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Clinical short communication

Spinal cord infarction with ipsilateral segmental neuropathic pain and flaccid paralysis. A functional role for human afferent ventral root small sensory fibres

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ABSTRACT

This paper illustrates the cases of two patients with an acute onset of right brachial neuropathic pain, flaccid paralysis and contralateral thermal and thermal pain hypoesthesia, without posterior column impairment nor pyramidal signs below the segmental lesion. MRI showed right sided spinal cord infarction, in the anterior spinal artery territory between C1 and C5 in one patient and between C3 and C7 in the other. Contact Heat Evoked Potentials and Quantitative Thermal Sensory testing are consistent with contralateral, but not ipsilateral, spinothalamic tract involvement. Electromyographic results established ipsilateral segmental denervation and somatosensory evoked responses were consistent with dorsal column sparing.

Unilateral anterior cervical spinal cord infarction may present with acute ipsilateral segmental neuropathic pain, lower motor neurone-type weakness, contralateral thermoanalgesia and no pyramidal signs. The ipsilateral pain provides novel evidence that in some instances, ventral roots can play a role in nociception in humans. The infarcted territory may result from occlusion of a sulcal commissural artery or a number of more proximal vessels (including a single or duplicated anterior spinal artery, vertebral arteries or feeding radicular arteries).

1. Introduction

Transient neuropathic pain often occurs [3,14] in the classical syndrome of anterior spinal artery infarction. The presenting symptoms of unilateral neuropathic pain, segmental lower motor neurone weakness without pyramidal signs, and contralateral spinothalamic dysfunction is under-recognised and often initially diagnosed as myelitis [16]. The mechanism responsible for.

the unilateral pain has not been determined. We consider the clinical, radiological and neurophysiological findings obtained from 2 cases of patients with infarction of the spinal cord in the distribution of the anterior spinal artery to support our hypothesis that the ventral roots may play a role in the genesis of ipsilateral segmental neuropathic pain in this syndrome.

2. Case reports

2.1. Case 1

A 22-year-old woman developed flaccid paralysis of elbow flexion and arm abduction that was accompanied by acute spontaneous burning pain in the right shoulder, proximal arm and neck. She had experienced a head and neck trauma one week before and was taking oral contraceptives. The pain improved over the next 5 h in association with ketoprofen, 200 mg IV. Upon admission to the hospital 4 days later, the patient was found to have normal cranial nerve function, thermal anesthesia in the left arm and leg and no spontaneous pain. Physical examination revealed paralysis of the right shoulder abduction and elbow flection and absent right biceps tendon reflex, but no weakness of left upper limb, or lower limbs, no pyramidal signs were noted. There was evidence of thermo-analgesia on the left below C4

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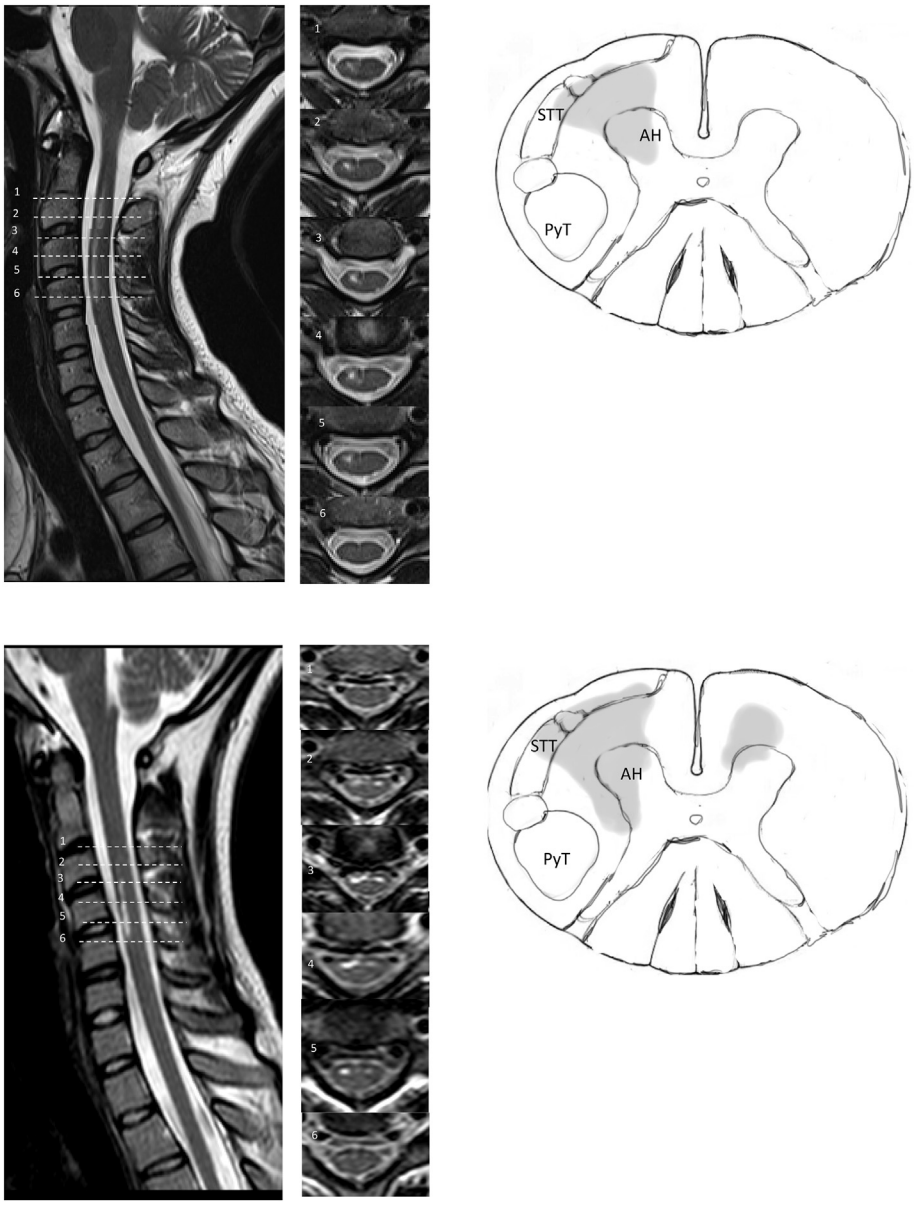

