

Stenting for carotid artery stenosis: Fractures, proposed etiology and the need for surveillance

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Purpose: Carotid artery stenting is a relatively new intervention for the treatment of carotid artery stenosis, and the long-term outcomes and complications are therefore yet to be determined. In one surgeon's practice, it was found that a stent fracture was the etiological factor for recurrent stenosis. A retrospective study was therefore performed with the hypothesis that carotid stent fractures are common. The aims were to determine prevalence of fractures in this surgeon's series, risk factors, and most importantly, clinical relevance.

Methods: Patients from one surgeon's private practice who had carotid stenosis deemed suitable for intervention (>80% asymptomatic, >70% symptomatic, 50% to 70% if an ulcerated lesion) and had suitable aortic and carotid morphology for carotid stenting between March 2004 and December 2006 were included. To enhance the quality of the measurement, two vascular surgeons and one radiologist examined the films independently to determine if there was a fracture present. Given that this was a retrospective study, there was no preconceived sample size determined.

Results: Fracture prevalence was found to be 29.2% or 14 out of 48 stents. Restenosis occurred in 21% of those stents with a detected fracture, after an average follow-up of 15 months. Several aetiological factors are proposed, with a finding in this series, of a strong and significant association between the presence of calcified vessels and the presence of fractures (odds ratio 7.7; standard error 5.6; 95% confidence interval 1.9-32.0, $P = .003$).

Conclusions: Although this is a small study, it demonstrates that carotid stent fractures do exist, and importantly, not all of them are benign. Therefore, the authors recommend regular surveillance with plain radiography in addition to duplex ultrasonography to enable early detection of fracturing. Following detection, institution of increased surveillance frequency and/or any appropriate intervention can be implemented, to aid in the prevention of complications resulting from restenosis should it become apparent. (J Vasc Surg 2008;47:1220-6.)

Carotid endarterectomy (CEA) is the current gold standard for the management of patients with high-grade carotid stenoses, being superior to best medical therapy, as established by several landmark-randomized trials.¹⁻³ However, since the first successful CEA by DeBakey in 1953, an alternative therapy has emerged during the last decade that has created new controversy – carotid artery stenting (CAS). This technique is reported to avoid certain complications of endarterectomy such as cranial nerve injury, wound complications, and discomfort of open surgery. However, CAS is not without its own complications.

The index case that prompted this series⁴ was noted accidentally during the deployment of a contralateral stent. The residual stenosis post-deployment was less than 10%. A 6-month surveillance scan of the stented side showed a stenosis of approximately 60%. The 9-month scan showed an increase in its severity, with stenosis of 70% and 3 months later, there was a further increase, with a stenosis of 90%. The degree of stenosis was calculated using the internal carotid artery to common carotid artery (ICA:CCA)

ratio. The stent was therefore removed surgically with a carotid endarterectomy without complication.

A retrospective analysis was therefore performed with the hypothesis that carotid stent fractures are common. The aims of this study were to determine the prevalence of stent fractures, the risk factors for fracturing and whether the presence of a fracture carries any clinical significance.

METHODS

Patients from one surgeon's practice who had carotid stenosis deemed suitable for intervention (>80% asymptomatic, >70% symptomatic, 50% to 70% if an ulcerated lesion) and had suitable aortic and carotid morphology for carotid stenting (appropriate access vessels, type I, II, and III arches except type III arches with a stenosis of the brachiocephalic or carotid orifice) between March 2004 and December 2006 were included. To enhance the quality of the measurement, two vascular surgeons and one radiologist examined the x-rays independently to determine if there was a fracture present. Given that this was a retrospective study, there was no preconceived sample size determined.

CAS was performed in the angiography suite, with an anesthetist present. Under local anesthesia, with minimal or no conscious sedation, percutaneous access was obtained using a Seldinger technique in the right common femoral artery, or rarely, through the left common femoral artery. Aspirin (100 mg/d) and clopidogrel were started 3 days before the procedure if the patient was not already taking it.

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Clopidogrel was continued after CAS (75 mg/d for 3 months), together with aspirin (100 mg/d indefinitely).

