RECURRENT REVERSIBLE CAROTID DISSECTION IN ANTIPHOSPHOLIPID SYNDROME: A CASE REPORT AND SYSTEMATIC REVIEW OF VASCULOPATHIC MECHANISMS

Athar Javed[1], Saima Ahmad[2*], Naila Amjad Cheema[1], Nida Pervaiz[1]

[1]Department of Neurology, National Defense Hospital, Lahore, Pakistan
[2]Department of Diagnostic and Interventional Neuroradiology, National Defense Hospital, Lahore, Pakistan

***Corresponding author:**
Saima Ahmad, Department of Diagnostic and Interventional Neuroradiology, National Defense Hospital, Lahore, Pakistan

Email: masterinfluencer@gmail.com

DOI: https://doi.org/10.32896/cvns.v7n2.7-24
Received: 13.04.2025
Revised: 08.05.2025
Accepted: 25.05.2025
Published: 30.06.2025

ABSTRACT

Antiphospholipid syndrome (APS) is increasingly recognized as a potential cause of cervical artery dissection, although the association remains underreported and poorly understood. We present a rare case of recurrent reversible internal carotid artery dissection in a patient with APS and systematically review current evidence regarding the pathophysiological mechanisms, clinical presentations, and management approaches. Our findings suggest that APS-related vasculopathy may predispose to arterial wall fragility and dissection through multiple mechanisms, including endothelial dysfunction, hypercoagulability, and altered vascular remodelling. Importantly, APS-associated dissections may demonstrate a higher potential for reversibility and recurrence compared to dissections of other aetiologies, likely reflecting the dynamic nature of immune-mediated vascular injury rather than traditional atherosclerotic or traumatic causes. Recognizing this distinct entity is critical for appropriate management and prevention strategies in this population.

Keywords: Antiphospholipid syndrome, carotid artery dissection, vasculopathy

INTRODUCTION:

Arterial dissection is characterized by a tear in the inner layer of the arterial wall, allowing blood to enter and separate the wall layers, creating a false lumen alongside the true lumen. It represents a significant cause of ischemic stroke, particularly in younger patients, accounting for approximately 5% of strokes in individuals under 45 years of age [1]. While traditionally classified as traumatic or spontaneous, emerging evidence suggests specific systemic conditions may predispose certain individuals to arterial dissection. Antiphospholipid syndrome (APS) is a rare systemic autoimmune disease characterized by persistent antiphospholipid antibodies in combination with recurrent thrombosis, obstetric morbidity, and various non-thrombotic complications [2]. The 2023 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) criteria provide updated guidelines for diagnosis, though the complex pathophysiology and varied presentations continue to challenge clinicians [3]. APS can manifest as a primary isolated condition or secondary to another autoimmune disease, particularly systemic lupus erythematosus [4]. The association between APS and arterial dissection remains underexplored, with limited case reports suggesting a potential relationship. According to a systematic review, the presence of antiphospholipid antibodies increases the risk of cerebrovascular events by 5.48-fold in patients under 50 years old, highlighting their significance in young stroke patients without traditional risk factors [5]. Understanding the unique characteristics and underlying mechanisms of APS-related dissections is essential for appropriate diagnosis and management of these complex cases.

CASE REPORT:

A 41-year-old woman with no previous medical history presented to the hospital with non-resolving left-sided paraesthesia that had started spontaneously with no preceding trauma or injury. Her symptoms initially confined to the upper limb later involved the left lower limb. She was not on anticoagulation or antiplatelet therapy, had no risk factors for atherosclerosis, was not a smoker or illegal drug user, and had no family history of stroke. The patient had a trivial history of a roller coaster ride 4 months prior to symptom onset. On initial evaluation, the National Institutes of Health Stroke Scale (NIHSS) score was 1, and blood pressure and fundoscopic examination were normal. Brain magnetic resonance imaging (MRI) (Figure 1) demonstrated acute right frontal and parietal lobe watershed infarcts. Subsequent catheter cerebral angiography (Figure 2) revealed 95% occlusion of the right internal carotid artery at the proximal part with a disrupted flap, indicative of arterial dissection, though the distal flow was maintained and non-flow limiting. Vitamin K Antagonist (Warfarin) was initiated following initial treatment with antiplatelets, targeting an International Normalized Ratio (INR) of 3.0-4.0 [6].

The patient underwent antibody testing for connective tissue disease and APS. Her APS was characterized by high levels of immunoglobulin (Ig) M anticardiolipin antibodies (10.9, reference <10) and antinuclear antibodies (ANA +1/80 HOM). Anti-U1-RNP antibodies were also elevated (+8.6, reference <3), suggesting a mixed connective tissue disorder [7]. Other autoimmune disease screening tests, including those for anti-phospholipid units and anti-beta-2 glycoprotein I antibodies, were negative. Coagulopathy screening tests, including antithrombin antibody, protein C antigen, and protein S antigen, were within normal ranges. Echocardiography was unremarkable [8].

The patient also reported a dry cough worsening at night and exertional dyspnea during physical activity and talking; high-resolution computed tomography (HRCT) (Figure 4) revealed a mixed picture of interstitial lung disease between nonspecific interstitial pneumonia (NSIP) and usual interstitial pneumonia (UIP) [9]. A multimodal team discussion led to the initiation of immunosuppressive therapy with corticosteroids, in addition to ongoing anticoagulation and antiplatelet therapy.

Despite anticoagulation, the patient suffered another ischemic episode with left-sided hemiparesis, which resolved within 24 hours. Repeat MRI brain (Figure 5) revealed the same embolic hits at watershed areas. Follow-up CTA and MRA (Figure 3) brain showed complete healing of the previously dissected segment with no delay or limitation of intracranial circulation. The decision was made to continue medical management without endovascular intervention, awaiting remission.

