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"Wrong-Way Eyes" in Supratentorial Hemorrhage

Michael S. Pessin, MD,* Lester S. Adelman, MD,+ Robert J. Prager, MD ± Ellen S. Lathi, MD,* and Dale J. Lange, MD*

A patient developed conjugate horizontal eye deviation to the side opposite a large frontal-perisylvian hemorrhage from a ruptured aneurysm. Autopsy examination confirmed the hemispheric locus of the hemorrhage and failed to disclose any lesion of the brainstem or thalamus to explain the "wrong-way" eye deviation.

Pessin MS, Adelman LS, Prager RJ, Lathi ES, Lange DJ: "Wrong-way eyes" in supratentorial hemorrhage. Ann Neurol 9:79-81, 1981

A patient is described who had the unexpected finding of conjugate horizontal eye deviation to the side opposite a large supratentorial hemorrhage ("wrong-way eyes").

A hypertensive 57-year-old woman was transferred to New England Medical Center Hospital in September, 1979, because of left hemiplegia. That morning she had developed a severe occipital headache and stiff neck, immediately followed by weakness, sweating, diarrhea, nausea, and vomiting. At another hospital a left hemiparesis with eyes deviated to the left was noted.

At her admission, blood pressure was 190/110 mm Hg with a regular pulse of 98. Her neck was supple. The general physical examination was normal. The patient was awake with her eyes open and blinking spontaneously, but she did not respond to verbal instructions. Her eyes deviated fully to the left in the horizontal plane, with no observable nystagmus. Both oculocephalic testing and icewater caloric stimulation of the right external auditory canal produced full horizontal conjugate eye excursion to the right. Her pupils were 3 mm in diameter and reacted normally to light stimulation. Visual threat from either side produced a blink response. The optic discs were flat, and retinal hemorrhages were not seen. Weakness of the left lower face was present. The gag reflex was intact. The right arm and leg moved spontaneously, and painful sternal rub produced a purposeful response by the right hand but decerebrate posturing on the left. Other sensory modalities could not be reliably tested. A flaccid, areflexic left hemi-

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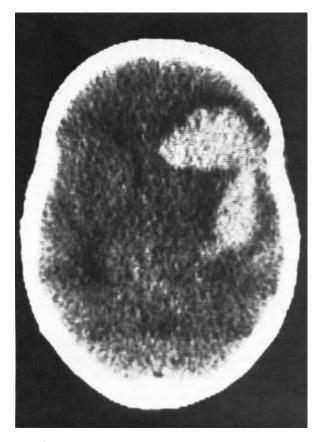


Fig 1. Axial nonenhanced CT scan at the level of the frontal horns.

plegia was present. Plantar responses were extensor bilaterally.

A computed tomographic scan (Fig 1) demonstrated a large area of hemorrhage in the right frontal and perisylvian regions with compression of the right lateral ventricle and a shift of midline structures to the left. Other scan slices showed blood in the right lateral and fourth ventricles.

Her eyes remained conjugately deviated to the left, but by afternoon both pupils were 4 mm in diameter and unreactive to light. Decerebrate posturing to minimal stimulation was present bilaterally. Her condition did not improve, and during the night she died.

Neuropathological Findings

Recent subarachnoid hemorrhage was present over the base of the brain, in the right sylvian fissure, and over the convexity of the right cerebral hemisphere. Three saccular aneurysms were found on the intracerebral arteries. The largest (0.7 cm) was at the bifurcation of the right middle cerebral artery in the sylvian fissure. A ragged tear measuring 0.3 cm in longest dimension was present on the lateral aspect of the sac, and this was presumed to be the source of the hemorrhage. Small unruptured aneurysms were found on both anterior cerebral arteries.

The brain was sectioned in the plane of the CT scan. A crescentic hemorrhage was found extending

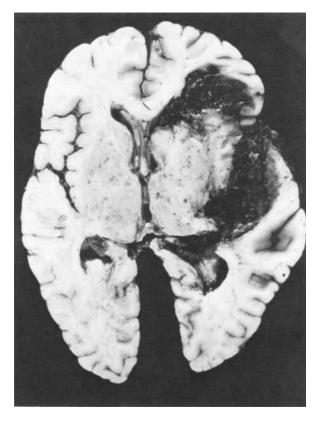


Fig 2. Section of the cerebral hemispheres in the plane of the CT scan, showing the intracerebral hemorrhage.

into the frontal white matter from the subarachnoid hemorrhage in the right sylvian fissure. It reached the right frontal horn anterior to the basal ganglia and dissected caudal to the thalamus. The thalamus was not involved, and, although a small portion of the caudal putamen was destroyed by the hemorrhage, the rest of the basal ganglia were intact (Fig 2). A small secondary midline hemorrhage was seen in the midbrain, but the pons was not involved. Microscopic sections confirmed the gross findings. The thalamus, pons, and basal ganglia (apart from the caudal putamen) were normal histologically.

