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A Spontaneous Cardioversion of Permanent Atrial Fibrillation to Sinus Rhythm Induced by Hyperkalemia

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Abstract **Introduction:** Atrial fibrillation (Afib), a common arrhythmia, can lead to significant complications, including stroke and heart failure. This case report examines a rare phenomenon: transient spontaneous conversion of permanent Afib to sinus rhythm in the context of acute-on-chronic kidney injury (AoCKD), hyperkalemia, and potential digoxin accumulation. **Case description:** Our case describes an 82-year-old female with long-standing permanent Afib, who was admitted to the hospital with progressive fatigue, nausea, vomiting, and anuric AoCKD. Laboratory results revealed severe hyperkalemia (7.0 mmol/L), metabolic acidosis, and uremic pericarditis. The patient's electrocardiogram demonstrated sinus rhythm, despite a known history of permanent Afib. After initiating dialysis, the electrolyte imbalance was corrected, and potassium levels normalized leading to Afib reemerging. **Discussion:** We hypothesized that hyperkalemia, possible accumulation of exogenous digoxin due to impaired renal clearance, and the role of endogenous digoxin-like factors (EDLFs) produced during AKI likely suppressed atrial ectopic activity and reentry circuits facilitating transient conversion of a permanent Afib to transient sinus rhythm. However, serum digoxin levels were not measured, limiting hypothesis confirmation. **Conclusion:** This case highlights the possible complex interplay of electrolyte imbalances, exogenous and endogenous digoxin accumulation, and AKI in Afib dynamics. Further research is needed to elucidate the role of EDLFs and their interaction with Afib and AKI. These findings emphasize the importance of comprehensive evaluation and management in patients with multi-system involvement.

Keywords: *Atrial Fibrillation, electrical cardioversion, EDLF, Digoxin, Hyperkalemia, AoCKD*

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1. Introduction

Atrial fibrillation (Afib) is a common supraventricular arrhythmia with significant clinical impact due to its association with increased morbidity and mortality. Despite its prevalence, the mechanisms underlying Afib are complex and not fully understood. The arrhythmia is initiated by ectopic electrical activity, often near the pulmonary veins (PVs), and sustained by reentry circuits and structural changes in the atrial substrate, such as fibrosis and dilation, It's characterized by uncoordinated atrial electrical activity and ineffective atrial contraction. [1] Traditionally classified by duration (paroxysmal, persistent, long-standing persistent, permanent), the latest 2023 ACC/AHA/ACCP/HRS guidelines introduce a stage-based framework emphasizing progression and early intervention Afib stages per 2023 guidelines:

Stage 1: At risk, with modifiable (e.g., hypertension) or nonmodifiable (e.g., age) risk factors. Focus: prevention.

Stage 2: Pre-AF, with structural or electrical changes (e.g., atrial enlargement). Focus: monitoring and intervention.

Stage 3: Active AF:

3A: Paroxysmal (resolves within 7 days).

3B: Persistent (>7 days).

3C: Long-standing (>12 months).

3D: Post-ablation (AF-free post-intervention).

Stage 4: Permanent AF, rhythm control not pursued. Focus: symptom and stroke management. [2]

The clinical presentation of AF is variable. Some patients are asymptomatic, while others may experience palpitations, fatigue, dyspnea, dizziness, and chest

discomfort. Complications include an increased risk of stroke, heart failure, and all-cause mortality. [3] This case report describes an elderly female with permanent atrial fibrillation (AF) who developed severe electrolyte and acid-base disturbances due to acute-on-chronic kidney failure. It explores the rare phenomenon of transient, spontaneous conversion of permanent AF to sinus rhythm, highlighting the interplay between hyperkalemia, possible accumulation of endogenous digoxin-like factors (EDLFs), and exogenous digoxin.

2. Case Description

An 82-year-old female was admitted to the ED with a 3-day history of progressive fatigue, nausea, vomiting, and inability to pass urine. According to a patient, the symptoms gradually progressed over the last couple of days, but as the nausea and vomiting progressed, she was unable to urinate for the past 24 hours. Her past medical history is significant with long-standing type 2 diabetes mellitus, chronic kidney disease, stage G3b, (eGFR = 32.6 mL/min/1.73 m²), chronic heart failure (NYHA III), atrial fibrillation (permanent form), and arterial hypertension, Grade II (ESH/ESC 2018). She was previously treated with Gliclazide, Metformin, Digoxin, Atorvastatin, Rivaroxaban, Spironolactone, Nebivolol, and Losartan. On admission, the patient appeared weak and could not communicate comprehensively with ED doctors. Because of that, the medical history was acquired from family members accompanying the patient. The vital signs showed normal HR—80 bpm, subfebrile fever T - 37.2, oxygen saturation and blood pressure within normal range.

