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Case Report

Detrimental Effects of Endovascular Intervention in Active Rheumatoid Vasculitis

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Balloon angioplasty can cause shear stress and tear of the vascular endothelium during mechanical dilatation, leading to increased inflammation and coagulation reactions of the vascular endothelium. Herein, a worst case of active rheumatoid vasculitis is described, where due to progressing ischemic necrosis of the leg, endovascular intervention was unavoidably performed in the presence of active rheumatoid vasculitis. After percutaneous balloon angioplasty, the patient developed recurrent thrombotic occlusion of the leg arteries, and finally, limb amputation resulted in despite vigorous treatment including medication, immunosuppression, catheter-directed thrombolysis, and post-thrombolysis anticoagulation. This case report indicates that endovascular intervention may be detrimental to the active rheumatoid vasculitis. Until the development of treatment guideline to prevent or control inflammatory reaction, endovascular intervention for the active rheumatoid vasculitis may not be appropriate as a first line therapy even though there is progressing ischemic necrosis.

Key Words: Endovascular intervention, Rheumatoid vasculitis, Thrombosis

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INTRODUCTION

Endovascular intervention has been widely performed for the treatment of arterial insufficiency as it is minimally invasive, requires less time compared to open surgery, and is associated with less morbidity. However, mechanical irritation of the arterial wall due to balloon dilatation induces shear stress and barotraumatic injury. This injury causes an inflammatory reaction in the endothelium and triggers coagulation reactions in the arterial lumen [1]. Rheumatoid vasculitis is a type of autoimmune vasculitis and is associated with a high mortality, making it one of the most serious extra-articular manifestations of rheumatoid arthritis [2]. One single-center study of 86 patients with rheumatoid vasculitis reported a 26% mortality rate over 5 years [3]. The vascular wall in cases of rheumatoid vasculitis is already in an inflamed state. Therefore, balloon dilatation of the arteries in patients with rheumatoid vasculitis carries a risk of aggravating inflammation and causing thrombosis. Currently, there are no guidelines for the endovascular intervention of stenotic or occlusive arteries caused by rheumatoid vasculitis. In this case study of the worst result, the detrimental effect of endovascular intervention in a 61-year-old woman with rheumatoid vasculitis is described and discussed.

CASE

Reporting of this case study was approved by the institutional review board, and informed consent was obtained from the patient. A 61-year-old woman with an ulcer on the left leg was transferred to our facility from another clinic. She was diagnosed with rheumatoid arthritis 2 years prior. She presented with a rheumatoid factor titer of >400 IU/

mL, C-reactive protein (CRP) level of 11.53 mg/dL, and involvement of both hands and feet affecting 9 joints in all. Her score on the 2010 American College of Rheumatology-European League Against Rheumatism classification criteria for rheumatoid arthritis was 8, which met the diagnostic criteria for rheumatoid arthritis [4]. She also presented with a deep cutaneous ulcer; foot gangrene with an amputated second toe; peripheral neuropathy, as evidenced by a nerve conduction study; foot drop; and nail fold infarcts on both toes and fingers (Fig. 1). Following a skin biopsy (Fig. 2) she was diagnosed with systemic rheumatoid vasculitis based on Scott and Bacon [5] criteria. Her ankle brachial indices (ABI) were 1.23 and 1.15 on the right and left sides, respectively. Her toe brachial indices (TBI) were 0.57 and 0.10 on either side. A computed tomography (CT) angiogram showed occlusion of the left dorsalis pedis artery (Fig. 3). In Inje University Haeundae Paik Hospital, after consulting with a rheumatologist, the patient administered 100 mg aspirin and 75 mg clopidogrel daily, 10 mg methotrexate weekly. After 3 days of intravenous corticosteroid (solumedrol 50 mg), she was administered oral glucocorticoid (deflazacort 12 mg) daily. However, there was deteriorating ischemic change in the left foot indicating refractoriness to medical treatment. Thus, rituximab 500 mg was administered intravenously. One week after the rituximab administration, endovascular intervention was performed due to progressing ischemic necrosis on her left foot. During the procedure, a 5-Fr sheath was inserted into the left common femoral artery in the antegrade direction, and the dorsalis pedis and posterior tibial arteries at the left ankle and foot were dilated with a 3-mm diameter balloon over a 0.035-inch guidewire (Fig. 4A, C). During the endovascular intervention, calf arteries were not treated, only the foot and ankle arteries were treated. After the procedure, completion angiography showed patent left dorsalis pedis and posterior tibial arteries at the left foot, and patent left popliteal, peroneal, anterior and posterior tibial arteries at the left calf (Fig. 4B, D, E). There was no intimal dissection or thrombotic occlusion in the treated arteries. One day after the intervention, the left TBI score increased from 0.10 to 0.52, and pain in her left foot decreased. The circulatory status of the leg was monitored by a daily assessment of ABI and TBI, and all post-intervention ABI and TBI scores of the left leg were >1.00 and >0.40, respectively, at every measurement. However, twelve days after the intervention, the patient complained of severe pain in her left leg, and the left ABI and TBI could not be measured. Angiography indicated total thrombotic occlusion of the anterior and posterior tibial arteries and peroneal artery at the left calf; then, catheter-directed thrombolysis was performed (Fig. 5A). A bolus of urokinase (230,000 IU or 4,400 IU/kg) was

