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# Severe Diabetic Ketoacidosis Complicated by RRT-Requiring Acute Kidney Injury in an Adolescent with Newly Diagnosed Type 1 Diabetes

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**Abstract**

Diabetic ketoacidosis (DKA) is a major and potentially life-threatening acute complication of type 1 diabetes mellitus (T1DM), particularly in children and adolescents. We report a 17-year-old male who presented with severe DKA as the first manifestation of previously undiagnosed T1DM. On admission, he was profoundly acidotic, markedly hyperglycemic, hemodynamically unstable, and anuric, with rising creatinine levels. Despite appropriate resuscitation and standard DKA therapy, renal function deteriorated to stage 3 AKI according to KDIGO, leading to the initiation of renal replacement therapy. During the initial phase of dialysis, the patient experienced several episodes of hypoglycemia, requiring insulin dose adjustment and closer glucose monitoring. Renal function gradually improved and patient was discharged in stable condition. This case highlights that DKA as the initial presentation of type 1 DM in adolescents can progress to severe AKI requiring dialysis, and emphasizes the importance of careful monitoring of renal function and glycemic management during renal replacement therapy in pediatric patients.



## 1. Introduction

Diabetic ketoacidosis (DKA) remains one of the most serious acute complications of type 1 diabetes mellitus (T1DM), contributing substantially to morbidity and mortality among children and adolescents worldwide. Despite advances in diagnostic strategies and public awareness, 15–70% of children still present with DKA at the time of T1DM diagnosis, with higher rates reported in regions where screening programs are limited and recognition of early hyperglycemic symptoms remains low [1, 2]. Adolescents constitute a particularly vulnerable group due to pubertal hormonal changes, psychosocial factors, and inconsistent healthcare engagement, all of which increase the likelihood of presenting with severe metabolic decompensation [1].

Acute kidney injury (AKI) is increasingly recognized as a common and clinically significant complication of pediatric DKA. Recent multicenter studies report AKI in 40–64% of children hospitalized with DKA, with approximately 15–20% progressing to severe AKI (KDIGO stage 2–3) [3, 4]. AKI in the context of DKA results from profound hypovolemia, severe acidosis, impaired renal perfusion, and inflammatory tubular injury, and may progress from prerenal dysfunction to intrinsic renal damage if not rapidly corrected. Importantly, several longitudinal studies have demonstrated that childhood AKI confers an increased long-term risk of hypertension and chronic kidney disease [5], underscoring the need for early recognition and timely management.

While most cases of DKA-associated AKI resolve with standard fluid and insulin therapy, progression to stage 3 AKI requiring renal replacement therapy (RRT) is rare in

children and has been described only in isolated reports. Existing literature highlights factors associated with severe AKI, including delayed diagnosis of T1DM, severe acidosis (pH <7.0), shock, and persistent oliguria/anuria, but data on pediatric patients who ultimately require hemodialysis remain limited [6, 7]. Consequently, the optimal timing and indications for initiating RRT in pediatric DKA-associated AKI are not well established.

This case report describes an adolescent with previously undiagnosed T1DM who presented with severe DKA complicated by stage 3 AKI requiring renal replacement therapy. We present this case to illustrate the diagnostic and therapeutic challenges associated with late recognition of T1DM, to highlight the progression from severe DKA to RRT-requiring AKI, and to emphasize the importance of early diagnosis, close monitoring of renal function, and monitoring for complications during dialysis.

## 2. Cases

A 17-year-old male adolescent was referred from a secondary hospital with decreased consciousness lasting 12 hours prior to admission. Two days earlier, he had developed progressive weakness, tachypnea, recurrent vomiting, and abdominal pain. Further history revealed previously unrecognized polyuria, polydipsia, polyphagia, and weight loss. There was no family history of diabetes mellitus. At the referring hospital, the patient presented with a pGCS score of E1V1M1, Kussmaul breathing, signs of shock, a blood glucose level of 559 mg/dL, and ketonuria. He was diagnosed with diabetic ketoacidosis and received fluid resuscitation totaling 2,380 mL over 9 hours, a continuous intravenous insulin aspart infusion at 1.48 IU/kg/hour, empiric antibiotics, and dobutamine as a vasopressor due to the unavailability of norepinephrine at that facility. No clinical or echocardiographic evidence of cardiac dysfunction was identified.

