

Single Case

# Surprising Hyperkalemia of 10.2 mmol/L in a Patient with Hyperglycemia: A Case Report

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## Keywords

Hyperkalemia · Hyperglycemia · Diabetes mellitus

## Abstract

Hyperkalemia is a life-threatening condition potentially leading to cardiac arrest. Here, we report a case of surprising severe hyperkalemia of 10.2 mmol/L in a diabetic patient with previously normal kidney function presenting without discernible clinical symptoms to our emergency department. The patient was admitted because of hyperglycemia of 32.8 mmol/L, which was detected during daily testing in her nursing home. The hyperkalemia was caused by pre-renal failure due to hyperglycemic polyuria which led to volume depletion, and worsened by a combination of potassium-sparing drugs and potassium supplementation. The patient was treated conservatively. Eighteen hours later, the serum potassium concentration was 4.6 mmol/L. The patient could be released 6 days later. To our knowledge, this is the highest described hyperkalemia treated conservatively and survived without cardiopulmonary resuscitation.

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## Background

A serum potassium concentration above 5.5 mmol/L is defined as hyperkalemia, which is a life-threatening condition eventually leading to cardiac arrest and is associated with a mortality rate of up to 41% in hospitalized patients [1–3]. To prevent and treat hyperkalemia, the underlying causes need to be understood and symptoms need to be interpreted correctly.

The four most common causes of hyperkalemia are impaired renal function, medication altering distal nephron potassium secretion, increased potassium release from cells, and high dietary intake of potassium [1, 4, 5]. Diabetes can cause alterations in plasma potassium via several mechanisms: insulin shifts K<sup>+</sup> ions from the extra- to the intracellular space, and insulin deficiency slows down this process. Additionally, the hyperosmolality resulting from hyperglycemia can directly lead to hyperkalemia via solvent drag [6, 7]. Hyperosmolality also causes polyuria. Polyuria either can cause hypokalemia due to an increase in distal nephron flow or can lead to volume contraction and prerenal failure and eventually result in hyperkalemia [7].

Usually, hyperkalemia causes clear clinical symptoms: nausea, palpitations, and weakness of the large muscles [8]. This is explained by the decreased resting membrane potential of cells, leading to altered excitability of all myocytes. Altered cardiomyocyte excitability, the main cause of the increase in mortality with hyperkalemia, is diagnosed via electrocardiogram (ECG). In mild hyperkalemia, the QRS complexes are broadened, while a sinus-wave pattern can be observed at severe stages [8].

A mainstay of emergency treatment of hyperkalemia is the initiation of dialysis [9]. Acute dialysis poses many risk factors, including the risk of acute vascular access, infection, severe hypotension, disequilibrium syndrome, and cardiac events [10]. Therefore, a conservative treatment of hyperkalemia is often preferred [9]. The conservative treatment approach consists of stimulation of both potassium uptake into cells and urinary potassium excretion [5].

Here, we report the case of an oligosymptomatic diabetic patient in whom hyperglycemia indirectly led to a hyperkalemia of 10.2 mmol/L. It was treated by implementing only conservative measures and is, to our knowledge, the highest described potassium value survived without cardiopulmonary resuscitation.

## Case Presentation

A 57-year-old female patient presented to our emergency department from her nursing home for hyperglycemia, which was detected during daily testing (32.8 mmol/L; 591 mg/dL). She had survived an insult to the basal ganglia due to a hypertensive emergency 2 years before (thus was not able to speak) and had insulin-dependent diabetes mellitus type 2. Secondary causes of arterial hypertension had been excluded. The kidney function had been measured as normal on her previous hospitalization 2 months before (baseline serum creatinine 0.66 mg/dL).

