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Cardiovascular Catastrophe in Catastrophic Antiphospholipid Syndrome: A Case Report

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Abstract: Catastrophic antiphospholipid syndrome (CAPS) is an exceedingly rare (< 1 % of APS cases) but life-threatening variant of antiphospholipid syndrome, marked by rapid, widespread thrombosis that can precipitate myocardial infarction, stroke, and multiorgan failure. We report a 72-year-old south Asian woman with established cardiovascular risk factors—rest angina, hypertension, type 2 diabetes, hyperlipidaemia, and arthritis—who presented with acute right lower-limb ischemia manifesting as pain, pallor, and coolness. CT angiography revealed complete occlusion from the distal right common femoral artery into the crural vessels and severe stenosis of the left tibial and peroneal arteries. Despite emergent thrombectomy, thrombolysis, full anticoagulation, high-dose corticosteroids, and intravenous immunoglobulin (IVIG), clinical deterioration ensued, culminating in fatal multiorgan thrombosis. This case underscores the aggressive and often refractory nature of CAPS, where even prompt triple-therapy intervention may prove insufficient. Clinicians must balance the urgent need for anticoagulation against haemorrhagic risk, particularly in advanced ischemia or infarction. Mortality in CAPS was historically around 50%, though recent series report reductions to ~30–35% with early multimodal therapy Heightened awareness and rapid institution of anticoagulation combined with immunosuppression and vascular intervention remain critical to alter the otherwise dismal prognosis.

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I. INTRODUCTION

A 72-year-old south asian woman with rest angina, arthritis, type 2 diabetes, hypertension, and hyperlipidaemia was brought to our emergency department for urgent thrombectomy after arriving from Egypt. She had initially complained of acute right lower limb pain with pallor and coldness at an outside facility. A history of multiple recurrent miscarriages raised clinical suspicion for an underlying prothrombotic condition.

Upon arrival, she was tachycardic (heart rate ~117 bpm), required high-flow nasal cannula oxygen to sustain 97% saturation, and exhibited somnolence with inability to follow commands. Both legs showed edema, and chest auscultation revealed bilateral rales consistent with pulmonary congestion. Neurologically, she displayed right-sided hemiparesis with 0/5 motor strength in the right upper and lower extremities; left-sided withdrawal to pain; sluggish but reactive pupils. Vascular examination showed absent distal pulses in the lower limbs on Doppler, with cold, mottled feet.

Laboratory evaluation revealed leukocytosis with neutrophilia, metabolic derangements including hyperglycemia, non-anion gap metabolic acidosis, elevated BUN, creatinine, and lactate. Cardiac enzymes (troponin, D-dimer) were elevated. ECG demonstrated anterior ST-depressions. ABG confirmed metabolic acidosis. Echocardiography showed mild systolic dysfunction (EF 45–

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50%) with global wall motion abnormalities. Arterial Dopplers identified thromboses in the left ulnar and radial arteries.

CT angiography of the abdominal aorta and bilateral iliofemoral arteries identified occlusion of the right lower extremity arterial supply—from the distal common femoral artery into leg vessels—and critical stenosis of the left tibial and peroneal arteries. Pulmonary CTA showed bilateral ground-glass opacities without emboli. Brain imaging revealed acute left caudate-putamen infarction with cerebral edema, evidence of early ischemia, extracranial occlusion of the left internal carotid artery, distal left PCA occlusion, and near-total obstruction of the right cavernous ICA.

Admitted to the MICU with left MCA syndrome, the patient underwent an M1 thrombectomy resulting in revascularization. Neurosurgical evaluation considered hemicraniectomy due to a left hemispheric infarct with mass effect, midline shift, and petechial hemorrhage—but deferred intervention due to poor neurological recovery potential.

On hospital day 2, she underwent right-lower-limb angiography and suction thrombectomy with catheter-based thrombolysis (EKOS), targeting the right common femoral, superficial femoral, and popliteal arteries. She received a low-dose heparin infusion. However, a follow-up angiogram revealed new occlusion of the left external iliac artery. Given the high risk of hemorrhagic conversion, further revascularization was withheld, and anticoagulation was stopped intraoperatively.

