

Expert Opinion

Alcohol Hangover Headache

Case History submitted by Randolph W. Evans, MD

Expert opinion submitted by Christina Sun, MD; Christine Lay, MD

Key words: alcohol hangover headache, migraine

(*Headache* 2007;47:277-279)

In his 1954 first novel, “Lucky Jim,” Sir Kingsley Amis describes the delayed effects of drinking port on the titular history lecturer upon awakening in the morning. “Dixon was alive again. Consciousness was upon him before he could get out of the way; not for him the slow, gracious wandering from the halls of sleep, but a summary, forcible ejection. . . . The light did him harm, but not as much as looking at things did; he resolved, having done it once, never to move his eyeballs again. A dusty thudding in his head made the scene before him beat like a pulse. . . . he sat up a little, and what met his bursting eyes roused to a frenzy the timpanist in his head.”

CASE

A few hours after drinking 3 glasses of any type of wine, a 35-year-old woman develops a moderately severe throbbing headache with light and noise sensitivity, nausea, diarrhea, thirst, fatigue, and difficulty with concentration. She may not feel completely well for 24 hours. However, if she has 2 or 3 gin and tonics,

From 1200 Binz #1370, Houston, TX 77004 (R.W. Evans); The Headache Institute, Roosevelt Hospital, New York, NY (Drs. Sun and Lay).

Address all correspondence to Dr. Randolph W. Evans, MD, 1200 Binz #1370, Houston, TX 77004, or Christina Sun, MD, and Christine Lay, MD, The Headache Institute, Roosevelt Hospital, 1000 Tenth Avenue, Suite 1c-10, New York, NY 10019.

she has no ill effects. She is healthy with no history of significant headaches.

QUESTIONS

What is the prevalence and cause of alcohol hangover headache (AHH)? What are the latency, features, and duration of the headache? Is the risk of development of AHH related to the type or amount of alcohol consumed? How can you distinguish between AHH and migraine triggered by alcohol? Are there any effective interventions or treatments for AHH?

EXPERT COMMENTARY

Alcohol hangover, or “veisalgia,” is a well-known and common phenomenon that generally occurs after heavy consumption of alcohol. The term “veisalgia” comes from the Norwegian *kveis*, which refers to the uneasiness following debauchery, and *algia*, the Greek term for pain. The alcohol hangover comprises a constellation of physical, cognitive, and psychological disturbances. Prominent physical symptoms include headache, anorexia, diarrhea, tremulousness, dizziness, fatigue, and nausea.¹ Heightened sympathetic nervous system activity manifested by tachycardia and sweating may also occur.² Cognitive and mood symptoms include decreased attention and concentration, decreased visuospatial skills and dexterity, as well as depression, anxiety, and irritability.

In a large epidemiological survey of headache in Danish 25- to 64-year-olds, the lifetime prevalence of hangover headache was 72%, making it the most common type of headache reported.³ The hangover headache is generally described as throbbing in nature. Usually, it occurs on the morning after alcohol consumption, when the blood alcohol concentration (BAC) is falling. Symptoms peak at about the time the BAC is zero and may continue for up to 24 h afterwards.² Though symptoms generally correlate with the amount of alcohol consumed, hangover is not entirely dose related.⁴ Paradoxically, hangover is much more common in light-to-moderate drinkers than in regular heavy drinkers.⁵⁻⁷

Though the mechanism of AHH is not fully understood, several physiological factors are likely to be contributory. Alcohol has a vasodilatory effect on intracranial vasculature, which results in throbbing headache pain. Endocrine and immune system disturbances, dehydration, and sleep disturbance also play a role in alcohol hangover.^{1,8} Alcohol has been shown to alter cytokine pathways by elevating levels of prostaglandin E2 and thromboxane B2,⁹ resulting in nausea, headache, and diarrhea. Dehydration also occurs as a result of the inhibitory effect of alcohol on antidiuretic hormone. The accumulation of acetaldehyde, the main metabolic product of ethanol, contributes to tachycardia, diaphoresis, skin flushing, nausea, and vomiting. Disrupted sleep, also a contributory factor to headache, can occur due to alcohol-induced changes in rapid eye movement (REM) cycles.

