

Chronic Subdural Hematoma Presenting as Ataxic Hemiparesis

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Abstract. A 57-year-old man was admitted for a typical left ataxic hemiparesis syndrome. He presented no vascular risk factor, with normal blood pressure. CT scan showed a frontoparietal right subdural hematoma. After surgery, the symptomatology disappeared completely. This observation reveals that subdural hematoma must be considered as a possible etiology of ataxic hemiparesis syndrome. The physiopathology is discussed.

The ataxic hemiparesis syndrome (AHS) was individualized by Fischer and Cole [3] in 1965. It is the association of a weakness of a lower limb to a dysmetria of the upper limb on the same side. It is usually known to be produced by a deep vascular lesion, lacunar infarct or small hematoma, in the basis pontis or in the internal capsule. It thus only requests medical management. We report one case in which the cause of this syndrome was a chronic subdural hematoma.

Report of a Case

A 57-year-old man who had been healthy and without known vascular risk factor, experienced over 5 days an unsteadiness of gait, and a clumsiness of the left hand. He complained of recent headaches, mainly in the morning. Two weeks later, persistence of these symptoms led to hospitalization in our department.

The patient was alert and well oriented. His blood pressure was 150/90 mm Hg. Examination revealed a mild left crural weakness, with a flexion graded 4/5, making walking difficult without assistance. Strength of the upper limbs and of the right lower limb was intact. Deep tendon reflexes were brisker in the left side and the left plantar response was extensor. The finger-nose and heel-knee-shin tests revealed a marked left dysmetria, rapid alternating movements were slow and awkward on the left side and the Holmes-Stewart phenomenon was present on the left. There was no dysarthria. The pupils, visual fields and extraocular movements were normal. The sensory examination was intact, with no sensory extinction phenomenon.

A computed tomography (CT) scan performed on the third day revealed a hypodense right frontoparietal subdural hematoma, with



Fig. 1. CT scan showing right subdural hematoma with mass effect on the right lateral ventricle.

mass effect on the right lateral ventricle (fig. 1). The cerebellum was intact.

On the next day, the patient underwent a right parietal craniectomy with removal of the hematoma. Three days after surgery, ataxia had completely disappeared, and only brisk deep tendon reflexes in the left limbs persisted.

Discussion

This clinical observation fits with the AHS described by Fischer and Cole [3]. The localizing value of the AHS is still under discussion. Previous reported observations of AHS concerned mostly small deep lesions in the white matter. When it was initially described in 1965 by Fisher and Cole (on pathological examination), it was ascribed to an ischemic lesion of the lower posterior arm of the contralateral internal capsule [3]. In 1978, Fischer [4] reported three anatomical observations of AHS with lesions at the junction of the upper and medial segments of the pons on the contralateral side. Later, this syndrome was reported in lacunar infarcts of the pons [11, 16], the internal capsule [5, 6, 13, 14, 18], and of the corona radiata [7, 15], and in hematomas of the pons [8, 19] and of the internal capsule [17]. The first case studied by CT scan, reported in 1980, revealed a lucency in the internal capsule. In further observations, CT scan or magnetic resonance imaging [11] confirmed previous anatomical data.

Observations of superficial lesions are much more uncommon. The first case was reported in 1986 by Mizon and Rosa [10] and concerned a patient in whom ataxic hemiparesis was secondary to a meningioma of the falx cerebri.

We think that in our case, the right subdural hematoma was responsible for the AHS, because of the simultaneity of the neurological signs and the CT scan findings, and especially because of the dramatic improvement of the symptomatology by the day after surgery. The AHS resulted from a widespread superficial lesion producing compression of both the frontal and the parietal lobes. It is, however, difficult to determine the level of dysfunction because of the existence of a mass effect on the deep structures. It was similar, in this point of view, to a case reported by Kuschner et al. [9], in which incoordination of a limb (without any weakness) was secondary to a frontoparietal tumor, accompanied by contralateral cerebellar hypometabolism.

It is well known that a supratentorial disease may produce cerebellar dysfunction. Recent studies of cerebellar blood flow in supratentorial ischemic strokes have, in many cases, revealed a functional hypometabolism affecting the cerebellar hemisphere contralateral to the cerebral infarct [12]. This phenomenon, named crossed cerebellar diaschisis (CCD), has also been described in patients with cerebral tumors [9]. Topographical correlations found that this syndrome is more frequent following widespread lesions involving both the parietal and

the frontal lobes, or in deep middle cerebral artery territory infarcts [12]. It is ascribed to a loss of excitatory input in the contralateral cerebellar hemisphere, caused by a dysfunction of corticopontocerebellar fibres. As it has been described in patients without motor impairment, it does not necessarily imply corticospinal fibre involvement [9]. A restricted distribution of the motor pathways lesions gives intact strength in one limb, and thus does not interfere with the demonstration of ataxia.

These data are in agreement with the anatomical findings: corticopontocerebellar pathways arise mostly in the frontal and parietal cortices, where they are more widespread and numerous than corticospinal motor fibres [2], before converging through the deep cerebral white matter, towards the internal capsule, the sublenticular and retrolenticular regions, and towards the pons via the cerebral peduncles. The AHS can probably be explained by a similar phenomenon and, as the CCD may be produced not only by deep but also by superficial cerebral lesions, although the latter appears to be an extremely uncommon etiology. It thus reveals that AHS must be considered as a clinical syndrome, with several possible topographical lesions.

Furthermore, this case is the first observation of an AHS produced by a subdural hematoma. This pathology must then be evoked when AHS occurs, and this observation shows that this syndrome is not always caused by a benign medical pathology with a spontaneous good prognosis. It may be ascribed to a neurosurgical lesion requiring emergency management. Therefore, a CT scan should always be promptly performed when this syndrome occurs, especially in the absence of vascular risk factor.

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