REVIEW ARTICLE

Richard P. Cambria, MD, Section Editor

Clinical relevance and treatment of carotid stent fractures

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Objective: **To review all published reports and investigate the clinical relevance and need for treatment of carotid stent fractures.**

Methods: **Electronic and hand-searching of the published literature and the Manufacturer and User Facility Device Experience (MAUDE) database.**

Results: **Thirteen articles were published. There are 10 case reports and 3 clinical studies. There are 26 reports of fractured stents in the MAUDE database. Fifty-five cases of carotid stent fractures are reported in total. A total of 201 carotid stents were examined in the 3 studies, and the incidence of fractures was 8.9% (18/201). Fractured stents were 22 Xact, 20 Acculink, 6 Precise, 2 Exponent, 1 Nexstent, 1 Genesis, 1 Symbiot, and 2 nonspecified nitinol self-expandable stents. Twenty-seven of the treated carotid lesions were atherosclerotic, 3 restenoses after carotid endarterectomy, 2 postradiational, 1 pseudoaneurysm, and 22 lesions of unknown pathology. Calcification was reported in 15 of the 27 atherosclerotic lesions (55.5%). Time from implantation to fracture ranged from 0 days (fracture during implantation) to 37 months. In 55% of the cases, stent fracture was associated with restenosis. Six patients presented with symptoms. Treatment was reported for 32 patients: 14 patients underwent de novo stent placement, 2 balloon angioplasty, 2 carotid endarterectomy, 2 bypass graft (1 vein, 1 polytetrafluoroethylene), 1 anticoagulation, and 11 patients were followed up.**

Conclusion: **Carotid stent fractures are mainly reported in self-expandable nitinol stents. Plaque calcification may be a risk factor for stent fractures. No difference was observed between open and closed-cell design. Stent fractures were often associated with restenosis and usually were asymptomatic. The actual incidence, clinical relevance, and optimal treatment remain to be clarified from larger prospective studies designed to investigate the issue. (J Vasc Surg 2010;51:1280-5.)**

Carotid artery stent (CAS) placement with embolic protection is an alternative treatment for patients with internal carotid artery stenosis who are at high risk for carotid endarterectomy. Most of the studies reporting on the long terms of CAS focus on clinical outcomes and restenosis rate. However, detailed information of the behavior of stents in the carotid artery is still lacking. The Food and Drug Administration (FDA) has long been concerned with the durability of stents. As the use of CAS expands and the number of reported incidences of complications related to this procedure grows, it seems necessary to assess the incidence and clinical implications of stent fractures after CAS. The aim of this study is to review all published reports and investigate the clinical relevance and need for treatment of carotid stent fractures.

METHODS

An electronic search of the published literature in PubMed from 1990 up to December 2009 was conducted. The terms used for this were stent fracture, stent breakage, carotid, stenosis. Further hand-searching of the relevant references of those articles retrieved from electronic searches was made. The Manufacturer and User Facility Device Experience (MAUDE) database was also searched (http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfMAUDE/search.CFM). The terms used were fracture and break for product problem and carotid stent for product class. Date reports received by FDA: 01/01/1997 to 12/29/2009. The obtained reports (67 in total) were reviewed, and those describing carotid stent fractures were included in the analysis. The reports that

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Table. Reported cases of carotid stent fracture

Author	*N*	*Carotid stenosis*	*Calcification*	*Stent type*
Valibhoy[2]	1	Atherosclerotic	—	Xact
Surdell[3]	1	Atherosclerotic	—	Nitinol
Ling[4]	14	Atherosclerotic	8	Xact (8) Precise (4) Exponent (2)
Diehm[5]	1	Atherosclerotic	—	Xact
Nazzal[6]	1	Atherosclerotic	—	Acculink
Varcoe[7]	1	Atherosclerotic	1	Precise
Habib[8]	1	Atherosclerotic	1	Acculink
Boehm[9]	2	Atherosclerotic, postradiation (CCA)	—	Nextstent, Genesis
Ross[10]	1	Postradiation		Xact
Abullage[11]	1	Restenosis		Xact
Saad[12]	1	Restenosis		Nitinol
deVries[13]	1	Pseudoaneurysm		Symbiot
Chang [14]	3	Nr	2	Xact (3)
MAUDE Database	26	Atherosclerotic (6) Restenosis (1)	5/6	Acculink (18) Xact (7) Precise (1)

