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High frequency of brachiocephalic trunk stent fractures does not impair clinical outcome

Edit Dósa, MD, PhD,[a] Balázs Nemes, MD, PhD,[a] Viktor Bérczi, MD, PhD, DSc,[b] Pál Kaposi Novák, MD, PhD,[b] Tamás Mirko Paukovits, MD, PhD,[a] Hunor Sarkadi, MD,[a] and **Kálmán Hüttl, MD, PhD,[a]** *Budapest, Hungary*

Objective: **Stenting is the preferred, minimally invasive treatment for innominate artery (IA) stenosis or occlusion. Stent fractures in the IA have not been assessed in larger cohorts. In this retrospective study, we examined the frequency and risk factors of IA stent fractures.**

Methods: **The final analysis included 32 patients (15 women; mean age, 59.4 ± 12.0 years) with 32 balloon-expandable stents (2000 to 2009). In 2010, the patients were asked to come back for a fluoroscopic examination of the implanted stents. Stent fractures and their relationship to atherosclerotic risk factors, lesion characteristics, postprocedural symptoms, and in-stent restenosis were analyzed. Fisher exact test and univariate Cox regression analysis were used in the statistical evaluation.**

Results: **Lesions were >20 mm in 14 patients (44%) or heavily calcified in 13 (41%). The mean follow-up time was 33.4 ± 21.0 months. Postprocedural symptoms were noted in nine patients (28%). Significant restenosis was detected in 22% of the implanted stents, and 11 stent fractures (34%) were found. The prevalence of heavily calcified lesions, postprocedural symptoms, and in-stent restenosis did not differ significantly between groups with and without fracture. Long lesions were associated with an increased incidence of stent fracture (hazard ratio, 5.09; 95% confidence interval, 1.33-19.48; $P = .017$). No correlation was observed between stent fractures and old age (≥70 years), female gender, smoking, hypertension, hyperlipidemia, or diabetes mellitus.**

Conclusions: **IA stent fractures are common but seem to have no effect on symptoms and in-stent restenosis rates. (J Vasc Surg 2014;59:781-5.)**

Innominate artery (IA) stenosis or occlusion can be asymptomatic or symptomatic. The cerebrovascular symptoms include signs of posterior circulation ischemia due to flow reversal in the right vertebral artery (subclavian steal syndrome) and, less commonly, transient ischemic attack (TIA) or stroke as a result of distal embolization. The right upper extremity symptoms are related to hypoperfusion or distal embolization.

Endovascular or surgical recanalization procedures are required in symptomatic patients.[1] Percutaneous transluminal angioplasty (PTA) combined with stenting was first reported in the 1980s, and since then, has become the primary treatment of IA stenosis. The efficacy of IA stenting has been validated in follow-up studies, which showed low complication and high primary patency rates.[2-5] Surgical reconstruction is preserved for patients with a high amount of calcium deposition or difficult anatomy.

From the Heart and Vascular Center[a] and the Department of Radiology and Oncotherapy,[b] Semmelweis University.

This work was supported by the János Bolyai Research Scholarship of the Hungarian Academy of Sciences (E.D.).

Author conflict of interest: none.

Reprint requests: Edit Dósa, MD, PhD, Heart and Vascular Center, Semmelweis University, Városmajor St 68, 1122 Budapest, Hungary (e-mail: dosaedit@yahoo.com).

The editors and reviewers of this article have no relevant financial relationships to disclose per the JVS policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

0741-5214/$36.00

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http://dx.doi.org/10.1016/j.jvs.2013.09.038

Recent studies have extensively investigated stent fractures in the lower limb, coronary, carotid, and vertebral arteries.[6-9] However, with the exception of individual case reports[10,11] and a small survey of nine patients,[12] no comprehensive data have been published on stent fracture prevalence in the IA. We hypothesized that IA stent fractures are common due to the proximity of the beating heart and movements of the shoulder girdles and the arms. The purpose of this retrospective study was to determine the frequency and risk factors of IA stent fractures. We also aimed to examine the effect of stent fractures on postprocedural symptoms and in-stent restenosis rates.

METHODS

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Semmelweis University Ethics Committee. All participants provided written informed consent. No compensation was provided for participation in the study; only travel expenses were covered.