Discussion

It is well known that supratentorial hemorrhage may produce contralateral hemiplegia with conjugate horizontal eye deviation away from the paralysis and toward the side of the lesion. By contrast, in a pontine lesion the eyes may conjugately deviate toward the side of the hemiplegia.

In 1967, Fisher [1] first called attention to the occurrence of conjugate eye deviation to the "wrong" side in supratentorial, hypertensive hemorrhage. He reported three patients with pathologically proved thalamic hemorrhage and a massive collection of blood within the third ventricle whose eyes had conjugately deviated away from the side of the lesion and toward the hemiplegia. No anatomical or physiological explanation for this finding was apparent. Keane [3] later corroborated Fisher's observation in three patients with pathologically verified supratentorial hemorrhage, all involving the thalamus. More recently [6] the same conjugate gaze abnormality was reported in three of eighteen patients with thalamic hemorrhage documented by CT scan. This uncommon gaze disorder has not been reported in association with hypertensive hemorrhage at other supratentorial sites [4], including the putamen [2].

In contrast to the other reported cases [1, 3, 6], our patient had neither a hypertensive thalamic hemorrhage nor a massive collection of blood in the third ventricle. Her parenchymatous hemorrhage resulted from rupture of a saccular aneurysm of the middle cerebral artery and was associated with subarachnoid as well as intraventricular blood. However, the parenchymal extension of the hematoma was a destructive mass lesion similar to a primary hypertensive hemorrhage.

We considered but dismissed several factors as the cause for this patient's gaze preference, including the possibility that the hematoma had acted as an epileptic (irritative) focus, forcing the eyes in the opposite direction. Although our patient did not have an electroencephalogram, no focal or generalized seizure activity was observed in the limbs, and nystagmoid jerking of the eyes-an expected feature of a frontal cortex seizure focus [5]-was absent. Furthermore, two of Keane's patients (Cases 1 and 3 [3]) had an electroencephalogram during the period of their conjugate gaze disorder, and only bilateral slowing was observed. The pathological examination ruled out the possibility that the patient's conjugate leftward eve deviation resulted from an additional destructive lesion either in the left hemisphere or in the pons. Thus, we can only document but cannot explain this observed conjugate gaze preference to the unexpected side in association with a supratentorial hemorrhage that spared the thalamus.

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Postictal Pleocytosis

James W. Schmidley, MD, and Roger P. Simon, MD

Six patients showed a transient and otherwise unexplained cerebrospinal fluid (CSF) pleocytosis following a flurry of generalized convulsions. Each had an obvious cause for repeated seizures. No evidence was found for an infectious, inflammatory, neoplastic, or other cause for the pleocytosis. All CSF specimens were clear and colorless, under normal pressure, and bacteriologically sterile. The maximal leukocyte count ranged from 9 to 80 per cubic millimeter and reached a maximum on the day after cessation of convulsions. No specimen contained more than 650 erythrocytes. Two patients initially had a mildly increased CSF protein; glucose values were unremarkable.

We propose that the pleocytosis in these patients was a result of frequently repeated generalized convulsions. The mechanism of postictal pleocytosis is uncertain. It may result from transient breakdown of the blood-brain barrier, which has been demonstrated after seizures in experimental animals. Although infectious causes must first be considered and rigorously searched for, it appears that seizures alone may cause a transient CSF pleocytosis.

> Schmidley JW, Simon RP: Postictal pleocytosis. Ann Neurol 9:81-84, 1981

Among a large number of patients admitted to a city hospital with repetitive generalized convulsions, we have encountered six in whom the cerebrospinal fluid (CSF) showed an otherwise unexplained mild, transient pleocytosis. While the occurrence of such postictal pleocytosis has occasionally been mentioned in the literature [4, 5, 8], the phenomenon is not well documented; its time course has not been reported in detail [15].

Case Summaries

Patients 1, 2, and 3 were young, healthy people without a history of tuberculosis or epilepsy, who took overdoses of isonicotinic acid (INH) and were admitted after repeated grand mal convulsions. All exhibited the profound metabolic acidosis characteristic of INH poisoning, and two showed the typical hyperglycemia [1]. Physical examinations and laboratory studies revealed no other pertinent abnormalities. When INH poisoning was suspected, pyridoxine was administered. Two patients were given

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