Dystonia-parkinsonism syndrome resulting from a bullet injury in the midbrain

Various cerebral lesions such as tumours, infection, and head trauma have been reported as causing parkinsonian syndromes. These lesions were localised in the basal ganglia, thalamus, corpus callosum, cerebral cortex, and midbrain. In the patient reported here the lesion was a bullet injury in the midbrain.

A 38-year-old man attempted suicide with a firearm. The bullet penetrated the medial part of the frontal bone and followed an oblique trajectory posteriorly and upwards to the left and reached the left midbrain (fig 1). The injury was followed by coma with right hemiplegia. Recovery of normal consciousness occurred progressively over 15 days.

His past medical history included a hip fracture at the age of 33 years and a traumatic amputation of the right fifth finger. Two years after the bullet injury an examination showed the following: hemiparesis predominating in his arm with brisk tendon reflexes and Babinski's sign; discrete asymmetry of his face due to central right facial paresis; horizontal diplopia in all directions provoked by a partial lesion of the third left cranial nerve; reduction of light touch, temperature, and pain sensation on the right half of the body, including the face, without impairment of deep sensitivity; resting tremor of 4-5 s each second occurring intermittently as sinusoidal waves lasting minutes to one hour, five to six times each day; intention tremor of the right arm and asynmetry of the right leg; right hemidystonia (fig 2) increasing in the upper limb during tremor. Tremor was spontaneous and activated by mental activity and somatosensory stimulations. Intravenous injection of piribedil, a dopaminergic agonist, led to the almost complete disappearance of tremor and dystonia after five days with functional recovery of walking, his arm being held close to his body with the elbow slightly flexed.

The left parietal somatosensory potential evoked by stimulation of the right median nerve was slowed at the subthalamic level. PET with fluorodopa labelled with fluorine-18 showed considerably reduced uptake in the left striatum, although uptake of lithium labelled with bromine-76 showed no functional change of D2 receptors on either side.

Combined treatment with levodopa, 100 mg/day, and 25 mg benserazide did not have any effect on tremor. Treatment with levodopa 300 mg and benzerazide 75 mg with 120 mg piribedil, however, was successful in eliminating tremor and dystonia without eliciting abnormal movements.

As reconstructed by CT, the trajectory of the bullet ended in the left cerebral peduncle. The symptoms resulting from this lesion included a right hemiparkinsonian syndrome, a kinetic cerebellar syndrome, and dystonia.

Tremor was a parkinsonian resting tremor with a rate of 4-6 Hz, with rhythmic bursts alternating in the agonist and antagonist muscles. This was associated with a decreased uptake of fluorodopa in the left striatum. This tremor has some atypical features compared with classical early parkinsonian tremor; however it persisted during a maintained posture with the right arm outstretched as well as during voluntary movement, and increased slightly when approaching the target point. It was intermittent, occurring in episodes lasting several minutes or hours with a wave-shaped amplitude.

The intermittent tremor was probably caused by a partial insult of striatal dopamine, as shown by PET. Akinessia of the right arm can be considered as a parkinsonian symptom. Right cerebellar kinetic syndrome associated with resting tremor, causing uncoodination and dysmetria, can be attributed to interruption of the dentatothalamic pathway and was more obvious after the tremor had been stopped by treatment.

Dystonia affecting the right hand and foot may have been due to the lesion of the red nucleus. Injuries to the red nucleus and substantia nigra have been correlated with dystonias in some patients. Leenders et al.6 reported that the causal malformation extended from the thalamus to the mesencephalon and included part of the red nucleus. The location of the bullet in our patient probably destroyed part of the red nucleus.

In conclusion, upper midbrain lesions such as those observed in our patient produce a complex syndrome consisting of hemidystonia, probably related to a lesion of the red nucleus, cerebellar kinetic syndrome due to the lesion of the dentatothalamic tract, and parkinsonian syndrome caused by injury to the substantia nigra and characterised by some particular features, particularly persistent tremor during movement and maintained posture. The follow up of this patient is short, but no fluctuations have been observed after three years of treatment with levodopa.

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