SUBSTANCE-RELATED AND ADDICTIVE DISORDERS

Alcohol Use Disorder

AUD

DSM-5-TR 303.90/305.00 | ICD-10-CM F10.10, F10.20

Compulsive alcohol use with impaired control, tolerance, and withdrawal that persists despite mounting medical and social harm.

LIFETIME PREVALENCE	TYPICAL ONSET	SEX RATIO	COURSE
~29% (US adults)	Late teens to mid-20s	~1.7:1 male:female	Chronic, relapsing-remitting

CLINICAL PICTURE

- Escalating quantity and frequency with failed cutdown attempts, morning drinking, and blackouts that patients reframe as ordinary social use.
- Tolerance shows as high-dose functioning; withdrawal emerges 6-24 hours after the last drink as tremor, sweating, tachycardia, nausea, and anxiety.
- Craving is cue-driven and situational, intensifying with stress, negative affect, and exposure to drinking environments or social pressure.
- Physical signs include hypertension, elevated GGT and MCV, spider angiomata, palmar erythema, gastritis, peripheral neuropathy, and unexplained trauma.
- Interpersonal fallout precedes medical presentation: absenteeism, DUIs, marital conflict, and role failure often bring the patient in before symptoms do.
- Comorbid insomnia, depressed mood, and anxiety typically improve substantially within 2 to 4 weeks of sustained abstinence.

DIAGNOSTIC CRITERIA AT A GLANCE

- Requires at least 2 of 11 problematic use indicators within 12 months, spanning impaired control, social impairment, risky use, and pharmacologic criteria.
- Severity is graded by symptom count: mild 2-3, moderate 4-5, and severe 6 or more, replacing the older abuse and dependence split.
- Craving, a strong desire or urge to drink, was added to the criterion set, while recurrent legal problems were removed from it.
- Tolerance and withdrawal are not counted when alcohol is taken solely under appropriate medical supervision, a rare situation for alcohol.
- Specifiers cover early remission (3 to under 12 months), sustained remission (12 months or more), and being in a controlled environment.

NEUROBIOLOGY & PHYSIOLOGY

- Alcohol potentiates **GABA-A** inhibition and blocks **NMDA** glutamate receptors; chronic use reverses this balance, driving withdrawal hyperexcitability.
- Mesolimbic dopamine release in the nucleus accumbens, amplified by endogenous opioid signaling at mu receptors, underlies reinforcement and craving.
- Repeated withdrawals kindle progressively severe episodes, raising seizure and delirium tremens risk with each successive detoxification.
- Allostatic shift recruits extended amygdala **CRF** and dynorphin systems, converting drinking from reward-seeking to negative-reinforcement relief.
- Heritability is roughly 50%, and **ADH1B** and **ALDH2** variants that produce flushing are strongly protective in East Asian populations.
- Chronic use causes thiamine depletion with Wernicke encephalopathy risk, cerebellar atrophy, hepatic steatosis to cirrhosis, and cortical volume loss.

PSYCHOLOGICAL MECHANISMS

- Negative reinforcement dominates late-stage use: drinking relieves withdrawal dysphoria, anxiety, and insomnia rather than producing euphoria.
- Alcohol expectancies for tension reduction and social ease, learned early from family and media, predict initiation and heavier adolescent consumption.
- Alcohol myopia narrows attention to immediate cues, impairing consideration of long-term consequences and inflating disinhibited behavior.
- Shame-based self-concept and stigma drive concealment, delayed help-seeking, and abstinence violation effects after a single lapse.
- Classical conditioning links people, places, and moods to craving, and cue reactivity persists for many months into abstinence.

DIFFERENTIAL & COMORBIDITY

- Substance-induced depressive and anxiety disorders resolve within weeks of abstinence, while independent disorders persist and need their own treatment.
- Screen with **AUDIT-C** or **AUDIT**, and differentiate from unhealthy drinking without disorder, bipolar disorder, and PTSD-driven self-medication.
- Comorbidity includes mood and anxiety disorders, PTSD, ADHD, tobacco use disorder, and other sedatives; polysubstance use raises overdose risk.
- Suicide risk is elevated roughly two to threefold, and acute intoxication increases impulsive attempts and the lethality of chosen means.
- Assess withdrawal severity with **CIWA-Ar**; scores above 8 to 10 warrant medication, and prior seizures or delirium tremens indicate inpatient detox.

Treatment EVIDENCE-BASED MANAGEMENT

PHARMACOLOGIC

- **Naltrexone 50 mg/day** oral or **380 mg IM monthly** is first-line, reducing heavy drinking days; check LFTs and avoid with any opioid agonist.
- **Acamprosate 666 mg TID** supports abstinence maintenance and is preferred in hepatic impairment; reduce the dose for CrCl of 30 to 50 mL/min.
- **Disulfiram 250 mg/day** works only with supervised dosing and high motivation, and the aversive reaction can itself be medically dangerous.
- Off-label **topiramate 200-300 mg/day** and **gabapentin 900-1800 mg/day** have supporting trial data, especially with comorbid anxiety or insomnia.
- Withdrawal management uses symptom-triggered **lorazepam** or **chlordiazepoxide** plus **thiamine 100 mg** given before any glucose load.

PSYCHOTHERAPY

- **Motivational interviewing** and brief interventions of 1 to 4 sessions reliably reduce consumption in risky drinkers who are not yet dependent.
- **CBT** with relapse prevention over 12 to 16 sessions targets high-risk situations, coping skills, and abstinence violation cognitions.
- **Contingency management** yields the largest short-term effect sizes, and the **community reinforcement approach** restructures competing reinforcers.
- **Behavioral couples therapy** improves both drinking outcomes and relationship functioning more than individual therapy delivered alone.
- **Twelve-step facilitation** produces abstinence rates equal to or better than CBT and durably increases mutual-help group engagement.

ADJUNCT & SUPPORTIVE

- **AA** and secular alternatives such as SMART Recovery provide free, sustained recovery capital, and active linkage clearly beats passive referral.
- Use **ASAM** criteria to match level of care, from outpatient counseling through medically managed inpatient withdrawal.
- Monitor with **AUDIT**, timeline followback, and biomarkers such as phosphatidylethanol, carbohydrate-deficient transferrin, and GGT.
- Treat comorbid insomnia behaviorally with **CBT-I** rather than sedatives, and correct thiamine, folate, and magnesium deficits.
- Screen for hepatitis C, vaccinate against hepatitis A and B, and coordinate hepatology follow-up for patients with advanced liver disease.

CLINICAL PEARLS

- Two or more of 11 criteria in 12 months makes the diagnosis; the count sets severity.
- Give thiamine before any glucose load to avoid precipitating Wernicke encephalopathy.
- Naltrexone works even while the patient is still drinking; abstinence is not required.

References

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NOTE

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