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AJNR Am J Neuroradiol 2002, 23 (9) 1577-1579
http://www.ajnr.org/content/23/9/1577

AJNR Am J Neuroradiol 23:1577–1579, October 2002

Case Report

Rapid Enlargement of a Posterior Communicating Artery Aneurysm after Guglielmi Detachable Coil Treatment of Ipsilateral Carotid Artery Aneurysms

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Summary: This case illustrates rapid aneurysm enlargement, presumably due to altered hemodynamics resulting from endovascular treatment of aneurysms on the same artery. We postulate that increased hemodynamic force directed to the inflow zone of the posterior communicating artery aneurysm was caused by the treatment of the two ophthalmic artery aneurysms. Originally, many of the flow vectors may have been directed into the larger ophthalmic segment aneurysm, located on the outside of the curve of the internal carotid artery. After treatment, flow may have been directed more smoothly around the carotid siphon and into the posterior communicating artery aneurysm.

The growth and rupture of saccular aneurysms involves hemodynamic, humoral, and structural factors (1–5). Changes in hemodynamic factors alone have been implicated in several instances of aneurysm growth or rupture. Many cases of de novo aneurysms and rapid aneurysm enlargement have been reported to occur after carotid ligation (6). We herein report the case of a patient with rapid enlargement of a posterior communicating artery aneurysm after embolization of two ipsilateral ophthalmic segment aneurysms with Guglielmi detachable coils (GDC; Boston Scientific Corporation/Target Therapeutics, Nattick, MA). We attribute the enlargement to changes in flow dynamics.

Case Report

A 45-year-old woman with chronic headaches presented after a fall. She had a long history of smoking with no other relevant medical history. She had no known familial history of aneurysms. Her CT and MR imaging studies of the head suggested multiple cerebral aneurysms. Diagnostic cerebral angiography performed at an outside hospital revealed multiple bilateral internal carotid artery aneurysms. Five aneurysms were present on the left internal carotid artery: a 12-mm cavernous, a 9-mm ophthalmic, a 3-mm ophthalmic, a 4-mm posterior communicating, and a 2-mm anterior choroidal artery aneurysm (Fig 1A and B). Three aneurysms were found on the right internal carotid artery carotid: a 3-mm cavernous segment, a 7-mm carotid cave, and a 2-mm anterior choroidal aneurysm. No aneurysms were found in the posterior circulation.

Endovascular treatment was considered as a treatment option; however, the close proximity of the multiple aneurysms along the internal carotid artery presented a possible obstacle to clip placement. After discussion with the patient and her family, we proceeded with endovascular treatment. We planned to treat the left ophthalmic artery and posterior communicating artery aneurysms.

The patient received clopidogrel bisulfate (75 mg administered orally every day) for 3 days before the procedure. The procedure was performed with the patient under general endotracheal anesthesia. Heparin was IV administered during the procedure to maintain a target activated clotting time value of 300 s. A Prowler 14 microcatheter (Cordis, Johson & Johnson, Miami, FL) with a Transend microguidewire (Meditech, Boston Scientific) was advanced through a 6F Envoy guide catheter under biplane road mapping. We initially attempted to catheterize the posterior communicating artery aneurysm, but it was difficult to control the catheter and guidewire mainly because of catheter looping in the cavernous aneurysm. We therefore proceeded to catheterize the larger, more distal of the left ophthalmic segment aneurysms. This aneurysm was successfully treated with GDCs. The smaller, more proximal of the two ophthalmic aneurysms was then accessed and also obliterated with GDCs. We considered treating the posterior communicating artery aneurysm at that point but elected to wait for the healing of the ophthalmic aneurysms to avoid any potential disturbance of the coils that were already in place.

One hundred forty days after initial GDC treatment, the patient returned for elective treatment of her unruptured left posterior communicating artery aneurysm and the largest right internal carotid artery aneurysm. Angiography of the left internal carotid artery showed marked interval enlargement of the left posterior communicating artery aneurysm (Fig 2). It measured 6×10 mm compared with approximately 3×4 mm on an angiogram obtained during the patient's initial treatment approximately 4.5 months earlier. The other aneurysms remained unchanged in appearance. The enlarged left posterior communicating artery aneurysm was successfully treated with GDCs with a steam shaped Prowler 14 through a 5F Envoy guide catheter. The contralateral carotid cave aneurysm was also treated. The patient tolerated this treatment without incident and was discharged in normal condition.

