Approach to Vascular Therapy for the Lower Legs: A Special Reference to Vascular Remodeling

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1 Introduction

We present our experiences with the diagnosis and endovascular treatment of lower leg arterial and venous diseases. First, we focus on the diagnosis and endovascular treatment of atherosclerotic disease. In particular, we discuss the therapeutic results for femoropopliteal arterial diseases, comparing bypass surgery and endovascular treatment, and examine the possibility of endovascular treatment for Trans-Atlantic Inter-Society Consensus (TASC) II C and D lesions, which are ordinarily contraindicated for endovascular treatment. Next, we present cases of iliofemoral venous disease. Differing from arterial diseases, venous disorders involve problems associated with venous valves and remodeling. We therefore discuss in detail the iliofemoral veno-occlusive diseases from the perspective of vascular remodeling and therapeutic options.

2 Artery

2.1 Pathogenesis of Arteriosclerotic Vascular Disease (AVD)

Atherosclerotic vascular lesions of the legs are related not only to ischemic damage to the leg tissue but also to a predisposition toward lethal systemic conditions, such as septicemia and acute embolic syndrome. Therefore, understanding the etiology of atherosclerotic vascular lesions is important. Hypertension, diabetes mellitus (DM), and hyperlipidemia, the so-called chronic adult diseases, are currently considered to represent risk factors for atherosclerotic vascular lesions. The underlying pathogenesis of atherosclerosis is an imbalance in lipid metabolism (Hansson & Hermansson, 2011), a maladaptive immune response, and chronic inflammation of the arterial wall. The main cause of the disrupted equilibrium among the accumulation (Hansson & Hermansson, 2011) and clearance of lipids and the immune responses is mediated by macrophage- (Moore & Tabas, 2011), neutrophil- (Zernecke, et al., 2008), T-cell- (Hansson & Hermansson, 2011) and dendritic cell-driven (Weber et al., 2008) pathways, and homeostasis is governed by chemokines and their receptors.

First, the absence of a confluent luminal elastin layer and the exposure of proteoglycans (Kwon et al., 2008; Weber & Noels, 2011) in the artery could induce the accumulation of LDL (low density lipoprotein). Second, an elevated level of circulating cholesterol transported by apolipoprotein B100 (ApoB100)-containing LDL generates atherosclerosis (Hansson & Hermansson, 2011). Third, LDA is exposed to oxidative modification, leading to chemokine secretion from endothelial cells (Weber & Noels, 2011). Fourth, the immune interaction among macrophages, neutrophils, T cells and dendrite cells leads to the accumulation of apoptotic cells, debris and cholesterol necrosis (Weber & Noels, 2011). Finally, fibroatheromatous plaques are composed of collagen and smooth muscle cells infiltrated by T cells and mast cells, creating a place where several inflammatory changes can occur (Weber & Noels, 2011) and resulting in atherosclerosis in the arterial wall.

2.2 Endovascular Treatment of ASD: Update

Endovascular treatment of atherosclerotic vascular lesions has been developing along with revolutionary advances in medical equipment. For common iliac or external iliac arterial lesions, endovascular treatments, including percutaneous transluminal angioplasty (or balloonplasty) and stent deployment, play major roles in restoring blood flow to the legs (Ichihashi, Higashiura et al., 2011; Mwipatayi, Thomas et al., 2011). However, controversy remains regarding the efficacy of endovascular treatment for vascular
lesions below the inguinal ligament (Conrad, Crawford et al., 2011; Baril, Chaer et al., 2010). To date, many comparative studies have examined balloon angioplasty (BAP) versus stent deployment (SD) (Chalmers, Walker et al., 2012) and BAP versus the use of a cutting balloon catheter (Canaud, Alric et al., 2008; Poncyljusz, Falkowski et al., 2013; Cardon, Jan et al., 2008; Cotroneo, Pascali et al., 2008; Garvin and Reifsnnyder, 2007). Chalmers et al. (2012) reported that at 12-month follow-up, there was no statistically significant difference in restenosis (stenting and PTA: 40.8%; PTA: 46.7% (p = 0.68)) for a long superficial femoral artery stenosis. There were fewer target lesion revascularizations in the patients randomized to stenting, but this result did not reach statistical significance (12.5 vs. 20.8%, p = 0.26). There was no difference in the rate of amputation. Patients in both groups reported an improved quality of life. Therefore, Chalmers et al. could not demonstrate the superiority of PTA plus stenting compared to PTA alone. Poncyljusz (Poncyljusz et al., 2013) reported that in the intention-to-treat analysis, the restenosis rates for superficial femoral artery stenosis at 2-month follow-up were 9 of 30 (30%) in the PTA group and 4 of 30 (13%) in the cutting balloon angioplasty (CBA) group (p = 0.117), and the ABI values at 12 months between the PTA and CBA groups were 0.77 +/- 0.11 versus 0.82 +/- 0.12, respectively (p = 0.039). Poncyljusz (Poncyljusz et al., 2013) could not demonstrate the superiority of CBT compared to PTA alone. Although techniques and devices for endovascular treatment have been developed, the promising results published previously do not yet appear to be sufficient to confirm the superiority of BAP and associated procedures over bypass surgery.

