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Case Report

Reversible Parkinsonism due to Chronic Subdural Hematoma: A Case Report

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Abstract

Although Chronic Subdural Hematoma (CSDH) is frequent in elderly patients, the CSDH can exceptionally cause a parkinsonism or aggravation of pre-existing parkinsonism. Only 27 cases reversible parkinsonism due to chronic subdural hematoma was reported in the literature.

Disappearance of the extra pyramidal symptoms followed craniotomy and removal of the CSDH suggest a cause-and-effect relation between the haematoma and the clinical symptomatology.

A case of a 62-year-old man with a two weeks history of parkinsonism caused by a CSDH reversible after surgery evacuation of the haematoma is reported.

CSDH is a rare cause of reversible parkinsonism after surgery. CT scan must be recognized in any acute Parkinsonism or any deterioration of pre-existing Parkinson disease to diagnose the Parkinsonism secondary to CSDH.

Keywords: Chronic subdural haematoma; Hemi-Parkinsonism; Surgery; Disappearance

Introduction

Chronic Subdural Hematoma (CSDH) is one of the most common types of traumatic intracranial hematoma, and often occurs in the elderly. Elderly patients with CSDH most commonly present with altered mental status and focal neurological deficit [1-5].

CSDH causing parkinsonism is a very rare event but has been reported in the literature [6-8]. CSDH is a rare cause of reversible secondary parkinsonism after surgery. Bostantjopoulou S [1] in 2009 can found only 27 cases of CSDH causing parkinsonism arising from a mass effect from CSDH. The good outcome after surgical evacuation suggests a cause-and-effect relation between the haematoma and the clinical symptomatology [9-11].

We described a case of reversible hemi-parkinsonism due to CSDH after surgery.

Case Presentation

A 62-year-old Moroccan right-handed man presented with hemiparesis and tremor in right hand extremity. He was healthy and he never had abnormal movements. He denied any medication or toxic abuse. His medical history showed that he has a minor head trauma one month ago. Three weeks ago, he presented headache, nausea and right hand and arm abnormal movement extremity tremor. One week later, he presented some mental deterioration with difficulty in walking and rigidity of his extremity, which was followed by right hemiparesis.

Neurological examination showed an alert and good healthy condition patient, with GCS at 15. Neurological examination showed hemiparesis. There was an evident moderate tremor (less than 2cm excursion) at rest, rigidity and bradykinesia in his right upper member. Tendon reflexes were symmetrically normal, but the right plantar response was in extension. No sensory deficits or cerebellar signs were present. Somatic examination showed no abnormalities. His vision and fundi were normal. The diagnosis was right parkinsonism.

Computed Tomography (CT) showed a chronic SHD on the left side, which markedly compressed the brain. A laboratory workup showed normal haematological and biochemical findings. He was operated on the same day. The haematoma was evacuated and drained via double parietal burr hole under local anaesthesia. The drain was removed on the third postoperative day. Postoperative course was unremarkable with regression of the right hemi-parkinsonism completely disappeared after neurosurgical evacuation of the hematoma without any anti-Parkinson drug with recovery of the hemiparesis. He was discharged from hospital in 7th day. No recurrence was noted with 12 months follow-up.

Discussion

CSDH are more common in the elderly. They may be precipitated by minor trauma, and there is increased incidence in alcoholics, epileptics, and patients receiving anticoagulant treatment. Trauma has been noted as a cause in 73% of subdural haematoma.

CSDH is difficult to diagnose by clinical manifestations only. Nonspecific neurologic symptoms and signs may lead physicians to make other diagnoses. Surgical treatment has been widely accepted as the most effective way to manage CSDH.

Parkinsonism consists of several extrapyramidal signs, characterized by tremor, muscle rigidity, and loss of postural reflexes, which may be caused by degeneration of dopaminergic neurons in the basal ganglia. The differential diagnosis of secondary parkinsonism includes a variety of underlying causes. Tumours sparing the basal

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ganglia or by normal pressure hydrocephalus with compression, distortion or hypoperfusion of the basal ganglia and their circuitry. In the rare case, parkinsonian syndromes may also be associated with CSDH [12-15].

Chronic SDH causing parkinsonism has been reported in the literature [1-6]. This event is very known and well documented in the literature. The hematoma can be bilateral or unilateral. The Parkinsonism can be bilateral or unilateral; homolateral or epsi lateral to the hematoma [1,7-9]. There were a few exceptional cases with hemi-parkinsonism in the ipsilateral side to the lesions (ipsilateral parkinsonism) whereas the most patients with secondary parkinsonism had causative lesions in the contralateral hemisphere [11].

Acute presentation of parkinsonism symptoms has been reported in 24 cases caused by chronic subdural hematomas with 11 cases due to bilateral chronic subdural hematomas [10,11].

Sunada and et al. [4] in 1996 could find only 20 cases of CSDH causing Parkinsonism including his case. The hematoma was bilateral in nine cases.

Bostantjopoulou S, [1] in 2009 reported a Medline search of relevant papers published since 1963 yielded 21 such cases [1]. Seventeen of these patients presented with de novo parkinsonism and 4 with worsening of pre-existing parkinsonism. Bilateral parkinsonism with a rapid onset of rigidity, bradykinesia and gait disturbances was reported in all seven published cases with parkinsonism due to bilateral CSH. Rest tremor was absent in two cases. Other common symptoms were headache and mental status changes. Our patient presented with a new onset, rapidly progressive, right parkinsonism, with tremor accompanied by headache and left pyramidal signs following a minor head trauma one month ago.

Sugie M., [3] reported a CSDH in the right hemisphere caused right-sided Parkinsonism through mechanical compression to the left hemisphere, which was shown in the SPECT image, in his case.

Hsieh TC and et al. [4] reported an incidental finding of SDH Tc-99m-TRODAT-1 SPECT that was originally done for evaluation of his Parkinsonism. He advised that neuroradiological study must be done if any aggravation of symptomatology in the patient followed for chronic Parkinson disease.

The mechanism causing parkinsonism in chronic SDH patients is not well understood. Various suggestions for the mechanism include mechanical pressure on the basal ganglia, either directly from the overlying hematoma or indirectly by torsion or displacement of the brain structure; midbrain compression caused by the uncal herniation through the tentorial notch, decreased numbers of dopaminergic receptors in the striatum due to mechanical pressure; and circulatory disturbances in the basal ganglia caused by displacement and compression of the anterior choroidal artery [1,5,9,13].

Disappearance of the extra pyramidal symptoms followed craniotomy and removal of the CSDH suggest a cause-and-effect relation between the haematoma and the clinical symptomatology [9]. Drug therapy was effective in all patients who were treated medically before surgery.

Conclusion

CSDH is a rare cause of secondary parkinsonism. Surgical treatment of those cases is associated with favourable outcome, without the need for antiparkinsonian medication. CT scan must be recognized in any acute Parkinsonism or any deterioration of pre-existing Parkinson disease to diagnose the Parkinsonism secondary to CSDH.

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