Case Reports

Neurological Deterioration in Diabetic Ketoacidosis – Is it Cerebral Edema or Something Else?

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Cerebral edema (CE) is the commonest cause of morbidity and mortality during the first day of treatment for (DKA) in pediatric patients, but if neurological deterioration occurs during treatment of diabetic ketoacidosis, other possibilities must also be considered. We present a 13 month old girl with DKA and meningococcal meningitis.

Keywords: *Cerebral edema, Diabetic ketoacidosis, Meningococcal meningitis.*

Cerebral edema is the commonest cause of morbidity and mortality during first day of treatment for DKA in pediatric patients(1). The commonest cause of abnormal neurology in a child with DKA is cerebral edema. But other possibilities like intracranial infection should be considered.

Case Report

A 13-month-old girl presented with 4 week history of intermittent fever, cough and lethargy. She was off food but was still drinking adequately; her mother had noticed that she was having more wet nappies than usual as well as some weight loss. There was no significant past medical history. There were no perinatal problems or developmental concerns. Immunizations were up to date. On initial examination she was afebrile, but had tachycardia (heart rate: 172 per minute). The respiratory rate was 40 per minute with oxygen saturation of 98% in air. She looked drowsy, tired, dehydrated with acidotic breathing. Further examination was unremarkable. Her blood glucose was 21.2 mmol/L and the blood gas showed severe metabolic acidosis with pH of 7.09 and base deficit of 25 mEq/L. Other investigations at this stage revealed low normal sodium of 135 mEq/L, potassium 3.6 mEq/L, urea 12.3 mg/dL, creatinine 0.65 mg/dL and CRP of 133 mg/L. Hemoglobin was 13.4 g/dL and white cell count was raised to 43.1×10^9/L with 31.4×10^9/L neutrophils. Blood film showed shift to left with toxic granulation in neutrophils. Urine was strongly positive for sugar and ketones. Her chest *X*-ray was normal. DKA therapy was commenced. A broad spectrum antibiotic was given in view of raised CRP, raised white blood cell count with neutrophilia and toxic granulation. During the course of treatment her biochemistry improved, blood glucose fell and pH improved but CRP rose. She appeared drowsy with GCS of 12-13; after about 20 hrs of treatment her GCS dropped to 10. She developed a petechial rash on her back, buttocks and legs, and also had neck stiffness. She was treated with mannitol and fluid restriction. Acyclovir was added to the treatment regimen. Brain CT revealed normal sized ventricles with good grey-white matter contrast. There was no evidence of cerebral edema or any other abnormality. Next morning her level of consciousness improved but the neck stiffness became more obvious. Lumbar puncture showed turbid CSF with 3200 white cells of which 90% were polymorphs. CSF protein was raised and Gram stain showed Gram negative intracellular diplococci. The blood and CSF culture did not grow any organisms but CSF DNA PCR was positive for group B meningococcus. She recovered completely with no long term sequelae.

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Discussion

Cerebral edema is the most likely cause of acute neurological deterioration in DKA, though in about 20% of acute neurological episodes other causes such as localized basilar edema, hemorrhage, thromboses, or infection found by computed tomography scan or on postmortem examination(2,3) may be seen. The management of other pathologies is often different from the management of cerebral edema.

Intracranial infections (meningitis/encephalitis) may be associated with DKA. There have been reports of DKA with Herpes simplex type-2 encephalitis(4), tuberculous meningitis(5) and group B streptococcal meningitis(6). We have not found another report of meningococcal meningitis with DKA.

Our case highlights the importance of making correct neurological diagnoses because of implications for the management. If intracranial infections, especially the fulminating ones caused by meningococcus, are not considered then the child may be managed on the lines of cerebral edema alone with devastating consequences.

The diagnosis was suspected clinically due to the presence of petechiae, and was confirmed by laboratory investigations. Our patient presented with moderate to severe ketoacidosis as a first presentation of diabetes. In our patient it was appropriate to suspect cerebral edema as a cause of neurological deterioration and treat it. It is likely that meningitis was associated with the severity of the DKA though symptoms suggesting the development of diabetes had been present for over four weeks prior to diagnosis. We recommend that meningitis should be considered if neurological status deteriorates during management of DKA.

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