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# Changes in Frontal Lobe Functioning in Alcohol-Dependent Patients Abstinent for at Least Three Months: A Prospective Observational Study from a Medical-College De-addiction Centre.

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## Abstract

***Background:*** Alcohol use disorder (AUD) is associated with structural and functional impairment of the frontal lobes, which govern executive function and behavioural control. The extent of frontal dysfunction during sustained abstinence, and the degree of accompanying recovery, remain incompletely characterised in real-world rehabilitation settings. ***Objective:*** To assess frontal-lobe functioning in alcohol-dependent patients maintaining abstinence for at least three months, using neuropsychological and structural neuroimaging measures. ***Methods:*** In this prospective observational study, 140 patients with alcohol dependence syndrome abstinent for at least 90 days were recruited by consecutive sampling at a medical-college de-addiction centre. Frontal-lobe function was assessed with standardised neuropsychological tests and structural change with brain computed tomography (CT). Data were analysed using chi-square tests, Pearson correlation, one-way ANOVA, and logistic and linear regression; a p-value below 0.05 was considered significant. ***Results:*** Participants were predominantly male (92.1%) and married (72.1%), with a mean age of 41.3 ± 9.2 years and a mean alcohol-use duration of 11.8 ± 6.4 years. Executive dysfunction was present in 62.1% and global frontal dysfunction in 62.9%; frontal cortical atrophy was detected in 39.3%. Longer alcohol use correlated with lower executive-function scores ($r = -0.44$, $p < 0.001$) and greater atrophy ($r = 0.38$, $p < 0.001$), whereas longer abstinence correlated with better executive function ($r = 0.41$, $p < 0.001$). Executive scores rose significantly across abstinence-duration groups ($F = 16.8$, $p < 0.001$). Severe executive dysfunction (OR = 3.40) and frontal atrophy were independent predictors of relapse risk. ***Conclusion:*** Chronic alcohol use is associated with substantial frontal-lobe structural and functional impairment, whereas sustained abstinence is associated with measurable neurocognitive recovery. Executive dysfunction and frontal cortical atrophy are key predictors of relapse, supporting the prognostic value of frontal-lobe integrity in AUD.

**Keywords:** Alcohol use disorder; Frontal lobe; Executive function; Abstinence; Neurocognitive recovery; Relapse.



## INTRODUCTION

Alcohol use disorder (AUD) is a chronic, relapsing neuropsychiatric condition characterised by compulsive alcohol intake, impaired control over consumption, and the emergence of negative emotional states during withdrawal. It represents a major global health burden: the harmful use of alcohol accounts for approximately 5.3% of all deaths worldwide and is among the leading preventable causes of neuropsychiatric morbidity [1]. The persistent and relapsing course of AUD reflects complex neurobiological changes affecting reward pathways, executive-control systems, and stress-regulation mechanisms.

Although chronic alcohol exposure produces widespread neurobiological alterations, the frontal lobes are particularly vulnerable because of their central role in executive function, decision-making, emotional regulation, and social cognition. The prefrontal cortex governs cognitive control, inhibitory control, working memory, and adaptive behaviour; its dysfunction manifests as impaired judgement, heightened impulsivity, and poor planning, which in turn reinforce maladaptive drinking behaviours [2,3].

Neuroimaging studies have consistently demonstrated structural brain changes associated with prolonged alcohol use, including reduced cortical thickness, loss of grey-matter volume, ventricular enlargement, and white-matter atrophy. These changes preferentially involve frontal cortical regions such as the dorsolateral prefrontal cortex, orbitofrontal cortex, and

anterior cingulate cortex, and correlate closely with executive and emotional-regulation deficits [4,5]. Functional imaging further shows impaired prefrontal engagement during tasks of inhibitory control, reward evaluation, and decision-making, with disruption of fronto-striatal and fronto-limbic connectivity contributing to compulsive drinking and relapse [2,7].

Neuropsychological investigations corroborate these findings, documenting deficits in working memory, attention, cognitive flexibility, planning, and problem-solving among alcohol-dependent individuals [3,4]. Importantly, such impairments frequently persist beyond detoxification and early abstinence, and their severity predicts treatment adherence, relapse vulnerability, and rehabilitation outcomes [11].

