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Spinal Cord Seizures: A Possible Cause of Isolated Myoclonic Activity in Traumatic Spinal Cord Injury: Case Report

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Summary

Spinal cord seizures are infrequently reported. They have been associated with intravenous dye placement, transverse myelitis and multiple sclerosis, but never with traumatic spinal cord injury (SCI). We report the case of a 48-year-old SCI male with complete C6 quadriplegia, and apparent spinal cord seizures. These seizures were characterised by myoclonus simplex activity involving the upper extremities only. The lower extremities were spared. The patient was conscious throughout the myoclonic activity and an electroencephalogram of the brain obtained during an event revealed no cortical epiliptiform activity. The seizures lasted approximately 30 seconds to a few minutes, and an acute increase in blood pressure and a decrease in pulse generally occurred 30 to 60 seconds prior to the event. Previously reported spinal cord seizures in multiple sclerosis were frequently treated with carbamazepine. In this case successful treatment was with diazepam. Spinal cord seizures may present in those with traumatic SCI. Benzodiazepines may be useful in the treatment of spinal cord seizures.
Key words: *Spinal cord injury; Seizures; Myoclonus; Benzodiazepines; Autonomic dysreflexia; Spasticity.*

Spinal cord seizures are an infrequently reported occurrence. Most reported cases have been associated with multiple sclerosis (Cherrick and Ellenberg, 1986; Espir and Millac, 1970; Ekbom *et al.*, 1968; Matthews, 1975; Twomey and Espir, 1980; Watson and Chiu, 1979). One case was documented with a spinal cord arteriovenous malformation and intravenous contrast dye administration. Another case was felt to be due to an ischaemic spinal cord event (Ekbom *et al.*, 1968). No cases have so far been reported to be associated with traumatic spinal cord injury (SCI). We describe the unique presentation of a case of apparent spinal cord seizures in a patient with traumatic SCI.

History

The patient is a 48-year-old male who suffered a C6 SCI in a motor vehicle accident. At the time of admission he was noted to have a C7 fracture with a bony fragment in the spinal

canal. Upon admission to rehabilitation he was neurologically graded as a C6 quadriplegic Frankel class A (Frankel *et al.*, 1969). The patient's clinical course was complicated by episodes of pneumonia. Nine months after his injury the patient developed periodic episodes of involuntary myoclonus of the upper extremities. The patient had myoclonus simplex activity which was arrhythmic and asymmetrical involving the elbow flexors and the shoulder girdle flexors. It involved the upper extremities bilaterally but did not involve the lower extremities. On physical examination the patient was afebrile with no significant neurological changes in his C6 quadriplegia. Each episode lasted from 2 to 5 minutes and consisted of multiple painful myoclonic jerks. The movements occurred in the upper extremities bilaterally, but did not involve the lower extremities. No alterations in the level of consciousness were noted during the episodes. Medications at the time included baclofen 20 mg p.o. q.i.d. Arterial blood gases drawn 2 days before the onset of these episodes revealed a low PCO2 of 4·53 kPa with a normal PO_2 of 8·66 kPa and a PH of 7·42 units. Blood chemistry studies revealed the following values: Na 141 mmole/L, K 4·0 mmole/L, Ca 2·37 mmole/L, PO_4 1·36 mmole/L, Mg 0·70 mmole/L and Glu 4·16 mmole/L. Venous CO2 was chronically low between 21 to 24 mmole/L. An electroencephalogram obtained during one of these episodes was normal.

Approximately 30 seconds to 1 minute before these myoclonic episodes the patient's blood pressure would rise from a baseline of 90/60 to 140/90 mHg and his pulse rate would drop from the mid 90's to the mid 60's. These episodes became more frequent. Consequently, during one episode 2·5 mg of diazepam was given intravenously. This abolished all myoclonic activity but the patient became flaccid and had a 30 second episode of apnoea. We then started him on oral diazepam and increased it slowly to 5 mg p.o. t.i.d. at which time these episodes ceased. When diazepam was weaned the myoclonic activity returned. Diazepam reinstituted at the previous dosage with complete resolution of myoclonic activity once the dosage reached 5 mg p.o. t.i.d.

