

MANUSCRIPT: COMPARING THE RELATIONSHIP OF DOMINANT A1 SEGMENT TO ANTERIOR COMMUNICATING ARTERY ANEURYSM

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Introduction:

The ACoA complex has a significant clinical implication and that is it is the most common site of intracranial aneurysm accounting for 23–40% and are considered the most frequent type of intracranial aneurysm in young adults, [1]. Multiple external factors together with predisposing genetic background, and contribute to intracranial aneurysm formation, [1,2]. Moreover, recently there has been a growing interest in the effects and consequences of the flow of blood and hemodynamics on the pathophysiology of intracranial aneurysm formation.

The cerebral vasculature in the anterior circulation in patients with anterior communicating artery aneurysms has long been observed to have an increased percentage of vascular anomalies like aplasia, hypoplasia, duplication, and fenestration of ACA segments. Among them, it has been reported by Rinaldo that hypoplasia of an A1 segment, is the most frequent anomaly, which occurs in 16 to 68% of patients with Acom aneurysms on the side of the larger or dominant A1 segment, [3]. When there is a dominant A1 supply which means one A1 dominantly supplies both anterior cerebral arteries (ACA), it is thought to have a causal relationship with the development of Acom aneurysms from increased shear stress on the arterial walls by jets of blood flow, [3,4,5]. This hemodynamic stress from unbalanced blood flow in the Acom and its contribution to aneurysm formation has not been well defined, [3-5]. Moreover, the exact relationship between this anatomical variant of the Acom complex and the rate of aneurysm formation and aneurysm rupture needs to be explored further. Thus, in this review, we address the various hemodynamics and geometrics involved in aneurysm initiation and formation seen with hypoplastic A1 segment and determine the relationship of this anatomical variation with Acom aneurysm formation.

Case discussion-

In our case series we operated total 10 patients, which Acom aneurysm on DSA and its dominant which exclusively filled by on A1 segment which suggestive that filling of the aneurysm was exclusively from the dominant side of the ACA.

For identify the dominant flow, needs DSA with cross clamping of the carotid artery.

All of these patients were posted for surgical clipping of the aneurysm with using of intraoperative fluorescent study by using indocyanine green dye for better guidance. Rule out other vessels clamped during the procedure by seeing patent flow beyond the clip. two of these patient developed anterior cerebral artery infarct which treated conservatively without added deficit.

Anatomic Configuration

Examples of DSA and CTA images of the 3 A1 configuration categories are shown in **Fig 1**, with examples of symmetric, dominant, and complete filling from A1s to A2s. All DSA studies are from the Cerecyte Coil Trial, including those with AcomA aneurysms and others with aneurysms of other vessels. The same criteria were used for the CTA studies, from patients having undergone CTA for acute stroke.

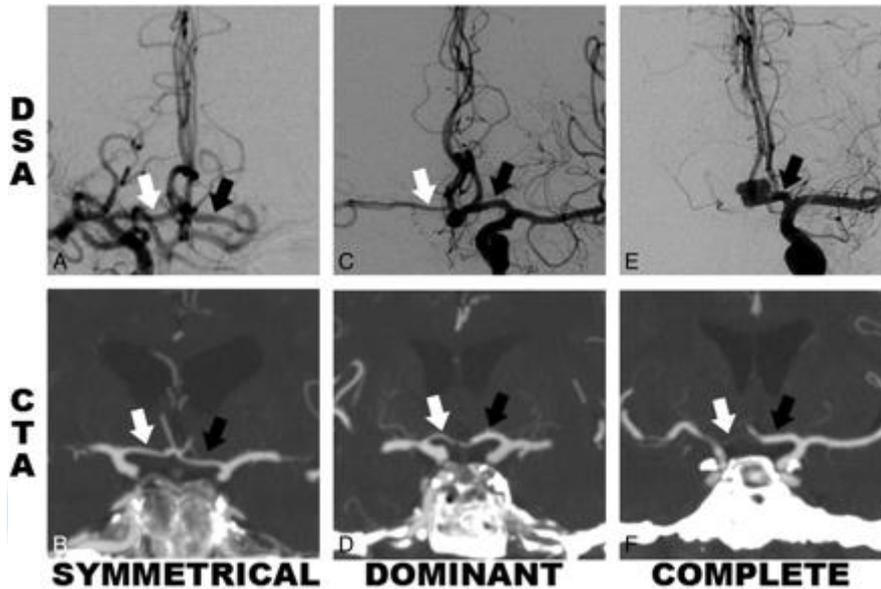


FIG1: xamples showing A1 segment categories with white and black arrows indicating right and left A1 segments, respectively, for the symmetric, dominant, and complete configurations. *A, C, and E*, DSA studies of patients with aneurysms, AcomA (*C and E*) and elsewhere (*A*). *B, D, and F*, CTAs used as the control group. The “symmetric” configuration from both DSA and CTA is seen in *A and B*, respectively. The “dominant” configuration is shown as a larger left A1 and smaller right A1 as shown in *C and D*, respectively. The “complete” configuration shows no evidence of the right A1 segment on *E* and a very small right A1 on *F*, with total or near-total supply to both A2s from the single A1.

