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**Case Report**

# Bilateral stroke: unpaired anterior cerebral artery infarct rare presentation

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## ABSTRACT

ACA territory is an uncommon site for occlusive vascular strokes. Small degree of asymmetry of anterior arteries is present in 80% of patients. Common variants of anterior cerebral arteries include aplasia or hypoplasia of A1 segment. Hypoplasia is found in 10% and aplasia in 1-2% of postmortem examinations. Of all ischemic strokes ACA territory strokes constitutes only 0.6 to 3%. Bilateral ischemic stroke in unpaired ACA is rare entity. We report a case of unpaired ACA due to hypoplasia of right ACA, presenting as stuttering hemiplegia with bilateral focal neurological deficit.

**Keywords:** Unpaired ACA, Bilateral stroke, Hypoplasia, Aplasia

## INTRODUCTION

Cerebrovascular infarction is a well-recognised clinical disorder but simultaneous bilateral infarction is relatively rare. Several morphological variations of the circle of Willis exist. In the Hodes et al.[1] autopsy series, only 18% of specimens of the circle of Willis were found to be anatomically normal and Krabbe-Hartkamp et al. reported that 42% of subjects showed a complete circle of Willis on magnetic resonance angiography. Anomalies of the ACA are not quite as rare as was previously believed. ACA may be hypoplastic on one side in 4-10% and aplastic in 1.3% of cases. Variations in the post-communical segment include unpaired (0.5-5%), bihemispherical (1%) and triplex (1-2%). Angio-MR demonstrated hypoplasia of A1 segment in 3% and of A2 segment in 2% of cases. In such cases the contralateral A1 segment of ACA is dilated and through dilated anterior communicating artery supplies blood to the entire area vascularized by anterior cerebral arteries. If a disturbance of the blood flow occurs in some variants of ACA in which one ACA provides blood for the both cerebral hemispheres, than that might lead to bilateral cerebral infarctions in ACA irrigational areas.[2]

## CASE REPORT

A male patient aged 65 years, came to the GGH Kurnool with sudden onset of weakness of right upper limb. On examination pulse rate, rhythm was normal, with low volume carotid pulse on left side with bruit, B.P was 180/100 in right brachial artery, 176/96 mm of Hg on left side. Nervous system examination showed right hemiplegia with upper motor facial palsy on right side. Other systems were normal. Diagnosed as ischemic stroke and further evaluated. Routine blood picture was normal, RBS 140 mg%, urea 38 mg%, serum creatinine 1 mg%, serum electrolytes were normal, ESR = 30 mm/hour, lipid profile showed hyper triglyceridemia, ECG showing L.V.H, 2D echo revealed concentric LVH,

CT scan brain was normal. Started on IV mannitol, antiplatelet agents, statins. MRI brain showed hyper intense lesions in left ACA territory. After 48 hours, power improved on right side but patient developed weakness of left upper limb and lower limb. Repeat MRI brain showed hyper intense lesions (Figure 1), in ACA territory both sides. CT angiogram revealed hypoplastic right ACA (Figure 2, 3), narrowing of left internal carotid (Figure 4). On Doppler study of neck vessels 80% to 90% narrowing of left internal carotid was seen. Patient improved slowly with treatment and is on regular follow up.

