Alcoholic Intracerebral Hemorrhage

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Six alcoholic patients developed extensive cerebral hemispheric hemorrhages with both intraventricular and subarachnoid blood. All patients had evidence of liver damage, low platelet counts, and abnormal prothrombin and partial thromboplastin times. Four patients presented with seizures; in two of the four, these seizures were initially diagnosed as alcohol withdrawal seizures. Four patients were comatose with lateralizing neurologic deficit; two patients were comatose without lateralizing neurologic deficit, suggesting a metabolic encephalopathy. In one patient there was delayed neurologic deterioration. In all six patients, computed tomography showed large diffuse cerebral hemispheric hemorrhages, prominent intraventricular blood, and breakthrough into the subarachnoid spaces, which was confirmed by necropsy findings. There was marked mass effect but minimal surrounding edema. All six patients died. In three, autopsy showed no evidence of aneurysm, vascular malformation, neoplasm, or amyloid angiopathy and no arteriolar hypertensive changes. (Stroke 1988;19:1565–1569)

Alcohol has known acute and chronic cardiovascular effects\(^1\)–\(^4\) that cause alcoholics to be at risk for stroke. Chronic alcoholics have decreased concentrations of liver-produced coagulation factors and platelet abnormalities that predispose them to hemorrhagic stroke. Chronic alcoholism may cause hypertension, and acute alcoholic episodes may precipitate an acute hypertensive state with return to normotension when the subject stops drinking alcohol. Based on the potential for hypertension and impaired coagulation, hemorrhagic stroke develops with increased frequency in alcoholics. I describe six patients with alcoholic nonhypertensive intracerebral hemorrhages (ICHs) that may have occurred as a consequence of the chronic and/or acute alcoholic state.

Case Reports

**Case 1.** A 52-year-old chronic alcoholic woman developed headache and vomiting; she suddenly became unresponsive. Findings were stupor, hyperventilation, left hemiplegia, bilateral Babinski's signs, and a fixed dilated right pupil. Her hemoglobin was 10 g/dl; stool guaiac's were positive. Platelet count was 70,000/mm\(^3\). Her prothrombin time was 14.5 (normal control 12) seconds and her partial thromboplastin time was 38 (normal range 23–34) seconds. She had impaired liver function (SGOT 300 IU, SGPT 450 IU, total bilirubin concentration 3.5 mg/dl, alkaline phosphatase value 190 IU). Computed tomography (CT) showed a massive right frontal-parietal hematoma with intraventricular and subarachnoid blood (Figure 1). Despite treatment with mannitol and corticosteroids, she did not improve neurologically; she developed pneumonia and died. Necropsy showed right intracerebral subarachnoid and intraventricular hemorrhage. There was no evidence of aneurysm, arteriovenous malformation, neoplasm, amyloid angiopathy, or hypertensive arteriolar vascular disease. The ICH had broken through into the subarachnoid spaces at multiple points, into the superficial frontal and parietal regions.

**Case 2.** A 45-year-old chronic alcoholic man had been drinking heavily for 2 weeks. He stopped drinking because of abdominal pain. Three days later, he had a generalized seizure; following the seizure, he developed left-sided weakness and became lethargic. During 8 hours, he became comatose and developed left hemiplegia with a fixed dilated right pupil. Laboratory studies showed a hemoglobin concentration of 9.5 g/dl, a white blood cell count of 17,000/mm\(^3\), a total bilirubin concentration of 4.2 mg/dl, a white blood cell count of 17,000/mm\(^3\), a total bilirubin concentration of 4.2 mg/dl, a white blood cell count of 17,000/mm\(^3\), a total bilirubin concentration of 4.2 mg/dl, a white blood cell count of 17,000/mm\(^3\). His prothrombin time was 16 (control 11.9) seconds and his partial thromboplastin time was 38 (normal range 23–34) seconds. She had impaired liver function (SGOT 300 IU, SGPT 450 IU, total bilirubin concentration 3.5 mg/dl, alkaline phosphatase value 190 IU). Computed tomography (CT) showed a massive right frontal-parietal hematoma with intraventricular and subarachnoid blood (Figure 1). Despite treatment with mannitol and corticosteroids, she did not improve neurologically; she developed pneumonia and died. Necropsy showed right intracerebral subarachnoid and intraventricular hemorrhage. There was no evidence of aneurysm, arteriovenous malformation, neoplasm, amyloid angiopathy, or hypertensive arteriolar vascular disease. The ICH had broken through into the subarachnoid spaces at multiple points, into the superficial frontal and parietal regions.