Patient selection and lesion characteristics. All patients who underwent IA stenting between January 1, 2000, and December 31, 2009, were included: 49 IA lesions were treated with stent implantation in 49 patients. Six of the 49 patients did not have a follow-up examination because of death caused by stroke in 1 patient, at 3 months, acute myocardial infarction in 2 patients, at 5 and 11 months, and cancer in 3 patients. The study excluded three patients who received self-expandable stents, and eight patients did not return for fluoroscopic examination.

The final analysis therefore included 32 patients with 32 balloon-expandable stents. The procedures were performed in the Heart and Vascular Center of Semmelweis University (Budapest, Hungary).

Lesions were considered long if their length was ≥20 mm. Lesions were defined as heavily calcified if calcification was present along the entire length of the IA by fluoroscopy.

Innominate artery stenting protocol. The preprocedural work-up included clinical data collection (symptoms, risk factors, which were described in detail previously,[13] and medical history), radial pulse palpation and blood pressure measurement in both arms, neurologic evaluation, and duplex scan of the neck arteries. In recent years, computed tomography (CT) or magnetic resonance angiography have become part of the preprocedural imaging protocol in patients with multivessel supra-aortic disease to evaluate which lesion should be treated first.

Acetylsalicylic acid (100 mg) was given orally for at least 3 days before the procedure and was continued permanently. Heparin (5000 IU) was administered intra-arterially at the beginning of the procedure. Blood pressure and electrocardiogram waves were monitored continuously. The procedures were carried out through femoral artery access. In patients with IA occlusion, the right brachial artery was also punctured.

The initial diagnosis was confirmed by thoracic aortography before the stent placement in all patients. A 4F pigtail catheter (Cordis Corp, Johnson & Johnson Co, Miami, Fla) was inserted into the aortic arch over a standard polytetrafluoroethylene-coated 0.035-inch wire (J-curved on one end, straight on the other end). Selective catheterization (H1, JB2, or SIM2; Cordis Corp) of the IA was performed in all patients, except those with IA occlusion.

A hydrophilic (standard or stiff) angled-tip 0.035-inch Terumo guidewire (Terumo Medical Corp, Tokyo, Japan) was advanced into the IA to pass through the stenosis or occlusion under fluoroscopic control. Thereafter, the 4F introducer sheath (Terumo Medical Corp) was exchanged for a long 7F sheath (Cordis Corp) over a standard or a hydrophilic stiff (Terumo) or a super-stiff 0.035-inch Amplatz wire (Boston Scientific Corp, Natick, Mass). We did not use embolic protection devices for the intervention.

The diameter of the patent segment and length of the lesion were measured on selective angiograms for accurate sizing of the stent. The indication for stent placement was a suboptimal PTA due to unfavorable lesion morphology (eg, heavily calcified lesions, occlusions) or a failed angioplasty. Different balloon and stent types were used, depending on the availability of devices and personal preference of the interventional radiologist. The angioplasty balloons were Wanda (size, 7-10 mm × 40 mm; Boston Scientific Corp) in eight patients and Pheron (size, 7-8 mm × 40 mm; Biotronik AG, Bülach, Switzerland) in seven. Direct stent implantation was performed without predilatation in 17 of the 32 patients. One Bard (3%) stent (size, 12 mm × 20 mm; C.R. Bard Inc, Murray Hill, NJ) and 31 Palmaz Genesis (97%) stents (size, 8-10 mm × 19-29 mm; Cordis Corp) were deployed. When it was necessary because of difficult access due to angulation at the origin of the IA or high-grade stenosis or occlusion, a long Mach 1 guiding catheter (Boston Scientific Corp) was used, and the stenosis was dilated first with a 3- to 4-mm coronary balloon over a 0.014-inch wire then with a 5- to 6-mm peripheral balloon, after which the stent was easily pushed through the lesion.

On completion angiography, the IA was imaged in a 30° to 45° right anterior oblique plane, the ipsilateral cerebral arteries in a posteroanterior 30° to 45° (right and left) anterior oblique and lateral planes, and the ipsilateral upper extremity arteries in a posteroanterior plane. Finally, the sheath(s) were removed and the punctured artery(ies) were compressed manually, followed by pressure bandage(s) overnight or, in more recent cases, the femoral artery was closed by an Angio-Seal percutaneous closure device (St. Jude Medical Inc, Little Canada, Minn). Patients were discharged 1 or 2 days after the procedure.

Technical success was defined as <30% residual stenosis without dissection or extravasation, whereas clinical success meant resolution of the symptoms.

