A most unusual case of right-sided ataxic hemiparesis with left trigeminal involvement is reported. Computerized tomographic scan revealed a small hemorrhage in the basis pontis. (Stroke 1987; 18:244-245)

A TAXIC hemiparesis is a cerebrovascular syndrome characterized by cerebellar-like ataxia, weakness, and pyramidal signs involving the limbs of the same side, the leg more than the arm. Although ataxic hemiparesis has been recognized as one of the lacunar syndromes, lesions other than lacunes, such as small pontine hemorraghes, have been reported as a possible causative lesion of the syndrome. 2,3

I report a most unusual case of a right-sided ataxic hemiparesis with left trigeminal involvement in which computerized tomographic (CT) scan revealed a small pontine hemorrhage in the basis pontis.

Subject and Methods

This 59-year-old hypertensive man experienced the sudden onset of dysarthria and weakness of the right arm and leg following occipital heaviness and nausea on February 2, 1985. The day after, he was admitted to the Neurological Institute of the University of Bologna. At admission his blood pressure was 180/110 mm Hg. He was alert, cooperative, and well-oriented. The neck was supple.

There were no pupillary abnormalities. There was fine horizontal nystagmus on right lateral gaze. Speech was slightly dysarthric, but swallowing was normal. The nasolabial fold on the right was flattened; mild right hemiparesis was present and more marked in the distal portion of the right leg. There was also moderate weakness of the left masster and temporal muscles; the jaw shifted to the left when he opened his mouth fully. Deep tendon reflexes were normal, but the Babinski sign was positive on the right. Sensation was impaired on the left side of the face, and corneal reflex was hypoactive on the left. There was right dysdiadochokinesis, and the finger-nose-finger test, shin-tapping test, and heel-shin test showed impairment on the right out of proportion with his weakness.

EEG, EKG, and laboratory data were normal. CT scan revealed a small hematoma in the half rostral basis pontis (Figure 1). Angiography of the vertebrobasilar system revealed no pathological findings. In the following days there was a rapid improvement. Nystagmus, dysarthria, and cerebellar signs disappeared. Sensation of the face returned to normal, and left masticatory weakness disappeared. Repeat CT scan on February 19 showed resolution of the high-density lesion in the pons. He was discharged with only minimal right hemiparesis 20 days after admission.

Discussion

Ataxic hemiparesis due to contralateral pontine hemorrhage has been reported and well-documented in the literature. 2,3 In the previously reported cases, CT scan showed the presence of a small hemorrhage in the base of the contralateral pons. In all the cases the recovery was complete or almost complete, and repeat CT scan showed the resolution of the lesion. 2,3 If the lesion extends laterally, as in the present case, the fifth cranial nerve can be also involved at the nuclear or intranuclear level. In 1981 Sakai et al reported a patient who experienced a right-sided ataxic hemiparesis with left masticatory involvement in which CT scan revealed an infarct in the left pons. 4 The authors could not explain why the exiting trigeminal sensory nerve was not impaired despite close proximity to the trigeminal motor nerve. In my case both sensory and masticatory fibers of the trigeminal nerve were affected. This finding has not been previously reported in patients affected by ataxic hemiparesis due to pontine lesion. Trigeminal impairment in ataxic hemiparesis should be carefully researched, especially when the lesion in the pons is more laterally located than has been classically described.
Ambrosetto Ataxic Hemiparesis Due to Pontine Hemorrhage

FIGURE 1. Unenhanced CT scan showing a small hemorrhage in the left basis pontis.

References


KEY WORDS • ataxic hemiparesis • pontine hemorrhage
Ataxic hemiparesis with contralateral trigeminal nerve impairment due to pontine hemorrhage.

P Ambrosetto

*Stroke*. 1987;18:244-245
doi: 10.1161/01.STR.18.1.244

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1987 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

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/18/1/244

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/