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Post-interventional immunosuppressive treatment and vascular restenosis in Takayasu's arteritis

M. C. Park, S. W. Lee, Y. B. Park, S. K. Lee, D. Choi[1] and W. H. Shim[1]

Objective. **To investigate the outcome of vascular interventions and the effect of post-interventional immunosuppressive treatment on the occurrence of vascular restenosis in patients with Takayasu's arteritis (TA).**

Methods. **Forty-two patients with TA who had undergone vascular intervention and had serial angiographies before and after intervention were enrolled. The demographic and clinical data were collected at the time when the interventions were performed, and the intervention modalities and post-interventional medical treatments were evaluated.**

Results. **Sixty-three interventions were performed in 42 patients. Twenty (31.7%) interventions restenosed 24.0 ± 21.9 months after intervention; the likelihood decreasing as time passed. Estimates of arterial patency after intervention were 90.1% at 1 yr, 75.5% at 2 yr, 68.4% at 3 yr, 61.6% at 5 yr and 49.3% at 10 yr. According to the log rank test, interventions that were performed during the stable stage of the disease ($P = 0.039$) and those that were followed by treatment with glucocorticoids and immunosuppressive agents ($P = 0.044$) were independent variables for the maintenance of arterial patency. Their hazard ratios were 0.30 and 0.41, respectively.**

Conclusion. **Restenosis occurred in 31.7% of TA patients after intervention. A lower restenosis rate was observed when the vascular interventions were performed at the stable stage and when post-interventional immunosuppressive treatment was implemented.**

KEY WORDS: Takayasu's arteritis, Vascular intervention, Restenosis, Immunosuppressive treatment.

Takayasu's arteritis (TA) is a chronic inflammatory disease of the large arteries, primarily affecting the aorta and its main branches [1–3]. The course of TA usually extends over years with varying degrees of activity; however, most patients with TA show features of active disease at the time of diagnosis and present with symptoms and signs associated with vascular insufficiency, reflecting the previous establishment of end-organ ischaemia [4–10]. Kerr *et al.* reported that more than 70% of patients with TA had active disease at the time of diagnosis [11], and in those with active disease the suppression of active arterial inflammation prior to the development of significant vessel damage improved prognosis [12–16]. In this regard, the role of glucocorticoids in suppressing systemic symptoms and controlling disease activity has been emphasized in the literature [5, 12, 13, 17]. Furthermore, the addition of immunosuppressive agents, such as methotrexate and azathioprine, to glucocorticoids has been reported as effective in controlling disease activity and halting angiographic progression [13–16, 18]. However, the established vascular stenosis is not usually reversed by medical treatment [11, 19], and surgical or endovascular interventions have been performed to relieve the critical vascular stenosis in cases with established lesions with favourable short-term outcomes [19–24].

Restenosis of vascular lesions that have previously been revascularized by surgical or endovascular interventions occur commonly in patients with TA; persistent inflammation at the site of intervention seems to be responsible for this phenomenon [11, 25–27]. Frequent vascular restenosis can cause cumulative end-organ damage and can therefore lead to serious complications, such as valvular heart disease, cerebrovascular accidents, congestive heart failure and ischaemic heart disease, all of which are strongly associated with poor prognosis in patients with TA [6–8]. Thus, maintaining arterial patency after vascular interventions is crucial in preventing further organ damage and improving the prognosis of patients with TA. In this regard, it is ideal that vascular interventions are deferred until disease activity is by medical treatment because aneurysm formation, graft dehiscence and graft occlusion are more likely to occur during active inflammation. However, most TA patients present with features of active disease at the time of their diagnosis [11] and, on occasion, vascular interventions cannot be delayed because critical stenosis of arteries can lead to poor prognosis. But it is unknown whether the suppression of persistent vascular inflammation at the site of intervention with glucocorticoids and immunosuppressive agents can reduce post-interventional vascular restenosis in patients with TA who present with critical vascular stenosis and impending organ ischaemia.

