https://doi.org/10.22502/jlmc.v14i1.584

Original Research Article

Risk Factors for Delirium Tremens Among Hospitalized Patients with Alcohol Withdrawal Syndrome: A Case-control Study

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ABSTRACT:

Introduction: Delirium tremens (DT) is the most severe complication of alcohol withdrawal and can be fatal if not recognized early. Identifying at-risk patients is challenging, especially in those presenting with seizures who are often too unwell to give a clear history. **Methods**: We conducted a retrospective case-control study at the Department of Psychiatry, Lumbini Medical College and Teaching Hospital. We included 124 patients—62 cases who developed DT and 62 age- and sex-matched controls with uncomplicated withdrawal. Data on demographics, clinical history, laboratory parameters, and vital signs were extracted from medical records. Statistical analysis was performed using PSPP 2.1.1. **Results**: A history of complicated withdrawal was strongly associated with DT (OR = 12.27, 95% CI: 2.72–55.35, p = 0.001). Patients with DT had significantly higher MCV (95.45 vs. 91.92 fL, p = 0.004), SGOT (200.61 vs. 102.50 U/L, p = 0.002), systolic BP (138.00 vs. 128.50 mmHg, p < 0.001), and diastolic BP (84.00 vs. 78.00 mmHg, p = 0.001). Platelet count (167,590.16 vs. 179,800.00 $\times 10^3/\mu L$, p = 0.01) and potassium (3.78 vs. 3.93 mEq/L, p = 0.049) were significantly lower among cases. No significant differences were observed for hemoglobin, bilirubin, SGPT, ALP, sodium, creatinine, urea, or pulse rate. **Conclusions**: A history of complicated withdrawal, elevated MCV and SGOT, higher blood pressure, lower platelets, and lower potassium were significantly associated with DT. These readily available parameters can help clinicians identify high-risk patients early and initiate timely preventive measures.

Keywords: Alcohol withdrawal; Case-control study; Delirium tremens; Risk factors

INTRODUCTION

Alcohol withdrawal syndrome (AWS) covers a wide range—from mild tremors to life-threatening autonomic instability.[1,2] The most severe end of that spectrum is delirium tremens (DT), which usually develops 2–3 days after alcohol cessation.[2,3] Patients become acutely confused, agitated, and hemodynamically unstable, with hallucinations, seizures, and dangerous swings in vital signs.[2,4] Left untreated, mortality used to hit 20%, but with benzodiazepines and supportive care, that figure now hovers around 1%.[5] Still, that 1% represents significant mortality or morbidity in terms of aspiration, injuries, and cardiac events.

Only a small fraction of withdrawing patients develop DT, but identifying them early is crucial.[2,6] Delayed recognition leads to complications like hyperthermia, arrhythmias, and prolonged ICU stays.[7] A retrospective study by Lukan et al. found that DT patients stayed six days longer in hospital and five extra days in the ICU.[4] Early prediction is tricky—especially in patients presenting with alcohol withdrawal seizures, who are often sedated, postictal, or too unwell to give a clear history.

Several studies have looked for predictors of

Submitted: *06 July 2026*
Accepted: *09 August 2026*
Published: *20 August 2026*

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How to cite this article:
Shakya RP, Maskey S, Maharjan S. Risk Factors for Delirium Tremens Among Hospitalized Patients with Alcohol Withdrawal Syndrome: A Case-control Study. J Lumbini Med Coll.2026;14(1):41-46. DOI: 10.22502/jlmc.v14i1.584. Epub: 2026 August 20.

DT, with variable results. Some found tachycardia, others found hyponatremia, hypokalemia, or thrombocytopenia.[5-7] But most of these studies focused on general AWS populations, not specifically on those who present with seizures—a group that is harder to assess and already at higher baseline risk.

Given this gap, we designed a case-control study to identify clinical and laboratory factors most strongly associated with DT development among hospitalized patients with diagnosis of alcohol dependence syndrome. Our goal is to help clinicians flag high-risk patients early, start timely treatment, and ultimately reduce both patient harm and healthcare costs.

