DIPLOPIA AND INVOLUNTARY EYE CLOSURE/Messert et al.


Diplopia and Involuntary Eye Closure in Spontaneous Cerebellar Hemorrhage

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SUMMARY Spontaneous cerebellar hemorrhage is of difficult clinical diagnosis. The causes can be varied, but the hemorrhage is most often associated with hypertensive cardiovascular disease. The neurological symptomatology is complex and often misleading. The diagnosis is mainly dependent on familiarity of the eye signs seen in this disease. Among these, the spontaneous unilateral eye closure is presented as an additional striking manifestation. The displacement of the brain stem by the hematoma is frequently associated with a seventh nerve palsy on the side of the hemorrhage. The patient, in an effort to obviate the diplopia caused by the gaze dissociations and extracranial motor palsies, has only the option to close the eye on the noninvolved side of the face, and thus the eye remaining open is on the side of the cerebellar hematoma. This paper presents reports of two patients with these symptoms.

THE CLINICAL DIAGNOSIS of spontaneous cerebellar hemorrhage is often a difficult one and few antemortem diagnoses are made. Reports by Fisher et al. and McKissock et al. have done much to describe the clinical aspects of the disease.

Spontaneous cerebellar hemorrhage in the majority of cases is associated with hypertensive vascular disease, and less frequently related to a number of other causes such as arteriovenous malformation, bleeding diathesis, infections of the central nervous system and trauma. The clinical spectrum of signs and symptoms can vary considerably from acute onset with coma and death within a few hours, to subacute and chronic courses simulating brain stem vascular infarctions or posterior fossa tumors.

The subacute cases are those presenting the greatest clinical challenge. The outstanding neurological manifestations are centered in the cranial nerves with diplopia, nystagmus, EOM abnormalities, paresis of conjugate gaze to one side, or even forced conjugate deviation to the other. A decreased corneal reflex on one side is frequently associated with a peripheral facial palsy on the same side.

The headache when unilateral, the facial palsy, the corneal reflex deficit, and the direction of gaze palsy all point to the side of the posterior fossa involvement and are homolateral to the hematoma.

The difficulty in diagnosis seems to reside in the rather unusual and paradoxical absence of expected manifestations. There is often no blood in the spinal fluid obtained by lumbar puncture. Significant neck rigidity is rare. There are few reports of observed papilledema. The pupils remain reactive, cerebellar ataxia, if present at all, is unimpressive and often absent, and often there are no paresis and no sensory change in the extremities. Thus, at this stage of the illness, the eye signs are by far the most characteristic features. The purpose of this report is to focus on the interesting phenomenon of the closure of one eye found early in the course.

This closure of one eye was noted by Fisher et al. and termed "involuntary closure of one eye." He thought the left eye was most usually involved and that it reflected brain stem damage. Lichtenstein, quoting Fisher, described "apparent ptosis of one eyelid" and stated that this was associated with an internal ophthalmoplegia. Photorphobia and/or reflex blepharospasm are interpretations of the eye closure in other case reports.

It is our interpretation that the closure of one eye is only the reflex avoidance of diplopia in these patients who have a marked and complex ophthalmoplegia of acute onset; the side of closure is simply determined by the presence of the associated facial palsy (Case 1). However, when the facial palsy is minimal or even absent, the eye closure can occur randomly on either side (Case 2).
Case Reports

Case 1
A 59-year-old hypertensive man had sudden onset of severe headache, vomiting and vertigo while playing golf on October 20, 1967. When admitted to a local hospital four days later, he was in acute distress, unable to sit up in bed, and vomiting. The blood pressure was 210/100 mm Hg. The patient was noted to present nystagmus on gaze to the left. He was transferred to the Veterans Administration Hospital (Madison, Wisconsin) on October 30, 1967.

On admission the patient was noted to keep his eyes closed most of the time, but was able to open them on command. Other abnormal neurological signs were: spontaneous nystagmus to the left with increase in nystagmus on attempted gaze to the left, mild paresis of abduction of the left eye on left lateral gaze, left peripheral facial palsy, corneal reflex decreased on the left, without any paresis in the extremities, and mild finger-to-nose dysmetria on the left. There was no stiffness of the neck or papilledema. The blood pressure was 250/130 mm Hg.

Over the next few days, as the patient became more comfortable, he was found to keep only his right eye closed, but was quite able to open it on command. He denied diplopia in spite of the obvious evidence of disjunction of the ocular axis in left lateral gaze.

Lumbar puncture on admission showed xanthochromic fluid and a pneumoencephalogram was consistent with a mass in the left posterior fossa. A craniotomy was performed on November 8, 1967, and a 20-cc hematoma was removed from the left cerebellar hemisphere. Within a week after surgery, nystagmus had receded, the patient was comfortable, and the right eye closure was not present any longer. The patient was discharged entirely asymptomatic.

Case 2
A 50-year-old white married man, with a two-year history of chronic myelogenous leukemia, was admitted for a routine evaluation of his anemia on September 17, 1968. On admission the patient was cachectic and ashen in appearance, but in no acute distress and quite alert. Laboratory evaluation showed the hematocrit to be 21, hemoglobin 7 gm percent, white count 110,000, and platelet count 26,000. He received two units of packed cells.

Just prior to leaving the hospital, he complained of severe right occipital headache. Vomiting occurred, accompanied by vertigo. The patient remained alert and oriented, but assumed a fetal position in bed. Both eyes were kept tightly closed most of the time, but on occasion he opened the left eye spontaneously. When asked to open his eyes he could cooperate, showing nystagmus in all directions of gaze. However, there were marked paresis of gaze to the right and forced conjugate deviation of the eyes to the left. On attempted gaze to the right there was marked dissociation of the ocular axis with paresis of abduction of the right eye and increase in nystagmus. Corneal reflexes were present and equal bilaterally. Pupils were normal. There was no evidence of facial weakness, neck stiffness or eyeground changes. Bilateral Babinski signs were present.

A diagnosis of right cerebellar hemorrhage and hematoma was made, but in view of the known hematological disorder, only supportive care was given. The eye closure was on the right. The patient died suddenly, about 24 hours after the onset of the acute headache, with signs and symptoms of craniocaudal deterioration and respiratory failure.

The postmortem examination revealed a large right intracerebellar hematoma with spread into the fourth ventricle and dislocation of the brain stem to the left (fig. 1).

Conclusions
In a disease of difficult clinical diagnosis manifested by complex eye signs, it seems worthwhile to call attention to the striking sign of unilateral eye closure. This sign, in the setting described in this paper, could possibly serve to alert the physician to the possibility of a cerebellar hemorrhage, lead to the critical evaluation of other presenting signs, and even point to the side involved.
References


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