The patient had been to our hospital 2 months previously for a urinary tract infection. During this stay, repeated hypertensive urgencies had been noted (arterial blood pressure

>180/110 mm Hg), as well as metabolic alkalosis (pH 7.37,  $\text{HCO}_3^-$  27.0 mmol/L) and hypokalemia (2.4 mmol/L). After successful antibiotic treatment for the urinary tract infection, the patient was discharged with a newly prescribed medication of ramipril 5 mg 1-0-1, spironolactone 200 mg 1-0-0, and daily potassium supplementation of 80.25 mmol (see online suppl. Table 1; for all online suppl. material, see [www.karger.com/doi/10.1159/000512590](http://www.karger.com/doi/10.1159/000512590) for list of all medications upon admission). The etiology of the hypokalemia was unfortunately not investigated. At discharge, the plasma potassium was 3.2 mmol/L. In the discharge report, the need for close ambulatory serum potassium monitoring was stated. For an overview of the patient history, please see the timeline according to CARE criteria (Fig. 1) [11]. After discharge, no ambulatory controls took place.

On the current admission, vitals were taken, an ECG was written, and a venous blood gas analysis was performed as a standard procedure. The patient was examined and severe dehydration was noted. The venous blood gas analysis showed an elevated blood glucose of 32.8 mmol/L (591 mg/dL) and an extreme serum potassium of 10.2 mmol/L (Table 1), and the ECG a flattened P wave with lengthening of the PR interval and a broadened QRS complex starting to merge with the T waves (Fig. 2a). At that point, both the intensive care physician and the nephrologist were informed, the patient was transferred to continuous monitoring, and an arterial blood gas analysis was obtained (Table 1). When arterial measurement verified the hyperkalemia, 1 L of isotonic saline, 40 mg furosemide, 100 mL 8.4% sodium bicarbonate, 10 mL 10% calcium gluconate, and 4 units of insulin were given intravenously (i.v.). Sixteen units of insulin were given subcutaneously. The ECG went promptly into a sinus rhythm within 20 min (Fig. 2b). One hour later, the serum potassium concentration was 8.8 mmol/L. Additional laboratory results (Table 2) revealed acute kidney injury (serum creatinine 5.90 mg/dL, blood urea nitrogen 224 mg/dL). Kidney ultrasound ruled out postrenal failure.

The patient was transferred to our intensive care unit. Four liters of isotonic saline i.v. were administered, prescribed under the hypothesis of prerenal kidney failure due to hyperglycemic polyuria. Serum potassium decreased from 10.2 to 4.6 mmol/L within 18 h of admittance (Fig. 3). The 24-h urine output was 3.4 L and serum creatinine returned to 0.83 mg/dL within 2 days. No dialysis was performed.

The patient could be released to her nursing home within 6 days of admittance. Potassium supplementation was paused. The need to monitor the serum potassium concentration in this patient more closely was communicated to her primary care physician.

## Discussion

We described the case of a patient hospitalized for diabetic decompensation which was complicated by hyperkalemia and acute kidney injury and handled conservatively. The hyperkalemia was of multifactorial origin: impaired renal function due to volume contraction in combination with medication altering distal nephron potassium secretion (RAAS [renin-angiotensin-aldosterone system] blockade) and dietary potassium supplementation.

The risk for cardiovascular mortality with hyperkalemia increases steeply starting from potassium concentrations of 5.5 mmol/L [12]. While there are reports of hyperkalemia of 11.4 mmol/L in a patient with a pacemaker and of 14 mmol/L under ongoing cardiac resuscitation, to our knowledge, 10.2 mmol/L is the highest survived K<sup>+</sup> value described to date in a patient without cardiac assist devices [13, 14]. Conservative management of severe hyperkalemia has been reported in patients with normal renal function [15]. In this case, we chose to treat the patient conservatively. Conservative treatment of hyperkalemia under the condition of good urinary output often is more rapid than the establishment of dialysis modalities. We believe this approach is feasible and reasonably safe to perform under intensive care conditions if the reestablishment of a sinus rhythm in response to calcium gluconate treatment is achieved rapidly (approx. within 10–20 min).

