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CASE REPORT / ПРИКАЗ БОЛЕСНИКА

Spontaneous coronary artery dissection as a cause of acute myocardial infarction with ST elevation

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SUMMARY

Introduction Spontaneous coronary artery dissection (SCAD) is defined as a dissection that has not occurred with atherosclerosis, trauma, or has not developed iatrogenically.

Case outline A 53-year-old man was admitted to the hospital due to chest pain and ischemic electrocardiographical changes. Coronarography was performed and 85% of the stenosis of the first diagonal branch (D1) was registered. During percutaneous coronary intervention (PCI), one drug-eluting was directly implanted into the D1. About three hours after the intervention, the patient developed an acute myocardial infarction with ST elevation (STEMI) and recoronarography was performed. The previously implanted stent in D1 was patent without thrombi. The subocclusive stenosis of the left anterior descending artery (LAD) was registered and PCI was performed. After implantation of the stents into the LAD, propagation of dissection towards left circumflex artery (LCx) was creating significant stenosis. Following the registration of the stenosis, PCI was performed on this branch. In order to determine the cause of acute STEMI, intravascular imaging was performed, seven days after last PCI. Optical coherence tomography showed an excellent stent apposition and expansion. In the area under the stents, in the proximal segment of LAD and LCx, showed duplication in the blood vessel wall. This duplication represents an unresorbed intramural hematoma as a consequence of SCAD.

Conclusion When performing coronarography on younger patients, on women in the peripartum and on patients with connective tissue, SCAD disorders should be kept in mind. The use of intravascular imaging could reduce the number of unrecognized SCAD.

Keywords: spontaneous coronary artery dissection; acute myocardial infarction with ST elevation; optical coherence tomography

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) represents a dissection that occurred without atherosclerosis, trauma or has not developed iatrogenically. There are two theories which describe how SCAD develops [1, 2]. According to the first theory, endothelial injury is accompanied by consequent penetration of blood, into the blood vessel wall [1]. According to the second theory, the primary event is the spontaneous bleeding from the vasa vasorum into the blood vessel wall [2]. SCAD causes 1–4% of all acute coronary events [3]. However, the true incidence and prevalence of SCAD are unknown, because they are often unrecognized. SCAD most commonly occurs in patients who do not have traditional risk factors for cardiovascular diseases [4]. The association with female sex, pregnancy, and oral contraceptives has been described [4, 5]. Systemic arteriopathies are also a predisposing factor and SCAD is most commonly associated with fibromuscular dysplasia. The trigger for the development of SCAD can be intense physical and emotional stress. The first step in the diagnostic algorithm is coronarography, which should be carefully performed to avoid the extension of dissection. In uncertain cases, intracoronary imaging may be helpful.

We present a case of a patient who developed acute myocardial infarction as a consequence of spontaneous dissection of the left anterior descending (LAD) branch of the left coronary artery.

CASE REPORT

A 53-year-old man was admitted to the emergency department due to chest pain and ischemic electrocardiographical (ECG) changes. Cardiovascular risk factors (smoking and obesity) were present, while physical examination did not show any irregularities. Electrocardiogram showed negative T wave in DI, aVL and V5–V6. High-sensitivity troponin was within the reference values. Transthoracic echocardiography initially and after transpulmonary contrast agent administration (Optison; GE Healthcare, Chicago, IL, USA) showed reduced systolic function with regional wall motion abnormalities (medio-apical segments of antero-lateral wall). Dual antiplatelet therapy per protocol for acute coronary syndrome was administered. Coronarography was performed and 85% stenosis of the first diagonal branch (D1) was registered (Figure 1A), while other coronary arteries were without