Despite these adverse effects, evidence indicates that neurobiological recovery may begin with sustained abstinence. Longitudinal studies have reported partial reversal of alcohol-related atrophy and gradual improvement in cognitive performance, reflecting neuroplastic processes such as synaptic remodelling and restoration of cerebral perfusion [14]. However, recovery trajectories are heterogeneous and are influenced by the duration and severity of alcohol use, age, nutritional status, and psychiatric comorbidity. The early abstinence phase is a dynamic period during which marked structural and functional changes can occur, yet restoration of higher-order executive function is often incomplete or unpredictable [7,14].

Because the integrity of frontal networks has emerged as a key predictor of relapse and recovery [11], understanding how frontal-lobe function changes during sustained abstinence is of considerable clinical importance. Nonetheless, few studies have examined these changes in real-world rehabilitation settings. The present study was therefore undertaken to assess frontal-lobe functioning in alcohol-dependent patients who had maintained abstinence for at least three months. By combining neuropsychological assessment with structural neuroimaging, the study aims to characterise the neurocognitive recovery associated with sustained abstinence and to inform rehabilitation strategies for AUD.

## MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Psychiatry and the affiliated de-addiction centre of Amaltas Institute of Medical Sciences, Dewas, Madhya Pradesh, India. Assessments and data collection were carried out in the inpatient and outpatient rehabilitation units over an 18-month period following approval by the Institutional Ethics Committee.

**Study population and sampling.** The study population comprised patients with alcohol dependence syndrome undergoing treatment and rehabilitation at the de-addiction centre who had maintained alcohol abstinence for at least 90 days. Consecutive sampling was used, with all eligible patients presenting during the study period enrolled until the target sample size was reached.

**Sample size.** The sample size was estimated using the formula $n = Z^2pq/d^2$, where $Z = 1.96$ (95% confidence level), $p$ = expected prevalence of frontal cognitive impairment (taken as 50%, the most conservative estimate), $q = 1 - p$, and $d$ = allowable error (10%). This yielded a minimum of 96 participants. To enhance statistical power, permit subgroup analysis, and accommodate non-response or incomplete data, the sample was increased to 140 participants.
Selection criteria. Patients aged 20–60 years with a diagnosis of alcohol dependence syndrome who had maintained abstinence for at least 90 days, were admitted or attending follow-up at the centre, and provided written informed consent were included. Patients were excluded if they had major neurological disorders (e.g., stroke, epilepsy, traumatic brain injury), major psychiatric disorders unrelated to alcohol use, or severe cognitive impairment precluding assessment, or if they declined consent.

**Data collection.** After written informed consent, a structured clinical proforma was used to record sociodemographic details (age, sex, occupation, marital status, socioeconomic status, and education) and a detailed alcohol-use history (age at initiation, duration of dependence, quantity and pattern of use, duration of abstinence, treatment history, and previous relapses). A psychiatric evaluation was performed to identify comorbidities and to assess mental status.

**Assessment of frontal-lobe function.** Frontal-lobe function was evaluated using standardised, validated neuropsychological instruments administered by trained clinicians. The domains assessed were executive functioning, attention and concentration, working memory, cognitive flexibility, planning and decision-making, and impulse control and behavioural regulation.

**Neuroimaging assessment.** Structural assessment of the frontal lobes was performed using computed tomography (CT) of the brain to detect cortical atrophy, ventricular enlargement, and focal lesions associated with chronic alcohol use.

**Ethical considerations.** The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants. Confidentiality of patient information was maintained, and participants were assured that declining to participate would not affect their treatment.

**Statistical analysis.** Data were entered into a standardised database and analysed using appropriate statistical software. Descriptive statistics (mean, standard deviation, frequency, and percentage) summarised sociodemographic and clinical variables. The chi-square test assessed associations between categorical variables; Pearson correlation examined relationships between continuous variables; and one-way ANOVA with post-hoc Tukey testing compared executive-function scores across abstinence-duration groups. Multivariate logistic regression identified predictors of relapse risk, and multiple linear regression identified predictors of executive and global frontal function. A p-value below 0.05 was considered statistically significant.

## RESULTS

A total of 140 patients with alcohol dependence syndrome who had maintained abstinence for at least three months and met the eligibility criteria were analysed. The findings are presented under sociodemographic characteristics, clinical characteristics, neuropsychological assessment, neuroimaging, association analysis, correlation analysis, overall clinical outcome, and predictors of outcome.

