Vascular ataxic hemiparesis: a re-evaluation

Thierry Moulin, Julien Bogousslavsky, Jean-Luc Chopard, Joseph Ghika, Thierry Crépin-Leblond, Valérie Martin, Philippe Maeder

Abstract

Ataxic hemiparesis is commonly considered as one of the "typical" lacunar syndromes. Using the prospective stroke registries from Lausanne and Besançon, 100 patients were selected consecutively (73% men, 27% women; age 64.7 (SD 13.6) years) with a first stroke and ataxic hemiparesis (hemiparesis or pyramidal signs and ipsilateral incoordination without sensory loss). Brain CT or MRI was performed on all patients. A primary haemorrhage was present in 5%, an infarct in 72%, isolated leukoaraiosis in 9%, and no apparent abnormality in 14%. The locations of lesions were the internal capsule (39%), pons (19%), thalamus (13%), corona radiata (13%), lentiform nucleus (8%), cerebellum (superior cerebellar artery territory) (4%), and frontal cortex (anterior cerebral artery territory) (4%). The clinical features of ataxic hemiparesis with different locations were almost identical. Only minor associated signs allowed the localisation of the lesions (paraesthesiae with a lesion in the thalamus; nystagmus or dysarthria with a cerebellar or pontine location). Crural paresis with homolateral ataxia was seen only with cortical paramedian frontal lesions. Presumed hypertensive small artery disease was not always found, but was still the leading cause of stroke, being present in 59% of the patients and in 62% of those with small deep infarcts. A potential source of embolism (arterial or cardiac) was found in one fourth of the patients. Therefore no definite association can be made between ataxic hemiparesis and lacunar infarction. In particular, so called uncommon lesion locations may not be rare. After extensive investigations a diagnosis of lacunar infarct can be retained in only slightly more than half of the cases.

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Several decades ago, French neurologists wrote on the clinical aspect of ataxic hemiparesis, though they did not use this term.¹⁻⁴ The first vascular case was reported by Nicolesco *et al*⁵ in a patient with an anterior cerebral artery territory infarct. Further

reports emphasised several lesion sites, including the thalamus and frontal lobe.⁶⁻⁹

Fisher introduced the term ataxic hemiparesis in 1978 to designate a clinical picture associating hemiparesis with ipsilateral ataxia.¹⁰ He emphasised that the lesion might be the result of an infarct in the pons, corona radiata, or internal capsule, and that it is suggestive of "lacunar infarction" (a small infarct in the territory of a deep perforator due to small artery disease).^{11,12} Initially described as a homolateral ataxia and crural paresis,¹³ ataxic hemiparesis is currently defined by the association of hemiparesis or corticospinal signs (weakness or hyperreflexia, Babinski's sign) with ipsilateral cerebellar incoordination. This picture has usually been explained by a lesion in the white matter, involving both the corticospinal and the cerebello-thalamo-cortico-ponto-cerebellar tracts^{10,12}; however, it has also been found to involve the thalamus or cortical areas.¹⁴⁻¹⁶ Ataxic hemiparesis is not the commonest "lacunar syndrome" (<10%).¹⁷⁻¹⁹

Our purpose was to study the features of ataxic hemiparesis and its association with lacunar infarction in a general population with first stroke.

Methods

We studied 100 patients with ataxic hemiparesis and first stroke. The patients were selected consecutively between 1986 and 1990 from all patients admitted to two primary care centres; all patients were part of prospective stroke registries (Lausanne and Besançon). All were examined by at least one of us. Selection criteria included: (a) hemiparesis with a motor deficit of mild or moderate severity with or without increased tendon reflexes and Babinski's sign. Involvement of the face, upper limb, and lower limb was assessed; (b) cerebellar type of incoordination, with dysmetria, defined as poorly controlled direction movement on finger to nose or finger to finger and heel to knee tests (with normal initiation and velocity but irregular acceleration or deceleration producing oscillations near the target with a series of secondary movements, eyes open or closed), rebound phenomenon (Steward-Holmes test), and dysidiadochokinesia; (c) no sensory deficit, but subjective dyesthesia or paraesthesia was allowed, as was dysarthria or gaze evoked nystagmus; (d) no evidence for aphasia, apraxia, other neuropsychological dysfunction or visual field defect.

