



Successful primary percutaneous coronary interventions in a patient with two consecutive ST-segment elevation myocardial infarctions and dual left anterior descending artery (type IV)

Uspešne primarne perkutane intervencije kod bolesnika sa dva uzastopna infarkta miokarda sa elevacijom ST-segmenta i dvostrukom prednjom descendentnom arterijom (tip IV)

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Abstract

Introduction. Dual left anterior descending (LAD) artery is a very rare inherited anomaly. It can be incidentally revealed during primary percutaneous coronary intervention (pPCI) and may produce difficulties in detecting and treating the culprit lesion. **Case report.** We presented a 52-year-old male patient with ST-segment elevation myocardial infarction (STEMI) of inferior wall, in whom dual LAD anomaly was revealed during pPCI: a short LAD artery originated from the left main coronary artery and a long LAD artery originated from the proximal part of the right coronary artery (RCA). A bare metal stent was successfully implanted in the place of the culprit lesion in RCA and ST-segment resolution was achieved in ECG. After two hours, the patient was referred again to the catheter lab due to new STEMI of anteroseptal wall. Another bare metal stent was implanted in new infarction related artery, this time it was proximal part of the short LAD. **Conclusion.** Careful and correct interpretation of ECG is very helpful in detection and treatment of the culprit lesion in cases with dual LAD.

Key words:

coronary vessels; congenital abnormalities; myocardial infarction; stents; reoperation; electrocardiography.

Apstrakt

Uvod. Dvostruka prednja descendentna arterija (PDA) je veoma retka urođena anomalija i može biti otkrivena slučajno tokom primarne perkutane koronarne intervencije (pPKI) što može stvoriti poteškoće u određivanju i lečenju infarktne lezije. **Prikaz bolesnika.** Prikazali smo muškarca, starog 52 godine, sa infarktomiokarda sa ST-elevacijom (STEMI) donjeg zida, kod koga je tokom pPKI otkrivena dvostruka PDA. Kratka PDA je imala ishodište iz levog glavnog koronarnog stabla, a dugačka PDA iz proksimalnog dela desne koronarne arterije (DKA). Na mestu infarktne lezije u DKA uspešno je implantiran metalni stent i postignuta rezolucija ST-segmenta u EKG-u. Nakon dva sata, bolesnik je vraćen u angiosalu zbog novog STEMI anteroseptalne lokalizacije. Drugi metalni stent implantiran je na mestu nove infarktne lezije, ovog puta u proksimalnom delu kratke PDA. **Zaključak.** Pravilna interpretacija EKG-a od velike je pomoći u detekciji i lečenju infarktne lezije kod retke anomalne dvostruke PDA.

Ključne reči:

aa. coronariae; anomalije; infarkt miokarda; stentovi; reoperacija; elektrokardiografija.

Introduction

The incidence of the dual left anterior descending (LAD) artery is uncommon and ranges from 0.01% to 0.03%. In 1983, Spindola et al. ¹ published a classification of this very rare inherited coronary artery anomaly. This classification in-

cluded four types of dual LAD. The first three types implied a variety of both short and long LAD arteries originating from the left coronary sinus or the left main coronary artery, and the fourth type implied that long LAD artery originating from the proximal part of the right coronary artery (RCA). In 2010, Manchanda et al. ² described the fifth type of dual LAD, where the

short LAD artery originates independently from the left coronary sinus, and the long LAD artery originates from the right coronary sinus. In 2012 a new, sixth type of dual LAD was proposed by Maroney and Klein³, where a unique route of long LAD artery was discovered. It arises from proximal RCA going underneath the right ventricular outflow tract to the anterior interventricular groove. Dual LAD anomaly can be a rare cause of ischemic heart disease or even sudden cardiac death, especially when the long LAD artery goes between large cardiac vessels, like aorta and pulmonary trunk, which can lead to artery compression during the high blood flow through those big arteries^{4,5}.

We presented a patient with type IV, dual LAD anomaly discovered during primary percutaneous coronary intervention (pPCI), who also had two consecutive STEMI in the span of two hours with culprit lesions in two different coronary arteries: the RCA and short LAD artery.

