Case Report

In-stent Thrombosis after Carotid Artery Stenting Despite Sufficient Antiplatelet Therapy in a Bladder Cancer Patient

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In-stent thrombosis (IST) after carotid artery stenting (CAS) is a rare but potentially devastating complication. We present a case of early IST after CAS despite sufficient antiplatelet therapy in a patient with bladder cancer. A 77-year-old man under preventive triple antiplatelet therapy underwent CAS without any intra- or periprocedural complications. However, the patient developed a large asymptomatic IST 6 days after CAS. Anticoagulant therapy with argatroban was reintroduced to treat IST concomitant with antiplatelet agents. Subsequently, the IST shrank and disappeared without any thrombotic symptoms. Malignancy is regarded as an acquired thrombophilic condition associated with a significant risk of thrombosis. In the field of coronary stents, cancer is associated with a significant increasing risk of IST. The cause of IST in our case was possibly related in hypercoagulable state because of the patient's cancer. Attention for IST should be paid in CAS cases with these risk factors, and repeated examination is recommended. **Key Words:** Cancer patient—carotid artery stenting—in-stent thrombosis.

Carotid artery stenting (CAS) is increasingly performed as an alternative to carotid endarterectomy in patients with extracranial internal carotid artery (ICA) stenosis. In-stent thrombosis (IST) after CAS is a rare but potentially devastating complication.[1] Antiplatelet therapy is regarded as essential to prevent thromboembolic complication and IST after CAS. We present a case of early IST after CAS despite sufficient antiplatelet therapy in a patient with bladder cancer.

Case Report

A 77-year-old man was referred to our hospital for the treatment of bladder cancer. The result of his urinary cytology was class V, suggesting urothelial carcinoma. He had experienced right-sided amaurosis fugax on several occasions and was diagnosed with severe stenosis of the right ICA. Duplex ultrasonography revealed an isoechoic carotid plaque causing stenosis of >90%, based on North American Symptomatic Carotid Endarterectomy Trial criteria (Fig 1). N-isopropyl-p-(123)I-iodoamphetamine single-photon emission computed tomography revealed no decrease in resting cerebral blood flow (Fig 2A) but

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Received June 20, 2012; revision received December 7, 2012; accepted December 21, 2012.

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1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2012.12.015

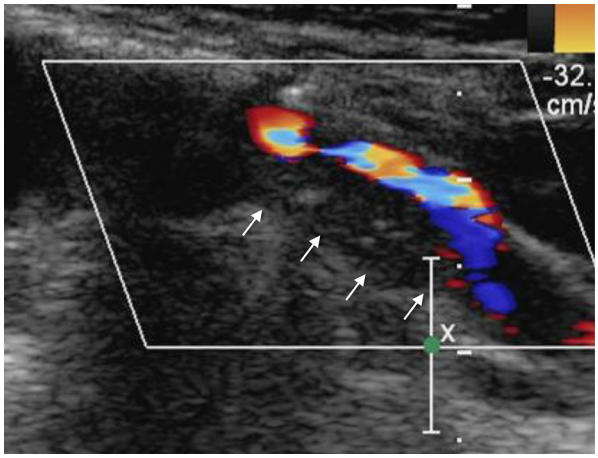

Figure 1. *Duplex ultrasonography of right carotid artery revealed isoechoic plaque* (arrows) *causing stenosis of >90%.*

revealed decreased cerebrovascular reactivity to acetazolamide (Fig 2B). Therefore, carotid revascularization was planned in advance of bladder cancer surgery. CAS was selected because of the high location of the cervical lesion. His blood examination revealed no abnormalities including prothrombin time and activated partial thromboplastin time. Aspirin (100 mg/day), clopidogrel (75 mg/day), and cilostazol (200 mg/day) were started >1 week before CAS as routine preventive administration. Inhibition of platelet aggregation was evaluated using VerifyNow (Accumetrics, San Diego, CA) on the day before CAS. Both aspirin reaction units (397; normal range >550) and P2Y12 reaction units (129; normal range 194-418) revealed sufficient inhibition.

