

Research Article

CARDIOEMBOLIC STROKE PRESENTING AS PARAPARESIS DUE TO AZYGOUS ACA INFARCT

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ABSTRACT

Cardioembolic stroke accounts for 14-30% of ischemic strokes patients. Cardioembolic infarctions are prone to early and long-term stroke recurrences. Anterior Cerebral Artery (ACA) territory infarcts constitute only 0.6% to 3% of all acute ischaemic strokes. Bilateral infarcts due to unpaired ACA occlusion are still rare. Cardioembolic Stroke leading to bilateral ACA infarct in a patient with azygous ACA has rarely been reported to literature. We report a patient with old left MCA infarct presenting with paraparesis. MRI brain was suggestive of bilateral ACA territory infarcts. MRI angiography brain revealed Azygous ACA. 2d Echocardiography revealed Ischemic Dilated cardiomyopathy with Left Ventricular (LV) apical Clot

Keywords: Cardioembolic Stroke, Azygous ACA, Bilateral ACA Infarcts, Left Ventricular Apical Clot

CASES

A 62 yr old right handed male presented with weakness of both lower limbs 2 days before admission to the hospital. He had past history of Left Middle Cerebral Artery (MCA) infarction with residual weakness in Right upper limb and lower limb with motor aphasia 2 years back. The weakness of right lower limb increased from the previous state such that patient could not walk or stand. On examination pulse was 84/min, BP was 110/70 mm hg.

On nervous system examination Motor Aphasia was present along with spasticity with hyperreflexia with contracture in Right upper and lower limb. Power in right Upper limb and lower limb was 3/5 & 2/5 respectively. Left upper & lower limb power was 5/5 & 3/5. Plantars were Bilateral Extensor. On respiratory system examination Bilateral basal crepitations were present. Cardiovascular system revealed no murmurs.



Figure 1: 2d Echo showing LV Apical clot

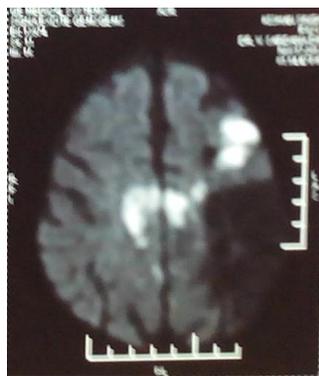


Figure 2: Diffusion weighted MRI BRAIN showing bilateral ACA territory infarcts



Figure 3: Magnetic Resonance Angiography Showing Anomalous ACA arising from Anterior communicating artery

Investigations revealed Hemoglobin of 12.2 gm/dl. Total leucocyte count was 12,310 cells/dl. Fasting Blood sugar was 96 mg/dl. Serum cholesterol was 144 mg/dl. HDL was 34 mg/dl. LDL 93 mg/dl.

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Triglycerides 84 mg/dl VLDL 16. Chest Xray was s/o cardiomegaly. Lung shadows were hazy in bilateral lower zones. ECG was s/o complete Right bundle branch block.

2d Echo done was suggestive of Regional wall motion abnormality (RWMA) at Apex, Distal two thirds of anterior wall and Interventricular septum Left ventricular ejection fraction 15-20% with severe LV diastolic dysfunction an LV apical clot (1.5cm) present (figure 1). MRI spine screening revealed degenerative changes with mild disc buldges at c4, c5-6 c6-7 without significant cord compression. MRI brain revealed Bilateral ACA territory acute infarcts with old Left MCA territory infarct (figure 2). MRI Brain Angiography revealed Azygous ACA – Bilateral A2 segments arising from anterior communicating artery supplied by left ACA, attenuated left MCA with pruning of peripheral branches (figure 3). No aneurysm or vascular malformation was seen.

Patient was started on Inj Enoxaparin (0.4 cc) subcutaneous BD and later shifted to oral Tablet Nicumolon 2mg OD. Tab Aspirin 150 mg Od, tab furosemide 20mg BD tab spironolactone 50 mg OD, tab isosorbid mononitrate 10 mg BD & Tab Ramipril 2.5 mg od. Tab metoprolol 12.5 mg BD was added after Respiratory symptoms and signs resolved.

Patient showed improvement in power from 2/5 to 3/5 in right lower limb and 3/5 to 4+/5 in left lower limb during 2 weeks of follow up. Patient was discharged on oral anticoagulants and antiplatelets and isosorbid mononitrate for IHD.

DISCUSSION

Cardioembolic strokes account for 14-30% of ischemic strokes. Owing to their large size, cardiac emboli flow to the intracranial vessels in most cases and cause massive, superficial, single large striatocapsular or multiple infarcts in the middle cerebral artery. Therefore, cardioembolic infarctions predominate in the carotid and middle cerebral artery distribution territories (Hart, 1992; Caplan, 1995).

Infarctions of the ACA accounted for 1.3% of all cases of stroke. Stroke subtypes included cardioembolic infarction in 45.1% of patients, atherothrombotic infarction in 29.4%, lacunar infarct in 11.8%. It is important to recognize the anomalies of the cerebral circulation. Review of literature reveals that accessory ACA is the most common anomaly with an incidence varying from 0.5 to 20%, followed by bihemispheric ACA (12 to 13.3%), unpaired or azygous ACA (0.3 to 3.2%) (Critchley, 1930). The true incidence of bilateral ACA infarction is unknown, with few cases reported in the literature.

A unilateral occlusion of the stem of the ACA proximal to its connection with the anterior communicating artery is usually well tolerated since adequate collateral flow comes from the ACA of the opposite side. Maximal disturbance occurs when both arteries arises from one ACA stem, in which case there will be infarction of the medial parts of both the cerebral hemispheres. This produces Paraparesis, These patients lie in bed unwilling to initiate any voluntary movement including speaking, appear to watch other people by following them with their eyes.

In our case MRI Angiography of Brain confirmed the presence of Azygous ACA with bilateral ACA territory infarction. However, we presume the stroke to be cardioembolic considering the presence of LV Clot on 2D echocardiography and recurrence of stroke in different territories. Transcranial Doppler which can be used to detect bubble signals in the desired artery as a confirmation for cardioembolic stroke was not available.

Conclusion

The case report highlights the presence anomalous azygous ACA which is a rare entity and cardioembolic stroke involving it leading to bilateral ACA territory infarction which is still rarer and more devastating. The case also highlights usefulness of MRI angiography in such cases.

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