

A life-threatening complication of Atkins diet

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In February, 2004, we saw a 40-year-old obese white woman who complained of dyspnoea. 5 days earlier, her appetite had decreased, and she had felt nauseous and had since vomited four to six times daily. She became increasingly short of breath, and presented to us as an emergency. She had strictly followed the low-carbohydrate high-protein Atkins diet, eating meat, cheese, and salads for the previous month. She took vitamins recommended by the diet: chromium picolinate, Atkins Basic3 (multivitamins; Atkins Nutritionals, Inc, USA), Atkins Essential Oils (omega fatty acids), Atkins Dieters' Advantage (electrolytes and extracts), and Atkins Accel (a "thermogenic" formula). As instructed by the original Atkins diet book,¹ she monitored her urine twice daily, with dipsticks strongly positive for ketones. She reported a weight loss of about 9 kg over this 1-month period.

On presentation to the emergency department, our patient was in moderate distress, with a respiratory rate of 20–30 breaths per min. On examination, her bowel sounds were hyperactive and she had mild epigastric tenderness. Otherwise, clinical examination was unremarkable with normal vital signs. Her body-mass index was 41.6 kg/m². Arterial blood gas showed: pH 7.19 (normal 7.40), pCO₂ 3.9 kPa (29 mm Hg; normal 40 mm Hg), and pO₂ 15.7 kPa (118 mm Hg; normal 100 mm Hg). The anion gap was high at 26 mmol/L and bicarbonate was low at 8 mmol/L (table). Serum glucose was 4.2 mmol/L; lipase was high at 326 U/L with normal amylase. White blood cell count was high at 13×10⁹/L. Urine analysis confirmed ketonuria. She was admitted to the intensive care unit, and was given an infusion of 5% dextrose with 150 mmol/L sodium bicarbonate at a rate of 30 mL/h. White blood cell count was normal on day 2; lipase increased to a peak of 848 U/L with a normal amylase, and returned to normal by day 3. Abdominal CT on admission showed a normal pancreas. Serum bicarbonate increased to 26 mmol/L by day 3. The patient was discharged without follow-up on day 4. She was asymptomatic without health problems when we spoke to her 3 months and 18 months later on the telephone.

The differential diagnosis of high-anion-gap metabolic acidosis includes ingestion of methanol, ethylene glycol, or salicylate, L- or D-lactate acidosis, and ketoacidosis due to diabetes mellitus, alcohol, or starvation. Our patient denied alcohol use; her serum osmolar gap was 0, which excludes the presence of unmeasured osmotic agents such as methanol or ethylene glycol; L-lactate

Test	Result	Normal
Anion gap	26 mmol/L	10–14 mmol/L
Sodium	144 mmol/L	136–145 mmol/L
Potassium	4.8 mmol/L	3.5–5 mmol/L
Chloride	110 mmol/L	96–106 mmol/L
Bicarbonate	8 mmol/L	24–30 mmol/L
Blood urea nitrogen	3.2 mmol/L	1.8–5.4 mmol/L
Creatinine	106 µmol/L	44–133 µmol/L

Table: Laboratory blood test results on admission

concentration was normal; and salicylate was undetectable. D-lactate acidosis was unlikely without antibiotic use or bowel surgery. Serum was positive for acetone, and β-hydroxybutyrate was high at 390 µg/mL (normal 0–44 µg/mL), consistent with ketoacidosis. Ketones are produced in the liver whenever there is decreased insulin during starvation. A low-carbohydrate diet such as Atkins can lead to ketone production;² in fact, the Atkins diet book recommends regular monitoring for ketonuria to confirm adherence to the diet.¹ Ketoacidosis has been described when this type of diet has been used to treat refractory childhood epilepsy.³ Stress may also worsen starvation-induced metabolic acidosis.⁴ We did not locate reports about such potentially life-threatening metabolic acidosis in healthy adults on the Atkins diet.⁵ In our patient, dehydration could have occurred from glycogen-bound water depletion and vomiting. However, this form of dehydration usually causes metabolic alkalosis in the absence of concomitant renal failure. Our patient had an underlying ketosis caused by the Atkins diet and developed severe ketoacidosis, possibly when her oral intake was compromised from mild pancreatitis or gastroenteritis. This problem may become more recognised because this diet is becoming increasingly popular worldwide.

References

- 1 Atkins RC. Dr Atkins' diet revolution. New York: Bantam Books, 1972.
- 2 Brehm BJ, Seeley RJ, Daniels SR, D'Alessio DA. A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. *J Clin Endocrinol Metab* 2003; **88**: 1617–23.
- 3 Ballaban-Gil K, Callahan C, O'Dell C, Pappo M, Moshe S, Shinnar S. Complications of the ketogenic diet. *Epilepsia* 1998; **39**: 744–48.
- 4 Toth HL, Greenbaum LA. Severe acidosis caused by starvation and stress. *Am J Kidney Dis* 2003; **42**: E16–19.
- 5 Astrup A, Larsen TM, Harper A. Atkins and other low-carbohydrate diets: hoax or an effective tool for weight loss? *Lancet* 2004; **364**: 897–99.