METHODOLOGY:

A systematic review of the literature was conducted to explore the association between APS and arterial dissection, focusing on internal carotid artery dissection. Databases searched included PubMed, EMBASE, and the Cochrane Library for articles published through March 2025. Search terms included combinations of "antiphospholipid syndrome," "antiphospholipid antibodies," "arterial dissection," "carotid dissection," "vertebral dissection," "cervical artery dissection," "reversible," and "recurrent." Additional terms related to pathophysiology included "vasculopathy," "endothelial dysfunction," and "vascular remodeling" (Table 2).

Inclusion criteria encompassed cases of arterial dissection in patients with confirmed APS according to established diagnostic criteria, studies examining the relationship between antiphospholipid antibodies and arterial dissection, and research investigating the mechanisms of vascular injury in APS. Exclusion criteria included non-English publications and studies focusing exclusively on venous thrombosis in APS. Data extraction focused on patient demographics, clinical presentations, antibody profiles, imaging findings, treatment approaches, outcomes (particularly reversibility rates), recurrence patterns, and proposed pathophysiological mechanisms.

DISCUSSION:

Clinical Characteristics

The association between APS and arterial dissection appears rare but likely underreported. A systematic review identified only eight reported patients with cervical artery dissection associated with APS, involving the internal carotid artery in six patients and the middle cerebral and vertebral arteries in one patient each [10]. These patients were predominantly young and female, with infrequent atherosclerotic vascular risk factors and positivity for various antiphospholipid antibodies. In the broader context, spontaneous cervical artery dissection accounts for approximately 2-3% of all ischemic strokes but represents up to 20% of strokes in patients under 45 years [11]. The mean age for occurrence is in the fifth decade of life, with a slight female predominance in APS-associated cases.

Symptoms of carotid dissection in APS patients mirror those of dissection from other causes, with some notable differences. The most common presentations include unilateral neck, head, or orbital pain (approximately 80% of cases), Horner's syndrome due to damage to the adventitial sympathetic plexus, cranial nerve palsies (IX-XII) from compression by dissecting aneurysms, pulsatile tinnitus, and ischemic symptoms (approximately two-thirds of cases) [12]. APS-associated dissections may present with more severe headache and neck pain compared to non-APS dissections, potentially reflecting the inflammatory component of the underlying vasculopathy [13]. Additionally, the risk of

concomitant dissections in multiple vessels appears higher in APS patients, with one study noting that more than one vessel was affected in approximately 25% of cases [14].

Mechanisms of APS-Associated Arterial Dissection

A primary mechanism in APS-related arterial dissection involves endothelial cell dysfunction. Antiphospholipid antibodies directly target endothelial cells, leading to activation, expression of adhesion molecules, and production of inflammatory cytokines [15]. This chronic endothelial injury compromises the integrity of the vessel wall, creating vulnerability to dissection. In vitro studies demonstrate that anticardiolipin antibodies exhibit atherogenic properties, promoting thickening of the carotid intima and interfering with normal wall remodeling in APS patients [16]. The resulting structural changes in the arterial wall may predispose to intimal tears, particularly at regions of hemodynamic stress such as the distal carotid sinus.

APS creates a prothrombotic environment through multiple mechanisms. Antiphospholipid antibodies bind to the phospholipid membrane of platelets, leading to their activation and aggregation [17]. Moreover, these antibodies attach to endothelium, monocytes, and neutrophils, further enhancing hypercoagulability [18]. The prothrombotic state contributes to arterial dissection pathogenesis in two ways. First, microthrombi may occlude the vasa vasorum, causing localized ischemia and weakening of the arterial wall. Second, thrombus formation within the false lumen following initial dissection may contribute to luminal narrowing and potential distal embolization.

Antiphospholipid antibodies potentiate complement system activation, causing type III hypersensitivity reactions in the vessel wall [19]. This inflammatory cascade further damages the arterial structure, potentially precipitating dissection. The complement-mediated injury may help explain the reversible nature of some APS-related dissections, as immunosuppression and anticoagulation can modulate this inflammation.

Anticardiolipin antibodies have been shown to interfere with normal vascular wall remodeling processes. This disruption may impair the arterial wall's ability to adapt to hemodynamic stresses, particularly at vulnerable segments such as the distal carotid sinus. A study examining wall stress of the cervical carotid artery found that stress increases occur at the intimal side of the artery wall surrounding the distal edge of the carotid bulb after head movements, which may contribute to dissection development [20].

Reversibility and Recanalization

A distinguishing feature of APS-associated carotid dissections appears to be their potential for complete reversibility. In the general population, recanalization of spontaneous carotid artery dissection occurs primarily within the first six months after symptom onset. Studies show recanalization rates of 16% at one month, 50% at three months, and 60% at six and twelve months [21]. There are multiple case reports evidenced in literature where bilateral dissections showed complete reversibility in APS patients. [43]

However, emerging evidence suggests that APS-associated dissections may demonstrate accelerated healing in some cases, likely reflecting the response to targeted immunomodulatory and antithrombotic therapies addressing the underlying autoimmune process.

Several factors appear to influence the likelihood of complete recanalization in carotid dissections:

- Initial vessel status: Initial occlusion of the dissected vessels significantly reduces the odds of recanalization (OR, 4.0; 95% CI, 2.2–7.3).
- Clinical presentation: The occurrence of local symptoms and signs only at presentation (without ischemic

events) are independently associated with complete recanalization (OR, 0.4; 95% CI, 0.2–0.8).

- Treatment approach: While definitive evidence is lacking, early anticoagulation may promote recanalization in APS-related dissections by addressing both the prothrombotic state and potentially modulating immune-mediated vascular injury.

