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ANGIOGRAPHIC MOYAMOYA IN CONSEQUENCE OF SECONDARY STENT OCCLUSION AFTER STENT-ASSISTED COILING OF THE ANEURYSM

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Abstract

Stent-assisted coil embolization is a widely accepted endovascular treatment of wide-neck aneurysms or when additional support is needed to keep the coils within the aneurysm sac. However, in-stent thrombosis and subsequent ischemic brain injury remains the major complication of these procedures in spite of aggressive antiplatelet therapy.

The study presents a case of a patient who underwent stent-assisted coiling of a saccular right middle cerebral artery aneurysm. Immediately after stent placement the patient was assigned to standard treatment with 5000 U of intravenous heparin, 600 mg of aspirin and 300 mg of clopidogrel. Thereafter, he was prescribed Plavix (clopidogrel) of 75 mg twice daily for 3 months and aspirin (acetylsalicylic acid) 300 mg daily lifelong. However, he discontinued medications 3 months after initial procedure.

Follow-up diagnostic cerebral angiography revealed complete occlusion of right middle cerebral artery at the proximal end of the stent, as well as blood filling of right distal middle cerebral artery branches from proximal vessels through newly developed moyamoya-like collateral vessels.

After reinitiating the dual antiplatelet therapy a six-month follow-up angiography demonstrated recanalization of the stent with involution of collateral vessels.

Moyamoya syndrome or angiographic moyamoya is known to be a compensatory formation of collateral vascular network in the base of the brain in response to the influence of various factors such as systemic disorders, radiation or other conditions. However, according to our data, this is the first case report of the development of angiographic moyamoya after intracranial stent thrombosis. We propose to include intracranial stent thrombosis as a possible rare cause of angiographic moyamoya along with other causes.

Keywords: *angiographic moyamoya, stent thrombosis, stent-assisted coiling, aneurysm.*

Introduction

Classical moyamoya disease is a progressive spontaneous bilateral stenotic occlusion of intracranial internal carotid artery and its proximal branches with compensatory formation of characteristic collateral vascular network in the base of the brain. It was first described in 1957 as “hypoplasia of the bilateral internal carotid arteries” [*Scott R, Smith E, 2009*]. Later International Classification of Diseases recognized “moyamoya” as the specific name for this condition [*Fukui M, 1997; Scott R, Smith E, 2009*].

Conditions with the characteristic moyamoya vasculopathy without associated risk factors are referred to “moyamoya disease”, whereas patients who also have well recognized associated conditions are categorized as having the “moyamoya syndrome” [*Scott R, Smith E, 2009*]. Terms “rui-moyamoya disease” in Japanese and “quasi-moyamoya disease” in English (synonym of “moyamoya syndrome” or “akin to moyamoya disease”) have also been used to describe these conditions [*Natori Y et al., 1997*]. Initially in 1986 T. Watanabe and N. Su-

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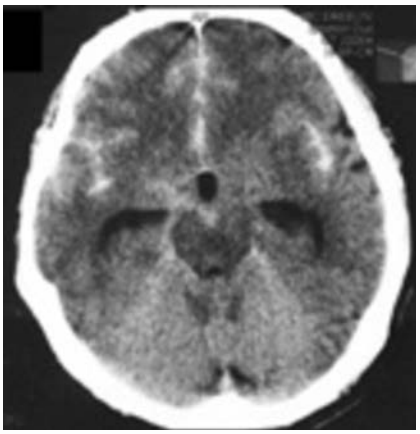

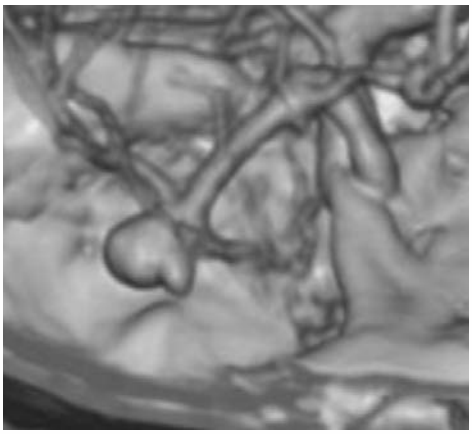

Figure 1. *Computed tomography of the patient revealed:* ***a)*** *massive subarachnoidal hemorrhage and hydrocephalus,* ***b)*** *saccular aneurysm of right middle cerebral artery*

