Endovascular treatment of a type-1 aortic dissection after coronary artery bypass grafting

Koroner arter baypas greftleme sonrası tip-1 aort diseksiyonunun endovasküler tedavisi

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ABSTRACT

Acute dissection is the most dangerous aortic disease with a high mortality, if remained untreated. The standard treatment varies as surgical, medical and endovascular depending on the morphology of the dissection and the presence of associated complications. Endovascular treatment options are gradually evolving and becoming more popular in surgical practice. In this article, we report a successful endovascular treatment of an acute type-1 dissection in a 62-year-old female case who underwent coronary artery bypass grafting two months prior.

Keywords: Aortic dissection; coronary artery bypass grafting; endovascular treatment.

Aortic dissection (AD) is a medical emergency caused by the tear of intima and continuous disruption of the media layer of the aortic wall. [1] The intimal tear allows the blood pressure to enter the aortic wall, forming a hematoma, which further separates the intima from the media layers through a false lumen. [2] This false lumen may progress forward in either direction. Aortic dissection has a high mortality rate varying between 36 and 72% and many die within 48 hours of the diagnosis, which further increases 1% per hour, if remained untreated. [1] Patients with AD typically present with a sudden onset of a sharp chest pain which is often described as tearing and ripping in nature. [3] However, silent and painless ADs have

ÖZ

Akut diseksiyon, tedavi edilmediği takdirde, yüksek mortalitesi olan en tehlikeli aort hastalığıdır. Standart tedavi, diseksiyonun morfolojisine ve eşlik eden komplikasyonların varlığına bağlı olarak cerrahi, medikal veya endovasküler olarak değişmektedir. Endovaskülar tedavi seçenekleri cerrahi uygulamada giderek gelişmekte ve daha fazla yaygınlaşmaktadır. Bu yazıda, iki ay önce koroner arter baypas greftleme yapılan 62 yaşında bir kadın olguda tip-1 aort diseksiyonunun başarılı endovasküler tedavisi sunuldu.

Anahtar sözcükler: Aort diseksiyonu; koroner arter baypas greftleme; endovasküler tedavi.

also been described in some cases.^[2,3] Less common presentations are related to the organ hypoperfusion, including peripheral ischemia (19%), syncope (13%), myocardial infarction (13%), cardiogenic shock (8,8%), and neurological findings (6.1%).^[4,5] The actual incidence is difficult to estimate with a ratio of 5 to 20/1.000.000 and one-third of patients with AD are initially misdiagnosed on admission.^[6,7] Aortic dissection is seen more frequently in males between the fourth and seventh decades of life.^[8]

In this article, we report a successful endovascular treatment of an acute type-1 dissection in a female case who underwent coronary artery bypass grafting



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(CABG) two months prior. An aortic stent was successfully placed in the ascending and arcus aorta without any impairment of cerebral and brachial blood circulation.

CASE REPORT

A 62-year-old woman, who had a CABG operation two months ago, was admitted to our outpatient clinic with a sudden onset of chest and back pain. The computed tomography angiography (CTA) showed type-1 AD, where the intimal entrance was compatible with the level of previous aortic cannulation site (Figure 1). The dissection also involved the arcus aorta, extending downward to 3 cm proximal to the renal arteries. Saphenous vein grafts were patent, as assessed by the intact passage of opaque materials.

Examination of the previous surgery remarks revealed no apparent problem during the aortic cannulation or a structural deformity of the aortic wall. Repeated echocardiography after the CABG operation showed no signs of AD. However, the entry point of the AD was compatible with the site of aortic cannulation. Therefore, the patient was diagnosed with a late traumatic type-1 AD secondary to the aortic cannulation during CABG operation.

The patient was planned for an endovascular treatment depending on her age, history of previous CABG operation, the anatomy of the AD, and patent saphenous vein grafts. The entry of the AD was 14 mm above the aortocoronary saphenous vein graft orifices. Transthoracic echocardiography revealed an ejection

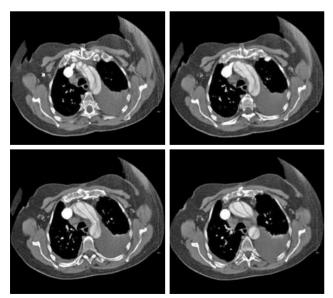


Figure 1. Computed tomography angiography slices showing the type-1 aortic dissection (Note the false lumen).

fraction of 45% with an aortic root (28 mm), ascending aorta (38 mm), and arcus aorta (31 mm) diameters. There were no valvular pathology and comorbidity. Biochemical blood test results were also within normal ranges.