We evaluated the outcomes of vascular interventions that had been performed in patients with TA and identified the independent variables associated with the maintenance of the arterial patency of revascularized lesions. Also, we sought to investigate whether post-interventional immunosuppressive treatment after surgical or endovascular interventions reduced vascular restenosis in TA.

Patients and methods

Patients

The medical records of 42 patients (four men and 38 women) with newly diagnosed TA who were seen at Yonsei University

Division of Rheumatology, Department of Internal Medicine, Institute for Immunology and Immunologic Diseases, Brain Korea 21 Project for Medical Science and [1]Division of Cardiology, Department of Internal Medicine, Yonsei Cardiovascular Center and Cardiovascular Research Institute, Yonsei University College of Medicine, Seoul, Korea.

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Correspondence to: Y. B. Park, Department of Internal Medicine, Yonsei University College of Medicine, 134 Shinchon-dong, Seodaemun-gu, Seoul, 120-752 Korea. E-mail: yongbpark@yumc.yonsei.ac.kr

Medical Center, Seoul, Korea, between January 1991 and June 2003 were reviewed retrospectively. All patients fulfilled the American College of Rheumatology classification criteria for TA [28] and had undergone surgical or endovascular interventions after diagnosis. We excluded patients who had been treated with glucocorticoids or immunosuppressive agents prior to intervention.

This study was approved by our ethics committee, and all study participants provided their signed informed consent.

Clinical assessment

At the time of intervention, mean disease durations from symptom onset and laboratory findings, including erythrocyte sedimentation rate (ESR, modified Westergren method, reference value <15 mm/h in men and <20 mm/h in women) and C-reactive protein (CRP, reference value <0.8 mg/dl) were evaluated. Disease activity was assessed according to the National Institutes of Health criteria for active disease [11]. These criteria included constitutional symptoms, such as fever and musculoskeletal symptoms, elevated ESR, features of vascular ischaemia or inflammation, such as claudication, diminished or absent pulse, bruit, vascular pain, blood pressure difference in the upper or lower extremities, and typical angiographic findings. New onset or worsening of two or more features defined active disease and a decrease in symptoms and signs or complete resolution of clinical features was indicative of stable disease.

Vascular interventions and angiographic assessment

The indications for surgical or endovascular interventions included renovascular hypertension, severe claudication of the extremities, cerebral infarct, and ischaemic heart disease. Intervention was considered successful if the residual stenosis was less than 30% of the luminal diameter with an increase of at least 50% from the pre-interventional diameter. The interventions that did not satisfy these criteria were excluded from analysis. Interventions performed after the age of 45 yr in men and 55 yr in women were also excluded because the risk of atherosclerosis increases significantly beyond these ages [29], preventing the clear discrimination of atherosclerotic lesions from vasculitic stenosis.

Aortic, coronary or peripheral angiographies were performed on all patients prior to intervention and post-interventional angiographies were followed to ascertain the patency of the revascularized lesions. Follow-up angiographies were performed routinely if the patients did not present new or recurrent symptoms suggesting restenosis. The schedule for routine follow-up was at 6–12 months after interventions and then biennially. In those with features of restenosis, follow-up angiographies were performed immediately. Mean angiographic follow-up duration was 33.7 ± 24.4 months (range 6–112 months). Restenosis was defined as a narrowing of the luminal diameter of more than 70% from the post-interventional diameter.

Statistical analysis

All statistical analyses were performed using SAS (version 8.1). Comparisons of disease activity, laboratory data, intervention modality, and post-interventional medical treatment modality between interventions that restenosed and those that remained patent upon follow-up angiography were performed using analysis of variance (ANOVA) or the independent *t*-test for continuous data and the χ^2 test for categorical data. Survival rates of arterial patency after intervention were calculated using the Kaplan–Meier method. A log rank test was performed to identify independent variables for vascular restenosis, and their hazard ratios were calculated using a Cox proportional hazards regression model. *P* values less than 0.05 were considered statistically significant.

