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Case Report

Unilateral Anterior Cerebral Artery A1 Segment Aplasia Associated With Acoma Aneurysm

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Abstract

The anterior cerebral artery A1 segment aplasia is an anatomical variation of the Willis circle. It is very rare and mostly associated with the anterior communicating artery aneurysm, but this relationship has still not been thoughtfully explored. The hemodynamic factors are speculated to be the main pathophysiological mechanism of the aneurysm formation. The risk of the aneurysm rupture in the setting of A1 aplasia is not well elucidated. Endovascular treatment may have good outcome in short term. Long term studies are needed to evaluate the recurrence and recanalization rate. Here, we describe a case of unilateral anterior cerebral artery A1 segment aplasia associated with a ruptured anterior communicating artery aneurysm in which the endovascular embolization was successful performed without any complication.

Keywords: Anterior cerebral artery A1 Aplasia, Anterior cerebral artery hypoplasia, Anterior communicating artery aneurysm, subarachnoid hemorrhage, Endovascular embolization.

INTRODUCTION

The A1 aplasia is rare variant of Circle of Willis anatomy which constitutes 6% [1]. This variation is often associated with Acoma aneurysm. In the literature, the incidence of anterior cerebral artery A1 segment aplasia with Acoma aneurysm has been reported in about 2% of patient population [2]. Hypoplasia occurs more frequently harboring 1% to 21% of a healthy population, and according to the previous studies, it's prevalence in association with Acoma aneurysm range from 24% to 90% [2-4]. In both cases, the right-sided localization is more common and the reason for this right-sided predominance is not yet understood [2, 5].

The relationship between the A1 segment aplasia and the aneurysm formation growth, and risk factor of rupture is not yet well understood. The hemodynamic factors associated with wall vessel biology are speculated to be the main pathophysiological mechanism of the aneurysm genesis [5]. Furthermore, the relation of other known risk factors of intracranial aneurysm formation such as smoking, family history, hypertension is not well documented in the literature. Lorenzo Rinaldo et al. assessed the relationship of A1 hypoplasia with the other known risk factors and found no statistically significant difference [6]. Besides the factors related to aplasia for the aneurysm formation, Acoma is known as a predilection location of an intracranial aneurysm and accounts for 23-40% of the intracranial ruptured aneurysm [7].

Endovascular treatment is more safe and effective than microsurgical clipping, and it is the first choice of intracranial aneurysm treatment. However, it demonstrated that unilateral A1 aplasia is associated with a high likelihood of long term aneurysm recurrence (>12 months; chi=6.49, p=0.01) [8]. The real pathophysiological process of aneurysm recurrence is still not clear.

In this present study, we reported a case of aneurysmal subarachnoid hemorrhage of Acoma associated with A1 segment aplasia treated by endovascular coil embolization.

CASE DESCRIPTION

A 46-year-old male with a medical history of hypertension presented with the worst headache,

dizziness 5 hours after symptom onset. On examination, he was alert with no focal neurological deficits. A computed tomography (CT) scan of the brain showed a Fisher grade 3 subarachnoid hemorrhage (SAH) in the bilateral lateral fissure cistern, suprasellar cistern, annular cistern, anterior pontine cistern, tentorium cerebelli, and part of the sulcus (Fig 1A). A CTA was performed for further diagnosis. It revealed an aneurysm of the anterior communicating artery and aplasia of the right anterior cerebral artery A1segment (Fig. 2A). After multidisciplinary team discussion, it was decided to perform a Digital Subtraction Angiography, after which the endovascular coil embolization was indicated. The patient's relatives were counseled for the procedure, the risks associated with it were informed, and written consent was obtained for the same.

The procedure was done under general anesthesia. The patient was in supine position on the operating table. The Seldinger technique was used to have access to the right femoral artery. A 6F guide sheath and guidewire were used to respectively explore both the internal carotid artery and left vertebral artery. The angiogram of the right internal carotid artery showed the right anterior cerebral artery was absent, whereas the left internal carotid showed the left anterior cerebral artery has a double trunk of A2, between them on the anterior communicating artery arose a saccular aneurysm measured 4.5 mm in width and 8 mm in height (Figure 2B, & 2C). Other cerebral vessels were normal.

After the angiogram was done, the catheter was pulled out; and the 6F catheter was sent to the stable position of the C2 segment of the left internal carotid artery. The Echelon 10 micro-catheter and synchro14 micro guide-wire were molded. The micro-catheter was inserted into the aneurysm cavity along the 6F catheter. After the angiogram confirmed the position of the micro-catheter in the aneurysmal sac, 9 coils were successively inserted in the aneurysm cavity. Post-coiling angiogram showed Modified Raymond-Roy Class II occlusion of the aneurysm with a normal filling of all vessels (Figure 2D). The micro-catheter and guiding catheter were withdrawn. The 6 F catheter was removed; hemostasis was achieved by manual compression. Immediate post-procedure CT revealed no hematoma or infarction. The patient was sent to the Department of the Intensive Care Unit. There was no postoperative complication during the hospital stay, and the patient was discharged in a stable condition. A Digital Subtraction Angiography (DSA) for control was scheduled after 6 months.

