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Research Article
doi: 10.61705/mqfksc47
Ace J. Addict., 7 (1): 46-54 | 2026

Effectiveness of a Structured Psychoeducational Intervention in Improving Sleep Quality, Anxiety and Depressive Symptoms among Patients with Alcohol Use Disorder: A Quasi-Experimental Study

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Key Words
alcohol use disorder; psychoeducation; sleep quality; anxiety; depression; quasi-experimental study

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Received: 30 Sep 2026
Accepted: 12 Oct 2026
Published: 26 Oct 2026

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ABSTRACT

Background: Poor sleep, anxiety and depressive symptoms persist after detoxification in alcohol use disorder (AUD) and predict relapse. Low-cost, non-pharmacological approaches suited to Indian de-addiction settings are poorly evaluated.

Aim: To assess whether a structured psychoeducational intervention, added to treatment as usual (TAU), improves sleep quality, anxiety and depressive symptoms in patients with AUD after detoxification.

Methods: A non-equivalent control group, pre-test/post-test/follow-up quasi-experimental study enrolled 120 detoxified adults with DSM-5 AUD (CIWA-Ar < 10) at [setting]. The intervention group (n = 60) received [six] structured group psychoeducation sessions over 4 weeks plus TAU; the control group (n = 60) received TAU. The Pittsburgh Sleep Quality Index (PSQI), Hamilton Anxiety Rating Scale (HAM-A) and Beck Depression Inventory-II (BDI-II) were administered at baseline (T0), 4 weeks (T1) and 8 weeks (T2). Analyses used baseline-adjusted ANCOVA and linear mixed-effects models.

Results:

Groups were comparable at baseline except PSQI, which was higher in the intervention group (12.48 ± 2.37 vs 11.37 ± 2.66; p = 0.017). In all, 108 participants (90.0%) completed T2. At 8 weeks, adjusted between-group differences favoured the intervention for PSQI (−2.92; 95% CI −3.76 to −2.08; partial η^2 = 0.31), HAM-A (−5.22; −6.87 to −3.58; partial η^2 = 0.27) and BDI-II (−5.70; −7.59 to −3.80; partial η^2 = 0.25; all p < 0.001). Group × time interactions were significant for all three outcomes (p < 0.001). Poor sleep (PSQI > 5) persisted in 75.5% vs 92.7% (p = 0.017). Relapse by 8 weeks was 26.4% vs 47.3% (RR 0.56; p = 0.041; NNT ≈ 5).

Conclusion: Structured psychoeducation added to TAU was associated with better sleep quality and lower anxiety and depressive symptoms over 8 weeks after detoxification. Randomised trials with longer follow-up are needed.

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INTRODUCTION

Alcohol use disorder (AUD) is one of the leading preventable causes of death and disability worldwide. The World Health Organization estimates that alcohol causes about 2.6 million deaths each year, and around 400 million people aged 15 years and above live with alcohol use disorders [1]. Alcohol was the leading risk factor for death and disability-adjusted life-years among people aged 15–49 years in the Global Burden of Disease analysis [2]. AUD is a chronic, relapsing condition with medical, psychiatric and social consequences [3]. In the United States, 12-month and lifetime prevalence of DSM-5 AUD were 13.9% and 29.1%, and fewer than one in five affected people ever received treatment [4]. In India, about 5.2% of people aged 10–75 years are estimated to need help for alcohol problems [5], and the National Mental Health Survey found a large treatment gap for alcohol use disorders [6].

Sleep disturbance is one of the most common and persistent problems in AUD. Alcohol initially shortens sleep onset, but chronic use and withdrawal disrupt sleep continuity, reduce slow-wave sleep and fragment REM sleep [7]. Insomnia affects a large share of patients entering treatment and often continues for months into abstinence [8]. A feed-forward allostatic model suggests that sleep disruption and negative emotional states sustain each other and drive further drinking [9]. Insomnia at treatment entry has predicted later relapse, in part because patients use alcohol to self-medicate sleeplessness [10].