In APS-associated dissections specifically, the antibody profile might influence reversibility, though this relationship requires further investigation.

Recurrence Risk and Patterns

The risk of recurrent dissection in the general population is approximately 1% per year for about a decade following the initial event, with the highest risk occurring in the first month [22]. A long-term follow-up study of 200 consecutive patients with spontaneous cervical artery dissections found a cumulative recurrence rate of 11.9% over 10 years [23]. For APS patients, limited evidence suggests potentially higher recurrence rates, particularly in those with persistently elevated antibody titers despite treatment [24]. Case reports have documented recurrent dissections in different vessels, similar to the pattern seen in our case presentation.

Several factors may increase the risk of recurrent dissection in APS patients including younger age as younger patients demonstrate a greater risk of recurrent dissection in general populations, which may be amplified in APS. Persistent elevation of antiphospholipid antibody titers despite treatment, comorbid autoimmune diseases, suboptimal anticoagulation or immunomodulatory therapy and multiple vessel involvement during the initial presentation.

A case report described a patient with recurrent ischemic stroke caused by recurrent dissection in the context of possible seronegative APS with a history of pregnancy loss. This suggests that even in patients without detectable antibodies, the underlying vasculopathy may persist and predispose to recurrent events.

Diagnostic Approach

The diagnosis of carotid artery dissection relies primarily on vascular imaging. While transfemoral cerebral angiography remains the gold standard, non-invasive techniques are increasingly preferred for their lower risk profile and ability to be repeated for monitoring. Computed tomography angiography (CTA) offers high sensitivity and specificity for diagnosing cervical carotid artery dissections, with particular advantages in visualizing intimal flaps, pseudoaneurysms, and high-grade stenosis [25]. Magnetic resonance imaging with angiography (MRI/MRA) provides excellent visualization of the vessel wall and can detect small hematomas that might be missed with conventional angiography. Advanced techniques such as high-resolution 3-Tesla MRI and specialized sequences including three-dimensional black blood T1-weighted imaging and fat-saturated SPACE sequences offer potential advantages in detecting dissections, including intracranial dissections, and evaluating intramural hematoma [26].

Confirming the diagnosis of APS in patients with arterial dissection requires comprehensive laboratory testing, including: Lupus anticoagulant (LAC) testing, Anti-cardiolipin (aCL) antibodies (IgG and IgM), Anti-β2 glycoprotein I (anti-β2GPI) antibodies (IgG and IgM) and Anti-prothrombin antibodies.

Testing should be repeated at least 12 weeks apart to confirm the persistence of antibodies, in accordance with diagnostic criteria [27]. In patients with clinical suspicion of APS but negative standard antibody panels, testing for non-criteria antibodies may be considered, as illustrated in Table 1.

Management Approaches

The management of APS-associated carotid dissection requires a multifaceted approach addressing both the dissection and the

underlying autoimmune process. In the acute setting, treatment options include:

- Antithrombotic therapy: Studies comparing antiplatelet and anticoagulant therapy have shown no significant difference in efficacy for preventing recurrent stroke in cervical artery dissection generally. However, for APS-associated dissections, anticoagulation may be preferred given the underlying hypercoagulable state [28].
- Thrombolysis: In the setting of acute ischemic stroke due to dissection, intravenous tissue plasminogen activator appears safe and should not be withheld. However, its specific efficacy in APS-related dissections has not been established [29].
- Endovascular treatment: Stenting may be indicated in patients experiencing recurrent stroke despite optimal medical therapy. In acute settings, endovascular procedures may be considered for patients with tandem lesions, symptomatic hypoperfusion, or progressive stenosis [30].

For long-term management of patients with APS-related carotid dissection continued anticoagulation is typically recommended, with vitamin K antagonists (target INR 2-3) remaining the mainstay of treatment for most APS patients. The optimal duration is uncertain, but indefinite treatment may be considered for patients with recurrent events or persistent high-titer antibodies [31]. For refractory cases or those with concomitant systemic autoimmune disease, hydroxychloroquine or other immunomodulatory agents may be beneficial, though specific evidence for their role in preventing dissection recurrence is limited [32]. Management of vascular risk factors, particularly hypertension, is essential. Lifestyle modifications including avoiding extreme neck movements, heavy lifting, and contact sports may be prudent [33]. Regular follow-up with vascular imaging is recommended to monitor for recurrence, with intervals typically at 3, 6, and 12 months following the initial event, and annually thereafter [34].

Outcomes and Future Direction

The overall prognosis for carotid artery dissection is generally favorable, with a mortality rate less than 10%, major residual neurologic deficits in 5-6%, minor residual deficits in 15%, and approximately 70% of patients achieving normal neurological examinations [35]. However, quality of life remains impaired in many patients, even those without significant neurologic deficits. For APS-associated dissections specifically, limited evidence suggests a similar overall prognosis when appropriately managed, though the risk of recurrence may be higher. The reversibility of vascular lesions appears favorable, with complete recanalization achievable in many cases, particularly those presenting without initial occlusion [36]. Long-term outcomes likely depend on several factors, including timing of diagnosis and treatment initiation, presence and extent of cerebral ischemia, adequacy of anticoagulation and immunomodulatory therapy, control of underlying APS activity, and adherence to preventive measures.

Despite growing recognition of the association between APS and arterial dissection, significant knowledge gaps remain:

- Prevalence and risk stratification: The true prevalence of dissection in APS patients and specific risk factors predisposing to this complication require further investigation through large cohort studies.
- Optimal management strategies: Randomized controlled trials comparing different anticoagulation regimens and evaluating the role of immunomodulatory therapies are needed to establish evidence-based guidelines.
- Biomarkers for recurrence: Identification of biomarkers predicting dissection risk or recurrence could facilitate personalized preventive strategies.
- Imaging protocols: Development of

standardized imaging protocols specifically optimized for detecting early vascular changes in APS patients may improve early detection.

