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Authors' response

We would like to thank and congratulate Dr Sanaei-Zadeh for his letter [1] regarding our article, recently published in "Cardiology Journal" [2].

The only reason for the omission was the possible simultaneous submission to both journals, or alternatively, as our paper was accepted in advance, his paper may be released after ours.

Anyway, the data of Sanaei-Zadeh et al. [3] are solid and their electrocardiography (ECG) analysis of methanol intoxication is impeccable.

The commonalities of both series are the presentation with sinus tachycardia, PR prolongation, and prolonged QT interval. As both series have determined, these ECG changes varied with the amount of methanol ingested, and maybe the way of ingestion. Most importantly, the time elapsed between ingestion and ECG recording seems to be crucial to capture those at higher risk. Full cardiac monitoring is recommended in both series. The discrepancy in the role of acidosis as a trigger for ECG changes, however, is more difficult to understand. While in our series severe acidosis predicted multiple ECG changes, in Sanaei-Zadeh's it did not.

Although acidosis appears to be consistently observed in methanol intoxication, an increase in anion gap or osmolality gap may not be observed consistently throughout the course of methanol intoxication. It is conceivable then that the differences on the role of acidosis could be associated with different timing on obtaining ECG recordings from the original intoxication.

It called our attention though, that in the study by Sanaei-Zadeh et al. [3], the authors did not report on methanol plasmatic concentrations which could be of interest for a multivariate analysis on the causes of high mortality in their series. In their table, they showed that the shorter the time from ingestion to first consult, the worse the evolution ($p = 0.07$). As some of these cases could be the result of suicide attempts, one would wonder whether the comatose state was the result of single substance intoxication or the consequence of multiple substances interactions. The same rationale would apply to explain the ECG changes. In our series, there was a case of Brugada pattern, however, and a careful analysis of the chart demonstrated that this patient was sedated with Propofol at the time of obtaining the ECG, thus the ECG change was attributed to a different substance than methanol.

Conflict of interest: None declared

References

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