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*Case Study*

# Case Study: Acute Inferior Myocardial Infarction in a 46-Year-Old Male with Large Ectatic Right Coronary Artery and Significant Thrombus Burden Post-Insecticide Exposure

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## A B S T R A C T

We report a case of a 46-year-old male, medically free and an ex-smoker, who presented with an acute inferior myocardial infarction (MI). Initial angiography revealed a severe ectatic right coronary artery (RCA) with a significant thrombus burden. Multiple interventions, including thrombus aspiration and ballooning, were unsuccessful. Despite initial setbacks, medical management with Aggrastat and heparin led to clinical improvement. This case highlights the challenges and management strategies for patients with ectatic coronary arteries and significant thrombus burdens, particularly when environmental factors such as exposure to insecticides may play a role.

**Keywords:** Acute myocardial infarction, coronary artery ectasia, thrombus burden, thrombus aspiration, glycoprotein IIb/IIIa inhibitors, angiography, intravascular ultrasound, insecticide exposure, pyrethroids organophosphates, carbamates

## Introduction

Acute myocardial infarction (MI) remains a leading cause of morbidity and mortality worldwide. Ectatic coronary arteries, characterized by abnormal dilatation, pose unique challenges in the management of acute coronary syndromes due to their propensity for thrombus formation and embolization[1]. Insecticides, commonly used in agricultural and household settings, have been associated with adverse cardiovascular outcomes. Chemical components such as pyrethroids organophosphates and carbamates can induce vasoconstriction, increase platelet aggregation and promote thrombus formation, thereby exacerbating underlying coronary anomalies[2-4].

Long-term and acute exposures to pesticides have been linked to heightened cardiovascular risk and endothelial dysfunction

through oxidative stress and inflammatory pathways[5-7]. This case study describes the clinical presentation, diagnostic challenges and management of a patient with an acute inferior MI and a severely ectatic RCA with a substantial thrombus burden, potentially triggered by acute insecticide exposure.

## Case Presentation

A 46-year-old male, with no significant medical history and a former smoker who quit four years ago after smoking for seven years, presented to the emergency department with acute onset of shortness of breath and severe chest heaviness. The symptoms began after he sprayed insecticide in his room, opened the window and subsequently, his wife unknowingly closed it. The patient went to sleep around 4 AM and awoke with the described symptoms. On clinical examination upon presentation, he was in visible distress, diaphoresis was noted and his vital signs revealed blood pressure of 135/85 mmHg, pulse rate of 92 beats per minute and oxygen saturation of 96% on ambient air. Physical examination of the cardiovascular and respiratory systems was otherwise unremarkable, with no audible murmurs, gallops or pulmonary rales. He had no prior family history of premature coronary artery disease, dyslipidemia or sudden cardiac death.

### Investigations and initial management

Upon arrival at the emergency room, an electrocardiogram (ECG) showed signs consistent with an inferior MI. The patient was promptly taken to the catheterization lab for further evaluation and management. Coronary angiography revealed a severely ectatic RCA with a significant thrombus burden, demonstrating a distinct area of luminal stenosis **(Figure 1A)**.

Attempts at thrombus aspiration were unsuccessful due to the size of the thrombus. Multiple ballooning attempts also failed to restore adequate blood flow. Intravascular ultrasound (IVUS) conducted during the initial procedure confirmed a markedly enlarged ectatic artery with lumen diameters measuring 8.7 mm and 8.6 mm **(Figure 1B)**. There was dropout of the ultrasound wave at the mid-segment due to the artery's exceptionally large diameter, as mapped along the longitudinal IVUS pullback view **(Figure 1C)**.

Pharmacological agents, including intracoronary Aggrastat (a glycoprotein IIb/IIIa inhibitor), adenosine and nitroglycerin, were administered. These interventions improved flow to the mid-distal RCA. However, there remained TIMI 0 flow (no flow) in the very distal RCA, which supplies the posterior lateral branch (PLB) and posterior descending artery (PDA), due to a well-organized large thrombus burden in that area. Consequently, the procedure was halted and the patient was placed on continuous Aggrastat and heparin infusion for 48 hours.

### Follow-up and subsequent management

The patient was brought back for a follow-up procedure 48 hours later. Repeat angiography showed improved flow to the distal RCA, PLB and PDA, although a large thrombus burden remained in the distal RCA. The patient continued on Aggrastat and heparin infusion for an additional three days. During his inpatient hospital stay, the patient exhibited significant and progressive clinical improvement, marked by a complete resolution of his chest pain alongside gradual stabilization and normalization of the ST-segment elevation on continuous ECG monitoring. Furthermore, tracking of cardiac biomarkers demonstrated a favorable response to the ongoing medical regimen; serum troponin levels, which had initially peaked at a high of 52,000 ng/L, progressively trended downward to 20,000 ng/L, directly confirming a meaningful reduction in active myocardial injury.

