Carotid Artery Dissection With Renal Infarcts
Two Cases

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Background Clinical features of carotid artery dissection include ipsilateral local signs, contralateral ischemic stroke, or both. We observed two patients in whom these features were associated with renal infarcts.

Case Descriptions A 57-year-old woman had painful Horner’s syndrome caused by a right internal carotid artery dissection. On days 3 and 4 she had acute abdominal pain, first on the right side and later on the left. The computed tomographic (CT) scan showed a left renal infaract. No aortic dissection or cardiac source of embolism was found. Transesophageal echocardiography showed a mild dys trophy of the ascending aorta and of the mitral valve. Cerebral angiography showed irregularities of the V3 segment of the left vertebral artery compatible with fibromuscular dysplasia. Erythrocyte sedimentation rate was 100 mm/h, and she complained of intense fatigue. She fully recovered within 3 months. A 53-year-old man had sudden severe abdominal pain followed by headache and difficulty in swallowing. He had 9th, 10th, 11th, and 12th cranial nerve involvement on both sides due to bilateral internal carotid artery dissections and pseudoaneurysms. CT scan showed a left renal infaract. Angiography showed extensive signs of fibromuscular dysplasia involving carotid, vertebral, renal, iliac, and mesenteric arteries as well as a dissection of the left renal artery. Erythrocyte sedimentation rate was 65 mm/h, and he complained of severe fatigue. His neurological signs returned to normal in 6 months.

Conclusions Renal infarct due to renal artery dissection may occur together with cerebral artery dissection. Acute abdominal pain, increased erythrocyte sedimentation rate, and intense fatigue are the warning symptoms. (Stroke. 1994;25:2488-2491.)

Key Words • carotid arteries • dissection • renal artery

With the advent of new technologies such as ultrasound with color flow imaging and magnetic resonance (MR) imaging and MR angiography, dissections of extracranial cerebral arteries are more easily recognized, and their clinical features and evolution are better defined.1-4 They include contralateral ischemic stroke,5-8 ipsilateral cervicofacial pain or headache,9 and cranial nerve involvement,10 which can occur in isolation or in association.

We recently observed two patients in whom the usual signs of carotid artery dissection were associated with an unusual clinical presentation including acute abdominal pain, increased erythrocyte sedimentation rate (ESR), and intense fatigue caused by renal infaracts.

Case Reports

Case 1
A 57-year-old right-handed woman had suffered from migraine with ophthalmic aura since the age of 24 years. While suffering from a migrainous attack resembling the previous ones, she had an unusual occipital headache and then noticed that she had symptoms consistent with right Horner’s syndrome. Neck ultrasound examination and transcranial Doppler showed that the right internal carotid artery was occluded with low flow in the middle cerebral artery. MR imaging and MR angiography showed dissection of the right internal carotid and vertebral arteries and no cerebral ischemia. She was treated with bed rest and continuous heparin infusion. On day 3, she had an acute severe pain in the right flank. Abdominal computed tomography (CT) showed a cortical infarct of the right kidney with a patent ipsilateral renal artery that was morphologically normal at angiography. The next day, she had an acute pain in the left flank, and a new CT scan showed a segmental infarction in the left kidney. Her blood pressure was normal. Another angiogram showed no abnormality of the aorta or renal or mesenteric arteries. Cerebral angiography confirmed the right internal carotid artery dissection and showed irregularities of the V3 segment of the left vertebral artery (Fig 1). Transesophageal echocardiography showed dysplasia of the ascending aorta (which was enlarged), a mild mitral valve prolapse, and a slight aortic valve incompetence with no endocarditis. Hemostasis and platelet count (400 000/mm³) were normal, as was the creatinine serum level. There was no proteinuria. Fibrin level was 9 g/L, ESR was 100 mm/h without dysglobulinemia or nuclear antibodies, and rheumatoid test was negative. White blood cell count was transiently elevated at the onset and spontaneously returned to normal. No giant cell arteritis could be seen in the right temporal artery biopsy, and fundi were free of cholesterol emboli. The patient had neither clinical nor histological (at skin biopsy) signs of Marfan’s or Ehlers-Danlos syndrome. She fully recovered in a few weeks except for severe fatigue that lasted 5 months.
Case 2

