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CASE REPORT

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The treatment of spontaneous coronary dissection extending to the Valsalva sinus during percutaneous coronary intervention with primary stenting

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Abstract

Aortocoronary dissections are among the rare complications of percutaneous coronary interventions. Occasionally, coronary dissections can be the triggering factor for aortocoronary dissection. In this case, the dissection in the coronary artery may extend to the Valsalva sinus and ascending aorta progressively. In this article, we present and discuss a 54-year-old male who underwent percutaneous coronary intervention for acute inferior myocardial infarction. The aetiology for right coronary artery occlusion appeared to be spontaneous coronary dissection which extended to the Valsalva sinus. After treatment with primary stenting, the 6-month follow up results are presented here.

Key words: spontaneous coronary dissection, percutaneous coronary intervention, Valsalva sinus dissection

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Introduction

Dissection of the sinus of Valsalva during percutaneous transluminal coronary angioplasty (PTCA) is a rare complication which generally occurs as an extension of coronary dissection. The expansion of dissection to the aorta wall may cause complications like nonfatal myocardial infarction, the need for immediate surgery or death [1–3]. In this paper, we report a case of extension of dissection to the sinus of Valsalva during primary intervention to the right coronary artery. The patient was admitted with acute inferior wall myocardial infarction due to spontaneous coronary dissection, which was treated with primary stenting.

Case report

A fifty-four-year-old male patient with no history of cardiovascular disease presented to the emergency room because of squeezing chest pain that started 2 hours prior to admission. The pain radiated to the left arm. Previous medical history was significant with hypertension. His blood pressure was 140/80 mm Hg and his pulse was 88/min on admission. A 1/6 short systolic murmur was heard at the apex. In electrocardiography, ST elevations were noted at leads D2, D3, and aVF derivations. In right sided electrocardiography, V2R, V3R, V4R, and V5R were found to be normal. His electrocardiography was normal. Creatine phosphokinase, creatine phosphokinase myocardial band, and troponin I values peaked at 290 U/L, 54 U/L, and 17 ng/ml, respectively. The patient was diagnosed with acute inferior myocardial infarction.

Because of ongoing chest pain, the patient was taken to the catheter laboratory. In selective coronary

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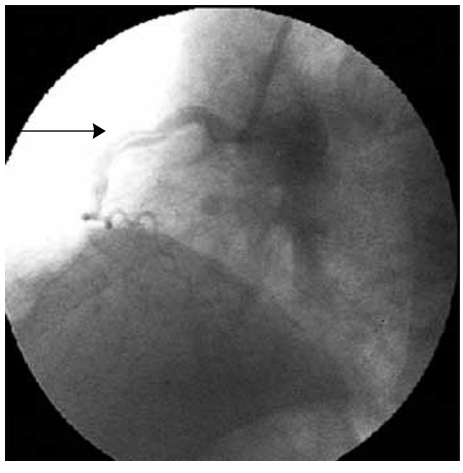

Figure 1. Spontaneous coronary dissection which causes acute inferior MI

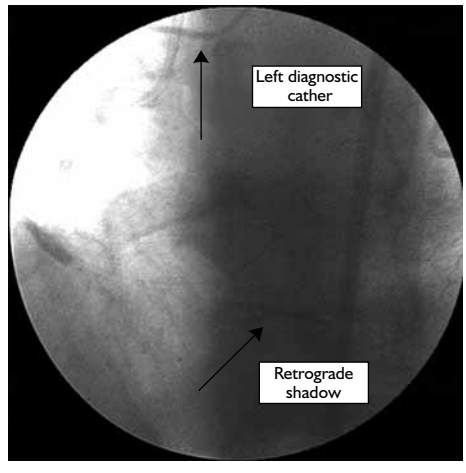

Figure 2. Differentiation of true and false lumen by screening of retrograd flow by contrlateral injection technique

