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SPECIAL EDITION

Controversies in Surgery for Thoracic Aorta

Surgical strategy for refractory aortitis

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Abstract
In some instances, we encounter cases suffered from inflammatory aortic diseases (aortitis) in Japan, some of which are at the active stages with systemic inflammation. Most of them are refractory with some technical difficulties of surgical treatment. The aortic wall, particularly, at the active stage, is too fragile to hold the surgical sutures. Consequently, the suture reinforcement with Teflon felt is required. In the late stage after surgery, false aneurysms on the suture line, that is, suture detachment potentially occur. To prevent such sequelae in the early and late phases, continuous (life-long) as well as perioperative inflammation control using corticosteroid as an initial drug and/or other immunosuppression agents. This decade, instead of the conventional open surgical repairs, endovascular treatments have widely spread, predominantly for stenotic aortic/arterial lesions. In particular, for more difficult patients suffered from more troublesome Behçet disease, endovascular treatments would have greater advantages to avoid more occasionally occurred pseudo-aneurysm on the other parts as well as the surgical suture lines. The key issues on surgical treatment for refractory aortitis are perioperative inflammation control including the long term with corticosteroid and/or immunosuppressive agents, appropriate open surgical or endovascular treatment approaches, and sufficient reinforcement of surgical suture lines.

Keywords Aortitis · Takayasu arteritis · Behçet disease · Surgical treatment · Endovascular treatment

Introduction

Aortitis, an inflammatory aortic disease, is difficult to interpret and define because its etiology is multifactorial [1, 2]. Its most common cause is non-specific and non-infectious vasculitis due to Takayasu arteritis in Japan, whereas giant cell arteritis is predominant in the western countries [2]. Behçet disease, Buerger disease, Kawasaki disease, ankylosing spondylarthritis, and Reiter's syndrome are also associated with some inflammatory aortic disease. In addition, this disease category comprehends inflammatory aortic diseases due to bacterial or viral infection such as syphilis, tuberculosis, lupus, human immunodeficiency disease, including mycotic aortic aneurysms. This article focuses on the surgical strategies for non-infectious refractory aortitis such as Takayasu arteritis and Behçet disease affecting the great vessels with the predominant prevalence in Japan.

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Takayasu arteritis (TA)

Clinical presentation, incidence, and etiology

TA is a non-specific inflammatory disease of unknown etiology and causes various types of aortic or arterial stenoses or dilatation [1–5]. Its clinical manifestations are varied and related to the vessel presenting the stenotic or occlusive lesions, such as the brachiocephalic arteries (pulseless disease) [6], descending thoracic or abdominal aorta (atypical coarctation) [7], renal arteries, coronary arteries, and pulmonary arteries. In addition, aortic aneurysm formation and aortic valve regurgitation (AR) with ascending aortic dilatation potentially develop [8]. As a characteristic issue, one of the clinical features is strong predominance in young women in Asian countries and Latin America, while it is very rare in Caucasian people [9]. There are also some differences in the affected aortic/arterial sites. In Japanese patients, the ascending aorta and aortic arch with its branches are more

frequently involved, while in the other countries, the abdominal aorta and renal arteries are most frequently affected [9, 10]. Subsequently, AR used to be the main cause of mortality in Japanese patients, while cerebrovascular accidents relating to hypertension in other Asian and Western countries [9–13]. TA is divided into five groups according to the involved arteries in the classification of the 11th International Conference on TA in 1994; aortic branches (Type-I), ascending aorta, arch and its branches (Type-IIa), ascending aorta, arch with its branches and thoracic descending aorta (Type-IIb), thoracic descending aorta, abdominal aorta and/or renal arteries (Type-III), only the abdominal aorta and/or renal arteries (Type-IV), and the combined features of Type-IIb and -IV (Type-V) [14]. In addition, according to the stage, this disease is subdivided into the early, acute, or active phase and the late, chronic, or inactive one, which is also important for decision-making of the initial treatment of TA. Nowadays, TA is widely recognized as a multifactorial disease. Although the etiology of TA remains uncertain, autoimmunity and genetic factors have been considered to be involved in its pathogenesis [15–17].

