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When Blood Pressure Pills Turn Deadly: A Telmisartan Tale: A Case of ARB's Induced Hyperkalemia

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Abstract: ***Angiotensin II receptor blockers (ARBs) are cornerstone agents in hypertension management, yet their suppression of aldosterone can mask acute metabolic crises when renal clearing capacity drops. We report an 81-year-old female on daily telmisartan for 20 years without biochemical surveillance. Following a 3-day history of acute watery diarrhea and vomiting, she presented with critical hyperkalemia (7.08mmol/L), severe metabolic acidosis (pH 7.105), an acute-on-chronic renal crisis (creatinine 2.70mg/dL), and an electrocardiogram ECG showing a lethal sine wave pattern. Despite these physiological parameters, she remained conscious and fully conversant (GCS E4V5M6). Immediate treatment with intravenous calcium gluconate stabilized the myocardial membrane and successfully reverted the cardiotoxic rhythm. Subsequent intensive care management with intermittent hemodialysis led to complete metabolic normalization, with her serum potassium stabilizing at 4.0mmol/L by discharge. This case highlights the critical importance of regular metabolic monitoring in geriatric patients on chronic renin-angiotensin-aldosterone system (RAAS) inhibitors, especially during intercurrent volume-depleting illnesses.***

Keywords: Telmisartan, Hyperkalemia, Acute Kidney Injury, Angiotensin II receptor blockers, Sine-wave electrocardiogram

1. Introduction

Telmisartan is a selective angiotensin II receptor antagonist widely prescribed for blood pressure control and macrovascular/renal protection [1, 2]. While effective, its downstream suppression of aldosterone can carry an insidious risk of severe hyperkalemia [4]. Because the baseline incidence of severe electrolyte disturbances following ARB monotherapy is under 2%, with average potassium elevations limited to 0.1-0.2 mmol/L [5], routine metabolic screening is frequently omitted once outpatient blood pressure goals are achieved.

Consequently, drug-induced hyperkalemia is often discovered incidentally [6] or during investigations for non-specific complaints like nausea, fatigue, or orthostatic hypotension [7]. However, in geriatric patients, age-related reductions in physiological renal reserve leave the body highly vulnerable. When acute hypovolemia occurs, the homeostatic pathways needed to upregulate aldosterone and excrete excess potassium are pharmacologically blunted by the chronic ARB therapy, allowing serum potassium to rapidly accumulate to a peri-arrest threshold [8].

2. Case Presentation

An 81-year-old female presented to the emergency department (ED) with a sudden onset of progressive dyspnea, central chest discomfort, and diffuse abdominal pain. Over the preceding three days, she had a frequent history of foul-smelling, loose, watery stools accompanied by recurrent vomiting and general weakness. Her medical history was significant for primary hypothyroidism, bronchial asthma, and essential hypertension managed with telmisartan 40 mg daily for over 20 years. Notably, she had never undergone secondary kidney function testing or electrolyte monitoring during her two decades of continuous therapy.

On physical evaluation, the patient was conscious, alert, and conversing normally with the medical team, demonstrating a Glasgow Coma Scale GCS score of E4V5M6. Initial vitals revealed a bradycardic pulse rate of 60 beats/min, respiratory rate of 20 breaths/min, blood pressure of 100/60 mmHg and an afebrile temperature of 98.6 F. Her oxygen saturation was 88% on room air, which corrected to 96% using 3L/mi supplemental oxygen via nasal prongs.

Point-of-care arterial blood gas (ABG) analysis showed severe, uncompensated metabolic acidosis: pH 7.105pCO2 31.7 mmHg, and HCO3 -9.7 mmol/L. Immediate emergency biochemistry confirmed a severe acute-on-chronic renal crisis:

- Serum Potassium K+: 7.08 mmol/L
- Serum Creatinine: 2.70 mg/dL
- Blood Urea: 63.3 mg/dL (BUN 29.58 mg/dL)
- Serum Sodium (Na+): 142 mmol/L

An emergency 12-lead ECG revealed absent P waves and profound widening of the QRS complexes actively merging into a classic, terminal sine wave pattern (Fig 1).