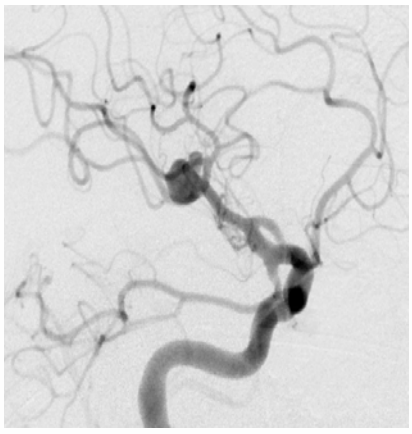

Figure 2. *Selective angiography of right internal carotid artery demonstrates saccular aneurysm of right middle cerebral artery trifurcation*

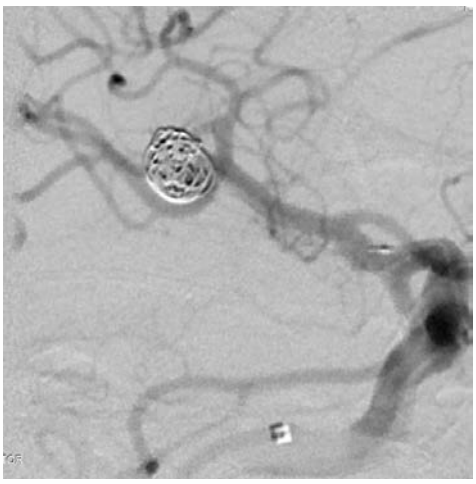

Figure 3. *Diagnostic cerebral angiography of right middle cerebral artery – obliteration of the aneurysm before coil detachment*

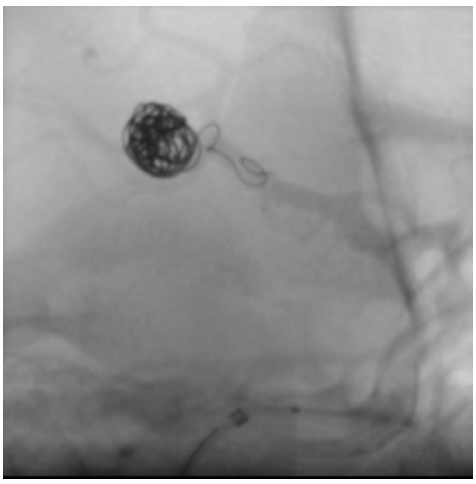

Figure 4. *Diagnostic cerebral angiography of right middle cerebral artery shows that some of the coil loops shifted into the middle cerebral artery lumen after coil detachment and microcatheter retrieval without digital subtraction*

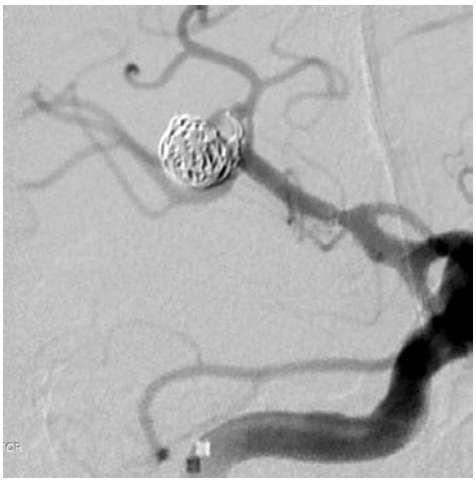

Figure 5. *Diagnostic cerebral angiography of right middle cerebral artery shows coils outside the lumen of artery by Neuroform stent with digital subtraction*

