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Reply

Resposta

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Dear Editors,

I thank Drs. Sookaromdee and Wiwanitkit for their comments on the manuscript entitled "Acute methanol poisoning"[1]. The readers argue that "it is difficult to judge that the case is an actual neurological problem due to methanol poisoning" and that "the high plasma methanol level, but normal osmolar gap, should be discussed".

Early in the course of methanol ingestion, the accumulation of methanol typically leads to an increase in the osmolal gap (OG). Later, as methanol is metabolized, the OG falls and the anion gap increases. A combination of a normal OG and elevated anion gap can be seen in the later stages of this process[2]. However, if the serum OG is low at baseline, this could obscure any increase in OG that is secondary to the accumulation of methanol[3]. Some patients with definite toxic alcohol ingestion have a normal OG[2,4]. Conversely, a high OG may be seen in processes other than toxic alcohol ingestion, including lactic acidosis, ketoacidosis, chronic kidney disease, the sick cell syndrome, and following the use of mannitol[3-5]. A patient with methanol intoxication could have either a normal or high OG and either a normal or high anion gap[3,4]. For these reasons, a normal OG cannot be used to rule out a toxic alcohol ingestion[3].

It is unknown whether the patient described in this case had a low OG at baseline before his intoxication as no prior laboratory testing was available. The time interval between his ingestion and presentation to the emergency department was unclear.

The patient described in this report had a plasma methanol level of 40 mg/dL. Similar levels have been lethal in some individuals. However, there are reports of patients who survived and had complete recovery following methanol poisoning despite concentrations as high as 920 mg/dL[6,7]. The readers point out that plasma methanol may be falsely positive in cases with ketoacidosis or hyperglycemia. Nevertheless, a diagnosis of ketoacidosis secondary to ethanol or hyperglycemia is unlikely in this patient. At the time of presentation, ethanol level was undetectable and glycemia was 132 mg/dL.

Bilateral necrosis of the lentiform nucleus, particularly of the putamen, is the most characteristic neuroimaging abnormality in acute methanol poisoning[8]. This is certainly not a pathognomonic finding, and it can also be seen in conditions such as Wilson's disease and Leigh's syndrome, among others[8]. However, methanol intoxication was clearly the underlying etiology in this patient.

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