

# Take steps to relieve ataxia in patients with alcohol use disorder

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**A**taxia is a well-known complication of chronic alcohol abuse, which is attributed to degeneration of the cerebellar vermis. However, effective treatment approaches, as well as the timing and level of recovery, remain unclear. One cross-sectional study found that long-term abstainers from alcohol had less severe ataxia than short-term abstainers,<sup>1</sup> suggesting that improvement is possible with continued sobriety. However, a recent longitudinal study contradicts this finding, reporting no improvement in ataxia in patients abstinent for 10 weeks to 1 year.<sup>2</sup>

**CASE REPORT**

**Unable to walk, heavy alcohol use**

Mr. G, a 59-year-old white male with a history of daily, heavy alcohol use, presents to the emergency room reporting that he has “not been able to walk right” for 3 weeks. He is in a wheelchair because of ataxia and difficulty balancing. He denies headaches, visual changes, weakness, numbness, and difficulty speaking or swallowing.

Mr. G reports drinking one 40-oz bottle of malt liquor and 2 pints of vodka per day for more than 40 years. His alcohol abuse led to homelessness, unemployment, and divorce. Despite heavy drinking, he denies signs of withdrawal, including shaking, sweating, seizures, and delirium.

Mr. G has no other medical conditions. He denies a family history of neurologic disorders or substance abuse.

His pulse is 100 beats per minute, respirations of 16 breaths per minute, temperature of 37°C, and blood pressure of 143/89 mm Hg. Physical examination reveals a wide-based gait.

Mr. G is admitted to the inpatient psychiatric unit to monitor and treat his alcohol withdrawal and to undergo further workup of the gait disturbance.

A head CT scan shows non-specific changes; an EEG also is within normal limits. Complete blood count, basic metabolic panel, liver function test, HIV test, acute hepatitis panel, thyroid function test, erythrocyte sedimentation rate, and vitamin B<sub>12</sub> tests are within normal ranges.

A full neurologic exam reveals a wide-based gait, impaired heel-shin test, and dysmetria on finger-nose-finger test. Mr. G is given a diagnosis of ataxia due to alcoholic cerebellar degeneration. Thiamine repletion is suggested.

**Treatment and outcome**

Mr. G continues on thiamine, 100 mg, twice daily, and oxazepam, 15 mg, as needed, to manage withdrawal symptoms. He receives gait training 3 times per week.

Approximately 10 days after admission, Mr. G is able to ambulate with a walker. Three weeks after admission, his gait has improved and he walks with a cane. (See the video at

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The authors report no financial relationships with any company whose products are mentioned in this article or manufacturers of competing products.

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One study showed that long-term abstainers from alcohol had less severe ataxia than short-term abstainers

CurrentPsychiatry.com for an illustration of this progressive recovery.)

After discharge, Mr. G is referred to an addiction psychiatrist and addiction psychotherapist for ongoing treatment of alcohol use disorder.

### Making the diagnosis

In a patient complaining of balance difficulties, consider ataxia secondary to cerebellar degeneration.

- **Take a complete history.** Ask about the onset and progression of ataxia.
- **Obtain a family history.** Some types of ataxia are genetic.
- **Perform a neurologic examination,** which may reveal signs of cerebellar deficits, particularly characteristic wide-based gait. These patients will have difficulty when walking in tandem. Other impairments on the neurologic exam that may raise suspicion for a cerebellar disorder include: impaired heel-shin test, impaired finger-nose-finger test (dysmetria), impaired rapid alternating movements (dysdiadochokinesia), nystagmus, impaired smooth pursuits, intention tremor, or speech abnormalities.
- **Perform head imaging, such as a CT scan or MRI.** In patients with ataxia secondary to alcohol abuse, imaging might reveal degeneration of the cerebellar vermis.
- **Perform laboratory tests,** such as inflammatory markers, vitamin levels, and thyroid function testing to detect possible toxic-metabolic or inflammatory causes.

Alcohol-induced ataxia can be diagnosed in patients with a history of heavy drink-

ing if the workup does not reveal another possible cause for the gait disturbance. Other less common deficits associated with alcohol-induced cerebellar injury include:

- dysarthria
- abnormal rate and force of movement
- limb ataxia.<sup>3</sup>

### Recommendations

- Be able to recognize the characteristic gait of patients with alcohol-induced ataxia.
- Provide thiamine supplementation.
- Refer patients to physical therapy.
- Educate your patients that their gait will not improve and may worsen if they continue to drink.
- Refer patients for ongoing treatment for alcohol use disorder, including medication management and psychotherapy.

Our experience suggests that patients with alcohol use disorder with cerebellar ataxia could have a good prognosis for ambulation. Improvement could occur over several weeks; it is unclear whether further gains can be expected with months or years of abstinence.

### References

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