Thiamine refractory Wernickes encephalopathy reversed with magnesium therapy

John Joseph Coughlan, Thomas Mross, Meghan Wafer, Richard Liston

Department of Medicine,
University Hospital Kerry,
Tralee, Ireland

Correspondence to
Dr John Joseph Coughlan,
jjcoughl@gmail.com

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DESCRIPTION

A 34-year-old man was presented to our emergency department in alcohol withdrawal.

Despite initial treatment with high-dose intravenous thiamine therapy he went on to develop nystagmus (video 1), ataxia, pass pointing, intention tremor and worsening confusion. He was diagnosed with Wernickes encephalopathy, an acute neuropsychiatric syndrome resulting from thiamine deficiency.¹

His serum magnesium levels were found to be low at 0.41 mmol/L (normal range 0.66–1.02 mmol/L). He was started on high-dose intravenous magnesium in addition to thiamine replacement and his neurological symptoms resolved once his serum magnesium levels had normalised (video 2).

He was discharged home and on review in clinic 4 weeks later, he had made a complete recovery.

Magnesium is an essential cofactor of an enzyme in the pentose phosphate pathway, transketolase,

whose activity is decreased in thiamine deficiency. Hypomagnesaemia may result in thiamine refractoriness in patients with Wernickes encephalopathy.²

Some studies have suggested that thiamine deficiency leads to Wernicke-Korsakoff syndrome only in patients whose transketolase has a reduced affinity for thiamine.³

Serum magnesium levels should always be checked in patients presenting with a history of excess alcohol who are at risk for developing Wernickes encephalopathy.

Learning points

- ▶ Patients with a history of excess alcohol often present with electrolyte abnormalities including hypomagnesaemia.
- ▶ Magnesium is an essential cofactor of transketolase, an enzyme whose activity is decreased in the context of thiamine deficiency.
- ▶ Magnesium levels should always be checked and supplemented if necessary in patients at risk of developing Wernickes encephalopathy.

Video 1 Our patient demonstrates bilateral vertically upbeat nystagmus.

Video 2 Our patient's nystagmus has improved, although a few beats remain at extreme lateral gaze.

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Contributors JJC was primarily responsible for drafting the clinical case report. TM and MW assisted with preparation of clinical videos and collection of patient information. RL provided clinical guidance and gave editorial feedback. All authors contributed to preparation of this clinical case report.

Competing interests None declared.

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