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Heart and Blood Vessels

Journal of the Cardiology Society of Serbia

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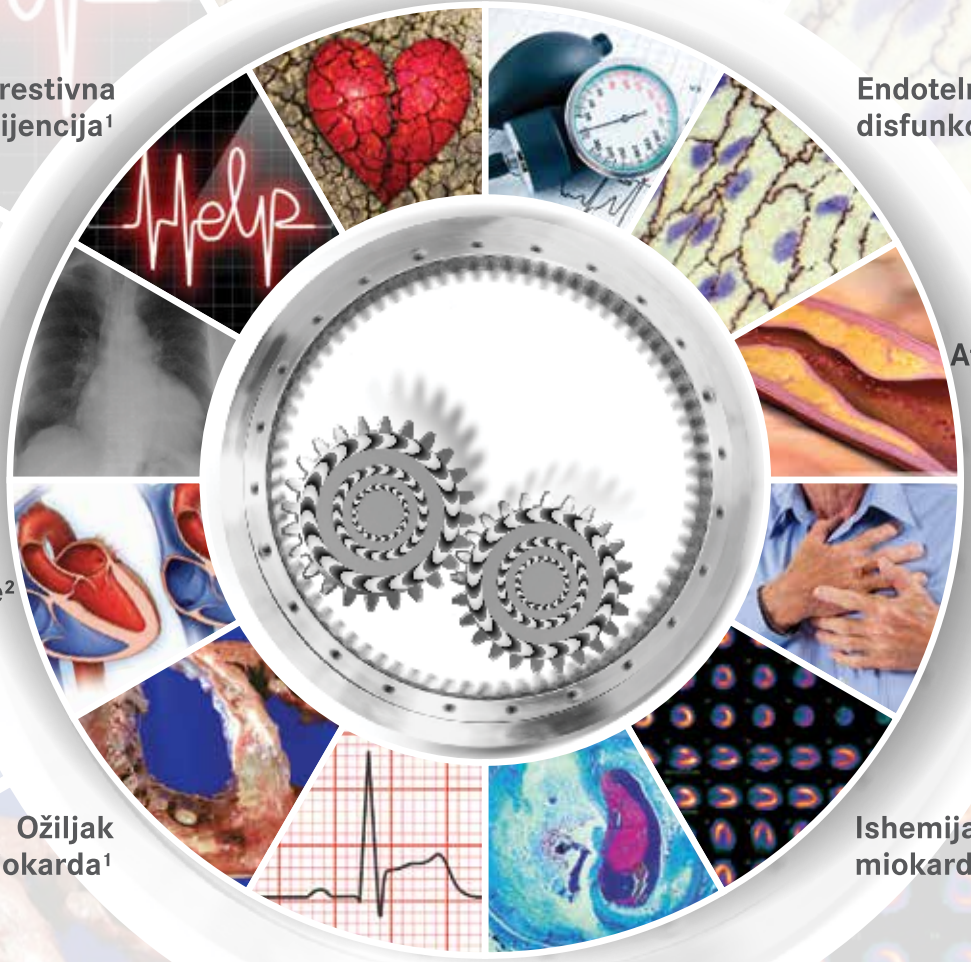

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Case report

Cardiac sarcoidosis detected with hybride imaging

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Abstract Sarcoidosis is a systemic granulomatous disease of unknown cause. Approximately one fourth of all patients with sarcoidosis have clinical manifestations of cardiac involvement. Granulomas may involve the pericardium, myocardium and endocardium of both the ventricles, as well as the atria. Cardiac involvement is associated with poor prognosis and is definitely confirmed with endomyocardial biopsy. Given the invasiveness and risks of the procedure, other alternative diagnostic methods are used. In diagnosis and clinical assessment of disease activity broad spectrum of analysis is used: clinical examination, laboratory analysis (ACE, chitotriosidase), multi-detector computer tomography (MDCT), hybrid imaging (positron emission tomography with computed tomography - PET/CT; positron emission tomography with magnetic resonance-PET/MR). In this case report we evaluate usefulness of hybrid imaging (FDG PET/CT) in detection of cardiac sarcoidosis.

Key words sarcoidosis, hybrid imaging

Introduction

Sarcoidosis is a systemic granulomatous disease of unknown cause. Approximately 25% of all patients with sarcoidosis have clinical manifestations of cardiac involvement, whereas myocardial granulomas can be identified in 25–79% of autopsy examinations[1,2]. Granulomas may involve the pericardium, myocardium and endocardium of both the ventricles as well as the atria[2].

In diagnosis and clinical assessment of disease activity broad spectrum of analysis is used: clinical examination, laboratory analysis (ACE, chitotriosidase), multi-detector computer tomography (MDCT), hybrid imaging (positron emission tomography with computed tomography- PET/CT; positron emission tomography with magnetic resonance-PET/MR).

Case report

A 59-year-old male complained of prolonged cough and shortness of breath. The patient had no previous history of smoking, alcohol abuse or past surgical experiences. After examination by a pulmonologist, MSCT of the lungs, determination of ACE and biopsy of nodular skin changes, the patient was diagnosed with sarcoidosis and treated with corticosteroids.

Five years after the diagnosis, and after intermittent corticosteroid therapy, the patient presented with exertional dyspnea, with mild limitation during ordinary physical activities. An electrocardiogram revealed a complete left bundle branch block and sinus tachycardia. Doppler echocardiography showed a severely dilated left ventricle (LV; 64 and 72 mm in systole and diastole, respectively), and a mildly dilated left atrium (41 mm). The LV ejection fraction (LVEF) was 30%. Carvedilol, losartan and furosemide were initiated, with clinical improvement.

In order to assess probable cardiac involvement in sarcoidosis, patient was sent to FDG PET/CT examination. FDG PET/CT examination was performed on a 64-slice hybrid PET/CT scanner (Biograph, Siemens Medical Solutions USA Inc.). Patient fasted for 8 hours before receiving an intravenous injection of 5.50 MBq of 18F-FDG per kilogram. PET/CT acquisitions started 60 min after tracer injection. A 3-dimensionalPET scan (6–7 fields of view-, 3 min/field, 30% overlap of beds, the bed width of ~16 cm) and low-dose non enhanced CT scan were acquired from the skull to the mid-thigh.

Low-dose CT, attenuation corrected PET, and combined PET/CT images were displayed for analysis on a Syngo Multi modality workplace (Siemens AG). “Positive” results were above the background level (blood pool and surrounding normal lung parenchyma and/or lymph node activity above the blood pool activity)[3,4]. Quantitative analysis of FDG uptake in the lesion was done based on the maximum standardized uptake value per focus (SUVmax). This value was calculated as the activity concentration measured at the end of the scan and corrected for individual body weight and dose injected as follows: tissue activity (counts/pixel/s) multiplied by the calibration factor and divided by an injected FDG dose (MBq/kilogram of bodyweight) [3,4]. FDG PET/CT examination revealed cardiac sarcoidosis in this patient, with focal pattern of uptake of FDG in myocardium, SUV max 5.8 (Figure 1).

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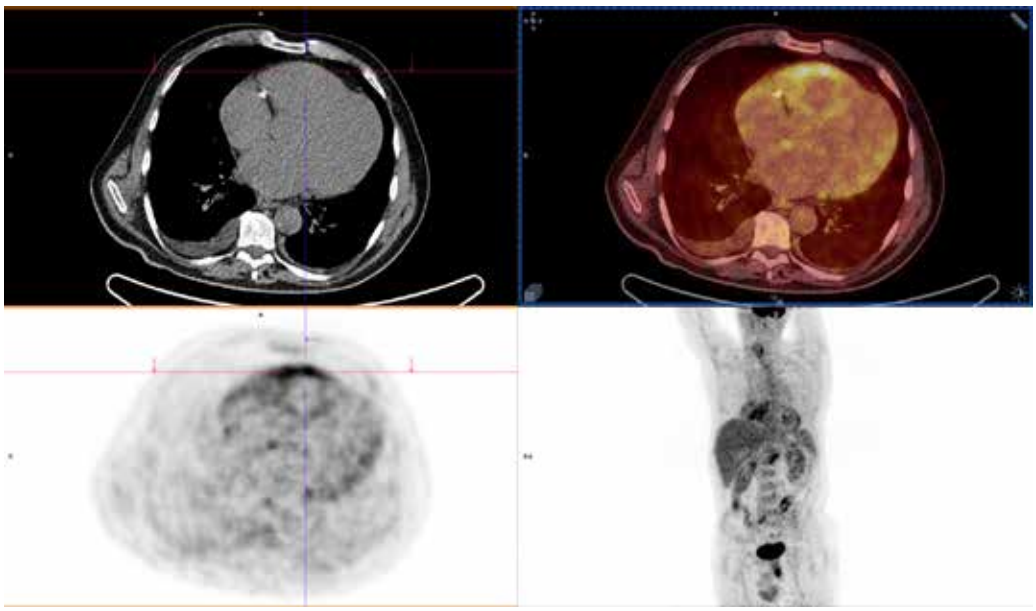

Figure 1. Cardiac sarcoidosis presented as focal FDG myocardial uptake on the FDG PET/CT examination

Discussion

Cardiac involvement in patients with sarcoidosis is being increasingly recognized and is associated with poor prognosis. Although environmental and genetic factors have been implicated in its pathogenesis, the etiology of cardiac sarcoidosis remains obscure.