Results

Modalities of interventions and medical treatment

Sixty-three interventions were performed in 42 patients. At the time of intervention, the mean age of the patients was 32.3 ± 11.3 yr (range 18–54 yr) and mean disease duration was 5.3 ± 4.1 months (range 0–19 months). The surgical intervention modalities included three endarterectomies (two for subclavian arteries and one for renal arteries) and 14 bypass grafts (seven for ascending aortas, four for descending aortas and three for abdominal aortas). The endovascular intervention modalities included eight percutaneous transluminal angioplasties (PTA, four for subclavian arteries, two for coronary arteries, one for common carotid arteries and one for renal arteries) and 38 PTAs with stent insertion (13 for subclavian arteries, 10 for common carotid arteries, nine for renal arteries, five for coronary arteries and one for superior mesenteric artery). High-dose glucocorticoids and immunosuppressive agents were given following 24 of the interventions. Prednisolone was started at a dose of 1 mg/kg/day for 1 month and tapered to a maintenance dose of 5–10 mg/day by 3–4 months. Oral methotrexate was added to glucocorticoids after 21 of the vascular interventions at a starting dose of 7.5 mg/week, and the dose was increased by 2.5 mg every 1–2 months to the maximal dose of 20 mg/week. Azathioprine was added to glucocorticoids after three of the vascular interventions at a starting dose of 1 mg/kg/day to the maximal dose of 2 mg/kg/day. High-dose glucocorticoids were given after 15 interventions without immunosuppressive agents according to the protocol described above. The remaining 24 interventions were not followed with either glucocorticoids or immunosuppressive agents. Aspirin and ticlodipine were administered after 54 and 47 interventions, respectively. Table 1 shows the univariate comparisons of demographic, clinical and laboratory characteristics at the time of intervention among the interventions followed by glucocorticoids and immunosuppressive agents, those followed by glucocorticoids alone, and those that were not followed by either glucocorticoids or immunosuppressive agent. The mean age, mean disease duration and laboratory parameters did not show a significant difference among the groups. However, the interventions that were performed in the active stage were more likely to be followed by glucocorticoids than those that were performed in the stable stage ($P < 0.05$). The addition of immunosuppressive agents was not significantly affected by disease activity.

Outcomes of vascular interventions

Restenosis of previously revascularized lesions occurred after 20 (31.7%) of the 63 interventions after an average of 24.0 ± 21.9 months (range 5–78 months) following intervention. Post-interventional restenosis were found in nine subclavian arteries, five common carotid arteries, two ascending aortas, two renal arteries, one coronary artery and one descending aorta. Table 2 shows the incidence of vascular restenosis on follow-up angiography according to the lag time from vascular intervention. Post-interventional restenosis occurred most frequently within the first year after intervention; the incidence tended to decrease as time passed.

Table 3 shows the results of the univariate analysis for comparisons of characteristics at the time of intervention between the interventions that restenosed and those that remained patent on follow-up angiography. The mean age, mean disease duration, laboratory parameters, modality of vascular intervention, and use

Table 1. Comparison of demographic, clinical, laboratory variables of the intervention groups with and without post-interventional medical treatments

	Followed by glucocorticoids and immunosuppressive agents ($n=24$)	Followed by glucocorticoids alone ($n=15$)	Followed by neither glucocorticoids nor immunosuppressive agent ($n=24$)	P
Mean age (yr)	32.0 ± 9.0	32.3 ± 11.1	33.1 ± 9.2	0.551
Mean disease duration (months)	5.7 ± 3.1	4.6 ± 2.4	5.3 ± 2.2	0.641
Disease activity				
Active	20	13	5	0.033
Stable	4	2	19	0.040
Laboratory parameters				
ESR (mm/h)	45.2 ± 29.0	50.1 ± 20.7	37.3 ± 34.5	0.103
CRP (mg/dl)	2.9 ± 1.5	2.7 ± 1.7	2.0 ± 2.1	0.366

Clinical, laboratory and disease activity-related variables were assessed at the time when the interventions were performed. Data were analysed by ANOVA.