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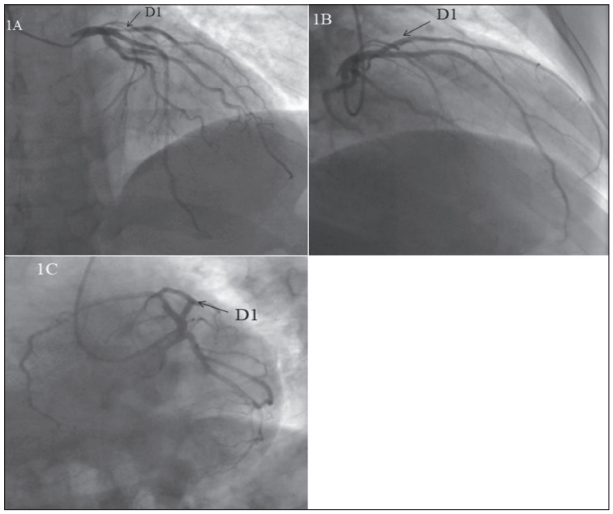

Figure 1. A: Significant stenosis of the first diagonal branch; B: the angiographic result after stent implantation on the first diagonal branch; C: the angiographic result after stent implantation on the first diagonal branch

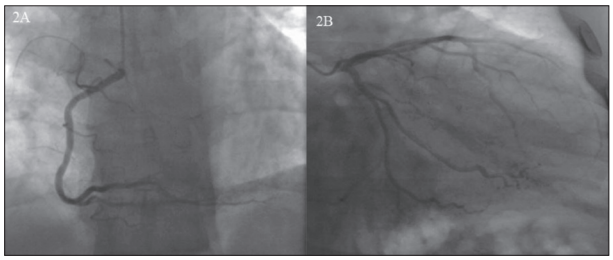

Figure 2. A: There is no significant stenosis on the right coronary artery; B: there is no significant stenosis on the left circumflex coronary artery

significant stenosis (Figures 2A and 2B). During percutaneous coronary intervention (PCI), one drug-eluting stent of 15 × 3.5 mm (XienceXpedition; Abbott Laboratories, Chicago, IL, USA) was directly implanted into the D1 (Figures 1B and 1C). About three hours after the intervention, the patient developed chest pain. On electrocardiogram, ST segment elevation V1–V5 was registered (Figure 3). Because the patient had cardiac decompensation, he was sedated, endotracheally intubated, and invasively mechanically ventilated. Due to suspected acute stent thrombosis, urgent recoronarography was performed, which showed patent previously implanted stent in D1, without thrombi (Figure 4A). However, a subocclusive stenosis was registered in the medial segment of the LAD, approximately 3.5 cm distal to the origin of the first diagonal branch (Figure 4A). During the PCI, two drug-eluting 28 × 3 mm and 28 × 3.5 mm stents (Synergy, Boston, MA, USA) were implanted, with the overlap technique, in the LAD, with optimal angiographic result (Figure 4B). After implantation of the stents into the LAD, significant stenosis of the circumflex branch of the left coronary artery (LCx) was registered (Figure 4B). This stenosis had not appeared during the previous coronarography and was not due to spasm. Drug-eluting 15 × 3 mm stent (Resolute; Medtronic, Dublin, Ireland) was implanted into the proximal segment of the LCx, with the optimal result (Figure 4C). In order to determine the cause of acute STEMI,

