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Case Reports

Antiphospholipid Syndrome Manifesting as Myocardial Infarction: A Case Report and Review of the Literature

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Abstract

Background: Antiphospholipid Syndrome (APS) is characterized by the presence of antiphospholipid antibodies, including lupus anticoagulant, anticardiolipin antibodies, and β2-glycoprotein I. These antibodies target phospholipids and associated proteins, leading to diverse clinical manifestations such as stroke, myocardial infarction, and deep vein thrombosis. Acute myocardial infarction caused by arterial thromboembolism is a rare first manifestation of APS.

Case Illustration: We present a case study of a 37-year-old female with a history of Deep Vein Thrombosis (DVT) and no identifiable risk factors, who, a decade ago and recently, experienced a sudden Myocardial Infarction (MI) due to arterial thrombosis. The angiographic appearance and the need for angioplasty were challenging at presentation, as the clot was migratory and had moved between coronary vessels. We conducted a thrombophilia evaluation due to the unusual site of thrombosis and the patient's age at presentation, which led us to establish the diagnosis of APS.

Conclusions: This case highlights the rare but essential association between APS and recurrent MI, as well as the unique angiographic behavior of APS-related coronary thrombi that can complicate percutaneous coronary intervention. While long-term vitamin K antagonist therapy remains standard for secondary prevention, clinicians should recognize APS as a potential cause of MI in young patients and anticipate procedural and therapeutic challenges beyond anticoagulation alone.

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Keywords: Antiphospholipid Syndrome, Acute Coronary Syndrome, Myocardial Infarction, Thrombosis.

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Introduction

Antiphospholipid Syndrome (APS) is an autoimmune disorder characterized by the presence of antiphospholipid (aPL) antibodies, which increase the risk of thrombotic events in both veins and arteries.[1-2] These antibodies, including Lupus Anticoagulant (LA), anticardiolipin antibodies (aCL), and β2-glycoprotein I (β2GPI), target phospholipids and associated proteins, leading to diverse clinical manifestations such as stroke, Myocardial Infarction (MI), and Deep Vein Thrombosis (DVT).[1,3-4] This case report underscores the complexities of managing acute myocardial infarction in the context of APS, exemplified by a 37-year-old woman with a history of DVT, presenting with severe chest pain and hypotension at Tehran Heart Center. Her clinical journey, marked by multiple thrombotic events and the eventual identification of aPL, highlights the intersection of APS with acute coronary syndromes and the imperative for comprehensive thrombophilia screening in similar cases. This report addresses the gap in understanding the relationship between thrombophilia and coronary artery disease, emphasizing the importance of greater clinical awareness and personalized treatment approaches to manage such complex clinical situations.

Case Illustration

A 37-year-old woman presented to the emergency department with an acute onset of severe chest pain and hypotension. Electrocardiography revealed ST-segment elevation in leads V1–V2, which corresponds to the septal wall region (Figure 1). The patient had a history of DVT 10 years ago, which had been treated with anticoagulation therapy. In the emergency department, her vital signs showed hypotension and tachycardia, and a physical exam revealed an S3 gallop sound and clear lungs. Emergency coronary angiography showed single-vessel disease with complete occlusion of the Left Anterior Descending (LAD) artery (Figure 2). Following successful wire passage through the

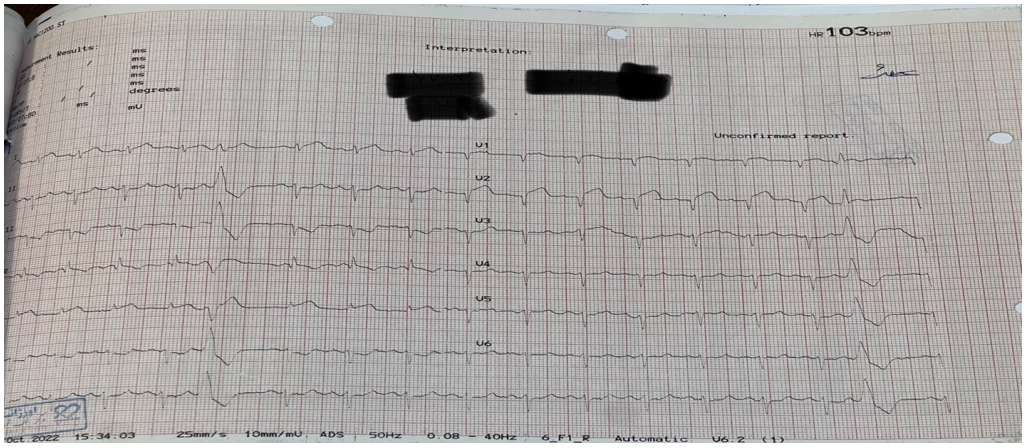

Figure 1. Electrocardiogram upon arrival of the patient to the emergency department.

