Retrospective Analysis of Surgery Versus Endovascular Intervention in Takayasu Arteritis
A Multicenter Experience

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Background—With recent advances in endovascular treatment, percutaneous endoluminal angioplasty has become particularly attractive for arterial lesions of Takayasu arteritis. However, data came from case reports or small series, and the long-term outcome has not been reported. The incidence of potential vascular complications after surgery or endovascular treatment is still to be determined.

Methods and Results—This retrospective multicenter study analyzed the results and outcomes of 79 consecutive patients with Takayasu arteritis (median age, 39 years; interquartile range [IQR], 25–50 years; 63 women [79.7%]) who underwent 166 vascular procedures (surgery, 104 [62.7%]; endovascular repair, 62 [37.3%]) for the management of arterial complications. After a follow-up of 6.5 years (IQR, 2.2–11.5 years), 70 complications were observed, including restenosis (n=53), thrombosis (n=7), bleeding (n=6), and stroke (n=4). The overall 1-, 3-, 5-, and 10-year arterial complication–free survival rates were 78% (IQR, 69%–88%), 67% (IQR, 57%–78%), 56% (IQR, 46%–70%), and 45% (IQR, 34%–60%), respectively. Among the 104 surgical procedures, 39 (37.5%) presented a complication compared with 31 of the 62 (50%) with endovascular repair. In multivariate analysis, biological inflammation at the time of revascularization (odds ratio, 7.48; 95% confidence interval, 1.42–39.39; P=0.04) was independently associated with the occurrence of arterial complications after the vascular procedure. Patients who experienced complications had higher erythrocyte sedimentation rates (P<0.001) and C-reactive protein (P<0.001) and fibrinogen (P<0.005) serum levels compared with those without complications.

Conclusions—The overall 5-year arterial complication rate was 44%. Biological inflammation increased the likelihood of complications after revascularization in patients with Takayasu arteritis. (Circulation. 2012;125:813-819.)

Key Words: angioplasty • inflammation • surgery • Takayasu arteritis • vasculitis

Takayasu arteritis (TA) is a chronic inflammatory arteritis of unknown cause affecting large vessels, predominantly the aorta and its main branches. Vessel inflammation leads to wall thickening, fibrosis, stenosis, and thrombus formation. Symptoms reflect end-organ ischemia. More acute inflammation can destroy the arterial media and lead to aneurysm formation. Although once thought to be a disorder that affected mostly young Asian women, TA has been identified in both sexes and many ethnic and racial groups worldwide.

Clinical Perspective on p 819

The goal of the treatment of TA is to relieve inflammation in the arteries and to prevent potential complications. Even with early detection and treatment, TA can be challenging to manage. First-line treatment of TA is immunosuppression, primarily with corticosteroids. With glucocorticoid treatment, remissions occur in 40% to 60% of all patients. About 40% of all steroid-resistant patients respond to the addition of cytotoxic agents. Approximately 20% of all patients are resistant to any kind of treatment. Therefore, aggressive medical and surgical treatment is required for patients suffering from major complications and a progressive disease course. Recent advancements in medical and surgical treatments, including endovascular interventions, have improved the prognosis of patients with TA. Initial reports, provided mainly from case reports or small series, have

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revealed interesting results of endovascular therapy for treating patients with TA.\textsuperscript{10–12} Furthermore, the long-term outcome, including that of the recently developed endovascular treatment, has not been reported.\textsuperscript{13} The incidence of potential vascular complications after surgery or endovascular treatment is still to be determined. In addition, the impact of adequate antinflammatory treatment before vascular intervention is unknown. Here, we report the results of a large multicenter study analyzing the outcome of vascular surgery and endovascular repair in the management of arterial complications of TA. Logistic regression was done to assess factors associated with complications after vascular intervention.

