SHORT REPORT

Ischaemic myelopathy associated with cocaine: clinical, neurophysiological, and neuroradiological features

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Abstract

Two patients with spinal infarction and one patient with the previously unreported complication of spinal transient ischaemic attack associated with cocaine misuse are reported. Spinal MRI documented an infarction in the territory of the anterior spinal artery in the first two patients and was completely normal in the patient with a transient ischaemic attack. Motor evoked potentials were abnormal in all three patients.

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Numerous well documented cases of cerebrovascular diseases associated with cocaine misuse have been reported¹⁻⁹ and, to the best of our knowledge, only three cases⁴⁻⁹ of ischaemic myelopathy induced by cocaine and no cases of spinal transient ischaemic attack have been previously described. We report clinical and neuroradiological findings together with a motor evoked potential (MEP) study in three patients with ischaemic myelopathy induced by cocaine, two with ischaemic infarct and the third with the previously unreported complication of spinal transient ischaemic attack.

Patients and methods

METHODS

MRI

Brain and spinal MRI was performed on a superconducting 0.5 tesla unit using multiplanar spin echo and fast spin echo to obtain T1 and T2 weighted images.

Motor evoked potentials

Central motor conduction was evaluated using magnetic stimulation of the motor cortex and spine through a 120 mm diameter circular coil. The stimulus intensity was 100% of the maximum output for cortical stimulation and 60% for radicular stimulation. To obtain preferential activation of each hemisphere, a clockwise

inducing current flow, as viewed from above, was used for the right motor cortex and an anticlockwise flow for the left motor cortex.¹⁰ Cortical stimulation was performed during voluntary contraction of about 20% of maximum voluntary contraction of the tested muscle. In patient 1, who presented with a severe tetraparesis, MEPs were recorded during maximum voluntary contraction from thenar and tibialis anterior muscles by surface electrodes and amplified with filter settings of 2 Hz and 5 kHz. The onset latencies of the MEPs obtained from cortical and paravertebral stimulation were measured. The central motor conduction time (CMCT) for thenar and tibialis anterior muscles was evaluated by subtracting the latency after cervical or lumbar stimulation from the latency after cortical stimulation. Control values were obtained after bilateral studies on 25 healthy subjects (13 men; mean age 43.7 (SD 18) years). Normal limits were defined by the mean +3 SD of the control values.

PATIENTS

Patient 1

A 22 year old man complained of cervical pain immediately after snorting cocaine and developed tetraplegia; he was urgently intubated and ventilated for the onset of an acute respiratory failure. Blood pressure was 140/90 mm Hg. A chest radiograph, ECG, EEG, and blood tests were normal. Neurological examination performed on the same day disclosed flaccid tetraplegia and dissociated sensory loss below the C2 level. Deep tendon reflexes in upper and lower limbs, plantar reflexes, and superficial abdominal reflexes were absent. Two months later neurological examination disclosed severe tetraparesis with pronounced spasticity, enhanced tendon reflexes in the upper and lower limbs, and bilateral Babinski's sign. Loss of pain and temperature sensation persisted below the C2 level. The patient showed only a minimal recovery of limb strength and of respiratory function during the subsequent 10 months and remained stable thereafter with severe tetraparesis.

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MRI obtained 48 hours after the onset of the illness showed a moderate swelling of the cervical cord on T1 weighted images with an abnormal increase in signal intensity extending from C2 to C7 on T2 weighted images. Three months later the MRI was repeated and showed abnormal linear hyperintensities in the spinal cord involving the anterior horns of the central grey matter extending from C2 to C7, thus suggesting an ischaemic lesion in the territory of the anterior spinal artery.

MEPs recorded two months after the onset of the illness documented an abnormality of CMCT for the upper and lower limbs. The latencies of the responses evoked through cervical and lumbar stimulation were within normal limits and excluded any peripheral slowing.

Patient 2

A 37 year old man presented with slight weakness of the arms and legs immediately after injecting himself with cocaine. The upper limbs recovered fully the next day. The patient could walk without support but a slight lower limb deficit remained. The patient also noted a constant urge to urinate. The patient was admitted to our department only one year later, when neurological examination disclosed spastic gait and enhanced tendon reflexes in the lower limbs with bilateral Babinski's sign. Strength and deep tendon reflexes were normal in the upper limbs. Position, vibration, temperature, and pain sensations were normal in the upper and lower limbs. Bladder control was normal.

MRI obtained one year after the onset of the illness disclosed an area of hyperintensity (gliosis) in the anterior horns of the central grey matter at the C4-C5 level corresponding to the lower territory of the anterior spinal artery.

MEPs obtained one year after the onset of the illness documented an abnormality of CMCT for the upper and lower limbs. The latencies of the responses evoked through cervical and lumbar stimulation were within normal limits and excluded any peripheral slowing.

Patient 3

A 33 year old man with a history of intravenous heroin and cocaine misuse developed tremor, pain, and weakness in the lower limbs a few minutes after an intravenous injection of cocaine. Blood pressure was 140/90 mm Hg. A chest radiograph, ECG, EEG, and blood tests were normal.

Neurological examination two hours after the onset of symptoms disclosed decreased pain and temperature sensation below T12 and mild symmetric weakness of the legs with absent plantar response. Twenty four hours later the clinical picture was normal.

MRI of patient 3 was normal both 24 and 72 hours after the onset of the symptoms.

MEPs obtained two hours after the onset of the symptoms documented an abnormality of CMCT for lower limbs alone (figure). The latencies of the responses evoked through cervical and lumbar stimulation were within

Patient 3. MEPs evoked by spinal and cortical stimulation recorded two hours (A) and 24 hours (B) after the onset of symptoms (spinal responses are not illustrated for the second test as the response latencies were unchanged). Values for central motor conduction time are indicated. The top traces show right thenar recordings; MEP latency after spinal stimulation and central motor conduction time are normal. The lower traces show right tibialis anterior recordings; MEP latency after spinal stimulation is normal, central motor conduction is prolonged on the first study (A) whereas the second study is completely normal.

normal limits and excluded any peripheral slowing. The patient was studied again 24 hours after the onset of symptoms, and at this time the neurophysiological findings were normal (figure).

Discussion

This study reports the first case of spinal cord transient ischaemic attack related to cocaine misuse together with two cases of spinal ischaemic infarct. The clinical findings of the patient with the transient attack were compatible with a hypoperfusion in the territory of the artery of Adamkiewicz, whereas the clinical findings in the other two patients were compatible with an ischaemic lesion in the territory of the anterior spinal artery. The route of cocaine administration was intravenous in two patients

and intranasal in the third. This suggests that, as for cerebral ischaemia complicating cocaine misuse, spinal ischaemia may follow any route of cocaine administration.

MRI showed the infarction in the two patients with the anterior spinal artery syndrome whereas it was normal in the patient with a transient ischaemic attack.

One salient finding in our study is the high sensitivity of MEPs in detecting spinal cord ischaemia determined by cocaine misuse. Because MRI in spinal cord stroke can be normal in the face of profound neurological deficits, especially if performed acutely,^{4 9 11} MEP study may be useful in showing spinal cord involvement, even in cases of spinal transitory ischaemic attack, and may help to localise the site of central motor pathway dysfunction.

As there is nothing distinctive from a clinical, neuroradiological, or neurophysiological point of view in ischaemic myelopathy induced by cocaine compared with most common forms of ischaemic myelopathy, this aetiology should be

considered in the differential diagnosis of any acute non-traumatic myelopathy, especially in young patients.

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