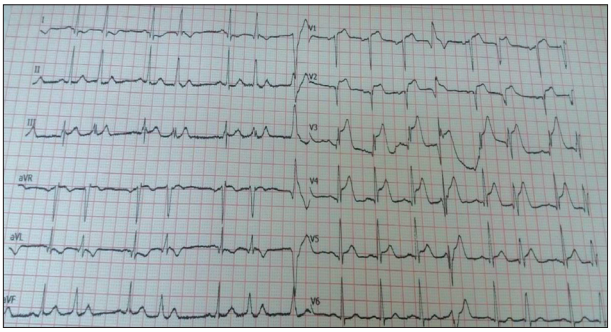

Figure 3. Electrocardiogram shows ST segment elevation V1–V5

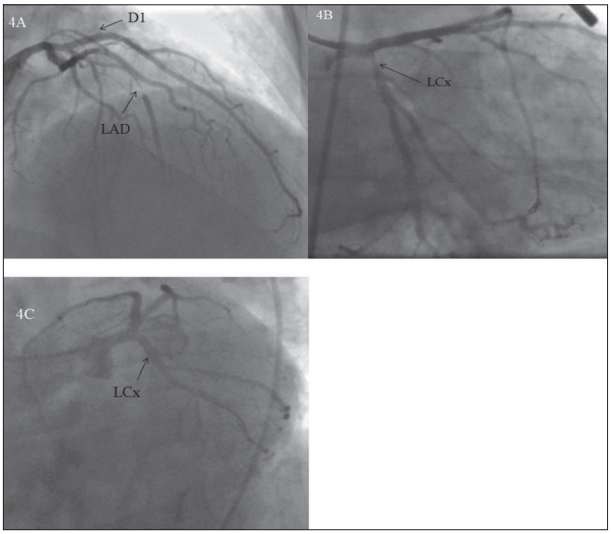

Figure 4. A: Angiography of the left coronary artery showing subocclusive stenosis in the medial segment of the left anterior descending artery (LAD); previously implanted stent in D1 was without thrombosis; B: after implantation of the stents into the left anterior descending artery, significant stenosis of the left circumflex artery was registered; C: the optimal result after percutaneous coronary intervention on the left circumflex artery (LCx)

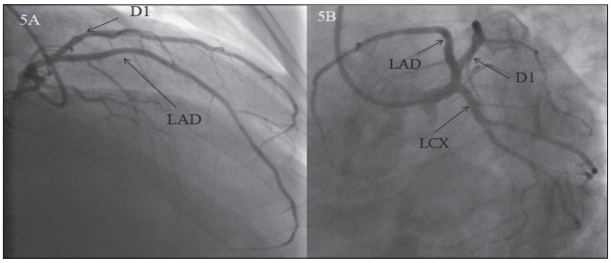

Figure 5. A: After seven days, optimal result in the area of implanted stents in D1 and left anterior descending artery (LAD); B: after seven days, optimal result in the area of implanted stents in D1, the left anterior descending artery, and the left circumflex artery (LCx)

intravascular imaging was performed, seven days after the previous PCI. Optimal result in the area of implanted stents in LAD and LCx were registered on coronarography (Figure 5). Optical coherence tomography (OCT) showed an excellent stent apposition and expansion, but also the duplication in the blood vessel wall in the area below the implanted stents, in proximal LAD and LCx. This duplication represents an unresorbed intramural hematoma as a consequence of SCAD (Figure 6). Since the cause of acute myocardial infarction was SCAD, the patient was switched

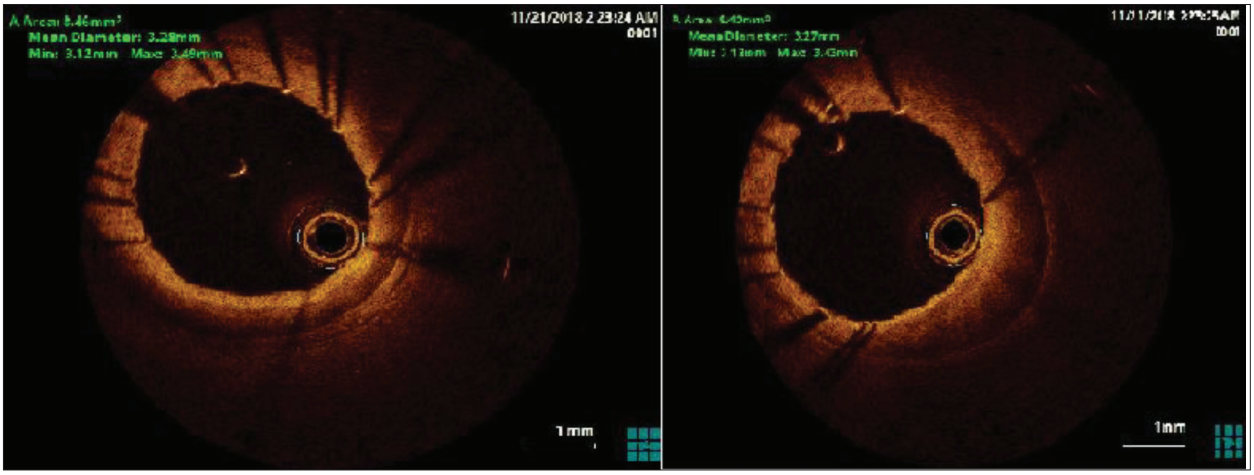

Figure 6. Optical coherence tomography under the stents in the proximal segment of the left anterior descending artery and the left circumflex artery reveals a duplication in the vessel wall; this duplication represents unresorbed intramural hematoma as a consequence of spontaneous coronary artery dissection