Symptom and diagnosis

In the active stage, prominent symptoms are non-specific or absent, such as fever, general malaise, arthralgia, neck pain, and weight loss, which make the initial diagnosis difficult. Clues for the diagnosis are hypertension, vascular bruits, asymmetric arm blood pressure, and other ischemic symptoms. Ishikawa's criteria have been widely used for the initial diagnosis; one obligatory criterion (age ≦ 40 years), two major criteria (left and right mid subclavian artery lesions) and nine minor criteria (high erythrocyte sedimentation rate, common carotid artery tenderness, hypertension, AR or annuloaortic ectasia and lesions of the pulmonary artery, left mid common carotid artery, distal brachiocephalic trunk, thoracic aorta and abdominal aorta) [18]. Non-invasive diagnostic modalities including CT-scans and magnetic resonance angiography have allowed an early and easy detection of this condition [19]. In addition, F-fluorodeoxyglucose positron-emission CT imaging allows the visualization of the distribution of lesions and inflammatory activity [20, 21]. In blood examinations, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) have generally been used to examine the systemic inflammation. It is necessary to differentiate TA from other similar diseases such as giant cell arteritis, atherosclerotic disease, congenital coarctation of the aorta, and Behçet disease.

Treatment

Medical treatment In the active TA phase, medical treatment with corticosteroids is the initial treatment after the confirmed diagnosis [22, 23]. The guidelines recommend 30 mg/day of adrenocorticosteroids as the initial dose for adult patients with active TA [24]. It is tapered at a rate of 5 mg every 2 weeks down to 10 mg, and thereafter at a rate of 2.5 mg every 2 weeks until withdrawal or to the minimum required dose to control inflammation, while monitoring ESR and CRP. With difficulty of its withdrawal or dose reduction, immunosuppressive drugs are used [24]. In case of persistent inflammation, even in the inactive phase, the administration of corticosteroids should be continued to control systemic inflammation with less than CRP of 1.0 mg/dL and/or ESR of 20 mm/h [24]. In addition, patients in the active TA are often complicated by thrombosis in the affected vessels [25]. Long-term aspirin therapy is recommended to prevent thrombus formation in vessels with endothelial damage [26]. Antihypertensive agents are also frequently used because more than 70% of patients have hypertension related to atypical aortic coarctation or renovascular hypertension [24].

Endovascular therapy With recent advances in endovascular treatment, this has become attractive, especially for inactive stenotic/occlusive arterial or aortic lesions due to TA. In general, the indication for percutaneous balloon angioplasty (PTA) was symptomatic stenosis of more than 70% or a peak systolic gradient of more than 50 mmHg across the stenotic lesion [27]. The first case of PTA for atypical coarctation (middle aortic syndrome) was reported in 1984 [28]. Without severe calcification, PTA is indicated as the first-line treatment for atypical coarctation [29, 30]. However, because of rigid and non-compliant stenoses, higher balloon inflation pressure is required to stretch and split the thick and more rigid fibrous tissue compared with atherosclerotic aortic stenosis. In fact, fatal rupture during PTA was also reported [31]. In difficult cases, PTA should be done with some cautions, otherwise it should be avoided. On the other hand, there has been so far only a few reports on endovascular aortic (aneurysm) repair (EVAR) as the initial treatment despite its positive use for inflammatory abdominal aortic aneurysm [32, 33]. In general, with the currently available devices, (T) EVAR is indicated mainly for distal arch to descending or abdominal aortic lesions. In TA, dilated aortic pathologies tend to occur predominantly in the ascending and aortic arch, apart from the stenotic aortic lesions. Furthermore, most patients are relatively young with low open-surgery risks. Subsequently, the incidences of (T) EVAR have been lower in the standard clinical settings. However, such a less invasive procedure has already been well established and obviously can be indicated safely, in particular, in patients with chronic TA stage.

Open surgical treatment In adult cases with TA, less than 20% of patients require open surgical treatment [34, 35].

