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REVIEW

# Prevalence of depression and anxiety symptoms by liver disease etiology

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Health care providers who care for patients with liver disease (LD) often come to learn that psychiatric comorbidities, such as depression (DEP) and anxiety (ANX) symptoms, are prevalent within this population. Over the past 10–15 years, a growing body of literature has provided evidence to support the association between LD and the presence of DEP and/or ANX.[1–5] These findings are often embedded within a heterogeneous mix of studies examining the association(s) between LD and health-related quality of life, predictors of increased morbidity and mortality among these patients, or clinical outcomes measured as part of liver transplant.[1–5]

The prevalence rates of DEP and ANX reported by many investigations also widely vary due to differences in the screening instruments and diagnostic thresholds used to define clinically relevant affective symptoms. For example, the prevalence of DEP symptoms in patients diagnosed with chronic LD was 63% in studies using self-report inventories, with far lower rates (4.5%) observed in studies using diagnostic clinical interviews.[2] This brief review aims to summarize the prevalence of both DEP and ANX symptoms in patients with LD, paying particular attention to screening measures and diagnostic thresholds used across studies. In addition, prevalence rates seem to vary across different etiology and histologic staging of LD.

## ALCOHOL-ASSOCIATED LIVER DISEASE (ALD)

DEP and ANX symptoms are highly prevalent in patients with alcohol use disorders,[4] and there is evidence to support an association between these comorbidities (Table 1). Rogal and colleagues reported the prevalence of ICD-9 DEP diagnoses to be 22% in patients with alcohol-associated liver disease (ALD) before liver transplant. Further, pretransplant DEP was also associated with post-transplant survival rates, length of hospitalization, and discharge to an outside facility.[8] In a prospective cohort study of 51 patients with ALD waitlisted for transplant,[6] the prevalence of self-report DEP and ANX symptoms was reported as 37.3% and 43.1%, respectively.

As part of a prospective longitudinal study, DiMartini and colleagues reported the lifetime prevalence of clinical DEP and ANX, per DSM-IV criterion, to be 20% and 13%, respectively, among 167 patients with ALD before liver transplant. The authors also showed that the lifetime prevalence of clinical DEP was the strongest predictor of survival, conferring a 2-time higher risk of mortality among patients with ALD.[7] In one of the only studies to exclusively examine self-reported ANX among patients with ALD awaiting transplant, Santos and colleagues reported the prevalence of ANX symptoms to be 29%.

**Abbreviations:** AIH, autoimmune hepatitis; ANX, anxiety; BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; CES-D, Center for Epidemiological Studies Depression Scale; CIDI, Composite International Diagnostic Interview; DEP, depression; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; GADI, Generalized Anxiety Disorder Inventory; HADS, Hospital Anxiety and Depression Scale; HADS, Hospital Anxiety and Depression Scale; HAM-D, Hamilton Depression Rating Scale; ICD-9, International Classification of Disease, Ninth Edition; LD, liver disease; MDD, major depressive disorder; PHQ-9, Patient Health Questionnaire, 9-Item Version; PBC, primary biliary cholangitis; PSC, primary sclerosing cholangitis; STAI, State-Trait Anxiety Inventory.

**TABLE 1** DEP and ANX in patients with ALD

| References, country | Study design | ALD patient (N) | Comparison group (N) | Assessment and cut-score | Prevalence DEP | Prevalence ANX |
|---|---|---|---|---|---|---|
| *Annema et al*[6] *Netherlands* | *Prospective cohort* | *51* | — | *STAI, ≥ 12* CES-D, ≥ 16 | *37.3* | *43.1* |
| *DiMartini et al*[7] *US* | *Prospective longitudinal* | *167* | — | *DSM-IV Diagnoses* | *20* | *13* |
| Rogal et al[8] US | Retrospect-analysis | 46 ALD w/DEP | 162 ALD w/o DEP | ICD-9 diagnoses | 22% vs. 19% | — |
| *Santos et al*[9] *Brazil* | *Prospective* | *< 80*[a] | — | *BAI, ≥ 8* | — | *29% with mild, moderate, or severe ANX* |

*Note:* Italicized = Studies that reported the prevalence of ANX, either in combination with- or independent of the prevalence of DEP.
[a]This study did not provide the N of patients with ALD, but instead indicated HCV to be the modal etiology of LD with N = 80 patients.
Abbreviations: BAI, Beck Anxiety Inventory; CES-D, Center for Epidemiological Studies Depression Scale; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; ICD-9, International Classification of Disease, Ninth Edition; STAI, State-Trait Anxiety Inventory.

