laboratory tests about thyroid function, autoimmune antibodies, and vasculitis indicators were all normal.

Digital subtraction angiography (DSA) showed a dissecting aneurysm of the C1 segment of right ICA and subtotal occlusion of the left ICA [Figure 1a and b]. Anterior communicating artery was open, and the left middle cerebral artery (MCA) territory got collateral blood flow from the right MCA [Figure 1c]. Severe bilateral stenosis (> 70%) was also revealed on the V2 segment of both vertebral arteries (VA) [Figure 1d-f]. Then angioplasty and stenting was performed for this young patient.

First, the vertebral angioplasty and stenting was performed and then the left ICA was stented because of the carotid sinus reaction. A dissecting stenosis on the initial segment of the left VA stenosis was found during the angiography before stenting. Firstly, one expanding stent was placed on the dissecting site, and then another two stents (4 mm × 60 mm and 4 mm × 40 mm) were delivered and deployed to cover the long stenotic lesion. A final angiography demonstrated an excellent stent placement across the stenotic lesion of the left VA and left vertebral angiogram revealed a good flow in vertebral and basilar arteries [Figure 1g].

After advancing the 8F guiding catheter within the C1 segment of the left ICA, a microwire (0.014 inches) was delivered through the subtotal occlusive site and predilation was performed with a balloon (2.5 mm × 15 mm) at 6 atm. Then, the stent (4 mm × 40 mm Xpert Stent System) was deployed over the stenosis and postdilation was performed with a balloon at 6 atm. Angiography after stenting showed the revascularization of the subtotal stenosis and good flow across the stent with complete disappearance of this stenosis [Figure 1h]. After the procedure, antiplatelet therapy (clopidogrel 75 mg and aspirin 100 mg daily) was sequentially administered for 3 months, and then aspirin is taken prophylactically. During the follow-up period of 3 years, this patient was normal at 3 months after discharged from our hospital, and no recurrent stroke occurred.

DISCUSSION

FMD is a noninflammatory, nonatherosclerotic vascular disease that commonly involves the renal and internal carotid arteries. The young patient has multiple vascular stenoses, but no cerebrovascular risk factors; therefore, cFMD can be diagnosed. The prevalence of symptomatic renal artery FMD is about 4 in 1000, and the prevalence of cFMD is probably half that.[1-4] FMD usually affects the females from 15 to 50 years of age and accounts for around 10% of cases of renal artery stenosis.[1-4]

Although the etiology of FMD is not well understood, several mechanisms have been proposed. For example,

