Author’s response to reviews

Title: Metabolic Encephalopathy secondary to Diabetic Ketoacidosis: A Case Report

Authors:

Maria Tomkins (maria.tomkins@ucdconnect.ie;mariatomkins200@gmail.com)
Richard McCormack (richardmccormack@nrh.ie)
Karen O'Connell (karenoconnell2@beaumont.ie)
Amar Agha (amaralagha@beaumont.ie)
Áine Merwick (ainemerwick@beaumont.ie)

Version: 1 Date: 07 May 2019

Author’s response to reviews:

7th May 2019

BMC Endocrine Disorders

Dear Editor,

Many thanks for accepting the manuscript titled “Metabolic encephalopathy secondary to diabetic ketoacidosis: A case report” for peer review. Please find below a detailed description of my response to the concerns raised by the reviewers.

Firstly, Reviewer 1 Tandy Aye, raised the following points which have been addressed as follows:

1. Advised to include a background regarding metabolic encephalopathy and whether it was a diagnosis of exclusion – Included in the Background section, page 4, line 65-74 and line 83.

2. Requested further information regarding the diabetes history and glucose control prior to admission – Included in the Case presentation, page 5, line 92-97
Secondly, Reviewer 2 Takao Ando, raised the following points which have been addressed as follows:

1. Requested a detailed description of the management of diabetic ketoacidosis – Included on page 10 line 195-200

2. Consideration of other causes of encephalopathy given the past medical history of depression, alcohol misuse, cannabis use, poor adherence and multiple sclerosis – extensive investigation of encephalopathy was undertaken in this case. The patient was highly-functioning in society prior to this episode of diabetic ketoacidosis, which directly correlated with encephalopathic symptoms. There was no evidence on admission of toxic substance ingestion or alcohol intake. Indeed, risk factors such as depression and poor medical compliance contributed to risk of DKA. Similarly, there was no evidence of progression of demyelination on neuroimaging however the presence of multiple sclerosis may have contributed to neurological vulnerability. These points are discussed and explained on page 3 lines 58-64, page 6 lines 118-120, page 7 lines 149-151 and page 14 lines 294-300

3. Concern over diagnosis of T1DM as the HbA1c was low on admission, request of more information regarding diabetes history, autoantibodies and consideration of other causes of ketoacidosis – A further description of the past diabetic control has been added on page 5 lines 92-97. The patient had a history of previous DKA as well as previous severe hypoglycaemic events. On discussing his control with his general practitioner, it is clear that he had very erratic control, rarely checked his sugars and had highly fluctuating capillary glucose readings when he did check. This HbA1c therefore reflects this erratic control.

The patient was diagnosed with T1DM in 1991 in childhood, age 9 therefore there is little doubt it was type 1; antibodies are not available as historically they were not commonly done in the early 1990s particularly when the diagnosis was very typical of type 1. The patient was insulin dependent from diagnosis and had at least two admissions with DKA, The glycaemic control was suboptimal at 8.5% (our target is less than 7%) but he had previous measurements which were higher.

Given this patient’s complex past medical history and socioeconomic status other precipitants of ketoacidosis should be addressed – These issues have been addressed on page 9, lines 165-169. Although starvation and alcohol intake may have contributed to ketoacidosis the patient met the diagnostic criteria for diabetic ketoacidosis so it is evident that ketoacidosis was the driving factor. Again, socioeconomic group and depression would have contributed to an increased risk of diabetic ketoacidosis.

4. The reviewer was concerned that the patient was not taking insulin for 2 years however this was a misinterpretation of the manuscript. The patient did not attend diabetes services for 2 years however was attending his family doctor during this period and was taking insulin during this period although compliance may have been suboptimal. This has been amended and clarified on page 5, line 92-97.
5. The reviewer requested that the post-encephalopathy course and discussion be shortened. Excerpts from both sections have been edited to resolve this issue. See page 11 paragraph one and three have been excluded.

I hope this edited submission meets the requirements listed above.

I confirm that all authors have reviewed and approved this amended submission.

Sincerely,

Maria Tomkins

mariatomkins200@gmail.com