Methods

Study Population

The present study is based on a retrospective analysis of 79 consecutive patients with TA followed up in the departments of internal medicine (Groupe hospitalier Pitie-Salpetriere, Paris, and Centre hospitalier regional Universitaire, Lille) and vascular medicine (Hôpital européen Georges-Pompidou [AP-HP], Paris) between 1992 and 2010. All the TA patients fulfilled the 1990 American College of Rheumatology criteria for the classification of TA.\textsuperscript{14} Potential confounding conditions (Cogan syndrome, Behçet disease, Kawasaki disease, syphilis, tuberculosis aortitis, vascular Ehlers-Danlos syndrome, Marfan syndrome, and neurofibromatosis) were excluded. The diagnosis of TA was based on clinical, biological, and imaging data (ie, vascular echocardiography, arteriography, angiographic computed tomography, and/or magnetic resonance angiography). For each patient, the following data were collected: age at diagnosis of TA and at the time of revascularization, sex, geographic origin, cardiovascular risk factors (ie, smoking, hypertension [systolic blood pressure >140 mm Hg and/or diastolic blood pressure >90 mm Hg], hypercholesterolemia [low-density lipoprotein cholesterol >130 mg/dL], overweight [body mass index >25 kg/m\textsuperscript{2}], diabetes mellitus), clinical features of TA (systemic and/or vascular presentations), blood pressure at the time of revascularization, inflammatory laboratory data (erythrocyte sedimentation rate and C-reactive protein and fibrinogen serum levels), arterial imaging, detailed surgical and medical treatment at time of revascularization, and outcome. Biological inflammation was defined as erythrocyte sedimentation rate >30 mm/h and C-reactive protein >6 mg/L.

Revascularization Procedure

The decision between surgery or endovascular procedure was made by the patient’s treating physician. Indications for surgery or endovascular repair included severe vascular claudication, hypertension with critical renal artery stenosis, cerebrovascular ischemia with critical stenosis of cerebral vessels, cardiac ischemia with confirmed coronary artery involvement, and critical aortic stenosis or aneurysm. Control of blood pressure after revascularization was defined as systolic blood pressure <140 mm Hg and diastolic blood pressure <90 mm Hg.

Complications of the Vascular Procedure

Criteria for complications of the vascular procedure included the onset or worsening of vascular symptoms that was not attributable to another condition, onset of a cardiovascular complication, bleeding, and vascular lesion(s) and/or worsening of preexisting vascular lesions (ie, restenosis, thrombosis, and/or aneurysms) detected on serial imaging studies (ie, Doppler sonography, angiographic computed tomography scan, and/or angiographic magnetic resonance imaging). Significant arterial restenosis was defined as >75% narrowing of the lumen. At each visit, criteria for disease activity were applied on the basis of symptom assessment, physical examination, and laboratory studies.\textsuperscript{2} Arterial imaging was performed every 6 months or sooner if there was uncertainty about disease progression.

Statistical Analysis

Data are summarized as frequencies and percentages for categorical variables. Quantitative variables are expressed as median and interquartile range (IQR). Differences between the surgical group and the endovascular group were tested with either the Fisher exact test or Wilcoxon test. Associations between complications at 12 months and variables were assessed with logistic random-effects regression models with patients as the random factor and variables as fixed factors to take into account the clustering of the observations. In practice, the glmmML routines in the R software package were used. All factors with a value of \( P < 0.1 \) in the univariate analysis were included in a multiple logistic random-effects regression model. Odds ratios (ORs) and their 95% confidence intervals (CIs) are presented as a measure of association. The association between vascular intervention and occurrence of a second vascular intervention or death was assessed with a univariate Cox proportional hazard model. The proportional hazards assumption for the Cox regression model fit was fulfilled. Survival curves were estimated by the Kaplan-Meier method. Follow-up estimation (median and IQR) was calculated with an inverse Kaplan-Meier method. All tests were 2 sided at the 0.05 significance level. All statistical analyses were carried out with the SAS 8.2 software package (SAS Inc, Cary, NC) and the R 2.10.1 software package (R statistical package; http://www.R-project.org).

