

CASE REPORT

Euglycemic diabetic ketoacidosis associated with uremia

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Abstract

Regular treatment of DKA patients is not feasible in cases associated with Uremia because of being urineless and prone to heart failure of these patients. Our results indicated that continuous renal replacement therapy is a better alternative.

KEYWORDS

DKA, euglycemia, uremia

1 | INTRODUCTION

As an acute complication of diabetes, diabetic ketoacidosis (DKA) is typically characterized with hyperglycemia (16.7–33.3 mmol/L, >55.5 mmol/L in rare case), elevated serum ketones, and acidosis.¹ However, DKA patients with normal blood glucose concentrations (<16.7 mmol/L) and low bicarbonate levels (<10 mmol/L) had also been reported, the pioneer of which is Munro et al in 1973.²

The optimal emergence treatment of DKA is fluids supply, which can increase blood volume and restore efficient influx of organs. However, in regarding to patients at the last stage of diabetic nephropathy relying on sustainable hemodialysis, rapid and large amount of fluids supply is not feasible since they have no urine and can easily get heart failure. In this group of patients, normal fluids/blood supply can only correct the acidosis for short time. Ketones in tissues will enter the blood and cause acidosis in 2–3 hours.

For these cases, continuous renal replacement therapy (CRRT) is a better option in many ways, including large amount fluids exchange, increased influx of organs, clearance of keto acids, lactic acid and inflammatory mediator in tissues, enhanced efficiency of essential molecules in blood, improved microcirculation, remission of tissue hypoxia. Up to now, there is no report of the treatment of DKA associated with uremia.

Our case report here indicate that normal dialysis could not correct DKA associated with uremia, while CRRT is a better available option.

2 | CASE REPORT

We describe a case of a 26-year-old female with type I diabetes (T1DM) of more than 10-years duration. This patient presented with severe abdominal pain started 1 day ago and had been performing continuous ambulatory peritoneal dialysis for 1 year. Ten years ago when this patient was diagnosed with T1DM after showing polydipsia without obvious trigger, she was treated with insulin injection. One year ago, this patient was hospitalized for uremia (late stage of chronic renal failure). Peritoneal dialysis catheter placement was performed and followed with regular peritoneal dialysis, after which this patient's clinical evolution was favorable.

The patient was hospitalized because of a 24-hours history of durative blunt abdominal pain. The associated symptoms included mushy stool diarrhea and cloudy peritoneal dialysate fluid. The patient had no sign of dark stool, chills, or fever. On physical examination, the patient showed signs of moderate anemia-like paleness. The patient was alert, with vital signs in normal range which include temperature at 36.5°C, pulse rate at 80 bpm, respiratory rate at 20 bpm,

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blood pressure at 100/60 mm Hg. The physical examination showed no jaundice-yellow with skin, sclera, and mucous membranes, no swelling of superficial lymph nodes. The breath sounds were clear without obvious adventitious sounds like dry/moist rales. Abdominal swelling caused by accumulation of fluid was observed. There was no observation of hepatosplenomegaly, tenderness, or rebound tenderness, as well as peripheral edema. The patient showed normal reflex action and no sign of pathological spread of reflexes.

The blood test results when hospitalized were as follows, leukocytes $11.8 \times 10^9/L$, hemoglobin 65 g/L, C-reactive protein (CRP) 140.94 mg/L, albumin 19.1 g/L, creatinine 528.0 $\mu\text{mol/L}$, blood glucose 15.0 mmol/L, and potassium 2.58 mmol/L. The peritoneal fluid was cloudy, and the analysis of which showed leukocytes at 80% and nucleated cell count at $2.93 \times 10^9/L$. The suspected bacterial infection was treated with intravenous and peritoneal administration of cefazolin and levofloxacin lactate upon hospitalization. Measures were taken to adjust the levels of blood glucose and blood pressure, correct anemia, maintain the level of electrolytes and pH balance. On Oct 18th, the drug resistance result from culture of peritoneal dialysis fluid indicated the infection of *Candida dubliniensis*. Treatment was then switched to intravenous Fluconazole and intraperitoneal influx of antifungal medication. At the meantime, peritoneal dialysis catheters were removed and blood dialysis was performed. On Oct 25th, the abdominal pain got worse and the patient showed obvious tenderness and rebound tenderness, which indicates intestinal obstruction. The intake of water and food was forbidden and gastrointestinal decompression was performed. In addition to the antifungal medication, Piperacillin/Tazobactam was prescribed against the infection. At 12 AM of Oct 29th, the patient felt chest tightness and shortness of breath. The blood pressure was taken at 80/50 mm Hg. The patient was in extremely low spirits. Obvious moist rales were noticed. Test of blood dialysis from Oct 27th read BNP > 10 000, CO_2CP 6.4 mmol/L, blood glucose 15 mmol/L. Beside the supply of fluid, dopamine was given to correct the risk of shock. Dialysis was emergency performed against acidosis. The CRP level was measured again at 175 mg/L, calcitonin at 4.48, leukocytes count at $16.67 \times 10^9/L$. Again, the abdominal pain got worse and the anti-infection reagent was switched to Meropenem. At noon, CO_2CP read at 6.4 mmol/L, pH at 7.17. Dialysis was performed in the afternoon and again on Oct 30th. On Oct 31st, CO_2CP read at 9.7 mmol/L, blood glucose at 15 mmol/L (hypoglycemia). Multiple blood gas tests indicated metabolic acidosis, whereas the level of lactic was normal. The blood glucose level increased although not as significant as seen in DKA. No urine was available to measure the level of ketones, and it is possible that urine ketones could be negative in DKA associated with chronic

kidney disease (CKD). Other local hospitals did not have the ability to test the blood ketones either. So the dialysis fluid was used as urine to test for ketones, which showed positive. This patient was then diagnosed as ketoacidosis, and CRRT was performed accordingly. Followingly, the acidosis was corrected and the blood sample was confirmed with ketoacidosis after sending out for test.

3 | DISCUSSION

Previous study indicate that DKA can be precipitated by infection.^{1,3} In our case, the patient was confirmed with infection of *Candida dubliniensis*. At the meanwhile, the patient also showed signs of intestinal obstruction, so we took measures of fasting, which could be another cause for DKA.^{3,4} Our case also indicated that in patients of DKA associated with uremia, ketones analysis could be performed using the dialysis fluid in case of no urine available. Most importantly, our report suggested that for DKA associated with uremia patients, hemodialysis was not able to correct acidosis. In our case, management strategies for DKA should be modified, which is consistent with previous findings that current management strategies for DKA is not suitable to patients with end-stage renal disease (ESRD).⁵ Moreover, consistent with previous reports,^{6,7} our example provided more evidences that CRRT can be an optimal strategy to treat DKA associated with uremia, which can serve as an alternative approach for DKA patients associated with ESRD.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest in association with the present study.

AUTHOR CONTRIBUTIONS

MZ and LL analyzed the data and wrote the paper. HX and HF assisted with the data collection and analyze. All authors read and approved the final manuscript.

ETHICAL APPROVAL

Informed consent was obtained from the patient regarding the report of her clinical scenario in an anonymous way.

DATA AVAILABILITY STATEMENT

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

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