

中文題目：紅斑性狼瘡併抗磷脂症候群導致心肌梗塞與缺血性腦中風：個案報告

英文題目：systemic lupus erythematosus and antiphospholipid syndrome with

myocardial infarction and ischemic stroke - A Case Report

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### ***Introduction:***

Antiphospholipid syndrome (APS) is an important clinical problem in patients with systemic lupus erythematosus (SLE). Arterial thrombosis in APS is fairly common and could involve coronary or cerebral arteries leading to myocardial infarction (MI) or stroke. However, APS involving multiple arterial branches of cardiovascular and central nervous system is rather rare. We reported a case of a 34-year-old female patient with SLE and APS who presented with non-ST-segment elevation myocardial infarction (NSTEMI) and ischemic stroke sequentially.

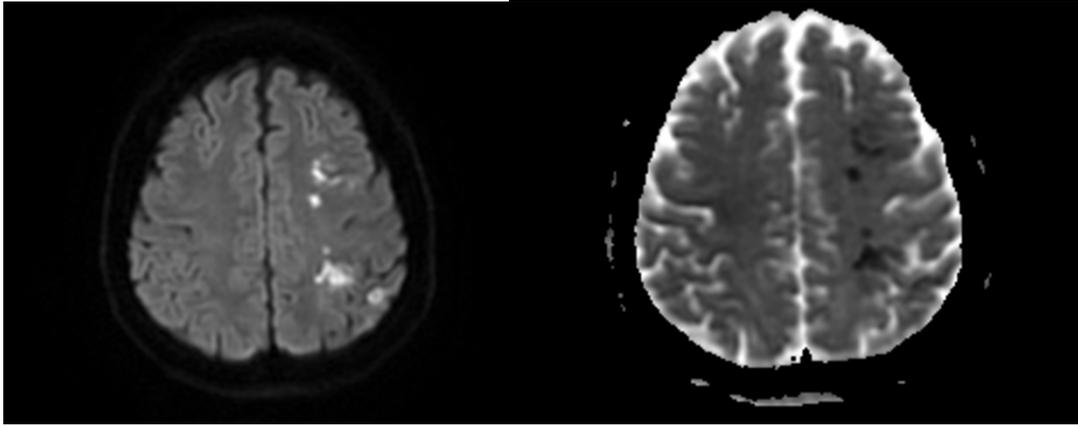
### ***Case Report:***

A 34-year-old female presented to our emergency room with acute cognitive impairment and Gerstmann syndrome (dysgraphia, dyscalculia, prosopagnosia and left-right disorientation). Two years ago, she underwent cardiac catheterization followed by a percutaneous coronary intervention (PCI) with an implantation of one stent in left anterior descending coronary artery (LAD). Afterwards, she was diagnosed with systemic lupus erythematosus (SLE) and took oral prednisolone 10mg per day. Unfortunately, she discontinued medication by herself 3 months before current admission. On admission, magnetic resonance imaging of brain revealed acute infarction in left anterior cerebral artery (ACA)/middle cerebral artery (MCA) and MCA/posterior cerebral artery (PCA) watershed. Her laboratory exams revealed elevated antinuclear antibody (ANA, 1:2560, speckled), nearly normal anti-ds-DNA level (10 IU/ml), low C4 level (8.56 mg/dL), high rheumatoid factor (57.7 IU/ml), slightly positive anti-ribosomal-P (23 EliAU/ml) and presence of antiphospholipid (aPL) antibodies (>150 Units) and lupus anticoagulant (1.94). Central nervous system (CNS) infection was less likely because of normal cerebrospinal fluid (CSF) cell count. She subsequently received pulse therapy with methylprednisolone [1,000 mg/day intravenously (IV) for three consecutive days] for SLE with antiphospholipid syndrome (APS) and CNS involvement. She was discharged from the hospital after cognitive function improved. However, four days later, she presented to our

emergency room again with chest pain. Cardiac enzyme was raised (troponin-I 0.092 ng/ml). Exercise electrocardiogram (EKG) disclosed positive finding. Her transthoracic echocardiography showed significant hypokinesia of the basoseptal wall of the left ventricle and an ejection fraction of 58.64%. An elective coronary angiography revealed in-stent restenosis (ISR, 70%) in the LAD stent. The stent was then dilated with drug-eluting balloon. The final angiographic result with well-deployed stents and thrombolysis in myocardial infarction (TIMI) III distal blood flow was achieved.

***Conclusion:***

Neuropsychiatric systemic lupus erythematosus (NPSLE) is a form of SLE arising from inflammation and/or thrombotic events in the nervous system. For symptoms reflecting inflammation or an underlying autoimmune process, high-dose glucocorticoids and intravenous cyclophosphamide remain the cornerstones. For symptoms reflecting an underlying thrombotic process such as APS, anticoagulation and antiplatelet agents are the mainstays of therapy, especially if aPL antibodies or APS are present. Patients with NPSLE are more likely to have positive aPL antibodies, and therefore are at higher risk of recurrent ischemic stroke. Similarly, SLE implies more thromboembolic hazards than conventional risk factors, even after standard therapy. Jinoos Yazdany et al found that patients with SLE had twofold to threefold higher risk of stroke and MI in a systematic review and meta-analysis. The study by Gurlek et al showed that a higher level of aCL antibodies predicted increased rate of restenosis after PCI. The systematic review and meta-analysis by Bundhun et al mentioned that SLE and APS are associated with significantly higher long-term ( $\geq 1$  year) adverse cardiovascular outcomes after PCI. However, multiple arterial territory involvement in SLE or APS was rather rare. Currently, only González-Pacheco et al reported a similar case of one healthy 28-year-old man who had no known coronary artery disease risk factors except APS and experienced acute left main coronary artery thrombosis and ischemic stroke. The patient was treated successfully with PCI, adequate anticoagulation, intravenous steroids and plasmapheresis. Alert diagnosis and prompt management for SLE combined with APS was necessary.



**Figure 1. Magnetic Resonance Imaging of Brain on August 2020.**

The image showed acute infarction in left ACA/MCA and MCA/PCA watershed with luxury reperfusion.