

# Intravenous Thrombolysis in a Young Patient with Acute Stroke due to Posterior Cerebral Artery Dissection

Ching-Hsiung Liu<sup>1</sup>, Chun-Liang Lin<sup>1</sup>, Sheng-Lan Yu<sup>2</sup>, Turrando Bih<sup>3</sup>, Liang-Wen Ding<sup>4</sup>

## Abstract

Infarcts of the territory of posterior cerebral artery (PCA) are common and their clinical presentations are well known. However, stroke in young adults originating at the PCA is relatively rare. We describe the case of a young female patient with right PCA infarcts that were probably caused by spontaneous arterial dissection. Dissection was successfully treated with intravenous recombinant tissue plasminogen activator. Herein, we discuss the mechanism, management, serial magnetic resonance angiography results, and functional outcome of treatment.

**Key Words:** Arterial dissection, posterior cerebral artery, thrombolytic therapy, subarachnoid hemorrhage, magnetic resonance angiography

*Acta Neurol Taiwan 2014;23:102-107*

## INTRODUCTION

Infarcts in the posterior cerebral artery (PCA) are not uncommon and characterized by visual field defects, contralateral somatosensory symptoms, and motor deficits<sup>(1)</sup>. The major cause of PCA infarction is a cardiac or intra-arterial embolism<sup>(2,3)</sup>. Vertebral or basilar arterial dissection may contribute to approximately 6% of PCA infarctions<sup>(3)</sup>. However, PCA infarcts resulting from dissection alone are rare<sup>(4,5)</sup>. To our knowledge, no published reports on an isolated infarction within the PCA caused by arterial dissection and treated with intravenous recombinant tissue plasminogen activator rtPA. Here, we report the case of a young female patient with right PCA infarcts probably

caused by spontaneous PCA dissection that occurred while riding a bicycle, which was treated with intravenous thrombolysis. The mechanism, management, serial magnetic resonance angiography results, and functional outcomes have also been discussed.

## CASE REPORT

A 32-year-old, unmarried, female athlete was admitted to our emergency room (ER) with a sudden severe headache on the right side of the head that originated from the occipital area and radiated to the fronto-temporal area, followed by weakness and numbness in her left limbs that started 40 min before admission after riding a bicycle for

From the Departments of <sup>1</sup>Neurology, <sup>2</sup>Radiology, <sup>3</sup>Emergency Medicine, and <sup>4</sup>Division of Critical Care Medicine, Department of internal medicine, Lotung Poh-Ai Hospital, Ilan, Taiwan, Republic of China.

Received February 11, 2013. Revised March 17, 2013.

Accepted July 11, 2014.

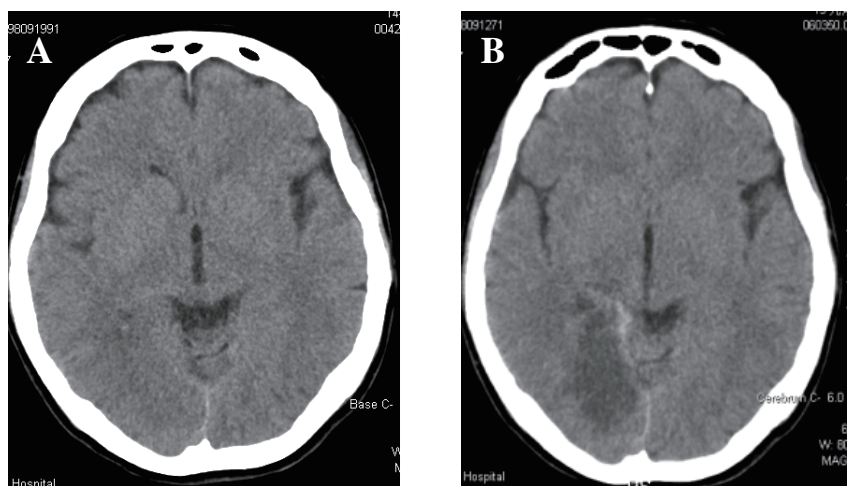
Correspondence to: Ching-Hsiung Liu, MD. Department of Neurology Lotung Poh-Ai Hospital, 83, Nan-Cheng Street Lotung Town, Ilan County, Taiwan, ROC.

