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Case Series

Postural orthostatic tachycardia syndrome in patients with alcohol use disorder: a case series

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ABSTRACT

Postural orthostatic tachycardia syndrome (POTS) is an under-recognized complication of chronic alcohol use and withdrawal. Autonomic dysfunction secondary to alcohol-related neuropathy may manifest as POTS during or after the deaddiction process. This case series describes eight patients who developed POTS while undergoing detoxification and deaddiction treatment. We retrospectively reviewed medical records of four patients admitted to Dr. B. C. Roy Hospital for alcohol deaddiction during 2024-2025 who presented with POTS symptoms during their treatment. All four patients (4 males; mean age 34.25 years) with chronic alcohol use disorder (mean duration 12.4 years) developed orthostatic intolerance symptoms during detoxification or early recovery phase. Tilt table testing or active stand tests confirmed inappropriate tachycardia (≥30 bpm increase) upon postural change. Symptoms significantly impacted rehabilitation outcomes, requiring integrated management approaches. POTS represents an important autonomic complication in alcohol deaddiction patients that requires systematic evaluation and may impact recovery outcomes. Recognition and management of POTS should be integrated into comprehensive alcohol rehabilitation programs.

Keywords: POTS, Alcohol deaddiction, Autonomic dysfunction, Alcoholic neuropathy, Orthostatic intolerance

INTRODUCTION

Alcohol use disorder (AUD) continues to pose a serious challenge to global public health, with its consequences rippling across millions of individuals and their families. Among its many physiological effects, prolonged alcohol consumption significantly disrupts the autonomic nervous system. Although the immediate symptoms of alcohol withdrawal are fairly well understood, how autonomic dysfunction evolves and persists into the recovery period remains an area that warrants deeper investigation.[1]

Postural orthostatic tachycardia syndrome (POTS) is defined by persistent symptoms of orthostatic intolerance alongside an abnormally large rise in heart rate — at least 30 bpm in adults — occurring within ten minutes of assuming an upright position, without any accompanying drop in blood pressure. Long-term alcohol use can damage the autonomic nervous system through several overlapping pathways, including direct toxic effects on nerve tissue, depletion of thiamine, increased oxidative stress, and impaired mitochondrial function. As a result of this cumulative autonomic damage, POTS may emerge as a recognizable clinical manifestation during the detoxification process and the early stages of recovery.[2,3]

This case series presents four patients admitted to B. C. Roy Hospital for alcohol deaddiction during 2024-2025 who presented with POTS as a complication of chronic alcohol.

Understanding this association is crucial for optimizing rehabilitation outcomes and providing comprehensive care during the recovery process.

CASE SERIES

This retrospective study analysed medical records of patients referred to Dr. B. C. Roy Hospital for alcohol deaddiction services. Individuals had no documented history of POTS or related autonomic complaints prior to referral. Patients with a provisional diagnosis of alcohol use disorder underwent diagnostic evaluation by an addiction specialist using a modified version of the structured clinical interview for DSM-IV to distinguish alcohol dependence from alcohol abuse and to determine the need for treatment. Baseline psychological status was assessed using the Beck depression inventory and Beck anxiety inventory. All four patients underwent medically supervised alcohol detoxification using standard benzodiazepine-based withdrawal protocols tailored to clinical severity, primarily employing chlordiazepoxide or lorazepam tapering regimens to prevent withdrawal complications. Detoxification was completed safely in all cases, with no episodes of seizures, delirium tremens, or significant medical instability. Comprehensive supportive care was provided throughout admission, including close monitoring of vital signs, hydration status, and withdrawal symptoms.

Patients evaluated for suspected POTS underwent a comprehensive clinical assessment, including a detailed medical history focused on cardiovascular and non-cardiovascular symptoms, followed by a physical examination. Orthostatic heart rate and blood pressure responses were assessed using an active standing protocol. POTS was diagnosed based on the presence of typical orthostatic symptoms and a sustained increase in heart rate exceeding 30 beats per minute within 10 minutes of standing, in the absence of orthostatic hypotension. Blood pressure was recorded at the beginning and end of the protocol. All patients subsequently underwent a standardised head-up tilt-table test with continuous 12-lead ECG monitoring. Following confirmation of POTS, management was initiated according to contemporary guidelines, emphasising both pharmacological and non-pharmacological strategies. Data were summarised using descriptive statistics and expressed as mean±standard deviation. This study was approved by the Research and Ethics Committee, Icare Institute of Medical Sciences and Research Haldia, with a waiver of individual consent.