### Sociodemographic characteristics

**Table 1. Age distribution of study participants**

Mean age = 41.3 ± 9.2 years

| Age group (years) | Frequency | Percentage |
|---|---|---|
| 20–30 | 18 | 12.9 |
| 31–45 | 65 | 46.4 |
| 46–60 | 57 | 40.7 |
| **Total** | **140** | **100** |

The mean age was 41.3 ± 9.2 years, and most participants (46.4%) were aged 31–45 years, followed by those aged 46–60 years (40.7%), indicating that alcohol dependence with sustained abstinence was most frequent among middle-aged individuals.

**Table 2. Gender distribution of study participants**

| Gender | Frequency | Percentage |
|---|---|---|
| Male | 129 | 92.1 |
| Female | 11 | 7.9 |
| **Total** | **140** | **100** |

The sample was predominantly male (92.1%), with females comprising only 7.9%, consistent with the epidemiology of alcohol dependence in the region.

**Table 3. Duration of alcohol use**

Mean duration = 11.8 ± 6.4 years

| Duration (years) | Frequency | Percentage |
|---|---|---|
| <5 | 19 | 13.6 |
| 5–10 | 44 | 31.4 |
| 11–15 | 38 | 27.1 |
| >15 | 39 | 27.9 |
| **Total** | **140** | **100** |

The mean duration of alcohol use was 11.8 ± 6.4 years; 31.4% of participants had used alcohol for 5–10 years, 27.9% for more than 15 years, and 27.1% for 11–15 years, while only 13.6% had a history shorter than 5 years.

**Table 4. Duration of abstinence**

Mean abstinence = 4.7 ± 1.9 months

| Duration (months) | Frequency | Percentage |
|---|---|---|
| 3–4 | 56 | 40.0 |
| 5–6 | 48 | 34.3 |
| >6 | 36 | 25.7 |
| **Total** | **140** | **100** |

The mean duration of abstinence was 4.7 ± 1.9 months; 40.0% of participants had been abstinent for 3–4 months, 34.3% for 5–6 months, and 25.7% for more than 6 months.

**Neuropsychological assessment of frontal-lobe function**

**Table 5. Distribution of executive dysfunction**

| Severity | Frequency | Percentage |
|---|---|---|
| None | 53 | 37.9 |
| Mild | 48 | 34.3 |
| Moderate | 29 | 20.7 |
| Severe | 10 | 7.1 |
| **Total** | **140** | **100** |

Executive dysfunction of some degree was present in 62.1% of participants: mild in 34.3%, moderate in 20.7%, and severe in 7.1%, while 37.9% showed no executive impairment.

**Table 6. Working-memory impairment**

| Category | Frequency | Percentage |
|---|---|---|
| Impaired | 76 | 54.3 |
| Normal | 64 | 45.7 |
| **Total** | **140** | **100** |

Working-memory impairment was present in 54.3% of participants, while 45.7% performed within the normal range.

**Table 7. Attention deficit**

| Category | Frequency | Percentage |
|---|---|---|
| Impaired | 68 | 48.6 |
| Normal | 72 | 51.4 |
| **Total** | **140** | **100** |

Impaired attention was observed in 48.6% of participants, and normal attention in 51.4%.

**Table 8. Cognitive flexibility**

| Category | Frequency | Percentage |
|---|---|---|
| Impaired | 81 | 57.9 |
| Normal | 59 | 42.1 |
| **Total** | **140** | **100** |

Cognitive-flexibility impairment was found in 57.9% of participants, while 42.1% showed normal cognitive flexibility.

**Table 9. Planning ability**

| Category | Frequency | Percentage |
|---|---|---|
| Impaired | 73 | 52.1 |
| Normal | 67 | 47.9 |
| **Total** | **140** | **100** |

Impaired planning ability was present in 52.1% of participants, with 47.9% performing normally.

**Table 10. Global frontal dysfunction**

| Category | Frequency | Percentage |
|---|---|---|
| Present | 88 | 62.9 |
| Absent | 52 | 37.1 |
| **Total** | **140** | **100** |

Global frontal dysfunction was identified in 62.9% of participants, while 37.1% showed no overall frontal-lobe impairment.

**Table 11. Frontal cortical atrophy**

| Grade | Frequency | Percentage |
|---|---|---|
| None | 85 | 60.7 |
| Mild | 38 | 27.1 |
| Moderate | 13 | 9.3 |
| Severe | 4 | 2.9 |
| **Total** | **140** | **100** |

Frontal cortical atrophy was present in 39.3% of participants: mild in 27.1%, moderate in 9.3%, and severe in 2.9%, while 60.7% showed no atrophy.