On arrival at our hospital, the patient appeared markedly emaciated (weight-for-height 72%) and remained critically ill, with a pGCS score of E2V3M5, Kussmaul respirations, cold extremities, a capillary refill time exceeding 3 seconds, and hypotension (80/30 mmHg; mean arterial pressure 47 mmHg). Dobutamine was discontinued, and norepinephrine was initiated as the appropriate vasopressor for shock management. Initial laboratory evaluation showed severe hyperglycemia (524 mg/dL), profound metabolic acidosis (pH 6.913; bicarbonate 2.1 mmol/L), leukocytosis (16,400/$mm^3$), thrombocytopenia (136,000/$mm^3$), elevated urea (76 mg/dL), and creatinine (2.38 mg/dL; estimated glomerular filtration rate 26 mL/min/1.73 $m^2$). Urinalysis demonstrated glucosuria and ketonuria. The patient was anuric, with urine output <0.2 mL/kg/hour. A diagnosis of severe diabetic ketoacidosis due to newly diagnosed type 1 diabetes mellitus, complicated by KDIGO stage 3 acute kidney injury, was established. Management was continued using a two-bag fluid protocol, intravenous insulin infusion, electrolyte correction, and empiric antibiotics.

On the second hospital day, the patient's level of consciousness improved to a pGCS score of E3V5M6, and metabolic parameters showed partial correction; however, anuria persisted beyond 24 hours. Serial arterial blood gas analyses are summarized in Table 1. Renal function worsened, with urea increasing to 115 mg/dL and creatinine to 5.10 mg/dL (eGFR 22.5 mL/min/1.73 $m^2$). Despite the absence of hyperkalemia or overt fluid overload, the patient had persistent metabolic acidosis, a doubling of serum creatinine within 24 hours, and sustained anuria. Pediatric nephrology consultation was obtained, and renal replacement therapy was initiated. Clinically, the AKI was considered to have progressed from a prerenal etiology to acute tubular necrosis, although fractional excretion of sodium and urine sediment analysis were not available for confirmation. Renal ultrasonography performed the following day showed normal-sized kidneys with preserved echogenicity, excluding congenital or chronic renal disease.

Intermittent hemodialysis was initiated on the third hospital day. Following the first session, urine output improved to 1.15 mL/kg/hour. During the initial dialysis sessions, the patient experienced several episodes of hypoglycemia, attributed to reduced insulin clearance and glucose shifts during dialysis. Trends in blood glucose levels during hospitalization are shown in Figure 1. Insulin dosing was adjusted accordingly, and blood glucose monitoring was intensified. A total of ten hemodialysis sessions were performed, initially three times per week for two weeks, followed by gradual tapering to twice weekly and then once weekly as renal function recovered. A detailed timeline of biochemical parameters is provided in Table 2. Renal function improved progressively, with a final serum creatinine of 0.57 mg/dL and an eGFR of 125 mL/min/1.73 $m^2$. Blood and urine cultures remained negative, and empiric antibiotics were discontinued on hospital day 10. The patient was discharged in stable condition, tolerating oral intake, and maintained on a basal–bolus insulin regimen. At three-month follow-up, HbA1c had decreased from 14.5% to 7.2%, and C-peptide levels were markedly low (0.06 ng/mL), consistent with type 1 diabetes mellitus.