Patients received 5000 units of heparin intravenously in total, which was administered during the diagnostic portion of the procedure. Blood pressure and electrocardiogram were monitored continuously. Thoracic arch aortography with angiography of the extracranial and intracranial carotid arterial tree was done routinely with lateral and anteroposterior views. A cerebral protection device was used in all cases and positioned a suitable distance beyond the distal end of the stenosis. Stenosis and vessel measurements were calculated using the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria of minimal luminal diameter compared with the distal internal carotid artery diameter and lesions were predilated with a 2 or 3 mm balloon. The sizing of the stent was calculated by computer software, and oversizing of the stent was 1 to 2 mm greater than the ICA diameter, except if the CCA was large, then no oversizing was included. Self-expandable stents were deployed and postdiluted with a balloon diameter that was calculated by computer software, and never greater than 5 mm. This was to prevent plaque protrusion through the stent struts with potential subsequent embolus, and also it was felt that the self-expanding nitinol stents were able to exert sufficient chronic outward force to negate the need for using a balloon more than 5 mm in diameter. Completion ipsilateral selective carotid (extracranial and intracranial) angiograms were obtained, and a residual stenosis of <20% was accepted to be technically satisfactory. Patients were transferred to the intensive care unit for neurovascular observation for 24 hours, and no heparin reversal was performed. Carotid duplex scan was performed at 1, 3, and 6 months, and yearly thereafter. After the discovery of stent restenosis with concomitant fracture in the index case, high-resolution digital images of plain radiographs were organized for all patients who underwent this procedure. Subsequent ultrasounds were performed every 6 months for detected fractures, and other stents continue to be under surveillance with plain films every 6 months. Stent restenosis was defined as more than 50% diameter reduction or the presence of occlusion. Widely accepted criteria for the interpretation of restenosis of a carotid stent are yet to be set in the published literature; a peak systolic velocity ratio (intra-stent/prestenotic) of greater than or equal to 2.0 was considered to be indicative of a restenosis of more than 50% diameter reduction, using the duplex ultrasound criteria for grading carotid stent stenosis of Zwiebel from the University of South Florida.⁵

Statistical analysis. All analysis was performed using SAS version 8.2 (SAS Institute Inc, Cary, NC, USA). Comparisons of proportions were made using χ^2 tests for equal proportion or Fisher exact tests where numbers are small. A two-sided *P* value of .05 was considered to be statistically significant.

Fracture classification. A Nitinol stent fracture classification has been proposed by the Cardiovascular Institute of the South (CIS) for Nitinol stent fracture standardiza-

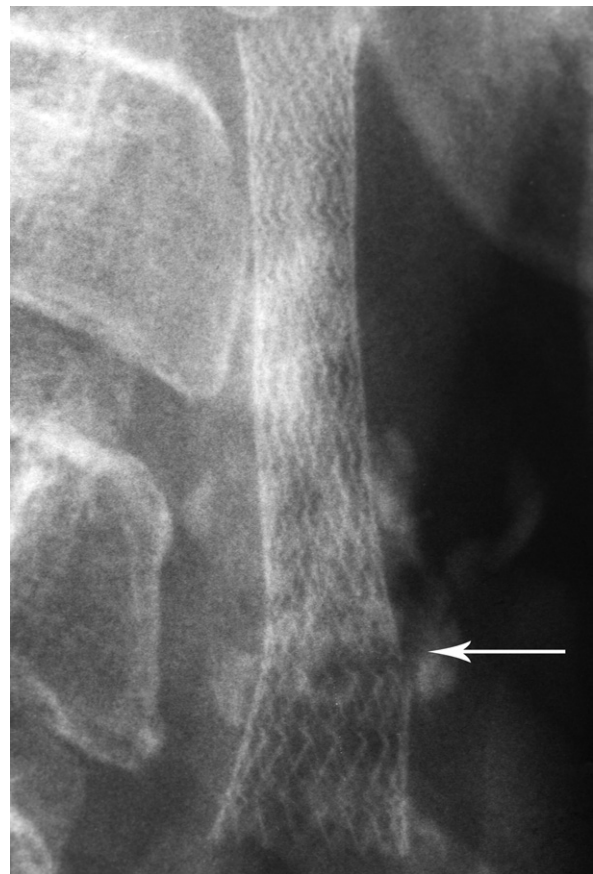


Fig 1. Type I fracture (*arrow*). This was an XAct 7-9 mm \times 40 mm stent placed in a 74-year-old male. Note the presence of adjacent calcification.

tion,⁶ which can be applicable to carotid stents. Type I fractures involve a single strut fracture only (Fig 1), type II fractures involve multiple Nitinol stent fractures that can occur at different sites (Fig 2), type III fractures involve multiple Nitinol stent fractures resulting in a complete transverse linear fracture but without stent displacement (Fig 3), and type IV fractures have a complete transverse linear fracture with stent displacement (Fig 4).