Discussion

Flow dynamics have been proved as important factors in aneurysm growth and rupture. Hemodynamic stress has been suggested as a necessary element in saccular aneurysm formation (2, 5). Flow dynamics in cerebral aneurysms have been studied in experimental models and with computer models. Flow patterns are complex and vary with the location and cardiac cycle. Cerebral aneurysms may typically have an inflow zone, a slow flow vortex zone, and an outflow zone (1, 7). Different flow hemodynamics exist in small and large aneurysms (8). Flow models in replicas of two ruptured posterior communicating artery aneurysms showed inflow at the

Received April 3, 2002; accepted after revision April 23.

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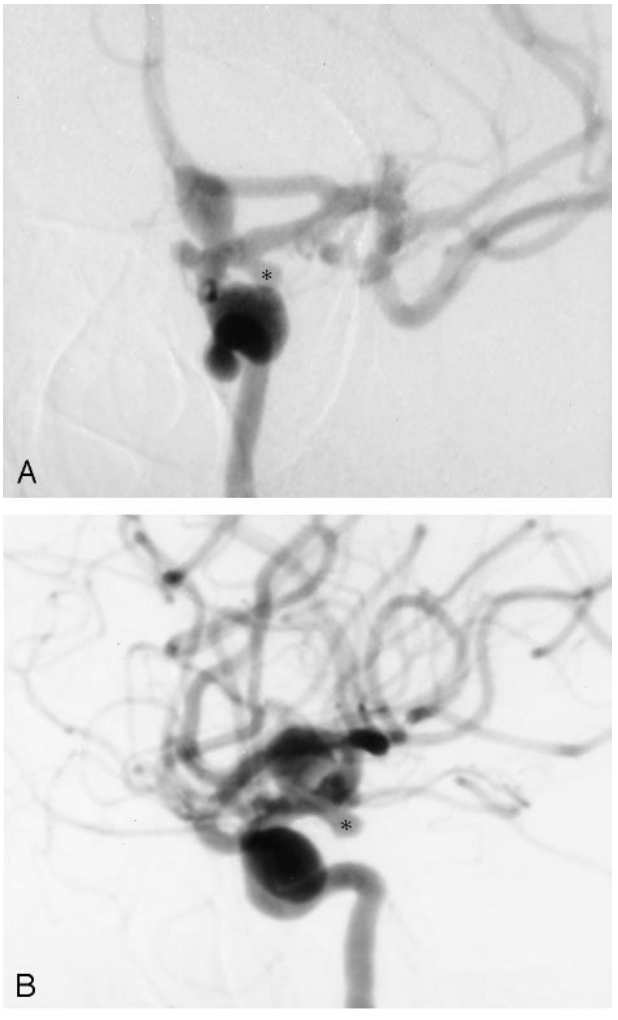

FIG 1. Angiograms from the case of a 45-year-old woman with chronic headaches who presented after experiencing a fall. Five aneurysms were present on the left internal carotid artery.

A, Anteroposterior view angiogram of the left internal carotid artery shows multiple aneurysms. These include a large cavernous carotid aneurysm, a small ophthalmic segment aneurysm, a large ophthalmic segment aneurysm, and a posterior communicating artery aneurysm (*asterisk*). A tiny anterior choroidal aneurysm is also present.

B, Lateral view angiogram of the left internal carotid artery. *Astserisk* indicates posterior communicating artery aneurysm.

distal neck, reverse vortical flow at the dome, and outflow at the proximal neck. The impact zone of the entering slipstream correlated with the point of rupture (9). Altering flow hemodynamics in a model replica of a fatal anterior communicating artery aneurysm affected the entrance of slipstreams into the aneurysm. Asymmetrical flow caused the entering slipstream to enter the aneurysm neck and impinge on the dome. Altered flow hemodynamics in this model also had implications regarding the success of MR imaging to identify the aneurysm (10). Numerous reports of de novo aneurysms after carotid ligation support these experimental findings and emphasize the importance of hemodynamics and asymmetrical flow in aneurysm formation (6). Enlargement of aneurysm remnants after surgical or endovascular treatment is also likely related to hemodynamic forces at residual or altered inflow zones (11, 12).

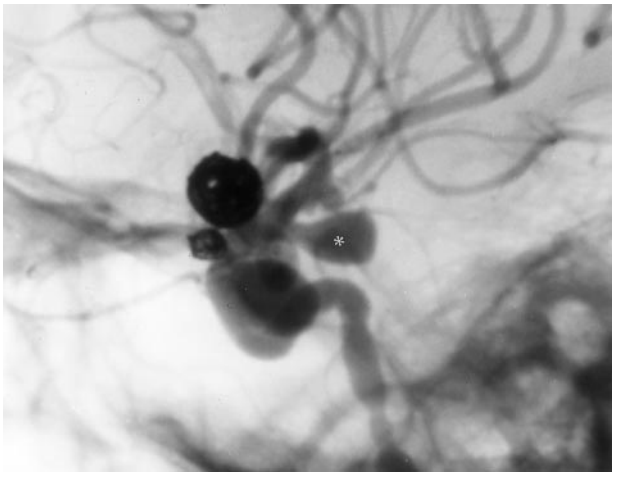

FIG 2. Four months after GDC treatment of the two ophthalmic segment aneurysms, lateral view angiogram of the left carotid artery shows marked interval enlargement of the posterior communicating artery aneurysm (*asterisk*, *compare with Fig 1B*). The treated ophthalmic aneurysms remain completely obliterated.

