The Syndrome of Unilateral Tuberothalamic Artery Territory Infarction

JULIEN BOGOUSSLAVSKY, M.D., FRANCO REGLI, M.D., AND GIL ASSAL, M.D.

SUMMARY The study of 3 personal cases and 5 published cases of unilateral infarct limited to the territory of the tuberothalamic artery suggests that this syndrome should be differentiated from the other thalamic syndromes. The onset is usually sudden, with moderate contralateral weakness. Sensory changes may be present but remain mild. The patients are aphasic, show perseverations and may be disoriented. In left-sided infarcts, transcortical aphasia, verbal and visual memory impairment and sometimes acalculia are found. In right-sided infarcts, hemispatial neglect, visual memory impairment and disturbed visuospatial processing are common. A decreased level of consciousness, disturbed ocular movements, severe motor weakness and delayed abnormal movements do not occur. Involvement of the ventral lateral and dorsomedial nucleus with sparing of the intralaminar nuclei, posterolateral formation and upper midbrain may explain this picture. The fact that the tuberothalamic artery arises from the posterior communicating artery, which often receives its supply from the carotid system, further justifies considering unilateral tuberothalamic infarcts as a syndrome.

Methods and Patients

We diagnosed tuberothalamic artery infarction from the CT scan pictures, according to the topographic description of Percheron.13 All 3 patients were examined by at least 3 neurologists and 1 neuropsychologist. A comprehensive battery of tests was done in patients 1 and 3, and a partial battery was administered to patient 2, because of marked speech disturbances. The following evaluation was done: orientation to time and place, spontaneous speech, verbal fluency, naming (Boston naming test16), repetition of isolated, or series (3–5 items) of, phonemes or words and sentences (3–15 words), auditory comprehension (Token test15), writing (spontaneous, dictation), reading (words, non-words, text, summary of text meaning), dichotic listening test, oral and written calculation, bucco-linguo-facial praxias, limb (symbolic, imitation) praxias, constructional praxia (spontaneous drawing of a cube, Rey complex Figure16), Benton Facial Recognition test17 and other visual gnosis tests (Ghent,18 Poppelreuter,19 Columbia20), orientation on a map of Switzerland, right-left discrimination, evocation of recent and remote events, verbal learning (Hebb’s recurring digits,21,22 Rey Auditory Learning16), visual learning (Corsi’s Block-tapping,22 delayed reproduction of Rey Complex Figure16), Wisconsin Card Sorting test,23 Stroop test,24 Luria’s conflicting tasks, sequential rhythms and sequential geometrical figures,25 and a nonverbal intelligence test (Raven progressive matrices26).

Case 1

A 45-year-old right-handed housewife was admitted after she suddenly experienced dizziness, vomiting, numbness in the right side of the face, clumsiness of the right hand and speech disturbances. Her past history revealed episodes of hypoglycemia and migraine, and she was treated with estrogens for primary ovarian insufficiency. She was suffering from familial type IV hypercholesterolaemia, and had a strong family history of coronary heart disease on her father’s side.

On neurological examination, the visual fields, the optic fundi, the pupils and oculomotoricity were normal. A slight light touch hypoaesthesia involving the right side of the face down to the C3 dermatome was present, without involving the mouth and tongue, and with symmetrical corneal reflexes. An “emotional” right facial weakness was present, with normal contraction on command but droop of the lips on the right side when smiling. The taste and the remainder of the cranial nerves were normal. There was a slight downward drift of the right upper limb on the stretched arm test performed with the eyes closed, but no objective weakness could be demonstrated, and the tone, tendon and cutaneous reflexes were normal. The coordination of all four limbs and the gait were normal. However, the patient tended to lose her balance when trying to jump on the right leg. Tactile, temperature, pain, postural and vibratory sensation were preserved, with normal graphesthesia, stereognosis and topoesthesia. Her blood pressure was 110/70 mm Hg with a regular pulse (80/min). No bruit was heard on the neck or precordium, and the general examination was normal.
Neuropsychological Examination

