Delayed In-Stent Occlusion Due to Stent-Related Changes in Vascular Geometry After Cerebral Aneurysm Treatment
—Case Report—

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Abstract
Stent-assisted coil embolization has been recently accepted as a treatment option for wide-neck or complex cerebral aneurysms. Delayed in-stent occlusion is described due to stent-related changes in vascular geometry. A 66-year-old man underwent stent-assisted coil embolization for an unruptured aneurysm of the vertebral artery. The treatment was successfully performed using the Enterprise stent. Follow-up angiography at 6 months showed asymptomatic in-stent occlusion. Three-dimensional analysis of the vascular geometry revealed that the left vertebral artery was straightened by 40° due to the stent placement. Such straightening of the vessel presumably caused kinking and occlusion of the vessel. Stent-related changes in vascular geometry may cause kinking of a vessel and result in occlusion after the treatment of cerebral aneurysms. Pre-treatment strategy may avoid this risk.

Key words: cerebral aneurysm treatment, delayed in-stent occlusion, stent placement, vascular geometry change

Introduction
Stent-assisted coil embolization has been recently accepted as a treatment option for wide-neck or complex aneurysms. Delayed in-stent occlusion has also been reported for the treatment of aneurysms. Most cases of delayed in-stent occlusions were thromboembolic occlusions due to cessation of double-antiplatelet therapy or progression of in-stent stenosis. We report a case of asymptomatic delayed in-stent occlusion after stent-assisted coil embolization due to changes in the vascular geometry caused by the stent placement.

Case Report
A 66-year-old man was referred to our hospital because of an incidentally discovered unruptured aneurysm with a maximum size of 8.6 mm on the left vertebral artery (VA). The aneurysm was located proximal to the posterior inferior cerebellar artery (PICA). The left anterior spinal artery (ASA) was also identified (Fig. 1A, B). The diameters of both VAs were similar. One week before the intervention, the patient was treated with 75 mg clopidogrel and 100 mg aspirin per day. Stent-assisted coil embolization was performed (Fig. 1C). A 28-mm Enterprise stent (Cordis Neurovascular, Miami, Florida, USA) was deployed to cover the neck of the aneurysm so that the distal end of the stent was located between the ASA and the PICA. The in-
In-Stent Occlusion Due to Changes in Vascular Geometry

Intervention was successfully performed. No immediate and peri-procedural complications occurred. The patient was discharged with no neurological deficit. The dual antiplatelet medications were continued. Although the patient remained neurologically intact, follow-up angiography at 6 months showed in-stent occlusion (Fig. 1D–F). The left VA was occluded from the branch of the left PICA. The distal end of the stent could be identified. Thereafter, we were able to reconstruct the vascular geometry of the occluded region. The vascular bending angles of the VA were 86°, 80°, and 46° in chronological order, which demonstrated that the vessel was gradually straightened. 

Fig. 1 A, B: Left vertebral (A) and three-dimensional (3D) rotational angiograms (B) on admission showing an unruptured aneurysm on the left vertebral artery (VA) proximal to the left posterior inferior cerebellar artery (PICA). The left anterior spinal artery (ASA) can be seen (arrows). C: Left vertebral angiogram after stent-assisted coil embolization showing the distal end of the Enterprise stent located between the PICA and the ASA (arrow). D–F: Follow-up left vertebral (D) and 3D rotational angiograms (E) at 6 months revealing in-stent occlusion. The left VA was occluded at the branch of the left PICA. The distal end of the stent can be seen (arrows). Right vertebral angiogram (F) showing the left ASA (arrow) supplied from the right VA via the union of both VAs. Viewing angiograms (D) and (F) together shows that the left VA is occluded between the PICA and the ASA.