In this case, the hyperkalemia was oligosymptomatic. Symptoms of hyperkalemia usually comprise muscular weakness and palpitations, symptoms the patient communicates [5]. The described patient could not communicate after having survived a stroke 2 years previously, and the hyperkalemia almost would have been missed. This case emphasizes the additional caution that should be given to patients with preexisting neurologic conditions [14].

Blockade of the RAAS is an independent risk factor for hyperkalemia [12]. While there currently is no clear recommendation on the frequency of testing intervals, in the described case, beginning treatment with RAAS blockade and potassium supplementation should have led to more frequent testing [3]. The hyperosmolar polyuria did in this case not lead to hypokalemia (see Introduction), as potassium-sparing medication was combined with potassium supplementation, resulting in a potassium load that distal nephron flux could not resolve.

A potential mechanism by which the patient might have had a survival benefit under such extreme hyperkalemia is the accompanying hyperglycemia. While it is an independent risk factor for hyperkalemia, it may have protected this patient from fatal cardiac consequences. The proposed mechanism, which has been discussed for vascular smooth muscle cells and cardiomyocytes in mice, rats, dogs, and the isolated perfused rabbit heart, is caused by (in our case hyperglycemic) hyperosmolality [16–19]. Hyperosmolality in these models has been hypothesized to be cardioprotective via two mechanisms: (1) it causes a lower excitability of the cardiomyocyte, changing the patterns of intracellular calcium mobilization, and (2) it alters the central control of cardiac responses [7, 18, 19]. In patients presenting without concurrent hyperglycemia, much lower potassium values have been described to be fatal [3, 12]. It is tempting to speculate that our patient was protected by the hyperosmolality caused by hyperglycemia.

In conclusion, we described a rare case of a survived hyperkalemia of 10.2 mmol/L. It presented surprisingly as hyperglycemia in an otherwise oligosymptomatic patient and was handled conservatively.

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## Statement of Ethics

All herein presented research was conducted in accordance with the World Medical Association Declaration of Helsinki. Full written and signed consent to publish the herein presented information has been given by the patient's guardian before submission.

## Conflict of Interest Statement

The authors report no competing interests.

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## Author Contributions

All authors treated the patient, analyzed and interpreted the patient data, and wrote and approved the final manuscript.

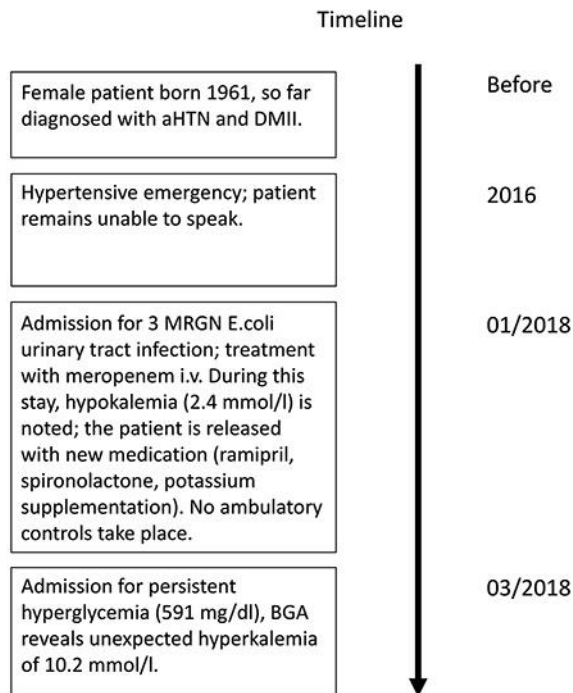
## Data Availability Statement

The data that support the findings of this study are fully available saved patient data at the University Hospital Hamburg, UKE. Data are available from the authors upon reasonable request if explicit repeated written consent of the patient's guardian is given.

