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BILATERAL PUTAMINAL HEMORRHAGE RELATED TO METHANOL POISONING

A COMPLICATION OF HEMODIALYSIS?

CASE REPORT

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SUMMARY - A case of acute methanol intoxication is presented, in which bilateral putaminal hemorrhage developed after hemodialysis. Even though the patient was initially comatose and profoundly acidotic, favorable outcome was achieved, with long-term neurologic impairments essentially restricted to mild crural paraparesis, retrograde amnesia, and marked visual deficit. A comparative literature review is evaluated.

KEY WORDS: methanol, putaminal hemorrhage, neurotoxicity, coma.

Hemorragia putaminal bilateral em intoxicação por metanol: uma complicação de hemodiálise? Relato de caso

RESUMO - Um caso de intoxicação aguda por metanol é relatado, no qual o paciente desenvolveu hemorragia putaminal bilateral após hemodiálise. Muito embora o paciente se encontrasse inicialmente comatoso e profundamente acidótico, a recuperação foi favorável, com comprometimento neurológico a longo prazo essencialmente restrito a paraparesia crural discreta, amnésia retrógrada e déficit visual acentuado. Uma revisão comparativa da literatura é avaliada.

PALAVRAS-CHAVE: metanol, hemorragia putaminal, neurotoxicidade, coma.

Acute methanol poisoning may be caused by accidental ingestion (mostly in children), or may be intentional. The latter is more frequently found in cases of suicide attempt, or, among alcoholics, when ethanol is replaced by methanol. Common commercially available sources of methanol are photocopying fluid, windshield washing fluid, wood alcohol, and perfume. In Brazil, the use of methanol as a substitute for gasoline has been occasionally associated with poisoning,

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when inadequately washed methanol containers are used for storage or transportation of a popular sugar cane brandy. The present report describes a condition in which ethanol ingestion was replaced by methanol.

CASE REPORT

MA, a 40-year-old male with a pre-morbid history of repeated alcohol consumption bouts was admitted to the emergency room with a one-day history of visual blurring followed by drowsiness. Shortly after admission he became comatose, with bilateral pupillary widening but no focal neurologic signs. Routine blood analysis was remarkable for metabolic acidosis (pH of 7.06 and bicarbonate of 3.9 mEq/L only). Toxicologic screening disclosed a very high blood methanol level (1.4 g/L).

While in the emergency room, the patient was immediately started on intravenous infusion of sodium bicarbonate, and 10% ethanol solution in 5% dextrose in water. A computed tomography (CT) scan of the head was unrevealing. Approximately 10 hours after arrival, he was started on hemodialysis. Throughout the procedure the patient was kept heparinized, and the activated thromboplastin time was maintained 1.5 times above normal. Dialysis was carried out in a single session, and was discontinued after normalization of blood methanol analyses.

While systemically stable, the patient failed to neurologically improve over the first week. At this point, a follow-up CT scan of the head disclosed bilateral putaminal hemorrhage (Fig 1A). Gradual neurologic improvement slowly developed over the following three weeks, and the patient was discharged from the hospital. At this point in time he had fully regained consciousness, presenting with bilateral blindness and mild crural paraparesis. There were no parkinsonian features.

Follow-up neurologic re-evaluation at six months revealed retrograde amnesia and slight visual improvement (insufficient to identify objects at a distance beyond 30 cm). Funduscopy disclosed bilaterally pale and atrophic optic disks. The remaining neurologic findings were essentially unchanged from the time of discharge. A repeat CT scan showed bilateral lucencies where the acute putaminal bleeding had been (Fig 1B).

COMMENTS

Neurologic changes. Methanol poisoning is uncommon, and this case presents with interesting findings. First, even though the acute assessment showed very high blood methanol levels, the final outcome was relatively benign, except for the visual impairment. While extrapyramidal signs are among the most frequent sequelae of methanol poisoning[5] and, as a general rule do not improve over time[3,7,8], the present case was free of such neurologic complication. Apraxia has also been reported in relation to methanol intoxication[6], but it was not found in the present case either.

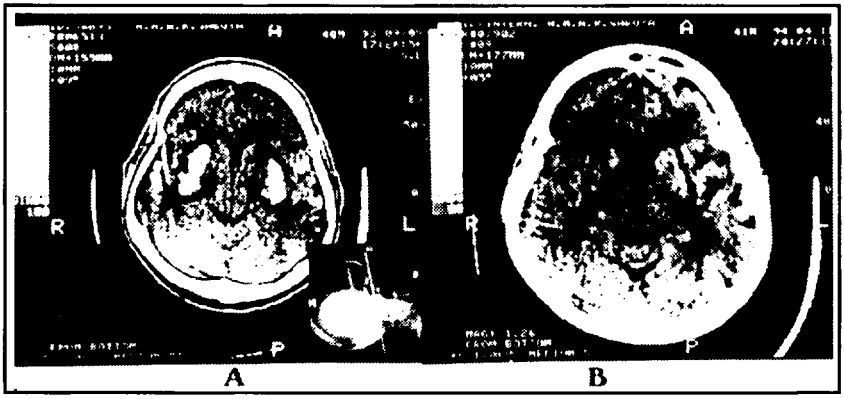

Fig 1. Computed tomography (CT) scans of the head: A - at seven days after admission. B - at six months. See text for description of CT findings.

Computed tomographic diagnosis. As to CT findings, early hypodense changes may be found, which are generally felt to be ischemic. In our case, however, only hyperdense changes were documented, at one week, which were consistent with deep bleeding. In a previous report[8], an association has been postulated between therapeutic dialysis and intracranial hemorrhage found on CT scan. Such association would be supported by the present findings.

Pathophysiology. With respect to pathophysiology and lesion location, a direct neurotoxic effect of methanol upon the optic nerves was also considered in the present case. In fact, direct ocular toxicity has been previously reported in such cases[4]. Alternatively, there seems to be a belief that putaminal neurons are particularly more vulnerable to methanol than other areas of the nervous system[5]. Early hypodense changes on CT scan may, indeed, represent putaminal edema and/or ischemia, with possible disruption of the blood-brain barrier.

Management. Treatment of acute methanol poisoning has been previously proposed by combining intravenous ethanol administration and hemodialysis[2], as was done in the present case. This proposition is because ethanol has greater affinity for alcohol dehydrogenase than does methanol. Since alcohol dehydrogenase plays a major role in initiating methanol metabolism[1], this management modality would appear to be sound. As described above, however, early disruption of the blood-brain barrier may be potentially associated with ischemia or edema. In either case, such disruption would facilitate bleeding under circumstances of hypocoagulability associated with heparinization. We therefore believe that routinely adopted procedures for therapeutic dialysis in these patients should be modified; that is, alternative strategies might take into account avoidance of heparinization if possible.

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