The ECG showed a regular sinus rhythm with LBBB, a widened QRS complex, and a prolonged PR interval (Figure 1). Laboratory investigations revealed (Table 1).

The insertion of the indwelling catheter did not result in a passage of urine, so an anuric state was confirmed. Despite the patient's dehydration and the need for increased intravenous fluid administration, we have decided to start a dialysis session, as our decision was supported by the presence of severe metabolic acidosis (pH—6.97, serum bicarbonate—5.1 mmol/L), ECG changes due to hyperkalemia (wide QRS complex, flattened P wave, prolonged PR interval), and bedside cardiac ultrasound revealed pericardial separation of approximately 13 mm, suggesting uremic pericarditis. In our comprehensive analysis, we determined that the presence of a urinary tract infection (UTI), was confirmed by the detection of bacteriuria and leukocyturia and the significant growth of Escherichia coli 10^7 in the urine culture.

The ongoing UTI triggered a cascade of physiological responses, including fever, nausea, and severe vomiting. These symptoms, in turn, significantly disrupted the patient's volemic status, leading to a state of dehydration, which led to an exacerbation of underlying chronic kidney disease and the development of an acute-on-chronic kidney disease (AoCKD). The first dialysis session went without complications; hemodynamic parameters during and after stayed within normal range. After dialysis, repeated VBG showed somewhat improved acid-base and electrolyte levels (Table 2).

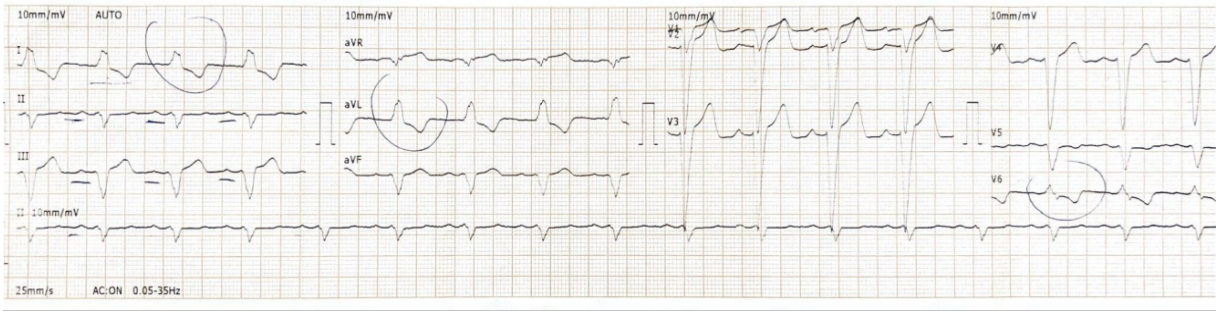

Figure 1. ECG on admission. Regular, sinus rhythm with LBBB, and a widened QRS complex, prolonged PR interval. (EGC speed 25mm/sec)

Table 1. Elevated Creatinine, Metabolic Acidosis, Hyperkalemia, Mild Leukocytosis

Parameter	Result	Reference Range
Creatinine	527.9 µmol/L	44–106 µmol/L (M), 36–90 µmol/L (F)
Urea	18.8 mmol/L	2.5–7.8 mmol/L
ABG		
- pH	6.97	7.35–7.45
- pCO2	21 mmHg	35–45 mmHg
- pO2	91 mmHg	80–100 mmHg
- Oxygen Saturation (sO2)	98.1%	>95%
- Potassium (K^+)	7.0 mmol/L	3.5–5.1 mmol/L
- Sodium (Na^+)	126 mmol/L	135–145 mmol/L
- Bicarbonate ($cHCO3^-$)	5.1 mmol/L	22–28 mmol/L
- Chloride (Cl^-)	96 mmol/L	98–107 mmol/L
- Lactate	7.2 mmol/L	0.5–2.0 mmol/L
CBC		
- WBC	12.93 x10³/µL	4.0–11.0 x10³/µL

Table 2. Metabolic Acidosis, Improved Potassium level

Parameter	Result	Reference Range
pH	7.29	7.35–7.45
Potassium (K+)	5.3 mmol/L	3.5–5.1 mmol/L
Sodium (Na+)	133 mmol/L	135–145 mmol/L
Chloride (Cl-)	101 mmol/L	98–107 mmol/L
Lactate (cLac)	4.5 mmol/L	0.5–2.0 mmol/L
Bicarbonate (cHCO3)	16.3 mmol/L	22–28 mmol/L

