METHANOL POISONING AND HEPARIN: A DANGEROUS COUPLE?

To the Editor—Bilateral cerebral ischemia of the basal ganglia is the most well known finding of methanol intoxication that can be identified on computed tomography and magnetic resonance imaging.1,2 Brain hemorrhage is a rare complication that has been related to systemic heparinization during hemodialysis.3,4 We report an unusual case of transformation of bilateral cerebral infarction into massive unilateral hemorrhage after hemodialysis without systemic heparin.

A 37-year-old man with a history of chronic alcohol abuse, was admitted to our hospital after ingesting an unknown amount of pure methanol. Three days earlier, he complained about abdominal pain, nausea, vomiting, photophobia, and blurred vision. Once admitted, his mental status rapidly deteriorated until coma, and he developed a generalized tonic-clonic seizure lasting 3 to 5 minutes.

The patient was transferred to our intensive care unit. Neurological examination showed fixed and dilated pupils, absence of corneal reflexes, and a Glasgow Coma Score (GCS) of 3. Although he breathed 50% oxygen, arterial blood analysis showed pH 6.85, plasma bicarbonate 2.6 mmol/L and pCO₂ 14 mmHg. Osmolality gap was 40 mOsm/L. The white-cell count was 38.8 × 10⁹/L, and hematocrit 43%. The coagulation profile including platelet count, prothrombin time, partial-thromboplastin time, and plasma fibrinogen level was within normal limits. The patient was intubated and mechanically ventilated. A cranial CT scan showed a bilateral hypodense area of infarction in the basal ganglia (Fig 1A). Toxico logical screening revealed a plasma methanol level of 59 mg/dL and an ethanol level of 0 mg/dL. The patient was started on intravenous ethanol, sodium bicarbonate, and folic acid. He underwent one 3-hr hemodialysis session (AN69 ST dialysis membrane) without standard heparin addition. However, the dialysis circuit was rinsed with 6,000 U of heparin dissolved in 1,000 cc of normal saline, and then flushed with 1,000 U of heparin dissolved in 500 cc of normal saline (boluses of 100 cc of this solution every 30 minutes).

Metabolic acidosis and patient’s neurological condition slightly improved after hemodialysis, the pupils returned to normal, and the GCS changed to 5. A coagulation profile drawn after hemodialysis showed a platelet count 319 × 10⁹/L, prothrombin time 59%, partial-thromboplastin time 7%, and plasma fibrinogen level 206 mg/dL. Four hours after hemodialysis while receiving fresh frozen plasma, he developed a sudden enlargement of his right pupil. Supportive measures were started, and a cranial CT scan revealed a massive hemorrhage into the right basal ganglia extending to the ventricular system, with diffuse cerebral edema (Fig 1B). The patient died 36 hours after admission, and he was organ donor.

Accidental or suicidal ingestion of methanol causes severe metabolic acidosis and usually ocular and central nervous system manifestations. The most characteristic radiological finding after methanol intoxication is bilateral necrosis of the basal ganglia, mainly of nonhemorrhagic origin.1,2,3 The basis for this anatomic specificity of methanol is unknown. Injury to the putamen probably represents a direct toxic effect of methanol metabolites, anoxia, and acidosis, that could be potentiated by a poor venous drainage of this area.4 Other features occasionally described, include edema, necrosis of subcortical white matter, cerebellar cortical lesions, subarachnoid and bilateral intracerebral hemorrhage.1,6 Bilateral brain hemorrhage is a rare complication that has been associated with hemodialysis. The role this procedure plays in the genesis of cerebral hemorrhagic changes in the setting of methanol poisoning is open to debate. Phang et al reported 6 of 21 patients and Giudicissi et al one case with brain hemorrhagic necrosis after hemodialysis, suggesting a deleterious role of systemic anticoagulation during hemodialysis.5,6 However, Patankar et al reported 1 of 4 patients with hemorrhagic changes before hemodialysis, and they inferred hemorrhage is unrelated to the procedure, although the coagulation profile are lacked in this case.7

Cerebral hemorrhage related to methanol poisoning is usually bilateral and not expansive. Large hemorrhages with extension into the ventricular system have been described8 although massive hemorrhagic transformation of cerebral ischemic lesions have not been previously reported. Our patient had a normal platelet count and coagulation times previous to hemodialysis, and it was per-

FIGURE 1. (A) Cranial CT scan showing a bilateral hypodense area of infarction in the basal ganglia. (B) Cranial CT scan showing diffuse cerebral edema and massive hemorrhage into the right basal ganglia with extension into the ventricular system, with large expansivity.
formed without systemic heparinization. Nonetheless, heparin was used in the dialysis circuit, being probably the cause of coagulation disturbance previous to hemorrhage. This case suggests avoidance of any use of heparin in cases of methanol intoxication.

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