Multiple acute infarcts in the posterior circulation

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Abstract

Objective—to evaluate clinical, radiological, and prognostic features of patients with multiple acute infarcts in remote arterial territories of the posterior circulation.

Design—Data analysis from a prospective acute stroke registry in a community based primary care centre using a standard protocol including MRI and MRA.

Results—In three and a half years, 27 of the 236 patients (11%) with posterior circulation stroke had multiple acute infarcts in the posterior circulation as shown by gadolinium enhancement on MRI. Eighteen patients had multiple infratentorial and supratentorial infarcts including the cerebellum and posterior cerebral artery territory, with coexisting brainstem involvement in seven patients. Fourteen patients had a rostral basilar artery syndrome and cerebellar signs; four patients had a visual field defect with cerebellar signs. Causes were vertebral (six) or basilar (four) artery atheromatosis, and cardioembolism (four). Seven patients had multiple acute infarcts in the posterior circulation of the cerebellum and lower brainstem. Brainstem and cerebellar signs were found in most patients (five); aetiologies were small vessel disease (four), cardioembolism (one), and vertebral artery dissection (one). Two patients with large artery atheromatosis had multiple acute infarcts in the posterior circulation in the brainstem and posterior cerebral artery territory. One month after stroke more than 25% of the patients were dependent or had died. There was no difference in the outcome between the three groups, and recovery was linked to the size of infarcts rather than to a high number of infarcts.

Conclusions—multiple acute infarcts in the posterior circulation usually involve the cerebellum. Simultaneous brainstem and posterior cerebral artery territory infarcts sparing the cerebellum are uncommon. They can be suspected clinically before neuroimaging, mainly when supratentorial and infratentorial infarcts coexist. This may be important, because different patterns of infarction are associated with different causes of stroke.

Methods

We studied all patients who were admitted to our population based primary care centre over 42 months with a first stroke in the posterior circulation. These patients represented a subsample of 800 patients included consecutively in the Lausanne Stroke Registry. The main criterion for inclusion into the study was the presence of more than one acute infarct involving remote arterial territories in the posterior circulation, shown by gadolinium enhancement on MRI.

The infarcts were demonstrated by high field MRI (Siemens Magnetom SP 63 1·5 Tesla) usually conducted within one week and in all patients within 13 days of stroke onset. Studies with MRI included T1, T2, and proton weighted images (5 mm thick slices); contrast images (T1 sagittal and transverse 5 mm slices) were obtained after injection of gadolinium. Three dimensional MRA of vertebral and basilar arteries (3D TOF, 64 slices of 0·8–1 mm) was also performed in all patients. Basilar artery, intracranial vertebral arteries, the proximal portion of cerebellar arteries, and the posterior cerebral arteries were studied. The extracranial segment of vertebral arteries was studied from the changes in the opacification of the corresponding vessel on MRA with Doppler ultrasound confirmation.