Case report

A 52-year-old male patient with chest pain and diaphoresis was admitted to the Emergency Department of the Military Medical Academy in Belgrade, Serbia. Pain had the character of tightening in the chest, spreading to the shoulders and upper arms and there was a time lapse from pain onset to admission of only one hour. The patient stated similar pain in the past. He was also treated for hypertension and diabetes mellitus type 2. He smoked 25 pack year of cigarettes.

His blood pressure was 120/80 mmHg and heart rate 67 beats *per* minute. Other physical findings on admission were unremarkable except for a pale, dewy skin.

Baseline ECG showed ST-segment elevation in the leads for the inferior wall, and reciprocal ST-segment depression in the D1 and AVL leads (Figure 1).

As *per* treatment protocol, before pPCI, the patient received oral administration of aspirin 300 mg, clopidogrel 600 mg and parenteral administration of unfractionated heparin 80 U *per* kilogram of body weight.

On coronary angiography, a short LAD artery was seen, arising from the left coronary sinus and exhausting in the middle segment, after the separation of a large septal trunk. In its proximal part, a significant, tubular-type, stenosis of about 80% of vessel lumen was seen (Figure 2).

The RCA had subtotal stenosis in the proximal segment just after the separation of a long artery which bended toward apical lateral region of the heart. That anomalous artery, which arose from the proximal part of the RCA appeared to be a long LAD artery in a very rare coronary artery anomaly called dual LAD. It had also a tubular stenosis of about 40–50% in its proximal part (Figure 3). According to ECG changes, the ST-segment elevation in the leads for the inferior wall, lesion of the RCA was recognized as the culprit lesion and after several balloon predilatations, a bare metal stent (dimension 3.5 × 15 mm) was implanted. The final coronarography effect was referred as excellent (TIMI 3 flow) (Figure 3).

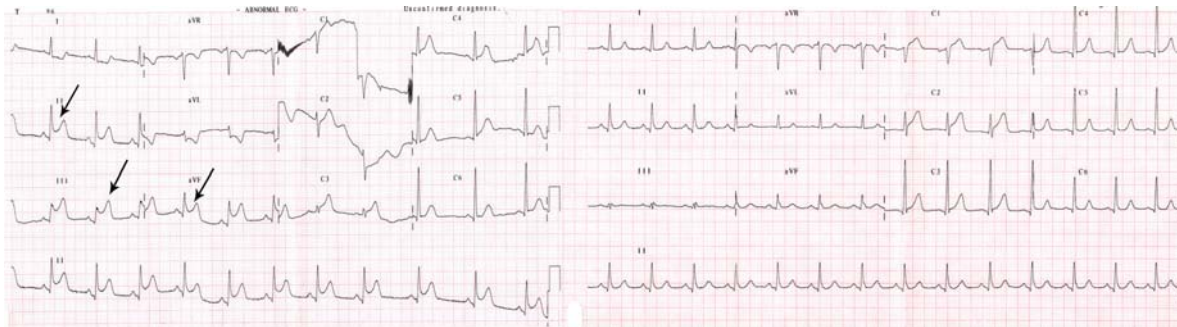
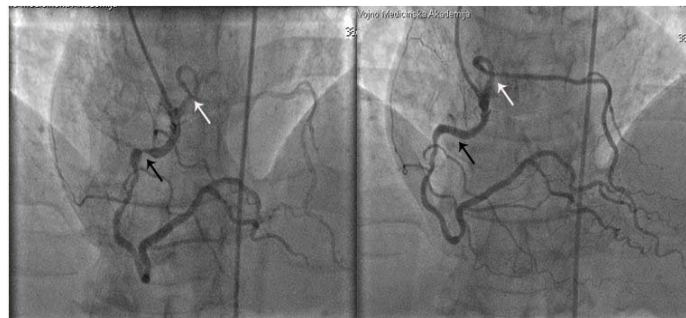


Fig. 1 – Left: ECG on admission: ST-segment elevation in the leads for the inferior wall (arrows); Right: Complete ST-segment resolution after performing primary PCI with stent implantation in the RCA as infarction related artery. ECG – electrocardiogram; PCI – percutaneous coronary intervention; RCA – right coronary artery.



