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# Enlarged anterior communicating artery masquerading as intracranial aneurysm. Case report

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Keywords
A1 hypoplasia, ACoA complex, anatomic variations, enlarged ACoA

## Abstract

**Background.** The anterior communicating artery (ACoA) complex consists of the ACoA, the pre-and post-communicating segments of the anterior cerebral artery, and the recurrent artery of Heubner. It is the most common site for anatomical variations in the circle of Willis. Such variations can mimic intracranial aneurysms.

**Case description.** A 30-year-old female presented with recurrent episodes of extreme headache and bilateral tinnitus. A brain computed tomography (CT) scan showed no significant lesions, while her CT-angiography (CTA) showed an enlarged vascular lesion at the ACoA, raising the suspicion for an ACoA aneurysm. A repeated CTA revealed a rare anatomical variation with a pattern of cross dominance in the ACoA complex; the left A1 and right A2 were dominant-enlarged, resulting in an enlargement of the ACoA. The presence of an ACoA aneurysm was hence excluded and the patient was managed conservatively. At 6-month follow-up, CTA showed no new findings.

**Conclusion.** ACoA enlargement can result from unequal hemodynamics around the ACoA complex, which may be mistaken for an aneurysm. A thorough study of the imaging data is of pivotal importance and may change the management strategy.

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## Introduction

The anterior communicating artery (ACoA) connects the two anterior cerebral arteries, dividing them into pre-communicating (A1) and post-communicating (A2) segments. ACoA, A1, A2 segments, and the recurrent artery of Heubner are collectively known as the AcoA complex [1]. Anatomical variations in this complex are common and are detected

in 58-85% of all patients with ACoA aneurysms [2]. A range of different arterial variations was indexed, including fenestration, duplication, trifurcation, hypoplasia, and aplasia [3]. Several efforts have been made to classify the ACoA complex variations as they may influence the surgical approach of any co-existing vascular disease [1].

Also, these anatomical variations can mimic intracranial aneurysms by the superimposition of a duplicated or a fenestrated vessel [2]. Furthermore, the net-result of the hemodynamic changes in these variations plays a considerable role in the development of several vascular pathologies, such as aneurysms and ischemic strokes [3]. Moreover, ACoA complex is the most common site for intracranial aneurysms, constituting up to 40% of all aneurysms, which can further ambiguate identifying these variations [4]. In this report, we present a case report of an enlarged ACoA masquerading initially as an ACoA aneurysm. To our knowledge, this is the first such case report of its kind in the literature.

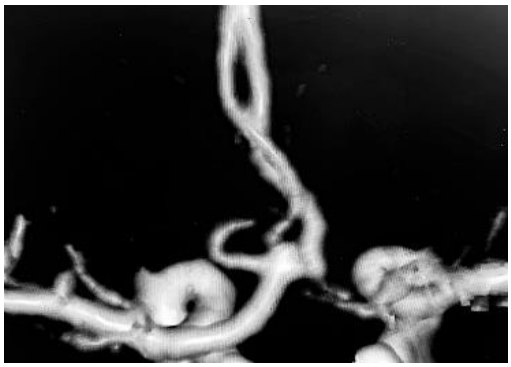
## Case scenario

An otherwise healthy, 30-year-old, female typist was referred by her general physician with a suspension of an unruptured ACoA aneurysm. She had a history of recurrent attacks of severe non-localized headaches associated with episodes of bilateral tinnitus, more on the left side, for two weeks. Each attack lasts for more than one hour with no exacerbating factors. The headache was rated as 8/10 by the patient, and it impacted her daily activities. The patient had an intact neurological examination. Her brain computed tomography (CT) scan revealed no significant lesions apart from a suspicious anterior interhemispheric lesion. The CT-angiography showed a pouched-out vascular lesion at the ACoA, raising suspicion for an ACoA aneurysm (Fig. 1A). As we had no endovascular facility at our institute, we opted to perform another CTA, aiming to obtain better-qualified images and more clear views. The new CTA showed a dominant left A1 and a hypoplastic right A1 (0.4 mm) segments of the anterior cerebral artery, which is a common variation especially encountered with ACoA aneurysms. Besides, a noticeable enlargement of the ACoA at its junction with the right anterior cerebral artery was found, most probably resulting from the inverted hemodynamics between the two sides of ACoA. The left A2 was normal, while the right A2 was enlarged. No intracranial aneurysms were detected (Fig. 1B-D). The patient was informed that she had a normal variation in the circulation rather than an ACoA aneurysm. Simple analgesics were prescribed, which helped the headache. The patient continued to have tinnitus. Therefore, a brain magnetic resonance imaging (MRI) was done to exclude the possibility of an arteriovenous fistula, which then came back negative. The patient was referred to an otolaryngologist, who diagnosed her with mild otitis media. At her 6-month follow-up visit, the patient's headache and tinnitus had resolved, and she had resumed her daily activities. The follow-up CTA and MRI studies revealed no new findings.