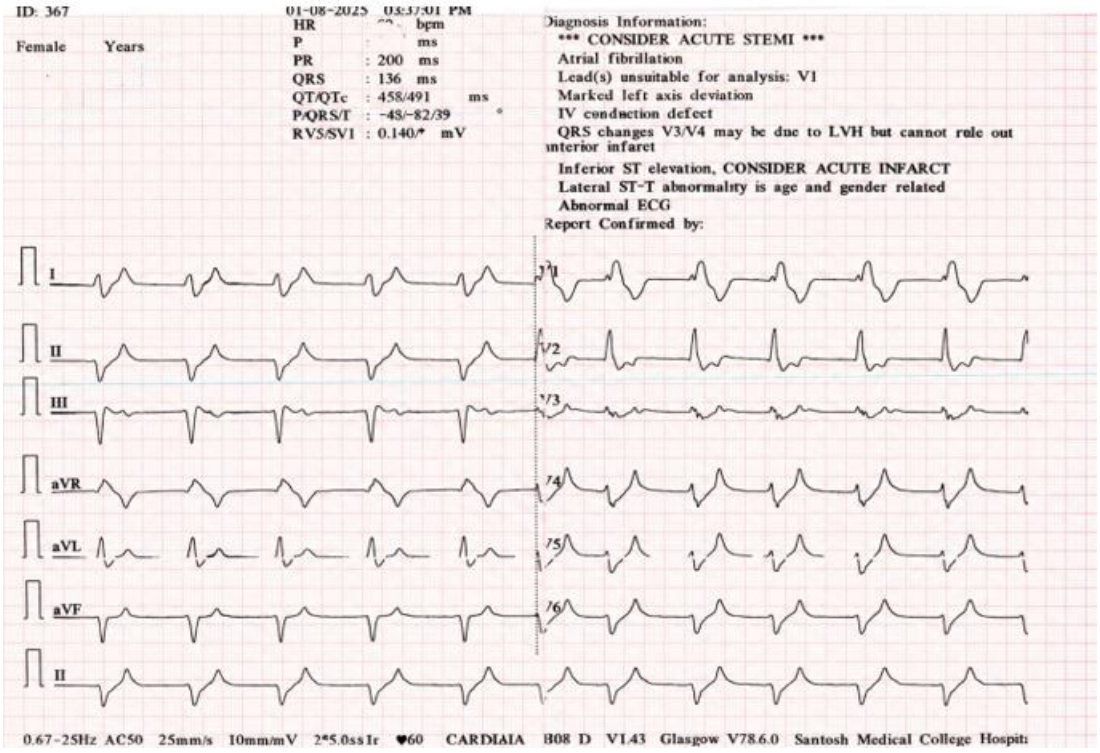

Figure 1: 12-lead ECG revealed absent P waves and profound widening of the QRS complexes towards sine wave pattern

An urgent abdominal ultrasound and non-contrast CT scan showed bilaterally shrunken kidneys with altered cortical echogenicity, indicating underlying, previously undiagnosed chronic kidney disease.

The emergency medicine team immediately activated the hyperkalemia management protocol. For myocardial membrane stabilization, 10 mL of 10% calcium gluconate was administered intravenously over 5 minutes under continuous cardiac monitoring. The cardiotoxic waveforms responded instantly, with the sine wave reverting back to a stable sinus rhythm with a normalized QRS interval duration (Fig 2).