METHODS

We conducted a retrospective case-control study at the Department of Psychiatry, Lumbini Medical College and Teaching Hospital, Palpa, Nepal. The study period was from April 11, 2026 to May 26, 2026. During this period, we reviewed the medical records of all patients admitted to our center in the past two years with a diagnosis of alcohol dependence syndrome according to ICD-11 criteria.8 Ethical approval was sought before starting the study from Institutional Review Committee (IRC) of Lumbini Medical College – Teaching Hospital (LMCTH): Reference no – 54/26/V1R0.

Cases were defined as patients who developed delirium tremens during their hospitalization. Controls were patients with uncomplicated alcohol withdrawal who did not develop DT. We included patients aged 18 to 70 years who had no evidence of delirium at the time of admission. We excluded those with concurrent substance use (other than nicotine), psychiatric comorbidities, or pre-existing medical or neurological conditions that could independently cause delirium. This was to ensure that any delirium we observed was truly due to alcohol withdrawal and not some other underlying pathology.

We enrolled 62 cases—this represented all patients who developed DT during the study period. For controls, we selected 62 patients with uncomplicated alcohol withdrawal from the same time period who had not been previously included. Controls were frequency-matched to cases by age group (18–28, 29–39, 40–50, >50 years) and sex. This gave us a final sample of 124 participants—62 cases and 62 controls.

The sample size calculation was based on pilot data, which showed that the proportion of controls with a history of complicated withdrawal (p_0) was 3.3%. Assuming an odds ratio of 11.0 for the primary exposure, the proportion of cases exposed (p_1) was calculated as 27.3%. With a 1:1 case-to-control ratio, a minimum of 27 cases and 27 controls were required to achieve 80% power at a 5% significance level. Our final sample of 62 cases and 62 controls therefore exceeded the minimum requirement. The sample size formula used was: $n = [Z_{\alpha/2}\sqrt{(2\bar{p}(1-\bar{p}))} + Z_{\beta}\sqrt{(p_0(1-p_0) + p_1(1-p_1))}]^2 / (p_1 - p_0)^2$ where $p_0 = 0.033$ (exposure in controls), $p_1 = 0.273$ (exposure in cases), $\bar{p} = (p_0 + p_1)/2 = 0.153$, $Z_{\alpha/2} = 1.96$ at 95% confidence level, and $Z_{\beta} = 0.84$ for 80% power.

We extracted data from medical records using a structured proforma. This included demographic information, clinical parameters, laboratory findings, and physiological measurements. All data were collected retrospectively from the patients' hospital charts.

Amount of alcohol consumed was recorded in units, where one unit equals 10 ml of pure ethanol. However, most patients in our study consumed locally brewed, non-standardized alcoholic beverages—primarily Local Raksi (distilled) and Jand (non-distilled). A nationwide survey of 284 homebrewed beverage samples in Nepal found that distilled spirits (Local Raksi) have a median ethanol concentration of 14% (IQR: 10–19%), ranging from 3% to 40%, while non-distilled beverages (Jand, Chhyang, Tongba) have a median of 5.20% (IQR: 3.60–9.80%), ranging from 1% to 18.90%.[9] More recent gas chromatography analysis of four homebrewed samples found ethanol concentrations ranging from 11.47% to 49.22%.[10] Given this wide variability, the amount of alcohol consumed was estimated based on patient self-report using a standard 300 ml stainless steel glass—a commonly used measure in local practice. However, we acknowledge that the lack of standardized alcohol concentration in homebrewed beverages introduces measurement uncertainty.

All statistical analyses were performed using PSPP 2.1.1 software. Continuous variables were compared using the independent t-test for normally distributed data or the Mann-Whitney U test for non-normal distributions. Categorical variables were analyzed using the Chi-square test or Fisher's exact test, as appropriate. A two-sided p-value < 0.05 was considered statistically significant. To quantify the magnitude of group differences, we calculated effect sizes—specifically, Cohen's d for parametric tests.

RESULTS

A total of 124 patients were included in this study—62 who developed delirium tremens (cases) and 62 who had uncomplicated alcohol withdrawal (controls). The two groups were successfully matched by age group and sex distribution, with no statistically significant differences in these baseline characteristics.

Table 1 summarizes the demographic characteristics of the study population. The difference in mean age between cases and controls was not statistically significant. Most patients in both groups were male reflecting the higher prevalence of alcohol use disorder among men. Marital status also did not differ significantly between the two groups.