E-mail: chl@mail.pohai.org.tw

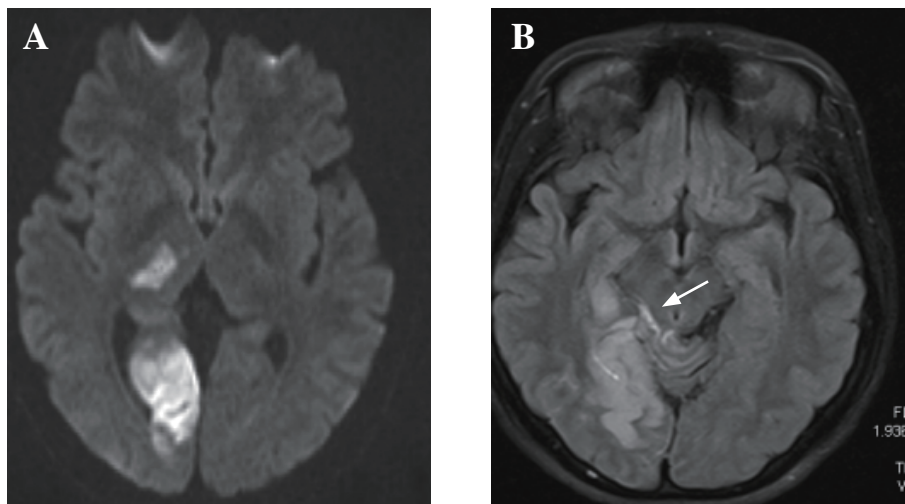
30 min. At the ER, she complained of persistent nausea and diffuse headache, especially in the posterior head region. She denied any history of head trauma prior to this event. Pregnancy test results were negative. Neurological examination showed left homonymous hemianopia, decreased muscle strength in her left limbs, and impaired sensory modality of pain and temperature on the left side of her face, left limbs, and left hemi-trunk. The patient also had impaired left limb coordination. The initial National Institute of Health Stroke Scale (NIHSS) score was 8 and brain computed tomography (CT) revealed no intracranial hemorrhage (Fig. 1A). Therefore, thrombolytic therapy with 0.7 mg/kg rtPA was started 2 h after stroke onset. On day 2 after admission, left limb muscle strength improved, but the patient was drowsy and complained of headache (NIHSS score, 5). Brain CT 24 h after thrombolytic therapy revealed minimal subarachnoid hemorrhage (SAH) on the right peri-pontine cistern and a right PCA infarct (Fig. 1B); however, intracranial hemorrhage was not indicated. Brain magnetic resonance imaging (MRI), including diffusion-weighted images, obtained 35 h after thrombolysis revealed right thalamus and right occipital infarction (Fig. 2A). Minimal ambient cistern SAH was noted on fluid-attenuated inversion recovery (FLAIR) image (Fig. 2B). Brain MR angiography (MRA) showed focal narrowing, followed by dilation of the proximal

portion of the right PCA and diffuse narrowing of the basilar artery (Fig 3A). The anterior and middle cerebral arteries were normal in caliber. The NIHSS score was 4. Brain MRA on day 4 after admission (107 h after thrombolysis) showed recanalization of the right PCA, and the string sign was noted on the images. In addition, increased stenosis was noted in the basilar artery (Fig. 3B).

