

A Case Report of Alcohol Use Disorder in Federal Medical Centre, Owo, Nigeria

Folorunso Timothy Oluwarotimi*, Adeniyi Kunle Ayomide, Okojie Ehinome Ehimare, Okonkwo Kenechukwu and Folorunso Ajibike Eunice

Federal Medical Centre, Ondo State, Nigeria.

*Correspondence:

Folorunso Timothy Oluwarotimi, Federal Medical Centre, Owo, Ondo State, Nigeria, E-mail: folorunsooluwarotimi@gmail.com.

Received: 02 Sep 2026; Accepted: 03 Oct 2026; Published: 14 Oct 2026

Citation: Folorunso Timothy Oluwarotimi, Adeniyi Kunle Ayomide, Okojie Ehinome Ehimare, et al. A Case Report of Alcohol Use Disorder in Federal Medical Centre, Owo, Nigeria. Japanese J Med Res. 2026; 4(4): 1-7.

ABSTRACT

Background: Alcohol use disorder is a chronic, relapsing condition that may simultaneously produce psychiatric, medical, traumatic and social harm.

Objective: We describe a patient whose escalating alcohol consumption culminated in alcohol-associated chronic liver disease, severe electrolyte abnormalities, recurrent falls, neglected distal humeral fracture and family disruption, followed by multidisciplinary rehabilitation.

Case Report: A 55-year-old man, trained chartered accountant and self-employed in a laundry business, had consumed alcohol for more than 30 years, with marked escalation during the preceding 3 years. He reported binge drinking, intake of approximately 5-8 units of gin on drinking days, craving, morning drinking, unsuccessful efforts to reduce intake, withdrawal symptoms, and continued use despite harm. Patient was earlier employed in a well-paid private establishment but lost job repeatedly thrice due to alcohol. He had severe episodes of quarrel with his wife, self-employed trader and children over the years before eventual separation from his wife and children 2 years before presentation. He however, denied cigarette use or other psychoactive substances. Physiological and behavioral dependence on alcohol were prominent. When he reduced or stopped drinking, he experienced poor sleep, restlessness, uneasiness and tremors. He described a desire to cut down, irritation when criticized, and guilt about his drinking and occasional eye-opener use. Alcohol use continued despite recognized recurrent falls, family separation and a serious upper-limb injury. His wife and children had distanced themselves because of his drinking, moved out of their matrimonial home and he had been living apart from them.

Approximately 2 years before the index admission, the patient was intoxicated while walking home in the rain. He began running, fell onto the right side of his body and immediately developed severe pain with inability to use the right upper limb. He was taken to hospital, stabilized and counselled for operative management, but he deferred admission and surgery. A persistent deformity around the right arm and elbow subsequently developed. X-ray done revealed malunion of a neglected distal humeral fracture. He is not known to have hypertension, diabetes mellitus, asthma or peptic ulcer disease but has positive family history of hypertension in his mother.

At admission, examination revealed a chronically ill man, icteric, wasted with mild bilateral pitting pedal edema. Cardiovascular examination revealed pulse rate of 98 beats/min, regular and of normal volume. Blood pressure of 130/90 mmHg, JVP was not elevated; S1 and S2 were heard, with a loud A2 and S4, but no murmur. The abdomen was full and moved with respiration and no area of tenderness. The liver was palpable approximately 2 cm below the right costal margin and liver span was 18 cm. There was no splenomegaly and the kidneys were not ballotable. Respiratory examination revealed vesicular breath sounds, no added sounds and SpO₂ was 97%.

There was no area of tenderness. Musculoskeletal examination showed a neurovascularly intact, deformed right upper limb and neurological examination was grossly intact. Mental-state examination showed a well-kempt, calm and largely cooperative man with neutral mood and congruent affect. Speech was spontaneous and coherent, and thought form was organized. He expressed ambivalence and conflicting thoughts about alcohol use but had no delusions, hallucinations or other psychotic phenomena. Concentration and

memory were intact, judgment was considered intact and insight was full.

