CASE REPORT

Challenging Management of Refractory Metabolic Acidosis and Acute Kidney Injury in a Child with Diabetic Ketoacidosis

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Abstract

Diabetic ketoacidosis (DKA) is the most serious complication of type I diabetes mellitus (DM) in children. Majority of these patients respond to fluid resuscitation, insulin, and supportive measures and rarely require renal replacement therapy. Here, we report the case of a young girl with DKA with severe refractory metabolic acidosis and acute kidney injury (AKI) and was successfully managed with renal replacement therapy.

Keywords: Hemodialysis, Peritoneal dialysis, Type I diabetes mellitus.

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Introduction

Diabetic ketoacidosis (DKA) is a leading cause of hospitalization, morbidity, and mortality in children. It occurs due to absolute or relative insulin deficiency and is defined as hyperglycemia, metabolic acidosis, and ketosis along with fluid-depleted state.[1] Metabolic acidosis in DKA occurs due to the production of keto-anions via hepatic fatty acid oxidation, the production of lactate due to adrenergic response to fluid depletion, and the accumulation of unmeasured anions due to acute kidney injury (AKI).[2] Metabolic acidemia leads to decreased cardiac contractility, affects oxygenation, and results in vital organ dysfunction. Therefore, it is important to correct the acidosis along with fluid correction and insulin therapy in these patients.[3,4] Due to volume depletion, prerenal AKI occurs in patients with DKA which generally is mild and resolves completely, while in some patients it is severe that it results in damage to renal parenchyma,[5] as in our patient who had severe AKI and required hemodialysis.

Case Description

A 13-year-old girl presented with abdomen pain and vomiting for 4 days and difficulty in breathing for the past 2 days. At presentation, she had tachypnea, tachycardia, low pulse volume, and hypotension. Her blood glucose level was high (>500) and urinary ketones (4+) were present. Blood gas analysis showed severe metabolic acidosis with an increased anion gap, and hence, diagnosis of type I diabetes mellitus (DM) with severe DKA was made. Other investigations showed serum sodium 145 mmol/L, potassium 4.6 mmol/L, urea 15 mg/dL and creatinine 0.9 mg/dL, hemoglobin 13 g/dL, platelet count 3.3 lac, and leukocyte count 22,000. Initially, fluid resuscitation (bolus at 20 mL/kg) followed by maintenance fluid as per Milwaukee Protocol (i.e., IVF volume for next 23 hours = 85 mL/kg + 100 mL/kg—bolus) and insulin @ 0.1 units/kg/hour were started. Repeated per abdominal examinations for hepatomegaly to avoid fluid overload were done. Fluid-electrolyte therapy and insulin infusions (later increased to 0.2 units/kg/hour) were dynamically adjusted according to the blood glucose and electrolyte values, but severe metabolic acidosis persisted (Table 1). All mechanical errors, related to drug or treatment charting or administration of fluids and insulin, were checked and were ruled out. Further, the clinical condition of the child worsened and she developed signs of shock and AKI. Hence, she was intubated and started on mechanical ventilation. Also, vasopressors (dopamine and adrenaline), intravenous antibiotics, and sodium bicarbonate were started. In view of persisting metabolic acidosis, AKI, and sick condition, peritoneal dialysis (PD) was started at around 38 hours of admission. After 12 hours of PD, improvement in blood pH and bicarbonate was observed (Table 1); hence, PD was continued for 72 hours after which blood pH was normalized (Table 1) and shock was resolved gradually over few days. The child was extubated on day 8 of admission. But the child further required 4 sessions of hemodialysis for persistent anuria and rising serum creatinine levels. Later, her urine analysis showed fungal infection for which she received antifungals for 14 days. The child was discharged after 1 month of hospital stay with normal creatinine levels. Now, the child is under regular follow-up and is doing well.

Discussion

As we know that fluid correction and insulin are the cornerstone treatments for DKA, however, few patients do not respond to this initial treatment. Refractory metabolic acidosis in such patients is detrimental for the vital organs and further leads to catecholamine refractory shock.[3,4] The role of bicarbonate is not clearly defined in these patients as shown in a systematic review done by Chua et al.[6] Our patient also did not respond to bicarbonate therapy, and hence, PD was started and the patient responded well. There are few case reports in which they have shown a rapid reversal of acidemia with renal replacement therapy in DKA in adult patients.[7,8]

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Table 1: Serial pH, HCO_3^-, electrolytes, and RFTs of patient

	pH	*HCO_3^-*	*Corrected sodium*	*Potassium*	*Chloride*	*Urea*	*Creatinine*
At admission	6.950	1.8	151	4.6	120	15	0.9
18 hours	6.934	1.7	167	2.4	123	33	3.0
At the time of starting PD	7.063	4.2	165	2.7	127	55	3.7
12 hours of PD	7.151	10.6	135	3.0	129	100	5.0
36 hours of PD	7.237	10.8	145	4.0	120	47	2.5
60 hours of PD (hemodialysis started)	7.374	14.6	144	4.5	116	154	6.5
After 4 sessions of hemodialysis	7.430	23.4	142	4.2	105	51	1.2

Acute kidney injury in these patients is another cause of increased morbidity and mortality and is suggested to have long-term implications on renal function, which is now been diagnosed early and managed with renal replacement therapy.[5,9,10]

Conclusion

Peritoneal dialysis should be considered as an early intervention in children with DKA in whom acidemia persists after fluid and insulin therapy. Also, renal functions should be monitored closely and AKI in such patients should be managed early by initiating renal intervention to prevent poor outcomes.

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