T); B) sagittal image from a multiplanar gradient recalled acquisition state pulse sequence, flip angle 25°, TR 640/TE 30.

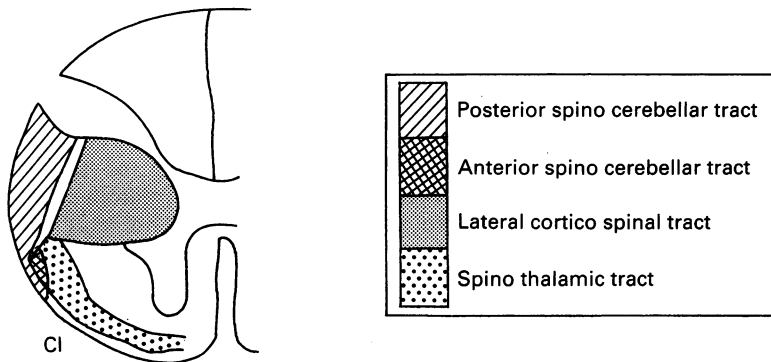
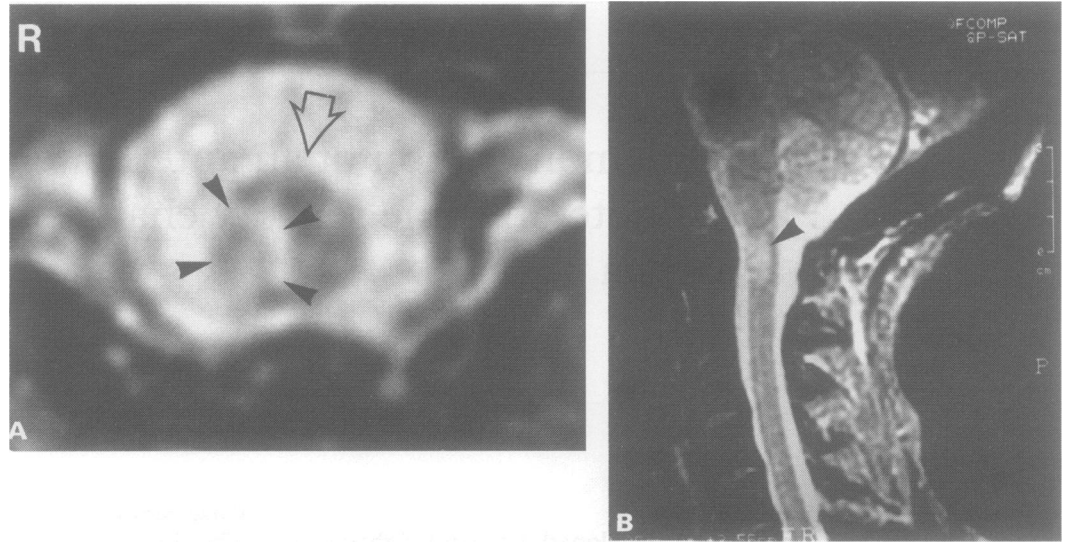


Figure 2 Diagram to show location of long tracts of the lateral column at C1. Adapted from Smith.¹² With permission of The Macmillan Press.

reflexes, reduced right-sided abdominal responses and a right extensor plantar response.

After a week (without treatment) her right-sided symptoms resolved completely over a few days, though the left-sided pain and temperature impairment remained. A month later, there had been little change, except she had developed a burning component to the left-sided sensory disturbance when the skin was lightly touched, and at times there was spontaneous burning discomfort, sometimes painful, which was felt predominantly in the left arm. Over the subsequent five months, these burning sensations slowly disappeared, but she had slight residual impairment of temperature appreciation affecting the left lower arm, lower trunk and leg.

During this period, she spontaneously reported two features which surprised her. First, she had experienced neither pain nor discomfort of any sort following surgery and subsequent removal of skin sutures from the left breast. Second, though she continued to have right-sided low back pain, for the first time she felt no pain on the left side or down the left leg.

MRI scanning during the first two weeks of her illness revealed a single, focal area of altered signal in the right anterolateral quadrant of the spinal cord at or just below the level of the foramen magnum, consistent with localisation at the spinal cord segment C1 (fig 1). The abnormal area did not cross the mid-

line, and it did not appear to extend posteriorly to involve the dorsal column nor probably the dorsal horn. No other abnormalities were detected on scanning the brain or cord. A repeat MRI scan a month later when her burning dysaesthesiae were present was unchanged.

