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EC NEUROLOGY
Case Report

An Unusual Presentation of a Child with Diabetic Ketoacidosis. Case Report

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Abstract

Diabetic ketoacidosis is well known in paediatric patients with both newly diagnosed and established type 1 diabetes.

We are presenting the case of a rare neurological presentation. A three years old female presented to the Emergency room in KSMC (King Saud Medical City), Children's hospital in Saudi Arabia with fever, rapid breathing, excessive urination and abdominal pain for two days, she had unrecordably high blood glucose with severe metabolic acidosis with a VBG reading as follows, pH 6.9, Bicarbonate 6.0, base excess -25, lactate 1.3 with wide anion gap. Urine had positive ketones 3 plus and glucose 4 plus.

Based on the presentation and lab work up the diagnosis of DKA (diabetic ketoacidosis) was made and she was started on the DKA management protocol as per endocrinology unit protocol in KSMC hospital. She encountered three episodes of generalised tonic-clonic convulsion in the PICU the day od admission and after few of DKA management and showed clinical improvement of metabolic acidosis and her level of conscious improved, she had sudden decrease in her level of conscious with mild metabolic acidosis (ph: 7.1, Bicarbonate 10, we notice on neurological examination she was found to have quadriparesis, power 1/5 and absent reflex all upper and lower limb with the involvement of the respiratory muscles. She was treated after full work up including MRI brain and Spine with conduction study as suspected acute critical polyneuropathy, improved.

In conclusion, children presenting with DKA may develop fatal neurological sequelae like cerebral oedema, although this is rare. Critical illness polyneuropathy and Guillain-Barré syndrome should be considered as a differential in acute weakness with DKA patient with consideration of other possibilities; early management can improve the outcome of the patient.

Keywords: *Diabetes Mellitus Type 1; DKA; Polyneuropathy; GBS*

Introduction

Diabetic ketoacidosis is highly prevalent in paediatric patients with both newly diagnosed and established type 1 diabetes. The incidence of type 1 diabetes (T1D) among youth is steadily increasing across the world. Up to a third of paediatric patients with T1D present with diabetic ketoacidosis, a diagnosis that continues to be the leading cause of death in this population [1].

Long-term cognitive consequences of type 1 diabetes and associated fluctuations in glycaemia during childhood and adolescence are well documented [2].

The most severe acute diabetes-related central nervous system complication in type 1 diabetes is cerebral oedema associated with diabetic ketoacidosis (DKA) [2], with 10 - 25% of affected children experiencing chronic central nervous system morbidity [3]. Although the frequency of DKA at diagnosis is relatively high (15 - 70% depending on age and geographic region), fulminate clinical cerebral oedema in this context is relatively rare, with an incidence rate of 0.5 - 0.9% [4]; hence, documented brain injury is also unique.

Other complications Deep vein thrombosis (DVT) is not uncommon in critically ill children who require central venous catheter placement as they introduce a foreign body, cause endothelial damage, and impair blood flow [5].

Abnormalities of haemostasis have been identified in patients with poorly controlled diabetes, although the mechanism is not entirely understood [6].

DKA is also characterized by elevated levels of inflammatory markers (CRP), cytokines (IL6, IL1 beta, TNF alpha) and complement activation [4]. This inflammatory state, combined with the disruption of the typical coagulation cascade, can place patients at increased risk of thrombosis and stroke during acute episodes of DKA.

Case Report

We are presenting a case of rare neurological presentation. A three years old female presented to the ER of KSMC (King Saud Medical City), Children's hospital in Saudi Arabia with fever, rapid breathing, excessive urination and abdominal pain for two days. Her parents have a consanguineous marriage, and her grandmother was a known case of insulin-dependent diabetes mellitus (IDDM) type 2. Our patient was diagnosed as a case of IDDM (insulin dependent diabetes) one year ago and she has been on insulin therapy that been given by her parents as was instructed by her Endocrinology physician mostly her mother, administer for her.

One week back before this presentation, she came to the ER with similar symptoms but responded well to the diabetic ketoacidosis management as per Endocrinology unit protocol in KSMC and she was discharged in stable condition.

However, during this presentation, she had unrecordably high blood glucose with severe metabolic acidosis with a VBG reading as follows, pH 6.9, Bicarbonate 6.0, base excess -25, lactate 1.3 with wide anion gap. Urine had positive ketones 3 plus and glucose 4 plus, diagnosis of DKA was made and she was started on the DKA management protocol as per the King Saud Medical City Endocrinology unit protocol. A brief history from the mother suggested, no missed dose, no expired medications. But She had, contact with the sick individual and she had flu-like symptoms.