The myocardium of the left ventricular free wall, particularly at the base of the heart, is most commonly affected, followed by the basal inter ventricular septum. Also, there appears to be a predilection for involvement of the conduction system[1,2,5]. Granulomas in the ventricular myocardium may lead to abnormal automaticity and re-entrant tachyarrhythmias manifesting as palpitations or syncope. Involvement of the conduction system may lead to bradyarrhythmias and syncope[5].

Congestive heart failure may result from widespread sarcoidosis of the myocardium with a decline in left ventricular ejection fraction and death[2].

Diagnosis of cardiac sarcoidosis remains challenging based on a broad and nonspecific set of signs and symptoms ranging from asymptomatic electrocardiographic findings to sudden death and progressively worsening heart failure and arrhythmias[1,5].

Cardiac sarcoidosis is definitely confirmed with endomyocardial biopsy[2,5]. Given the invasiveness and risks of the procedure, other alternative diagnostic methods are used.

Diagnosis and clinical assessment of disease activity usually implies clinical examination with laboratory analysis (ACE, chitotriosidase) and imaging procedures.

MDCT is the most available and consequently most used. However, molecular imaging such as FDG PET/CT or PET/MR are more informative.

The advantage of FDG PET/CT is providing information of precise anatomic localization of the disease and the functional changes at the cellular level. Additionally, the whole-body study protocol is especially important in order to evaluate the extent of this multisystem disease[1].

Hybrid imaging is more expensive than CT scan, but the higher expense is well balanced by its ability to demonstrate activity/inactivity in lesion, which is not possible with morphologic examinations. Radiation burden of a high resolution or a contrast-enhanced CT scan is accompanied by a greater radiation burden than the one necessary in the PET/CT scan[7]. When using a low dose, the mean effective dose was reduced from 8.10 to 5.50 mSv. The blinded analysis of the image quality showed no clinically significant degradation of the lower-dose studies[7].

Further studies using PET/MR may further decrease the radiation dose, and bring additional information about this pathology.

Taken together, for a definitive diagnosis and management all patients with cardiac sarcoidosis will likely benefit from an echocardiogram and either cardiac MR or FDG PET/CT or PET/MR[2,5]. FDG PET/CT imaging could be performed to establish baseline disease activity, assess need for initiation of medical therapy and monitor response to treatment over time. Alternatively, in patients with contraindication to performing MR, FDG PET/CT could be used as first line imaging.

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Sažetak

Otkrivanje sarkoidoze srca putem hibridnog imidžinga

Isidora Grozdić Milojević[1,2], Dragana Šobić Šaranović[1,2], Vera M. Artiko[1,2]

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Sarkoidoza je sistemska granulomatozna bolest nepoznatog uzroka. Otprilike jedna četvrtina svih pacijenata sa sarkoidozom ima kliničke manifestacije sarkoidoze srca. Granulomi mogu zahvatiti perikard, miokard i endokard obe komore, kao i pretkomore. Sarkoidoza srca je povezana sa lošom prognozom i definitivno se potvrđuje endomiokardijalnom biopsijom. S obzirom na invazivnost i rizike procedure, u dijagnostici srčane sarkoidoze koriste se druge alternativne dijagnostičke metode. U dijagnozi i kliničkoj proceni aktivnosti bolesti koristi se širok spektar analiza: klinički pregled, laboratorijske analize (ACE, hitotriosidaza), multidetektorska kompjuterizovana tomografija (MDCT), hibridne dijagnostičke procedure (pozitronska emisiona tomografija sa kompjuterskom tomografijom - PET / CT; pozitronska emisiona tomografija sa magnetnom rezonancom-PET / MR). U ovom prikazu slučaja biće prikazana korisnost FDG PET / CT u otkrivanju srčane sarkoidoze.

Ključne reči: sarkoidoza, hibridni imaging

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Case report

Double-kissing crush of the complex left main bifurcation lesions in a patient with acute coronary syndrome and cardiogenic shock

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Abstract Percutaneous coronary intervention of distal unprotected left main bifurcation lesions is technically very challenging, and associated with lower procedural success rate and an increased rate of major adverse cardiac and cerebrovascular events at short- and long-term follow-up, in comparison to coronary artery bypass graft surgery. Herein, we present in detailed a double kissing crush stenting of distal unprotected left main true and complex bifurcation lesion in 69-years old female patient with acute coronary syndrome and cardiogenic shock. Therefore, our patient needed urgent coronary angiography and „ad hoc" percutaneous coronary intervention of the „culprit" lesion due to life-threating condition. This article is focused on advantages of this 2-stent technique over other techniques in the treatment of the distal unprotected left main bifurcation lesions, and point out its limitations as well. Performing double kissing crush stenting in the distal unprotected left main true and complex bifurcation lesions is much more challenging than other stenting technique, but if it is performed by an experienced interventional cardiologist, the efficacy and safety of the procedure exceeds its potential disadvantages, which is of utmost importance for patient benefit.

Key words Acute coronary syndrome, Unprotected left main bifurcation lesion, Double kissing crush, Percutaneous coronary intervention

Introduction

Percutaneous coronary intervention (PCI) of distal unprotected left main (ULM) bifurcation lesions is technically very challenging, and associated with lower procedural success rate and an increased rate of major adverse cardiac and cerebrovascular events (MACCE) at short- and long-term follow-up, in comparison to coronary artery bypass graft surgery (CABG)[1-3]. There are several techniques for this type of lesion, including provisional stenting, T-stenting, T-stenting and small protrusion (TAP), Culotte and double-kissing (DK) Crush stenting[4-5]. Herein, we present a DK-Crush stenting of distal ULM true and complex bifurcation lesion in our patient with acute coronary syndrome and cardiogenic shock. We emphasize advantages of this 2-stent technique over other techniques in the treatment of the distal ULM bifurcation lesions, and point out its limitations as well.

Case Report

A 69-years old female patient was admitted to our hospital due to typical chest pain and dyspnea onset 2 hours ago and ischemic changes on ECG with ST elevation in leads aVR and V1, and horizontal and down-sloping ST depression in all other leads (Figure 1). The patient was hemodynamically unstable with arterial blood pressure (BP) 70/40 mmHg, and therefore, both vasopressor (norepinephrine 2-4 μg/min iv. infusion) and inotropic stimulation (dopamine 2-5 μg/kg/min iv. infusion) were given. The patient was unconscious, intubated, and on mechanical ventilation. Echocardiography findings showed impaired left ventricle function, with ejection fraction (EF) 30%, and hypo-akinesia of medial and apical segments of interventricular septum and anterior wall, and hypokinesia of both lateral and inferior wall. The patients had several comorbidities: arterial hypertension, hyperlipoproteinemia, and poorly regulated insulin-dependent diabetes mellitus (>20 years) with glycated hemoglobin (HbA1C) 7.8%. In the Coronary Care Unit (CCU), the patient was treated with loading dose of dual antiplatelet therapy (aspirin 300 mg and ticagrelor 180 mg) through a nasogastric tube. Due to clinical and ECG signs of acute coronary syndrome (ACS) and hemodynamic instability, the patient was planned for urgent coronary angiography and PCI.
Initial coronary angiogram was performed with the femoral artery approach using standard Judkins tech-

