The established medical treatment for patients with symptomatic BA stenosis includes antiplatelet drugs, sometimes in combination with systemic anticoagulation. However, the natural history of BA stenosis remains poor with a reported annual risk of stroke of 20% in the basilar territory. If the symptoms are refractory to medical management, the therapeutic options are limited. Bypass surgery at the BA is difficult and associated with high morbidity. Percutaneous angioplasty is associated with a significant complication rate, including vessel dissection and thromboembolic events caused by plaque disruption. There is a high restenosis rate after percutaneous angioplasty. Due to recent advances in stent technology, angioplasty and stent placement have emerged as an alternative treatment option for this condition. Yet interventional procedures involving protracted forced straightening of severely elongated arteries may pose a risk in themselves.

In this paper, we report a fatal complication in a patient with symptomatic high-grade BA stenosis in combination with a severe kinking at the site of stenosis. The postmortem findings and technical aspects of the intervention are discussed.

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**Abbreviations used in this paper:** BA = basilar artery; PCA = posterior cerebral artery.

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**Case Report**

**History and Presentation.** This 59-year-old male patient was referred to our institution with a history of several transient ischemic attacks in the past 6 months. His symptoms included left-sided weakness and speech disturbances, occurring acutely and lasting for < 30 minutes, and gradually resolving after several minutes. Four months before presentation at our institution, the patient had suffered a brainstem infarct. During medical treatment with aspirin (150 mg daily) and clopidogrel (75 mg daily) the patient experienced further recurrent, transient ischemic brainstem attacks presenting as episodes of tetraplegia. A diagnostic digital subtraction angiogram confirmed the presence of a severe (> 80%) mid-BA stenosis with a length of 18 mm as well as a severe vessel kink (Fig. 1a and b). The vertebral arteries showed atherosclerotic changes but no significant stenosis. Both posterior communicating arteries were hypoplastic; hence no significant perfusion from the anterior circulation was found. Given the continuing clinical symptoms of the patient an interventional treatment attempt was offered.

**Operative Course.** After the induction of anesthesia, a 6 Fr sheath was placed into the patient’s right femoral artery and a guide catheter was navigated into the right vertebral artery. To prevent embolic events, 5000 IU of...
Fatal hemorrhage after treatment of a basilar artery stenosis

Heparin was administered in addition to the continuing antiplatelet medication. A 0.014-inch exchange wire was advanced across the stenosis, and navigated into the left PCA. This navigation was complicated due to the tortuosity of the posterior circulation. A 3 × 15-mm Gateway percutaneous angioplasty balloon (Boston Scientific) was advanced over the exchange wire and carefully inflated. During this procedure, some straightening of the BA was observed. A control angiogram showed a partial reopening of the vessel (Fig. 1c and d).

After removal of the balloon, a 4.5 × 20-mm stent (Wingspan, Boston Scientific) was placed across the stenosis without complication. However, the stent could not be deployed in the stenosis due to high internal friction of the entire system. During the attempt the BA was markedly stretched and straightened by the stent and guidewire. The attempt to deploy the stent was abandoned after several minutes, and the stent with the microwire was retracted into the guide catheter. Because of the significant risk of restenosis of the unprotected intracranial stenosis, the decision was made to deploy a smaller stent. A new microwire was deployed across the stenosis. This time the wire was navigated into the right PCA because the patient’s anatomy suggested that this might ease the tension on the stent during its deployment. At this point during fluoroscopy the BA was observed to move, and massive extravasation of contrast media was observed (Fig. 2). A perforation was suspected at the BA tip or in the right proximal PCA.

The effects of heparin were reversed by administration of protamine (15 mg), and a 4 × 7-mm balloon (Hyperglide, Boston Scientific) was placed into the mid-BA and inflated for several minutes to facilitate clotting. During this period, the patient became hypertensive and subsequently suffered a cardiac arrest from which he was successfully resuscitated in the angiography room. The balloon was removed, and a postinterventional CT confirmed massive extravasation of contrast media and a subarachnoidal hemorrhage. Although an extraventricular drainage catheter was placed,
the patient did not sustain tolerable intracranial pressures and died 18 hours after the intervention.

Postmortem Examination. The postmortem examination confirmed extensive subarachnoid blood and generalized brain swelling. It revealed a 3-mm rupture of the BA proximal to the stenosis at the transition zone to the severely indurated vessel (Fig. 3 left). No sign of a perforation was found at the tip of the BA or in the PCA. Massive atheromatous disease was found in the posterior circulation, most pronounced at the site of stenosis in the mid-segment of the BA. The BA vessel wall showed high-grade subintimal thickening, fibrosis, and cholesterol deposition (Fig. 3 right), and increased vessel wall diameter.

Discussion

Patients with a high-grade intracranial stenosis who remain symptomatic despite adequate medical treatment represent a therapeutic dilemma. Without further treatment, these patients have a poor prognosis with a high risk of stroke or death. The new generation of self-expanding stents for intracranial applications appears to be flexible enough to be navigated through the tortuous vessels and across tight stenoses in these patients. However, the interventional treatment of these lesions remains dangerous and several studies have reported fatal complications during percutaneous angioplasty or stent-assisted angioplasty of the BA. Most of these complications were caused by periprocedural thromboembolic events and dissections; a few studies report vessel rupture as a consequence of manipulation or perforation of devices.2,3,6

In the presented case the postmortem findings revealed that the rupture was not caused by a microwire perforation at the right proximal PCA origin, as concluded from the angiogram. Instead, the rupture point was found proximal to the stenosis in the transition zone between normal-appearing and indurated vessel wall. This area was not directly exposed to balloon angioplasty or the manipulation with the microwire or stent. Fluoroscopy during intervention showed a straightening of this area during microwire manipulation, angioplasty, and the stent placement attempt. The vessel rupture was presumably a result of the sustained forced straightening and overstretching by the repetitive passage of the stenosis during the intervention. This resulted in a rupture of the BA segment due to the elongation and rigidity of the atherosclerotic vessel wall.

Therefore it may be concluded that severe kinking is an additional risk factor in percutaneous angioplasty and stent placement in a patient with intracranial stenosis and may cause vessel rupture without direct perforation. Care should be taken to minimize the manipulation of the vessel before stent placement.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Address correspondence to: Jan Gralla, M.D., Department of Neuroradiology, West Wing, John Radcliffe Hospital, Headley Way, Headington Oxford, OX3 9DU, United Kingdom. email: jan.gralla@insel.ch.