Service de Neurologie,
Hôpital J Minjoz,
CHU Besançon,
France
T Moulin
J-L Chopard
T Crépin-Leblond
V Martin

Service de Neurologie,
CHU Vaudois,
Lausanne,
Switzerland
J Bogousslavsky
J Ghika
P Maeder

Correspondence to:
Dr Julien Bogousslavsky,
Service de Neurologie,
CHU Vaudois, CH-1011
Lausanne, Switzerland.

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Table 1 Distribution of risk factors and type of stroke

Risk factor	No lesion leukoaraiosis (n = 23)	Deep infarct (n = 53)	Brainstem infarct (n = 14)	Superficial infarct (n = 6*)	Primary haemorrhage (n = 4*)	Total (n = 100)
Age (y)	69.9	63.6	62.2	61.6	59.5	64.7
(SD)	(9.9)	(13.6)	(14.1)	(21.6)	(15.8)	(13.5)
Male/female	16/7	40/12	10/5	5/1	2/2	
(% male)	(69.5)	(76.9)	(66.6)	(83.3)	(50)	(73)
Hypertension	19†	28	11†	3	1	(62)
(%)	(83)	(53)	(79)	(50)	(25)	
Diabetes	3	9	3	1	0	(16)
(%)	(13)	(17)	(21)	(16)	00	
Smoking	6	21	5	4	3	(39)
(%)	(26)	(40)	(36)	(67)	(75)	
Hypercholesterol	6	13	3	1	1	(24)
(%)	(26)	(25)	(21)	(16)	(25)	
PCV >45%	11	28	5	2	2	(48)
(%)	(47)	(53)	(36)	(33)	(50)	
Previous TIAs	1	9	3	1	1	(15)
%	(4)	(16)	(21)	(16)	(25)	

*One primary haemorrhage was considered as an intrinfarct haematoma.

†Percentage among patients with brainstem infarct or without lesion differed significantly ($p > 0.05$) from other patients. Hypercholesterol = Hypercholesterolaemia; PCV = packed cell volume.

The protocol of investigations and analysis of associated factors followed the guidelines of the Lausanne stroke registry, as reported in detail previously.¹⁹ Risk factors included hypertension; blood pressure higher than 160/90 mm Hg at least twice before the stroke; diabetes mellitus; known fasting blood glucose >6 mmol/l before the stroke; hypercholesterolaemia; fasting blood cholesterol higher than 6.5 mmol/l, and current cigarette smoking. All patients had Doppler ultrasound and B mode echotomography, ECG, and standard blood and urine tests. Angiography, trans-thoracic/trans-oesophageal echocardiography, and transcranial Doppler were performed in selected patients.

In selected instances CT and MRI were performed at least once within one month of the stroke; all emergency CTs were followed by another CT or MRI four to 10 days later. The topographic diagnosis of infarct or primary haemorrhage was made from lesion mapping templates developed in the Lausanne stroke registry and elsewhere.^{20, 21} The results of neuroimaging were classified into different groups according to anatomical locations and vascular territories: (a) no lesion visible; (b) leukoaraiosis, (c) posterior part of posterior limb of internal capsule (territory of anterior choroidal artery); (d) thalamus (terri-

tory of thalamogeniculate arteries); (e) frontal cortex (superficial territory of anterior cerebral artery); (f) pons (territory of paramedian arteries); (g) corona radiata/centrum ovale (involving the territory of medullary branches of the superficial branches of middle cerebral artery); (h) lentiform nucleus and adjacent part of internal capsule (territory of lenticulostriate arteries). The location and volume of lesions were determined independently by at least two of us (including a neurologist and a radiologist).²²

Presumed infarct aetiologies were separated into the following groups as previously defined¹⁹: large artery disease (>50% stenosis in the appropriate large artery); potential cardiac source of embolism; small artery disease (hypertension or diabetes mellitus, in the absence of a potential arterial or cardiac source of embolism, and with a <15 mm infarct limited to the territory of a deep perforator on CT); other aetiologies (dissection, haematological disorders, etc); and undetermined.

Follow up data was obtained from the outpatient clinic.

Statistical studies were carried out with a descriptive univariate analysis χ^2 test corrected by Fisher's test, and analysis of variance (ANOVA) for volume measurement.

Results

GENERAL CHARACTERISTICS

There were 51 patients from Lausanne and 49 from Besançon, representing 4% of strokes in each centre. These two groups were identical in sex, age distribution, risk factors, aetiologies, and location of lesions. There was a preponderance of men (73%). Mean age was 64.7 (SD 13.6) (table 1).

