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The use of continuous hemodiafiltration in a patient with diabetic ketoacidosis

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Abstract A variety of fatal complications are associated with diabetes mellitus. Among these, diabetic ketoacidosis (DKA) figures largely in fatalities in young diabetics. Although hyperosmotic diuresis in DKA causes extreme fluid loss, acute renal failure is less common than expected in DKA. We treated a case of severe DKA with associated coma, acute respiratory failure, and acute renal failure in a 24-year-old man who had been diagnosed with type 1 diabetes mellitus at age 19. The comatose patient had been intubated before transfer to our hospital for intensive care. Despite infusion with isotonic saline and insulin, metabolic acidosis was refractory. On day 2, urine output decreased and pulmonary congestion developed, so we started continuous veno-venous hemodiafiltration (CVVHDF), which was effective against the metabolic acidosis; urine output increased gradually. CVVHDF was withdrawn on day 7, and the patient's renal function recovered completely. He was discharged from the intensive care unit (ICU) on day 14.

Key words Diabetes mellitus · Complications · Continuous veno-venous hemodiafiltration

Introduction

Diabetic ketoacidosis (DKA) may occur in patients with poorly controlled diabetes mellitus (DM). Pneumonia, gastroenteritis, and urinary tract infection, as well as other infections, most commonly precipitate DKA. Other stressors, including emotional problems, hot weather, trauma, pancreatitis, and alcohol or drug abuse may also trigger DKA. The clinical symptoms of DKA depend on the degree of hyperosmolality, volume depletion, and metabolic acidosis. Generally, there is fluid loss of 5 to 7l, and increased serum creatinine and urea nitrogen. It is rare, however, for the condition to become so severe that measures to redress acute renal failure are required. Although the reason is not clear, hyperosmotic diuresis polyuria seems to mitigate the development of acute renal dysfunction. Nevertheless, DKA can precipitate acute renal failure. We report here our experience with a comatose patient with DKA with refractory metabolic acidosis and acute failure of the respiratory system and kidneys. Continuous veno-venous hemodiafiltration (CVVHDF) effectively treated the renal failure and refractory metabolic acidosis in this patient.

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Case report

A comatose 24-year-old man (height, 168 cm; weight, 100 kg), who had, 5 years earlier, been diagnosed with type 1 DM, was admitted to our hospital with respiratory insufficiency and acute renal failure due to DKA, requiring intensive care. Although he was using insulin, compliance with the treatment was poor (HbA1c, 10.8%). The onset of the present episode was marked by loss of appetite and vomiting after he contracted a common cold. He initially sought treatment at another hospital and was admitted. Laboratory tests indicated severe metabolic acidosis (pH, 6.844; HCO_3^-, 3.8 mmol·l^{-1}; base excess (BE), 29.6 mmol·l^{-1}; and anion gap, 38.7 mmol·l^{-1}), hyperglycemia (484 mg·dl^{-1}), and renal dysfunction (urea nitrogen, 16.1 mg·dl^{-1}; creatinine, 1.62 mg·dl^{-1}). Upon diagnosis of DKA, isotonic saline and insulin were administered. The next day, he complained of shortness of breath and showed symptoms of hypoxia before falling into a coma. The trachea was intubated and mechanical ventilation was started. Subsequently, he was transferred to our hospital.

Table 1 shows the laboratory results for samples taken after admission to our hospital. On day 1, although his blood glucose level was 183 mg·dl^{-1}, blood gas analysis indicated metabolic acidosis. Chest X-ray

Table 1. Laboratory test results

	Day 1	Day 2	Day 3	Day 5	Day 7	Day 12
Potassium (mmol·l^{-1})	4.5	3.9	2.8	3.4	3.6	4.2
Urea nitrogen (mg·dl^{-1})	19	25	22	23	25	24
Creatinine (mg·dl^{-1})	2.06	3.93	4.35	3.69	3.09	1.00
Urinalysis						
Glucose	2+	+		±	–	2+
Protein	4+	+		+	+	2+
Ketone body	2+	–		–	–	–
Glucose (mg·dl^{-1})	184	210	104	238	208	102
HbA1c (%)	10.0					
Blood gas						
PH	7.259	7.093	7.320	7.375	7.472	7.536
$PaCO_2$ (mmHg)	20.9	21.6	31.7	47.8	38.8	40.0
BE (mmol·l^{-1})	–16.1	–21.6	–9.0	1.8	3.3	9.7
HCO_3^- (mmol·l^{-1})	8.4	6.5	6.5	27.3	28.4	33.1
CCr (ml·min^{-1})	53.5	8.2		19.8	63.2	