Posterior circulation infarcts, other parenchymal changes, and relevant arterial lesions were recorded and classified into previously defined categories. The nine following topographic categories were considered:
Virchow-Robin
vertebral/basilar
bral
weighted
images)
images
ritories), midbrain, thalamus (posterior cere-
thalamic-subthalamic
tory
enhancement.
imals was also
recorded, including anterior circulation
infects, focal areas of hypertensive signal, and
diffuse leukoaraiosis. Arterial
lesions were
classified into seven categories: dolichoecstatic
vertebral/basilar arteries, vertebral artery
stenosis or occlusion, basilar artery stenosis or
occlusion, and occlusion of major branches of
the basilar artery (including posterior cerebral
artery). Acute infects were defined as
ischaemic lesions greater than an enlarged
Virchow-Robin perivascular space (which
demonstrates a high signal intensity on T1
weighted images and a high signal on T2
weighted images) and showing gadolinium
enhancement. An uninterrupted lesion visible
on two or more neighbouring slices on MRI
was considered to be an infect in a single arte-
torial territory, even if it involved more than one
of our nine topographic categories. Thus
infects involving simultaneously two different
contiguous anatomical structures (for exam-
ple, brainstem plus cerebellum or thalamus
plus mesencephalon) but corresponding to a
single vascular territory, and infarction over-
lapping adjacent territories (for example, pos-
terior inferior cerebellar artery and superior
cerebellar artery) were considered a single
lesion. Bilateral infect in the thalamoperforate
territory (paramedian, thalamic-subthalamic
artery) from the P1 segment of the posterior
cerebral artery was also classified as a single
lesion,26 as was infect in the posterior cere-
bral artery territory involving the superficial
(occipital-temporal) and deep (mesencephalo-
thalamic) region. Thus multiple acute infects
in the posterior circulation corresponded to
involvement of two or more of the three main
sequential segments of the posterior circula-
tion defined in the New England Medical
Center classification:17 proximal (medulla
and posterior inferior cerebellar artery cerebel-
hum fed by the intracranial vertebral arteries
and their branches); middle (pons, lower mid-
brain, and anterior inferior cerebellar artery
cerebellum fed by basilar artery and branches);
and distal (upper midbrain, thalamus, superior
cerebellar artery cerebellum and temporal and
occipital lobes fed by the distal basilar artery
and superior cerebellar artery, penetrating and
posterior cerebral artery branches). Infects
with a largest diameter ≤ 15 mm were classi-

cated as small.

Neurological and neuropsychological fea-
tures were assessed by at least two of the
authors including a senior neurologist (JB)
within five days of stroke. We analysed clinical
features and information about risk factors
defined following published guidelines:23
including hypertension (blood pressure higher
than 160/90 mm Hg at least twice before
stroke), diabetes mellitus (fasting blood glu-
cose concentrations above 6.0 mmol/l known
to exist before stroke), regular cigarette smok-
ing, hypercholesterolaemia (fasting blood
cholesterol above 6.5 mmol/L), venous packed cell
volume, heart disease (including angina
pectoris, old myocardial infect, chronic non-
valvar atrial fibrillation), and previous tran-
sient ischaemic attack(s). Apart from MRI and
MRA, systematic investigations included brain
CT on admission, three lead ECG monitoring
for at least 24 hours after admission, 12 lead
ECG, standard blood tests, Doppler ultra-
sonography with frequency spectral analysis,
and B-mode echotomography of the origin of
the carotid arteries and vertebral arteries.
Additional catheter cerebral angiography using
the Selddinger method was performed in two
patients, and transthoracic/transoesophageal
echocardiography was performed in 20
selected patients with presumed cardio-
vascular ischaemic stroke.

We considered the following potential
causes of stroke: (a) in situ atherosclerosis or
artery to artery embolism (large artery disease)
in patients with risk factors who had a stenosis
of at least 70% of the lumen diameter in the
appropriate large artery as shown by three
dimensional images on MRA or conventional
angiography using the NASCET method;
(b) small artery disease was presumed in
patients with longstanding hypertension or
diabetes mellitus (in the absence of potential
arterial or cardiac sources of emboli) and a
small (< 15 mm) infect limited to the terri-
tory of deep perforators; (c) potential cardiac
cardiac sources of embolism (mainly non-valvar atrial
fibrillation, left ventricular akinesic segment,
and other less common sources) were consid-
ered according to previously defined crite-
ria; (d) other and undetermined. The
evolution within the first four weeks after
stroke was recorded in all patients.

Results
Twenty seven (11%) of 236 patients with ver-
tebrobasilar ischaemic stroke had multiple
acute infects in the posterior circulation.
There were nine women (33%) and 18 men
(67%). The median age was 66 (range 32 to
87) years.

