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Case Report

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CHILD WITH TYPE 2 DIABETES MELLITUS COMPLICATED WITH DKA

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ABSTRACT

Background: DKA is a complication that *only* occurs in patients with type 1 diabetes. This is not true. DKA does occur in type 2 diabetes but it is rarely occurs in the absence of a precipitating event. ***Case presentation:*** We present case report describes the first case of an 11-year-old boy with newly diagnosed type 2 DM as the first presentation who also had DKA and symptoms of cerebral edema. We illustrate the clinical course and our experience in managing this case. **Conclusion:** DKA is not common among patients diagnosed with type 2 DM; however, it should not be ruled out. If diagnosed and treated properly, these complications can be easily resolved.

KEY WORDS: Diabetes mellitus, ketoacidosis, cerebral edema, pediatrics

INTRODUCTION

Diabetic ketoacidosis (DKA) is commonly described in individuals with type 1 diabetes mellitus (DM) (Leonid Barski, 2013).[1] However, it can also occur in individuals with type 2 DM during conditions that induce catabolic stress, such as infections, surgery, and trauma, or later in disease progression, in which beta-cell function is lost (Leonid Barski, 2013).[1] DKA results from the relative or absolute deficiency of insulin and an increase in counter-regulatory hormones, such as glucagon, cortisol, catecholamines, and growth hormone (Lee HJ1, 2017).[2]

Cerebral edema (CE) is an uncommon but potentially devastating consequence of DKA. It is far more common among children with DKA than it is among adults (George S Jeha, 2017).[3] Symptoms typically emerge during treatment for DKA, but they may also be present prior to initiation of therapy (George S Jeha, 2017).[3]

This case report describes the first case of an 11-year-old boy with newly diagnosed type 2 DM as the first presentation who also had DKA and symptoms of cerebral edema. We illustrate the clinical course and our experience in managing this case.

CASE REPORT

The patient was an 11-year-old obese boy who was admitted with complaints of dizziness and shortness of breath. Ten days prior to admission, his family noted that he was intermittently confused, was experiencing dizziness, and had a decreased appetite. He had a history of polyuria and polydipsia.

On physical examination, the patient was conscious but agitated with moderate to severe dehydration and rapid, shallow respirations. The source of infection was revealed a small abscess on his right buttock that was discharging pus and had a small area of induration. Additionally, acanthosis nigricans was noted (Figure 1).

The patient's height was 160 cm (>90–95th percentile), weight was 89 kg (>97th percentile), and body mass index was 34.8 kg/m^2 (obese). His respiratory rate was 30 breaths/min, pulse was 124 beats/min, and blood pressure was 175/100 mmHg. He also had a high body temperature (38.8°C, axillary). The patient's characteristics as evaluated at admission can be viewed in

Table 1. The patient was admitted to the pediatric intensive care unit, and insulin infusion with intravenous fluids was initiated.

By the sixth hour of treatment, the patient had become disoriented and showed a decrease in his Glasgow coma scale score. Brain edema was suspected, and mannitol (0.5 g/kg) was immediately administered. He was intubated using the pressure-regulated volume-control mode. Cerebral computed tomography was performed immediately, and it confirmed brain edema (Figure 2).

Despite the continuation of dextrose infusion accompanied by insulin infusion, acidosis persisted. Continuous renal replacement therapy (CRRT) was therefore initiated. By 24 hours following CRRT initiation, the patient showed some improvement. He was weaned from mechanical ventilation and returned to full consciousness.

The patient was slowly weaned off fluid and insulin infusions, and subcutaneous insulin was started to control his serum glucose level. Further investigation revealed that the patient's laboratory values confirmed the presence of type 2 DM based on normal endogenous insulin and negative antibodies (Table 2).

The patient began treatment with oral metformin to improve insulin resistance, which maintained his serum glucose level within the normal range. Finally, he was discharged on oral metformin after showing complete recovery 6 days after admission (4 days in the pediatric intensive care unit for CRRT and 2 days on the pediatric ward with subcutaneous insulin and metformin 500 mg with meals twice daily.).

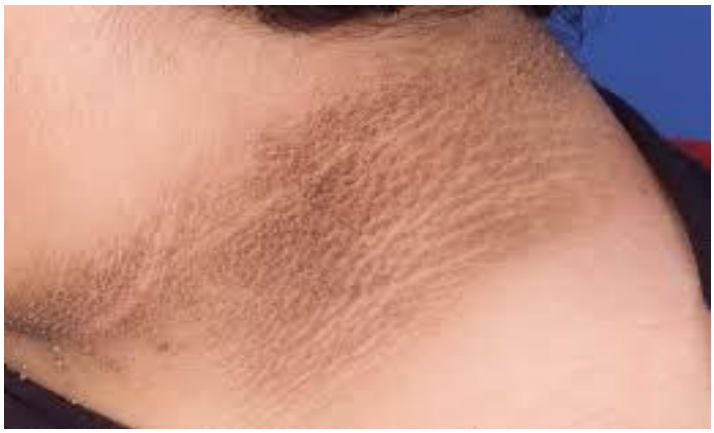

Figure 1.Acanthosis nigricans was noted on the patient's neck at admission.