Results

Characteristics of Patients With TA

Table 1 reports the baseline characteristics of the 79 predominantly female patients (79.7%) with TA included. Their median age at diagnosis of TA was 39 years (IQR, 25–50 years). Their median age at time of the arterial procedure was 45 years (IQR, 31–55 years). Patients originated mainly in Europe (48.1%) and to a lesser extent in North Africa (21.5%) or Africa (10.1%). Cardiovascular risk factors included arterial hypertension (n = 39), smoking (n = 21), hypercholesterolemia (n = 15), overweight (n = 14), and diabetes mellitus (n = 9). Vascular symptoms were reported by 46 patients (58.2%). The main presenting features of TA were arterial claudication of extremities (n = 31) typically of the lower limbs (58.1% of these patients), absence or asymmetry of upper- or lower-extremity pulse(s) (n = 12), new-onset hypertension (n = 11), vascular bruits (n = 10), asymmetry of blood pressure (>15 mm Hg; n = 10), stroke (n = 6), and angina pectoris (n = 5). General symptoms were present in 10 patients (12.6%): fever in 3, arthralgia in 3, erythema nodosa in 3, scleritis in 2, and myalgia in 1. Study of associated diseases revealed that 1 patient had Crohn disease and 1 had sarcoidosis. Increased acute-phase proteins were measured in 34 patients (43%) with an erythrocyte sedimentation rate and C-reactive protein and fibrinogen serum levels of 29 mm/h (IQR, 13–53 mm/h), 6 mg/L (IQR, 4–21 mg/L), and 3.8 g/L (IQR, 3.1–5.3 g/L), respectively.

The 79 patients with TA underwent 166 vascular procedures, including 104 bypass surgeries (62.7%) and 62 percutaneous angioplasties (37.3%). Of the 62 patients with TA who had endovascular procedures, 42 (67.7%) had stenting. The type and territory of arterial lesions requiring the 166 vascular procedures are detailed in Table 2. Stenosis or occlusion and aneurysms represented 137 (82.5%) and 29 (17.5%) of the 166 arterial lesions, respectively. Overall, 31
Cardiovascular risk factors (n=79), n (%)  
- Hypertension* 39 (49.4)
- Smoking 21 (26.6)
- Hypercholesterolemia† 15 (18.9)
- Overweight‡ 14 (17.7)
- Diabetes mellitus 9 (11.4)

Vascular symptoms, n (%) 46/79 (58.2)  
- Arterial claudication 31 (67.4)
- Absence of pulse 12 (26.1)
- New-onset hypertension 11 (23.9)
- Asymmetry of blood pressure§ 10 (21.7)
- Vascular bruises 10 (21.7)
- Stroke 6 (13.0)
- Angina pectoris 5 (10.8)

Biology  
- ESR, mm/h 29 (13.5–53)
- C-reactive protein, mg/L 6 (4–21)
- Fibrinogen, g/L 3.8 (3.1–5.3)

ESR indicates erythrocyte sedimentation rate. Values are median (interquartile range) when appropriate.

*Hypertension was defined as systolic blood pressure >140 mm Hg and/or diastolic blood pressure >90 mm Hg.
†Hypercholesterolemia was defined as low-density lipoprotein cholesterol >130 mg/dL.
‡Overweight was defined as body mass index ≥25 kg/m².
§Asymmetry of blood pressure was defined as blood pressure difference >15 mm Hg.

(18.7%) were aortic, 24 (14.4%) were subclavian, 22 (13.2%) were renal, 19 (11.4%) were carotid, and 16 (9.6%) were iliac arterial lesions. Treatments at the time of the revascularization are summarized in Table 2. There were 166 vascular procedures: 104 surgeries (62.7%) and 62 endovascular interventions (37.3%). The 104 surgical revascularizations included 68 prosthetic bypasses (65.4%) and 36 vein bypasses (34.6%). The main surgical procedures included carotid-subclavian, carotid-axillary, subclavian-humeral, aorta-iliac, aorta-femoral, aorta-subclavian, aorta-aorta, aorta-renal, and celiac-mesenteric bypass.

Medical therapy at the time of the revascularization consisted of corticosteroids in 73 of the 166 vascular procedures (43.9%) and immunosuppressants in 32 (19.3%), including methotrexate (n=27), mycophenolate mofetil (n=3), and azathioprine (n=2). In addition, patients received low-dose aspirin (95 of 166, 57.2%), anticoagulants (12 of 166, 7.2%), and statins (39 and 166, 23.5%).