## DISCUSSION

This prospective observational study examined frontal-lobe functioning in 140 alcohol-dependent patients who had maintained abstinence for at least three months, integrating neuropsychological assessment with structural neuroimaging. The findings demonstrate a high burden of frontal dysfunction alongside measurable structural change, together with evidence of abstinence-related recovery.

The mean duration of alcohol use was 11.8 ± 6.4 years, and longer use correlated significantly with frontal cortical atrophy ($r = 0.38$) and with lower executive-function scores ($r = −0.44$) and greater global cognitive impairment ($r = 0.42$). These observations are consistent with reports that chronic alcohol exposure produces cumulative, dose-related grey-matter loss, particularly in prefrontal and anterior cingulate regions [5,8]. Pfefferbaum and colleagues showed that grey- and white-matter loss accelerates with age in chronic alcoholics and that older individuals sustain greater frontal volume deficits [5,6]. Treatment-naive and psychosocially preserved samples show comparable prefrontal grey-matter reductions related to lifetime alcohol intake [9,10], underscoring that structural injury is not confined to severely affected patients.

Neuropsychological testing revealed executive dysfunction in 62.1% of participants and global frontal dysfunction in 62.9%, with additional deficits in working memory (54.3%), cognitive flexibility (57.9%), planning (52.1%), and attention (48.6%). This pattern accords with Oscar-Berman and Marinković and with Moselhy and colleagues, who described impaired executive function, memory, and emotional regulation arising from disruption of interconnected frontal and limbic systems [3,4]. Sullivan and Pfefferbaum similarly attributed these deficits to degradation of frontocerebellar and fronto-limbic circuitry [7].

Structural imaging identified frontal cortical atrophy in 39.3% of participants, and atrophy was significantly associated with both age ($p = 0.02$) and executive dysfunction ($p = 0.01$; $r = −0.31$). The coupling of structural and functional deficits is supported by early quantitative imaging work [8] and by studies linking smaller frontal grey-matter volumes to poorer clinical outcome and shorter time to relapse [11,12].

Sustained abstinence was associated with progressive improvement: executive function ($r = 0.41$), working memory ($r = 0.36$), attention ($r = 0.29$), and global frontal function ($r = 0.39$) all correlated positively with abstinence duration, and executive scores increased across abstinence-duration groups (ANOVA $F = 16.8$, $p < 0.001$). These findings mirror longitudinal evidence of non-linear grey-matter recovery during abstinence [14]. Nevertheless, overall cognitive impairment persisted in 68.6% of participants, indicating that recovery during early abstinence is incomplete and heterogeneous.

Multivariate analysis identified duration of alcohol use, executive dysfunction, and frontal atrophy as independent predictors of relapse risk, with severe executive dysfunction the strongest predictor (OR = 3.40). This is concordant with work showing that brain morphology at treatment entry and during early abstinence predicts relapse propensity [12,13], and with the broader model in which frontal structural integrity governs the clinical course of AUD [2]. Strengths of this study include the integrated neuropsychological–imaging design and an adequate sample size; principal limitations are the use of CT rather than volumetric MRI and the absence of longitudinal follow-up, both of which should be addressed in future work.

## CONCLUSION

This study demonstrates that alcohol dependence is associated with substantial impairment of frontal-lobe function that persists into sustained abstinence. A high proportion of participants showed deficits in executive function, working memory, attention, cognitive flexibility, and planning, and a considerable minority showed frontal cortical atrophy on imaging, reflecting the chronic neurotoxic effects of alcohol.

Longer alcohol use was associated with greater cognitive impairment and structural damage, whereas longer abstinence was associated with progressive recovery of frontal-lobe function, highlighting the benefit of sustained sobriety. Executive dysfunction and frontal cortical atrophy emerged as significant predictors of relapse risk, underscoring the clinical value of assessing frontal-lobe integrity in AUD.

Early identification of frontal dysfunction and targeted cognitive rehabilitation may improve treatment response and reduce relapse. Overall, the findings support early intervention, sustained abstinence, and multidimensional neurocognitive assessment in the management of alcohol use disorder, and argue for long-term follow-up and structured rehabilitation to promote functional recovery.

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