### 2.3 Comparison of the Mid-term Results between Endovascular Treatment and Bypass Surgery

Al-Nouri reported that failed intervention for superficial femoral artery occlusive disease has an impact on the preservation of ischemic limbs (Al-Nouri, Krezalek et al., 2012). Therefore, treating superficial femoral arterial lesions is important in salvaging critically ischemic legs. Among the previously reported comparative studies for BAP, the BASIL study only revealed proof of superiority or even efficacy of BAP over bypass surgery for infra-inguinal vascular disease (Bradbury, Adam et al., 2010; Moxey, Brownrigg et al., 2012). According to a randomized study comparing bypass surgery and endovascular treatment for superficial femoral arterial lesions, BAP appears superior to bypass surgery with artificial grafts in terms of patency of the superficial femoral vascular lesions (Bradbury, Adam et al., 2010). Meanwhile, our retrospective study comparing PTA and saphenous vein bypass surgery (Figures 1, 2; Tables 1, 2, unpublished data) revealed that the results for endovascular treatment of femoropopliteal TASCII C/D lesions showed that the 1-year patency rates were between 65 and 77% in the PTA and 73 and 75% in the PTA plus stent deployment groups, compared to bypass surgery.

There was no statistically significant difference in the survival (PTA: 79.3% at 2 years, bypass: 72.6% at 2 years, P=0.94) and patency (PTA: 70%, bypass: 78.1%, P=0.29) rates between PTA and bypass surgery in patients with TASCII C/D femoropopliteal lesions. In addition, there was a statistically significant difference in the limb salvage rates between endovascular treatment and bypass surgery in patients with TASCII C/D femoropopliteal lesions (PTA: 76.9%, bypass: 96.8%, P=0.039). There was no statistically significant difference in the run off scores (ROSs) between the limb salvage and non-limb salvage subgroups (limb salvage: median 7.0, non-salvage: median 3.75, P=0.44) in the endovascular treatment group (Table 2).
Figure 1: Comparison of the patency rates between the PTA and bypass surgery groups

Figure 2: Comparison of the survival rates between the PTA and bypass surgery groups
### Table 1: Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>PTA</th>
<th>Bypass</th>
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<tbody>
<tr>
<td>Patients</td>
<td>57</td>
<td>26</td>
<td>31</td>
</tr>
<tr>
<td>Age</td>
<td>70.3</td>
<td>74.1</td>
<td>67.1</td>
</tr>
<tr>
<td>Male</td>
<td>45</td>
<td>18</td>
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</tr>
<tr>
<td>Female</td>
<td>12</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Fontaine</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>8</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>III</td>
<td>18</td>
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<tr>
<td>IV</td>
<td>31</td>
<td>15</td>
<td>16</td>
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<tr>
<td>TASC</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>C</td>
<td>12</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>D</td>
<td>44</td>
<td>13</td>
<td>31</td>
</tr>
<tr>
<td>ROS</td>
<td>Average</td>
<td>4.5</td>
<td>4.02</td>
</tr>
<tr>
<td></td>
<td>Median</td>
<td>4</td>
<td>4</td>
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<tr>
<td>Technical Success rate</td>
<td>25 (96.1%)</td>
<td>31 (100%)</td>
<td></td>
</tr>
<tr>
<td>Average follow-up periods (month)</td>
<td>13.4 (0.7 - 45.9)</td>
<td>14.7 (1.2 - 47.1)</td>
<td></td>
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</table>