Follow-up. Follow-up visits were scheduled at 4 weeks and at 6 and 12 months after the stenting, and annually thereafter. The postprocedural examinations included clinical data collection, radial pulse palpation, blood pressure measurement in both arms, and a duplex scan. On the basis of these evaluations, patients in whom significant IA in-stent restenosis or reocclusion was suspected had no palpable right radial pulse, had a blood pressure difference (≥30 mm Hg) between arms, or right subclavian steal syndrome. The suspected IA in-stent restenoses or reocclusions were verified with CT angiography in symptomatic patients.

In 2010, the patients were asked to come back for an additional follow-up visit, when a fluoroscopic evaluation was also performed in addition to the above-mentioned examinations. The patients were told that X-rays usually have no side effects in the typical diagnostic range for this examination. The high-magnification fluoroscopic examinations were done in the angiography suite (AXIOM Artis FA; Siemens Medical Solutions AG Company, Erlangen, Germany) with the following parameters: 7.5 fps, 100 to 125 kV, and 550 to 800 mA.

To visualize the implanted stents, three cine loops with a length of three cardiac cycles were recorded in posteroanterior, right, and left anterior oblique 30° to 45° projections. The postprocessing was performed on a Leonardo Workstation (Syngo 2003; Siemens Medical Solutions). The fluoroscopic images were analyzed by two experienced interventional radiologists (K.H., B.N.) in consensus.

Stent fractures were defined according to a nitinol stent fracture classification that has been proposed by the Cardiovascular Institute of the South (Houma, La).[14] Type I is a single-strut fracture, type II fractures represent multiple stent fractures that can occur at different sites, type III is a complete transverse fracture without stent

displacement, and type IV fractures have a complete transverse linear fracture with stent displacement.[8,14]

Statistical analysis. Statistical analysis was performed with SPSS 21.0 software (IBM Corp, Armonk, NY). The relationship between stent fracture and other postprocedural variables was analyzed using the Fisher exact test. A univariate Cox regression model was used to identify independent baseline predictors of stent fracture. All analyses were two-tailed, and values of $P \leq .05$ were considered as statistically significant.

RESULTS

Patient data. The gender distribution of the patients was almost equal (17 men, 15 women), and their average age was 59.4 ± 12.0 years (range, 28-83 years). Major symptoms included signs of posterior cerebral ischemia due to vertebrobasilar insufficiency in 10 patients (31%), TIA in 4 (13%), amaurosis fugax in 3 (9%), and right upper extremity claudication in 15 (47%). No patients had significant (≥70%) right common or internal carotid artery stenosis. None of those who presented with TIAs and amaurosis fugax had significant carotid artery stenosis or unstable carotid plaques on any side. According to the medical history, 10 patients (31%) had ischemic heart disease and nine (28%) had lower extremity arterial disease. Atherosclerotic risk factors included smoking in 20 patients (63%), hypertension in 22 (69%), hyperlipidemia in 19 (59%), and diabetes mellitus in 5 (16%).

Lesion characteristics. The stented lesions were de novo stenoses in 29 patients (91%) and recurrent stenoses after previous PTA in three (9%). The degree of stenosis or restenosis was 60% to 89% in 27 patients (85%) and 90% to 99% in two (6%). Three patients (9%) had IA occlusion <1 cm in length; one occlusion was an orifice lesion, and two were confined to the body of the IA. Lesions were long in 14 patients (44%). Heavily calcified lesions were detected in 13 patients (41%). Three (9%) of the 32 lesions were circumferentially calcified.

Early postprocedural period (≤30 days). The technical success rate was 100%. No neurologic complications occurred in the periprocedural period. An arteriovenous fistula developed in one patient at the femoral puncture site, and one brachial artery thrombosis was observed. Both patients were treated surgically. The 30-day stroke, stroke mortality, and overall mortality rates were zero. All patients reported improvement or resolution of the preprocedural symptoms.

Follow-up period. The mean follow-up time was 33.4 ± 21.0 months (range, 2-73 months). Only one patient had less than 6 months of follow-up. In-stent restenosis was observed in seven patients (22%), and three of them had no palpable right radial pulse. The blood pressure difference between the two arms was >30 mm Hg in all patients, and duplex scan showed subclavian steal syndrome in five patients.

