

## A Case of Bilateral Ptosis Associated with Cerebral Hemispheric Lesions

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**Abstract:** Ptosis occurs in a variety of disorders including myasthenia gravis, oculomotor palsy, Horner's syndrome and brain stem disorders. There are also supranuclear lesions causing blepharoptosis. The latter disorders are reflex blepharospasm, apraxia of eyelid opening and Meige's syndrome. Since the total number of bilateral ptosis associated with cerebral hemispheric lesions is very few, whether the responsible lesions are located in the nondominant hemisphere or bilateral hemispheres are still controversial. We report here a case of bilateral cerebral ptosis that occurred in association with cerebral infarction of the nondominant hemisphere.

**Key Words:** *blepharoptosis, cerebral infarction*

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### CASE REPORT

The case was a right-handed 73-year-old male, a retired clerk.

Chief complaints: Bilateral ptosis.

Family history: Nothing contributory.

Past history: He was told he had essential hypertension at the age of 44, but was not treated with any medications.

Present illness: At age 67, he experienced his first stroke which caused left hemiparesis and dysarthria. He was admitted to a nearby hospital with a diagnosis of cerebral infarction. After he received rehabilitation therapy he left the hospital without any neurological deficits. Since then he was taking antihypertensive and antihyperlipidemic medications. When he was 70, he again experienced left

facial weakness, left hemiparesis, and difficulty in opening his eyes. After this attack, he had occasional headaches. He received physiotherapy which improved his left hemiparesis. However, he continued to have difficulty in opening his eyes which made him visit our hospital.

Physical examinations on admission: His height was 162 cm, and the body weight was 51.5 kg. His B.P. was 150/90 mmHg, pulse 84/min, regular, and temperature was 35.3°C. He had no anhidrosis. Other general physical examinations were unremarkable.

Neurological examinations: He was awake and alert. He was oriented and able to perform simple calculations. His general fund of knowledge was normal. His tone of speech was monotonous, and had a mild dysarthria. He always had bilateral ptosis without any fluctuations during the whole day. The pupils were equal and reactive to light and accommodations. The extraocular movements were full (Fig. 1). He had no apraxia of opening

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Fig. 1: He always had symmetrical bilateral ptosis without fluctuations during the whole day. The extraocular movements were full.

the eyelids, and no blepharospasms were noted. He had a slight mask-like face. He had no weakness of orbicularis oculi, nor orbicularis oris. He had some difficulty in wrinkling the forehead bilaterally. The nasolabial fold was shallow on the left side. The gag reflex and palatal movements were normal. He had a mild weakness in all the four extremities without laterality. No rigidity of the neck was noted and he had no difficulty in elevating his neck. His gait was slightly wide based, but otherwise no ataxia was noted in the finger to nose and the heel to shin tests. The sensory systems were normal. The deep tendon reflexes were moderately increased bilaterally. Extensor toe signs were negative bilaterally.

#### LABORATORY DATA

Urinalysis, CBC and electrolytes were all within the normal limits. Uric acid was 5.0 mg/dl. The total cholesterol was 186 mg/dl, and triglyceride was 67 mg/dl. FBS was 108

mg/dl. Other serum chemistry studies were also all within the normal limits. Antiacetylcholine esterase receptor antibody was negative. Chest x-p, abdominal films and ECG were all normal. Pulmonary function tests were normal. His EEG was normal. Repetitive nerve stimulations revealed no waxing, nor waning of M-waves. A tension test was negative, and no objective improvement of bilateral ptosis, nor any increase in the muscle strength was noted by an intravenous edrophonium injection. The cranial CT scan revealed multiple low density areas in the right and left internal capsules, and the white matters near the anterior horns of the lateral ventricles and also in the frontotemporal subcortical areas bilaterally (Fig. 2A, 2B). No lesions were noted in the brain stem.

#### HOSPITAL COURSE

From the results, myasthenia gravis was ruled out. His bilateral ptosis was considered to be due to his cerebral infarctions from

