Postcoital Carotid Artery Dissection Associated with Acute Cerebral Infarction: A Case Report

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Abstract- Carotid arterial dissections may result from spontaneous or traumatic causes. Postcoital arterial dissections have been reported in both the vertebral and coronary arteries. We report a rare case of spontaneous dissection from the common carotid artery to the internal and external carotid arteries after sexual intercourse, leading to fulminant middle cerebral artery (MCA) territory infarct. Although we restored the flow in the proximal carotid artery with stenting, brain swelling with hemorrhagic transformation was still noted in the region of the right MCA.

Key Words: Acute cerebral infarction, Carotid dissection, Postcoitus

Acta Neurol Taiwan 2009;18:267-271

INTRODUCTION

Cervical artery dissections (CAD) can result from either a primary intimal tear with secondary dissection into the medial layer, or via a primary intramedial hemorrhage. An intimal tear will form intramural hematoma that may compromise the original vessel lumen, thrombus formation, and leading to cerebral infarction⁽¹⁾. In addition to intrinsic factors like connective tissue disorders and generalized arteriopathy, mild mechanical traumas, including cervical manipulative therapy⁽²⁾, or sudden neck movements and stretching may cause CAD.

In the previously published literature, there were some case series reported arterial dissections following sexual intercourse, including dissection involving the aorta⁽³⁾, coronary⁽⁴⁾, vertebra^(1,5,6) and middle cerebral arteries (MCA)⁽⁷⁾. Postcoital CAD has been reported and it may be resulted from strenuous action of the neck during sexual activity. We presented a case of spontaneous dissections from the common carotid artery (CCA) with extension to the external carotid artery (ECA) and internal carotid artery (ICA) after sexual activity, which led to fulminant MCA territory infarct.

CASE REPORT

A 58-year-old man had a history of myocardial infarction following angioplasty and stenting 2 years

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Received November 19, 2008. Revised January 5, 2009. Accepted April 25, 2009.

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ago. The patient had taken anti-platelet and antihypertensive agents regularly. Following sexual intercourse, the patient was found to have a change of consciousness with left hemiplegia by his wife, and was then brought to the emergency department in 1 hour of the episode around 0130 in the morning. On arrival, blood pressure was 140/66 mmHg with a regular pulse rate of 48 beats/min and respiration was 21 breaths/min. The Glasgow coma scale was E2M6V2, left hemiplegia, right-sided gaze preference, and left-sided hemineglect and hemianopia, but no Horner's sign was noted. Initial National Institute of Health Stroke Scale (NIHSS) score was 29. Emergent brain CT 90 minutes after event onset showed focal swelling of the right MCA territory (Fig. 1). Intravenous thrombolytic therapy was not indicated in this patient for a high NIHSS score and hypodensity over more than one-third of the MCA territory shown on head CT. After detailed explanation of the risk and benefit of intra-arterial thrombolysis to his family, they agreed to perform emergent cerebral angiography to evaluate the

possibility of thrombolysis.

Emergent cerebral angiography was performed three hours after the episode, which revealed a tapered, flamelike dissection from the right mid-CCA with extension to the right ECA and ICA (Figs. 2A-B). A tapered stenosis with distal occlusion at the right ICA siphon was seen. There was no evidence of acute coronary lesion or septum defect upon angiography. Thereafter, two Boston Carotid Wall stents were deployed from the proximal CCA to the ICA under a protection filter wire (Fig. 2C). Balloon angioplasty was also performed with successful recanalization of the right anterior cerebral artery flow (Thrombolysis In Myocardial Infarction (TIMI) II to III), but failed to open the M1 portion of the right MCA. A follow-up head CT after the procedure showed hemorrhagic transformation with perfusion defect at the right MCA territory (Fig. 3). Decompres-sive craniectomy was performed immediately. During this admission, the laboratory studies, including hemogram and biochemistry, and coagulation profiles were all within normal

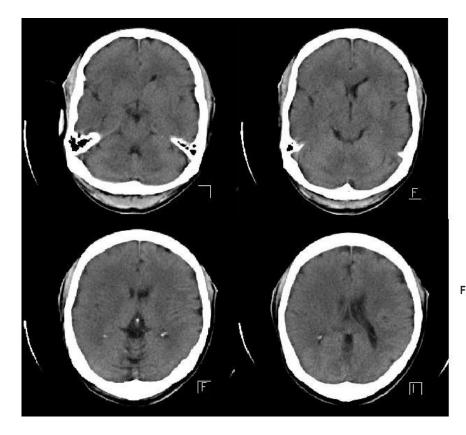


Figure 1. Head computed tomography shows early infarct sign at the right middle cerebral artery territory with effacement of sulci, loss of insular ribbon and poor differentiation of gray-white matter. Mass effect is noted in the right cerebral hemisphere without significant midline shift.

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limits. (Table) The patient had received tracheotomy and was discharged one month later after successful weaning of mechanical ventilator. His consciousness was clear with left hemiplegia, Modified Rankin Scale 4, and NHISS 11.

