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Cerebrovascular Episode after an Acute Alcoholic Binge

Authors

Sourya Acharya[1], Samarth Shukla [2], Prasad Gurjar[3], Rasika Thakre [3]

[1]Prof, Dept of Medicine
[2]Professor, Dept of Pathology
[3]Resident, Dept of Medicine
JN Medical College, DMIMS Univ. Sawangi (Meghe) Wardha 442004 Maharashtra
Corresponding Author
Dr. Sourya Acharya
Dept of Medicine, JN Medical College DMIMS Univ. Sawangi (Meghe)
Wardha 442004 Maharashtra
Email: *souryaacharya@yahoo.co.in*

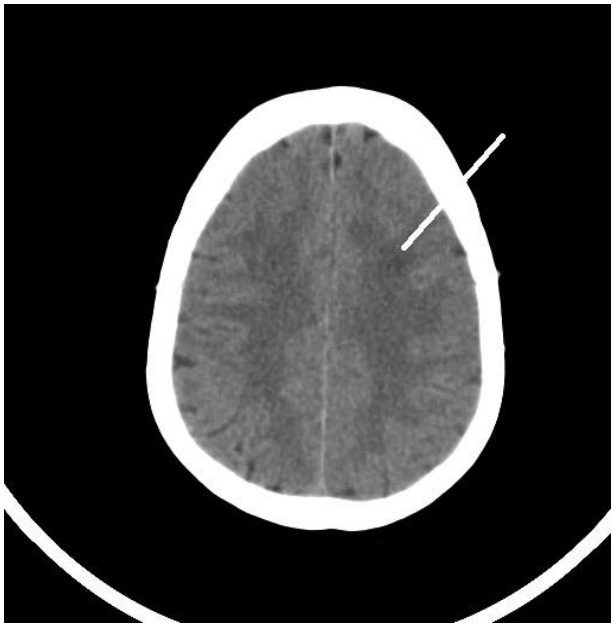
A 40 year old male patient presented to us with stuporous condition .He was accompined by relatives. According to history he was a chronic alcoholic and had an episode of binge drinking on the previous night. There was no history of vomiting, convulsion, fever. He was a non diabetic and non hypertensive.

General examination revealed Pulse of 80/min regular, Blood pressure 150/100mm of Hg. There was no icterus, pallor, cyanosis. JVP was normal.

Central nervous system examination – patient was stuporous, GCS – E3M4V3, pupils normal size reacting to light , dolls eye reflex present, patient was not moving right side of the body on deep painful stimuli and left plantar was extensor . There was no signs of meningeal irritation.

CVS, RS and per abdomen examination was normal. Investigations revealed blood sugar of 100mg%. Hemogram was normal. Serum electrolytes, kidney and liver functions were normal. A CT scan of brain revealed small hypodensity in left high convexity frontoparital lobe with adjacent gyral enhancement suggestive of acute infarct.

Patient was treated with g 25% dextrose, 100mg thiamine intravenously, aspirin, IV mannitol. Patient's mentation improved after 2 days. Then he was subjected to physiotherapy for hemiparesis and was referred to the deaddiction center for councelling. He was discharged with a normal mentation and residual hemiparesis after 5 days and awaits followup.



CT image revealed small hypodensity in left high convexity frontoparital lobe with adjacent gyral enhancement suggestive of acute infarct .

DISCUSSION

The role of alcohol consumption as an independent risk factor for ischemic brain infarction has remained unclear. Both mortality and morbidity from ischemic brain infarction seem to be increased among heavy alcohol drinkers. A synergistic effect of alcohol consumption and hypertension on the risk of ischemic stroke has been observed.[1,2] Studies claim that there are convincing evidence to support such an effect. [3,4] A case-control study showed that heavy alcohol ingestion within the 24 hours preceding the onset of stroke was a risk factor for ischemic brain infarction. [4] Cardioembolic stroke in particular could well be precipitated by binge drinking, because alcoholic intoxication may precipitate untoward effects on circulation and cardiac rhythm.

The risk factors for ischemic brain infarction differ by subtype of stroke. Recent heavy drinking of alcohol to increase the risk of cardioembolic stroke.A study concluded that alcohol intake within 24 hours (>40 g) and 1 week (>300 g) before the onset of stroke were both found to be significant and independent risk factors for cardioembolic stroke in men. [5] Previous case-control studies have not shown recent heavy alcohol intake to be a risk factor for cardioembolic stroke. Atrial fibrillation is a common cause of embolic stroke. Alcohol may account for a third of the new cases of atrial fibrillation, and atrial fibrillation may occur with acute and chronic alcohol ingestion. [5,6]

Recent heavy drinking of alcohol may trigger cardiogenic brain embolism. There are several plausible mechanisms that could explain the effect. First, heavy drinking of alcohol precipitates cardiac arrhythmias. It is also conceivable that some subjects may show genetically greater sensitivity to the arrhythmogenic effects of alcohol than others. An experimental study demonstrated that subjects prone to alcohol-induced atrial fibrillation seem to develop an exaggerated sympathetic reaction during even a modest (blood alcohol 1.5/1000) acute alcoholic intoxication. [7]

Alcohol ingestion may aggravate sleep apneas in subjects with sleep apnea syndromes. Sleep apnea syndromes are characterized by repeated long apneic episodes, arterial hypoxia, cardiac dysfunction, and arrhythmias. The arrhythmias may predispose to the formation of intracardiac thrombi and also dispatch existing thrombi into circulation.

Finally, the syndrome of acute alcohol withdrawal and troublesome hangover may result in vomiting causing a spontaneous Valsalva maneuver, which facilitates the entrance of paradoxical emboli via atrial septal defects and patent foramen ovale into the cerebral circulation.

In conclusion, the present study suggests that recent heavy drinking, including episodic and binge drinking, may trigger cardiogenic brain embolism. This is a new item on the list of hazards caused by heavy drinking of alcohol. Light drinking does not trigger cardiogenic brain embolism, and former heavy drinking is not a risk factor for cardioembolic stroke.

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