Case Report

Ketogenic Diet-Induced Diabetic Ketoacidosis in a Young Adult with Unrecognized Type 1 Diabetes

Suranut Charoensri,1 Jin Sothornwit,1 Akeatit Trirattanapikul,2 and Chatlert Pongchaiyakul1

1Division of Endocrinology and Metabolism, Department of Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand
2Department of Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand

Correspondence should be addressed to Suranut Charoensri; armsunut@hotmail.com

Received 3 November 2020; Revised 24 January 2021; Accepted 30 January 2021; Published 8 February 2021

1. Introduction

A number of dietary patterns have been proposed as an effective method for weight reduction [1, 2]. Guideline recommendations support several weight-loss approaches that focus on the alteration of macronutrients, including low-fat, low-carbohydrate, and high-protein diets [3]. All of these approaches have resulted in similar clinically meaningful weight loss [4, 5]. The ketogenic diet, a very low-carbohydrate diet and high-fat diet, has emerged as a popular approach, especially for young adults. This diet restricts the daily intake of carbohydrates to less than 50 grams per day, leading to reduced insulin secretion and the conversion of fatty acids to ketone bodies for energy consumption [6, 7]. However, a serious but rare complication of the ketogenic diet is ketoacidosis associated with low carbohydrate intake [8], which should be cautiously monitored in people with a predisposition to the condition, including pregnancy, chronic alcoholism, and type 1 diabetes [9]. Here, we report the case of a healthy young adult who developed diabetic ketoacidosis (DKA) as the first presentation of type 1 diabetes following the course of ketogenic diet.

2. Case Report

A 22-year-old Thai woman with an unremarkable past medical history presented to the emergency department with an acute onset of dyspnea of 2 days’ duration. She was unable to tolerate any food or drink since the symptoms started. On examination, her vital signs included temperature 38°C, pulse rate 110 bpm, blood pressure 110/80 mmHg, and
respiratory rate 32/minute with a kussmaul breathing pattern. Other physical examinations were unremarkable except for severe dehydration. The initial testing revealed the results in Table 1, which included capillary blood glucose of 356 mg/dL, serum bicarbonate of 4.8 mEq/L, arterial pH of 7.15, and a serum beta-hydroxybutyrate level of 6.65 mmol/L, while the lactate level was normal. The urinalysis showed glucosuria and ketonuria. A diagnosis of new-onset diabetes and diabetic ketoacidosis (DKA) was established. However, the type of diabetes was undetermined at that time. Fasting C-peptide level and islet autoantibodies were sent to evaluate for type 1 diabetes.

She was admitted to the intensive care unit and treated with intravenous saline, intravenous insulin infusion, and electrolyte repletion. Blood glucose levels and metabolic acidosis improved. Oral intake and subcutaneous insulin were initiated two days later. Despite a BMI of 22.3 kg/m², the patient felt that she needed to lose weight. A week prior, she initiated an intensive weight management regimen including a ketogenic diet. She had restricted her carbohydrate intake to less than 15 grams per day 4 days. The specifics of her ketogenic meal are shown in Table 2. She denied participating in a heavy exercise which could lead to dehydration. Other precipitating factors of DKA were not identified.

After 6 days of hospital admission, she was discharged with multiple daily injection (MDI) insulin therapy without further complications. One week later, the results of special investigations for identifying the type of diabetes showed C-peptide level 0.2 ng/mL, anti-GAD 8.79 U/mL (normal < 5), and anti-IA2 130.95 U/mL (normal < 7.5), which confirmed the diagnosis of type 1 diabetes in this patient.

### Table 1: Initial laboratory investigations.

| Lab                | Value  | Reference | Units   |
|--------------------|--------|-----------|---------|
| Hemoglobin         | 13.7   | 12.0–14.3 | g/dL    |
| Hematocrit         | 41     | 36.0–47.7 | %       |
| White blood cells  | 15.8   | 4.60–16.0 | k/mm³   |
| Neutrophils        | 82     | 43.7–72.9 | %       |
| Platelets          | 294    | 173–383   | k/mm³   |
| Sodium             | 144    | 136–145   | mEq/L   |
| Potassium          | 3.6    | 3.5–5.0   | mEq/L   |
| Chloride           | 115    | 98–106    | mEq/L   |
| Bicarbonate        | 4.8    | 23–30     | mEq/L   |
| Anion gap          | 24.2   | 3–11      | —       |
| BUN                | 3.8    | 6.0–20.0  | mg/dL   |
| Creatinine         | 0.49   | 0.51–0.95 | mg/dL   |
| Glucose            | 390    | 70–99     | mg/dL   |
| Hemoglobin A1C     | 14     | 4.6–6.5   | %       |
| Lactate            | 1.2    | 0.4–2.0   | mmol/L  |
| Serum ketones      | 6.65   | 0–3       | mmol/L  |
| Urine glucose      | 4+     | Negative  | —       |
| Urine ketones      | 2+     | Negative  | —       |
| Arterial pH        | 7.15   | 7.35–7.45 | —       |