The patient was well-oriented in time and space. She complained of concentration difficulty and memory disturbances. Her spontaneous speech was quantitatively reduced, with short sentences (5 words maximum) and slowed output. Occasional phonemic paraphasias were present ("anassin" instead of "assassin") and less frequently, semantic paraphasias ("clock" instead of "watch"). Naming was impaired (46/76, Boston naming test) and verbal fluency was reduced (14 names of animals in 9 minutes). The dichotic listening test did not show any asymmetry. Verbal comprehension (31/36, Token test) was only mildly impaired, despite slowness of answering. The repetition of isolated, or series (up to 5) of phonemes and words and of sentences (up to 15 words) was normal. Words, non-words, and text reading were normal. Spontaneous writing was reduced but without paragraphia or syntactic disturbances. Writing on dictation was unimpaired. Oral calculation was markedly impaired, even for simple arithmetical operation (0/6), with tendency to perseverate. Written calculation was similarly impaired, with loss of the arithmetical programs for addition, subtraction, multiplication and division. Spontaneous drawing of a cube was preserved, although slowed. Bucco-linguo-facial praxias were normal, and limb praxias did not show any significant disturbance (4/4 on symbolic gestures, 5/6 on imitation gestures). Visual and visuo-spatial gnosias were normal (Ghent: 35/36, Columbia: 10/12, orientation on a geographic map). Marked difficulties were present on evocation of recent events, and visual as well as verbal learning difficulties were present (Hebb's recurring digits: maximal span of 5, failure of span + 1 learning; Corsi's Block-tapping: maximal span of 5, failure of span + 1 learning). Perseverations were present on execution of Luria's conflicting tasks, sequential rhythms and sequential geometrical figures. On Stroop test, naming was markedly slowed (1'48'', 1 mistake, 7 autoconnections) as compared with word reading (1'55'').

Case 2

A 74-year-old right-handed, hypertensive man was admitted after he suddenly experienced speech disturbances, followed a few hours later by mild right-sided weakness. On examination, visual acuity, visual fields, ocular movements and pupils were normal. Optic fundi revealed moderate retinal vascular sclerosis. Facial, buccal and lingual sensation were preserved, with symmetrical corneal reflex. A slight "emotional" right-sided facial paresis was present, with symmetrical contraction on command. The taste and the remainder of the cranial nerves were normal. The strength was normal and symmetrical in the four limbs, but on the stretched arm test, there was a slow drift with pronation tendency of the right arm. Fine motility of the fingers was slowed on the right, and the tone of the right arm and leg was slightly increased, without tendon reflexes asymmetry or abnormality. Abdominal reflexes were present and plantar reflexes were down-going. Coordination was normal in the four limbs, except moderate dysdiadochokinesia in the right upper limb. The gait was normal. Tactile, temperature, pain, postural and vibratory sensations were normal and symmetrical, with normal 2-point discrimination, graphesthesia, stereognosia and absence of sensory extinction. Blood pressure was 160/100, but general examination was normal for his age.

