# Oxcarbazepine induced jerky see-saw nystagmus

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**Authors' contributions**
Study concept and design: Nanković and Habek. Acquisition of data: Adamec, Nanković, Zadro, Hajnšek, Habek. Analysis and interpretation of data: Adamec, Nanković, Zadro, Hajnšek, Habek. Drafting of the manuscript: Adamec. Critical revision of the manuscript for important intellectual content: Adamec, Nanković, Zadro, Hajnšek, Habek. Administrative, technical, and material support: Adamec, Nanković, Zadro, Hajnšek, Habek.

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Sir,

A 43-year-old female presented to the emergency department because of dizziness, drowsiness and generalized urticaria that occurred the day prior. Patient's history revealed protracted delivery with neonate asphyxia, following which she had been diagnosed with epilepsy at age seven when she experienced her first generalized tonic-clonic seizure. Her MRI showed ulegyria and hypoxic-ischaemic changes characteristic for perinatal damage. Her last visit to an epilepsy clinic was eighteen days before. At that time a change in her therapy was made with gradual initiation of oxcarbazepine (start dose of 300 mg QD in the evening for 5 days, than 300 mg BID for 5 days, than 300 mg in the morning and 600 mg in the evening for 5 days) and tapering of levetiracetame. Her therapy, at the time of her presentation to the emergency department, was (in total daily doses): oxcarbazepine 900 mg, sodium valproate 1000 mg, levetiracetame 750 mg, clonazepam 4.5 mg, lamotrigine 100 mg. Neurologic examination revealed a jerky see-saw nystagmus (SSN) more pronounced on the right eye (Video 1). Brain CT scan was normal. Oxcarbazepine and lamotrigine were discontinued and carbamazepine was gradually introduced. The carbamazepine was chosen because this drug controlled her seizures very well in the past, however pediatric neurologist chose to switch from carbamazepine to levetiracetame at one point. On follow-up, a week later, she didn't have nystagmus and didn't experience dizziness and drowsiness any more but still had residual urticaria on her face and neck.

SSN is a torsional-vertical nystagmus in which one eye elevates and intorts, while the other depresses and extorts. Jerky SSN is reportedly caused by lesions in the chiasmal or parasellar area thus resulting from disruption of the visual pathway (1). However, SSN was described in association with vascular and inflammatory lesions of the rostral midbrain, pons and cerebellum (1, 2). Based on these reports, SSN can result from dysfunction of the medial longitudinal fasciculus (MLF) that coordinates input to the oculomotor and trochlear nuclei from the vestibular nuclei (2).

One of the most common side-effects of oxcarbazepine therapy is nystagmus and it is related to its mono-hydroxy-derivative levels in serum (3). The therapeutic effect of oxcarbazepine results from its effect of blocking neural ion channels. We hypothesize that that same effect caused SSN in our patient blocking neurons that are part of the MLF. As nystagmus is a well described side-effect of antiepileptic therapy and given the fact that nystagmus in our patient commenced on initiation and disappeared on discontinuation of oxcarbazepine, we report on a yet undescribed case of jerky SSN caused by oxcarbazepine.

References:


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Video 1. Jerky see-saw nystagmus (SSN) more pronounced on the right eye.