Table 2 presents the clinical parameters and their associated odds ratios. A previous history of complicated alcohol withdrawal was significantly more common among patients who developed DT. The odds of developing DT were approximately 12 times higher among patients with a previous history of complicated withdrawal compared with those without such a history. This finding indicates a strong and statistically significant association between prior complicated withdrawal and the development of DT.

In contrast, no statistically significant associations were observed between DT and the type of alcohol consumed, amount of alcohol consumed, or duration of alcohol consumption. The duration since the last alcohol intake could not be reliably estimated using an odds ratio because there were no controls who had their last alcohol intake more than 24 hours before presentation.

The comparison of biochemical parameters between cases and controls is presented in **Table 3**. Missing values were imputed using the mean of available values for each variable, resulting in complete data for all 62 cases and 62 controls. For Case #35, all missing laboratory values were imputed using the case means.

Among the biochemical parameters, patients with DT had significantly higher mean corpuscular volume, serum glutamic oxaloacetic transaminase, systolic blood pressure, and diastolic blood pressure compared with controls. In contrast, platelet count and serum potassium level were significantly lower among cases. No statistically significant differences were observed between cases and controls for hemoglobin, total serum bilirubin, serum glutamic pyruvic transaminase, alkaline phosphatase, sodium, creatinine, urea, or pulse rate.

Table 1. *Demographic characteristics of the study population (N=124)*

Variable	Category	Control (n=62)	Case (n=62)	Statistics
Age (years)	Mean ± SD	41.21 ± 10.80	44.08 ± 9.71	t=1.28, p=0.20
Age group	18–28	4 (6.45%)	4 (6.45%)	p=0.22*
	29–39	15 (24.19%)	15 (24.19%)	
	40–50	27 (43.55%)	27 (43.55%)	
	>50	16 (25.81%)	16 (25.81%)	
Sex	Male	58 (93.55%)	58 (93.55%)	p=1.00*
	Female	4 (6.45%)	4 (6.45%)	
Marital Status	Married	57 (91.94%)	61 (98.39%)	p=0.17*
	Single	5 (8.06%)	1 (1.61%)	

**Fisher's exact test*

Table 2. *Clinical Parameters of the Study Population (N=124)*

Variable		Control n (%)	Case n (%)	OR (95% CI)	Statistics
Past complicated withdrawal	Yes	2 (3.23%)	18 (29.03%)	12.27 (2.72–55.35)	0.001*
	No	60 (96.77%)	44 (70.97%)	Reference	
Type of alcohol	Commercial	5 (8.06%)	2 (3.23%)	2.63 (0.49–14.04)	0.42*
	Home made	57 (91.94%)	60 (96.77%)	Reference	
Amount (>15 units)	Yes	26 (41.94%)	26 (41.94%)	1.00 (0.49–2.04)	χ^2=0, p=1
	No	36 (58.06%)	36 (58.06%)	Reference	
Duration (>20 years)	Yes	18 (29.03%)	28 (45.16%)	2.01 (0.95–4.26)	χ^2=3.43, p=0.06
	No	44 (70.97%)	34 (54.84%)	Reference	
Last intake >24 hrs	Yes	0 (0.00%)	2 (3.23%)	—	
	No	62 (100.00%)	60 (96.77%)	—	

**Fisher's exact test*

Table 3*: Biochemical and Physiological Parameters of the Study Population (N=124)*

Variables	Cases (Mean ± SD)	Controls (Mean ± SD)	Statistics	Effect Size
Hemoglobin (g/dL)	13.12 ± 1.91	13.35 ± 3.10	t=-0.78, p=0.22	Small
MCV (fL)	95.45 ± 10.86	91.92 ± 7.10	t=2.94, p=0.004	Medium
Platelet (×10³/µL)	167,590.16 ± 43,160.70	179,800.00 ± 49,200.00	t=-2.56, p=0.01	Small
Total bilirubin (mg/dL)	1.43 ± 0.67	1.40 ± 0.55	U=1,892.50, p=0.80	Negligible
SGPT (U/L)	96.72 ± 55.01	88.50 ± 65.00	U=1,723.50, p=0.21	Small
SGOT (U/L)	200.61 ± 136.97	102.50 ± 72.00	t=3.17, p=0.002	Medium
ALP (U/L)	116.46 ± 53.31	100.50 ± 38.00	U=1,750, p=0.15	Small
Sodium (mEq/L)	140.56 ± 5.08	140.40 ± 3.50	t=0.31, p=0.76	Negligible
Potassium (mEq/L)	3.78 ± 0.48	3.93 ± 0.38	t=-1.99, 0.049	Small
Creatinine (mg/dL)	0.97 ± 0.36	0.96 ± 0.28	t=0.61, p=0.54	Negligible
Urea (mg/dL)	28.37 ± 7.63	26.50 ± 7.00	t=1.57, p=0.12	Small
Pulse rate (/min)	90.55 ± 9.75	88.50 ± 10.00	t=-0.84, p=0.40	Negligible
Systolic BP (mmHg)	138.00 ± 8.96	128.50 ± 9.40	t=4.15, p<0.001	Medium
Diastolic BP (mmHg)	84.00 ± 7.62	78.00 ± 7.20	t=3.56, p=0.001	Medium