Neuropsychological Examination

The patient was well-oriented in time and place. A detailed neuropsychological assessment was not possible, because of the marked disturbances of language. Spontaneous speech was moderately reduced, with slowed output and shortened sentences (8 words maximum). Phonemic and semantic paraphasias were present, as well as perseverations and occasional neologisms, with preserved syntax and absence of dysarthria. The frequency of the paraphasias rendered the patient’s speech very difficult to understand. Naming was impaired (17/76, Boston naming test). Production of automatic series (days of the week, months) was preserved in anterograde order, but paraphasias and perseverations appeared in retrograde order. Verbal fluency was reduced (4 names of animals in three minutes), with perseverations. The dichotic listening test...
test did not disclose any left-right ear asymmetry. Verbal comprehension was moderately impaired (25/36 Token test), and the repetition of isolated, or series (up to 5) of phonemes or words and of sentences (up to 15 words) was fully preserved. The reading of a text was normal, although slow, but his account of the story showed appearance of numerous paraphasias, perseverations, and neologisms. Spontaneous writing showed paragraphias and neologisms, with preserved copying. Writing on dictation was impaired, with paragraphias and perseverations of letters and words. Spelling of words was disturbed (2/5). Oral calculation was impaired (1/5). Written calculation showed preservation of addition and subtraction, but multiplication and division were impaired. Spontaneous drawing of a cube was normal. Bucco-linguo-facial praxias were preserved and limb praxias (symbolic and imitation) showed only occasional perseverations with autocorrection. Visual gnosias were preserved (Ghent: 33/36, Poppelreuter: 3/4), with preserved orientation on a geographic map. Learning of series of visual and verbal items was markedly impaired but was not quantified, because the patient did not cooperate well enough. Evocation of recent or remote events was disturbed by the speech disturbances. On execution of Luria’s conflicting orders, sequential rhythms and sequential geometrical figures, perseverations appeared, with selfcorrection attempts and refusal to continue the tests.

Standard blood and urine tests were normal. EKG showed moderate ischemic changes with left ventricular hypertrophy. A CT scan performed 2 days after admission showed an isolated lucency in the anterior aspect of the thalamus on the left side, suggesting a recent infarct in the tuberothalamic artery territory (fig. 2).

Case 3
A 68-year-old insulin dependent diabetic right-handed man suddenly experienced transient blurring of vision with persisting left-sided weakness and feeling of intense fatigue. On admission, visual acuity was moderately depressed in both eyes (right: 0.5; left: 0.7) with diabetic retinopathy on optic fundi examination. The visual fields were full, but left-sided extinction appeared on bilateral simultaneous stimulation. The pupils and ocular movements were normal. Light touch and pain sensation were slightly decreased in the left side of the face, with a decreased left corneal reflex. A slight lower facial paresis was present, either on voluntary or emotional contraction. Bilateral hypoaucusia was present, but the remainder of the cranial nerves showed no abnormality. The strength and tone were normal in the 4 limbs, but the right arm drifted slowly downward and pronated on the stretched arm test. The fine movements of the fingers were slightly slowed on the left side, with dysdiadochokinesia and moderate imprecision on finger-to-nose movements. No hypermetria or rebound phenomenon were found. The tendon reflexes were decreased distally in the 4 limbs. Abdominal reflexes were decreased on the left side. The left plantar reflex was upgoing, but was flexor on the right. Tactile, pain and temperature sensations were slightly decreased on the left side of the body, with a tendency to extinction on simultaneous bilateral stimulation, but with normal graphesthesia.
and stereognosis. Vibratory and postural sensations were decreased distally in the 4 limbs, with some degree of imbalance on Romberg’s manoeuvre and tandem gait. Blood pressure was 160/90 mm Hg, with a pulse of 72/min. The general examination was unremarkable.