STROKE TYPE AND TOPOGRAPHY

No lesion was found in 14 patients, but 10 of these patients had only one CT and none had MRI. In nine patients, the only abnormality was leukoaraiosis, which was found in nearly a quarter of the patients. At least one lesion was found in 77 patients (table 2). An infarct was found in 94% of these cases (72/77) and a

Table 2 Type of stroke

Type of stroke	Number
No visible lesion	14
Leukoaraiosis	9
Total	23
Infarcts	72
Superficial infarct	5
ACA pial territory	2(*)
SCA pial territory	3
Deep infarct	53
Brainstem infarct	14
Pons (upper and medial part)	14
Primary haemorrhage	5
Lentiform nucleus	2
Corona radiata	1
Pons	1
Lobar (intrinfarct haematoma; ACA)	1*
Total	100

*One primary haemorrhage was considered as an intrinfarct haematoma.

ACA = Anterior cerebral artery; SCA = superior cerebellar artery.

Table 3 Location of stroke

Location	Number (%)	Volume (ml)
Frontal cortex (ACA territory)	3 (4)	—
Corona radiata: anterior part	10 (13)	0.71 +
Corona radiata: posterior part	9 (12)	0.45
Internal capsule	21 (39)	0.39
Lentiform nucleus	6 (8)	1.24*
Thalamus	10 (13)	0.12**
Pons	15 (19)	0.18**
Cerebellum (SCA territory)	3 (4)	—
Total	77 (100)	0.43

ACA = Anterior cerebral artery; SCA = superior cerebellar artery.

* $p < 0.05$; ** $p < 0.01$, ANOVA test.

haemorrhage in 6% (one intrinfarct haematoma located in anterior cerebral artery territory, four primary haematomas located in the corona radiata (one patient), pons (one patient) or lentiform nucleus (two patients)). There were 67 small deep infarcts (53 in the anterior circulation and 14 in the posterior circulation) and five superficial infarcts.

The main location of lesion was the posterior part of the internal capsule (30/77; 39%) (table 3). As CT section variability sometimes rendered the lesion more difficult to localise, we considered that the lower part of the internal capsule (21/30) and the superior part of the internal capsule located in the posterior part of corona radiata near the ventricular body (9/30) corresponded to the same territory. The volume of infarcts in these two areas was very close (0.39–0.45 ml) and the clinical aspects were also similar. The second most common location was the pons, in 19% (15/77), followed by the anterior part of the corona radiata (remote from the ventricular wall) in 13% (10/77), and thalamus in 13% (10/77) (involving the territory of the thalamo-geniculate artery (9/10) except in one patient with a thalamotuberal infarct). Two locations were rare: in 8% (6/77), the lesion was in the lentiform nucleus (medial lenticulostriate arteries territory), being a primary haemorrhage in two of the six patients; superficial infarcts were found in 8% (6/77) of the patients, three infarcts involving the anterior cerebral artery territory (one patient with intrinfarct haematoma), and three the superior cerebellar artery territory.

The volume of lesions was calculated only for deep lesions (mean 0.43 ml). The statistical analysis of the volume according to different

locations showed an expected difference, in that smaller volumes were found in the pons and thalamus and larger volumes in the lentiform nucleus (table 3).

Brain MRI was performed in 23 patients; in half of them it confirmed the location of the lesion seen on CT, whereas in 39% of the cases it identified a new lesion. Brain MRI showed a double lesion in 10.5% (8/77) of the patients (pons and internal capsule in five patients and corona radiata and internal capsule in three).

RISK FACTORS AND STROKE AETIOLOGY

Tables 1 and 4 summarise the risk factors and aetiology. The prevalence of risk factors in the small deep infarct group were: hypertension (62%), smoking (39%), and hypercholesterolaemia (24%). Diabetes was less frequent (16%). A potential arterial (10%) or cardiac (12%) source of embolism was present in 22% of the patients. The commonest presumed cause of stroke was hypertensive small artery disease (59% overall). In deep and brainstem infarcts, this frequency increased (62%); several locations were associated with an even higher prevalence of small artery disease (thalamus (70%) and pons (80%)).

Presumed aetiologies were similarly distributed among the different subtypes and locations of stroke, except for embolic events, which were more common with superficial infarcts (50%) and with infarcts in the anterior part of the corona radiata (40%).

