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Case Report

Ruptured de novo posterior communicating artery aneurysm associated with arteriosclerotic stenosis of the internal carotid artery at the supraclinoid portion

Abenamar Sámano, Tatsuya Ishikawa, Junta Moroi, Shingo Yamashita, Akifumi Suzuki, Nobuyuki Yasui

Department of Surgical Neurology, Research Institute for Brain and Blood Vessels Akita, Akita City, Japan

E-mail: *Abenamar Sámano - superabenamar@hotmail.com; Tatsuya Ishikawa - teddyish@akita-noken.jp; Junta Moroi - moroi@akita-noken.jp; Shingo Yamashita - yamashita@akita-noken.jp; Akifumi Suzuki - akifumi@akita-noken.go.jp; Nobuyuki Yasui - yasui@akita-noken.go.jp
*Corresponding author

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Abstract

Background: Several de novo intracranial aneurysms have been described related to changes in hemodynamics after therapeutic occlusion of internal carotid artery (ICA); however, de novo aneurysms related to a supraclinoid arteriosclerotic stenosis of the ICA have not been described yet. Authors consider that it is important to bear in mind the possibility of developing an aneurysm in these special conditions.

Case Description: The evolution of a 62-year-old patient with subarachnoid hemorrhage, intraparenchymal frontal hematoma with some atypical circumstances that were presented together as well as the treatment he received are shown in this report. We can see this patient suffered a right thalamic hemorrhage at the age of 51 years; this condition was associated to a severe atherosclerotic stenosis of right supraclinoid ICAy. A long term had elapsed since the diagnosis of the stenosis and the discovery of a ruptured ipsilateral de novo supraclinoid internal carotid artery-posterior communicating artery (ICA-PcomA) aneurysm.

Conclusions: It seems like both conditions: the atherosclerotic supraclinoid ICA which tells of an Samano *et at*: Ruptured De Novo PcomA Aneurysm Associated with Arteriosclerotic Stenosis of Supraclinoid ICA. Altered vessel environment coupled to a long exposure time, hemodynamic changes, unbalance in the wall sheer stress could all of them lead to the development of the de novo aneurysm.

Key Words: Aneurysm, atherosclerosis, de novo, intracranial carotid artery, stenosis, subarachnoid hemorrhage

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INTRODUCTION

Aneurysms arising at the bifurcation of the posterior communicating artery (PcomA) from the internal carotid artery (ICA) are very common, comprising a quarter of all aneurysms. Variations of the circle of Willis are well known and could have a primordial role in the development of berry aneurysms associated with other risk factors such as genetics, changes in wall shear stress, smoking, hypertension and sex.[4]

Hemodynamic changes have been considered as one of the major reasons for de novo aneurysms.[2] Agenesis or hypoplasia of the ICA as well as therapeutic ICA occlusion are known to promote hemodynamic effects and subsequent aneurysm formation.[1] However, our investigations have identified few patients in whom arteriosclerotic ICA occlusion has been considered related to de novo aneurysm development.[5] The main purpose of this report is to describe the case of a patient with ruptured de novo ICA-PcomA aneurysm that was discovered just adjacent to the stenotic segment due to atherosclerosis already identified 11 years ago. We also discuss the possible factors for de-novo aneurysm formation.

CASE DESCRIPTION

A 62-year-old man experienced sudden onset of severe headache, dizziness and vomiting and visited our hospital 2 hours after onset. Neurological examination revealed disturbed consciousness (Glasgow coma scale 9) with hemiparesis of the left side. He underwent CT, demonstrating subarachnoid hemorrhage (SAH) in the basal cisterns as well as a significant amount of hematoma in the right sylvian fissure and frontal lobe [Figure 1]. The patient had a history of hypertensive right Samano *et at*: Ruptured De Novo PcomA Aneurysm Associated with Arteriosclerotic Stenosis of Supraclinoid ICA. Thalamic hematoma 11 years earlier, and we discovered that head CT [Figure 1] and cerebral angiography had been performed. Cerebral angiography showed a severely stenotic supraclinoid segment of the ICA on the right side [Figure 2]. Stenosis of the ICA was asymptomatic, and the distal segment of the stenotic ICA has been perfused by collateral blood flow through the PcomA, while the right A1 segment appeared hypoplasic and both A2 segments were filled from the left ICA [Figure 2]. However, no evidence of any aneurysms was identified in those angiographic studies. He was hospitalized for 60 days in 1999 and discharged with sequelae of slight left-sided hemiparesis.

