Spontaneous Carotid Dissection

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Abstract

Spontaneous internal carotid dissection typically occurs in young or middle-aged patients. It is associated with a high rate of recovery and low mortality. Anticoagulant and antiplatelet drugs fail to demonstrate a significant improvement when compared with placebo. A 38 year-old woman with spontaneous dissection of the internal carotid artery diagnosed by MRI is reported. She had an excellent recovery without treatment and no recurrent attack occurred after one year of follow-up. The clinical manifestations, diagnostic tests and treatment of spontaneous internal carotid dissection are briefly reviewed in this communication.

Dissection of the internal carotid or vertebral arteries is a well recognized cause of ischemic cerebrovascular disease in western countries(1). The incidence is increasing due to advances in non-invasive imaging techniques such as MRI, MRA and ultrasonography(2). In Thailand, dissection of extracranial vessels is not a well-known entity. We herein report a case of spontaneous dissection of the internal carotid artery diagnosed by MRI.

CASE REPORT

A 38-year old woman developed a continuous dull headache in the right fronto-temporal region, 7 days before admission. During this period, she suffered three episodes of transient visual impairment in the right eye. The visual symptoms were characterized by "soap-bubble appearance" which lasted about 5 minutes. One day prior to admission, the intensity of headache increased and she had noticed numbness and weakness on the left side of her body and face. On the day of hospitalization, she was nauseated and confused. Ten days before admission she had a mild upper respiratory tract infection which subsided within 2 days after receiving amoxycillin and paracetamol. Her past medical record was unremarkable. She denied any history of hypertension, hyperlipoproteinemia,

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diabetes mellitus, oral contraceptive pill or neck trauma. On physical examination, the patient was afebrile and had normal respiration. Neurovascular examination revealed a blood pressure of 110/70 mmHg in the supine position and a normal examination of her cardiac and peripheral vascular system. However, a bruit at her right carotid bifurcation was detected. The mental status examination was normal. She had left sided weakness of grade IV/V with left facial weakness of upper motor neuron type and decreased pain sensation on the left side of her body and face. The deep tendon reflexes were normal on the right and hyperreflexia on the left. Plantar reflex revealed dorsiflexion on the left.

CT-scan of the brain showed a right fronto-parieto-temporal low density lesion according to the teritory of the middle cerebral artery. Four-vessel angiography revealed "flame-shaped" occlusion of the right internal carotid artery 1 cm above the carotid bifurcation (Fig. 1). The stenotic lesion had a smooth surface. No evidence of atherosclerosis, fibromuscular dysplasia or a vasculitic lesion of the cranial vessels were detected angiographically. The axial MRI of cervical vessels revealed high signal intensity in T_1W_1 which repressented clotted blood in a totally occluded right internal carotid artery above the site of occlusive lesion allocated by angiogram. At the site of the partially occluded region, crescentic high signal intensity in T_1W_1 indicated intramural clot formation within the arterial wall and a narrowed signal voided lumen represented a patent compressed vascular lumen was observed (Fig. 2). The MRI was compatible with dissection of the right internal carotid artery. Laboratory work-up for associated vascular disease included CBC, VDRL, ANA, PT, PTT, lipid profiles, ESR, and lupus anticoagulant were unremarkable. The electrocardiogram and echocardiogram were also within normal limits. The patient received aspirin 300 mg per day and physical therapy. The headache subsided within one week. After three weeks, the weakness had improved to nearly normal and the left sided numbness had disappeared. After one year of follow-up, no recurrent attack has occurred and the patient leads a normal active live.

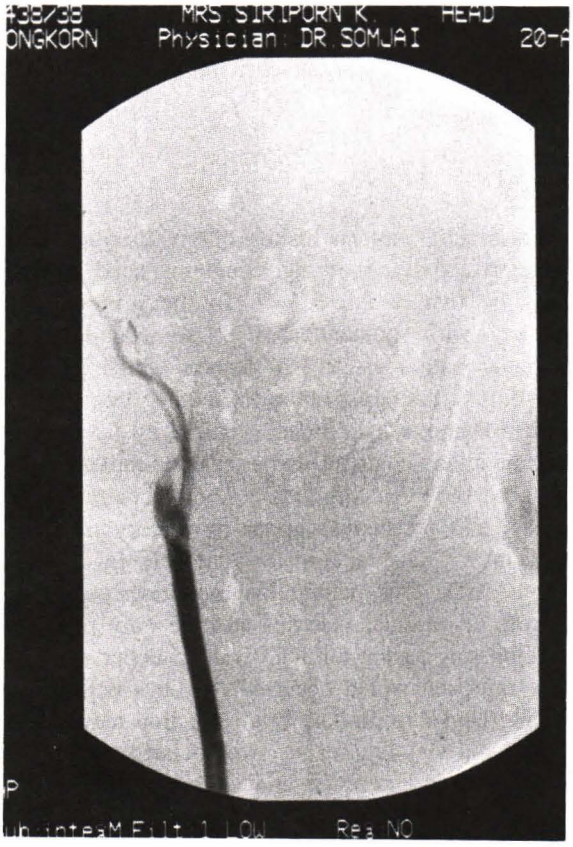

Fig. 1. A cerebral angiogram revealed "flame-shaped" occlusion of the right internal carotid artery 1 cm above the carotid bifurcation.

