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Chronic Subdural Haematomas and Parkinsonian Syndromes

R. G. Wiest[1], **J. M. Burgunder**[1], and **J. K. Krauss**[2]

[1] Department of Neurology, Inselspital, University of Berne, Berne, Switzerland
[2] Department of Neurosurgery, Inselspital, University of Berne, Berne, Switzerland

Summary

We describe three men with parkinsonian syndromes caused or aggravated by chronic subdural haematomas. A 63-year-old man developed tremor at rest, rigidity and bradykinesia one week after he fell and hit his head. A 70-year-old patient suffering from tardive dyskinesia and drug-induced parkinsonism experienced deterioration of his bradykinetic symptoms over two weeks. There was no history of trauma. The third patient, a 82-year-old man with idiopathic Parkinson's disease had a marked increase of his leftsided parkinsonian symptoms. Again, there was no history of trauma. In all three patients chronic subdural haematomas were demonstrated by computed tomography. Evacuation of the chronic subdural haematoma resulted in disappearance respectively improvement of the movement disorder. Diagnostic evaluations appear to be delayed and initial misinterpretations are frequent. The findings of our report and review of the literature point out that a favourable outcome after appropriate surgical treatment is achieved in most instances.

Keywords: Chronic subdural haematoma; movement disorder; parkinsonism; Parkinson's disease; surgery.

Introduction

The differential diagnosis of secondary parkinsonism includes a variety of underlying causes [13]. Occasionally, parkinsonism is caused by supratentorial tumours sparing the basal ganglia or by normal pressure hydrocephalus with compression, distorsion or hypoperfusion of the basal ganglia and their circuitry [15, 16]. In the rare case, parkinsonian syndromes may also be associated with subdural haematomas. We present three patients with a causal relationship between parkinsonism and chronic subdural haematoma (CSH). In one patient CSH was the cause of levodopa-unresponsive parkinsonism which was completely reversible after evacuation of the haematoma. In two other patients, one with Parkinson's disease (PD) and one with drug-induced parkinsonism, CSH caused marked deterioration of parkinsonian symptoms.

Patients and Methods

The three cases reported here were seen over a period of six years. Patient 1 was treated at the Department of Neurosurgery at the University of Berne, Switzerland, and patients 2 and 3 at the Department of Neurosurgery at the University of Freiburg, Germany. During this six-year period 4 patients out of 250 patients with CSH had CSH-related movement disorders. The fourth patient with CSH-induced dystonia has been reported previously.[4]

Case 1

This 63-year-old man had a history of oligophrenia. Otherwise, he was healthy and he never had abnormal movements. One week after he fell and hit his head, he insidiously developed tremor and rigidity of his extremities which was followed by rigidity of his neck. The movement disorder progressed over the next two weeks and he also displayed bradykinetic symptoms. He was suspected to suffer from idiopathic PD and treatment with levodopa (3×250 mg daily) was initiated. However, no improvement of his symptoms was achieved. He was referred for further evaluation.

On admission to our hospital he had a typical parkinsonian appearance with hypokinetic symptoms and tremor at rest clearly pronounced on the left side. He was demented. He was oriented about his person and vaguely about the situative context, but not to time. He showed little interest in daily routines and communication. Speech was dysarthric and slow. Salivation was increased. Neurological examination showed positive glabellar and peri-orbicular taps and marked neck rigidity. He was hypomimic and his spontaneous movements were reduced. He had no papilloedema. Muscle strength was normal in all limbs. There was severe rigidity of his left arm and leg. Tendon reflexes were more pronounced on his left side. Plantar responses were extensor. He displayed moderate bradykinesia. His stance was unstable and flexed with a tendency to fall to the left. No sensory deficits or cerebellar signs were present. He could write and draw, but was remarkably slow. A cranial computed tomography (CT) scan revealed a rightsided CSH (Fig. 1). There was marked compression of the right hemisphere with severe shift of the midline. He was operated on the same day. The haematoma was evacuated and drained via a parietal burr hole under general anaesthesia. The operation was uneventful and he recovered rapidly. The drain was removed on the first postoperative day. His parkinsonian symptoms were considerably improved and levodopa therapy was stopped. He was discharged on the third postoperative day. Four weeks later the parkinsonian symptoms had disappeared