Laboratory testing showed total bilirubin 45.1 $\mu\text{mol/L}$, conjugated bilirubin 14.8 $\mu\text{mol/L}$, aspartate aminotransferase 61 IU/L, alanine aminotransferase 42 IU/L, total protein 51 g/L, albumin 30 g/L, sodium 128 mmol/L and potassium 2.6 mmol/L. Ultrasonography demonstrated a coarse micronodular hepatic echotexture and hepatomegaly (17.2 cm). He received a benzodiazepine taper, vitamin supplementation, medical and gastroenterology review, orthopaedic assessment and an intensive multidisciplinary rehabilitation programme involving psychiatry, clinical psychology, occupational therapy, nursing, social work and family engagement. Interventions included relapse-prevention work, rational-emotive behavioural techniques, time and financial management, 12-step recovery activities, relaxation training and sponsor identification. He reported 11 weeks of abstinence, later underwent surgery on the affected upper limb, and described improving family communication and reunion with his wife and children. Conclusion: This case illustrates how severe alcohol use disorder can create a reinforcing cycle of medical illness, injury and psychosocial deterioration. Integrated addiction, medical, orthopaedic and psychosocial care can support meaningful early recovery, although long-term follow-up remains essential.

Keywords

Alcohol use disorder, Alcohol-associated liver disease, Hypokalaemia, Distal humeral fracture, Rehabilitation, Multidisciplinary care.

Introduction

Alcohol use disorder (AUD) is characterised by impaired control over alcohol consumption and persistent use despite adverse physical, psychological or social consequences. Its effects may extend far beyond intoxication, producing withdrawal, nutritional and electrolyte disturbances, liver injury, accidental trauma, family disruption and occupational decline. Contemporary diagnostic and treatment frameworks therefore require a broad biopsychosocial assessment rather than a narrow focus on the amount of alcohol consumed [1,2].

Cloninger's neurogenetic typology distinguishes a later-onset 25years, milieu-limited Type 1 pattern, commonly associated with anxiety, higher harm avoidance and lower novelty seeking, high reward dependence, from an earlier-onset, male-limited Type 2 pattern characterised by greater novelty seeking and antisocial traits [3,4] and intolerance of uncertainty and may contribute to

anxiety-driven drinking in some patients [5]. These constructs do not replace the diagnosis of AUD [6].

Alcohol-associated liver disease spans steatosis, steatohepatitis, fibrosis and cirrhosis. Because continued alcohol exposure drives progression, effective management requires treatment of both the liver disease and the underlying AUD. Major hepatology guidance supports abstinence and coordinated care that integrates liver or medical treatment with addiction, psychological and social interventions [7-12].

The present case is notable for the convergence of severe AUD, alcohol-associated chronic liver disease, profound hypokalemia, an intoxication-related neglected distal humeral fracture, social implications, loss of job and major family consequences. It also demonstrates the practical value of a multidisciplinary rehabilitation programme in a resource-constrained setting. The report is structured in accordance with the CARE case-report framework [12].

Chest X-ray, 12-lead resting ECG and echocardiography were essentially normal Full blood count Gastroenterology assessment

Table 1: Clinical timeline.

Time	Event
More than 30 years before presentation	Alcohol use began; initially described as modest.
Approximately 3 years before presentation	Marked escalation in quantity and loss of control, with binge drinking, craving, withdrawal symptoms, morning drinking and progressive family disruption.
Approximately 2 years before presentation	Intoxication-related fall with right distal humeral fracture. Operative care was recommended but deferred; chronic deformity developed.
17 February 2026	Later follow-up records identified this as the last reported alcohol use.
19 February 2026	Psychiatric and medical admission/review. Diagnoses included alcohol dependence/AUD, chronic liver disease, hypokalemia and neglected distal humeral fracture. Diazepam taper and vitamin supplementation were prescribed; urgent medical assessment and multidisciplinary referrals were arranged.
February-April 2026	Inpatient and subsequent outpatient rehabilitation involving psychiatry, clinical psychology, occupational therapy, nursing and social work. Family meetings, relapse-prevention work, time and money management, 12-step activities and relaxation training were undertaken.
17 April 2026	Clinical psychology and occupational-therapy reviews documented good motivation, review of assignments, psychobehavioural time-management work, therapeutic sharing and progressive muscle relaxation.
6 May 2026	Patient reported approximately 11 weeks of abstinence. Sponsors and a relapse-response plan were identified; family reconciliation and medical follow-up were discussed.
Later follow-up; exact date not visible	Patient reported surgical treatment of the right upper limb at a private hospital, continued abstinence and improving communication with his children. Repeat liver tests and dietary review were advised.