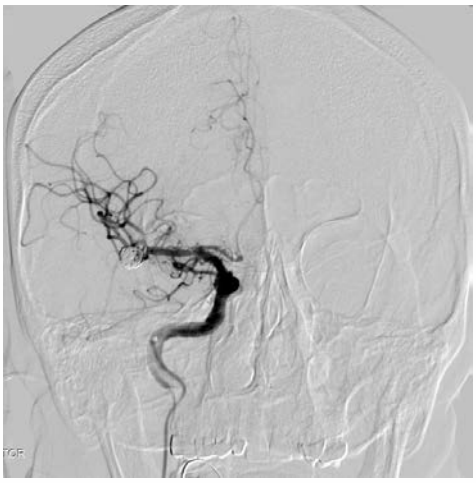

Figure 6. *Final selective angiographies of right internal carotid artery revealed sufficient flow of contrast medium through major middle cerebral artery branches*

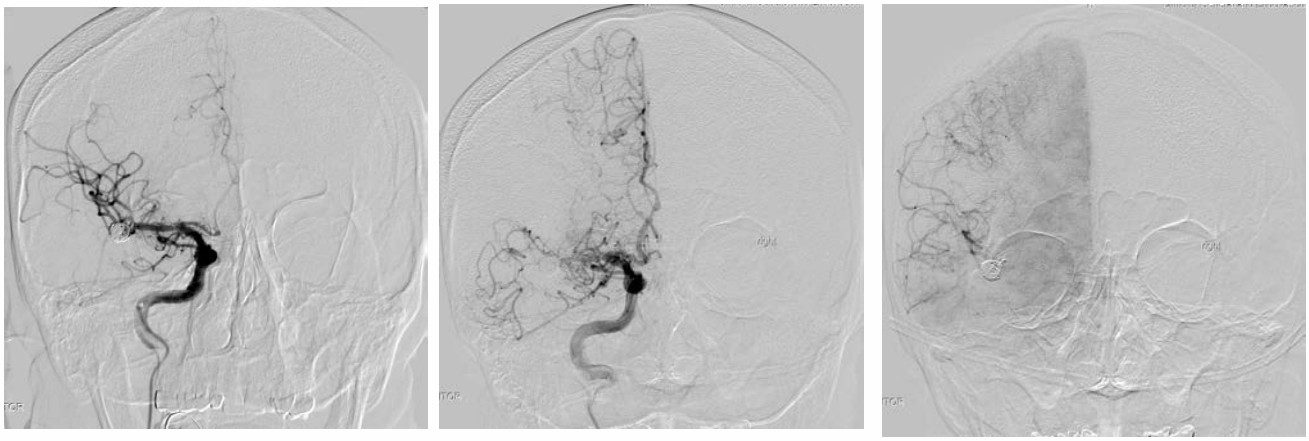

FIGURE 7. *Right internal carotid angiography:* ***A)*** *Early arterial phase. Normal anterograde flow in the left internal carotid artery and its branches. Absence of filling of M2 segment of right middle cerebral artery as well as development of moyamoya-like vascular network at the right side.* ***B)*** *Late arterial phase. Absence of filling of M2 segment of right middle cerebral artery, development of moyamoya-like vascular network at the right side.* ***C)*** *Capillary phase. Filling of right middle cerebral artery from moyamoya and cortical collaterals*

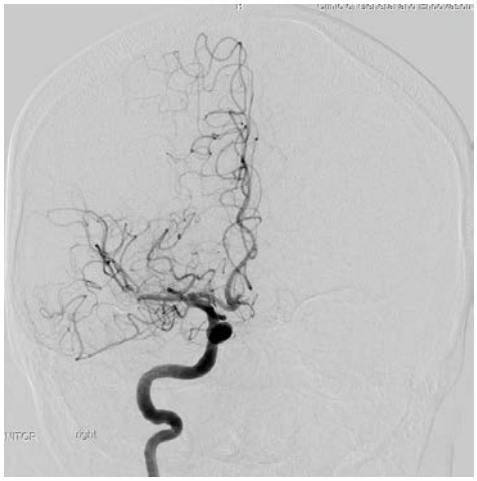

FIGURE 8. *Six-month follow-up angiography showed recanalization of M2 segment*

zuki proposed "rui- or quasi-moyamoya disease", which includes: 1) unilateral involvement, 2) stenosis or occlusion of the proximal portion of middle cerebral artery with development of Moyamoya vessels, 3) association of vascular malformations, 4) known causes, and 5) other causes [*Watanabe T, Suzuki N, 1986*]. Natori Y. and co-authors proposed that patients demonstrating both stenotic changes in the terminal portion of internal carotid artery and the formation of moyamoya vessels in association with some other disorders should be classified as "angiographic moyamoya" [*Natori Y et al., 1997*].