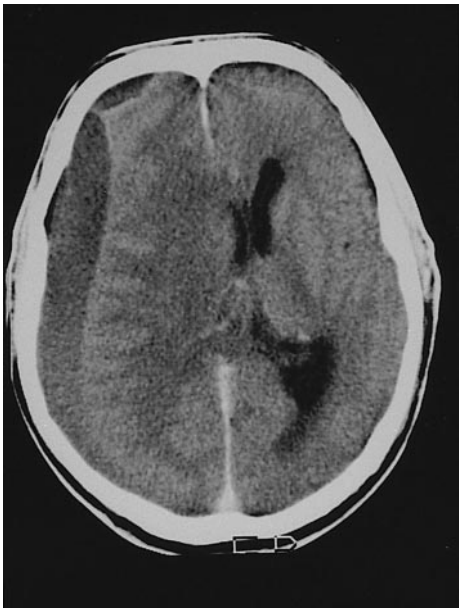

Fig. 1. Axial computed tomography scans of a 63-year old man show a chronic subdural haematoma over the right hemisphere with marked compression and midline shift resulting in predominantely leftsided parkinsonism

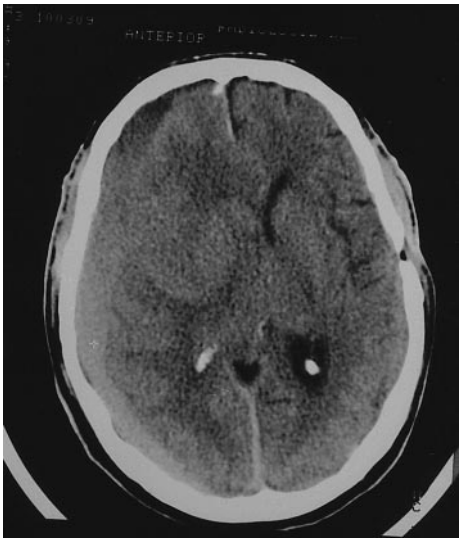

Fig. 2. Axial computed tomography scans of a 82-year-old patient with Parkinson's disease reveal a rightsided subacute/chronic subdural haematoma with marked compression resulting in an increase of parkinsonian symptoms and signs, more pronounced on the left side

completely. His condition remained unchanged during the next six months.

Case 2

This 70-year-old man had a history of chronic alcoholism. At the age of 53, he was hospitalised because of a psychotic episode with paranoid ideation and delusions. Treatment with perphenazine and amitriptyline was started. Under this medical regimen his psychotic symptoms improved, and he was discharged home. He continued to take perphenazine at a dosage of 50 mg daily for several years. During the subsequent course, he developed a parkinsonian syndrome and orofacial dyskinesias. The parkinsonian syndrome was characterized by akinetic symptoms, bilateral rigidity and postural instability. The orofacial dyskinesias were pronounced when talking. Several attempts to withdraw the neuroleptic medication resulted in paranoid delusions and increase of the dyskinesias. At the age of 70 years, two weeks prior to his hospitalization in the Department of Neurosurgery, an increase of his akinetic symptoms was noted. In particular, there was a marked reduction of spontaneous movements and deterioration of his bilateral bradykinesia. Furthermore, gait ignition failure was more pronounced, and there was marked increase of retropulsion. Also, his leftsided rigidity became more severe. Within two days prior to admission, he developed urinary incontinence and he became progressively obtunded. A CT scan of his head demonstrated bilateral CSH resulting in marked compression of both hemispheres. On admission he presented with the clinical picture of akinetic mutism. He underwent emergency evacuation and drainage of the CSHs via bilateral burr hole trephinations. Postoperatively, his neurological symptoms improved within three days. The drains were removed on the fourth postoperative day. Eight days postoperatively, he had marked hypomimia. Rigidity and bradykinesia were more marked on the right side. He was able to ambulate without help, however, he displayed gait ignition failure and postural instability. Furthermore, he demonstrated orofacial dyskinesias, spastic dysphonia and simple vocal tics consisting of throat clearing noises. During the following weeks his condition improved further, and he regained his pre-operative level of function.