Flow Configuration

Figure 2 illustrates the concept of using the contrast injection filling and dilution phases during selective angiography to determine the A1 flow configuration. Representative still frames are shown after injection of contrast into the left ICA and right ICA in the same patient showing injection (**Fig 2 A, -B**), early dilution after injection pressure in the neck is removed (**Fig 2 C, -D**), and late dilution (**Fig 2 E, -F**). When we inject into the right ICA (**Fig 2 A, C, and E**) for this patient, both A2 segments maintain equal contrast throughout the dilution period. When we inject into the left ICA (**Fig 2 B, D, and F**), the contrast within both A2 segments is rapidly diluted by blood from the right A1. This exemplifies right A1 dominance with some contribution from the left A1.

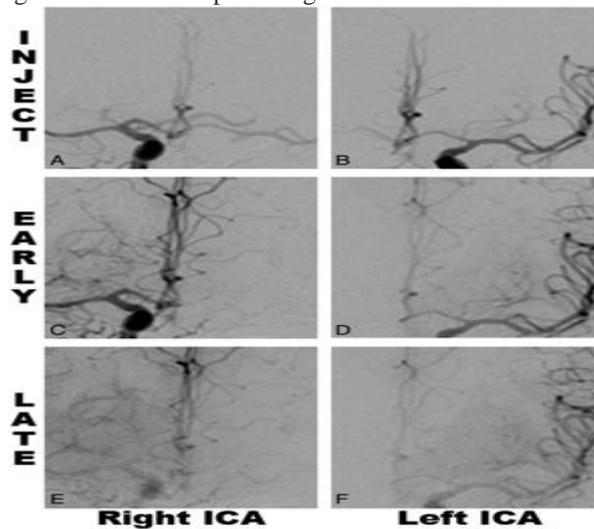


Fig 2.

A1 flow configuration on DSA from a single patient with right A1 dominance. *A–F*, Selective images from angiography after injection of contrast into the right ICA (*A*, *C*, and *E*) and left ICA (*B*, *D*, and *F*) for the same patient. During the pressure of injection into the ICA in the neck (*A* and *B*), contrast is seen filling both A2s from each A1. Following release of the pressure of injection, contrast continues showing the early dilution phase (*C* and *D*), maintaining both A2s filling from the right A1 (*C*), while there is substantial dilution of left A2 (*D*), injected from the left ICA. The late phase in both *E* and *F* shows all vessels as dilute. Effectively, there is a mixed supply of left A2 from both A1s, giving the classification of the configuration as “right dominant.”

Discussion:

AcoA aneurysms have relatively complex anatomical structures, and have anatomical variations, with critical adjacent neurovascular structures which require close attention to these critical structures during exposure to surgical clipping. Variations in the AcoA and the relationship between bilateral A1 and A2 are also important factors in the formation of aneurysms. Numerous studies have summarized the relationship between the AcoA and adjacent vessels and aneurysm formation, [3-6]. Factors such as fenestration deformity of the AcoA, size ratio, and the presence of A1 on the dominant side, have provided important clues in the genesis of AcoA aneurysms formation, [7]. The interaction of A1 segment hypoplasia with other known risk factors for aneurysm growth and rupture of ACoA aneurysms, however, is less well understood.

The natural history of saccular intracranial aneurysms can be divided into two phases, first an initiation phase and second progression phase with eventual progression to rupture, [8]. Initiation of aneurysm formation can be explained by alteration of the normal hemodynamics of blood flow acting together with structural defects of the vessel wall, [8,9]. The hemodynamic theory states that a major event in the initiation of an aneurysm is the impingement of a stream of or jets of blood flow on the vessel wall; that being greatest at the arterial bifurcation as supported by pathological glass model experiments, [10]. Hemodynamic factors include the effects of pulsatile blood flow, hypertension, and a local increase in circulation coupled with structural degeneration of the vessel wall believed in most instances to be acquired, [10,11]. This abnormal hemodynamic stress accounts for medial degeneration at the apex of a vessel bifurcation, [11].