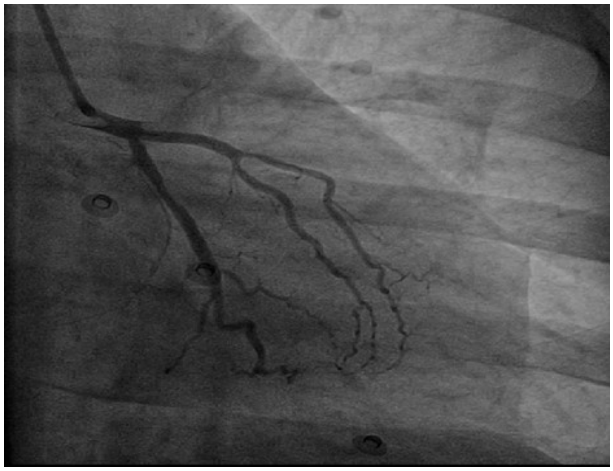

Figure 2. Coronary angiogram revealed that the Left Anterior Descending (LAD) artery is occluded from the ostioproximal.

affected artery, a significant clot was identified as the cause of the LAD artery occlusion. Although a thrombectomy was performed, the chest pain persisted, leading us to place a stent in the proximal LAD. However, shortly after the initial treatment, the patient experienced a recurrence of severe chest pain, similar in intensity to her initial symptoms. Continuous monitoring revealed the development of ventricular tachycardia. Synchronized cardioversion was promptly and successfully delivered with a 200-joule electrical shock. Following this intervention, a repeat examination of the coronary arteries revealed a new occlusion in the proximal segment of the Left Circumflex (LCx) artery and the mid part of the LAD, as depicted in Figure 3. The thrombus in the LAD artery appeared to have migrated and now obstructed the LCx artery. A wire was promptly navigated through the LCx occlusion, and a decision was made to deploy a stent at this location. Following balloon dilation to expand the arteries, a drug-eluting stent was placed in the proximal LAD and LCx, achieving successful reperfusion with TIMI grade 3 flow. The final angiograms from both the initial LAD intervention and the subsequent LCx Percutaneous Coronary Intervention (PCI) demonstrated adequate stent apposition, no residual stenosis in the treated segments, and restoration of distal flow (TIMI Flow = 3) without evidence of thrombus or dissection (Figure 4). Following the post-dilation of the LCx stent, the patient experienced a recurrence of chest pain. A subsequent coronary angiogram revealed a significant stenosis in the midsection of the Ramus Intermedius (RI) artery (Figure 4), which was treated with balloon angioplasty. After these interventions, the patient was transferred to the Coronary Care Unit (CCU) without reporting any further chest pain and with stable vital signs. However, laboratory

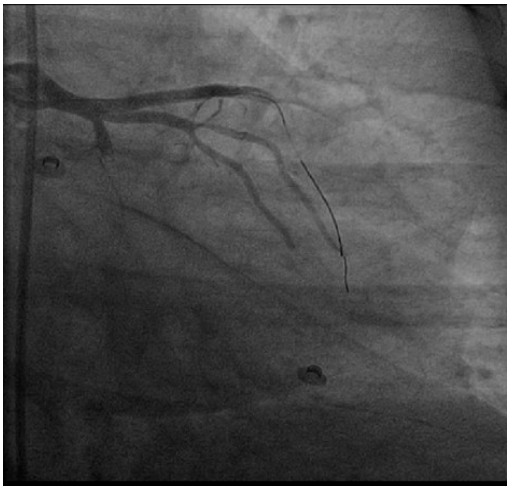

Figure 3. Coronary angiogram revealed left circumflex (LCx) artery occlusion.

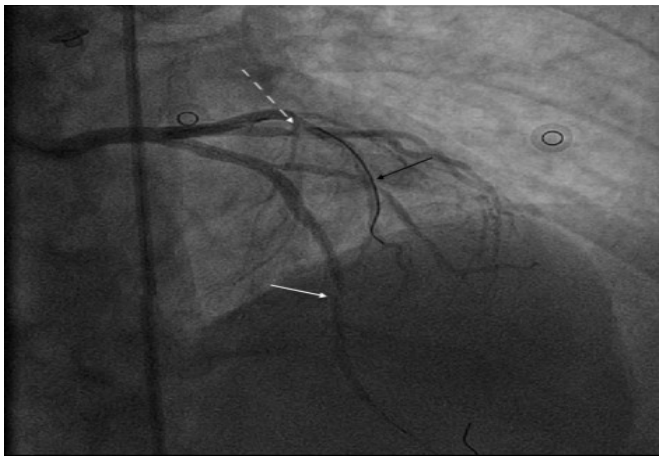

Figure 4. Coronary angiogram showed significant stenosis of the mid portion of the Ramus Intermedius (RI) artery. Black arrow: LCx is patent; there is a wire in LCx. White arrow: The LAD is patent; the wire is shown in the LAD. Dashed arrow: Significant stenosis in RI.

