Hemiataxia-hypesthesia: a thalamic stroke syndrome

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Abstract

Six patients had isolated hemiataxia and ipsilateral sensory loss, as a manifestation of thalamic infarction in the thalamogeniculate territory. Acute hemiataxia-hypesthesia was not found in 1075 other patients from the Lausanne Stroke Registry who were admitted during the same period. Stroke onset was progressive in five patients and immediately complete in one. Five patients had an objective sensory loss. In two patients this affected light touch, pain and temperature sense, and in another three light touch, pain temperature, position and vibration sense. One patient had a purely subjective sensory disturbance. The sensory deficit cleared or was clearing although the ataxia persisted in all patients. On lesion mapping on CT or MRI, all patients had involvement of the lateral part of the thalamus (ventral posterior nucleus and ventral lateral nucleus). The presumed causes of stroke were cardioembolism in one patient, posterior cerebral artery occlusion in one patient and meningovascular syphilis in one patient, hypertensive small vessel disease in two patients, and undetermined in one patient. Hemiataxia-hypesthesia is a new stroke syndrome involving the perforating branches to the lateral thalamus, but in which small vessel disease may not be the leading cause.

Though not specific, few neurological syndromes are usually associated with small infarcts in the territory of deep perforating branches. A pure sensory stroke (PSS) is usually due to a small infarct involving the thalamus. A pure motor hemiparesis (PMH) is most commonly the result of infarction in the internal capsule or the basis pontis. In the majority of patients with ataxic hemiparesis (AH), the infarct is located in the pons, corona radiata or internal capsule.

The neurologic picture associating hemisensory disturbance with hemiataxia on the same side of the body has not been reported in detail. Six patients with isolated hemiataxia and ipsilateral sensory loss as a manifestation of small thalamic infarction were studied.

Methods

By a systematic search of the Lausanne Stroke Registry,⁵ six patients with isolated hemiataxia (limb incoordination with dysmetria, hypermetria, intention tremor, and dysidiadochoki-

nesia) were found and ipsilateral sensory disturbance among 1075 patients with a first stroke who were admitted consecutively to our centre between 1986 and 1990.

Patients with associated hemiparesis, visual disturbances or neuropsychological dysfunction were excluded. All patients were examined by at least one of the authors. The patients with deep sensory loss were observed using several clinical tests for ataxia, to exclude sensory disturbance as the main cause for incoordination. Proprioceptive ataxia is defined as poorly controlled direction of finger to nose and heel to knee tests but without intention tremor or oscillations, and impairment of limb placement, which are critically compensated by visual cues.⁶

CT (with and without contrast) or MRI was performed within 36 days of stroke. The anatomical location of infarct was assessed following the lesionmapping templates developed in our centre and elsewhere.^{7,8} The size of infarcts (maximum diameter) was determined by the authors independently.

All patients had Doppler ultrasound, electrocardiogram, and standard blood and urine tests. Angiography was performed in two patients, echocardiography in three patients and CSF analysis in three patients.

Hypertension (blood pressure higher than 160/90 mm Hg measured at least twice before the stroke), diabetes mellitus (known fasting blood glucose > 6 mmol/l before the stroke), cigarette smoking, hypercholesterolemia (> 6.5 mmol/l) and other clinical features including heart disease were recorded.

Follow up data were obtained through our outpatient clinic between one and 12 months after stroke.

Results

Patients

All six patients had a thalamic infarct on the side opposite to their symptoms. They belonged to a group of 30 patients with thalamic infarction on CT or MRI who were seen during the study period. There were four men and two women, with a median age of 67 (range 38-82) (table 1). Four patients had at least two risk factors—ie, hypertension, diabetes mellitus, cigarette smoking, or hypercholesterolemia. One (patient 4) had no known vascular risk factors (table 2).

Clinical Findings

Stroke onset was progressive over two to 24 hours in all patients but one (patient 2) who

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Table 1 Clinical findings

Patient/Age/Sex	Side	Onset	Headache	Hypesthesia	Ataxia	Follow-up (months)
1/38/M	R	P (6h)	+	FU*	UL	12
2/68/M	L	P (7h)	+	FUTL**	UL	1
3/42/M	L	P (2h)	—	FUL	UL	1/2
4/82/F	L	I	+	FUL*	UL	4
5/76/M	R	P (24h)	—	FUL	UL	1/2
6/66/F	L	P (12h)	+	L (sj)	UL	1

R = right; L = left; P = progressive; I = immediately complete; sj = purely subjective; F = face; U = upper limb; L = lower limb; T = trunk; * = vibration and position sense spared; ** = vibration and position sense disturbed only in lower limb.

