Vascular ataxic hemiparesis: a re-evaluation

Thierry Moulin, Julien Bogousslavsky, Jean-Luc Chopard, Joseph Ghika, Thierry Crèpin-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 (paraesthesias 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.

(Keywords: ataxic hemiparesis; lacunar infarction)

Several decades ago, French neurologists wrote on the clinical aspect of ataxic hemiparesis, though they did not use this term. Further reports emphasised several lesion sites, including the thalamus and frontal lobe. Fisher introduced the term ataxic hemiparesis in 1978 to designate a clinical picture associating hemiparesis with ipsilateral ataxia. 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). Initially described as a homolateral ataxia and cranial paresis, 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; however, it has also been found to involve the thalamus or cortical areas. Ataxic hemiparesis is not the commonest “lacunar syndrome” (<10%).

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 heel and 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 dysdiadochokinesia; (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.
Vascular ataxic hemiparesis: a re-evaluation

Table 1  Distribution of risk factors and type of stroke

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>No lesion leukoaraiosis (n = 23)</th>
<th>Deep infarct (n = 53)</th>
<th>Brainstem infarct (n = 14)</th>
<th>Superficial infarct (n = 6*)</th>
<th>Primary haemorrhage (n = 4*)</th>
<th>Total (n = 106)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>69-9</td>
<td>63-6</td>
<td>62-2</td>
<td>61-6</td>
<td>59-5</td>
<td>64-7</td>
</tr>
<tr>
<td>(SD)</td>
<td>(9-9)</td>
<td>(13-6)</td>
<td>(14-1)</td>
<td>(21-6)</td>
<td>(15-8)</td>
<td>(15-3)</td>
</tr>
<tr>
<td>Male/female</td>
<td>16/7</td>
<td>40/12</td>
<td>10/5</td>
<td>5/1</td>
<td>2/2</td>
<td>1/6</td>
</tr>
<tr>
<td>(% male)</td>
<td>(69-5)</td>
<td>(76-9)</td>
<td>(66-6)</td>
<td>(83-3)</td>
<td>(50)</td>
<td>(73)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>19*</td>
<td>28</td>
<td>14*</td>
<td>3</td>
<td>1</td>
<td>2 (62)</td>
</tr>
<tr>
<td>(% 3)</td>
<td>(83)</td>
<td>(53)</td>
<td>(79)</td>
<td>(50)</td>
<td>(25)</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>3</td>
<td>9</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>16</td>
</tr>
<tr>
<td>(%)</td>
<td>(13)</td>
<td>(17)</td>
<td>(21)</td>
<td>(16)</td>
<td>(0)</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>6</td>
<td>21</td>
<td>5</td>
<td>4</td>
<td>3</td>
<td>39</td>
</tr>
<tr>
<td>(%)</td>
<td>(26)</td>
<td>(40)</td>
<td>(36)</td>
<td>(67)</td>
<td>(75)</td>
<td></td>
</tr>
<tr>
<td>Hypercholest</td>
<td>6</td>
<td>13</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>24</td>
</tr>
<tr>
<td>(%)</td>
<td>(26)</td>
<td>(25)</td>
<td>(21)</td>
<td>(16)</td>
<td>(25)</td>
<td></td>
</tr>
<tr>
<td>PCV &gt;45%</td>
<td>11</td>
<td>28</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>48</td>
</tr>
<tr>
<td>(%)</td>
<td>(47)</td>
<td>(53)</td>
<td>(36)</td>
<td>(33)</td>
<td>(50)</td>
<td></td>
</tr>
<tr>
<td>Previous TIA</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>15</td>
</tr>
<tr>
<td>%</td>
<td>(4)</td>
<td>(16)</td>
<td>(21)</td>
<td>(16)</td>
<td>(25)</td>
<td></td>
</tr>
</tbody>
</table>

*One primary haemorrhage was considered as an intrasaffact haemotoma.

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 (territory 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 defined16: 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 χ2 test corrected by Fisher’s test, and analysis of variance (ANOVA) for volume measurement.

Table 2  Type of stroke

<table>
<thead>
<tr>
<th>Type of stroke</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>No visible lesion</td>
<td>14</td>
</tr>
<tr>
<td>Leukoaraiosis</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>23</td>
</tr>
<tr>
<td>Infarcts</td>
<td>72</td>
</tr>
<tr>
<td>Superficial infarct</td>
<td>5</td>
</tr>
<tr>
<td>ACA pial territory</td>
<td>2(*)</td>
</tr>
<tr>
<td>SCA pial territory</td>
<td>3</td>
</tr>
<tr>
<td>Deep infarct</td>
<td>53</td>
</tr>
<tr>
<td>Brainstem infarct</td>
<td>14</td>
</tr>
<tr>
<td>Pons (upper and medial part)</td>
<td>14</td>
</tr>
<tr>
<td>Primary haemorrhage</td>
<td>5</td>
</tr>
<tr>
<td>Lentiform nucleus</td>
<td>2</td>
</tr>
<tr>
<td>Corona radiata</td>
<td>1</td>
</tr>
<tr>
<td>Pons</td>
<td>1</td>
</tr>
<tr>
<td>Lobar (intrasaffact haemotoma; ACA)</td>
<td>1*</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
</tr>
</tbody>
</table>

*One primary haemorrhage was considered as an intrasaffact haemotoma.

