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Efficacy of stent angioplasty for symptomatic stenoses of the proximal vertebral artery

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Abstract

Background: To evaluate the safety and efficacy of stent angioplasty in the treatment of symptomatic arteriosclerotic stenoses of the proximal vertebral artery (VA).
Methods: Thirty-eight symptomatic stenoses of the vertebral origin were treated with flexible balloon-expandable coronary stents. Angiographic and clinical follow-up examinations were obtained in 26 patients at a mean of 11 months.
Results: The immediate post-procedural angiographic results showed no residual stenosis in 33 vessels and mild residual stenoses in five vessels. Periprocedurally, there were two asymptomatic technical complications and one TIA. During follow-up re-stenosis could be detected in 10 cases (36%), and vessel occlusions in two patients. Two stents were broken. One of the restenosis caused a TIA within the follow-up period.
Conclusions: Flexible balloon-expandable coronary stents proved to be save and effective in preventing vertebrobasilar stroke but were incapable to preserve the proximal vertebral artery lumen. For the VA origine an adequate stent, self-expanding, bioresorbable, or drug-eluting has to be found.

Keywords: Atherosclerosis; Stent; Angioplasty; Proximal vertebral artery stenosis

1. Introduction

Stenoses of the proximal vertebral artery (VA) are often incidentally discovered by angiography or other examinations of the brain supplying arteries such as duplex sonography. The frequency of which such asymptomatic lesions are identified concurs with previous descriptions of the benign course of unilateral vertebral artery pathology [1]. The data on incidence is also equivocal. Arteriosclerotic stenoses of the proximal VA are less frequent than of the carotid artery, perhaps by a factor of 2 [2]. The "New England Medical Center Posterior Circulation Registry" [3] recorded 407 patients with vertebrobasilar symptoms including 20% ($n = 80$) with a stenosis of the V_1 segment. In 52.5% of these patients a stenosis of the contralateral VA was also evident. Hennerici et al. [4] described a population of 426 patients with cerebrovascular risk factors in which 2.3% ($n = 10$) had a stenosis of the VA origin and 7% ($n = 30$) an aplasia or occlusion of a VA without clinical symptoms of stroke or TIA.

The morphology of the stenoses and the good collateral blood supply in the vertebrobasilar system determine the relatively benign course of this disease [6]. The risk of cerebral infarction caused by this disease is undetermined [6]. Moufarrij et al. [5] evaluated the course of 96 patients with a

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stenosis of the proximal VA during 4.6 years: Nineteen percent ($n = 18$) developed initial symptoms of a vertebrobasilar ischemia and only two patients suffered from a brain stem infarct. Nevertheless proximal VA stenoses may be identified as the cause of basilar thrombosis (>10% of our patients). Vertebrobasilar infarcts constitute 20% of all cerebral infarcts. Half of these strokes are caused by emboli from other arteries or cardiac sources. The other half are due to diseases of the vertebrobasilar arteries [7]. Wityk et al. [3] identified stenoses of the proximal VA as the sole causative factor in 38 from 407 cases (9%) with symptoms of a vertebrobasilar ischemia. According to these authors, arterio-arterial embolic causes were thought to be more frequent than hemodynamic ones. Other authors [8] attached greater significance to hemodynamic factors, e.g. bilateral stenoses, as the main reason for non-cardiac causes of ischemia of the vertebrobasilar circulation.

The risk for cerebral ischemia is increased if a stenosis of the proximal VA is combined with a proximal stenosis of the contralateral vertebral or the basilar artery [3]. Evidence-based data on treatment with anticoagulation or antiplatelet agents in the prophylaxis of stroke in this disease are not available. More invasive therapeutic strategies must be considered in symptomatic stenoses of the VA combined with contralateral stenosis, occlusion, or hypoplasia. Medical prophylaxis does not influence the prognosis in these cases [9]. Both surgical and endovascular procedures are described in the literature. Surgical therapy (bypass, transposition, or thrombendarteriectomy) should not be the first choice because of high non-ischemic complication rate and high invasivity [10]. The disadvantages of the percutaneous transluminal angioplasty (PTA) of stenoses of the proximal VA without stent placement are early relapses and vessel dissections during treatment [11]. Hatano et al. [12] emphasized the elastic resistance of these stenoses of the VA, which complicates PTA. If a vessel dilation is performed more vigorously the risk of a dissection is increased. PTA combined with primary stent implantation (stent-PTA) is a concept to overcome the disadvantages of PTA alone.