RESULTS

Patient demographic details. Sixty-one carotid arteries were treated in 54 patients. Eleven patients were lost to follow-up (two patients had bilateral stents, and nine patients had a unilateral stent), leaving a total of 48 stents in 43 patients. Patient demographic details and comorbid conditions are displayed in Table I. High resolution digital images were obtained and specific coned views of the stents were requested. Where possible, the carotid stent was placed parallel to the x-ray receptor, with an antero-posterior and lateral views obtained. It was requested that cervical vertebrae be excluded from the line of projection, however, this was not always adhered to. Despite this, the quality of the

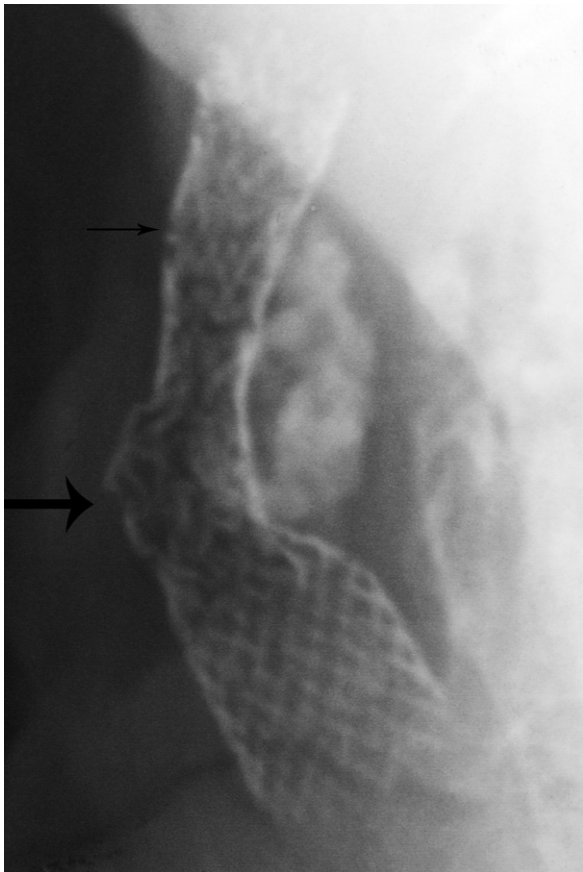


Fig 2. Type II fracture. The presence of two or more fractures, with obvious discontinuity of the stent margin (*arrows*). This is a Precise 8 mm × 40 mm stent placed in a 76-year-old female. The adjacent calcification is causing an obvious lateral displacement of part of the stent (*large arrow*).

images was good enough to enable three independent examiners to detect a fracture.

Primary atherosclerotic lesions were treated in 41 patients and recurrent stenosis after previous CEA in two (7%) patients. A history of stroke was present in three (7%) patients and previous transient ischemic attacks or amaurosis fugax in 21 (49%) patients. Nineteen (44%) had asymptomatic disease. Technical success was achieved in 48 cases. Five out of 48 patients experienced morbidity associated with the procedure; one retroperitoneal bleed required operative intervention, two patients suffered a transient ischemic attack (which resolved completely in 12 hours), and one patient suffered a stroke with residual disability. One patient died from myocardial infarction on postoperative day ten.

