

Sustained downgaze as the only remained sign after regaining consciousness in hepatic encephalopathy

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Abstract

Sustained downgaze mostly occurs in association with lesions affecting the dorsal midbrain. We report sustained downgaze in a patient with hepatic encephalopathy. The sustained downgaze existed for seven more days after she regained her consciousness. The persistent downgaze even after regaining full consciousness indicates localized pretectal dysfunction rather than diffuse encephalopathy as the mechanism of sustained downgaze in our patient. The ocular motor dysfunction in hepatic encephalopathy may be due to localized dysfunction of the brainstem.

INTRODUCTION

Sustained downgaze mostly occurs in lesions affecting the dorsal midbrain. However, this eye sign has also been reported in subarachnoid hemorrhage, seizure, hepatic failure, hypoglycemia, and hypoxic encephalopathy, even without structural damage to the pretectal area.^{1,2} We report sustained downgaze as the only persistent finding in a patient with hepatic encephalopathy after regaining the consciousness.

CASE REPORT

A 64-year old woman presented to the emergency room with altered consciousness for 15 hours. She had suffered from chronic B-viral hepatitis without proper management. She was not on any medication. She had a recent history of melena. On neurologic examination, she was stuporous, but pupils were equal at 2 mm and reactive. She also showed conjugate downward deviation of the eyes with intact vestibulo-ocular reflexes in both horizontal and vertical directions. She recovered her consciousness the next day and stayed alert, but could not voluntarily open her eyes. When her eyelids were raised manually, sustained downgaze was observed (Figure 1A). She had no tremor or myoclonus. Laboratory findings showed anemia (hemoglobin, 10.1 g/dl, reference: 10.7-14.6), thrombocytopenia of 87,000/uL (reference: 143,000-376,000), slightly prolonged prothrombin time (12.9 sec, reference: 9.8-12.2), and normal liver enzymes.

Ammonia level was within normal range (21 uMol/L, reference: 0-54). Cerebrospinal fluid study was also normal. Her T1-weighted MRI revealed high signal intensities in bilateral globus pallidus, which had not been found on her previous MRI three years before (Figure 1B). Abdomen CT additionally disclosed cirrhotic liver with esophageal varices. The sustained downgaze existed for seven more days after she regained her consciousness. She received oral lactulose therapy (10 ml three times a day) with a diagnosis of hepatic encephalopathy due to a recent gastrointestinal bleeding. On the eighth day, she was able to open her eyes and her sustained downgaze disappeared.

DISCUSSION

To our knowledge, this is the first case with sustained downgaze as the only persistent sign after regaining consciousness from hepatic encephalopathy. Altered mental status and sustained downgaze in this patient may have been ascribed to hepatic encephalopathy based on the liver cirrhosis on her abdomen CT and clearing of sustained downgaze with intake of lactulose. The hepatic encephalopathy had probably been precipitated by gastrointestinal bleeding.

Patients with hepatic encephalopathy may show nystagmus on lateral gaze, sustained downgaze or downward and lateral ocular deviation, vertical skew deviation, ocular bobbing³, or dysconjugate gaze.⁴ In most patients with hepatic

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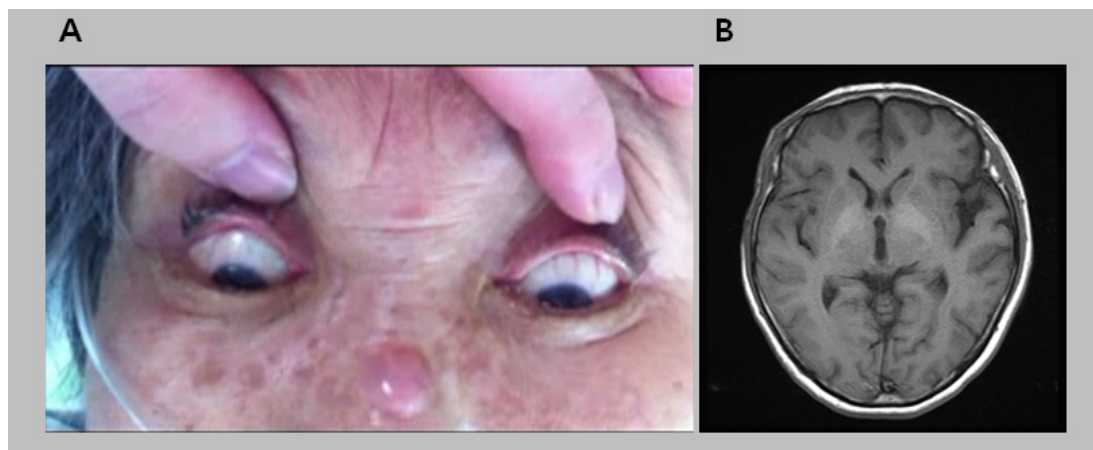


Figure 1. (A) Symmetric downward eye deviation on manual opening of the eyes. (B) Brain MRI of the patient. T1-weighted MRI shows high signal intensities in bilateral globus pallidus.

encephalopathy, the eye signs are accompanied by coma and disappear with resolution of other neurological signs.^{3,5,6} In view that both the eye signs and diffuse cerebral dysfunction develop rather simultaneously, the eye signs in hepatic encephalopathy can be explained by bilateral depression of cerebral gaze pathways. However, previous reports have shown that the eye signs in hepatic encephalopathy may be from brainstem dysfunction rather than due to bilateral depression of the cerebral gaze pathways. One study showed that 4 of 51 patients with hepatic coma had no reflexive horizontal eye motions.⁷ Another report described resolution of ocular bobbing after treatment of encephalopathy in a patient with hepatic coma.³ These reports indicate that hepatic coma may affect the brainstem tegmentum.

Our patient provides further evidence that the eye signs in hepatic coma may be due to localized brainstem dysfunction rather than diffuse encephalopathy. The sustained downgaze existed for seven more days after she regained her consciousness, and it was the only persistent sign. Similarly, a previous report described a patient with ocular divergence that lasted three more days after the patient became alert.⁴ Hepatic encephalopathy may, on rare occasions, be associated with ocular motor dysfunction, which is also indicative of brainstem dysfunction. Preservation of other functions, such as pupillary reflexes, posture, and respiration, subserved by the brainstem at the similar level also points to metabolic derangements as the mechanism of the ocular motor dysfunction in such patients.⁴

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