Turkish Neurosurgery 2005, Vol: 15, No: 4, 196-199

The Association of A1 Segment Hypoplasia/Aplasia with Anterior Communicating Artery Aneurysms: A Radiological Study

A1 Segment Hipoplazisi/Aplazisi ile Anterior Komunikan Arter Anevrizması Birlikteliği: Bir Radyolojik Çalışma

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ABSTRACT

OBJECTIVE: To determine the incidence of A1 segment hypoplasia/aplasia in a Turkish population and to discuss its importance in etiology of anterior communicating artery aneurysms (AComAA).

METHODS: Cerebral diagnostic digital subtraction angiography (DSA) data from our institution between 2002 and 2004 were analyzed retrospectively. The patients were divided into aneurysm and control groups. All patients who had a brain tumor, arteriovenous malformation, arteriovenous fistula or aneurysms other than AComAA were excluded from the study. From 182 patients studied, 75 had AComAA. The control group included 107 patients without any vascular pathology.

RESULTS: The incidence of A1 segment hypoplasia/aplasia was 41.33% in patients with AComAA and 10.28% in control group. In the aneurysm group, right A1 hypoplasia and right A1 aplasia were established in 12% of the patients. Left A1 hypoplasia was seen in 9.33% and left A1 aplasia in 8% of the patients. In the control group, the most frequently observed anomaly was right A1 hypoplasia at 7.48%. Right A1 aplasia, left A1 hypoplasia and left A1 aplasia were found at the same frequency of 0.93%.

CONCLUSION: A1 segment hypoplasia/aplasia plays an important role in occurrence of AcomAAs. In the literature, the incidence of A1 hypoplasia/aplasia in AcomAAs ranges between 50% and 80%. In our series, we found a lower value than previous series at 41.33%. This may be related to the genetic features of Turkish population and the hypoplasia criteria we employed.

KEY WORDS: Anterior communicating artery, Aneurysm, A1 hypoplasia, A1 aplasia

ÖZ

AMAÇ: Bir grup Türk populasyonunda A1 hipoplazi/aplazi insidansını belirlemek ve bu durumun anterior kommunikan arter anevrizmalarının (AComAA) etiolojisindeki önemini vurgulamak.

YÖNTEM: 2002-2004 yılları arasında hastanemizde yapılmış olan diagonistik serebral dijital substraksiyon anjiografi (DSA) verileri retrospektif olarak incelendi. Bu hastalar kontrol ve anevrizma olarak iki gruba ayrıldı. Çalışmadan beyin tümörü, arteriovenöz malformasyon, arteriovenöz fistül veya AComA harici anevrizması olan hastalar çıkarıldı. 182 hastadan 75'inde AComAA sı saptandı. Kontrol grubunu herhangi vasküler patolojisi olmayan 107 hasta oluşturdu.

BULGULAR: AComAA sı olan hastalarda A1 hipoplazi/aplazi oranı %41.33, kontrol grubunda ise %10.28 olarak saptandı. Anevrizma grubunda sağ A1 hipoplazisi ve sağ A1 aplazisi %12 hastada tespit edildi. Sol A1 hipoplazisi %9.33, sol A1 aplazisi %8 olguda saptandı. Kontrol grubunda sağ A1 hipoplazisi %7.48, sağ A1 aplazisi, sol A1 hipoplazisi ve sol A1 aplazisinin birlikte insidansı %0.93 olarak değerlendirildi.

SONUÇ: A1 segment hipoplazi/aplazi, AComAA'nın oluşumunda önemli rol oynamaktadır. Literatürde yayınlanmış olan serilerde, A1 segment hipoplazisi/aplazisinin AComAA'ndaki insidansı %50 ile %80 arasında değişmektedir. Bizim serimizde daha önceki çalışmalara göre bu insidans daha düşük bulunmuştur ve % 41.33'tür. Bu durum Türk populasyonun genetik özelliklerine ve bizim kullandığımız hipoplazi kriterlerine bağlı olabilir.

ANAHTAR SÖZCÜKLER: Anterior kommunikan arter, Anevrizma, A1 hipoplazisi, A1 aplazisi

Received : 08.09.2005
Accepted : 18.10.2005

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INTRODUCTION

The association between AComAA and anomalies of the anterior part of Willis polygon is well known (1,2,5,8,9,11,12,13,15). These anomalies include hypoplasia or aplasia of A1 segment of anterior cerebral artery, duplex A1, A1 fenestration, azygos A2 and decreased angle between A1 and A2 (2,3,5,6,7,14). The most frequently seen is hypoplasia of A1 segment, frequently seen with AcomAAs. How these anomalies predispose to aneurysm formation remains unknown, but it has been proposed that anomalous arterial structure disrupts normal blood flow conditions causing turbulence, which increases arterial wall stress and thereby leads to aneurysm formation (8,10). There is no large prospective study in regard to these entities. On the other hand, the only series pointing out the incidence of the association of A1 hypoplasia/aplasia in the Turkish population was performed by Karazincir et.al. (5) and they found an incidence of 50%. Our study is the second one conducted in a small series consisting of a Turkish population.

MATERIALS AND METHODS

This study was performed retrospectively. Cerebral diagnostic DSA data between 2002 and 2004 from our institution were analysed. We excluded all factors that could lead to 'de novo' aneurysm formation. We didn't include patients who had a brain tumor, an arteriovenous malformation, an arteriovenous fistula or aneurysms other than AComAA. The patients' imaging data were evaluated by an experienced neuroradiologist. There are no clear criteria for A1 hipoplasia in the literature. We considered A1 hypoplastic if there was more than 50% difference in the diameter between both A1s. In some cases, vasospasm may have lead to misinterpretation of our results, so we ruled it out by the absence of vasospasm in neighboring arteries, constant A1 diameter from internal cerebral artery up to AComA, and by performing control angiography three months later.