Complications After the Vascular Procedure  
A detailed analysis of the 70 complications (42.2%) after the 166 vascular procedures is summarized in Table 3. Procedure-related complications were classified as early (ie, <30 days) and late (>30 days). The time between revascularization and complications was 6 months (IQR, 1–36 months), with one third occurring within the first 3 months. Among the 104 surgical procedures, 39 (37.5%) presented a complication compared with 31 (50%) of the 62 endovascular repair. Of the 42 patients who had an endovascular procedure with stenting, 20 (47.6%) experienced a vascular complication. Main complications included restenosis (n=53, 75.7%) and, to a lesser extent, thrombosis (n=7, 10%), bleeding (n=6, 8.6%), and stroke (n=4, 5.7%). Of the 6 surgical thromboses, 4 and 2 occurred with prosthetic and vein graft reconstruction, respectively. The main sites of arterial lesions affected by a complication were the aorta in 17 (24.3%), renal artery in 14 (20%), carotid artery in 10 (14.3%), subclavian artery in 9 (12.8%), iliac artery in 9 (12.8%), and mesenteric artery in 6 (8.5%). Compared with surgery, there was no difference in terms of laboratory data or medical treatment at time of revascularization with an endovascular procedure. The complication rate when there was evidence of biological inflammation was 61.5% and 64% for endovascular repair and surgery, respectively.
Outcome
The time of follow-up after the vascular procedure was 6.5 years (IQR, 2.2–11.5 years). Four deaths (1 after surgery and 3 after endovascular repair) were due to severe sepsis (n=2), stroke (n=1), and mesenteric ischemia (n=1). The 1-, 3-, 5-, and 10-year survival rates were 97% (IQR, 94%–100%), 96% (IQR, 91%–100%), 96% (IQR, 91%–100), and 92% (IQR, 84%–100%), respectively. The overall 1-, 3-, 5-, and 10-year arterial complication–free survival rates were 78% (IQR, 69%–88%), 67% (IQR, 57%–78%), 56% (IQR, 46%–70%), and 45% (IQR, 34%–60%), respectively (Figure 1). The 5- and 10-year arterial complication–free survival rates were 60% and 57% versus 49% and 29% for the surgery group versus the endovascular repair group, respectively. Biological inflammatory parameters at the time of the vascular procedure were significantly different according to the occurrence of a complication (Figure 2). Patients who experienced complications after the vascular procedure had higher erythrocyte sedimentation rates (45 mm/h [IQR, 22–88 mm/h] versus 24 mm/h [IQR, 7–34 mm/h]; \(P<0.001\)) and C-reactive protein (10 mg/L [IQR, 6–46 mg/L] versus 5 mg/L [IQR, 4–17 mg/L]; \(P<0.001\)) and fibrinogen (4.0 g/L [IQR, 3.8–6 g/L] versus 3.2 g/L [IQR, 2.5–3.9 g/L]; \(P<0.005\)) serum levels at time of revascularization compared with those without complications, respectively (Figure 2).

Table 3. Main Complications After 166 Vascular Procedures Performed in 79 Patients With Takayasu Arteritis

<table>
<thead>
<tr>
<th></th>
<th>All (n=70)</th>
<th>Surgery (n=39)</th>
<th>Endovascular Repair (n=31)</th>
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<tbody>
<tr>
<td>Early complications (n=21), n (%)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restenosis</td>
<td>15 (21.4)</td>
<td>8 (20.5)</td>
<td>7 (22.6)</td>
</tr>
<tr>
<td>Bleeding</td>
<td>5 (7.1)</td>
<td>3 (7.7)</td>
<td>2 (6.4)</td>
</tr>
<tr>
<td>Stroke</td>
<td>1 (1.4)</td>
<td>0</td>
<td>1 (3.2)</td>
</tr>
<tr>
<td>Late complications (n=49), n (%)†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restenosis</td>
<td>38 (54.3)</td>
<td>18 (46.1)</td>
<td>20 (64.5)</td>
</tr>
<tr>
<td>Thrombosis</td>
<td>7 (1.0)</td>
<td>6 (15.4)</td>
<td>1 (3.2)</td>
</tr>
<tr>
<td>Stroke</td>
<td>3 (4.3)</td>
<td>3 (7.7)</td>
<td>0</td>
</tr>
<tr>
<td>Bleeding</td>
<td>1 (1.4)</td>
<td>1 (2.6)</td>
<td>0</td>
</tr>
<tr>
<td>Death, n (%)</td>
<td>4 (5.7)</td>
<td>1 (2.6)</td>
<td>3 (9.6)</td>
</tr>
</tbody>
</table>

