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Case Reports

Acute ST-Elevation Myocardial Infarction in a 25-Year-Old Female with Polycystic Ovary Syndrome: A Cardiometabolic Risk in Women of Reproductive Age

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Abstract

Background: Acute myocardial infarction is relatively rare in young patients. The age of onset gradually decreases due to multiple risk factors. The causes of Myocardial Infarction (MI) among patients aged less than 45 can be divided into four groups: atheromatous coronary artery disease, non-atheromatous coronary artery disease, hypercoagulable states, and MI related to substance misuse.

Case Illustration: A 25-year-old female came to the emergency department with chest discomfort for the past 40 minutes, has a history of diabetes for the past 3 years, and a Polycystic Ovary Syndrome (PCOS) history. Her vital signs show elevated Blood Pressure (BP) 150/100 mmHg. ECG showed sinus rhythm with ST elevation in the anterior leads. Random Blood Glucose (RBG) was 477 mg/dL, High Sensitivity (HS)-troponin 403 ng/L, and blood ketone 3.1 mmol/L. She was initially treated with: ticagrelor 180 mg, Acetosal 320 mg, insulin 4 u/hour. She was diagnosed with ST-Segment Elevation Myocardial Infarction (STEMI) and Diabetic Ketoacidosis (DKA). Coronary angiography revealed 95% stenosis in proximal Left Anterior Descending (LAD) and was treated as the culprit lesion, while 85% stenosis in mid Right Coronary Artery (RCA) was considered as the residual stenosis. Primary Percutaneous Coronary Intervention (PCI) was initiated at the proximal LAD, and post-PCI angiography showed a good result with TIMI 3 flow to the distal LAD.

Conclusions: PCOS increases cardiovascular risk primarily by promoting insulin resistance and metabolic dysfunction. Young individuals suspected of elevated cardiovascular risk should undergo a comprehensive cardiometabolic evaluation. PCI remains the cornerstone treatment for STEMI across all ages due to its well-established mortality benefit.

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Keywords: CAD, PCOS, LAD, DM

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Introduction

Acute Myocardial Infarction (AMI) is relatively rare in younger individuals, with reported incidence rates ranging from 2% to 10%.[1-2] Although younger patients generally have a more favorable prognosis and the condition occurs less frequently in individuals under the age of 45, AMI can still be life-threatening and lead to lasting disability.[3]

In young adults, AMI can result from a variety of causes, broadly categorized into four main groups: atheromatous and non-atheromatous Coronary Heart Disease (CHD), hypercoagulable states, and substance abuse. In young individuals, the development of atheromatous CHD is linked to the same common risk factors seen in adults. Smoking is highly prevalent, affecting up to 92% of young patients, especially those under 40 years old. Non-atheromatous causes include congenital coronary artery abnormalities, though rare, which can cause myocardial infarction in young adults. One such condition, myocardial bridging, can lead to significant ischemia and MI. Hypercoagulable states such as antiphospholipid syndrome, which often affects young adults in their 30s, are associated with recurrent arterial and venous thrombosis. It can occur as a primary condition or secondary to autoimmune diseases like systemic lupus erythematosus. Patients with this syndrome frequently show increased platelet adhesiveness and early signs of atherosclerosis. Substance abuse, particularly cocaine use, significantly increases the risk by inducing coronary vasospasm, with other substances like amphetamines and cannabis. Excessive alcohol intake has been connected to MI in young people, although its exact role is unclear.[4]

Case Illustration

A 25-year-old woman presented to the emergency department with acute chest discomfort lasting for 40 minutes. Her past medical history was notable for type 2 diabetes mellitus, diagnosed three years earlier, and polycystic ovary syndrome (PCOS). A comprehensive clinical assessment was performed to evaluate her condition. On examination, her vital signs were as follows: heart rate 100 beats per minute, respiratory rate 20 breaths per minute, blood pressure 150/100 mmHg, and body temperature 36.0°C. Physical examination revealed no significant abnormalities.

An Electrocardiogram (ECG) was performed, revealing a sinus rhythm with significant ST-segment elevation in the anterior leads. (Figure 1).