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FIGURE 1. Computed tomograms, 52-year-old chronic alcoholic woman (Case 1). Top left: right frontal-parietal hyperdense nonenhancing lesion with (top right) extension into ganglionic and thalamic region. Bottom left: lateral ventricles are dilated with evidence of ventricular compression by extensive right hemispheric hematoma. Bottom right: intraventricular and cisternal blood.

blood in the lateral ventricles breaking through into the cortical sulcal spaces in multiple regions. There was evidence of transtentorial herniation. There was no evidence of aneurysm, vascular malformation, neoplasm, amyloid angiopathy, or hypertensive vascular disease.
Summary of Clinical Findings

The clinical and CT findings in these six patients are summarized in Table 1. Five patients were in their 40s. Five were men and one was a woman. None had a history of hypertension; none had elevated blood pressure when initially examined, and none were hypertensive during their hospital course. All six patients had biopsy evidence of fatty liver or cirrhosis. All were anemic with low platelet counts and abnormal prothrombin and partial thromboplastin times. Four patients initially had seizures; in two, this initial seizure was believed to be an
alcohol withdrawal seizure, but both patients showed a decreasing level of consciousness with focal deficit. Four of the six patients were comatose without focal deficit, pupillary abnormalities, or abnormal eye movements, suggesting a metabolic encephalopathy. In only one of the six patients was there delayed (8 hours later) neurologic deterioration, which included findings consistent with transtentorial herniation. In this patient necropsy showed evidence of transtentorial herniation and intraventricular bleeding.

CT showed a characteristic pattern consisting of widespread cerebral hemispheric hemorrhage that appeared as a mottled, heterogeneous, high-density lesion. There was prominent cortical sulcal space and basal cisternal blood. The subarachnoid space hemorrhage was quite prominent and diffuse. Intraparenchymal blood was seen in four patients. The hemispheric hemorrhage was associated with marked mass effect. There were CT findings consistent with subfalcine and transtentorial herniation.

All six patients died. Autopsy was performed in three patients and showed diffuse and extensive cerebral hemispheric hemorrhage without evidence of an underlying aneurysm, vascular malformation, neoplasm, amyloid angiopathy, or hypertensive disease.

**Discussion**

Previous studies have reported that alcohol may predispose to hemorrhagic stroke\(^5\,^7\); chronic alcohol ingestion is associated with hypertension. One study reported that the association of alcohol with stroke is reduced after adjusting for hypertension\(^8\); however, in another study alcohol was a major risk factor for ICH despite blood pressure control.\(^9\) The risk of hemorrhagic stroke was directly correlated with the amount of alcohol consumed, and the relation held for subarachnoid hemorrhage and ICH.\(^5\,^10\) If alcohol consumption is decreased, the risk of hemorrhagic stroke is reduced. None of these six patients had a history of hypertension or were hypertensive when the ICH developed. My clinical data do not allow me to quantify alcohol consumption with the coagulation disturbances or to compare the risk of hemorrhagic stroke with a control group.

If hypertension is not implicated as a major risk factor for hemorrhagic stroke, it is likely that the impaired coagulation mechanism (low platelet count with abnormal prothrombin and partial thromboplastin times) was related to the ICH. The reduced number of platelets and impaired platelet function would weaken the blood vessel wall, and combined with impaired coagulation factors, this predisposes alcoholics to ICH. All these patients had low platelet counts and abnormal prothrombin and partial thromboplastin times. In alcoholics with hepatic damage, there may be decreased concentrations of clotting factors, increased fibrinolysis, and laboratory evidence of disseminated intravascular coagulation.\(^11\)

In patients with hypertensive ICH, the characteristic locations include the putamen, thalamus, pons, or cerebellum;\(^12\) the intracranial bleeding does not usually break through into the subarachnoid space, whereas in nonhypertensive patients with ICH (e.g., aneurysm, vascular malformation, amyloid angiopathy) the hemorrhage is more commonly located in
the subcortical hemispheric white matter and breaks through into the subarachnoid spaces. In hypertensive ICH, CT shows a homogeneous hyperdense nonenhancing lesion, usually with a surrounding hypodense region representing edema. Although many patients with ICH have erythrocytes in the cerebrospinal fluid, CT rarely shows subarachnoid blood. All six alcoholic patients with cerebral hemispheric hemorrhage showed cisternal and cortical sulcal blood; however, autopsy showed no other identifiable cause of the ICH. In one patient there was delayed neurologic deterioration consistent with transtentorial herniation. Without an early CT at the time the initial neurologic dysfunction developed and a repeat CT after the delayed neurologic deterioration, I could not precisely determine the mechanism of the delayed deterioration. Potential pathologic mechanisms include worsening of the edema, herniation, and intraventricular extension of the bleeding. The infrequent finding of delayed neurologic deterioration in these six alcoholic patients is consistent with the low incidence reported in ICH series.

Based on the location, size, and the CT pattern of the alcoholic hemorrhage, it is most unlikely that this represented a hemorrhagic contusion or a traumatic ICH. The CT pattern was also not consistent with a hemorrhagic cerebral infarction. The intracranial blood was widely dispersed within the subarachnoid spaces. There was blood in the superficial cortical and subcortical white matter, with extension into the basal ganglia and thalamus. Despite the large size and prominent mass effect (with evidence of subfalcine and transtentorial herniation), surrounding hypodense areas representing edema were not seen. This lack of CT-visualized edema is contrasted with the findings of prominent edema in other large nonalcohol-related ICHs. None of these six alcoholic patients showed multiple ICHs. I suggest that chronic alcohol exposure may lead to massive cerebral hemorrhage. Other studies using large sample sizes and quantitative features of alcohol ingestion and coagulation may settle the significance of this finding.

References

Key Words: • alcoholism • blood coagulation • blood platelets • cerebral hemorrhage
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