ORIGINAL RESEARCH PAPER

General Medicine

BILATERAL ANTERIOR CEREBRAL ARTERY INFARCT – A RARE CASE.

KEY WORDS:

Dr Tadar Nyedar Jr Medicine, Department of Medicine and, All India Institute of Medical Sciences, Rishikesh, India

Dr Bharathi Mohan Jr Medicine, Department of Medicine and, All India Institute of Medical Sciences, Rishikesh, India

Dr Ujjawal Kumar Shriwastav Jr Medicine, Department of Medicine and, All India Institute of Medical Sciences, Rishikesh, India

Dr. Dhiri Ranjan Pradhan Jr Radiology, Department of Medicine and, All India Institute of Medical Sciences, Rishikesh, India

INTRODUCTION

Stroke is prevalent across patient populations and is the fifth commonest cause of death if considered separately from other cardiovascular diseases, thus accounting for significant morbidity and mortality. Infarcts involving the territory of the anterior cerebral artery (ACA) are uncommon due to significant collateral blood supply from anterior communicating artery. Infarction of the ACA territory accounts for only 0.3% to 4.4% of cerebral infarctions reported. Bilateral ACA infarction is even rarer. (1,2) We would like to present a case of 60 years old male who has presented to us with bilateral ACA infarct.

Case Report

60 years old right-handed male, from Bijnor (Uttar Pradesh) who is a farmer by occupation; a chronic smoker (40 pack-years) with occasional alcohol intake history; without any diagnosed comorbidities presented to us with acute onset weakness of bilateral lower limbs on 20th November after returning from his work in fields. The weakness was sudden on onset started from the left lower limb with the inability to stand up and progressed in a period of three to four hours to involve both the lower limb completely after which the patient could not move his lower limbs. The patient was bedridden with normal bowel and bladder activities and intact higher mental function. Two days before admission, the patient was found to exhibit altered behaviour in the form of inability to obey command and loss of speech. Both the events were not associated with a history of fever, headache, vertigo, dizziness, loss of consciousness, abnormal body movements, chest pain, palpitation, shortness of breath or any history of trauma.

The patient presented to the emergency department of AIIMS, Rishikesh on 5th December 2021 with a GCS of E4V1M6 and altered behaviour. On general physical examination, the patient was found to have a thin/emaciated look and some signs of dehydration. His vitals were; RR- 18/min, BP 160/85 mmHg, Temperature – 98.2 F, Pulse 95 beats/min, SpaO2 96% on Room air and random blood sugar was 119 mg/dl. His respiratory, cardiovascular and abdominal examinations were within normal limits. Positive findings on CNS examination; Loss of speech, reduced tone in all the four limbs, power of 1/5 in bilateral upper limbs and 0/5 in bilateral lower limbs with both plantar reflexes showing extensor response, hyperreflexia in bilateral knee jerk with a normal response on others. The sensory examination could not be assessed. Routine blood investigations and chest X-ray were within normal limits. 2D echo was done by the Cardiology team which did not reveal any abnormalities. CE-MRI brain and spine with MR angiography revealed the presence of bilateral anterior cerebral artery territory infarct with old ischemic changes in the left cerebral hemisphere. Bilateral carotid artery doppler showed the presence of soft plaque in the right common carotid artery. The patient was stabilized in the medicine high dependency unit (HDU) and the patient was started on antiplatelet therapy, anti-hypertensive and regular chest, limb physiotherapy. The patient clinically improved with final GCS of E4V5M6, bilateral upper limb power 3/5 and lower limb power 2/5, thus the patient was discharged to continue advised medications and physiotherapy.

Table 1: Investigations

	05/12/2021	08/12/2021
Hb (g/dl)	15.6	14.3
TLC (x1000/cumm)	8.82	7.69
DLC (N/L/M)	77/13/7	90/6/3.9
Platelet count (Lacs/cumm)	1.8	1.52
Blood urea	60.5	
Serum creatinine	1.02	
Na	134.6	
K	4.45	
Ca	9.41	
Uric acid	5.30	
Phosphorus	4.58	
Total bilirubin	0.67	
Direct bilirubin	0.13	
SGOT/SGPT	51/67	
ALP	134	
GGT	35	
Total protein	6.4	
Albumin	3.4	
Globulin	3	
PT/INR	9.4/0.86	
Viral markers (HBsAg, Anti-HBC, Anti-HIV antibodies)	Non-reactive	

Other investigations:

HbA1c: 5.3 %

Thyroid profile: FT3: 1.01 ng/ml, FT4: 7.64 ug/dl, TSH: 1.79 uIU/ml

Lipid profile: Total cholesterol: 241, Triglyceride: 106, HDL: 67.8, LDL: 240.

Vitamin B12: 175 pg/ml

CSF examination: 0.5ml colorless, total count – 5 cells/cumm (100% monomorphs), sugar – 109 mg/dl, protein – 67.4, ADA – 0.40 U/L, Gene X pert – No MTB detected

Screening 2D ECHO: No LVRMWA/LVEF – 60%/All chambers normal/Valves normal/No vegetations, clots or pericardial effusion

CE MRI Brain with Cervical Spine and Whole spine screening:

- Attenuated bilateral ACA with acute infarct in bilateral ACA territories

- Old infarcts in deep and superficial watershed territories of left cerebral hemisphere

MR Angiography of brain and neck:
- Right A1 ACA shows diffuse narrowing with reduced flow related signal in right A2 and A3 ACA.
- Left ACA is showing absent flow related signal in A1 onwards.
- Left vertebral artery is hypoplastic.

Bilateral Carotid artery doppler:
- Few athero-calcific changes are visualized in arteries
- A soft plaque of size (11.3mm) is seen for a length of 9mm in right common carotid artery.