Nine patients (28%) were symptomatic: two showed signs of posterior cerebral ischemia, three had right upper extremity claudication, and four complained of dizziness. Five of the nine symptomatic patients had in-stent restenosis. The cause of symptoms in the other four patients was degenerative neck disease. Three of the five patients with symptomatic in-stent restenosis have already undergone, and two have been scheduled for reintervention.

Table I. Vascular calcification, presence of in-stent restenosis, and postprocedural symptoms in patients with and without innominate artery (IA) stent fracture

Characteristic	*Stent, No. (%)*		P *value*
	Fractured (n = 11)	*Nonfractured (n = 21)*	
Heavy calcification	7 (64)	6 (29)	.072
In-stent restenosis	2 (18)	5 (24)	1.000
Postprocedural symptoms	3 (27)	6 (29)	1.000

Fluoroscopic examination. Altogether, 11 stent fractures (34%) were detected. Fractures were type I in 1, type II in 4, type III in 2, and type IV in 4 patients, with 63% of the fractures predominantly in the proximal portion of the stents.

Factors associated with stent fracture. Heavily calcified lesions were more common in patients with IA stent fracture; however, the difference did not reach the statistically significant level ($P = .072$; Table I). The prevalence of in-stent restenosis did not differ significantly between the groups with and without fracture (Table I). In case of postprocedural symptoms, the statistical analysis did not reveal a significant difference between patients with and without stent fracture (Table I). The Cox regression analysis showed that the presence of long lesions was associated with an increased incidence of stent fracture (hazard ratio, 5.09; 95% confidence interval, 1.33-19.48; $P = .017$; Table II). No correlation was found between stent fractures and age ≥70 years, female gender, smoking, hypertension, hyperlipidemia, or diabetes mellitus (Table II).

DISCUSSION

Stent fracture has long been recognized as a complication of endovascular procedures. Fractures, although with varying frequencies and consequences, have been reported in all sites where stents are deployed to resolve luminal narrowing.[6-12] With the exception of a few individual case reports[10,11] and a small survey of nine patients,[12] this is the only study that has examined the frequency of stent fracture in a larger cohort of patients with IA stenting. Our retrospective assessment of a group of 32 patients has demonstrated that fracture is a common complication of IA stenting. We have also found that lesion length directly correlates with an increased fracture risk, amounting to a 34% overall fracture rate in the current series. In our survey, however, the high prevalence of stent fracture did not have a significant effect on symptoms and in-stent restenosis rates.

Table II. Univariate analysis of predictors of stent fracture after innominate artery stenting

	Univariate Cox regression analysis	
Variables	*HR (95% CI)*	P *value*
Age ≥70 years	0.63 (0.14-2.95)	.561
Female gender	1.15 (0.35-3.78)	.821
Smoking	0.52 (0.16-1.72)	.287
Hypertension	1.32 (0.35-4.96)	.686
Hyperlipidemia	1.61 (0.47-5.53)	.449
Diabetes mellitus	0.61 (0.08-4.80)	.640
Heavy calcification	3.02 (0.88-0.35)	.078
Long lesion	5.09 (1.33-19.48)	.017[a]

CI, Confidence interval; *HR*, hazard ratio.
[a]Statistically significant.

Although IA PTA without stent placement can be accomplished with high technical success (96%-100%) and low complication rates (0%-6%),[2-4] stenting has a clear advantage over PTA in patients with complex lesions.[1,5,15-20] Parallel with the increase in the number of IA stent implantations, the first case reports of stent fractures have also emerged.[10,11] Stent fracture is most frequently seen after revascularization of vessels, such as the femoropopliteal (15%-37%) and superficial femoral arteries (2%-65%), where generally longer self-expandable stents are used.[6] Rupture of balloon-expandable stents is also often detected in the vertebral, iliac, and renal arteries, among others, with a cumulative incidence ranging between 8% and 33%, depending on the site.[6] In the present study, we found a 34% overall fracture rate in the IA, which is comparable to the number of structural defects observed in other supra-aortic branches.[6,8]

External mechanical forces and structural design of the stents can both be responsible for the relatively high frequency of stent fractures in the IA. We can assume that movements of the shoulder girdles and the arms can result in an increased mechanical stress on the IA stents.[21] In addition, the continuous expansion and contraction caused by the proximity of the beating heart are also presumed to contribute to the nearly 30% stent fracture rates described in the suprathoracic vessels.[6,8]