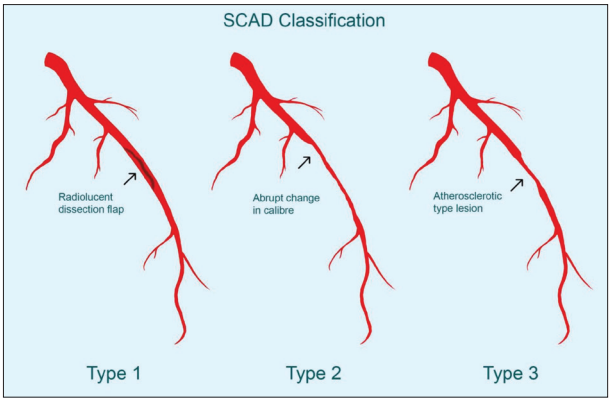

Figure 7. Angiographic spontaneous coronary artery dissection classification system

to clopidogrel, according to recommendations [6]. After six months, exercise stress testing on treadmill (Bruce protocol) was performed. The patient achieved maximal effort (up to 10 METs), without clinical and ECG signs of coronary flow reduction.

Written consent to publish all shown material was obtained from the patient.

DISCUSSION

There are three types of SCAD (Figure 7) [7]. Type 3 is the rarest angiographic manifestation of SCAD, occurring in 3.4% of patients, while the most common is type 2, in about 67.5% of patients [3]. In uncertain cases, intravascular imaging may be helpful. Despite the great importance of intracoronary imaging, caution is needed because of possibility for extension of coronary dissection by using a wire or diagnostic catheter. Seven days after the first coronary event we performed OCT. In the area below the implanted stents in the proximal LAD and LCx, we recognized duplications in the blood vessel wall corresponding to the unresorbed hematoma as a result of SCAD. The lesion in D1 was not recognized as a SCAD type 3. The stent was implanted into the proximal part of D1 and thus prevented the spread of the intramural hematoma further into the diagonal branch. Intramural hematoma propagated in the LAD distal to the first diagonal branch. After the first stent was implanted into the medial part and then the second one into the proximal part of the LAD, intramural hematoma was pushed proximally to the ostium of LCx.

A Spanish multicenter prospective SCAD registry, including 318 patients with SCAD, showed that the artery most frequently involved was the LAD (44%), predominantly affecting the distal segments (39%) and its branches (54%) [8]. In our case, the LAD was also affected by SCAD.

Patients with SCAD were divided into four groups. The first group consisted of patients with hereditary connective tissue disorders associated with arterial wall defect (Marfan syndrome) [9]. The second group represented patients with underlying atherosclerosis, especially men at an average of 55 years [9]. The third group included women in the peripartum period, while the last group consisted of patients with idiopathic SCAD [10, 11]. According to this classification, our patient can be classified into the second group – he is a male, 53 years old, obese, and smoker. It is also important to emphasize that SCAD can be associated with exposure to physical effort, chest trauma, and certain drugs (cocaine, cyclosporine, 5-fluorouracil, and oral contraceptives) [11]. Our patient on the day of the hospital admission was exposed to hard physical effort. Treatment of SCAD includes medical treatment, PCI, and surgical myocardial revascularization. Considering the high risk of complications during PCI, conservative treatment is favored. Also, because of the spontaneous healing of SCAD and the resorption of intramural hematoma in about 70–97% of patients, conservative treatment is advised [12]. A Canadian SCAD cohort study was a multicentric, prospective, observational study of patients with non-atherosclerotic SCAD from 22 centers in North America [13]. The majority of these patients (84.3%), who were being treated conservatively, had good survival rate. However, patients with ongoing ischemia and hemodynamic instability are