Table 2. Incidence of post-interventional vascular restenosis on follow-up angiography according to the lag time from vascular interventions

	Vascular interventions	
Lag time	Followed by angiography n	Restenosed n (%)
<1 yr	12	6 (50.0%)
1–2 yr	18	8 (44.4%)
2–3 yr	9	3 (33.3%)
3–4 yr	7	1 (14.3%)
4–5 yr	8	1 (12.5%)
5–6 yr	3	0 (0%)
>6 yr	6	1 (16.7%)
Total	63	20 (31.7%)

of aspirin or ticlodipine were not different between the two groups, but disease activity and post-interventional immunosuppressive treatment showed significant differences in vascular outcome. Seventeen (44.7%) of the 38 interventions performed during the active stage restenosed, and three (12.0%) of the 25 interventions performed at the stable stage restenosed. Restenosis occurred more frequently after interventions performed during the active stage than after interventions performed at a stable stage ($P<0.05$). Two (8.3%) of the 24 interventions that were followed by glucocorticoid and immunosuppressive agent treatment restenosed, and six (40.0%) of the 15 interventions that were followed by glucocorticoids alone restenosed. Twelve (50.0%) of the 24 interventions that were not followed by either glucocorticoids or immunosuppressive agents restenosed. A significantly lower restenosis rate was found after interventions that were followed by glucocorticoids with immunosuppressive agents compared with those that were followed by glucocorticoids alone and those that were not followed by medical treatment ($P<0.05$). The difference between the latter two cases was not significant.

Independent variables for arterial patency

Estimates of arterial patency after intervention were 90.1% at 1 yr, 75.5% at 2 yr, 68.4% at 3 yr, 61.6% at 5 yr and 49.3% at 10 yr. According to the log rank test with the Kaplan–Meier method, vascular interventions that were performed at the stable stage of the disease ($P<0.05$) and those that were followed by glucocorticoids and immunosuppressive agents ($P<0.05$) had a significantly higher patency rate (Fig. 1). A Cox proportional hazards regression model revealed that the hazard ratio for the occurrence of restenosis of the former was 0.30, while that of the latter was 0.41. Demographic data, laboratory findings (at the time of intervention), the site of involved arteries, and intervention modality were not significantly associated with the occurrence of post-interventional vascular restenosis.

Table 3. Comparison of demographic, clinical, laboratory, intervention-related and medical treatment-related variables between the interventions that restenosed and the interventions that remained patent

	Restenosed ($n=20$)	Patent ($n=43$)	P
Mean age (yr)	31.9 ± 7.7	32.6 ± 12.0	0.883
Mean disease duration (months)	5.5 ± 2.0	5.0 ± 3.2	0.591
Disease activity			
Active	17	21	<0.001
Stable	3	22	0.004
Laboratory parameters			
ESR (mm/h)	48.5 ± 30.2	37.4 ± 23.7	0.116
CRP (mg/dl)	2.5 ± 2.0	1.9 ± 1.6	0.219
Modality of intervention			
Surgical	4	13	0.297
Endarterectomy	1	2	0.689
Bypass graft	3	11	0.275
Endovascular	16	30	0.297
PTA	4	4	0.214
PTA with stent insertion	12	26	0.593
Medical treatment			
Glucocorticoid with immunosuppressive agent	2	22	0.002
Glucocorticoid alone	6	9	0.314
No glucocorticoids	12	12	0.024
Aspirin	17	37	0.595
Ticlodipine	13	34	0.187

Clinic and disease activity-related variables were assessed at the time when the interventions were performed. Data were analysed by independent t-test or χ^2 test.

Discussion

It is well recognized that restenosis of vascular lesions that have been revascularized previously by intervention can occur in patients with TA [25–27]. In this study, 31.7% of revascularized lesions restenosed after intervention, and the incidence of restenosis was highest within the first year and decreased as time passed. This time-dependent decrement is similar to that observed in atherosclerosis, but the restenosis rate is much higher than that seen in cases with atherosclerosis [30–33].