## NONALCOHOLIC FATTY LIVER DISEASE

NAFLD is the hepatic manifestation of metabolic syndrome. It is well established that DEP is associated with aspects of metabolic syndrome (eg, obesity).[10] Similarly, a growing body of research supports the association between NAFLD and increased DEP, though far fewer studies report on the prevalence of ANX among patients with NAFLD[1] (Table 2).

Xiao and colleagues reported the pooled prevalence of DEP to be 18.2% across 11 studies using NAFLD samples. Several cross-sectional investigations of patients with NAFLD also report on the prevalence of DEP and ANX, though rates widely vary. For example, Weinstein and colleagues reported the prevalence of DEP to be 27.2% in NAFLD when using self-reported diagnoses paired with confirmatory medical record review. Further, patients with NAFLD were shown to have a significantly higher prevalence of DEP compared with those with hepatitis B (HBV; 3.7%) but not hepatitis C (29.8%).[12] In a large population-based study of 567 patients with NAFLD,[14] Youssef and colleagues showed that 14% of patients endorsed moderate to severe DEP on the Hospital Anxiety and Depression Scale (HADS), while 53% reported subclinical/mild symptoms of DEP. Similar findings were also shown for ANX, as 25% of patients with NAFLD reported moderate/severe symptoms, while 45% reported subclinical ANX symptoms.[14]

Evidence also supports an association between DEP/ANX and the histological staging of NAFLD. For example, Youssef and colleagues reported that patients endorsing moderate/severe DEP were 3.6 times more likely to present with more severe NAFLD compared with patients endorsing subclinical/no DEP. This finding was also observed in a large, population-based study of 112,797 Korean men and women, whereby the severity of NAFLD was significantly associated with self-reported DEP, even after covariate adjustment.[11] Regarding ANX, Choi and colleagues reported that the prevalence of *both* state- and trait-ANX (ie, here state-ANX is defined as ANX one feels in the moment while trait-ANX is ANX one generally feels overall) increased in patients with more severe NAFLD, but only among women.[15]

## HCV-RELATED LD

DEP and ANX symptoms are known to be prevalent in patients with HCV,[16] and they often occur at a higher rate compared to patients with HBV or healthy controls.[17–19] In a meta-analysis of 12 studies that included 130,039 patients,[19] the pooled prevalence of DEP was 24.5% among those diagnosed with HCV compared with 17.2% of non-HCV controls (Table 3). The authors also reported that patients with HCV had

**TABLE 2** DEP and ANX in patients with NAFLD

| References, country | Study design | NAFLD patient (N) | Comparison group (N) | Assessment and cut-score | Prevalence DEP | Prevalence ANX |
|---|---|---|---|---|---|---|
| Jung et al[11] Korea | Cross-sectional | 112,797 | — | CES-D ≥ 16 | 11.1% (FLI—normal) | — |
| | — | — | — | — | 8.9% (FLI—mild) | — |
| | — | — | — | — | 9.7% (FLI—Mod/severe) | — |
| Weinstein et al[12] US | Cross-sectional | 184 | 190 HBV 504 HCV | Self-Report + MR Confirm | 27.2% NAFLD 3.7% HBV 29.8% HCV | — |
| Xiao et al[13] Singapore | Meta-analysis | 2,041,752 | — | Various | 18.2% pooled estimate | — |
| *Youssef et al*[14] *US* | *Cross-sectional* | *567* | — | *HADS-D (1–7, ≥ 8) HADS-A (1–7, ≥8)* | *14% (clinical ≥ 8) 53% (Subclinical 1–7)* | *25% (clinical ≥ 8) 45% (subclinical 1–7)* |

*Note*: Italicized = Studies that examined both depression and anxiety symptoms.
Abbreviations: CES-D, Center for Epidemiological Studies Depression Scale; FLI, Fatty Liver Index; HADS, Hospital Anxiety and Depression Scale; MR, medical record.

twice the risk of DEP compared with healthy controls.[19] Carta and colleagues showed the lifetime prevalence of major depressive disorder (MDD), assessed through structured clinical interview, was 32.6% for HCV, 15.5% for HBV, and 12.8% for healthy controls. Similarly, the lifetime prevalence of panic disorder was 8.9% for patients with HCV compared with 7.9% for HBV and 2.8% for healthy controls. Associations between HCV and generalized ANX disorder or social phobia were not significant.[17] Two population-based studies in the United States[18] and Germany[20] reported the prevalence of DEP as 29.7% and 25.9%, respectively, though neither survey investigation measured ANX.

