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); aphasias in 8 patients (2 with right-sided lesions, 3 with left-sided lesions, 3 with bilateral lesions), including nonfluent 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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Multiple 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. 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. 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). 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).
**Clinical Manifestations**

**Left-Sided Infarcts**

All 3 patients had an acutely impaired state of consciousness, and all had apathy 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, exotropy or hypotropy). 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 apathy 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 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 categorical clues); in the remaining 2 patients, there was memory impairment only in the visuospatial domain. 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.

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### 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><strong>Left-Sided Infarcts</strong></td>
<td></td>
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<tr>
<td>1/M/31 A, VA, DM, VL, LD, midbrain</td>
<td>Verbal anterograde</td>
<td>Severe</td>
<td>Horizontal and vertical palsy, skew deviation, right hypotropopy</td>
<td>None</td>
<td>Confabulations, paraphasias</td>
<td>Executive dysfunction, apathy, mild hemiparesis</td>
<td></td>
</tr>
<tr>
<td>3/F/59 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, apathy, mild hemiparesis, and hemisensory loss</td>
<td></td>
</tr>
<tr>
<td>9/F/78 A, DM, LD</td>
<td>Verbal anterograde</td>
<td>Sleepy</td>
<td>None</td>
<td>Ideomotor</td>
<td>Nonfluent aphasia</td>
<td>Executive dysfunction, dyscalculia, apathy</td>
<td></td>
</tr>
<tr>
<td><strong>Right-Sided Infarcts</strong></td>
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<tr>
<td>4/M/48 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, apathy, mild hemiparesis, and hemisensory loss</td>
<td></td>
</tr>
<tr>
<td>5/M/35 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, apathy, mild hemiparesis, hemiataxia</td>
<td></td>
</tr>
<tr>
<td>6/F/48 A, VA, DM, LD, midbrain</td>
<td>Global severe anterograde</td>
<td>Normal</td>
<td>Vertical palsy, oblique diplopia</td>
<td>Visuoconstructional</td>
<td>Slight dysarthria</td>
<td>Executive dysfunction, anosodiaphoria, apathy, mild hemiparesis, limb dystonia</td>
<td></td>
</tr>
<tr>
<td>8/F/65 A, VL, DM, LD, midbrain</td>
<td>Severe anterograde</td>
<td>Severe</td>
<td>Left hemianopsia</td>
<td>Visuoconstructional</td>
<td>Paraphasia</td>
<td>Executive dysfunction, apathy, mild hemiparesis</td>
<td></td>
</tr>
<tr>
<td>10/F/64 A, VA, DM, LD, midbrain</td>
<td>Severe anterograde</td>
<td>Sleepy, athymornia</td>
<td>Vertical palsy</td>
<td>None</td>
<td>Paraphasia</td>
<td>Executive dysfunction, abulia, apathy</td>
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<tr>
<td>12/M/65 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, apathy</td>
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<tr>
<td><strong>Bilateral Infarcts</strong></td>
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<tr>
<td>2/F/65 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, apathy, asterixis, limb dystonia</td>
<td></td>
</tr>
<tr>
<td>7/M/68 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, apathy, disinhibition</td>
<td></td>
</tr>
<tr>
<td>11/M/68 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>
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</tbody>
</table>

Abbreviations: A, anterior nucleus; DM, dorsomedial nucleus; LD, laterodorsal nucleus; VA, ventroanterior nucleus; VL, ventrolateral nucleus.
visuospatial learning scores were zero or close to zero for spontaneous recall and were not improved by categorical clues) in all patients, both in the verbal and visuospatial domains. Executive dysfunction and apathy were also found in all patients. Aphasias disturbances with word-finding difficulty, semantic, and phonemic paraphasias were present in 2 patients. All had complex eye movement disturbances (horizontal and vertical palsy, skew deviation). One had limb dystonia and asterixis. Dysarthria and dyscalculia were found in 1 patient.

Bilateral involvement was associated with severe memory loss and behavioral changes, including hypersexuality, 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 hospital 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 horizontal 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 visuospatial memory disturbances, which persisted for several weeks; however, both recovered completely. Behavioral (frontal-like) disturbances, which were present on hospital admission, disappeared completely in all patients.

**Bilateral Infarcts**

All patients remained severely disabled, regardless of age. They all presented with severe coma on hospital admission and showed long-lasting (more than 1 month) disturbances of consciousness. Two had vertical gaze palsy, which evolved after several months into persistent paresis. They all showed severe, long-lasting (several months or even several years) global memory and behavioral (frontal-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, in one third of the individuals the territory of the polar thalamic artery is taken over, unilaterally or bilaterally, by the paramedian thalamic artery. The respective anatomical structures may be affected at once.

In the early 1980s, French authors described cases of combined polar and paramedian thalamic infarcts. In their seminal clinicopathological study of 28 patients with paramedian thalamic and midbrain infarcts, Castaigne et al described 5 patients with lesions touching both territories. One patient (patient 7) with bilateral infarcts had severe global retrograde and anterograde amnesia, apraxia, dysgraphia, and a vertical gaze palsy. This case fits well with our patients with bilateral lesions. Barbizet et al described a patient with bilateral lesions who showed severe amnesia without disturbance of vigilance and oculomotoricity. Michel et al described a patient with combined left-sided infarction who showed a peculiar deficit uniquely in verbal anterograde memory. Our present series thus represents an extension 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 dominated by “palipsychism” and is in most cases associated with severe perseverative behavior and increased sensitivity to interference, anterograde memory disturbance, intrusions, naming difficulties, dysarthria, hypophonia, and apathy. Left-sided thalamopolar infarction is associated with subcortical aphasia, while right-sided infarction has been linked to hemineglect and impaired visuospatial processing.

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

As expected, the clinical picture of this combined polar-paramedian thalamic stroke in our study corresponded largely to the sum of the clinical picture of both the polar and paramedian syndromes with (1) amnesia, often severe and combined with various neuropsychological deficits, (2) a state of altered consciousness and behavioral frontal signs, and (3) eye movement disturbances, most frequently vertical gaze palsies. There were a number of less frequently associated manifestations, such as transient mild hemiparesis or hemisensory loss, hemiataxia, dysarthria, limb dystonia, and asterixis. In most of these patients, a cardioembolic origin of the lesions...
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, mamillary 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. The other structure is the dorsomedial nucleus of the thalamus. While somewhat controversial, severe memory disturbances have long been associated with damage to this structure. 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