RESULTS

A hundred and eighty-two patients fulfilled the criteria and were included into the study. The ages ranged between 3 and 89, with a mean of 51.89. Seventy-five patients had AComAA, with a mean age of 55.72. The total A1 hypoplasia/aplasia incidence was 41.33%. The most frequent anomalies were right A1 hypoplasia and aplasia which occurred in 12% of the patients, left A1 hypoplasia in 9.33 % and left A1 aplasia in 8% of the patients. One hundred and seven patients formed the control group, with a mean age of 49.23. The incidence of A1 hypoplasia/aplasia was 10.28%. The most frequent anomaly was right A1 hypoplasia 7.48% while right A1 aplasia, left A1 hypoplasia and left A1 aplasia were found at the same frequency of 0.93%. (Table I) (Figure 1)

DISCUSSION

Rupture of intracranial aneurysm followed by subarachnoid hemorrhage (SAH) is a serious disorder with high morbidity and mortality. Therefore, aneurysmal SAH represents a serious public health problem. Intracranial aneurysms are classified on the basis of their etiology (traumatic, infectious, or spontaneous) or morphological appearance (berry or fusiform). The pathophysiology of cerebral aneurysms remains unclear despite extensive studies but several possible mechanisms had been proposed. It was Forbus, who first recognized the gaps in tunica media of cerebral arteries and suggested that they represent sites of aneurysm origin. However, further research revealed that these gaps are rather intersections into the muscle layer and that the artery wall is actually stronger in these areas. Mechanical stress seems to play the main role in aneurysm formation (9). It has long been estimated that aneurysms develop at the sites of arterial branching where wall stress is maximal. Hoi et al. found that aneurysmal wall stress is proportional to length of

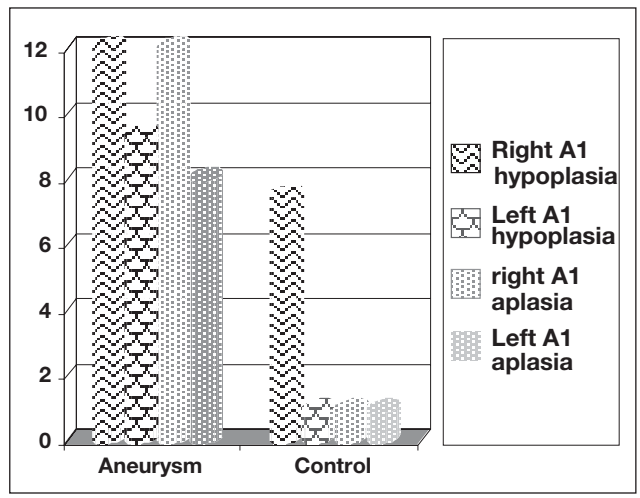

Figure 1: Graphic showing distribution of A1 hypoplasia/aplasia in AcommAA and control groups.

Table I: The Incidence of A1 Hypoplasia/Aplasia in the Present Study

	Number of patients	Mean age	A1 hypoplasia/ aplasia	Right A1 hypoplasia	Left A1 hypoplasia	Right A1 aplasia	Left A1 aplasia
Aneurysm group	75	55.72	41.33%	12 %	9.33%	12%	8%
Control group	107	49.23	10.28%	7.48%	0.93%	0.93%	0.93%

aneurysmal neck and reversibly proportional to third power of parent artery radius in their model (4). In this study, we analyzed the association, between AComAAs and hypoplasia/aplasia of the A1 segment. Yaşargil observed an 80% incidence of A1 hypoplasia in cases of AComAA (17). Wilson documented A1 hypoplasia in 85% of AComAAs in his autopsy study (16). Angiographic studies reveal a lower incidence. Karazincir found A1 hypoplasia in %50 (5) and Kasuya in 45.2% (6) of patients with an AComAA. However, in our study this incidence was determined as 41.33%.

The mechanical stress theory explains common sites of aneurysmal development especially in cases of disturbed flow conditions, but cannot fully explain all cases of aneurysm formation. Another possible mechanism of aneurysm development is remodeling of the vascular extracellular matrix. There is evidence that the aneurysm wall is a dynamic structure that renews rapidly. The most important angiogenetic factors are VEGF, NO, and MMP's (5). Higher levels of these factors in comparison to a normal artery wall were found in an aneurysmal wall. These factors, especially MMP's, lead to weakening of the aneurysmal wall, growth and subsequent rupture. This theory in association with the mechanical theory can explain most cases of aneurysm formation, especially if there is no disturbance of blood flow. In our study, we found that 58.5% of patients that have a AComAA do not have an underlying vascular pathology. This means that arterial wall remodeling is more important than mechanical factors, but we think that large prospective studies are necessary to prove this hypothesis.

As we mentioned above, there is a lower incidence of A1 hypoplasia/aplasia in our study in comparison with previous ones. We think that this difference is due to genetic features of the Turkish population and the A1 hypoplasia criteria, we employed. Compared with the control group, there is distinct evidence of A1 hypoplasia/aplasia in the aneurysm group, so we agree with other authors that such anomalies predispose to aneurysm formation.

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