Transbrachial Carotid Artery Stenting Can Prevent Renal Cholesterol Embolism

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Abstract

A 78-year-old man was admitted for the treatment of internal carotid artery stenosis. The left internal carotid artery was occluded and stenosis of the right internal carotid artery was progressive. The patient had a history of renal cholesterol embolism associated with percutaneous peripheral intervention. Stenting of the right internal carotid artery was successfully performed via the right brachial artery, and cholesterol embolism was not noted after the procedure. This case suggests that carotid artery stenting (CAS) performed via a brachial approach is less likely to elicit cholesterol embolism than CAS performed via a femoral approach.

Key words: carotid artery stenting, cholesterol embolism, brachial approach

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Introduction

Renal cholesterol embolic disease is a rare disease caused by the embolization of debris from atheromatous plaque, primarily aortic atheromatous plaque (1-3). One of the major precipitating factors includes arterial catheter manipulation involving atheromatous plaque in the aorta (1-3). Usually, carotid artery stenting (CAS) is performed via the femoral artery (femoral carotid artery stenting) because large catheters must be placed in the common carotid artery. We herein describe the case of a patient with internal carotid artery stenosis treated with CAS via the right brachial artery (bCAS) who had a history of renal cholesterol embolism (CE) associated with percutaneous peripheral intervention (PPI). CE was not noted after the procedure.

Case Report

A 78-year-old man presented with dizziness, and an examination revealed occlusion of the left internal carotid artery and stenosis of the right internal carotid artery. The patient was known to have an abdominal aortic aneurysm and a history of renal cholesterol embolic disease after PPI performed for arteriosclerosis obliterans 10 months prior to the

carotid artery stenting. A CT image showed calcification involving the aortic arch, abdominal aorta and common iliac arteries (Fig. 1). An abdominal aortic aneurysm, occlusion of both femoral arteries and stenosis of the left common iliac artery were also noted. The left brachial artery was punctured and a guidewire was advanced to the left femoral artery. Stents were deployed in the left common iliac and superficial femoral arteries. A total of 44 mL of contrast medium was used, and successful revascularization was obtained. However, eosinophilia and an increased serum creatinine level were noted after PPI, with values of 1,430/ μ L and 2.3 mg/dL, respectively. A urinalysis showed no hematuria, and blood tests for ANA, HBS and HCV were negative. Renal CE was suspected, and a small amount of prednisolone was prescribed, after which both the eosinophilia and renal dysfunction improved (Fig. 2). One month after PPI, the patient noticed a rash and nodule on his left thigh, which disappeared spontaneously.

Seven months before the current admission, the patient was treated with percutaneous coronary intervention (PCI). Diagnostic coronary angiography was performed one week prior to PCI, and the total amount of contrast medium used was 15 mL, including diagnostic angiography and intervention. Both procedures were undertaken from the right radial artery. Following PCI, an increase in the proportion of

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Figure 1. 3D-CT angiography and CT showing calcification involving the aortic arch (A, C) and ascending aorta (B), thoracic aorta (D) and suprarenal abdominal aorta (E, F). Calcification is most evident in the aortic arch (A, C).

Figure 2. Changes in the serum creatinine level and number of eosinophils. After PPI and PCI, we observed an increase in the number of eosinophils and serum creatinine level. Renal dysfunction and eosinophilia were not noted following the use of CAS via the brachial approach. PPI: percutaneous peripheral intervention, PCI: percutaneous coronary intervention, CAS: carotid artery stenting

eosinophils and level of serum creatinine were noted (Fig. 2).

On admission, the patient appeared well with no definitive neurological abnormalities. A complete blood count showed a hemoglobin level of 9.8 g/dL and a white cell

count of 6,560/ μ L, with 82% neutrophils, 11% lymphocytes, 2% eosinophils and 5% monocytes. The serum creatinine level was 1.2 mg/dL, and other laboratory findings were normal. Carotid duplex sonography revealed progression of the right ICA stenosis from 61% to 75% on ECST measure-

Figure 3. Brain MRI and SPECT performed before stenting. Diamox SPECT showed the steal phenomenon in both cerebral hemispheres, with more severe findings in the left hemisphere (upper row). MR images showed old cerebral infarcts (lower row).

ment compared with the findings obtained 10 months earlier, with an increase in the peak systolic velocity of the most stenotic lesion from 177 cm/s to 394 cm/s. A brain MR image showed only old cerebral infarcts in the left hemisphere (Fig. 3); however, Diamox-SPECT showed hemodynamic impairment of both hemispheres, with more severe findings on the left side (Fig. 3). The patient's dizziness was considered to be attributable to the impaired cerebral perfusion, and revascularization was necessary. Because the contralateral internal carotid artery was occluded, stenting was indicated instead of carotid endarterectomy. As revascularization of the femoral and iliac arteries via stenting was successful, it was possible to perform CAS via the femoral artery; however, we decided to perform CAS via the right brachial artery in order to avoid manipulation of the aorta and iliac artery with the catheter with the intention of preventing renal CE.

Operating procedure: Two antiplatelet agents, aspirin (100 mg/day) and clopidogrel (75 mg/day), were administered orally prior to the stenting. A 4-Fr sheath was placed in the right brachial artery and a 4-Fr Simmons-type diagnostic catheter was navigated to the right common carotid artery. After the 4-Fr diagnostic catheter was placed in the external carotid artery over the wire, the 4-Fr diagnostic catheter was replaced with a 0.038-inch, 260-cm guidewire. The 4-Fr sheath was removed and a 6-Fr ultralong sheath (Flexor® Shuttle® Guiding Sheath, Cook, Bloomington, USA) was advanced over the wire and placed in the right

common carotid artery (Fig. 4). The procedure after placement of the ultralong sheath was similar to that used with a femoral approach. The carotid stenosis was successfully resolved (Fig. 4), with no clinical evidence of CE during or after bCAS (Fig. 2). The patient was discharged on day 14 with no neurological deficits.

