Case Report

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Bilateral Anterior Cerebral Artery Thrombosis - A Mimicker of Basilar Artery Thrombosis: A Case Report.

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Abstract:

We present a rare case of bilateral anterior cerebral artery (ACA) territory infarction with akinetic mutism, quadriparesis and frontal lobe release signs along with limbic dysfunction mimicking a basilar artery thrombosis. The etiology was thrombosis of the anterior communicating artery with propagation or embolism of the thrombus to both A2 segments of the anterior cerebral artery and restricted or absent blood flow in the branches arising from the A2 and A3 segments causing extensive infarction. We also aim to review the anatomy of the ACA to understand localization of lesions.

Key words: Bilateral cerebral infarction, Aneurysm, Anterior cerebral artery, Magnetic resonance angiography

Introduction

Bilateral anterior cerebral artery vascular territory infarction is very rare. Here, we present an elderly male with bilateral infarction due to aneurysm of the anterior communicating artery.

Case Report:

A 76-year-old healthy male and a heavy smoker, presented with acute onset loss of responsiveness. On clinical evaluation he was mute, non-responsive, quadriparesis with decorticate posturing and spontaneous eye opening. Conjugate eye movements were noted as though he was looking about the room, but he did not track moving objects. Pupils were equal and reactive with intact dolls eye movement and corneal reflex. Gag reflex was equivocal with a difficult assessment. Tendon reflexes were asymmetrical and exaggerated more in the lower limbs compared with the upper limbs. Both plantar were extensor with no clonus. Frontal lobe release reflexes (snout and grasp reflexes) were present. Communication with eye movements or eye blinks was unsuccessful. There was no response to tactile stimulation except grimacing to extreme pain. His blood pressure was high (newly diagnosed), further corroborated with the electrocardiogram and echo cardiogram showing left ventricular hypertrophy secondary to chronic hypertension. There was no evidence of carotid or cardiac embolization on carotid duplex and echocardiogram. A CT scan of the brain at 4 hours of onset of symptoms revealed age-related cerebral atrophy and small vessel disease with old lacunar infarctions of bilateral basal ganglia. There was no evidence of any early CT changes suggesting an infarction or intracerebral hemorrhage or subarachnoid hemorrhage. However, a repeat scan at 17 hours showed bilaterally dense A2 segment with acute infarction of bilateral anterior cerebral artery territory. There was no evidence of sinus venous thrombosis or intracerebral or subarachnoid hemorrhage on the second non-contract CT as well.

An electroencephalogram showed mild generalized slowing (6-8Hz) with no evidence of encephalopathy or non-convulsive status epilepticus. The patient was also tested for COVID-19 as ischemic strokes are a rare presentation and the patient had been visited by his son who had arrived from overseas after the onset of the COVID pandemic just after the lockdown was imposed and was living with him.

An MRI, MRA and MRV time of flight was performed on a 3 Tesla machine on day 6. MRI showed high signal intensity in T2 FLAIR, T2W

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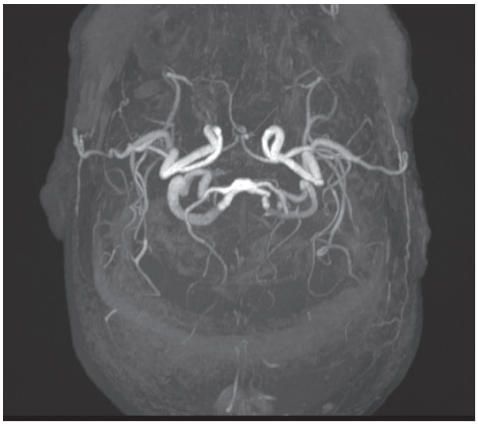
and DWI images in the bilateral ACA territory with diffusion restriction in these areas as well as involvement of the corpus callosum. MRA showed an aneurysm in the anterior communicating artery, measuring 2.5mm by 3.5 mm facing left, with the neck of the aneurysm measuring 1.8mm (Fig 1). The A2 segment of the right ACA was opacified normally (possible interval recanalization) and the A2 segment of the left ACA was not visualized. Both internal carotid arteries and middle cerebral arteries were normal in course and caliber. The basilar artery showed a normal course and caliber and divided into normal sized posterior cerebral arteries. The posterior communicating arteries on each side were normally developed. The right vertebral artery was prominent than the left, however both vertebral arteries were normal in course and caliber. MRV study was normal.