Historical data have shown that interferon (INF)-based antiviral therapy for patients with HCV is frequently associated with treatment-induced DEP and ANX.[22] Schaefer and colleagues reported the prevalence of DEP during INF treatment to be 30%–70% based on 10 studies, though prevalence rates varied by type of screening instrument. For example, it was estimated that MDD developed in 15%–45% of patients treated with INF,[22] as assessed by diagnostic interview. Similarly, the authors estimated the

**TABLE 3** DEP and ANX in patients with HCV-related LD

| References, country | Study design | HCV patient (N) | Comparison group (N) | Assessment and cut-score | Prevalence DEP | Prevalence ANX |
|---|---|---|---|---|---|---|
| Boscarino et al[18] US | Cross-sectional | 4781 | — | PHQ-8, ≥ 10 | 29.7% | — |
| *Carta et al*[17] *Italy* | *Cross-sectional* | *135* | *76 HBV 540 control* | *CIDI* | *32.6% lifetime-HCV 15.1% lifetime-HBV 12.8% lifetime-control* | *8.9% lifetime panic-HCV 7.9% lifetime panic-HBV 2.8% lifetime panic-control* |
| Erim et al[20] Germany | *Cross-sectional* | 81 | — | BDI, ≥14 HADS-A, ≥7 | 25.9% | — |
| *Gallach et al*[21] *Spain* | *Intervention* | *145* | — | *HADS ≥7* | *13.1% at baseline* | *8.3% at baseline* |
| *Schaefer et al*[22] | *Review* | — | — | *Various* | *30%–70% 15%–45% MDD* | *11%—45%* |
| Younossi et al[19] | Meta-analysis | 130,039 | 127,506 | Various | 24.5% pooled estimate 17.2% of non-HCV controls | — |

*Note:* Italicized = Studies that examined both DEP and ANX.
Abbreviations: BDI, Beck Depression Inventory; CIDI, Composite International Diagnostic Interview; GADI, Generalized Anxiety Disorder Inventory; HADS, Hospital Anxiety and Depression Scale; INF, Interferon (INF)-alpha treatment; PHQ-8, Patient Health Questionnaire, 8-Item Version.

**TABLE 4** DEP and ANX in patients with other etiologies of chronic LD

| References, country | Study design | Etiology LD patient (N) | Comparison group (N) | Assessment and cut-score | Prevalence DEP | Prevalence ANX |
|---|---|---|---|---|---|---|
| Huang *et al*[23] China | Prospective | 209 HBV | — | HAM-D ≥ 20 | 5.3% | — |
| | — | — | — | HAM-D 7-19 | 23.9% | — |
| Huet et al[24] Canada | Cross-sectional | 116 PBC | — | BDI ≥10 | 44.8% | — |
| *Janik et al*[25] *Poland* | *Cross-sectional* | *140 AIH* | *170* healthy | *PHQ-9* STAI | *29% w/ moderate to severe symptoms* | *Measured, but rates not reported* |
| van Os et al[26] Netherlands | Cross-sectional | 92 | — | BDI ≥10 | 42% | — |
| | — | PBC and PSC | — | *DSM-IV* | — | — |
| | — | — | — | MDD | 3.7% | — |
| *Schramm et al*[27] *Germany* | *Cross-sectional* | *103 AIH* | *1939 Healthy* | *PHQ-9* | *5.9% w/DEP symptoms* | *8.3% w/ moderate ANX* |
| | — | — | 3720 Healthy | *GAD-7* | *10.8% w/ MDD* | *4.2% w/ severe ANX* |
| *Yilmaz et al*[28] *Turkey* | *Cross-sectional* | *41 HBV* | *36 inactive HBV and 53 healthy* | *HADS-D > 10* | *60% HBV* | *48.7% HBV* |
| | — | — | — | *HADS-A > 8* | *47.2% inactive HBV* | *33.5% inactive HBV* |
| | — | — | — | — | *25% Healthy* | *20.7% Healthy* |

*Note:* Studies that examined both DEP and ANX.