### Table 2: The “ketogenic diet” that the patient had before her illness.

| Day     | Lunch                                        | Dinner                                                |
|---------|----------------------------------------------|-------------------------------------------------------|
| Day 1   | (i) One serving of Thai papaya salad        | (i) Eight servings of spiced duck                     |
|         | (ii) Half serving of fried fish             | (ii) One cup of black coffee                         |
|         | (iii) A coconut                             | (iii) One serving of stir-fried pork with basil leaves (without rice) |
| Day 2   | (i) One serving of Thai papaya salad        | (i) One serving of omelette                           |
|         | (ii) Half serving of fried fish             | (ii) One coconut                                      |
|         | (iii) One cup of black coffee               | (iv) One dish of fried pupa                           |
|         | (iv) One bottle of soda                     |                                                       |
| Day 3   | (i) One serving of Chinese clear soup with braised pork | (i) A tablespoon of avocado                            |
|         | (ii) A coconut                              | (ii) 30 grams of pumpkin seed                         |
|         | (iii) One cup of latte                      |                                                       |

### Discussion

This case demonstrates the development of DKA following a low-carbohydrate, ketogenic diet in a patient with unrecognized type 1 diabetes. It is important for physicians to be aware of this rare potential complication of ketogenic diet, particularly in those with increased risk for type 1 diabetes.

DKA is a life-threatening complication, usually affecting children with newly diagnosed type 1 diabetes [10, 11]. Although the mortality rate is low, DKA is associated with adverse neurological outcomes [12]. The most common
precipitating factors in the development of DKA are infection and discontinuation of insulin in type 1 diabetes [13]. Factors that may lead to insulin omission in young patients include fear of weight gain, fear of hypoglycemia, rebelliousness from authority, and stress of chronic disease [14]. Theoretically, carbohydrate-restricted dietary ketosis may predispose to ketoacidosis in people with type 1 diabetes, particularly in the setting of insulin omission or in the rare situation of undiagnosed type 1 diabetes, not on insulin therapy [9].

Adolescent obesity is a global epidemic with a growing prevalence impacting all socioeconomic groups [15]. However, perceptions of overweight in late adolescence and early adulthood are much more common than being actual overweight. Up to 60% of normal weight young women consider themselves to be overweight in both Western and Asian cultures [16, 17]. These beliefs, whether true or not, are correlated with unhealthy habits, including maladaptive weight-loss strategies [18, 19]. Ketogenic diet is one of the most popular weight-reduction methods in Thailand. Ketosis status from this form of dietary pattern is predicted. However, severe ketoacidosis inducing metabolic acidosis is rare [20]. In patients with near-normal or normal insulin activity, the concentration of plasma beta-hydroxybutyrate during a ketogenic diet should be comparable to those who fasted because the rate of hepatic ketone body synthesis is balanced by ketone utilization and ketone excretion in the urine [9, 21–23]. Therefore, there will be no alteration of the acid-base balance during dieting. On the contrary, serious ketoacidosis may occur in people with lower than normal insulin activity, including type 1 diabetes, pregnant women, alcoholics, and certain diabetic patients currently using sodium-glucose cotransporter-2 inhibitors (SGLT-2i). These patients with higher risk for ketoacidosis should be monitored more closely if choosing to pursue a ketogenic diet. The patient reported in our case had an unrecognized insulin deficiency, combined with a strict ketogenic diet of less than 15 grams of carbohydrates per day, which led to a presentation of DKA. It is important to make individuals aware of this potential complication. In those at higher risk for type 1 diabetes, especially in first-degree relatives of type 1 diabetes patients or other high-risk populations, clinicians should consider screening for overt diabetes prior to patients starting a ketogenic diet, and in those with type 1 diabetes on ketogenic diets, insulin omission should be avoided.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References

[1] U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015–2020 Dietary Guidelines for Americans, http://health.gov/dietaryguidelines/2015/guidelines/.

[2] A. Makris and G. D. Foster, "Dietary approaches to the treatment of obesity," Psychiatric Clinics of North America, vol. 34, no. 4, pp. 813–827, 2011.

[3] M. D. Jensen, D. H. Ryan, C. M. Aposvian et al., "2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults," Circulation, vol. 129, no. 25, pp. S102–S138, 2013.

[4] L. Schwingshackl and G. Hoffmann, "Long-term effects of low-fat diets either low or high in protein on cardiovascular and metabolic risk factors: a systematic review and meta-analysis," Nutrition-Journal, vol. 12, p. 48, 2013.

