SHORT REPORT

Neurophysiological findings and MRI in anterior spinal artery syndrome of the lower cervical cord: the value of F-waves

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Abstract

Ten hours after onset of an anterior spinal artery syndrome (ASAS) F-waves could not be obtained from the paralysed left hand muscles. F-waves in the right hand were present and motor nerve conduction studies were normal on both sides. One week later paralysis of the right hand together with paraparesis occurred. Two days later F-waves from the right hand had disappeared. Three weeks later MRI confirmed the diagnosis of ischaemic spinal cord infarction.

Occlusion of the anterior spinal artery initially impairs its entire distribution area at the level concerned, including the boundary zone fed from the pial arterial plexus (vasa corona). Due to increasing peripheral collateral supply, a conical longitudinal infarction area finally remains, confined within the vulnerable central grey matter.¹ Infarction in the middle or lower cervical cord may be associated with pain in the neck, shoulders, and arms, followed by tetraplegia, dissociated sensory loss, and bladder and bowel dysfunction.² With occlusion of one of the two branches of the central (sulcal) artery, an incomplete Brown-Séquard syndrome results, with ipsilateral paresis and a contralateral dissociated sensory disturbance.^{3,4}

The F-wave is a potential of variable form and of small, unstable amplitude, following with some delay the compound muscle action potential (CMAP) in response to electrical stimulation of the peripheral nerve.^{5,6} Even in healthy subjects not every stimulus elicits an F-response;⁷ its occurrence depends on the functional state of the first and second motor neuron.^{8,9}

In our case the ASAS developed like a "progressive stroke" so that we could observe its progress by repeated electroneurophysiological and MRI investigation.

Case report

A 67 year old woman with no history of vascular disease was admitted to hospital with paralysis of the left hand and intermittent instability of gait, approximately 10 hours after onset of symptoms. Neurological

examination on 20 October 1988 showed flaccid paralysis of the left intrinsic hand muscles. Moreover, extension and flexion of fingers and hand were not possible. Hypoalgesia was detected on the back of the right hand and on the palm of the left hand. Deep tendon reflexes in the upper extremities and both patellar reflexes were intact, but the Achilles tendon reflexes were doubtful. There was no Babinski sign and no significant disturbance of gait.

Motor nerve conduction studies at admission showed normal findings in the median and ulnar nerves, apart from an increased threshold of excitability of the motor axons in these nerves and missing F-waves in the left thenar and hypothenar muscles on 50 consecutive supramaximal stimuli. The low sensory nerve action potentials (sNAP) in the median, ulnar and sural nerve indicated a distal sensory neuropathy, particularly considering the SSEP and H-reflex findings (table, part a).

Four days later further paresis appeared in the right hand and in both legs. At this time the biceps brachii and supine jerks remained intact, triceps, patellar and ankle jerks were abolished. She could not walk and had urinary retention.

Nerve conduction studies on the 26 October showed severe reduction of the CMAP in the left thenar on stimulation of the median nerve and extreme reduction of CMAP in the left hypothenar on stimulation of the ulnar nerve. There were no F-waves on the right hand side, despite normal motor conduction velocities and CMAPs in the median and ulnar nerves. Neither voluntary nor spontaneous activity in the left and right thenar and hypothenar muscles. The H-reflex was abolished, the occurrence of F-waves on peroneal nerve stimulation was reduced, both due to acute involvement of the corticospinal tract at the level of the cervical cord (table, part b).

