CASE REPORT

Indian Journal of Diabetes and Endocrinology
Volume 7 Number 1/January – June 2025

Successful Outcome of Severe Diabetes Ketoacidosis in an Adult with Cerebral Edema, Acute Renal Failure, and Rhabdomyolysis: Case Report

Manoj Agrawal[1], Vibha Yadav[2], Rishi Shukla[3], Neha Agrawal[4]

HOW TO CITE THIS ARTICLE:

Manoj Agrawal, Vibha Yadav, Rishi Shukla, et al. Successful Outcome of Severe Diabetes Ketoacidosis in an Adult with Cerebral Edema, Acute Renal Failure, and Rhabdomyolysis: Case Report. Ind Jl of Diabetes and Endo. 2025; 7(1): 25-28.

ABSTRACT

Diabetic ketoacidosis (DKA) is the most serious hyperglycaemic emergency seen in patients with type 1 diabetes mellitus (T1DM). Treatment associated complications such as cerebral edema, acute renal failure, rhabdomyolysis, and dyselectrolytemia further increase the risk of morbidity and mortality. Cerebral edema is the principal cause of mortality in DKA (in children) while rhabdomyolysis with acute renal failure is rarely reported, particularly in adults. Lack of awareness of these complications leads to delays in therapeutic intervention. We herein report the successful management of a 20-year-old male who presented with Severe DKA with cerebral edema, hypophosphatemia, rhabdomyolysis, acute renal failure, and persistent metabolic acidosis.

KEYWORDS:

• Acute renal failure • Cerebral edema • Case report • Diabetes ketoacidosis in Adults • Rhabdomyolysis

INTRODUCTION

Cerebral edema and rhabdomyolysis are rare complications that occur majority in children less than 3 years of age with severe diabetic ketoacidosis.[1] These complications are very well described in various literature for the paediatric population but are not well understood in adults except in some case reports,[2,3] The incidence of cerebral edema in DKA in the pediatric population is 0.5-1% while the adult is less than 0.03% in DKA admitted patients. When it is associated with acute renal failure then treatment becomes very challenging.

AUTHOR'S AFFILIATION:

[1] Fellow, Department of Pediatric Endocrinology, Regency Hospital, Kanpur, Uttar Pradesh, India.
[2] Fellow, Department of Pediatric Endocrinology, Regency Hospital, Kanpur, Uttar Pradesh, India.
[3] Consulatant Endocrinologist, Regency Hospital, Kanpur, Uttar Pradesh, India.
[4] Fellow, Department of Pediatric Endocrinology, Regency Hospital, Kanpur, Uttar Pradesh, India.

CORRESPONDING AUTHOR:

Manoj Agrawal, Fellow, Department of Pediatric Endocrinology, Regency Hospital, Kanpur, Uttar Pradesh, India.
E-mail: jmanoj957@gmail.com

➢ **Received:** 03-06-2025 ➢ **Accepted:** 19-07-2025

We present a case of a 20 years old adult who presented to us with severe diabetes ketoacidosis, refractory shock, hypokalaemia, hypophosphatemia, and microscopic hematuria with an acute renal injury who was successfully discharged after 10 days of treatment. This highlights the importance of the identification of rare complications of diabetic ketoacidosis and treatment at the right time can prevent mortality.

Case Summary: A 20-year-old male who brought to the emergency room with unconsciousness and severe metabolic acidosis and respiratory distress. He has lost weight of 5 kg in the last one months with a history of polyuria, and polydipsia with an acute history of vomiting for the last 2 days.

On Initial examination, the patient was unconscious, had a severe dehydration, dry mucosa, capillary oxygen saturation (CRT) of more than 2 seconds, a saturation of 95 percent, and a pulse rate of 40 beats/min with a blood pressure of 160/90 mmHg. Respiratory rate was 34 breaths/minute with Kussmaul"s breathing.