Table 2: Available investigation results and interpretation.

Domain	Investigation	Result	Clinical interpretation
Liver biochemistry	Total bilirubin	45.1 µmol/L	Elevated.
	Conjugated bilirubin	14.8 µmol/L	Elevated.
	AST (SGOT)	61 IU/L	Mild elevation.
	ALT (SGPT)	42 IU/L	Mild elevation; AST predominance.
	Total protein	51 g/L	Low.
	Albumin	30 g/L	Low; compatible with impaired hepatic synthetic function and/or poor nutrition.
Electrolytes/ renal	Sodium	128 mmol/L	Hyponatraemia.
	Potassium	2.6 mmol/L	Marked hypokalaemia requiring prompt assessment and correction.
	Chloride	104 mmol/L	Within commonly used adult reference limits.
	Bicarbonate	26 mmol/L	Within commonly used adult reference limits.
	Urea	1.9 mmol/L	Low; may reflect low protein intake, reduced hepatic urea synthesis or dilution.
	Creatinine	94 µmol/L	No biochemical evidence of renal impairment from this value alone.
Metabolic	Fasting blood glucose	94 mg/dL	Within the normoglycaemic range.
Lipid profile	Total cholesterol	112 mg/dL	Low-normal.
	LDL cholesterol	74 mg/dL	Interpret in clinical context.
	HDL cholesterol	38 mg/dL	Low for an adult man under many laboratory reference standards.
	Triglycerides	115 mg/dL	Interpret in clinical context.
Urinalysis	Appearance; specific gravity; pH; leukocytes	Clear/yellow; 1.030; 8.0; trace	Concentrated urine with alkaline pH and trace leukocytes; clinical significance uncertain without microscopy or culture.
Abdominal ultrasonography	Liver	Coarse parenchyma with echodense micronodules; liver span 17.2 cm	Chronic parenchymal liver disease with hepatomegaly.
	Kidneys	Left 9.6 x 4.2 cm; right 10.7 x 4.1 cm	Measurements documented; no structural interpretation was provided.

Repeat Electrolytes and liver function tests 15/08/2026.

Table 3 Repeat Electrolytes and liver function tests 15/08/2026.

Domain	Investigations	Results	Interpretation
Electrolytes	Sodium	142mmol/L	Within commonly used adult reference limits.
	Potassium	4.0mmol/L	Within commonly used adult reference limits.
	Chloride	114 mmol/L	Within commonly used adult reference limits.
	Bicarbonate	28 mmol/L	Within commonly used adult reference limits.
	Urea	3.9 mmol/L	Within commonly used adult reference limits.
	Creatinine	104 µmol/L	Within commonly used adult reference limits.
Liver biochemistry	Total bilirubin	7 µmol/L	Within commonly used adult reference limits.
	Conjugated bilirubin	3 µmol/L	Within commonly used adult reference limits.
	AST (SGOT)	8 IU/L	Within commonly used adult reference limits.
	ALT (SGPT)	12 IU/L	Within commonly used adult reference limits.
	Total protein	78 g/L	Within commonly used adult reference limits.
	Albumin	36 g/L	Within commonly used adult reference limits.



included viral serology for HBsAg, anti-HCV and RVS which were non-reactive. Liver enzymes such as ALT, AST and bilirubin were elevated, total protein and albumin were low but others were normal.

The liver is normal in size and outline with a span of 14.9 cm, it has a mild homogeneous increase in parenchymal echogenicity. The vascular channels, intrahepatic and extrahepatic ducts are within normal limits. No obvious intrahepatic mass is seen. The gallbladder, pancreas, para-aortic areas, spleen and both kidneys were sonographically normal. There is no ascites.

