THE AMERICAN JOURNAL of MEDICINE®

# The Eyes Are Useless When the Mind Is Blind: A Rare Case of Anton-Babinski Syndrome in Hepatic Encephalopathy

*To the Editor:*

Anton-Babinski syndrome is a condition in which patients deny vision loss despite objective evidence. They further confabulate to support their stance. Cortical blindness is the loss of vision caused by a unilateral or bilateral lesion in the occipital cortex in the presence of normal anterior visual pathway. Anton syndrome is most commonly due to bilateral infarction of posterior cerebral artery, but it can arise from any condition resulting in cortical blindness. It is not known why only some patients with cortical blindness end up having Anton-Babinski syndrome.

No case has been reported about Anton-Babinski syndrome in association with cortical blindness and hepatic encephalopathy. This is likely due to only 12 worldwide cases that have linked hepatic encephalopathy to cortical blindness. All 12 cases have been reported in Europe and Asia.[1-3]

## CASE DESCRIPTION

A 44-year-old Hispanic woman, with a history of cryptogenic liver cirrhosis diagnosed in 2012, presented with her relatives after they realized she was unable to see. The patient's family stated that she had been running into objects over the past 2 weeks, worsening over the past 2 days. She also exhibited intermittent disorientation and confusion over the same time period. Upon questioning, the patient denied acknowledgment of any symptoms and attempted to demonstrate that she was able to see.

Upon initial neurological evaluation, she was cooperative, awake, and oriented only to person. She was unable to track her family members as they moved across the room while conversing with them. Physical examination was significant for asterixis and lower-extremity edema bilaterally. No signs of meningismus or trauma were found. Abdominal examination did not reveal fluid wave, spider angiomata, or other stigmata of the liver cirrhosis.

The ophthalmologic examination showed profound decreased visual acuity bilaterally; however, the patient was able to identify sources of light and object movements in the visual field. Loss of threat reflex was exhibited; however, pupillary reflexes were preserved, as well as extraocular movements. A dilated funduscopic examination and tonometry were unremarkable, and no optokinetic nystagmus was elicited.

The laboratory data were consistent with her history of cirrhosis, and an ammonia level was elevated. Computed tomography of the head without contrast failed to show any lesion able to explain the symptoms and findings.

Based on the ophthalmologic examination and these results, emergent anatomic pathologies of vision loss were ruled out, as well as psychogenic visual impairment. Thus, the diagnosis of cortical blindness was made.

Treatment entailed a combination of lactulose and rifaximin, and within 24-36 hours the patient had significant improvement in her visual and mental capacity. She was discharged home after 72 hours hospitalization with no neurologic symptoms.

## DISCUSSION

Although hepatic encephalopathy is common in decompensated liver cirrhosis, vision loss rarely occurs as an accompanying symptom. Early recognition of acute vision loss in hepatic encephalopathy could help avoid unnecessary expensive ophthalmologic and neurologic work-up.

Few cases have reported transient cortical blindness in hepatic encephalopathy.[1-3] However, the case above details the unaware vision loss by the patient, which has not been expressed in previous studies.

To date, this is the first case of Anton-Babinski syndrome in association with hepatic encephalopathy. Furthermore, this is the first case in the US detailing cortical blindness as a manifestation of hepatic encephalopathy.

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**Funding:** None.
**Conflict of Interest:** None.
**Authorship:** All coauthors have contributed substantially enough to deserve authorship in this manuscript, participating actively, intellectually, as well as in the critical review and analysis of the data while crafting/elaborating the manuscript.

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0002-9343/$ -see front matter Published by Elsevier Inc.

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http://dx.doi.org/10.1016/j.amjmed.2016.12.024

## ACKNOWLEDGMENT

We are indebted and grateful to Salvador Cruz-Flores (Chief, Department of Neurology, Texas Tech University Health Sciences Center, El Paso) for his helpful advice, guidance, and constructing suggestions that improved this clinical communication.

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