BE, Balloon expandable; *CAS,* carotid stenting; *CE,* carotid endarterectomy; *CTA,* computerized tomographic angiography; *DSA,* digital substraction angiography; *DUS,* duplex ultrasound; *MAUDE,* Manufacturer and User Facility Device Experience; *PTA,* percutaneous transluminal angioplasty; *Nr,* not reported; *SE,* self-expandable.

did not provide adequate data and those describing fracture of other parts of the device were excluded.

Carotid stent fractures were classified according to the Nitinol stent fracture classification proposed by the Cardiovascular Institute of the South (CIS) for Nitinol stent fracture standardization.[1] Type I fractures involve a single strut fracture only, type II fractures involve multiple Nitinol stent fractures that can occur at different sites, type III fractures involve multiple Nitinol stent fractures resulting in a complete transverse linear fracture but without stent displacement, and type IV fractures have a complete transverse linear fracture with stent displacement.

RESULTS

Thirteen articles were published. There are 10 case reports and 3 clinical studies. There are 26 reports of fractured stents in the MAUDE database. Fifty-five cases of carotid stent fractures are reported in total. Twenty-seven of the treated carotid lesions were atherosclerotic, 3 restenoses after carotid endarterectomy, 2 postradiational, 1 pseudoaneurysm, and 22 lesions of unknown pathology. Calcification was reported in 15 of the 27 atherosclerotic lesions (55.5%). All but one (Genesis; Johnson & Johnson, New Brunswick, NJ) were related to self-expandable stents. Fractured self-expandable stents included 22 Xact (Abbott Laboratories, Abbott Park, Ill), 20 Acculink (Abbott Laboratories), 6 Precise (Cordis, Miami Lakes, Fla), 2 Exponent (Medtronic Vascular, Santa Rosa, Calif), 1 Nexstent (Boston Scientific, Natick, Mass), 1 Symbiot self-expanding covered stent (Boston Scientific), and 2 nonspecified nitinol self-expandable stents. Twenty-three of the 52 specified fractured self-expandable stents (44%) had closed-cell design. There are 6 type I, 11 type II, 9 type III, and 6 type IV fractures; the rest are not described. Time from implantation to fracture ranged from 0 days (fracture during implantation) to 37 months. There are six reported cases of fracture during implantation, all associated with Acculink stents. In 55% of the cases (25/45), stent fracture was associated with restenosis. Six patients presented with symptoms. Two presented with contralateral stroke, two with neck hematoma, one had drop attacks during head movement, and in one patient, symptoms were not described. In a case of stent fracture during implantation, the filter embolic protection device was captured by broken stent struts during retrieval. Treatment was reported for 32 patients: 14 patients underwent de novo stent placement, 2 balloon angioplasty, 2 carotid endarterectomy, 2 bypass graft (1 vein, 1 polytetrafluoroethylene), 1 anticoagulation, and 11 patients were followed up (Table).

Three clinical studies reported on the incidence of carotid stent fractures presenting conflicting results: Ling et al performed a retrospective study of patients from one surgeon's private practice. They reported an incidence of stent fractures of 29.2% (14 of the 48 stents examined). The presence of calcified vessels was significantly associated with the presence of fractures. Fractures occurred 7.7 times more frequently in calcified lesions.[4] Varcoe et al reported an incidence of 1.9% of carotid stent fractures (1 of the 53 stents examined). It was a type I fracture. They also noted that another six stents had an irregular tiled appearance, similar to fish scales, that can be confused for stent fracture.[7] Chang et al evaluated 100 stents with neck films; there were 50 closed-cell stents and 50 open-cell stents. They found three stent fractures that occurred in closed-cell Xact stents placed >1 year prior to examination (3 of 100 or 3% overall incidence, 12% for Xact stents). No other

Table. Continued.

