Insomnia: The Neglected Component of Alcohol Recovery

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How often do alcoholics give the excuse that they drink to get to sleep? And how often do newly sober alcoholics discover in their sleeplessness, that one more thing, for which they had depended on alcohol, was its soporific effect?

Insomnia is extremely common during alcohol recovery and is associated with an increased risk of relapse [1]. Insomnia also has a significant impact on a patient's cognition, mood, and ability to participate in alcohol treatment[2][3]. Though it is easy to assess, this syndrome is frequently neglected by clinicians who are underwhelmed by this complaint from their newly sober patients. Even when insomnia is recognized, no treatment is offered in a majority of cases out of a mistaken belief that all sleep medications are addictive [4].

We believe that increased recognition and management of insomnia could lead to better outcomes in treating alcohol dependence. We will attempt to summarize succinctly the current evidence for our stance and argue for more aggressive efforts to identify and treat insomnia in early recovery.

Alcohol is hypothesized to disrupt sleep through its effects on numerous pathways. Alcohol dependence causes significant changes to glutamatergic and GABAergic pathways extensively involved in the neurobiology of sleep [5]. Alcohol dependence yields reduced melatonin levels in the early part of the night [6]. Murine models have shown that gene-directed circadian variation influences alcohol consumption [7].

Alcohol has a clear impact on sleep architecture [8]. Objectively, alcohol-dependent subjects have been shown to have increases in sleep-onset latency (SOL) and percentage of slow-wave sleep (%SWS), as well as suppression of REM sleep [5]. In early recovery sleep efficiency (SE), %SWS, REM latency, and REM density are reduced and SOL was prolonged [8]. Subjective data is limited. One study reported rates of insomnia as high as 50 percent in alcohol withdrawal [9]. A longitudinal study measuring changes in the PSQI, a well validated self report sleep measure, over 12 weeks, revealed little improvement in the score[10].

Sleep problems in recovering alcoholics are not simply inconvenient or uncomfortable. They have been associated with relapse. On polysomnogram (PSG), persistent abnormalities in REM density and latency at 14 weeks are linked to relapse. Likewise other measures, including increased SOL, reduced %SWS, and reduced SE increase relapse chances [8]. On self-report measures, increased SOL predicts relapse, and subjective complaints of sleep fragmentation appear to be better predictors of relapse than PSG findings [5, 11].

Large numbers of alcoholics admit to using alcohol to help them fall asleep [10]. Insomnia in and of itself is associated with poor quality of life, cardiovascular morbidity, and depression. Thus is it reasonable to postulate both that alcoholics in early sobriety might turn to an old friend to get to sleep and that treating their sleep problems should result in reduced relapse risk. Only a few studies have considered this possibility, however.

A randomized controlled trial comparing zopiclone (an enantiomer of eszopiclone) to lorazepam found improvements in certain sleep parameters in subjects in the benzodiazepine (BZD) arm, while the non-benzodiazepine benzodiazepine-receptor agonist (BzRA) subjects reported better daytime function [11]. Complicating matters, however, is the fact that drugs typically used to treat insomnia – BZDs and BzRAs alike – have abuse potential. Clinicians are hesitant to prescribe them to patients with addiction histories, let alone those newly in sobriety.

Alternate medication strategies to benzodiazepine receptor agonists exist. A placebo-controlled four-week trial of trazodone (200 mg) in 16 patients showed improvements in CGI and HAM-D, as well as wake time after sleep onset (WASO) as measured by PSQI[12]. A large NIAAA-funded placebo-controlled trial of trazodone in alcoholics showed significant subjective improvements in sleep, as measured by PSQI scores, in the trazodone arm as compared to placebo. The sleep improvements accrued over three months, with PSQI scores reverting to placebo levels after trazodone discontinuation [13]. This study raised concerns about reduced abstinence in trazodone-treated patients, however. Patients on trazodone had both a lower percentage of abstinent days and a higher rate of return to heavy drinking after discontinuation of the drug.

Among other medications studied for sleep induction in alcoholics in early recovery is gabapentin. Cleared renally, gabapentin is generally considered safe in patients with alcohol-related liver damage. While its administration is associated with some reduction in rates of relapse, the literature regarding its effect on improving sleep in early recovery is equivocal [14-17]. One placebo-controlled trial actually showed worsening of sleep parameters in patients on gabapentin [18]. Small, open-label trials of quetiapine and magnesium have also shown promise [19, 20].

Avoiding medications altogether, psychological treatments such as CBT have demonstrated potential for treating insomnia in alcoholic patients, with reduced depression and anxiety as well as better sleep[21]. Open-label trials have reported benefit from bright-light therapy[22]. With their low side-effect profiles, these interventions offer safer treatment option for these patients.

Although the literature describing alcohol-induced sleep problems is substantial, research into managing these problems is sparse. Subjective insomnia complaints correlate poorly with PSG data. A lack of standardized sleep scales for research makes matters worse. Of the many different scales that have been used, some have unproven validity. Not all studies include the relapse measures, essential to any trial enrolling patients with a history of alcohol dependence. Future trials should remedy these shortcomings by using properly validated sleep scales and consistently including relapse measures.

The challenge of objective measurements and standards...
notwithstanding, patients themselves are well able to describe their subjective distress, poor sleep factoring prominently in it. Insomnia in early alcohol recovery thus offers an easily recognized symptom that we believe should be aggressively addressed. Moreover, evidence-based treatment options for insomnia in early alcohol recovery should be systematically evaluated to determine if they can reduce relapse risk.

Alcoholics in early recovery have enough to contend with. We clinicians should do what we can to help them get a good night’s sleep.

References