DISCUSSION

Stroke is prevalent across patient populations and is the fifth commonest cause of death if considered separately from other cardiovascular diseases, thus accounting for significant morbidity and mortality. It can be categorized as ischemic, haemorrhagic, or subarachnoid. Ischemic strokes present with clinical syndrome pertaining to effect of reduced blood supply to particular areas of brain. Infarcts involving the territory of the anterior cerebral artery (ACA) are uncommon due to significant collateral blood supply from anterior communicating artery. Infarction of the ACA territory accounts for only 0.3% to 4.4% of cerebral infarctions reported. Bilateral ACA infarction is even rarer. (1,2)

The ACA emerges from the anterior clinoid segment of the internal carotid artery which then continues anteromedially towards the longitudinal fissure, where the anterior communicating artery form anastomosis between two ACA. The ACA itself often divides into five segments, usually labelled as A1 through A5, or as proximal (A1), ascending (A2, A3), and horizontal segments. A significant feature of the ACA is its robust anastomotic complex which leads to low rate of infarctions in its territory. Notably, infarctions simultaneously affecting both cerebral hemispheres may also be present among ACA stroke cases. These are rare and characteristically occur because of clinically significant anatomical variations affecting both ACA's at any point along its course. The most recognizable patterns are the azygos, bihemispheric, and ACA with hypoplastic or absent A1 segment.

Hypertension, hypercholesterolemia, diabetes mellitus, and smoking are known risk factors that underlie varied processes which ultimately result in atherosclerosis of large and small arteries. Atherosclerosis is the primary cause of ACA infarct. Cardiac embolism from different sources, including atrial fibrillation, intracardiac thrombus, valve disease, and tumors are other significant causes of ACA infarction. Some reports suggest that cardiac emboli are more frequently the cause of ACA as compared to MCA and posterior cerebral artery (PCA) infarcts. (3,4) Another significant mechanism of ACA stroke is arterial dissection which is rare and found in Japanese young population. (3,5) Among the other less common mechanisms, vasculitis, coagulopathic states and vasospasm are some of them. Vasospasms has been found to be triggered by subarachnoid haemorrhage and pituitary apoplexy and this mechanism has been correlated with both unilateral and bilateral ACA infarcts (6). However, in some aetiology remains unknown.

There are varied presentations in people who are found to have acute stroke of ACA territory as it depends on whether the ACA itself or its branches are involved. Size also remains one of the determining factor to influence on the clinical manifestation. Nevertheless, the most common presentation (in 86-90%) is found to be motor deficit involving the lower extremities contralateral to the infarct site. (7) Heubner's artery and medial striate artery infarcts are associated with contralateral face and arm weakness, resulting from damage to the anteromedial caudate nucleus, anterior limb of the internal capsule, and anterior perforated substance. Other motor disorders related to ACA infarction include hypometria, bradykinesia, global akinesia, loss of reciprocal coordination, parkinsonian gait, tremor, dystonia, and motor neglect. Isolated sensory deficits are less common. Abulia, agitation, motor perseveration, memory impairments, emotional lability, or incontinence, as well as anosognosia, are among the neuropsychologic features associated with ACA infarction. Bilateral ACA infarction is rare. One study involving 48 patients with ACA infarction had only 2 cases, with a mean age at presentation of 40. (7) Paraparesis and akinetic mutism were also documented in the context of bilateral ACA stroke. Headaches also correlate with ACA infarction, specifically in instances of arterial dissection.

In our case, patient is an old age and had presented to us with paraplegia and mutism of recent onset. The risk pathogenesis of the phenomenon was concluded to be due to atherosclerosis as directly evidenced by diffuse narrowing of right A1 ACA and absent flow in left ACA in MR angiography and indirectly evidenced by presence of athero-calcific plaque in right common carotid artery.

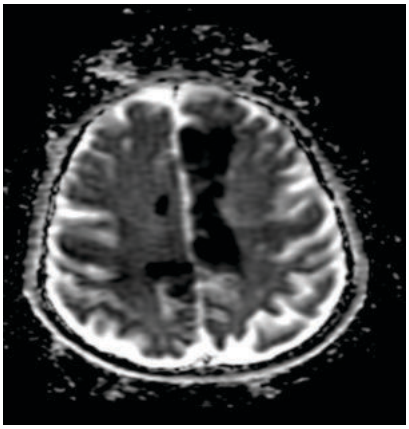

Figure A. DWI images showing areas of diffusion restriction with corresponding low values on ADC maps

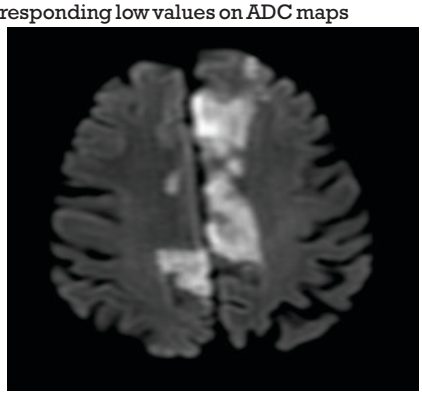

Figure B. DWI images showing areas of diffusion restriction in bilateral ACA territory- suggestive of acute infarct

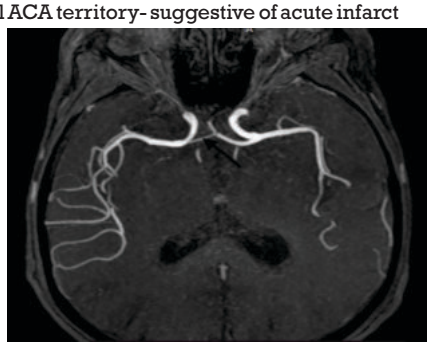

Figure C. MR angiography showing diffuse narrowing of A1 segment of right ACA.

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