Time to fracture (months)	*Diagnosis*	*Type of fracture*	*Restenosis*	*Symptoms*	*Treatment*
6	DSA	III	+	−	CE
6	DSA	III	+	Nr	CAS
4 to 37 (mean, 18)	Plain radiography, DUS	I:3 II:6 III:2 IV:2	3/14	Nr	Nr
12	DUS	III	+	−	None
12 days	DSA	II	+	−	Anticoagulation
23	Multiplanar radiography, selective DUS	I	−	−	None
0	Intraoperative DSA	II	−	−	None
4	CTA	IV	+	+	CAS
17	CTA	III	+	−	CAS
5	DUS	III	+	−	CE
7	CTA	IV	+	−	CAS
3	CTA	IV	+	−	None
7	CT	—	−	+	PTFE graft
>12	Multiplanar radiography	Nr	Nr	Nr	Nr
0-24 (mean, 9)	DSA (9) Intraoperative DSA (6) Plain radiography (2)	I (2) II (3) III (2) IV (1)	13/20	4/20	CAS (10) PTA (2) Vein graft (1) None (7)

stents were fractured. Two of the three stent fractures were associated with the presence of calcified plaque noted on plain films.[14] A total of 201 stents were examined in these 3 studies, and 18 of them were reported fractured (8.9%).

DISCUSSION

Stent fractures have been described in the aorta; the vertebral, coronary, renal, pulmonary, femoral, popliteal, and tibial arteries, the biliary tract; hemodialysis fistulas; and coronary saphenous vein grafts.[15-25] Fractures in the femoropopliteal segment are associated with high restenosis rates and reduced patency.[21] Stent fracture has been reported to occur in areas with flexible arteries and in those with repetitive movements, such as the knee area. Carotid artery is lately recognized as a potential site of stent fracture because of the rotation and flexion forces.

Factors contributing to stent fracture include external mechanical forces such as arterial shortening, axial rigidity, kinking, and compression.[26] Implantation of a carotid stent was shown to be associated with a substantial reduction of arterial compliance of the extracranial arteries. Pulsatile blood flow through the stent can transmit extensive internal stress, contributing to stent fracture, especially near the heart or great vessels.[27] However, in vitro examination of nitinol stents concluded that strains imposed by pulsatile motion are not sufficiently high to cause fatigue, and are therefore not principally responsible for stent fractures observed in the superficial femoral artery.[28] The length of the stented segment has also been proposed as a factor in stent fracture. The longer the stented segment and the more overlapping stents are used, the more liable the stents are to fracture.[21]

The carotid bifurcation is located in a highly mobile part of human anatomy, such as the femoropopliteal segment. The neck may rotate around the central axis of the cervical spine and perform flexion and extension over the length of cervical vertebrae. Head and neck movements expose a carotid stent to forces of axial and crush deformation.[29] Vos et al determined the effect of head movement on in situ carotid stents using three-dimensional time-of-flight magnetic resonance angiography. They found that carotid artery loses its natural flexion and extension and rotational flexibility following CAS.[30] Furthermore, the ex vivo flexibility properties of the stents themselves are lost when placed in vivo. Therefore, the stented segment of the artery appears as a stiff, inflexible unit in all head positions. The unstented segments of the vessel are left to accommodate head movements, resulting in sharp angulations of up to 85 degrees at the junctions between the stent and artery.[30] The carotid artery is only partially able to accommodate the changes in geometry that result from physiologic movements of the head, which may lead to kinking at the distal end of the stent. Also, they found that the common carotid artery and the internal carotid artery are subjected to considerable torsion shear with head turning. These vessels present a physiological tendency to twist around each other, because of the differences in outflow resistance between the internal and external carotid arteries, creating a further torque and shear force. All of these shear forces lead to friction at both ends of the stent, place ongoing stress on the stent, and can possibly be a contributing cause to fracture.[30] Additionally, elastic recoil owing to pharyngeal muscle contraction and external musculoskeletal compression are added

forces that are expected to contribute to stent fracture in the carotid artery.[6] Swallowing results in a medial crush of the stented artery due to contraction of the pharyngeal constrictor muscles.[29]