Biology‡

<table>
<thead>
<tr>
<th></th>
<th>All (n=70)</th>
<th>Surgery (n=39)</th>
<th>Endovascular Repair (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESR, median (IQR), mm/h</td>
<td>29 (14–51)</td>
<td>26 (13–50)</td>
<td>30 (21–83)</td>
</tr>
<tr>
<td>C-reactive protein, median (IQR), mg/L</td>
<td>7 (4–21)</td>
<td>5 (4–25)</td>
<td>8.1 (4–19)</td>
</tr>
<tr>
<td>Fibrinogen, median (IQR), g/L</td>
<td>3.8 (3–5.3)</td>
<td>3.7 (3–4)</td>
<td>4 (3–6)</td>
</tr>
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Treatments, n (%)

<table>
<thead>
<tr>
<th></th>
<th>All (n=70)</th>
<th>Surgery (n=39)</th>
<th>Endovascular Repair (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steroids</td>
<td>28 (40)</td>
<td>18 (42.8)</td>
<td>10 (31.2)</td>
</tr>
<tr>
<td>Immunosuppressants</td>
<td>20 (28.6)</td>
<td>11 (28.2)</td>
<td>9 (29.1)</td>
</tr>
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</table>

ESR indicates erythrocyte sedimentation rate; IQR, interquartile range.
*Early complications are procedure-related complications occurring at 30 days after the procedure.
†Late complications are procedure-related complications occurring >30 days after the procedure.
‡Biology and treatments were recorded at the time of revascularization.

Figure 1. Arterial complication–free survival.

Figure 2. Biological inflammatory parameters at the time of the vascular procedure and according to the occurrence of a complication. Erythrocyte sedimentation rate (ESR) level, C-reactive protein serum level, and Fibrinogen serum level.
Factors Associated With Complications After the Vascular Procedure

In univariate analysis, the use of anticoagulants (OR, 30.89; 95% CI, 5.28–180.69; \(P<0.001\)), aspirin (OR, 4.85; 95% CI, 1.1–21.46; \(P=0.043\)), and immunosuppressants (OR, 11.69; 95% CI, 2.8–48.88; \(P<0.001\)) and biological inflammation (OR, 4.99; 95% CI, 1.26–19.77; \(P=0.04\)) were associated with the occurrence of complications (Table 4). In multivariate analysis, biological inflammation (OR, 7.48; 95% CI, 1.42–39.39; \(P=0.04\)) was independently associated with the occurrence of complications (Table 4).

Discussion

Our experience is derived from a large cohort of patients with TA who underwent vascular procedures. Our aim was to analyze the long-term outcomes of revascularization procedures in patients with TA and to assess factors associated with postprocedural complications. The most striking conclusions drawn by this study are that the overall 5-year arterial complication rate was 44% and that biological inflammation at the time of revascularization increases by 7 times the likelihood of complications in patients with TA.

