Angioplasty and stenting in the carotid and vertebral arteries

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Summary

Carotid and vertebral artery percutaneous transluminal angioplasty and stenting are new, experimental techniques. Their potential uses are discussed and the results and complications reported to date are reviewed.

Keywords: percutaneous transluminal angioplasty; stents

Treatment of carotid artery stenosis

Atherosclerotic carotid stenosis is responsible for 10% of ischaemic strokes but until recently there has been uncertainty about the benefit of treating carotid stenosis. However, two randomised controlled trials, the North American Symptomatic Carotid Endarterectomy Trial (NASCET)¹ and the European Carotid Surgery Trial (ECST),² have now convincingly established the benefit of carotid endarterectomy in preventing strokes in symptomatic patients. Both trials showed that the very high rate of stroke in patients with recently symptomatic severe carotid stenosis of 70% or more treated medically was significantly reduced by surgery. However, surgery has disadvantages including the use of general anaesthesia, damage to cranial and superficial cutaneous nerves from the incision, and problems from the scar. Carotid percutaneous transluminal angioplasty (PTA) avoids these disadvantages, can be used to treat surgically inaccessible lesions, and involves a shorter hospital stay. Interest has therefore developed in using PTA, with or without stenting, in the carotid artery, although neither technique has gained general acceptance because of uncertainty about the risks and benefits.

The main concern is that carotid PTA may result in a stroke at the time of the procedure.^{3,4} This may be caused by the guidewire or balloon catheter dislodging thrombus or a fragment of the atherosclerotic plaque, which embolises to the cerebral circulation. PTA also causes dissection which may occlude the vessel lumen, resulting in critical cerebral ischaemia, or promote thrombus formation and cerebral embolism.^{3,4} Despite these risks, a number of centres in Europe and North America have excellent early results of carotid PTA, although there are very few data on long-term outcome. Data on carotid angioplasty, available from several series totalling over 500 patients, give a similar rate of procedure-related stroke or death during carotid PTA as found as a result of carotid surgery in NASCET and ECST. The mean stroke rate at the time of the procedure for all the published carotid PTA series together is 1.5% for minor or non-disabling stroke and 2.1% for major stroke or death, resulting in an overall rate of 3.6%.⁵ Other potential complications of carotid PTA include groin haematoma and reaction to contrast used during the procedure.

Another concern about carotid PTA is that re-stenosis may result in symptom recurrence which, in the case of embolic stroke could be catastrophic for the patient. Re-stenosis after peripheral and coronary PTA may be associated with significant symptoms. However, claudication and angina are primarily related to a reduction in flow, while cerebral ischaemia from carotid stenosis is usually thromboembolic. Re-stenosis after carotid PTA may remain asymptomatic if it produces a smooth vessel lumen and embolisation does not occur.

Our current knowledge about the short- and long-term safety and efficacy of carotid PTA is limited by the lack of data from randomised controlled trials of carotid PTA versus carotid endarterectomy. The patients in the published series may have been highly selected with respect to degree of stenosis, eccentricity of the lesion, comorbidity from other pathology and so on, making it impossible to compare the low immediate complication rate of carotid PTA with surgery. There is no long-term follow-up of the efficacy of the procedure. The results from the only on-going trial, the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS), in which the majority of patients have had simple angioplasty without stenting,⁶ should be available early in 1998. Until data from randomised trials are available, cerebrovascular PTA will remain an experimental procedure.

Carotid stenting in the management of carotid stenosis

It is unclear whether the routine use of stents has anything to add to simple balloon PTA in the management of carotid stenosis. It has been suggested that

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Case 1 Internal carotid artery stenosis treated with carotid PTA and stenting

A 77-year-old man presented with a left hemisphere infarct from which he had made a full recovery. Carotid Doppler and magnetic resonance angiography showed a 99% stenosis of the origin of the left internal carotid artery which was referred for PTA. The lesion (figure 1) was crossed with a Compass wire and dilated with a 6 x 40 mm balloon. After dilation there was some subintimal dissection with calcified plaque and a residual stenosis of 70% (figure 2). Because of the inadequate result, a Strecker 6 x 40 mm stent was placed across the residual stenosis with excellent result (figure 3). The patient was heparinised for 24 h, then started on dipyridimole and continued on aspirin. The patient was asymptomatic during the procedure. Six-month Doppler follow-up showed no haemodynamically significant stenosis. He remains asymptomatic eight months later.

Figure 1 Stenosis of the internal carotid artery

Figure 2 Following PTA: subintimal dissection, calcified plaque and a 70% residual stenosis

Figure 3 After stent insertion, no residual stenosis

stenting should be the interventional treatment of choice for carotid stenosis because the initial results are superior.⁷⁻⁹ Stents are beginning to be used in the endovascular management of carotid stenosis in some centres in North America. The ideal stent used in the carotid artery should be radio-opaque and easily visualised. There should be little or minimal shortening when the stent is deployed, to enable accurate placement. The stent should be available in a range of sizes and should be non-thrombogenic. Stents which are available for use in the carotid artery include those which are balloon expandable (eg, 'Palmaz' and 'Strecker') and those which are self-expandable (eg, the 'Wall stent'). Preference for the type of stent used varies according to the individual radiologist.