Case 3

This 82-year-old man had a ten year history of idiopathic PD. Initially, a 5-Hz tremor at rest of his left arm was noted. In the further clinical course he also developed tremor at rest of his right arm. Subsequently, akinetic symptoms dominated the clinical picture. Because of postural instability with marked propulsion, he suffered from several falls. When he was 81 years old, a left subdural haematoma was evacuated and drained via a burr hole trephination at another hospital. His parkinsonian symptoms were responsive to dopaminergic therapy. He was treated with levodopa (total dosage 375 mg daily), bromocriptin (total dosage 15 mg daily), and biperiden. About a week prior to admission, a marked increase of his leftsided parkinsonian symptoms was noted, and he was barely able to walk. He then became progressively apathetic and had several episodes of vomiting. A CT scan showed a right-sided subacute/chronic subdural haematoma with marked compression of the right hemisphere and a midline shift of more than 15 mm (Fig. 2).

The patient was admitted to the emergency room. Because he was not able to co-operate, he was intubated and sedated. The haematoma was evacuated and drained via two burr holes. Postoperatively, his condition improved rapidly. The drains were removed on the third postoperative day. He fully recovered to the level of his previous condition 10 days after surgery.

Discussion

Subdural haematomas in the rare case may cause various movement disorders such as dystonia [6, 7, 21], chorea [3, 9, 14] and parkinsonism [1, 2, 4, 5, 10–12,

17, 19, 22–29, 31, 32]. Most frequently, movement disorders have been described to be associated with chronic subdural haematomas, while only exceptionally acute or subacute subdural haematomas were reported [31]. Several cases of CSH-related parkinsonism have been reported previously. It remains completely unclear, however, how frequently subdural haematomas may cause movement disorders. Overall, such a relationship appears to be rather rare. According to our experience worsening of pre-existing or new movement disorders occur in about 2% of patients with CSH.

Reviewing the literature we have identified 16 patients with parkinsonism associated with CSH for whom data are available. The data are summarized in Table 1. Seven other patients with parkinsonism secondary to CSH reported in the Japanese, Italian and Portuguese literature [5, 19, 27, 29] have been mentioned by Pau *et al.* and by Sunada *et al.* [22, 28].

Clinical symptoms of CSH become evident within days to weeks after trivial head injury. CSH more commonly occurs in elderly individuals. Clinical symptoms are variable and may mimick other focal lesions, resembling those of stroke, transitory ischaemic attack or tumour, or present as general deterioration with headache, nausea, loss of urinary function or confusion. In the rare cases with parkinsonian syndromes, parkinsonism was characterized most frequently by hypomimia, bradykinesia and tremor (Table 1). In about two thirds of patients a complete parkinsonian syndrome was present. Other signs and symptoms were present in several instances. Frequently, however, patients complained only about mild headache and some cases presented with pure parkinsonism.

In the 16 previous patients with CSH-related parkinsonism (13 men and 3 women; age range: 38 to 83 years, mean: 66 years), unilateral CSH were described in 10 instances and bilateral CSH in 6 instances. Fifteen of the 16 patients underwent surgical treatment by burr hole trephination or craniotomy. All patients who were treated surgically showed rapid improvement of parkinsonism after evacuation of the haematoma. In 11 cases, complete remission of all symptoms was achieved. Four patients had residual symptoms after surgery. Incomplete remissions tended to occur more frequently in patients with bilateral haematomas but were not correlated with the size of the haematoma. One patient reported by Hageman and Horstink, recovered spontaneously without further treatment [11]. In this case, spontaneous resorption of the CSH was demonstrated by repeated CT imaging 3 months later. Improvement of parkinsonism in the postoperative course is seen within days and usually remains stable. Pre-operative levodopa therapy was reported to ameliorate parkinsonism in two previous patients [26, 28], but it was not effective in patient 1 of the present series. Worsening of pre-existent parkinsonism by CSH has received only very little attention. Harding reported on a PD patient with unexplained deterioration of parkinsonian symptoms in whom a unilateral CSH was detected [12]. As described here CSH also caused marked deterioration of drug-induced parkinsonism and PD in two of our patients. In these cases removal of the CSH resulted in recovery of the parkinsonian symptoms to the premorbid level prior to manifestation of the CSH.