Discussion

CE involves the embolization of distal small arteries by cholesterol crystals and other contents of atherosclerotic plaque, causing end-organ damage (1-3). The mechanism of CE is considered to be migration of the content of atheromatous plaque into organs (1-3), and precipitating factors reportedly include vascular surgery, angiography, angioplasty, stenting and antithrombotic therapy (1-3).

Our patient presented with renal dysfunction after PPI. In general, the causes of renal dysfunction after angiography or endovascular treatment can be categorized into those associated with prerenal and renal failure (4). Hypovolemia due to restriction of oral intake before the procedure or the use of NSAIDs can lead to prerenal failure. Meanwhile, with respect to intrinsic renal failure, contrast-induced nephropathy and CE can complicate the catheter procedure (4). Of these causes, CE presents with eosinophilia. Contrast-induced nephropathy should be considered in association with PPI; however, the total amount of contrast material used during PPI in our patient was as low as 44 mL. Therefore, it is dif-

Figure 4. Carotid artery stenting via the right brachial artery. (A) Right common carotid angiography showing a stenosed ICA with an ulcer. (B) The left MCA was markedly opacified through the Acom because the left ICA was occluded. (C) A guiding catheter (ultralong sheath) was placed via the right brachial artery. (D) Finally, favorable dilatation was achieved. ICA: internal carotid artery, MCA: middle cerebral artery, Acom: anterior communicating artery

difficult to attribute the patient's renal failure to contrast-induced nephropathy. It is also difficult to explain the eosinophilia as being due to contrast material-induced nephropathy (5).

In the present case, pathological findings were lacking; however, the diagnosis of renal arterial cholesterol embolism was sufficiently deserved based on the patient's eosinophilia, increased serum creatinine level and prior history of PPI involving the aorta and iliac arteries, in which atherosclerotic changes were severe. Although obtaining a histopathological diagnosis is the only method of confirming the diagnosis of CE, poor healing at the sampling site has been reported, and the indications for biopsy should be carefully evaluated (3). Our patient presented with a rash and nodule on his left thigh one month after PPI, which may have been a dermatological manifestation of CE; however, confirmation from a dermatologist was lacking. Jucgla et al. studied 26 patients with histologically-proven CE and reported that four patients presented with skin lesions owing to CE more than one month after the precipitating factor (range, 42-126 days) (6). The skin lesions noted in our patient were consistent with the dermatological manifestations of CE.

The mechanisms underlying the development of eosinophilia in patients with CE are not fully understood; however, a foreign body reaction inflammatory response triggered by cholesterol crystals, which are insoluble in body fluid, is a

contributing factor (7). When cholesterol emboli lodge in small arteries of 150-200 μ m diameter, polymorphonuclear leukocytes and eosinophils infiltrate the affected arterioles, and an endothelial inflammatory reaction occurs in a stepwise pattern (7). Renal CE can lead to acute, subacute and chronic renal failure based on the amount of cholesterol emboli (3). In this case, the patient's renal dysfunction was relatively mild, and we believe that a small amount of plaque debris in the aorta lodged in the kidneys, leading to mild renal dysfunction and eosinophilia. This stepwise pattern could explain the delayed appearance of the skin lesion on the leg.

The distribution of atherosclerotic plaque is rather uniform as a result of consistent hemodynamic stress associated with human anatomy (8). Common sites of atheromatous plaque formation include the infrarenal abdominal aorta, iliac and femoral bifurcations and carotid bifurcations (8), with the upper extremity arteries much less frequently involved (8). Perdue et al. studied 26 patients with histologically-proven CE and reported that most atheromatous microemboli originated in the infrarenal or terminal portion of the aorta or proximal large branches (9). Therefore, it is crucial to avoid the abdominal aorta and iliac arteries in order to prevent CE. In the current patient, the use of PPI and PCI prior to bCAS caused eosinophilia and renal dysfunction, and the aorta was involved in both procedures.

Brachial CAS can avoid the aorta completely if the lesion is on the right side, as in the present case. Compared with femoral CAS, in which a guiding catheter is placed from the femoral artery to the common carotid artery, bCAS is less likely to elicit CE due to the avoidance of common sites of atheromatous plaque.

Patients with a history of CE, who are rich in atheromatous plaque, are expected to develop CE easily with additional catheter manipulation. The present case is novel in that it shows that successful avoidance of CE in a patient with a history of CE is possible using a different approach route, i.e., the brachial approach, in which atheromatous plaque is much less abundant compared to the femoral approach.

There are very few previous reports of the rate of CE according to the approach route: the brachial or femoral artery. To our knowledge, there is only one report describing successful percutaneous transluminal coronary angioplasty via the right brachial artery in a patient with CE owing to transfemoral cardiac angiography (10). The authors emphasized the use of the brachial approach to avoid vessel plaque.

In conclusion, we herein reported a case of successful CAS performed via the brachial approach in a patient with CE. This case suggests that CE can be avoided with a brachial approach; however, more case reports are required to fully evaluate the nature of bCAS with respect to the prevention of CE.

The authors state that they have no Conflict of Interest (COI).

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