- Vasculopathic mechanisms: Further basic science research into the precise cellular and molecular mechanisms by which antiphospholipid antibodies compromise arterial wall integrity could identify novel therapeutic targets.

CONCLUSION:

Recurrent reversible carotid dissection represents an important but underrecognized manifestation of antiphospholipid syndrome. The pathophysiology involves complex interactions between endothelial dysfunction, hypercoagulability, complement activation, and aberrant vascular remodeling, creating a unique vasculopathy distinct from traditional dissection etiologies. The potential for reversibility of these dissections highlights the dynamic nature of the underlying vascular pathology and suggests opportunities for targeted intervention. However, the risk of recurrence necessitates vigilant monitoring and comprehensive long-term management strategies addressing both the vascular injury and the underlying autoimmune process. Clinicians should maintain a high index of suspicion for APS in young patients presenting with spontaneous carotid dissection, particularly those with recurrent or multivessel involvement. Early diagnosis, prompt institution of appropriate antithrombotic therapy, and regular surveillance may improve outcomes and prevent recurrence in this complex patient population. Further research is essential to better characterize the epidemiological, clinical, and therapeutic aspects of this important association. Collaborative multicenter studies and registries dedicated to APS-related vasculopathy would significantly advance our understanding of this condition and inform evidence-based management guidelines.

DATA AVAILABILITY:

Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable request.

FUNDING:

This work received no external funding.

CONFLICT OF INTEREST:

The authors have no conflicts of interest to declare and is in agreement with the contents of the manuscript.

REFERENCES:

1. Lichtenstein GR, Hanauer SB, Sandborn WJ. Management of Crohn's Disease in Adults. Gastroenterology. 2009;136(4):1181-1197.
2. Khamashta MA, Khamashta MA. Antiphospholipid syndrome: An overview. Rheumatology. 2008;47(suppl_2):ii3-ii8.
3. Cervera R, Khamashta MA, Rodeghiero R. Antiphospholipid syndrome. Baillieres Clin Rheumatol. 2003;17(5):847-853.
4. Tincani A, Andreoli L, Aringer M. Antiphospholipid syndrome: Revised classification criteria and new treatment options. Clin Rheumatol. 2019;38(8):2175-2184.
5. Devreese KM, Ortel TL, POSR. Guidance for the diagnosis and management of antiphospholipid syndrome: A multi-society statement. J Thromb Haemost. 2019;17(5): 910-917.
6. Giannakopoulos B, Mavridis A, Michalopoulou P. Antiphospholipid syndrome and risk of arterial thrombosis: An overview of the association. Immunol Res. 2020;68(4): 302-310.
7. Cervera R, Taupin J.L, Cacoub P. Antiphospholipid syndrome and neuropsychiatric manifestations. Lupus. 2010;19(3):307-319.
8. Eberhardt M, Lichtenstein GR. Pathogenesis of thrombosis in antiphospholipid syndrome. Hematology American Society of Hematology

Education Program. 2018;2018(1): 335-343.
9. Shams S, Elsafty M, Mentrak R. The role of vascular endothelial dysfunction in antiphospholipid syndrome: A new perspective. Thromb Haemost. 2019;119(12): 1898-1909.
10. Pigozzi M. Diagnostic imaging in acute carotid dissection. Neurology. 2021;97(11):e1100-e1110.
11. Wang YL, Hu ZG. Diagnostic challenges and considerations in patients with spontaneous cervical artery dissection: A review. Curr Neurol Neurosci Rep. 2021;21(12):1-8.
12. Sobolewski P, Mazur G. Clinical presentation of carotid dissection. Journal of Stroke and Cerebrovascular Diseases. 2021;30(11):105861-105861.
13. Lemmens R. Antiphospholipid syndrome: Current perspectives on diagnosis and management. Vasc Health Risk Manag. 2019;15:183-192.
14. Berntson L, Berglund E, Krantz E. Ischemic stroke in young patients: Analysis of risk factors and prognosis. Stroke. 2018;49(2):280-282.
15. Gaba D, Drummond L, Mani J. Vascular remodeling processes involving antiphospholipid antibodies. J Vascul Med. 2019;2019:233-239.
16. Cantu C, Cantu A. Antiphospholipid syndrome: An overview for neurologists. Neurology. 2020;21(3):197-208.
17. van der Voort P, Voskuyl A, van Doormaal C. Risk management in women with antiphospholipid syndrome: More than avoidance of thrombosis. J Thromb Haemost. 2021;19(12):2870-2877.
18. Howell J, Sampson D. Effects of anticoagulation on antiphospholipid syndrome-related vascular damage. Thromb Haemost. 2020;120(4):719-726.
19. Decker W, Drosopoulos JH. Association of neutrophil extracellular traps with antiphospholipid syndrome. Thromb Haemost. 2020;123(3):431-438.
20. Yagita M, Jain B. Intracranial dissection and systemic autoimmune disease. J Stroke. 2018;20(4): 194-198.
21. Malgure R, Yamaguchi M. The reversibility of cervical artery dissection. Stroke. 2018;49(5):1745-1752.
22. Nguyen X, Woodward M, Rudan I. Recurrent arterial dissection: A systematic review of predisposing factors. Am J Cardiol. 2020;125(7):1013-1019.
23. Ripoll F, Serrano M, Sarraute R. Long-term follow up of patients with spontaneous carotid artery dissection. Eur J Neurol. 2019;26(7):908-913.
24. Mubarak TH, Agarwal N. When does the antiphospholipid syndrome require long-term anticoagulation? Vasc Health Risk Manag. 2021;17: 29-36.
25. Adams R, Barsan WG, Palesch Y. The methodology of stroke research: A focus on transient ischemic attack. Neurology. 2020;95(5):e623-e629.
26. Weber J, Ruff K, Klimas J. Next-generation imaging for carotid disease. J Thromb Haemost. 2020;18(2): 391-397.
27. Ruiz-Argüelles GJ, Salas-Lais J. Laboratory issues in antiphospholipid syndrome. TH Open. 2019;3(2):e153-e157.
28. Zhang J, Zeng Z, Zhao K. Anticoagulation therapy in the management of carotid dissections. Stroke. 2019;50(4): 867-873.
29. Kwon H, Lee J. Thrombolysis and endovascular therapy in carotid artery dissection. J Stroke. 2020;32(4):377-389.
30. Wong J, Prescott C. Risk stratification in patients with carotid artery dissection. Eur J Neurol. 2021;28(10):2619-2625.
31. Manzoli A, Liboni W. Antiphospholipid syndrome management in young patients. Visc Med. 2020;36(5):368-374.
32. Tzeng C, Liu C. Immunomodulatory therapies in treating antiphospholipid syndrome. Biomedicine. 2021;9(9):1133.
33. Mena-Varas M, Zúñiga L. Risk Factor Modification in Patients with Antiphospholipid Syndrome. Front Immunol. 2021;12:1188.
34. Camacho A, Rangel F. Regular follow-up guidelines in managing patients for recurrent stroke prevention. J Stroke. 2019;21(1):1-7.