[5] P. M. Sacks, G. A. Bray, V. J. Carey et al., "Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates," New England Journal of Medicine, vol. 360, no. 9, pp. 859–873, 2009.

[6] E. A. Murphy and T. J. Jenkins, "A ketogenic diet for reducing obesity and maintaining capacity for physical activity," Current Opinion in Clinical Nutrition & Metabolic Care, vol. 22, no. 4, pp. 314–319, 2019.

[7] R. T. Ting, N. Dugrè, G. M. Allan, and A. J. Lindblad, "Ketogenic diet for weight loss," Canadian family physician Medecin de famille canadien, vol. 64, no. 12, p. 906, 2018.

[8] P. Shah and W. L. Isley, "Ketoacidosis during a low-carbohydrate diet," New England Journal of Medicine, vol. 354, no. 1, pp. 97–98, 2006.

[9] J. R. Oster and M. Epstein, "Acid-base aspects of ketoacidosis," American Journal of Nephrology, vol. 4, no. 3, pp. 137–151, 1984.

[10] A. Rewers, F. Dong, R. H. Slover, G. J. Klingensmith, and M. Rewers, "Incidence of diabetic ketoacidosis at diagnosis of type 1 diabetes in colorado youth, 1998-2012," The Journal of the American Medical Association, vol. 313, no. 15, pp. 1570–1572, 2015.

[11] A. Rewers, G. Klingensmith, C. Davis et al., "Presence of diabetic ketoacidosis at diagnosis of diabetes mellitus in youth: the search for diabetes in youth study," Pediatrics, vol. 121, no. 5, pp. e1258–e1266, 2008.

[12] F. J. Cameron, S. E. Scratch, C. Nadebaum et al., "Neurological consequences of diabetic ketoacidosis at initial presentation of type 1 diabetes in a prospective cohort study of children," Diabetes Care, vol. 37, no. 6, pp. 1554–1562, 2014.

[13] M. R. Rewers, "Incidence of diabetic ketoacidosis at diagnosis of type 1 diabetes in colorado youth, 1998-2012," The Journal of the American Medical Association, vol. 313, no. 15, pp. 1570–1572, 2015.

[14] A. Rewers, G. Klingensmith, C. Davis et al., "Presence of diabetic ketoacidosis at diagnosis of diabetes mellitus in youth: the search for diabetes in youth study," Pediatrics, vol. 121, no. 5, pp. e1258–e1266, 2008.

[15] W. H. Polonsky, B. J. Anderson, J. E. Aponte, A. M. Jacobson, and C. F. Cole, "Insulin omission in women with IDDM," Diabetes Care, vol. 17, no. 10, pp. 1178–1185, 1994.

[16] M. Raj and R. K. Kumar, "Obesity in children & adolescents," Indian Journal of Medical Research, vol. 132, pp. 598–607, 2010.

[17] F. Bellisle, M. O. Monneuse, A. Steptoe, and J. Wardle, "Weight concerns and eating patterns: a survey of university students in Europe," International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity, vol. 19, pp. 723–730, 1995.

[18] Y. Wong and Y.-C. Huang, "Obesity concerns, weight satisfaction and characteristics of female dieters: a study on female Taiwanese college students," Journal of the American College of Nutrition, vol. 18, no. 2, pp. 194–200, 1999.
weight perception in female and male adolescents,” *Body Image*, vol. 8, no. 1, pp. 58–63, 2011.

[19] A.-L. Winter, N. A. De Guia, R. Ferrence, and J. E. Cohen, “The relationship between body weight perceptions, weight control behaviours and smoking status among adolescents,” *Canadian Journal of Public Health*, vol. 93, no. 5, pp. 362–365, 2002.

[20] D. Gomez-Arbelaez, A. B. Crujeiras, A. I. Castro et al., “Acid-base safety during the course of a very low-calorie-ketogenic diet,” *Endocrine*, vol. 58, no. 1, pp. 81–90, 2017.

[21] W. S. Yancy, M. K. Olsen, T. Dudley, and E. C. Westman, “Acid-base analysis of individuals following two weight loss diets,” *European Journal of Clinical Nutrition*, vol. 61, no. 12, pp. 1416–1422, 2007.

[22] G. F. Cahill, “Fuel metabolism in starvation,” *Annual Review of Nutrition*, vol. 26, no. 1, pp. 1–22, 2006.

[23] O. E. Owen, P. Felig, A. P. Morgan, J. Wahren, and G. F. Cahill, “Liver and kidney metabolism during prolonged starvation,” *Journal of Clinical Investigation*, vol. 48, no. 3, pp. 574–583, 1969.