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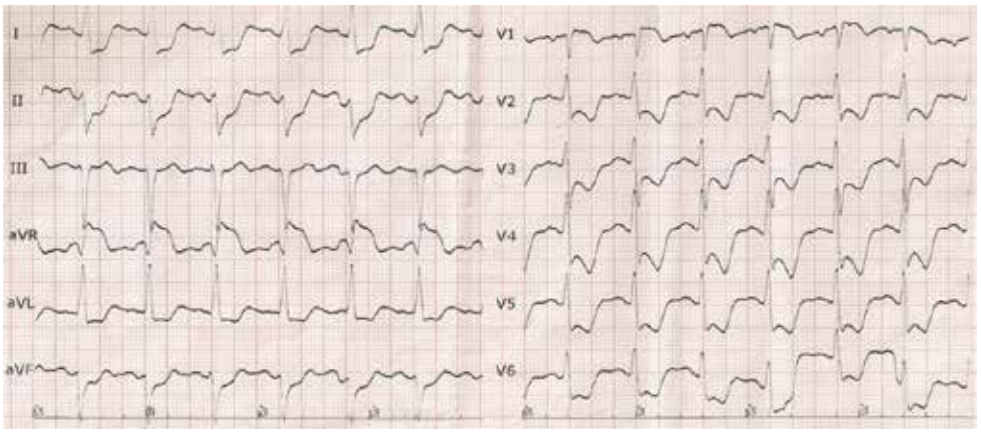

Figure 1. ECG on admission to Coronary Care Unit (CCU): Note the elevation of ST segment of 5.0 mm in lead aVR and 2.0 mm in lead V1 (STaVR > STV1), and horizontally and downsloping depression of ST segment with negative T waves in all other leads. These ECG changes implicates that the culprit lesion is located in the left main (LM), and the patient is at very high-risk for intrahospital mortality due to high amplitude od ST elevation in lead aVR.

nique with 7-French catheters. The culprit lesion was a true bifurcation lesion located in the distal segment of left main (LM) and ostial and proximal segments of both left circumflex artery (LCx) and left anterior descending artery (LAD), with Thrombolysis in Myocardial Infarction (TIMI) 2 flow in both arteries (Figure 2.A). Also, there was a significant stenosis in the medial segment of LAD and distal segment of LCx, as well as in the proximal and medial segments of right coronary artery (RCA) (Figure 2.B,C).

Analyzing the lesion complexity, we emphasized several key characteristics according to EBC consensus document[4,5], which defined our stenting strategy:

1. This was a true bifurcation lesion: 1,1,1 according to Medina classification;
2. The LCx reference diameter was >2.5 mm and it was around 3.0 mm, but it was smaller than LM diameter;
3. There was a significant stenosis at the LCx ostium extending more than 5 mm into the artery;
4. The angle between LAD and LCx was >70 degree (around 90 degree); and
5. Because of significant and long ostial stenosis, we assumed that it would be difficult to access the LCx, and that there was a high-risk of LCx compromise if we first stent the LM towards the LAD.

Therefore, our decision was to perform DK-Crush technique. Culotte stenting was not our first choice due to significant mismatch between the LM and the LCx reference diameters and wide bifurcation angle (>70 degree). After wiring both LAD and LCx with Asahi Sion Blue (Asa-

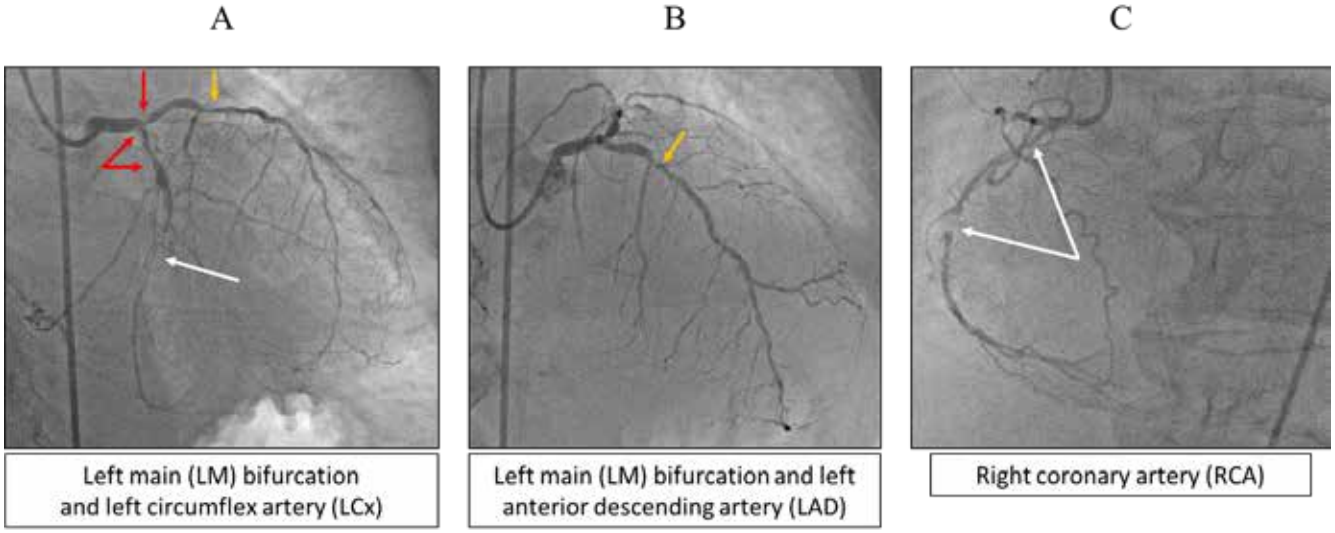

Figure 2. Initial coronary angiogram. A. Postero-anterior (PA) projection with caudal 20 degree angulation shows that the culprit lesion was a true bifurcation lesion located in the distal segment of left main (LM) and ostial and proximal segments of both left circumflex artery (LCx) and left anterior descending artery (LAD), with Thrombolysis in Myocardial Infarction (TIMI) 2 flow in both arteries (red arrows); B. PA projection with cranial 40 degree angulation shows a significant stenosis in the medial segment of LAD (yellow arrow), as well as in the distal segment of LCx (white arrow); and C. Left anterior oblique (LAO) projection 30 degree shows a significant stenosis in the proximal and medial segments of right coronary artery (RCA) (white arrows)

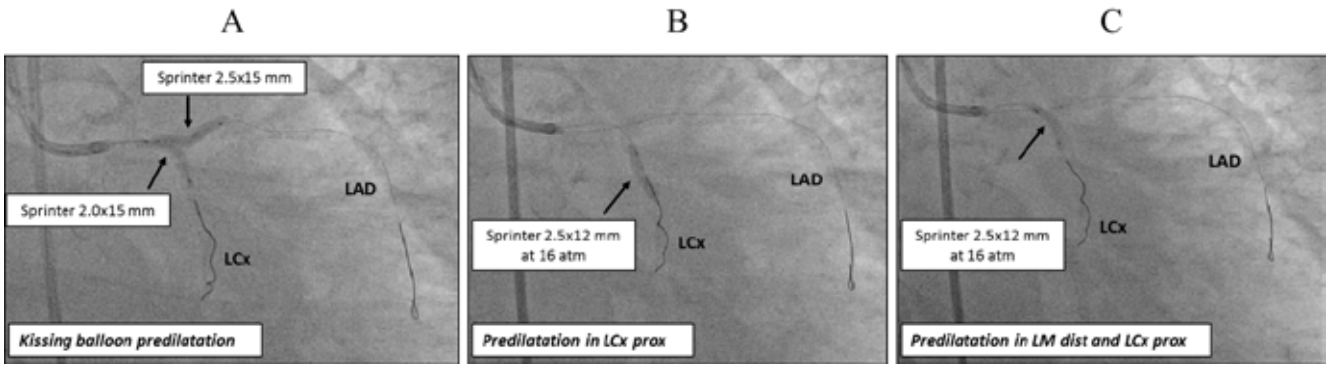

Figure 3. Balloon predilatations of the left main (LM), left anterior descending artery (LAD) and left circumflex artery (LCx). A. Kissing balloon predilatation of the LM bifurcation with 2 semi-compliant (SC) balloons; B. Predilatation in the proximal segment of LCx with SC balloon; C. Predilatation in the ostial segment of LCx and LM with SC balloon.

hi Intecc), kissing balloon predilatation of the LM bifurcation were performed first, with 2 semi-compliant (SC) balloons: Sprinter (Medtronic) 2.5x15 mm in LM-LAD at 14 atm and Sprinter (Medtronic) 2.0x15 mm in LM-LCx at 16 atm (Figure 3A). Additionally, multiple predilatations of LCx were performed in its ostial and proximal segments with SC balloon Sprinter (Medtronic) 2.5x12 mm at 16 atm (Figure 3.B,C). Thrombolysis in myocardial infarction (TIMI) 3 flow was achieved, and arterial BP was increased up to 85/50 mmHg. After that, stenting of the LCx with minimal protrusion in the LM was performed (Figure 4.A,B). Figure 4A shows positioning of the stent in LCx, and in the same time positioning of the SC balloon Sprinter (Medtronic) 3.5x20 mm in the LM towards the LAD. Figure 4B shows implantation of the stent Synergy (Boston Scientific) 3.0x20 mm at 16 atm in the LCx with minimal protrusion in the LM. The next step was removing guidewire and balloon from the LCx, and after that, the SC balloon Sprinter (Medtronic) 3.5x20 mm was inflated in the LM-LAD at 14 atm crushing the proximal part of the stent in the LM (Figure 4C). Additionally, the proximal optimization (POT)-crush in the LM with the same SC balloon at 20 atm was also performed (Figure 4D).