In the pediatric field, 80% of patients need open surgery predominantly for stenotic/occlusive lesions, because 70% of these patients are in the active TA phase and on steroid therapy [35]. The major surgical procedures are aortic graft replacement, aorto–aorto bypass, and aorto–branch bypasses to the cervical, coronary, renal, and visceral arteries. Cerebrovascular ischemic symptoms are common in the young patient population because of the high incidence of involvement of the arch-vessel in Japan [10]. Aorto–cervical bypass originating in the ascending aorta and anastomosed to the carotid bulb using saphenous vein grafts is recommended [36]. For aortic coarctation, an aorto–aortic or axillo–iliac/femoral artery bypass a prosthetic graft was the most commonly performed procedure [37]. Coronary artery involvement is reportedly at 10% [38]. The coronary ostium is most frequently involved [39]. In the previous experience of the National Cerebral and Cardiovascular Center, coronary artery disease was found in 41.3% ; coronary artery bypass grafting in 10 and coronary ostial endarterectomy in 9 [40]. Recently, successful percutaneous coronary intervention with drug-eluting stents was reported for proximal coronary disease in TA [41, 42].

Aneurysm formation is one of the major sequelae related to the prognosis in TA [43]. In Japan, the incidence of aneurysmal formation was 22.2–31.9%, while its rupture rate seems to be lower than that of atherosclerotic aneurysms [44]. AR associated with ascending aortic or root dilation is the major issue in Japan, which require concomitant root and aortic valve replacement using a composite valve-graft conduit [45–47]. Prosthetic valve and/or valve-graft detachment including coronary false aneurysm is the most serious sequela of this type of surgery in the long term, particularly in patients with persisting inflammation [45–47]. Technically, a "mini-skirt" technique, in which the stiff sawing ring of prosthetic valve is not directly attached to the annulus, is recommended for active-staged TA and other inflammatory diseases because of inflammatory fragility of the aortic annulus (Fig. 1) [46–48]. Compared with aortic valve replacement alone or standard composite valve-graft root replacement, the mechanical stress to the fragile inflamed annulus is expected to be less. On the other hand, aortic valve-sparing surgery (AVS) is not highly recommended for inflammatory root pathologies due to TA [48, 49]. The author experienced some redo cases with remarkable inflammatory changes of the aortic valves such as thickening and shortening leaflets after initially successful AVS, particularly, in cases with active TA. Without active inflammation of the aortic wall and valve, AVS is considered for selected patients and the outcome has been favorable in seven patients (one case with active TA) in the author's private experience. In aortic arch pathology due to TA, in our experiences, conventional total arch replacement was performed safely using the standard selective cerebral perfusion with deep hypothermia [50]. For descending and thoracoabdominal dilated aortic pathologies, standard prosthetic graft replacement was also carried out with favorable outcome [51].

Outcome

In the natural history of TA, four significant predictors exist: (1) major complications of Takayasu retinopathy, hypertension, AR, and aneurysm, (2) progressive course, (3) age, and (4) calendar year of diagnosis [32]. The 15-year survival was 66.3/96.4% with/without major complications, 67.9/92.9% with/without progressive course, 58.3/92.7% for

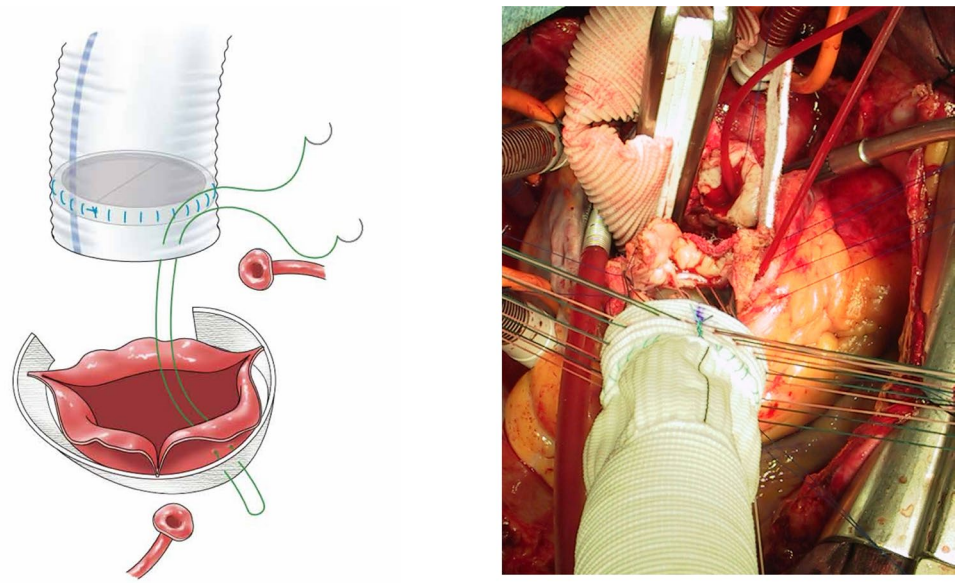

Fig. 1 Composite valve-graft root replacement (CVGRR) with a mini-skirt technique and suture reinforcement with an outside Teflon felt strip. Left: a schema, right: an operative finding

