If Nothing Goes Right, It Goes to the Left: A Case of Left Anterior Cerebral Artery Stroke Due to Right Internal Carotid Artery Stenosis

Dear Editor,

Symptomatic carotid disease with more than 50% stenosis usually causes neurological symptoms in the form of minor stroke or transient ischaemic attacks in the ipsilateral anterior circulation territory and the most common location of plaque is in the carotid bifurcation and proximal internal carotid artery (ICA).[1] The mechanism of stroke is an artery to artery embolism, thrombosis or hemodynamic fluctuations. Azygos anterior cerebral artery (AACA) is a variant of Willisian circle anatomy characterised by absence of the anterior communicating artery (ACom) and the bilateral anterior cerebral arteries (ACAs) join to form a single trunk which travels superiorly in the interhemispheric fissure and has implications in the arterial haemodynamics. We report a case of left ACA territory infarction caused by right ICA stenosis in a patient with azygos ACA.

Case Report

A 55-year-old gentleman with past history of hypertension and dyslipidaemia for the last 5 years, poorly compliant to treatment, with acute onset right-sided weakness of predominantly lower limb along with speech disturbance, presented outside the window period. On examination, the patient had apathy, global aphasia, right upper motor neuron facial weakness and right-sided weakness predominantly in the lower limb. CT brain at admission showed evolved infarct in the left ACA territory involving the left parasagittal frontal lobe [Figure 1]. CT angiogram showed right ICA hypodense plaque causing about 80% stenosis at origin and azygous ACA with occlusion of the left division of azygos ACA [Figure 1]. Cardiac evaluation including 2D-echo and 24 hours Holter monitoring were normal. MR plaque imaging of the right ICA plaque showed lipid-rich core without any ulceration or intraplaque haemorrhage. The patient was managed with dual antiplatelets, statins and antihypertensives. In view of the symptomatic right carotid stenosis, carotid revascularisation was considered. Over the next 1 month, the patient improved to NIHSS of 9 and modified Rankin scale of 3. He is planned for right carotid endarterectomy.

Discussion

Anterior circulation comprises of ACA and middle cerebral

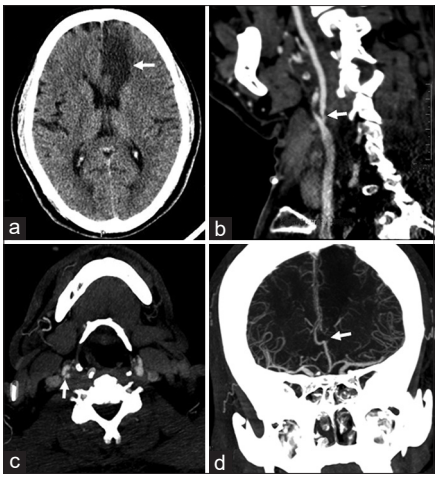

Figure 1: CT brain with angiogram of the patient. (a) Axial CT brain on admission showing left mesial frontal hypodensity (arrow) suggesting an evolved ACA territory infarct. (b) CT angiogram of extracranial vessels in sagittal section showing right proximal (white arrow) ICA plaque causing 80% stenosis. (c) CT angiogram axial section at the level of carotid bifurcation showing hypodense plaque (white arrow) in the right proximal ICA. (d) CT angiogram coronal section of intracranial vessels showing azygos ACA and left division occlusion (arrow)