Figure 1. Brain Magnetic resonance angiography revealed an aneurysm in the anterior communicating artery (arrow), measuring 2.5mm by 3.5mm facing left, with neck of aneurysm measuring 1.8mm.



In summary MRI revealed an unruptured anterior communicating artery aneurysm with thrombosis of the A2 segment of the left anterior cerebral artery with possible interval recanalization of the A2 segment of the right ACA. A CT angiogram could not be performed as the patient's condition continued to deteriorate.

All other biochemical parameters were within normal range. As the window for thrombolysis was missed, he was commenced on aspirin and atorvastatin. With significant deterioration by day 10 he was commenced on palliative care with family consensus. He succumbed on day 14. A postmortem study was not performed.

Discussion:

To localize and understand the various presentations of bilateral ACA territory infarctions knowledge of the vascular anatomy of the ACA is essential. The ACA is divided in to 3 segments – A1, A2 and A3. The Horizontal A1 extends from the origin of the ACA to its junction with the anterior communicating artery (AComA). The medial lenticulostriate artery arises from the A1 segment. The A2 segment extends from the AComA to its bifurcation into the pericallosal and the calloso-marginal arteries and its branches include orbitofrontal, frontotemporal and the recurrent artery of Heubner. The recurrent artery of Heubner is a lenticulostriate branch arising from the proximal A2 segment. The A3 segment refers to the cortical branches supplying the anterior two-thirds of the medial hemispheric surface and a small superior area over the convexities.

In summary the ACA supplies the medial surface and the adjacent rim of the lateral convexity of the entire frontal and parietal lobes, the anterior limb of the internal capsule, the inferior half of the head of the caudate nucleus, portions of the inferior putamen and globus pallidus, parts of the hypothalamus, the anterior column of the fornix and the anterior part of the corpus callosum(1)(2).

The corpus callosum is uncommonly involved in infarcts as it gets its blood supply from a branch of the anterior communicating artery (median callosal or subcallosal artery), from the pericallosal artery (distal part of the anterior cerebral artery) and from the posterior pericallosal artery (usually a branch of the posterior cerebral artery).

Bilateral infarction of the ACA vascular territory is rare occurring in 0.6% to 3% of all strokes (3). In a case series of 1490 cases only two patients had bilateral infarctions (3). Unilateral infarctions are usually due to embolic phenomena from the heart or the internal carotid artery or due to contralateral or ipsilateral occlusion of the internal

carotid artery or local thrombosis caused by vasculitis (4). Bilateral ACA territory infarctions are usually due to a rupture of an aneurysm of the anterior communicating artery or from thrombosis of the pre-communicating part of the anterior cerebral artery combined with an agenesis of the contralateral pre-communicating part of the ACA (1). Bilateral infarctions from cardiac or carotid embolism are extremely rare (1). Bilateral ACA territory infarction presents as motor deficits of the lower extremities and disturbed consciousness mimicking a basilar artery occlusion (5)(6) due to ischemic dysfunction of the limbic system, specially the cingulate gyrus.

Our patient had an unruptured berry aneurysm. Two CT brains done within 17 hours did not show any evidence of a subarachnoid hemorrhage and the aneurysm was most likely due to chronic hypertension for which he had not taken appropriate medical care. Vasculature a sperm RA was within normal limits except for the unruptured small berry aneurysm in the (AcomA). From the clinical presentation the most likely etiology would have been a thrombus in the anterior communicating artery and the aneurysm propagating or embolizing into the A2 segment of both ACA. With both the A2 segments occluded initially the recurrent arery of Huebner and the pericallosal and the callosomarginal arteries arising from the A2 segment would have been occluded or had restricted flow thus leading to infarction of the corpus callosum, the anterior limb of the internal capsule, the inferior half of the head of the caudate nucleus, portions of the inferior putamen and globus pallidus, parts of the hypothalamus, the anterior column of the fornix and the anterior part of the corpus callosum. This leads to the clinical manifestation of quadriparesis, akinetic mutism and frontal lobe release signs along with other features of limbic system dysfunction mimicking a basilar artery thrombosis.

Conclusion:

Bilateral ACA infarction, a very rare presentation is usually fatal unless interventional therapy is tried. A meticulous decision on early CT angiogram (CTA) in rare and unusual presentations is essential for definitive diagnosis of bilateral ACA occlusion or basilar thrombosis enabling lifesaving timely thrombolysis or thrombectomy.

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