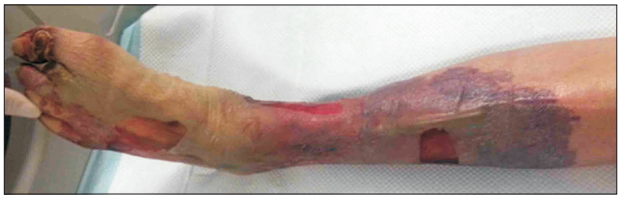

Fig. 1. Pre-intervention image of the left leg. There is a deep cutaneous ulcer, amputated second toe, foot drop, and nail fold infarcts on the toes.

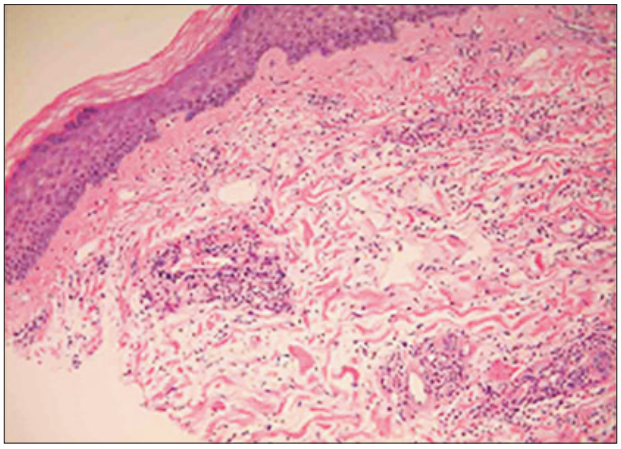

Fig. 2. Skin biopsy at the left calf biopsy revealed neutrophilic dermatitis with dermal neutrophilic infiltration with karyorrhexis and leukocytoclastic vasculitis involving small and medium-sized dermal vessels. The findings are consistent with rheumatoid neutrophilic dermatitis and vasculitis (H&E stain, ×200).

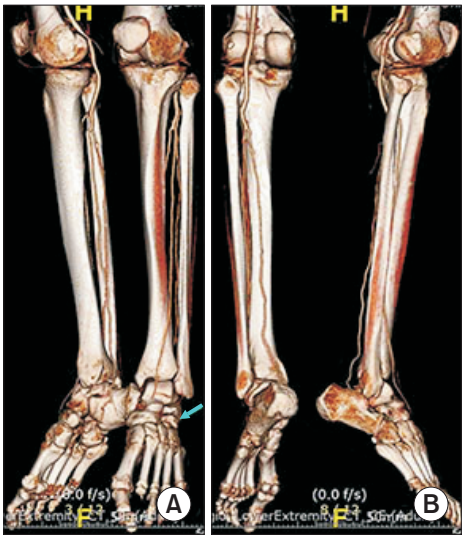

Fig. 3. Computed tomography angiographic findings; (A) anterior view (arrow), (B) posterior view. Occlusion of the left dorsalis pedis artery is visible.