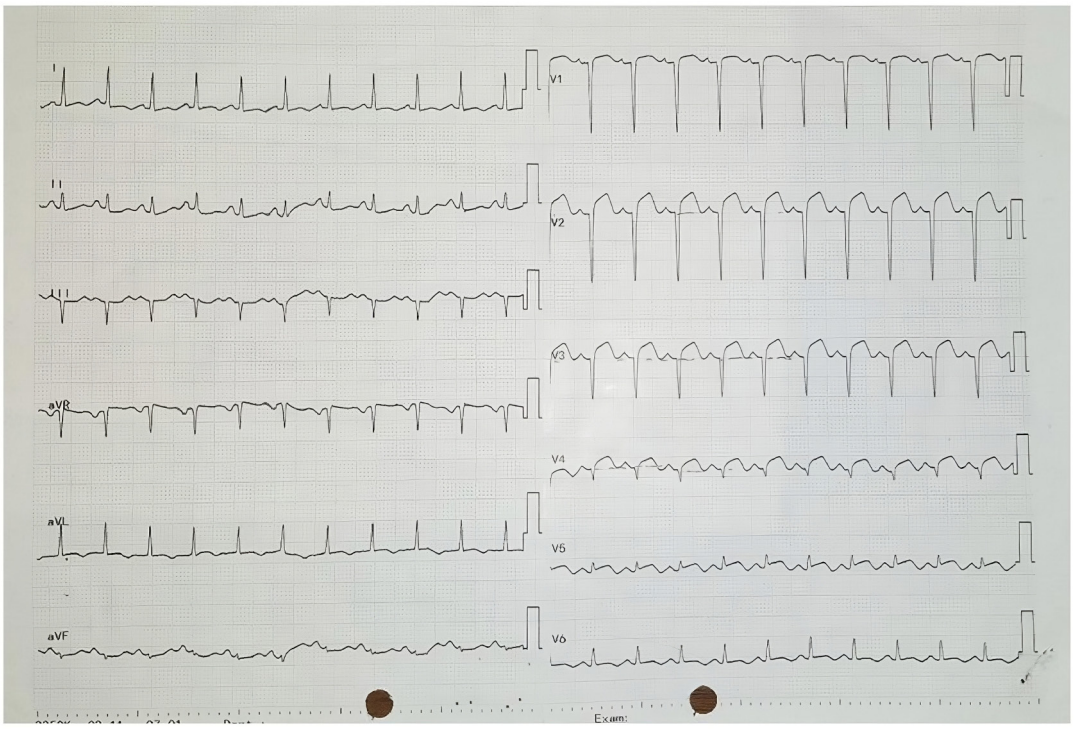

Figure 1. Electrocardiogram showing sinus tachycardia and anterior STEMI (ST-Elevation Mycocardial Infarction).

Immediate treatment was initiated. She received a loading dose of ticagrelor 180 mg along with acetylsalicylic acid 320 mg, atorvastatin 80 mg, and morphine 1 mg due to pain. Her RBG was 477 mg/dL, therefore, insulin administration was deferred until serum potassium levels were determined. The patient was admitted to the cathlab for primary Percutaneous Coronary Intervention (PCI).

Laboratory findings revealed elevated High Sensitivity (HS)-troponin 403 ng/L, blood ketone 3.1 mmol/L, confirming a diagnosis of Diabetic Ketoacidosis (DKA). The urea level was 25 mg/dL, Natrium 132 mmol/L, Kalium 4.2 mmol/L, and Chloride 95 mmol/L. An insulin infusion was commenced at a rate of 4 units per hour to manage hyperglycemia and facilitate correction of ketoacidosis.

Coronary angiography revealed significant multi-vessel coronary artery disease. A critical 95% stenosis was localized to the proximal LAD, which was identified as the culprit lesion responsible for the patient's acute myocardial ischemia. Furthermore, a significant residual stenosis of 85% was noted in the mid RCA. This secondary lesion was designated for future, staged percutaneous coronary intervention (Figure 2).

Following PCI, post-procedural angiography demonstrated successful revascularization with TIMI grade 3 flow to the distal LAD (Figure 3).