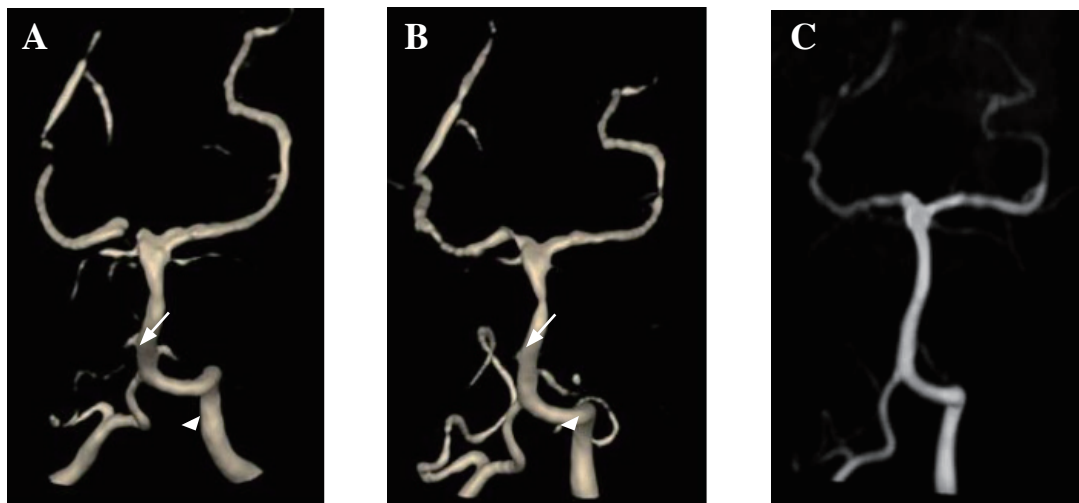
The focal string sign of the right proximal PCA, serial dynamic flow changes of the right PCA, and SAH on brain MRA indicated right PCA dissection. The clinical presentation of the acute occipital headache during exercise was compatible with the diagnosis of right PCA dissection. The patient had no stroke risk factors such as diabetes mellitus, hypertension, or hyperlipidemia on admission, and no history of migraine or contraceptive use. Results of laboratory assays, including anti-nuclear antibody, anti-cardiolipid antibody, lupus anticoagulant, anti-thrombin III antibody, fibrinogen, protein C, protein S, and homocysteine levels, were within normal range. Cardiac assessment, including echocardiography and 64-cut cardiac CT, revealed only mitral valve prolapse. No vegetative thrombus or abnormal shunting was observed. The patient was discharged 4 weeks after admission with good performance of activities of daily living (NIHSS score, 3). One year after stroke onset, the patient could



**Figure 1.** Computed tomography results of a 32-year old female stroke patient. (A) No subarachnoid or intracranial hemorrhage on admission. (B) Follow-up, non-contrast computed tomography 24 h after thrombolysis shows subarachnoid hemorrhage in the right peri-pontine cistern with associated hypo-attenuation in the medial aspect of the right occipital lobe.



**Figure 2.** Magnetic resonance imaging results of the patient 35 h after thrombolysis. (A) Axial diffusion-weighted image reveals right thalamus and right occipital infarction. (B) The axial view of a fluid-attenuated inversion recovery image showing right ambient cistern subarachnoid hemorrhage (arrow).



**Figure 3.** Serial magnetic resonance angiography with reconstruction after thrombolysis. (A) Three days after admission: Focal narrowing, followed by interruption and dilation of the proximal part of the right posterior cerebral artery (PCA) (arrow); diffuse narrowing near the top of the basilar artery (arrowhead). (B) Five days after admission: Partial recanalization of the right PCA with string sign (arrow). However, the upper part of the basilar artery is narrower on this image than on the images obtained 3 days after admission (arrowhead). (C) One year after discharge: Right PCA with almost fully patent flow and normal caliber of the previously narrowed basilar arterial segments.

perform daily activities, including riding a bicycle. During follow-up examination, only partial left homonymous hemianopia was noted with a NIHSS score of 1. Brain

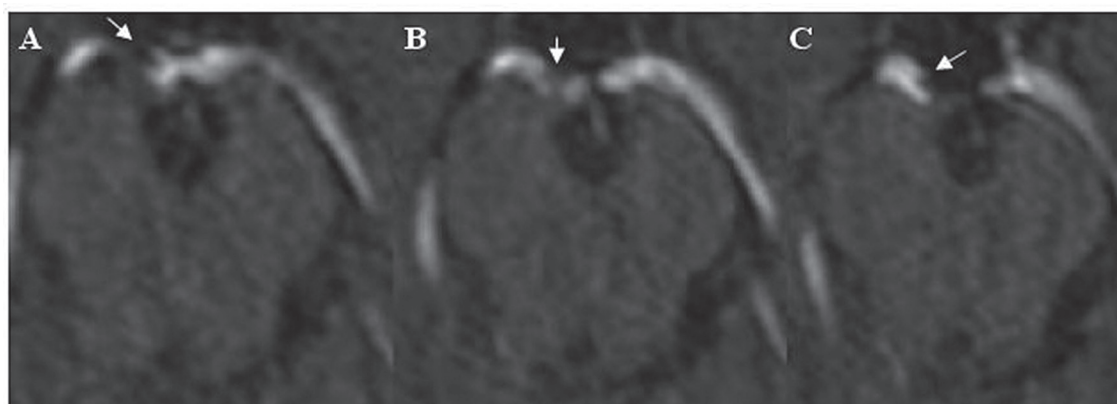
MRI and MRA obtained at the same time revealed that the old insult in the right PCA and the PCA flow were almost fully patent (Fig. 3C).