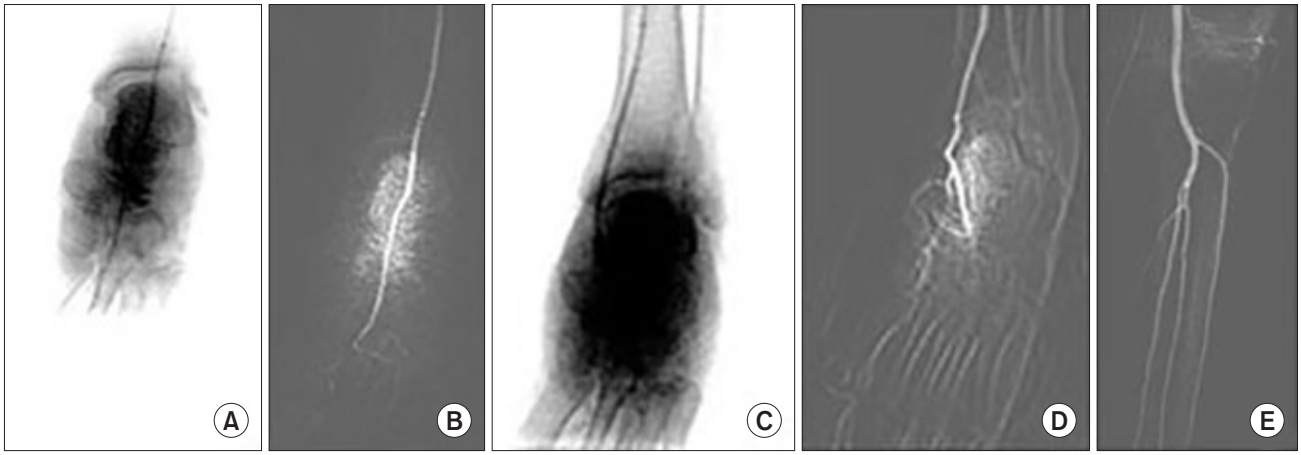

Fig. 4. Endovascular intervention of the left foot. There was no intimal dissection or thrombotic occlusion in the treated arteries at the left foot and ankle. (A) Balloon angioplasty of the left dorsalis pedis artery. (B) Patent left dorsalis pedis artery after balloon angioplasty. (C) Balloon angioplasty of the left posterior tibial artery at the left ankle and foot. (D) Patent left posterior tibial artery after balloon angioplasty at the left ankle and foot. (E) Patent left popliteal, peroneal, anterior and posterior tibial arteries at the left calf.

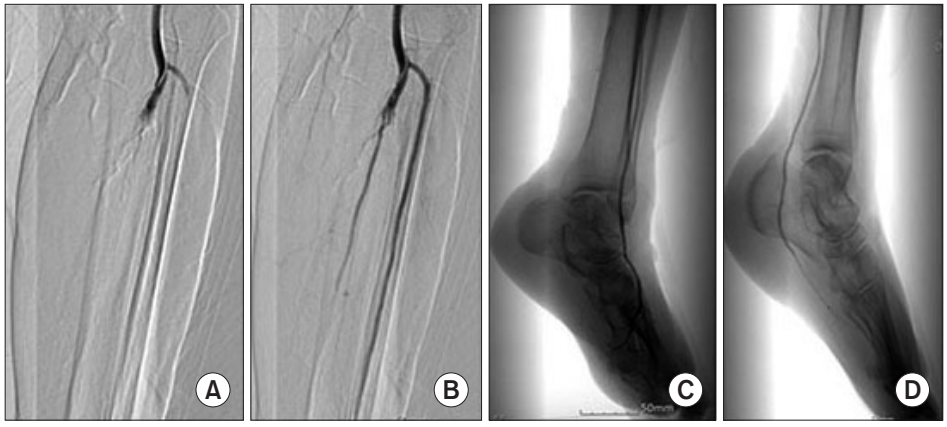

Fig. 5. Thrombolysis of the left calf arteries and catheter-directed thrombolysis. (A) Occlusion of the anterior and posterior tibial arteries and peroneal artery at the left calf is noted. Catheter is placed at the tibioperoneal trunk. (B) After thrombolysis with urokinase, recanalization of the anterior and posterior tibial arteries is seen. (C, D) Angiography using catheter in the anterior tibial and posterior tibial arteries shows patent dorsalis pedis and posterior tibial arteries at the left foot, respectively.

injected in the proximal portion of the tibioperoneal trunk for 10 minutes via a catheter. Following this procedure, a continuous infusion of urokinase (230,000 IU/h or 4,400 IU/kg/h) was performed for 10 hours. Subsequent to the thrombolysis procedure, recanalization was observed in the anterior and posterior tibial arteries at the left calf and dorsalis pedis and posterior tibial arteries at the left foot (Fig. 5B-D). ABI and TBI scores of the left leg were 0.86 and 0.31, respectively. After catheter-directed thrombolysis, low-molecular-weight heparin (dalteparin sodium, 5,000 IU subcutaneous injection daily) was added to the existing medication (methotrexate, aspirin, clopidogrel and