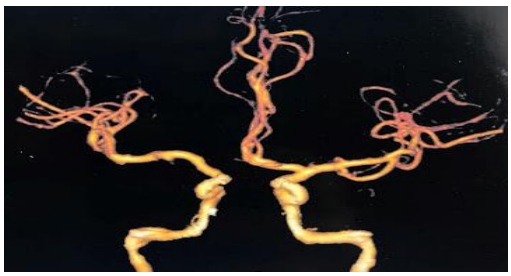
**Figure 1.** Imaging of an ACoA variation mimicking an aneurysm. (A): A cerebral CT angiography, 3D reconstructed image showing a suspected ACoA aneurysm. (B, C) different views of a second CTA, confirming the absence of an intracranial aneurysm. Instead, an enlarged ACoA was noted accompanied by an enlargement of both the left A1 and right A2. (D) An artistic depiction of the ACoA arterial complex demonstrating the inverted hemodynamics. The vascular configuration showing an enlarged ACoA (*), LA1, and RA2 with a hypoplastic RA1 and a normal-sized LA2 with no evidence of aneurysm formation.

ACoA; Anterior communicating artery, ICA; Internal carotid artery, MCA: middle cerebral artery, R; Right, L; Left. M1: sphenoidal or horizontal segment of the MCA. A1: Pre-communicating segment of the anterior cerebral artery. A2: Post-communicating segment of the anterior cerebral artery.

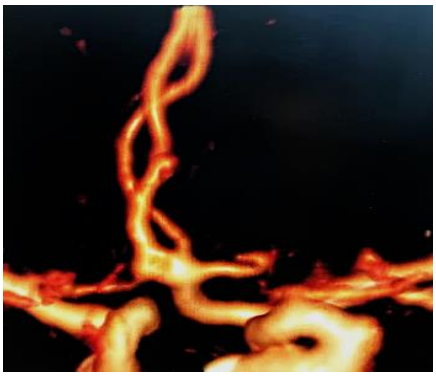
**A.**



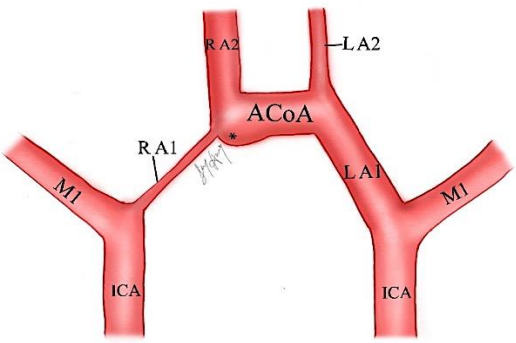
**B.**



**C.**



**D.**




## Discussion

The anterior cerebral artery plays a vital role in the blood supply to the anterior collateral circulation as it gives off penetrating striatal arteries that supply the anterior hypothalamus, septum pellucidum, and parts of the corpus striatum [5]. Vascular anomalies of the ACoA complex are not uncommon and are mostly encountered in the A1 segment [6]. A unilateral hypoplastic A1 segment is one of the most common anomalies in the circle of Willis with an incidence of 3% [3,7]. Marinković et al. described two types of A1 hypoplasia based on carotid angiogram namely, mild and extreme hypoplasia [8]. Mild hypoplasia is diagnosed when the diameter of the A1 segment is more than 1 mm, whereas extreme hypoplasia is defined by an A2 segment diameter of less than 1 mm [8]. Hypoplasia of one A1 segment is compensated by the dominance of the contralateral A1 segment [6]. These variations are often asymptomatic or found incidentally, chronic headache is, however, a possible presentation in patients with arterial hypoplasia [9]. The A1 segment is a rare site for ischemic strokes, representing only 0.6-3% of all stroke etiologies [10]. The risk for ischemic stroke is further aggravated by the presence of such hypoplasia [5,10]. Furthermore, the presence of A1 hypoplasia increases the risk of aneurysm formation; a phenomenon that can be explained, in part, by alteration in ACoA complex hemodynamics [1]. In this case, the extreme hypoplastic right A1 segment was compensated by the dominance of the left A1 segment, which supplied the right A2 segment through the ACoA. These variations manifested radiologically as an enlarged ACoA with a pouched-out appearance leading to a suspicion of an ACoA aneurysm. Moreover, the presence of such inverted hemodynamics around the ACoA complex may render the distal circulation more prone to ischemic stroke or aneurysm formation [7]. Thereafter, follow-up by annual imaging is important in determining patient's prognosis and could confirm or exclude the stability of such anomaly [10]. Knowing such variations is also essential in planning surgical and endovascular approaches, as these variations can require a different management strategy [3]. The purpose behind presenting this case is to highlight their propensity to pose a diagnostic dilemma, especially when catheter angiography is not feasible. Moreover, our case demonstrates how cautious the surgeon should be in reviewing the patient's radiology while planning the appropriate management.

## Conclusion

Anterior communicating artery enlargement can result from unequal hemodynamics around the anterior communicating complex, which may be mistaken for an aneurysm. A thorough study of the imaging data is of pivotal importance and may change the management strategy.

### Abbreviations

ACoA - Anterior communicating artery
CT - Computed tomography
CTA - Computed tomography angiography
MRI - Magnetic resonance imaging.

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