Abbreviations: AIH, autoimmune hepatitis; BDI, Beck Depression Inventory; *DSM-IV*, *Diagnostic and Statistical Manual, Fourth Edition*; GADI, generalized anxiety disorder-7; HADS, Hospital Anxiety and Depression Scale; HAM-D, Hamilton Depression Rating Scale; MDD, major depressive disorder; PHQ-9, Patient Health Questionnaire, 9-Item Version; PBC, primary biliary cholangitis; PSC, primary sclerosing cholangitis; STAI, State-Trait Anxiety Inventory.

prevalence of ANX during INF treatment to be 11%–45% across 7 studies.[22] However, when comparing the prevalence rates of DEP and ANX between INF and direct-acting antiviral agents in patients with HCV, research suggests that direct-acting antiviral agents do not seem to be associated with treatment-inducted DEP or ANX.[21] Gallach and colleagues showed that the prevalence of self-reported baseline DEP and ANX was 13.1% and 8.3%, respectively. However, no differences in DEP and ANX rates were found either during or after 12-week direct-acting antiviral agent treatment, suggesting that this antiviral therapy did not significantly impact psychological symptoms during the trial or after sustained virologic response.[21]

## OTHER ETIOLOGIES OF LD

This brief review also considers DEP and ANX across several other etiologies of LD: primary biliary cholangitis, primary sclerosing cholangitis (PSC), autoimmune hepatitis (AIH), and HBV (Table 4).

While examining the effect of fatigue on quality of life in patients with primary biliary cholangitis, Huet and colleagues found self-reported DEP to be ~45%.[24] Similarly, in a Dutch sample of 92 patients with either primary biliary cholangitis or PSC, self-reported DEP was found to be 42%, though only 3.7% of patients met DSM-IV criteria for MDD.[26] While neither study measured symptoms of ANX, one recent investigation provided qualitative findings on PSC and ANX.[29] Ranieri et al[29] adopted a phenomenological approach to understand the subjective narratives of 22 patients with PSC who participated in 4 weeks of virtual focus groups. ANX was a frequent theme described by patients and often emerged at the time of PSC diagnosis. Further, ANX-based themes included the following concerns: risk of early mortality, reduced quality of life, worsening physical health, the need for further medical testing and/or transplant, and missed opportunities to celebrate developmental and lifetime achievements of their children.[29]

Several cross-sectional investigations assessed the prevalence of self-reported DEP and ANX in patients with AIH compared with age-matched and sex-matched controls.[25,27,30] Schramm and colleagues reported that the frequency of self-reported DEP and moderate ANX among 103 German patients with AIH to be twice that observed in the German general population (ie, 5.9% vs. 2.6% DEP and 8.3% vs. 4.4% ANX). Further, 10.8% of patients with AIH met criteria for MDD and 4.2% presented with severe ANX, which were respectively, 5 and 4 times greater than rates in the general population (ie, 1.9% MDD and 1.1% ANX).[27]

The prevalence of DEP in patients with HBV has been examined,[12,17,23,28,31,32] though limited data on rates of ANX are available.[17,28] Comparative investigations commonly show that the prevalence of DEP in patients with HBV is lower than that in patients with HCV,[12,17,32] a finding that extends to both self-report–diagnosed and clinician-diagnosed DEP. At least 1 investigation examined rates of self-reported DEP and ANX in patients with HBV.[28] Yilmaz and colleagues showed that 60% of patients with HBV patients endorsed DEP compared with 47.2% of patients with inactive HBV and 25% of healthy controls. Similarly, 48.7% of patients with HBV endorsed ANX, followed by 33.5% and 20.7% of those with inactive HBV or healthy controls.[28]

## SUMMARY

DEP and ANX are prevalent in patients with LD. These associations persist even when considering multiple LD etiologies. Subsequently, DEP and ANX should not be underestimated in patients with LD, particularly since there is a growing body of evidence that both comorbidities can negatively impact patient-centered outcomes. Numerous well-validated, brief measures for assessing DEP and ANX in patients with LD are widely available, and both psychotropic medication management and/or psychological counseling are well-known treatments for these comorbidities. However, more targeted DEP and ANX treatment studies for patients with LD are needed at this time, particularly investigations that focus on the efficacy of affective symptom treatment within samples of patients diagnosed with varying LD etiologies.

### CONFLICTS OF INTEREST

The authors have no conflicts to report.

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