Heavy calcification of the carotid artery has been reported as an important predictor of complications following carotid artery stenting. The calcification impedes complete expansion of the stent. This can lead to an acute fracture of the stent, sometimes responsible for in-stent restenosis.[4,31] The presence of calcification exerts an external loading force on the stent. Because of the irregularity of calcified plaques, focal pressure is placed on certain parts of a stent in situ, and this could be exacerbated by neck movements. A calcified vessel also creates a more rigid artery and a point of fixation on the stent in situ, necessitating that the proximal and distal unstented segments of artery accommodate in-neck movement. This creates regional friction, increasing the risk of fracture.[4] Plaque calcification is reported in 55.5% of the atherosclerotic cases with stent fractures.

Fatigue and fracture of stents originate at stress concentration regions that are under alternating external loading modes (pressure, bending moments, and torsion moments). The external loading produces tensile background stresses, which accumulate material defects.[32] The microscopic defects, after many load cycles, produce microcracks, which enlarge to macrocracks under favorable environmental and stress conditions. The rate of microcrack enlargement depends mainly on the material of which the stent is made. Once a crack has reached a critical size, there can be an abrupt final fracture that severs the structural continuity of the stent.[33]

From the structural point of view, the stent is a cylindrical shell. Calcification plaques form on this shell and can cause severe stress concentrations at locations where the plaques end. The plaques have a different (typically higher) elastic modulus from the stent. Such elastic mismatch leads to stress concentrations. Pulsative internal blood pressure, but also bending and torsion, can cause very high tensile stresses in the vicinity of the plaque-stent boundary.[34] Fatigue cracks can initiate, enlarge, and finally become critical after a sufficient number of pulses.

Stents with closed-cell design have overlapping or fully connecting struts, as compared with stents with open-cell design, which have both connecting and nonconnecting struts. The former have higher surface coverage, but there are no studies demonstrating a difference in fracture rates between the two designs in other arterial beds. In carotid arteries, similarly, there is no difference in fracture rates.

Detecting stent fractures may be more challenging than anticipated. Stent fractures may not be adequately detected with the unmagnified anteroposterior and lateral radiographic views. Plain radiographic images have an uncertain sensitivity and specificity even at magnified levels.[4] Scrutinizing the fine struts of these small stents requires magnification and removal of interference from overlying bony structures.[7] Open-cell stents may present an irregular tiled appearance similar to fish scales that can be easily confused for stent fracture.[7] Doppler ultrasound is the common examination performed in the follow-up after CAS, but it is an indirect imaging method measuring flow rates, and there is no possibility to directly visualize the stent. Stent fractures cannot be identified by ultrasound.[9] In patients with suspected restenosis by ultrasound, it is recommended to use computed tomography angiography to evaluate the stents' lumen and depict the reason for the restenosis.[9] Rotational angiography without contrast can identify carotid stent fractures because it provides the possibility to magnify and to remove the interference of bony structures by changing the viewing angle. A proposed algorithm for the detection of carotid stent fractures could be the following: intraoperatively, during completion angiography, a magnified picture of the stent should always be obtained, and it should be carefully inspected for strut fractures, especially in heavily calcified lesions. This picture will be used as a future reference image. In the postoperative follow-up, in addition to duplex ultrasound, unmagnified anteroposterior and lateral radiographic views should be performed at 12 months and yearly thereafter. Patients with ultrasonographically-detected restenosis and patients with suspected stent fracture in the plain radiography should be subjected to rotational angiography without contrast.

