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Case Report Open Access

A Novel Technique for Carotid Artery Stenting: Double Stenting with Sequential Balloon Angioplasty: A Case Report and Technical Note

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Abstract

Cerebral embolism due to the plaque particles caused by stent implantation and balloon dilation is the most commonly seen complication of the carotid artery stenting procedure. After the stent implantation and/or balloon dilation, plaque fragmentation and protrusion through the interstices of the stent may cause cerebral embolism, periprocedural stent thrombosis, 30-day stroke or late in-stent stenosis. To reduce the plaque fragmentation and prevent plaque protrusion inside the stent, routine use of the double stenting with sequential balloon angioplasty as a novel technique, may be a solution.

Keywords: Carotid artery stenosis; Endovascular technique; Stent

Introduction

Carotid artery stenting (CAS) is a viable endovascular treatment modality alternative to carotid endarterectomy. Brain embolism due to the plaque particles caused by stent implantation and balloon dilation is the most commonly seen complication of the CAS procedure. The frequency of symptomatic or asymptomatic ischemic events seen on diffusion-weighted MRs can be reduced by using proximal or distal embolic protection devices [1,2]. After stent implantation and/or balloon dilation, plaque protrusion inside the stent may cause periprocedural stent thrombosis, 30-day stroke or late in-stent stenosis [3,4]. To reduce plaque fragmentation and prevent plaque protrusion inside the vessel lumen, the CAS technique was changed in our clinic. Almost all CAS procedures are performed using double stenting with sequential balloon dilation under distal protection. In this technical note, double stenting with sequential balloon dilation for CAS is presented and discussed.

Case Report and Technique

A 74-year-old male patient was referred to our clinic for evaluation for CAS. Doppler ultrasonography (US) and magnetic resonance angiography (MRA) showed ulcerated, calcified and eccentric plaque causing 50%-60% stenosis in the proximal part of the left internal carotid artery (ICA). The patient had a history of right transient hemiparesis, which had occurred three months previously. He was on medicine for hypertension and Type 2 diabetes mellitus. Daily, 100 mg of acetylsalicylic acid (Aspirin; Bayer healthcare, Germany) and 75 mg of clopidogrel (Plavix; Bristol-Myers Squibb/Sanofi Pharmaceuticals, NY, USA) were prescribed and the CAS was planned for approximately 10 days later. Before the procedure, Aspirin and Plavix sensitivity were tested with Verify Now (Accumetrics, San Diego, CA, USA). The patient was sensitive enough and one day later, the procedure was accomplished. Before the procedure, a diffusion-weighted MR was performed to detect any new ischemic lesion caused by the CAS procedure. Diagnostic angiograms obtained under local anesthesia showed 50% stenosis consistent with the Doppler US and MRA (Figure 1). Heparin (5000 IU) was administered intravenously (IV) and then the left common carotid artery was catheterized with a long shuttle sheath (Cook Inc., Bloomington, IN, USA) with the assistance of an exchange, hydrophilic 0.035-inch guide wire; a distal protection filter (Emboshield NAV6, Abbott, Redwood City, CA, USA) was placed inside the ICA, 4-5 cm away from the stenosis. A hybrid tapered carotid stent, Cristallo Ideale 9-6×30 mm in size (Invatec, Medtronic, and Santa Rosa, CA, USA) was implanted and a 4×20 mm monorail balloon was used for dilation after stent implantation. A second, identical carotid stent, the same size as the first stent, was deployed inside the second stent for covering and preventing the plaque protrusion inside the first stent. And then, a 5×20 mm monorail balloon dilation was applied and the opening was optimal without any residual stenosis (Figure 1). During the second balloon dilation, systolic blood pressure dropped from 155 to 90 mmHg and the pulse fell from 76 to 48/m. Atropine (0.5 mg) was administered IV urgently and at the end of the procedure the systolic blood pressure and pulse were 110 mmHg and 82/m, respectively. The filter was extracted and there was no debris inside the filter. The entrance site, the common femoral artery was closed with 6-F closure device (Angio-Seal, St. Jute Medical, Minnesota, USA) for hemostasis. The physical examination showed no neurological deficit. A heparin IV (750 IU/h) was ordered for 24 h and a diffusion-weighted MR was performed one day later. There were no new ischemic lesions. The patient was discharged without any neurologic problems. Aspirin was recommended indefinitely and Plavix for 6 weeks. The patient was neurologically symptom-free and Doppler US performed in the 1st, 3rd and 6th months showed no in-stent stenosis.