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

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
haemorrhage in 6% (one infarct)
a haematomata 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 post-
erior 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 su-
perior part of the internal capsule located in the
posterior part of corona radiata near the ven-
tricular 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 hemor-
rhage 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
infranuclear haematomata), and three the su-
perior 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 cap-
Sule 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 hypercholestero-
laemia (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 pres-
umed 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 dis-
ease (thalamus (70%) and pons (80%)).

Presumed aetiologies were similarly distrib-
uted among the different subtypes and loca-
tions of stroke, except for embolic events,
which were more common with superficial
infaracts (50%) and with infarcts in the an-
terior 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 dysdiadochokineses were
found in only one quarter of the patients.
Face-upper limb-lower limb (FUL) hemi-
paresis was almost as frequent with pontine
(53%) and capsular lesions (57%). A partial
motor deficit (FU, UL or F/UL) was more
common with lentiform nucleus (67%) in-
volved, 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 radi-
ata (50%), lentiform nucleus (50%), and thal-
amus (40%). In infarcts involving the anterior

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

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>No lesion (n = 23)</th>
<th>Deep infarct (n = 53)</th>
<th>Brainstem infarct (n = 14)</th>
<th>Superficial infarct (n = 6*)</th>
<th>Primary haemorrhage (n = 4*)</th>
<th>Total (n = 100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undetermined</td>
<td>2</td>
<td>0</td>
<td>3</td>
<td>(33)</td>
<td>(22)</td>
<td>19</td>
</tr>
<tr>
<td>Large artery disease</td>
<td>2</td>
<td>6</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Embolic heart disease</td>
<td>1</td>
<td>8</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>AF</td>
<td>2</td>
<td>7</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Akinesia</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>MVP</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Hypertensive arteriopathology</td>
<td>16</td>
<td>30</td>
<td>10</td>
<td>-</td>
<td>1</td>
<td>59</td>
</tr>
</tbody>
</table>

*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.
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

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**Table 5 Clinical features and stroke location**

<table>
<thead>
<tr>
<th>Location</th>
<th>No motor deficit</th>
<th>FUL</th>
<th>FUL/UL</th>
<th>UL/UL</th>
<th>No F</th>
<th>Isolated ataxic hemiparesis</th>
<th>Paraesthesia</th>
<th>Dysarthria</th>
<th>Static ataxia</th>
<th>Nystagmus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal cortex (ACA)</td>
<td>0</td>
<td>0</td>
<td></td>
<td>0/0/3</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>1</td>
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<tr>
<td>territory</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Corona radiata</td>
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<td>4</td>
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<td>0/2/1</td>
<td>5</td>
<td>6</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>anterior part</td>
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<td>(30)</td>
<td>(30)</td>
<td>(30)</td>
<td>(60)</td>
<td>(60)</td>
<td>(10)</td>
<td>(4)</td>
<td>(10)</td>
<td>(30)</td>
</tr>
<tr>
<td>Corona radiata</td>
<td>0</td>
<td>4</td>
<td>0/3</td>
<td>0/2/1</td>
<td>5</td>
<td>6</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>posterior part</td>
<td>(44)</td>
<td>(30)</td>
<td>(26)</td>
<td>(22)</td>
<td>(44)</td>
<td>(44)</td>
<td>(11)</td>
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<td></td>
</tr>
<tr>
<td>Internal capsule</td>
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<td>12</td>
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<td>0/2/1</td>
<td>5</td>
<td>11</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>0</td>
</tr>
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<td>(57)</td>
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<td>(52)</td>
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<td>(9)</td>
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<td></td>
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</tr>
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<td>Leniform nucleus</td>
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<td>3</td>
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</tr>
<tr>
<td>Thalamus</td>
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<td>(20)</td>
<td>(60)</td>
<td>(10)</td>
<td>(80)</td>
<td>(20)</td>
<td>(30)</td>
<td>(30)</td>
<td>(0)</td>
</tr>
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<td>Pons</td>
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<td>3</td>
<td>3</td>
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<td>3</td>
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</tr>
<tr>
<td>Cerebellum (SCA) territory</td>
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<td>1/0/0</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(67)</td>
<td>(33)</td>
<td>(16)</td>
<td>(36)</td>
<td>(42)</td>
<td>(18)</td>
<td>(30)</td>
<td>(34)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
<td>36</td>
<td>9/15</td>
<td>2/7/3</td>
<td>28</td>
<td>32</td>
<td>14</td>
<td>23</td>
<td>26</td>
<td>3</td>
</tr>
<tr>
<td>(2/5)</td>
<td>(47)</td>
<td>(31)</td>
<td>(16)</td>
<td>(36)</td>
<td>(42)</td>
<td>(18)</td>
<td>(30)</td>
<td>(34)</td>
<td>(3)</td>
<td>(30)</td>
</tr>
</tbody>
</table>