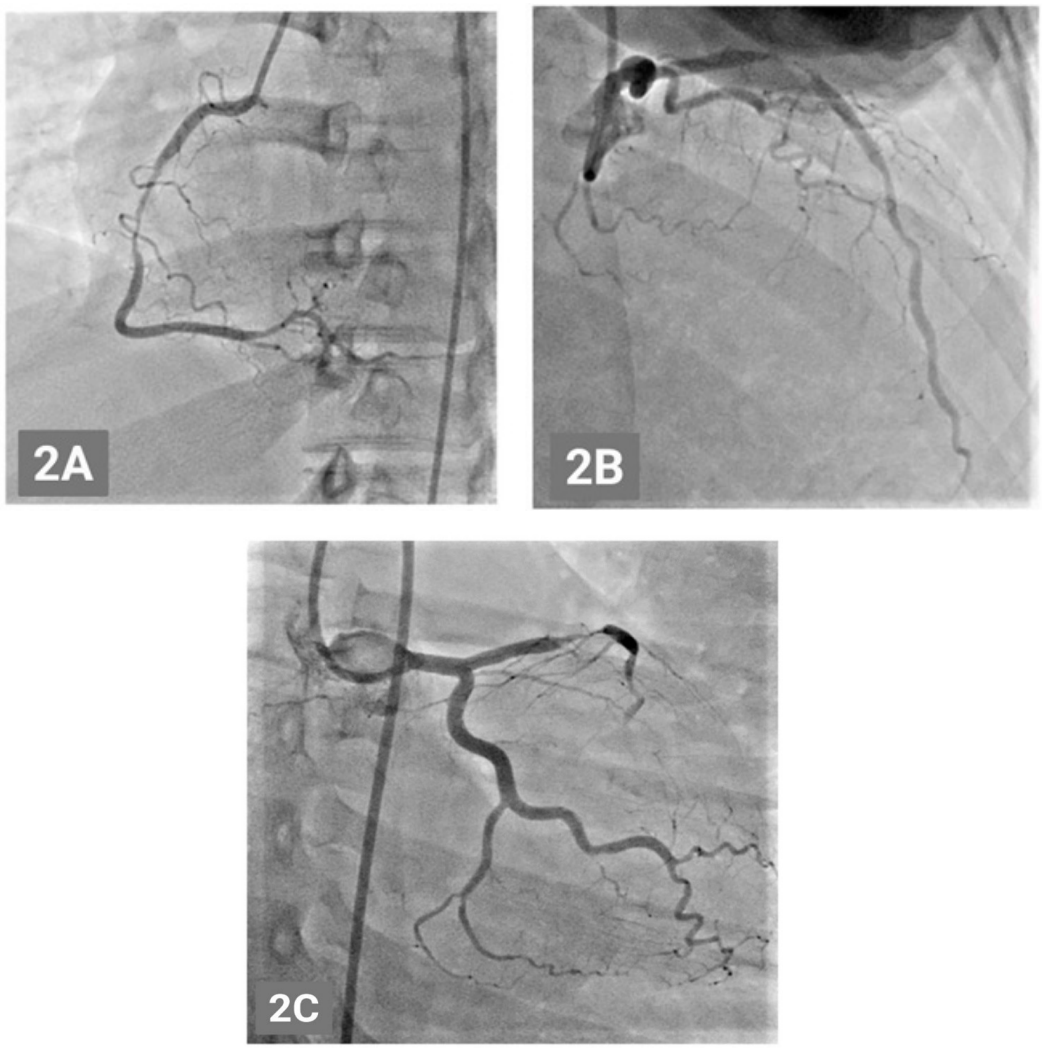

Figure 2. Coronary angiography views revealing multi-vessel stenosis. (A) Right coronary artery, RAO CRA view. (B) Left anterior descending, RAO CRA view. (C) Left anterior descending, RAO CAU view.