Towards the end of the third week of treatment a slight improvement occurred and the condition stabilised. At this point severe bilateral atrophy of the intrinsic hand muscles was apparent; less severe atrophy had developed in the forearms and the right peroneal muscles. The biceps brachii and supine reflexes were normal, although the triceps reflex were absent. Patellar reflexes were normal although the ankle jerks were

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Neurophysiological findings

Nerve	Median		Ulnar		Peroneal		Tibial	
	L	R	L	R	L	R	L	R
a) 20 October 1988 (paresis of left intrinsic hand muscles)								
mNCV (m/s)	50	—	50	—	51	46	—	47
CMAP (mV)	6	—	10	—	10	13	—	22
sNCV (m/s)	43*	—	37*	—	—	—	—	46**
sNAP (µV)	8*	—	3*	—	—	—	—	1**
F-wave								
occurrence (%)	0	70	0	60	60	60	—	—
latency (ms)	0	28	0	29	53	52	—	—
H-reflex								
amplitude (µV)	—	—	—	—	—	—	500	300
latency (ms)	—	—	—	—	—	—	35	35
b) 26 October 1988 (development of tetraparesis)								
mNCV (m/s)	45	56	60	49	54	52	—	47
CMAP (mV)	1.5	7	0.1	9	10	11	—	15
sNCV (m/s)	41*	—	—	—	—	—	—	46**
sNAP (µV)	8*	—	—	—	—	—	—	1**
F-wave								
occurrence (%)	0	0	0	0	20	10	—	—
latency (ms)	0	0	0	0	52	52	—	—
H-reflex								
amplitude (µV)	—	—	—	—	—	—	0	0
latency (ms)	—	—	—	—	—	—	—	—
c) 29 November 1988 (after stabilisation of the condition)								
mNCV (m/s)	0	0	0	0	49	48	—	—
CMAP (mV)	0	0	0	0	6	1.6	—	—
sNCV (m/s)	43*	42*	37*	41*	—	—	—	—
sNAP (µV)	8*	3*	3*	3*	—	—	—	—
F-wave								
occurrence (%)	0	0	0	0	60	60	—	—
latency (ms)	0	0	0	0	53	52	—	—
H-reflex								
amplitude (µV)	—	—	—	—	—	—	—	—
latency (ms)	—	—	—	—	—	—	—	—

L = left.
R = right.
— = not examined.
0 = no response.
CMAP = compound muscle action potential.
sNAP = sensory nerve action potential.
*finger-wrist (stimulation ring electrodes, registration surface electrodes).
**stimulation sural nerve at the ankle using surface electrode; registration tibial nerve in popliteal fossa using unipolar needle electrodes.

diminished. Both were more pronounced on the right. There was severe weakness of the right and less severe weakness of the left leg, and she could not walk. There were hypoalgetic and hypaesthetic areas on the trunk and on the upper extremities but bladder function was restored.

The last nerve conduction studies were carried out in the sixth week after admission when the condition had stabilised. No CMAP could be evoked in either thenar or hypothenar on both sides. The orthodromic sensory nerve conduction studies of the median and ulnar nerves were unchanged. In the first dorsal interosseus and abductor digiti minimi muscles widespread pathological spontaneous

Figure 2 Axial view of the cervical spinal cord in T2 weighted MRI; ventrally an area of increased signal intensity is visible (arrow).

activity was detected, whereas voluntary activity was absent. Examination of the lower extremities showed normal F-response occurrence and amplitude in both the extensor digitorum brevis muscles, in spite of a severe reduction of the CMAP in these muscles (decreased M/F ratio, M = CMAP), indicating an increased excitability of the second motor neuron (table, part c). There were no signs of denervation or reinnervation in the severely atrophic right anterior tibial muscle (atrophy was due to muscle inactivity because of the central paresis).

The SSEP on 2 December (six weeks after admission) showed normal amplitude and latency of the brachial plexus and cortex response on median nerve stimulation at the wrist on both sides. There was reduced amplitude of the lumbar response on left tibial nerve stimulation, and no lumbar response on right tibial nerve stimulation. Latency of cortical response on the right tibial nerve stimulation was longer than on the left; latencies of the cortical response on both sides were still normal. These findings correlated with the distal sensory neuropathy mentioned above.

