



HYPERMAGNESEMIA CAUSING REVERSIBLE ATRIOVENTRICULAR CONDUCTION DELAY: A CASE OF SYMPTOMATIC BRADYCARDIA FROM OTC CATHARTICS



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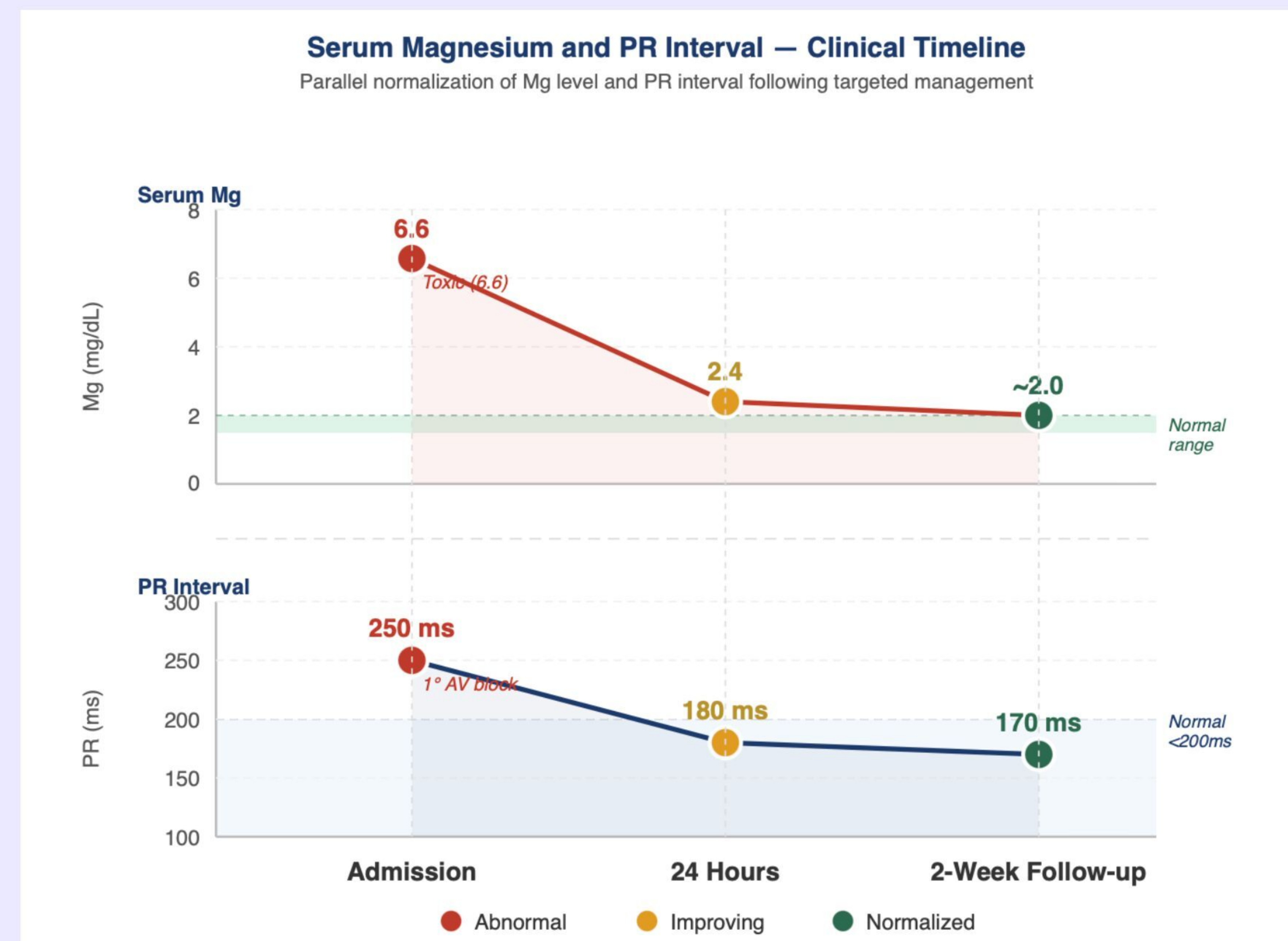
Introduction

Hypermagnesemia is uncommon, linked to renal failure (reported in 5–7% cases), yet clinically significant toxicity can arise with only mild renal impairment when gastrointestinal transit is prolonged. Cardiac conduction is particularly vulnerable, producing AV nodal delay, bradycardia, and even complete heart block. We report a case of syncope from reversible AV block caused by hypermagnesemia, where prompt recognition permitted straightforward, rapid therapy and excellent outcome.

