UNSUCCESSFUL PRIMARY PCI FOR POSTPARTAL DISSECTION OF ALL THREE MAJOR CORONARY ARTERIES

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Coronary artery dissection is rare but very dangerous condition which can result in myocardial infarction. It mostly occurs in young postpartum women. The left anterior descending artery is affected in 75% of cases and in some extremely rare cases dissection may include all three coronary arteries. Treatment guides are still not consistent. While some authors recommend medical treatment other recommend surgical one. Case report. We present a 36-year-old female patient who was admitted to our hospital with intermittent chest pain and dyspnea that occurred two hours before admission. After premedication with Aspirin 300 mg per os, Clopidogrel 300 mg per os and Enoxaparin 30 mg iv, the pain disappeared. Chest pain was repeated after five days. Coronary angiography revealed spiral dissection in medial segment of LAD. After PCI attempt, dissection progressed and ended fatal. Pathological finding was intimal dissection of the coronary arteries with loss of some parts of internal elastic lamina. It is interesting that in the literature there is no consistent opinion about therapeutic approach to SDCA and we hope that this case report will contribute to elucidating the problem.

Key words: acute spontaneous coronary artery dissection, fibromuscular dysplasia, PCI treatment of SCAD

Introduction

Spontaneous coronary artery dissection (SCAD) is generally considered a rare event which, if not recognized and treated on time, can be fatal. SCAD is an unusual cause of acute coronary syndrome which is not associated with conventional risk factors. It was first described in 1931 in a 42-year-old healthy woman who died shortly after the first symptoms appeared (1). It is difficult to estimate real epidemiology trends due to the limited number of reported cases. However, approximately 500 cases of SCAD have been described in literature. The majority of patients (71.9%) were women and 28.1% were men. SCAD has been reported in 10% of women younger than 50 years presenting with acute coronary syndrome. Same studies have shown that the prevalence of SCAD increases with ageing of the population, so its highest prevalence (76%) is among patients under the age of 40 (2). However, some latest reports showed that SCAD is not such a rare manifestation. In the population of women who underwent coronary angiography, 29.3% had SCAD (3). One third of them were young, healthy women in the last trimester of pregnancy or shortly after delivery, as Koul et al. reported. Most of them presented in the postpartum period (78%) and the rest of them during pregnancy. SCAD usually occurs within two weeks of delivery, but in some patients it could present very late (10-12 weeks postpartum). The left anterior descending artery (LAD) is affected in 75%, the right coronary artery (RCA) in 20%, the left circumflex artery (LCx) in about 4%, and the left main coronary artery (LMCA) in less than 1% of the cases (4). In extremely rare cases, SCAD may include all three coronary arteries simultaneously (5).

Case report

We present a case of a 36-year-old woman who was admitted to our hospital with intermittent chest pain and dyspnea that occurred two
hours before admission. Blood pressure was 120/80 mm Hg with heart rate 100/min. It was tenth postpartum day after an uncomplicated spontaneous vaginal delivery. She had two successful pregnancies (7 and 3 years before) and was on hormone fertility stimulation therapy in the last three years. Two months before delivery she was taking Hexoprenaline (0.5 mg per day) and Verapamil 80 mg (per day). She had been an active smoker for 10 years, (approximately 20 cigarettes per day) without other risk factors for coronary artery disease.

Baseline electrocardiogram (ECG) showed negative T waves in D2, D3 and aVF, V2-V6 leads which suggested anterior-lateral and inferior ischemia (Figure 1A). Troponin I level was 1.10 ng/ml. Echocardiography revealed akinetic apex of the left ventricle and estimated ejection fraction was 63%. After premedication with aspirin 300 mg per os, clopidogrel 300 mg per os and enoxaparin 30mg iv, the pain disappeared. Chest pain was repeated after five days with ECG changes - ST elevation in D2, D3, aVF, V2-V6 leads (Figure 1B). Troponin I level was 3.14ng/ml. The patient was immediately transferred to invasive coronary unit for further diagnosis and treatment.

Coronary angiography revealed spiral dissection of LAD and LCx (Figure 2A) with 80-90% diameter stenosis in the medial segment of LAD (Figure 2B). The lesion in the medial segment of LAD had the appearance of a sharp narrowing of the artery lumen with irregularities.

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Figure 1. ECG showing: negative T waves in D2, D3 and aVF, V2-V6 leads (A); ST elevation in D2, D3, aVF, V2-V6 leads (B)
Right coronary artery catheterization revealed dissection at the ostium (Figure 1C). Dissection continued to spread proximally from both LAD and LCx towards the LMCA. Because the patient was hemodynamically unstable with signs of cardiogenic shock and bradycardia, we placed contrapulsatory intra-aortic balloon pump and temporary pace maker. Stents implantation was done from the medial segment of LAD to the LMCA (Figures 2D, 3A, 3B). After stent placement in the LMCA, patient was disoriented and lost consciousness, with Thrombolysis in Myocardial Infarction (TIMI) 2 coronary flow in both LAD and LCx. After the implantation of another stent in the medial segment of LAD due to the uncovered dissection (Figure 3C), no-reflow phenomenon and consequently cardiac arrest developed (Figure 3D). Cardiopulmonary resuscitation was unsuccessful.

An autopsy was done.

Pathological finding was intimal dissection of the coronary arteries with the loss of some parts of internal elastic lamina which is labeled as fibromuscular dysplasia (Figure 4).