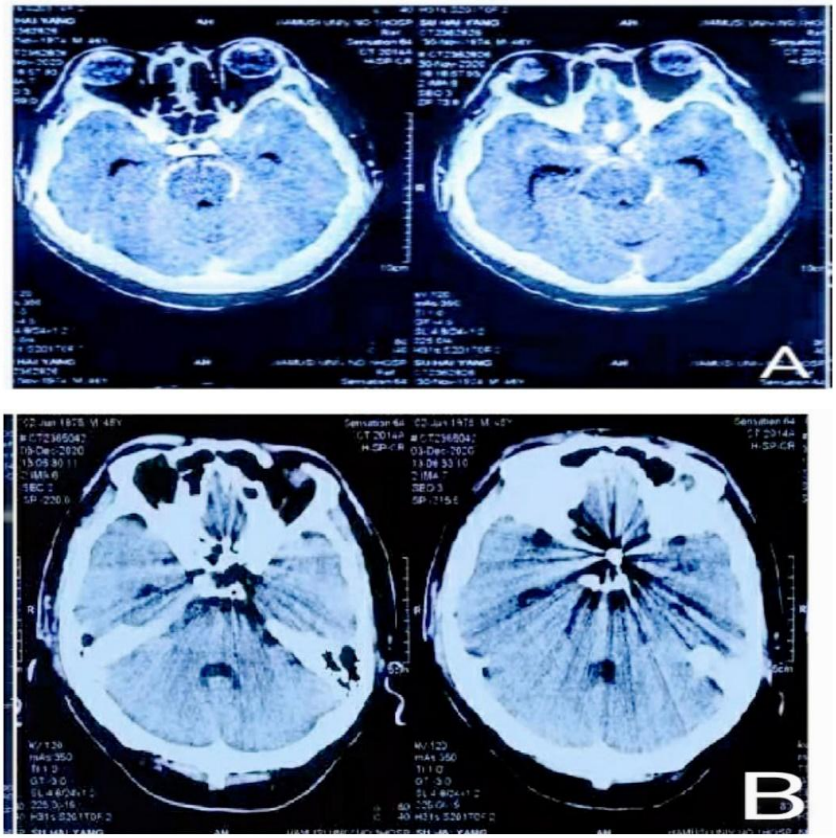

Figure-1: Computed tomography. (A) showed a high density in supra sellar, prepontine, ambient cistern which mean a subarachnoid hemorrhage. (B) post-procedural CT control appear no particularity