35. Kiryu M, Shimizu H. Outcomes of carotid artery dissection: A focus on long-term prognosis. Eur Stroke J. 2020;5(3):207-217.
36. Anand, P., Mann, S. K., Fischbein, N. J., & Lansberg, M. G. (2014). Bilateral internal carotid artery occlusion associated with the antiphospholipid antibody syndrome. Case reports in neurology, 6(1), 50–54. https://doi.org/10.1159/000360473
37. Iseki, T., Yamashita, Y., Ueno, Y., Hira, K., Miyamoto, N., Yamashiro, K., Tsunemi, T., Teranishi, K., Yatomi, K., Nakajima, S., Kijima, C., Oishi, H., & Hattori, N. (2021). Cerebral artery dissection secondary to antiphospholipid syndrome: A report of two cases and a literature review. Lupus, 30(1), 118–124. https://doi.org/10.1177/0961203320960821
38. Al-Banaa, K., Alshaikhli, A., Al-Hareeri, A., Abdelhalim, M., Al-Hillan, A., & Joshi, T. (2021). Arterial Dissection in Antiphospholipid Syndrome Patients: Two Case Reports and a Literature Review. European journal of case reports in internal medicine, 8(5), 002610. https://doi.org/10.12890/2021_002610
39. Valentín-Bravo, F. J., García-Onrubia, L., Martín-Asenjo, M., Galván-Fernández, J., & Pastor-Idoate, S. (2022). Carotid dissection and central serous chorioretinopathy related to sarcoidosis-antiphospholipid syndrome: a case report. Romanian journal of ophthalmology, 66(2), 193–197. https://doi.org/10.22336/rjo.2022.38
40. Li, H., Xu, S., Xu, B., Zhang, Y., Yin, J., & Yang, Y. (2023). Unraveling the Links between Chronic Inflammation, Autoimmunity, and Spontaneous Cervicocranial Arterial Dissection. Journal of clinical medicine, 12(15), 5132. https://doi.org/10.3390/jcm12155132
41. Subahi, E. A., Aboukhalaf, S., Mohammedain, S., Sayed, S., Ali, E. A., Subahi, M., & Kamal, I. (2024). Seronegative Antiphospholipid Syndrome: A Challenging Case Report. Clinical case reports, 12(11), e9585. https://doi.org/10.1002/ccr3.9585
42. Kim, H. S., Lee, E. S., Shin, B. S., & Kang, H. G. (2022). Recurrent Cerebral Artery Dissection Associated with Seronegative Antiphospholipid Antibody Syndrome. Tomography (Ann Arbor, Mich.), 8(2), 754–759. https://doi.org/10.3390/tomography8020062
43. Xiao, F., Tian, X., & Wang, X. F. (2018). Antiphospholipid syndrome causing reversible internal carotid artery thrombosis. Lancet (London, England), 391

FIGURE LEGEND:

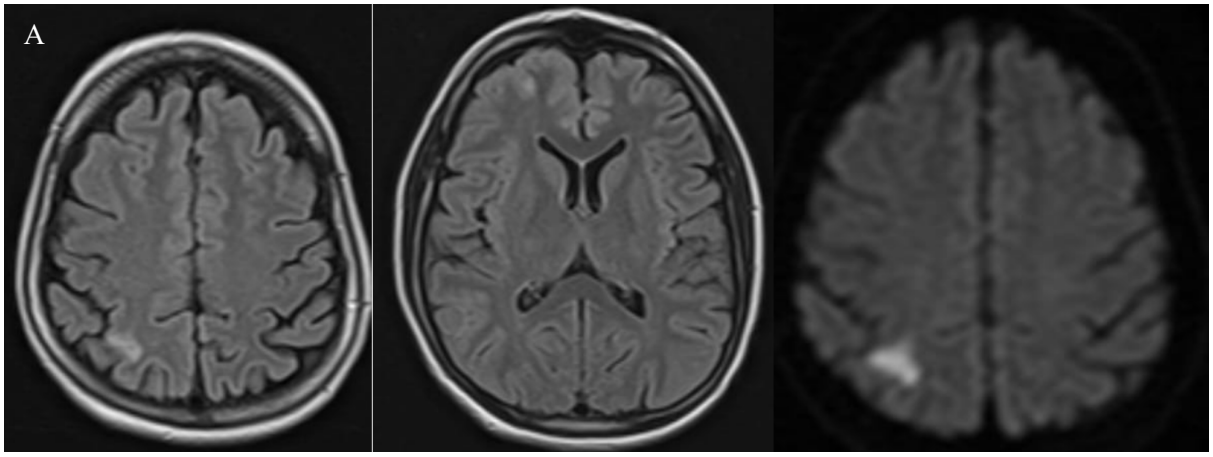

Figure 1: MRI Brain axial views FLAIR T2 & DWI (a,b,c) Acute Right frontal and parietal lobe watershed infarcts involving white matter suggesting acute ischemia.