Anxiety and depressive disorders frequently co-occur with AUD. Large epidemiological surveys show strong associations between AUD and independent mood and anxiety disorders [11, 12]. Relapse after treatment remains common, and post-treatment problems predict it [13]. The links run in both directions: negative affect can motivate drinking, while heavy drinking produces depressive and anxious symptoms [14, 15]. Many alcohol-induced depressive symptoms fall within weeks of abstinence [16], but a substantial group continues to have clinically significant anxiety and depression after detoxification, which predicts earlier return to drinking [17]. Anxiety and AUD share pathways of stress reactivity and avoidance [18], and neurobiological models place negative affect at the centre of the withdrawal–relapse cycle [19].

Approved medicines for AUD exist but are underused [20, 21], and recent work stresses the need for accessible behavioural treatments that address the full clinical picture [22]. Cognitive-behavioural therapy has moderate effects on substance use outcomes [23] and adds benefit to pharmacotherapy [24]. Large trials such as Project MATCH and COMBINE showed that structured psychosocial treatments achieve meaningful improvement [25, 26]. In India, a brief lay-counsellor-delivered treatment reduced harmful drinking in primary care, showing that structured, low-intensity psychological care can work in resource-limited settings [27].

Psychoeducation is a structured, didactic and interactive process that gives patients accurate information about their illness and its treatment, and teaches skills to manage it [28]. It improves outcomes in schizophrenia and bipolar disorder [29–31] and reduces depressive and anxiety symptoms in the general population [32]. For sleep, behavioural approaches such as stimulus control and sleep hygiene are recommended as first-line treatment for insomnia in AUD [33], and cognitive-behavioural therapy for insomnia improves sleep in patients recovering from alcohol dependence [34]. A meta-analysis found that insomnia treatments improve sleep in AUD, although effects on drinking were less clear [35]. Stigma about alcohol dependence is high, and education that frames AUD as a treatable health condition may support engagement [36].

Despite this evidence, few studies have tested a single, structured psychoeducational package that addresses sleep, anxiety and depressive symptoms together in the early post-detoxification period, particularly in Indian de-addiction settings. This study aimed to assess the effectiveness of a structured psychoeducational intervention, added to treatment as usual, on sleep quality (primary outcome), anxiety and depressive symptoms (secondary outcomes) among patients with AUD. We hypothesised that the intervention group would show greater improvement than the control group at 4 and 8 weeks.

Methods

Study design and setting

This was a quasi-experimental study with a non-equivalent control group and pre-test, post-test and follow-up measurements. It was conducted at [name of de-addiction centre / department of psychiatry, city] between [month year] and [month year]. Reporting follows the Transparent Reporting of Evaluations with Nonrandomized Designs (TREND) statement [37].

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Participants

Adults aged 21–60 years with a DSM-5 diagnosis of AUD [38] were eligible after completing inpatient detoxification, defined as a Clinical Institute Withdrawal Assessment for Alcohol–Revised (CIWA-Ar) score below 10 [39]. Other inclusion criteria were an Alcohol Use Disorders Identification Test (AUDIT) score of 20 or more [40], ability to understand [Hindi/English], and written informed consent. Exclusion criteria were [other substance use disorder except tobacco, psychotic or bipolar disorder, cognitive impairment, serious medical illness, current hypnotic or antidepressant start within 2 weeks, and current structured psychotherapy].

To avoid contamination, participants were allocated by [ward / admission period]: those admitted to [Ward A / during period 1] formed the intervention group and those admitted to [Ward B / during period 2] formed the control group. Consecutive eligible patients were enrolled until each group reached 60.

Sample size

Sample size was calculated with G*Power 3.1 [41] for the between-group difference in PSQI global score at 8 weeks. Assuming a medium effect (Cohen's d = 0.6), two-sided $\alpha = 0.05$ and 80% power, 45 participants per group were required. Allowing for 25% attrition, 60 participants per group (120 in total) were enrolled. [Confirm the assumed effect size and its source.]

Intervention

The intervention group received a structured psychoeducational programme in addition to treatment as usual. It comprised [six] group sessions of [45–60] minutes delivered over 4 weeks by [a trained psychiatric nurse / clinical psychologist], in groups of [6–10], with a printed take-home booklet. Sessions combined short teaching, discussion, demonstration and homework, and drew on motivational [42], sleep-hygiene [43] and relapse-prevention [44] principles:

1. AUD as a health condition: effects of alcohol on brain, body, sleep and mood; myths and stigma.
2. Sleep and alcohol: why sleep is poor after stopping; sleep hygiene, stimulus control and a regular sleep–wake schedule.
3. Anxiety: recognising symptoms; breathing retraining and progressive muscle relaxation.
4. Low mood: the link between drinking and depression; activity scheduling and challenging unhelpful thoughts.
5. Relapse prevention: cravings, high-risk situations, refusal skills and coping plans.
6. Family and follow-up: role of family support, medication adherence and when to seek help; review and booster plan.