Figure 2. Coronary angiography series showing: LAO CRAN, (LJ 4.0; 6 F) LCA where we can see sharp lumen narrowing in medial to distal segment of LAD, there are no any other changes in coronary arterial system (A); LAO CAUD, (RJ, 4.0; 6F) RCA, dissection of ostial part of RCA (B); RAO CAUD: First projection of LCA with guing catheter (GEBU 3.75) where spiral dissection is present in the main stem of LAD and ACx (C); BMW wire placed in LAD and ACx. First stent was implanted in the medial part of LAD (D)
Figure 3. Coronary angiography series show: Second stent placed in the proximal part of LAD and overlapped with the first one. TIMI flow in LAD was 3 (A); Stent placed in the main stem (B); Dissection progressed to aorta and flow in distal part of LAD was lost. We placed one more stent in the proximal part (C); Last projection before exitus letalis (D).

Figure 4. Pathological findings show: Coronary artery dissected with blood. HE, Ob.x4 (A); Thickened intima and disrupted internal elastic lamina. Media destroyed with blood. Autofluorescence, obj.x10 (B)
Discussion

All SCAD patients have been traditionally divided into three groups: patients with coronary artery disease (CAD), women in the last trimester of pregnancy or shortly after delivery, and the idiopathic group. SCAD related to pregnancy is usually labeled as p-SCAD (6). The first peripartum case of SCAD was reported in 1952 (1). Etiology of SCAD remains unclear. De Maio showed that only 30% of patients had risk factors for atherosclerosis but they found that both multiparity and advanced age are related to SCAD diagnosis. However, Hering et al. showed that atherosclerosis is more often the cause of SCAD. They reported that rupture of atherosclerotic plaque occurred in 35 of 42 patients with SCAD (3). Intimal thickening caused by hormone level increase during pregnancy is also reported to be the possible cause of pregnancy associated SCAD as Gammal showed (7). He showed significant intimal thickening in female rats exposed to synthetic estrogen and progestosterone. Literature review of histological finding showed that the main findings are: eosinophiles infiltrate, cystic medial necrosis, intimal tear, but, in some cases, there were no pathological findings. Role of eosinophiles in SCAD has been well described in literature. Basso et al. described hematoma between tunica media and adventitia. Study from 1996 showed that SCAD was the cause of acute myocardial infarction or sudden cardiac death. In all cases women were involved. Histology findings included eosinophilic infiltrate in four cases, cystic medial necrosis in one case and in the rest of them there was no histological finding (8). Significantly different eosinophile presence in SCAD group was shown when compared to control group. The explanation of possible relation between eosinophiles and dissection is well documented (9). Connective tissue disorders have been lately described by Harrison, our case can be diagnosed as intimal fibromuscular dysplasia (11).

The treatment of SCAD can include medical therapy and revascularization procedures such as percutaneous coronary intervention or coronary artery bypass graft surgery. The decision which treatment to use depends on the clinical presentation and the extent of coronary dissection. Conservative treatment with antiplatelet medications, antithrombin agents, nitrates, and beta blockers may be a reasonable approach in asymptomatic, stable patients with limited dissections (12,13). The literature today abounds with data which have described PCI as successful main treatment of SCAD (14). However, in all these cases dissection occurred in one vessel, mostly LMCA, unlike our case where dissection occurred in all three arteries simultaneously (15). For dissection involving several coronary vessels, beating heart surgery is a recommended first line treatment since it can restore blood flow distal to the dissection. This intervention also provides reinforcement for false and true channel (16). Tweet et al. showed that patients with a single lesion in the left or right coronary artery had significantly better outcome when treated with an early aggressive strategy. Early treatment with CABG or percutaneous coronary intervention following the diagnosis of SCAD leads to less need for further intervention (17).

If we consider this case, we are sure that the operator (the author of this paper) was not up to the task. It is correct that we immediately set up a balloon pump and temporary pacemaker to provide cardiac output and hemodynamic support. Should the patient be immediately referred to an emergency surgery unit? (In our center there is no such possibility). Was it a basic mistake to place stents in the LAD from the distal segment to the main stem? Considering this case, knowing that most SCAD have fundamentally less valuable media layer and that it usually progresses proximally and distally through the vessel wall, we believe that it is correct to put a stent in the LMCA toward the LAD and than refer the patient to immediate surgery in other center. We hope that the comments of readers of this text will help interventionalists in better understanding of this potentially fatal event and point out the best therapeutic approach in these and similar cases.

Conclusion

This case report shows one of the types of fibromuscular dysplasia as a cause of spontaneous dissection of the coronary arteries and acute coronary syndromes (ACS) in the post-partum period. It also draws attention to this population of patients experiencing ACS and high probability of SDCA as a main cause of ACS. It is interesting that in the literature there is no consistent opinion about therapeutic approach to SDCA and we hope that this case report will contribute to elucidate the problem.
References


SPONTANA DISEKCIJA SVE TRI KORONARNE ARTERIJE KAO UZROK AKUTNOG INFARKTA MIOKARDA

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Spontana disekcija koronarnih arterija je redak, ali vrlo opasan događaj, koji može da se završi infarktom miokarda. Najčešće se javlja kod mladih žena pred kraj ili neposredno nakon porođaja. Leva koronarna arterija zahvaćena je u 75% slučajeva, a u nekim, vrlo retkim slučajevima, disekcija može da zahvati sve tri koronarne arterije.

Ključne reči: spontana disekcija koronarnih arterija, fibromuskularna displazija, PCI tretman SDKA

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