## DISCUSSION

Intracranial dissection is an under-diagnosed cause of stroke among younger patients<sup>(6)</sup>, which should be considered in young adults with no specific risk factors for atherosclerotic stroke. Diagnosis of intracranial dissection requires presentation of the typical clinical manifestation of intracranial dissection and significant indications of dissection on brain images. Nearly all patients with arterial dissections present with neck pain or headaches, as the cervical and intracranial arteries are heavily innervated by pain fibers<sup>(7)</sup>. The neuroradiological findings of conventional angiography in arterial dissection include a long tapered arterial stenosis or tapered occlusion, intimal flap, contrast medium retention in the late angiographic phase, double lumen, and dissection aneurysm<sup>(8-10)</sup>. Noninvasive diagnostic tools such as brain MRI and MRA can be used to confirm the diagnosis of arterial dissection. Findings of cerebral arterial dissection on MRI, including intramural hematoma with increased external diameter of the artery on T1-weighted images and intimal flap and double lumen on axial source images of MRI<sup>(9)</sup>. MRI and MRA have replaced conventional angiography as the initial imaging study of choice for diagnosing cerebral arterial dissection<sup>(9,10)</sup>.

These techniques are often used for subsequent monitoring of changes in the arterial lumen<sup>(6,7)</sup>. In our case, sequential brain MR source images demonstrated the characteristic double lumen sign (Fig 4). However,

conventional catheter angiography is important for diagnosing suspected cerebral arterial dissection in young stroke patients despite negative noninvasive imaging results. Hemianopic visual and hemisensory symptoms are the most common complaints of patients with PCA stenosis. The mechanisms of PCA stenosis or occlusion-related PCA infarction include thrombosis and cardiac embolism<sup>(1-3)</sup>, but PCA dissection is rare<sup>(3,4,11)</sup>. A recent report indicated that the incidence of spontaneous intracranial arterial dissection (SCIAD) was 8.8% in intracranial cerebral infarctions, and there was only one case of PCA dissection among the SCIAD patients<sup>(12)</sup>. The cause of intracranial arterial dissection remains unknown<sup>(6,8)</sup>. Dissections may be caused by arterial stretching and tearing, which causes bleeding within the arterial wall, and complicated by SAH<sup>(8)</sup>. Differential diagnosis related to cerebral or cervical arterial dissection is variable; connective tissue and vascular disorders have been associated with dissection, including fibromuscular dysplasia<sup>(13)</sup>, homocystinuria<sup>(14)</sup>, and reversible cerebral vasoconstriction syndromes<sup>(15)</sup>. Arterial dissection should be considered in young stroke patients with negative screening results for atherosclerotic lesions, those who participate in vigorous athletic or other activities that involve neck stretching, or those who have undergone chiropractic or other neck manipulation<sup>(4,5,8)</sup>. The present patient who was diagnosed with right PCA infarcts experienced a sudden occipital headache, followed by impaired coordination and numbness of the left limbs after



**Figure 4.** Serial axial source images of brain magnetic resonance imaging in the patient after thrombolytic treatment (A) and (C), The arrows point to the double lumen sign in the right proximal and distal posterior cerebral artery (PCA) on day 3 after admission. (B), The arrowhead points to focal interruption of the PCA.

riding a bicycle for approximately 30 min. Thus, arterial dissection can be considered in a young adult with sudden onset ischemic infarction with SAH on exercise without a stroke history or stroke risk factors; however, SAH post rtPA cannot be fully ruled out even if the probability is low. The incidence of intracerebral hemorrhage as a complication of thrombolytic therapy in ischemic stroke patients within 36 h is 6.4%, but no SAH was reported<sup>(16)</sup>. Trivial head trauma secondary to a stretch or shearing injury of the PCA may have resulted in right proximal PCA dissection. Moreover, spontaneous PCA dissection with secondary diffuse narrowing of the basilar artery after SAH has previously been reported<sup>(17)</sup>, consistent with the image study results in the present case. We cannot exclude the possibility of a basilar arterial dissecting aneurysmal lesion extended to the right PCA; however, based on the site of SAH on the right peri-pontine cisterna, PCA dissection was more likely.

