Divergence Paralysis Due to a Small Hematoma in the Tegmentum of the Brainstem

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There has been considerable controversy concerning divergence paralysis, an entity described as early as 1883 by Parinaud. We recently observed a patient with divergence paralysis, who on CT scan presented a small hematoma in the tegmentum of the brainstem. This case may support the theory that the center for divergence exists in the upper brainstem.

Key Words: Divergence paralysis, hematoma, brainstem

Divergence paralysis (DP) was first described as a clinical entity by Parinaud in 1883, and the most complete description of this condition was made by Duane in 1899 (Dunnigton 1923). However, there is not only considerable controversy concerning the nature of DP, but also some doubt as to the validity of DP as a clinical entity (Dunnigton 1923; Chamlin and Davidoff 1951; Urist 1952). Jampolsky contend ed that DP and bilateral sixth nerve palsy, two clinical entities were inseparable and “that it would be more fruitful to consider so called cases of DP to be in reality cases of bilateral sixth nerve palsy (Scheiman et al. 1986).” This concept was supported by an electro-oculographic study, which showed impaired abduction in three cases of diagnosed DP even though other signs of bilateral lateral rectus palsy were absent (Kirkham et al. 1972). Many authors, however, do not agree to Jampolsky’s total rejection of the entity of DP, and accept DP as an important clinical entity (Scheiman et al. 1986).

DP has been observed in a variety of conditions; chorea, syphilis, encephalitis, multiple sclerosis, head trauma, increased intracranial pressure, brain tumor, vascular lesions of the brainstem, and cerebral hemorrhage (Dunnigton 1923, Leboysohn 1926, Robbins 1941, Lippmann 1944, Savitsky and Madonick 1946, Chamlin and Davidoff 1950, Deutsch 1950, Lyle 1954, Cunningham 1972, Rutkowski 1972). However, DP due to a localized hemorrhage is rare in the literature and we could find no report of it on CT brain scan.

CASE REPORT

A 62-year-old man was admitted to Severance Hospital for an abrupt onset of vertigo, vomiting, and diplopia. He was relatively healthy until 10 days prior to admission, when he began to have a frequent cough and blood-tinged sputum. One day before admission, vertigo, vomiting, and diplopia developed abruptly during defecation in the morning. He visited a local clinic, where his blood pressure was measured as 230/170 mmHg. Then he was transferred to Severance Hospital.

There was a long history of untreated hypertension. Three years prior to this admission, pulmonary tuberculosis was diagnosed and treated for one year.

On admission his temperature was 36.6°C, pulse 80, respiration 20, and blood pressure 160/100. On physical examination the patient appeared acutely ill. Coarse breathing sounds were noted over both lung bases with moist rales. Heart and abdomen were normal. No lymphadenopathy was found. Rectal examination was negative. On neurologic examination, the patient was alert and oriented with fluent speech. The ocular movements were full in all directions. But uncrossed diplopia was present beyond 70 cm and did not change on lateral gaze. Increasing distance fixation produced wider separation of the images. The pupils were equal and reacted to light and in accommodation. There was no end-point nystagmus. The remaining cranial nerves were normal. Muscle strength
was normal and sensation was intact. The patient could not perform the finger-to-nose test on either side. The tendon reflexes were hyperactive on both extremities but the plantar responses were flexor.

A brain CT scan revealed a small increased density localized just to the right of the midline in the tegmentum of the upper pons, extending to midbrain (Fig. 1). This finding was indicative of a hypertensive hemorrhage in that area. Chest PA and lateral views showed mass-like densities in the right middle lobe and the superior segment of the left lower lobe. Chest CT scan revealed multiple nodular densities scattered in both lung fields. Cytologic examination of the sputum was positive for malignant cells on three occasions even though bronchoscopic biopsy revealed only chronic nonspecific inflammation. Three sputum cultures for acid-bacilli were positive.

On the tenth hospital day, the patient was discharged against medical advice, and at that time he showed no improvement in the uncrossed diplopia.

DISCUSSION

DP is difficult to differentiate from minimal bilateral sixth nerve palsy. Bedrossian (1958) stated that “when one reviews the reported cases of DP, one is impressed with the fact that accurate fields of fixation have been neglected in most cases, and many cases of mild bilateral sixth nerve palsy are misdiagnosed as DP.” He urged clinicians to assess comitancy carefully and to look for end-point nystagmus before reaching a diagnosis of DP. If a mild noncomitancy is present or nystagmus exists, a diagnosis of sixth nerve palsy may be more appropriate (Bielschowsky 1935; Bedrossian 1958; Scheiman et al. 1986).

The case reported here has all of the characteristic clinical features of the entity commonly designated as DP. The acute onset, uncrossed diplopia, which was present beyond 70 cm and was not changed on lateral gaze, no end-point nystagmus, and no limitation of eye movements bear out this diagnosis. A brain CT scan showed a small hematoma just to the right of the midline in the tegmentum of the brainstem, which probably was the cause of DP in this case.

There has been considerable speculation regarding the site of a specific divergence center that could be responsible for DP (Holden 1921; Howard 1931; Saitsky and Madonick 1946; Lyle 1954; Cunningham 1972). Some investigators insisted that divergence simply represents a relaxation of convergence (Scobee and Green 1946). However, Bruce (1935) suggested that divergence is an active process and argued from a teleologic basis that it would be inconceivable that convergence would be a neural mechanism without an antagonistic system. Several studies (Adler 1953; Breining and Moldaver 1955) have provided electromyographic data supporting the concept of an active process of divergence. Many authors (Dunnington 1923; Bielschowsky 1935; Bruce 1935) proposed a
brainstem center for divergence near the abducens nuclei. Lippmann (1944) supported the theory of a mesencephalic center for divergence with one case of tumor of vermis cerebelli. However, Bender and Savitsky (1940) reported one case of DP with an autopsy finding of a small cavemos hemangioma in the periaqueductal central gray matter at the level between the superior and inferior colliculi. Their hypothesis of a mesencephalic center might be supported by Mays' report (1984), in which divergence cells were found in the mesencephalic reticular formation just dorsal or dorsolateral to the oculomotor nucleus. The divergence center in our case is probably located in the tegmentum of the brainstem.

REFERENCES