Most symptoms due to carotid stenosis are thromboembolic rather than haemodynamic. During PTA the atherosclerotic plaque is ruptured. Re-endothelialisation subsequently occurs, replacing the irregular plaque surface by a smoother, less thrombogenic surface. Thus an incomplete dilation may not be important if PTA alters the thrombogenicity of a lesion. Furthermore, we have shown that a process of remodelling occurs in most carotid arteries in the year after PTA, resulting in improvement in residual stenosis of up to 49%.¹⁰ Stents may not therefore be necessary in the majority of patients in whom balloon angioplasty is straightforward. However, where simple balloon angioplasty does not produce a good result, carotid stents may be useful in improving the anatomical outcome. These situations include stenoses in which the lesion is heavily calcified so that the stenosis does not dilate well after balloon inflation, and when angioplasty has resulted in significant dissection ('bail-out stenting'). Our vascular team also uses stents when the initial angioplasty has reduced the stenosis by less than 20%, as in our experience these do not remodel.

Case 2 Recurrent vertebral artery stenosis treated with PTA and stenting

A 73-year-old woman presented in December 1994 with a cerebellar infarction. She made a full recovery and catheter angiography in March 1995 showed bilateral vertebral artery stenosis with normal carotid arteries. The left vertebral artery was very atretic and the stenosis unsuitable for angioplasty. The right artery stenosis was suitable for angioplasty. This was performed in April 1995 and a good result achieved. She continued on aspirin. She had no further symptoms until May 1996 when she had a sudden onset of dizziness, vertigo and nausea which lasted for a hour. Catheter angiography showed re-stenosis of the right vertebral artery (figure 4) and it was decided to redilate and stent the stenosis. The lesion was crossed with a 014 guidewire. A 4×20 mm balloon was inflated to deploy a 4×18 mm arteriovascular engineering microstent across the stenosis with excellent result (figure 5). She was heparinised and then warfarinised for three months. She remains asymptomatic 12 months later.

Figure 4 Vertebral artery origin re-stenosis

Figure 5 After stent insertion

Carotid stenting appears to be relatively safe, with complication rates similar to those reported for simple PTA. Peri-procedure complications in almost 500 patients with carotid artery stenosis treated with stenting have been reported by a group in the US as a mean rate of major stroke or death of 3.9% and a mean rate of minor stroke of 2.2%.¹¹ Long-term results have not been reported. There is concern that carotid stents may stimulate excessive neo-intimal hyperplasia, that subsequent carotid surgery, if needed, may be made difficult, and that some types of stents may collapse as a result of minor pressure on the neck. Stents also add considerably to the expense of the procedure. Further research is therefore needed and randomised controlled trials comparing carotid stenting and surgery are currently being planned.

Other uses of carotid stents

The treatment of carotid artery dissection is another potential use of carotid stents. In this condition, which is responsible for about 5% of strokes in patients under the age of 60 years, the intima of the artery develops a tear, either spontaneously or after trauma, and forms a 'flap' within the vessel lumen. Symptoms include Horner's syndrome, carotidynia (pain in the neck in the carotid distribution) and cerebral ischaemia. There are no randomised controlled trials of management of carotid dissection, but the alternatives are anticoagulation for a variable (and uncertain) period, surgical bypass procedures and proximal vessel ligation. The surgical options are usually only employed if ischaemic symptoms recur on anticoagulation, or if imaging shows progression of the thrombus or aneurysm formation. Carotid artery stents could be used instead of surgery. Theoretically, the stent will hold the dissection flap against the vessel wall, maintaining a patent lumen and preventing arterial occlusion secondary to the flap. There are a few case reports of the use of a stent to occlude dissecting carotid aneurysms.¹² Trials of the management of carotid dissection, including the use of stents, need to be done.¹³

Stents in the management of vertebral artery stenosis

The value of stents in the treatment of vertebral artery stenosis is uncertain. Surgery for vertebral artery stenosis is rarely carried out because surgical access to the vessel is poor. There is very little information available about the natural history of vertebral artery stenosis but it is reasonable to assume that vertebro-basilar ischaemia distal to severe stenosis of the vertebral arteries will recur as frequently in the vertebrobasilar circulation as in the carotid. Stenoses often occur at the vertebral artery origin which is easily accessible to endovascular techniques and so endovascular management may become the treatment of

choice for these lesions. Series including 82 vertebral arteries treated with PTA show a procedure-related complication rate of 3.7% for major stroke. There were no reported minor strokes.¹⁴⁻¹⁶ The stenoses treated were in the proximal and mid portion of the vertebral artery. There is no long-term follow-up in most of these patients and there are no trials comparing surgery with medical care. Results from CAVATAS comparing angioplasty with or without stenting with medical care for vertebral artery stenosis are not yet available.⁶

Difficulty in achieving an adequate dilation of the origin of the vertebral artery with PTA and a possible high re-stenosis rate at this site (three out of 34 arteries in one series of which two were symptomatic,¹⁶ and three out of four in our experience, one of which was symptomatic) have encouraged the use of stents at this site. In one series of three patients who developed re-stenosis after vertebral PTA and who subsequently underwent repeat PTA with stenting, angiographic follow-up one year after stenting showed no re-stenosis.¹⁷ Vertebral artery origin stenosis may be analogous to ostial renal artery stenosis. At this site, conventional PTA has a high failure rate due to elastic recoil but it appears that this problem may have been overcome by the use of stents.¹⁸

Conclusion

The endovascular treatment of carotid and vertebral artery disease is a new and exciting area with increasing potential uses of both simple balloon angioplasty and stents. However, their use remains experimental and the results of randomised controlled trials are required before they can enter general use.

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