

# Enlarged Ulcer-like Projection and Acute Arterial Embolism in the Lower Limbs of a Patient with Chronic Stanford Type B Aortic Dissection

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## ABSTRACT

We report a rare case of thromboembolism in the lower limbs in a 62-year-old male who presented with chest and back pain and was diagnosed with Stanford type B acute aortic dissection with a patent false lumen. Computed tomography revealed a thrombosed false lumen and ulcer-like projection, without expansion of the aortic dimension. He was discharged on the 12th day. However, on the 52nd day of illness, he presented with chest and back pain, elevated C-reactive protein and an enlarged ulcer-like projection on computed tomography. Seven days later, he experienced sudden left leg pain and sensory disturbance. Arteriography revealed right popliteal artery occlusion. Thrombus aspiration and percutaneous transluminal angioplasty were performed for recanalization. Aortic dissection with a false lumen was speculated as the embolization source and surgery was performed. His symptoms resolved after surgery?

**Key words:** Aortic dissection, Ulcer-like projection, Arterial embolism, Lower limbs.

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## INTRODUCTION

Acute aortic dissection requires prompt diagnosis and treatment since the mortality rates are extremely high. In Japan, which has the most rapidly aging population in the world, the incidence of acute aortic dissection as a typical arteriosclerotic vascular disease is assumed to increase in the future. In uncomplicated Stanford type B acute aortic dissection, medical therapy is generally recommended, which is classified as Class I.<sup>1</sup> Acute aortic dissection with a thrombosed false lumen has a better prognosis as compared with acute aortic dissection with a patent false lumen. However, it has been reported that an ulcer-like projection that occurs during the acute stage of aortic dissection may be a risk factor for expansion of the aortic dimension in the chronic phase. Invasive treatment should be considered for an ulcer-like projection that may enlarge and develop into an aneurysm, resulting in re-dissection and rupture.<sup>2</sup>

In some patients with aortic dissection, vascular endothelial disorder and severe inflammation-related activation of the coagulation system may occur at the false lumen in the aortic dissection, since the tunica media of the aortic wall is dissected. Coagulation and fibrinolytic systems are typically activated by the enlarged thrombosed false lumen and are complicated by disseminated intravascular coagulation in the chronic phase.<sup>3</sup> It is thought that coagulation disorder is particularly common at the ulcer-like projection and is associated with thrombosis and expansion of the aortic dimension during the chronic phase. The incidence of ischemia in the lower limbs as a complication of aortic dissections is 7-18%. This phenomenon is often complicated by extensive dissection, as in DeBakey type I; thromboembolism is rarely the cause.<sup>4</sup>

In the present case study, we report a patient who presented with an enlarged ulcer-like projection in the descending aorta and thromboembolism in the lower limbs during the chronic phase of Stanford type B aortic dissection.

## CASE REPORT

A 62-year-old male received treatment for hypertension (Diagnosed in his 40s) in a local clinic. At 6:00 a.m. on the hospitalization day, the patient suddenly experienced subjective chest and back pain that did not subside. He was transferred to our hospital by ambulance at 8:05 a.m. The results of physical examination at presentation were as follows: height, 165 cm; weight, 62.5 kg; body mass index, 23.9; consciousness, clear; blood pressure, 135/86 mmHg without laterality; pulse, 90 beats/min and regular; respiratory rate, 20 times/min; temperature, 36.8°C. No bruit was heard in the neck and no abnormalities were detected in the chest, abdomen and extremities.

His blood test results were as follows: white blood cell count, 8570/m<sup>2</sup>; C-reactive protein, 0.14 mg/dL; prothrombin time, 21.3 sec; activated partial thromboplastin time, 39.1 sec; fasting blood glucose, 433 mg/dl; antithrombin activity, 103%; fibrin degradation product, 5 µg/ml; D-dimer, 0.66 µg/ml.

Electrocardiography demonstrated sinus rhythm with a heart rate of 90 bpm, a normal axis and ST segment depression in leads I, aVL and V5-6. On portable plain chest radiography, an enlarged mediastinal shadow was observed. Echocardiography showed normal contractility of the left ventricle. Neither aortic incompetence nor pericardial fluid were detected. Contrast-enhanced computed tomography of the chest and abdomen revealed aortic dissection with thrombotic occlusion from the distal branch of the left subclavian artery to the 10<sup>th</sup> thoracic vertebral level. The greatest dimension of the vessel was 43 mm (Figure 1). The anterior spinal artery appeared from a true lumen.