DISCUSSION

This case-control study identified several factors significantly associated with the development of delirium tremens among hospitalized patients with alcohol withdrawal syndrome. The strongest predictor was a history of complicated alcohol withdrawal, with nearly 12-fold higher odds of developing DT. We also found that elevated MCV, elevated SGOT, lower platelet count, lower serum potassium, and higher systolic and diastolic blood pressure were significantly associated with DT. These findings align with much of the existing literature and have practical implications for clinical practice.

A previous history of complicated alcohol withdrawal emerged as the most robust predictor in our study (OR = 12.27, 95% CI: 2.72–55.35, p = 0.001). This is consistent with a large body of evidence. A systematic review and meta-analysis by Goodson et al. identified prior DT or withdrawal seizures as one of the most important predictors of subsequent complicated withdrawal.[11] Similarly, Kim et al. found that a history of DT was a strong predictor among patients presenting with alcohol withdrawal seizures.[2] The biological plausibility is straightforward—patients who have experienced severe withdrawal in the past are likely to have undergone kindling, a process where repeated withdrawal episodes increase the severity of subsequent episodes.[12,13] This finding underscores the importance of taking a thorough history of prior withdrawal complications at the time of admission.

We found significantly lower platelet counts in the DT group compared with controls (167,590.16 ± 43,160.70 vs. 179,800.00 ± 49,200.00 ×10³/µL, p = 0.01). This is consistent with the findings of Eyer et al., who studied 827 patients with AWS and found that lower platelet count was associated with DT.[14] Berggren et al. similarly reported that thrombocytopenia occurring early in withdrawal predicted progression to DT or seizures.[15] The mechanism is not entirely clear, but chronic heavy alcohol use suppresses bone marrow function, particularly thrombopoiesis. Platelet levels may therefore reflect the severity of long-term alcohol-related bone marrow suppression, which in turn correlates with overall disease severity and withdrawal risk.

Patients who developed DT had significantly lower serum potassium levels compared with controls (3.78 ± 0.48 vs. 3.93 ± 0.38 mEq/L, p = 0.049). This finding aligns with the studies by Eyer et al. and Berggren et al., both of which identified hypokalemia as a predictor of severe withdrawal.[14,15] Wood et al. also found that lower potassium levels were associated with DT in their cohort of 637 patients.[16] Hypokalemia during withdrawal may result from several mechanisms—increased sympathetic activity, poor oral intake, gastrointestinal losses from vomiting or diarrhea, or the effects of alcohol-induced malnutrition. Regardless of the underlying mechanism, our finding suggests that monitoring serum potassium levels could help clinicians identify patients at higher risk for DT.

Elevated SGOT was significantly associated with DT in our study (200.61 ± 136.97 vs. 102.50 ± 72.00 U/L, p = 0.002). This is consistent with the work of Wood et al., who found that higher AST and higher GGT were associated with DT.[16] Liver enzyme elevation reflects hepatocellular injury, which is common in chronic heavy drinkers. The degree of liver dysfunction may serve as a proxy for the overall severity of alcohol-related organ damage, which in turn predicts withdrawal severity.

