The Syndrome of Combined Polar and Paramedian Thalamic Infarction

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**Background:** Occlusion of the polar or the paramedian arteries of the thalamus usually leads to distinct infarcts with specific clinical and imaging correlates. However, vascular variation is such that in up to one third of humans, the polar artery is missing and its territory taken over by the paramedian arteries.

**Objective:** To provide attention to the corresponding stroke syndrome of combined polar and paramedian thalamic infarction.

**Methods:** We studied combined polar-paramedian thalamic infarction in 12 patients (6 right-sided lesions, 3 left-sided lesions, and 3 bilateral lesions) who were selected from 208 consecutively registered patients with thalamic strokes in the Lausanne Stroke Registry.

**Results:** The clinical manifestation included executive dysfunction, apathy, and memory impairment in all patients, with eye movement disturbances in 10 patients (5 with right-sided lesions, 2 with left-sided lesions, 3 with bilateral lesions); acutely impaired consciousness in 11 patients (5 with right-sided lesions, 3 with left-sided lesions, 3 with bilateral lesions); aphasic disturbances in 8 patients (2 with right-sided lesions, 3 with left-sided lesions, 3 with bilateral lesions), including non-fluent aphasia in 1 patient (with left-sided lesions); dysarthria in 5 patients (4 with right-sided lesions, 1 with bilateral lesions); constructional apraxia in 5 patients (with right-sided lesions); mild hemiparesis in 4 patients (2 with right-sided lesions, 2 with left-sided lesions); dyscalculia in 3 patients (1 with left-sided lesions, 1 with right-sided lesions, 1 with bilateral lesions); limb dystonia or asterixis in 2 patients (1 with right-sided lesions, 1 with bilateral lesions); mild hemisensory loss in 2 patients (1 with right-sided lesions, 1 with left-sided lesions); hemiataxia in 1 patient (with right-sided lesions); and ideomotor apraxia in 1 patient (with left-sided lesions). Follow-up showed severely disabling, persistent amnesia in 7 patients (4 with right-sided lesions, 3 with bilateral lesions) and persistent eye movement dysfunction in 5 patients (2 with right-sided lesions, 1 with left-sided lesions, 2 with bilateral lesions). The most common etiology appeared to be cardioembolism, followed by artery-to-artery embolism and presumed small-artery disease.

**Conclusions:** Key features of this syndrome included amnesia preceded by a period of altered consciousness, and vertical eye movement disturbances. The severe and persistent amnesia may be due to coexisting damage to the anterior and dorsomedial nuclei.

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**Thalamic Infarcts**

Thalamic infarcts account for 11% of vertebrobasilar infarcts and, on the basis of clinicoradiological-anatomical studies, can be divided into 4 groups based on the 4 main arterial territories. Inferolateral infarcts are the most common (45%). The second (35%) and third (12.5%) most frequent thalamic infarcts involve the territory of the paramedian or the polar arteries, respectively, with clinical syndromes that are well recognized. The paramedian artery syndrome includes acutely lost or decreased consciousness (usually transient), frequently followed by neuropsychological and eye movement (upward gaze) disturbances but few motor or sensory deficits; the polar artery syndrome is mainly characterized by neurobehavioral deficits, which, depending on the side of the lesion, usually affect language or visuospatial functions and, less commonly, memory.

However, the vascular supply to the thalami is far from homogenous. Indeed, in one third of individuals, the territory of the polar thalamic artery is taken over, unilaterally or bilaterally, by the paramedian thalamic artery. The clinical syndrome that results from such a combined polar-paramedian thalamic stroke has received little attention.

**Methods**

At the time of study, the Lausanne Stroke Registry included 208 patients with thalamic stroke. Patients with previous stroke or mul-
ultiple or coexisting brain lesions were excluded. Among the 208 patients, we identified 12 patients (5.8%) in whom the lesion was confined to the territories of both the polar and paramedian arteries, either unilaterally (3 left-sided, 6 right-sided) or bilaterally (3 patients). The computed tomographic scans and magnetic resonance images of these patients were mapped onto templates (Figure 1 and Figure 2) according to our previously reported procedure.10,11 All patients had been examined clinically by at least 1 of us (J.B.) and had undergone a comprehensive neuropsychological examination using the Lausanne battery of tests.12 Severity of memory impairment was defined operationally as an inability to lead an independent life.