The patient was sedated under general anesthesia and intubated in the angiography suite. Cerebral perfusion was monitored with electroencephalography (EEG).[9] The right femoral artery was explored and a 7F sheath was inserted. A 0.035-inch hydrophiliccoated guidewire (Glidewire®, Terumo Med Corp., NJ, USA) and a pigtail catheter (Optitorque®, Terumo Med Corp., NJ, USA) were progressed successively up to the ascending aorta without any resistance. An aortography view revealing the true lumen was obtained (Figure 2). The right brachial artery was cannulated percutaneously. A 0.035-inch hydrophilic-coated guidewire (Glidewire®, Terumo Med Corp., NJ, USA) was progressed through the brachiocephalic branch of the aortic root to the aortic lumen (Figure 2). Left carotid artery was also inserted using a 0.035-inch hydrophilic-coated guidewire (Glidewire®, Terumo Med Corp., NJ, USA) which was progressed through the left carotid branch of the aortic root to the aortic lumen (Figure 2). These guidewires and a peripheral balloon catheter (Renma®, Terumo Med Corp., NJ, USA) were kept available to secure the main trunks of the arcus

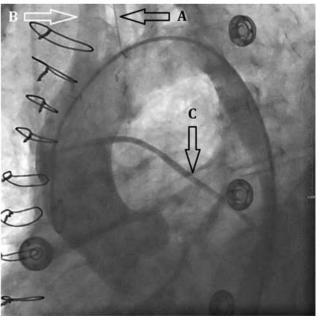


Figure 2. Aortography showing the aortic root, aortic arch, and its branches

A: Guidewire inside the left carotid artery; B: Guidewire inside the brachiocephalic artery; C: Temporary pacemaker.

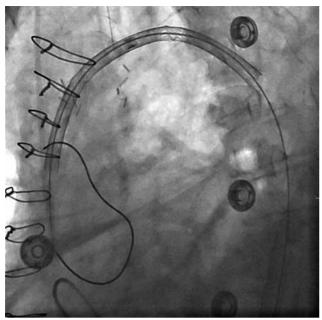


Figure 3. Aortography showing the deployment of the stent into the ascending aorta.

aorta from the occlusion in a case of an undesired displacement of the aortic intimal flap, while expanding the aortic stent.

A temporary transvenous pacing catheter (Medtronic Inc., Minneapolis, MI, USA) was introduced through the right femoral vein and tested inside the right ventricle (Figure 2). After the injection of 5000 IU unfractionated heparin, a 0.035-inch support catheter (CXI®, Cook Vascular, Vandergrift, PA, USA) was introduced and progressed through the pigtail catheter.

A dissection stent system (DJUMBODIS®, Saint Come Chirurgie, Marseille, France) with its own delivery catheter was, then, progressed to the ascending aorta (Figure 3). The stent was expanded initially at the level of intact aortic wall segment between the intimal tear and the aortocoronary saphenous vein ostia (Figure 4a). The temporary hypotension was constituted by setting the temporary pacemaker at 150 bpm. The mean blood pressure (BP) reduced to 10 mmHg during the cardiac arrest and the stent was fully opened in one minute of period (Figure 4b). A mixture of radioopaque and physiological saline (200 mL) were used to inflate the expanding balloon. A temporary pacemaker was closed and the normal sinus rhythm was achieved without a need for synchronized electric cardioversion. Normal BP was reconstituted with the infusion of positive inotropic agents. Although the EEG soon after the cardiac arrest showed a decrease, it was recovered quickly with the increased BP and almost reached the preoperative level at baseline.

A pigtail catheter was re-introduced over the support catheter and an aortography was repeated. It demonstrated that the entry disappeared and the dissected false lumen was pushed outwards to the original site (Figure 5). No intervention was, thus, needed for the previously secured arcus trunks, as the brachiocephalic trunk and the left common carotid artery circulations were completely normal (Figure 5). Renal functions were unaffected and the urine output was 70 mL/h during the process. All the catheters were withdrawn and the femoral arteriotomy was repaired with a 5-0 polypropylene suture. The patient who was hemodynamically stable was taken to the intensive care unit.

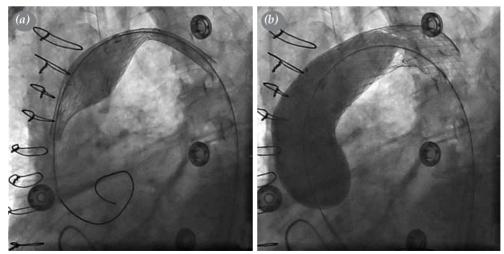


Figure 4. (a) The initial partial expansion. (b) The further proximal expansion pushes the intimal dissection flap back to the aortic wall (Note that the balloon protruding longitudinally out of the stent line under an excessive force.).

The patient was extubated postoperative eighth hour without any trouble. Blood urea nitrogen and creatinine levels were within normal ranges. The mean urine output was 90 mL/h. Repeated A control computed tomography performed on the next day showed the disappeared entry, circulating arcus branches, lost false lumen, and the correct position of the stent (Figure 6). Celiac and the renal arteries were circulating through the true lumen, and therefore, the dissected lumen distal to the stent was decided to be followed. All distal pulses were palpable. The patient was discharged on the sixth day after the intervention and scheduled for follow-up visits.