tests revealed a significantly elevated cardiac enzyme level, with a troponin T level of 47,300 ng/L, indicating substantial myocardial damage. Notably, despite the patient's denial of diabetes mellitus or dyslipidemia, laboratory results showed elevated total cholesterol (220 mg/dL), Low-Density Lipoprotein (LDL) cholesterol (170 mg/dL), and a hemoglobin A1c level of 11%, suggesting poorly controlled glycemic levels. Additional laboratory tests were conducted to determine the possibility of a thrombophilia disorder due to the patient's history of DVT and recent acute myocardial infarction caused by a clot. The results showed positive findings for anticardiolipin IgM (19 U/ml), anti-beta2 glycoprotein-1 antibody IgM (22 U/ml), and lupus anticoagulant (62 U/ml). These tests were repeated 12 weeks later, yielding consistent results. Based on these findings, we initiated an intensive antithrombotic treatment regimen to reduce the risk of future thrombotic events. The patient was prescribed a combination of Acetylsalicylic Acid (ASA, also known as aspirin), clopidogrel, and warfarin for one month, followed by clopidogrel and warfarin for an additional year. Long-term warfarin therapy was planned to be continued indefinitely to manage the risk of arterial thrombosis, with a target International Normalized Ratio (INR) of 2.5-3.5 to ensure optimal anticoagulation.

Discussion

APS is characterized by a state of hypercoagulability secondary to circulating antiphospholipid antibodies, which promote thrombus formation via several mechanisms. These antibodies induce endothelial dysfunction, activate platelets, and stimulate coagulation cascades, leading to increased fibrin generation and impaired fibrinolysis. As a result, patients with APS are at heightened risk for migratory and recurrent arterial thromboses, even within coronary vessels during interventional procedures.[5-6]

This case illustrates the procedural dilemmas posed by APS during PCI, particularly the need to balance anticoagulation to prevent new thrombus formation without precipitating bleeding complications or stent thrombosis. Initial management involved attempts at thrombus aspiration, which proved unsuccessful. Due to ongoing hemodynamic instability, coronary stenting was selected to restore flow. Alternative strategies, such as intracoronary thrombolytics, could be considered. Using IIb/IIIa inhibitors and follow-up angiography could also be considered, but was less optimal given the patient's instability.

Thrombotic events associated with APS can affect arteries and veins, leading to diverse symptoms and sequences of thrombotic events

Table 1. A review of the literature on patients with antiphospholipid syndrome who experienced myocardial infarction

	Semczuk-Kaczmarek et al.	Prandi et al.	Marti et al.	Qiu et al.	Cranley et al.	González-Cordero et al.	Ayan et al.	Smukowska-Gorynia et al.	Snipelisky et al.	Pervez et al.	Faizal et al.
Year	2020	2022	2014	2023	2018	2019	2019	2015	2013	2019	2021
Gender	Male	Male	Male	Male	Female	Male	Male	Female	Male	Female	Female
Age	25	51	47	35	51	26	23	43	55	27	46
Past medical history	SLE	No	Inferior MI, PTE, APS	ORIF (lumbar)	APS	No	No	Hx of heavy bleeding	No	Chronic pain syndrome	No
Other risk factors	Overweight (BMI = 29)	Smoking	Hyperco-agulability	Smoking	No	No	Smoking, Family history of premature CAD	Smoking	No	No	No
History of VTE	Yes	No	Yes	No	No	Yes	No	No	No	Yes	No
Known case of APS	Yes	No	No	No	Yes (CAPS)	No	No	No	No	Yes	No
MI localization	Anterior	Anterior	Inferior	Inferior	Inferior	Anterolateral	Anterolat-eral	Anterolateral. Anterior-Inferior (recurrent)	Lateral	Anterior (v4 through v6) and inferior (II, III, aVF)	Anterior-wall
Number of vessels	2	2	1	1	0	1	1	2	1	1	1