BE, base excess; CCr, creatinine clearance

revealed focal infiltration in the left lung field. White blood cell count and C-reactive protein level were elevated, and antibiotics (cefmetazole sodium, 2 g·day^{-1} and clindamycin 0.6 g·day^{-1}) were administered. Insulin was infused at 8–10 U·h^{-1}, and acetate Ringer's solution was given at 1000 ml·h^{-1} for the first hour, followed by 500 ml·h^{-1} during the next 2 h. Fluid replacement with isotonic saline was continued, and the metabolic acidosis seemed to be ameliorated. Blood pressure, heart rate, and central venous pressure were all within control parameters. On day 1, urine output did not fall and, in the evening, urine was negative for ketonuria. Urine output progressively decreased, however, in spite of the fluid replacement therapy, and the metabolic acidosis worsened. On day 2, general edema developed, and a chest X-ray revealed pulmonary edema. Brain computed tomography (CT) did not reveal any abnormalities. The serum creatinine value was increased, to 3.93 mg·dl^{-1}, so we applied CVVHDF at the following settings: blood flow, 100 ml·min^{-1}; replacement volume, 20 ml·min^{-1}; and dialysate, 10 ml·min^{-1}. The metabolic acidosis was corrected after the initiation of CVVHDF (Fig. 1). On day 6, urine output had increased to 1000 ml·day^{-1}, and CVVHDF was withdrawn on the next day. On subsequent days, urine output continued to increase (Fig. 2). As the patient's renal function returned, respiratory functions also gradually recovered. Tracheal extubation was carried out on day 10. After showing completely uncomplicated recovery of renal function, the patient was discharged from the ICU on day 14.

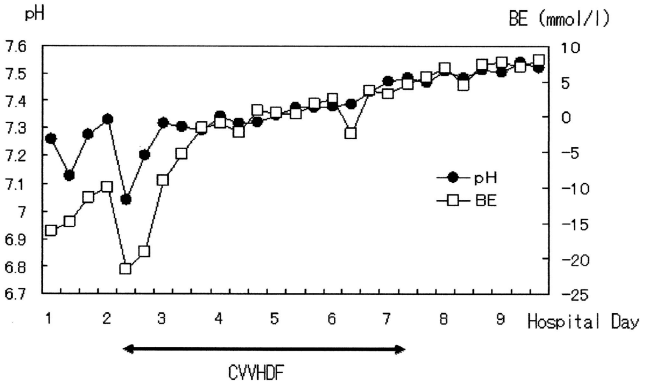

Fig. 1. Time course of metabolic acidosis during patient's stay in the intensive care unit (ICU). The metabolic acidosis was corrected after the initiation of continuous veno-venous hemodiafiltration (*CVVHDF*). CVVHDF was withdrawn on day 7. *BE*, base excess

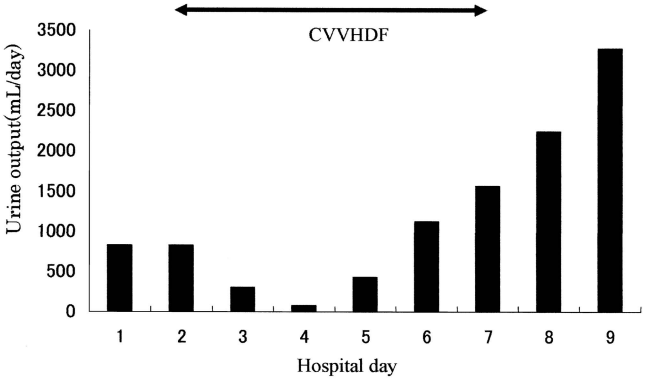

Fig. 2. Patient's urine output while in the ICU. Urine output increased after the initiation of continuous veno-venous hemodiafiltration (CVVHDF). On day 6, urine output had increased to 1000 ml·day^{-1}, and CVVHDF was withdrawn on the following day

Discussion

We treated a comatose patient who had refractory metabolic acidosis, acute respiratory failure, and acute

renal failure due to DKA. CVVHDF was effective in treating the severe metabolic acidosis: the patient returned to consciousness and, marking renal recovery, residuals were no longer detected in urine samples.