The pathomechanisms leading to parkinsonism in CSH remain unclear. Direct mechanical compression of the basal ganglia, vascular disturbances and metabolic changes may be involved. Distortion and compression of the upper mesencephalon and the basal ganglia may lead to a disturbance of the nigrostriatal axis at different levels: the substantia nigra, nigrostriatal pathways and the striatum [16]. The ambiguous response of CSH to treatment with levodopa is most likely related to the site of the functional lesion along the nigrostriatal axis. Patients with compromised nigrostriatal outflow but with intact striatal dopaminergic receptors might respond to pharmacological therapy, whereas those with compression and dysfunction of the striatal dopaminergic receptors might not. Reduction of dopaminergic receptors has been reported in a patient with parkinsonism secondary to an intracranial tumour who did not benefit from levodopa therapy [8]. Vascular disturbance as indicated by impaired perfusion and decreased oxygen extraction and utilisation as a result of local increased tissue pressure onto the basal ganglia may also be relevant [18, 20]. However, it is important to note that only few patients with CSH develop CSH-related parkinsonism. It is unclear, why such movement disorders occur in some patients but not in others, in particular with regard to the fact that morphological imaging studies may be practically identical. Possibly, patients with CSH-related parkinsonism may have preclinical nigrostriatal dysfunction, which can not compensate further insults to the basal ganglia and therefore result in clinical symptoms [11, 31].

Trosch and Ransom documented a rare late effect of

Table 1. *Parkinsonism Associated with Chronic Subdural Haematomas*

Author	Age at onset, sex	CSH	Parkinsonian symptoms	Other symptoms	Responsiveness to levodopa	Outcome after surgery
Samiy [24] 1963	52, M	left	Tre, Rig, Gt (right > left)	none	not given	complete remission
Cohn [4] 1977	62, M	right	Tre, Hyp	headache, confusion, fatigue	not given	complete remission
Sandyk [25] 1982	66, M	left	Tre, Hyp, Bra (left > right)	headache, confusion, fatigue	yes	complete remission
Sandyk and Kahn [26], 1983	38, F	right	Tre, Hyp, Bra, Rig, Gt (left > right)	headache, confusion	not given	complete remission
Glatt *et al.* [10] 1983	74, M	bilateral	Hyp, Bra, Rig, Gt	memory deficit	not given	complete remission
	72, M	bilateral	Tre, Hyp, Bra, Rig, Gt	none	not given	marked improvement
Harding [12] 1984	78*, F	left	Tre, Hyp, Bra, Rig, Gt	confusion, incontinence	decreased response to levodopa in preexistent PD	recovery to premorbid function
Accardi *et al.* [1] 1985	48, M	bilateral	Tre, Bra, Rig	headache, fatigue	not given	complete remission
Peppard *et al.* [23] 1986	73, M	bilateral	Tre, Hyp, Bra, Rig, Gt	none	not given	complete remission (complicated course due to pneumocranium)
	61, M	right	Hyp, Bra, Rig, Gt	none	not given	complete remission
Krul and Wokke [17], 1987	83, M	bilateral	Tre, Hyp, Bra, Rig, Gt	incontinence	not given	partial remission
Pau *et al.* [22] 1989	60, F	right	Bra, Rig	headaches, lethargy	not given	partial remission
Ammenomori *et al.* [2], 1989	58, M	right	Tre, Hyp, Bra, Rig, Gt	headache	not given	complete remission
Tybor and Kotwica [32] 1992	75, M	bilateral	Tre, Hyp, Bra	headache	not given	complete remission
Hageman and Horstink [11] 1994	66, M	left	Tre, Hyp, Bra	none	not given	spontaneous remission
Sunada *et al.* [28] 1996	75, M	left	Tre, Hyp, Rig, Gt	confusion, hemiparesis (right)	yes	complete remission
Wiest *et al.* 1998	63, M	right	Tre, Bra, Rig, Hyp, Gt (left > right)	none	no	complete remission
	70*, M	bilateral	Increase of preexistent Bra, Rig, Gt	incontinence, somnolence	not given	recovery to premorbid function
	82*, M	right	Increase of preexistent Tre, Bra, Gt (left > right)	apathy	decreased response to levodopa	recovery to premorbid function