### Table 2: Results of Limb Salvage Rates

<table>
<thead>
<tr>
<th></th>
<th>Limb salvage rate</th>
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<tbody>
<tr>
<td>Non Limb Salvage cases</td>
<td></td>
</tr>
<tr>
<td>PTA group</td>
<td>6/26 (limb salvage rate: 76.9%)</td>
</tr>
<tr>
<td>Bypass group</td>
<td>1/31 (limb salvage rate: 96.8%)</td>
</tr>
<tr>
<td>p</td>
<td>0.039</td>
</tr>
<tr>
<td>ROS</td>
<td></td>
</tr>
<tr>
<td>PTA group</td>
<td></td>
</tr>
<tr>
<td>Limb salvage cases</td>
<td>average value: 6.91 (3 - 10)</td>
</tr>
<tr>
<td></td>
<td>median value: 7.0</td>
</tr>
<tr>
<td>Non limb salvage cases</td>
<td>average value: 3.59 (1 - 7)</td>
</tr>
<tr>
<td></td>
<td>median value: 3.75</td>
</tr>
</tbody>
</table>

### 2.4 Treatment of Peripheral Arterial Lesions below the Knee

For peripheral arterial lesions below the knee, the efficacy of endovascular treatment is more controversial than for infrainguinal arterial lesions. However, a recent study (Iida, Soga et al., 2013) of 884 patients with infrapopliteal arterial lesions, showed that with balloon angioplasty, freedom from major adverse limb events (MALE)+perioperative death (POD) was 82 +/- 1% and 74 +/- 2% at 1 and 5 years, respectively. The risk factors associated with MALE+POD were age >=80 years (adjusted hazard ratio [HR], 0.4; P < .001), non-ambulatory status (HR, 2.0; P < .001), albumin <3.0 g/dL (HR, 1.4; P < .0001), Rutherford 6 (HR, 2.2; P < .001), C-reactive protein >=3.0 mg/dL (HR, 2.1; P < .001), and below-the-ankle disease (HR, 2.0; P < .001) (Iida, Soga et al., 2013). The 1- and 5-year amputation-free
survival rates were 71 +/- 2% and 38 +/- 3%, respectively (Iida, Soga et al., 2013). These data could support the usefulness of endovascular treatment for infrapopliteal arterial lesions. The cutting balloon catheter (Canaud, Alric et al. 2008) and subintimal arterial flossing with antegrade-retrograde intervention (SAFARI)(Zhuang, Tan et al., 2011; Spinosa, Harthun et al., 2005; Gandini, Pipitone et al., 2007) have the potential to improve the patency rate compared to simple balloon angioplasty.

3 Vein

3.1 Iliofemoral Veno-occlusive Disease: Definition

Iliofemoral veno-occlusive disease (IVD) is manifested by varicose veins, venous ulceration, edema, venous eczema, and lipodermatosclerosis. The main cause of the above manifestations is venous hypertension as a result of venous stasis with secondary inflammation. In an experimental study using venous hypertension caused by arteriovenous fistulae, Takase et al. found that the venous valve leaflets were occupied with granulocytes, monocytes, T-lymphocytes, and endothelial cells expressing high levels of P-secretin and intercellular adhesion molecule 1 (ICAM-1) (Takase, Pascarella et al., 2004). These data suggest that venous hypertension and the sequential degeneration of the venous valves generate IVD through apoptosis of the venous valves (Takase, Pascarella et al., 2004).

3.2 Mechanism of Venous Valve Movement (Figure 3)

Venous valves are moved by venous pressure rather than blood flow, so complete closure of the venous valves is required to prevent valve degeneration (Qui, Quijano et al., 1995; Lurie, Kistner et al., 2003). Venous flow is usually pulsatile, and 20 cycles of valve movement per minute occur during standing (Bergan, Schmid-Schonbein et al., 2006). In addition, the venous leaflets do not touch the vascular sinus wall when the venous valves are completely open (Bergan, Schmid-Schonbein et al., 2006). This characteristic is responsible for preventing venous sclerosis through shear stress and the cytokine cascade. Considering the dynamic movement of the venous valves, Lurie et al. (Lurie., et al., 2003) reported that the valve cusps move in four phases constituting the valve cycle. The vortical stream behind the valve cusps plays a role in valve function and prevents stasis inside the valve pocket (Lurie, Kistner et al., 2003). Thus, the central jet may facilitate outflow (Lurie, Kistner et al., 2003).