The patient was hospitalized in our coronary care unit after being diagnosed with Stanford type B acute aortic dissection with a patent false lumen. Bed rest and antihypertensive therapy were initiated. Target systolic blood pressure was set as 120 mmHg or less. The level of bed rest subsequently increased. Computed tomography scans on the 5<sup>th</sup> day of illness demonstrated no expansion of the aortic dimension; however, a thrombosed false lumen and the development of an ulcer-like projec-

tion were observed (D-dimer, 1.26 µg/ml; C-reactive protein, 6.08 mg/dl; Figure 2). Systolic blood pressure was less than 120 mmHg, due to oral antihypertensive drugs. During electrocardiogram monitoring, atrial fibrillation was not found. The patient was discharged on the 12<sup>th</sup> day of illness (D-dimer, 2.75 µg/ml; C-reactive protein, 2.52 mg/dl). Contrast-enhanced computed tomography following discharge, on the 30<sup>th</sup> day of illness, showed no expansion of the aortic dimension, but increased thrombus formation in the false lumen and an enlarged ulcer-like projection were observed (D-dimer, 1.20 µg/ml; C-reactive protein, 1.24 mg/dl; Figure 3).

Computed tomography scanning was performed during regular follow-up; on the 50<sup>th</sup> day of illness, progression of thrombus formation at the false lumen and reduction of the ulcer-like projection were observed (D-dimer, 1.83 µg/ml; C-reactive protein, 0.62 mg/dl). On the 52<sup>nd</sup> day of illness, the patient presented to our hospital with subjective chest and back pain. There were no remarkable changes detected compared to the previous computed tomography scans and D-dimer levels (1.58 µg/ml). However, C-reactive protein level was increased to 5.92 mg/dl and the patient was hospitalized for follow-up. On the 58<sup>th</sup> day of illness, he suddenly experienced subjective left leg pain and sensory disturbance. Contrast-enhanced radiography of the lower limbs was immediately performed and right popliteal arterial occlusion was detected by arteriography (D-dimer, 2.25 µg/ml; Figure 4). We did not choose thrombolysis because this treatment dissolves the pseudoluminal thrombus of the aorta and may enlarge the dissection.

Thrombus aspiration and percutaneous transluminal angioplasty were performed and recanalization was successfully achieved. Urokinase (60,000 unit/day) was continuously administered due to persistent thrombus on imaging. Pathological findings of the aspirate showed red thrombus (Figure 5). Ischemic symptoms in the lower limbs subsequently resolved. The ulcer-like projection in the aorta was speculated as the source of embolization. Following surgery for aortic dissection, no embolization was detected. The patient's symptoms were resolved and he was discharged.

Consent was obtained from the patient regarding publishing clinical information and data was presented in a way that ensured individual anonymity.

## DISCUSSION

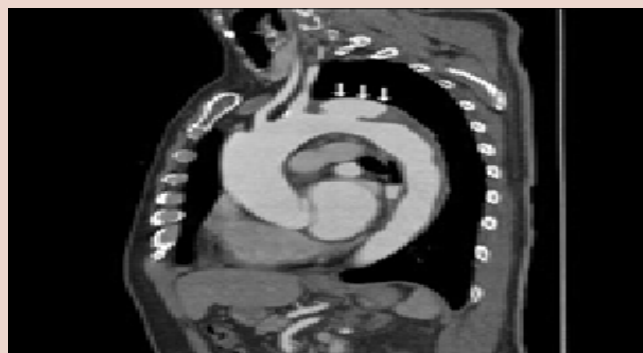
An ulcer-like projection, which protrudes into the obstructed false lumen on contrast media enhanced imaging, can be detected by imaging modalities such as angiography and contrast-enhanced CT. During dissection with a thrombosed false lumen, the false lumen disappears while communication occurs between the false lumen and the true lumen, leading to dissection with ulcer-like projection and dissection with a patent false lumen due to enlargement of the false lumen longitudinally, resulting in dissection with a patent false lumen.<sup>5</sup>



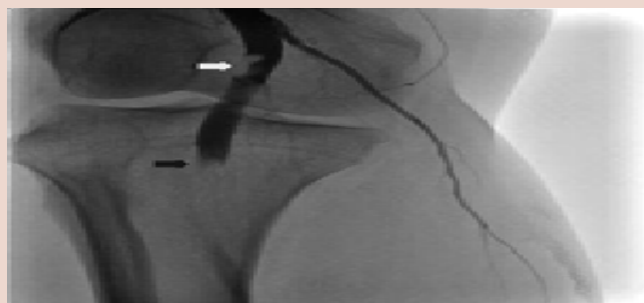
**Figure 1:** Dissection was performed from the Aortic Arch to the Descending Aorta, demonstrating that the False Lumen was occluded by Thrombi.



**Figure 2:** A thrombosed False Lumen and Onset of Ulcer-like Projection was observed (Arrow).



**Figure 3:** The Number of Thrombi in the False Lumen Increased and an Enlarged Ulcer-like Projection was Detected (Arrow).