Neuropsychological Examination

The patient was well-oriented in time and place, with a normal level of consciousness. His spontaneous speech showed no abnormality, with normal output. Naming was within normal ranges (60/75 Boston naming test). The repetition of isolated, or series of, phonemes or words and of short and long sentences was fully preserved. Auditory comprehension showed very mild difficulties (32/36 Token test). Spontaneous writing and writing on dictation were normal, except for neglect of the left side of the page. The reading of words, non-words and a text was normal, with a good oral summary of the text meaning. On dichotic listening test, left-sided extinction was prominent (right ear: 25/30, left ear: 0/30 with 10/10 on monaural stimulation). Oral calculation was preserved (4/5), but written calculation was markedly impaired (0/4) because of loss of the correct spatial disposition of the operations and right-sided neglect. Bucco-linguo-facial praxies and limb (symbolic, imitation) praxies were normal. Spontaneous drawing of a cube was impaired (loss of perspective). Recognition of faces was preserved (43/54, Benton test). Tests for visual gnosis showed normal results (35/36, Ghent; 10/12, Columbia). The copy of Rey complex figure was impaired (16 points, centile 0). Orientation on a map was preserved, and no right-left confusion was found. Verbal learning was insufficient (Hebb’s recurring digits: span of 5, failure of span + 1 learning; 15 words of Rey: total of 22: 5-3-5-5-4, centile 0). Visual learning was more impaired (Corsi’s block-tapping: span of 4, failure of span + 1 learning; learning of 15 signs: total of 18: 2-3-4-5-4; 11/15 on recognition; delayed reproduction of Rey complex figure: immediate reproduction: 6 points, centile 0, and delayed reproduction: 2 points, centile 0). The Wisconsin card sorting test was much disturbed (0 series realized) and marked slowing was present on the Stroop test (1’39”). The results of the Raven PM 38 test were insufficient (15 points, centile 0).

Standard blood and urine tests were within normal ranges, except elevated cholesterol (8.2 mmole/l). EKG showed a left anterior hemiblock. The CSF was clear, with normal proteins (390 mg/l); 1 white cell/cm³ and negative VDRL and FTA-Abs. Somatosensory evoked potentials (median nerve stimulation) showed an altered morphology and reduced amplitude on the right parietal recording. A CT scan done one week after admission showed an isolated lucency in the anterior part of the right thalamus, suggestive of an infarct in the territory of the right rubrothalamic artery (fig. 3).

Discussion

The rubrothalamic artery has also been called the polar artery of the thalamus, the anterior thalamo-subthalamic paramedian artery, the anterointernal optic artery, and the premamillary pedicle. It originates from the middle third of the posterior communicating artery, but may be absent in 30–40% of the population, in which case its territory is supplied by the posterior thalamo-subthalamic paramedian artery originating from the first segment of the posterior cerebral artery (P1 or basilar communicating artery). When the rubrothalamic artery is present, it supplies the reticular nucleus, the mammillo-thalamic tract, part of the ventral lateral nucleus and of the dorsomedial nucleus, and the lateral aspect of the thalamic pole, but not the anterior nucleus. Three other systems supply the thalamus (fig. 4): 1) The posterior thalamo-subthalamic paramedian artery (or deep interpeduncular profunda artery, thalamoperforate or retro-mamillary pedicle) is often unpaired and originates from P1. It supplies the paramedian part of the upper midbrain and of the thalamus (intralaminar nuclei, dorsomedial nucleus, and the lateral aspect of the thalamic pole, but not the anterior nucleus). Three other systems supply the thalamus (fig. 4): 1) The posterior thalamo-subthalamic paramedian artery (or deep interpeduncular profunda artery, thalamoperforate or retro-mamillary pedicle) is often unpaired and originates from P1. It supplies the paramedian part of the upper midbrain and of the thalamus (intralaminar nuclei, dorsomedial nucleus, internal part of the ventral posterior nucleus); 2) The thalamogeniculate pedicle originates from the posterior cerebral artery after the level of the posterior communicating artery (P1) and includes the thalamic infero-lateral arteries and the pulvinar infero-lateral arteries. It supplies most of the ventral posterior nucleus, a part of the ventral lateral nucleus, and of the pulvinar. 3) The posterior choroidal arteries leave the posterior cerebral artery at the same level as the thalamogeniculate branches and separate into a mesencephalo-posteromedial system and a hippocampo-posterolateral system, which supply the thalamic region, part of the substantia nigra and

![Case 3, CT scan. Infarct of the anterior part of the right thalamus.](image-url)

Suggestive clinical syndromes have been reported in unilateral infarcts in these three territories (table 1). The infarcts in the thalamogeniculate pedicle territory are the most common ones, and typically produce a sensory stroke which may or may not evolve towards a Dejerine-Roussy syndrome. The infarcts in the posterior thalamo-subthalamic paramedian artery territory are often bilateral, because this artery is often a single branch coming off the basilar communicating artery (butterfly-shaped infarcts). When there are 2 arteries, the infarct is unilateral. It typically gives rise to decreased consciousness, neuropsychological impairment, and oculomotor disturbances, sometimes delayed abnormal movements. Infarcts limited to the posterior choroidal system are very uncommon, and seem to produce partial visual field defects.