Attendance was recorded at each session. [Fidelity checks, facilitator training and the booklet should be described or provided as supplementary material.]

Control group

The control group received treatment as usual (TAU): [routine pharmacological management, brief advice by the treating team and scheduled outpatient review]. After the final follow-up, the control group was offered the psychoeducational booklet and [a condensed session].

Data collection and outcome measures

A structured proforma recorded age, sex, education, marital status, occupation, residence, socioeconomic status (modified Kuppuswamy scale) [45], age at first drink, duration of AUD, AUDIT score, tobacco use, family history of AUD, prior detoxification attempts and CIWA-Ar score at entry.

The primary outcome was sleep quality, measured by the Pittsburgh Sleep Quality Index (PSQI). It yields a global score of 0–21; a score above 5 indicates poor sleep [46]. Secondary outcomes were anxiety, measured with the clinician-rated Hamilton Anxiety Rating Scale (HAM-A; 0–56) [47], and depressive symptoms, measured with the Beck Depression Inventory-II (BDI-II; 0–63) [48]. Relapse, defined as [any drinking / heavy drinking] by 8 weeks [by self-report confirmed by a family member], was an exploratory outcome. Outcomes were measured at baseline after detoxification (T0), at 4 weeks at the end of the intervention (T1) and at 8 weeks of follow-up (T2) by [an assessor who was not involved in delivering the intervention].

Statistical analysis

Data were analysed with [software and version]. Continuous variables are reported as mean ± SD and categorical variables as n (%). Baseline groups were compared with the independent t-test and the chi-square or Fisher's exact test. Within-group change was tested with the Friedman test and paired t-tests (T0 vs T2). The main between-group analysis was analysis of covariance (ANCOVA) on T1 and T2 scores, adjusted for the baseline score [49], with partial η^2 as effect size. A linear mixed-effects model with a random intercept for participant, fixed effects for group, time and group × time, fitted by restricted maximum likelihood, used all available observations; the interaction was tested with a likelihood-ratio test.

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Sensitivity analysis imputed missing T2 values by last observation carried forward. Proportions with poor sleep and relapse were compared with the chi-square or Fisher's exact test, and relative risk and number needed to treat were calculated. Pearson correlations assessed associations between changes in outcomes. Two-sided $p < 0.05$ was considered significant.

Ethics

The study was approved by [Institutional Ethics Committee name, approval number, date] and [registered with the Clinical Trials Registry–India, CTRI/____]. It was conducted in accordance with the Declaration of Helsinki [50]. All participants gave written informed consent, and confidentiality was maintained throughout.

Results

Participant flow and baseline characteristics

Of 120 participants enrolled, 115 (95.8%) were assessed at 4 weeks and 108 (90.0%) at 8 weeks. Loss to follow-up was 7 of 60 (11.7%) in the intervention group (1 before T1, 6 before T2) and 5 of 60 (8.3%) in the control group (4 before T1, 1 before T2). Completers had higher baseline BDI-II scores than non-completers (24.64 vs 19.33; $p = 0.016$), but did not differ in age, AUDIT, PSQI or HAM-A scores. Intervention participants attended a mean of 5.13 ± 0.85 of 6 sessions; 48 (80.0%) attended at least 5.

1. Mean age was 38.62 ± 8.52 years in the intervention group and 39.80 ± 10.57 years in the control group; 95.0% of each group were men (Table 1). The groups did not differ significantly in any sociodemographic or clinical variable. Tobacco use was less frequent in the intervention group (68.3% vs 83.3%; $p = 0.088$).

