Young Patient With Aphasia, Monoparesis, Facial Drop, Facial Sensory Loss, Diplopia, Dysarthria, and Vertical Gaze Palsy

Daniel Strbian, MD, PhD; Sophia Sundararajan, MD, PhD

Case Description

We present a case of a 40-year-old male without previous medical history and medication who denied tobacco smoking or alcohol abuse. He led a healthy lifestyle, and there were no cardiovascular risk factors in his family history. In October 2012, he experienced acute onset of horizontal diplopia while playing with his child. During the transport to the emergency room, he developed a left-sided facial drop and dysarthria. On arrival, he was immediately moved to the computed tomography (CT) room as a candidate for intravenous thrombolysis and was examined by a stroke neurologist. At that time, the patient’s level of consciousness was slightly drowsy, but he was orientated. His pupils were equal with normal reactions to light. He had horizontal diplopia in either direction, more pronounced on the right side. The right eye was nasally deviated. There was no problem with vertical eye movements. He had facial sensory loss on the left side, left-sided facial drop causing dysarthria, and difficulty with word finding. There were no sensorimotor findings in the upper extremities. He was able to raise his right lower extremity, but could not hold it in the upright position for 5 seconds, and the Babinski sign was present on the right side. The National Institutes of Health Stroke Score was 6.

With the suspicion of posterior circulation stroke, a noncontrast computed tomographic head scan and CT angiography were ordered. There was no pathology on noncontrast computed tomographic scan. Before CT angiography findings were available, the patient’s symptoms progressed. He became mute but understood verbal commands and developed a vertical gaze palsy. There was confusion on the localization of the lesion. On the one hand, right-sided weakness and aphasia suggested a left middle cerebral artery syndrome; however, left facial drop and facial sensory loss did not fit this. On the other hand, diplopia with eye movement abnormalities suggested a brain stem lesion.

After considering contraindications, the patient received full-dose of intravenous alteplase 1 hour and 14 minutes after symptom onset. CT angiogram showed the basilar artery to be open, but detailed radiologist’s report was not available at that time. During the alteplase infusion, the patient’s condition improved. He was able to speak in full sentences with paraphasic errors and had continued dysarthria. His left-sided facial drop improved, and there were no facial sensory symptoms. He had still diplopia; the right eye was adducted but had conjugate eye movement to the left. He had inability to abduct the right eye and a vertical gaze palsy. His right lower extremity weakness resolved, and the National Institutes of Health Stroke Scale improved to 3.

According to the radiologist, CT angiography results showed no occlusion or other pathology in the carotid, vertebral, and basilar arteries. Because of the suspicion of brain stem ischemia, MRI was ordered. What do you expect to be the finding and why? (see below for Answer).

During the emergency room follow-up, the patient’s condition improved markedly, and the only residual symptom was diplopia when looking in the distance. He was moved to the stroke unit, where his neurological symptoms resolved. The speech therapist did not find any abnormalities, and a neuropsychologist detected mild cognitive findings, which subsequently resolved during a short sick leave. The patient returned to his normal life soon after discharge. In the comprehensive diagnostic work-up, the only abnormality detected was elevated low-density lipoprotein cholesterol (128 mg/dL).

Answer

MRI showed 2 small diffusion-weighted imaging–positive lesions suggestive of acute ischemia in left thalamus; no other abnormalities were seen.

Discussion

This case demonstrates neuroanatomic challenges. One does not typically consider thalamic lesions in case of aphasia or vertical gaze palsy. Although aphasia, with or without hemiparesis, is a typical finding in middle cerebral artery infarcts, vertical gaze palsy is usually associated with lesions of the mesencephalic rostral interstitial nucleus of the medial longitudinal fasciculus, the interstitial nucleus of Cajal, the posterior commissure, and the periaqueductal gray matter.

The thalamus is part of the diencephalon and its nuclei supply crucial input and output to the cerebral cortex. It coordinates information from major afferent, somatomotor, and limbic...
system pathways and from the reticular formation. Its vasculature supply comes from 4 arterial systems; 3 of which originate from the vertebrobasilar system: (1) the paramedian (thalamosubthalamic) artery comes off the first segment of posterior cerebral artery (P1 segment); (2) the inferolateral (thalamogeniculate) artery, from the second segment of the posterior cerebral artery (P2 segment); and (3) the posterior (medial and lateral) choroidal arteries, also arise from the P2 segment. The polar (tuberothalamic artery) is derived from the posterior communicating artery.