GENERAL AND NEUROLOGICAL VASCULAR
FEATURES
Nine patients (26%) had a history of
ischaemic heart disease and one patient (No
26) had non-valvar atrial fibrillation (table 1).
Eleven patients (41%) had hypertension, six
patients (22%) smoked cigarettes regularly,
four patients (15%) had hypercholester-
olaemia, four patients (15%) had diabetes, 12
patients (44%) had multiple risk factors, and
six patients (22%) had none of these risk
factors. Two patients (8%) had a patent foramen
ovale. Overall, age, sex, and risk profiles were
similar to those of the patients from the
Lausanne Stroke Registry in general.25 Three
patients (11%) had had transient ischaemic
attack(s) (five months, one week, four years
before stroke) in the posterior circulation.\textsuperscript{28} Sixteen patients (59\%) had a sudden non-progressive onset of symptoms, whereas stroke was progressive in 11 (44\%) patients (over 2–24 hours in five patients, over more than 24 hours to 15 days in six patients).

**TOPOGRAPHY OF INFARCTS (TABLES 1, 2, 3, AND 4)**

Our patients had from two to seven infarcts (median 3–6) including cerebellum (25), brainstem (17), occipital lobe (13), or thalamus (11). Cerebellar infarcts were bilateral in 80\% of the patients (20/25), brainstem infarcts occurred in 24\% (4/17) and occipitothalamic infarcts were found in 8\% of patients (2/24). In the 25 patients with cerebellar infarction the following vascular territories were involved: posterior inferior cerebellar artery in 22 patients (Nos 1–12, 14, 15, 18–20, 22–25, 27), bilaterally in 10; superior cerebellar artery in 15 patients (Nos 1, 3, 5, 6, 10–16, 18, 23, 24, 26), bilaterally in five; anterior inferior cerebellar artery in five patients (Nos 4, 5, 10, 12, 24). In patients with brainstem infarction, the territory of the paramedian and circumferential arteries was involved in 13 patients (Nos 1, 4, 5, 7, 9, 14, 17–19, 21, 22, 23, 25), and the territory of the short circumferential arteries from the P2 segment of the posterior cerebral artery was involved in four patients (Nos 10, 11, 17, 24). The occipital lobe was affected in 13 patients (Nos 1, 2, 3, 6, 8, 12–14, 18, 21, 23, 26, 27), bilaterally in only one (No 6). The thalamus was involved in 11 patients (Nos 1–3, 10, 11, 12, 15–18, 20), bilaterally in two (Nos 16, 17) (in six patients the infarct was localised to the territory of the thalamogeniculate arteries\textsuperscript{26} (inferolateral pedicle\textsuperscript{29}), in five patients to the paramedian territory\textsuperscript{29} (thalamic-subthalamic arteries\textsuperscript{29}). No patient had a medullary infarct.

We identified three clinicotopographic patterns of multiple acute posterior circulation infarcts (tables 1, 2, and 3):

1. **Multiple infratentorial and supratentorial infarcts of cerebellum and posterior cerebral artery (or proximal and/or middle + distal multiple acute infarcts in the posterior circulation (Nos 17, 27)):** 18 (67\%)

Seven patients (39\%) also had a brainstem infarct (table 1). Most of the patients (78\% = 14/18) clinically had a rostral basilar artery syndrome\textsuperscript{30} (including hemianopia in seven patients) and cerebellar signs; four other patients had a visual field defect and cerebellar signs (table 4). A history of ischaemic heart disease (39\%) and hypertension (33\%) was common. Contrast angiography was normal in one patient (No 15) without risk factors. The presumed cause of stroke was large artery disease in nine patients: focal (two) atheromatosis of the basilar artery; multifocal atheromatosis (one), and focal stenosis (one) or occlusion (two) of one intracranial vertebral artery. One patient (No 8) had a tandem intracranial atheromatous stenosis with diffuse narrowing of the basilar artery associated with a stenosis of the posterior cerebral artery. One patient (No 11) had an occlusion of the proximal right vertebral artery and a distal focal stenosis of the left vertebral artery; one patient (No 23) had coexisting focal stenosis of the basilar artery and left vertebral artery. Cardioembolism was the likely aetiology in five patients with a history of myocardial infarct with akinetic left ventricular segment, and non-valvar atrial fibrillation. A 48 year old man (No 12) died two days after stroke: he had developed headache, vomiting, left sensory-motor hemiparesis, and ataxia, with rapid coma. Brain MRI showed multiple, large and bilateral infarcts in all three cerebellar and both posterior cerebral artery territories. Brain MRA suggested an occlusion in the distal portion of the basilar artery and left posterior inferior cerebellar artery, with otherwise intact arteries. Although transthoracic echocardiography was normal, a cardioembolic mechanism was suspected. Aetiology remained unknown in three patients, and uncertain in two patients with only a patent foramen ovale (No 24, 27). One month after stroke 13 patients (72\%) were independent, and four (22\%) were still dependent.