MRI of the skull and the cervical spinal cord one day after development of tetraparesis on 25 October 1988 showed no pathological findings. On two further MRIs (14 November and 13 December 1988) an area of high signal intensity was demonstrated ventrally at C5-T1 level in the T2-weighted images (fig 1 and 2).

Cerebrospinal fluid was normal on three occasions (21 and 26 October, 11 November 1988).

Discussion

In our case the ASAS developed atypically during five days, similar to a “progressive stroke”. The classic dissociated sensory disturbance below the level of the lesion was absent. We could find areas of hypalgesia in both upper limbs and trunk, whereas pain sensation in the largest area of the trunk and in the legs was preserved. This can be explained by the destruction of the anterior white commissure, and the sparing of the marginal zone of the lateral columns,¹ which contain the

Figure 1 Median sagittal view of the spinal cord in T2 weighted MRI; ventrally an area of increased signal intensity extending from C5 to T1 is distinguishable (arrows).

spinothalamic tracts (fig 2). Initial phenomena of sensory irritation presented in the form of paraesthesiae rather than pain.

Inflammatory signs indicating transverse myelitis were absent in three consecutive CSF examinations. Consequently, the MRI signals at C5-T1 were to be attributed to an ischaemic infarction. This conclusion is supported by the fact that the area of high signal intensity remains strictly confined to the distribution of the anterior spinal artery (fig 1 and 2).

Controversial reports about the ability of MRI to reveal ischaemic infarctions in the spinal cord have been recently published.^{10,11} Markusse *et al* reported a case of ASAS in which MRI failed to show any abnormality of the spinal cord immediately and three months after the onset of ASAS. Successful demonstration of an infarction by MRI seems to depend on the time of its application, as shown in our case.

The F-response is particularly easily recorded from the intrinsic hand and foot muscles.⁵ The frequency of F-responses in ulnar nerve varies between 50–93% of stimuli in healthy subjects, indicating a relatively high probability of an F-response being produced with each stimulus.⁷ It may be assumed that the F-response results from a recurrent discharge in motor neurons following their antidromic activation.^{6,12} Thus F waves, as well as somatosensory and motor evoked potentials and the H-Reflex, represent a simple electrophysiological measure for assessing the proximal segment of a nerve.¹³

In ASAS, F-waves are affected at a very early stage as our case shows. We could demonstrate a loss of F-waves approximately 10 hours after the onset of paralysis of the hand. It must be assumed, however, that the state of inexcitability of the anterior horn cells begins already simultaneously with the ischaemic attack.

A loss of F-waves was also observed in the acute phase of cerebrovascular disturbances.⁹ After development of spasticity the F-waves are more persistent.⁸ Small infarctions in the motor cortex cause monoparesis with loss of F-responses. Such infarctions are more frequent than occlusions of a sulcal artery and can already be demonstrated within hours or days in cranial MRI or CT.

Hysteric monoparesis may also occur acutely; the F-response is normal in this condition, as we have seen in several cases.

In ASAS the ischaemic process involves neither the spinal ganglion nor the dorsal column, thus abnormal sensory nerve conduc-

tion studies and SSEP findings are absent.¹⁴ Apart from a rapid increase of the threshold of excitability of the peripheral motor axon, motor nerve conduction studies remain initially normal, until Wallerian degeneration has reached the proximal stimulation point. Then a considerable reduction of CMAP becomes visible. The Wallerian degeneration of thin nerve fibres starts as early as 25 hours after the neuronal lesion and progresses at the speed of 25 cm per day.¹⁵ Needle EMG shows pathological spontaneous activity at later stages, beginning in paraspinal muscles.¹⁶

Although nonspecific, the loss of F-responses is the unique early objective finding of ASAS of the lower cervical cord. This finding can have a diagnostic value especially in cases with an atypical course, as in the one presented. The diagnosis of an anterior spinal artery syndrome can be confirmed nowadays by MRI, whereas in the past only necropsy confirmation was possible.

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