**Table 1.** Serial arterial blood gas analysis during hospitalization.

| Measurement Order | pH | $pCO_2$ (mmHg) | $HCO_3$ (mmol/L) | BE (mmol/L) |
|---|---|---|---|---|
| 1 | 6.91 | 10.3 | 2.1 | -30 |
| 2 | 7.15 | 19.5 | 6.8 | -22 |
| 3 | 7.28 | 16.2 | 7.8 | -19 |
| 4 | 7.25 | 15.4 | 6.8 | -21 |
| 5 | 7.39 | 16.6 | 10.2 | -15 |
| 6 | 7.25 | 32.2 | 14.6 | -13 |
| 7 | 7.38 | 20 | 12 | -13 |
| 8 | 7.37 | 37.9 | 22.1 | -3 |

**Table 2.** Timeline of renal function and urine output during hospitalization.

| Hospital Day | Urea (mg/dL) | Creatinine (mg/dL) | eGFR (mL/min/1.73 $m^2$) | Urine Output (mL/kg/hour) | Renal Replacement Therapy |
|---|---|---|---|---|---|
| Day 1 | 76 | 2.38 | 26 | <0.2 | No |
| Day 2 | 115 | 5.1 | 22.5 | <0.2 | No |
| Day 3 | 107 | 3.8 | 30 | 1.15 | Yes |
| Day 6 | 107 | 3.8 | 30.21 | 0.8 | Yes |
| Day 8 | 174 | 6.32 | 18.1 | 0.6 | Yes |
| Day 10 | 80 | 3.4 | 33.7 | 1 | Yes |
| Day 13 | 56 | 1.61 | 39.2 | 1.2 | Yes |
| Day 16 | 14 | 0.38 | 188 | 1.5 | Yes |
| Day 19 | 66 | 0.83 | 138 | 1.9 | Yes |
| Day 22 | 39 | 0.72 | 100 | 1.2 | Yes |
| Discharged | 32 | 0.57 | 125 | 2.01 | Yes |

