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# Cerebral Ptosis- A Masquerade to Glasgow Coma Scale

## Case Report

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### List of abbreviations

GCS- Glasgow Coma Scale; CT- Computed Tomography; NIHSS- National Institute of Health Stroke Scale; MRI- Magnetic Resonance Imaging; MCA- middle cerebral artery; ICU- Intensive Care Unit

### Introduction

The Glasgow Coma Scale (GCS) was first described by Graham Teasdale and Bryan Jennett in 1974. It is still widely used as a clinical scale to assess a patient's depth of impaired consciousness and coma following an acute brain injury [1]. The GCS has scores between 3 and 15, 3 being the worst and 15 the best. It measures three parameters: eye response (E), verbal response (V), and motor response (M).

Ptosis can be unilateral or bilateral, partial or complete, occurs in cases of cerebral ischemic or hemorrhagic stroke, without involvement of brainstem or without involvement of ocular mechanism can be termed as cerebral ptosis [2]. It is associated with higher frequency of gaze preference to side of lesion as well as upgaze limitation compared to patients without cerebral ptosis [2]. Fewer studies are conducted to ascertain the GCS score in patients with cerebral ptosis. We describe a case where cerebral ptosis caused a documentation of spuriously low GCS and highlight the importance of detailed ptosis examination in acute cerebrovascular accidents patients.

### Case description

A 69-year-old male presented to the emergency room in the window period of acute ischemic stroke with complaints of left sided weakness with slurred speech. GCS on arrival was 15/15 with National Institute of Health Stroke Scale (NIHSS) of 8. Magnetic Resonance Imaging (MRI) showed acute ischemia involving the right MCA (middle cerebral artery) with M1 cutoff and no distal flow (Figure 1)

Patient underwent mechanical thrombectomy and was admitted to Intensive Care Unit (ICU) for neuromonitoring. An Improvement in motor power was noted on the left side with power of -3/5 with GCS of E4V5M6. 48 hrs. post thrombectomy GCS of the patient was noted to be E1V5M6 despite promptly following verbal commands. With low GCS, emergent computer tomography (CT) of brain was performed which showed subtle hyperintensities in the right posterior peninsular white matter suggestive of minimal hemorrhages within the infarct with mild mass effect on the right lateral ventricle with no midline shift. (Figure 2)

**Figure 1:** MRI showing Right MCA cutoff (Arrow pointing).

**Figure 2:** CT brain- showing wedge shaped hypodensity involving right frontal, temporal and posterior front- parietal regions-likely infarct with mild mass effect on right lateral ventricle.

**Figure 3:** Bilateral ptosis noted in the patient. Note the palpebral fissure length is 0mm.

Conservative strategies such as anti-edema measures along with supportive care were continued. The patient continued to have GCS of E1V5M6. Repeat CT brain done on day 5 showed similar findings of the previous. Diagnosis of cerebral ptosis was done according to Manconi's criteria.

GCS as a prognostic indicator of neurological deterioration was not considered in this case. Conservative management was continued and by day 15, improvement in ptosis was noted.

## Discussion

The Glasgow Coma Scale (GCS) has been used as a standard tool to describe the extent of impaired consciousness. The scale assesses patients based on three aspects: eye-opening, motor, and verbal responses [3]. Assessing each of these separately provides a clear picture of a patient's neurological state.

Assessment of responsiveness with the Glasgow Coma Scale is widely used to guide early management of patients with any kind of acute brain injury [3]. Decisions to be taken in more severely impaired patients include emergent management such as securing the airway and triaging to determine the need of immediate patient transfer to initiate the appropriate neurological intervention. Decisions in less severely impaired patients include the need for neuroimaging, admission for observation or discharge [3]. Serial Glasgow Coma Scale assessments are done in monitoring the clinical course of a patient and to decide on their management. In a comparative study by Gennarelli et al, it was demonstrated that the existence of a continuous, progressive association between increasing mortality after a head injury and decreases in GCS Score from 15 to 3 [4]. Even though, GCS is one of the most powerful clinical tools to prognosticate, neither the GCS score nor any single tool alone should be used to predict a patient's outcome. This is because the prognostic implications of the score are influenced by factors such as diagnosis,need of mechanical ventilation, age of the patient and clinical indices such as pupillary dysfunction and conditions such as cerebral ptosis.

