

## Ptosis in Patients with Cerebral Strokes

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### Abstract

**Introduction:** Cerebral ptosis is, rare but its frequency in patients with strokes has not received systematic study.

**Objective:** To determine the frequency of ptosis in patients with acute hemispheric stroke and to identify stroke features associated with ptosis.

**Methods:** Eyelid function was studied in 48 consecutive patients with acute hemispheric stroke and 40 age-matched subjects with no known neurological disease. All underwent comprehensive neuro-ophthalmologic and general neurological examination within 48 hours of admission, including measurement of palpebral fissures, marginal reflex distance, and range of upper lid movement.

**Results:** 12 cases (25%) had ptosis, which was bilateral in 2 and unilateral in 10. In one patient with large hemispheric infarction, complete bilateral or asymmetric ptosis was the first sign of imminent herniation, preceding pupillary dilation and ocular motor deficits.

**Conclusion:** Ptosis occurs frequently in patients with hemispheric strokes, especially in association with right hemispheric lesions. Complete bilateral ptosis is usually caused by large infarctions and may be a premonitory sign of an impending herniation.

**Keywords:** Ptosis; Cerebral Stroke; Neuro-ophthalmologic Examination; CT Scan

### Introduction

Ptosis is rare with strokes but though reported in literature, systematic study is lacking for this important sign of neurological malfunctioning. Ptosis in acute stroke, not explained by oculomotor nerve or sympathetic dysfunction has been termed cerebral ptosis [1] and may be more common than previously recognised [2]. The precise anatomical basis for this type of ptosis including complete bilateral ptosis (CBP) remains speculative but has been described most frequently in cerebral strokes in general and massive right hemispheric lesions [3] in particular. Why this occurs and the potential clinical value of this phenomenon has not been described clearly.

### Aim of the Study

To determine the frequency of ptosis in patients with acute hemispheric stroke and to identify stroke features associated with ptosis. This study further characterizes this phenomenon, particularly with reference to its time course and relationship to cerebral herniation syndromes.

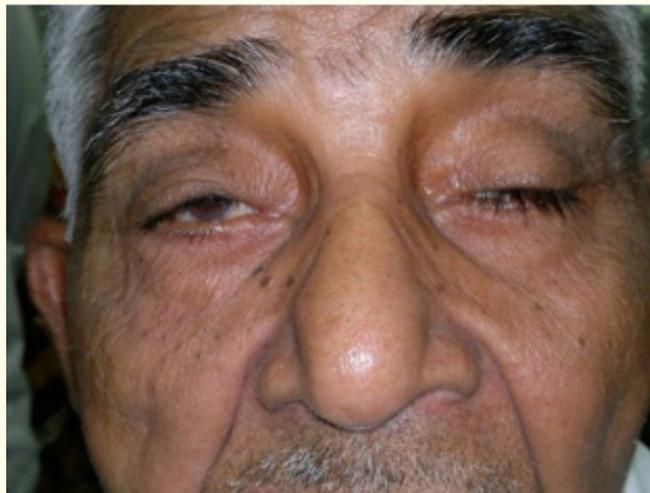
## Methods

This study includes 48 consecutive patients with acute hemispheric stroke and 40 age-matched subjects with no known neurological disease. Patient who had haemorrhagic stroke or TIA, too drowsy to cooperate with the neuro-ophthalmologic examination, had signs of brain stem disease, levator dehiscence and dermatochalasis were excluded. Cerebral ptosis was diagnosed when unable to open eyelids to show any underlying sclera, alert enough to follow a simple command promptly, and able to adequately move other orofacial musculature on command. The simple command was to have the patient show 2 fingers of the right hand to the examiner. All patients admitted to the neurology/neuro surgical unit with stroke were examined. All underwent comprehensive neuro-ophthalmologic and general neurological examination within 48 hours of admission, including measurement of palpebral fissures, marginal reflex distance, and range of upper lid movement.

