Pitfalls In The Endovascular Treatment Of Takayasu’s Disease: Should They Ever Be Used And When

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RAPID EXPANSION OF OPPORTUNITIES FOR EFFECTIVE TREATMENT OF LARGE VEIN STENOSIS, THROMBOSIS, AND OCCLUSION

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DISCLOSURE OF CONFLICTS OF INTEREST

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I do not have any relevant financial relationship with any commercial interests.

Takayasu’s arteritis (T.A.) is an inflammatory arterial disease of the systemic nature with progressive destruction of arterial wall affecting entire aorta and its major branches to cause various occlusive symptoms. With its 'progressive' nature, TA takes a lifelong course from early inflammatory stage to late fibrotic/occlusive stage among the majority often with waxing and waning symptoms.

Therefore, bypass surgery is NOT a panacea to relieve the occlusive lesion to cause acute/chronic arterial insufficiency permanently but remains vulnerable for the future involvement to T.A. especially when done during the remission period along relatively early stage of the T.A. Hence, a new approach of less invasive nature was mandated to bridging the gap till the disease reaches to its late/occlusive stage among the majority often with waxing and waning symptoms.

We assessed the results of balloon angioplasty and stenting on N=24 patients in 'inactive' chronic stage to treat the T.A. as an interim measurement if not a semi-permanent solution to restore the hemodynamic status and to relieve clinical symptoms especially for the condition of multiple vessel involvement during 11 year period. (January 1995 to December 2005) as previously reported (Lee BB, Laredo J, Neville R, Villavicencio JL: Endovascular Management of Takayasu Arteritis: Is It a Durable Option? Vascualr, Vol. 17, No. 2, pp.1–10, 2009)

We made a further extended assessment (average of 76.2 months follow up) on the long term efficacy of endovascular approach.

Open surgical bypass has been able to relieve most of the occlusive lesion to cause acute/chronic arterial insufficiency. But, unfortunately, proper identification of an 'unaffected' area of disease-free artery required for bypass surgery is often difficult, if not impossible. Besides, when 'inactive' stage flares up to full blown 'active' stage, previously healthy area of the vessel wall, selected for the bypass surgery, would become vulnerable to subsequent risk of the involvement.
All 24 patients (6F=15, M=9, age range 21-50 years, mean = 28.9 years) met the clinical diagnostic criteria confirming a diagnosis of T.A. with the appropriate combination of obligatory, major and/or minor criteria of signs and symptoms. All were confirmed for the chronic inactive stage to become candidates for the endovascular treatment.

Clinical staging of the disease (chronic inactive stage) as well as the extent of its involvement (single or multiple vessels) was established by Duplex ultrasonography, CT scan and/or contrast-enhanced MRI in addition to laboratory markers erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP).

The main indication for the therapy includes, clinically significant ischemia involving one or more vascular beds, renovascular hypertension, cerebrovascular ischemia, and upper extremity ischemia.

Intervention was made to a single vessel lesion with critical stenosis over 75%, or involvement of at least three cerebrovascular vessels with greater than 50% luminal diameter narrowing.

All lesions were treated with angioplasty alone or with angioplasty and stenting.

Indications for stenting were the presence of a post-angioplasty flow-limiting dissection, residual stenosis greater than 30%, and pressure gradient greater than 5 mm Hg.

A total of N=90 lesions were identified in the N=24 patients. N=35 lesions met the criteria for endovascular treatment and underwent subsequent angioplasty with or without stenting (angioplasty alone; N=18, and angioplasty with stenting; N=17).

Arteries treated included: renal arteries (N = 16), subclavian/innominate arteries (N = 11), carotid arteries (N= 5), and abdominal aorta (N= 3).

Among the N=35 lesions that underwent endovascular treatment, N=26 lesions achieved excellent to good target lesion revascularization with no residual stenosis (N=16) or only minimal residual stenosis (N=10).

N=5 lesions had only moderate target lesion revascularization with a greater than 30% residual stenosis and the rest (N=4) had poor target lesion revascularization with a greater than 70% residual stenosis.

Satisfactory hemodynamic correction of the pressure gradient by the procedure occurred in N=30 with equalization of pre- and post stenotic pressures. Lesions with moderate (N=1) and poor (N=4) target lesion revascularization had minimal to no reduction of the pressure gradient.

The degree of clinical/functional improvement in general, however, did not correlate with the degree of anatomical and hemodynamic improvement.

Recurrence of symptoms and stenosis was observed in both the angioplasty alone group and angioplasty and stenting group. Within the 48 month follow up period, a total of N=8 lesions developed restenosis in the angioplasty alone group (N=18) compared with N=3 in the angioplasty and stenting group (N=17). All the lesions with recurrent stenosis underwent successful reintervention with relief of symptoms and without complication in its majority (N=8).
An additional retrospective review (January 1995 to December 2009) on same N=24 patients for further 'extended' period for 5 years revealed more recurrence of the stenosis in both the angioplasty alone group (12/18) and angioplasty and stenting group (7/17) : N=12 (N=4 in addition to initial N=8) among angioplasty alone group (N=18); N=7 (N=4 in addition to initial N=3) among angioplasty and stenting group.

Among N=4 of new recurrences among the angioplasty alone group, N=2 were at de novo site while other N=2 were on same site of previous recurrence. N=3 responded to angioplasty alone with good symptomatic relief while N=1 required additional stenting.

All N=4 among angioplasty and stenting group developed at de novo site and N=2 responded to the angioplasty while other N=2 required the stenting.

All recurrent lesions to cause the symptoms also can be successfully treated with repeat angioplasty and/or stenting. Endovascular therapy is therefore, a recommendable option to T.A. in chronic inactive stage in particular as a durable treatment modality.

Conclusions

Takayasu Arteritis (T.A.) lesions in inactive chronic stage can be managed safely with angioplasty alone or with angioplasty and stenting, to providing excellent to good clinical improvement in the majority seen at first follow up (46.8 months) and also through further extended follow up period (76.2 months).

All recurrent lesions to cause the symptoms also can be successfully treated with repeat angioplasty and/or stenting.

Endovascular therapy is therefore, a recommendable option to T.A. in chronic inactive stage in particular as a durable treatment modality.

Thank you for your attention!
Retrospective review was done on N=24 patients in ‘inactive’ chronic stage T.A. who were treated with angioplasty alone or stenting combined during 11 year period. (January 1995 to December 2005)

All 24 patients (F=15, M=9, age range 21-50 years, mean = 28.9 years) met the clinical diagnostic criteria confirming a diagnosis of T.A. with the appropriate combination of obligatory, major and/or minor criteria of signs and symptoms.

All were confirmed for the chronic inactive stage to become candidates for the endovascular treatment.

All patients underwent angiography and all identified lesions were classified using the new arteriographic (Numano) criteria based on involvement of specific arterial beds.

N=11 patients underwent additional Gadolinium-DTPA enhanced MRI and 3-D MRI studies for further assessment of transmural activity of T.A.

The main indication for the therapy includes, clinically significant ischemia involving one or more vascular beds, renovascular hypertension, cerebrovascular ischemia, and upper extremity ischemia.

Intervention criteria: a single vessel lesion with critical stenosis over 75%, or involvement of at least three cerebrovascular vessels with greater than 50% luminal diameter narrowing.