Clinical Outcome of Aggressive Thiamine Treatment in an Underdiagnosed Neuropsychiatric Disorder

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A 73-year-old man with a medical history of hypertension, gout, and atrial fibrillation with a 40-year history of alcohol abuse (0.5 pints of vodka per day) presented to the emergency department after a fall down two steps. On trauma assessment, the patient was found to have two fractured ribs as well as a small pneumothorax, and was therefore admitted for observation. On hospital day 3, the patient became increasingly agitated and delirious, with a steady decline in mental status. Concern for alcohol withdrawal prompted administration of oral thiamine (100 mg) and an increase in the dosage of chlordiazepoxide from 50 mg to 100 mg.

Over the following 2 weeks, the patient became increasingly disoriented with worsening cognitive impairment and ataxia. Two-point restraints and mittens were placed on the patient after several attempts by him to pull out his nasogastric tube and the intravenous (IV) line from his arm. On hospital day 16, psychiatry was consulted for the management of delirium characterized by gradual cognitive deterioration and worsening behavioral disturbances. On initial psychiatric evaluation, the patient appeared confused, as he was alert only to person but not to place or time. The patient demonstrated disorganized thought process, speech, and behavior, in addition to visual hallucinations. Throughout the interview, the patient showed evidence of cognitive impairment as well as confabulation. Ophthalmologic abnormalities, such as nystagmus and bilateral lateral rectus palsy, were not discovered upon examination.

To further assess cognitive ability, a Montreal Cognitive Assessment (MOCA) was performed at bedside during the initial psychiatric evaluation, and the patient scored an 8 of 30 (a score of ≤25 reveals cognitive impairment)[1] (**Table 1**).

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Disclosure: The authors have no relevant financial relationships to disclose.

doi: 10.3928/00485713-20150626-02

TABLE 1.

Montreal Cognitive Assessment Score Prior to and During High-Dose[a] Intravenous Thiamine Treatment[b]

Day	MOCA Score (out of 30 points)
0	8
1	10
2	15
3	19

Abbreviation: MOCA, Montreal Cognitive Assessment Score.
[a]500 mg.
[b]Assessment was performed at the bedside and was conducted and scored in the same fashion each day.

DIAGNOSIS

Wernicke-Korsakoff Syndrome

The patient's clinical presentation and extensive history of alcohol abuse was highly suggestive of Wernicke-Korsakoff syndrome (WKS). Due to the gradual yet significant decline in the patient's cognitive, behavioral, and functional ability, as well as the morbidity and mortality associated with WKS, aggressive treatment with IV thiamine (500 mg 3 times daily for 3 days) was immediately started after initial psychiatric evaluation.

To evaluate the effect of aggressive treatment with thiamine on the patient's cognitive functioning, a MOCA was performed consecutively for 3 days (the duration of the IV thiamine treatment). On day 1 the patient's MOCA score was 10 of 30; on day 2 it was 15 was of 30; and on day 3 it was 19 of 30 (**Table 1**). In addition to moderate improvement in cognitive ability, the patient appeared more alert and oriented to his surroundings and demonstrated significantly less disorganized thought process, behavior, and speech. Physical and occupational therapy led to improvement in his ataxia. The patient, however, continued to exhibit evidence of confabulation, although considerably less than prior to the aggressive thiamine treatment. His cognitive functioning by the third day of aggressive IV thiamine treatment had significantly improved, gradually returning to his baseline cognitive functioning.

DISCUSSION

This case shows the importance of aggressive thiamine treatment in WKS. The patient's initial clinical presentation and progressive cognitive and neurologic decline, which were initially misinterpreted solely as delirium, were actually early signs consistent with WKS.

Based on subjective and objective analysis using the MOCA, the patient demonstrated improvement in ataxia and cognitive functioning over a 3-day period of aggressive thiamine treatment. Even though the patient's MOCA score continued to indicate cognitive impairment (≤25), improvement in cognitive ability over a 3-day period demonstrates the beneficial effects of thiamine treatment.

The dosage, frequency, and duration of treatment is based on studies conducted on patients with WKS, as well as those who are at risk for developing this syndrome.[2,3] The initial dose of 100 mg of oral thiamine given on hospital day 3 was insufficient in preventing further cognitive deterioration and reducing the morbidity and mortality associated with WKS. In fact, those who show clinical signs and symptoms consistent with WKS should be treated with 500 mg of thiamine.

Due to the fact that the IV thiamine was discontinued after 3 days of treatment, it is unclear if there would have been a complete reversal of WKS if treatment had been continued for a longer duration. Prior to administration of a high dose of thiamine (500 mg), the patient was being treated with a low dose of thiamine (100 mg for 13 days), and thus it is unclear if the patient would have shown more significant improvement in cognitive functioning if aggressive high-dose thiamine had been administered earlier in hospitalization. Future research should be conducted to further examine the optimal duration of thiamine treatment, as well as the clinical effects of delaying thiamine treatment.

In summary, thiamine deficiency, often seen in chronic alcoholism, may result in WKS. Even though there is a significant morbidity and mortality associated with WKS, this syndrome is frequently underdiagnosed and clinically undertreated. Thus, aggressive treatment with high-dose IV thiamine (500 mg) is clinically important in improving the clinical outcome in patients with WKS.

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