![Figure 3: Mechanism of venous valve movement. Venous flow: Normally pulsatile; Venous valves open and close with movement; 20 times per minute; When the valve leaflets are fully open, they do not touch the sinus wall. All surfaces of the valve are exposed to shear stress.](image-url)
3.3 Shear Stress (Figure 4 and 5)

Shear stress is generated and mediated in endothelial cells by a network of signaling pathways that can promote gene expression. A lack of shear stress on the venous sinus leaflets generates inflammation and thrombotic conditions. A strong correlation exists between endothelial cell dysfunction and areas of low mean shear stress and oscillatory flow with flow reversal (Traub and Berk, 1998). Disturbed flow and the associated low-shear stress generally upregulate endothelial cell genes and proteins that promote atherogenesis (Chiu and Chien, 2011). In the venous system, disturbed flow resulting from reflux, outflow obstruction, and/or stasis leads to venous inflammation and thrombosis and hence the development of CVD (Chiu and Chien, 2011).

**Figure 4:** Normal Shear Stress. Under shear stress due to stable venous flow, there is a release of factors from endothelial cells that inhibit coagulation, leukocyte migration, and smooth muscle proliferation.

**Figure 5:** Shear Stress and Static Flow. Meanwhile, under opposite shear stress, some factors promote the growth of smooth muscle and the activation of leukocyte factors.
3.4 Classification of Venous Disease

Venous disease can be classified by the CEAP. CEAP stands for Clinical severity, Etiology, Anatomy, and Pathophysiology. Clinical signs in the affected legs are categorized into seven classes, designated C0 to C6 (Table 3). Leg symptoms associated with CVD include aching, heaviness, swelling, and skin irritation. Limbs categorized in any clinical class may be symptomatic (S) or asymptomatic (A). CVD encompasses the full spectrum of signs and symptoms associated with classes C0 to C6, whereas the term “chronic venous insufficiency” is generally restricted to disease of greater severity (i.e., classes C4-C6). Thus, varicose veins in the absence of skin changes are not indicative of chronic venous insufficiency (Porter and Moneta, 1995; Eklof, Rutherford et al., 2004).

<table>
<thead>
<tr>
<th>C</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>C0</td>
<td>No visible or palpable signs of venous disease.</td>
</tr>
<tr>
<td>C1</td>
<td>Telangiectasies of reticular veins</td>
</tr>
<tr>
<td>C2</td>
<td>Varicose veins: distinguished from reticular veins by a diameter of 3 mm or more</td>
</tr>
<tr>
<td>C3</td>
<td>Edema</td>
</tr>
<tr>
<td>C4</td>
<td>Changes in skin and subcutaneous tissue secondary to CVD, now divided into 2 subcategories to better define the differing severity of venous disease:</td>
</tr>
<tr>
<td>C4a</td>
<td>Pigmentation or eczema</td>
</tr>
<tr>
<td>C4b</td>
<td>Lipodermatosclerosis or atrophie blanche</td>
</tr>
<tr>
<td>C5</td>
<td>Healed venous ulcer</td>
</tr>
<tr>
<td>C6</td>
<td>Active venous ulcer</td>
</tr>
</tbody>
</table>

Table 3: Refinement of C classes in CEAP. CEAP classes C5 and C6 has been likened to the impairment associated with heart failure.

3.5 Current Topics in Venous Ulceration Associated with Chronic Venous Disease

Among the complications associated with chronic venous disease, a venous ulcer on the lower extremity is a major problem due to difficulty in managing infections, inflammation, and pain (Mosti, 2013). As for familial heredity, Serra et al. (Serra et al., 2012) performed a genetic study in patients with chronic venous insufficiency and verified that varicose veins (saphenofemoral junction reflux) were linked to the candidate marker D16S520 on chromosome 16q24, which may account for the linkage to FOXC2. Previous studies have reported that matrix metalloproteinases (MMP) and neutrophil gelatinase-associated lipocalin (NGAL) might have a role in the healing process in patients with chronic venous ulcers (Rayment et al., 2008; Pukstad et al., 2010). In a biochemistry study for venous ulcer patients, Serra et al. (Serra, et al., 2013) reported that enzyme-linked immunosorbent assay (ELISA) tests revealed significantly higher levels of MMP-9 and NGAL in the plasma and wounds from patients with ulcers compared to patients without ulcers (p < 0.01). In addition, Serra reported that Western blot analysis demonstrated increased expression of MMP-9 and NGAL from biopsies from patients with ulcers compared to patients without ulcers (p < 0.01). Therefore, Serra speculated that MMP-9 and NGAL have a role as biomarkers for healing in patients with a venous ulcer (Serra et al., 2013). In addition, Serra reported that subantimicrobial doses of doxycycline seem to generate chronic venous leg ulcer healing through the inhibition of MMP-9, NGAL and VEGF (Serra et al., 2013).