DISCUSSION

The clinical consequences of internal carotid artery dissection (ICD) depend on whether there is dissection that extends subintimally where it will cause luminal stenosis which will compromise blood supply to distal branches of the artery or it dissects subadventitially which will result in a sac-like out-pouching of the adventitia and produce a mass effect on nearby structures such as cranial nerves or sympathetic fibers[3,4]. The most frequent clinical manifestations of ICD are headache, focal cerebral ischemia and oculosympathetic paresis[2,4]. Headache is an initial manifestation in 47 per cent of patients with ICD[5]. It usually is described as focal unilateral throbbing or steady sharp pain in the orbital-periobital region or around the mastoid process or frontal or temporal region of the affected side[5]. It may precede other symptoms and last from less than 1 hour to 3 months with median duration of 3 days[5]. The

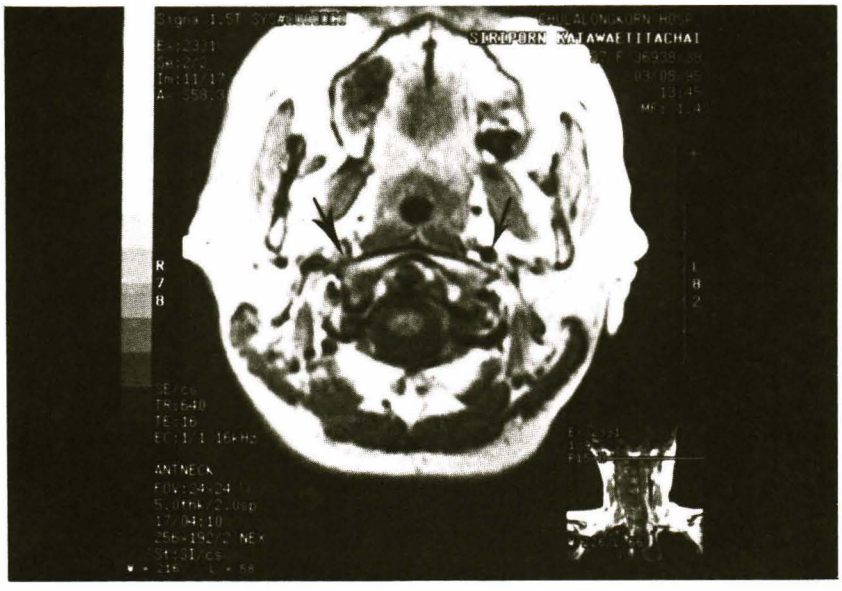

Fig. 2. MRI at the site of dissection revealed the partially occluded right internal carotid artery with the high signal intensity in T_1W_1 which indicated a clot in the dissected arterial wall and a narrowed signal voided lumen which represented partial compressed vascular lumen (large arrow). Normal left internal carotid artery (small arrow).

long interval between the onset of headache and the onset of focal cerebral ischemia differentiate headache associated with internal carotid dissection from other forms of ischemic stroke[6]. Focal cerebral ischemic symptoms range from a transient ischemic attack to massive infarction may be found in 82.5 per cent of cases[7] and appear in the first week after the initial manifestations[4,7]. Oculosympathetic paresis (unilateral Horner's syndrome without anhydrosis) results from an involvement of post-ganglionic sympathetic fiber around the internal carotid artery (ICA) and may be found in 36-58 per cent of cases[2,8]. It tends to last longer than other manisfestations[2,8]. Other signs and symptoms include carotid bruit, pulsatile tinnitus, ipsilateral neck pain, amaurosis fugax[9]. Cranial nerves may also be involved especially CN IX, X, XI, XII[10,11]. However, cranial nerves V, VII, III, IV, VI involvement are rare[10,11]. This patient had headache and amaurosis fugax as initial presentation as well as carotid bruit followed by a clinical syndrome of carotid occlusion.