The patient received aspirin and clopidogrel along with continued low-dose heparin for NSTEMI. Supportive care included IV furosemide and BiPAP for pulmonary edema. Hematology was consulted given multiple arterials thromboses, and a presumptive diagnosis of catastrophic antiphospholipid syndrome (CAPS) was made. Laboratory testing confirmed elevated anti-β2-glycoprotein I IgG and anticardiolipin IgG antibodies despite normal IgA/IgM isotypes.

She was promptly treated with pulse methylprednisolone for three days and IVIG (400 mg/kg/day for four days). Heparin was discontinued on day 3 due to hemorrhagic transformation, and both anticoagulation and antiplatelet agents were withheld. Despite these intensive interventions, her clinical status deteriorated steadily, and she succumbed on day 6.

This rewritten case reflects current understanding of CAPS an extremely rare but often fatal hypercoagulable syndrome necessitating urgent multidisciplinary treatment including anticoagulation, immunomodulation, and vascular intervention.

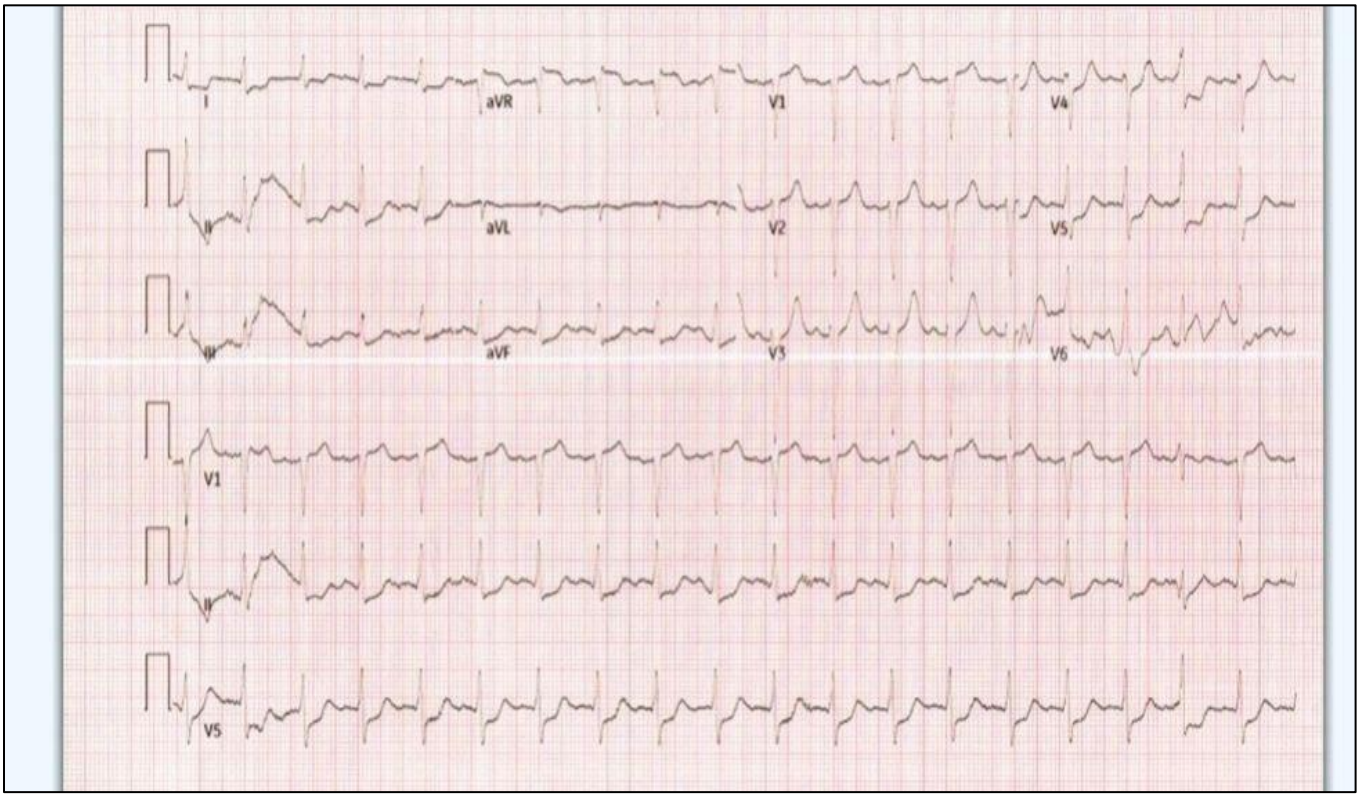

Fig 1. Sinus Tachycardia with Fusion Complexes, Marked ST Abnormality, Possible Inferior Subendothelial Injury

