

Elevated LDL Secondary to Multiple Myeloma: A Case Report

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**Abstract**

Introduction: Multiple myeloma is a rare cause of elevated LDL. We describe a case of a patient with marked increase in LDL, with subsequent improvement after diagnosis and treatment of multiple myeloma.

Case Presentation: A 72-year-old male was referred to our lipid clinic for marked increase in cholesterol levels. Historically, his LDL ranged from 87-133 mg/dL over a span of 15 years without treatment. Routine assessment in 2017 showed his LDL increased to 252 mg/dL, which was confirmed on repeat testing. Atorvastatin 80mg daily was initiated. As part of his initial lipid clinic labs a 24-hour urine was ordered to assess for nephrotic syndrome, which revealed significant proteinuria, and the patient was referred to nephrology. Subsequent urine electrophoresis demonstrated serum free light chains (1,474 mg/dL) and elevated monoclonal lambda protein (2,073 mg/dL) concerning for multiple myeloma. Hematology/oncology then diagnosed the patient with IgG lambda multiple myeloma. Four weeks after addition of ezetimibe at initial lipid clinic visit, his LDL had decreased to 95 mg/dL, and both medications were continued. He started chemotherapy, achieving a very good partial response with reduction in IgG and monoclonal proteins. Most recent labs after reduction in paraproteins show further improvement of his LDL, now 42 mg/dL.

Discussion/Conclusion: Though patients with multiple myeloma typically present with nonspecific symptoms, elevated LDL is not known to be a common initial indicator of disease. Our patient achieved reduction in his lipids with medical therapy, and subsequent improvement with the treatment of previously undiagnosed multiple myeloma.

**Categories**

CV Team Case

**Program Name**

The Ohio State University Wexner Medical Center

## Managing Flecainide Toxicity: A case report of toxicity in the setting of acute renal injury

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### Abstract

#### Introduction

Flecainide is a common antiarrhythmic drug used to treat ventricular and supraventricular arrhythmias. Although rare, flecainide toxicity can occur and is known to have potentially lethal effects. Toxicity is particularly concerning as there is no specific antidote available. Given this, it is essential for clinicians to understand the potential factors which can precipitate flecainide toxicity.

#### Case Presentation

A 73 year old female was admitted to the hospital with pneumonia. A few weeks prior to presentation, her electrophysiologist increased her dose of flecainide for breakthrough episodes of atrial fibrillation. During hospitalization she was started on levofloxacin for pneumonia treatment. Although she has known renal insufficiency, her creatinine levels were noted to be elevated from baseline during admission, however, were improving by discharge. She was discharged to home on levofloxacin. Two weeks later she was readmitted to the hospital with symptoms of weakness, fatigue, lightheadedness, ataxia, nausea, and diplopia. On admission she was noted to be hyperkalemic and her kidney function had worsened. An EKG showed a wide QRS complex and intermittent failure to capture of her pacemaker (see Figures 1 & 2). Treatment was initiated with IV fluids and alkalization. She was diagnosed with flecainide toxicity and was treated with supportive measures and alkalization while waiting flecainide clearance. Within 48 hours her QRS duration had returned to baseline and she was discharged home after six days in the hospital.

#### Discussion

Flecainide is a Vaughn Williams Class IC antiarrhythmic medication that is typically well-tolerated and effective. It is a sodium channel blocker which works on phase 0 of the cardiac action potential. Pharmacokinetically it works by use-dependence. On EKG, Flecainide's effects can potentially cause prolongation of the PR, QRS, and QT intervals.

