POST TRAUMATIC PARKINSONIAN SYNDROME: A CASE REPORT

PRATAP CHAND R.

SUMMARY

Parkinsonian syndrome attributed to cranio-cerebral trauma is rare. Two young adult males developed Parkinsonism following severe head injury. The clinical features and response to therapy are described.

INTRODUCTION

Parkinsonian syndrome secondary to cranio-cerebral trauma is a controversial subject. Of the 163 reports of traumatic Parkinsonism which includes over 200 individual cases, only 2% have had sufficient evidence to implicate trauma as the etiological factor.[1] Clinical criteria for the diagnosis of this rare entity include cerebral trauma of sufficient severity to produce mid-brain damage and a definite interval between the trauma and onset of Parkinsonism.[2] Two cases that fit the above criteria are described.

CASE REPORT

Case I

In 1977, at the age of 20 years, a Malay male suffered a head injury in a motor vehicle accident and was admitted to a local hospital in coma. According to his parents, it took about 30 days for recovery of consciousness; at this time, they noticed that he had weakness of the left half of his body and loss of memory for a period of three weeks before and 30 days after the accident.

He left hospital and two months later he noticed the onset of involuntary movements in the left upper limb, maximum at rest and absent in sleep. This increased in intensity over the next two months and then remained unchanged. The left-sided weakness gradually improved over the next two years. In view of the persistent involuntary movements, he presented to the university's Neurology Clinic in March 1984. There was no history of similar illness in his family nor of exposure to opiates, phenothiazines, butyrophenones and dopamine depletors such as reserpine and tetrabenazine.

Physical examination in March 1984 revealed normal higher mental functions and cranial nerves. Motor power was 5/5 in all groups. He had brady-kinesis, cogwheel rigidity and a coarse, biplanar, distal, resting tremor in his left upper limb which disappeared on movement. Deep tendon reflexes were bilaterally exaggerated in all extremities, more on the left than the right side and glabellar tap sign was positive. Touch, pain and temperature sensations were impaired by 40% on the left half of the body. He had no other neurological deficits.

Pratap Chand R., MBBS, DM (Neurology)
Consultant Neurologist and Lecturer
Department of Medicine
Hospital Universiti, Universiti Sains Malaysia
16150 Kubang Kerian
Kelantan, Malaysia

Hemogram, urinalysis, liver function tests (LFT), blood sugar, blood urea and skull X-ray were normal. Blood and cerebrospinal fluid (CSF) VDRL tests were non reactive. Serum copper was 17 μmol/l, urinary copper excretion was 0.2 μmol/24 hours, serum ceruloplasmin was 2 μmol/l and slit lamp examination showed no evidence of Kayser Fleischer (KF) rings. He was treated with trihexyphenidyl 6 mg daily. He showed some improvement on this and L-Dopa was added to the regimen at a dose of 500mg twice daily. He could not tolerate higher doses of L-Dopa due to side effects of nausea, vomiting, postural hypotension and disturbed sleep. Two months later there was a clear improvement in both bradykinesis and cogwheel rigidity. The tremor though much reduced in intensity was still present.

Case 2

This 20-year-old Chinese male had been admitted to the General Hospital, Penang in an unconscious state following a head injury sustained in a motor vehicle accident in November 1981.

His case notes revealed that at admission he had been in a "state of altered consciousness, responding to pain by semipurposive movements". He had a left abducens and left lower motor neuron facial palsies and a crossed right hemiplegia. Skull X-ray had shown a linear fracture of the left parietal bone. With conservative treatment, he made a gradual recovery and regained full consciousness after 40 days. He had retrograde amnesia for a month and post traumatic amnesia for 40 days.

He left hospital and made a gradual partial recovery from the right hemiplegia over the next six months. At this time, he noticed the onset of involuntary movements in his right upper limb. This progressed over the next three months to involve his right lower limb and had remained unchanged since then. There was no history of addiction to opiates, nor of exposure to drugs capable of inducing Parkinsonian features.

He presented to the university's Neurology Clinic in May 1984 for the above complaint. Physical examination at this time revealed mask-like facies and overt depression. Higher mental functions were otherwise normal. He had residual, partial left abducens and left lower motor neuron facial palsies and sensorineural deafness in the left ear. Motor power was grade 4/5 in the right upper and lower limbs. There was cogwheel rigidity, bradykinesis and a biplanar resting tremor involving the tongue, right upper and lower limbs. Deep tendon reflexes were exaggerated in all extremities, more on the right than the left side and the glabellar tap sign was positive. The right plantar reflex showed a positive Babinski response and he had 50% impairment of touch, pain and temperature sensations on the right half of the body. He had no other neurological deficits.

Hemogram, blood sugar, blood urea, LFT and skull X-ray were normal. Blood and CSF VDRL were non reactive. The slit lamp examination showed no KF ring, serum ceruloplasmin was 1.8 μmol/l, serum copper was 16.8 μmol/l and the 24 hour urinary copper excretion was 0.35 μmol. He showed a good response to trihexyphenidyl 6mg/day and was totally free of the tremor, rigidity and bradykinesis one month later.

DISCUSSION

Since the report by Deschamps,[3] there have been 163 communications implicating trauma as an aetiological factor for Parkinsonism. A review of the literature by Grimberg in 1934 pointed out that many were reports in which Parkinsonism seemingly followed peripheral trauma to the extremities.[4] He stated that this syndrome could occur only if the trauma had also produced brain injury. Schwab and England concluded that only 2% of the reported cases merit the title of 'Traumatic Parkinsonism'. These included three cases reported by Lindenberg in whom there were clinical and post-mortem evidence of traumatic lesions in the substantia nigra.[1,5]

The conspicuous absence of Parkinsonism as a sequel to devastating head injuries in World Wars I and II and the fact that it has not so far been reported in patients younger than 40 years, have raised speculations about the existence of this entity.[1,6] In both the cases presented here, the head injury and the subsequent Parkinsonism occured at the age of 20 years. The craniocerebral trauma was sufficiently severe to produce head injury as judged by the period of unconsciousness and the neurological deficits pointed to brain-stem damage. It is also worth noting that both patients had hemiplegia on the same side as the rigidity and tremor, and that the Parkinsonian tremor made its appearance when the hemiplegia was improving. There were intervals between the trauma and the onset of Parkinsonian features of three months and six months in Case 1 and Case 2 respectively. It is difficult to explain the progression of symptoms in traumatic Parkinsonism reported by earlier authors.[2,7] In the present cases, the disease had remained static after an initial progression. This non progression is more in keeping with Parkinsonism of traumatic etiology. The therapeutic response to trihexyphenidyl and L-Dopa is also noteworthy. The above cases seem to fit the clinical criteria for a post traumatic Parkinsonian syndrome and the present report serves to emphasise that the entity though exceedingly rare, exists.

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