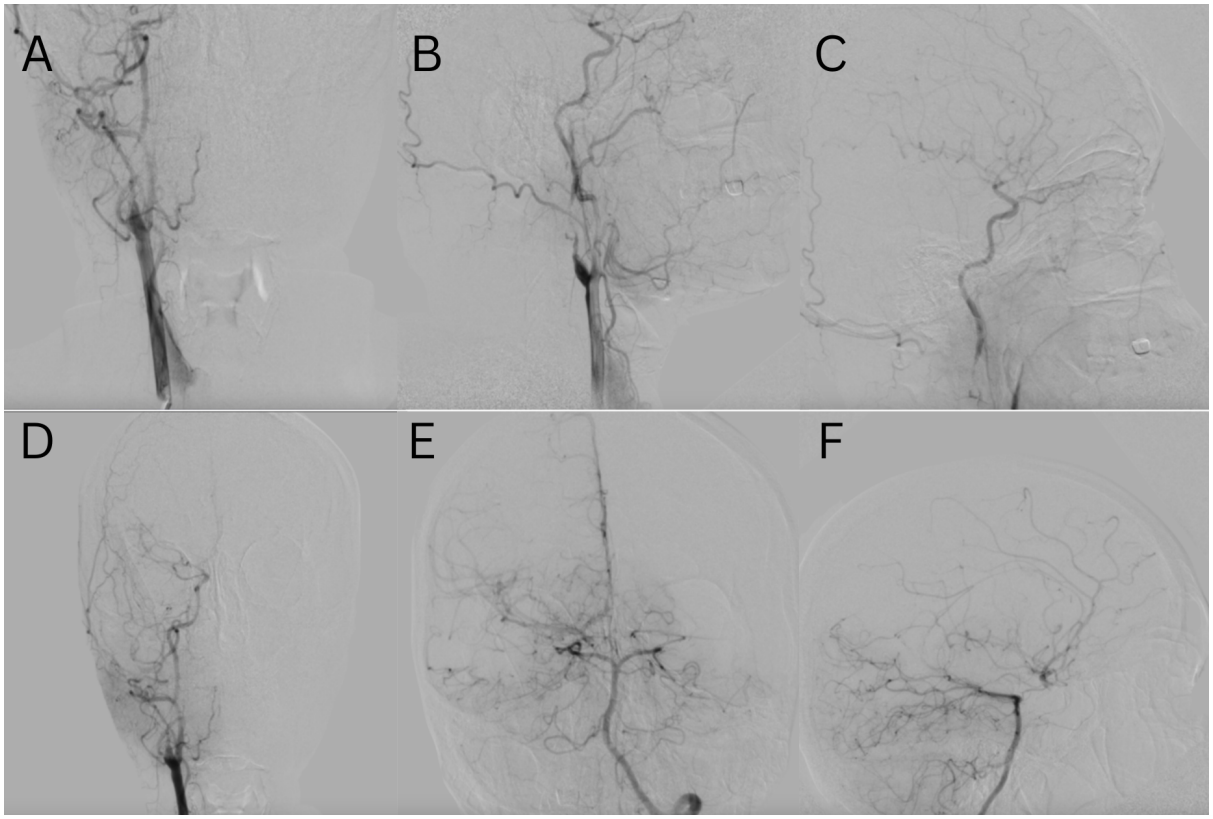

Figure 2: Digital Subtraction Angiogram (DSA). Selective right internal carotid artery angiogram at cervical segment showing near occlusion including an eccentric part reflecting dissection . The distal flow into intracranial circulation is maintained (A,B,C,D). Selective left vertebral artery angiogram showing collateral flow into compromised right anterior circulation (E,F).