The Initial blood glucose was 270 mg/dl with blood ketone (beta-hydroxybutyrate) level of 6.4 mmol/l, dyselectronemia (Blood investigation outlined in Table 1)

Given clinical and biochemical findings, a diagnosis of severe DKA with cerebral edema was kept. Normal saline bolus at 20 ml/kg was given over one hr, followed by maintenance fluid with dehydration correction fluid replacement (with added potassium) and insulin infusion (0.1 IU/kg/hr). Mannitol was given in view of cerebral edema, and some improvement in blood gas parameters was observed after 24 hr of therapy (Outlined in Table 1). However, the patient did not show any clinical improvement with worsening of GCS.

Parameter	At Admission	After 24 hr	Before dialysis	After Dialysis
Blood glucose (mg/dl)	270	220	180	190
Blood ph	6.9	7.1	7.21	7.34
Bicarbonate (mEq/L)	6.4	13.1	15.2	22
Blood ketone (mmol/L)				
Na (mmol/l)	131	151	154	
K (mmol/l)	3.3	3.1	2.1	3.7
Creatinine (mg/dl)	0.9	2.5	3.2	2.1
Phosphorus (mg/dl)		<0.9		3.1
BE	-25.4	-14.1	-11.8	-3
Lactate (mmol/l)		2		

At 24 hours of admission, renal function was deteriorated (creatinine 3.1mg/dl), with hypernatremia (Na 151mmol/l), hypokalaemia (3.1mmol/l), and decreased urine output, despite of dehydration correction. At the same time, serum phosphorus came out to 0.9 mg/dl (2.8-4.5 mg/dl) with urine routine microscopy showing microscopic hematuria, an early sign of rhabdomyolysis due to tissue injury.[4,5] To prevent hypophosphatemia and rhabdomyolysis phosphorus supplements were added with potassium in intravenous fluid.

Renal function continued to worsen despite fluid resuscitation and supportive management.

Hence hemodialysis was initiated under the guidance of a Nephrologist.

After homodialysis patient improved clinically over the next 12 hr by regaining consciousness and a further improvement in acidosis which was stalled after initial improvement. Finally, the patient shifted to subcutaneous insulin over the next day with a resolution of diabetic ketoacidosis.

At the 10th day patient was discharged on a basal-bolus regimen with the diabetes education.

DISCUSSION

This index case highlights that timely identification of complications and management with proper fluid therapy are key factors for the successful management of diabetic ketoacidosis.

Lethal complication of diabetes ketoacidosis like acute renal injury and cerebral edema can occur due to refractory metabolic acidosis, persistent dehydration, and hypoxia-induced tissue injury, which is very rare in adults

except in some case reports.[6,7]

Cerebral edema in type 1 diabetes can occur due to too rapid correction of osmolarity and due to tissue hypoxia in paediatric population but pathophysiology in an adult is not well understood. Mannitol has a protective role at cellular level to prevent cytotoxic injury and it reduces intracranial pressure effectively.

Clinical symptoms of cerebral edema vary according to the severity from encephalopathy, and seizure to respiratory coma and death. In this patient, severe diabetic ketoacidosis, and acute renal injury was precipitating cerebral edema to develop. If cerebral edema occurs along with refractory metabolic acidosis, electrolyte disturbances, and acute renal injury then management will be challenging. In review if literature, there was four case who treated with Renal replacement therapy due to acute renal injury in diabetes ketoacidosis. In most cases renal injury due to noncompliance of therapy and due to refractory metabolic acidosis.[5-7]

The index patient has cerebral edema, hypokalaemia, hypophosphatemia, microscopic hematuria, and acute renal injury. Mannitol played important role in prevention of irreversible brain injury. Although mannitol can cause osmotic diuresis and hypernatremia (Seen in index patient) which again precipitate intravascular volume depletion so appropriate fluid therapy to prevent dehydration is vital. Along with it, supplements of phosphorus and potassium in intravenous maintenance fluid can prevent insulin-induced electrolyte imbalance.

Nontraumatic rhabdomyolysis may occur in severe Diabetes ketoacidosis due to phosphate depletion, which can be identified in the early phase by detecting microscopic hematuria.[8-10] In this patient renal functions worsened even though after appropriate fluid therapy hence haemodialysis was done to normalize refractory metabolic acidosis. Resolution of acidosis after dialysis leads to improvement in cerebral edema and the patient was successfully shifted to subcutaneous basal-bolus insulin. In Index patient, the severe clinical picture and worsening occur probably due to delayed arrival at hospital.