Median nerve somatosensory evoked potentials from both hands, visual evoked potentials, chest x ray, blood count, ESR, auto-antibody titres, lupus anticoagulant screen, and serological tests for syphilis were normal. Her spinal fluid showed normal protein content and cell count, but electrophoresis showed local synthesis of IgG with oligoclonal bands, which were absent in serum.

Discussion

This patient demonstrates the presence of a partial, right-sided Brown-Séquard syndrome. For the first time, this can be correlated during life with a focal lesion in the right anterolateral quadrant of the spinal cord at the level of C1, as shown by MRI scanning. The clinical features, scan appearance and CSF findings are consistent with a single inflammatory lesion, as seen in an initial episode of multiple sclerosis.⁶

There are a number of conclusions which can be drawn from this patient. She proves during life that pain and temperature appreciation are indeed subserved by pathways that travel in the opposite anterolateral quadrant. This confirms observations that could only be inferred during life, since hitherto it has only been possible to clarify post-mortem the precise site of the disease process or surgical lesion. Consistent with the loss of spinothalamic function is the demonstration of analgesia. This analgesia was evident for two different sorts of pain. First, there was apparent *prevention* of the superficial pain that would have been expected after surgery, presumably because the operation site fortuitously included an area where there was pre-existing spinothalamic sensory impair-

ment. Second, there was selective abolition on the left side of the longstanding, deep, musculoskeletal pain that had been present before sensory loss occurred.

Later she experienced allodynia (that is, pain due to a non-noxious stimulus, such as light touch) and spontaneous burning pain. These features, well recognised in central pain states, developed some days after the onset of her symptoms. From observations on patients with acute, traumatic spinal cord injuries, when the time course of sequelae can easily be ascertained, a delay of days, or sometimes weeks or months, is often encountered and attributed to central neural dysfunction, the mechanisms of which are uncertain.⁷

Although quantitative testing was not undertaken, some subtle alteration of light touch on the same side as the spinothalamic impairment was described, with diminution and a "thick" sensation being reported. Ipsilateral (right-sided) sensory changes were not encountered. Impairment of light touch contralateral to the lesion (and also ipsilateral hyperpathia) have occasionally been observed both in humans and in experiments on animals. As with the development of dysaesthesiae in central pain states, the mechanisms of these sensory abnormalities may depend on release of central inhibition with subsequent poor modulation and wide irradiation, the variable clinical phenomena being related to the degree of hemicord involvement from disease or surgically-induced lesions.⁸

Despite the evidence in animals of ascending nociceptive tracts that travel in the dorsal columns, in particular spinocervical and post-synaptic dorsal column pathways,⁹ these columns appeared to be intact on MRI scanning of the present patient. The preserved somatosensory evoked potentials elicited by median nerve stimulation again suggest the dorsal columns were intact, since the evoked volley is transmitted through dorsal column/medial lemniscal pathways.¹⁰ Thus it is unlikely that tracts within the dorsal columns were implicated in the sensory disturbances that the patient experienced.

The fact that trigeminal sensation remained completely intact confirms that the descending nucleus and tract of the fifth nerve, which descend to C1 and probably more caudally, must lie posterior and presumably in the substantia gelatinosa of the

spared dorsal horn. In addition, second-order trigeminal fibres must not only remain posterior but ascend to the brainstem before crossing the midline.¹¹ This anatomical arrangement not only accounts for sparing of facial sensation in this patient, but is the rationale for the operation of medullary tractotomy which provides spinothalamic facial sensory loss with preservation of light touch.

Finally, the transient motor features are consistent with involvement of descending corticospinal and possibly ascending spino-cerebellar fibres. Clinico-anatomical studies in humans have shown that in the high cervical region these fibres are in close proximity to the spinothalamic tract. These meticulous histological studies on large numbers of patients enabled Smith to summarise diagrammatically the location of the long tracts of the lateral column at different spinal levels.¹² The location of these tracts at C1 is shown in fig 2 for comparison with the MRI scan appearances of this patient.

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