Retrograde Emboli in An Occluded Arteriovenous Graft in Percutaneous Transluminal Angioplasty complicated Acute Basilar Artery Occlusion

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Abstract

We reported the case of a 65-year-old man with chronic renal failure. After percutaneous transluminal angioplasty for an occluded arteriovenous graft, acute loss of consciousness occurred suddenly. Emergent neurological consultation resulted in activation of an acute stroke protocol. Intra-arterial thrombolysis with recombinant tissue plasminogen activator was performed, resulting in recanalization. Echocardiography, carotid Doppler sonography, and vascular intervention excluded other possible etiologies. Retrograde thromboemboli in the occluded arteriovenous graft was highly suspected. (J Intern Med Taiwan 2014; 25: 15-19)

Key Words: Occluded arteriovenous graft, Retrograde thromboemboli, Basilar artery occlusion, Intra-arterial thrombolysis

Introduction

Basilar artery occlusion (BAO) is an uncommon form of acute stroke that leads to death or long-term disability unless immediate recanalization is achieved. Previous reports showed a mortality rate of 85%–95% without recanalization, even with anticoagulant and fibrinolytic therapy1. The incidence of BAO due to local thrombosis or artery-to-artery thromboemboli originating from arteriosclerotic lesions is >50%. Other factors include cardiac emboli or vertebral artery dissection. In addition, embolic occlusions of the BA are often distal, but atherothrombotic occlusion frequently affects the proximal or middle segments of BA2. Types of the BA occlusive disease include isolated BA disease (44.8%), posterior circulation atherosclerosis (41.4%), and embolism of BA (13.8%)3. In patients with acute BA stroke, recent studies showed local intra-arterial thrombolysis or mechanical recanalization as safe methods for recanalization4.

We reported the case of a patient with end-stage renal disease (ESRD) in whom percutaneous transluminal angioplasty (PTA) was performed for an occluded arteriovenous (AV) graft. However, acute stroke occurred; retrograde embolism was highly suspected and led to total occlusion of the distal BA. Intra-artery thrombolysis with recombinant tissue...
plasminogen activator (rt-PA) was performed, and the occluded BA was recanalized.

Case

A 65-year-old man with ESRD presented with a history of coronary artery disease. A stent was placed over the left anterior descending coronary artery and hemodialysis was performed via left forearm loop AV graft. The patient was transferred to our hospital for emergent thrombectomy for acute thrombosis in the AV graft. During surgery, total occlusion of the AV graft to the venous anastomosis was noted and an attempt to pass a 4-Fr Fogarty catheter failed. Angioplasty was therefore performed. The AV graft was punctured 2 cm below the arterial anastomosis and a 6-Fr insert (Supersheath Set, Medikit, Tokyo, Japan) was inserted. Angiogram again revealed acute thrombosis in the AV graft in addition to the retrograde contrast medium flow into the left axillary artery (Figure 1).

Sudden loss of consciousness and ipsilateral pupil dilatation then occurred. Immediately, brain computed tomography (CT) was performed, but it disclosed no obvious intracranial hemorrhage or apparent brain infarction (Figure 2). Emergent neurological consultation for acute stroke was required. Acute stroke protocol was activated.

Emergent angiography was also performed. Selective left vertebral artery angiography demonstrated total occlusion of the left distal vertebral artery to distal BA. Intra-artery rt-PA (4 mg) was administered via a catheter placed at the proximal portion of the left vertebral artery. Treatment resulted in recanalization of the occluded BA within

Figure 1. Retrograde contrast medium to the axillary artery (black arrow) revealed total occlusion of the venous site of the AV graft.

Figure 2. Brain CT revealed only brain atrophy and no obvious hemorrhage.
Retrograde Emboli Resulted Acute Basilar Artery Occlusion

Echocardiography showed atrial fibrillation, but no left heart thrombus was noted. Carotid Doppler ultrasound scan showed no obvious atherosclerotic change. Twenty-four hours after thrombolysis, National Institutes of Health Stroke Scale score reached E3M5V4, and the level of consciousness had improved but did not recover fully. Follow-up brain CT after 24 h revealed no hemorrhage or obvious ischemic area.

During hospitalization, the level of consciousness gradually was improved, but dysarthria, conscious disorder, facial weakness, and dysphonia were observed. Based on the clinical characteristics of this case, prognosis seemed poor. One month after initial presentation, the sequelae of the occluded BA persisted.

Discussion

Determining the etiology of acute BAO can be challenging. It is the most important to distinguish the atherosclerotic disease from the retrograde embolism. Multiple factors contribute to the pathogenesis of atherosclerosis disease and embolization, including dyslipidemia, endothelial dysfunction, inflammatory and smoking. However, patients who possess the mechanical interventions including guidewire manipulation or vascular surgery have the major risk for embolization, like our case. Furthermore, patients with thromboembolism or atherosclerotic disease have the similar therapy strategy and good outcome under combination of antithrombotic therapy, lipid-lowering therapy, blood pressure control, smoking cessation and glycemic control.

In situ changes in atherosclerosis could be found via carotid doppler sonography or intensive vascular intervention. In our case, there was no significant atherosclerotic plaques in situ over basilar artery in angiography. Cardiogenic emboli (including atrial fibrillation and post valve replacement) is excluded by advanced echocardiography or transesophageal echocardiography. In addition, cardiogenic emboli occur most often in the common carotid artery. A hypercoagulable state may also be indicative of acute BAO. In the case reported here, retrograde flow to the left subclavian artery.

Figure 3. Contrast medium through the left vertebral artery showed total occlusion of the distal part of basilar artery.

Figure 4. Basilar artery occlusion was recanalized (black arrow) after intra-arterial thrombolysis with recombinant tissue plasminogen activator. The bilateral posterior cerebral artery is visible.
(distal) was induced by manual injection of contrast to reveal a totally occluded AV graft, which may have been venous back flow. Because of the inadequacy of this procedure, retrograde emboli-related BAO occurred.

In order to prevent this kind of an event, a test dose of contrast (5–10 mL) can be administered to the fistula to confirm total occlusion. Alternatively, PTA can be performed to treat severe stenosis. If total occlusion recurs, careful PTA wiring can be attempted; postPTA thrombolysis is required if successful; if not, surgical intervention (including patchy enlargement or bypass surgery with polytetrafluorethylene graft) is again necessary.

Our approach to this case of BAO included noninvasive methods (Doppler sonography and CT angiography) and vascular intervention. When patients present with acute brain stem symptoms but negative findings on brain CT, vascular intervention is important. In accordance with precious reports, intra-arterial fibrinolysis with rt-PA was attempted first in our case. After fibrinolysis, recanalization of the occluded vessel was achieved (Figure 4).

References


經皮血管成型術引發逆流性血栓且併發基底動脈阻塞

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摘要

一名65歲男性，有慢性腎衰竭病史並長期接受血液透析治療，此次因左前臂人工洗腎透析管路阻塞而來本院行經皮冠狀動脈血管成型術治療。在行血管成形術時，病患突然發生意識喪失且單側瞳孔擴張，經緊急會診神經內科醫師評估並安排腦部電腦斷層懷疑為急性缺血性腦中風，並從病患臨床表現及當時正接受侵入性醫療行為等情況，高度懷疑為依附於人工洗腎透析管路內之血栓逆流而造成之基底動脈阻塞。該病患經心臟內科施行經動脈內注射血栓溶解劑 (recombinant tissue plasminogen activator (rt-PA)) 後，病患隨即回復意識及神經學部分功能，但日後追蹤病患仍存有言語表達模糊不清等後遺症存在。

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