

# Psychiatric Fantasies and Urological Realities

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## Description

Acute Myocardial Infarction (AMI), commonly known as a heart attack, is a critical cardiovascular event typically associated with older adults and those with significant risk factors such as hypertension, diabetes, and smoking. However, occurrences of AMI in young adults, especially those with underlying genetic predispositions like Familial Hypercholesterolemia (FH), present unique challenges in diagnosis and management. This case report explores the clinical presentation, diagnostic workup, and treatment strategies in a young adult with FH who experienced an AMI.

## Case presentation

Mr. A, a 32-year-old male, presented to the emergency department with a sudden onset of severe chest pain radiating to his left arm and associated with diaphoresis. His medical history was significant for FH, diagnosed during childhood due to elevated LDL cholesterol levels and a family history of premature coronary artery disease. Despite being on statin therapy (atorvastatin 40 mg daily) since adolescence, his LDL cholesterol remained persistently elevated (LDL-C >190 mg/dL).

On examination, Mr. A appeared anxious and diaphoretic. His blood pressure was 140/90 mmHg, heart rate 110 bpm, and oxygen saturation 98% on room air. Cardiac auscultation revealed normal heart sounds with no murmurs. Initial electrocardiogram showed ST-segment elevation in leads II, III, aVF, and V4-V6, consistent with acute inferior and lateral myocardial infarction.

## Diagnostic workup

Following the ECG findings, urgent coronary angiography was performed, revealing a complete occlusion of the proximal Right Coronary Artery (RCA). Percutaneous Coronary Intervention (PCI) was successfully performed with stenting of the RCA. Subsequent laboratory investigations showed elevated cardiac biomarkers, including troponin I (peak level 12 ng/mL), confirming the diagnosis of AMI.

Further investigations included lipid profile analysis, which indicated markedly elevated LDL cholesterol levels (LDL-C >250 mg/dL). Genetic testing confirmed a heterozygous mutation in the LDL receptor gene, consistent with FH. Additional risk factors such as smoking (10 pack-years) and a sedentary lifestyle were also identified.

**Treatment and management:** In the acute phase, Mr. A received dual antiplatelet therapy (aspirin and clopidogrel) and high-intensity statin therapy (atorvastatin 80 mg daily) post-PCI. He was also started on beta-blockers and angiotensin-converting enzyme inhibitors (ACE inhibitors) for secondary prevention of cardiovascular events. Lifestyle modifications, including smoking cessation counseling, dietary modifications (low-fat diet), and a structured exercise program, were emphasized.

Long-term management focused on aggressive lipid-lowering therapy to achieve target LDL cholesterol levels (<70 mg/dL) despite the genetic predisposition. Periodic follow-up visits included monitoring of lipid levels, adherence to medications, and assessment of cardiovascular risk factors.

## Outcome

During subsequent clinic visits, Mr. A demonstrated significant improvement in symptoms and adherence to the prescribed treatment regimen. Repeat lipid profile showed a gradual decline in LDL cholesterol levels with continued statin therapy and lifestyle modifications. Serial echocardiograms revealed preserved left ventricular function without evidence of new ischemic changes.

## Discussion

This case underscores the importance of recognizing and managing AMI in young adults with FH, a genetically determined disorder associated with elevated LDL cholesterol levels and premature atherosclerosis. Despite aggressive lipid-lowering therapy initiated during adolescence, our patient experienced an AMI, highlighting the residual cardiovascular risk in FH despite treatment.

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The early onset of AMI in FH patients necessitates a comprehensive approach to risk reduction, including intensive lipid-lowering therapy, lifestyle modifications, and close monitoring for cardiovascular events. Genetic testing plays a crucial role in confirming the diagnosis of FH and guiding therapeutic strategies aimed at reducing cardiovascular morbidity and mortality.

## Conclusion

In conclusion, this case report illustrates the clinical challenges and management strategies in a young adult with FH who presented with AMI. Early recognition, aggressive lipid-lowering therapy, and comprehensive secondary prevention are essential in optimizing outcomes and reducing the burden of cardiovascular disease in this high-risk population.

## Key takeaways

1. Acute myocardial infarction can occur in young adults with familial hypercholesterolemia despite early initiation of statin therapy.
2. Genetic testing is valuable in confirming the diagnosis of FH and guiding personalized treatment approaches.
3. Comprehensive management, including intensive lipid-lowering therapy and lifestyle modifications, is crucial for reducing cardiovascular risk in FH patients.

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