Particular attention was paid to the time course of the ptosis and to the appearance of oculomotor nerve dysfunction or pyramidal signs ipsilateral to the infarction (i.e. right-sided weakness or extensor plantar response). Cranial computed tomographic (CT) scans were reviewed and clinical courses recorded

## Results

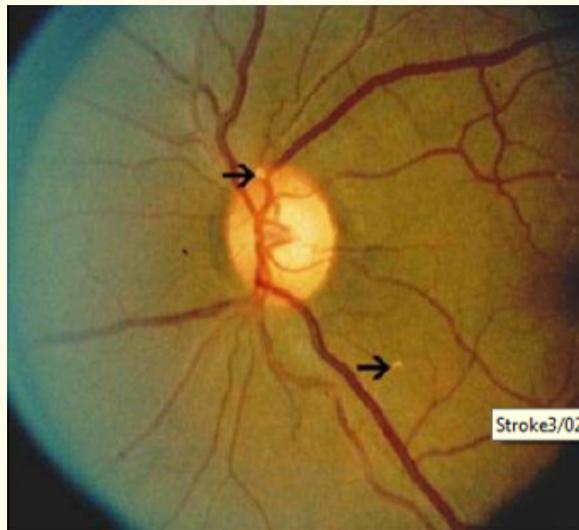
48 patients admitted in Bokaro General Hospital during 2003 - 2004 with stroke were the cohort of this study. Male: Female ratio was 3:1, mean age 56.4 yrs (38 - 70 yrs). 12 cases (25%) had ptosis, which was bilateral in 2 and unilateral in 10. All patients initially had Glasgow Coma Scale scores of 15. In two patients with large hemispheric infarction, complete bilateral or asymmetric ptosis was the first sign of imminent herniation, preceding pupillary dilation and ocular motor deficits. This case had left visual field deficits, right gaze preference or deviation, impaired up gaze, left facial weakness upper motor neurone distribution but with some involvement of frontalis), leftward tongue deviation, dense left hemi paresis (with at best a flicker of leg movement), and left hemi sensory loss with varying degrees of visuospatial inattention. Initial CT scans showed a hyper dense middle cerebral artery sign in all patients, and some scans showed early signs of ischemia exceeding 50% of this territory. In 2 patients, the etiology was due to right internal carotid artery occlusion. In 1 patient, a cardio embolic source was suggested.



**Figure 1:** Persistent cerebral ptosis (left) following ischemic stroke.



**Figure 2:** Persistent cerebral ptosis (right) with full ocular movement following ischemic stroke, Subconjunctival haemorrhage following anticoagulant therapy.



**Figure 3:** Hollenhorst plaque; proximal source of arterial or cardiac emboli.

### Discussion

The central caudal nucleus of the oculomotor nerve comprises the motor neuronal pool for the levator palpebrae superioris [4]. Isolated focal lesions of this midbrain region have been associated with CBP but are rare. Larger lesions are more commonly associated with nuclear ophthalmoplegia [4]. Thus, it is easy to understand how the mass effect of a large hemispheric infarction could cause distortion and dysfunction of this midbrain structures area anterior to the motor strip. Considering the observations in these studies, it would

seem that sub cortical and possibly more medial structures (as suggested by the patient with the anterior cerebral artery occlusion) may produce ptosis. The delayed appearance of ptosis in our patients has not been reported before and suggests that the shift of brainstem structures from an expanding swollen mass is responsible, rather than the infarction itself. In our 3 patients who died, CBP occurred 5 to 24 hours before the appearance of contra lateral motor or brainstem clinical signs. This may further suggest that CBP is an important clinical warning that clinicians should recognize [2]. The feature that makes CBP recognizably different from drowsiness is the patient's obvious attempts to raise the eyelids, which is accompanied by vigorous frontalis muscle contractions. This feature has been termed weak asymmetric forehead with fallen lids [5]. Large left hemispheric infarctions are invariably accompanied by global dysphasia, which affects receptive language; thus, because of patients' inability to understand the request to open their eyelids, the ptosis is difficult to recognise.

### Conclusion

Ptosis occurs frequently in patients with hemispheric strokes, especially in association with right hemispheric lesions. Complete bilateral ptosis is usually caused by large infarctions and may be a premonitory sign of an impending herniation. Recent studies have noted that ptosis precedes signs of herniation [2], which also was observed in the current series. The clinical value in recognizing CBP in patients with massive hemispheric infarctions is that it invariably is associated with progression toward herniation. When signs of herniation such as pupillary dilatation develop, the prognosis is extremely poor [5]; therefore, recognition of a clinical sign that precedes other more established signs of herniation could represent an opportunity for early intervention. Current options for massive hemispheric edema are aggressive osmotic therapy with hypertonic saline or mannitol, induced cooling to 33°C to 34°C, and decompressive craniectomy.

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