Table 1. Baseline characteristics of participants (n = 120)

Characteristic	Intervention (n = 60)	Control (n = 60)	p
Age, years, mean ± SD	38.62 ± 8.52	39.80 ± 10.57	0.501[a]
Male sex, n (%)	57 (95.0)	57 (95.0)	1.000[c]
Education: illiterate / primary / secondary / higher secondary / graduate, n	6 / 10 / 17 / 16 / 11	7 / 10 / 17 / 14 / 12	0.993[b]
Married, n (%)	35 (58.3)	35 (58.3)	0.319[b]
Unemployed, n (%)	8 (13.3)	11 (18.3)	0.855[b]
Residence: urban / semi-urban / rural, n	27 / 12 / 21	33 / 8 / 19	0.472[b]
SES lower or upper lower, n (%)	22 (36.7)	22 (36.7)	0.721[b]
Age at first drink, years	19.38 ± 3.03	19.12 ± 3.31	0.646[a]
Duration of AUD, years	11.27 ± 6.19	12.05 ± 7.56	0.536[a]
AUDIT score	26.80 ± 4.74	26.82 ± 4.66	0.985[a]
Tobacco use, n (%)	41 (68.3)	50 (83.3)	0.088[b]
Family history of AUD, n (%)	25 (41.7)	30 (50.0)	0.464[b]
Prior detoxification attempts	1.28 ± 1.17	1.13 ± 1.00	0.451[a]
CIWA-Ar at entry	4.92 ± 1.86	4.73 ± 2.05	0.609[a]

[a] Independent t-test. [b] Chi-square test across all categories of the variable. [c] Fisher's exact test. SES, socioeconomic status (modified Kuppuswamy scale).

Sleep quality, anxiety and depressive symptoms over time

At baseline, PSQI was higher in the intervention group (12.48 ± 2.37 vs 11.37 ± 2.66; $p = 0.017$), while HAM-A and BDI-II scores did not differ (Table 2). All participants had poor sleep (PSQI > 5) at baseline. By 4 and 8 weeks, mean scores on all three measures were lower in the intervention group, with unadjusted effect sizes of $d = 0.49–0.55$ for PSQI, 0.98–1.01 for HAM-A and 0.82–0.93 for BDI-II.

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Table 2. Outcome scores by group and time point (mean ± SD)

Outcome	Time	Intervention	Control	t	p	Cohen's d
PSQI	T0 (n = 60/60)	12.48 ± 2.37	11.37 ± 2.66	2.43	0.017	0.44
PSQI	T1 (n = 59/56)	8.71 ± 3.33	10.25 ± 2.92	−2.63	0.010	−0.49
PSQI	T2 (n = 53/55)	8.00 ± 3.16	9.82 ± 3.40	−2.88	0.005	−0.55
HAM-A	T0	19.63 ± 5.41	20.82 ± 5.67	−1.17	0.244	−0.21
HAM-A	T1	12.02 ± 6.40	18.34 ± 6.53	−5.24	<0.001	−0.98
HAM-A	T2	11.38 ± 6.83	18.35 ± 6.97	−5.25	<0.001	−1.01
BDI-II	T0	23.07 ± 6.83	25.15 ± 7.60	−1.58	0.117	−0.29
BDI-II	T1	15.76 ± 8.05	22.34 ± 8.02	−4.39	<0.001	−0.82
BDI-II	T2	14.15 ± 8.66	22.56 ± 9.47	−4.81	<0.001	−0.93

T0, baseline; T1, 4 weeks; T2, 8 weeks. n shown as intervention/control. Independent t-test; d uses the pooled SD. Lower scores indicate better sleep and fewer symptoms.

Within-group change

Both groups improved over time on all outcomes (Friedman test, $p < 0.001$ for each), but improvement was larger in the intervention group (Table 3). From baseline to 8 weeks, PSQI fell by 4.57 points in the intervention group and 1.65 points in the control group; HAM-A by 7.83 and 2.67; and BDI-II by 9.21 and 3.31 points.

