Acute alcohol intoxication can present with mood changes, disinhibition, impaired judgment, reduced attention and concentration, incoordination, slurred speech, unsteady gait, etc. These symptoms worsen with increasing blood alcohol concentration. Cerebrovascular accidents can manifest with similar clinical features. A U-shaped relationship has been identified between alcohol consumption and ischemic stroke, whereas a more linear relationship has been found between alcohol use and hemorrhagic stroke. Heavy drinking is associated with an increase in the occurrence of intracerebral hemorrhage (ICH) and poor outcomes. Alcohol consumption is associated with elevated blood pressure (BP) through alteration of sympathetic nervous system, renin–angiotensin system, and vascular responses. It further influences inflammatory processes as well as vascular incidences. Hypertensive ICH (HICH) is the most common type of ICH accounting for 75% of primary ICH. We report a case of alcohol dependence syndrome who manifested with a hemorrhagic stroke.

A 38-year-old man was brought to our hospital in a drowsy condition at around 5 p.m. in the background of binge drinking of around 750 ml whiskey on the previous night. He had a dependent pattern of alcohol use for the past several years and was consuming 300–360 ml of whiskey daily for the last 2 weeks. Clinically, he was hemodynamically stable (pulse rate – 84/min, BP – 136/84 mmHg) and responding to verbal commands. He was not oriented to time and place and had gait ataxia. A psychiatric evaluation revealed reduced motor activity, incoherent speech, and ill sustained attention and concentration. There were no delusions or perceptual abnormalities. His MMSE score was 18/30 and CIWA-Ar -04. He was provisionally diagnosed a case of acute alcohol intoxication.

His hematological and biochemical investigations showed random plasma glucose – 105 mg/dL, mean corpuscular volume – 102.3fL, liver function tests (serum glutamic-oxaloacetic transaminase/prothrombin time [PT]/gamma-glutamyl transferase – 117/181/442 IU/L, serum Bilirubin – 1.1 mg/dL), electrolytes (Na/K - 149/4.9 mEq/L), PT – 13.5 s, and international normalized ratio – 1.05. Other investigations including arterial blood gas analysis, chest radiograph, electrocardiograph, and urinalysis were normal.

In view of persistence of what seemed like features of acute intoxication even after the expected period of the last drink, with significant cognitive impairment in the absence of other features of alcohol withdrawal or delirium tremens, an urgent noncontrast computed tomography (NCCT) of the brain was advised which showed ICH (Right) involving basal ganglia with intraventricular extension [Figure 1].

Around after 4 h, he developed alcohol withdrawal (CIWA-Ar 18, RASS score-4+). He was managed with intravenous (IV) lorazepam (symptom triggered protocol) and other supportive medications and IV fluids. The next morning, he developed high-grade fever (102.4 F), tachycardia, and reduced O$_2$ saturation. He had papilledema and GCS was 10/15. Chest radiograph showed patchy opacities in bilateral lower and middle lobes. He was kept on mechanical ventilator support. Repeat NCCT of the brain was suggestive of increased size of hemorrhage with obstructive hydrocephalus [Figure 2]. Urgent burr hole craniotomy was done.

The next morning, the patient had cardiac arrest, which responded to a resuscitation attempt and he was maintained on dopamine and noradrenaline infusion. After around 6 h, he had another cardiac arrest but could not be revived. The autopsy report...
was suggestive of ICH-right caudate lobe with intraventricular extension as a cause of death.

Heavy alcohol consumption can predispose an individual to HICH.\(^8\) Labeling an individual as “alcoholic” may result in underdiagnosis of such grave clinical condition or misdiagnosis as intoxication. As a result, such cases may not receive adequate clinical attention and care. This case highlights such common practice and error in case management which can be avoided by sensitizing and training the physicians in this aspect. Furthermore, it is the responsibility of the consultation–liaison psychiatrist to be cautious about such presentations and manage them in collaboration with other specialties.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the legal guardian has given his consent for images and other clinical information to be reported in the journal. The guardian understands that names and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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There are no conflicts of interest.

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