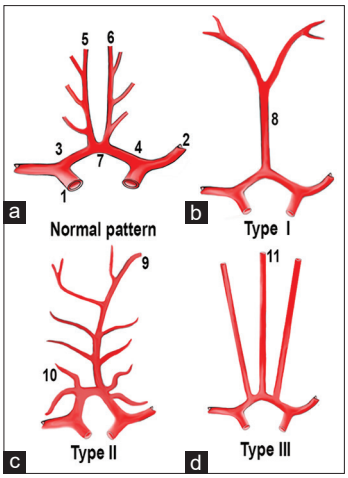

Figure 2: Schematic depiction of normal ACA and anomalous variants. 1 – terminal ICA, 2 – left MCA M1, 3 – right A1 ACA, 4 – left A1 ACA, 5 – right A2 ACA, 6 – left A2 ACA, 7 – Acom artery, 8 – true azygos ACA, 9 – left A2 azygos (bihemispheric) ACA, 10 – rudimentary right A2 ACA and 11 – accessory third median azygos ACA. (a) Normal ACA pattern. (b) Type I variant showing true azygos ACA from which ACA branches supply both hemispheres. (c) Type II variant showing bihemispheric ACA where most of the major branches to the bilateral hemispheres arise from one A2 ACA and contralateral rudimentary A2 ACA. (d) Type III variant showing an accessory ACA arising from Acom artery

artery (MCA) formed from ICA. The ACAs are connected by the ACom which is a primary collateral along with the posterior communicating artery in cases of occlusions or stenosis in the vascular network.[2] Arteria termatica or AACA was first described by Wilder with a reported incidence of less than 2%.[3] AACA is characterised by A1 segments of bilateral ACAs joining to form a single stem (A2 segment) which ascends in the interhemispheric fissure as a result of the persistence of the embryonic median artery of the corpus callosum. The anatomical variations in the ACA were described by Baptista.[4] Type I anomaly is the true azygos ACA, from which ACA branches supply both hemispheres. Type II anomaly is the bihemispheric ACA, where both right and left ACA are present, but most of the major branches to the bilateral hemispheres arise from one ACA and the other one is rudimentary. In type III, an accessory ACA arises from Acom [Figure 2]. Our patient had a Type I anomaly.

The left ACA territory infarct could be explained by right ICA stenosis due to the coexistent true azygos ACA with probable embolisation from the right ICA plaque as the mechanism causing the infarct and thus the aetiology of stroke is large artery atherosclerosis. In a patient with high-grade carotid stenosis, with infarct in the opposite hemisphere, carefully analysing the vascular anatomy may give a clue to the aetiology which can help in the management of the patient.

Conclusion

AACA is a rare variant, which can produce unilateral or bifrontal infarctions.[5] The anatomical variations in the ACA should be carefully looked into in cases of ACA territory strokes that affect the arterial haemodynamics and a proper etiological evaluation aids in the optimal management of the patient.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Acknowledgements

We thank Mr. Krishnakumar N for his assistance in obtaining high quality images.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Harikrishnan Ramachandran, Harini Pavuluri, Sapna Erat Sreedharan, P.N. Sylaja

Department of Neurology, Comprehensive Stroke Care Program, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram, Kerala, India

Address for correspondence: Dr. P.N. Sylaja, Professor of Neurology, Comprehensive Stroke Care Program, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum - 695 011, Kerala, India.
E-mail: sylajapn@hotmail.com

References

1. Abbott AL, Paraskevas KI, Kakkos SK, Golledge J, Eckstein H-H, Diaz-Sandoval LJ, *et al*. Systematic review of guidelines for the management of asymptomatic and symptomatic carotid stenosis. Stroke 2015;46:3288-301.
2. Moreira de Sousa CS, Mendes de Miranda CLV, Avelino MC, Bastos BB, Moreira de Sousa RS, Mendes de Miranda CVV. Thrombosis of the azygos anterior cerebral artery. Case Rep Radiol 2017;2017:5409430.
3. Baptista AG. Studies on the arteries of the brain. Neurology 1963;13:825-35.
4. Beyhan M, Gökçe E, Karakuş K. Radiological classification of azygos anterior cerebral artery and evaluation of the accompanying vascular anomalies. Surg Radiol Anat 2020;42:1345-54.
5. Khaliq A, Gul P, Gul P. Azygos anterior cerebral artery causing Bifrontal infarcts: A case report. PJNS 2018;13:20-2.

Submitted: 26-Apr-2021 **Accepted:** 06-Aug-2021 **Published:** 13-Jan-2022

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

DOI: 10.4103/aian.aian_307_21