CLINICAL PATTERNS (TABLE 5)

Ataxia was present in both the upper and lower limb in 98% of the patients. Dysmetria and hypermetria were present in all patients, but hypotonia or dysdiadochokinesis were found in only one quarter of the patients. Face-upper limb-lower limb (FUL) hemiparesis was almost as frequent with pontine (53%) and capsular lesions (57%). A partial motor deficit (FU, UL, or F/U/L) was more common with lentiform nucleus (67%) involvement, followed by the anterior part of the corona radiata (30%), the thalamus (20%), and the pons (17%). The frequency of facial weakness was similar with lesion of the pons (60%), anterior part of the corona radiata (50%), lentiform nucleus (50%), and thalamus (40%). In infarcts involving the anterior

Table 4 Relationship between aetiology and stroke type (n (%))

Aetiology	No lesion leucoarosis (n = 23)	Deep infarct (n = 53)	Brainstem infarct (n = 14)	Superficial infarct (n = 6*)	Primary haemorrhage (n = 4*)	Total (n = 100)
Undetermined	4 (17)	9 (17)	3 (22)	2 (33)	1 (25)	19
Large artery disease†	2 (9)	6 (11)	1 (7)	0	1 (25)	10
Embolic heart disease	1 (4)	8 (15)	0	3 (50)	0	12
AF	—	7	—	2	—	9
Akinesia	1	1	—	—	—	2
MVP	—	—	—	1	—	1
Hypertensive arteriopathy	16 (70)	30 (57)	10 (71)	1 (17)	2 (50)	59

*One primary haemorrhage was considered as an intra-infarct haematoma; †>50% stenosis in the appropriate artery or dissection.

AF = Atrial fibrillation; MVP = mitral valve prolapse.

Table 5 Clinical features and stroke location

Location	No motor deficit	FUL	FU/UL	F/U/L	No F	Isolated ataxic hemiparesis	Paraesthesia	Dysarthria	Static ataxia	Nystagmus
Frontal cortex (ACA territory)	0	0	0	0/0/3 (100)	3 (100)	3 (100)	0	0	1 (33)	0
Corona radiata anterior part	0	4 (40)	0/3 (30)	0/2/1 (30)	5 (50)	6 (60)	1 (10)	3 (30)	3 (30)	0
Corona radiata posterior part	0	4 (44)	3/0 (30)	0/1/1 (26)	2 (22)	4 (44)	0	4 (44)	1 (11)	0
Internal capsule	0	12 (57)	5/1 (29)	0/2/1 (14)	3 (14)	11 (52)	3 (14)	2 (9.5)	6 (29)	0
Lentiform nucleus	0	2 (33)	0	1/0/3 (67)	3 (50)	3 (50)	1 (17)	1 (17)	1 (17)	0
Thalamus	2 (20)	4 (40)	0/2 (20)	0/2/0 (20)	6 (60)	1 (10)	8 (80)	2 (20)	3 (30)	0
Pons	0	8 (53)	0/5 (30)	1/1/0 (17)	6 (40)	3 (20)	0	10 (67)	10 (67)	1 (7)
Cerebellum (SCA territory)	0	2 (67)	1/0 (33)	0/0/0	0	1 (33)	0	1 (33)	1 (33)	2 (67)
Total	2 (2.5)	36 (47)	9/15 (31)	2/7/3 (16)	28 (36)	32 (42)	14 (18)	23 (30)	26 (34)	3 (4)

F = Face; U = upper limb; L = lower limb; ACA = anterior cerebral artery; SCA = cerebellar artery.

cerebral artery territory, the clinical picture was characteristic, with crural paralysis and homolateral brachial ataxia. In half of the patients with thalamic infarct, pyramidal signs were hardly apparent (only increased tendon reflexes or Babinski's sign).

Minor associated signs such as ataxia when standing or dysarthria (67% of patients) suggested a pontine lesion. In thalamic infarct, paraesthesia or pain was frequent (80%). When the internal capsule was involved, paraesthesia was less common (14%), while ataxia and dysarthria was found in one quarter of the patients (23%). On the other hand, no associated disturbance was found in 42% (32/77) of our patients (mainly with involvement of the corona radiata (60%; 6/10), internal capsule (50%; 15/30), or lentiform nucleus (50%; 3/6).

SHORT TERM EVOLUTION

The initial evolution of stroke followed the classical pattern of lacunar infarction with progressive or initially fluctuating onset in one third of the patients, with no differences between stroke type (table 6). At one month, no patient had died. Some weakness remained in 20% of patients, and ataxia persisted in more than 60%. Most patients had no functional disability (38%) or had minor sequelae without limitation of previous activities

(53%). Nine patients had sequelae which limited activities. A severe disability persisted in all patients with infarcts in the anterior cerebral artery territory.