All four patients (4 males; mean age 34.25±2.32 years) with chronic alcohol use disorder (mean duration 12.4±4.61 years) developed orthostatic intolerance symptoms during detoxification or early recovery phase.

Demographic features and orthostatic cardiovascular responses

Table 1 summarises the demographic characteristics and orthostatic haemodynamic responses of the four patients included in the analysis. All patients were male, with ages ranging from 23 to 48 years. During the active stand test, resting supine heart rates ranged from 64 to 78 beats per minute, increasing markedly upon standing to values between 100 and 118 beats per minute at 10 minutes. The mean heart rate increment (ΔHR) during active standing ranged from 36 to 40 beats per minute, fulfilling diagnostic criteria for POTS in all cases. Importantly, systolic and diastolic blood pressures remained relatively stable during postural change, with no patient demonstrating a significant orthostatic blood pressure drop.

Findings from the laboratory tilt-table test were consistent with active stand results. Supine heart rates recorded during tilt testing ranged from 67 to 88 beats per minute and increased to 97–120 beats per minute during head-up tilt. The ΔHR during tilt testing ranged from 30 to 39 beats per minute, again meeting POTS criteria. Blood pressure values during tilt remained preserved, confirming the absence of orthostatic hypotension. Overall, both testing modalities demonstrated reproducible orthostatic tachycardia without hypotension across all patients, supporting the clinical diagnosis of POTS in this cohort.

Table 2 summarises the clinical profile and illness trajectory of postural orthostatic tachycardia syndrome (POTS) in four patients with alcohol use disorder, illustrating a spectrum of autonomic involvement across different stages of disease. All patients presented with classic orthostatic symptoms, including dizziness, palpitations, light-headedness and near-syncope, which typically became apparent during or shortly after alcohol detoxification, suggesting unmasking of autonomic dysfunction during withdrawal. Older patients with longer duration and heavier alcohol exposure demonstrated more advanced manifestations, as reflected by biochemical abnormalities and neurological features. The 48-year-old patient exhibited laboratory evidence of alcohol-related organ damage and nutritional deficiency, alongside peripheral neuropathy, indicating established autonomic neuropathy with partial symptom reversibility following treatment. In contrast, the 27-year-old patient, despite younger age, showed severe functional impairment driven by profound malnutrition, requiring prolonged rehabilitation and combined pharmacological therapy, with only partial recovery. The 39-year-old patient represented an earlier stage of illness, with intermittent symptoms predating admission and minimal biochemical abnormalities, responding rapidly to conservative measures and achieving complete resolution with sustained abstinence.

The youngest patient demonstrated prominent anxiety-related symptom amplification, highlighting the interaction between autonomic dysfunction and psychiatric comorbidity.

Overall, the table highlights a progression from subclinical and reversible POTS to persistent autonomic dysfunction in alcohol use disorder, influenced by duration of alcohol exposure, nutritional status, and timely intervention.

Table 1: Demographic features and orthostatic cardiovascular responses.

Demographic features			Active-stand test						Head up tilt-table test			
Patient number	Age (years)	Sex	Supine HR	Supine BP	Standing HR at 10 minutes	Standing BP at 10 minutes	Delta HR	Supine HR	Supine BP	Standing HR at 10 minutes	Standing BP at 10 minutes	Delta HR
1	48	M	64	100/60	100	98/74	36	68	112/70	107	109/70	39
2	27	M	72	118/64	112	104/62	40	75	124/72	106	113/68	31
3	39	M	69	104/70	107	97/70	38	67	107/68	97	105/68	30
4	23	M	78	114/70	118	100/68	40	88	116/74	120	108/70	32

BP-Blood pressure, HR-heart rate

Table 2: Clinical characteristics, laboratory findings, and management of patients with alcohol-related POTS.