to be treated with PCI. In our case, acute STEMI complicated with progredient heart failure indicated primary PCI, which was performed with optimal angiographic results. Data from the literature states that successful PCI is associated with (1) implantation of long stents covering the 5–10 mm proximal and distal edge of the intramural hematoma, (2) direct stent implantation without prior balloon predilatation, (3) balloon dilatation without stent implantation, (4) fenestration of the intramural hematoma by cutting the balloon and decompression of the false lumen, (5) multistent approach by firstly sealing distal and proximal ends with stents, before stenting the middle, and (6) use of bioresorbable stents [14, 15, 16]. Balloon dilation with low-pressure (up to 4 atm) inflations should be used to decrease the risk of perforation [17]. Coronary artery bypass grafting has been described as a treatment strategy for SCAD in patients with left main stenosis and also after technical failure of attempted PCI. According to experts, the basis of medical therapy are acetylsalicylic acid and beta-blockers. Administration of the P2Y12 inhibitors is controversial for patients not treated with PCI. In patients treated with PCI, dual antiplatelet therapy with clopidogrel is recommended [18]. After OCT analysis, which confirmed the SCAD of the LAD, our patient was switched to clopidogrel. Intrahospital survival of these patients is good; however, recurrent spontaneous coronary artery dissections are common. During the follow-up period, our patient was without symptoms of angina. After six months, exercise stress testing on treadmill (Bruce protocol) was performed and the patient achieved maximal effort (up to 10 METs), without clinical and ECG signs of coronary flow reduction. When performing coronarography on younger patients, women in the peripartum and patients with connective tissue disorders, SCAD should be kept on mind, especially if the angiographic finding indicates a possible type 2 or 3 spontaneous dissection. In the future, intravascular imaging could reduce the number of unrecognized and inadequately treated SCAD.

Conflict of interest: None declared.

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Спонтана дисекција коронарне артерије као узрок акутног инфаркта миокарда са *ST* елевацијом

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САЖЕТАК

Увод Спонтана дисекција коронарне артерије (СДКА) дефинисана је као дисекција која није повезана са атеросклерозом, траумом, нити је настала јатрогено.

Приказ болесника Мушкарац старости 53 године је примљен у болницу због болова у грудима и електрокардиографски регистрованих исхемијских промена. Урађена је коронарографија и нађено је 85% сужење прве дијагоналне гране. У истом акту је урађена перкутана коронарна интервенција, са директном имплантацијом леком обложеног стента у прву дијагоналну грану. Око три сата после интервенције код болесника се развио акутни инфаркт миокарда са *ST* елевацијом и урађена је коронарографија. Претходно имплантирани стент у првој дијагоналној грани био је без тромбозе. Нађена је субоклузивна стеноза леве предње силазне артерије те је урађена перкутана коронарна интервенција. После имплантације стента у леву предњу силазну артерију регистовано је сужење на левој циркумфлексној артерији као последица пропагације дисекције. Урађена је перкутана коронарна интервенција и на овој грани. После седам дана, у циљу дефинисања узрока акутног инфаркта миокарда са *ST* елевацијом, урађен је интраваскуларни имиџинг. Оптичка кохерентна томографија показала је одличну апозицију и експанзију стентова. У подручју испод стентова у проксималној левој предњој силазној артерији и левој циркумфлексној артерији региструје се дупликатура у зиду крвног суда. Ова дупликатура представља нересорбовани интрамурални хематом као последицу СДКА.

Закључак Када се изводи коронарографија, код младих људи, жена у перипарталном периоду и болесника са болестима везивног ткива треба мислити на СДКА. Уз интраваскуларни имиџинг, треба настојати да се смањи број непрепознатих СДКА.

Кључне речи: спонтана дискеција коронарне артерије; акутни инфаркт миокарда са *ST* елевацијом; оптичка кохерентна томографија