Therapeutic Intervention

Management was multidisciplinary. The initial treatment plan combined withdrawal management, medical evaluation and rehabilitation. A fixed-dose diazepam taper was prescribed, initial 10 mg twice-daily phase for 4 days; followed by 10 mg nightly for 6 days followed by 5 mg nightly for 4 days. Vitamin B-complex supplementation was also prescribed. An initial prescription for aripiprazole 10 mg nightly was given.

The orthopaedic team counselled the patient and relatives on surgical intervention following initial stabilization but analgesics were given such as Arthrotec (diclofenac sodium 75 mg and Misoprostol 300 µg) twice daily and co-codamol (codeine 8 mg and paracetamol 500 mg) two tablets twice daily. Surgery was successfully done when the patient had completed the psychological rehabilitation.

Rehabilitation was multidisciplinary and evolved from admission-based care to outpatient follow-up. Psychiatry coordinated the programme, while clinical psychology, occupational therapy, nursing and social work addressed relapse prevention and reintegration. Interventions included psychoeducation,

motivational interviewing, review of homework assignments, cognitive and rational-emotive behavioural techniques, and stress, anxiety, trigger and uncertainty-management strategies, time management in activities of daily living, budgeting and redirection of funds away from alcohol, progressive muscle relaxation, therapeutic sharing, 12-step recovery activities and planning for alternative non-alcoholic responses in social situations. Nutritional counselling and physiotherapy were also documented, while acamprosate 333 mg was considered for relapse prevention.

Family and social interventions were prominent. The team explained that rehabilitation was a continuing process rather than a short detoxification admission. The patient was encouraged to identify at least three sponsors; he named a younger brother and the clinical psychologist and was advised to add another reliable supporter. He developed a plan to disclose his abstinence to previous drinking companions, request water or soft drinks in social settings, structure the vulnerable period after work and contact a sponsor or caregiver promptly if a lapse occurred. Reconciliation with his wife, improved communication with his children and mobilisation of family support in Lagos were repeatedly discussed.

Follow-up and Outcomes

At outpatient psychology and occupational-therapy reviews on 17 April 2026, he was well groomed, calm, cooperative and motivated. Assignments from previous sessions were reviewed, and the programme reinforced psychobehavioural time management, budgeting, 12-step recovery, therapeutic sharing and relaxation training.

By 6 May 2026, he reported that his last alcohol use had occurred on 17 February 2026, corresponding to approximately 11 weeks of abstinence. He remained engaged with the rehabilitation team and articulated concrete relapse-prevention strategies. He also

planned continued medical review, repeat liver-function testing and abdominopelvic ultrasonography. Repeat ultrasonography dated 15 June 2026 documented a liver span of 14.9 cm, mild homogeneous parenchymal echogenicity and no ascites, compared with the earlier 17.2-cm hepatomegaly and coarse micronodular appearance. This interval improvement coincided with reported abstinence, although causality cannot be established from a single case. In a later multidisciplinary review, he reported undergoing surgery for the right upper-limb injury, maintaining abstinence and re-establishing communication with his children. He was counselled to avoid alcohol and unregulated herbal mixtures, seek dietary guidance and continue medical, psychological and rehabilitation follow-up.

Long-term biochemical recovery, functional outcome after orthopaedic surgery, sustained abstinence beyond the recorded follow-up period and formal assessment of quality of life were appreciable.



Discussion

The case described severe AUD, illustrating the concept of “evil in the bottle”. This case demonstrated a myriad of problems caused by alcohol consumption: impaired control, craving, withdrawal symptoms, hazardous use, continued use despite physical and interpersonal harm, unsuccessful attempts to reduce drinking and recurrent morning use [1]. The clinical and laboratory findings were consistent with alcohol-associated chronic liver disease; long-term heavy alcohol exposure, icterus, hepatomegaly, elevated bilirubin, aspartate aminotransferase predominance, hypoalbuminaemia, low total protein and a coarse micronodular hepatic echotexture on ultrasonography. This case demonstrates the cumulative and interacting consequences of severe AUD. The patient did not present with a single isolated complication; rather, years of escalating alcohol exposure produced a linked pattern of withdrawal symptoms, hazardous use, recurrent falls, chronic liver injury, electrolyte abnormalities, family separation and delayed orthopaedic care. The title, “Evil in the Bottle”, reflects the patient-level reality that the harm extended across biological, psychological and social domains.

