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Comorbidity of Generalized Anxiety Disorder and Alcohol Use Disorders among Individuals Seeking Outpatient Substance Abuse Treatment

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Abstract

The present study sought to address a gap in the literature by providing preliminary evidence of the prevalence and clinical characteristics of comorbid generalized anxiety disorder (GAD) and alcohol use disorders (AUD) in a front-line outpatient substance abuse clinic. Of 39 outpatients meeting criteria for an AUD, nearly half (46%) also met criteria for current GAD. The onset of GAD occurred prior to AUD in 67% of comorbid cases, with an average time lag of 12.5 years among individuals with primary GAD. Participants with comorbid GAD-AUD endorsed higher levels of worry severity and worry-reduction alcohol expectancies, and 55.6% of comorbid participants had a history of suicide attempts. Groups did not differ on anxiety sensitivity, social anxiety, or depression. Comorbid participants were more likely to indicate that worry interfered with their substance abuse treatment, and to indicate interest in concurrent treatment targeting their worry. Study findings provide initial evidence that GAD may be a prevalent and relevant factor among individuals with AUD seeking outpatient substance abuse treatment.

Keywords

Generalized anxiety disorder; Alcohol use disorders; Comorbidity; Worry; Outpatient substance abuse treatment

Although co-occurring anxiety and alcohol problems continues to generate interest among researchers (cf. Stewart & Conrod, 2008), the connection between generalized anxiety disorder (GAD) and alcohol use disorders (AUD) has received comparatively little direct study. Prior studies have shown that GAD is associated with considerable impairment (Kessler et al., 2001), chonicity (Ballenger et al., 2001), and lack of adequate treatment (Grant et al., 2005). Considering these factors, it is plausible that individuals with this disorder may turn to alcohol as a means to self-medicate. In a seminal early review of alcohol and anxiety disorder comorbidity by Kushner et al. (1990), it was shown that between 8.3% - 56.2% of inpatient alcoholics, with a median prevalence of 22.9%, met the criteria for GAD. However, investigators (e.g. Kushner et al., 2005; Schuckit & Hesselbrock, 1991; Stein, 2001) raised concerns about the source of these elevated rates of co-occurrence, suggesting that the apparent link between GAD and AUD was an artifact of sampling among individuals experiencing acute

alcohol withdrawal. This postulation was based on shared symptoms between GAD and withdrawal and resolution of GAD following inpatient alcohol treatment.

Nevertheless, some converging data indicate that the GAD-alcohol problem link may exist independent of the sampling confound created by investigating GAD in alcoholic inpatients. Burns and Teeson (2002) found that 12-month AUD was associated with a significant, elevated likelihood to have comorbid GAD (OR = 3.3, 95% confidence interval = 1.9 - 5.6). Also, two independent investigations with large, nationally representative samples (*N*s = 5,877 and 43,093, respectively) each suggested that individuals with GAD may use alcohol to self-medicate symptoms of the disorder (Bolton et al., 2006; Grant et al., 2005; Robinson et al., 2009). To date, however, there has been no publication of a focused examination of this comorbidity. The purpose of the present study is to fill a gap in the literature by providing preliminary data on the prevalence and nature of co-occurring GAD and AUD among individuals seeking outpatient substance abuse treatment.

Method

Participants

Assessment batteries were administered to 110 individuals involved in one of three separate four-week intensive outpatient substance abuse treatment programs (IOPs) as part of a parent study on the impact of anxiety on treatment participation. Compensation for the study included $10 for completing a self-report assessment battery and another $10 for participating in a psychiatric interview. Treatment clinicians were instructed to wait between 14 and 28 days before patients could be recruited for participation, to reduce any impact of acute substance withdrawal on self-report ratings and diagnostic presentation of anxiety (Driessen et al., 2001). Informed consent was obtained prior to study participation, and all study procedures were approved by the local Institutional Review Board (IRB). The only exclusion criterion in the naturalistic study was a lack of English language understanding. The present post-hoc study is based on selected assessments from the complete, IRB-approved parent study.

