

AZYGOS ANTERIOR CEREBRAL ARTERY CAUSING BIFRONTAL INFARCTS: A CASE REPORT

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Abstract

The azygos anterior cerebral artery (ACA) is an uncommon anatomical variant. There is absence of the anterior communicating artery (ACom) and bilateral anterior cerebral arteries (ACA) form a common trunk and travels superiorly in the interhemispheric fissure. It can result in bilateral frontal infarcts in case of thromboembolism. Therefore its correct diagnosis is of grave importance for better patient clinical outcome. Cerebrovascular infarction is a common condition, however simultaneous symmetrical bilateral infarction secondary to arterial occlusion is a rare entity. We report a case of a 50 years male with thromboembolic event in azygos anterior cerebral artery resulting in bifrontal cerebral infarcts with associated hypoplasia of right cerebral artery. Normally the paired ACAs are connected by the anterior communicating artery to complete the anterior portion of the circle of Willis. These arteries then supply the medial surface of each cerebral hemisphere. However in azygos ACA the ACom is absent and bilateral ACA has common A2 segments. In humans the incidence of unpaired ACA is rare with an incidence between 0.3-2 % .

Keywords:

Azygos anterior cerebral artery, bifrontal infarcts, frontal lobes.

Introduction

The azygos anterior cerebral artery (ACA), with an incidence of <1%¹ is a rare anatomical anomaly in the circle of Willis. In this vascular anomaly, the distal segments (A2) of both ACAs are represented by a single vessel from which all branches supplying the medial aspect of cerebral hemispheres and corpus callosum originate. The clinical significance of an AACA is the alteration of arterial hemodynamics of the frontal lobe. It is also associated with other malformations like agenesis of the corpus callosum, arterio-venous malformations, hydranencephaly and aneurysms of the azygos ACA (AACA) are even rarer².

Case Report

A 50 years old male patient was admitted in the emergency department reporting severe headache, dysarthria and motor weakness in lower limbs since 48hours. His past medical history was remarkable for hypertension and was on antihypertensive drugs. He was nondiabetic/ non smoker and past surgical history

was insignificant. There was no familial hyperlipidemia. He had a positive family history of hypertension. However he always complained of headache for which he had been taking antihypertensives and analgesics. On clinical examination his blood pressures were 180/100mmhg, pulse 80/min and temperature 37°C. Neurological examination showed GCS score of 11. Lower limb weakness was noted with response to painful stimuli (M +4). He was confused and delivered inappropriate speech (V+3). His eye movements were spontaneous (E+4). The remainder of the physical examination was unremarkable. He went through plain CT scan (prime aquilion 128 slice Toshiba) that showed bilateral symmetrical hypodense areas in paramedian frontal lobes (fig 1a and b) and possibility of venous infarct was raised due to bilateral and symmetrical involvement however no evidence of any hemorrhage was noted. Patient was further advised CT angiography which included early phase images for arteries and delayed images were taken for venous sinuses. The venogram was normal without any evidence of dural venous sinus thrombosis. 3D images of CT arteriography showed azygos anterior cerebral artery (AACA) with

endoluminal thrombus and hypoplasia of right anterior cerebral artery (fig 2), reducing the vascular flow completely, determining an ischemic stroke in the frontal lobe bilaterally and anterior region of the corpus callosum. His carotid Doppler USG showed a calcified plaque in right carotid bulb causing 29% area stenosis. No soft plaque was noted

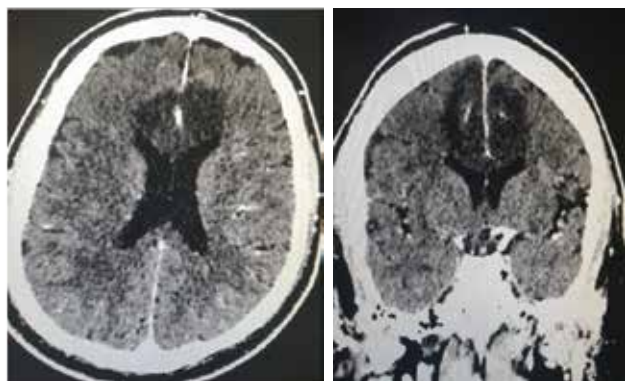


Fig 1a and 1b. Axial and coronal reconstructed images of CTA showing non enhancing hypodense ischemic infarcts in medial frontal lobes and anterior corpus callosum.