She did show some respond at all to the DKA management, and her condition stabilized, her VBG improved PH 7.1, HCO_3: 10, She encountered three episodes of generalised tonic-clonic convulsion in the PICU same day of admission, her level of conscious improved but, after few dys from stabilization she was deteriorated in the form of a progressive decrease in GCS. Finally, she required ventilatory support in PICU, on neurological examination she had generalized weakness (quadriparesis), power 1/5 and absent reflex all upper and lower limb with the involvement of the respiratory muscles. Multiple neuroimaging was done (MRI brain and spine with contrast), which was unremarkable (Figure 1). Her CSF examination showed WBC of 435/cmm; protein was 79.9 mg/dl, RBC was 2.0 mg/dl. EEG revealed diffuse cortical dysfunction; Amylase was 212 U/L and lipase 2004 U/L, which were suggestive of pancreatitis. The liver enzymes had a steady rise but the other lover functions were normal. NCV was done showed [2] repeated VBG (venous blood gas). PH: 7.3, PCO_2: 35 mmHg. HCO_3: 18 meq/l, base excess: +1 mmol/l.

She was covered by antibiotics as suggestive by infectious team given her clinical situation improved and level of conscious improved. Still, still, she cannot move her limbs, a dose of IVIG suspecting autoimmune disorder; however, autoimmune workup was normal that included ANA, pANCA, cANCA, LKM were all normal.

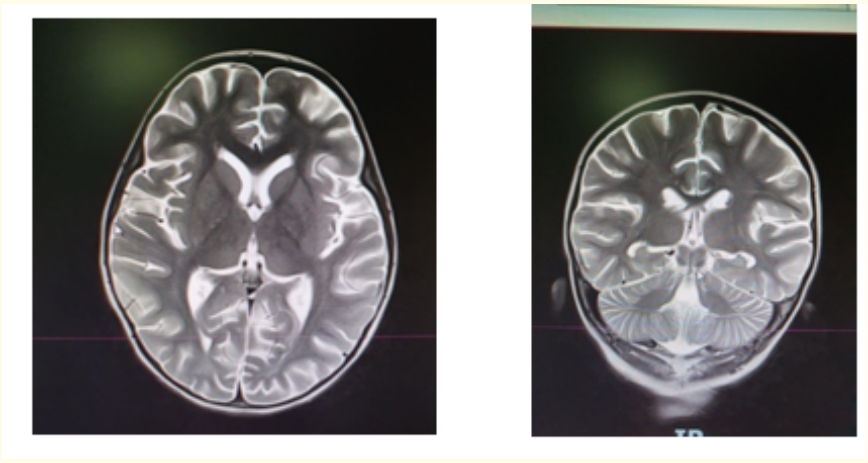

Figure 1: *MRI brain T2 axial and coronal cut and spine normal.*

She had enterovirus from the nasopharyngeal aspirate, and she was positive for MRSA from the bronchial wash.

She came out slowly from her DKA status, and her neurological function improved, and she was extubated to NIV then to NC and then finally to room air. The patient was discharged home and came for neurology clinic follow up and NCV test was done after 6 months showed (Figure 2) with a repeated dose of IVIG 1 gm/kg.

Patient's weakness improved she is able to walk normally, prepared ready to raise her arm to 90 degrees, flex arm and minimal extension of both arms, shoulder adduction and minimal abduction, examination normal power and reflex in both lower limb and upper 3/5 shoulders and elbow and normal hand movement, DTR +1. She continues on physiotherapy.

Discussion

There is no existing literature documenting such a case. This is the first reported case in children complicated with acute quadriplegia of unclear pathogenesis was confirmed.