Discussion

Many prospective, randomized, controlled trials demonstrated the no inferiority of CAS compared to CEA, in both symptomatic and asymptomatic patients [5]. Despite the operator experiences and evolving technology that have decreased CAS complications, patients still are under considerable high complication rates due to some factors including lesion features [6,7]. Especially ulcerated, echo lucent, fibro fatty vulnerable plaques and plaques causing severe stenosis are more

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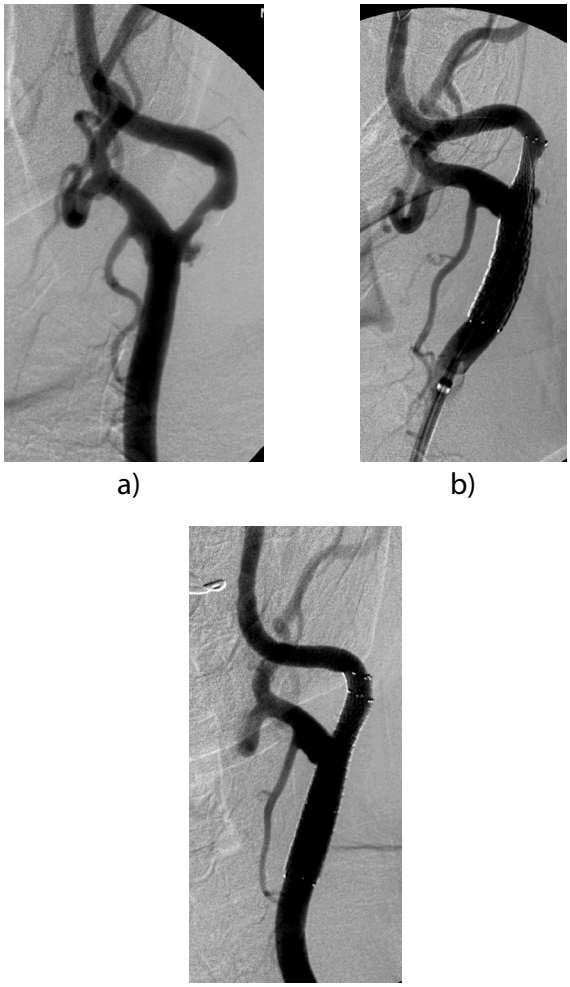

Figure 1a-c: Digital subtraction angiogram showing the ulcerated, eccentric, short, 50% stenosis in the proximal part of the left internal carotid artery next to the bifurcation (a). After stent implantation and dilation with a balloon 4-mm in diameter, a residual stenosis is seen (b). Figure c shows no residual stenosis after second stent insertion and dilation of a 5-mm balloon.

prone both to disruption and to causing cerebral embolism during the stenting and balloon dilation procedure, in spite of using protection devices [8]. In some cases, it is possible that the disrupted plaque particles may fill the protection filter and cause symptomatic neurologic complications as well as asymptomatic brain lesions, both of which may be visible on diffusion-weighted MR [9,10]. It is possible to decrease the embolic debris rate by using more metal coverage for plaque. Closed-cell design carotid stents have more metal coverage than open-cell ones. But even their increased coverage is insufficient to prevent plaque particles from escaping between stent wires or meshes. Implantation of another stent inside the first, in telescopic fashion as done in the treatment of wide-necked or fusiform cerebral aneurysms [11], is one method of increasing the metal coverage, as was done in the current case. But sequential balloon dilation also appears to be important to decrease the fragmentation of plaque. After the second stent insertion, the final desired opening of the stenosis is obtained by dilation of the final balloon to a diameter of 5 or 6 mm. If predilation is required, the balloon diameter must not exceed 3 mm. After the first stent insertion, dilation must be performed with a 4-mm balloon. Sequential dilation during the procedures appears to provide less disruption of the plaque, resulting in decreased production of embolic particles. So fewer distal embolic lesions are expected to be encountered than would have been possible with a single stent. In addition to periprocedural stroke, stroke occurring between 24 hours and 30 days after the procedure is also a major complication [3]. This suggests that stroke occurring as a complication of CAS cannot be attributed solely to technical problems.

The cause of late stroke after CAS is unknown; however, plaque protrusion may be one of the potential causes. The frequency of plaque protrusion into the stent after CAS is very low (4%) on angiography, but Shinozaki et al reported that the frequency of plaque protrusion into the stent was 7.8% by intravascular US in 77 consecutive CAS patients. Using angiography they saw plaque protrusion in only 2 patients [12]. In addition to plaque protrusion inside the stent, the other two major causes of the late in-stent stenosis seen in CAS cases are the neointimal proliferation caused by a self-expanding stent and small post-procedural stent dimensions resulting from inefficient balloon dilation [13]. Plaque protrusion may also cause acute stent thrombosis and subacute in-stent stenosis, which are rarely seen (only 0.04% to 2% of all cases) devastating complications [4]. The prevention of plaque protrusion inside the stent is important to decrease the restenosis, acute stent thrombosis and 30-day stroke rate. A way to decrease the 30-day stroke and restenosis rates which are caused by plaque protrusion inside the stent is to jail the plaques with an additional second stent, as was done in this case. I refer to this procedure as double stenting. In conclusion, double stenting and sequential balloon dilation with distal protection devices may be a new and potentially effective and promising technique for CAS to decrease cerebral embolism, periprocedural stent thrombosis, 30-day stroke, and late in-stent stenosis which are due to plaque fragmentation and plaque protrusion.

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