Table 3. Within-group change from baseline to 8 weeks (completers)

Outcome	Group (n)	Mean change T0–T2 (95% CI)	Paired t	p	d_x	Friedman χ^2 (df = 2)
PSQI	Intervention (53)	4.57 (3.96 to 5.17)	14.84	<0.001	2.04	70.83
PSQI	Control (55)	1.65 (1.12 to 2.19)	6.07	<0.001	0.82	25.11
HAM-A	Intervention (53)	7.83 (6.62 to 9.04)	12.66	<0.001	1.74	67.65
HAM-A	Control (55)	2.67 (1.62 to 3.72)	5.00	<0.001	0.67	28.64
BDI-II	Intervention (53)	9.21 (7.91 to 10.50)	13.94	<0.001	1.91	71.42
BDI-II	Control (55)	3.31 (1.99 to 4.63)	4.93	<0.001	0.66	22.08

Positive change indicates improvement. d_x, standardised mean change (mean change / SD of change).

Between-group effects adjusted for baseline

After adjustment for baseline scores, the intervention group had lower PSQI, HAM-A and BDI-II scores at both 4 and 8 weeks (Table 4). At 8 weeks the adjusted difference was −2.92 points for PSQI, −5.22 for HAM-A and −5.70 for BDI-II, with large effect sizes (partial η^2 0.25–0.31). Mixed-effects models using all 343 observations confirmed significant group × time interactions for all three outcomes. Results were unchanged in the last-observation-carried-forward sensitivity analysis (n = 120): PSQI −2.85 (−3.64 to −2.06), HAM-A −5.37 (−6.95 to −3.78) and BDI-II −5.86 (−7.62 to −4.09); all $p < 0.001$.

Table 4. Baseline-adjusted between-group differences (intervention minus control)

Outcome	Time	ANCOVA adjusted difference (95% CI)	F (df)	p	Partial η^2	Mixed model group × time β (95% CI)	LR χ^2 interaction (df = 2)
PSQI	T1	−2.55 (−3.35 to −1.76)	40.10 (1, 112)	<0.001	0.264	−2.63 (−3.46 to −1.80)	51.22, p < 0.001
PSQI	T2	−2.92 (−3.76 to −2.08)	47.77 (1, 105)	<0.001	0.313	−2.92 (−3.77 to −2.06)	
HAM-A	T1	−4.89 (−6.26 to	50.15 (1,	<0.001	0.309	−4.90 (−6.41 to	52.81, p <

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doi: 10.61705/mqfksc47
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Outcome	Time	ANCOVA adjusted difference (95% CI)	F (df)	p	Partial η^2	Mixed model group × time β (95% CI)	LR χ^2 interaction (df = 2)
		−3.52)	112)			−3.39)	0.001
HAM-A	T2	−5.22 (−6.87 to −3.58)	39.69 (1, 105)	<0.001	0.274	−5.34 (−6.89 to −3.79)	
BDI-II	T1	−4.13 (−5.63 to −2.63)	29.77 (1, 112)	<0.001	0.210	−4.13 (−5.93 to −2.34)	41.18, p < 0.001
BDI-II	T2	−5.70 (−7.59 to −3.80)	35.56 (1, 105)	<0.001	0.253	−6.06 (−7.90 to −4.22)	

ANCOVA adjusted for the baseline score of the same outcome. Mixed model: random intercept per participant, all available observations, β relative to the T0 group difference.

Clinical categories and relapse

The proportion with poor sleep fell to 75.5% in the intervention group and 92.7% in the control group at 8 weeks (p = 0.017; Table 5). At 8 weeks, 41 of 53 intervention participants (77.4%) had HAM-A scores below 18, compared with 30 of 55 controls (54.5%). Minimal depressive symptoms (BDI-II 0–13) were present in 28 (52.8%) and 10 (18.2%), respectively. Relapse by 8 weeks occurred in 14 of 53 intervention participants (26.4%) and 26 of 55 controls (47.3%) (RR 0.56; absolute risk reduction 20.9%; NNT 4.8; p = 0.041).

Among completers, reductions in PSQI correlated with reductions in HAM-A (r = 0.45) and BDI-II (r = 0.38), and HAM-A change correlated with BDI-II change (r = 0.41; all p < 0.001). Number of sessions attended was not associated with PSQI improvement (Spearman ρ = −0.11; p = 0.44).