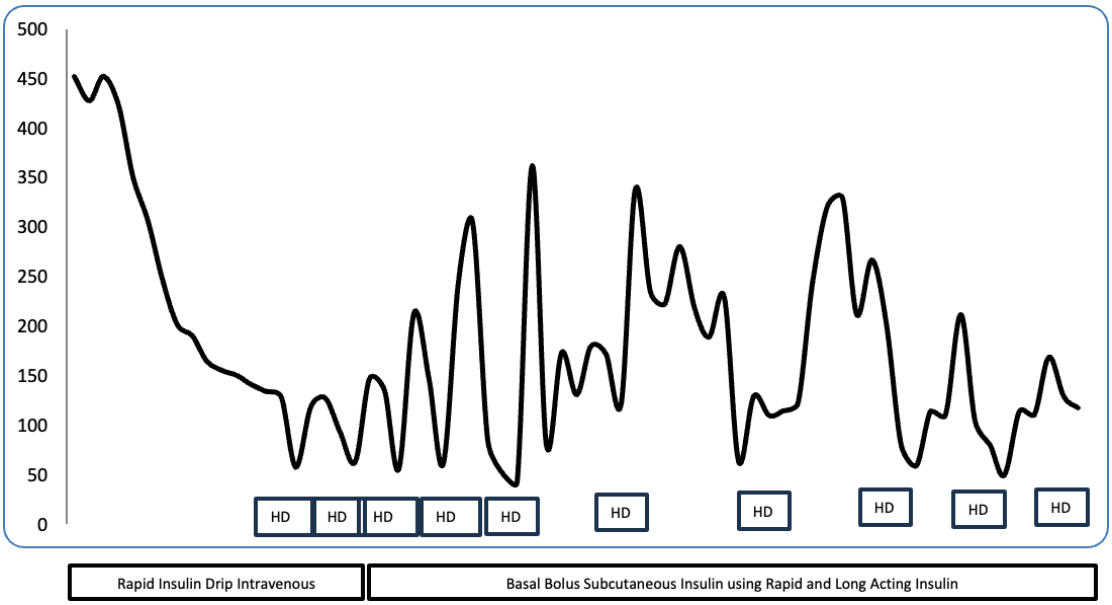


**Figure 1.** Trend of blood glucose levels during hospitalization. Serial capillary blood glucose measurements are shown in chronological order. Marked glycemic variability occurred during the acute phase of severe diabetic ketoacidosis, with recurrent hypoglycemia predominantly during hemodialysis (HD) sessions and transient hyperglycemia during transition from intravenous insulin infusion to subcutaneous basal–bolus insulin therapy. Glycemic stability improved following insulin dose adjustment and renal recovery.

## 3. Discussions

### *3.1. Mechanism of AKI in This Specific Patient*

This case describes an adolescent with newly diagnosed diabetic ketoacidosis who progressed to severe acute kidney injury requiring renal replacement therapy, as well as the challenges of glycemic management during hemodialysis. Such presentations remain uncommon, as only a small proportion of children with DKA develop severe AKI necessitating dialysis. Huang et al. reported that 43–52% of children with DKA develop AKI, while approximately 18% progress to severe AKI (KDIGO stage 2–3), and only a minority require renal replacement therapy [4]. The clinical course in this patient therefore

provides insight into the severe end of the DKA spectrum in adolescents.

At presentation, marked hyperglycemia (blood glucose >500 mg/dL), leukocytosis, and an elevated neutrophil-to-lymphocyte ratio reflected profound dehydration and a systemic inflammatory response. These findings are consistent with those reported by Suraphan et al., who demonstrated that blood glucose levels >500 mg/dL, leukocyte counts >15,000/mm$^3$, and NLR >5.5 were significantly associated with an increased risk of AKI in pediatric DKA [8].

The pathophysiology of AKI in this patient was likely multifactorial. In the early phase, severe dehydration due to osmotic diuresis and reduced oral intake led to hypovolemia and impaired renal perfusion, consistent with prerenal AKI. However, the absence of renal recovery despite adequate fluid resuscitation, together with persistent anuria, progressive creatinine elevation, and granular casts on urine sediment, suggested progression to intrinsic AKI in the form of acute tubular necrosis (ATN). Renal ultrasonography demonstrated normal kidney size and structure, making chronic or obstructive kidney disease unlikely. This pattern is consistent with previous reports showing that prolonged renal hypoperfusion during severe DKA can result in transition from prerenal AKI to ATN [7, 9]. Overall, the clinical and laboratory findings in this patient support a diagnosis of ATN following sustained prerenal injury.

### *3.2. Rationale and Timing of Dialysis*

The patient was evaluated by the pediatric nephrology team following persistent anuria despite adequate fluid resuscitation and ongoing conservative management, accompanied by refractory metabolic abnormalities. Considering the overall clinical course, renal replacement therapy was initiated. This approach is consistent with recommendations that emphasize initiating RRT based on the presence of life-threatening disturbances in fluid, electrolyte, or acid–base balance, persistent anuria or oliguria, and the risk of systemic complications related to uremia, rather than isolated creatinine thresholds.

This case illustrates that, in severe DKA, improvement of metabolic parameters does not necessarily parallel recovery of renal function. Delaying renal replacement therapy in this setting may therefore contribute to further clinical deterioration. RRT was discontinued after serial assessments demonstrated recovery of intrinsic renal function, reflected by improving urine output and stabilization of metabolic parameters, and when dialysis was no longer required to meet the patient's clinical needs, in accordance with KDIGO recommendations [10, 11].

### *3.3. Dialysis-Related Hypoglycemia and Prevention Strategies*

A notable and relatively uncommon finding in this case was the occurrence of recurrent hypoglycemia during hemodialysis in a patient with newly diagnosed type 1 diabetes mellitus. This phenomenon is likely related to reduced insulin clearance in the setting of acute kidney injury, as the kidneys play a substantial role in insulin metabolism and degradation.