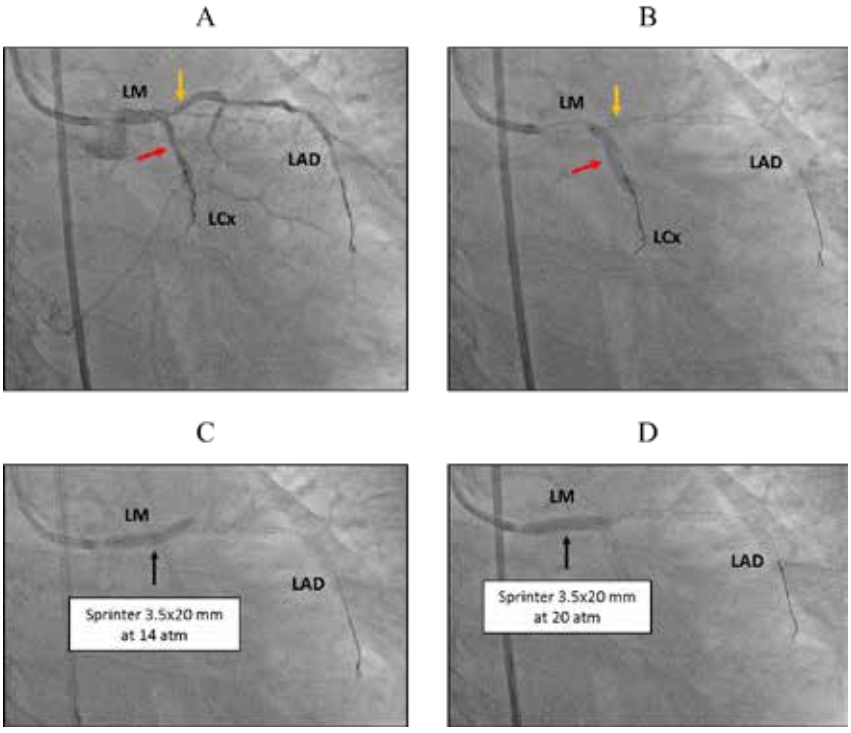

Figure 4. A. Positioning of the stent Synergy (Boston Scientific) 3.0x20 mm (red arrow) in the left circumflex artery (LCx) with minimal protrusion in the left main (LM), and in the same time positioning of the uninflated semi-compliant (SC) balloon Sprinter (Medtronic) 3.5x20 mm (yellow arrow) in the LM towards the left anterior descending artery (LAD); B. Implantation of the stent Synergy (Boston Scientific) 3.0x20 mm at 16 atm (red arrow) in the LCx with minimal protrusion in the LM. The uninflated SC balloon Sprinter (Medtronic) 3.5x20 mm (yellow arrow) is located in the LM towards the LAD; C. After removing guidewire and balloon from the LCx, the crush of the proximal part of the implanted stent in the LM was performed with SC balloon Sprinter (Medtronic) 3.5x20 mm in the LM-LAD at 14 atm; and D. The proximal optimization (POT)-crush in the LM was performed with the same SC balloon at 20 atm.

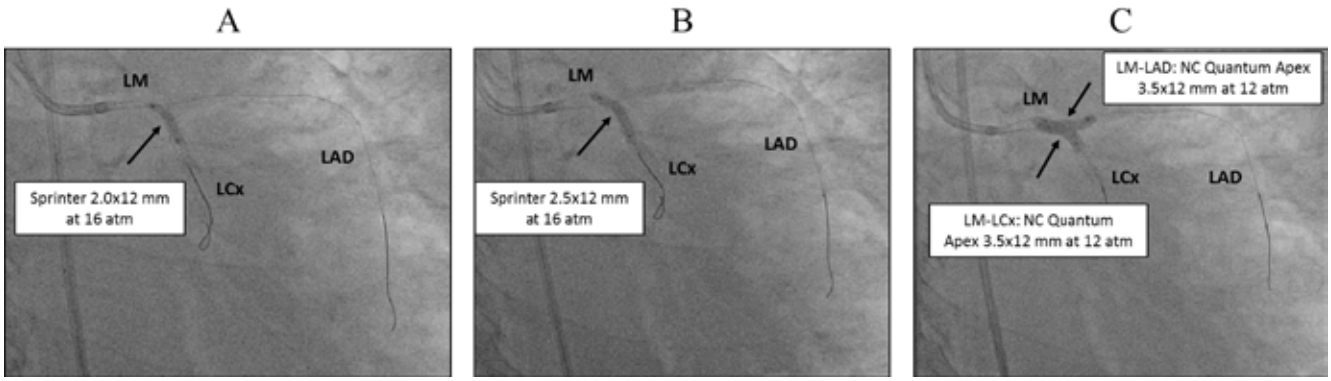

Figure 5. A. and B. After rewiring of the left circumflex artery (LCx) through the proximal strut of the crushed stent, LCx strut dilatation was performed with semi-compliant (SC) balloons Sprinter (Medtronic) 2.0x12 mm and Sprinter (Medtronic) 2.5x12 mm, both at 16 atm; C. First kissing balloon inflation (KBI) was performed with 2 non-compliant (NC) balloons: NC Quantum Apex (Boston Scientific) 3.5x12 mm in LM-LAD at 12 atm and NC Quantum Apex (Boston Scientific) 3.5x12 mm in LM-LCx at 12 atm. LM - left main; LAD - left anterior descending artery

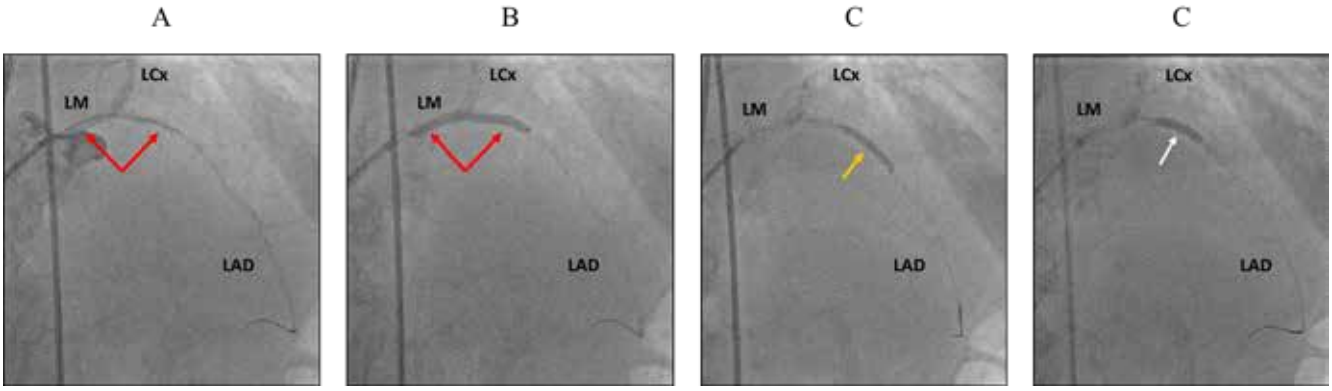

Figure 6. A. Positioning of the stent CID (Alvimedica) 3.5x38 mm (red arrows) from the left main (LM) ostium towards the proximal segment of left anterior descending artery (LAD), after removing the guidewire from the LCx; B. Implantation of the stent CID (Alvimedica) 3.5x38 mm (red arrows) from the LM ostium towards the proximal segment of LAD at 14 atm; C. Another stent Promus Premier (Boston Scientific) 3.0x20 mm (yellow arrow) was implanted in the medial segment of LAD at 14 atm; and D. Postdilatation between two stents in the LAD with non-compliant (NC) balloon Quantum Apex (Boston Scientific) 3.5x12 mm at 16 atm (white arrow). LCx - left circumflex artery

After rewiring of the LCx through the proximal strut of the crushed stent with Asahi Sion Blue (Asahi Intecc), LCx strut dilatation was performed with SC balloons Sprinter (Medtronic) 2.0x12 mm and Sprinter (Medtronic) 2.5x12 mm, both at 16 atm (Figure 5.A,B). First kissing balloon inflation (KBI) was performed with 2 non-compliant (NC) balloons: NC Quantum Apex (Boston Scientific) 3.5x12 mm in LM-LAD at 12 atm and NC Quantum Apex (Boston Scientific) 3.5x12 mm in LM-LCx at 12 atm (Figure 5C). Then, after removing the guidewire and balloon from the LCx, stent CID (Alvimedica) 3.5x38 mm was implanted from the LM ostium towards the proximal segment of LAD at 14 atm (Figure 6.A,B). Additionally, another stent Promus Premier (Boston Scientific) 3.0x20 mm was implanted in the medial segment of LAD at 14 atm, and postdilatation between two stents in the LAD with NC balloon Quantum Apex (Boston Scientific) 3.5x12 mm at 16 atm was performed (Figure 6.C,D).