We also found higher MCV in the DT group (95.45 ± 10.86 vs. 91.92 ± 7.10 fL, p = 0.003). MCV is a well-established marker of cumulative, chronic heavy alcohol exposure.[12] Patients with elevated MCV have likely been drinking heavily for a longer period, which may explain their higher risk of DT. This is consistent with the idea that more severe, long-term alcohol use leads to more severe withdrawal syndromes. The combination of elevated MCV and elevated SGOT suggests that patients who develop DT have more advanced alcohol-related liver disease and longer cumulative exposure.

An interesting finding was the significantly higher systolic and diastolic blood pressure in the DT group (systolic: 138.00 ± 8.96 vs. 128.50 ± 9.40 mmHg, p < 0.001; diastolic: 84.00 ± 7.62 vs. 78.00 ± 7.20 mmHg, p = 0.001). Wood et al. similarly found that systolic blood pressure >150 mmHg was associated with DT.[16] The elevated blood pressure in our cases likely reflects the increased sympathetic nervous system activity that characterizes severe alcohol withdrawal.[13] However, we did not find a significant difference in pulse rate between the two groups. This was somewhat unexpected, as tachycardia is a hallmark of autonomic hyperactivity during withdrawal. One possible explanation is that some patients had already received benzodiazepines before their pulse was recorded. Timing of measurement, hydration status, and individual autonomic variability may also have played a role.

We did not find statistically significant associations between DT and the type of alcohol consumed, amount of alcohol consumed, or duration of alcohol consumption. This contrasts with some earlier studies. Monte et al. found that higher amount of alcohol consumption was associated with DT,[17] and Wright et al. identified heavier drinking as a risk factor.[1] The lack of association in our study may be due to several reasons. First, self-reported alcohol consumption is notoriously unreliable, and patients may underreport or overreport their intake. Second, our sample size, though adequate for detecting large effects, may have been insufficient to detect smaller differences in drinking patterns. Third, our population was relatively homogenous—most patients were long-term local alcohol users—which may have limited our ability to detect differences in drinking patterns.

Our findings have practical value for clinicians. Patients with a history of complicated alcohol withdrawal should be monitored closely and treated with adequate doses of benzodiazepines.[2,6] The presence of thrombocytopenia, hypokalemia, elevated MCV or SGOT, and raised blood pressure should raise suspicion for impending DT. Such patients warrant cautious and aggressive management to prevent progression to full-blown delirium. The ASAM clinical practice guideline on alcohol withdrawal management recommends that patients with risk factors for severe withdrawal receive higher initial doses of benzodiazepines and closer monitoring.[12]

Early identification of high-risk patients can reduce complications, shorten hospital stays, and lower healthcare costs. Lukan et al. found that patients who developed DTs had six days longer hospital stays and five extra days in the intensive care unit.[4] By identifying these patients early, we can potentially reduce the morbidity and economic burden associated with DT.

Limitations

This study has several limitations. First, it was retrospective, so we relied on medical records for data quality and completeness. Despite imputation of missing values for some variables, the accuracy of the data depends on the quality of documentation in the original charts. Second, our sample size, though adequate for detecting large effects, may have been insufficient to detect smaller differences in some parameters. Third, the study was conducted at a single tertiary care center, which may limit the generalizability of our findings to other settings or populations. Fourth, we could not adjust for all potential confounders, such as nutritional status, concurrent infections, or the timing and dosage of benzodiazepine administration. Fifth, the imputation of missing values, while necessary for complete-case analysis, introduces some degree of uncertainty into our estimates. Sixth, the amount of alcohol consumed was based on patient self-report using a standard 300 ml glass, but the actual ethanol concentration of homebrewed beverages varies widely—from 3% to 40% for distilled spirits (Local

Raksi) and 1% to 18.90% for non-distilled varieties (Jand, Chhyang, Tongba). This variability may have affected our ability to detect associations between alcohol quantity and DT. Finally, our findings are based on a Nepalese population, and the patterns of alcohol use and withdrawal may differ from those in other countries.

CONCLUSIONS

In conclusion, a history of complicated withdrawal, elevated MCV, elevated SGOT, lower platelet count, lower potassium, and higher blood pressure were significantly associated with the development of delirium tremens in patients with alcohol withdrawal syndrome. These parameters can help clinicians identify high-risk patients early and initiate appropriate preventive measures. Future prospective studies with larger sample sizes are needed to validate these findings and develop a clinical prediction rule for DT in this population.

Funding: None

Competing Interesting: None

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