Echocardiography, 3-lead echocardiography, echocardiography monitoring, and ultrasound examination of the neck and intracerebral arteries were performed on all patients. For follow-up examination, consisting of a clinical neurological examination and a comprehensive neuropsychological examination, all patients were seen at least twice at intervals ranging from 2 months to 8 years.

RESULTS

Twelve patients (6 men, 6 women; 6 right-sided lesions, 3 left-sided lesions, 3 bilateral lesions; mean ± SD age, 54.5 ± 23.5 years) met the template criteria for combined polar-paramedian thalamic infarction (Table).10,11 The lesions in all 12 patients were drawn and are shown in Figure 1 and Figure 2.

Risk factors (hypertension [blood pressure >160/90 mm Hg], hypercholesterolemia [cholesterol level >239.75 mg/dL, 6.2 mmol/L], diabetes mellitus [fasting glucose level >120.6 mg/dL, 6.7 mmol/L], and active smoking) were present in 8 patients. The suspicion of cardiac or artery-to-artery embolism and microangiopathy as the etiology was based on the results of cardiac monitoring, echography, Doppler ultrasound, and laboratory tests. The most common etiology appeared to be cardioembolism (9 of 12 patients; 5 had patent foramen ovale [3 with septal aneurysm] and the remaining 4 had atrial fibrillation), followed by artery-to-artery embolism (2 of 12 patients) and presumed, isolated small-artery disease (1 of 12 patients).
## Table. Clinical Information of the 12 Patients