angiography, the proximal of the right coronary artery (RCA) was noted to be dissected, thrombosed and totally occluded (Figure 1). No lesion was detected in the other coronary arteries. In left ventriculography, there was hypokinesis in the inferior region. Percutaneous coronary intervention to the lesion in RCA was planned subsequently. A 7 French right guiding catheter (Medtronic Inc., Minneapolis, MN, USA) and BMW 0.014 guide wire (Guidant Inc., St. Paul, MN, USA) were inserted to the RCA. For the distinction of true lumen from false lumen, selective imaging of the left coronary artery was performed with a 7 French left guiding catheter (Medtronic Inc, Minneapolis, MN, USA) via a puncture from the left femoral artery. Distinction between the real and false lumen was provided by following the retrograde flow of an opaque substance into the RCA (Figure 2). PTCA (4 × 12 mm) and subsequent stenting (4 × 22 mm, Microport Medical Co., Ltd, Shanghai, China) were performed to the dissected lesion. TIMI 3 flow was obtained by the provision of complete clearness. However, contrast pooling was observed in the localized region in Valsalva sinus when the lesion was controlled by administering an opaque substance during the procedure (Figure 3). It was thought that the dissection extended into the Valsalva sinus. The patient had no complaints. Primary stenting (4 × 22 mm ve 4 × 14 mm, Microport Medical Co., Ltd, Shanghai, China) was performed consecutively to RCA ostium to limit the dissection in the Valsalva sinus. The procedure was finished without apparent complication. In the follow-up, the patient's vital signs were stable and he was not in any pain. It was observed with computerized tomography that dissection did not expand and was localized in the Valsalva sinus. One day after

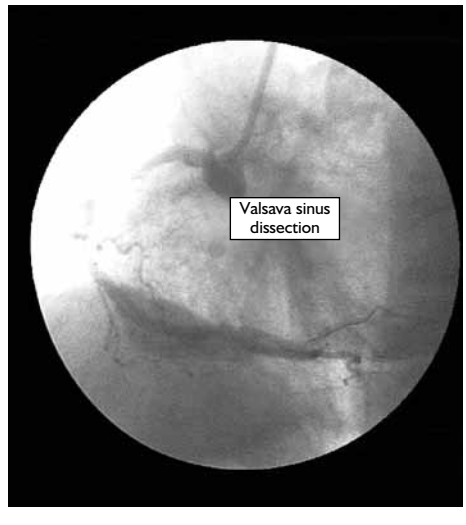

Figure 3. The extending of coronay dissection to sinus Valsalva during the operation

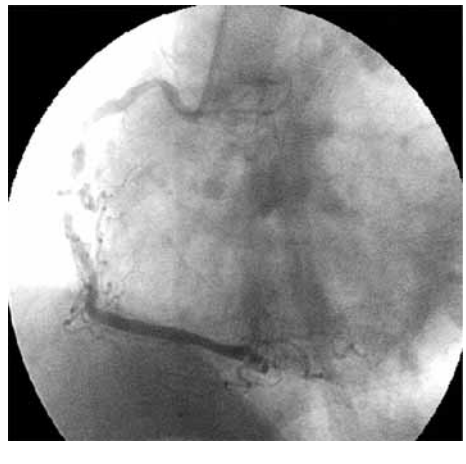

Figure 4. Control coronary angiography performed at sixth month

the procedure, in the root of aorta was free of dissection by the echocardiography. Ejection fraction was determined to be 45%. The patient was discharged after administration of beta-blocker; angiotensin converting enzyme inhibitor, statin, clopidogrel, and acetyl salicylic acid. The patient had no complaints at the 1st, 3rd, and 6th month follow-ups. His functional capacity was class 1–2. No aorta regurgitation was encountered. Stents were found to be patent in the control angiography performed in the 6th month and no dissection was observed in the aorta root in aortography (Figure 4).

Discussion

Spontaneous dissection of coronary arteries is a rare reason for acute coronary syndromes. Although the pathogenesis is not exactly known, haemorrhage in the media of the artery wall following primary separation of the vasa vasorum is a potential explanation [4]. Spontaneous coronary dissections are generally lethal; they can rarely be observed during coronary angiographies and can be seen as rarely as once in 1000 cases [5]. The aetiology is not yet clear and abnormalities in collagen synthesis are thought to be the cause [6]. It is generally seen in young women especially during pregnancy, postpartum period and after oral contraceptive intake. It is considered that microstructural changes formed in the systemic arteries due to haemodynamic and hormonal factors might be the cause [7]. Plaque rupture causes separation of the intima from media in patients with atherosclerotic coronary artery disease and may conclude with localized dissections [8]. Clinical conditions such as hypertension, vasculitis, and connective tissue diseases might accompany spontaneous coronary dissection [4, 5]. Our case was hypertensive, and we think that the plaque in the coronary artery was ruptured and caused the dissection. There were no clinical findings to raise suspicion of underlying connective tissue disease.