We performed 3-dimensional CT angiography (3D-CTA), revealing the known stenotic supraclinoid segment of the ICA as well as the presence of a berry aneurysm in the right ICA-PcomA just after the stenotic segment of the ICA [Figure 3]. Given the poor condition of the patient and the imaging findings, he underwent wide craniotomy and clipping for the PcomA aneurysm and evacuation of the intraparenchymal hematoma. The ICA proximal to the aneurysm appeared to be stenotic due to deep arteriosclerotic changes on visual inspection [Figure 4]. The patient recovered well from hemiparesis, and received a ventriculo-peritoneal shunt for hydrocephalus. He was discharged and returned home in an independent state, although slight left-sided hemiparesis and dysarthria remain as sequelae.

DISCUSSION

Considering that the prevalence of asymmetrical Willis' ring could be as high as 64% in general population, it's supposed that only around the 40% of the total population would present an "ideal" Willis' circle. This asymmetrical condition in the vessels has been described as a risk factor in the aneurysms development.[2,7]

It is well known that changes in the hemodynamic flow of ICA caused by hypoplasia or occlusive treatments

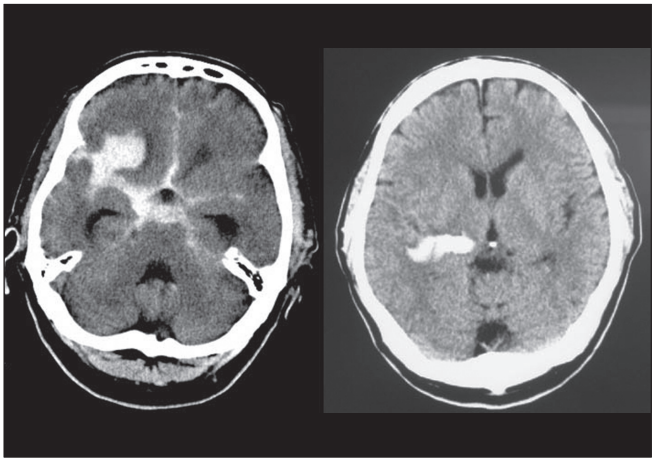

Figure 1: Left: CT 2 hours after onset of symptoms. A large collection of blood in the subarachnoid space and intraparenchymal hematoma can be seen. Right: CT taken in 1999 following right thalamic hemorrhage, it was the reason for the first hospitalization

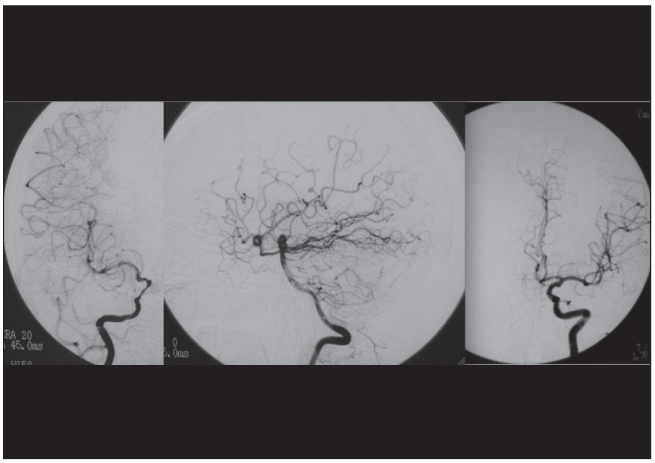

Figure 2: Anteroposterior view of right ICA angiography (left), lateral view of right vertebral angiography (center) and anterioposterior view of left ICA angiography (right) performed in 1999. The severely stenotic segment of right supraclinoid ICA is clearly apparent (left), along with a well-developed collateral circulation through PcomA (center). The right A1 segment remained hypoplasic while both A2 segments are filled with the flow that comes from left ICA (right)