After second rewiring with Asahi Sion Blue (Asahi Intecc) of the LCx through the distal strut of the crushed stent, final KBI was performed in the same way as the first one with the higher pressure in the balloon located in the LCx atm (Figure 7A). After that, the POT with NC Quantum Apex (Boston Scientific) 4.5x12 mm at 14 atm, and additionally flaring with the same NC balloon at 20 atm were performed in the ostial segment of LM, in order to improve optimal stent expansion and strut apposition in the artery (Figure 7.B,C).

Figure 8 shows the final angiogram with good result and TIMI 3 flow in both LAD and LCx arteries. Arterial BP was increased up to 110/70 mmHg immediately after PCI, with vasopressor (norepinephrine 2-4 µg/min iv. infusion) and without inotropic stimulation for the next several days. Maximal value of high-sensitive troponin T was 9372 ng/L, while brain natriuretic peptide was >5000 pg/ml.

However, 400 ml of contrast agent was used for the intervention in this patient with cardiogenic shock, and therefore, contrast-induced nephropathy occurred. Seventy-two hours after PCI, serum creatinine level increased from 86 up to 272 µmol/L, while estimated glomerular filtration rate (eGFR) decreased from 58 up to 15 ml/min/1.73 m^2. The patient was received 6 hemodialysis for the next 6 days. Fourteen days after PCI, serum creatinine level decreased up to 108 µmol/L, while eGFR increased up to 48 ml/min/1.73 m^2. The patient was hemodynamically stable, with arterial BP of 110/70 mmHg, without

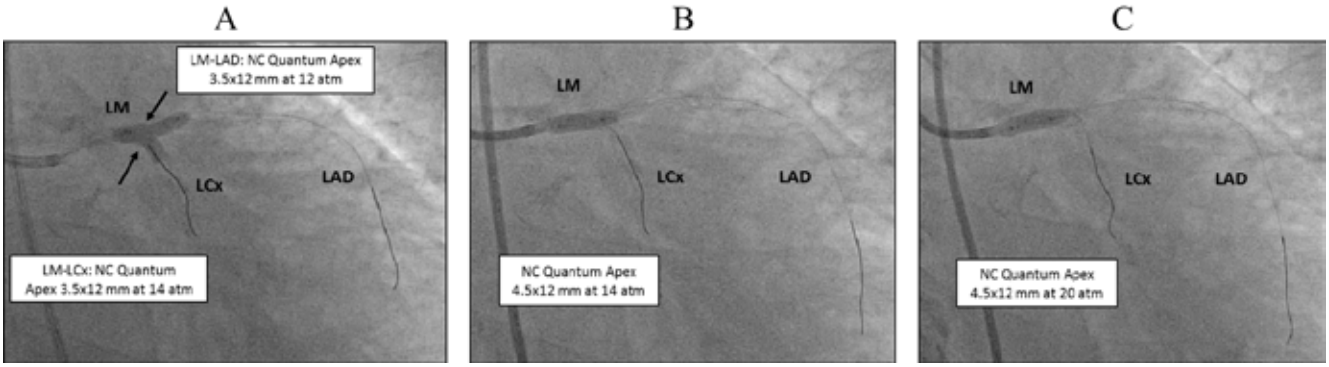

Figure 7. A. Final kissing balloon inflation (KBI) was performed with 2 non-compliant (NC) balloons: NC Quantum Apex (Boston Scientific) 3.5x12 mm in LM-LAD at 12 atm and NC Quantum Apex (Boston Scientific) 3.5x12 mm in LM-LCx at 14 atm; B. The proximal optimization (POT) in the LM with NC Quantum Apex (Boston Scientific) 4.5x12 mm at 14 atm; and C. Flaring with the same NC balloon at 20 atm were performed in the ostial segment of LM, in order to improve optimal stent expansion and strut apposition in the artery. LM - left main; LAD - left anterior descending artery; LCx - left circumflex artery

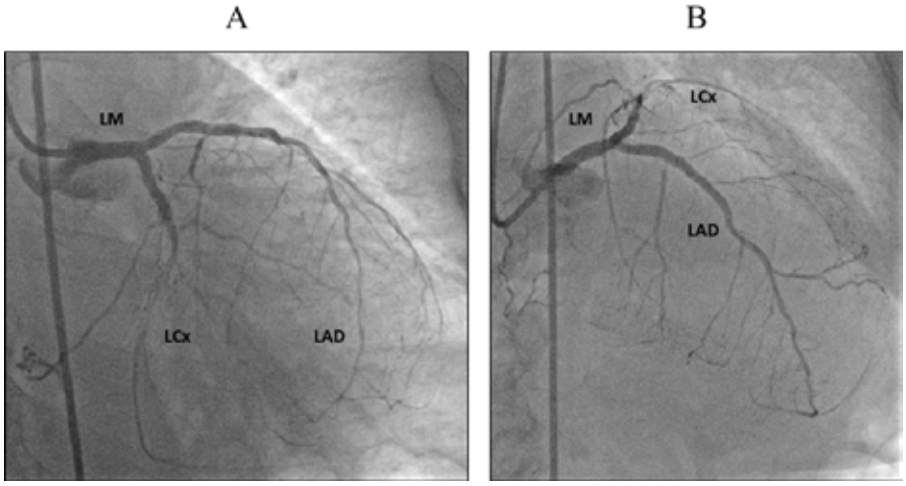

Figure 8. Final coronary angiogram shows good result and TIMI 3 flow in both left anterior descending (LAD) and left circumplex (LCx) arteries. A. Postero-anterior (PA) projection with caudal 20 degree angulation; and B. PA projection with cranial 40 degree angulation. Significant stenosis in the distal segment of LCx, as well as in the proximal and medial segments of right coronary artery (RCA) did not treat during index procedure. LM – left main

vasopressor and inotropic stimulation. Therefore, she was discharged from our hospital, and planned for outpatient visit 1 month later.

Discussion

The present case demonstrates detailed approach to the DK-Crush technique for stenting of distal ULM true bifurcation lesion in hemodynamically unstable patient with acute coronary syndrome and cardiogenic shock. Therefore, our patient needed urgent coronary angiography and „ad hoc" PCI of the „culprit" lesion due to life-threating condition. Current European Society of Cardiology (ESC) guidelines recommend PCI as an alternative to CABG surgery in selected patients with ULM coronary artery disease[6]. Previous studies and registries have been demonstrated that 1-stent strategy with drug-eluting stent (DES) placement (especially new-generation DES) for ostial or body/shaft lesions of the ULM was non-inferior in comparison to CABG in terms of long-term MACCE[7-12]. However, the majority of patients with ULM coronary artery disease have involvement of the distal bifurcation, which is associated with worse long-term outcomes after stenting compared with ostial/shaft lesions treatment[8,13]. In DELTA-2 registry, 85% of patients had involvement of the distal ULM bifurcation which was treated with provisional stenting in majority of cases, while 2-stent strategy was performed in only 20% of them[6]. In this registry, PCI of ULM was associated with lower risk for cerebrovascular events, but with higher risk for target vessel revascularization (TVR), in comparison to CABG at long-term follow-up[9]. However, there were no differences in death and myocardial infarction (MI) between PCI DELTA 2 cohort and CABG DELTA 1 cohort[8-9]. Similarly, in the recent EXCEL (Evaluation of XIENCE Versus Coronary Artery Bypass Surgery for Effectiveness of Left Main Revascularization) trial, 80% of patients had distal ULM bifurcation lesion with low or intermediate SYNTAX (Synergy Between Percutaneous Coronary Intervention With Taxus and Cardiac Surgery) scores ($\leq$32), which were also treated most commonly with provisional stenting using new-generation DES (everolimus-eluting stent, XIENCE, Abbott Vascular)[12]. In this trial, PCI of ULM with

new-generation DES was non-inferior to CABG with respect to composite rate of death, MI or stroke at long-term follow-up, but TVR rate at 30 days was higher with PCI[12]. Contrary, in the NOBLE (Nordic-Baltic-British Left Main Revascularization Study) trial, in which around 80% of patients also had distal ULM bifurcation lesion and treated with provisional stenting using first-generation DES, PCI of ULM was associated with a higher composite rate of death, MI, stroke, or repeat revascularization at 5 years than CABG[11]. Therefore, PCI of distal ULM bifurcation lesion remains a matter of debate[14].