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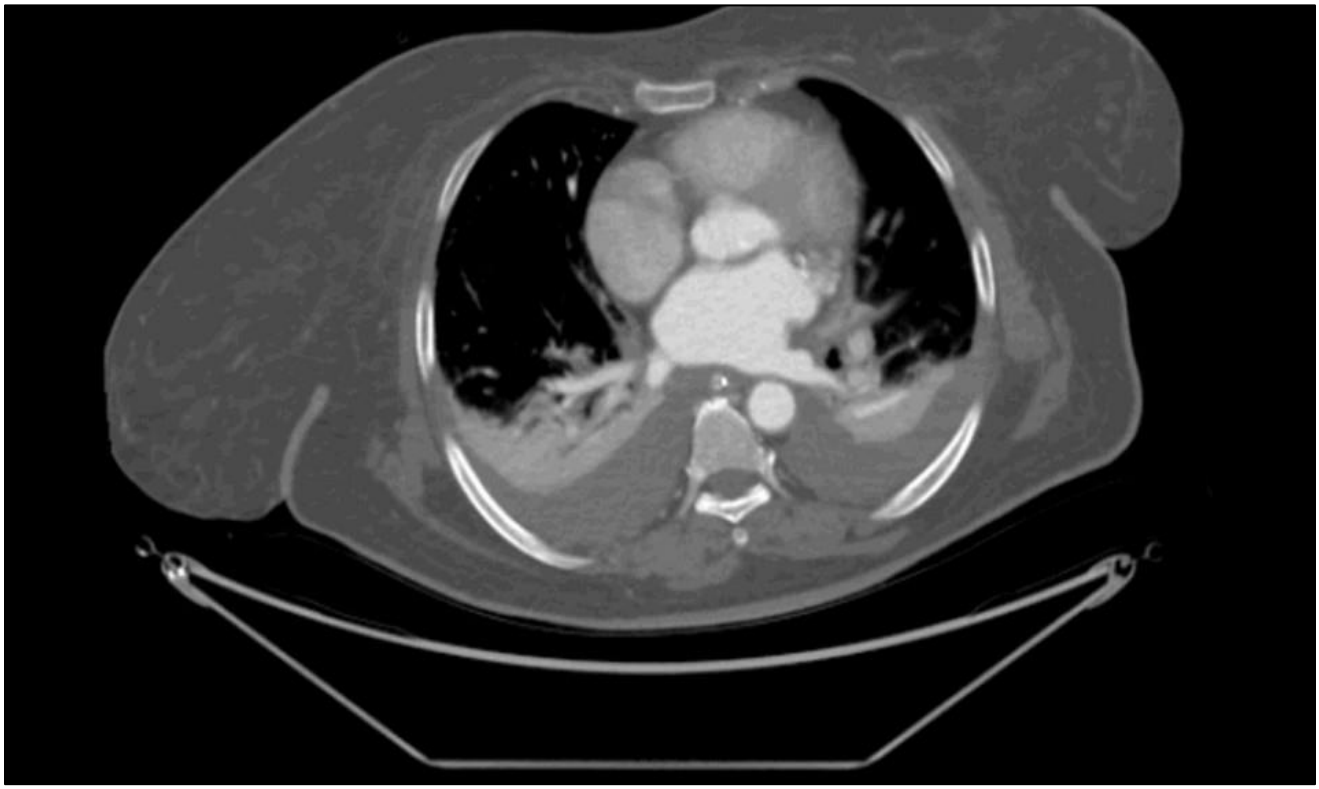

Fig 2. CT Abdomen Aorta and Bilateral Iliofemoral w wo Contrast Showed Occlusion of the Right Lower Extremity Arteries from the Distal Right Common