On the other hand, no study has been devoted to infarcts of the tuberothalamic artery territory. We

TABLE 1 Reported Syndromes in Unilateral Thalamic Infarction

<table>
<thead>
<tr>
<th>Territory of thalamic infarction</th>
<th>Origin of artery</th>
<th>Reported clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior thalamo-subthalamic paramedian artery</td>
<td>Initial segment of the PCA = basilar communicating artery (P1)</td>
<td>consciousness, confusion, behavioral changes7, asterixis30, motor hemineglect8, memory impairment7, language disturbances (dominant thalamus)9, contralateral cerebellar dysfunction34, faciobrachial tactile hypoesthesia8, upgaze palsy7, Weber's &amp; Claude's third nerve fascicular syndromes7, delayed abnormal movements7</td>
</tr>
<tr>
<td>Thalamo-geniculate pedicle</td>
<td>Second segment of the PCA (P2)</td>
<td>homisensory syndromes: Dejerine-Roussy syndrome,1,2 pure sensory stroke,4 cheiro (podo) oral/pseudo radicular variants4, motor hemineglect (non-dominant thalamus)37, contralateral cerebellar dysfunction38, ataxic hemiparesis39, language disturbances (dominant thalamus)40</td>
</tr>
<tr>
<td>Posterior choroidal arteries</td>
<td>Second segment of the PCA (P3)</td>
<td>homonymous horizontal sectoranopia41, lateral geniculate body</td>
</tr>
</tbody>
</table>

\[ \downarrow = \text{decreased} \]
could identify only 5 published cases of isolated unilateral infarcts in this territory, with CT scan and neurological and neuropsychological assessment (table 2). Percheron\textsuperscript{13} mentioned a personal case with pathological study, but the clinical picture was unknown. Three more cases studied by CT scan were reported,\textsuperscript{11, 12} but they had an associated infarct in the ipsilateral occipital lobe which obscured the symptomatology. Bilateral infarcts limited to this territory also seem to be very rare.\textsuperscript{43, 44} In the cases where the tuberothalamic artery is absent, its territory is supplied by the posterior thalamo-subthalamic paramedian artery and infarcts then involve the whole paramedian area, unilaterally\textsuperscript{45} or bilaterally.\textsuperscript{46}

The study of our 3 cases and of 5 published cases with a unilateral infarct limited to the tuberothalamic artery territory (table 2) shows that delineation of a suggestive syndrome is possible. The onset is usually sudden, with mild to moderate contralateral motor weakness or clumsiness (86%). A contralateral "emotional" facial weakness with preserved voluntary contraction is frequently found, as formerly reported.\textsuperscript{11} Contralateral sensory changes may occur (43%), but involve only the superficial modalities of sensation and remain mild. Neuropsychological dysfunction is present in 100% of the cases. The patients are withdrawn, but not drowsy and show apathy, lack of spontaneity and perseverations, especially in the cases with right-sided infarction. They can be disoriented (28%). In infarcts of the dominant thalamus, speech disturbances are associated with visuo-verbal memory impairment, and sometimes acalculia. The language disturbances do not seem to be different from those described in other types of thalamic infarction involving the lateral formation\textsuperscript{48} or the posterior paramedian area,\textsuperscript{8, 32, 33} and suggest transcortical aphasia: speech is reduced, with anomia and some phonemic/semantic paraphasias and occasionally neologisms (case 2), with slightly impaired or normal comprehension and normal repetition. The memory disturbances involve both the verbal and visual items, with learning difficulties. In infarcts of the non-dominant thalamus, contralateral hemispatial neglect may be prominent, with memory impairment involving mainly the visual learning and altered visuospatial processing with constructional dyspraxia.