Discussion

The existence of ataxic hemiparesis is controversial, dysmetria sometimes being attributed to corticospinal dysfunction itself.²³ Furthermore, its definition has been variable and at times confused with other clinical syndromes such as dysarthria-clumsy hand.^{10 24} This probably explains our difficulty in comparing the previously reported series with each other. Overall, 4% of our patients with first stroke had ataxic hemiparesis, which corresponds with published findings.^{17 18 25-28}

Although ataxia has usually been considered to be severe in over two thirds of cases,²⁹ it was often moderate in our patients, nearly always involving the arm and leg with equal intensity. Some authors proposed that partial hemiparesis, nystagmus, and dysarthria are suggestive of an infratentorial lesion,³⁰ whereas facial sparing and lack of dysarthria and paraesthesia would be associated with a hemispherical lesion.²⁹⁻³¹ Actually, minor associated signs were common in our patients—for example, paraesthesia with thalamic infarct and dysarthria or gait ataxia with a pontine infarct. Hemiparesis involved the face, arm, and leg in half our patients with capsular, corona radiata or pontine stroke, but facial sparing was not uncommon, mainly with lentiform nucleus lesions. Sudden onset without progression occurred more often in our patients than in previous reports.^{17 19 26 32} Overall, short term recovery was good, especially in patients with small deep infarct or haemorrhage.^{14-18 26} The poorest functional outcome may correspond to anterior cerebral artery territory infarct with large cortical involvement.³³

The prevalence of risk factors in our group with small deep infarcts was similar to previous reports, except for diabetes, which was higher in our series.²⁸ Presumed hypertensive small artery disease is the main cause of small

Table 6 Clinical course, outcome, and stroke type (n (%))

Aetiology	No lesion leukoaraiosis (n = 23)	Deep infarct (n = 53)	Brainstem infarct (n = 14)	Superficial infarct (n = 6*)	Primary haemorrhage (n = 4*)	Total (n = 100)
Onset:						
Sudden	16 (69)	32 (60)	12 (86)	6 (100)	4 (100)	70
Progressive	5 (22)	13 (25)	1 (7)	—	—	19
Fluctuating	2 (9)	8 (15)	1 (7)	—	—	11
Evolution:						
No sequelae	9 (39)	18 (34)	7 (50)	3 (50)	1 (25)	38
Moderate	12 (52)	30 (57)	7 (50)	1 (17)	3 (75)	53
Severe	2 (9)	5 (9)	0	2 (33)	0	9

One primary haemorrhage was considered as an intrinfarct haematoma.

deep infarcts (lacunar infarct).^{17 18 26–29 34} Sixty two per cent of our cases fulfilled the criteria for lacunar infarct, which was particularly common in the thalamus and the pons. On the other hand, potential cardiac or arterial sources of embolism were not uncommon, mainly with superficial infarcts and infarcts in the anterior part of the corona radiata.²¹ In infarcts of the corona radiata, a haemodynamic mechanism in the end zone territory has been suggested.²¹ Low flow could be a consequence of several conditions, such as vascular atheroma, cardiac failure, hypovolaemic state, and hyperviscosity. No haemodynamic factor or trigger could be identified in our patients.

In previous CT studies, lesions have been detected in 30–70% of the cases.^{18 35 36} Size and location are probably determinant, so that infarcts could remain undetected when small or located in the brainstem.^{14 18} Only 14% of our patients had no visible lesion, however. In these cases, associated disturbances (nystagmus, gait ataxia, or dysarthria), partial hemiparesis, and presumed hypertensive arteriopathy were common. Only a few cases with a double lesion have been reported.^{37 38} In a larger series using MRI, one sixth of the cases examined for possible lacunar infarct showed double lesions,³⁵ as in our study. We found that ataxic hemiparesis can develop after different types of stroke, including haemorrhage, superficial infarct, or deep infarct. Haemorrhage as a cause of lacunar syndrome has been reported in 5% of the cases,^{3 35} in agreement with our own findings. Most haemorrhages involve the lenticulostriate region,²⁹ and other locations (internal capsule,³⁹ capsulothalamic region,⁴⁰ and pons^{41 42}) are rare. In our study, lenticulostriate haemorrhages were larger than the other haemorrhages associated with ataxic hemiparesis. On clinical grounds, it was not possible to distinguish ataxic hemiparesis due to haemorrhage from ataxic hemiparesis due to infarct.