Although provisional stenting is currently recommended for most non-LM bifurcation lesions, a recent study by Chen et al. showed a worse outcome at 1-year follow-up with this 1-stent strategy of distal ULM true bifurcation lesion compared with DK-Crush 2-stent strategy[13,15-19]. This result was driven by higher rates of target vSessel MI and definite or probable stent thrombosis (ST), as well as rates of target lesion revascularization (TLR), especially at the LCx ostium, in the provisional group compared with DK-Crush group[13]. Similar findings were observed in the DKCRUSH-III trial in which DK-Crush stenting was superior to another 2-stent Culotte strategy in the treatment of distal ULM true bifurcation lesions, also driven by lower rates of definite or probable ST, as well as rates of TVR and TLR, especially at the LCx ostium, as in DK-CRUSH V trial[15,20]. This increased 3-year rate of ST in the Culotte group led to a more frequent MI and cardiac deaths, especially in patients with complex distal ULM bifurcation lesion with wide bifurcation angle (≥70%), and SYNTAX score ≥23[20-21]. There are several reasons for the superiority of DK-Crush over both provisional and Culotte stenting in the treatment of distal ULM true bifurcation lesions. First, DK-Crush with first KBI immediately after balloon crush provides better stent expansion at the LCx ostium[13,15,20]. Second, final KBI and POT with NC balloon at high pressure during DK-Crush stenting further improves stent expansion and strut apposition at both LAD and LCx ostium, as well as in the ULM[15,20]. Therefore, DK-Crush ensures fully coverage of the LCx ostium with less metal overlap and stent distortion, resulting in lower rates of in-stent restenosis, TLR and TVR, as well as ST and target vessel MI, in comparison to Culotte stenting[15,20]. Additionally, it has been demonstrated that DK-Crush stenting of distal ULM bifurcation lesion with wide angle ≥70% was associated with much better efficacy in terms of MACCE and better safety in terms of ST, compared with Culotte stenting[15,20]. Regarding provisional approach of the distal ULM true bifurcation lesion, bail-out LCx stenting through ULM stent cells may be difficult or result in imprecise placement, incomplete stent expansion and strut apposition, or edge dissection[13]. According to previous findings, the European Society of Cardiology (ESC) recommends DK-Crush stenting as a preferred strategy for the treatment of distal ULM true bifurcation lesion (Class of Recommendation IIB, Level of Evidence B)[6].

However, DK-Crush is a very complex technique with many steps, and therefore, requires additional procedural time and large amount of contrast dose usage[22]. This could be very unfavorable in patients with cardiogenic shock due to renal hypoperfusion resulting in higher risk for contrast-induced nephropathy which is a well-known strong predictor of in-hospital morbidity and mortality[23]. Therefore, it is necessary to take into account both benefit and risk for DK-Crush stenting in these patients, and this benefit-risk ratio largely depends on the operator skill and experience[22].

Performing DK-Crush in distal ULM true and complex bifurcation lesions is much more challenging than Culotte and/or provisional stenting, but if the PCI is performed by an experienced interventional cardiologist, the efficacy and safety of the procedure exceeds its potential disadvantages, which is of utmost importance for patient benefit.

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Sažetak

"Double-Kissing Crush" tehnika kompleksne bifurkacione lezije na glavnom stablu leve koronarne arterije kod pacijenta sa akutnim koronarnim sindromom i kardiogenim šokom

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Perkutana koronarna intervencija bifurkacione lezije glavnog stabla leve koronarne arterije je tehnički veoma izazovna i zahtevna procedura, i udružena je sa manjim periproceduralnim uspehom i većom učestalošću neželjenih kardiovaskularnih i cerebrovaskularnih događaja tokom kratkoročnog i dugoročnog perioda praćenja, u poredjenju sa aorto-koronarnom bajpas operacijom srca. U ovom radu detaljno objašnjavamo "double kissing crush" stent tehniku lečenja prave i kompleksne bifurkacione lezije glavnog stabla leve koronarne arterije kod 69-godišnje bolesnice sa akutnim koronarnim sindromom koji je komplikovan kardiogenim šokom. Zbog toga je našoj bolesnici bila potrebna hitna koronarna angiografija i „ad hoc" perkutana koronarna intervencija lezije odgovorne za nastanak ovog životno-ugrožavajućeg stanja. Ovaj rad je fokusiran na prednosti "double kissing crush" stent tehnike u odnosu na druge tehnike u lečenju bifurkacione lezije glavnog stabla leve koronarne arterije, ali takodje ukazuje i na njena postojeća ograničenja. Izvođenje ove stent tehnike u lečenju prave i kompleksne bifurkacione lezije glavnog stabla leve koronarne arterije mnogo je zahtevnije od ostalih tehnika stentiranja, ali ako je izvodi iskusan interventni kardiolog, efikasnost i bezbednost ove tehnike prevazilazi njene potencijalne nedostatke, što je od najveće važnosti za dobrobit pacijenta.

***Ključne reči**: Akutni koronarni sindrom, Bifurkaciona lezija glavnog stabla, Double kissing crush stent tehnika, Perkutana koronarna intervencija*

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Prikaz slučaja

Značaj rane dijanoze urođenih trombofijija za prevenciju trombotskih dogadaja

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Sažetak Infarkt miokarda pre 65 godine u žena, predstavlja ranu koronarnu bolest. Odsustvo konvencionalnih faktora rizika za kardiovaskularne bolesti u pacijenata sa trombotičnim dogadajima usmerava ispitivanje u parvcu stečenih trombofilija. Isključivanjem mogućnosti stečenih trombolfija, ispitivanje treba sprovesti na prisutnost urođenih trombofilja, radi njihove što ranije dijagnoze i adekvatne terapije radi sprečavanja trombotskih događaja kako u arterijskom tako i venskom vaskularnom koritu.

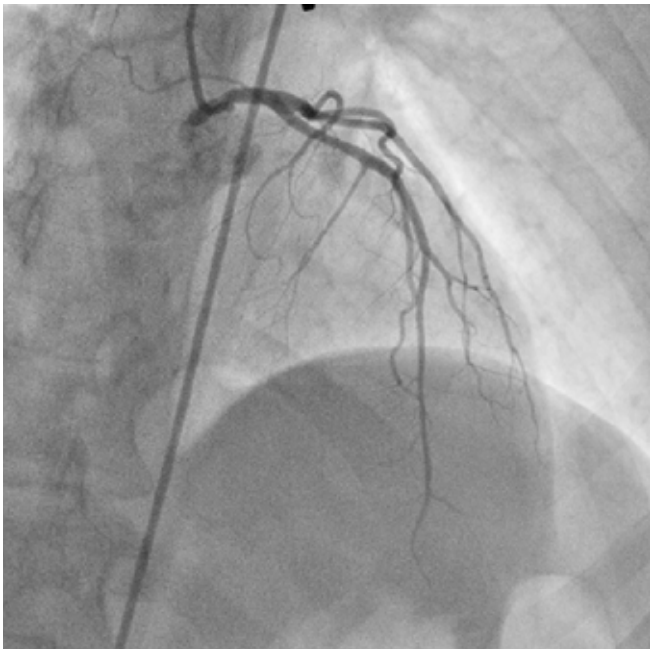
Ključne reči: infarkt miokarda sa ST elevacijom, urođena trombofilija

Uvod

Pojava akutnog infarkta miokarda u mlađoj životnoj dobi, pre 45 godine je retka. Ateroskleroza u tim slučajevima nije glavni patofiziološki mehanizam nastanka bolesti, ukoliko nema nekog ekstremno povišenog konvencionalnog faktora rizika za kardiovaskularne bolesti (KVB), kao kod familijarne hiperlipoproteinemije[1,2]. Treba tragati za drugim uzrocima infarkta miokarda: zloupotreba droga (naročito kokaina), pojava hiperkoagulabilnih stanja (naslednih trombofilija), embolijskih fenomena, endokarditisa, i dr[2,3]. Naravno, ne sme se zaboraviti ni aditivni učinak svih pomenutih faktora i stanja. Kardiovaskularne bolesti su multifaktorijalne, njihovi klinički entiteti su koronarna bolest, moždani udar, periferna arterijska bolest.