Darker colored alcoholic drinks contain congeners, which are the natural byproducts of alcohol fermentation. They impart flavor, color, and aroma to alcoholic beverages. There is a higher association with hangover headache in drinks with congeners compared with drinks without these substances. Whiskey, bourbon, and red wine have a higher concentration of congeners than other alcoholic beverages have, such as vodka and gin. In addition, congeners may be proinflammatory.¹⁰

It may be difficult to differentiate between AHH and migraine triggered by alcohol consumption. Both are throbbing in nature, and often associated with nausea, vomiting, photophobia, and phonophobia, and are usually made worse by movement. Those who have

a family history of headaches, headaches starting in childhood or adolescence, or multiple triggers in addition to alcohol are likely to have migraine. People with headaches occurring only in the setting of alcohol consumption probably do not have migraine, although with time, this diagnosis can change. Though the differentiation between the 2 types of headache may be difficult at times, the treatments are often similar.

Though multitudes of "hangover cures" including dietary supplements, prescription medications, and over-the-counter medications have been proposed, a systematic review of randomized controlled trials failed to show that any conventional or complementary intervention is effective for preventing or treating alcohol hangover, though encouraging findings exist for γ -linolenic acid from *B. officinalis* (a yeast based combination preparation) and tolfenamic acid.¹¹ The administration of tolfenamic acid, a prostaglandin inhibitor, at the time of alcohol ingestion had a small prophylactic effect in reducing hangover severity in a single small randomized controlled trial.¹² Tolfenamic acid is part of a group of anthranilic acids, which form a subclass of nonsteroidal antiinflammatory drugs (NSAIDs) with specific action as prostaglandin inhibitors.

Despite the lack of evidence supporting any interventions for hangover prevention, a number of measures are thought to lessen the effects of AHH. First, drink in moderation and sip beverages slowly. Eating greasy foods before alcohol consumption helps to slow or delay the absorption of alcohol. Also, eating honey and tomato juice, both rich in fructose, allows the body to metabolize alcohol more effectively. Drinking a lot of water or fluids that have minerals and electrolytes will also help to alleviate the dehydration associated with alcohol consumption. Caffeine may also provide some headache relief, as a result of its vasoconstrictive action. However, caffeine intake should be balanced by increased water consumption, as caffeinated beverages can lead to further dehydration. With the guidance of a physician, certain NSAIDs are also likely to be effective in lessening the effects of AHH, due to their action as prostaglandin inhibitors. Mefenamic acid and meclufenamate, which are anthranilic acids, may be the NSAIDs of choice, as tolfenamic acid is not available in the United States.

The woman described in the above case is most likely to be suffering from AHH. Her sensitivity to wine is probably due to the presence of congeners, especially in red wine. The absence of any other significant headache history makes the diagnosis of migraine much less likely.

Conflict of Interest: None

REFERENCES

1. Wiese JG, Shlipak MG, Browner WS. The alcohol hangover. *Ann Intern Med.* 2000;132:897-902.
2. Swift R, Davidson D. Alcohol hangover: Mechanisms and mediators. *Alcohol Health Res World.* 1998;22:54-60.
3. Rasmussen BK, Olesen J. Symptomatic and non-symptomatic headache in a general population. *Neurology.* 1992;42:1224-1231.
4. Ylikahri RH, Leino T, Huttunen MO, et al. Effects of fructose and glucose on ethanol-induced metabolic changes and on the intensity of alcohol intoxication and hangover. *Eur J Clin Invest.* 1976;6:93-102.
5. Harburg E, David D, Cummings KM, Gunn R. Negative affect, alcohol consumption and hangover symptoms among normal drinkers in a small community. *J Stud Alcohol.* 1981;42:998-1012.
6. Crofton J. Extent and costs of alcohol problems in employment: A review of British data. *Alcohol Alcohol.* 1987;22:321-325.
7. Gunn R. Hangovers and attitudes toward drinking. *Q J Studies Alcohol.* 1973;34:194-198.
8. Calder I. Hangovers. *BMJ.* 1997;314:2-3.
9. Kangasaho M, Hillbom M, Kaste M, Vapaatalo H. Effects of ethanol intoxication and hangover on plasma levels of thromboxane B2 and 6-ketoprostaglandin F1 alpha and on thromboxane B2 formation by platelets in man. *Thromb Haemost.* 1982;48:232-234.
10. Wiese J, McPherson S, Odden MC, Shlipak MG. Effect of opuntia ficus indica on symptoms of the alcohol hangover. *Arch Intern Med.* 2004;164:1334-1340.
11. Pittler MH, Verster JC, Ernst E. Interventions for preventing or treating alcohol hangover: Systematic review of randomised controlled trials. *BMJ.* 2005;331:1515-1518.
12. Kaivola S, Parantainen J, Osterman T, Timonen H. Hangover headache and prostaglandins prophylactic treatment with tolfenamic acid. *Cephalalgia.* 1983;3:31-36.