Table 2 Risk factors/Presumed aetiology

Patient	Risk factors	Heart disease	Angiography	Aetiology
1	CS, HC, DM	—	—	Meningovascular (syphilis)
2	HT	—	PCA occlusion	Large artery disease
3	HT, CS, HC	Intra-atrial thrombus	—	Cardioembolism
4	—	—	—	—
5	HT, HC	—	—	Small vessel disease
6	HT, DM	—	—	Small vessel disease

CS = cigarette smoking; DM = diabetes mellitus; HC = hypercholesterolemia; HT = hypertension; PCA = posterior cerebral artery.

had a transient impairment of consciousness at onset (see table 1). All patients were examined within 48 hours of stroke and none reported improvement before admission. Sensory disturbance was the first symptom, usually starting with prickling in the face (predominantly peri-oral) and distally in one or both limbs. Patient 3 complained of leg pain as the first symptom that later progressed to the arm. All patients had hemiataxia contralateral to the thalamic infarct, with a "cerebellar-type" of incoordination on the finger to nose, finger to

finger, and heel to knee tests (defined as movements with normal initiation and velocity, but with irregular accelerations and decelerations producing oscillations on getting close to the target with a series of secondary corrective movements around the target, with the eyes either open or closed.⁹ Dysdiadochokinesia and loss of dynamic tonus (rebound phenomenon) were also present on that side.

Five patients had sensory loss. In two patients this involved light touch, pain and temperature sense and light touch, pain, temperature, position and vibration sense in the other three. These sensory disturbances involved the entire hemibody in one patient (patient 2), spared the trunk in three patients (patients 3–5) and were limited to the face and arm in patient 1. Patient 6 had a purely subjective sensory disturbance (lower limb parasthesias). Patient 2 had a transient asterix involving the hand contralateral to infarct. The remaining neurological examinations were normal in all patients, including tendon reflexes, muscular strength, and plantar responses.

Imaging Findings

CT showed the infarct in five patients, and in one patient the lesion was visible only on MRI (see fig). In patient 3 the infarct was visible on CT, although MRI showed a larger lesion. On lesion mapping on CT or MRI, all infarcts involved the ventral posterior nucleus and the ventral lateral nucleus (thalamogeniculate or inferolateral territory).¹⁵ The adjacent part of the internal capsule seemed to be affected in

Figure Topography of infarcts on CT (patients 1–4, 6) or MRI (patient 5). ■ Thalamogeniculate arteries territory.

two patients (patients 1 and 6). In patient 2, there was also a silent small infarct in the ipsilateral occipital lobe. The maximum diameter of infarcts varied between 7.5 and 12.5 mm.

Aetiology

The presumed cause of stroke was cardio-embolism in patient 3 who had an intra-atrial thrombus diagnosed by transesophageal echocardiography. Posterior cerebral artery occlusion was found on angiography in patient 2. Hypertensive small vessel disease was likely in two patients who had hypertension (patient 5) or hypertension and diabetes (patient 6), in the absence of potential arterial or cardiac sources of emboli. Patient 1 had meningovascular syphilis with abnormal CSF. The cause of stroke remained unknown in patient 4 (Refer to table 2).

Evolution

Patient 3 recovered completely within 15 days; in another patient (patient 4), some degree of ataxia and sensory loss persisted for four months after the stroke. In both patients, the sensory deficit cleared or improved before ataxia. In the other patients, the hemiataxia persisted nearly unchanged (at ½, one and 12 months) despite complete sensory recovery.

