

## No Alarm on ECG: A Case of Severe Asymptomatic Hyperkalemia with No Significant ECG Changes

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### Abstract

Hyperkalemia usually produces characteristic ECG changes due to its effects on myocardial membrane potential. When these accompany high serum potassium levels, they indicate a medical emergency. However, ECG findings do not always correlate with biochemical severity—sometimes even profound hyperkalemia presents with a normal ECG. We present a case of a 47-year-old woman with chronic kidney disease who exhibited a serum potassium level of over 8 mmol/L yet remained asymptomatic with a normal ECG. This case highlights the diagnostic limitations of ECG in hyperkalemia and emphasizes that management should be guided by biochemical results and clinical context rather than ECG appearance alone.

**Keywords:** Hyperkalemia, ECG, Chronic Kidney Disease, T waves, Acute Kidney Injury  
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### Introduction

#### Case Report

A 47-year-old woman, hypertensive for 12 years, with decompensated chronic liver disease secondary to autoimmune hepatitis (biopsy-proven), asthma for 7 years, and chronic kidney disease secondary to chronic tubulointerstitial nephritis for 4 years (baseline serum creatinine 2.5 mg/dL), presented for a routine nephrology follow-up.

Her recent laboratory report from a private laboratory revealed a **serum potassium of 8.4 mmol/L**. She denied symptoms such as fatigue, weakness, paresthesia, palpitations, nausea, or vomiting. On examination, she was **vitaly stable** with a pulse of 78/min and blood pressure of 130/70 mmHg. She was pale with mild pedal edema. Abdominal examination revealed splenomegaly and shifting dullness; neurological examination was normal.

Given the high potassium level, she was referred to the emergency department. Repeat electrolytes from the hospital laboratory confirmed **serum K<sup>+</sup> 8.1 mmol/L**. An ECG was performed, which showed normal sinus rhythm with no hyperkalemic changes.

Pseudohyperkalemia was suspected; hence, another sample was drawn in a lithium heparin tube using a large-bore needle with minimal tourniquet time and without fist clenching. The repeat potassium level was again **8.4 mmol/L**, confirming true hyperkalemia.

## Hyperkalemia & ECG

### Laboratory Findings

Parameter	Result	Normal Range
Hemoglobin	9.3 g/dL	12 – 15
Platelets	155 × 10 <sup>3</sup> /mm <sup>3</sup>	150 – 425
Sodium	132 mmol/L	135 – 145
<b>Potassium</b>	<b>8.4 mmol/L</b>	3.5 – 5.4
Chloride	110 mmol/L	90 – 110
Urea	138 mg/dL	10 – 50
<b>Creatinine</b>	<b>4.77 mg/dL</b>	0.6 – 1.1
Uric Acid	8.6 mg/dL	2.4 – 6.2

**Urine analysis:** yellow, pH 6.0, specific gravity 1.015, protein ++, no RBCs or bacteria.

### Medication History

- **Triforge** (Amlodipine/Valsartan/Hydrochlorothiazide) 10/160/12.5 mg once daily
- **Spironolactone** 100 mg once daily for ascites secondary to DCLD  
She also reported frequent intake of milk and baked potatoes.

Both medications were discontinued immediately. She was admitted to the ICU for cardiac monitoring and urgent treatment.

### Initial Management

- **IV calcium gluconate** for cardiac membrane stabilization
- **Nebulized salbutamol** to promote intracellular potassium shift
- **Regular insulin 10 IU** in 25% dextrose IV
- **Sodium polystyrene sulfonate 30 g** for potassium elimination

After 8 hours, serum potassium fell to **5.5 mmol/L**, but it rebounded to **6.8 – 7.8 mmol/L** within the next 8 hours, while the patient remained asymptomatic and ECG remained normal.

The **Depotash protocol** was then initiated:

- IV insulin 10 IU in 100 mL 25% D/W every 6 hours
- IV calcium gluconate (10%) every 12 hours
- Sodium polystyrene sulfonate 30 g with lactulose every 12 hours
- Salbutamol nebulization every 4 hours

Over the next 48 hours, her potassium normalized to **4.7 mmol/L**. Serial ECGs remained normal throughout. After three consecutive normal potassium readings, she was discharged with a potassium-restricted diet. Follow-up after two days revealed **serum K<sup>+</sup> 4.2 mmol/L**.

### Discussion

Hyperkalemia (K<sup>+</sup> >5.5 mmol/L) is a potentially life-threatening electrolyte disorder that affects cardiac excitability. Elevated extracellular potassium reduces the resting membrane potential, impairing depolarization and conduction. Classically, ECG changes progress from tall peaked T waves to PR prolongation, P-wave flattening, QRS widening, sine-wave pattern, and ultimately asystole or ventricular fibrillation [7].

## Hyperkalemia & ECG

However, multiple studies have shown that ECG changes are not reliably correlated with potassium levels. Montague et al. found that only half of hyperkalemic patients exhibited typical ECG changes [1]. Raffee et al. reported normal ECGs in 25% of patients with  $K^+ \geq 5.5$  mmol/L [4]. Case reports by Khattak et al. and Haykal et al. described patients with life-threatening hyperkalemia but minimal ECG abnormalities [2,9].

Possible explanations include:

1. **Rate of Potassium Rise:** Gradual accumulation, as in chronic kidney disease, allows myocardial adaptation [3].
2. **Metabolic Modifiers:** Acidosis, hyponatremia, or hypocalcemia may mask expected ECG patterns [7].
3. **Pharmacologic and Structural Factors:** Drugs such as  $\beta$ -blockers, digoxin, and antiarrhythmics, and comorbidities like uremia or structural heart disease, alter conduction [5].
4. **Individual Variability:** Differences in ion-channel reserve and myocardial responsiveness produce variable ECG presentations [8].

In this patient, chronic renal dysfunction and gradual potassium rise likely allowed cardiac adaptation, explaining the absence of ECG changes. Her asymptomatic presentation also reflects slow biochemical progression.

Clinicians should therefore not rely solely on ECG to gauge hyperkalemia severity. Laboratory confirmation remains the diagnostic gold standard, and treatment should not be delayed while awaiting ECG confirmation. Management must address both the biochemical derangement and its underlying cause.

Early management includes:

- **Calcium salts** for cardiac stabilization
- **Insulin – glucose therapy** and  **$\beta_2$ -agonists** to shift potassium intracellularly
- **Cation-exchange resins** or **dialysis** for potassium removal
- **Cessation of potassium-retaining agents** (e.g., ACE inhibitors, ARBs, spironolactone)
- **Dietary restriction** of potassium-rich foods

Our patient improved rapidly after discontinuation of potassium-retaining medications and standard hyperkalemia management. Her case highlights the risk of underestimating biochemical hyperkalemia in CKD patients when ECG is normal.

## Conclusion

Severe hyperkalemia can present without ECG abnormalities, especially in patients with chronic kidney disease and gradual potassium accumulation. A normal ECG should never reassure clinicians in the presence of critically elevated serum potassium. Laboratory testing remains the definitive diagnostic tool, and treatment must be initiated based on biochemical evidence rather than ECG appearance. Early recognition and management are essential to prevent fatal arrhythmias.

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