Previous studies have shown that insulin requirements may decrease significantly in patients with DKA and impaired renal function undergoing renal replacement therapy, increasing the risk of hypoglycemia if insulin doses are not carefully adjusted [12]. In addition, hemodialysis itself can lower blood glucose levels through multiple mechanisms, including glucose transfer into the dialysate, altered insulin and glucose distribution, and blunted counter-regulatory hormonal responses during dialysis, particularly when dialysates with low or normal glucose concentrations are used [13]. These mechanisms likely explain why hypoglycemic episodes in this patient predominantly occurred during dialysis sessions.

These observations underscore the need for frequent glucose monitoring, individualized insulin dose adjustments, and proactive strategies to prevent hypoglycemia in pediatric patients undergoing dialysis in the setting of acute metabolic derangements.

### *3.4. Comparison With Similar Published Pediatric Cases*

In most pediatric patients with DKA, AKI is mild to moderate and resolves with fluid resuscitation and insulin therapy. Huang et al. reported that while nearly half of children with DKA develop AKI, only a small fraction progress to severe AKI requiring renal replacement therapy [4]. Similar findings were reported by Pachapure et al., who observed that dialysis was required in only a minority of children with DKA-associated AKI [14].

Compared with these reports, the present patient exhibited a more severe clinical course, characterized by rapid progression to KDIGO stage 3 AKI, lack of response to conservative management, and the need for hemodialysis. Although isolated case reports have described AKI in severe DKA [15, 16], the occurrence of recurrent intradialytic hypoglycemia in patients with newly diagnosed type 1 diabetes remains rarely reported. This case therefore expands the existing literature by highlighting the complex interaction between renal dysfunction, insulin metabolism, and dialysis in severe pediatric DKA.

### *3.5. Limitations*

This report has several limitations. As a single case report, the findings cannot be generalized. Biomarkers of tubular injury were not available, and AKI classification relied on clinical findings and conventional laboratory parameters. In addition, detailed assessment of insulin pharmacokinetics during hemodialysis was not performed, and long-term renal outcomes beyond early follow-up remain limited.

### *3.6. Implications and Future Directions*

The novelty of this case lies in the combination of DKA as the initial presentation of type 1 diabetes mellitus, rapid progression to dialysis-requiring AKI in an adolescent, and recurrent hypoglycemia during hemodialysis. Given evidence that AKI during DKA increases the risk of subsequent microalbuminuria and diabetic kidney disease [1], this case highlights the importance of long-term renal follow-up after recovery from the acute episode.

Further studies are needed to identify predictors of severe AKI in pediatric DKA, to clarify optimal timing for initiation of renal replacement therapy, and to develop standardized strategies for glycemic management during dialysis in acute metabolic conditions. Longitudinal research is also required to better define the long-term renal outcomes of children who experience severe AKI during DKA.

## 4. Conclusions

This case highlights the potential severity of AKI in severe diabetic ketoacidosis in newly diagnosed type 1 diabetes may progress to renal replacement therapy-requiring AKI. In contrast with most published pediatric DKA cases which AKI is transient and resolves with conservative management, this patient exhibited a more severe and prolonged renal courses. This emphasized that persistent anuria and deteriorating renal function despite adequate treatment should prompt early nephrology consultation and consideration for renal replacement therapy. Recurrent hypoglycemia during dialysis emphasizes the important of glucose monitoring during dialysis with more frequent bedside glucose check or temporary dose reduction during dialysis sessions in patients with impaired renal clearance. This case emphasized the importance of early detection, careful indication for renal replacement therapy in AKI, closed monitoring for glucose during dialysis. Future studies are needed to identify predictors of severe AKI in pediatric DKA, to define optimal threshold for initiating renal replacement therapy, and to establish standardized strategies for insulin management during dialysis.

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