2. **Multiple infratentorial infarcts of cerebellum and lower brainstem (or proximal + middle multiple acute infarcts in the posterior circulation (Nos 17, 27)):** seven (26\%)

These patients showed brainstem signs sometimes combined with cerebellar ataxia (table 4). Presumed aetiology was hypertensive small artery disease in four patients (57\%). A 51 year old patient (No 4) had IgM anticardiolipin antibodies without any other potential cause of stroke. Another patient (No 5) had a proximal occlusion of the right vertebral artery related to a subintimal dissection detected on conventional angiography. The likely cause of stroke was cardioembolism in a 55 year old woman with coronary heart disease and an akinetic left ventricular segment. All but one patient was independent one month after stroke (table 2).

3. **Multiple infarcts of brainstem and posterior cerebral artery (or proximal + middle multiple acute infarcts in the posterior circulation (Nos 17, 27)):** two (7\%)

Only two patients had multiple supratentorial and infratentorial infarcts including pons and both posterior cerebral artery territories, but sparing the cerebellum (table 3). These patients had brainstem signs (table 4); MRA showed basilar artery or vertebral artery atheromatosis in both patients. In patient No 17 there was an extension of a thrombus from the intracranial vertebral artery to the proximal basilar artery.

**CONCOMITANT VASCULAR DISEASES**

No patient had had a recent ischaemic event in the anterior circulation, and none had an old infarct in the posterior circulation. A carotid stenosis was found in one patient, who had had an episode of amaurosis fugax six years before the present stroke (No 10). An asymptomatic carotid stenosis was detected in four patients, including an intracranial stenosis in one patient (No 8). Nine patients (Nos 3–9,
Table 1 Infratentorial and supratentorial multiple acute infarcts in the posterior circulation (cerebellum, posterior cerebral artery and brainstem; proximal and/or middle + distal (Nos 17, 27))

<table>
<thead>
<tr>
<th>Topography</th>
<th>Large artery lesion</th>
<th>Cardiac lesion</th>
<th>Stroke mechanism</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>2</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>4</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>5</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>6</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Dependent</td>
</tr>
<tr>
<td>7</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>8</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>9</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>10</td>
<td>None</td>
<td>Akinesia + aneurysm</td>
<td>Cardioembolism</td>
<td>Dependent</td>
</tr>
<tr>
<td>11</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>12</td>
<td>None</td>
<td>None</td>
<td>Cardioembolism</td>
<td>Dead</td>
</tr>
<tr>
<td>13</td>
<td>None</td>
<td>None</td>
<td>?</td>
<td>Independent</td>
</tr>
<tr>
<td>14</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>15</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>16</td>
<td>None</td>
<td>Parietal thrombus</td>
<td>Cardioembolism</td>
<td>Dependent</td>
</tr>
<tr>
<td>17</td>
<td>None</td>
<td>None</td>
<td>?</td>
<td>Independent</td>
</tr>
<tr>
<td>18</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
<tr>
<td>19</td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Dependent</td>
</tr>
<tr>
<td>20</td>
<td>None</td>
<td>Patent foramen ovale</td>
<td>Paradoxical embolism</td>
<td>Independent</td>
</tr>
<tr>
<td>21</td>
<td>None</td>
<td>Atrial fibrillation</td>
<td>Cardioembolism</td>
<td>Independent</td>
</tr>
<tr>
<td>22</td>
<td>None</td>
<td>Patent foramen ovale</td>
<td>Paradoxical embolism</td>
<td>Independent</td>
</tr>
</tbody>
</table>