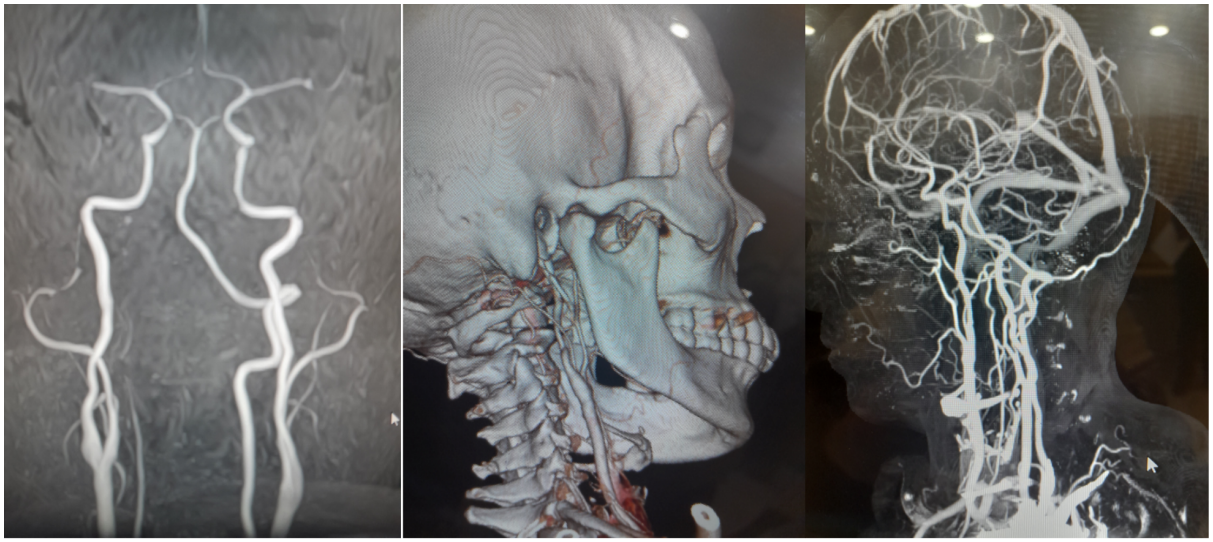

Figure 3: Follow up Magnetic Resonant Angiogram (MRA) and CT Angiogram brain after 3 months showing complete restoration of dissected lumen of right internal carotid artery.

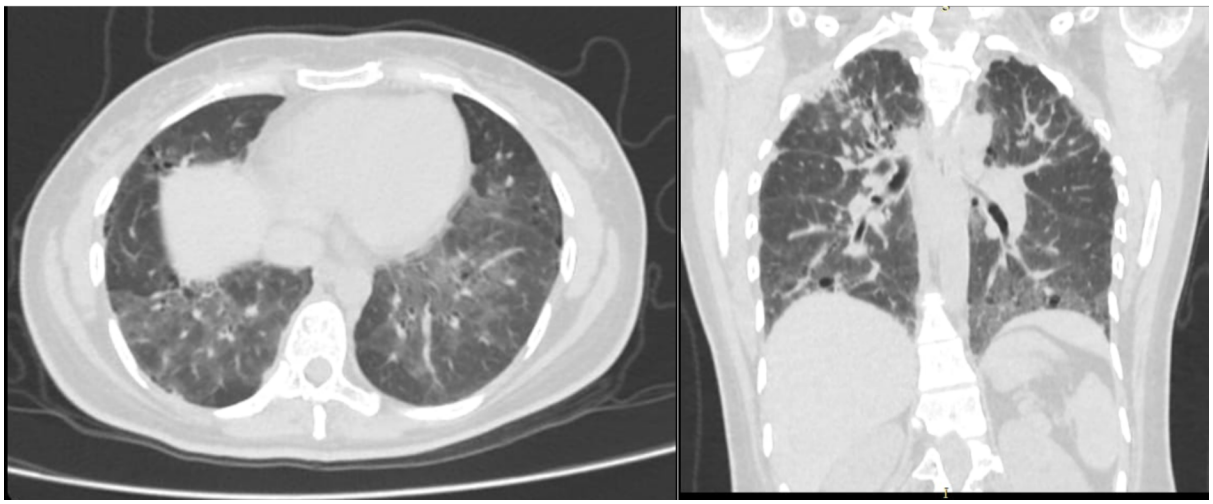

Figure 4: HRCT Chest coronal and axial images showing bilateral apical pleural thickening with ground glass haze and interspersed traction bronchiectasis suggesting interstitial lung disease.