The experiences of our group with stent angioplasty using coronary stents in the treatment of stenosis of the proximal VA are presented in this paper.

2. Material and methods

Thirty-six patients with a total of 38 arteriosclerotic stenosis of the proximal VA were treated by stent-PTA between November 1997 and June 2001 in our hospitals. Nine were female and 27 male (mean age was 61 years, range 44–75 years). Twenty-one stenoses were right-sided, and 17 were on the left. In 34 cases a contralateral stenosis, occlusion, or hypoplasia of the VA was associated. In five cases distal intracranial stenoses of the vertebrobasilar arteries were associated, possibly a contributing factor for cerebral ischemia or TIA. All patients gave informed consent for the endovascular treatment. High flexible balloonexpandable coronary stents made of stainless steel were used for stent angioplasty in all cases (S 670, Medtronic Vascular, Santa Rosa, CA, USA or Velocity, Cordis, Miami Lakes, FL, USA).

The stenoses were assessed by means of digital subtraction angiography. Stenoses ≥70% by NASCET criteria were included [13]. A stenosis with recurrent neurological symptoms despite a sufficient oral anticoagulation with an international normalized ratio (INR) between 2.0 and 3.0 or antiplatelet medication with aspirin or clopidogrel was the absolute indication for treatment. Relative indications were a symptomatic proximal VA stenosis associated with a stenosis, occlusion, or hypoplasia of the contralateral VA and an angiographic relapse after PTA without neurological symptoms (Table 1a). The treatment was applied to the dominant VA, or in case of bilateral disease, the more tightly stenosed VA. Twenty-one patients had cerebral infarcts in the vertebrobasilar territory and 15 had vertebrobasilar TIA. Two cases were not categorized as above: case 15, a stenosis of a vertebral artery terminating at the posterior inferior cerebellar artery (PICA) combined with occlusion of the contralateral VA, and case 27 a high-grade relapsing stenosis of a previously treated contralateral VA (see case 3). Patients with proximal vertebral artery stenoses and normal contralateral vertebral artery were not included in this study and were treated by medical therapy (oral anticoagulation with an INR between 2.0 and 3.0 or aspirin 300 mg/day or clopidogrel 75 mg/day). In order to eliminate other causes for vertebrobasilar strokes, a German Stroke Guideline orientated workup was performed in each patient before the intervention (including transesophageal echocardiography and a protein C global assay to exclude cardiac embolic sources and coagulopathies in all patients).

All interventions were performed using local anaesthesia. Anticoagulation was initiated with heparin leading to a sufficient elevation of activated partial thromboplastin time (aPTT) >90 s. No stenosis had to be pre-dilated to position the stent. On average, the stent-PTA balloon was dilated for 15 s with nominal pressure of 6–12 atm within the vessel.

For prophylaxis of stent-thrombosis [14], all patients received 100 mg aspirin per day orally for lifetime and, aside from the first six patients, who were treated with ticlopidin, 75 mg clopidogrel per day for 6 weeks. All patients were examined by a neurologist before and after the intervention. MRI brain scan was performed within 48 h post-interventionally (T_2- and diffusion-weighted).

3. Results

The grade of stenosis, results, and complications of stent-PTA, and the results of follow-up examinations are presented in Table 1b. All interventions were successful; in five cases a residual stenosis ≤20% persisted. Intraprocedural dissections were encountered in two cases not causing clinical symptoms and were treated by additional stent placement. One patient developed a transient worsening of symptoms after the proce-