LAD = large artery disease.
Multiple acute infarcts in the posterior circulation

Table 2  Cerebellum and lower brainstem multiple acute infarcts in the posterior circulation (proximal + middle (Nos 17, 27))

<table>
<thead>
<tr>
<th>Topography</th>
<th>Large artery lesion</th>
<th>Cardiac lesion</th>
<th>Stroke mechanism</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>Anticardiolipin antibodies</td>
<td>Independent</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>LAD (dissection)</td>
<td>Dependent</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>SAD</td>
<td>Independent</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>SAD</td>
<td>Independent</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>SAD</td>
<td>Independent</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>SAD</td>
<td>Independent</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>Akinesia</td>
<td>Cardioembolism Independent</td>
</tr>
</tbody>
</table>

SAD = small artery disease; LAD = large artery disease.

11, 20–22) had leukoaraiosis. Two patients (Nos 10, 16) with multiple infratentorial and supratentorial infarcts, possibly of cardioembolic origin, also had a dolichoectatic basilar artery. We found no patient with arteritis or another angiopathy. One patient (No 8) with peripheral arteriopathy and one patient (No 11) with aortocoronary bypass were under anticoagulant therapy at the onset of stroke. No patient was under antiaggregant therapy at the time of the index stroke.

Discussion

Haemorrhagic transformation of infarct was only seen in one non-anticoagulated patient who had three infarcts (No 10). One patient (No 5) with bilateral large cerebellar infarcts and a pontine infarct developed a compression of the fourth ventricle with hydrocephalus. He improved after external ventricular drainage. One month after stroke, 20 patients (74%) were independent, six were dependent, and one had died. This contrasted with only 19 (9%) of the 209 patients with one single infarct being dependent at one month (P < 0.01). Of 20 patients (Nos 1, 2, 4, 5, 6, 8, 10–18, 20, 23, 24, 26, 27; 74%) with large infarcts (>3 cm, >2 slices on MRI), six (30%) had a poor functional outcome and one died. On the other hand, 12 of 18 patients (67%) with more than two infarcts were independent one month after stroke.

Table 3  Brainstem and posterior cerebral artery territory multiple acute infarcts in the posterior circulation (proximal + middle (Nos 17, 27))

<table>
<thead>
<tr>
<th>Topography</th>
<th>Large artery lesion</th>
<th>Cardiac lesion</th>
<th>Stroke mechanism</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Dependent</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>None</td>
<td>LAD</td>
<td>Independent</td>
</tr>
</tbody>
</table>

LAD = large artery disease.
tory on MRI or CT, Struck et al\textsuperscript{11} reported that 11 had associated infarcts involving other cerebellar, brainstem, and supratentorial territories, but the number of patients without an MRI was not specified. Caplan et al\textsuperscript{17} reported 10 patients with occlusive disease of the proximal vertebral artery and embolism to the posterior circulation: in five subjects MRI showed supratentorial and infratentorial infarcts involving distinct territories. Tettenborn et al\textsuperscript{12} reported that of 22 patients with postoperative brainstem and cerebellar infarcts, 10 had multiple infarcts in the posterior circulation. However, these studies did not discuss the topographic, clinical, and prognostic features associated with multiple infarcts. More recently, two reports emphasised that multifocal brainstem\textsuperscript{13} or cerebellar\textsuperscript{17} infarction has been largely overlooked, but without a systematic study of acute multiple posterior circulation infarcts.

Most of our patients (93%) had a cerebellar component of acute multiple posterior circulation infarcts, often bilateral, which involved the posterior inferior cerebellar artery territory more often than in reported series of "pure" cerebellar infarcts.\textsuperscript{9,22,34,36} A supratentorial component of multiple acute posterior circulation infarcts was present in 90% of the patients and brainstem infarction in two thirds.