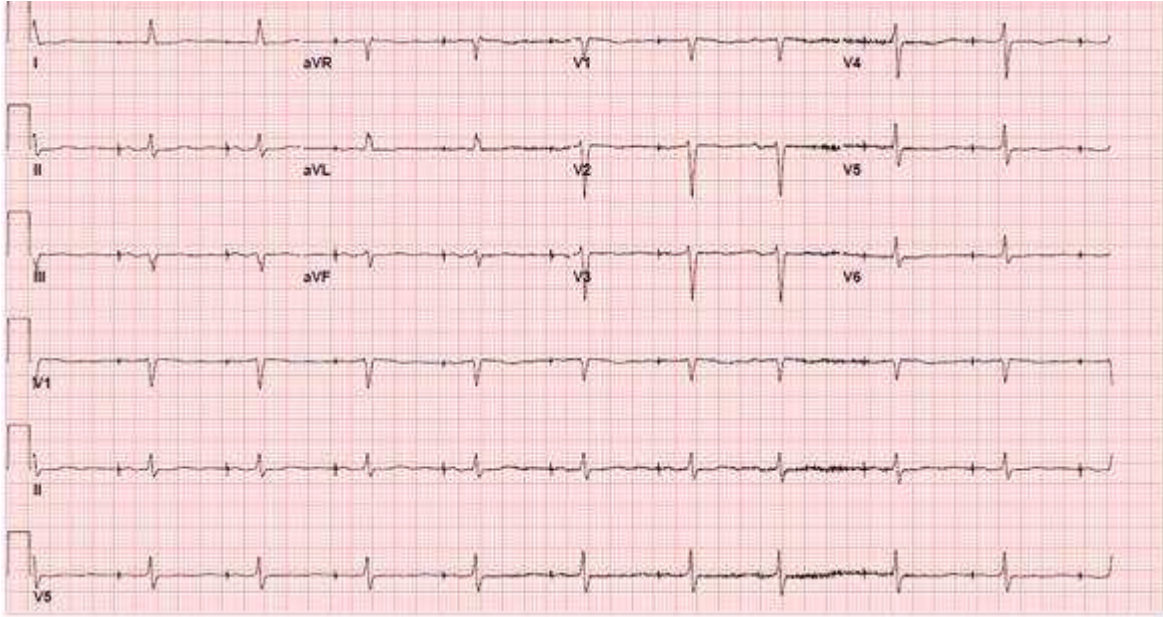
Flecainide has a narrow therapeutic window. Although levels are obtainable, they are not readily available so therefore the diagnosis of flecainide toxicity is often made based on clinical presentation. Common signs and symptoms of flecainide toxicity include increased QRS duration, prolonged PR interval, paresthesia, ataxia, sedation, seizure, and coma.

Given that there is no antidote for flecainide and it is not dialyzable, the current treatment for toxicity is to institute supportive measures and use high-dose sodium bicarbonate. Although the mechanism by which this works is not fully understood, it is thought that sodium bicarbonate dissociates flecainide from the sodium channel binding-site.

#### Conclusion

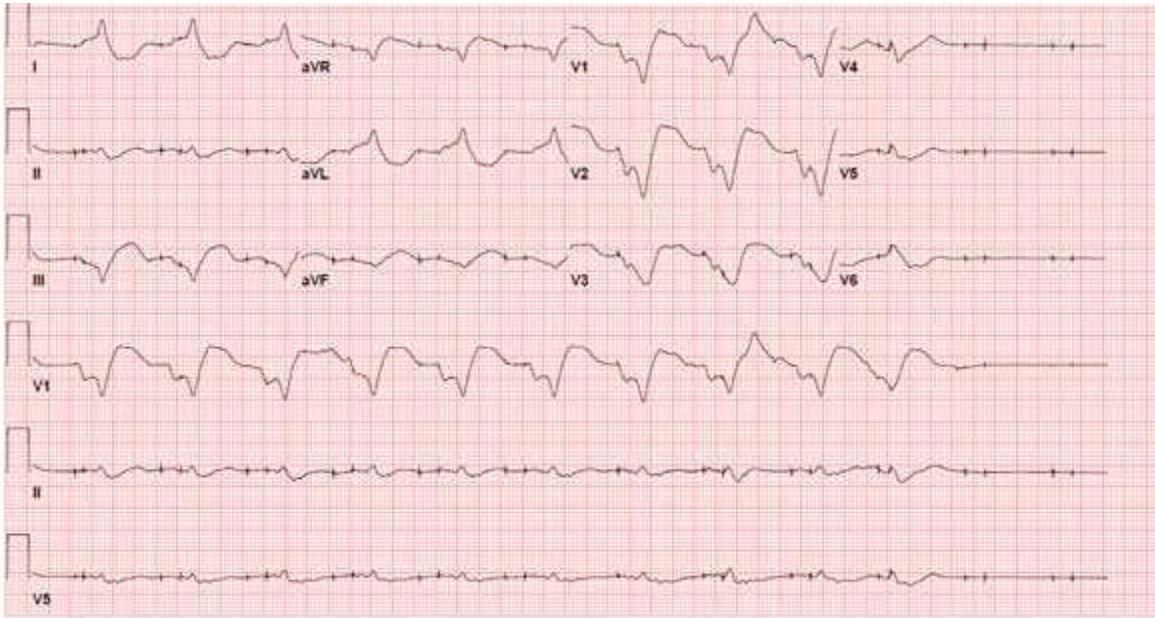
Although flecainide toxicity is rare, this case illustrates the potentially detrimental effects that acute illness, especially acute renal injury can have on drug levels. The pharmacokinetics of flecainide need to be considered by clinicians when managing flecainide toxicity, given there is no specific antidote to reverse the effects. Management of toxicity should focus on supportive measures and alkalization until the medication has been eliminated from the patient's system.

#### Figure 1



Baseline EKG performed at a routine cardiology visit

Figure 2



Initial EKG on hospital presentation.

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Riverside Methodist Hospital