A Case of an Acute Type A Aortic Dissection with Left Coronary Artery Malperfusion that was Successfully Stented as a Bridge to Surgery

Tatsuya Shigematsu, Hideki Okayama, Shinsuke Kido, Kenshou Matsuda, Tetsuya Aono, Yuta Tanaka, Tetsuya Kosaki, Saki Hosokawa, Go Kawamura, Yoshitaka Kawada, Go Hiasa, and Tadakatsu Yamada

Although acute type A aortic dissection with obstruction of the coronary artery is frequently fatal, some case reports have shown the effectiveness of stenting as a bridge to definitive surgery. We report a case of a 72-year-old woman referred to our hospital with acute onset chest pain. Her history included stent grafting for an abdominal aortic aneurysm three months before surgery, and percutaneous coronary intervention for the left circumflex artery and right coronary artery one year prior to admission. Electrocardiogram findings indicated ST-segment elevation. Emergency coronary angiography showed a 99% stenosis at the proximal part of the left anterior descending artery. Intravascular ultrasound study revealed a hematoma located from the proximal part of the left anterior descending artery to the left main trunk. After implanting a drug eluting stent from the left main trunk to the left anterior descending artery, computed tomography revealed an acute type A aortic dissection. Ascending aortic replacement and coronary artery bypass grafting were performed. Successful implantation of the stent at the left coronary artery was a bridge to surgery for the patient.

KEY WORDS: acute type A aortic dissection, stent, surgery

I. Introduction

Acute type A aortic dissection (AAAD) is frequently a fatal condition. This is especially true concerning cardiac tamponade or coronary malperfusion due to the extension of the medial dissection. AAAD with obstruction of the left coronary artery shows unstable hemodynamics and has a poor prognosis. Some case reports have already shown the effectiveness of stenting as a bridge to surgery in AAAD with coronary malperfusion, which is often fatal. We report a case of AAAD with left coronary artery malperfusion that was successfully stented as a bridge to surgery.

II. Case report

A 72-year-old woman was referred to our hospital with sudden onset of severe chest pain. She had a medical history of hypertension, dyslipidemia, stent grafting for abdominal aortic aneurysm three months previously, and percutaneous coronary intervention for the left circumflex artery and right coronary artery one year before admission. Her chest pain persisted at the time of arrival at our hospital, and she also presented with nausea. Initial vital signs were as follows: blood pressure, 136/93 mmHg, heart rate, 105 bpm, respiration rate, 18 breaths per minute, oxygen saturation, 96%. Her peripheral pulses were present and symmetric. On physical examination, she had an early diastolic murmur, and no abnormalities were found in the lungs or abdomen. Initial laboratory results were as follows: white blood cell count, 11,200 per µl; hemoglobin, 12.8 g per dL; platelets, 14.6×10⁴ µL; creatine kinase, 111 U per L with MB fraction 5 U per L; troponin- I, 11.2 pg per ml. Twelve-lead electrocardiogram (ECG) showed ST-segment elevation on leads V1–V5 with complete right bundle branch block rhythm at a rate of 101 beats per minute (Fig. 1A). A bedside transthoracic echocardiogram (TTE) showed severe hypokinesis of the anterior-anteroseptal wall without pericardial effusion. Other findings were an ejection fraction of 40%, a mild aortic regurgitation without dilatation. Emergency coronary angiography was carried out on suspicion of ST elevation myocardial infarction through a right radial approach. Right coronary angiography showed severe hypokinesis of the anterior-anteroseptal wall without pericardial effusion. Other findings were an ejection fraction of 40%, a mild aortic regurgitation without dilatation. Emergency coronary angiography was carried out on suspicion of ST elevation myocardial infarction through a right radial approach. Right coronary angiography showed severe hypokinesis of the anterior-anteroseptal wall without pericardial effusion.
'swinging lumen' (Fig. 1B and C). Intravascular ultrasound revealed a hematoma that was located from the proximal part of the LAD to the left main trunk (LMT), which compressed the lumen of the LAD (Fig 2). Since we thought that her AAAD involved the left coronary artery, we chose stenting from LMT to the LAD as a lifesaving procedure. We used a drug eluting stent (Resolute Onyx® 3.5 mm × 26 mm) placed from the distal to the proximal part of the LAD. A second drug eluting stent (Resolute Onyx® 3.5 mm × 18 mm) was implanted from the LMT to the proximal part of the LAD (Fig. 3A). After placing the stent, the intravascular ultrasound study showed that the distal stent edge was located at the distal end of the hematoma and the proximal edge was located at the ostial of the LMT. Final angiography confirmed a thrombolysis in myocardial infarction (TIMI) 3 flow and we finished the percutaneous coronary intervention procedure (Fig. 3B). The time required for reperfusion was 80 min.