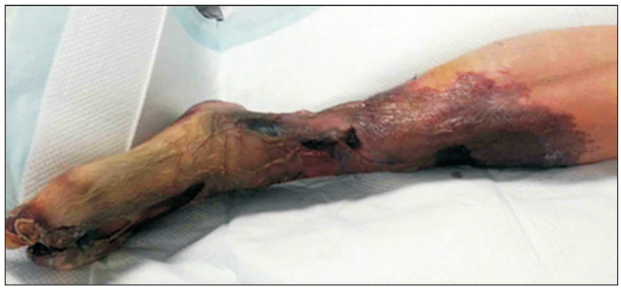

Fig. 6. Progression of gangrene of the left leg after thrombolysis. Gangrene progressed and ultimately resulted in amputation.

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deflazacort). However, the gangrene continued to progress (Fig. 6), and ABI and TBI scores decreased. Ischemic necrosis of the whole left foot and mid-calf developed, and an above-knee amputation was performed eight days after the thrombolysis.

DISCUSSION

Rheumatoid vasculitis is a systemic autoimmune vasculitis that mainly affects small to medium-sized blood vessels [2]. Autoimmune vasculitis, such as rheumatoid vasculitis, can cause chronic wounds that do not respond to vascular intervention and standard local wound care [6]. In this case, the skin biopsy finding, rheumatoid dermatitis and vasculitis, suggests that the cutaneous manifestation of rheumatoid arthritis may also contribute to the worsening condition of the patient's left leg. However, it has been reported that purpura, livedo reticularis, atrophie blanche and skin ulcer are the typical manifestations of rheumatoid dermatitis, but ischemic necrosis of the underlying soft tissue is not a cutaneous manifestation [7]. Therefore, it is speculated that, before the endovascular intervention, the progression of ischemic necrosis of the left leg is mainly due to arterial insufficiency. Rheumatoid vasculitis has high morbidity and mortality, making it one of the most serious extra-articular manifestations of rheumatoid arthritis [2]. Advances have been made in the treatment of rheumatoid arthritis using various disease-modifying anti-rheumatic drugs, biological agents, monoclonal antibodies, and corticosteroids [8]. Until now, there is no consensus on the indications, timing, and outcomes of revascularization for vasculitis, and treatment is often guided by empiricism [2,9]. However, it has been reported that in patients with an autoimmune vasculitis, Takayasu arteritis, active state of the disease is a risk factor for restenosis after revascularization procedure, and both bypass surgery and endovascular procedures are associated with high failure rates and frequent operative complications [10]. In this case, the CRP level of the patient was 11.53 mg/dL indicating active inflammatory status of the rheumatoid vasculitis. However, because the ischemic necrosis of the left leg deteriorated progressively and the patient presented refractoriness to pharmacologic treatment, the less invasive treatment, endovascular intervention, was unavoidably performed in the presence of active rheumatoid vasculitis in this patient. Several efforts to protect the arteries from thrombotic complications after endovascular intervention have been previously reported. One prospective study showed that, following successful intervention of the femoral and popliteal arteries, adventitial infusion of dexamethasone to the treated segment improved clinical outcomes [11]. In a large retrospective study with 57,041 patients, comparing mono-antiplatelet therapy with aspirin versus dual antiplatelet therapy with aspirin plus thienopyridine antiplatelet agent showed that dual antiplatelet therapy was associated with prolonged survival in patients with critical limb ischemia undergoing lower extremity revascularization, including both bypass surgery and endovascular intervention. However, in the current case, despite vigorous treatment with aspirin, clopidogrel, immunosuppressive agents, such as methotrexate, solumedrol, deflazacort and rituximab, and post-thrombolysis low molecular weight heparin, the patient developed progressing thrombotic occlusion after endovascular intervention and eventually resulted in limb amputation.

This case report of the worst result indicates that endovascular intervention may be detrimental to the active rheumatoid vasculitis. The negative result of this case report may be helpful for physicians in clinical decision making regarding endovascular intervention for inflammatory vascular diseases. Until the development of treatment guideline to prevent or control inflammatory reaction, endovascular intervention for the active rheumatoid vasculitis may not be appropriate as a first line therapy even though there is progressing ischemic necrosis.

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