Since no cases with pathological verification are available, except one with unknown symptomatology,\textsuperscript{13} clinical-anatomic correlations are limited to speculations based on other types of thalamic infarcts or diseases, or on electrocaigation studies. Language disturbances in the setting of a thalamic lesion have been attributed to involvement of the ventral lateral nucleus,\textsuperscript{40, 47-49} putamin,\textsuperscript{40} and or dorsomedial nucleus.\textsuperscript{46, 50-52} In a case of unilateral left posterior thalamic injury, the patient had left-appearance extinction and left-hemiparesis, followed by right-sided neglect and left-sided apraxia.

### Table 2: Unilateral Infarction of the Tuberothalamic Artery Territory: Summary of Clinical Features

<table>
<thead>
<tr>
<th>Authors</th>
<th>Sex</th>
<th>Age</th>
<th>Risks factors</th>
<th>Side</th>
<th>Onset</th>
<th>Sensory changes</th>
<th>Neuropsychological examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cohen et al\textsuperscript{9} 1980</td>
<td>♂</td>
<td>62</td>
<td>DM</td>
<td>L</td>
<td>right-sided weakness and speech disturbances (sudden)</td>
<td>(+) phonic paraphasias, anomia, slight of comprehension, normal repetition</td>
<td></td>
</tr>
<tr>
<td>Archer et al\textsuperscript{10} 1981</td>
<td>♂</td>
<td>57</td>
<td>?</td>
<td>L</td>
<td>transient right-sided weakness and persisting speech disturbances (sudden)</td>
<td>- paraphasias, ↓ of comprehension, normal repetition, perseverations, ↓ visual/verbal memory</td>
<td></td>
</tr>
<tr>
<td>Graff-Radford et al\textsuperscript{11} 1984 (case 3)</td>
<td>♂</td>
<td>75</td>
<td>HT</td>
<td>L</td>
<td>subacute change of personality, depression</td>
<td>- disorientation, aspontaneity of anomia, normal repetition, ↓ visual/verbal memory</td>
<td></td>
</tr>
<tr>
<td>Graff-Radford et al\textsuperscript{11} 1984 (case 5)</td>
<td>♀</td>
<td>64</td>
<td>HT multiple aneurysms</td>
<td>R</td>
<td>left-sided weakness and hemineglect (sudden)</td>
<td>- disorientation, normal language, ↓ nonverbal memory, ↓ visuospatial processing</td>
<td></td>
</tr>
<tr>
<td>Biller et al\textsuperscript{12}</td>
<td>♂</td>
<td>29</td>
<td>lupus anticoagulant</td>
<td>L</td>
<td>transient right-sided weakness and numbness, and dysarthria, followed by progressive memory impairment</td>
<td>- disorientation to time, ↓ memory, normal speech but monotone voice, omission of words when reading</td>
<td></td>
</tr>
<tr>
<td>Present study (case 1)</td>
<td>♀</td>
<td>45</td>
<td>hypercholesterolaemia</td>
<td>L</td>
<td>dizziness, vomiting, right-sided facial numbness and hand clumsiness, speech disturbances (sudden)</td>
<td>(+) phonic paraphasias, aspontaneity of speech, anomia, normal comprehension and repetition, ↓ visual/verbal memory, perseverations, acalculia</td>
<td></td>
</tr>
<tr>
<td>Present study (case 2)</td>
<td>♂</td>
<td>74</td>
<td>HT</td>
<td>L</td>
<td>sudden speech disturbances followed by right-sided weakness</td>
<td>- paraphasias and neologisms, anomia, slight ↓ of comprehension, normal repetition, ↓ visual/verbal memory, perseverations</td>
<td></td>
</tr>
<tr>
<td>Present study (case 3)</td>
<td>♂</td>
<td>68</td>
<td>DM</td>
<td>R</td>
<td>fatigue, transient blurring of vision, persisting left-sided weakness (sudden)</td>
<td>(+) normal language, multimodal left-sided hemineglect, constructional apraxia, visual more than verbal memory impairment, &quot;frontal&quot; syndrome</td>
<td></td>
</tr>
</tbody>
</table>