The initial results of endovascular treatment of the carotid, subclavian, and renal arteries in TA were first reported in 1980, predominantly from Asian countries.\(^{10,11,15–17}\) Several reports came from small series. Data were encouraging, with initial success rates ranging from 74% to 100% and short-term restenosis occurring in \(<25\%\) of cases.\(^{10–12,18,19}\) Consistently, the overall restenosis rate in our study was 31%. Renal arterial restenosis accounted for the highest rate of complications after endovascular treatment; it occurred in up to one third of patients of our series. Such a complication significantly increased the morbidity of patients with TA, especially renal insufficiency and severe hypertension. To further explore the long-term outcome of surgery and endovascular procedures in patients with TA, we analyzed the association between vascular intervention and the occurrence of a second vascular intervention or death using a Cox proportional hazard model. This analysis allows us to study the association between the type of vascular intervention and the time to failure. The overall 1-, 3-, 5-, and 10-year arterial complication–free survival rates were 78%, 67%, 56%, and 45%, respectively. Among the 104 surgical procedures, 39 (37.5%) presented a complication compared with 31 of the 62 (50%)...
with endovascular repair. Surgery has been associated with low mortality and morbidity except for surgery on an aortic aneurysm, especially a ruptured aneurysm. Consistent with our data, the major surgical procedures in the Japanese nationwide surveys were aortocervical bypass, cervicosubclavian bypass, aortic replacement, aortocoronary bypass, replacement of aortic aneurysm, aortoaoartic bypass, aortorenal bypass, reconstruction of renal vessel, and nephrotoomy. Kieffer et al reported satisfactory early and long-term outcome in 24 patients with TA who underwent surgery for renal artery stenosis. Renal artery revascularization was unilateral in 46% and bilateral in 54%. During the 61.3-month follow-up, repeated renal artery revascularization was required in only 4 patients. Hypertension was cured in 63%, improved in 31%, and unchanged in 6%. On the other hand, Sharma et al published results of endovascular therapy for renovascular hypertension in 66 patients with TA. Technical success was seen in 89%. The stenosis decreased, systolic pressure gradient decreased, blood pressure improved, and drug requirement decreased. At 22 months of follow-up, the restenosis rate was 16%. However, the mean follow-up period was quite short. Technical problems related to the underlying disease are frequently encountered. Chronic inflammation involving all layers of the vessel wall, extensive periarterial fibrosis, thickening, and adhesions result in tough, noncompliant, rigid vessel walls. Furthermore, the results of a recent US cohort study were rather discouraging. Both endovascular and surgical revascularizations were initially successful, but restenosis occurred in 78% of the lesions treated with an endovascular procedure and 36% of those subjected to surgery. Interestingly, in our series, vascular complications were more likely to occur when biological inflammation was observed at the time of the vascular procedure, regardless of the type of vascular treatment. Biological inflammation at the time of revascularization increased by 7 times the likelihood of complications in patients with TA. Usually, surgery or endovascular repair is recommended at a time of quiescent TA disease to avoid complications. Matsuyama et al found that increased levels of inflammatory cytokines within the arterial lesions of TA induce production of matrix metalloproteinase (MMP)-3 and MMP-9 from infiltrated mononuclear cells and/or smooth muscle cells, which results in the destruction of elastic fibers in the arterial media. They observed a positive correlation between both MMP-3 and MMP-9 levels and disease activity score, and the elevated levels of MMP-3 and MMP-9 improved when patients entered remission. Numano et al advocated that the inflammation caused by TA would result in atherosclerotic lesions of the aorta and arteries. Hypertension, frequently found in patients with TA, may accelerate the atherosclerotic process in the long run. To improve the prognosis in patients with TA, it is therefore mandatory to prevent some potential complications caused by atherosclerotic disorders.

We acknowledge some limitations in our study. Our analysis was performed as a retrospective review of a cohort of patients whose revascularization was performed at different time courses of their disease, and we cannot rule out that the 2 groups could differ in their baseline characteristics. We were unable to collect complete longitudinal data on patients who were seen on only an intermittent consultation basis. Prospective enrollment and data collection from the time of diagnosis would be ideal but are more difficult to achieve with rare diseases.

The strengths of our study include a large cohort of patients with TA who have undergone surgical or endovascular revascularization and have a long-term outcome. The median time of follow-up in our series was 6.5 years, higher than observed in previous studies. We also provided a comprehensive standardized approach to the collection of data. Strikingly, our results are likely to be clinically meaningful for clinicians in charge of patients with TA.

Conclusions

We presented here the first study on the long-term outcome of revascularization procedures in patients with TA. The overall 3- and 5-year rates of arterial complication were 33% and 44%, respectively. Our study suggests that biological inflammation at the time of revascularization increased the likelihood of complications in patients with TA.

Disclosures

None.

References


**CLINICAL PERSPECTIVE**

Aggressive medical and surgical treatment is required for patients with Takayasu arteritis suffering from major complications and a progressive disease course. With recent advances in endovascular treatment, percutaneous endoluminal angioplasty has become particularly attractive for arterial lesions of Takayasu arteritis. However, the data have come from small series, and the long-term outcome has not been reported. We conducted a retrospective study to report the long-term outcome of 79 consecutive patients with Takayasu arteritis who underwent 166 vascular procedures (surgery, 104 [62.7%]; endovascular repair, 62 [37.3%]) for the management of arterial complications. The overall 1- and 3-year rates of arterial complication were 22% and 33%, respectively. In multivariate analysis, we found an independent association of biological inflammation (odds ratio, 7.48; 95% confidence interval, 1.42–39.39) with the occurrence of postprocedural arterial complications. Our study suggests that biological inflammation at the time of revascularization increased the likelihood of complications in patients with Takayasu arteritis.

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