The most common lesion associated with ataxic hemiparesis was a small deep infarct.^{28 29} As in recent studies,^{26 28–30 43–48} we found that the most common location was the posterior limb of the internal capsule, usually also involving the upper part of the internal capsule near the ventricular body. By contrast with a recent study,⁴⁹ we suggest that these infarcts may involve the anterior choroidal artery territory as they commonly extended into the posterior limb of the internal capsule. Identification of the supply to this area requires further study.^{48 50} In ataxic hemiparesis, few reports have documented involvement of the anterior part of the corona radiata as in our series; such infarcts were clearly located at some distance from the ventricular body, corresponding to the territory of the white matter medullary arteries,^{21 46 47 51} although part of this area could also be supplied by the lateral lenticulostriate arteries.²⁰ In the brainstem, pontine involvement in the territory of the basilar paramedian arteries is common.^{10 35–38 41 52} We found it to be the second

most frequent location of lesion. Dysarthria and gait ataxia were suggestive of this location. Clinical variants have also been reported, with bilateral motor dysfunction,⁵³ trigeminal nerve impairment,⁵⁴ or slight ataxia in the contralateral lower extremity.⁵⁵ We had no patient with a midbrain infarct.⁵⁶

Although ataxic hemiparesis was usually due to a small deep infarct, superficial infarcts were not uncommon and involved the anterior cerebral artery or superior cerebellar artery territories. Infarcts of the anterior cerebral artery territory with ataxic hemiparesis have commonly been reported.^{5 33} In these cases, the corticopontocerebellar pathway (Türck's tract) may be affected within the frontal lobe, explaining "cerebellar" ataxia.³³ The clinical features in our patients with anterior cerebral artery infarcts were characteristic, with crural weakness and homolateral brachial ataxia. Interestingly, these features were first reported as typical of a lacunar syndrome, with a lesion in the pons.¹³ Our findings do not support this. Infarcts in the superior cerebellar artery territory have rarely been reported with ataxic hemiparesis,⁵⁷ more often with dysarthria clumsy hand syndrome.^{56 58 59}

Thalamic lesions with ataxic hemiparesis have rarely been reported,^{4 6 55 60–63} but we found this location to occur quite often. The lateral part of the thalamus was the most commonly involved area. The dentatorubrothalamic tracts may be affected within the ventrolateral part of the thalamus, so that in thalamic infarct with ataxic hemiparesis, cerebellar ataxia may be explained by the interruption of this afferent pathway. The lack of reports of ataxic hemiparesis from thalamic infarct may be because corticospinal signs are usually slight; they are probably due to ischaemia or oedema compressing the adjacent corticospinal tract.^{4 60} Another possible explanation of mild hemiparesis in thalamic infarct is associated infarction of the adjacent internal capsule, as the thalamogeniculate branches may sometimes contribute to the supply to the innermost part of the posterior limb of the internal capsule.⁶⁴ Because of commonly associated sensory disturbances, ataxic hemiparesis may be less frequent than "hypoesthetic ataxic hemiparesis" or "painful ataxic hemiparesis".^{44 56 60 61} In "hemiataxia-hypoesthesia syndrome", also associated with small lateral thalamic infarcts, no sign of corticospinal dysfunction is present.⁶⁵ These four syndromes, ataxic hemiparesis, hemiataxia-hypoesthesia syndrome, painful ataxic hemiparesis, and hypoesthetic ataxic hemiparesis might be explained by variations in the blood supply to the capsulothalamic region.⁶⁶ When the blood supply is predominantly through the posterior circulation (thalamogeniculate arteries), infarction may be limited to the lateral thalamus, with hemiataxia-hypoesthesia syndrome or painful ataxic hemiparesis. On the other hand, when the blood supply is mainly through the anterior circulation (anterior choroidal artery), infarction may involve both the thalamus and the internal capsule,

leading to hypaesthetic ataxic hemiparesis.⁶⁷

Our findings suggest that there is no definite association between ataxic hemiparesis and lacunar infarct due to small artery disease. Uncommon locations of lesion occur—for instance, in the thalamus, cerebellum, lentiform nucleus, and cerebral cortex. Minor associated signs are the best predictors of a specific anatomical location, but they are often lacking. As ataxic hemiparesis has no aetiological, anatomical, or pathophysiological specificity, we think that it generally requires a comprehensive radiological and aetiological evaluation.

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