The main clinical presentation in our 20 patients with acute multiple supratentorial and infratentorial infarcts (groups 1 and 3) was a partial and reversible rostral basilar artery syndrome\textsuperscript{17,38} in all but one patient, with cerebellar signs and a visual field defect in about half of the patients. No patient had clinical evidence for involvement of only one single vascular territory and none of those with a presumed small artery disease (group 2) had a lacunar syndrome.\textsuperscript{17}

Stroke was seldom preceded by transient ischaemic attack compared with other posterior circulation infarcts (18\%,\textsuperscript{38} 23\%,\textsuperscript{39} 57\%).\textsuperscript{48} Stroke onset was not progressive in about two thirds of the patients, without significant difference between the aetiological subgroups (artery to artery embolism, cardioembolism, local thrombosis, small artery disease). This suggests that infarcts mostly came on simultaneously.

### Table 4: Topography, clinical features, and outcome of 27 patients with multiple acute infarcts in the posterior circulation

<table>
<thead>
<tr>
<th>Vascular territory</th>
<th>No TIA</th>
<th>Onset</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infratentorial and supratentorial MAPCI (cerebellum, PCA and brainstem)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 PICA, l + m; SCA, l + m; p; PCA; Th (ii)</td>
<td>7, b</td>
<td>s</td>
<td>HH, CNP (VII), limb and trunk ataxia, dystagnus</td>
</tr>
<tr>
<td>2 PICA, l + m; SCA, l; Th (ii); PCA</td>
<td>4, b</td>
<td>s</td>
<td>HH, dystagnus, dysarthria, M + S (l, u, l), limb and trunk ataxia, asterixis</td>
</tr>
<tr>
<td>3 PICA, m; SCA, m; PCA</td>
<td>6, b</td>
<td>p</td>
<td>HH, vertikal gaze palsy, dysarthria, limb ataxia</td>
</tr>
<tr>
<td>6 PICA, l + m; SCA, l + m; PCA</td>
<td>6, b</td>
<td>p</td>
<td>HH, dystagnus, dysarthria, limb ataxia, CNP (asydnergia, visual hallucinations, aachromatopsia)</td>
</tr>
<tr>
<td>8 PICA, m; PCA</td>
<td>2, b</td>
<td>s</td>
<td></td>
</tr>
<tr>
<td>10 PICA, l; AICA; SCA, l; m (sc); Th (ii)</td>
<td>3, u</td>
<td>s</td>
<td>Somaalonge, vertege palsy, CNP (III), dysarthria, dystagnus, M (l, u, l), limb ataxia</td>
</tr>
<tr>
<td>11 PICA, l + m; SCA, l + m; m (sc); Th (ii)</td>
<td>6, b</td>
<td>p</td>
<td>CPN (III), dysarthria, limb and trunk ataxia, NPsy (anesia, apathy, confusion)</td>
</tr>
<tr>
<td>12 PICA, l; AICA; SCA, l + m; PCA; Th (ii)</td>
<td>3, b</td>
<td>s</td>
<td>Somaalonge/coma, dystagnus, M + S (l, u, l), limb and trunk ataxia</td>
</tr>
<tr>
<td>13 SCA, l + m; PCA</td>
<td>2, b</td>
<td>s</td>
<td>HH, limb and trunk ataxia</td>
</tr>
<tr>
<td>14 PICA, l; PCA, l + m; PCA</td>