In patients with atherosclerosis, the outcome after surgery seems to be more favourable than after endovascular procedures. Serruys *et al.* [34] evaluated the outcomes of 1205 patients who were

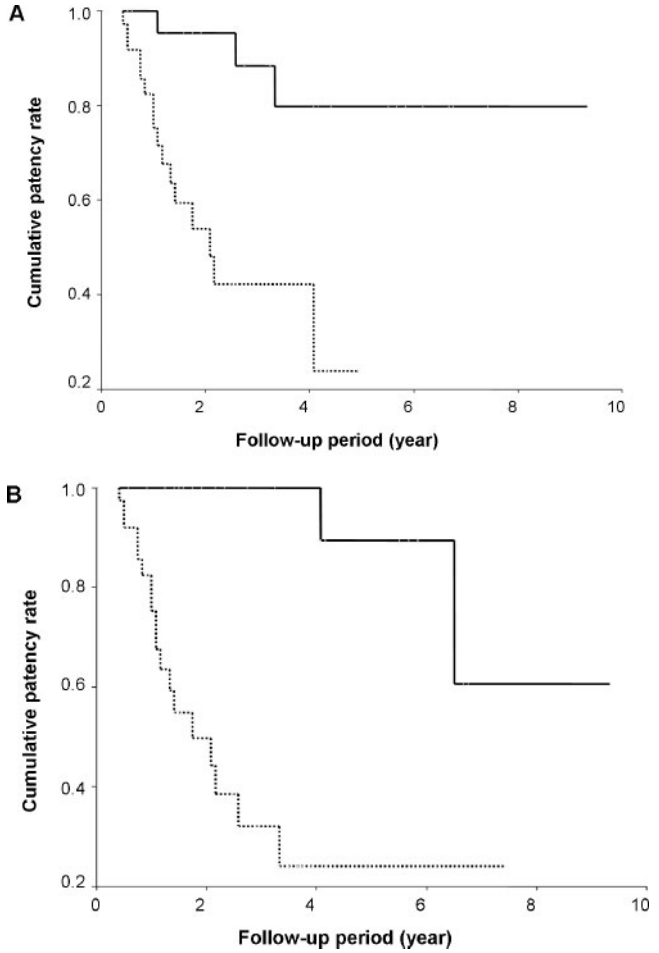

FIG. 1. Comparison of patency rates of revascularized lesion after interventions. (A) Patency rate of revascularized lesions was higher after interventions performed at the stable stage (continuous line) than after interventions at the active stage (dashed line) ($P = 0.005$). (B) Patency of revascularized lesion was well maintained when glucocorticoid with immunosuppressive agent had been added after intervention (continuous line) compared with interventions followed by glucocorticoid alone (dotted line) ($P = 0.015$).

randomly assigned to undergo stent implantation or bypass surgery and found that 16.8% of patients in the stent group underwent a second revascularization, compared with 3.5% from the surgery group. Stables *et al.* [35] suggested that the use of coronary stents has reduced the need for repeat revascularization compared with balloon angioplasty, though the rate remains significantly higher than in patients managed with bypass surgery. Similar results favouring surgical revascularization were reported in patients with TA. Liang *et al.* [27] described the results of angiographic follow-ups of 52 vascular interventions in patients with TA who were in clinical remission and found that the restenosis rates were as high as 35% after surgery and 57% after endovascular intervention. Although the short-term benefits of endovascular interventions in TA, such as the relief of vascular symptoms, are well defined [19–24], surgery seems to have more favourable outcomes than endovascular interventions if the maintenance of vascular patency is considered more important as a long-term outcome [25–27]. But, in contrast to these previous findings, the intervention modality did not affect the vascular outcome in this study. We found that the more important determinants of vascular restenosis were active disease at the time of intervention and the absence of post-interventional immunosuppressive treatment. This may be because many of our patients had active vascular inflammation at the time they had undergone vascular intervention. Indeed, the restenosis rate after interventions that were performed during active disease was significantly higher than that of those performed during remission in this study, and the outcome of the latter cases was comparable to that of interventions performed in atherosclerosis cases [30–36]. Although the exact explanation for the higher restenosis rate in the active disease group is unclear, active vascular inflammation at the site of intervention might cause restenosis after intervention, as previously suggested [11, 25–27]. This finding thus raises the possibility that the suppression of vascular inflammation with glucocorticoids and/or immunosuppressive agents may reduce restenosis in patients with TA receiving vascular interventions.