In this series of 48 carotid artery stents, 29% ($n = 14$) of the carotid stents implanted had a fracture detected on follow-up plain radiography (Table II). The mean time to plain radiographs was 18 months (range 4.0 to 37 months). Only a single stent was used in each procedure using one of three stent types: Xact (Abbott, Abbott Park, Ill), Precise

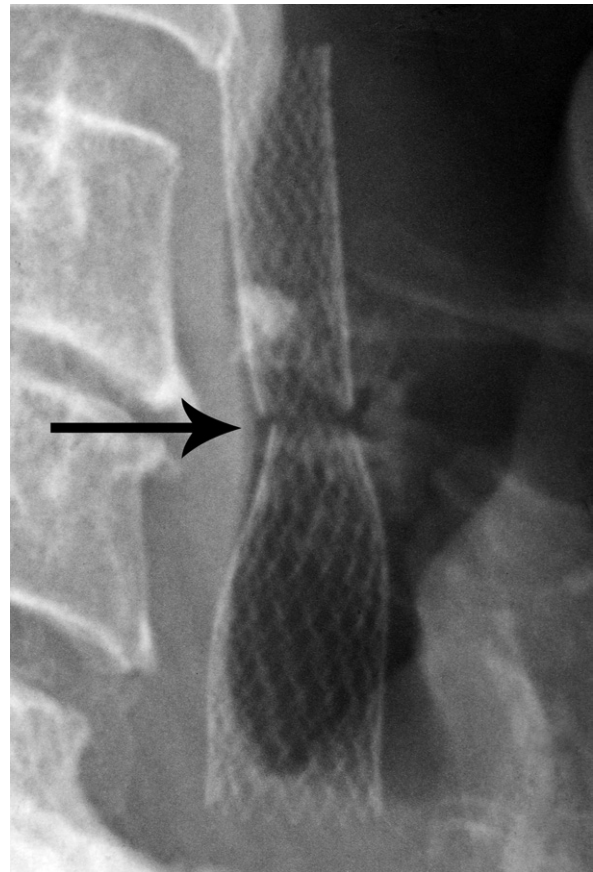


Fig 3. Type III fracture. Transverse linear fracture without stent displacement (*arrow*). This is an Xact 8-10 mm × 40 mm stent placed in a 82-year-old male. Note the localized calcification.

(Cordis, Miami Lakes, Fla), or Exponent (Medtronic Vascular, Santa Rosa, Calif). The same surgeon performed the procedure in all cases.

Out of the 48 stents implanted, 14 (29%) had a fracture detected at a mean radiographic follow-up of 18 months. Duplex sonography was performed in all patients to assess stent patency. Three out of the 14 fractures showed increased velocity ratios >2.0 . (Table III). The mean follow-up period for duplex scans was 14.0 months. Table IV illustrates the velocities on ultrasound according to fracture type. (Table IV).

In determining the risk factors for fracture, the presence of local calcification in the area where the stent was deployed was examined. Eight out of the 14 fractured stents were associated with local calcification in the region of the deployed stent (examples of which are shown in Figs 1 to 3), whereas five 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 (odds ratio 7.7; standard error 5.6; 95% confidence interval 1.9-32.0). The χ^2 $P = .003$. (Table V). The presence of angulation of more than 45 degrees in the stented segment

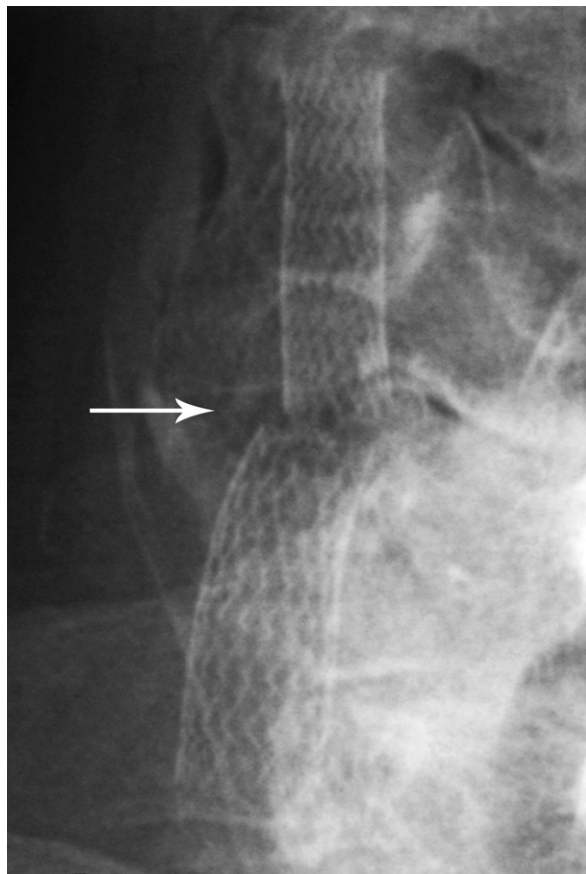


Fig 4. Type IV fracture. Transverse linear fracture with stent displacement (*arrow*). This is an Xact 8-10 mm × 40 mm placed in a 76-year-old male.

