Dear Editors,

We appreciate the opportunity to revise our manuscript. The point-by-point reply to the reviewers' comments can be found below. We would like to thank the reviewers for their positive and constructive comments. The changes we have made are highlighted in red in the revised manuscript. The following is a table of the revisions made.

Yours Sincerely,
On behalf of all authors

Comments	Revisions
Introduction	
The authors highlight the relationship between DKA and cerebral edema. However, there is no discussion of the relationship between diabetes and vascular disease including increased stroke risk.	We have revised it as you suggested in Line 73-76 and added citations (reference #1-4).
BICAO is listed as a possible cause of stroke. However, this was seen on initial MRI, is it possible that stroke was related to chronic condition and not to acute DKA.	Thank you for your comment. However, the clinical course of BICAO is often chronic due to collateral circulation (Line 181-183, reference #6-7, #14). Our clinical inferential process has been described in Line 230-244. We have modified the title of our manuscript due to BICAO is a sufficient risk factor for stroke. We also modified the abstract in Line 39-43, Line 59-61, Line 69-70 to describe DKA acts as additional stroke risk factor for patient with BICAO, HTN, T1DM.
This patient suffered two prior strokes, it would be beneficial to discuss the risk of recurrent stroke in dependent of DKA.	Thank you for your comment. We have revised it as you suggested in Line 73-76 and added citations (reference #1-4).
Case Presentation	
The authors state that the patient was admitted for rehabilitation 3 months after suffering a stroke. There is no discussion of the reason for this lag and what the patients state of health was in the 3 months between stroke and admission.	Actually, she had no lag for rehabilitation. She received health care in another hospital before she was transferred to our hospital. We have described it in Line 95-100.
It is unusual for DKA to develop in a hospitalized patient, it would be helpful for the authors to comment on the suspected etiology.	Thank you for bringing this to our attention. We have described it in Line 193-194, 213-218 and added citations (reference #17).
It is unusual for mild DKA to require a prolonged time course for correction (3 days).	It’s unusual for this situation. However, we’ve excluded the potential factors such as infection or

Was there an explanation for this delay? Were other possible causes for persistent metabolic acidosis such as sepsis evaluated for?	sepsis before the second stroke. We described it in Line 220-229. (reference #36).
Discussion	
It is implied in the discussion that DKA caused this stroke. Is there evidence to support this?	The possible mechanisms of DKA related to ischemic stroke have been presented in Line 201-212. However, the clinical course of BICAO is often chronic due to collateral circulation in Line 181-183. (reference #6-7, #14)
How is the risk of acute inflammation of DKA different from the chronic vascular disease of Type 1 DM?	The acute inflammations of DKA are different from the chronic vascular disease. We have described it in detail in Line 174-176 and 201-212.
Is BICAO a sufficient risk factor for stroke without the addition of DKA risk?	Thank you for your comment. We have revised the statement in Line 78-81, Line 181-188 and added reference #6-8, #14. We have modified the title of our manuscript due to BICAO is a sufficient risk factor for stroke. We also modified the abstract in Line 39-43, Line 59-61, Line 69-70 to describe DKA acts as additional stroke risk factor for patient with BICAO, HTN, T1DM.