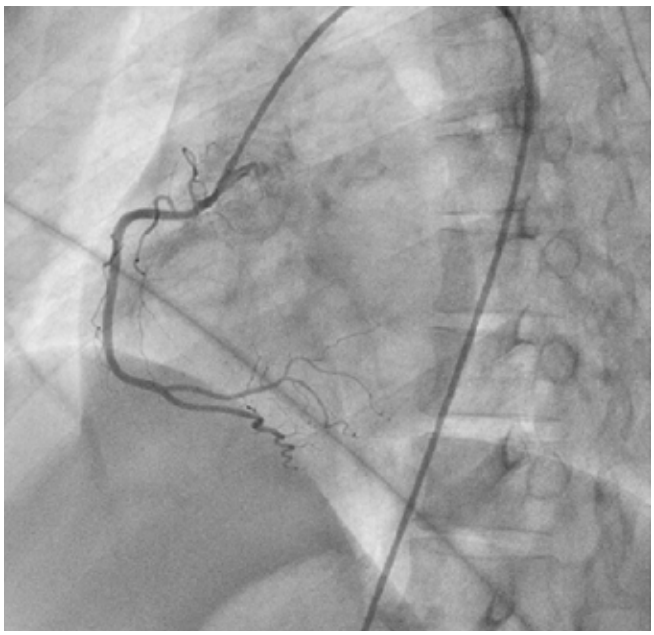
Prikaz bolesnika

Pacijentkinja stara 45 godina, majka dvoje dece, sa tipičnom kliničkom slikom i elektrokardiogrfskom prezentacijom STEMI infarkta anterolateralne lokalizacije, primljena u koronarnu jedinicu, 1 čas od početka bola u grudima. U elektrokardiogramu je registrovana elevacija ST spojnice za 3-5 mm u D1, aVL, V5, V6, te su prisutni q-zupci u V1-3 i ST depresija 3-5mm u inferiornim odvodima (slika u ogledalu)(Slika 1). U biohemijskom nalazu značajne su bile vrednosti TnI od 1.9 ng/mL po dolasku i 50 ng/mL nakon 12h sati (što je ujedno i maksimalno izmerena vrednost). Ukupni holesterol iznosio je 4,11 mmol/L, LDL 2,6 mmol/L, HDL 1.1 mmol/L, trigliceridi 0.9 mmol/L, glikemija 4,8 mmol/L. Ostali biohemijski parametri su bili u fiziološkim granicama. Zbog nedostupnosti katerizacione laboratorije u datom momentu, primenjena je farmakološka reperfuziona terapija selektivnim trombolitikom alteplazom. U toku 60 minuta od početka primene terapije dolazi do prestanka bola u grudima potom rezolucije ST elevacija za više od 50% u odnosu na početnu vrednost. Na rutinskoj ranoj koronarografiji (5h od primene fibrinolitičke terapije) epikardne koronarane arterije su bile bez vidljivih suženja sa TIMI III protokom i bez vidljivih anomalija (Slika 2 i 3). Ehokardiogram pokazuje samnjenu ejekcionu funkciju leve komore, EF 48%, FS 26%, sa segmetnim poremećajem kinetike po tipu akinezije apikalnog segmenta septuma i apikomedijalnog segmenta anteriornog zida. Color Doppler krvnih sudova vrata bez znakova ateroskleroze karotidnih arterija. Test na urođenu trombofiliju pokazuje rezitenciju na protein C.



Slika 2. Desna koronarna arterija bez suženja

Adresa za korepsondenciju: Gabrijela Stojković, Opšta bolnica Leskovac, E-mail: gabikardio@gmail.com



Slika 3. Leva koronarna arterija bez suženja i glatkih ivica

Diskusija

Infarkt miokarda pre 65 godine u žena, predstavlja ranu koronarnu bolest koja ce se javlja u grupi vrlo visokorizičnih i visokorizičnih pacijenata za KVB[2]. Međutim, bolesnica je bila bez ijednog glavnog faktora rizika za KVB (pušenje, arterijska hipertenzija, dislipidemija, dijabates melitus), sa normalnom bubrežnom funkcijom samim tim i niskim rizikom za ubrzanu aterosklerozu, što je potvrđeno kolor Dopplerom krvnih sudova vrata (screening testom rane ateroskleroze u mladih osoba) koji je takođe bio bez znakova ateroskleoroze. Nije bilo stečenih trombofilnih stanja (gojaznost, smanjena fizika aktivnist, vožnja avionom duža od 5h i kontraceptivna terapija, poremećaj menstrualnog cikusa), i bez dijagnostikovane autoimune bolesti, i bez bilo kakvog simptoma ili znaka ovih oboljenja. Na taj način isključena je mogućnost sekundrane trombofilije koja bi bila odgovorna za nastanak akutnog infarkta miokrda. Sa druge strane pozitivna porodična anamneza rane arterijske tromboze, (otac preboleo moždani udar u pedesetoj godini života) predstavlja snažan modifikator rizika sa reklasifikacionim potencijalom svrstavajući je u grupu visokog rizika za kardiovaskularne događaje**.** Podatak duboke venske tromboze u ličnoj anamnezi pre 6 godina, je sugesisao na opštu sklonost ka trombozi – trombofiliji[4]. Jasna porodična anamneza rane tromboze i ponovljene tromboze u mlađoj životnoj dobi indikuju test na urođene trombofiliju. Test je pokazao rezistenciju aktiviranog faktora VIII (FVIIIa) na aktivirani protein C (APC). Protein C je najvažniji sistem sa antikoagulantnim i fibrinoliznim potencijalom. Aktivirani protein C rezistencija (APC) je najčešća nasledna trombofilija, koja je prisutna kod 20-40% svih pacijenata sa dubokom venskom trombozom[4,5]. Ovaj defekt je prisutan u oko 4-5% zdravih individua belaca, mada se u nekim sredinama ovaj procenat kreće i do 15%[3-5]. Neophodno je naglasiti da negativan test u pravcu trombofilije ne isključuje njeno postojanje. Testiranje obuhvata samo trombofilije koje su nam u ovom trenutku poznate4). Akutnu trombozu kod ovih bolesnika treba lečiti heparinom i oralnom antikoagulantnom terapijom (OAT). Profilaktična antikoagulantna terapija primenjuje se kod homozigota i heterozigota sa rezistencijom na aktivni protein C i kod bolesnika sa dva ili više trombotska dogadaja koji ukazuje na povećan rizik od tromboza (5,6). Trajna oralna antikoagulantna terapija inhibitorom vitamin K, neophodna je kako bi se prevenirali trombotski događaji kako u arterijskom tako i u venskom koritu (6). Pozitivna porodična anamneza ranih trombotskih događaja ne sme ostati neispitana i prepuštana terapiji slučaja. Testiranje na urođene trombofilije u slučajevima neočekivane koronarne bolesti u mlađem životnom dobu, predstavlja važan protokol u prevenciji neželjenih trombotskih dogadjaja. U slučaju dokumentovane nasledne trombofilije linijski srodnici treba da budu pregledani i /ili praćeni i testirani.

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Abstract

The role of early diagnosis of inherited thrombophilia in prevention of thrombotic events

Gabrijela Stojković[1], Dejan Đokić[1], Jelena Stojanović[1], Olivera Božić[1], Miodrag Miladinović[1], Aleksandar Stanković[1], Ana Đorđević Dikić[2,3], Branko Beleslin[2,3]

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Myocardial infarction before the age of 65 in women is considered as an early manifestation of coronary artery disease. The absence of conventional risk factors for cardiovascular diseases in patients with thrombotic events may suggest acquired thrombophilia. Excluding the possibility of acquired thrombophilia, the examination should be performed for the presence of inherited thrombophilia, in order to prevent thrombotic events in both the arterial and venous vascular bed by early diagnosis and adequate therapy.

Key words: *myocardial infarction with ST-elevation, inherited thrombophilia*

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Review article

Electrocardiographic changes in patients with hypertrophic cardiomyopathy: Relevance for athletic screening

Vojislav Giga, Milorad Tešić

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Abstract Hypertrophic cardiomyopathy is one of the most prevalent inherited cardimyopathies and according to the US data is the leading cause of sudden cardiac death in athletes. Athletes with HCM are asymptomatic in majority of cases and have normal physical phindings, however over 90% of them have abnormal ECG, so it is important to recognize these ECG patterns.