Table I. Patient demographics

	Number (%)
Mean age (y)	74
Male/female	37 (86)/6 (14)
Smoking	24 (56)
Hypertension	34 (79)
Diabetes mellitus	13 (30)
IHD	22 (51)
Asymptomatic	19 (44)
TIA or amaurosis fugax	21 (49)
Stroke	3 (7)
Bilateral carotid stent	5 (12)
Contralateral occlusion	4 (9)
Ipsilateral previous CEA	2 (5)

IHD, Ischemic heart disease (angina, acute myocardial infarction, coronary artery bypass graft, percutaneous transluminal coronary angioplasty); TIA, transient ischemic attack; CEA, carotid endarterectomy.

with the head held in neutral position did not have a statistically significant affect on the presence of fractures.

There was no statistically significant association between fractures and smoking, hypertension, diabetes mellitus or sex of the patient, and a multivariate analysis was not

Table II. Fracture prevalence (mean radiographic follow-up of 18 months)

Type I	Type II	Type III	Type IV
3 (21%)	6 (43%)	3 (21%)	2 (14%)

Table III. Fracture effect on restenosis

Fractured (n = 14)		No fracture (n = 34)	
No restenosis	Restenosis	No restenosis	Restenosis
11 (79%)	3 (21%)	31 (91%)	3 (9%)

Table IV. Results of follow-up duplex scans^a

Peak systolic velocity (cm/s)	Type I	Type II	Type III	Type IV
<150	1	6	1	2
150-300	2	0	1	0
>300	0	0	1	0
Total	3	6	3	2

^aUsing duplex ultrasound criteria for grading carotid stent stenosis from University of South Florida.

Table V. Physical risk factors for fracture

	Odds ratio for fracture	Standard error	Confidence interval	P value
Calcification	7.7	5.6	1.9-32.0	.003
Angulation	3	2.4	0.6-14.3	.21

Table VI. Fracture location

Lower third	Middle third	Upper third
1 (9.1%)	6 (54.5%)	4 (36.4%)

feasible due to the small numbers in this study. Regarding the type of stent used 8 out of 34 (24%) of Xact stents, 4 out of 11 (36%) Precise stents and 2 out of 3 (66%) Exponent stents had a fracture detected. There was a trend towards fractures occurring more frequently in the longer stents, however, this was not statistically significant ($P = .28$).

The location of the fracture has been categorized into occurring in the lower, middle or upper third of the stent. It is difficult to categorize the location of the fractures in relation to its anatomical location such as the ICA, carotid bulb, or CCA, given that these fractures are seen on plain x-rays. Most fractures were located in the middle third. (Table VI).

DISCUSSION

Stent fracture is not a new phenomenon. Reports of fracture are noted in Nitinol stents implanted in the periph-

eral arterial tree, esophagus, biliary tract, colon, heart, renal, iliac, popliteal and subclavian, and brachiocephalic vessels.⁷⁻¹⁸ The complication of a Nitinol carotid stent fracture has only been reported as a case study involving the index case of this surgeon's series.⁴

This series has demonstrated that the presence of calcification in the internal carotid artery has a strong association with the occurrence of fractures in the stent; a calcified internal carotid artery is eight times more likely to have a fracture. Currently in the literature, there is no classification for the severity of calcification of the carotid artery. As a consequence, the statement of a calcified vessel in this series is purely a qualitative statement, referring to the presence of visible calcium seen on plain radiographs in the region of the implanted stent. The authors speculate that the presence of calcification exerts an external loading force on the stent, which given the irregularity of such plaques, places focal pressure on certain parts of a stent in situ; this would be exacerbated by neck movements. A calcified vessel also creates a more rigid artery and, hence, a point of fixation on a stent in situ, necessitating that the proximal and distal unstented segments of artery accommodate for changes in neck movement. This creates regional friction and, hence, increases the risk of fracture.