Although anticoagulants are recommended for extracranial artery dissection, this treatment is controversial in intracranial arterial dissection<sup>(4,6,8)</sup>. Intracranial arterial dissections can cause infarction in relation to thrombus formation, propagation, and embolization within the dissected lumen. Anticoagulant therapy may prevent intra-luminal thrombus formation and embolization<sup>(4)</sup>. Furthermore, in the few reported cases of extracranial spontaneous internal carotid arterial dissection treated by thrombolysis with rtPA<sup>(18,19)</sup>, no new or worsened focal neurological sign, SAH, or dissected artery rupture was observed<sup>(19)</sup>. Early brain MRI (FLAIR) or lumbar puncture for cerebrospinal fluid analysis may be performed early to exclude SAH in patients with suspected dissection who will receive thrombolytic therapy. However, no differences in outcomes were noted with thrombolytic therapy in cerebral arterial dissection with infarction or occlusive ischemic infarction<sup>(20)</sup>. One observational study showed that patients with ischemic stroke due to spontaneous artery dissection treated with intravenous thrombolysis had a similar or even better outcome than patients not treated with rtPA<sup>(21)</sup>. Intraventricular rtPA administration is feasible without complications after SAH and may prevent vasospasm and hydrocephalus after SAH<sup>(22,23)</sup>. Blood coagulation is in a hyper-functional state early after SAH to induce secondary lowering of fibrinolytic activity. Plasma rtPA

activity increased within 24 h of SAH to dissolve fibrin deposits in the blood vessels to maintain blood flow after suspected cerebral vasospasm<sup>(24)</sup>. The mechanism of rtPA in treating cerebral arterial dissection with SAH may be by maintaining normal blood flow in injured vessels and preventing secondary vasospasm.

In the present case, although minimal SAH was noted on brain CT after thrombolytic therapy, no change in NIHSS score was noted. Nevertheless, good functional outcome was noted after 6 months.

## CONCLUSIONS

Intracranial arterial dissection is an under-diagnosed cause of stroke in younger patients, and should be considered if the patient has no atherosclerosis risk factors. MRA may be used as a noninvasive diagnostic tool for intracranial dissection. The intracranial arterial dissecting aneurysm may rupture and cause SAH. The use of intravenous thrombolysis in acute stroke patients with arterial dissection remains controversial, but tends to be more beneficial. Thrombolysis may not be withheld in patients with cerebral arterial dissection-related acute ischemic stroke. Physicians should evaluate the risk-benefit ratio of thrombolytic therapy for individual patients. We successfully treated a patient with PCA dissection with intravenous thrombolysis, and our findings may help in the therapeutic decisions for such cases.

## REFERENCES

1. Brandt T, Steinke W, Thie A, Pessin MS, Caplan LR. Posterior cerebral artery territory infarcts: clinical features, infarct topography, causes and outcome: Multicenter results and a review of the literature. *Cerebrovasc Dis* 2000;10:170-182.
2. Yamamoto Y, Georgiadis AL, Chang HM, Caplan LR. Posterior cerebral artery territory infarcts in the New England Medical Center Posterior Circulation Registry. *Arch Neurol* 1999;56:824-832.
3. Kumral E, Bayulkem G, Ataç C, Alper Y. Spectrum of superficial posterior cerebral artery territory infarcts. *Eur J Neurol* 2004;11:237-246.
4. Caplan LR, Estol CJ, Massaro AR. Dissection of the posterior cerebral arteries. *Arch Neurol* 2005; 62:1138-