**Figure 4:** Arteriography of the Lower Limb. Thrombus (White arrow) and Occlusion (Black arrow) of the Right Popliteal Artery were detected.



**Figure 5:** Histological Findings of Arterial Embolism in the Lower Limbs. Red Thrombus was detected, whereas Organized Thrombus was not.

In addition, it has been reported that ulcer-like projections that occur in the proximal descending aorta are a risk factor for subsequent aorta-related adverse events.<sup>6</sup> In the ulcer-like projection, thrombus in the false lumen may indicate an unstable status with regard to mobility, as the embolism in the false lumen is not sufficiently organized, leading to the risk of floating thrombus flowing into the vascular cavity and subsequent embolism.

A common cause of embolism in the lower limbs is myocardial infarction and atrial fibrillation. It has also been suggested that a thrombus attached to a plaque in the ascending aorta and aortic arch may be the source of embolization when thrombi in the aorta cause embolism.<sup>6</sup>

In the present patient, sinus rhythm was observed on electrocardiography without atrial fibrillation during hospitalization and echocardiography demonstrated normal contractility of the left ventricle and no enlargement of the left atrium. In addition, there were no plaques with apparent thrombi in the ascending aorta on computed tomography images. It was speculated that thrombi within the ulcer-like projection in the descending aorta may be the source of the embolus.

Few studies have focused on alterations in the coagulation system during aortic dissection in the chronic phase. However, according to a study by Nakajima *et al.* patients with residual dissection and a maximum dimension of 45 mm or more exhibited significantly increased levels of thromboglobulin, thrombin-antithrombin III, thrombin-antithrombin complex, D-dimer and 2plasmin inhibitor-plasmin complex in comparison with patients without residual dissection or patients with residual dissection and a maximum dimension of <45 mm.<sup>7</sup>

It was reported that there was no significant difference in the morphology of the false lumen when using platelets versus thromboglobulin as a thrombus formation marker.<sup>8</sup> Furthermore, as a coagulation marker, prothrombin fragment 1+2 demonstrated a significant correlation with the aortic dimension and thrombin-antithrombin complex and D-dimer were increased in patients with partial thrombosis of a patent false lumen, as compared with patients with a thrombosed false lumen.<sup>8</sup>

As these cases are rare, numerous questions remain. However, we hypothesize that the diameter of the vessel is large during the chronic phase of aortic dissection, which may lead to coagulation disorder in patients with partial thrombosis of a patent false lumen.

The C-reactive protein level was elevated, which is known to be an indicator of thrombus formation in the false lumen in patients with acute aortic dissection.<sup>9</sup> It has been reported that the course and degree of the peak value of C-reactive protein may be a risk factor for long-term prognosis (Mortality and aortic events) of hospitalized patients with type B aortic dissection.<sup>10</sup> It has also been reported that the peak value of C-reactive protein and the uptake of fluorodeoxy glucose at the site of dissection on positron emission tomography may be useful prognostic indicators.<sup>11</sup> Therefore, persistent inflammation of the dissected aortic wall may be associated with prognosis. In the present case, the greatest dimension of the site of aortic dissection was 43 mm, which did not subsequently expand, whereas a slight increase in C-reactive protein and D-dimer persisted in the chronic phase. Therefore, his enlarged ulcer-like projection and thromboembolism might have been caused by inflammation and a coagulation disorder.

Embolectomy is considered a treatment for arterial embolism in the lower limbs associated with acute aortic dissection and antithrombotic drug therapy is used for the prevention of recurrence. However, for patients in whom multiple relapses occur and patients who have difficulty using antithrombotic drugs, endovascular stent-graft placement and blood vessel prosthesis implantation are considered. In addition, the plaque that is

the source of thromboembolism may rupture, leading to a penetrating aortic ulcer, intramural hematoma or dissection. Type B aortic dissection is generally initially managed medically, with surgical intervention reserved for those who develop complications related to the dissection.<sup>1</sup> However, there is increasing interest in endovascular treatment of type B aortic dissection prophylactically before the onset of malperfusion, which has the potential to reduce the incidence of late complications.<sup>12</sup> In the present patient, surgical intervention was chosen, as endovascular treatment for complicated type B dissection was not adapted to the Japanese insurance at that time.

In conclusions, an ulcer-like projection complicating type B aortic dissection in the chronic phase may become a source of thromboembolism and rupture due to expansion of the aortic dimension and branch occlusion. When coagulation disorder increases during follow-up, it may be necessary to consider endovascular treatment.

## CONCLUSION

Aortic dissection with an ulcer-like projection in the chronic phase may cause thromboembolism.

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## CONFLICT OF INTEREST

The authors declare no conflict of interest.

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