When a dominant pattern of flow in the anterior cerebral arteries is present, the dominant A1 ends in a physiological bifurcation with increased flow past the apex going both in the ipsilateral A2 segment and into the contralateral A2 segment via the anterior communicating artery, [12]. It would seem probable that the aneurysm neck is found in this location, [12].

Anatomically, AcomA aneurysms are commonly termination type, when one A1 predominantly supplies both sides; [13]. In this configuration, a straight jet of A1 blood is directed into a relatively wide-neck aneurysm with both A2s oriented perpendicular to the dominant A1 vessel, [13]. It is hypothesized that an asymmetric A1 anatomic configuration is an important component in the development of such aneurysms, suggesting a causal relationship, [14]. It is apparent the role of hemodynamic factors in aneurysm formation.

Blood flow through a vessel can be modeled mathematically by using the Poiseuille equation relating flow, F ; to vessel length, L ; the pressure drop across the vessel, ΔP ; the blood viscosity, η ; and the vessel diameter, d :

$$F \propto \frac{\Delta P \cdot d^4}{\eta \cdot L}$$

For 2 vessels of similar length and pressure difference, a 2-fold difference in vessel diameter results in a 16-fold difference in flow, [15]. Above this cutoff value, the difference in flow between the vessels very rapidly decreases. Most interesting, the Poiseuille equation describing vessel resistance and relative flow is limited to where blood flow is laminar. Therefore, when one considers the complicated vessel branching geometry around the AcomA, the vessel diameter may become less critical in determining the subsequent flow patterns, [14,15]. The A1 to A2 flow dominance was classified on a 3-point scale: **symmetric**, no clear dominance of the inflow contribution of 1 A1 segment over the other; **dominant**, 1 A1 segment contributes more inflow to an A2 than the contralateral segment; and **complete**, no detectable inflow contribution from the contralateral segment, [16]. Initial categorization was achieved based on the vessel diameter. If the A1 segment diameters were similar, with the difference between the 2 less than half of the larger A1 diameter, they were classified initially as symmetric, [16]. Similarly, if the A1 segments showed asymmetry, with the difference between the 2 is greater than half, the smaller vessel was classified as hypoplastic and the larger, one as dominant. If 1 A1 segment was not apparent or was barely detectable on angiography, the case was categorized as complete. Note that; categorization was refined by careful inspection of the early and late dilution phases for A2 during the angiographic series. This assessment of dilution of contrast from A2s during the angiographic series allows a partly physiologic method of cataloging flow patterns, more than just the size of A1s, [15,16].

There are several hypotheses as to the precise mechanisms by which this configuration predisposes to aneurysm formation, including wall shear stress, A1/A2 bifurcation angles, and flow patterns influenced by the vessel geometry, [3,11]. Perhaps all of these factors interact. The key observation lies in realizing that the stresses of hemodynamic foci of laminar and turbulent flow occur cyclically, at a frequency of approximately 60–75 times per minute every hour of every day, month, and year of one’s life, [17]. Thus, a higher risk of aneurysm development, growth, and rupture risk may exist for A1 dominance, [17].

Progression and rupture of aneurysm: While hemodynamic stress and structural changes may lead to an aneurysm formation, it has been suggested that the factors leading to an aneurysm's progression differ from those of its initiation. Once the aneurysm has formed or once the outpouching of the vessel wall has begun secondary to the combination of hemodynamic stress and wall degeneration, its further development appears unrelated to the pattern of flow, [17,18]. Turbulence within the aneurysm sac and

vibration of its wall has been proposed as potential mechanisms of growth and rupture and are different from the flow-dependent phenomena of formation, [18,19].

From a surgical perspective, during clipping of acom aneurysms with a hypoplastic A1, the significance of arterial infarct due to vasospasm or arterial injury should not be undermined. Meticulous clinical follow-up in the postoperative period of such cases should be performed by surveillance CT scan. Moreover, we need to be vigilant of any cerebral infarction in post-operative cases of surgical clipping in the face of hypoplastic A1 with potential arterial injury and subsequent poor collateral circulation distal to the hypoplastic segment of A1 in the anterior cerebral artery.

Conclusion:

Thus our results are in union with the hypothesis that hypoplastic A1 is significantly associated with acom aneurysm formation and progression based on hemodynamic studies. We also propose an attentive and watchful approach to patients in the immediate postoperative period of clipping of Acom Aneurysm with hypoplastic A1 and early post-operative surveillance CT head if necessary.

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