Target vessel	LAD and the left marginal artery	LM and ostial LAD	RCA	Posterior descending RCA	Slow flow in RCA	Proximal LAD	Distal LAD	LAD, CX, proximal LAD (recurrent)	RCA	Proximal LAD	Distal LAD
LVEF	47	40	N/A	N/A	N/A	<30	35-40	45	50	Preserved	N/A
Reccurrent MI during admission	No	No	No	Yes	No	Yes	No	Yes	Yes	No	No
Pharmacother spy during admission	DAPT, eptifibatide	Eptifibatide, DAPT	Heparin, abciximab, enoxaparin, DAPT	TT, tirofiban (discontinue), bivalirudin (replaced by fondaparinux), methylprednisolone	LMWH, warfarin, corticosteroid replacement therapy (due to adrenal crisis)	LMWH, nitrates, enalapril, metoprolol succinate, DAPT, eptifibatide	Warfarin, heparin, and antiischemic medications	DAPT, statin, eptifibatide ACE II inhibitor, beta-blocker, abciximab, TNG, UFH (replaced by LMWH), methylprednisolone followed by prednisone	Warfarin	TT	Enoxaparin, switched to oral anticoagulation with warfarin
Post-discharge treatment	TT	LMWH, clopidogrel and warfarin	DAPT, acenocoumarol, and atorvastatin	TT	TT, corticosteroid replacement	TT, hydroxychloroquine, high-dose statin, carvedilol, and enalapril	Not mentioned	TT, beta-blocker, ACE II inhibitor, pantoprazole, statin, and prednisone	Warfarin	TT	Enoxaparin was switched to oral anticoagulation with warfarin

SLE: Systemic Lupus Erythematosus; MI: Myocardial Infarction; PTE: Pulmonary Thromboembolism; APS: Antiphospholipid Syndrome; ORIF: Open Reduction and Internal Fixation; CAPS: Catastrophic Antiphospholipid Syndrome; CAD: Coronary Artery Disease; Hx: History; DAPT: Dual Antiplatelet Therapy; TT: Triple Therapy (Oral Anticoagulant Such As Warfarin + DAPT); LMWH: Low Molecular Weight Heparin; ACE II: Angiotensin-Converting Enzyme II; TNG: Trinitroglycerin; UFH: Unfractionated Heparin; RCA: Right Coronary Artery; LAD: Left Anterior Descending; LM: Left Main Artery.

depending on the number of vessels involved or the affected organ. The leading cardiac symptoms of APS include valve disease, myocardial infarction, and intracardiac thrombus. Table 1 demonstrates a review of the literature on the topic. The reported cases span young to middle-aged adults (early 20s to mid-50s) with a slight male predominance, and only a minority had a prior APS diagnosis or classical cardiovascular risk factors. Smoking and a history of venous thromboembolism were the most frequent additional risks. Our patient also had diabetes mellitus and dyslipidemia despite her denial in her past medical history. Diabetes and high cholesterol significantly increase cardiovascular risk and promote endothelial dysfunction in APS, thereby raising the likelihood and severity of arterial thrombosis and atherosclerosis progression. These conditions act synergistically with APS to accelerate vascular events, such as myocardial infarction and stroke, necessitating the aggressive control of both blood glucose and lipids.[7] Infarcts involved both anterior and inferior territories, most often affecting the LAD or right coronary arteries, with single-vessel disease predominating. Left ventricular function ranged from preserved to severely reduced, and recurrent infarction during the same admission occurred in several patients. Management varied but typically combined dual antiplatelet therapy and anticoagulation, often triple therapy with warfarin, alongside steroids or immunomodulation when catastrophic APS or adrenal crisis was suspected. These findings underscore the heterogeneous presentation of APS-related myocardial infarction and highlight the importance of early recognition and aggressive, tailored antithrombotic therapy to prevent recurrence and improve outcomes. A study by Cervera[1] investigated the prevalence of arterial thrombosis in a cohort of 1000 individuals diagnosed with APS. The results showed that 5.5% of these patients experienced MI, whereas only 2.8% of them presented with MI as the initial manifestation of APS.[1] Furthermore, Cervera et al. reported that antiphospholipid antibodies were present in approximately 11% of patients with MI, highlighting the potential role of APS in the pathogenesis of MI.[1] One of the most challenging issues in managing patients with APS is treating those with vascular thrombosis. In our case, the patient presented with acute MI, and Initial thrombus aspiration was attempted as a clot removal strategy; however, it failed to restore adequate coronary flow. Given the ongoing instability and the critical nature of the lesion, stent implantation was selected to promptly re-establish vessel patency,