- 1143.
5. Sherman P, Oka M, Aldrich E, Jordan L, Gailloud P. Isolated posterior cerebral artery dissection: report of three cases. *AJNR Am J Neuroradiol* 2006;27:648-652.
  6. Chen M, Caplan LR. Intracranial dissections. *Front Neurol Neurosci* 2005;20:160-173.
  7. Caplan LR. Dissections of brain-supplying arteries. *Nat Clin Pract Neurol* 2008;4:34-42.
  8. Shimoji T, Bando K, Nakajima K, Ito K. Dissecting aneurysm of the vertebral artery: Report of seven cases and angiographic findings. *J Neurosurg* 1984;61:1038-1046.
  9. Provenzale JM. MRI and MRA for evaluation of dissection of craniocerebralarteries: lessons from the medical literature. *Emerg Radiol* 2009;16:185-193.
  10. Debette S, Leys D. Cervical-artery dissections: predisposing factors, diagnosis, and outcome. *Lancet Neurol* 2009;8:668-678.
  11. Takenouchi T, Shimozato S, Fujiwara H, Momoshima S, Takahashi T. Posterior cerebral artery dissection on a serial magnetic resonance angiography. *Brain Dev* 2012;34:396-399.
  12. Shimoyama T, Kimura K, Iguchi Y, Shibasaki K, Watanabe M, Sakai K, Sakamoto Y, Aoki J. Spontaneous intracranial arterial dissection frequently causes anterior cerebral artery infarction. *J Neurol Sci* 2011;304:40-43.
  13. de Bray JM, Marc G, Pautot V, Vielle B, Pasco A, Lhoste P, Dubas F. Fibromuscular dysplasia may herald symptomatic recurrence of cervical artery dissection. *Cerebrovasc Dis* 2007;23:448-452.
  14. Takagi H, Umemoto T. Homocysteinemia is a risk factor for aortic dissection. *Med Hyptheses* 2005; 64:1007-1010.
  15. Mawet J, Boukobza M, Franc J, Sarov M, Arnold M, Bousser MG, Ducros A. Reversible cerebral vasoconstriction syndrome and cervical artery dissection in 20 patients. *Neurology* 2013; 81:821-824.
  16. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995;333:1581-1587.
  17. Padovani R, Fabrizi A, Sabattini L, Gaist G. Benign arterial dissections of the posterior circulation. *J Neurosurg* 1991;75:69-72.
  18. Derex L, Nighoghossian N, Turjman F, Hermier M, Honnorat J, Neuschwander P, Trouillas P. Intravenous tPA in acute ischemic stroke related to internal carotid artery dissection. *Neurology* 2000;54:2159-2161.
  19. Georgiadis D, Lanczik O, Schwab S, Engelter S, Sztajzel R, Arnold M, Schwarz S, Lyrer P, Baumgartner RW. IV thrombolysis in patients with acute stroke due to spontaneous carotid dissection. *Neurology* 2005;64:1612-1614.
  20. Zinkstok SM, Vergouwen MD, Engelter ST, Lyrer PA, Bonati LH, Arnold M, Mattle HP, Fischer U, Sarikaya H, Baumgartner RW, Georgiadis D, Odier C, Michel P, Putaala J, Griebbe M, Wahlgren N, Ahmed N, van Geloven N, de Haan RJ, Nederkoorn PJ. Safety and functional outcome of thrombolysis in dissection-related ischemic stroke: a meta-analysis of individual patient data. *Stroke* 2011;42:2515-2520.
  21. Budimkić MS, Berisavac I, Beslać-Bumbaširević L, Savić O, Stanarčević P, Ercegovac MD, Pađen V, Jovanović DR. Intravenous thrombolysis in the treatment of ischemic stroke due to spontaneous artery dissection. *Neurologist* 2012;18:273-276.
  22. Varelas PN, Rickert KL, Cusick J, Haccin-Bey L, Sinson G, Torbey M, Spanaki M, Gennarelli TA. Intraventricular hemorrhage after aneurysmal subarachnoid hemorrhage: pilot study of treatment with intraventricular tissue plasminogen activator. *Neurosurgery* 2005;56:205-213.
  23. Ramakrishna R, Sekhar LN, Ramanathan D, Temkin N, Hallam D, Ghodke BV, Kim LJ. Intraventricular tissue plasminogen activator for the prevention of vasospasm and hydrocephalus after aneurysmal subarachnoid hemorrhage. *Neurosurgery* 2010;67:110-117.
  24. Ji Y, Meng QH, Wang ZG. Changes in the Coagulation and Fibrinolytic System of Patients with Subarachnoid Hemorrhage. *Neurol Med Chir (Tokyo)* 2013. [Epub ahead of print]