CONCLUSION

An appropriate intravenous fluid, correction of electrolyte imbalance, and insulin replacement are cornerstones of the treatment of diabetic ketoacidosis. Mannitol should be kept at the bedside of patients with cerebral edema, as early therapy can prevent irreversible injury. Along with it, acute kidney injury and nontraumatic rhabdomyolysis are very rare in an adult patient with DKA but timely diagnosis and intervention at an early stage can prevent life-threatening complications.

Conflict of Interest: The author declares no conflict of interest

Consent for publication: Consent for publication has been taken.

Acknowledgments: none

Author Contributions: MA was involved in patient management, revised manuscript, and approved manuscript. VA was involved in management. NA was involved in revising and approving the manuscript, and RS was involved in patient management and revised and approved the manuscript.

REFERENCES

1. Joseph Wolfsdorf, Nicole Glaser, Mark A. Sperling; Diabetic Ketoacidosis in Infants, Children, and Adolescents: A consensus statement from the American Diabetes Association. *Diabetes Care* 1 May 2006; 29 (5): 1150–1159. https://doi.org/10.2337/dc06-9909
2. Troy, P.J., Clark, R.P., Kakarala, S.G. *et al.* Cerebral edema during treatment of diabetic ketoacidosis in an adult with new onset diabetes. *Neurocrit Care* **2**, 55–58 (2005). https://doi.org/10.1385/NCC:2:1:055
3. Huang Z., Xu L, Li F., Deng W., Li Y. Fulminant type 1 diabetes mellitus with rhabdomyolysis: have we overlooked the situation? Diabetes Res Clin Pract. 2010 Dec; 90(3): e47-9. doi: 10.1016/j.diabres.2010.07.010. Epub 2010 Aug 25. PMID: 20800305.
4. Bajpai A., Agrawal N. Hyperglycemic emergency, Diabetes management and pathophysiology MedEclasses course in Pediatric Endocrinology, Available at learning. Grow society.in
5. sErica Zarse, Michelle Knoll, David D. Williams, Kelsee Halpin, Gouri Kallanagowdar, Sarah Tsai; 1028-P: Acute Kidney Injury in Youth with Diabetes Admitted with Isolated Diabetic Ketoacidosis, Isolated Hyperglycemic Hyperosmolar State,

and Hyperosmolar Diabetic Ketoacidosis: A Single Center Experience. *Diabetes* 1 June 2022; 71 (Supplement_1): 1028–P. https://doi.org/10.2337/db22-1028-P.

6. Kawata H., Inui D., Ohto J., Miki T., Suzue A., Fukuta Y., Nishimura M. The use of continuous hemodiafiltration in a patient with diabetic ketoacidosis. J Anesth. 2006; 20(2): 129-31. DOI: 10.1007/s00540-005-0385-2. PMID: 16633773.
7. Singh H., Krishna R.H.S., Jain A., Sharma N. Peritoneal dialysis for the rescue in critical refractory metabolic acidosis in diabetic ketoacidosis. J Family Med Prim Care. 2019 May; 8(5): 1792-1793. DOI: 10.4103/jfmpc.jfmpc_138_19. PMID: 31198761; PMCID: PMC6559115.
8. David W. Miller, Corey M. Slovis, Hypophosphatemia in the emergency department therapeutics, The American Journal of Emergency Medicine,Volume 18, Issue 4,2000, Pages 457-461, ISSN 0735-6757, https://doi.org/10.1053/ajem.2000.7347
9. J. Møller-Petersen; P. Thorgaard Andersen; N. Hjørne; J. Ditzel (1986). *Nontraumatic rhabdomyolysis during diabetic ketoacidosis., 29(4), 229–234.* doi:10.1007/bf00454881
10. Agha A., Nawaz S., Saleem M., Jones S., Ahmed A. Haemofiltration as a treatment option in refractory life-threatening diabetic ketoacidosis. Endocr Abstr 2015; 38: p. 88.