Dissection in RCA is generally encountered during cardiac catheterization and percutaneous coronary intervention, and the cause is usually a guiding catheter [9–11]. The procedures relating to dissections are generally mild and localized. They recover spontaneously or easily with stenting without any sequela. However, the guiding catheter may cause dissection to progress anterograde to the ostium and retrograde to the distal part of RCA [1]. Important factors determining the course of dissection are; the structural properties of the guiding catheter, the anatomic features of the coronary arteries, and the experience and technical knowledge of the surgeon [12]. Atherosclerotic plaques at proximal RCA cause loss of elasticity in half of the patients, so RCA becomes more susceptible to procedural traumas. RCA has an angled view in most patients. Since the guiding catheter is generally placed at angle points, dissection development during manipulation becomes easier [12]. The increase in coronary flow pressure after contrast injection might cause the dissection to expand [1]. Exposure of the coronary artery to the sheer effect in both phases due to the blood flow in RCA, both in systole and diastole, and also having less collateral than left artery, explain the higher prevalence of spontaneous dissection in the RCA than in the LAD [2]. Age, hypertension, and calcifications in the coronary artery are reasons that may cause development of dissection. Our case was that of a 54-year-old who had been recommended medication due to hypertension but did not take his drugs regularly. His body constitution was not marfanoid. No drug abuse or exhausting exercise was noted in his history. Laboratory tests to search for the presence of connective tissue diseases were negative. In angiography there was no calcification in the coronary artery ostium; however, RCA had an angled takeoff. We propose that in our case the prolonged procedure, manipulations, and repeated contrast injections might have caused the spread of spontaneous dissection to the Valsalva sinus in addition to the structural features of RCA.

Coronary artery dissections that invade the aorta wall can be treated with different methods, i.e. conservative treatment [1, 13], stenting [2, 3, 13], or surgery [2, 3, 13]. Perez- Castellano et al. observed that localized dissections of the Valsalva sinus spontaneously recovered within one month of medical treatment. It was stated that this situation was partially dependent on the anatomic structure of the Valsalva sinus. The upper limit of each aortic sinus contains circumferential supravalvular fibres enriched with collagen fibres, which is why dissections are generally limited to the Valsalva sinus [1]. In this case, we estimated that successful ostial stenting procedure had as much a role as the structure of the Valsalva sinus in the treatment of the Valsalva sinus dissection.

One of the most important points in the stent application for the treatment of dissections originating from the RCA and extending to the aorta is the distinction between false and true lumens. The assessment of false lumens as true lumens may result in failure of the procedure, and may even lead to serious complications. In these clinical conditions, monitoring the retrograde flow in the dissected coronary artery by administering an opaque substance to the counterlateral coronary artery with a puncture from the femoral artery facilitates the distinction. We used this method for the treatment of our patient.

Dunning et al. showed that limited dissections extending to the aorta can be treated with successful stenting of the entry point of the coronary dissection. However, they stated that the surgical procedure had to be applied in dissections extending 40 mm over the coronary ostium [2]. In addition, coronary stenting may heal aortocoronary dissection by preventing it from spreading. Therefore, a surgical procedure may not be necessary [1–3]. In our case, the dissection was localized in the Valsalva sinus; therefore, we did not consider surgery. We think that successful percutaneous coronary intervention after distinction between the true and false lumens caused the dissection to be localized in the Valsalva sinus and saved the patient from a serious complication.

In conclusion, spontaneous coronary dissections may extend to the Valsalva sinus and cause vital complications. Coronary stenting procedure is one of the successful treatment methods in these conditions. However, the accurate distinction of real lumens from false lumens during the stenting procedure of the dissected lesions is important.

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