In spontaneous ICD, there is no history of major injury or previous vascular diseases. However, in some patients history of trivial injury such as sport activity, sneezing, coughing, head turning may be responsible[2,7,9]. In the minority of patients with spontaneous ICD, some underlying vascular pathology or risk factors which include fibromuscular dysplasia, Marfan's syndrome, Ehlers-Danlos's syndrome, syphilis, cystic medial degeneration of vessel, hypertension smoking will be observed[2,9]. Migraine[12], oral contraceptive pill[9] and preceeding upper respiratory tract infection[13] have a controversial role in spontaneous ICD. This patient had no underlying risk factors or vascular diseases and the only possible precipitating factor for ICD was an upper respiratory infection which occurred ten days before the attack. Upper respiratory tract infection may cause ICD by trivial injuries from associated coughing.

Angiography is the gold standard in the diagnosis of ICD[4]. However, the specific pattern "double lumen" will be found only in a minority of cases[4]. The most common angiographic finding is luminal stenosis[4]. Non-invasive imaging techniques which can be used to diagnose and follow-up the course of this disease, are MRI and ultra-

sonography[14-16]. This patient had a luminal stenosis seen on angiography and had a diagnostic MRI.

Spontaneous ICD typically occurs in young or middle aged patients[2,17]. The disease is slightly more common in females[2,17]. Spontaneous ICD is associated with a high rate of recovery and low mortality[2]. The rate of recurrent dissection is 2 per cent in the first month[18]. Afterward it will be 1 per cent per year[18]. The recurrent dissection seldom takes place in the previously affected vessel[18]. No known risk factor is associated with recurrent dissection[18].

Treatment of ICD is still controversial due to its good prognosis. Anticoagulant and antiplatelet drugs have failed to demonstrate a significant improvement when compared with placebo groups [2]. However, it is still reasonable to use anticoagulants, in patients with incomplete, progressive cerebral ischemia or angiography demonstrated distal embolization[2]. In this patient, the occlusion was complete and the stroke syndrome was stabilized. She was given antiplatelet and the long term follow-up for one year revealed a good result. Surgical treatment may have a role in the group that develops recurrent cerebral ischemia while receiving anticoagulant therapy. Reported vascular surgical techniques are: cervical carotid ligation, aneurysm resection and ICA reconstruction as well as cervico-to-intracranial ICA bypass[19].

SUMMARY

A 38 year-old woman presented with severe unilateral headache and amaurosis fugax on the right. Three days later she developed ischemic cerebrovascular disease in the distribution of her right middle cerebral artery. CT-scan revealed ischemia in the distribution of right middle cerebral artery. A four-vessel angiogram showed occlusion of right internal carotid artery 1 cm above the carotid bifurcation. MRI revealed a high signal intensity in T_1 W_1 which represented a blood clot in the arterial wall and partially occluded vascular lumen. This suggested dissection of the internal carotid artery. She had no evidence of previous injury or vascular disease. Spontaneous carotid dissection was diagnosed and she received aspirin 300 mg per day and physiotherapy. The recovery was excellent and she has had no recurrent attack after one year of follow-up.

(Received for publication on May 17, 1996)

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การเซาะของหลอดเลือดแดงคาโรติดชนิดเกิดขึ้นเอง

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รายงานผู้ป่วยหญิงไทย อายุ 38 ปี มาด้วยอาการปวดศีรษะ และตามองไม่เห็นชั่วคราวด้านขวา 3 วันต่อมา ผู้ป่วยเกิดอาการสมองขาดเลือดจากการอุดตันของหลอดเลือดมิลเดิลซีรีบรัลด้านขวา การตรวจด้วยคอมพิวเตอร์สมอง พบสมองขาดเลือดในบริเวณที่เลี้ยงด้วยหลอดเลือดมิดเดิลซีรีด้านขวา การตรวจหลอดเลือดที่ไปเลี้ยงสมองสี่หลอดโดยทางรังสีวิทยา พบการอุดตันของอินเตอร์นัลคาโรติดด้านขวาบริเวณ 1 เซ็นติเมตรเหนือแขนงแยกของหลอดเลือดคาโรติด การตรวจด้วยเอ็มอาร์ไอ พบลักษณะของลิ่มเลือดในผนังหลอดเลือดและรูของหลอดเลือดบริเวณดังกล่าวบางส่วนยังไม่อุดตัน ซึ่งเข้าได้กับลักษณะของการเซาะของหลอดเลือดอินเตอร์นัลคาโรติด ผู้ป่วยรายนี้ไม่มีประวัติอุบัติเหตุ และไม่มีประวัติโรคหลอดเลือดมาก่อน ผู้ป่วยได้รับการวินิจฉัยว่ามีการเซาะของหลอดเลือดคาโรติดชนิดเกิดขึ้นเอง และได้รับการรักษาด้วยแอสไพริน 300 มิลลิกรัมต่อวัน ร่วมกับการทำกายภาพบำบัด ผู้ป่วยฟื้นตัวได้ดี และไม่มีอาการเกิดขึ้นซ้ำอีกหลังติดตามการรักษาครบ 1 ปี

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