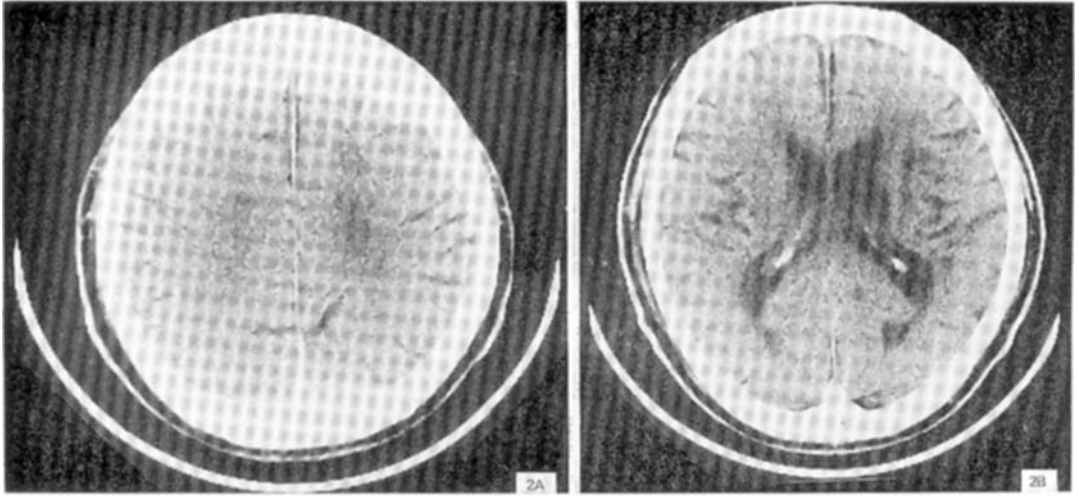


Fig. 2A, 2B: The cranial CT scan revealed multiple low density areas in the right and left internal capsules, the white matters near the anterior horns of the lateral ventricles, and also in the frontotemporal subcortical areas bilaterally.

the clinical history and low density areas in the cerebral hemispheres by CT studies, since the extraocular movements were full, and there were no brain-stem symptoms and signs other than bilateral ptosis. The responsible lesions of his bilateral ptosis were attributed to supranuclear lesions, i.e. multiple cerebral infarctions. The risk factors of cerebral infarctions were investigated. His risk factors were hypertension, and hyperlipidemia, which were treated with nifedipine 10 mg twice a day and probucol 250 mg twice a day. This treatment improved his headache.

#### DISCUSSION

It has been known that bilateral ptosis may occur in association with a cerebral hemispheric lesion. In 1974, Caplan reported 25 cases of blepharoptosis that occurred after acute strokes.<sup>1</sup> He commented that most of cerebral ptosis previously reported may be mistakenly attributed to oculomotor palsy caused by increased intracranial pressure in the cases of brain tumors, and other disorders. He classified the 13 cases of blepharo-

ptosis which occurred after a cerebral infarction as a clinical entity, since it could not be explained by either a third nerve or sympathetic dysfunction. He developed a hypothesis that there might be a hemispheric center whose lesion could cause this bilateral ptosis.

In 1977 Nutt reported 2 cases of bilateral blepharoptosis after a cerebral infarction of the nondominant hemisphere.<sup>7</sup> Since these patients had frontal release signs, he considered that bilateral cortical lesions caused this manifestation, as pseudobulbar palsy is caused by bilateral hemispheric lesions.

In 1978 Krohel and others reported a case of bilateral blepharoptosis which occurred to a patient with a recent right frontal infarction, who had had a left frontal infarction.<sup>4</sup> In this case the author considered that the bilateral frontal lesions were causing bilateral blepharoptosis.

Recently Lepore reported 13 cases of bilateral blepharoptosis in association with cerebrovascular accidents in the right frontotemporal areas.<sup>5</sup> All the patients had left hemiparesis, left hemisensory loss, left facial palsy and left conjugate gaze dysfunction.

Eleven of the 13 cases had dysarthria, 8 cases had deviation of the tongue to the left, 8 cases had left homonymous hemianopsia, and 5 cases had high cortical dysfunction. Among the 13 cases, 3 cases had no abnormal findings in the left cerebral hemisphere by CT scan examinations and all the 13 cases had a strong relationship between the right hemisphere lesions and bilateral blepharoptosis. In addition there is a tendency that the right hemisphere lesions often cause conjugate gaze paresis,<sup>2</sup> and reflex blepharospasms which were supranuclear movement disorders of the eyelids caused by the nondominant hemisphere lesions.<sup>3</sup> Therefore, elevation of the eyelids may be ruled by the nondominant hemisphere similar to the conjugate gaze function. Moreover, there are two cases of left hemisphere arteriovenous malformations who had bilateral ptosis, which improved after surgery.<sup>6</sup>

Our case had bilateral cerebral disorders from both the neurological manifestations as well as CT findings. Since there were no abnormalities of the pupils, extraocular movements, nor sympathetic disturbances, we attribute his bilateral blepharoptosis to supranuclear lesions; however, it is not certain whether the responsible lesions can be located in the right hemisphere alone or in both hemisphere. It is interesting to note that the history of our patient is similar to

Lepore's cases. Bilateral blepharoptosis of our patient occurred in association with left facial palsy and left hemiparesis. There is a strong possibility that the lesions in a non-dominant hemisphere can cause bilateral blepharoptosis. Our patient is one of the rare cases of bilateral blepharoptosis associated with nondominant hemispheric lesions and it is important to help elucidating the pathophysiological mechanisms of supranuclear blepharoptosis.

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