



## Acute Type B Aortic Dissection in a Patient with Previous Endovascular Abdominal Aortic Aneurysm Repair

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Endovascular aortic repair (EVAR) was relatively safe, and became a widely performed procedure. If aortic dissection (AD) occurred in patient with previous EVAR, it could cause fatal complications like endograft collapse. Surgical treatment was limited in this situation for comorbidities and complex anatomies. Here we report a rare case of acute type B AD developed following trans-radial coronary intervention in a patient with previous EVAR of abdominal aortic aneurysm, which was treated with thoracic EVAR.

**Key Words:** Aorta, Aneurysm, Dissection, Endovascular procedures

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### INTRODUCTION

Since its first introduction by Parodi et al. in 1991 [1], endovascular aortic repair (EVAR) was widely performed, accounting nearly half of abdominal aortic aneurysm (AAA) repair [2]. Recently, the indication for EVAR and thoracic EVAR (TEVAR) was expanded into various disease entities, including acute or chronic aortic dissection (AD), abdominal and thoracic aortic aneurysm, traumatic blunt aortic injury, aortic intramural hematoma, and penetrating aortic ulcer [3]. Although safe, several complications could be developed after EVAR, such as access site complications, endoleak, endograft migration or collapse [4]. Among them, acute AD is one of rare complications after EVAR. Incidence of retrograde AD after TEVAR was approximately 2.4% [5]. Especially, there are very few reports about acute type B AD in post EVAR status [5-11]. Here we report a rare case of acute type B AD following transradial coronary intervention in patients with previous EVAR.

### CASE

A 73-year-old male patient presented with acute chest pain. It was a new-onset symptom which worsened one week ago in intensity and duration. Electrocardiography on admission was normal sinus rhythm without pathologic ST-T change.

In past medical history, he had suffered from emphysema for four years. EVAR was performed for the growing AAA (60 mm) by Endurant stentgraft (Medtronic, Santa Rosa, CA, USA) following percutaneous coronary intervention (PCI), with two drug-eluting stents (DESs) on distal left anterior descending artery (LAD) and proximal right coronary artery (RCA) in December 2014 with interval of one week.

Diagnostic coronary angiography showed that previous stents in distal LAD and proximal RCA were both patent but de novo lesion was newly observed at distal left circumflex artery (LCX). Ad hoc PCI with DES was done via right radial route without complications.

One hour after the PCI, the patient suddenly complained

of a severe back pain and blood pressure (BP) was increased over 180/100 mmHg. Immediate CT aortography revealed AD, Stanford type B, DeBakey type III, from distal to the origin of left subclavian artery to proximal end of previous aortic stentgraft (Fig. 1). We did not perform ABI test because the patients did not complained of any discomfort of both lower leg extremities and both iliac arteries were intact on CT.

We chose medical treatment rather than surgical or endovascular treatment because AD was uncomplicated type B dissection and significant organ malperfusion was not noticed. Clopidogrel was discontinued and tight BP & heart rate (HR) control with intravenous labetalol and nicardipine was started to maintain BP 100/60 mmHg and HR 60/min.

Three days later, he complained of worsening abdominal pain and urine output was decreased, suspicious of organ malperfusion. Follow-up CT aortography also showed intact superior mesenteric artery and both renal arteries. However slight hypoperfusion in liver right lobe, bowel and both kidneys was noticed due to decreased distal flow.

So we decided to perform TEVAR (Fig. 2). Because 4 vessel angiography showed intact both vertebral arteries with favorable collateral flows, a 24F VALIANT Thoracic Captivia 36x207 mm (Medtronic) was implanted covering left subclavian artery through femoral approach. A vascular plug 14 mm was implanted at left subclavian artery through 8F Vista brite IG catheter from transradial route. Final angiography revealed successful isolation of AD entry site, no visible aneurysmal sac, no endoleak, and patent left carotid artery.

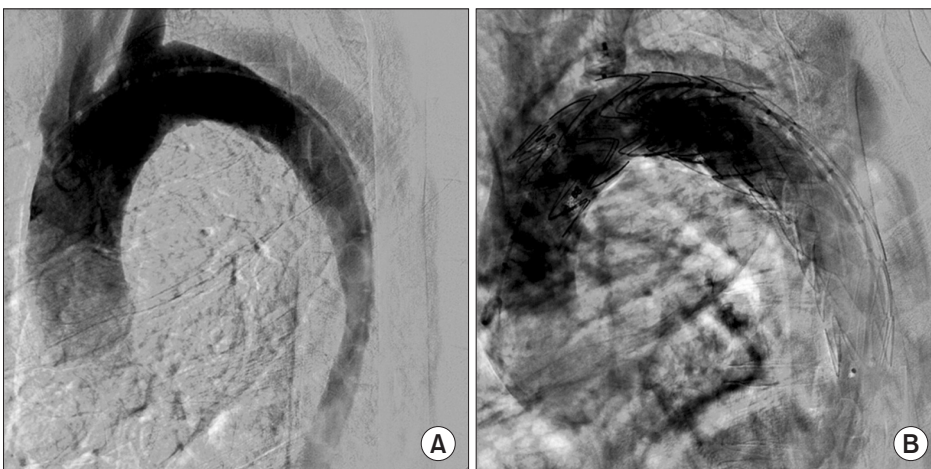
Clopidogrel was resumed to prevent coronary stent thrombosis. Abdominal pain and oliguria was improved. Surveillance CT aortography at 1 week showed well implanted stentgraft at aortic arch and descending aorta with slightly thrombosed false lumen (Fig. 3). The patient did not complained of any discomfort related with left subclavian artery such as arm claudication or weak radial pulse.