The design of the implanted stent itself can substantially influence the likelihood of fracture. Most of our patients received Palmaz Genesis stents, and only one individual was treated with a Bard stent. Although a survey by Tsutsumi et al[22] found that Palmaz stents were less likely to rupture than S670 balloon-expandable coronary stents deployed in the vertebral arteries, the literature contains a large amount of data regarding the proclivity of Palmaz stents to deformation and fracture.[6]

Our study found that heavily calcified lesions were more common in patients with IA stent fracture; however, the difference did not reach the statistically significant level. A survey conducted on internal carotid artery stents has found that calcification of the vessel wall showed strong association with stent fracture.[8] The risk of stent fracture was eight times higher in the calcified than in the noncalcified arteries.[8] The most widely accepted explanation for this finding is that calcification causes changes in the regional wall stiffness that may lead to excessive focal pressure on certain parts of the stent, hence exacerbating fracture.

Our study revealed a significant direct correlation between the length of the stenotic segment and the probability of fracture. Results of previous reports appear to indicate that the lesion length and, consequently, the stent length has a significant influence on the frequency of stent fracture,[6] likely because longer stents are subject to higher radial forces, especially in their midsegment.[23] These forces over a period of time may lead to metal fatigue and eventually fracture.[23]

The fractures can be associated with higher in-stent restenosis rates and recurrence of symptoms, but the available literature data are quite inconsistent in this regard. Tsutsumi et al[22] examined the occurrence of vertebral artery stent fracture in a retrospective cohort of 12 patients and detected rupture in half of the deployed coronary stents; however, no patients had in-stent restenosis or postprocedural ischemic symptoms. Symptomatic in-stent restenoses or reocclusions caused by IA stent fractures also have been reported.[10,11] We identified in-stent restenosis in 22% and postprocedural symptoms in 28% of our patients after IA stenting but found no significant correlation between the presence of fracture and the recurrence of stenosis or clinical symptoms.

The limitations of the present study leave some of our findings incomplete: First, patients were retrospectively enrolled into the study, and the results represent an experience acquired in a single institution.

Second, owing to the retrospective nature of patient selection, follow-up protocols were not uniform, and complete follow-up data were not available in all patients.

Third, given the small number of nine patients who had preprocedural CT angiography, digital subtraction angiography and fluoroscopy were used to assess vascular calcification, both of which are poor imaging modalities for this purpose.

Fourth, the series included only three short (<1 cm) chronic total occlusions, indicating that the results of this study do not necessarily apply to chronic total occlusions.

Fifth, Palmaz stents were implanted in most patients; thus, we cannot discern the performance of different stent designs, such as covered balloon-expandable stents, which are more frequently used for these lesions.

Most of the fractured stents remained asymptomatic, but validation by independent cohorts might be necessary to reach definite conclusions on the clinical and therapeutic consequences of IA stent fractures.

CONCLUSIONS

Even if stents deployed in the brachiocephalic trunk possess a relatively high fracture risk, especially in patients with long lesions (≥20 mm), the occurrence of IA stent

fracture does not significantly correlate with clinical outcome.

We thank Levente Lukács, MD, for his work in collecting patient data and Péter Vargha for his assistance with statistical analysis.

AUTHOR CONTRIBUTIONS

Conception and design: VB, KH
Analysis and interpretation: ED, VB, BN, KH
Data collection: TP, HS
Writing the article: ED, VB, PK
Critical revision of the article: ED, VB, BN, PK, TP, HS, KH
Final approval of the article: ED, VB, BN, PK, TP, HS, KH
Statistical analysis: ED
Obtained funding: Not applicable
Overall responsibility: ED