There is a striking disparity in carotid stent fracture rates between the three published clinical studies. The group from Dartmouth reported a 3% total incidence of stent fractures, with all fractures occurring to Xact stents.[14] The group from Westmead, Sydney reported an incidence of 1.9%,[7] with a single fracture that occurred in a Precise stent. Contrarily, the group from Perth reported a remarkably higher incidence of 29.2%; 14 fractures occurring in 8 Xact, 4 Precise, and 2 Exponent stents.[4] This disparity is difficult to explain. Plaque calcification could be a factor affecting the reported incidence of fractures. The study from Perth provides adequate data regarding plaque calcification: 8 out of the 14 fractured stents were associated with local calcification in the region of the deployed stent, whereas 5 out of the 34 stents without a fracture had localized calcification. The presence of calcification resulted in a 7.7-times greater odds for a fractured stent.[4] The study from Dartmouth reports that two of the three stent fractures were associated with the presence of calcified plaque noted on plain films but does not provide any data about the total number of patients with calcification.[14] The study from Sydney does not provide any data about plaque characteristics. Moreover, 21% of the patients had radiation-induced stenosis and postendarterectomy restenosis, considered uncalcified lesions.[7] It is possible that the latter two studies included a smaller number of patients with significant plaque calcification; consequently, the number of stent fractures was decreased. Another factor that could affect the results is the type of stent used. The

two stents most frequently fractured, Xact and Acculink, were not used in the study from Sydney.

The clinical relevance of carotid stent fractures in association with restenosis and stroke has not been clarified yet. In this review, we found that 55% of the reported fractures that provided adequate data (25/45) were associated with restenosis. However, in most cases, restenosis was initially found and, during further investigation, stent fracture was discovered. We do not exactly know what percentage of carotid stent fractures lead to restenosis. Ling et al reported that, although this series of carotid arterial stent fractures had a fracture rate of 30%, the majority were benign at the time of this writing; only 3 of these had restenosis >50% on surveillance ultrasound scans at an average of 12 months postimplantation. Type IV fractures, which by definition are displaced, did not have restenosis detected.[4] However, not all fractures are benign and can progress to a clinically significant stenosis. Furthermore, we do not know how many of the restenoses produce symptoms. In our review, we found six patients that presented with symptoms, and only two of them had suffered an ipsilateral ischemic stroke.

To date, there is limited information regarding optimal management of carotid stent fractures. In asymptomatic patients without restenosis, follow-up seems to be a reasonable treatment. Eleven of the 32 patients with available data regarding treatment were followed up. When fracture is complicated with thrombus formation, anticoagulation is needed. In this case, restenting should be avoided because it might be associated with significant risk of distal embolization. The presence of neurologic symptoms or in case of severe in-stent restenosis, restenting of the lesion can be performed by a bridging nitinol self-expanding stent. This is an acceptable approach for long-term restenosis rather than early stent fracture with thrombus formation.[6] However, the insertion of another stent would further stiffen the carotid segment, increasing the susceptibility to further stent fractures. De novo carotid stenting was the procedure most often performed in these patients. Fourteen of the 32 underwent carotid stenting, and 2 underwent balloon angioplasty. In the presence of impedance to the blood flow, neurologic deficit, stent penetrating the carotid wall, or dissection of the carotid artery, open surgery with stent removal is an alternative option.[6] Successful endarterectomy was reported in two cases after carotid stent fracture.[2,10] Two cases of bypass graft using vein and polytetrafluoroethylene have also been described.[13]

After consideration of the potential for stent fractures in patients that underwent CAS and the uncertainty regarding its incidence, clinical outcome, and treatment, it is necessary to investigate the issue in large prospective studies in patients treated only for atherosclerotic disease, taking into account the plaque type, the specific type of stent, and the carotid anatomy using specific radiographic and ultrasonic assessment.

AUTHOR CONTRIBUTIONS

Conception and design: GS, AD
Analysis and interpretation: GS, AK, CK
Data collection: GS, AK, CK
Writing the article: GS, AD, AG
Critical revision of the article: GS, AD AG
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