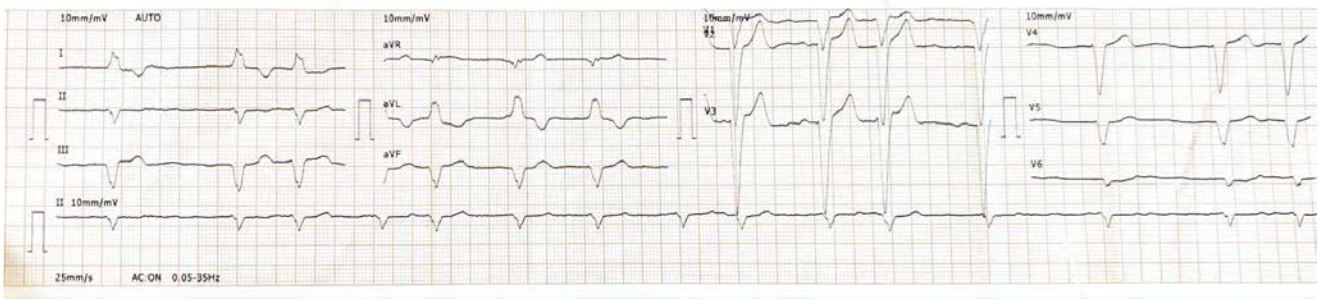

Figure 2. ECG approx. 24 hours after admission, serum potassium - 5.0 mmol/L. Irregularly irregular rhythm with LBBB, no P waves, and a widened QRS complex. (EGC speed 25mm/sec)

Table 3. Normal Potassium Level, Mild Metabolic Acidosis

Parameter	Result	Reference Range
Creatinine	134.0 µmol/L	44–106 µmol/L (M) 36–90 µmol/L (F)
pH	7.30	7.35–7.45
Potassium (K+)	3.7 mmol/L	3.5–5.1 mmol/L
Sodium (Na+)	141 mmol/L	135–145 mmol/L
Chloride (Cl-)	110 mmol/L	98–107 mmol/L
Bicarbonate (cHCO3)	20.6 mmol/L	22–28 mmol/L

Acknowledging the fluid loss associated with dialysis, aggressive rehydration was implemented post-procedure. Within approximately six hours, administering 2–2.5 liters of IV normal saline successfully restored urine output, resolving the anuric state. What has to be noted is that upon admission and afterward, 3 consecutive ECGs conducted with the patient showed signs of hyperkalemia: flattened P waves, a wide QRS complex, and prolonged PR interval. If the patient's long-standing history of permanent atrial fibrillation had been unknown, it would have been challenging to identify atrial fibrillation based on the initial ECG in the emergency department or subsequent ECGs conducted after admission. The ECGs demonstrated typical signs of hyperkalemia with a regular rhythm. Gradual potassium reduction, combined with cardiac membrane stabilization using intravenous calcium gluconate—achieved through dialysis and intravenous insulin with glucose (administration of furosemide was precluded due to the patient's anuric state)—resulted in the resolution of hyperkalemia-related ECG changes and the re-establishment of the irregularly irregular rhythm characteristic of atrial fibrillation (during which serum potassium was 5.0 mmol/L) (Figure 2).

The echocardiography revealed significant left ventricular hypertrophy, and reduction of the global systolic function of the left ventricle, EF - 52%. Inability to access diastolic function due to Afib. Significant left atrial dilation diameter of 51mm (Normal < 4.1 cm in men or < 3.9 cm in women), a right atrial major dimension of 41 (normal 3.4-5.3 cm), a minor dimension: of 63 (normal 2.6-4.4 cm), the right ventricle of normal size and normal function. Pericardium: Circular separation was observed, with a maximum of 12 mm along the anterolateral wall. After an appropriate treatment in the internal medicine and intensive care department which included 2 more sessions of dialysis, hydration therapies, acid-base, and electrolyte balance monitoring and correction, the patient´s subjective, objective, and laboratory results improved (Table 3).

After which the patient was discharged from the hospital.