DISCUSSION

Acute AD is the most common fatal disease related to the aorta, if remained untreated. [10] According to its pathological and anatomical features, ADs of the ascending aorta (type A) carries a higher risk of mortality than the ADs of the descending aorta (type B). [11] The most common entry area susceptible to the initial disruption in the ascending aorta (type A) is the portion 2 or 3 cm above the coronary artery orifices. [8] Another general site is the isthmus side of the aorta, distal to the left subclavian artery (type B). [10] The dissection has the tendency to progress in either direction, until the intramural blood re-disrupts the intima distally and re-enters the main circulation of the true aortic lumen. [3]

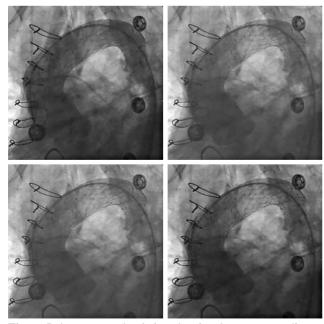


Figure 5. A post-procedural view showing the stent expanding to the planned lodge.

The dissection may sometimes remain thrombosed and this subintimal hematoma forms a blind sac without a re-entry tear. However, in the majority of patients, AD inevitably extends forward through the arcus aorta into the thoracic aorta and abdominal aorta.

In our case, the diagnosis was initially made as a late traumatic AD due to the aortic cannulation procedure two months ago, as the entry tear was consistent with the previous aortic cannulation site. However, a spontaneous acute dissection was not excluded. Irrespective of the definite diagnosis, treatment modality was mainly established based on the CABG operation history and the overall condition of the patient. Although the ascending ADs are best treated with surgical repair, patients, who are ineligible for surgery, as in our case, have a relatively poor prognosis.[13] The international registry of acute aortic dissection (IRAD) revealed that the 28% of the patients with type A dissections were considered ineligible for conventional open surgery.[13] Inappropriate conditions as the poor overall health status, advanced age, the

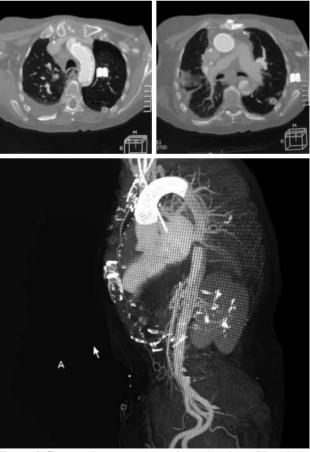


Figure 6. Computed tomography angiography views (Next day).

presence of neurological symptoms, and previous open heart surgery, as in our case, make the endovascular approach favorable. Adhesions also constitute a major concern in re-do surgeries. Adhesions also constitute a major concern in re-do surgeries. The risk of trauma to the bypass grafts during re-do surgery secondary to the CABG makes these patients having more risk than the others. Fortunately, recent improvements in endograft and stent technology have enabled surgeons to behave less invasive in the ascending AD patients who are poor candidates for surgery, particularly. Therefore, endovascular repair option has become the treatment of choice in this patient population having a previous cardiac surgery.

In the treatment of type-1 AD, two different stent types can be used: a bare stent or an endograft.[12] In our case, a bare stent was used rather than a branched endograft, as the previous one has the capability of providing in-stent blood passage directly to the cranial branches emerging from the arcus aorta.[13] Although bare stents have a higher incidence of endoleaks and further aneurysmatic dilatation, a continuous perfusion of the main aortic trunks should be the primary concern in the acute period.[12] Another point is the presence of a landing zone which is described as the segment between the saphenous vein graft orifices and the aortic entry tear. [13] In this case, the landing zone length above the saphenous vein grafts was appropriate for the stent expansion. Of note, there are two types of bare stents according to the expansion mechanisms: a balloon expandable or self-expandable stents. In our case, a balloon expandable stent was preferred rather than a self-expandable stent, as the latter has a poor control of radial force operated with a bidirectional expanding balloon. Uncontrolled radial force may overexpand the stent, leading to the displacement of the dissection flap which may subsequently create an obstruction at the orifices of the main trunks originating from the arcus aorta.[12] As a result, it should also be kept in mind that the constitution of the temporary pacemaker in the overdrive mode has a significant impact on the proper positioning of the stent during expansion.

In conclusion, although endovascular repair of the type-1 AD is still a rare entity in the literature, it can be successfully applied in selected cases who are ineligible for the conventional surgical treatment. Low mortality and morbidity rates make endovascular and hybrid approaches more favorable than the open surgery in medical practice.

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