<td>4, b</td>
<td>s</td>
<td>HH, dysarthria, limb and trunk ataxia</td>
</tr>
<tr>
<td>15 PICA, l + m; SCA, m; Th (pm)</td>
<td>4, b</td>
<td>p</td>
<td>Stpor, M (t, u, l), NPsy (apathy, subcortical aphasia), asterixis, dysarthria</td>
</tr>
<tr>
<td>16 SCA, m; Th (pm)</td>
<td>2, b</td>
<td>s</td>
<td>Stpor, vertege palsy, CNP (III, IV), limb and trunk ataxia, M (u), NPsy (dementia)</td>
</tr>
<tr>
<td>18 PICA, l; SCA, m + l; p; PCA; Th (pm)</td>
<td>7, b</td>
<td>p</td>
<td>HH, Horners syndrome, M (u, l), dysarthria, NPsy (confusion, apathy, anosmia)</td>
</tr>
<tr>
<td>20 PICA, l + m; Th (pm)</td>
<td>3, b</td>
<td>s</td>
<td>Somaalonge vertege palsy, TP (m, u), NPsy (confusion), dysarthria</td>
</tr>
<tr>
<td>23 PICA, l + m; SCA, l; PCA</td>
<td>4, b</td>
<td>s</td>
<td>HH, &quot;one and a half&quot; palsy, CNP (V, VII), dysphagia, dysarthria, M + S (l, u, l), limb ataxia</td>
</tr>
<tr>
<td>24 PICA, l + m; SCA; AICA; m (sc)</td>
<td>3, b</td>
<td>s</td>
<td>Ske deviation, M (u), trunk ataxia, dysarthria, NPsy (apathy, emotional lability, apraxia)</td>
</tr>
<tr>
<td>26 SCA, m; PCA</td>
<td>2, b</td>
<td>s</td>
<td>HH, dysarthria, limb and trunk ataxia</td>
</tr>
<tr>
<td>27 PICA, l; PCA; Th (ii)</td>
<td>3, u</td>
<td>s</td>
<td>HH, trunk ataxia, NPsy (alexia, aachromatopsia, amnesia), S (l, u)</td>
</tr>
<tr>
<td><strong>Cerebellum and lower brainstem MAPCI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 PICA, l; AICA; p</td>
<td>2, u</td>
<td>s</td>
<td>Saccadic horizontal pursuit, CNP (VIII, cochlear), M (u, l), limb and trunk ataxia</td>
</tr>
<tr>
<td>5 PICA, l + m; AICA; p</td>
<td>3, b</td>
<td>+, p</td>
<td>Somaalonge/coma, dystagnus, CNP (V, VI, VII), dysarthria, limb and trunk ataxia, S (l, u)</td>
</tr>
<tr>
<td>7 PICA, l + m; p</td>
<td>3, b</td>
<td>s</td>
<td>Somaalonge horizontal pursuit, CNP (VII, limb and trunk ataxia)</td>
</tr>
<tr>
<td>9 PICA, m; p</td>
<td>2, b</td>
<td>s</td>
<td>Dysarthria, M (ataxic hemiparesis)</td>
</tr>
<tr>
<td>19 PICA, l; p</td>
<td>2, b</td>
<td>p</td>
<td>Dysarthria, M (l, u, l)</td>
</tr>
<tr>
<td>22 PICA, m; p</td>
<td>2, u</td>
<td>s</td>
<td>Saccadic horizontal pursuit, dysarthria, M (l, u, l)</td>
</tr>
<tr>
<td>25 PICA, l; p</td>
<td>2, u</td>
<td>s</td>
<td>CNP (V, VI), trunk ataxia</td>
</tr>
<tr>
<td><strong>Brainstem and PCA territory MAPCI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17 p; m (sc); Th (tt, pm)</td>
<td>5, b</td>
<td>+, p</td>
<td>Vertical gaze palsy, Horners syndrome, pseudo—VI, M (ataxic hemiparesis), NPsy (visual and auditory hallucinations), dystagnus</td>
</tr>
<tr>
<td>21 p; PCA</td>
<td>3, b</td>
<td>s</td>
<td>Dysarthria, M (ataxic hemiparesis)</td>
</tr>
</tbody>
</table>