CAS was performed with distal filter protection. Activating clotting time was 328 seconds after 7000 U of heparin was administrated intravenously. Digital subtraction angiography (DSA) revealed a high-grade ICA stenosis (Fig 3A). A FilterWire EZ (Boston Scientific, Natick, MA) was deployed distal to the stenosis. Predilatation was performed with a 3.5-mm diameter Sterling balloon catheter (Boston Scientific). After a Carotid Wallstent (Boston Scientific) placement, postdilatation was performed with a 4.5-mm diameter Sterling balloon catheter. DSAs after CAS revealed adequate dilatation without IST in both lateral and anteroposterior view (Fig 3B-D). The patient was neurologically intact. Continuous heparin (500 U/h) was intravenously administrated after CAS and stopped on the next day after carotid ultrasonography confirmed the patency of the stent.

Six days after CAS, computed tomographic angiography (CTA) performed as a routine follow up examination revealed an asymptomatic but large IST (Figs 4A, 4G). Because antiplatelet therapy had been confirmed sufficient before CAS, anticoagulant therapy was restarted using continuous intravenous administration of argatroban (60 mg/day). Nine days after CAS, CTA revealed slight shrinkage of the IST (Figs 4B, 4H). Therefore, 3 mg of warfarin was orally administered and continuous intravenous anticoagulation was stopped several days later. International normalized ratio of prothrombin time was maintained from 1.7 to 2.0. Fourteen days after CAS, carotid ultrasonography showed apparent shrinkage of the IST (Figs 4C, 4I). Seventeen days after CAS, CTA revealed further shrinkage of the IST (Figs 4D, 4J). A detailed hematologic examination revealed no blood coagulopathy including protein C deficiency, protein S deficiency, antiphospholipid antibody syndrome, or homocystinemia. The patient was discharged without any neurologic symptoms. Six weeks after CAS, carotid

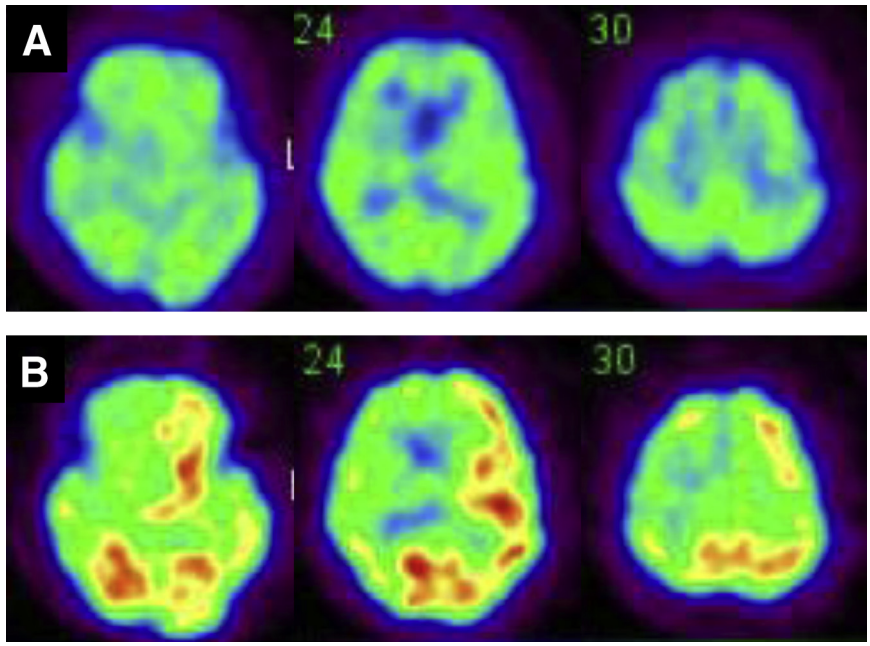

Figure 2. *N-isopropyl-p-(123)I-iodoamphetamine single-photon emission computed tomography showed no decrease in resting cerebral blood flow (A), but decreased cerebrovascular reactivity to acetazolamide (B) at the right internal carotid artery territory.*