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

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

<table>
<thead>
<tr>
<th>Antiology</th>
<th>No lesion</th>
<th>0.5 lesion</th>
<th>1.0 lesion</th>
<th>2.0 lesion</th>
<th>Total (n = 100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset:</td>
<td>Sudden</td>
<td>16</td>
<td>32</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Progressive</td>
<td>5</td>
<td>13</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Fluctuating</td>
<td>2</td>
<td>8</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Evolution:</td>
<td>No sequella</td>
<td>9</td>
<td>18</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>12</td>
<td>30</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>2</td>
<td>5</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

One primary haemorrhage was considered as an infarct-infarct haematoma.
deep infarcts (lacrimal infarct).\textsuperscript{17, 20, 26–29, 54} 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.\textsuperscript{20} In infarcts of the corona radiata, a haemodynamic mechanism in the end zone territory has been suggested.\textsuperscript{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.\textsuperscript{3, 35, 36} Size and location are probably determinant, so that infarcts could remain undetected when small or located in the brainstem.\textsuperscript{14, 15} 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 arteriolopathy were common. Only a few cases with a double lesion have been reported.\textsuperscript{37, 38} In a larger series using MRI, one-sixth of the cases examined for possible lacunar infarct showed double lesions,\textsuperscript{39} as in our study. We found that atactic 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.\textsuperscript{3, 35} Most haemorrhages involve the lenticulostriate region,\textsuperscript{29} and other locations (internal capsule,\textsuperscript{39} capsulothalamic region,\textsuperscript{40} and pons\textsuperscript{41–43}) are rare. In our study, lenticulostriate haemorrhages were larger than the other haemorrhages associated with atactic hemiparesis. On clinical grounds, it was not possible to distinguish atactic hemiparesis due to haemorrhage from atactic hemiparesis due to infarct.

The most common lesion associated with atactic hemiparesis was a small deep infarct.\textsuperscript{14, 15} As in recent studies,\textsuperscript{20, 26–29, 42–48} 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,\textsuperscript{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.\textsuperscript{48, 50} In atactic 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.\textsuperscript{21, 46–47, 51} Although part of this area could also be supplied by the lateral lenticulostriate arteries.\textsuperscript{20, 38–39, 41–45} 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,\textsuperscript{53} trigeminal nerve impairment,\textsuperscript{54} or slight ataxia in the contralateral lower extremity.\textsuperscript{55} We had no patient with a midbrain infarct.\textsuperscript{56}

Although atactic 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 atactic hemiparesis have commonly been reported.\textsuperscript{43, 55} In these cases, the corticopontocerebellar pathway (Türck's tract) may be affected within the frontal lobe, explaining "cerebellar" ataxia.\textsuperscript{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 5\% of the cases.\textsuperscript{35} Our findings do not support this. Infarcts of the superior cerebellar artery territory have rarely been reported with atactic hemiparesis,\textsuperscript{57} more often with dysarthria clumsy hand syndrome.\textsuperscript{54, 58–60}

Thalamic lesions with atactic hemiparesis have rarely been reported,\textsuperscript{43, 54, 55, 56–63} 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 atactic hemiparesis, cerebellar ataxia may be explained by the interruption of this afferent pathway. The lack of reports of atactic 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.\textsuperscript{43, 50} 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.\textsuperscript{44} Because of the commonly associated sensory disturbances, atactic hemiparesis may be less frequent than "hypeaesthetic atactic hemiparesis" or "painful atactic hemiparesis".\textsuperscript{44, 56–58} In "hemiataxia-hypaesthesia syndrome", also associated with small lateral thalamic infarcts, no sign of corticospinal dysfunction is present.\textsuperscript{65} These four syndromes, atactic hemiparesis, hemiataxia-hypaesthesia syndrome, painful atactic hemiparesis, and haepaesthesia atactic hemiparesis might be explained by variations in the blood supply to the capsulothalamic region.\textsuperscript{66} When the blood supply is predominantly through the posterior circulation (thalamogeniculate arteries), infarction may be limited to the lateral thalamus, with hemiataxia-hypaesthesia syndrome or painful atactic 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 hypoaesthetic 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.