Key words hypertrophic cardiomyopathy, electrocardiogram, athletes

Hypertrophic cardiomyopathy (HCM) is one of the most prevalent inherited cardiomyopathies in general population with an incidence of 1:500[1], that is responsible for 5% of all sudden cardiac deaths (SCD)[2]. In elite athletes the prevalence of the disease is significantly lower 1:1500[3], however according to US data it is responsible for more than 1/3 of all SCD in this population[4]. HCM was cause of death in 11% of British atheltes[5]. Overall, these data underscore the importance of screening for HCM in athletes.

Athletic screening comprises of detailed history, including history of sudden cardiac death in family members, physical examination and 12 leads electrocardiogram (ECG). Sympotms suggestive of HCM are fatique, dyspnea, anginal chest pain and syncope. However, in some patients, the first presentation of the disease is SCD.

Typical physical finding of systolic murmer over left sternal border is present in only 21% of patients with known disease (4). On the other hand over 90% of patients with HCM have pathological ECG.

Electrocardiographic changes in patients with hypertrophic cardiomyopathy

According to the Italian experience, introduction of 12-lead ECG in athletic screening resulted in significant decrease in SCD in this population, predominantly due to the identification of patients with congenital cardiomyopathies and channelopathies.

Correct interpretation of ECG enables identification of the athletes with risk of SCD. However, physicians should be familiar with sport related ECG changes (bradicardia, first degree AV block, etc) in order to avoid further unnecessery investigations.

First official guidelines for ECG interpreation in athletes were published by European Society of Cardiology in 2010[7]. Due to the large amount of published data in the field and expansion of the scientific knowledge in sports cardiology several revisons of ECG interpretation in athletes were published subsequently. The major intention of these revisions was to decrease false positive results, but to maintain adequate sensitivity by correctly differentiating physiological ECG changes in athletes from pathological. In the mixed group of 4297 white and 1208 black athletes, and 103 athletes with known HCM[8], the prevalence of false positive results was 21.5% according to 2010 ESC criteria, 9.6% according to Seatlle criteria[9] and 6.6% based on refined Seatlle criteria[8]. All three criteria identified patients with HCM with sensitivity of 98.1%, suggesting that implementation of revised guidelines resulted in increased specificity with preserved senitivity.

HCM is characterized by different ECG changes such as negative T waves, ST segment depression, pathological Q waves, left bundle branch block, left axis deviation, left atrial enlargement.

Negative T waves are defined as negative T waves more than 1mm in depth in two or more leades except in leads D3, aVR and V1. In patients with confirmed HCM prevalence of negative T waves is 54-62% in white[10,11] and 77% black patients[12]. On the other hand, the prevalence of negative T waves in inferior or/and lateral leads in healthy white athletes is very low (2%)[13], but its prevalence is significantly higher in black athletes 8-10%[12]. Presence of negative T waves in inferior, lateral and inferolateral leads in athletes should always raise suspecion of HCM. Negative T waves in leads V4-V6 are suggestive of apical form of HCM. For the confirmation of apical HCM magnetic resonance imaging is frequently required, since some cases may be missed by transthoracic echocardiography. Deep negative T waves in lateral and inferolateral leads are almost always related to pathological conditions, whreas isolated deep negative T

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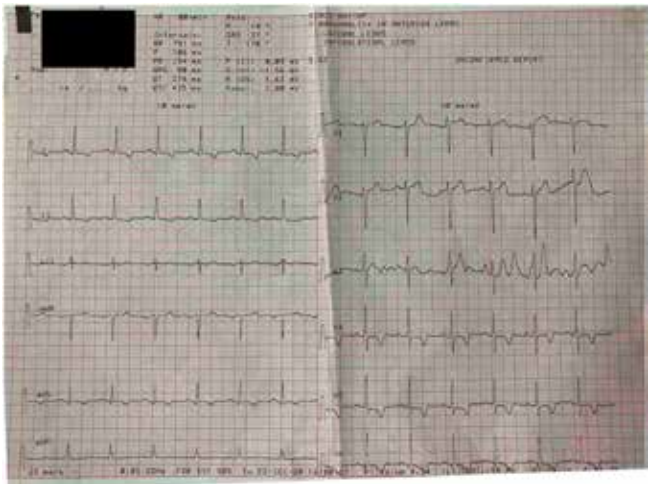

Figure 1. ECG in patient with HCM, note the presence of deep negative T waves predominatly in lateral leads

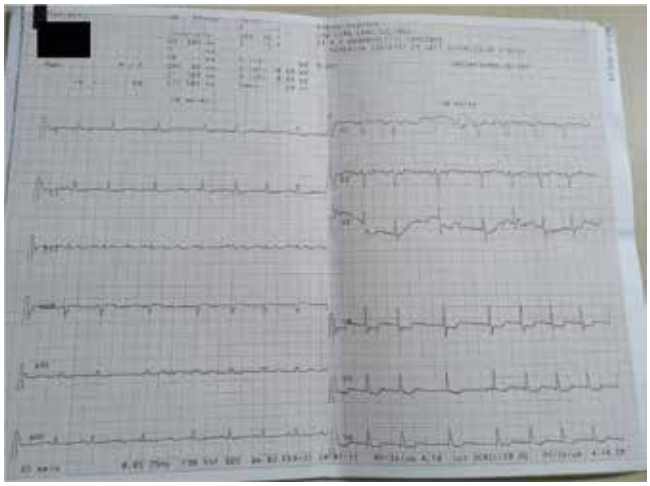

Figure 2. ECG in patient with HCM and atrial flutter. Please note ST segment depression in leads V4-V6 without voltage criteria for LV hypertrophy, and pronounced negative portion of atrial P waves in lead V1

waves in inferior leads are rarely sign of underlying pathological condition, but further workup is also required.

ST segment depression is extremely rare finding in healthy athletes with the prevalence of less than 1%[11,12]. The prevalence of ST segment depression (Figure 2), defined as ST segment depression of more than 0.5mm in two or more leads, is around 50% in patients with HCM[14].

Pathological Q waves are frequent finding in patients with HCM, detected in 42% of asymptomatic patients[15]. Abnormal Q is defined as Q wave of more than 3mm in depth and of more than 40 msec in duration. In the presence of deep narrow Q waves (less than 40 msec in duration), which is common finding in lean highly trained athletes, pathological Q waves are characterized by the Q/R ratio more than 0.25. The presence of pathological Q waves in athletes requires further investigation. Similarly, further work up is required in all the athletes with **complete left bundle branch block** (prevalence of 2% in patients with HCM)[14] or **slow interventricular conduction** (QRS duration more than 140 msec).

Left axis deviation, between -30 to -90 is extremly rare finding in athelets (< 1%)[12,16]. Left atrial enlargement (P wave duration of more than 120 msec in leads I and II with negative portion of P wave in V1 of more than 1mm in depth and ≥ 40 msec in duration in lead V1) is considered to be abnormal in athletes.

Isolated voltage criteria for LV hypertrophy (Lion-Sokolov criteria sum of S wave in V1 and R in V5 or V6 more than > 35mm) is highly prevalent finding in athletes 64%[13,17] and represents increased left ventricular mass due to the physical training. Isolated voltage criteria for LV hypertrohy (without any other ECG changes such as left atrial anlargement, axis deviation) in asyptomatic athletes, with normal physical findings and without history of SCD in family members do not require further investigation. However, isolated voltage criteria for LV hypertrophy can be the only ECG finding in less than 2% of patients with confirmed HCM, but these patients have more benign prognosis with low risk for malignant rhythm disturbances[18].

All athletes with ECG changes suggestive of HCM require diagnosis confirmation by imaging studies including transthoracic echocardiography or/and magnetic resonance imaging. Currently, athletes with confirmed diagnosis are disqualified from further high level training and compatition.

Athletes with pathological ECG but normal imaging studies including magnetic resonance imaging represent challenging cases. These patients could continue training with regular clinical and imaging follow up. Physicians should be aware that electrocardiographic chages preceed morphological changes in patients with cardiomyopathies[18-20].

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Sažetak

Elektrokardiografske promene kod hipertrofične kardiomiopatije: Značaj za skrining sportista

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Hipertrofična kardiomiopatija predstavlja jednu od najčešćih naslednih kardiomiopatija i prema podacima iz SAD vodeći je uzrok naprasne smrti kod mladih sportista. Vrlo česte sportisti sa hipertrofičnom kardiomiopatijom su bez simptoma i sa urednim fizikalnim nalazom. Međutim, patološke promene na 12-kanalnom elektrokardiogramu su prisutne i kod 90% bolesnika sa HCM, tako da je poznavanje elektrokardiografskih promena važno za identifikaciju ovih sportista.

Ključne reči: *hipertrofična kardiomiopatija, elektrokardiogram, sportisti*

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