Structured psychiatric interviews were conducted at 2 of the 3 study sites, resulting in 56 participants with both self-report and diagnostic data from which the sample in the current study were drawn. Among the 56 individuals completing the self-report battery and diagnostic interview, 39 individuals were diagnosed with current AUD (*n*, alcohol abuse = 5; *n*, alcohol dependence = 34). The sample included in the study analyses consisted of all individuals with an AUD. Demographic details are presented in Table 1.

Measures

The Structured Clinical Interview for DSM-IV-TR Axis I: Patient Edition (SCID; First, Spitzer, Gibbon, & Williams, 2002) was used to determine clinical diagnoses among participants in the study. Clinical symptoms were assessed utilizing the Penn State Worry Questionnaire (PSWQ; Startup & Erickson, 2006; α = .87), the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992; α = .89), the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987; α = .97), and the Beck Depression Inventory (BDI; Beck & Steer, 1987; α = .92). Alcohol expectancies were measured using the Worry-Reduction Alcohol Expectancy Scale (Smith & Tran, 2007; α = .97). To determine participants' interest in treatment for worry, a set of investigator-designed questions were administered to evaluate the impact of worry on treatment. The study questions included, “How much does worry get in the way of your treatment?” (0 = ‘Not at All’, 4 = ‘Extremely’), “Are you interested in treatment for worry?”(Yes/No), and, “When do you think would be the best time to get treatment for worry?” (1 = ‘Now, while in treatment for this addiction’, 2 ‘Later, after this addiction treatment is completed’, 3 ‘I'm interested, but I'm not sure when I would be ready’).

Results

Prevalence of Comorbid GAD and AUD

Among participants diagnosed with an AUD, 18 (46.2%) were also diagnosed with current GAD (see Table 1). Most participants with comorbid GAD-AUD (n = 16, 88.9%) had concurrent alcohol dependence. Comorbid participants were also more likely to have additional DSM-IV diagnoses of panic disorder with agoraphobia, sedative-hypnotic dependence, stimulant dependence, and opioid dependence (ps < .037). No other differences emerged between groups on co-occurring DSM-IV disorders. Among comorbid individuals, GAD co-occurred with AUD independent of any other anxiety disorder in 5/18 (27.8%) of cases. Race/ethnicity differences between these groups were marginally significant (p = .074).

Age of onset Lag

The mean (*SD*) age of onset for GAD among participants in the study sample was 17.56 (7.41). Average AUD onset among individuals with comorbid GAD-AUD was 24.28 years (*SD* = 12.32). The average age of onset for AUD participants without GAD was slightly higher than for those with comorbid GAD (*M* = 27.62 [*SD* = 12.62]). Evaluation of the time lag between the onset of GAD and AUD among participants with both disorders showed that GAD was primary in 12/18 (66.7%) of cases. GAD and AUD occurred in the same year for one case. The time lag among GAD-AUD participants in whom GAD was primary ranged between 1 and 49 years (*M* = 12.5 [*SD* = 13.1]). Alternatively, AUD occurred an average of 5.6 years (*SD* = 4.45) prior to GAD among the five participants in whom the AUD was primary (lag range = 1 to 13 years).

Clinical Symptoms

Group comparisons of clinical symptoms (See Table 2) with a Bonferroni-corrected alpha level for this set of tests (.0125) showed that GAD-AUD participants had higher levels of worry severity than AUD participants (t = -4.079[37], p < .0001). Cohen's d (Cohen, 1988) effect size for the group difference in worry severity = 1.32. The groups did not significantly differ in social anxiety or depressive symptoms (ts = -1.150[37] to -1.682[34], ps < .10). The t-test result from group comparison of anxiety sensitivity was less than .05 (t[36] = -2.158, p = .038), but after application of the corrected alpha for clinical symptom comparisons this difference was not statistically significant.

Clinical Severity

As a proxy for overall clinical severity, a group comparison of suicidality was conducted. Chi-square analysis showed a significant difference between these groups on suicidal behavior (χ^2 = 4.13, p = .042, Φ = .33). Specifically, 10/18 (55.6%) of comorbid participants acknowledged a history of attempted suicide, compared to 5/21 (23.8%) of AUD participants without GAD.

