Letters to the Editor should be typed doubled-spaced (including references) with conventional margins. The overall length is limited to five manuscript pages, including not more than one figure or one table.

Case of Methanol Poisoning: Final Words

To the Editor:

I would like to comment on a recent Letter (1), in which Shahangian tried to defend his data concerning a case of methanol poisoning reported by him in a previous issue (2). When we examined his data, we noticed that the formate concentrations as reported were pharmacokinetically unattainable and we suggested in a Letter to the Editor (3) that Dr. Shahangian must have committed a substantial error in his analysis (possibly 10- to 100-fold). In his response (4), Dr. Shahangian admitted that “we were not aware of a substantial increase in the fluorescence intensity ...” Furthermore, he stated that, when he corrected for that error and re-analyzed one of his samples, formate concentrations were found to be 2 mmol/L as compared with 17 mmol/L when he used his original method (last set of data in reference 4 and formate values in Figure 1 of reference 2). Thus, the error in the sample that he re-analyzed was 8.5-fold (equivalent to a margin of error of 750%)!

Whether his error was 8.5-fold, as he admitted, or 10- to 100-fold as we suggest, the fact remains that the blood formate concentrations reported in his paper (2) are inaccurate and completely unreliable and that any conclusions based on such data are of questionable validity at best.

References


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Ed. note: Dr. Makar asked for an opportunity to respond to ref 1. With this response their debate is closed.

To the Editor:

I read with great interest the Case Report (1) concerning the formic and lactic acidosis in a patient with methanol intoxication. The described patient had extreme metabolic acidosis on arrival, with plasma lactate of 8 mmol/L and methanol of 74 mmol/L. The lactate then increased to 23 mmol/L.

These authors suggest that the steep increase in lactate was due to the metabolic effects of formate. They also comment that the initially high lactate probably didn't result from ischemia and hypotensive shock, because their patient was adequately perfused throughout. I would like to draw attention to the possible metabolic effect of seizure the patient had on arrival. Seizures can cause extremely high lactate (see the following tabulation, Case A), which, fortunately, in otherwise healthy subjects is quite rapidly metabolized. My interpretation of the results would be that the initially high lactate could have been the effect of seizure, with then superimposed effects of formate.

I would also like to point out that a measurement of serum osmolality and a calculation of osmolal gap (measured plasma osmolality minus osmolality calculated as [2(Na' + K) + glu] + [urea]) in a patient with severe metabolic acidosis could channel the diagnostic thinking into the possibility of ethanol or methanol intoxication (Case B) and speed up the diagnosis. Patients with ethanol intoxication can also present with severe metabolic acidosis (1, 2).

To the Editor:

Dilution of Specimens for Assays of Cholesterol in High-Density Lipoprotein

To the Editor:

Gorba (1) reported an apparent decrease in the HDL-cholesterol value of over 20% (620 vs 810 mg/L) after the sample was prediluted with isotonic saline. We reported (2) an easy way to overcome this problem. Diluting serum with an equal volume of a 50 g/L solution of bovine albumin in ammonium acetate solution (pH 7-8; 0.1 mol/L) leads to almost the same results as obtained with native sera. Our precipitation reagent is phosphotungstic/MgCl₂, and cholesterol in the supernate was determined enzymically.

We found the following orthogonal