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CVA in Patient with Systemic Sclerosis on Aspirin Therapy: A Case Report

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Background

Systemic sclerosis is an autoimmune disease characterized by immune system dysfunction, vasculopathy and fibroblast dysfunction leading to excess collagen deposition in the skin and internal organs.[1] Patients with this condition are at higher risk of acute cerebral vascular events, but it is difficult to develop strategies for prevention due to our limited understanding of the pathophysiology of disease.[2]

Case

A 76-year-old female with a history of systemic sclerosis presented to the emergency department with acute onset slurred speech, facial droop, and left arm pain with a National Institutes of Health (NIH) Stroke Scale of 1. She was outside the thrombolytic window. Other history includes hypertension and hyperlipidemia; both were well controlled. MRI confirmed right frontal lobe ischemic stroke with additional ischemic foci in the temporal and parietal lobe. CTA neck and echocardiogram were negative for emboli source, and LDL was within normal limits. She was started on dual antiplatelet therapy (DAPT). The patient's condition improved and by day 2 the only residual symptom was facial droop. She was discharged on DAPT with plans to transition to PLAVIX monotherapy after 90 days.

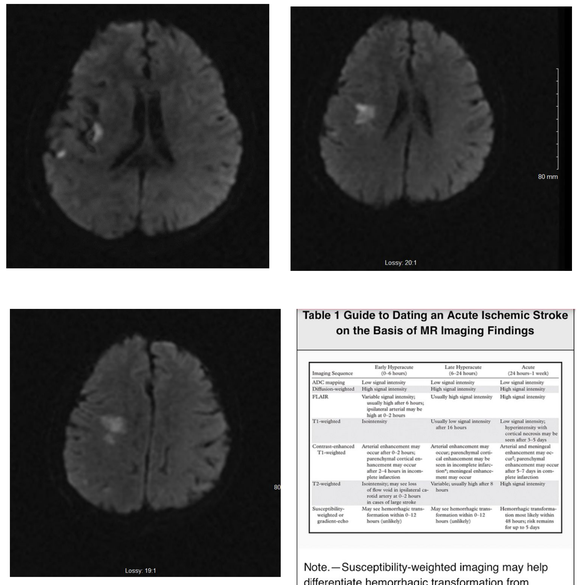

Figure 1. T1 weighted MRI. Hypointense signals from right frontal lobe extending towards base of ganglia (A), with additional ischemic foci in the temporal (B) and parietal lobes (C).

Table 1 Guide to Dating an Acute Ischemic Stroke on the Basis of MR Imaging Findings

Imaging Sequence	Early Hyperacute (0–6 hours)	Late Hyperacute (6–24 hours)	Acute (24 hours–1 week)
ADC mapping	Low signal intensity	Low signal intensity	Low signal intensity
Diffusion-weighted	High signal intensity	High signal intensity	High signal intensity
FLAIR	Variable signal intensity; usually after 6 hours; ipsilateral cortical may be high at 0–2 hours	Usually high signal intensity	High signal intensity
T1-weighted	Isointensity	Usually low signal intensity after 16 hours	Low signal intensity; hyperintensity with cortical necrosis may be seen after 3–5 days
Contrast-enhanced T1-weighted	Arterial enhancement may occur after 0–2 hours; parenchymal hemorrhagic transformation may occur after 2–4 hours in incomplete infarction	Arterial enhancement may occur; parenchymal corticomeningeal enhancement seen in incomplete infarction*; meningeal enhancement may occur	Arterial and meningeal enhancement may occur; parenchymal enhancement may occur after 5–7 days in complete infarction
T2-weighted	Isointensity; may see loss of flow void in ipsilateral cortical artery at 0–2 hours in cases of large stroke	Variable; usually high after 8 hours	High signal intensity
Susceptibility-weighted or gradient-echo	May see hemorrhagic transformation within 0–12 hours (unlikely)	May see hemorrhagic transformation within 0–12 hours (unlikely)	Hemorrhagic transformation may occur within 48 hours; risk remains for up to 5 days

Note.—Susceptibility-weighted imaging may help differentiate hemorrhagic transformation from cortical necrosis, and diffusion-weighted imaging findings may be falsely negative in patients with hyperacute or acute posterior circulation or lacunar stroke. In the hyperacute stages, a large area of enhancement in the deep gray matter or basal ganglia may indicate impending hemorrhagic

Table 1 shows guidelines that can be used to date and classify acute ischemic strokes[3]