Fig. 1. A. An MRI of the cervical spine from patient 1 taken five days after the onset of symptoms. The left panel presents a sagittal view of the cervical spine (T2) with a hyperintense narrow longitudinal area extending from C2 to the C5 level. The middle panel presents transverse images at distinct levels according to the number references. Drawings based on the Neuroanatomy Atlas of D.E. Haines illustrating sections of the cervical spinal cord with superimposed dashed lines that represents the boundaries of the largest lesion obtained on MRI are presented in the right panel. B. An MRI of the cervical spine from patient 2 taken thirty days after the onset of symptoms. Left, sagittal view of cervical spine (T2) with a hyperintense narrow longitudinal area extending from C3 to C4–5 level. Middle panel with transverse sections at various levels according to the number references. Right: drawing of the cervical spinal cord based as in A, with superimposition of the largest lesion (dashed) obtained on MRI.

level, but response to touch was normal and there was no sphincter dysfunction. An MRI of the brain was normal. An MRI of the cervical spine (Fig. 1 top) showed a non-enhancing hyperintense signal in the anterior right quadrant of the spinal cord between C1 and C5. The cervical roots were of normal calibre and showed no obvious signs of pathology. There were no signs of vertebral artery dissection up to the basilar artery junction. An initial Electromyography (EMG) showed no activation of right biceps, deltoids and infraspinatus muscles. The cerebrospinal fluid (CSF) was normal. Contact Heat Evoked Potentials (CHEPS) derived from stimulation of the forearms were normal on the right and absent on the left (Fig. 2). Somatosensory evoked responses (SEPs) from the median nerves were normal and symmetric. EMG showed positive sharp waves and fibrillations in right biceps, deltoid and infraspinatus muscles. Quantitative thermal testing (QST) (TSA-II Medoc, Israel) showed thermal anesthesia (0° to 50 °C) on the left. The patient reported only weak “tingling” when the stimulus reached 50 °C. Thermal thresholds were normal on the right (see Fig. 2). Biochemical, hematological and coagulation studies, including S and C proteins were normal. She received IV methylprednisolone 500 mg daily for five days, without change in the weakness. At follow-up the clinical signs were unchanged one year later.

2.2. Case 2

A 24-year-old female on oral contraceptives, developed acute occipital headache, severe cervical pain radiating to the right arm above the elbow and marked weakness of the right shoulder and arm. Neurological examination revealed no pyramidal signs and that her perception of thermal and pinprick sensations was decreased on the left below C3. When admitted at the emergency room, an MRI of the brain was found to be normal and MRI angiography did not reveal carotid or vertebral artery dissection. An MRI of the cervical spine obtained two days later revealed a T2 hyperintense lesion on the right anterior quadrant of the spinal cord extending between C3 and C7 with a small area of gadolinium enhancement (Fig. 1 bottom). An additional smaller hyperintense area in the left anterior horn extended from C4 to C6. CSF was normal. She received IV methylprednisolone and then plasmapheresis with no improvement. An EMG obtained four months later revealed positive sharp waves and fibrillations in right C5-C6 territory. The sensory examination was unchanged. Eighteen months after the onset there was residual weakness of the right deltoid and biceps muscles and persistence of a decrease in the perception of pain and temperature below the C3 spinal level, thought to be due to a lesion