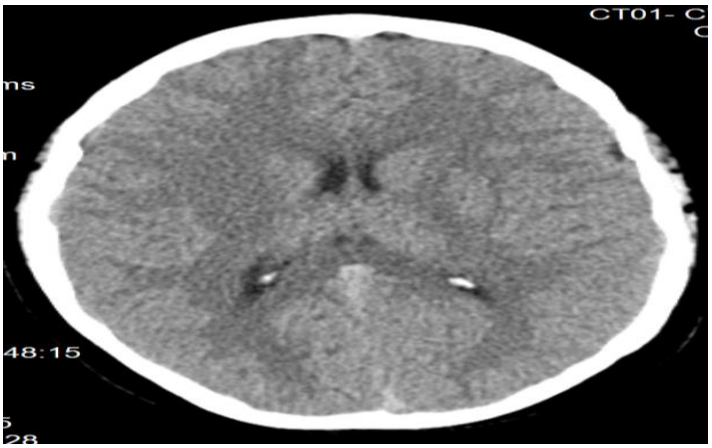

Figure 2.Cerebral computed tomography (CT) of the patient's brain showing no edema or hemorrhage.

Table 1. Laboratory values for parameters tested at admission

Parameter	Laboratory value	Reference range
pH[a]	7.04	7.35-7.45
PCO_2[b] (mmHg)	14.6	35-45
HCO_3[c] (mmol/L)	7.3	22-26
Base excess (mmol/L)	-24	+\-2
Glucose (mmol/L)	27	70-106
Na^{+}[d] (mEq/L)	129	132-142
K^{+}[e] (mEq/L)	3.4	3.5-5
Creatinine (mg/dL)	69	.6-1.1
Urea	3.7	17-43
WBC[f]	15.9	
C-reactive protein (mg/L)	90	<3

[a]PH: potential of hydrogen
[b]PCO_2: partial pressure of carbon dioxide
[c]HCO_3: bicarbonate
[d]Na^{+}: sodium
[e]K^{+}: potassium
[f]WBC: white blood cell count

Table 2.Laboratory values obtained after the patient recovery

Parameter	Laboratory value	Normal range
Insulin (mlU/L)	5.41	(2.60-37.60)
C-peptide (nmol/L)	0.438	(0.26-1.03)
Anti-glutamate decarboxylase (anti-GAD) antibody	Negative	
Anti-islet cell antibody	Negative	
HbA1C[a]	10.7%	4-6%
[a]HbA1C: glycated hemoglobin		

DISCUSSION

American and European retrospective studies have shown that approximately 20% to 30% of patients with DKA had type 2 diabetes (Newton CA1, Diabetic ketoacidosis in type 1 and type 2 diabetes mellitus: clinical and biochemical differences., 2004).[4] DKA is one of the most serious acute metabolic complications of diabetes (Kitabchi AE1, 2009)[5], and it occurs less often in patients with type 2 DM than in those with type 1 DM because these patients are thought to be insulin-resistant rather than insulin-deficient. A study performed in Japan reported an annual incidence of 2.37 cases per 100,000 persons in children aged 0 to 14 years. Therefore, the incidence of DKA in patients with type 2 DM seems to be far lower than that in patients with type 1 DM in the overall diabetic population (Kamata Y1, 2016).[6]

When DKA occurs, common precipitating factors are discontinuation of medication, infection, myocardial infarction, inadequate insulin therapy, or newly diagnosed DM (Association, 2001).[7]

Patients with type 2 DM who present with DKA are more likely to be obese and negative for autoimmune markers (Newton CA1, Diabetic ketoacidosis in type 1 and type 2 diabetes mellitus: clinical and biochemical differences., 2004).[8] We presented the first reported case of type 2 DM with persistent severe DKA and CE in a pediatric patient and reviewed the acute care management of the suspected CE. CE is the most frequent and serious complication of DKA in children. Clinically apparent CE occurs in approximately 1% of childhood DKA cases and is associated with high mortality and neurological morbidity (Faich GA, 1983).[9] The pathogenesis of CE, however, is not understood; investigators have attributed it to cellular swelling as a result of rapid osmolar changes occurring during intravenous infusions. Several studies, however, have shown no relationship with the volume or sodium content of the infusion or any association with the rate of change in serum glucose concentration (Glaser N1 & Pediatrics, 2001).[10] Early detection of CE with proper intervention can prevent permanent brain damage (Edge JA1, 1999).[11]