Conditions associated or coexisted with angiographic moyamoya have been widely reported in the literature. They can be either congenital or acquired systemic or isolated disorders, as well as caused by trauma, radiation or other impacts [*Natori Y et al., 1997; Scott R, Smith E, 2009*].

The true etiology for the development of angiographic moyamoya is still not understood [*Houkin K et al., 2012*]. Genetic factors appear to play a major role in moyamoya development, and underlying defects in regulation of specific extracellular proteins have effects on cerebral vessels in susceptible persons, resulting in moyamoya phenotype when particular environmental triggers are present [*Scott R, Smith E, 2009*].

The study presents a case of angiographic moyamoya, developed as a result of intracranial stent thrombosis and proposes to include this condition as another causative event for angiographic moyamoya.

CASE REPORT

A 45-year-old patient was admitted to the clinic of General and Endovascular Neurosurgery of Yerevan State Medical University with the diagnosis of ruptured saccular aneurysm of right middle cerebral artery territory and prior subarachnoid hemorrhage of I grade according to Hunt-Hess classification [*Hunt W, Hess R, 1968*]. Initial computed tomography (Fig. 1) and cerebral angiography (Fig. 2) confirmed the presence of a bilobed aneurysm at the right middle cerebral artery trifurcation area as a source of subarachnoid hemorrhage. The aneurysm was obliterated with Axium (EV3, Covidien, USA) and GDC Matrix (Boston Scientific, USA) coils using standard endovascular technique (Fig. 3).

The procedure was complicated by shifting of the last coil loop into the lumen of the parent vessel while microcatheter was withdrawn (Fig. 4). A decision was made to place Neuroform (Boston Scien-

tific, USA) 2.5×15 *mm* stent with the aim to trap the coil loop both in the aneurysmal sac and afferent vessel (Fig. 5). Immediately after stent placement 5000 *U* of intravenous heparin, 600 *mg* of aspirin and 300 *mg* of clopidogrel were administered through nasogastric tube. The stent was placed without complications. Final selective angiography revealed sufficient flow of contrast medium through major middle cerebral artery branches (Fig. 6) and obliteration of about 90% of aneurysmal sac without vessel dropout (Fig. 5). After uneventful hospital course the patient was discharged without neurological deficit. He was prescribed Plavix (clopidogrel) 75 *mg* twice daily for 3 months and aspirin (acetylsalicylic acid) 300 *mg* daily lifelong.

Follow-up diagnostic cerebral angiography was scheduled after 3 months. However, the patient was lost to follow-up and was admitted to our department 14 months later. Moreover, the patient was not compline with the postoperative antiplatelet therapy and apparently discontinued medication 3 months after initial procedure.

During the re-admission to the hospital neurologic examination revealed minimal left sided hemiparesis, other physical and laboratory findings where normal. Diagnostic cerebral angiography revealed complete occlusion of right middle cerebral artery at the proximal end of the stent, complete obliteration of the aneurysm, as well as filling of right distal middle cerebral artery branches from proximal vessels through newly developed moyamoya-like collateral vessels (Fig. 7). No complications where observed during and after the procedure. The patient was discharged without changes in general and neurological conditions.

After reinitiating the dual antiplatelet therapy a six-month follow-up angiography demonstrated recanalization of the stent with the involution of collaterals (Fig. 8).

It is known that thromboembolic complications after stent-assisted coiling usually result in various degrees of ischemic brain injury, which leads to mild or severe deficit or even death. However, the compensatory changes can rarely lead to the formation of moyamoya-like vascular collateral network with the establishment of sufficient blood supply to the middle cerebral artery region, probably with existed genetic predisposition to the development of moyamoya. As far as we know this is the first case report of the development of angiographic moyamoya after intracranial stent thrombosis. We propose to include intracranial stent thrombosis as a possible rare cause of angiographic moyamoya along with other causes.

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