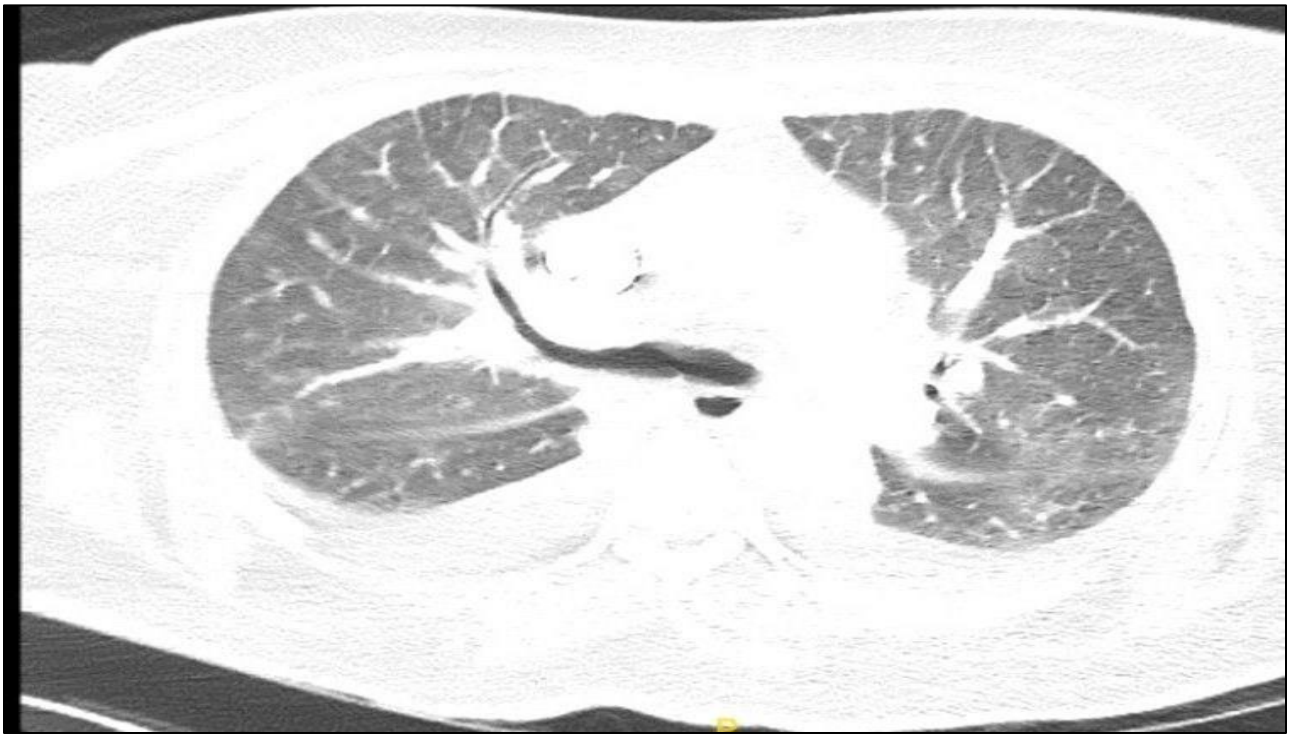

Fig 3 CT Pulmonary Angiography Revealed Bilateral Ground-Glass Opacities

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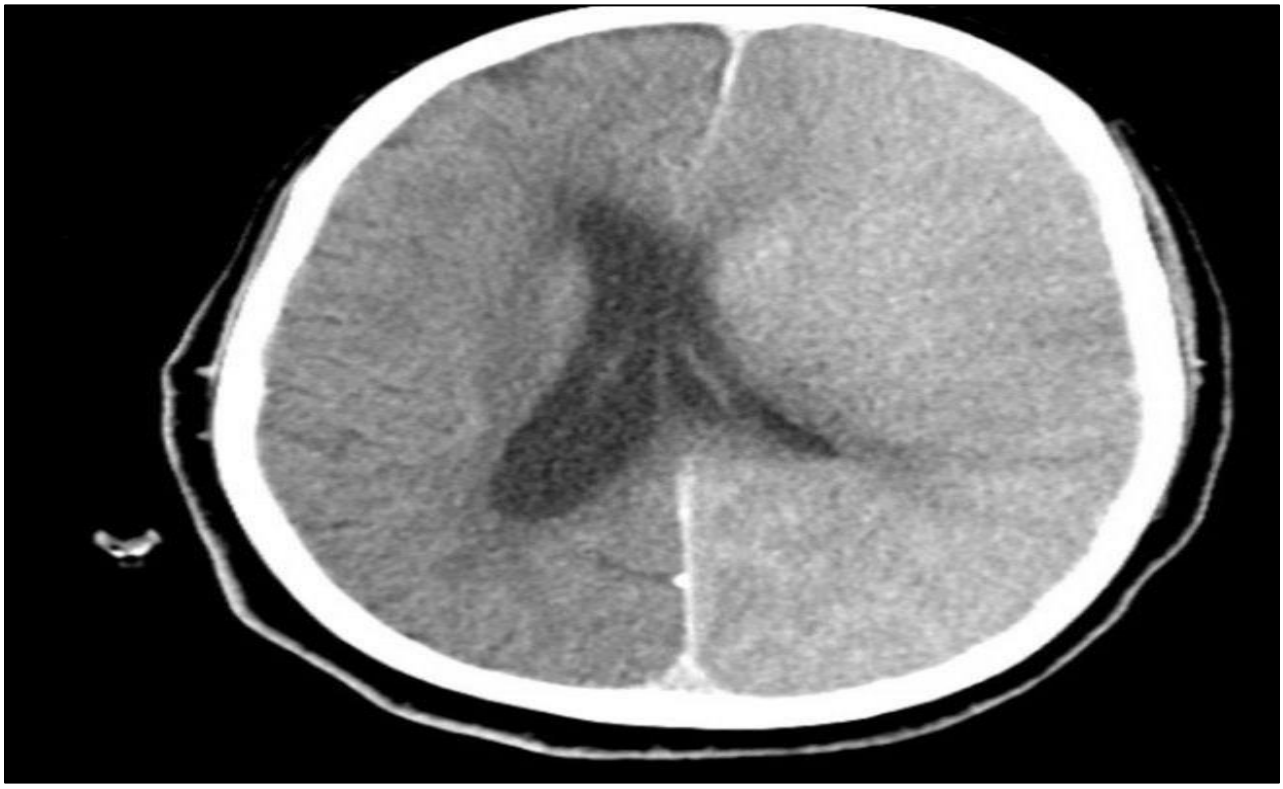

Fig 4 CT Head Showed Large Territory Left Cerebral Hemispheric Infarct and Stable Mass Effect with Midline Shift to the Right of Approximately 6 mm

II. DISCUSSION

Management of APS especially when cardiovascular complications arise requires careful stratification and tailored therapy, with unique considerations in older patients. Bernardi et al. emphasize that older adults often face a dual challenge higher thromboembolic risk and increased vulnerability to bleeding [1]. This comprehensive review underscores the need for individualized, multidisciplinary management in older adults with APS, balancing thrombotic prevention against bleeding risk, and pursuing advanced therapies when conventional measures fall short. Warfarin and other vitamin K antagonists remain the cornerstone of thrombotic APS therapy DOACs are discouraged in patients with arterial events or triple-positive antibody profiles due to heightened recurrence rates [2] Adjunctive treatments including hydroxychloroquine, statins, and vitamin D are recommended for refractory or complex cases, while targeted immunotherapies **(**e.g. rituximab, belimumab, anti-TNF, complement inhibitors, and β_2-glycoprotein I peptide agents) are emerging as promising options. Treatment strategies typically parallel standard APS management, with anticoagulation at the core [3] . In select cases, especially where autoimmune inflammation predominates, immunosuppressive therapies—including