The diagnosis of severe AUD was strongly supported by the clinical history. He exhibited impaired control, craving, withdrawal, hazardous use, continued consumption despite physical and interpersonal harm, repeated desire to reduce intake and morning drinking. Modern terminology favours AUD over the older labels of alcohol abuse or dependence, with severity based on the number of diagnostic features present [1]. The patient's history also included the four classic CAGE domains—cutting down, annoyance by criticism, guilt and eye-opener use—which constitute a brief alcohol screening construct rather than a stand-alone diagnostic test [13]. The case also underscores the value of obtaining a detailed drinking history: frequency alone would have underestimated severity because the patient denied daily use and yet had multiple dependence features and substantial consequences.

Alcohol-associated chronic liver disease was supported by hepatomegaly, icterus, hyperbilirubinaemia, AST predominance, hypoalbuminaemia and a coarse micronodular ultrasonographic appearance, suggesting that the liver disease had progressed beyond simple steatosis. Current hepatology guidance emphasizes that alcohol-associated liver disease exists on a spectrum and that treatment of AUD, sustained abstinence and appropriate liver assessment are central to disease modification [6-8]. However, the precise liver stage cannot be determined from the available data. The absence of documented ascites, encephalopathy, variceal bleeding and splenomegaly does not by itself exclude cirrhosis, while INR, alkaline phosphatase, gamma-glutamyl transferase and elastography were not documented. Viral serology for hepatitis B and C and retroviral disease was negative.

Hypokalemia was particularly of concern. A potassium concentration of 2.6 mmol/L may increase the risk of weakness and cardiac arrhythmia, while hyponatraemia may complicate withdrawal and medical stabilisation; disturbances of potassium, sodium and other

electrolytes are well described in chronic alcohol use disorder [14]. In patients with possible liver disease, benzodiazepines remain first-line agents for clinically significant alcohol withdrawal, but the regimen should be individualised, monitored and adjusted for hepatic impairment and withdrawal severity [15]. This necessitated acute medical review, electrocardiographic monitoring, parenteral thiamine administration and repeat electrolyte results [16].

Alcohol-related intoxication is associated with falls and fracture risk, and the patient directly linked the humeral injury to impaired judgement and unsteady behavior while intoxicated [17]. Delayed treatment resulted in chronic deformity and prolonged functional limitation. Adult distal humeral malunion or neglected fracture is technically challenging and may require corrective reconstruction, internal fixation or arthroplasty depending on the fracture pattern, joint condition and patient factors [18-20]. Although later surgery was reported, the lack of radiographs, operative details and objective functional measurements prevents evaluation of the orthopaedic outcome.

The most instructive aspect of this case was the integrated rehabilitation model. Medical or liver treatment without addiction care would have left the principal driver of injury untreated, while detoxification alone would not have addressed liver surveillance, fracture disability, social isolation and family rupture. Reviews and meta-analyses support simultaneous, multidisciplinary management of AUD and alcohol-associated liver disease, including coordinated medical, psychiatric, psychological and social interventions [9-11]. Cognitive-behavioural approaches also have evidence across alcohol and other substance use disorders, including within combined behavioural and pharmacological treatment strategies [21,22]. The patient's early abstinence, active participation, sponsor identification, structured relapse-prevention plan and improved family contact are clinically encouraging, although AUD remains a relapsing disorder and longer follow-up is required.

Conclusion

Severe AUD may act as a common cause linking chronic liver disease, metabolic abnormalities, traumatic injury and family breakdown. This case highlights the importance of recognising the full syndrome rather than treating each complication in isolation. Early medical stabilisation, sustained abstinence support, liver assessment, definitive injury care and coordinated psychiatric, psychological, occupational and social rehabilitation should be pursued together. The patient's early abstinence and functional engagement were promising, but continued follow-up is necessary to ensure recovery is sustained.