DM = diabetes mellitus; HT = hypertension; (+) = slight; + = marked; - = absent; R = right; L = left; ↓ = decrease.
lamo-subthalamic paramedian artery territory infarct, which was pathologically studied, we found that the language disturbances and the impairment of verbal memory correlated well with involvement of the dorsomedial nucleus, in the absence of significant involvement of the ventral lateral nucleus, pulvinar and mamillothalamic tract. In the present case, involvement of the dorsomedial nucleus also seems to correlate the best with the dysphasic and memory disturbances, because of the arterial supply topography of the tuberothalamic artery. The mamillothalamic tract is usually supplied by this artery and may also participate in the memory disturbances. Involvement of the ventral lateral nucleus may also account for at least some of the aphasic deficits, and probably explains the occurrence of “emotional” facial paresis and contralateral neglect in nondonminant side infarction. Contralateral neglect may also be produced by a lesion of the intralaminar nuclei, but these nuclei are not supplied by the tuberothalamic artery. The occasional presence of only mild sensory changes indicates that the ventral posterior nucleus is mostly spared. Initial transient or mild contralateral weakness probably corresponds to continuous involvement of the internal capsule by edema or pressure during the acute ischemic phase, and it is not similar to the severe hemiplegia produced by peduncular involvement in infarcts of the posterior thalamo-subthalamic paramedian or circumferential arteries, or to the hemiplegic syndrome of the posterior cerebral artery.

Many of the neuropsychological disturbances found in this syndrome do not markedly differ from those reported in unilateral infarcts of the posterior thalamo-subthalamic paramedian artery territory, probably because of vascular supply overlap in structures like the dorsomedial nucleus. However, infarcts of the tuberothalamic artery territory should be differentiated from those of the posterior thalamo-subthalamic paramedian artery territory, because they do not produce altered consciousness, supranuclear upgaze palsy, intra-axial third nerve palsy, severe contralateral hemiplegia or delayed abnormal movements, as they do not involve the intralaminar nuclei and the upper midbrain. Moreover, as the posterior communicating artery — from where the tuberothalamic artery originates — is filled by the internal carotid artery in as many as 40% of the hemispheres, recognition of this syndrome among the other syndromes of thalamic infarction is important, because it may occasionally be related to carotid system disease. One case of thalamic infarction was reported after carotid artery occlusion, but the configuration of the posterior cerebral artery was not described. An angiographic study of the pattern of the circle of Willis in thalamic infarction still remains to be done.

In summary, we believe that tuberothalamic artery territory infarcts can be differentiated from the other types of thalamic infarcts by their clinical aspects and CT scan pictures. However, since no cases with clinic-anatomic correlation have yet been reported, we think that the hypotheses advanced for the explanation of the symptomatology should, for the moment, be accepted with reservation, because they are based on pathologic studies of other thalamic syndromes.

Addendum

Another case of unilateral left infarction in the territory of the tuberothalamic artery has been reported by Gorelick et al (Gorelick PB, Hier DB, Benevento L, Levitt S, Tan W: Aphasia after left thalamic infarction. Arch Neurol 41: 1296–1298, 1984), with a detailed analysis of speech disturbances.

Acknowledgment

We would like to thank Andrew Kertesz, MD, University of Western Ontario, for his helpful comments on the manuscript.

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The syndrome of unilateral tuberothalamic artery territory infarction.
J Bogousslavsky, F Regli and G Assal

*Stroke*. 1986;17:434-441
doi: 10.1161/01.STR.17.3.434

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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