A Case of High-Grade Basilar Artery Stenosis with Spontaneous Recovery

Spontan Düzelme ile Seyreden Bir Yüksek Dereceli Baziler Arter Stenoz Olgusu

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ABSTRACT

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High-grade basilar artery stenosis was detected in a 31-year-old man that presented with right hemiparesis and dysarthria. The exact etiology of the stenosis could not be determined. The patient was treated medically and remained asymptomatic during a 6-months follow-up period, and follow-up angiograms showed a dramatic improvement in the high-grade stenosis. This report aimed to draw attention to the importance of follow-up for high-grade basilar artery stenoses with medical therapy prior to using endovascular approaches, especially in young patients with a stable clinical course and unknown etiology.

Key Words: Basilar artery, endovascular treatment, stenosis.
INTRODUCTION

The management of basilar artery (BA) stenosis is a challenging problem for neurologists dealing with stroke. Anticoagulation, though not confirmed to be superior to antiplatelets in prospective, randomized trials, has been the choice of treatment, especially in patients showing a progressive clinical deterioration. The development of endovascular methods has offered a promising treatment option for cases with high-grade BA stenosis, in which the prognosis is usually unfavorable if left untreated. Whatever the cause, dissection or atherothrombosis, we still lack scientific data regarding the efficacy of available treatment options - antiplatelets, anticoagulants, endovascular methods or combination of these - in these cases. This report emphasizes the need for controlled trials in managing BA stenosis by presenting a case with high-grade BA stenosis, in which the patient not only stayed asymptomatic, but also the stenosis showed a dramatic improvement under medical therapy.

CASE

A 31-year-old man was referred to our clinic because of right-sided weakness and mild dysarthria. Symptoms started 10 days prior to his admission with a sudden onset of weakness in the right arm and leg and difficulty in articulation. He complained of occipital headache radiating to the neck during the previous week. He was admitted to another medical facility where his blood pressure was recorded as 230/120 mmHg. No abnormality was detected on his admission cranial computed tomography. He was diagnosed as ischemic stroke and set on acetylsalicylic acid and amlodipine therapy. His symptoms gradually improved over the following days and he became symptom free on the 5th day of his admission. He was then referred to our institution for further evaluation.

His neurologic examination, in our center, was normal except for a right-sided extensor plantar response. He was a heavy smoker and a diagnosis of hypertension was established a year ago, but he did not comply with the recommended medication. Laboratory investigations including blood chemistry, cell count, erythrocyte sedimentation rate and electrocardiography were normal, except for high cholesterol, triglyceride and homocysteine levels. The lipoprotein(a) level, markers for connective tissue diseases and hematologic parameters including fibrinogen, lupus anticoagulant, protein C, protein S, antithrombin III and activated protein C resistance were within normal levels. Transthoracic and transesophageal echocardiography did not reveal any pathology.

Cranial magnetic resonance imaging (MRI) showed a subacute, left pontine infarction (Figure 1). As a suspicion for BA stenosis was raised by magnetic resonance angiography, the patient was further evaluated with conventional angiography, which demonstrated a short segment of high-grade stenosis of the BA, just after the branching of anterior inferior cerebellar arteries (Figure 2). The rest of the angiographic investigation was normal and no signs suggestive of vasculitis or dissection were detected. The stenotic region was further evaluated with T1-weighted fat suppressed MRI sequences, but no evidence for dissection was present.

As the clinical picture of the patient was stable, angiographic intervention was deferred and the patient was set on anticoagulant therapy. A statin for the dyslipidemia,
an angiotensin converting enzyme inhibitor for the hypertension and a vitamin complex for the hyperhomocysteinemia were added to the therapeutic regimen. The patient stayed asymptomatic for the following 6 months. In his reevaluation, no new ischemic lesions were detected in his cranial MRI. His control angiography revealed a marked improvement in the high grade stenosis, with only a residual slight irregularity of arterial contours in the area of stenosis (Figure 3). Anticoagulation was stopped and the patient was discharged on anti-platelet therapy.