Discussion

Paroxysmal neurological disturbances have often been described in multiple sclerosis (Cherrick and Ellenberg, 1986; Espir and Millac, 1970; Ekbom *et al.*, 1968; Matthews, 1975; Twomey and Espir, 1980; Watson and Chiu, 1979). Many authors have felt these to be seizures of spinal cord origin (Cherrick and Ellenberg, 1986; Espir and Millac, 1970; Ekbom *et al.*, 1968; Matthews, 1975; Twomey and Espir, 1980; Watson and Chiu, 1979). These spinal cord seizures have only rarely been described in conditions other than mutiple sclerosis (Uhl *et al.*, 1981; Castaigne *et al.*, 1968). To our knowledge they have never been associated with traumatically induced SCI. Findings in animals suggest that the spinal circuit possesses the ability to acquire new neuronal patterns which can lead to spinal cord seizures (Yu *et al.*, 1984).

In most case reports the neurological activity was localised clinically, as it was in our patient (Espir and Millac, 1970; Ekbom *et al.*, 1968; Matthews, 1975; Twomey and Espir, 1980; Watson and Chiu, 1979; Tokokura *et al.*, 1976; Uhl *et al.*, 1981; Yu *et al.*, 1984; Castaigne *et al.*, 1968). Generally the myoclonus is confined to one or two limbs. These seizures may be associated with painful sensations (Watson, 1979). The patient had a normal electroencephalogram throughout one episode of myoclonic activity. This has been previously reported in patients with spinal cord seizures attributed to multiple sclerosis (Cherrick and Ellenberg, 1986; Matthews, 1975).

This patient had possible contributing factors for developing spinal seizure activity. First he had a low arterial PCO2 which may provoke seizures by contributing to vasoconstriction and reducing the blood flow to the brain and spinal cord

(Tokokura *et al.*, 1976). The patient had mild hypomagnesaemia; this has been implicated in the production of cerebral seizures, but usually with rapid changes produced by such conditions as severe fluid losses, not present in this patient (Erickson, 1980). Hypomagnesaemia has been described in SCI (Broughton, 1972). It is often transient and insufficient to produce symptoms. Finally, our patient developed signs consistent with autonomic dysreflexia prior to the seizures. Yarkony, *et al.*, (1986) have documented autonomic dysreflexia as a cause of cortical seizure activity in SCI injured above T-6. It follows that autonomic dysreflexia may cause spinal cord seizures as well as cortical seizures. Other causes of spinal cord seizures have been reported, such as hyperventilation, tactile stimulation, truncal movement, exercise, and hot baths (Ekbom *et al.*, 1968). These types of stimulation have also been implicated in causing autonomic dysreflexia (Erickson, 1980). Whether the autonomic dysreflexia causes spinal seizures or was a part of the seizure activity is open to conjecture.

Carbamazepine has been the drug of choice for treatment of spinal cord seizures associated with multiple sclerosis. Up to now benzodiazepines have not been documented in the treatment of spinal cord seizures, but successful treatment in our case was with diazepam. Diazepam is a documented effective antiseizure medication (Browne and Penry, 1973). It is also an effective antispasticity medication in SCI patients (Young and Delwaide, 1981). Diazepam was chosen because we felt that spasticity may have been contributing to episodes of autonomic dysreflexia which were related to the myoclonic activity. With diazepam treatment they did not recur. Doses were increased gradually so that cognitive and pulmonary side effects could be minimised.

Because diazepam has been frequently used to control spasticity in traumatic SCI, many unrecognised spinal cord seizures may have been diagnosed as spasticity and been treated successfully with diazepam. Physicians treating traumatically induced SCI should recognise that these patients are at risk from the development of spinal cord seizures.

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