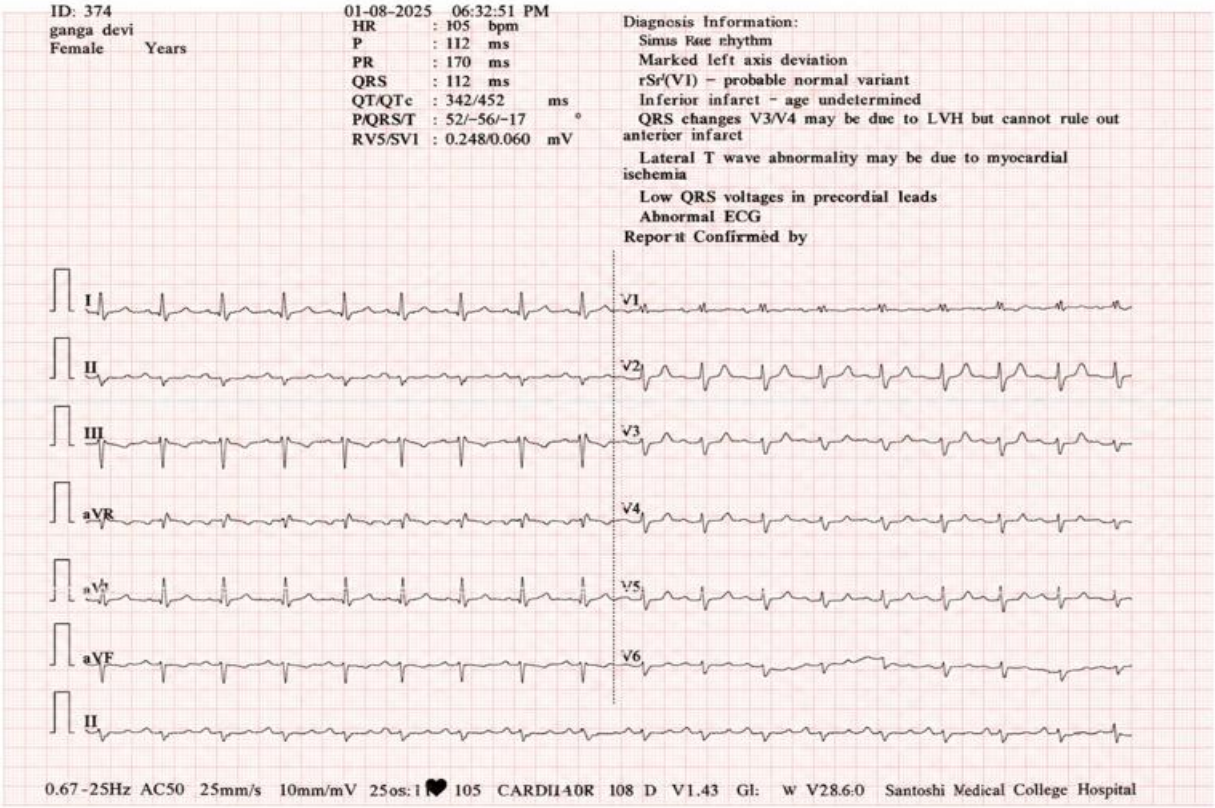

Figure 2: 12-lead ECG revealed sinus tachycardia and nonspecific lateral T-wave abnormalities, with no acute ST-elevation changes.

Intracellular potassium translocation was then driven using a glucose-insulin infusion (10 Units regular insulin in 100 mL of 25% Dextrose) paired with sequential nebulization's of salbutamol. To manage her severe cellular acidemia, a slow intravenous infusion of 200 mEq of 8.4% sodium bicarbonate was provided.

The patient was admitted to the intensive care unit, and telmisartan was permanently discontinued. Due to persistent metabolic acidosis and kidney injury, an emergency hemodialysis catheter was secured, and she was initiated on intermittent hemodialysis, which successfully lowered her post-dialysis serum potassium to 5.22 mmol/L. Over her hospital course, she received supportive critical care, including broad-spectrum antibiotics for secondary acute pyelonephritis, sequential enemas, and oral sodium bicarbonate tablets.

Her biochemical parameters normalized steadily: her serum creatinine down-trended from 2.70 mg/dL to 1.56 mg/dL, and her serum potassium stabilized at a safe, physiologic baseline of 4.0 mmol/L. Following complete recovery, she was safely discharged with a comprehensive outpatient follow-up plan.

3. Discussion

ARBs control hypertension by blocking angiotensin II from binding to its receptor, which downregulates the synthesis and release of aldosterone from the adrenal cortex [1, 4]. Because aldosterone is the primary hormone regulating potassium elimination in the collecting duct, its long-term inhibition shifts the physiological burden of potassium clearance onto simple glomerular filtration [3, 5]. While healthy kidneys accommodate this shift, this geriatric patient possessed chronic, shrunken kidneys that had gone unmonitored for twenty years, severely reducing her baseline reserve [6].

When the patient experienced acute watery gastroenteritis, the resulting volume depletion triggered a prerenal acute kidney injury. Under normal physiological conditions, the body would compensate for this hypovolemic drop by stimulating a substantial rise in aldosterone to retain sodium and water [5]. However, because her distal nephrons were locked in an absolute, medication-induced aldosterone blockade, her kidneys completely lost the capacity to clear potassium, prompting an ultra-acute serum rise to 7.08 mmol/L [8].

This case underscores that severe hyperkalemia can present as an ultra-acute cardiorespiratory emergency rather than an incidental laboratory finding, even when the patient remains completely conscious and alert [7]. The direct myocardial toxicity of this rapid rise caused her severe intraventricular conduction delay, generating the classic sine wave pattern [8].

Clinicians must maintain awareness that intravenous calcium gluconate does not lower serum potassium concentrations; instead, it shifts the threshold potential of the myocyte membrane to restore membrane excitability and prevent lethal ventricular fibrillation [8]. This critical stabilizing step provided the necessary clinical window to safely execute definitive insulin-shifting therapies and organize urgent renal replacement therapy [8].

4. Conclusion

Chronic telmisartan therapy in elderly individuals carries an insidious risk of hidden cardiotoxicity if biochemical surveillance is neglected [4]. Age-related drops in renal reserve can cause a common, transient gastrointestinal illness to rapidly escalate into a life-threatening hyperkalemic emergency when the primary endocrine pathway for potassium excretion is pharmacologically blocked [5, 8]. Early recognition of characteristic hyperkalemic ECG signs is a life-saving skill that must be prioritized over waiting for formal laboratory data. Clinicians must maintain a high index of suspicion for drug-induced electrolyte disturbances in long-term antihypertensive users, implementing periodic metabolic screening before an acute systemic insult occurs [7].

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