age > 35/≤ 35 years, and 79.9/96.5% in the early/late period. In the multivariate analysis, major complications, progressive course, and high ESR were independent predictors for the prognosis. It was described that the long-term survival was excellent up to 10 years and was not affected by disease activity or the use of steroids [33]. Even after successful PTA for arterial or aortic stenotic lesions, some pressure gradients tend to remain. Restenosis occurs reportedly in 15–20% during the mid-term follow up. To prevent restenosis, stenting is currently added, especially for ostial lesions, long segment lesions, or incomplete dilatation following PTA [52–54]. The long-term impact of PTA remains uncertain because of no reports on the longer period outcome. Only for atypical coarctation, the satisfactory long-term survival after surgical treatment was reported in a series of 33 patients followed up from 1960 to 2004 [55]. The presence of postoperative residual hypertension influenced the event-free and survival rates. Empirically, at open bypass surgery, large-size grafts of more than 12 mm in diameter should be used to relieve the stenosis sufficiently and to prevent residual proximal hypertension. Even after successful open surgery, anastomotic false aneurysms (anastomotic detachment) occur anytime in the long term. The previous report from Japan demonstrated the incidence of anastomotic false aneurysm of 8.5% (22/259) [56]. However, in recent cases with established surgical techniques using synthetic suture material, its incidence has decreased to 1.8% at 10 years and 3.5% at 20 years. In general, to prevent this complication, reinforcement of sutures using a Teflon felt or felt strip and careful suppression of persisting inflammation with corticosteroids or immunosuppressive agents are highly recommended [44–48]. Recently, successful TEVAR for dilated lesions due to TA was reported [32]. However, re-intervention for ruptured aneurysm after this type of endoluminal treatment was also reported [33]. The long-term efficacy of the endovascular aneurysmal repair remains uncertain even for standard pathology in younger patients suffered from TA. Particularly, in the inflammatory lesions due to TA, a positive but cautious approach might be necessary.

Finally, with active or inactive systemic inflammation, it is essential to suppress inflammation pharmacologically, determining the CRP level and ESR rate [48]. The inflammation due to TA would eventually result in atherosclerotic lesions of the aorta and arteries [57]. Hypertension is also associated with a high incidence of TA, which accelerates atherosclerosis in the long term.

Behçet's disease (BD)

Clinical presentation, incidence, and etiology

BD is a multi-systemic inflammatory disorder with gastrointestinal, ophthalmological, neurological, cardiovascular, musculoskeletal, and urogenital involvement [58, 59]. BD is also seen in young adults aged 20–40 years old predominantly in the Asian countries including Japan. The etiology remains unknown, but the HLA-B51 genotype is a risk factor associated with more severe disease.

Symptom and diagnosis

BD consists of recurrent ulcers in oral and genital mucosae and relapsing uveitis with unknown causes. The diagnosis is made with Diagnostic Criteria for Behçet's disease by Behçet's Disease Research Committee, Ministry Health, Welfare, and Labour, Japan [58, 59]. This disease is a multi-systemic inflammatory disorder with gastrointestinal, ophthalmological, neurological, cardiovascular, musculoskeletal, and urogenital involvement. Vascular lesions occur in 25–30%. Both arteries and veins are involved in most. The most common arterial site involved in BD is the abdominal aorta, followed by the pulmonary artery, femoral, popliteal, and carotid artery. Most of thoracic aorta diseases are involved in the aortic root with aortic regurgitation like TA. The incidence of arch to descending thoracic aneurysm is low.

Treatment

Medical treatment BD is treated primarily with immunosuppressive agents, particularly in the perioperative periods [58, 59]. Large-vessel arteritis is treated with corticosteroids in combination with azathioprine or cyclophosphamide.