Although not demonstrated with statistical significance in this series, the presence of angulation, particularly of the distal internal carotid artery may also have an association with fracturing of the stent. Flors Vos et al¹⁹ used three-dimensional time-of-flight magnetic resonance angiography to determine the effect of head movement on in situ carotid stents. They found that following CAS, the carotid artery loses its natural flexion/extension and rotational flexibility. Furthermore, the ex vivo flexibility properties of the stents themselves are lost when placed in vivo. As a result, the stented segment of the artery appears as a stiff, inflexible unit in all head positions, leaving the unstented segments to accommodate head movements and resulting in sharp angulations of up to 85 degrees at the junctions between the stent and artery.

Furthermore, they found that the CCA and ICA are subjected to considerable torsion shear with head turning. In addition to this, there is also a physiological tendency for these vessels to twist around each other due to 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, placing ongoing stress on the stent and hence can possibly be a contributing cause to fracture.

All four fracture types were detected in this series, with type II being the most prevalent. The development of these fractures may be initiated right at the beginning of the stent manufacturing process; Nitinol stents must undergo multiple heat treatments, laser cutting, etching, electropolishing, surface finishing, and fatigue testing.²⁰ Microscopic investigations show that the strut surface can develop small microcracks on the surface of the struts, which naturally will progress when exposed to other forces when in situ.

Also, due to its configuration at thermal conditioning, once the stent is implanted into body temperature, it undergoes a small negative dilation²¹ of less than 1%, which according to Stankiewicz, may increase the tensile stresses developed ahead of a crack and result in decreased fracture toughness and fatigue-crack growth resistance.

The design of the stent may substantially influence the likelihood of fractures.²²⁻²³ In our series, all three of the stents used had fractures detected, but further analysis is limited by the small study size. In the femoral stenting in obstructions (FESTO) study, Scheinert et al found that stent design affected the frequency and severity of stent fractures.

Many stents are designed to withstand a straight cyclic fatigue stress, which in vivo, does not exist. This is because the stent in vivo is subject to deformation from multiaxial sources relating to the effect of the body on the stent and the stent on the body. In an attempt to increase the conformability and flexibility of carotid stents, manufacturers have tried to decrease the number of bridges between rings, and vary the shape of these bridges, employing non-straight bridges to improve flexibility. However, the smaller number of connections of these open cell designs are prone to compression and elongation, contributing to a higher likelihood of fracture.²⁴ On the contrary, closed cell stent designs employ a dense scaffolding to create a more rigid but less flexible stent, increasing its strength but also its brittleness. The latter may lead to fracture when exposed to various biomechanical forces such as external compression. The carotid bifurcation is not protected by the axial skeleton or skull, and is easily exposed to such external forces.

The stent durability is also influenced largely by the physiological loading that it must withstand. After only 30 days in vivo, the carotid stent has faced about 3 million pulsatile cardiac cycles. Fracture rates of up to 30% have been reported in stents that are in great vessels close to the heart.¹¹ Further, the cardiac cycle imparts in vivo cyclic stresses through continued expansion and contraction of the carotid arteries. In addition, the ICA is a dynamic free-floating vessel, being only tethered at the proximal and distal ends. High velocity flow rates in the carotid arteries would lend to greater movement of the "free-floating" vessel, causing greater elongation and contraction of the vessel and possibly contribute to stent fracture.

Clinical relevance. Although this series of carotid arterial stent fractures had a fracture rate of 30%, the majority were benign at time of writing; only three of these had restenosis >50% on surveillance ultrasound scans at an average of 12 months postimplantation. High-grade (>75% diameter reduction) after CAS and CEA is generally thought to be a clinically significant lesion that can progress to a stroke.²⁵

It is interesting to note that the type IV fractures, which by definition are displaced, did not have restenosis detected at average follow-up in this series. It was a type III fracture that required surgical removal, having progressed rapidly over 6 months from a 60% stenosis to a 90% stenosis.

Although no direct association can be drawn from this small series, it does nevertheless demonstrate that not all fractures are benign and can progress to a clinically significant stenosis.

A review of the existing Nitinol stent fracture literature including that for aortic endografts, popliteal arteries, and coronary stenting (Allie et al⁶) has consistently described clinical sequelae related to the fracture in all reports. Motivated by the unclear impact of the phenomenon of stent fractures in the sirolimus-coated cordis self-expandable stent (SIROCCO) trial, Scheinert²² initiated the FESTO study. He found that stent fractures were associated with restenosis of >50% in one third of fractures, and reocclusion in another third. Clearly, primary patency rate of the stented segment was significantly lower for patients with stent fractures (41.1% vs 84.3%, $P < .00001$), demonstrating its clinical importance and relevance.