Alcohol Expectancies

Comparison between groups on worry-reduction alcohol expectancies showed that GAD-AUD participants had significantly higher levels of beliefs that alcohol reduces worry (t[37] = -3.328, p = .003). Cohen's d for this statistically significant difference = 1.05.

Treatment Impact of Worry

Comparison of GAD-AUD and AUD groups on the question, “How much does worry get in the way of your treatment?” showed that comorbid individuals had higher levels of treatment interference from worry (t[37] = -2.942, p = .006, d = .93). Chi-square analyses revealed that GAD-AUD participants were more likely to indicate greater than moderate (scale rating >2)

treatment interference from worry ($\chi^2 = 3.94$, $p = .047$, $\Phi = .32$). Comorbid individuals also had higher interest in treatment for worry ($\chi^2 = 8.20$, $p = .004$, $\Phi = .46$). Among the 15 GAD-AUD participants who expressed interest in treatment for worry, the majority (60%) expressed a preference for concurrent treatment.

Discussion

The results of this inquiry showed that GAD was present in elevated rates among study participants, with nearly half (46%) of individuals with an AUD also meeting criteria for this anxiety disorder. This rate of comorbidity, although relatively high, was within the range (8.3% - 56.2%) reported by Kushner et al. (1990) in their review of anxiety and alcohol use disorders. These prevalence findings are especially notable when evaluated in light of the sampling methods used in the current study, which were designed to circumvent the sampling confound presented by patients experiencing anxiety from alcohol withdrawal by evaluating an outpatient sample 2-4 weeks post-treatment initiation. Further support was provided by the age-of-onset analyses, which showed that GAD occurred prior to AUD in 67% of comorbid cases, and that the time lag between GAD and AUD onset among participants with primary GAD was 12.5 years.

Marginally significant race/ethnic differences in the presence of GAD among individuals with an AUD in this treatment-seeking sample, based on an underrepresentation of African-American/Black individuals, were also intriguing. This finding differs from prevalence rates from the NESARC epidemiological study, which showed that African-American/Black individuals were almost twice as likely as White individuals to have comorbid GAD and AUD (Smith et al., 2006). It is unclear based on the present data whether this disparity is a result of treatment-seeking status, or perhaps treatment access for this racial/ethnic group in the present study. Further inquiry with larger treatment-seeking samples can help clarify this inconsistency.

The current study also showed specificity in the symptoms differentiating AUD individuals with and without comorbid GAD. While these groups differed in level of worry severity, the results for anxiety sensitivity, social anxiety, and depression were not statistically significant. Moreover, the effect size for the difference in worry severity showed that this symptom was predictably salient for individuals with GAD.

Results from analyses of suicidality may have been the most notable. In particular, the finding that greater than half (55.6%) of individuals in this sample with co-occurring GAD and AUD attempted suicide is an elevated rate that deserves further inquiry. Recent findings have highlighted the link between anxiety disorders and suicide (e.g. Sareen et al., 2005), and these findings provide preliminary evidence that GAD coupled with AUD may be a potentially lethal combination.

Group differences in both worry-reduction alcohol expectancies and treatment interference from worry were also clinically significant. The results for worry-reduction alcohol expectancies are in line with prior research suggesting that individuals with GAD may self-medicate to cope with the symptoms of this anxiety disorder (Bolton et al., 2006; Grant et al., 2005). While finding that GAD-AUD individuals had greater levels of beliefs that consuming alcohol reduces worry does not explicitly test this hypothesis, the results do provide indirect support in this limited research area.

The impact of worry from the client's perspective may be best expressed, however, by comorbid participants' elevated ratings of treatment interference from, and interest in treatment for, this clinical symptom. It was also noteworthy that most of those who expressed interest in treatment for worry estimated that the best time for this treatment was during their treatment for substance