A 53-year-old man had a past history of hypertension for which he had been treated for 10 years. He complained of fatigue for 3 weeks. One day, he suddenly had both a left parietal headache and abdominal pain around the umbilicus. For the next few days, the abdominal pain was less severe, but he had dysphagia and dysphonia. Two weeks later, he was hospitalized. Neurological examination showed a left myosis and lower cranial nerve palsies (9th, 10th, and 12th on the left side and 11th on the right side) as well as right carotid bruit. Blood pressure was normal. MR imaging and MR angiography showed dissection of both internal carotids without cerebral infarction. The abdominal CT scan showed an infarct of the right kidney (Fig 2) and no dissection of the aorta. Angiography showed ectatic iliac arteries and dissection of both renal arteries and internal carotid and right vertebral arteries (Fig 2). Fibromuscular dysplasia (FMD) involved all these arteries and the mesenteric and celiac arteries as well (Fig 2). There were large pseudoaneurysms of both internal carotid arteries and of the origin of the right vertebral artery (Fig 2). Transesophageal echocardiography showed no dissection of the thoracic aorta and no cardiac source of embolism. ESR was 65 mm/h and fibrin level was 9 g/L. Creatinine serum level was 110 \( \mu \)mol/L, and there was no proteinuria. There were no clinical or histological (on skin biopsy) signs or familial history of Marfan's or Ehlers-Danlos syndrome. He recovered almost completely in 6 months except for severe fatigue after 1 year.

Discussion

These two cases are remarkably similar in the association between extracranial internal carotid artery dissections and renal infarcts, which occurred simultaneously in one patient and 2 days apart in the other. In both cases, carotid dissection manifested itself mainly by local signs, with painful Horner’s syndrome in one and lower cranial nerve palsies in the other. There was no sign of cerebral or retinal ischemia. The MR imaging and angiographic patterns were typical of carotid dissection.

In both cases acute abdominal pain led to recognition of the renal infarcts, bilateral in case 1 and unilateral in case 2. None of the infarcts had a cardiac source of emboli at transesophageal echocardiography, aortic dissection, or atrial fibrillation that could have embolized in the renal artery. The most likely cause is a renal artery dissection as shown in the second patient. In our first patient, however, renal arteries were normal at angiography, and an extensive workup was performed to disclose another possible cause. None was found, and the exact mechanism of the renal infarcts remains obscure.

An inflammatory syndrome with increased ESR, fibrin level, and C reactive protein was present in both patients. This raised the possibility of a connective tissue disease for which, however, no biological evidence was found. Moreover, the favorable long-term prognosis without specific treatment is against such an underlying condition. A most likely explanation could be an inflammatory response to the renal infarct, although an acute self-limited arteritis cannot be ruled out. Such an occurrence has already been reported in one patient with renal infarct, FMD, and dissection of the renal artery and no cerebral artery involvement.11
The simultaneous occurrence of several nontraumatic arterial dissections suggests the presence of an underlying arterial wall disease such as elastic tissue diseases or FMD. Typical angiographic signs of extensive FMD were present in the second patient, whereas the first exhibited only minimal signs of an arterial wall disease with slight enlargement of the ascending aorta and an irregularity with caliber of the vertebral artery in the V3 segment.

FMD of cervical arteries is found in up to 30% of cases in series of nontraumatic carotid dissections. FMD of renal arteries is also occasionally found in patients who have extracranial artery dissection, although the large series from the Mayo Clinic of FMD of the renal artery did not find an association with FMD of carotid arteries.

FMD is also the main cause of isolated nontraumatic renal artery dissection, although one should be cautious in diagnosing FMD in patients with nonatherosclerotic stricture of the renal artery, which could also be due to previous silent dissection. In a series of 24 patients from the Mayo Clinic with isolated nontraumatic renal artery dissection, the authors found recent hypertension in 71% of patients, abdominal pain in 42%, headache in 25%, and hematuria in 21%. The majority of patients had aneurysm of renal arteries (96%), renal infarct (92%), and FMD (92%). In this series, there was a strong association between renal artery dissection and FMD. In none of the patients, however, was an associated extracranial carotid or vertebral artery dissection noted.

Our two cases show that extracranial carotid artery dissection can be associated with renal artery dissections and subsequent symptomatic renal infarcts. The association in time of multiple dissections at cervical and systemic sites, along with apparent FMD, elevated ESR, and intense fatigue, provides evidence that trauma is not the pathogenesis of the dissections and that some very active process (not just other indolent processes) may be involved. This underlying pathological condition could be acute progression of FMD, possibly triggered by an unknown inflammatory process. More should be learned from pathological studies when opportunities arise in the future.

Such cases indicate that occurrences of acute abdominal pain, elevated ESR, and fatigue (and probably acute hypertension) in patients presenting with the usual signs of carotid artery dissection should point to
the possibility of a renal infarct and raise the question of a widespread arterial wall disease such as FMD.

References

1. Hart RG, Easton JD. Dissections of cervical and cerebral arteries. 
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