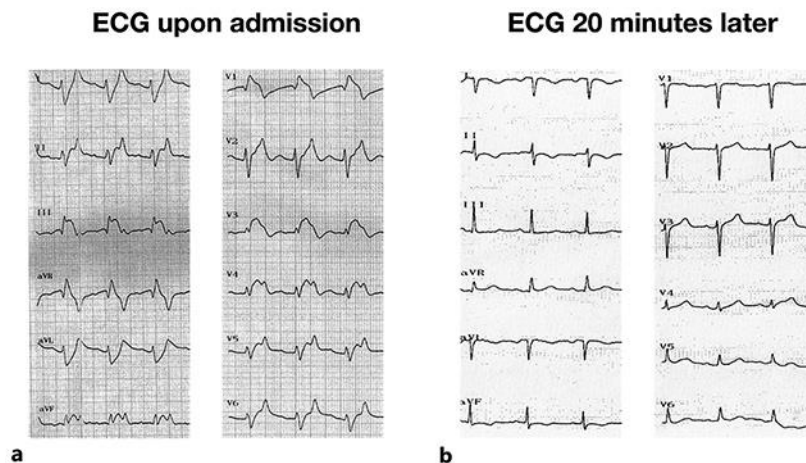
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**Fig. 1.** Timeline according to CARE criteria. aHTN, arterial hypertension; DMII, diabetes mellitus type 2; MRGN, multiresistant gram-negative; BGA, blood gas analysis.



**Fig. 2.** **a** ECG of the patient on admission, showing a broadened QRS complex and elevated T waves. **b** ECG of the patient after i.v. calcium gluconate, showing a sinus rhythm.

## Evolution of Hyperkalemia

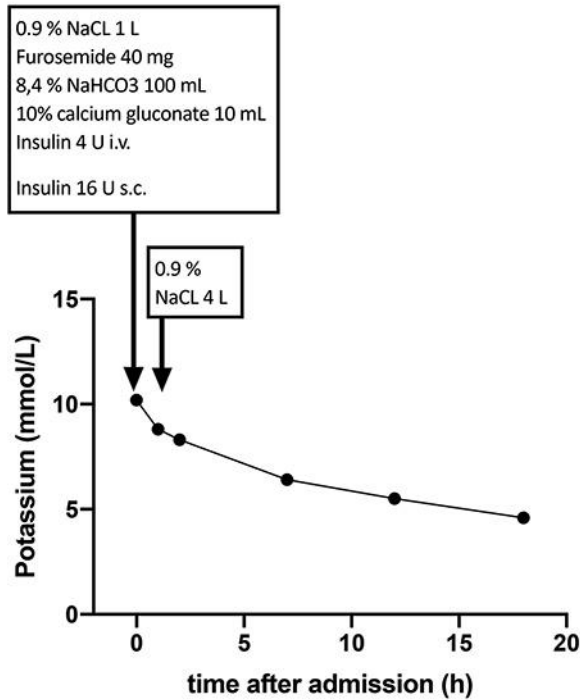


Fig. 3. Evolution of hyperkalemia over time and medication administered.



**Table 1.** Arterial and venous blood gas analysis (BGA) on admission

	Venous BGA	Arterial BGA
pO <sub>2</sub> , mm Hg	43.5	71
pCO <sub>2</sub> , mm Hg	44.1	43.3
pH	7.28	7.27
Bicarbonate, mmol/L	20.5	19.9
Hemoglobin, g/dL	11.2	10.1
Sodium, mmol/L	146	146
Potassium, mmol/L	10.2	10.2
Calcium, mmol/L	1.4	1.39
Chloride, mmol/L	113	114
Lactate, mmol/L	1.6	1.5
Glucose, mmol/L	32.5	32.8

**Table 2.** Laboratory results on admission showing acute kidney injury

	Normal range	Patient's result
Hemoglobin, mmol/L	7.64–9.5	6.8
Leukocytes, ×10 <sup>9</sup> /L	3.8–11.0	23
Thrombocytes, ×10 <sup>9</sup> /L	150–400	255
Blood urea nitrogen, mmol/L	1.67–3.34	37.4
Creatinine, μmol/L	44.2–88.4	521.6
C-reactive protein, μmol/L	to 14.75	17.7