Fig. 2 – Left and middle: coronary angiogram, “spider” and caudal view, showing the short LAD artery (arrow) with a significant stenosis in its proximal part. Right: short LAD artery after stent implantation (arrow). LAD – left anterior descending.



**Fig. 3 – Left: coronary angiogram, cranial view, with the RCA and significant stenosis as culprit lesion (black arrow) and the anomalous long LAD artery with mild proximal stenosis (white arrow). Right: RCA after stent implantation in culprit lesion (black arrow) and the long LAD artery (white arrow).
RCA – right coronary artery; LAD – left anterior descending.**

Soon after pPCI, the patient was without chest pain, and ECG showed complete ST-segment resolution (Figure 1).

Two hours after the intervention, while recovering in intensive care unit, the patient had chest pain again, with identical propagation and diaphoresis. ECG revealed ST-segment elevation in AVL and V1 to V3 (Figure 4).

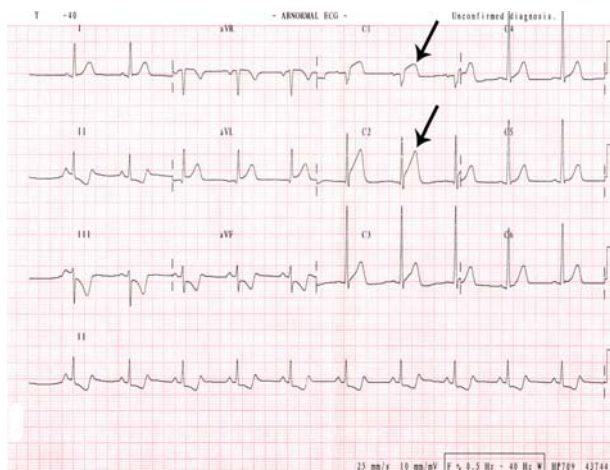


Fig. 4 – ECG two hours after the primary PCI on the RCA, ST-segment elevation in the leads for the anteroseptal wall (arrows) suggesting new culprit lesion in the short LAD artery.

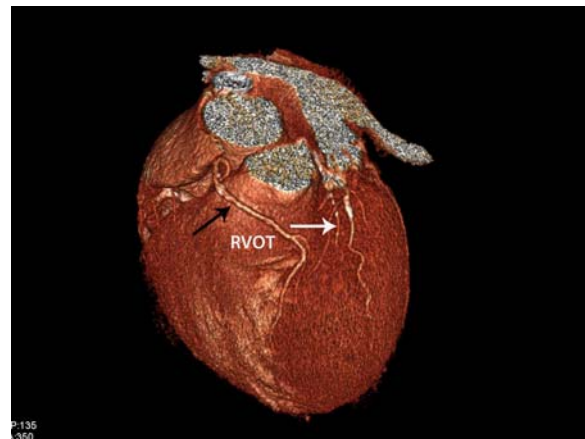
ECG – electrocardiogram; PCI – percutaneous coronary intervention; RCA – right coronary artery; LAD – left anterior descending.

The patient was returned to the catheter lab and another pPCI was performed. Thrombus morphologic lesion was noticed in the place of the previous stenosis in the proximal segment of the short LAD artery originating from the left coronary sinus, so we implanted another bare metal stent in this new culprit lesion. An excellent coronarography effect was achieved with TIMI 3 flow. A control angiography of recently stented RCA was performed and TIMI 3 flow remained.

After this intervention, chest pain disappeared, and complete ST-segment resolution was observed on ECG.