Hyperhomocysteinemia (HHc) is a factor associated with cardiovascular and cerebrovascular disease (Vollset et al., 2001; De Bree et al., 2002; Boysen et al., 2003). Sam et al. (Sam et al., 2003) de-
scribed the relationship between chronic venous ulceration and HHc as a thrombophilic disorder. Additionally, de Franciscis et al. (de Franciscis et al., 2013) found that homocysteine-lowering therapy with folic acid improved wound healing in patients with chronic venous leg ulcers. They also reported that low molecular weight heparin has a role in chronic venous ulcer healing, possibly due to the antithrombotic and anticoagulant effects (Serra et al., 2013; Serra et al., 2011).

### 3.6 Endovascular Treatment for Chronic Venous Occlusive Disease

Standard anticoagulation decreases the risk of pulmonary embolism and thrombus propagation, but does not treat the occlusion itself. Up to half of all patients with an iliofemoral DVT treated by anticoagulation alone subsequently develops post-thrombotic syndrome (PTS) (Kahn et al., 2008; Baldwin et al., 2013). PTS is associated with significant morbidity (leg swelling, pain, and ulceration), resulting in poor quality of life and lifelong socioeconomic implications (Kahn et al., 2008; Baldwin et al., 2013).

Endovascular treatment with catheter-directed thrombolysis is a good treatment option for not only restoring the occluded vein but preventing the occurrence of PTS. A Cochrane review suggested a significant reduction in the rate of PTS from 65 to 48 percent with any thrombolysis (Watson & Armon, 2004), and a more recent systematic review reported that systemic thrombolysis reduced PTS from 57 percent to 27 percent with CDT (Alesh et al., 2007).

Stent deployment for a residual stenotic lesion after catheter-directed fibrinolytic therapy is controversial (Park et al., 2013). May-Turner syndrome is characterized by left common iliac vein stenosis due to extrinsic compression by the left common iliac artery. After catheter-directed thrombolysis is performed, residual stenosis in May-Turner syndrome is a good indication for stent deployment to prevent the recurrence of deep vein thrombosis (Park et al., 2013). Warner et al. reported that functional outcomes, including pain relief, edema, and return to work, can improve after CDI and stent deployment in patients with an iliofemoral deep vein thrombosis (Warner et al., 2013). However, for DVTs located below the inguinalis, CDT and stent deployment cannot always improve the long-term patency and prevent the occurrence of PTS (Jackson et al., 2005).

Therefore, the indications for endovascular treatment for patients with DVT in our institute are as follows: DVT due benign or malignant disease; recent (within 2 weeks) occurrence of symptoms related to a DVT; no contraindication to thrombolysis, such as brain lesions, previous cerebrovascular attack, and recent surgical procedure; stent deployment for residual stenosis of the common/iliac vein after CDT; stent deployment for lesions below the inguinal level.

In the prone position, venographies via a popliteal vein catheter revealed thrombosis from the left popliteal vein to the external iliac vein and various collateral pathways (a-c). We performed fibrinolytic therapy using urokinase (120,000 units/day via an indwelling catheter) and anticoagulation therapy (10,000 units via the dorsal pedal vein).