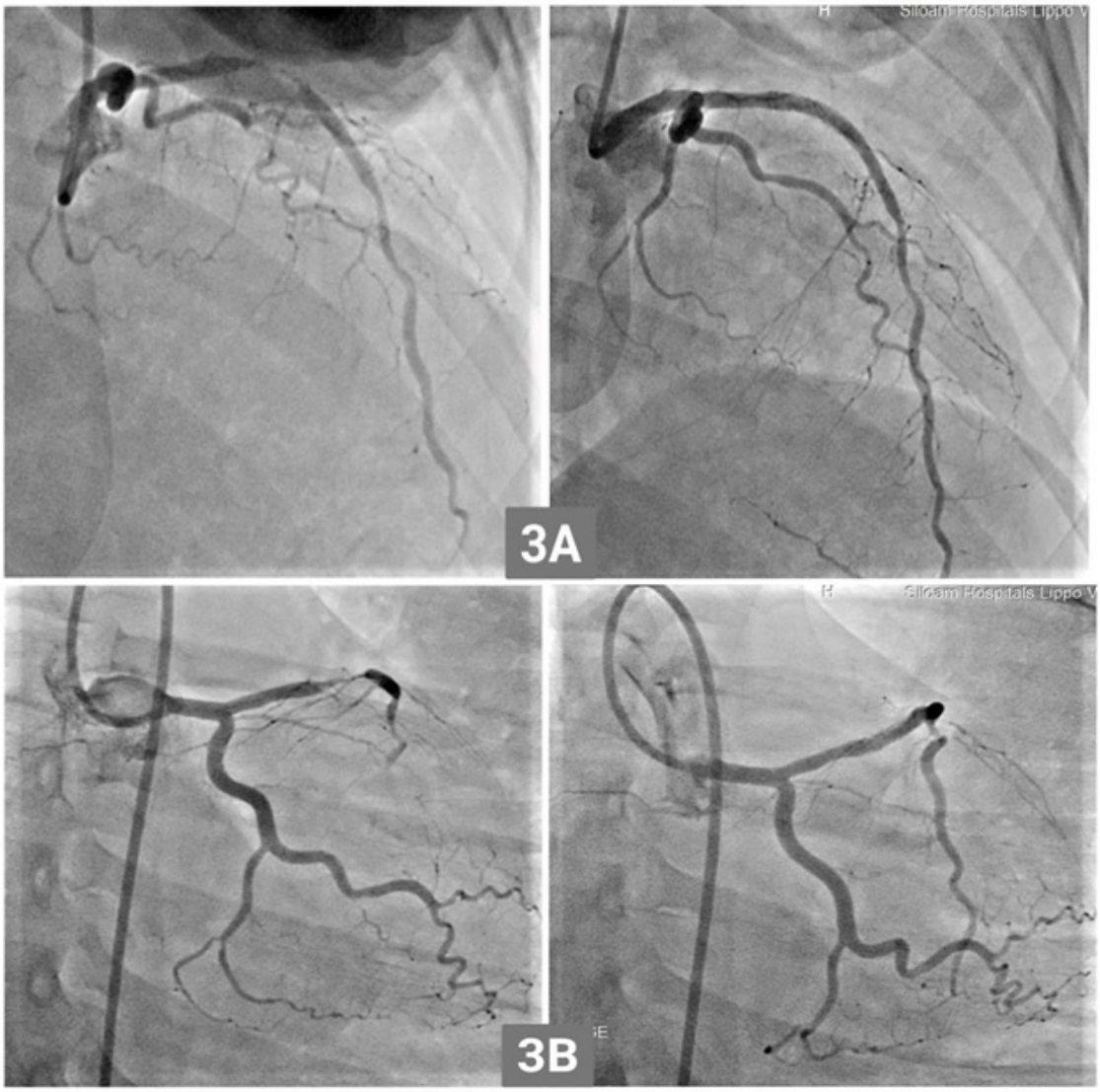

Figure 3. Post-percutaneous coronary intervention (PCI) results in the Left Anterior Descending Artery (LAD). (A) Left anterior descending, RAO CRA view & post-PCI. (B) Left anterior descending, RAO CAU view & post-PCI.

Femoral access was preferred due to the spasm of the radial artery. The lesion was predilated with a balloon (®Pantera pro-2.5x20 mm) inflated to 18 atm. Drug Eluting Stent (DES ®Supraflex Cruz 3.5x20 mm) was inserted at proximal LAD, inflated 16 atm. Door-to-balloon time was 90 minutes, and the duration from the onset of symptoms to coronary artery recanalization was 130 minutes. The patient was stable after PCI. Post-revascularization ECG was done, showing anterior Q waves with V2-V6 T inversion. (Figure 4).

