

Inverse Ocular Bobbing in a Patient With Encephalitis Associated With Antibodies to the N-methyl-D-aspartate Receptor

A 30-YEAR-OLD WOMAN presented with headache, fever, disorientation, and recent memory disturbance. Brain magnetic resonance imaging showed fluid-attenuated inversion recovery hyperintense abnormalities in both hippocampi, without abnormal findings in other areas of the brain or brainstem.¹ The patient subsequently developed tonic convulsions, restlessness, anxiety, and hypoventilation that led to the use of sedation and mechanical ventilation. While in the intensive care unit, inverse ocular bobbing and skew deviation were transiently observed (**Figure**) (a video is available at <http://www.archneuro.com>). Antibodies to NR1/NR2 heteromers of the N-methyl-D-aspartate receptor were identified in her serum and cerebrospinal fluid. After immunotherapy and removal of an ovarian teratoma, all symptoms started to improve and the patient was able to return to her job 1 year later.

COMMENT

Inverse ocular bobbing, or ocular dipping, consists of a slow, spontaneous downward eye movement with fast return to midposition. In



Video available online at www.archneuro.com

contrast to ocular bobbing, which is most often attributed to large, destructive pontine lesions, ocular dipping may be observed in anoxic coma or following prolonged status epilepticus and is thought to be a marker of diffuse, rather than focal, brain damage.² The encephalitis associated with antibodies to the N-methyl-D-aspartate receptor of-



Figure. The position of the patient's eyes showed skew deviation when the inverse ocular bobbing resolved.

ten associates with central hypoventilation and, much less frequently, with vertical gaze paresis or cranial nerve dysfunction, all suggesting involvement of the brainstem.³ In the current case with head magnetic resonance images that did not show any signal abnormality in the brainstem, the association of ocular dipping with hypoventilation and skew deviation suggests that the diffuse encephalopathy in addition to involvement of the brainstem ocular pathways were responsible for the abnormal ocular movement.

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Additional Information: Video is available at <http://www.archneuro.com>.

REFERENCES

1. Shimazaki H, Ando Y, Nakano I, Dalmau J. Reversible limbic encephalitis with antibodies against the membranes of neurones of the hippocampus. *J Neurol Neurosurg Psychiatry*. 2007;78(3):324-325.
2. Leigh RJ, Zee DS. *The Neurology of Eye Movements*. 4th ed. New York, NY: Oxford University Press; 2006:674-675.
3. Dalmau J, Tuzun E, Wu HY, et al. Paraneoplastic anti-N-methyl-D-aspartate receptor encephalitis associated with ovarian teratoma. *Ann Neurol*. 2007; 61(1):25-36.