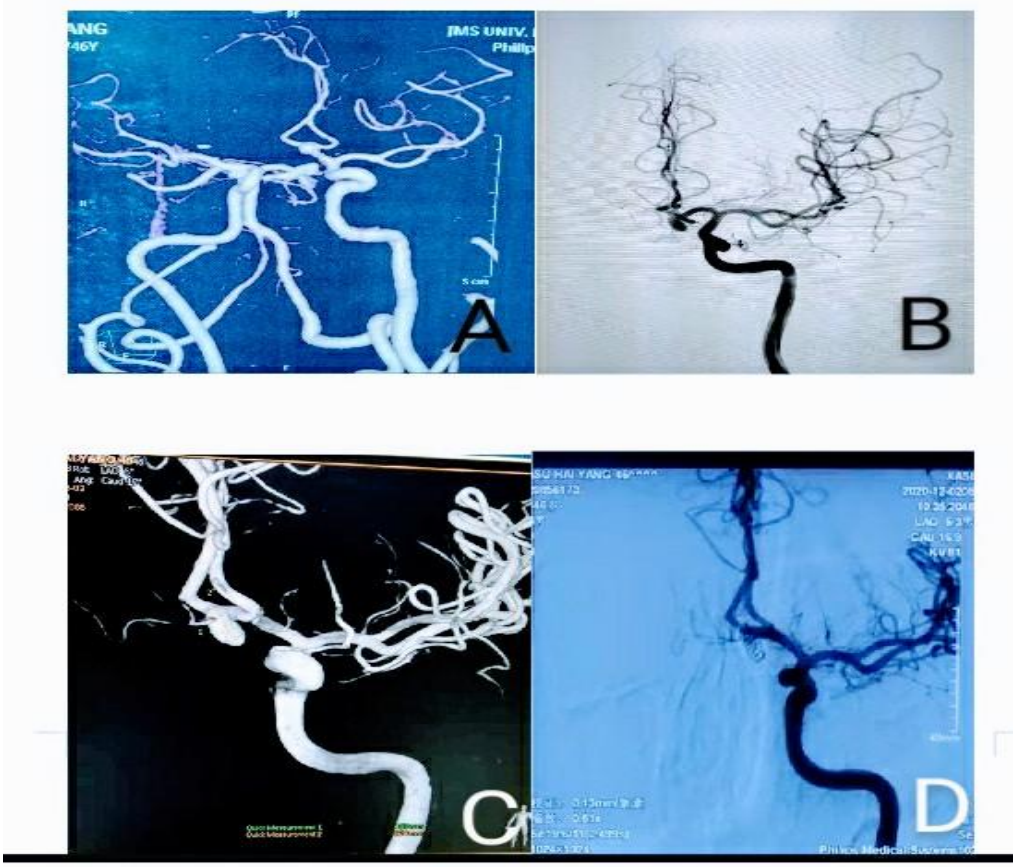

Figure-2: computed Tomography Angiography (A) showed an anterior communicating artery aneurysm and absence of right A1 segment of anterior cerebral artery. Digital subtracted Angiography (B, C) showed the Acoma aneurysm measured 4.5 mmx 8 mm. (D) post-coiling angiogram showed Modified Raymond-Roy Class II occlusion of the aneurysm

DISCUSSION

The A1 segment aplasia of the anterior cerebral artery is very sparsely. The early knowledge of the prevalence of this anatomical variation is derived from an autopsy report and angiogram [9]. It is known to be associated with the anterior communicating artery aneurysm. Kransy *et al*., found the prevalence to be 2% [2]. In the literature, hypoplasia was more reported, higher than occurs aplasia. Hao Chen *et al*., Reported 14.1% of the prevalence of Acoma aneurysm with A1 segment hypoplasia [5]. Fan Yang reported 49.8% [3]. These very fewer studies report on aplasia can be explained by the scarcity of this predisposition. In most reports, aplasia was considered as a grading scale of hypoplasia, therefore, many studies assess aplasia and hypoplasia together.

The A1 segment aplasia is located on the right side in our case. Many authors reported this right-sided predominance in comparison with the left side [2, 5, 8]. The main reasons for this right side preponderance disturbance are still unknown. Studies must be conducted to explain this right-sided predominance.

The main pathophysiological mechanism of the Acoma aneurysm formation in the setting of A1 segment aplasia has not been well elucidated. It's thought to be related to the hemodynamic factors. A1 aplasia may increase the blood flow across the Acoma, therefore may induce aneurysm formation. The flow impingement on the apex of bifurcations of vessels are involved in the aneurysm initiation by causing a morphological and functional change on the vessel wall. The wall shear stress on the impingement point is high and related to the aneurysm formation [10-13]. These hemodynamic factors may influence the process of wall remodeling by affecting the apoptosis of smooth muscle cells, the inflammatory response of endothelium, and enzyme secretion [10]. Studies assessing especially the relationship of A1 segment aplasia and the known risk factors of the aneurysm were not found in the literature. smoking, hypertension and family history are factors which have been studied to contribute to the aneurysm formation in case of A1 hypoplasia [6]. From this report, we conclude that the A1 aplasia and known risk factors of aneurysm seem to be an independent risk of aneurysm formation. We think also the existence of both may increase the aneurysm occurrence rate, but that must be confirmed in future studies.

The main cause of nontraumatic subarachnoid hemorrhage is a rupture of an intracranial aneurysm,

and it's known to be related to a high mortality and morbidity rate. Acoma aneurysms represent 23-43% of ruptured intracranial aneurysms [7, 14]. The A1 aplasia is speculated to lead to hemodynamic disturbance which is already known as a risk factor of aneurysm rupture. Despite the hemodynamic factor, the aneurysm size and hypertension are known for its high risk of aneurysm rupture [7, 14]. In our case, the patient has a history of hypertension and the aneurysm measured 4.5 mm in width and 8 mm in height. This result confirmed the report of the previous study. It still remains unknown, the real risk of rupture of Acoma aneurysm in the setting of A1aplasia.

The endovascular embolization shows a good outcome and is the first choice of aneurysm treatment [8]. In our case, endovascular embolization was successful performed without any procedural or peri-procedural complication, and this during all the patient hospital stay. Studies are needed to assess the risk of recanalization and recurrence of Acoma aneurysm in the sitting of A1 aplasia.

CONCLUSIONS

The association of anterior cerebral artery A1 segment aplasia and Acoma aneurysm is very rare and still not well understood. More Studies are needed in the future to attain insight into the pathophysiological mechanism, the risk of rupture, and the recurrence of Acoma in the setting of A1 aplasia.

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