Discussion

AMI in young adults is predominantly caused by premature atherosclerosis, with plaque rupture or erosion accounting for nearly 90% of cases with comorbidities including diabetes mellitus, metabolic syndrome, hypertension, chronic kidney disease, and smoking. The remaining 10% arise from non-atheromatous etiologies.[5] New studies emphasize the rising importance of non-traditional risk factors, such as chronic inflammation, autoimmune disorders, hypercoagulability, and substance abuse, in causing acute MI among younger populations.[6]

Historically, AMI in young women was considered uncommon, primarily due to the presumed protective effects of estrogen.[7] However, this perspective is challenged by the presence of PCOS, a common endocrinological disorder affecting 6–13% of women of reproductive age worldwide.[8] This endocrinological disorder is increasingly acknowledged as a contributor to heightened cardiovascular risk factors. A meta-

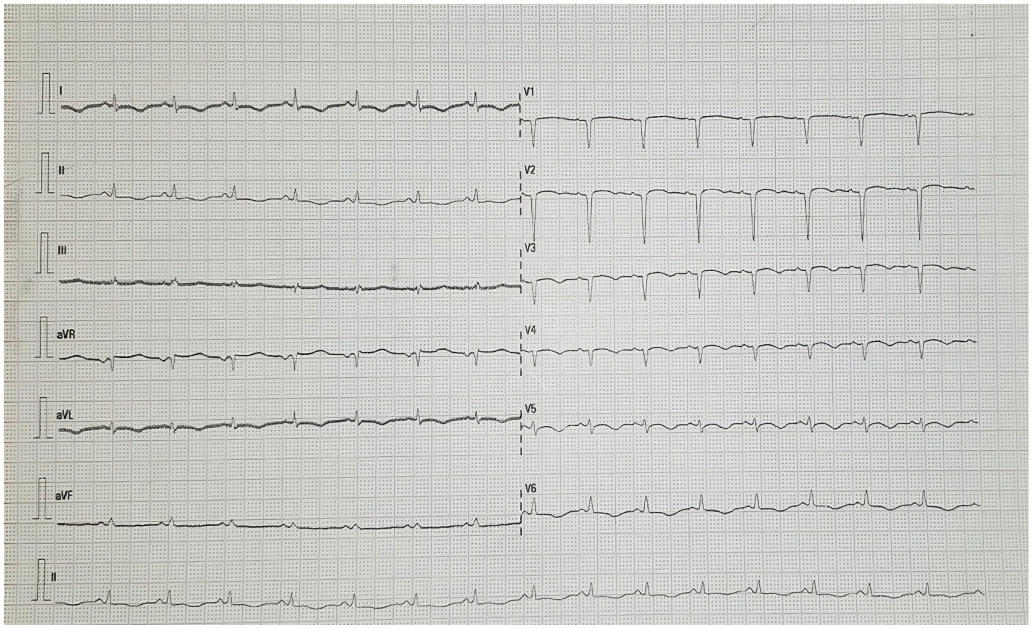

Figure 4. Electrocardiography post Percutaneous Coronary Intervention (PCI).

analysis of 104,392 subjects demonstrated that PCOS is independently associated with increased Cardiovascular Disease (CVD) risk, particularly coronary heart disease (odds ratio [OR] 1.44; 95% confidence interval [CI]: 1.13–1.84).[9-10.]

PCOS is characterized by hyperandrogenism, insulin resistance, and metabolic disturbances such as dyslipidemia, hypertension, and obesity—all established traditional cardiovascular risk factors.[11] Elevated free testosterone levels have been correlated with higher systolic blood pressure and adverse lipid profiles, further aggravating endothelial dysfunction. Importantly, women with PCOS frequently develop type 2 Diabetes Mellitus (DM).[12] DM is a complex metabolic disorder characterized by chronic hyperglycemia due to defects in insulin secretion, insulin action, or both.[13] In this case, insulin resistance is most likely driven by the presence of PCOS. Hyperandrogenism, often resulting from PCOS, impairs insulin signaling both directly and indirectly by altering fat distribution and promoting a proinflammatory state. Furthermore, a feedback loop exists in which hyperinsulinemia stimulates additional androgen production. This complex interplay contributes to the metabolic and reproductive abnormalities characteristic of PCOS.[14]

The link between DM and CVD is multifaceted and involves both macrovascular and microvascular dysfunction. Chronic hyperglycemia contributes to endothelial dysfunction by impairing nitric oxide bioavailability, promoting oxidative stress, and triggering inflammatory pathways. These effects lead to inflammation and injury of blood vessels, accelerating atherosclerosis development—a key factor in coronary artery disease, stroke, and peripheral vascular disease.[15]