The clinical manifestation of infarction depends on the affected vascular territory and associated part of the thalamus. The most common infarcts affect the inferolateral thalamus (inferolateral or thalamogeniculate artery) and the paramedian thalamus (paramedian or thalamosubthalamic artery), whereas the anterior thalamus (polar or tuberothalamic artery) and posterior thalamus (posterior choroidal arteries) are less commonly affected.

Thalamogeniculate Territory

Patients usually develop a complete thalamic syndrome with sensory and motor deficits, as well as abnormal movements. The sensory deficit is usually the initial manifestation, followed by motor deficits, whereas abnormal movements appear later and are dominant findings. Some patients develop sensorimotor stroke with paresthesias, or numbness on one side of the body with hemiparesis, increased reflexes, and with Babinski sign. Others may present with motor abnormalities and third cranial nerve involvement with ptosis, diplopia, and ophthalmoparesis. A minor portion of patients even have a pure sensory deficit limited to the arm. Speech disturbances, memory impairment (anterograde amnesia in all cases), and disturbances of consciousness can also occur, but the latter is rare with lesions in this territory.

Paramedian or Thalamosubthalamic Artery Territory

Most patients have sensorimotor abnormalities, and some of them develop third cranial nerve involvement with diplopia, ophthalmoparesis, ptosis, and mydriasis. A smaller number of patients have pure motor abnormalities along with third cranial nerve involvement and abnormalities of vertical gaze. The principal clinical features are decreased level of consciousness, altered social skills and personality, aphasia (with lesions on the left), spatial deficits (with lesions on the right), short-term memory loss (anterograde amnesia), vertical gaze paresis, impaired convergence, and skew eye deviation.

Polar Artery Territory

These patients commonly have a pure motor deficit, sometimes associated with motor aphasia and others have anterograde amnesia. Some patients develop sensorimotor deficits with anterograde amnesia, whereas others develop isolated third cranial nerve involvement with ptosis and diplopia.

Posterior Choroidal Artery Territory

Patients with infarction in the distribution of the posterior choroidal artery can present with a pure motor deficit but also with sensorimotor deficit. They usually have speech disturbances (motor aphasia and dysarthria).

Thalamic stroke can lead to acute aphasia, hemiplegia, hemisensory loss, and decreased consciousness. Patients commonly experience mutism in the early stage, and this tends to improve to a verbose, paraphasic, jargon, as was the case in the presented patient. Thalamic aphasia is reminiscent of fluent paraphasic aphasias, but the presence of decreased comprehension can give a clue to the posterior location. Anomia is usually severe and impaired syntax can also be part of the clinical presentation. During repetition, the patients’ verbal output is better than conversational speech. Repetition is one of the most important tests to assess in extrapyramidal aphasias, as it is relatively spared compared with other language functions.

Comprehensive language test batteries for the diagnosis of aphasia usually includes spontaneous language (telling a story, describing a picture), language comprehension (phoneme discrimination, pointing to objects, body parts, colors, or actions, and following commands), naming of objects (body parts, colors, or actions, repetition of syllables, easy and difficult words, short and long sentences), comprehension and reading (letters, pseudowords, words, sentences, and paragraphs), and writing (spontaneous, by dictation, by copy of letters, pseudowords, words, sentences, or paragraphs).

**TAKE-HOME POINTS**

- When some symptoms do not fit the presumed vascular territory, rethink the neuroanatomy.
- Aphasia can be caused by lesions in different part of the brain.
- Aphasia, amnesia, vertical gaze palsy, skew deviation, and decreased level of consciousness suggest a lesion in paramedian vascular territory of thalamus.
- Progressive and fluctuating symptoms are common, especially in posterior circulation stroke.

**Disclosures**

None.

**Suggested Reading**


**KEY WORDS:** aphasia ■ diagnosis ■ thalamus
Young Patient With Aphasia, Monoparesis, Facial Drop, Facial Sensory Loss, Diplopia, Dysarthria, and Vertical Gaze Palsy
Daniel Strbian and Sophia Sundararajan

Stroke. 2013;44:e146-e147; originally published online October 15, 2013; doi: 10.1161/STROKEAHA.113.002268

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/44/11/e146

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/