Continued Confusion After Resolution of Diabetic Ketoacidosis

Ryan Rogers DO
Lehigh Valley Health Network, Ryan.Rogers@lvhn.org

Gretchen A. Perilli MD
Lehigh Valley Health Network, gretchen_a.perilli@lvhn.org

Philip Dunn DO
Lehigh Valley Health Network, Philip.Dunn@lvhn.org

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Continued Confusion After Resolution of Diabetic Ketoacidosis

Ryan Rogers, DO, Gretchen Perilli, MD, and Philip Dunn, DO

Lehigh Valley Health Network, Allentown, Pennsylvania

Introduction

Diabetic Ketoacidosis (DKA) is a complication of an uncontrolled type I diabetic. After resolution of the ketosis, the polyuria, polydipsia, nausea and encephalopathy usually subside. DKA poses risks for developing several cerebral complications including cerebral edema, seizure activity and CVA but intracranial hemorrhages (ICH) have been reported in rare case among children.

Case Presentation

A 20-year-old type one diabetic female, found unresponsive by family, and brought into ER. Vital signs were BP 91/39, HR 135, RR 32, O2 100% on room air and febrile at 101.7. Patient had incoherent vertebral responses; PERRL, only withdrew extremities to painful stimuli. She had a glucose > 1400, BHBA of 8.86, 40-60 ketones in urine and PH of <6.94, PCO2 <20 and HCO3 of <5.0. In addition to fluid resuscitation, she was started the DKA protocol of regular insulin 0.1 U/kg, a biarbonate infusion, intubated and transferred to the MICU. The bicarbonate infusion was discontinued when PH was >7.2, and when her AG closed she was transitioned to basal/bolus insulin as Lantus and Humalog. After resolution of DKA, sedation was held and she remained encephalopathic and not following commands. A CT scan of the head revealed multiple bilateral parenchymal hematomas in her frontal and parietal lobes, MRI confirmed hemorrhages with the largest being 3.1 x 1.9 cm with surrounding edema and regional mass effect. Days later after close observation, patient followed commands, extubated, and transferred to low level.

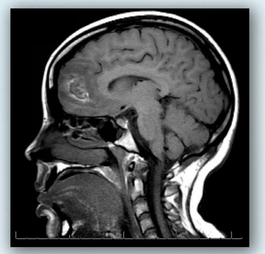

Image 1. MRI of the brain pre-contrast, sagittal T1 view, showing left frontal lobe intracranial hemorrhage.

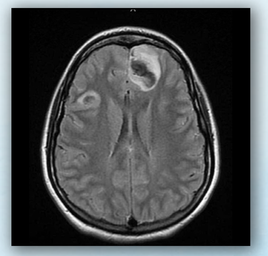

Image 2. MRI of the brain pre-contrast, axial view with flair, showing large left frontal lobe intracranial hemorrhage measuring 3.1 x 1.9 cm and smaller right frontal lobe lesion.

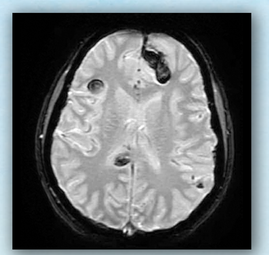

Image 3. MRI of the brain pre-contrast, axial view SWI, showing left frontal, right frontal, right splenium and left temoporal lobe intracranial hemorrhages.

Discussion:

Mortality rates from DKA range from 2-5%, mostly from medical illnesses precipitating the metabolic derangements, including infections and infarctions.[3] In DKA, several cerebral complications including cerebral edema/ischemia, comas and seizsures occur. ICH's are a rare complication and infrequent in adults but several cases, among children, have been published.[2] Several theories regarding the causes have been propoed. An increase in vWF and decreased levels of protein C /free protein S activity causing a prothrombic state have been shown during DKA and its treatment.[5] A reduced blood volume from dehydration, higher blood viscosity, and acidosis causes hyperventilation leading to vertebral vasoconstriction, could potentially result in cerebral ischemia and infarction.[1] Hyperglycemia and acidosis have been theorized to cause endothelial damage allowing RBS's to leak from blood vessels.[5] The combination of ischemia and permeability of arteries potentially causes cerebral infarctions transforming into ICH's.[6] A way to prevent such complications is early detection. Once the diagnosis of DKA is made, using coma cales, which have been used to evaluate and treat other encephalopathies, could prompt a CT scan of the head, leading to early diagnosis.[4] In conclusion, when altered mental status is presented with DKA, further investigative studies should be pursued, rather han assuming the encephalopathy is from the metabolic derangements.

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