Whether the long-term patency of carotid artery stenting is better than the natural history of the disease and/or surgical endarterectomy is yet to be determined. The clinical impact of carotid stent fractures should not be underestimated, especially given the evidence of poor patency of fractures in the superficial femoral artery stents. This is exemplified by the concern that the American Food and Drug Administration has displayed, having mandated aggressive surveillance for stent fractures in all prospective stent-based device trials.

The question is then raised about when a lesion should receive intervention if a restenosis is detected. Lal et al²⁶ recommend intervention in asymptomatic lesions >80% in carotid restenosis, reasoning that it is difficult to discern which lesion will remain asymptomatic. As an aside, Lal et al report an in-stent restenosis of >80% in five out of 22 (23%) patients in their series, which was diagnosed by surveillance ultrasonography at 15 months. It would be interesting to determine the prevalence of carotid stent fractures in that series.

Limitations. The sensitivity and specificity of plain radiography in detecting all types of stent fractures is unknown, even with magnified views. Thus, the potential for missing possible fractures is present, even though the films were examined by three independent observers. Fluoroscopy carries significant risks, and the value of CT angiography and ultrasonography likewise is yet to be determined.

In relation to the presence of calcification, analysis is limited by the qualitative description of calcification being either present or absent in the region of where the stent is deployed. A prospective trial would be required to use an imaging modality such as CT angiography to accurately quantify the location and severity of calcification, including whether it is circumferential or localized.

There are also limitations due to the type of study being a single centre, retrospective, nonrandomized case-control study. Eleven out of 54 patients were lost to follow-up, which may have an impact on the accuracy of the analysis of complications in this series given that it represents twenty percent of the study size.

Furthermore, the ability to draw conclusions regarding risk factors for fracturing, and long term sequelae is limited by the small sample size of this series (which precluded a multivariate analysis) and short follow-up period for ultrasonography. Nevertheless, the objective of this article was to inform clinicians of a potential complication of carotid stenting and recommend continued long-term surveillance.

CONCLUSION

Not all carotid stent fractures are benign. The authors believe it is worthwhile to include plain radiographs in addition to the regular surveillance of carotid stents with duplex ultrasonography to enable early detection of fracturing and, hence, institution of any appropriate intervention to aid in the prevention of sequelae such as recurrent stenosis, should it become apparent. The latter is ever more so important in the carotid artery, which has little room for error, if any at all.

AUTHOR CONTRIBUTIONS

Conception and design: AL, KS

Analysis and interpretation: AL

Data collection: AL, TG

Writing the article: AL, TG

Critical revision of the article: AL, KS, PM, TG

Final approval of the article: KS, PM

Statistical analysis: AL

Obtained funding: AL, PM

Overall responsibility: KS

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INVITED COMMENTARY

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Stent fracture is something that has been identified in nearly every region in which stents have been implanted. In particular, the use of stents within the vascular system has increased dramatically over the past 10 years. It is only with extended use and evaluation that identification of stent fractures within the superficial femoral artery was recognized. These stent fractures have been associated with extended lengths of treatment, extensive calcification of the vessels, and an increased incidence of re-stenosis within the vessels. To date, no extensive evaluation of stents within the carotid distribution has been performed until the present study.

This study in particular brings out several important points. The first of these is that the incidence of stent fractures within the carotid distribution is likely higher than previously expected. Sec-

ondly, calcification within the internal carotid artery radically increases the risk of stent fracture and third, there is yet an undetermined relationship between stent fracture and recurrent stenosis within the carotid artery. Several other points deserve emphasis as well.

Despite the fact that only a short segment of the carotid artery is treated with these stents, a significant percentage of these stents fractured. Second, although these stents involve modifications of existing stents, the modifications made to accommodate the anatomy of the carotid circulation are inadequate to completely alleviate the risks of stent fracture and to accommodate the increased motion noted in this vascular region.

The findings of this current study present yet another note of caution for those treating the carotid artery with interventional means. These findings also point out the importance of the need for further studies to evaluate the short-term and long-term function of interventional therapy in carotid bifurcation disease.

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