Endovascular therapy Open surgical therapy carries a higher risk of recurrent false aneurysm on the anastomotic sites between the native aorta/artery and the prosthetic graft and the native aorta/artery in BD [60–64]. Consequently, TEVAR and PTA in conjunction with immunosuppressive therapy are recommended more for aneurysmal aortic or arterial lesions.

Open surgical treatment After surgery, anastomotic false aneurysm formation (detachment of anastomosis) occur at much higher incidence [65–72]. To prevent such lethal sequelae, more meticulous reinforcement to the anastomotic sites is required. In redo root surgery after prosthetic aortic valve replacement or composite valve-graft conduit repair, sufficient reinforcement of the aortic valve annulus with single or double Teflon felt strips is needed (Fig. 2) [73–76]. Aortic allograft (homograft) is another option to prevent anastomotic detachment [77]. Heart transplantation is an ultimate surgical option in redo cases after multiple surgeries [78]. In addition, careful inflammation control is required for favorable outcome without anastomotic false

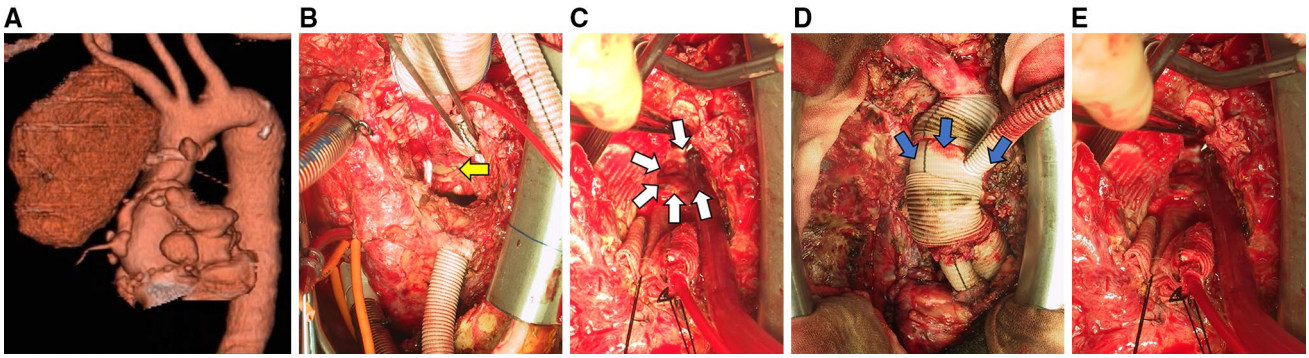

Fig. 2 A case of Behçet disease requiring re-redo CVGRR using a mini-skirt technique with suture reinforcement with outside and inside Teflon felt strips. **a** Spontaneously occurred a pseudo-aneurysm on the ascending aorta and a pseudo-aneurysm due to detachment of the root sutures beneath the left coronary artery and detachment of the bilateral coronary buttons. **b** A yellow arrow: an origin of the ascending aortic pseudo-aneurysm. **c** White arrows: detachment of the root sutures for the previous CVGRR. **d** Blue arrows: reinforcement of the aortic valve annulus. **e** CVGRR and hemiarch replacement with graft wrapping of the suture lines to prevent suture detachment

aneurysm or aneurysmal formation/rupture of the other aortic sites.

Outcome

In more troublesome BD, valve detachment after aortic valve replacement occurred in 4 (40%) of ten patients [74, 75]. Proximal anastomotic detachment occurred in 1 (20%) of the first five patients who underwent composite graft root replacement using the standard technique, whereas there was no detachment in the later five patients operated on with the mini-skirt technique [73]. At other anastomotic sites such as those for coronary and distal aortic reconstruction, no anastomotic false aneurysms occurred, presumably due to routine external Teflon felt reinforcement [73, 76].

Conclusions

Recent advances of medical and surgical treatment including endovascular interventions have improved the prognosis of patients suffered from refractory inflammatory vasculitis due to TA or BD. The incidences of potentially occurred vascular complications after surgical interventions including endovascular treatments have decreased due to careful medical control of systemic inflammation and appropriate surgical techniques for fragile cardiovascular tissues due to active inflammation.

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