Severe Diabetic ketoacidosis in a Newly Diagnosed Child with Type 2 Diabetes Mellitus: A Case Report

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Introduction

Diabetes mellitus (DM) is a chronic metabolic disease that affects carbohydrate, fat, and protein metabolism [1]. Various types of DM could affect children, including type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus (T2DM), Maturity Onset Diabetes of the Young (MODY), and neonatal diabetes (NDM) [2]. The commonest type among children is type 1; however, type 2 incidence is remarkably increasing, which is probably related to increasing obesity among children [3-5]. Type 2 Diabetes mellitus (T2DM) usually affects obese children and adults, especially those having a positive family history. T2DM is characterized by the presence of hyperglycemia with high or normal insulin secretion from the beta cells of the pancreas at early stages of the disease labeled as “insulin receptor resistance” with gradual loss of insulin production with time [6-10]. Patients usually present with milder symptoms of polyuria, polydipsia, polyphagia and loss of weight in comparison to T1DM [11]. Diabetic ketoacidosis (DKA) is characterized by the presence of hyperglycemia, ketosis with ketonuria, and metabolic acidosis. DKA is a common acute complication of T1DM, but could also happen in T2DM. Hyperosmolar hyperglycemic non-ketotic coma is another acute complication of T2DM [12]. Cerebral edema is an uncommon but rather a serious consequence of DKA [13]. It could develop spontaneously or during treatment of DKA, especially during the first 4 to 12 h of initiation of treatment, but still could occur up to 24 h of initiation of treatment [14]. It rarely occurs before initiation of treatment or after 24 h of initiation of treatment. Usually headache, dizziness, agitation are the earliest signs, but other symptoms such as altered level of consciousness, age-inappropriate incontinence or sustained heart rate deceleration are early symptoms that often appears even before remarkably changes on brain computed tomography (CT) [15]. We aimed to report a 12 y old obese child presented with type 2 DM associated with severe metabolic acidosis of DKA and cerebral edema.

Case Report

An obese 12 y old Egyptian boy, previously medically free, presented to the emergency room (ER) of King Abdulaziz university hospital,
with two weeks’ histories of, shortness of breath, polyuria, polydipsia & nocturia and mild weight loss. He was in his usual health state until 14 days’ prior of presentation, when he started to have loss of appetite, fatigability, polyuria, polydipsia & nocturia that waked him up to 6 times per night. He also had palpitations, non-bilious vomiting, with moderate generalized abdominal pain. Initially, there were no histories of a headache, seizures, irritability, loss of consciousness, fever, cough, chest pain, joint pain or skin rash. Unfortunately, his family has not brought him till lately, when his symptoms were deteriorating with a headache; dizziness, agitation and altered level of consciousness with changes in sensorial and cognitive functions were all present at the time of presentation. His nutritional history was of unhealthy “junk” food with no regular exercise “sedentary lifestyle”. He had no other remarkable past medical or surgical histories. His family history revealed, no parental consanguinity, but a positive family history of obesity in parents, his grandfather and many family members have type 2 diabetes. His initial examination revealed severely dehydrated with Kussmaul breathing. Vital signs were the temperature of 36.8°C, heart rate of 124 beats/min, high blood pressure of 175/100, respiratory rate of 30/min with peripheral oxygen saturation of 98%. Weight was 89 kg (+7.57 Standard Deviation Score (SDS)), height was 160 cm (+1.4 SDS), and body mass index (BMI) of 34.77 kg/m² (+3.97 SDS). Neurologically he was agitated with frequent attempts of removing the intravenous cannula, confused, talking with inappropriate words, not age matched. Glasgow coma scale (GCS) of 13/15, normal tone and reflexes. He also had acanthosis nigricans at his neck and thigh folds (Figure 1). His first few hours after admission to the pediatric intensive unit, he became disoriented, GCS deteriorated down to 8/15, with bilaterally reactive pupils, no gag reflex, O₂ saturation started to drop down to 84% on room air, for which patient was ventilated electively (Table 1).

Figure 1: Acanthosis Nigricans on the neck of an obese 12 y old patient with newly diagnosed type 2 diabetes mellitus.