**TIA** = transient ischaemic attack; **No** = number of infarcts; **u/b** = unilateral, bilateral; **s** = same/other vascular territory; **NPsy** = neurological disturbance; **M5** (l, u, l) = motor weakness (motor sensory disturbance (face (l), upper (u), lower (l) limb); **HH** = homonymous hemianopia; **CNP** = cranial nerve palsy (III-XII).

Vascular territories—Cerebellum: PICA, m/l = posterior inferior cerebellar artery, media/lateral branch; AICA = anterior inferior cerebellar artery; SCA, m/l = superior cerebellar artery, media/lateral branch; Thalamos = th; u = thalamous subthalamic artery (polar artery); Th, pm = paramedian artery (thalamo-subthalamic artery); th, il = inferolateral artery (thalamolencephalitic artery). Occipital lobe: PCA = cortical branches of posterior cerebral artery. Mesencephalon: m; sc = short circumferential arteries from PCA (P2 segment). Pons: p = paramedian, short and long circumferential arteries.

Multiple acute infarcts in the posterior circulation

No particular risk factor correlated with a poor outcome. There was no difference in the outcome between patients with multiple infratentorial and supratentorial infarcts including the posterior cerebral artery, patients with multiple infratentorial infarcts of the cerebellum and brainstem, and the two patients with multiple infarcts of the brain stem and posterior cerebral artery territory. Nevertheless, multiple acute posterior circulation infarcts were clearly associated with a worse prognosis than single infarcts in the posterior circulation, as 26% of patients were dead or dependent one month after stroke. However, regardless of distribution of the infarcts, 75% of our patients with multiple acute infarcts in the posterior circulation were functionally independent one month after stroke. Moreover, there was the same proportion of patients with a good outcome in the groups with either more or less than five infarcts. On the other hand, the patients with large infarcts had a poorer outcome, suggesting that size rather than a large number of infarcts correlates with a poor outcome.

Besides atheroma and embolism, multiple infarction may be related to angiopathies (isolated angiitis, amyloid angiopathy, infectious arteritis, polyarteritis nodosa, Wegener's granulomatosis, giant cell arteritis, Behcet's disease, hypersensitivity vasculitis), coagulopathies, and familial conditions (CADASIL, MELAS), but none of our patients had any of these conditions. Atheromatosis of the large arteries was the most frequent presumed cause of infarcts in our series (4%, table 1), independently of the topography of the lesions. This is not surprising as there was a preponderance of elderly men. Several studies showed that occlusion of rostral basal territory branches (posterior cerebral artery, distal basal artery, and superior cerebellar artery) and intracranial vertebral artery/posterior inferior cerebellar artery is usually embolic, intrinsically atherosclerotic disease being more uncommon. As previously underlined in single infarction in the posterior circulation, the proximal segment of the vertebral artery was also suspected to be the embolic source in most of our patients with supratentorial and infratentorial infarcts, suggesting that extracranial vertebral artery disease may play an important part in posterior circulation stroke. On the other hand, no "referential" localisation of atheromatosis along the basilar artery was found in our patients, and none of them showed a midbasilar occlusion, which may sometimes be associated with a benign clinical course. Atheromatosis may also occur the origin of vertebral artery and basilar artery branches, particularly involving the smaller penetrating vessels rather than larger branches (cerebellar or anterior spinal arteries). Small artery disease was uncommon (15%) in our patients, although it was relatively more frequent in the patients with multiple, small infarcts (60%).

The frequency, location, and severity of carotid atheroma in patients with vertebrobasilar occlusive disease is variable (40%, 54%). We did not find a correlation between posterior circulation and carotid artery atheroma, suggesting that it may develop independently in each of these locations.

Nearly 20% of our patients had a potential cardiac source of embolism. This seems similar to what has been reported for ischaemic stroke in general, including unselected patients with posterior circulation infarcts. Indeed cerebellar and posterior cerebral artery territory infarcts are commonly due to cardioembolic embolism. In 15% of our patients, the cause of stroke remained unknown. However, transoesophageal echocardiography was performed in only a few patients and atherosclerotic plaques in the aortic arch were not ruled out.

Overall, our findings suggest that embolism, from an arterial or a cardiac source, is the main aetiology of multiple acute infarcts in the posterior circulation. Whether embolism causes multiple territory infarction by proximal occlusion, or whether one or several distal haemodynamic infarction, or it is the result of multiple emboli or a single embolus breaking up and dispersing into different sites is not clear from our study.

Our findings emphasise the heterogeneity of clinical, topographic, and aetiological aspects of multiple acute posterior circulation infarcts. This suggests that early recognition of these types of infarct may have implications for emergency therapy options in the acute phase of stroke, including acute stroke trials assessing fibrinolytic or anti-ischaeamic drugs.

296