M Male; *F* female; *CSH* chronic subdural haematoma; *Tre* tremor; *Hyp* hypomimia; *Bra* bradykinesia; *Rig* rigidity; *Gt* gait disturbance.
* Deterioration of pre-existing parkinsonian symptoms and signs.

mechanical compression secondary to bilateral haematomas progressing to central herniation in a 66-year-old man [30]. This patient developed persisting levodopa-responsive parkinsonism after a delay of several weeks after removal of the haematomas. Recently, Turjansky *et al.* presented the case of a 36-year-old woman with an acute right sided temporal haematoma and overlying subdural haematoma with anatomical distortion and brainstem compression [31]. The patient developed a hemiparkinsonian syndrome which was ipsilateral to the haematoma. An 18F-dopa PET study was performed which demonstrated reduced uptake in the left putamen, contralateral to the side of the haematomas.

In all patients with a rapidly progressive course of new-onset parkinsonism or otherwise unexplained deterioration of pre-existent parkinsonian syndromes CT studies should be performed to exclude curable causes

of secondary parkinsonism. The prognosis of CSH-related parkinsonism is favourable after adequate surgical treatment. Early recognition of CSH in these patients can prevent inefficacious drug regimes and will limit time of hospitalisation. While clues such as headaches or additional neurological symptoms and signs are evident in several patients with CSH-related parkinsonism such symptoms can also be absent.

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Comments

This article describes three cases where parkinsonian syndromes were caused by chronic subdural haematomas. An extensive literature research and discussion of the pathophysiological mechanisms is added. The article is well written, well researched, but basicly is just a report of 3 more cases to be added to about 25 previously published cases in about 20 references. The experience reported here could be of some interest to neurosurgeons but also to neurologists who are mainly involved in the primary care of patients with movement disorders.

This paper would renew the well-known fact that chronic subdural haematoma has been described as a chameleon, able to mimic chronic psychosis, dementia, stroke and also parkinsonian disease. This was discovered when after the advent of computertomography hundreds of patients in chronic mental institutions where screened by CT-scan and certain types of organic brain disease were discovered as causes of these diseases, such as fronto-basal meningioma or chronic subdural haematoma. The paper could help to alert neurosurgeons and neurologists and remind them that in seemingly classic

parkinsonian syndrome a CT-scan should be performed at the beginning of any investigation or treatment. However, this is also not quite new, since in many psychiatric and neurological hospitals it is now standard to have at least one baseline computed tomography or MRI in all brain diseases when the patient is seen for the first time.

J. Schramm, C. Schaller

With great interest I was studying this paper and I have also discussed it with out neurological colleagues here in Graz.

The subject of functional movement disorders caused by the mechanical power of a chronic subdural haematoma was demonstrated in 3 cases; this was also published by Samii 1963, Sandyk 1983 and Harding 1984 in case of deterioration of the symptoms in parkinson patients and that one should think about such a cause like chronic subdural haematoma in the diffential diagnosis. The main goal of this paper should be to outline the importance of radiological investigation with CT and MRI in patients with worsening of extrapyramidal disorders. We could not find MRI control investigations or functional neuro-imaging with PET, although it was mentioned in the text, but not performed. This the paper is worth while because of the clinical relevance as well as the complex problem of the interdisciplinary observation and treatment of Parkinson patients.

G. Pendl

Correspondence: Joachim K. Krauss, M.D., Department of Neurosurgery, University of Berne/Inselspital, 3010 Berne, Switzerland.