Case

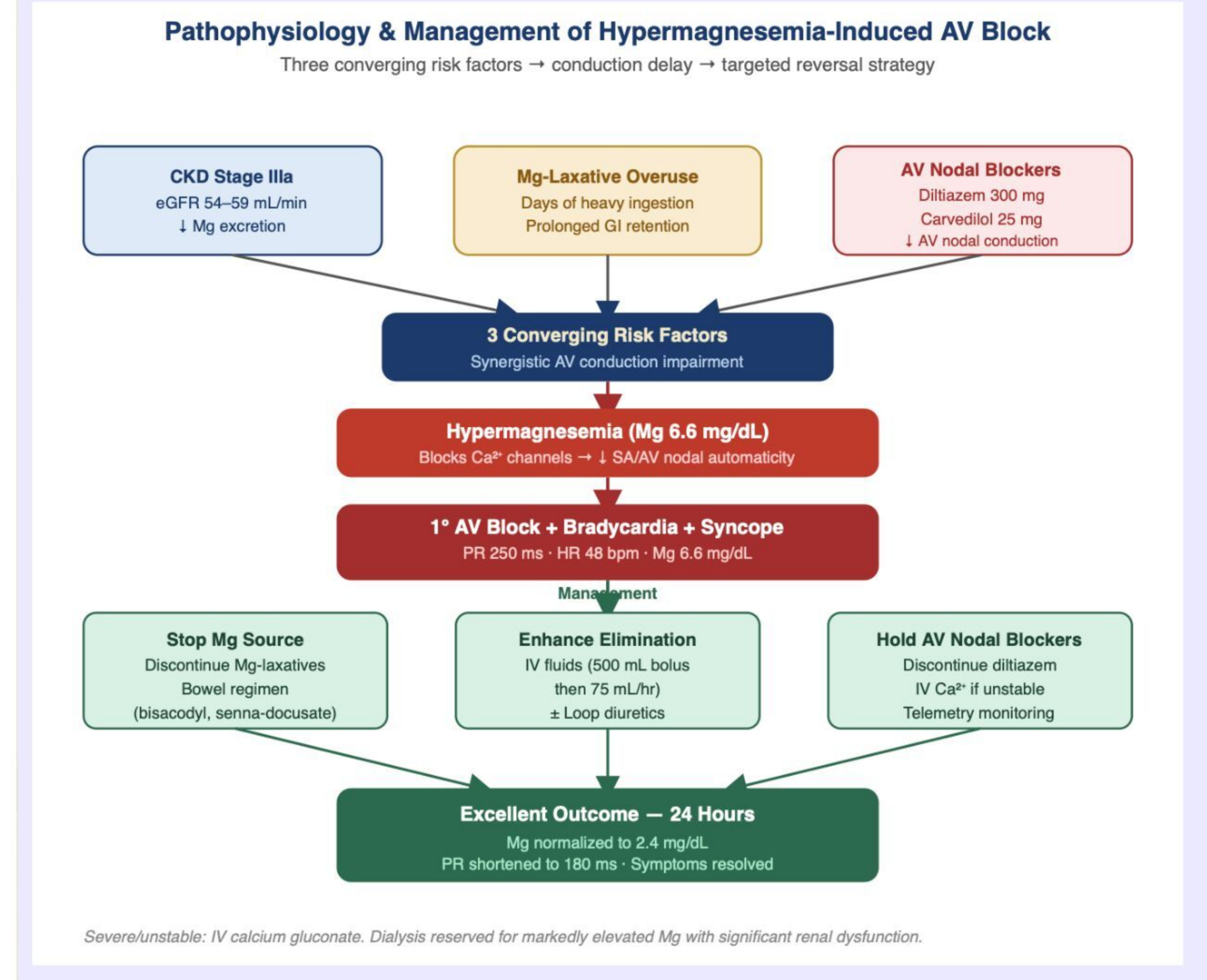
A 71-year-old transgender man with resistant hypertension, type 2 diabetes, Hashimoto's thyroiditis, and CKD stage IIIa (eGFR 54–59 mL/min) experienced syncope preceded by lightheadedness and diaphoresis while ambulating. EMS documented bradycardia (HR 48 bpm) with hypotension responsive to atropine. In the ED, he was pale but alert; vitals were stable (BP 130/70 mm Hg, HR 55 bpm). Head CT was unremarkable. ECG demonstrated a new first-degree AV block (PR 250 ms; QT/QTc 458/472 ms) relative to prior tracings. Laboratory testing revealed hypermagnesemia (Mg 6.6 mg/dL; ref 1.5–2.0) with creatinine 1.4 mg/dL. He reported several days of heavy ingestion of magnesium-containing Laxatives for constipation.

Case



Home medications included carvedilol 25 mg, diltiazem 300 mg, hydralazine 10 mg, losartan 100 mg, and weekly testosterone. Diltiazem was discontinued, and the patient received isotonic fluids (500-mL bolus then 75 mL/hr), an aggressive bowel regimen (bisacodyl, Senna- docusate), and continuous telemetry. Echocardiography showed no structural disease. Within 24 hours, he had 3 bowel movements; serum magnesium normalized to 2.4 mg/dL, with PR interval shortening to 180 ms (QTc 438 ms) and complete resolution of symptoms. He was discharged with magnesium-laxative avoidance and AV nodal blocker review. Subsequent cardiology follow-up 2 weeks later showed a normal ECG and heart rate.

Mechanism



Discussion

Hypermagnesemia can cause reversible bradycardia and AV block even in mild CKD, especially with excess magnesium-laxative use and concurrent AV nodal blockers. Diltiazem and carvedilol slow AV conduction, an effect amplified by elevated magnesium. The parallel improvement in magnesium (6.6→2.4 mg/dL) and PR interval (250→180 ms) supports a transient, exposure-related cause. Management includes stopping magnesium, promoting elimination (fluids, catharsis ± diuretics), and holding AV nodal blockers; calcium or dialysis is reserved for severe cases. Early recognition enables rapid recovery and avoids unnecessary interventions