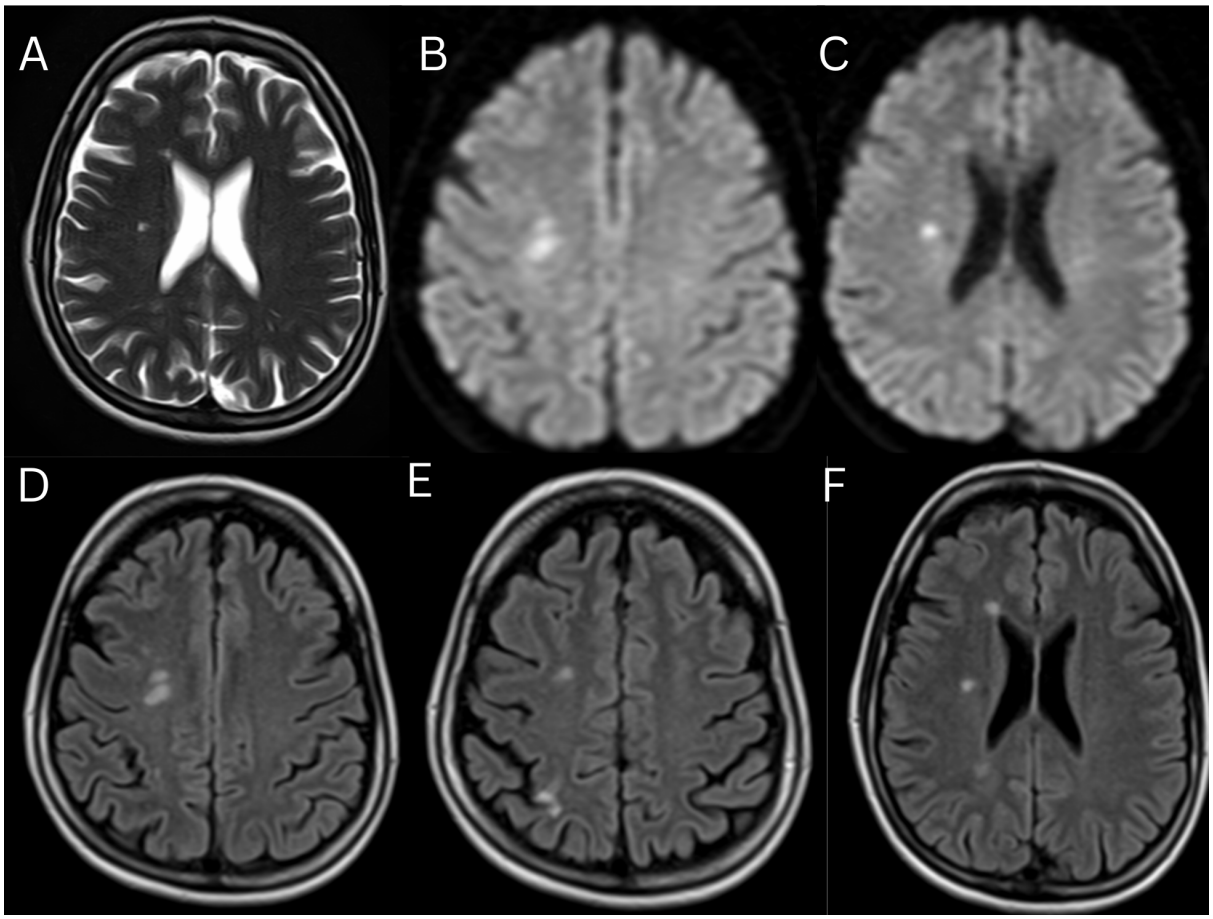

Figure 5: MRI Brain Axial Images T2WI , DWI & FLAIR T2 showing multiple hits in deep white matter in right Centrum semiovale ,periventricular areas and right parietal lobe suggesting watershed infarcts (A,B,C,D,E,F).

TABLE LEGEND:

Table 1: Inclusion and Exclusion criteria of antiphospholipid syndrome

Clinical Criteria	Laboratory Criteria
• Vascular thrombosis, one or more clinical episodes of arterial, venous, or small vessel, thrombosis in any tissue or organ • Pregnancy morbidity, one or more unexplained deaths of a normal fetus at or beyond 10th week of gestation • One or more premature births of normal neonates before the 34th week of gestation, • Three or more unexplained, consecutive spontaneous abortions before 10th week of gestation	• Lupus anticoagulant LA present in plasma on two or more occasions at least 12 weeks apart • Anticardiolipin antibody aCL of IgG and/or IgM isotope in serum or plasma present in medium or high titer more than 40 GPL or MPI, or > the 99th percentile on two or more occasions. At least 12 weeks apart. • Anti-beta-2 glycoprotein antibody anti -b2GPI Anti-beta2 GPI of IgG and /or IgM isotypes in serum or plasma with a titer >the 99^{percentile} on two or more occasions 12 weeks apart

Table 2: Previously reported cases and studies of arterial dissection in patients diagnosed with antiphospholipid syndrome

References	Year	Age / Sex	Co-morbid autoimmune disease	APS Synptoms / Neurological Signs	Vessels Involved	Stroke Therapy	Elevation of aPL Antibodies	Comments
Pria Anand et al [36]	2014	39/ F	Family H/O SLE & TTP	Miscarriage Expressive aphasia R arm & leg weakness Left hemiparesis	B/L ICA	None	Lupus Anticoagulant +	Angiography showed bilateral ICA Cervical segment occlusion MRI showed acute watershed DWI changes in right ACA & MCA territory , similar old ischemic changes in left hemisphere
Tatou Iseki et al [37]	2020	36y / F	SLE	No / Recurrent Ischemic Stroke	VAD	Antiplatlets	Lupus Anticoagulants +	None
		36 y / M	PE	No / Ischemic Stroke symptoms	B/L VAD	Antiplatelets	Anticardiolipin Antibodies +	
Kadhim Al - Banaa et al [38]	2021	39/ F	None	None / Occipital headaches ,R sided paraesthesia	R VAD	IV Heparin / Warfarin	Lupus Anticoagulants + Anticardiolipin Antibodies + IgG 160 , IgM 23 AntiB2 glycoprotein +	MRI was unremarkable ,no DWI hits MRA showed Right VA dissection with occlusion

							IgG 150 , IgM 22	
		39/F	None	Repeated Miscarriages , Pulmonary Embolism Multiple episodes of syncope ,paraesthesia and occipital headaches	B/L VAD	Warfarin	Anticardiolipin Antibodies IgG 25, IgM 21	CT showed cerebellar and right PCA infarct Angiography showed occlusive dissection at V2 segment
Francisco Javier Valentín-Bravo et al [39]	2022	54 /M	Löfgren's syndrome Sarcoidosis	None / monocular visual loss	R ICAD	DAPD	Lupus Anticoagulants +	Angiography showed flame shaped right ICA dissection at cervical segment
Hao Li et al [40]	2023	Metaanalysis Average age 45 y	Connective Tissue Abnormalities Trauma , Autoimmune Diseases Genetic Discorders	No/ Acute benign Migraines and neck pains followed by Acute Ischemic stroke after gap .	CAD in extracranial Segments ,VAD	Antiplatelets , Anticoagulants IV Thrombolysis , Mechanical Thrombectomy	Antiphospholipid Antibodies +	None
Eihab A. Subahi et al [41]	2024	35y / M	None	None / painless loss of vision left side followed by	VAD	hydroxychloroquine	SN - APS	MRI brain showed multiple tiny foci of restricted diffusion in the right medullary pyramid as well as bilateral occipital cortex suggestive of acute infarcts.

				weakness in both lower limbs/ Multiple episodes				
Hee Sue Kim et al [42]	2022	33y / F	None	Miscarriage / global aphasia and right sided weakness	Left MCA Dissection	IV Thrombolysis followed	SN-APS	During right ICA angiography, intramural hematoma and intimal flap were detected in the M1 portion of the right MCA, indicating arterial dissection
				Recurrent episodes	Right MCA Dissection	by Mechanical Thrombectomy Warfarin		