Postmortem Pathological Examination of a Case One Month after Carotid Artery Stenting

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Key words: carotid stenosis, neointima, pathology, stenting, thrombosis

Summary

There have been few pathological reports on human carotid stenting.

A 73-year-old diabetic male with 80% stenosis in the left carotid bifurcation. Despite no neurological events, he died of pneumonia one month after. Findings pathological examination revealed: successful dilatation without deformity of the stent, eccentric compression and partial disruption of the plaque by struts, thrombi around disrupted plaque, and neointimal growth.

Findings were generally compatible with those reported on coronary stenting. Case accumulation is required to clarify an exact pathological process and establish a procedure to reduce complications.

Introduction

Stenting is increasingly being used in patients with carotid stenosis^{1,2}. However there have been few reports on pathology after carotid stenting, except for study on the specimen obtained by post-stenting endarterectomy³.

As far as we know, this is the first case report of postmortem, pathological study on human

carotid stenting. We will discuss and compare the findings with previously published reports on pathology of coronary stenting or post-stenting carotid endarterectomy.

Case Report

A 73-year-old male. History of diabetes, hypertension, and myocardial infarction. No history of transient ischemic attack or stroke. While waiting for coronary bypass surgery, magnetic resonance angiography detected and angiography confirmed 80% stenosis in the left internal carotid artery (ICA) origin, but no thrombus or ulceration (figure 1A). He underwent carotid stenting.

Carotid stenting: Antiplatelets and intravenous heparin had been given. The stenosis was predilated with a 4.0 mm x 40 mm balloon (SAVVY™, Cordis Inc.) at 6 atm for 40 seconds, then stented with a 30 mm-long Palmaz™ stent mounted over a 5 mm x 40 mm balloon (PowerFlex™, Cordis Inc.) at 6 atm for 40 seconds. Next, the site was afterdilated with a 6 mm x 40 mm balloon (PowerFlex™, Cordis Inc.) at 6 atm for 40 seconds. The stenosis was successfully dilated (figure 1B). Heparin was replaced with oral anticoagulants.

Although no neurological event occurred, he

Figure 1 Angiogram during stenting. A) Eighty-percent stenosis was detected in the origin of the left internal carotid artery (ICA) without mural thrombosis or ulceration. B) By stenting and afterdilatation, sufficient luminal gain was obtained.

subsequently developed severe pneumonia and died one month after stenting. An autopsy was performed.

A specimen from the left ICA with stent was fixed in 10% buffered formalin and embedded in glycol methacrylate (GMA), then sliced with a tungsten carbide hard knife into serial cross sections of 5-micrometer thickness. Hematoxylin-eosin (H-E), Elastica van Gieson (EVG), and Masson's trichrome stain were performed on each section.

Under the low magnification (figure 2A), the diameter of the lumen was 5.5 mm at the ICA; dilated enough without restenosis. The plaque consisted mainly of lipid-rich soft components and was eccentrically compressed by struts. There was neither medial disruption nor arterial dissection.

Under the high magnification, where the stent strut simply contacted with native intima without compression or disruption (figure 2B), neointima was found over the strut. The neointima mainly consisted of thrombi around the

strut and overlying thin fibrous cap. The thrombi consisted of platelets and fibrin, and a small number of erythrocytes. Infiltration of lymphocytes, macrophages, and smooth muscle cells were found. The fibrous cap mainly consisted of smooth muscle cells and extracellular matrices. The endothelialization was completed.

Conversely, at the shoulder of the atheroma (borderzone of the atheroma and the residual lumen), the strut disrupted the native intima and contacted directly to the lipid components (figure 2C). At this site, a larger volume of thrombi around the struts was discovered. Infiltration of the lymphocytes, macrophages, and smooth muscle cells was few. The organization of thrombi was poor. Many cholesterol crystals and the granulomatous foreign body type giant cell were found around the thrombi. The endothelialization was completed also in this site.

Discussion

Although there have been few pathological studies after carotid stenting, published studies have been done on coronary pathology. Some of the pathological findings were: compression and disruption of plaque by stent struts, thrombi associated struts and disrupted plaque, infiltration of inflammatory cells and neointimal growth, infiltration of multi-nucleated giant cells, and disruption of media and arterial dissection^{4,5}.

In our case, the findings were generally compatible with these reports. However, at the shoulder of the atheroma, where struts penetrated and disrupted the lipid-rich soft plaque, a relatively larger volume of thrombi was found.

In coronary arteries, the lipid-rich soft plaque was reported to be vulnerable to rupture, which causes thrombotic occlusion and leads to acute coronary syndrome^{6,7}.

In carotid arteries, although controversy still

Figure 2 Photomicrographs of stented left carotid artery. A) At low magnification (x10). Here, the lipid-rich atheromatous plaque was compressed toward the vessel wall, and sufficient luminal gain was obtained. There were no medial disruption or arterial dissection. B) High magnification of figure 2A (x100). At a site the strut simply contacted with native intima, the neointima was identified. It consisted of a small volume of thrombi, lymphocytes, macrophages, smooth muscle cells and extracellular matrix. C) Also high magnification (x200). At the shoulder of the atheroma, the strut disrupted native intima and directly contacted with underlying lipid components. Cholesterol crystals and relatively larger volume of thrombi were found. The organization of thrombi was poor.

remains, disruption of unstable and soft plaque was reported to cause acute thrombotic occlusion^{8,9}. At this site, rupture of the soft plaque by struts, and exposure of the atheromatous debris to the lumen rather than thrombogenicity of struts, seemed to cause thrombosis⁶.

In this case the thrombi did not influence the diameter of lumen. However, as mass thrombi are a potential cause of acute luminal occlusion or distal embolism which lead to serious cerebral ischemia, deployment strategies that reduce the damage to plaque may be required.

On the other hand, one month after stenting, neointima was identified over the strut. Reports on human coronary pathology indicated that neointimal growth began about 10 days after and was almost complete in one or two months^{4,5}.

The time course of neointimal growth was

generally compatible with those reports. As for coronary arteries, it was suggested that arterial injury during stenting and subsequent luminal thrombosis could potentially increase neointimal growth and late restenosis^{4,5}. According to a study on post-stent carotid endarterectomy, restenosis was due to intimal hyperplasia³. It is difficult to presume whether restenosis would occur in this case. But considering that the composition and growth time course of the neointima was generally compatible with those reported in human coronary arteries, it is reasonable to assume, even in this case, further neointimal growth and restenosis would occur from the site where strut penetrated the atheroma and caused thrombosis. Deployment strategies to reduce arterial injury and thrombosis may be needed, not only to avoid acute complication, but also to reduce late restenosis.

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