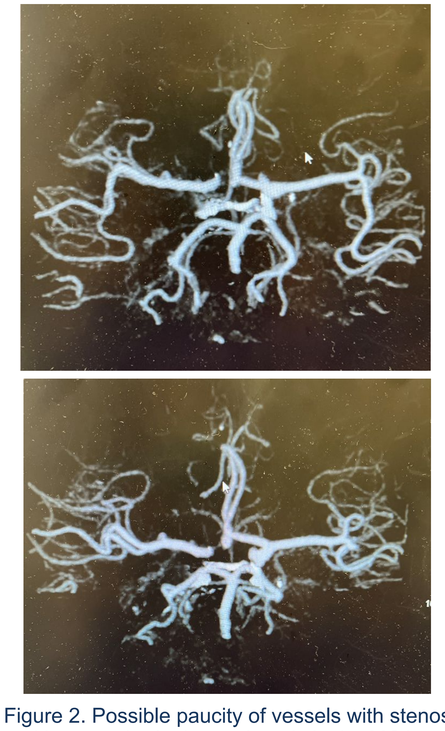

Figure 2. Possible paucity of vessels with stenosis and intracerebral atherosclerosis in the MCA territories.

Discussion

This case illustrates that the risk of cerebrovascular accident in a patient with a history of systemic sclerosis may be increased. Other contributing risk factors in this patient include a history of hypertension and hyperlipidemia. However, the patient was not on antihypertensives because blood pressure was controlled outpatient without them. Permissive hypertensive was allowed but no longer present following dual antiplatelet therapy (DAPT). Her hyperlipidemia was controlled with statins and the lipid panel showed LDL in normal range. For these reasons we suspect systemic sclerosis to be the most likely cause of CVA in this patient. Patients with this disease have a higher neurovascular and cardiovascular burden which puts them at risk for CVA.[4] Although, instructions have been made to manage the cutaneous manifestations and other findings associated with this disorder, very little research has investigated the neurovascular findings.[5] There are no clear guidelines for prevention of CVA in this population, however, long-term aspirin therapy is a consensus practice. Unfortunately, aspirin therapy failed for this patient, and she will eventually be transitioned to PLAVIX monotherapy after DAPT. The POINT and CHANCE trials indicated that DAPT provided the most benefit for the first 21 days after acute CVA.[6,7] However, due to the suspected paucity of stenosis, we felt the patient would benefit from 90 days of DAPT, followed by monotherapy.

Conclusion

Clinicians must be aware of the increased neurovascular burden associated with systemic sclerosis and recommendations should be made to help guide clinical decision making. Further research should be done regarding pathophysiology and prevention of CVA in patients with systemic sclerosis.

References

References:

1. Pattanaik D, Brown M, Postlethwaite BC, Postlethwaite AE. Pathogenesis of Systemic Sclerosis. Front Immunol. 2015;6:272. Published 2015 Jun 8. doi:10.3389/fimmu.2015.00272
2. Ungprasert P, Sanguankeo A, Upala S. Risk of ischemic stroke in patients with systemic sclerosis: A systematic review and meta-analysis. Mod Rheumatol. 2016;26(1):128-131. doi:10.3109/14397595.2015.1056931
3. Allen LM, Hasso AN, Handwerker J, Farid H. Sequence-specific MR imaging findings that are useful in dating ischemic stroke. Radiographics. 2012;32(5):1285-1299. doi:10.1148/rg.325115760
4. Cen X, Feng S, Wei S, Yan L, Sun L. Systemic sclerosis and risk of cardiovascular disease: A PRISMA-compliant systemic review and meta-analysis of cohort studies. Medicine (Baltimore). 2020;99(47):e23009. doi:10.1097/MD.0000000000023009
5. Pope JE, Denton CP, Johnson SR, Fernandez-Codina A, Hudson M, Nevskaya T. State-of-the-art evidence in the treatment of systemic sclerosis. Nat Rev Rheumatol. 2023;19(4):212-226. doi:10.1038/s41584-023-00909-5
6. Pan Y, Elm JJ, Li H, et al. Outcomes Associated With Clopidogrel-Aspirin Use in Minor Stroke or Transient Ischemic Attack: A Pooled Analysis of Clopidogrel in High-Risk Patients With Acute Non-Disabling Cerebrovascular Events (CHANCE) and Platelet-Oriented Inhibition in New TIA and Minor Ischemic Stroke (POINT) Trials [published correction appears in JAMA Neurol. 2019 Sep 30;:] [published correction appears in JAMA Neurol. 2021 Aug 16;:null]. JAMA Neurol. 2019;76(12):1466-1473. doi:10.1001/jamaneurol.2019.2531
7. Johnston SC, Elm JJ, Easton JD, et al. Time Course for Benefit and Risk of Clopidogrel and Aspirin After Acute Transient Ischemic Attack and Minor Ischemic Stroke. Circulation. 2019;140(8):658-664. doi:10.1161/CIRCULATIONAHA.119.040713

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