<table>
<thead>
<tr>
<th>Patient/ Sex/Age, y</th>
<th>Anatomical Lesions</th>
<th>Memory Impairment</th>
<th>Impairment of Consciousness</th>
<th>Ocular Impairment</th>
<th>Apraxia</th>
<th>Language</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/31</td>
<td>A, VA, DM, VL, LD, midbrain</td>
<td>Verbal anterograde</td>
<td>Severe</td>
<td>Horizontal and vertical palsy, skew deviation, right hypotropia</td>
<td>None</td>
<td>Confabulations, paraphasias</td>
<td>Executive dysfunction, aphathy, mild hemiparesis</td>
</tr>
<tr>
<td>3/F/59</td>
<td>A, DM, VL, LD, midbrain</td>
<td>Verbal anterograde</td>
<td>Sleepy</td>
<td>Left hemianopsia, exotropia, vertical palsy</td>
<td>None</td>
<td>Paraphasias</td>
<td>Executive dysfunction, aphathy, mild hemiparesis, and hemisensory loss</td>
</tr>
<tr>
<td>9/F/78</td>
<td>A, DM, LD</td>
<td>Verbal anterograde</td>
<td>Sleepy</td>
<td>None</td>
<td>Ideomotor</td>
<td>None</td>
<td>Executive dysfunction, dyscalculia, aphathy</td>
</tr>
<tr>
<td>4/M/48</td>
<td>A, VL, DM, LD</td>
<td>Moderate visuospatial</td>
<td>Sleepy</td>
<td>Hemianopsia, conjugate right gaze deviation</td>
<td>Constructional</td>
<td>Dysarthria</td>
<td>Executive dysfunction, dyscalculia, aphathy, mild hemiparesis, and hemisensory loss</td>
</tr>
<tr>
<td>5/M/35</td>
<td>A, VA, DM, LD, midbrain</td>
<td>Severe visuospatial</td>
<td>Sleepy</td>
<td>Horizontal and vertical palsy</td>
<td>Constructional</td>
<td>Dysarthria</td>
<td>Executive dysfunction, hypersexuality, jocularity, aphathy, mild hemiparesis, hemiataxia</td>
</tr>
<tr>
<td>6/F/48</td>
<td>A, VA, DM, LD, midbrain</td>
<td>Global severe anterograde</td>
<td>Normal</td>
<td>Vertical palsy, oblique diplopia</td>
<td>Visuoconstructual</td>
<td>Slight dysarthria</td>
<td>Executive dysfunction, anosodiaphoria, aphathy, mild hemiparesis, limb dystonia</td>
</tr>
<tr>
<td>8/F/65</td>
<td>A, VL, DM, LD</td>
<td>Severe anterograde</td>
<td>Sleepy</td>
<td>Left hemianopsia</td>
<td>Visuoconstructual</td>
<td>Paraphasia</td>
<td>Executive dysfunction, aphathy, mild hemiparesis</td>
</tr>
<tr>
<td>10/F/64</td>
<td>A, VA, DM, LD, midbrain</td>
<td>Severe anterograde</td>
<td>Sleepy, atymnoria</td>
<td>Vertical palsy</td>
<td>None</td>
<td>Paraphasia</td>
<td>Executive dysfunction, abulia, aphathy</td>
</tr>
<tr>
<td>12/M/65</td>
<td>A, VA, VL, DM, LD, midbrain</td>
<td>Moderate anterograde</td>
<td>Sleepy</td>
<td>Vertical palsy</td>
<td>Constructional</td>
<td>Dysarthria</td>
<td>Executive dysfunction, aphathy</td>
</tr>
<tr>
<td>2/F/65</td>
<td>A, VA, DM, VL, LD, midbrain</td>
<td>Severe anterograde</td>
<td>Severe, coma</td>
<td>Horizontal and vertical palsy, skew deviation</td>
<td>None</td>
<td>Confabulations, paraphasia, word-finding difficulty</td>
<td>Executive dysfunction, aphathy, asterixis, limb dystonia</td>
</tr>
<tr>
<td>7/M/68</td>
<td>A, VA, DM, midbrain</td>
<td>Severe anterograde</td>
<td>Severe, coma</td>
<td>Vertical palsy, bilateral ptosis</td>
<td>None</td>
<td>Dysarthria, word-finding difficulty</td>
<td>Executive dysfunction, aphathy, disinhibition</td>
</tr>
<tr>
<td>11/M/68</td>
<td>A, VA, DM, LD, midbrain</td>
<td>Severe anterograde</td>
<td>Severe, coma</td>
<td>Vertical palsy</td>
<td>None</td>
<td>Paraphasia</td>
<td>Executive dysfunction, hypersexuality, hyperphagia</td>
</tr>
</tbody>
</table>

**Abbreviations:** A, anterior nucleus; DM, dorsomedial nucleus; LD, laterodorsal nucleus; VA, ventroanterior nucleus; VL, ventrolateral nucleus.

## CLINICAL MANIFESTATIONS

### Left-Sided Infarcts

All 3 patients had an acutely impaired state of consciousness, and all had aphathy and executive dysfunctions. Memory was impaired in all patients but only in the verbal domain. Phonemic paraphasias were present in 2 patients and nonfluent aphasia in 1. All but 1 patient had complex eye movement disturbances (horizontal and vertical palsy, skew deviation, exotropia or hypotropia). Dyscalculia and ideomotor apraxia was observed in 1 patient. A rapidly regressive, mild hemiparesis was found in 2 patients, 1 with a slight hemisensory loss affecting touch and pinprick.

### Right-Sided Infarcts

All had aphathy and executive dysfunctions and an impaired memory. In 4 patients, this memory impairment was global anterograde (verbal and visuospatial) and severe (Rey auditory verbal learning and Rey visuospatial learning scores were zero or close to zero for spontaneous recall and were not improved by categorical clues); in the remaining 2 patients, there was memory impairment only in the visuospatial domain. Five patients had an acutely impaired state of consciousness. Five patients had complex eye movement disturbances (horizontal and vertical gaze palsy, skew deviation). There was constructional apraxia in 5 patients, dysarthria in 4, and dyscalculia in 1. A rapidly regressive mild hemiparesis was found in 4 patients, 1 with a slight hemisensory loss affecting touch and pinprick, 1 with hemiataxia, and 1 with limb dystonia.