abuse. Together, these results suggest for the first time that clients perceive that worry has a negative impact on substance abuse treatment. Future work could clarify the impact of worry on treatment, as it is unclear whether worry alone, worry about clinical symptoms, or worry-reduction alcohol expectancies create this treatment obstacle. For example, Kushner et al. (2009) found that tension-reduction alcohol expectancies significantly moderated the efficacy of a treatment for panic disorder among clients with an AUD. Considering the group differences in worry-reduction alcohol expectancies in this study, and previous findings showing that purposeful self-medication with alcohol is prevalent among individuals with GAD (e.g. Robinson et al., 2009), the question of whether it is worry per se, or worry-reduction alcohol expectancies, that negatively impacts treatment begs examination. Upon determination of how worry interferes with treatment, appropriate recommendations for how to best treat this symptom in the context of substance abuse treatment can be made. Moreover, based on finding that the majority of patients in this study with comorbid GAD and AUD had additional co-occurring anxiety disorders (e.g. panic disorder), the most efficient treatment approach may be to treat worry as part of a larger protocol designed to treat comorbid alcohol and anxiety disorders more generally.

Study Limitations and Future Directions

These novel additions to the literature provide a preliminary basis for enhanced focus on worry in substance abuse treatment. However, it is necessary to replicate and extend these results, so that a firm empirical grounding supports these efforts. Toward this end, it is important to consider the current study's findings in the context of its limitations. First, the small sample size of the current pilot study suggests that these findings should be considered preliminary insights into comorbid GAD-AUD. Future studies in this area should include a larger sample. Second, the study data were cross-sectional, so that longitudinal, follow-up data were not collected. Additional research with prospective designs would address this concern. Third, the data on clinical symptoms, alcohol expectancies, and treatment impact of worry were collected via self-report. This methodology, although straightforward, can be subject to response distortion. Fourth, and finally, the presence of state anxiety was not measured during the 2-4 week timeframe between treatment onset and study participation. Future efforts should more closely monitor anxiety across this period to rule out the presence of anxiety due to alcohol withdrawal.

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Table 1

Demographics of Study Sample [a]

Variable	AUD	Comorbid GAD-AUD	*p*
N	21 (53.8%)	18 (46.2%)	
Age			*ns*
Range	21-67	19-60	
Mean (SD)	40.00 (13.51)	39.11 (12.45)	
Sex			*ns*
Male	8 (38.1%)	9 (50.0%)	
Female	13 (61.9%)	9 (50.0%)	
Race			.074
African-American/Black	8 (38.1%)	2 (11.1%)	
Caucasian/White	13 (61.9%)	16 (88.9%)	
Marital Status			*ns*
Never Married	9 (42.9%)	7 (38.9%)	
Married	5 (23.8%)	8 (44.4%)	
Separated/Divorced	5 (23.8%)	3 (16.7)	
Widowed	2 (9.5%)	0 (0.0%)	

[a] Age comparison was conducted using a two-tailed independent samples *t*-test. Sex, race, and marital status comparisons were conducted using exact two-tailed chi-square tests.

Table 2

Clinical Symptom Comparison of AUD Participants With and Without Comorbid GAD[a]

Variable	Group	*N*	M	*SD*	95% Confidence Interval	Min-Max (Range)[b]	*t*	df	*p*-value[c]
Worry severity (PSWQ)	AUD	21	46.29	13.58	40.48 - 52.10	22-80 (58)	-4.079	37	<.0001
	GAD-AUD	18	62.78	11.31	57.56 - 68.00				
Anxiety Sensitivity (ASI)	AUD	20	25.33	10.56	20.70 - 29.96	9-57 (48)	-2.158	36	0.038
	GAD-AUD	18	33.76	13.54	27.50 - 40.20				
Social Anxiety (LSAS)	AUD	19	49.55	32.42	34.97 - 64.13	6-134 (128)	-1.682	34	0.102
	GAD-AUD	17	67.56	31.28	52.69 - 82.43				
Depression (BDI)	AUD	21	18.29	11.77	13.26 - 23.32	4-53 (49)	-1.15	37	0.257
	GAD-AUD	18	22.72	12.29	17.46 - 27.98				

[a] Clinical symptom comparisons were conducted using two-tailed independent samples *t*-tests.

[b] Minimum, maximum scores and ranges obtained in the study sample.

[c] *p*-values were assessed for significance based on a Bonferroni-corrected alpha level from the total number of planned tests (.05/4 = .0125). Significant results using this corrected alpha are bolded.