Ptosis or blepharoptosis is defined as an abnormally low-positioned eyelid [2]. Ptosis can be congenital or acquired with neurogenic, myogenic, mechanical and traumatic causes [2]. Cerebral ptosis which is a rarer cause of acquired ptosis, can be partial or complete, unilateral or bilateral without brainstem involvement [5]. Diagnosis of cerebral ptosis is made by inclusion criteria of Manconi as described in Table 1 above.There have been many theories regarding the mechanism of insult leading to cerebral ptosis.The study done by Manconi et al postulated that supranuclear disruption of ocular and motor nerve pathways is the reason for oculomotor dysfunction and cerebral ptosis [6]. In unilateral hemispheric involvement, eyelid dysfunction can be seen with insult of frontal and parietal lobes as cerebral ptosis. These eyelid abnormalities are more commonly described with right hemispheric involvement as described by Vegda et al [2]. Another study done by Johnston et al, reported resolution of cerebral ptosis secondary to resolution of parietal dysfunction which was seen in the right hemispheric infarct [7]. The right cerebral cortex is postulated to be responsible for the tonicity of levator palpebrae superioris activity [2]. In a study done by Averbuch-Heller et al it was reported that bilateral ptosis was the first sign of imminent herniation.

However, the role of bilateral cerebral ptosis to be used as a clinical tool to predict impending deterioration needs further research and more reporting.

In our patient with right hemispheric involvement but with no signs of herniation, we asked a few questions to derive the information about his neurological status such as he was asked to tell his name, address, day/date and other personal details. He was also asked to follow simple commands such as protruding his tongue, holding the examiner's fingers and lifting his left arm and leg (nonparetic side).

**Table 1: Manconi's Criteria**

| Observations | Patient |
|---|---|
| Age | 69 |
| Gender | Male |
| MRI brain | Right MCA infarct with M1 occlusion |
| Brainstem | Not involved |
| Verbal and motor response | present |
| pupils | Reactive to light, bilateral equal |
| Length of palpebral fissure | 0mm |
| Amplitude of levator excursion | 0mm |
| Frontalis activity | present |
| Marginal reflex distance | 0mm |
| Blepharospasm | no |
| Apraxia on eye opening | no |
| Facial palsy | no |
| Ptosis onset posts 48 hrs. of symptom onset | yes |



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Other parameters such as vertical length of palpebral fissure, amplitude of levator excursion, blepharospasm, apraxia of eyelid opening, gaze preference, horizontal or vertical gaze palsy, pupillary size, symmetry and pupillary light reaction, and presence of facial palsy were examined and noted as done in the study by Vegda et al [2].

Thus, we concluded that our patient had cerebral ptosis according to the Manconi criteria.

Repeat CT brain did not show any increase in midline shift or cerebral edema. Conservative management was continued and patients' clinical status improved on day 15.

In our patient there was no correlation noted between cerebral ptosis and worsening neurological status and hence cerebral ptosis should not be confused with reduced consciousness of the patient.

## Conclusion

Cerebral ptosis which can occur unilateral or bilaterally, is a rarely described sign, physiological basis of which still remains unknown. Paying attention to this often-ignored neurological finding secondary to fewer cases reported, may probably be useful in prognosticating the outcome. Assessment of the patient and accurately identifying the confounding factors in various neurological scores such as Glasgow Coma Score is very vital and needs more research.

### Clinical significance

Clinical significance of this case report includes many learning points such as, firstly, whenever there is a drop in GCS score of a patient with hemispheric stroke, the possibility of cerebral ptosis needs to be considered. Second, using alternative neurological assessment tools in clinical suspicion of cerebral ptosis needs to be explored. Finally, appropriate education of the bedside nurse regarding the cerebral ptosis phenomenon is essential and the need to assess other responses in GCS, like in our patient, is important to identify worsening neurological status.

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