Patient no.	Age (years)	Sex	Symptoms of POTS	Laboratory/biochemical analysis	Management
1	48	Male	Dizziness, palpitations, near-syncope, tunnel vision on standing	Macrocytic anaemia (MCV 104 fL), elevated GGT (286 U/L), low thiamine (58 nmol/L)	IV thiamine followed by oral supplementation, folate and multivitamins, increased fluid intake, compression stockings, gradual mobilisation, propranolol 20 mg twice daily
2	27	Male	Severe orthostatic intolerance, fatigue, palpitations, light-headedness, exertional breathlessness	Severe malnutrition (BMI 17.6), Elevated transaminases with AST>ALT ratio, very high GGT	Nutritional rehabilitation, IV thiamine, vitamin B complex, alpha-lipoic acid, midodrine 5 mg TID, fludrocortisone 0.1 mg daily, graded physiotherapy
3	39	Male	Light-headedness, palpitations on standing, gastrointestinal symptoms (diarrhoea, early satiety, nausea)	Elevated transaminases with AST>ALT ratio, very high GGT	Fluid and salt loading, compression garments, propranolol 20 mg twice daily, motivational enhancement therapy, anxiety management
4	23	Male	Light-headedness, tremulousness, palpitations, anxiety exacerbated on standing	Elevated transaminases with AST>ALT ratio, low folate, vitamin D deficiency	Optimisation of sertraline, propranolol 20 mg twice daily, graduated exercise therapy, motivational enhancement therapy, integrated psychiatric care

POTS-Postural orthostatic tachycardia syndrome, AST-aspartate transaminase, ALT-alanaine transaminase, GGT-gamma glutamyl transferase, BMI-body mass index, MCV-mean corpuscular volume

DISCUSSION

This case series highlights POTS as an under-recognised manifestation of chronic alcohol use, emerging across a spectrum of disease severity and often becoming clinically evident during detoxification. The temporal relationship between alcohol withdrawal and symptom onset suggests that cessation of alcohol may unmask latent autonomic dysfunction that had been partially compensated during active drinking. Chronic alcohol exposure exerts toxic effects on both central and peripheral components of the autonomic nervous system, leading to impaired cardiovascular reflexes and abnormal heart rate regulation on assuming an upright posture. Several pathophysiological mechanisms likely contribute to the development of POTS in individuals with alcohol use disorder. Alcohol-related autonomic neuropathy is well documented and is mediated by direct neurotoxicity, oxidative stress, and mitochondrial dysfunction affecting small unmyelinated and thinly myelinated autonomic fibres. Nutritional deficiencies, particularly thiamine deficiency, further exacerbate neuronal injury by impairing glucose metabolism and axonal transport, as demonstrated in patients with macrocytosis and low thiamine levels. These processes collectively result in impaired sympathetic vasoconstriction in the lower extremities and splanchnic circulation during standing, leading to excessive venous pooling and a compensatory tachycardic response characteristic of POTS.[4-8]

In addition, chronic alcohol use is associated with altered baroreflex sensitivity and dysregulation of the renin–angiotensin–aldosterone system, contributing to reduced effective circulating volume. Malnutrition and low body

mass index, as observed in younger patients, may further amplify hypovolaemia and autonomic instability. The presence of gastrointestinal dysmotility in some cases reflects diffuse autonomic involvement, supporting a systemic neuropathic process rather than isolated cardiovascular dysfunction.[9,10]

Psychiatric comorbidities, particularly anxiety and depressive disorders, appear to modulate symptom perception and severity. Hyperadrenergic states driven by anxiety may worsen orthostatic tachycardia, while misattribution of early symptoms to psychological causes can delay diagnosis. Importantly, the heterogeneity in clinical course observed in this series suggests that early-stage autonomic dysfunction may be reversible with sustained abstinence, nutritional rehabilitation, and conservative measures, whereas prolonged alcohol exposure leads to persistent or only partially reversible POTS.[11-14]

Overall, these findings underscore the need for heightened clinical awareness of alcohol-related autonomic dysfunction and routine orthostatic assessment in patients undergoing deaddiction. Early recognition and targeted management may prevent progression to chronic POTS and improve functional outcomes in this vulnerable population.

CONCLUSION

This case series demonstrates that POTS represents a clinically significant complication in patients undergoing alcohol deaddiction treatment. The syndrome likely reflects chronic alcohol-induced autonomic neuropathy that becomes clinically apparent during withdrawal and early recovery phases. Healthcare providers working in addiction medicine should maintain awareness of POTS as a potential complication and consider systematic screening for orthostatic intolerance in patients with prolonged heavy alcohol use.

Further research is needed to determine the prevalence of POTS in alcohol deaddiction populations, identify risk factors for its development and persistence, elucidate the underlying pathophysiological mechanisms, establish optimal screening and diagnostic approaches, and develop evidence-based treatment protocols. Longitudinal studies examining the natural history of alcohol-related autonomic dysfunction during recovery would provide valuable prognostic information.

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