### Bilateral Infarcts

All 3 patients had an acutely impaired state of consciousness, which was more severe and longer lasting than in those with unilateral lesions. Anterograde memory was severely impaired (Rey auditory verbal learning and Rey
visuospatial learning scores were zero or close to zero for spontaneous recall and were not improved by categori-
cal clues) in all patients, both in the verbal and visuos-
patial domains. Executive dysfunction and apathy were
also found in all patients. Aphasic disturbances with word-
finding difficulty, semantic, and phonemic paraphasias
were present in 2 patients. All had complex eye move-
ment disturbances (horizontal and vertical palsy, skew
devation). One had limb dystonia and asterixis. Dysar-
thria and dyscalcuia were found in 1 patient.

Bilateral involvement was associated with severe
memory loss and behavioral changes, including hyper-
sexuality, jocularity, dysphoria, hyperphagia, and apathy.
None of the 12 patients reported pain.

FOLLOW-UP

Left-Sided Infarcts

The evolution was quite satisfactory. All recovered from
the initial drowsiness within a week or less. Persistent
horizontal or, more commonly, vertical gaze paresis could
be corrected by the use of prisms; however, in 1 case, slight
vertical paresis persisted. Verbal memory disturbances
persisted in all patients for several weeks to 3 months;
however, all patients recovered completely. Behavioral
(frontal-like) disturbances, which were present on hos-
pital admission, disappeared completely in all patients.

Right-Sided Infarcts

The evolution was less satisfactory. All recovered from
the initial drowsiness within a week or less. Four patients
had severe, persistent global anterograde memory deficits. These
patients were confined to nursing homes. Persistent hori-
zontal or, more commonly, vertical gaze paresis could
be corrected by the use of prisms; however, in 2 cases, slight
vertical paresis persisted. Two patients had only visuo-
spatial memory disturbances, which persisted for several
weeks; however, both recovered completely. Behavioral
(frontal-like) disturbances, which were present on hos-
pital admission, disappeared completely in all patients.

Bilateral Infarcts

All patients remained severely disabled, regardless of age.
They all presented with severe coma on hospital admis-
sion and showed long-lasting (more than 1 month) dis-
turbances of consciousness. Two had vertical gaze palsy,
which evolved after several months into persistent pares-
sis. They all showed severe, long-lasting (several months
or even several years) global memory and behavioral (front-
al-like) disturbances and remained severely dependent,
requiring institutional or private care. None were able to
return to their former social or professional activities.

COMMENT

Because of the great variability of the vascular supply of
the thalamus,2,9,12,14-17 in one third of the individuals the
territory of the polar thalamic artery is taken over, uni-
laterally or bilaterally, by the paramedian thalamic ar-
tery.7,8 The respective anatomical structures2,15,16,18-20 may
be affected at once.

In the early 1980s, French authors5,9,9,21,22 described
cases of combined polar and paramedian thalamic in-
farcts. In their seminal clinicopathological study of 28
patients with paramedian thalamic and midbrain in-
farcts, Castaigne et al9 described 5 patients with lesions
touching both territories. One patient (patient 7) with
bilateral infarcts had severe global retrograde and an-
terograde amnesia, apraxia, dysgraphia, and a vertical gaze
palsy. This case fits well with our patients with bilateral
lesions. Barbizet et al18 described a patient with bilateral
lesions who showed severe amnesia without disturb-
ance of vigilance and oculomotoricity. Michel et al18 de-
scribed a patient with combined left-sided infarction who
showed a peculiar deficit uniquely in verbal antero-
grade memory. Our present series thus represents an ex-
tension and consolidation of the clinical pattern of this
combined syndrome described briefly in the past.