In the current case, the patient's history of PCOS combined with poorly controlled diabetes mellitus presents a unique intersection of metabolic and cardiovascular risks. While substance abuse was ruled out based on clinical presentation and history, the prothrombotic milieu associated with PCOS likely contributed to the acute event.[16] Furthermore, hyperandrogenism in PCOS exacerbates insulin resistance and systemic inflammation, leading to endothelial injury and the promotion of atherosclerosis. This chronic inflammatory state impairs endothelial function, which is a key precursor to plaque development and instability.[17] PCOS often coexists with obesity, which at the cellular level induces a chronic inflammatory state that damages vascular walls and impairs endothelial function. This sustained inflammation promotes venous wall fibrosis and dysfunction, leading to impaired blood flow and subsequent venous stasis.[18] Collectively, these pathological changes contribute to the components of Virchow's triad—endothelial injury, hypercoagulability, and blood flow abnormalities — thereby increasing the risk of thrombosis in this case.[19]

The coexistence of DKA and STEMI poses an added complexity. DKA is known to provoke systemic inflammation and a prothrombotic state with increased Von Willebrand factor and decreased free protein S and protein C activity, ultimately

impairing myocardial perfusion and increasing the risk of adverse cardiovascular outcomes.[20]

In this case, femoral access was preferred due to the spasm of the radial artery. Door-to-balloon time was 90 minutes, and the duration from the onset of symptoms to coronary artery recanalization was 130 minutes. The patient was stable after PCI and started a routine medications consisting of 80 mg of acetosal, 90 mg of ticagrelor, 40 mg of atorvastatin, 2.5 mg of bisoprolol after PCI.

STEMI should be managed according to standard protocols, regardless of patient age. Urgent reperfusion via primary PCI remains the cornerstone of treatment across all age groups due to its proven mortality benefit. Compared to other cases, this case follows the same management approach, with the only additional consideration being the implementation of the DKA protocol.

It is important to note that women and diabetic patients are more likely to present with atypical symptoms, leading to potential diagnostic delays.[21] A comprehensive cardiometabolic evaluation should be performed even in young individuals who are suspected of being at increased cardiovascular risk. Timely identification reduces the risk of associated complications.

Conclusion

This case emphasizes the importance of comprehensive cardiovascular risk assessment even in young females. PCOS increases cardiovascular risk primarily by promoting metabolic disturbances. DKA triggers systemic inflammation and a prothrombotic state, together with endothelial dysfunction due to hyperglycemia, leading to impaired blood flow and venous stasis—key elements of Virchow's triad. The diagnosis might be challenging in some cases due to the presence of atypical symptoms, which are more commonly observed in female and diabetic patients, potentially leading to delayed recognition. STEMI should be treated according to established standard protocols regardless of the patient's age, with primary PCI as the urgent and vital treatment due to its proven impact on reducing mortality across all age groups. Future investigations, including advanced vascular imaging, may provide deeper insights into the structural and functional vascular changes in such patients, guiding personalized therapeutic strategies.

List of Abbreviations

AMI Acute Myocardial Infarction
CAD Coronary Artery Disease
CAU Caudal
CRA Cranial
CVD Cardiovascular Disease
DES Drug-Eluting Stent
DKA Diabetic Ketoacidosis
DM Diabetes Mellitus
ECG Electrocardiogram
IVUS Intravascular Ultrasound
LAD Left Anterior Descending artery
LCX Left Circumflex artery
MI Myocardial Infarction
PCI Percutaneous Coronary Intervention
PCOS Polycystic Ovary Syndrome
RAO Right Anterior Oblique
RBG Random Blood Glucose
RCA Right Coronary Artery
SCAD Spontaneous Coronary Artery Dissection
STEMI ST-Elevation Myocardial Infarction

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors Contributions

E.A.K. collected the clinical, laboratory, and imaging data. Contributed to the literature review, case analysis, discussion, and assisted with manuscript formatting and editing. N. N. W. supervised the clinical aspects of the case, contributed to the literature review, and provided expert review of the manuscript drafts. W. W. S. participated in case analysis and assisted with manuscript formatting and editing. G. L. T. contributed to case analysis and supported manuscript formatting and editing. All authors have read and approved the final manuscript.

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Conflict of Interest

The author declared no conflict of interest.

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Generative AI and AI-Assisted Technologies in the Writing Process
Generative AI tools were utilized to support the preparation of this manuscript. Perplexity AI was used to assist in searching for up-to-date and relevant case reports and literature. ChatGPT (OpenAI) was used to help refine wording, improve clarity, and correct grammar and spelling. All content was critically reviewed and edited by the authors to ensure accuracy, originality, and adherence to ethical standards.

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