**DISCUSSION**

This report describes a patient presenting with left pontine infarction and high-grade BA stenosis. Despite intensive investigations the etiology of the stenosis could not be determined. The presence of hypertension, dyslipidemia, hyperhomocysteinemia and history of smoking are all in favor of an atherosclerotic process, but the lack of any plaque formations in the extracranial circulation and the rest of the intracranial circulation in conventional angiography are against such a diagnosis. The possibility of an isolated intracranial atherosclerosis is rather speculative, bearing in mind that the patient was not of Asian descent. Taking into consideration the young age of the patient, the presence of occipital headache at the time of onset of symptoms and the remarkable improvement in the follow-up angiography, the most plausible explanation for the stenosis is a dissection. The presence of the above listed risk factors may have predisposed to the formation of dissection, which was suspected in the initial admission of the patient to our center, but could not be proven by T1-weighted fat suppressed MRI sequences. Still we must admit that, none of the angiographic gold standard findings such as double lumen appearance or endothelial flap could be demonstrated and therefore the possibility of cryptogenic embolism or in-situ thrombosis could not be excluded, both of which possibly would lead to recanalization either spontaneously or under anticoagulation during a period of 6 months.

The most extensive information regarding the follow-up in patients with BA stenosis comes from the retrospective and prospective arms of the Warfarin-Aspirin Symptomatic Intracranial Disease Study (1,2). The retrospective study included 28 symptomatic cases of BA stenosis all having an atherosclerotic origin (1). 33% of the patients on aspirin and 11% of the patients on warfarin had a stroke in the same territory of stenosis during a median follow-up period of 13.8 months. The potential benefit from anticoagulation in basilar artery stenosis was also observed in the subgroup analyses of the prospective arm (n=112); patients with BA stenosis on aspirin treatment were approximately 2 times more likely to suffer from a stroke (ischemic or hemorrhagic) or vascular death when compared to patients treated with warfarin (2). However this superiority of warfarin over aspirin was lost when the analyses were restricted to recurrent ischemic events in the territory of the symptomatic artery.

The cumulative stroke rate -same or a different territory- in BA stenosis secondary to atherosclerosis ranges from 5%-15%/year (1-4). Patients with a stenosis greater than 80% had a substantially higher risk for stroke recurrence (1). On the other hand data on follow-up of patients with BA dissection is scarce. In 1 series 84% of the patients had a good or excellent outcome, whereas in another series nearly half of the patients had permanent disabling deficits (5,6). Patients with BA dissection not only present with ischemic stroke but also with subarachnoid hemorrhage, and spontaneous healing of BA dissection is not an uncommon finding in the series reported in the literature (7,8).

The technical developments in interventional radiology have opened a new era for the treatment of patients with BA stenosis. The Stenting of Symptomatic Atherosclerotic Lesions in The Vertebral or Intracranial Arteries Study, included 17 cases of BA stenosis, of whom 2 (12%) developed stroke within 30 days of stent placement (9). Additionally, registry results and case series report successful results with balloon angioplasty and stent applications in BA stenosis (10-13). In fact, most of the patients who have undergone BA stenting were cases with lesions due to atherosclerosis either unresponsive to maximal medical therapy or had contraindications for anticoagulation. Despite successful results, the lack of randomized trials comparing the efficacy of stenting vs. medical therapy and the risks such as obliteration of the ostia of small penetrators or rupture leading to subarachnoid hemorrhage make BA stenting more challenging compared with extracranial stent placements.

As such a dramatic improvement is highly unexpected for an atherosclerotic plaque, a dissection was accepted as the most plausible explanation for the stenosis in our patient although it could not be proven by radiologic in-
vestigations. Whatever the cause, the best therapy for these patients is not known. The concept that BA disease is associated with a very poor prognosis is being altered by the recently published prospective hospital registries (14). Still, there is no data regarding the best medical choices, and the timing of endovascular methods is also a subject of debate. Currently the management of the patients depends on their clinical status under medical therapy; endovascular approaches like stenting are reserved for patients with progressive course despite maximum medical therapy. Whether the statins and angiotensin converting enzyme inhibitors could have a beneficial effect in angiographic improvement of the lesion, taking into consideration the accumulating evidence for these agents in stabilizing and even reversing the vascular plaque burden and whether they should be added to the therapeutic regimen are all subjects of further studies (15,16). The dramatic reversal of the lesion in our patient draws attention to the importance of follow-up of neurologically stable high-grade BA stenosis patients with medical therapy before endovascular approaches.

REFERENCES


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