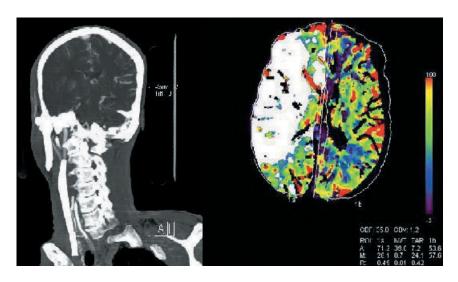
DISCUSSION

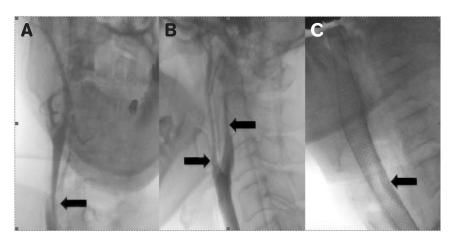
Coital activity is similar to the Valsalva maneuver which may increase intrathoracic, central venous and right atrial pressure. These may precipitate paradoxical embolism through a patent foramen ovale⁽⁸⁾. Besides, migraine variant and transient vasoconstriction of intracranial arteries associated with cerebral infarct during orgasm have been reported^(9,10).

In this patient, neither a history of migraine nor coital cephalgia was noted before. There was no evidence of patent foramen ovale in angiographic and echocardiographic studies. A vivid dissection of the right CCA, ICA and ECA was found by angiography. The spontaneous CCA dissection often results from an extension of the aortic arch dissection⁽¹¹⁾, and dissection of both CCA and ECA had been reported after a traumatic kick to the neck region⁽¹²⁾. Our patient did not have aortic arch dissection, but his dissection originated at the mid-CCA and extended to ICA and ECA. The strenuous sexual activity related mechanical stress, associated with increased intra-arterial pressure during orgasm may

Figure 2. (A) Dissections at the right common carotid artery (arrow: false lumen). (B) Dissections at the right internal carotid artery (right arrow) and external carotid artery (left arrow) (Arrow: tapered, flamelike dissection). (C) After stenting of the right common carotid artery (arrow: false lumen)

Figure 3. Brain computed tomography (left) show acute infarction in the right middle cerebral artery territory status post stenting in the right CCA and ICA. Intracerebral hemorrhage is noted in the right striatocapsular region and thalamus with midline shift to the left. Perfusion scan of the head computed tomography (right) after right carotid artery stenting shows perfusion defect at the right middle cerebral artery territory and preserved perfusion at the right anterior cerebral artery territory.





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Table. Laboratory	examinations
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	Result	Normal range
Hemogram		
Hb	13.8 (g/dl)	13-17 (g/dl)
WBC	8.66 (10³/µl)	3.8-10.4 (10³/µl)
Platelet	162 (10³/µl)	140-400 (10³/µl)
Biochemistry		
Sodium	138 (mmol/L)	135-148 (mmol/L)
Potassium	4 (mmol/L)	3.5-5.3 (mmol/L)
AST	21 (IU/L)	<37 (IU/L)
BUN	26 (mg/dl)	7-22 (mg/dl)
Cr	0.9 (mg/dl)	0.7-1.2 (mg/dl)
Total cholesterol	119 (mg/dl)	<200 (mg/dl)
Triglyceride	79 (mg/dl)	<200 (mg/dl)
HDL-cholesterol	43 (mg/dl)	>35 (mg/dl)
Coagulation profile		
PT	10.7 (second)	8-12 (second)
INR	1.09 (second)	
APTT	24.9 (second)	32-46 (second)

cause stress in the injured intima, and then induced CAD. To the best of our knowledge, this is the first reported case of postcoital dissections involved the CCA, ICA, and ECA.

The management strategy of this type of extensive arterial dissection is dependent on changes in the clinical course of each individual patient. Conservative medical management with anticoagulant or antiplatelet agents may be used in stable or asymptomatic patients with a non-fluctuating course⁽¹³⁾. Emergent angioplasty and stenting should be performed in the patients with impending stroke or when medical treatment fails, i.e. persistence or progression of symptoms^(14,15). Surgical intervention was not considered in our patient because of the distal location of the carotid dissection and the extensive dissection length beneath to the proximal MCA. Endovascular treatment with stenting is safe and technically effective in management of CAD with a favorable clinical outcome in some cases^(16,17).

The cause of cerebral hemorrhage might be due to

hyperperfusion after carotid stenting in this patient. Hyperperfusion syndrome (HPS) was first described by Sundt et al. with the presentation of focal seizure and intracerebral hemorrhage a few days after carotid endarterectomy⁽¹⁸⁾, although successful recanalization of the stenotic vessels. The possible pathophysiology may be due to the failure of normal cerebral autoregulation, secondary to long-standing changes in perfusion pressure. Maximal dilation of cerebral arterioles for long periods of time causes a loss of cerebral blood flow autoregulation in areas of chronically hypoperfused brain tissue and can result in hemorrhage and/or edema.19 The incidence of HPS after endovascular treatment of the carotid arteries was reported below 5%^(19,20) and happened between the 5th and 7th days after the procedure⁽¹⁸⁾. Patients with preexisting brain lesions, either previous or acute stroke, are at a higher risk of developing HPS⁽¹⁹⁾. In this patient, we failed to open the occluded MCA, so the possibility of hemorrhage after HPS was relatively low. Another possibility is hemorrhagic transformation, which may develop in about onethird of all stroke patients within the first week of ischemic stroke, but is rare in stroke secondary to CAD⁽²¹⁻²³⁾.

In conclusion, we report a rare case of CCA, ECA and ICA dissections after sexual activity with a large MCA infarct. Although postcoital CAD is a very rare cause of neck vessel dissections, its rapid progressive course can lead to massive cerebral infarction. In addition to the conservative medical treatment with anticoagulants or antiplatelet agents, angioplasty or stenting can be used to reopen the dissected vessels.

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