3D Image of CTA showing azygos ACA and hypoplastic right ACA.

Discussion

The term arteria termatica (also called azygos ACA) was introduced in 1885 by Yasargil, Wilder was the first to describe the fusion of both A2 segments of ACA to form a single artery¹.

The unpaired, distal A2 segment of the anterior cerebral artery is named azygos. Unpaired pericallosal artery or azygos pericallosal artery are also used for the

same anatomical variants. Although the presence of unpaired ACA is common in lower mammals, it is uncommon in humans².

Baptista defined three ACA variations in the ACA. In type I anomaly also called as true azygos ACA, there is only one azygos ACA from which all major ACA branches spread to both the hemispheres. Type II anomaly is 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 anomaly, a third artery (accessory ACA) arises from anterior communicating artery³. In our case it was type II ACA which was confirmed by 3D CT angiography.

Diagnosis of this rare anatomical variation by angiographic studies has very important role to play in the patient diagnosis and treatment of potential neurological and neurosurgical conditions⁴.

Anterior cerebral artery territory infarctions are uncommon compared to middle and posterior cerebral artery. Large number of the patients with ischemic stroke showed that the frequency of infarctions confined to the ACA territory ranges from 1% to 4.4%. Moreover, isolated occlusion of the ACA in only 1%-3.7% of patients with cerebral ischemia⁵. Bilateral ACA infarction is even rarer. Twenty-seven cases of ACA territory infarction were reported among 1490 cases of cerebral infarction in the Lausanne Stroke Registry; however, there were only two cases of bilateral ACA territory infarction⁶. In our case the initial diagnosis of venous infarct was given due to bilateral symmetrical involvement of medial frontal cortex however CT venography didn't show any venous infarction and angiography further showed AACA making a final diagnosis but after a delay of 24hours.

Bilateral ACA territory infarction is usually due to vasospasm that occurs as a complication of subarachnoid haemorrhage caused by the rupture of aneurysms of the anterior communicating arteries or distal ACAs. However, in the case of an anomaly in the anterior part of the circle of Willis, thrombosis or embolism can lead to bilateral infarction⁷. In our case bifrontal infarcts were due to anomaly of the anterior part of circle of Willis rather than subarachnoid hemorrhage.

According to Brian et al, simultaneous bilateral cerebral infarction 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 when investigating unusual presentations of cerebrovascular infarction⁷. In our case the initial CT scan was reported as venous infarct and possible superior sagittal sinus thrombosis was suspected to be the main culprit which however was normal on subsequent CT Venography study.

Association of aneurysms with azygos ACA is noted, though rarely with only few cases reported in the literature. The mechanism of aneurysm formation in azygos ACA could be due to hemodynamic stress or because of a congenitally anomalous artery⁸

Azygos ACA were reported to be associated with aneurysms which were mostly saccular in nature. Non-saccular aneurysms of azygos ACA have also been reported, which require complex clip application or multiple clips⁹.

In our case thrombolytic therapy (tPA) was not started initially as the patient presented late. Patient was given aspirin and kept on antihypertensives. Later on patient was given anticoagulant. During his stay he was given stroke care. Patient had lower limb paresis and urinary incontinence on discharge however he was well oriented to time, place and person. He was advised to continue drugs, physiotherapy, perform exercises and take low fat diet.

Conclusion

In conclusion, this case highlights the problems faced by the radiologists in diagnosing and clinicians in treating the patients of arterial / venous territory infarcts, as it can make a huge impact on the treatment options. The treatment options had the potential to cause more damage than already sustained if not diagnosed properly. This case also highlights the need for early access to high-level diagnostic imaging. The sharing of this very rare anatomical variant will alert fellow radiologists bearing in consideration the alteration of arterial hemodynamics of the frontal lobe therefore saving patients time and producing a better outcome.

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Abdul Khaliq; data collection, data analysis, manuscript writing, manuscript review

Palwasha Gul; data collection, data analysis, manuscript writing, manuscript review

Pari Gul; concept, data collection, data analysis, manuscript writing, manuscript review