References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed, text revision (DSM-5-TR). Washington, DC: American Psychiatric Association Publishing. 2022.
2. World Health Organization. Mental Health Gap Action Programme (mhGAP) guideline for mental, neurological and substance use disorders. 3rd ed. Geneva: World Health Organization. 2023.
3. Cloninger CR, Bohman M, Sigvardsson S. Inheritance of alcohol abuse: cross-fostering analysis of adopted men. *Arch Gen Psychiatry*. 1981; 38: 861-868.
4. Cloninger CR. Neurogenetic adaptive mechanisms in alcoholism. *Science*. 1987; 236: 410-416.
5. Carleton RN. Fear of the unknown: one fear to rule them all?. *J Anxiety Disord*. 2016; 41: 5-21.
6. Jophlin LL, Singal AK, Bataller R, et al. ACG Clinical Guideline: Alcohol-Associated Liver Disease. *Am J Gastroenterol*. 2024; 119: 30-54.
7. European Association for the Study of the Liver. EASL Clinical Practice Guidelines: Management of alcohol-related liver disease. *J Hepatol*. 2018; 69: 154-181.
8. Crabb DW, Im GY, Szabo G, et al. Diagnosis and treatment of alcohol-associated liver diseases: 2019 practice guidance from the American Association for the Study of Liver Diseases. *Hepatology*. 2020; 71: 306-333.
9. Elfeki MA, Abdallah MA, Leggio L, et al. Simultaneous management of alcohol use disorder and liver disease: a systematic review and meta-analysis. *J Addict Med*. 2023; 17: e119-e128.
10. Shroff H, Gallagher H. Multidisciplinary care of alcohol-related liver disease and alcohol use disorder: a narrative review for hepatology and addiction clinicians. *Clin Ther*. 2023; 45: 1177-1188.
11. Winder GS, Fernandez AC, Mellinger JL. Integrated care of alcohol-related liver disease. *J Clin Exp Hepatol*. 2022; 12: 1069-1082.
12. Gagnier JJ, Kienle G, Altman DG, et al. The CARE guidelines: consensus-based clinical case reporting guideline development. *J Med Case Rep*. 2013; 7: 223.
13. Ewing JA. Detecting alcoholism: the CAGE questionnaire. *JAMA*. 1984; 252: 1905-1907.
14. Baj J, Flieger W, Teresiński G, et al. Magnesium, calcium, potassium, sodium, phosphorus, selenium, zinc, and chromium levels in alcohol use disorder: a review. *J Clin Med*. 2020; 9: 1901.
15. American Society of Addiction Medicine. The ASAM Clinical Practice Guideline on Alcohol Withdrawal Management. *J Addict Med*. 2020; 14: 1-72.
16. Sullivan JT, Sykora K, Schneiderman J, et al. Assessment of alcohol withdrawal: the revised Clinical Institute Withdrawal Assessment for Alcohol scale (CIWA-Ar). *Br J Addict*. 1989; 84: 1353-1357.
17. Ke Y, Hu H, Zhang J, et al. Alcohol consumption and risk of fractures: a systematic review and dose-response meta-analysis of prospective cohort studies. *Adv Nutr*. 2023; 14: 599-611.
18. Stavrakakis IM, Ntontis Z, Kastritsi O, et al. Distal humerus fracture malunion in adults: a case report and a review of the literature. *Case Rep Orthop*. 2022; 2022: 6041577.

-
19. Morrey ME, Morrey BF, Sanchez-Sotelo J, et al. A review of the surgical management of distal humerus fractures and nonunions: from fixation to arthroplasty. *J Clin Orthop Trauma*. 2021; 20: 101477.
 20. Holte AJ, Dean RE, Chang G. Distal humerus fractures: review of literature, tips, and tricks. *JSES Rev Rep Tech*. 2024; 4: 639-646.
 21. Magill M, Ray L, Kiluk B, et al. A meta-analysis of cognitive-behavioral therapy for alcohol or other drug use disorders: treatment efficacy by contrast condition. *J Consult Clin Psychol*. 2019; 87: 1093-1105.
 22. Ray LA, Meredith LR, Kiluk BD, et al. Combined pharmacotherapy and cognitive behavioral therapy for adults with alcohol or substance use disorders: a systematic review and meta-analysis. *JAMA Netw Open*. 2020; 3: e208279.