## DISCUSSION

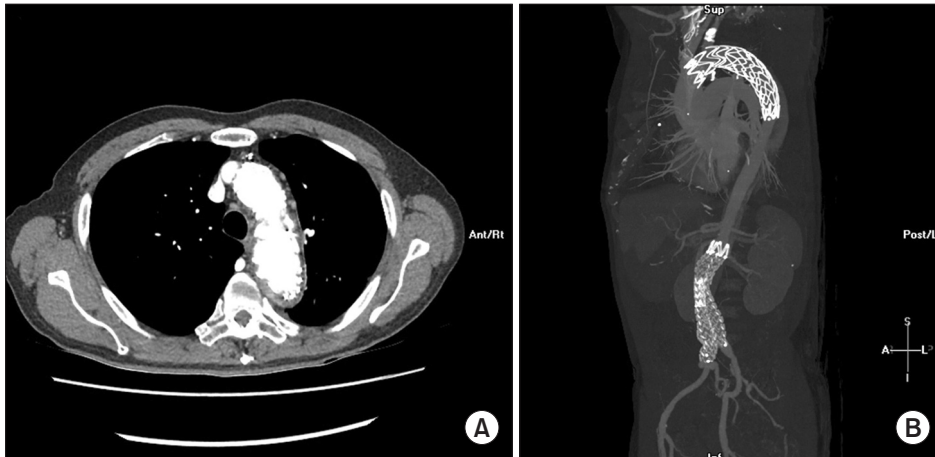
Several cases regarding acute type B AD in patients with



**Fig. 1.** Baseline CT aortography. (A) CT aortography axial image. Type B AD was originated just distal to left subclavian artery. (B) 3D reconstructive image. Type B AD was propagated into proximal edge of previous EVAR graft. CT, computed tomography; AD, aortic dissection; EVAR, endovascular aortic repair.



**Fig. 2.** TEVAR. (A) Aortography before TEVAR. True lumen was compromised distal to Lt subclavian artery. And false lumen was enhanced with contrast. (B) Aortography after TEVAR. True lumen was expanded and contrast enhancement of false lumen was almost disappeared. TEVAR, thoracic endovascular aortic repair.



**Fig. 3.** Surveillance CT aortography. (A) CT aortography axial image. Dissecting flap was disappeared. (B) CT aortography 3D-reconstruction image. TEVAR graft was well positioned. CT, computed tomography; TEVAR, thoracic endovascular aortic repair.

EVAR were reported previously. There was discrepancy in onset, treatment and prognosis. Girardi et al. [6] reported retrograde type B AD which was developed postoperative day 2 in 1999. It was treated by conservative medical therapy. However surgical repair for enlarging thoroabdominal aneurysm due to AD was performed four months later. Haulon et al. [7] reported acute type B AD five months after operation in 2003. In this case, an AD significantly compressed concave true lumen and the endograft was occluded apparently. The patient expired from acute aortic. Iyer et al. [5] reported acute type B AD 11 weeks postoperatively in 2009. Similar to our case, the site of the tear was right at the distal aspect of the origin of the left subclavian artery. However in this case, AD resulted in endograft collapse and occlusion. Reexpansion of a collapsed abdominal stentgraft was achieved by endografting of the proximal entry tear.

As described, some mechanisms for type B AD after EVAR were suggested by previous researchers. First of all, AD may be spontaneous without precipitating factors. Second, AD could occur due to technical aspect or device-related in early postoperative period [6]. Direct wire injury at thoracic aorta and device oversizing could precipitate AD. Also previous AD cases were mostly developed after EVAR with proximal bare stent with anchoring barbs. Third, AD may be related with pre-existing aortic defects like aortic calcification, penetrating ulcer and aortic ectasia. Biomechanical interaction with these defects and device

could result in type B AD [11].

Our case was developed just after right transradial PCI. During transradial PCI via right radial artery, the wires and catheters barely reach the aorta at left subclavian artery level. With thorough review, we suspected that guidewire or guiding catheter could injure the aorta during the retrieval because it was too soon after the PCI to be spontaneous. Aortic pathology such as severe calcification might also amplify the iatrogenic injury.

Even though, some cases were managed with medical therapy alone successfully in previous reports, type B AD could be devastating in patients with EVAR. The endovascular graft limited a reentry tear, resulting in the absence of an outflow for the false lumen. In consequence, the pressure in the false lumen exceeded the true lumen pressure, resulting in the compromised true lumen and fatal device collapse.

In conclusion, we report the acute type B AD after right transradial PCI in patient with previous EVAR graft. It was presumed to be initiated by wire or catheter injury and amplified due to underlying aortic pathology such as calcification. We performed a TEVAR to cover the proximal entry tear and the patient was successfully stabilized. We should be cautious during the procedure, especially in patients with aortic pathology. Acute type B AD with previous EVAR graft could be potentially fatal. Therefore close monitoring is mandatory with aggressive treatment when necessary.

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