All patients with suspected CE should be managed in an intensive care unit (ICU). Intravenous (IV) mannitol should be readily available at the bedside to permit prompt treatment if symptoms suggesting CE develop. Treatment for CE consists of the following (JI1., 2014)[12]: Reduced rate of fluid administration (e.g., reduce fluids to the maintenance rate, using an isotonic solution such as normal saline). The dose may be repeated in 2 hours, if there is no initial response. The recommendation for mannitol is based upon the suggested beneficial effect of mannitol in several case reports (Roberts MD1, 2001).[13]

Intubation and mechanical ventilation may be required for patients with impending respiratory failure. However, aggressive hyperventilation (beyond the baseline hyperventilation present in most patients with DKA) is not recommended because it may decrease cerebral blood flow to a sufficient extent to cause cerebral ischemia and actually increase the extent of brain injury from CE in any form (Marcin JP1 & Commitee., 2002).[14]

Imaging with head CT may be useful for excluding other causes of neurologic deterioration (e.g., intracranial hemorrhage or cerebrovascular thrombosis), but should not delay treatment. These other causes are probably rare in this population (JI, 2014).[15]

CONCLUSION

DKA is not common among patients diagnosed with type 2 DM; however, it should not be ruled out. We reported a rare case of an 11-year-old with type 2 DM complicated with DKA and CE. If diagnosed and treated properly, these complications can be easily resolved.

REFERENCES

1. Leonid Barski, M. I. (2013, april). Comparison of Diabetic Ketoacidosis in Patients With Type-1 and Type-2 Diabetes Mellitus. Retrieved from amarican juornal of medical sciences: http://amjmedsci.org/article/S0002-9629(15)30697-2/abstract?cc=y=.
2. Lee HJ1, Y. H. (2017, feb). Factors Associated with the Presence and Severity of Diabetic Ketoacidosis at Diagnosis of Type 1 Diabetes in Korean Children and Adolescents. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/28049242.
3. George S Jeha, M. (2017). Cerebral edema in children with diabetic ketoacidosis. Retrieved from UpToDate: http://www.uptodate.com/contents/cerebral-edema-in-children-with-diabetic-ketoacidosis.
4. Newton CA1, R. P. (2004, sep 27). Diabetic ketoacidosis in type 1 and type 2 diabetes mellitus:

clinical and biochemical differences. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/15451769.
5. Kitabchi AE1, U. G. (2009, july). Hyperglycemic crises in adult patients with diabetes. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/19564476.
6. Kamata Y1, T. K. (2016, Jun 29). Distinct clinical characteristics and therapeutic modalities for diabetic ketoacidosis in type 1 and type 2 diabetes mellitus. Retrieved from PubMed: https://www.ncbi.nlm.nih.gov/pubmed/27499457.
7. Association, A. D. (2001, Jan 24). Hyperglycemic crises in patients with diabetes mellitus. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/?term=American%20Diabetes%20Association%5BCorporate%20Author%5D.
8. Newton CA1, R. P. (2004, sep 27). Diabetic ketoacidosis in type 1 and type 2 diabetes mellitus: clinical and biochemical differences. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/15451769.
9. Faich GA, F. H. (1983, May). The epidemiology of diabetic acidosis: a population-based study. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/6405612.
10. Glaser N1, B. P., & Pediatrics, P. E. (2001, jan 25). Risk factors for cerebral edema in children with diabetic ketoacidosis. The Pediatric Emergency Medicine Collaborative Research Committee of the American Academy of Pediatrics. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/11172153.
11. Edge JA1, F.-A. M. (1999, oct 8). Causes of death in children with insulin dependent diabetes 1990-96. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/10490436.
12. JI, W. (2014). The International Society of Pediatric and Adolescent Diabetes guidelines for management of diabetic ketoacidosis: Do the guidelines need to be modified? Retrieved from uptodate: http://www.uptodate.com/contents/cerebral-edema-in-children-with-diabetic-ketoacidosis/abstract/45?utdPopup=true.
13. Roberts MD1, S. R. (2001, sep 2). Diabetic ketoacidosis with intracerebral complications. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/15016193.
14. Marcin JP1, G. N., & Commitee., A. A. (2002, dec). Factors associated with adverse outcomes in children with diabetic ketoacidosis-related cerebral edema. Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/12461495.
15. JI1., W. (2014, jan 15). The International Society of Pediatric and Adolescent Diabetes guidelines for management of diabetic ketoacidosis: Do the guidelines need to be modified? Retrieved from pubmed: https://www.ncbi.nlm.nih.gov/pubmed/24866064.