corticosteroids, IVIG, or plasma exchange—may be considered, though data remain limited. In summary [4], aPL-mediated thrombosis in APS arises from a cascade of anticoagulant dysregulation, cellular activation, and immune-mediated vascular inflammation, often precipitated by an additional physiological or environmental insult.

III. CONCLUSION

This case report highlights the extensive and life threatening cardiovascular complication. This patient had elevated troponin and the patient also required urgent thrombectomy for PAD which suggests systemic thrombosis affecting both large and small vessels leading to myocardial infarction or ischemia. The multiple thrombosis in peripheral and cerebral arteries demonstrates the severity of thrombotic burden in CAPS. The systemic nature of the condition often leads to widespread vascular occlusion involving various organ systems. The acute ischemic stroke coupled with NSTEMI suggests how CAPS can lead to both cerebral and myocardial infarctions simultaneously, a rare occurrence with thrombotic disease. This patient was managed with thrombectomy and thrombolysis, followed by pulse- dose steroids and intravenous immunoglobulin (IVIG) for CAPS, revealing the complex nature of management.

Anticoagulation management is particularly difficult due to the risk of hemorrhagic conversion, emphasizing the need for careful balance when treating patients with CAPS-related thrombosis.Recognizing the potential for cardiac involvement in CAPS may help in early identification and intervention, improving the overall prognosis in such critically ill patients.

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