Ideally, vascular interventions are deferred until disease activity is controlled by medical treatment because aneurysm formation, graft dehiscence and graft occlusion are more likely to occur during active inflammation [19, 20]. On the other hand, Ishikawa *et al.* [7, 8] suggested that the presence of serious complications, such as valvular heart disease, cerebrovascular accidents, congestive heart failure, ischaemic heart disease and renovascular hypertension, was a significant predictor of mortality in patients with TA, and Kerr *et al.* [11] reported that histologically active vasculitis was present in 44% of specimens obtained from patients with TA who were in clinical remission. Considering this, delaying intervention until active inflammation is controlled may be harmful in cases involving critical stenosis and impending organ ischaemia, which should be resolved immediately. In these cases, prompt intervention is needed, although this approach may result in a higher rate of restenosis, to prevent further organ damage, which is associated with a poor prognosis.

The roles of glucocorticoids and immunosuppressive agents in TA have been investigated widely. Amelioration of systemic symptoms was achieved in more than half of glucocorticoid-treated patients with TA [4, 10]. Meanwhile, immunosuppressive agents have been reported as effective in controlling disease activity, even halting the progression of angiographic lesions [5, 12, 13, 17]. However, reports regarding whether glucocorticoids arrest angiographic progression are conflicting, and previous studies have shown that glucocorticoids alone in active TA are successful in arresting disease progression in fewer than 50% of patients, necessitating the use of additional immunosuppressive agents [11, 14, 37, 38]. In this study, the administration of glucocorticoids and immunosuppressive agents was performed in a manner similar to standard protocols used in previous studies [11–16], and we found that glucocorticoids used in conjunction with immunosuppressive agents significantly reduced the risk of vascular restenosis. Although interventions followed by glucocorticoids alone showed a lower restenosis rate than those not followed by medical treatment, glucocorticoids alone did not reduce the rate of restenosis significantly in this study. This result, along with those from previous studies, suggests that glucocorticoids alone may be insufficient to prevent vascular restenosis; thus, the addition of immunosuppressive agents to glucocorticoid treatment is needed to maintain arterial patency after intervention.

This study was performed in a retrospective manner and has some potential limitations: (i) heterogeneous treatment modalities and the treatment durations present a risk of significant bias when assessing the effects of the medical treatments on vascular outcome; and (ii) although the addition of immunosuppressive agents to glucocorticoids was allocated in a random manner, the initiation of glucocorticoids was allocated based on the clinical and laboratory findings, which also can cause a bias influencing the differences in vascular outcomes. In order to clarify these issues, prospective controlled studies will be needed to better identify the

effects of the medical treatment and disease activity on vascular outcome.

In practice, it is important to revascularize the stenosis with surgical or endovascular interventions to prevent further organ damage in patients with TA. However, the maintenance of arterial patency is also important, as frequent restenosis of vascular lesions may lead to serious complications and a decrease in quality of life. In cases of mild and non-urgent stenosis, controlling vascular inflammation with medical treatment prior to vascular intervention seems to be beneficial. However, prompt revascularization procedures are needed in cases with severe stenosis or impending organ ischaemia, with more favourable outcomes expected if they are followed by post-interventional immunosuppressive treatment.

Rheumatology	Key messages
	• Vascular restenosis occurred after 31.7% of interventions in patients with Takayasu's arteritis. • A lower restenosis rate was observed when the interventions were performed at the stable stage and when post-interventional immunosuppressive treatment was implemented.

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