Fig. 2 A: Three-dimensional fused vascular images before (green), immediately after (blue), and 6 months after the treatment (brown). The anterior spinal artery can be seen (blue arrow). The distal end of the stent was also identified. Change in the vascular geometry before and just after stent placement was observed (black arrow). Additionally, change in the vascular geometry just after stent placement and 6 months after the treatment was observed (red arrow) by focusing on the distal end of the stent. The vascular bending angles were measured three dimensionally. The angles were 86°, 80°, and 46° in chronological order, which demonstrated that the vessel was gradually straightened. B: This image shows possible changes in vascular geometry. The red curved line shows the centerline of the vertebral artery before the treatment (green). The blue curved line shows the centerline of the vertebral artery 6 months after the treatment. In the region of occlusion, a blue line was drawn by connecting the distal end of the stent and both occlusion edges. The vascular geometry was changed from the red curved line to the blue curved line by stent placement during the follow-up period of 6 months, which might have resulted in the kinking (black arrow) and occlusion of the vessel.

Neurol Med Chir (Tokyo) 53, March, 2013
Discussion

The 3D analysis of the vessel showed that the vessel angle was straightened by 40° at 6 months after the treatment. Stent-related changes in vascular geometry have been observed in the treatment of cerebral aneurysms. Vascular geometry was changed by stent placement in anterior communicating artery aneurysms, and the angle of change ranged from 7.60° to 74.88°, with a mean of 29.95°. Change in vascular geometry also occurred after stent placement for bifurcation aneurysms, and the vascular angles changed by a mean of 18° on immediate angiograms and 34° on follow-up angiograms. In our case, the vessel angle changed by 40° at 6 months after treatment, which is consistent with previous findings. On the other hand, no cases of in-stent occlusion were mentioned in the previous studies.

The 3D analysis of the vessel revealed that the change in vascular geometry caused the kinking and occlusion of the vessel. Delayed in-stent occlusion rarely occurs after the treatment of cerebral aneurysms. Delayed in-stent occlusion was found in 1 of 110 patients who received stent-assisted coil embolization for intracranial aneurysms, and manifested as neurological symptoms. The occlusion occurred after the cessation of double-antiplatelet therapy. Delayed in-stent stenosis occurred in 7 of 156 cases (4.5%). Most were asymptomatic and resolved without additional intervention. Two cases (1.3%) of in-stent occlusion were observed at 5 and 15 months, and both remained asymptomatic. One of those cases was caused by progression of the in-stent stenosis.

In-stent occlusion can occur due to in-stent thrombosis, progression of in-stent stenosis, or stent-related changes in vascular geometry as we have proposed. We continued dual antiplatelet therapy in our case, so the occlusion was not related to in-stent thrombosis. Although little is known about the etiology of in-stent stenosis of a non-stenotic vessel during treatment of cerebral aneurysms, neointimal hyperplasia is believed to be a possible cause. Progression of in-stent stenosis can result in in-stent occlusion. In our case, we could not find any in-stent stenosis at the non-occluded portion of the vessel. Therefore, progression of in-stent stenosis was unlikely to have caused the in-stent occlusion, although we cannot deny this possibility due to the lack of images during the follow-up period of 6 months. 3D analysis demonstrated that drastic vascular change could structurally cause kinking of the vessel, and such kinking could lead to occlusion of the vessel. Although we cannot definitively conclude that the change in vascular geometry caused the in-stent occlusion, we can presume that the vascular change caused structural kinking of the vessel, which resulted in the in-stent occlusion. We consider that vascular change was the most likely cause in this case among the possible mechanisms of in-stent occlusion mentioned above. This possibility should be taken into account during aneurysm treatment using stents. Some previously reported cases of in-stent occlusion may have been caused by changes in vessels induced by stent placement. However, detailed 3D analysis of the vascular geometry is essential to show whether such vascular changes caused the in-stent occlusion.

In our case, in-stent occlusion might have been avoided if we had deployed the stent from the proximal end of the acute bending portion of the VA, thus avoiding kinking of the vessel and in-stent occlusion. A small vessel tends to be bent more and the closed-cell Enterprise stent tends to bend a vessel more than the open-cell Neuroform stent (Stryker Neurovascular, Fremont, California, USA). Therefore, if a parent vessel has an acute bending angle and a small diameter, endovascular surgeons should be careful to prevent kinking and occlusion of the vessel. In such a case, using the Neuroform stent, if available, instead of the Enterprise stent will cause less change to the vessel and may reduce the risk of in-stent occlusion.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper. K. Kono received financial support from the Japan Labour Health and Welfare Organization as research funds to promote hospital functions. All authors who are members of The Japan Neurosurgical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

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