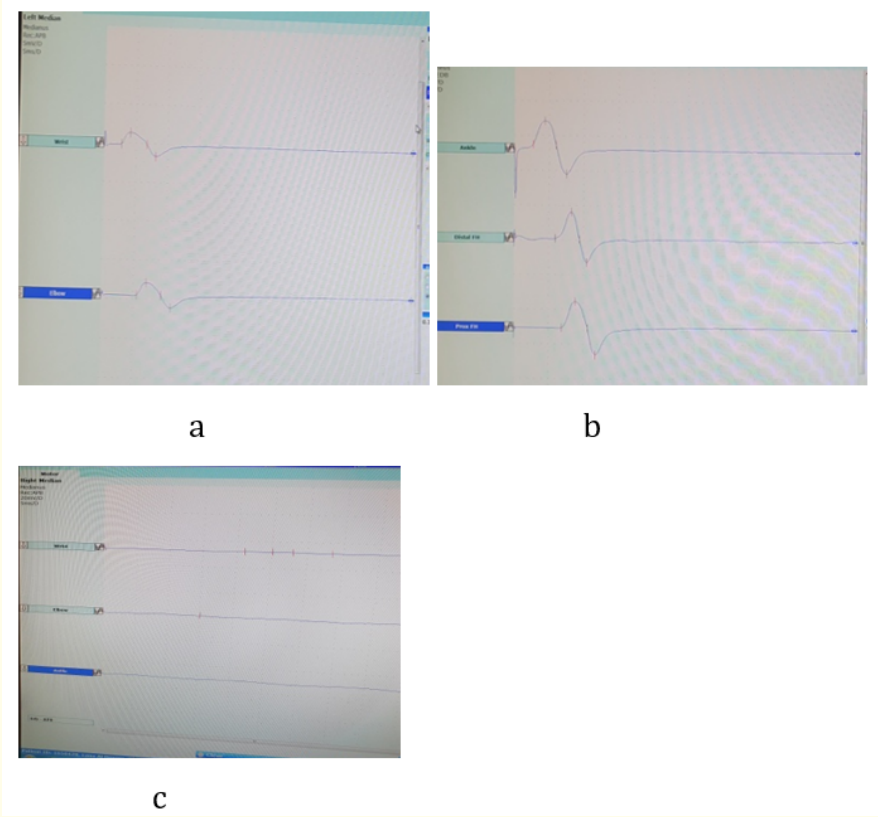

Figure 2: *Showed Nerve conduction study that showed improved function in lower limb compared to upper limb still low voltage with high stimuli > 30. In c) you see the initial study that showed prominent CMAP amplitude reduction was observed in wrist and elbow nerves, complete block in the upper limb.*

Neurological manifestation associate with severe DKA, transient focal cerebral oedema and impaired mental state at presentation with new-onset type 1 diabetes in children was reported 2. We demonstrate that alterations occur most markedly in cerebral white matter, particularly in the frontal lobes, and are most prominent in the youngest children with the most dramatic acidemia.

Other complication hypokalaemic periodic paralysis with normotension, alkaline urine, hypercalciuria and metabolic alkalosis is seen in hyperplasia of the juxtaglomerular apparatus with severe hyperaldosteronism and moderate to a marked increase in renin activity is a well-known complication in diabetes. A tendency for magnesium deficiency in patients with diabetes mellitus is well-established. Hypomagnesemia has been reported in 63.3% of hospitalised diabetic hypokalemic patients [7]. The mechanism responsible for magnesium deficiency in patients with diabetes is not entirely known.

There is a significant relationship between the levels of magnesium and potassium in serum in a patient with acute limb weakness [8]. Whang., *et al*. showed that 42% of hospitalised patients who had hypokalemia also had hypomagnesemia.

There were few cases reported with weakness associated with electrolyte disturbance, but the exact mechanism is unknown [9].

In our patient she had been diagnosed with IDDM and with her presentation to the emergency was treated as DKA but was challenging to control her symptoms possibly due to viral infection that may exacerbate the symptoms. Still, suddenly she develops weakness, her electrolyte was checked daily, there was no abnormality detected that could explain the acute weakness her range of potassium level was 4.2 - 5 mmol/dl. Her CSF analysis was suggestive of meningitis-encephalitis that was the impression of an infectious team. However, all cultures were negative, was treated by antibiotics, but that cannot explain the acute weakness.

We think the abnormal CSF finding can be seen in GBS (pleocytosis and high protein) and that may explain the acute weakness, acute polyradiculoneuropathy with a variable clinical presentation. Strict diagnostic criteria are essential for patient care. Pleocytosis had been seen in GBS patient [9]. Although NCV was not supportive, the nerve conduction test showed an absent response in upper and lower limb even in high frequency [10].

In our patient presented with positive enterovirus was from Nasopharyngeal swab not CSF even though was treated as encephalitis, but can this be complicated with acute flaccid paralysis which is symmetric, not known and not reported before. It is foremost essential to make this recognition since dysphagia along with flaccid paralysis, indicates a high risk of possible respiratory failure and these patients should be monitored closely.

Fever may be seen as a symptom of neurological disease or could have contributed further to the outcome. Therefore, we feel that also in DKA, fever control is of pivotal importance to prevent further damage due to fever.

Our patient treated as acute polyneuropathy, received IVIG in PICU and after discharge given another dose of IVIG. Clinically imported, still mild weakness of left upper limb that showed some progress with physiotherapy, she walking normally, feeding well, her sugar controlled, the right upper limb improved but still a mild distal weakness of the left arm and +1 deep tendon reflex. Again, weakness in a patient with GBS can take time till back to normal function.

The exact aetiology of the paralysis could not be determined. Still, three possibilities were considered, but the most likely is Critical illness polyneuropathy again we should think of another possibility like hypokalemia but in our patient was not the cause, Guillain-Barré syndrome still possible and had been reported before in adult patient. This is the second reported case in general population of acute flaccid weakness as complication in insulin-dependent diabetes, the first one was in Adult [11].

Conclusion

In conclusion, children presenting with DKA may develop fatal neurological sequelae like cerebral oe oedema, although this is rare. Critical illness polyneuropathy and Guillain-Barré syndrome should be considered as a differential in acute weakness with DKA patient with consideration of other possibilities, early management can improve the outcome of the patient.

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