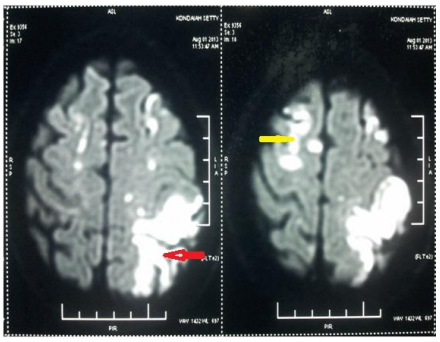

**Figure 1: MRI brain showing hyper intense signals in ACA territory both sides.**

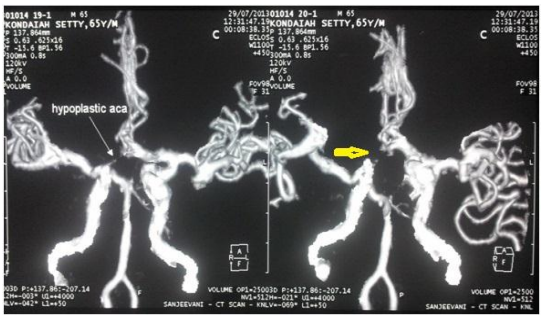


**Figure 2: CT angiogram showing hypoplastic right ACA (white & yellow arrows).**

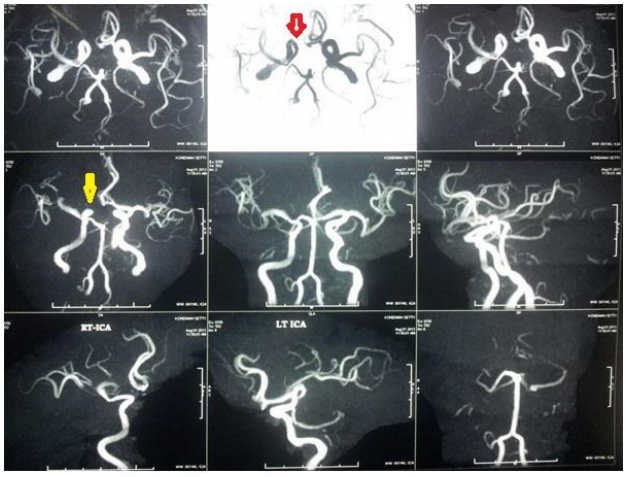


**Figure 3: CT angiogram showing hypoplastic right ACA (red and yellow arrows).**

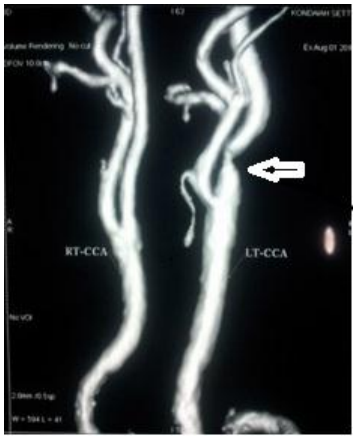


**Figure 4: Narrowing of left internal carotid artery (white arrow).**

## DISCUSSION

The ACA is a major vessel responsible for the blood supply to the interhemispheric region. ACA territory accounts for only 0.3% to 4.4% of cerebral infarctions reported, bilateral ACA infarct being even rarer.[3] Many anatomic variations of ACA are found angiographically. But three distinct patterns are well recognised: accessory ACA, bihemispheric ACA and unpaired or azygous ACA. The relative rarity of ACA territory infarcts can be highlighted by the fact that Nakajima[4] experienced only 27 cases of ACA occlusion from 1977 to 1989 of which only 4 were bilateral. Bogousslavaski found ACA occlusion in only 1.8%.of which no case was with bilateral infarcts. In 2004, Yamaguchi et al. reported a case with a patient presenting with lower limb weakness and magnetic resonance angiography demonstrating bilateral anaplastic ACAs.[5]

In our case patient is having hypoplastic ACA on right side (Figure 2, 3) with narrowed internal carotid on left side (Figure 4) cause for the cerebral vascular compromise leading to the focal neurological deficit with bilateral infarcts in ACA territory (Figure 1).

If anterior communicating artery is congenitally atretic, or if atheromatous lesion occurs in distal anterior cerebral artery, TIAs and strokes can occur. Occlusion can be either at precommunal segment ($A_1$)/stem connecting ICA to anterior communication artery or post-communal segment ($A_2$)/distal to anterior communicating artery.

Unilateral occlusion of the stem of the ACA proximal to its connection with the anterior communicating artery ($A_1$ segment) is usually well tolerated since adequate collateral flow comes from the ACA of the opposite side. Branch occlusion of the ACA produce only fragments of the total syndrome, as would be expected, usually produces a spastic weakness or cortical sensory loss in the opposite foot and leg.[6] Maximal disturbance occurs when both arteries arises ($A_2$ segment) from one ACA stem due to contralateral $A_1$ segment aplasia in which

case there will be infarction of the medial parts of both the cerebral hemispheres. This produces para paresis with weakness more marked peripherally, cortical sensory loss, urinary incontinence and abulia and aphasic symptoms. These patients lie in bed unwilling to initiate any voluntary movement including speaking.[7] Complete infarction due to occlusion of one ACA distal to the anterior communicating artery results in a sensory-motor deficit of the opposite foot and leg and a lesser degree of paresis of the arm with sparing of the face.[6] Urinary incontinence, contra lateral grasp and sucking reflexes and Para tonic rigidity (Gegenhalten) may be evident. Foot drop is a constant finding and difficulty in walking is even greater than expected from the weakness present because apraxia of gait is an additional problem and may be combined.

In conclusion, simultaneous bilateral cerebral infarction can be the result of a unilateral anterior cerebral artery occlusion and this can potentially mimic a space-occupying lesion. Anomalies of cerebral vasculature are not as rare as is usually believed and this should be borne in mind while investigating unusual presentations of cerebrovascular infarction.

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