RECURRENT SPINAL SHOCK LIKE SYNDROME A possible convulsive disorder?

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ABSTRACT

The case of a 7-month-old boy who had 5 episodes mimicking a spinal shock and followed up over 4 years is reported. Taking into consideration the normal neurological development the effect of anticonvulsive drugs and after excluding other hypothesis a convulsive disorder is advanced as the most plausible explanation.

INTRODUCTION

Injuries to the spine occur infrequently in infants. The gravity of these lesions makes it mandatory to suspect spinal trauma in the presence of acute paralysis. We report here a case of what we think a convulsive disorder mimicking a spinal shock syndrome in a seven months old male.

CASE REPORT

A 7-month-old male was admitted to the emergency room on January 86 for severe respiratory distress of two hours duration.

On examination he had severe retraction, irregular breathing and absence of diaphragmatic movement. He was cyanosed, drowsy, irritable and afebrile. Blood pressure was 105/60, Pulse 140 and the chest was full of crepitations.
Neurological examination showed complete quadriplegia, severe axial hypotonia and complete areflexia. No sensory level could be determined but minimal response to painful stimuli was obtained on the face. Spontaneous head movement was possible and pupils were pinpoint without reaction to light.

The patient was intubated immediately and transferred to the intensive care unit for respiratory assistance. Till the day of admission the baby was healthy, developing normally and exclusively breast-fed. There was no history of perinatal insult, any significant disease or hospitalization before.

Symptoms started 2-3 hours earlier when the baby was playing with his 8 years old sister. The family was not sure that he sustained a trauma and there was no external sign indicating a possible falling down.
After intubation the patient was given 0.25mg/kg manitol, 4mg dexamethazone and considered as a possible case of head and spinal injury.
Skull and spinal X-rays were normal, as routine investigations including complete blood count, serum electrolytes, and liver enzymes.

Eleven hours later, the patient was fully conscious, pupils were pinpoint but reactive to light, he could move both 4 limbs but still hypotonic. He remained afebrile and was extubated few hours later. He was kept in pediatric department for three days and discharged in good condition with the diagnosis of spinal trauma without fracture.

Less than 24 hours after discharge the patient was readmitted with the same picture. There was no notion of trauma, insect bite or intoxication. He was afebrile, had severe respiratory acidosis and hypercapnia. Respiratory assistance was needed for 24 hours. Routine investigations including serum calcium, phosphorus and magnesium were normal, no toxics were detected in urine. A brain C.T. Scan showing C1-C2 was also normal. The patient recovered completely after 24 hours. Dynamic cervical spine X-rays were normal. He was discharged with a hard collar cast with the diagnosis of recurrent spinal cord injury without radiological evidence.

Six days later the patient was admitted to pediatric department with the same symptoms. Fundi were normal. Lumbar puncture show normal CSF. Repeated potassium levels were normal as other routine laboratory

investigations. Hospital course was identical to previous episodes.
The patient was kept for one week for observation in hospital and no abnormality was detected.
A possible convulsive disorder was evoked but a standard EEG was considered normal. No treatment was given on discharge.
One month after the first attack the patient was admitted with identical symptoms. The same hospital course was noted. Blood ketons were negative, serum ammonia, lactic acid, liver enzymes, and repeated potassium levels were again normal.
Since the first attack four weeks earlier no mental regression was noted and the patient continued to thrive well. At 8 months age weight was 8kg and HC 43 cm. He was discharged on valproic acid 30mg/kg/day and the collar was taken off.

Six days after starting progressively valproic acid the patient was admitted with respiratory distress and quadriplegia. The duration of symptoms was shorter (6 hours). He was intubated to maintain airway but no respiratory assistance was needed. The mother had noted uprolling eye movement at the beginning of respiratory symptoms. Valproic acid levels were not available and clonazepam was added. The patient was discharged 24 hours later in good condition. On follow up one episode of abnormal eye movement followed by hypotonia was noted when medication were not given (accidentally) for three days, otherwise the patient remained symptom free. He started to walk at 13 months and other developmental milestones were normal. Clonazepam was stopped at 12 months of age and valproic acid at 20 when the development of the child was considered normal.
At the age of 4 years his physical and neurological examinations were normal and no sequella was clinically detectable.

DISCUSSION

Spinal column injury in children is uncommon (1). Spinal cord injury without radiological abnormalities is a well described entity in pediatric age (2, 3). The notion of trauma is not always easy to verify.

The syndrome of spinal shock consists of transient loss of all motor, sensory and segmental reflex functions below the level of injury (4).

The presence of spinal shock can mimic complete cord trans-section, so the true deficit and prognosis can not be known until spinal shock resolves.

This usually occurs within 24 hours of injury (4).
The clinical presentation of our patient was suggestive of spinal shock caused by a high cervical injury. As the notion of trauma was not clearly defined, other causes of non-traumatic paralysis were sought. Organophosphorous intoxication may cause myosis, severe respiratory distress and a variety of central nervous symptoms (5). Others causes like lathyrism, Guillain Barre Syndrome, poliomyelitis, acute hemorrhage due to vascular malformation (6), scorpion or snake bites (7) were excluded by history and clinical progression. Possible metabolic diseases including periodic paralysis were unlikely (8). Recurrent spinal cord injury without a clearly defined traumatic episode was reported in a young child (3).

This patient had a total of five episodes mimicking spinal shock, 2 of them occurred in spite of immobilization by a hard collar cast.
The absence of trauma in 4 episodes and the non-recurrence after anticonvulsant therapy made us doubtful to consider these episodes as merely post traumatic.
The standard EEG could be normal in convulsive disorders. More prolonged registration could be helpful but they were not available.
Finally the non-recurrence of these episodes under anticonvulsant, the normal neurological development, the absence of a clearly defined trauma make the hypothesis of a benign convulsive disorder mimicking a spinal shock plausible. A post-ictal symptomatology of an uncommon convulsive disorder mimicking a spinal shock syndrome is another theoretical possibility.

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