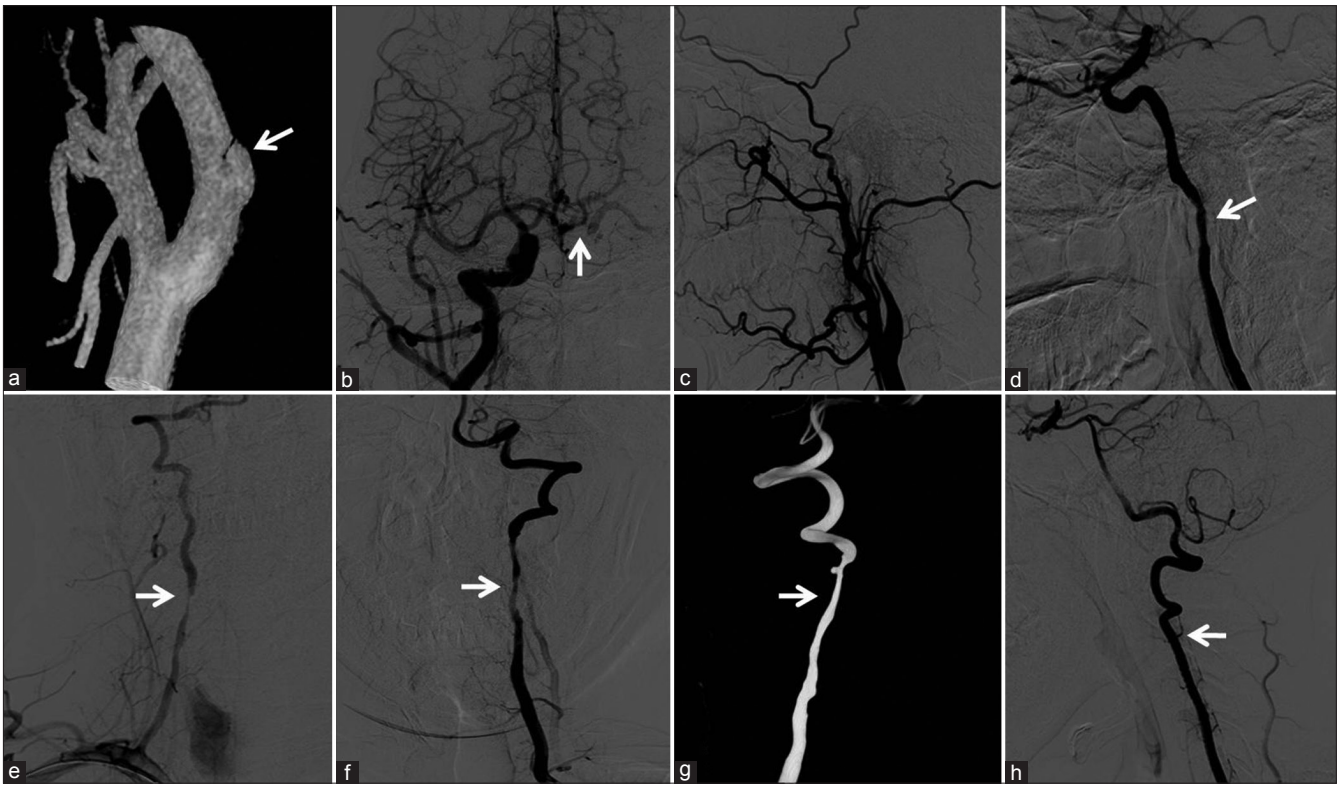

Figure 1: (a) The dissecting aneurysm on the C1 segment of right internal carotid artery; (b) "rat tail sign" of the left internal carotid artery before predilation; (c) the anterior communicating artery was open and the left middle cerebral artery territory got collateral blood flow from the right middle cerebral artery; (d-f) severe bilateral stenosis on the V2 segment of both VAs; (g) left vertebral arteries after stenting; (h) left internal carotid artery after balloon predilation

genetic predisposition, hormonal factors, and arterial wall ischemia.[1,4] The pathogenesis of FMD remains unclear. A number of theories have been proposed, including the environmental factors such as smoking and estrogen, as well as genetic factors; however, about 10% of patients with FMD have an affected family member.[5]

cFMD may be asymptomatic or associated with a variety of nonspecific symptoms, including headache, tinnitus, vertigo, lightheadedness, and syncope.[1] The clinical manifestations of cFMD are variable and depend on a number of factors, including the distribution of vascular bed involvement and the type and severity of the vascular lesions.[6,7] The more specific neurologic syndromes of TIA, amaurosis fugax, stroke, Horner's syndrome, and cranial-nerve palsies may be the first presentation of FMD involving the carotid or VA.[8] And the most feared and serious sequela of cFMD include TIA, stroke, subarachnoid hemorrhage, and cervical artery dissection. It shows that FMD is present in about 15-20% of patients with a spontaneous dissection of carotid or VA.[9] And multiple cervical artery dissections are more common in patients with an underlying arteriopathy, such as FMD.[10]

Noninvasive imaging modalities for diagnosing FMD include Doppler ultrasound, computed tomographic angiography, and magnetic resonance angiography. The accepted gold standard for the diagnosis of cFMD is DSA. "String of beads pattern" in the pathological carotid or renal arteries is an important and most common angiographic finding and is present in over 90% of cases. However, it was not observed in this present case. This invasive test should be considered for those symptomatic patients in whom intervention is contemplated or for cases in which there is uncertainty about the patient's diagnosis or severity of the disease.[11]

Medical therapy and revascularization are the two major treatment options for cFMD patients. As previously discussed, antiplatelet therapy is the mainstay of the medical therapy. For symptomatic patients with carotid or vertebral artery FMD, who have suffered a dissection, angioplasty with stenting may be performed. The indications for intervening in cFMD are for those in whom antiplatelet or anticoagulant therapy is contraindicated or less effective and for those cFMD patients with pseudoaneurysm formation, usually the result of a prior dissection.[12]

The patient was successfully treated with angioplasty with stenting. During the follow-up period, no adverse events and complications were observed suggesting that angioplasty with stenting may be a safe and effective treatment method for this condition.

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Conflicts of interest

There are no conflicts of interest.

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