secure the lesion, and minimize the risk of further flow compromise. The LCx occlusion could represent either a) procedure-related embolization of thrombotic material from the LAD during PCI or b) de novo in-situ thrombosis related to the hypercoagulable state of APS. The immediate temporal relationship to LAD manipulation and the abrupt angiographic appearance favor an embolic complication, whereas APS likely enhanced the thrombotic milieu and prolonged the occlusion. We have acknowledged both possibilities, but consider procedure-related embolization on a background of APS the most plausible explanation.[8] Consequently, we opted for a triple antithrombotic therapy regimen consisting of ASA, clopidogrel, and warfarin for an initial period of one month, followed by clopidogrel and warfarin for one year. After that, we decided to continue warfarin therapy indefinitely, with a target INR of 2.5-3.5. However, a significant challenge in treating APS patients is the use of warfarin or Direct Oral Anticoagulants (DOACs), which requires careful consideration and monitoring to balance the risk of thrombosis with the risk of bleeding.

Initially, it is essential to stratify APS patients into high-risk and low-risk groups. A history of arterial thrombosis or triple positivity for LA, aCL, and β2GPI[9] typically characterizes high-risk patients. The Trial of Rivaroxaban in Antiphospholipid Syndrome (TRAPS) revealed that rivaroxaban was associated with an increased incidence of adverse events in high-risk APS patients without providing any significant benefits and, instead, introducing additional risks.[10] Consequently, the 2019 European Society of Cardiology guidelines recommend against the use of DOACs in patients with APS, regardless of the APS subtype or the number of positive tests. Furthermore, a meta-analysis revealed that DOACs significantly increased the risk of subsequent arterial thrombosis, with a 5-fold higher risk than vitamin K antagonists (VKAs)[9], leading to a recommendation to use VKAs instead of DOACs for anticoagulation treatment and secondary prophylaxis of arterial thrombosis in APS patients.[11] A comprehensive review of case reports underscores the difficulties in managing APS in patients who have experienced MI. Despite aggressive treatment regimens, including triple therapy, a significant proportion of patients (3 out of 7) experienced recurrent MI. This high recurrence rate highlights the complexity of achieving optimal outcomes in patients with APS and MI. It emphasizes the critical importance of post-discharge treatment choices in long-term disease management. Established guidelines recommend indefinite treatment with VKAs for patients with APS.[11]

Conclusion

In conclusion, personalized management strategies are essential for patients with thrombophilia conditions. The recurrence of MI despite aggressive treatment underscores the complexities of treatment and the need for ongoing, tailored management. Further research is necessary to refine therapeutic approaches and improve outcomes for patients with APS who have cardiovascular complications.

List of Abbreviations

ACE II	Angiotensin-Converting Enzyme II
aCL	Anticardiolipin
aPL	Antiphospholipid
APS	Antiphospholipid Syndrome
ASA	Acetylsalicylic Acid
β2GPI	β2-glycoprotein I
BMI	Body Mass Index
CAD	Coronary Artery Disease
CAPS	Catastrophic Antiphospholipid Syndrome
CCU	Coronary Care Unit
CX	Circumflex Artery
DAPT	Dual Antiplatelet Therapy
DOACs	Direct Oral Anticoagulants
DVT	Deep Vein Thrombosis
Hx	History
INR	International Normalized Ratio
LA	Lupus Anticoagulant
LAD	Left Anterior Descending
LCx	Left Circumflex
LDL	Low-Density Lipoprotein
LM	Left Main Artery
LVEF	Left Ventricular Ejection Fraction
LMWH	Low-Molecular Weight Heparin
MI	Myocardial Infarction
ORIF	Open Reduction And Internal Fixation
PCI	Percutaneous Coronary Intervention
PTE	Pulmonary Thromboembolism
RCA	Right Coronary Artery
RI	Ramus Intermedius
SLE	Systemic Lupus Erythematosus
TIMI	Thrombolysis in Myocardial Infarction
TNG	Trinitroglycerin
TRAPS	The Trial of Rivaroxaban in

	Antiphospholipid Syndrome
TT	Triple Therapy (Oral Anticoagulant Such As Warfarin + DAPT)
UFH	Unfractionated Heparin
VTE	Venous Thromboembolism

Ethical Clearance

The participant in this case report has provided written informed consent to publish her clinical information, and any identifying information has been removed or anonymized to protect her privacy.

Publication Approval

All authors consent to the publication of this manuscript.

Authors Contributions

SS Conceptualized, drafted, and revised the manuscript critically for important intellectual content; SN drafted and revised the article; AM drafted and revised the article; HA conceptualized, designed, and critically revised the article for important intellectual content. All authors approved the final version to be published.

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Conflict of Interest

None.

Availability of Data and Materials

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Copyright/Permissions for Figures

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