3. Discussion

Atrial fibrillation (AF) refers to a supraventricular tachyarrhythmia that is characterized by a diffuse and disorganized atrial electrical activity that replaces normal sinus node function and results in ineffective mechanical atrial contraction. the underlying electrophysiological abnormality involves three principal components: the occurrence of "triggers" initiating focal ectopic activity in the atria or PVs, structural and functional abnormalities ("drivers") that promote and maintain reentry, and finally the presence of an abnormal substrate that helps to perpetuate the AF. [1,5] Potassium, the primary intracellular cation (100–150 mmol/L), plays a crucial role in maintaining the resting membrane potential (Vm) through the inward potassium current. During atrial fibrillation (AF), hyperkalemia can significantly alter electrical activity by disrupting the delicate balance

between intracellular ([K^+]i) and extracellular potassium ([K^+]o). [4] Even a slight increase in extracellular potassium levels leads to a substantial change in the [K^+]i/[K^+]o ratio, resulting in a decrease in the resting membrane potential, which can further destabilize cardiac electrophysiology. The cardiac muscle is the most prominent organ subjected to hyperkalemia, and the increased serum potassium ion leads to conduction abnormality and impaired myocardial contraction. Atrial tissues are more sensitive to hyperkalemia than the sinus node, ventricular tissues, or the bundle of His. [3] As the potassium ion level increases, the sinus node continues to propagate electrical activity to the ventricles without depolarizing the atrial muscle, called sino-ventricular rhythm. [3]

We suppose that the patient continued intake of exogenous digoxin even when the acute kidney injury progressed and oliguria-anuria worsened, which could lead to a serum accumulation of exogenous digoxin.

Digoxin exerts its effects on cardiac electrophysiology through direct and indirect mechanisms. Directly, it slows the sinus node rate by decreasing automaticity, prolongs the AV node refractory period to control ventricular rate during atrial fibrillation, and increases myocardial contractility by elevating intracellular calcium levels. Indirectly, digoxin enhances the vagal tone and suppresses sympathetic activity, further contributing to bradycardia and slowed AV nodal conduction. These properties make digoxin effective in managing atrial fibrillation and heart failure, although its narrow therapeutic index necessitates careful monitoring to avoid toxicity. [7] Endogenous digoxin-like factors (EDLFs) are steroid-like molecules produced within the body that inhibit the Na^+/K^+-ATPase pump, similar to the action of digoxin. [6]

In the context of acute kidney injury (AKI), the kidneys and adrenal glands are believed to be primary sources of EDLFs. Elevated levels of these factors have been described in pregnancy complications such as preeclampsia, which involves acute kidney injury and volume overload. [6]

The mechanism by which EDLFs may suppress atrial fibrillation (AF) involves the inhibition of the Na^+/K^+-ATPase pump, leading to increased intracellular sodium and calcium levels. This alteration can stabilize myocardial conduction and suppress ectopic activity. Additionally, EDLFs may exert vagal-like effects, increasing vagal tone and further inhibiting atrioventricular (AV) nodal conduction, thereby altering atrial electrophysiology. [6] The correlation between AKI and elevated EDLF levels suggests that severe AKI could lead to a transient increase in EDLF levels. This, combined with hyperkalemia's effects, might explain the temporary suppression of AF and the appearance of a regular rhythm. [6] We hypothesize that the observed spontaneous conversion of atrial fibrillation (AF) to sinus rhythm in this patient could be multifactorial, influenced by electrolyte abnormalities like hyperkalemia (suppressing re-entry rhythms and/or rapid focal ectopic firing (particularly in patients with enlarged atrium)), [4] potential accumulation of endogenous digoxin-like factors (EDLFs), and the exogenous digoxin due to impaired renal clearance due to acute-on-chronic kidney injury (AKI).

While the above-written discussion might seem intriguing, this is still stated as a hypothesis. The correlation between acute kidney injury and endogenous digoxin-like factors (EDLFs) remains poorly understood due to the limited available research. Additionally, other potential contributing factors were not adequately investigated in this specific case. For instance, the patient's serum digoxin level was never measured, which could potentially confirm our hypothesis.

4. Conclusion

This case highlights a rare and poorly understood phenomenon of spontaneous conversion of permanent atrial fibrillation (AF) to sinus rhythm, associated with severe hyperkalemia, acute-on-chronic kidney disease (AoCKD) with anuria, and potential endogenous and exogenous digoxin accumulation, that likely and collectively contributed to the suppression of ectopic activity and reentry pathways, resulting in transient sinus rhythm.

While the findings are intriguing, they remain hypothetical due to the limited understanding of EDLF mechanisms and their correlation with AKI/AoCKD. Furthermore, the absence of serum digoxin level measurement limits the confirmation of the proposed hypothesis. This case underscores the need for further research to explore the relationship between EDLFs, electrolyte disturbances, and AF dynamics, particularly in complex clinical settings like AoCKD.

As a rare and not well-studied condition, this case contributes to the existing literature but calls for cautious interpretation and acknowledgment of its limitations.

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