Table 5. Clinical categories at 8 weeks and relapse

Outcome	Intervention n/N (%)	Control n/N (%)	p
Poor sleep (PSQI > 5) at T1	49/59 (83.1)	55/56 (98.2)	0.009[c]
Poor sleep (PSQI > 5) at T2	40/53 (75.5)	51/55 (92.7)	0.017[b]
HAM-A < 18 (mild or none)	41/53 (77.4)	30/55 (54.5)	0.022[b]
BDI-II 0–13 (minimal)	28/53 (52.8)	10/55 (18.2)	<0.001[b]
BDI-II 29–63 (severe)	5/53 (9.4)	16/55 (29.1)	0.019[b]
Relapse by 8 weeks	14/53 (26.4)	26/55 (47.3)	0.041[b]

[b] Chi-square test. [c] Fisher's exact test. Relapse RR 0.56, NNT 4.8.

Discussion

In this quasi-experimental study, a structured psychoeducational programme added to treatment as usual was associated with better sleep quality and lower anxiety and depressive symptoms over 8 weeks after detoxification. The benefit appeared by the end of the 4-week programme and was maintained, or slightly larger, at follow-up. Adjusted differences of about 3 PSQI points, 5 HAM-A points and 6 BDI-II points are clinically meaningful, and effects were consistent across ANCOVA, mixed-effects and sensitivity analyses.

The control group also improved, as expected. Sleep, anxiety and depressive symptoms tend to improve naturally in early abstinence [7, 16], and many alcohol-induced symptoms resolve within weeks [17]. The comparison with a concurrent control group is therefore essential: the added improvement in the intervention group goes beyond the natural course of early recovery. Despite this, most participants in both groups still had poor sleep at 8 weeks, consistent with reports that insomnia persists for months after drinking stops [8, 10].

The effect on sleep agrees with evidence that behavioural sleep strategies improve sleep in AUD. Cognitive-behavioural therapy for insomnia improved sleep in recovering patients [34], and a meta-analysis found that insomnia treatments improve sleep in AUD [35]. Our programme was lighter than full CBT-I, with one dedicated sleep session and repeated reinforcement, but it included core components such as sleep hygiene and stimulus control [33, 43]. This low intensity is relevant for

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doi: 10.61705/mqfksc47
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Indian settings where trained therapists are scarce [6, 27].

The larger effects on anxiety and depression are consistent with meta-analytic evidence that brief psychoeducation reduces these symptoms [32] and with the benefit of psychoeducation in other psychiatric disorders [29–31]. Education about the effects of alcohol on mood may reduce self-blame and stigma [36], while relaxation and activity-scheduling skills give patients alternatives to drinking for coping with distress [18, 23]. Changes in sleep, anxiety and depression were moderately correlated, which fits models in which poor sleep and negative affect reinforce each other [9, 19].

Relapse was lower in the intervention group. This finding is exploratory, but it is plausible: insomnia and negative affect predict relapse [10, 13, 17], and relapse-prevention skills were part of the programme [44]. Similar gains have been reported with structured psychosocial interventions in large trials [25, 26] and in Indian primary care [27]. Psychoeducation could complement medication, which remains underused [20, 21, 24].

Strengths and limitations

Strengths include a concurrent control group, three time points, validated instruments, low attrition and analyses that used all available data. The main limitation is the non-randomised design: allocation by [ward/period] may introduce selection bias and unmeasured confounding. Baseline PSQI was higher in the intervention group, which may allow greater regression to the mean, although adjustment for baseline addressed this in part [49]. Blinding of participants was not possible, and BDI-II and PSQI are self-reported. Group sessions provided extra attention that TAU did not, so non-specific effects cannot be excluded. Completers had higher baseline depression scores. The sample was mostly male and from one centre, which limits generalisability to women. Follow-up of 8 weeks is short, relapse was self-reported, and drinking quantity was not measured with tools such as the timeline follow-back.

Implications and future research

Structured psychoeducation is inexpensive, can be delivered by nurses or counsellors and fits into existing inpatient and outpatient de-addiction care. Randomised controlled trials with attention-matched controls, longer follow-up (6–12 months), objective sleep measures, biochemical confirmation of abstinence and more women are needed. Studies should also test which components drive benefit and whether booster sessions sustain it.

Conclusion

Added to treatment as usual, a structured psychoeducational intervention was associated with improved sleep quality and reduced anxiety and depressive symptoms over 8 weeks in patients with alcohol use disorder after detoxification. It is a feasible, low-cost addition to de-addiction care that merits evaluation in randomised trials.

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