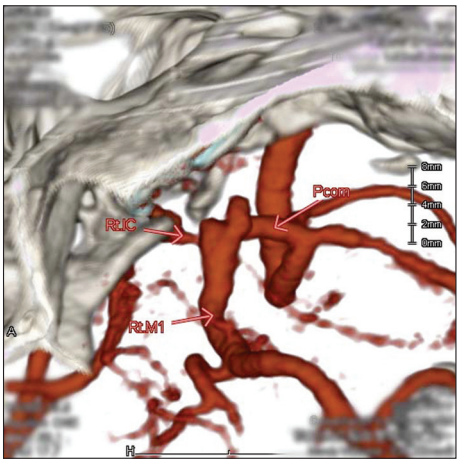

Figure 3: Colored 3D-CTA showing the clearly stenotic segment just beside the right anterior clinoid process, along with de novo ICA aneurysm at the origin of the PcomA. Rt.IC, right internal carotid artery; Rt.M1, middle cerebral artery; Pcom, posterior communicating artery

Figure 4: Left: pre-clipping picture. Atherosclerotic ICA (black-arrow), neck of aneurysm (yellow-arrow). The PcomA (white-arrow) and the anterior choroidal artery (green-arrow) are seen. Center: Schematic drawing of the surgical field. Right: post-clipping photograph. A well-developed PcomA is coming into the ICA

Figure 5: Upper: Typical flow coming from ICA (solid arrow) to PcomA (thin arrow) in the usual anatomical way. Bottom: in this patient the main flow in coming from the PcomA (solid arrow) to ICA territory and a weak flow is coming from ICA through the stenotic segment (thin arrow). The collision point of both coming flows could produce a turbulence effect that insults the walls of ICA

when the whole ICA must be sacrificed could be directly related to intracranial aneurysms.[1,2] These hemodynamic aneurysms are often found in the anterior communicating artery or in the contralateral ICA. This report shows a case of a right ICA-PcomA de novo aneurysm 11 years after the diagnosis of an ipsilateral arteriosclerotic stenosis of supraclinoid ICA. As far as we investigated we could only find one case report illustrating a de novo aneurysm related to an ipsilateral occluded ICA. In this report Ogasawara *et al*.[8] described in 1995 a case of a 58-year-old female patient whom developed a ruptured true PcomA aneurysm 13 years after surgical occlusion of the ipsilateral ICA. She presented SAH as a consequence of a ruptured intracranial ICA aneurysm 13 years before and received a right superficial temporal artery-middle cerebral artery anastomosis and surgical occlusion of the right cervical ICA. In their report, hemodynamic factors played a dominant role to grow the true PcomA aneurysm.

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These hemodynamics changes have been extensively studied lately with the computational fluid dynamics modeling systems and it has been found that in the supraclinoid ICA and in the infundibulae portion of PcomA the presence of regions of low wall shear stress (WSS) surrounded by areas of high WSS play a preponderant roll in the rise, growth and rupture of PcomA aneurysms.[3,7]

In our patient, we theorize that in the first stage the ICA's lumen was narrowed by the atherosclerotic plates, provoking the weakening of its flow giving rise to a stronger reversal flow coming from PcomA to supraclinoid ICA and the collision of both unpaired flows [Figure 5] caused a turbulent effect that could produce large variations in the WSS that led to the formation of de novo aneurysm. In addition we suppose that arteriosclerotic degeneration of the artery, such as breakdown of the elastic fibers, may have been partially related to aneurysm formation.[6]

CONCLUSIONS

In conclusion, we would like to stress that when a patient with arteriosclerotic ICA occlusion and good collateral

blood flow through the circle of Willis is young, as in our patient, the possibility of de novo aneurysm should be considered. Close, careful, multidisciplinary follow-up is necessary for such patients.

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