REFERENCES

1. Eisenhauer AC. Subclavian and innominate revascularization: surgical therapy versus catheter-based intervention. Curr Interv Cardiol Rep 2000;2:101-10.
2. Motarjeme A. Percutaneous transluminal angioplasty of supra-aortic vessels. J Endovasc Surg 1996;3:171-81.
3. González A, Gil-Peralta A, González-Marcos JR, Mayol A. Angioplasty and stenting for total symptomatic atherosclerotic occlusion of the subclavian or innominate arteries. Cerebrovasc Dis 2002;13:107-13.
4. Hüttl K, Nemes B, Simonffy A, Entz L, Bérczi V. Angioplasty of the innominate artery in 89 patients: experience over 19 years. Cardiovasc Intervent Radiol 2002;25:109-14.
5. Paukovits TM, Lukács L, Bérczi V, Hirschberg K, Nemes B, Hüttl K. Percutaneous endovascular treatment of innominate artery lesions: a single-centre experience on 77 lesions. Eur J Vasc Endovasc Surg 2010;40:35-43.
6. Rits J, van Herwaarden JA, Jahrome AK, Krievins D, Moll FL. The incidence of arterial stent fractures with exclusion of coronary, aortic, and non-arterial settings. Eur J Vasc Endovasc Surg 2008;36:339-45.
7. Chakravarty T, White AJ, Buch M, Naik H, Doctor N, Schapira J, et al. Meta-analysis of incidence, clinical characteristics and implications of stent fracture. Am J Cardiol 2010;106:1075-80.
8. Ling AJ, Mwipatayi P, Gandhi T, Sieunarine K. Stenting for carotid artery stenosis: fractures, proposed etiology and the need for surveillance. J Vasc Surg 2008;47:1220-6; discussion: 1226.
9. Varcoe RL, Mah J, Young N, So SS, Vicaretti M, Swinnen J. Prevalence of carotid stent fractures in a single-center experience. J Endovasc Ther 2008;15:485-9.
10. Periard D, Haesler E, Hayoz D, Von Segesser LK, Qanadli SD. Rupture and migration of an endovascular stent in the brachiocephalic trunk causing a vertebral steal syndrome. Cardiovasc Intervent Radiol 2008;31(Suppl 2):S53-6.
11. Al-Natour M, Renno A, El-Fedaly M, Abbas J, Nazzal M. Stent fracture in the brachiocephalic trunk [published online ahead of print March 19, 2013]. Vascular doi: 10.1177/1708538113478775.
12. Usman AA, Resnick SA, Benzuly KH, Beohar N, Eskandari MK. Late stent fractures after endoluminal treatment of ostial supraaortic trunk arterial occlusive lesions. J Vasc Interv Radiol 2010;21:1364-9.
13. Dósa E, Hirschberg K, Apor A, Járányi Z, Entz L, Acsády G, et al. Echolucent or predominantly echolucent femoral plaques predict early restenosis after eversion carotid endarterectomy. J Vasc Surg 2010;51:345-50.
14. Allie DE, Hebert CJ, Walker CM. Nitinol stent fractures in the SFA. Endovasc Today 2004;1:22-34.
15. Przewlocki T, Kablak-Ziembicka A, Pieniazek P, Musialek P, Kadzielski A, Zalewski J, et al. Determinants of immediate and long-term results of subclavian and innominate artery angioplasty. Catheter Cardiovasc Interv 2006;67:519-26.
16. Brountzos EN, Malagari K, Kelekis DA. Endovascular treatment of occlusive lesions of the subclavian and innominate arteries. Cardiovasc Intervent Radiol 2006;29:503-10.
17. Zaytsev AY, Stoyda AY, Smirnov VE, Scherbyuk AN, Kondrashin SA, Artukchina EG, et al. Endovascular treatment of supra-aortic extracranial stenoses in patients with vertebrobasilar insufficiency symptoms. Cardiovasc Intervent Radiol 2006;29:731-8.
18. van Hattum ES, de Vries JP, Lalezari F, van den Berg JC, Moll FL. Angioplasty with or without stent placement in the brachiocephalic artery: feasible and durable? A retrospective cohort study. J Vasc Interv Radiol 2007;18:1088-93.
19. Criado FJ. Endovascular techniques for supra-aortic trunk intervention. Perspect Vasc Surg Endovasc Ther 2007;19:231-7.
20. Patel SN, White CJ, Collins TJ, Daniel GA, Jenkins JS, Reilly JP, et al. Catheter-based treatment of the subclavian and innominate arteries. Catheter Cardiovasc Interv 2008;71:963-8.
21. Lee CE, Shaiful AY, Hanif H. Subclavian artery stent fracture. Med J Malaysia 2009;64:330-2.
22. Tsutsumi M, Kazekawa K, Onizuka M, Kodama T, Matsubara S, Aikawa H, et al. Stent fracture in revascularization for symptomatic ostial vertebral artery stenosis. Neuroradiology 2007;49:253-7.
23. Nair RN, Quadros K. Coronary stent fracture. A review of the literature. Cardiac Cath Lab Director 2011;1:32-8.

Submitted Jul 20, 2013; accepted Sep 19, 2013.