Fig. 1  An ECG showing ST-segment elevation on leads V1-V5 with complete right bundle branch block rhythm (A). Left coronary angiography showing severe stenosis at the proximal part of the LAD at the initial phase of injection (B, arrows). At the late phase of the injection, anterior descending artery was dilated (C, arrows).

Fig. 2  The intravascular ultrasound study revealed that the hematoma was located from the proximal part of the LAD to the LMT (A and B, asterisk) and compressing the lumen of the LAD. (C and D, asterisk).
Computed tomography showed that the AAAD originated from the aortic arch and extended retrogradely above the sinuses of Valsalva and anterogradely to just below the proximal end of the stent graft for abdominal aortic aneurysm (Fig. 4A). Although the extended hematoma compressed the left main coronary ostium, coronary flow to the left coronary artery was sufficiently maintained by stent implantation (Fig. 4B). After confirming the diagnosis, the cardiovascular surgery team conducted an ascending aortic replacement and coronary artery bypass grafting using a saphenous vein graft (SVG). Distal end of SVG was anastomosed to LAD seg 8 and proximal end of SVG was anastomosed to the graft of the ascending aorta. The procedure was performed successfully without complication and the patient was discharged in a stable condition. Computed tomography that was performed before discharge revealed the patency of the SVG.

III. Discussion

We reported a case of AAAD with left coronary artery malperfusion that was successfully stented as a bridge to surgery. Despite of the development of improved diagnostic imaging and surgical strategies, AAAD is frequently fatal. Surgical outcomes are gradually improving, but the in-hospital mortality of surgical patients remains around 10%\(^2\). In patients with acute coronary
syndrome, missing AAAD increases mortality. Hirata et al. reported that ECG findings were normal in only 27% of AAAD patients. Neri et al. reported that the coronary artery was involved in 11.3% of AAAD cases, which had a negative effect on prognosis. Kawahito et al. showed that mortality due to AAAD with or without coronary malperfusion is significantly different (20–33% vs 12–15%). Acute myocardial infarction (AMI) occurs in 1–2% of patients with AAAD. A possible mechanism associating AAAD and AMI involves coronary ostium being compressed by the expanding false lumen. In this scenario, the dissection flap can partially occlude the ostium of the coronary artery, which lead to coronary thrombosis and subsequent AMI.

The dissection can extend directly into the coronary arteries from the aorta, and as in the present case, the right coronary artery is more often affected than the left coronary artery. Coronary angiography often reveals obstruction of the coronary artery caused by its compression due to false lumen expansion, termed ‘swinging lumen’ and this phenomenon might be an important sentinel of AAAD. In the present case, as a result of injecting contrast medium, vessel lumen pressure was raised which exceeded pressure from adventitia caused by the expanding hematoma. Thus, angiography revealed partial lumen dilation just after injection. Because left coronary artery involvement in cases of AAAD is a catastrophic condition, surgical treatment is challenging and may not be successful. Tominaga et al. suggested that treatment using a coronary perfusion catheter to stabilize a coronary artery compression by the expanding a hematoma will help maintain adequate coronary perfusion and reduce myocardial ischemia until surgical repair. Barabas et al. reported a case of emergent LMT stenting in AAAD complicated with anterior myocardial infarction, indicating that stenting for a collapsed LMT can be lifesaving and can serve as a bridge to surgery. After that case report was published, other case reports have shown effectiveness of stenting as a bridge to definitive surgery, similar to the case presented here.

In the present case, although coronary stent occlusion due to perioperative discontinuation of antiplatelet agents was also taken into account for coronary bypass grafting, if on the assumption that the coronary artery bypass grafting will be conducted, we might as well have used bare metal stent and implanted the stent only LMT as a bridge to surgery. In addition, some reports described that a technique of placing the coronary stent slightly out into the aorta and removing the implanted stent intraoperatively of the coronary bypass grafting also might be useful because no need of antiplatelet therapy and good patency of the bypass graft.

IV. Conclusion
AMI due to AAAD is a potentially fatal condition and surgical treatment of patients is challenging. We reported on a case of AAAD with left coronary artery malperfusion that was successfully stented as a bridge to definitive surgery, which resulted in a successful outcome for the patient.

Conflicts of interest
The authors state that they have no conflict of interest.

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