DM can bring about a variety of fatal complications. Myocardial infarction and cerebrovascular diseases are the commonest causes of death for DM sufferers. These complications are usually related to age and the duration of DM. In younger patients, DKA is associated with a large number of fatalities. Even when 71 of fluid are lost due to hyperosmotic diuresis in DKA [1,2], however, acute renal failure is rare. Soler et al. [3] have reported 16 deaths in 258 patients with DKA: only 1 of these patients developed acute renal failure. Similarly, Biegelman [4] reported 2 cases of acute renal failure in 32 deaths among 340 patients with DKA and Tunbridge [5], concluding that 74 of 448 deaths in diabetics under age 50 were due to ketoacidosis, reported that only 2 of these patients had developed acute renal failure.

The factors that contribute to acute renal failure in DKA are not clear. Some authors have concluded that rhabdomyolysis contributes to the development of the acute renal failure, because most patients with acute renal failure show symptoms of rhabdomyolysis. Plasma creatinine phosphokinase (CPK) results for our patient were within the normal range, so rhabdomyolysis was not likely to have been implicated in his acute renal failure. The metabolic acidosis continued in spite of good glucose control, because of severe cell damage. We assumed that CVVHDF had been effective in removing the harmful substances released from the damaged cells.

Our patient had been living alone and, because of a personality problem, complied only poorly with the necessary treatment for DM. In the episode reported here, onset was marked by loss of appetite and vomiting. On admission for intensive care, he was comatose and suffering from refractory metabolic acidosis and renal dysfunction. In spite of fluid replacement, his urine output decreased and serum creatinine and urea nitrogen values increased. To compensate for the decreasing urine output, we increased the rate of fluid infusion, and this led to general edema and pulmonary congestion. While the reason for these manifestations was not clear, it was likely that he had vitamin B1 deficiency and Wernicke's encephalopathy, because of his lifestyle.

We were concerned not to bring about a disequilibrium syndrome that would worsen the coma, so, because the osmolarity change with CVVHDF is slower than with hemodialysis, we selected CVVHDF as the renal replacement therapy. The optimal timing of CVVHDF is not clear. In general, however, early introduction and high-volume replacement are most effective for the recovery of renal function. We initiated CVVHDF when urine output decreased.

To treat our patient's severe DKA, adequate fluid replacement and plasma glucose control with insulin infusion were essential. With these procedures, and the early introduction of CVVHDF, we effectively treated the patient's refractory metabolic acidosis, and he was discharged with no remaining complications.

References

1. Kitabchi AE, Umpierrez GE, Murphy MB, Barrett EJ, Kreisberg RA, Malone JI, Wall BM (2001) Management of hyperglycemic crises in patients with diabetes. Diabetes Care 24:131–153
2. Chiasson JL, Aris-Jilwan N, Belanger R, Bertrand S, Beauregard H, Ekoe JM, Fournier H, Havrankova J (2003) Diagnosis and treatment of diabetic ketoacidosis and the hyperglycemic hyperosmolar state. CMAJ 168:859–866
3. Soler NG, Bennett MA, FitzGerald MG, Malins JM (1973) Intensive care in the management of diabetic ketoacidosis. Lancet 7810:951–954
4. Biegelman PM (1971) Severe diabetic ketoacidosis (diabetic "coma"): 482 episodes in 257 patients; experience of 3 years. Diabetes 20:490–500
5. Tunbridge WM (1981) Factors contributing to deaths of diabetics under fifty years of age. On behalf of the Medical Services Study Group and British Diabetic Association. Lancet 8246:569–572