Structural and humoral factors are also important factors in aneurysm formation and growth. Structural factors, in particular the disruption of the internal elastic lamina, are important and possibly necessary conditions in aneurysm genesis (13). Structural considerations also may impact treatment, with apposition of the internal elastic lamina at the neck possibly being an important factor. In this patient with multiple aneurysms, structural factors may have facilitated the rapid aneurysm enlargement. Multiple aneurysms occur in approximately one-fifth to one-third of the population. Cigarette smoking and female sex are two factors associated with increased risk for multiple aneurysms, both of which were present in this case (14, 15). Also, the large number of aneurysms in this patient suggests a possible underlying structural abnormality, or weakness, in the cerebral blood vessels. Underlying structural weakness may have exaggerated the effect of the hemodynamic change.

Conclusion

To our knowledge, this is the first report of rapid aneurysm enlargement due to altered flow dynamics from endosaccular obliteration of proximal aneurysms. This report emphasizes the importance of hemodynamic factors in aneurysm growth and the need for the neurointerventionalist to consider the possible hemodynamic effects of endovascular intervention on the growth or development of other, untreated, aneurysms. Diagnostic modalities, such as flow-sensitive MR imaging, may assist in these determinations in the future (Feinberg et al, submitted for publication).

References

1. Gonzalez CF, Cho YI, Ortega HV, Moret J. **Intracranial aneurysms: flow analysis of their origin and progression.** *AJNR Am J Neuroradiol* 1992;13:181–188
2. Strother CM. **Understanding the natural history of saccular aneurysms.** *AJNR Am J Neuroradiol* 1998;19:1183–1184
3. Ferguson GG. **Physical factors in the initiation, growth, and rup-**

ture of human intracranial saccular aneurysms. *J Neurosurg* 1972; 37:666–677
4. Misra BK, Whittle IR, Steers AJ, Sellar RJ. **De novo saccular aneurysms.** *Neurosurgery* 1988;23:10–15
5. Stehbens WE. **Etiology of intracranial berry aneurysms.** *J Neurosurg* 1989;70:823–831
6. Maiuri F, Spaziante R, Iaconetta G, Signorelli F, Cirillo S, Di Salle F. **"De novo" aneurysm formation: report of two cases.** *Clin Neurol Neurosurg* 1995;97:233–238
7. Ostergaard JR, Hog E. Incidence of multiple intracranial aneurysms: influence of arterial hypertension and gender. *J Neurosurg* 1985;63:49–55
8. Tenjin H, Asakura F, Nakahara Y, et al. **Evaluation of intraaneurysmal blood velocity by time-density curve analysis and digital subtraction angiography.** *AJNR Am J Neuroradiol* 1998;19:1303–1307
9. Imbesi SG, Kerber CW. **Analysis of slipstream flow in two ruptured intracranial cerebral aneurysms.** *AJNR Am J Neuroradiol* 1999;20: 1703–1705
10. Kerber CW, Imbesi SG, Knox K. **Flow dynamics in a lethal anterior communicating artery aneurysm.** *AJNR Am J Neuroradiol* 1999;20: 2000–2003
11. Imbesi SG, Kerber CW. **Analysis of slipstream flow in a wide-necked basilar artery aneurysm: evaluation of potential treatment regimens.** *AJNR Am J Neuroradiol* 2001;22:721–724
12. Graves VB, Strother CM, Partington CR, Rappe A. **Flow dynamics of lateral carotid artery aneurysms and their effects on coils and balloons: an experimental study in dogs.** *AJNR Am J Neuroradiol* 1992;13:189–196
13. Yong-Zhong G, van Alphen HA. **Pathogenesis and histopathology of saccular aneurysms: review of the literature.** *Neurol Res* 1990; 12:249–255
14. Kojima T, Waga S. **More than one aneurysm on the same artery.** *Surg Neurol* 1984;22:403–408
15. Qureshi AI, Suarez JI, Parekh PD, et al. **Risk factors for multiple intracranial aneurysms.** *Neurosurgery* 1998;43:22–27