Reviewer's report

Title: Hyperglycemia-related central pontine demyelinization after a binge-eating attack in a patient with type-2 diabetes: a case report

Version: 0 Date: 19 Dec 2017

Reviewer: Joseph D. Burns

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Pliquett and colleagues submit for review a report of a case of central pontine myelinolysis that occurred in the setting of severe hyperglycemia and in the absence of significant sodium shifts. The report is valuable in that it replicates the findings of other similar reports and has the potential to make an even stronger case than previous reports that hyperglycemia is the culprit. The following are suggestions for improving the current manuscript draft:

1. The case on a clinical basis for CPM should be better made and illustrated by providing a more detailed neurologic exam, especially at admission, peak severity, and discharge, and follow-up. The information provided in the current draft does not demonstrate to the reader the clinical picture nearly well enough.

2. The MRI data would be better if the ADC maps were shown to demonstrate true restricted diffusion.

3. The case for hyperglycemia-mediated increased in serum osmolality should be made as strongly as possibly. Can the authors calculate serum osmolality (or use the measured value) over the admission and plot it against time in a single graph that also does the same for the serum sodium and glucose concentrations? The rate of rise of the serum sodium concentration is especially important to show. Incorporation of pre-admission data that shows definitive sudden increase in serum osmolality around the time of symptom onset (or preceding by a few days to a week) would be very interesting if the data are available as it would be conclusive proof that a sudden rise in serum osmolality due to glucose and not sodium is the culprit. Graphing these data would be much more useful than what the authors show, which is an expected decrease in serum glucose concentration during the hospital admission.

4. If the level of consciousness was indeed normal at discharge, and he was just encephalopathic, how do the authors explain this? The pontine lesion would not necessarily be expected to cause this. Was there more widespread osmotic myelinolysis, such as in the corpus callosum? Other explanations?
5. Cirrhosis was probably a risk factor for the development of CPM in this patient. While his glucose intake preceding admission was quite dramatic, I suspect that it was the combination of this plus cirrhosis that led to CPM -- many other patients have probably had similar sugar ingestion binges without CPM. The authors should discuss this in the discussion section.

6. The conclusion, both in the abstract and body should emphasize that the case demonstrates that CPM can be caused by sudden, severe, and sustained hyperglycemia, especially when another risk factor (in this case, cirrhosis) is present. This is the real value of the case. There are plenty of other, better reasons for diabetic patients to modify their sugar intake and adhere to prescribed insulin regimens.

Are the methods appropriate and well described?
If not, please specify what is required in your comments to the authors.

Yes

Does the work include the necessary controls?
If not, please specify which controls are required in your comments to the authors.

Yes

Are the conclusions drawn adequately supported by the data shown?
If not, please explain in your comments to the authors.

Yes

Are you able to assess any statistics in the manuscript or would you recommend an additional statistical review?
If an additional statistical review is recommended, please specify what aspects require further assessment in your comments to the editors.

Not relevant to this manuscript

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