| Parameter                  | At presentation | During hours of admission | Few days of admission | Normal range |
|----------------------------|-----------------|---------------------------|-----------------------|--------------|
| PH                         | 6.997           | 7.02                      | 7.40                  | 7.37-7.45    |
| Serum bicarbonate          | 7.2             | 6.4                       | 23                    | 21-26        |
| Base excess                | -26             | -20                       | -2                    | UP TO 2      |
| O₂ sat %                   | 98              | 84                        | 99                    | 94-100       |
| Random glucose1            | 27              | 18                        | 8                     | 3.9-6.7      |
| Urine ketone               | +3              | +2                        | negative              | negative     |
| Serum sodium1              | 129             | 150                       | 143                   | 136-145      |
| Serum potassium1           | 3.4             | 4.4                       | 3.8                   | 3.5-5.1      |
| Serum chloride1            | 101             | 102                       | 102                   | 98-107       |
| Serum Urea1                | 12.3            | 5.8                       | 3.7                   | 2.5-6.4      |
| Serum Creatinine2          | 140             | 87                        | 80                    | 53-115       |
| HbA1C                      | 10.72%          |                           |                        | 4.2-6.3%     |
| Serum Phosphate1           | 0.71            | 0.82                      | 1.27                  | 0.8-1.58     |
| Serum Albumin3             | 1.01            | 1.02                      | 0.87                  | 0.70-1       |
| Serum magnesium1           | 43              | 40                        | 42                    | 40.2-47.6    |
| Cortisol7                  | 439             |                           |                       | 118.60-618   |
| Insulin like Growth Factor 1 (IGF1)14 | 192    |                           |                       | 143-693      |
| Testosterone7              | 12.6            |                           |                       | 8.4-28.7     |
| White blood cell count (WBC)9 | 9.9         |                           | 7.86                  | 4.5-13.5     |
| Red blood cell count (RBC)10 | 6.44         |                           | 2.93                  | 4-5.40       |
| Hemoglobin (Hb)11          | 15.9            |                           | 7.1                   | 12-15        |
| Hematocrit (HCT) %         | 46.3            |                           | 22.1                  | 35-49        |
| Mean cell volume (MCV)12   | 71.9            |                           | 75.4                  | 80-94        |
| Mean cell hemoglobin (MCH)13 | 24.7        |                           | 24.2                  | 32-36        |
| Platelets (PLT)9           | 350             |                           | 388                   | 150-450      |
| Thyroid stimulating hormone4 | 1.37         |                           | 0.27-4.2              |
| Free Thyroxin5             | 15              |                           | 12-22                 |
| Insulin6                   | 5.41            |                           | 2.60-37.60            |
| Connecting peptide7        | 0.438           |                           | 0.16-1.68             |
| Lactic acid                | 1.2             |                           | 1.2                   | 0.4-2        |
| Amylase8                   | 29              | 29                        | 29                    | 25-115       |
| Lipase8                    | 388             | 388                       | 388                   | 73-393       |
| Serum Calcium1             | 2.42            | 2.39                      | 2.40                  | 2.12-2.52    |
| Islet Cell Autoantibodies  | Negative        |                           |                       |              |
| Glutamic Acid Decarboxylase Autoantibodies | Negative | | |

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The literature review revealed that DKA could occur in patients with T2DM, but usually at a higher blood glucose level than in DKA patients with T1DM. This was the result of a retrospective review in a tertiary center of 69 patients (between 9 and 18 years of age) who presented with DKA, of which 13% had type 2 diabetes mellitus [16]. In another retrospective review of 100 patients, 50 of which had T2DM & the other 50 had T1DM, that aimed to compare the characteristics of youth patients with those types of DM at diagnosis, >25% of type 2 DM had DKA. The study also revealed increased prevalence of obesity, acanthosis nigricans, and hypertension among patients with type 2 DM [17]. Regarding cerebral edema development in DKA patients, a retrospective study that aimed to assess the risk factors for cerebral edema of 61 children with DKA & in whom has developed cerebral edema, has found that children who had low partial pressures of arterial carbon dioxide (PaCO₂) and high serum urea nitrogen concentrations at presentation and who were treated with bicarbonate were at increased risk for cerebral edema [18]. Although some theories have been suggested to be the cause of developing cerebral edema, even with the best application of guidelines, cerebral edema still can occur, thus the best way to prevent it is by preventing the development of DKA [14]. We have reported this case to enrich the knowledge of pediatricians to consider the possibility of type 2 DM in obese children and adolescent presented with DKA. As most of physicians, when they face a child with severe DKA consider type1 DM while in this case severe DKA was a presentation of type 2 DM.

**Conclusion**

Early presentation of children and adolescent with diabetes is crucial to prevent acute complication such as diabetic ketoacidosis. Community awareness of signs and symptoms of diabetes has to be raised up through educational programs, diabetes awareness campaign and school educational tutorials.

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| Insulin Autoantibodies | Negative |
|------------------------|----------|
| 1: (mmol/L) 2: (µmol/L) 3: (g/L) 4: (µIU/L) 5: (Pmol/L) 6: (IU/L) 7: (mmol/L) 8: (U/L) 9: (K/uL) 10: (M/uL) 11: (g/dL) 12: (IL) 13: (pg); 14: (ng/mL) |

Table 1: Values of various parameters at admission.
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