In further course of hospitalization, there were no complications and cardiac specific enzymes activity completely returned to normal values. Echocardiography at discharge, seven days after admission, showed mild hypokinesia of the middle and basal part of the septum with left ventricular ejection fraction of 50%. The patient was discharged home in good condition

and referred to further follow-up. One year after pPCI, the patient underwent dobutamine stress echo examination which was negative for ischemic heart disease. A few months later, he complained of mild chest discomfort, but on examination in emergency unit, serial ECGs were without changes, serum cardiac specific enzymes activities were in normal value range, arterial blood pressure was 130/80 mmHg and the rest of physical examination was unremarkable. Having in mind his current health state and the previous medical history we decided not to expose him to invasive coronary angiography, but to emergency multi-detector computed tomography (MDCT) coronary angiography. It revealed that both of implanted stents were without signs of occlusion or restenosis, the lesion in the long LAD artery was estimated as unchanged, and culprit lesion was not seen. Using this imaging we recognized the dual LAD to be of type IV because the long LAD artery did not have trajectory under, but above the right ventricular outflow tract (Figure 5).



**Fig. 5 – MDCT coronary angiography performed one year later. Long LAD artery originates from the proximal RCA and goes above RVOT toward anterior interventricular groove (black arrow), suggesting type IV dual LAD. Short LAD artery is also seen (white arrow).
MDCT – multidetector computed tomography; LAD – left anterior descending; RCA – right coronary artery; RVOT – right ventricular outflow tract.**

Discussion

Acute coronary syndrome in patients with anomalous coronary arteries has been reported as specific and sometimes

difficult to treat when performing PCI^{6,7}. In the presented patient with STEMI of the inferior wall a very rare coronary artery anomaly, called dual LAD, was incidentally revealed during the primary PCI. He also had multivessel coronary disease which could have made treatment decision difficult, especially if a patient had more diffuse ECG changes or myocardial infarction of another localization. A short LAD artery could sometimes look as acutely blocked infarction related artery and trying to open it may lead to perforation. Additionally, early separation of a long LAD artery from the RCA also might be missed on coronarography due to catheter malposition, or due to the separate source of the anomalous artery from the right or left coronary sinus⁸. The presented patient had inferior wall myocardial infarction, which was clear due to ST-segment elevation on ECG in the leads for inferior wall. Although the RCA was not occluded and had TIMI 3 flow, significant proximal lesion was recognized as culprit lesion. After two hours, the patient developed another STEMI, but this time, ST-segment elevation was in the leads for anteroseptal region, which guided us to the short LAD artery, with its proximal lesion, as infarction related artery and not the long LAD artery which also had borderline stenosis. This suggests that ECG guided primary PCI could be of enormous help in revealing a culprit lesion in STEMI patients with anomalous coronary arteries, especially in patients with multivessel coronary disease. What was a rupture trigger for the second culprit lesion remains unclear, but it is understandable that plaque in the short LAD artery was highly unstable and complex interplay of variables in and outside the plaque could cause rupture with subsequent thrombosis. Acute myocardial infarction is a state of increased inflammatory and hemodynamic activities which can cause further plaque destabiliza-

tion, arterial spastic reaction, intraplaque hemorrhage and plaque disruption^{9,10}. It is possible that selective injection of contrast fluid, during PCI, can cause microscopic foci of endothelial loss or even erosions on the unstable plaque surface which can lead to thrombotic cascade. An interesting question could be asked in this case: What if anteroseptal STEMI had appeared first in our patient and anomalous long LAD artery had not been seen on coronarography due to separate ostium in the right coronary sinus or due to catheter malposition in the RCA? Because the short LAD artery diminished after giving large septal trunk, thinking of occlusion and trying to open it could have had dangerous consequences and at least it would have consumed a precious time.

Furthermore, very rare anomalies of coronary arteries may be a problem if urgent cardiac surgery is required¹¹.

This case was interesting to us due to several reasons: incidental revelation of one of the rarest coronary anomalies; development of two consecutive STEMIs of two separate regions (first inferior, and second was anteroseptal) within two hours; correct interpretation of culprit lesions sites among several hemodynamically significant stenosis of the coronary arteries including the anomalous LAD artery, and successfully performed PCI with excellent recovery of the patient.

Conclusion

Dual LAD artery is very rare anomaly which might be incidentally revealed during primary PCI. In these patients, if multivessel coronary disease exists, recognizing culprit lesion is more complicated which can be amended by careful interpretation of ECG changes together with understanding the anomalous arteries' trajectories.

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