The day after treatment, restoration of the vein was observed, but stenosis of the common femoral vein was seen (arrow in d). Balloon dilation was attempted, but residual stenosis was still seen (arrow in f). Five days after treatment, venography showed thrombotic occlusion of the left superficial vein (g and h) and residual stenosis of the left common iliac vein (arrow in i). Residual stenosis of the common femoral vein could have driven the relapse of thrombotic occlusion of the superficial vein. On clinical follow-up, this patient was taking warfarin to prevent the development of thrombosis. No leg swelling was noted after the procedure.
Figure 6: 72-year-old man with a left leg DVT (1): CEAP C3
Figure 7: 64-year-old woman with a DVT due to inguinal lymph node removal for melanoma (1): CEAP C3. Contrast-enhanced CT images showed thrombotic occlusion of the left lower leg from the left common iliac vein above the popliteal vein (arrows in all of the images). Venography in the supine position showed flow in the superficial vein (d), but severe stenosis was observed in the common iliac vein (e). We tried to perform fibrinolytic and anticoagulant therapies, but restoration of the iliac vein could not be achieved (f, g). Balloon dilation and stent deployment were not effective, resulting in the recurrence of thrombotic occlusion of the left iliac vein (h). On clinical follow-up, this patient was taking warfarin to prevent the further development of thrombosis. No leg swelling was noted after the procedure until the patient eventually died from melanoma.
Figure 8: 66-year-old man with metastatic lymph nodes from colon cancer that compressed the right external iliac vein: CEAP C4a. CT (a) showed a bulky metastatic lymph node compressing the right external iliac vein (arrow in a). Venography in the prone position showed thrombotic occlusion of the right common iliac and superficial veins. An indwelling catheter for metastatic liver disease was also seen (yellow arrows in the b and c images). Nine days after fibrinolytic and anticoagulant treatment, restoration of the occluded veins was achieved (d). Stent deployment at the stenotic site was performed to prevent the recurrence of a thrombotic occlusion of the iliac vein (e).
Figure 9: 58-year-old man with intractable right sciatic pain. On the MRI images, the left common iliac vein was compressed by the right common iliac artery (arrow in a and b), and the sacral nerve root (white arrow in b) was compressed by a varicose vein (yellow arrow in b). Varicose formation was driven by compression of the left common iliac vein. The intractable right sciatic pain may have been induced by direct compression of the sciatic nerve by the varicose vein (Hu and Wu et al., 2010).

Figure 10: 70-year-old woman with an arteriovenous fistula and May-Turner syndrome: CEAP C4a. (a) Aortography showed fine vessel proliferation around the left iliac artery and early visualization of the left iliac veins. (b)Venography showed an occlusion of the left common iliac vein. (c) Left common iliac arteriography showed no fine vessel proliferation or early venous drainage. However, deep vein thrombosis occurred after arterial embolization. Therefore, we speculate that venous stasis after shunt occlusion led to DVT formation.
Figure 11: 77-year-old woman with an arteriovenous fistula and with DVT (1): CEAP C5. (a) Left common iliac arteriography showed fine vessel proliferation around the left inguinal region and early visualization of the left common iliac vein. Occlusion of the left common iliac vein was also observed. (b) Venography via the left popliteal vein showed an occlusion of the left common iliac vein. (c) After treatment, venography showed recanalization of the left common iliac vein.

The photos before (left) and after (right) treatment for DVT show that the swollen left lower leg shrank significantly. Skin pigmentation was noted after treatment (right).
Figure 12: 88-year-old woman with an arteriovenous fistula and DVT (1): CEAP C6. Aortography showed fine vessel proliferation around the left internal iliac artery and early visualization of the left iliac vein. (b) Venography showed occlusion of the left common iliac vein. (c) After balloon PTA and stent deployment, venography showed recanalization of the left common iliac vein.

Figure 13: The photos before (left) and after (right) treatment for DVT show that the swollen left lower leg shrank significantly. Skin pigmentation and a healing skin ulcer were noted after treatment (right).

4 Summary

Vascular approach to lower legs is now challenging and developing treatment option for restoring both the occluded arterial or venous peripheral disease. On peripheral arteriosclerosis disease, endovascular therapy including PTA(percutaneous transluminal angioplasty) and stent deployment seem to be effective at the above knee. However, endovascular treatment for below the knee is not sufficient to reach the outcome of long term patency results of bypass surgery. Meanwhile, on peripheral venous disease including deep vein thrombosis, anticoagulant and conservative therapies have a major role of treating venous dis-
ease. On view of preventing post thrombotic syndrome, catheter directed therapy seem to be effective to restore the occluded venous disease despite high level evidence of revealing the effectiveness of catheter directed therapy has not been verified.

Acknowledgement

Special thanks to Yukiko Baba for illustrating the figures.

References