Discussion

The first so-called "thalamic stroke syndrome" was reported by Jules Déjérine and Gustave Roussy in 1906.¹⁰ This was defined as a prominent and persistent sensory loss affecting both superficial (touch, pin and temperature) and deep (position and vibration sense) modalities and involving the entire hemibody opposite to a thalamic infarct; a mild and rapidly covering hemiparesis could be associated; later, intolerable pain and choreoathetotic movements could develop in the affected limbs. Several case reports with different manifestations of thalamic infarction have been reported since the original paper by Déjérine and Roussy, but little attention has been paid to the neurologic picture demonstrated by our patients.^{11–14}

Isolated thalamic infarction may be classified into specific syndromes related to four main arterial territories.¹⁵ These include: thalamogeniculate or inferolateral infarction, which is characterized by complete hemicorporeal sensory loss (with possible sparing of proprioception), isolated or associated with some degree of weakness or ataxia; tuberothalamic infarction, with neuropsychological dysfunction and transient corticospinal signs; posterior choroidal infarction, with partial hemianopia as the main abnormality; and paramedian infarction, with vertical eye movement disorders, and transient and fluctuating consciousness disturbances followed by neuropsychological dysfunction.

Reviewing all previous cases of thalamic infarcts from published reports, we found two cases of patients with a clinical presentation similar to our six patients.^{16 17} The association

hemiataxia-hypesthesia, however, has never been emphasised. Caplan *et al*¹⁶ reported a 60 year old hypertensive and diabetic man, with progressive left hemicorporeal sensory disturbance (pin, touch, and position sense) associated with an ipsilateral cerebellar-type of incoordination and dysarthria. Strength, reflexes, and plantar responses were normal. A patient with thalamic hematoma manifested by sensory loss and ipsilateral ataxia was reported by Azouvi *et al*,¹⁷ and ataxia was imputed to proprioceptive loss. The neurologic picture, however, was poorly characterised. In these two cases, the lesion involved the lateral part of the thalamus. In a group of 18 patients with thalamogeniculate territory infarction studied previously at our centre,¹⁵ sensory loss was associated with some degree of ataxia in six patients. Isolated hemiataxia and ipsilateral sensory loss have been reported by the authors in relation to an anterior choroidal artery infarct involving the posterior limb of the internal capsule immediately adjacent to the thalamus, which probably disrupted part of the thalamic radiations.¹⁸

We think that isolated hemiataxia and ipsilateral sensory loss (hemiataxia-hypesthesia) is clearly distinct from the Déjérine-Roussy syndrome. In our patients, no motor weakness was present, whereas the ataxia was the main disturbance. It is also remarkable that in all but one patient, the sensory loss improved or cleared before hemiataxia.

Sensory loss in our patients may be easily explained by involvement of the ventral posterior nucleus on the appropriate side.¹⁹ The explanation for thalamic ataxia may be more controversial. Some authors have advocated that it may be due to associated hemiparesis or proprioceptive loss.^{10 20 21} None of our patients, however, had hemiparesis. Also, the incoordination had no feature suggestive of proprioceptive ataxia, and it was not linked to deep sensory loss as it persisted despite recovery of sensory disturbances. A patient with lateral thalamus hemorrhage and sensory loss associated with ipsilateral hemiataxia and hemiparesis was followed for over three years by Garcin.²² Ataxia also persisted after sensory loss and weakness recovered. He referred to the "thalamic cerebellar-type of ataxia" already quoted by Vincent in 1908.²³ Involvement of the ventral lateral nucleus, with interruption of dentatorubrothalamic fibres, may be a possible cause for ataxia in our patients who all showed involvement of that nuclear group on CT or MRI. In patients with ataxic hemiparesis and internal capsule involvement, the ataxia has been linked to the interruption of reciprocal fibres connecting the ventral lateral nucleus and the precentral cortex.^{24–27} This emphasises the possibility that the ventral lateral nucleus of the thalamus may play a key role in the genesis of "thalamic ataxia".

Crossed cerebellar diaschisis,^{28 29} describes a metabolic depression in the cerebellar hemisphere contralateral to supratentorial infarction. It could be a functional counterpart to this anatomical hypothesis.¹⁸ Although crossed cerebellar diaschisis has been reported in

patients with capsular or thalamic stroke, however, the correlation with clinical ataxia has only been anecdotal.^{30 31}

We suggest that acute hemiataxia-hypesthesia may be highly suggestive of small strokes involving the lateral part of the thalamus (thalamogeniculate territory) or the immediately adjacent internal capsule (anterior choroidal artery territory). Contrary to ataxic hemiparesis, pure motor hemiplegia or pure sensory stroke, hypertensive small artery disease does not seem to be the main cause of this syndrome, as this cause could be retained in only two of the six infarcts in our patients and in two of the seven infarcts previously reported.^{15 16}

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