In thalamopolar infarction, the clinical dysfunction is
mainly neurobehavioral. In the acute phase, it is domi-
nated by “palipsychism” and is in most cases associated
with severe perseverative behavior and increased sensi-
tivity to interference, anterograde memory disturbance,
intrusions, naming difficulties, dysarthria, hypophonia,
and apathy.23 Left-sided thalamopolar infarction is asso-
ciated with subcortical aphasia, while right-sided infarc-
tion has been linked to hemineglect and impaired visuo-
spatial processing.2,8

Unilateral paramedian infarction leads to acute loss
of or decreased consciousness, correlating with involve-
ment of the intralaminar nuclei and the rostral mid-
brain reticular formation, and to eye movement distur-
bances, primarily upward gaze limitation due to the
concomitant involvement of the thalamic-midbrain junc-
tion. At least one third of all paramedian infarctions are
bilateral2 because frequently a unilateral paramedian
pedicle supplies the paramedian region bilaterally. This
leads to persisting, profound attentional deficits and con-
fusional states. The present literature review suggests that
these 2 thalamic syndromes (ie, thalamopolar syn-
drome and paramedian syndrome) show some overlap
in their clinical manifestations. However, the core fea-
ture of the thalamopolar syndrome is neurobehavioral,
with lateralized deficits according to the side of the le-
sion, while the core features of the paramedian syn-
drome are impaired vigilance and eye movement distur-
bances. “Frontal” signs and transient amnesia may be
colorful to both, especially with bilateral involvement.

As expected, the clinical picture of this combined pola-
paramedian thalamic stroke in our study corresponded
largely to the sum of the clinical picture of both the po-
lar and paramedian syndromes with (1) amnesia, often
severe and combined with various neuropsychological
deficits, (2) a state of altered consciousness and behav-
ioral frontal signs, and (3) eye movement disturbances,
most frequently vertical gaze palsy. There were a num-
ber of less frequently associated manifestations, such as
transient mild hemiparesis or hemisensory loss, hemi-
ataxia, dysarthria, limb dystonia, and asterixis. In most
of these patients, a cardioembolic origin of the lesions

1215

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was suspected on the basis of atrial fibrillation or persistent foramen ovale with or without septal aneurysm. There are, however, a few puzzling aspects of this combined stroke syndrome. Anterograde memory disturbances have been reported in both the polar and paramedian syndromes, but they are usually mild and recovery is good. Moreover, occasional severe amnesia, when due to unilateral thalamic lesions, has been associated with left-sided lesions. We found severe anterograde amnesia in all our patients with bilateral involvement and in 4 with right-sided, combined strokes. Moreover, recovery from amnesia was very incomplete in all patients with bilateral strokes and in 4 patients with unilateral, right-sided strokes. A retrospective study of combined polar and paramedian thalamic infarctions, like ours, does not allow for a true direct comparison of the different thalamic syndromes. However, comparison with the reported clinical manifestation and course suggests that the severe anterograde amnesia and the subsequent incomplete recovery may be the most important findings of combined polar-paramedian thalamic stroke. We tentatively propose an explanation for this finding.

Of all the structures or circuits implicated in memory, 2 involve the thalamus. One is the Papez circuit, which is composed of the hippocampus, fornix, mammillary body, mammillothalamic tract, anterior thalamic nuclei, and cingulated gyrus. This circuit has been strongly implicated in memory function. The anterior thalamic nuclei are part of the vascular territory of the polar artery. Pure amnesia has been reported from lesions involving these nuclei. Of the vascular territory of the polar artery. Pure amnesia has been reported from lesions involving these nuclei. Most, if not all, of the dorsomedial nucleus is supplied by the paramedian artery. Although these 2 structures (ie, the anterior nuclei and the dorsomedial nucleus) are anatomically quite close, they appear to belong to 2 different circuits, both implicated in memory functions, with distinct arterial supply. We believe, on the basis of the relatively good recovery from amnesia secondary to either a polar or a paramedian stroke, that these circuits may mutually act in a compensatory way in case of lesion. We suggest that since both nuclei are affected in combined polar-paramedian stroke, amnesia is more severe and less prone to recovery.

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Analysis and interpretation of data: Perren, Clarke, and Bogousslavsky. Drafting of the manuscript: Perren. Critical revision of the manuscript for important intellectual content: Perren, Clarke, and Bogousslavsky. Statistical analysis: Perren. Study supervision: Clarke and Bogousslavsky.

REFERENCES