## Discussion

Coronary artery ectasia (CAE) represents a rare and challenging anatomical anomaly characterized by diffuse or localized abnormal dilatation of the coronary segments. Managing acute coronary syndromes in the presence of CAE is notoriously difficult due to disturbed, sluggish blood flow dynamics, marked blood stasis and a very high propensity for massive intracoronary thrombus formation. As documented by[8], the drastically increased vessel diameter complicates standard mechanical revascularization strategies, as devices such as aspiration catheters and balloon angioplasty often fail to effectively capture or compress massive, well-organized thrombus burdens. These diagnostic and anatomical characteristics are clearly illustrated in Figure 1, where baseline angiography demonstrates the localized stenosis **(Figure 1A)** and IVUS imaging confirm the massive luminal diameter reaching up to 8.7 mm **(Figure 1B, 1C)**.

In this particular case, mechanical interventions initially proved unsuccessful, necessitating a shift toward intensive pharmacological support. The administration of intracoronary and continuous intravenous glycoprotein IIb/IIIa inhibitors (such as Aggrastat) alongside heparin played a pivotal therapeutic role. Glycoprotein IIb/IIIa inhibitors block the final common pathway of platelet aggregation, which is critical when managing large, refractory thrombotic loads where mechanical tools fall short[9].

Beyond the anatomical complexities of CAE, a particularly compelling aspect of this presentation is the temporal relationship between acute indoor insecticide inhalation and the onset of myocardial infarction. While environmental and occupational pesticide exposures have traditionally been studied in the context of chronic morbidity and mortality—such as cancer, neurological disorders and long-term cardiovascular risks[5,6]—acute toxicological triggers are increasingly recognized in precipitating sudden cardiac events. Household insecticides commonly contain active ingredients like pyrethroids organophosphates or carbamates. These chemical agents are capable of inducing severe endothelial dysfunction, provoking profound systemic oxidative stress and promoting localized vascular inflammation[10,11,7].

Furthermore, acute exposure to these neurotoxic and vasoactive compounds can stimulate sympathetic hyperactivity and direct smooth muscle contraction, triggering acute coronary vasospasm. In a patient with pre-existing, subclinical coronary ectasia, a toxicant-induced vasospasm combined with a hypercoagulable state creates a "perfect storm" for rapid stasis-mediated thrombosis. The inhalation of concentrated aerosols in a poorly ventilated room, followed by prolonged nocturnal exposure, likely initiated a cascade of acute vasospasm and accelerated platelet clumping within the already dilated and vulnerable right coronary artery. This dual mechanism—underlying structural ectasia compounded by acute toxic environmental exposure—underscores the critical need for clinicians to meticulously evaluate environmental and occupational history when encountering atypical or unprovoked acute coronary syndromes.

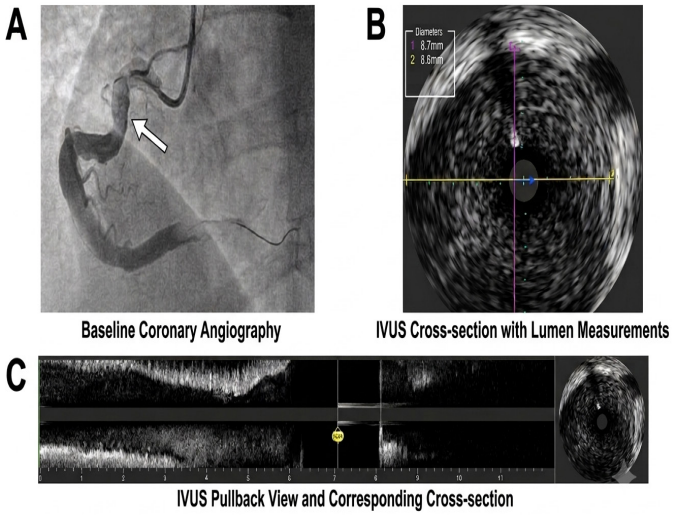


**Figure 1:** Multimodal imaging of the coronary vessel. (A) Baseline coronary angiography showing a distinct stenosis (white arrow). (B) Intravascular ultrasound (IVUS) cross-sectional image with marked lumen diameter measurements (8.7mm and 8.6mm). (C) IVUS longitudinal pullback map showing the full length of the imaged vessel

## Conclusion

This case highlights the complexities and potential strategies in managing acute MI in patients with ectatic coronary arteries and significant thrombus burdens. When mechanical interventions encounter limitations due to adverse vascular anatomy and high thrombotic volume, continuous pharmacological therapy with glycoprotein IIb/IIIa inhibitors and full anticoagulation offers a reliable alternative strategy. Moreover, the role of acute environmental exposures, such as heavy indoor insecticide inhalation, in precipitating acute coronary events should not be underestimated in clinical practice. Incorporating thorough toxicological and environmental exposure assessments into routine history-taking is essential for identifying unusual triggers of acute coronary syndromes. Further studies are warranted to develop optimized treatment protocols for this unique subset of patients and to elucidate the precise pathways by which chemical insecticides induce acute coronary events.

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