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Case report

MINOCA caused by plaque erosion

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Abstract

Introduction. According to clinical studies, the prevalence of acute myocardial infraction with nonobstructive coronary arteries (MINOCA) is around 5%. MINOCA can be triggered by various atherosclerotic and nonatherosclerotic causes with heterogenous pathophysiological mechanisms. Plaque disruption is frequent at MINOCA patients and it implies plaque rupture, plaque erosion, and calcium node. We presented a case of a 32-year-old male patient with MINOCA caused by plaque erosion.

Case report. A 32-year-old male patient was admitted due to chest pain, ECG abnormalities, and elevated cardio specific enzymes. Urgent coronary angiography was performed and non-significant 30% stenosis was found in proximal LAD. OCT was performed in LAD revealing plaque erosion with white thrombus masses, as well as a 57% stenosis, with minimal lumen area of 7.91mm^2. According to OCT findings, it was decided to proceed with medical treatment. During follow-up, the patient was without symptoms. Functional tests on ischemia were normal after one year of the incident. After two years of the incident, CT coronarography registered a mild 25% stenosis in the proximal LAD.

Conclusion. In the diagnostics and treatment of the patients with MINOCA, it is vital to use a multimodal imaging. Plaque erosion is one of the possible causes of MINOCA, firstly because of the possibility to trigger vasospasm with consequential impaired flow. The usage of intracoronary imaging is paramount to adequately analyze the morphology in the culprit area.

Kew words myocardial infraction with nonobstructive coronary arteries, optical coherent tomography, plaque erosion

Introduction

According to the clinical studies, the prevalence of acute myocardial infraction with nonobstructive coronary arteries (MINOCA) is around 5%[1]. These patients have a better prognosis when compared with the patients where the cause of the acute myocardial infraction is the obstruction of coronary arteries[2].

MINOCA can be triggered by various atherosclerotic and nonatherosclerotic causes with heterogenous pathophysiological mechanisms[2,3]. MINOCA is considered a working diagnosis and it should be searched for the basic cause of clinical presentation.

Plaque disruption is frequent at MINOCA patients and it implies plaque rupture, plaque erosion, and calcium node. Plaque disruption can also be a trigger of a thrombosis which causes an acute myocardial infraction with a distal embolization, coronary vasospasm or, in some cases, it can cause transit complete thrombosis with a spontaneous thrombolysis.

Coronary angiogram can imply plaque disruption, but the final diagnosis can be made only by the usage intracoronary imaging. Optical coherent tomography (OCT) is considered to have an advantage due to its higher resolution.

We presented a case of a 32-year-old male patients with MINOCA caused by plaque erosion.

Case Presentation

A 32-year-old male patient was admitted due to the chest pain, ECG abnormalities, and elevated cardio specific enzymes. Symptom onset was six hours prior the hospital admission. It was manifested with a severe chest pain and sweating. The patient was a smoker, and it was considered as the only risk factor. Physical examination was unremarkable. Electrocardiogram (ECG) recorded by emergency medical service (EMS) registered ST elevation in precordial leads (Figure 1.). Urgent coronary angiography was performed and non-significant 30% stenosis was found in proximal left anterior descending (LAD), prior the bifurcation with strong septal branch with TIMI 3 flow (Figure 2A and 2B).

OCT was performed in LAD in order to analyze the morphology of the lesion in the proximal part. Plaque erosion in 16mm of length with white thrombus masses was registered, distal reference diameter beneath the

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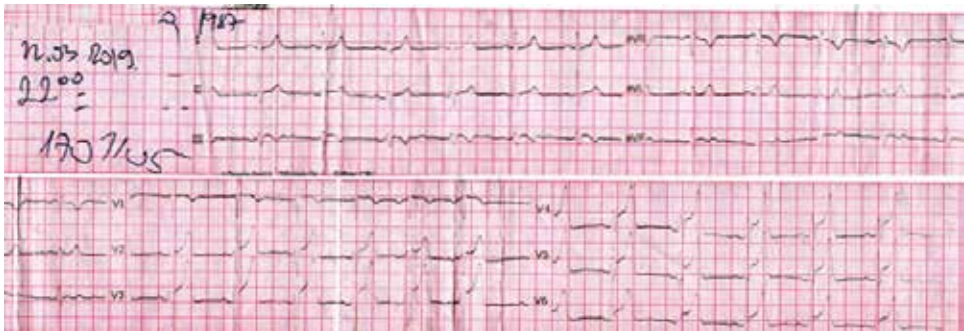

Figure 1. ECG recorded by the EMS, showing elevation in precordial leads

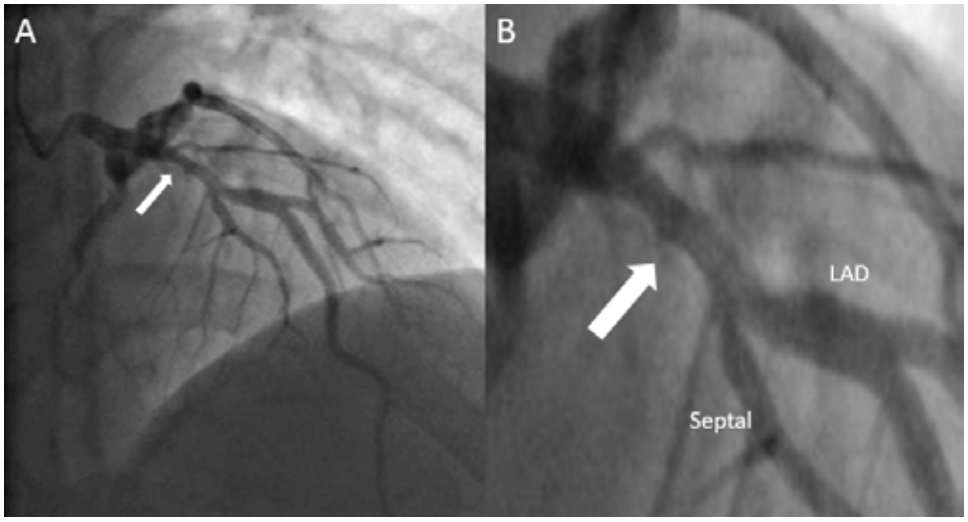

Figure 2A. Cranial projection of the LCA, arrow pointing to the 30% stenosis in proximal LAD; **2B**. Proximal LAD stenosis in close-up

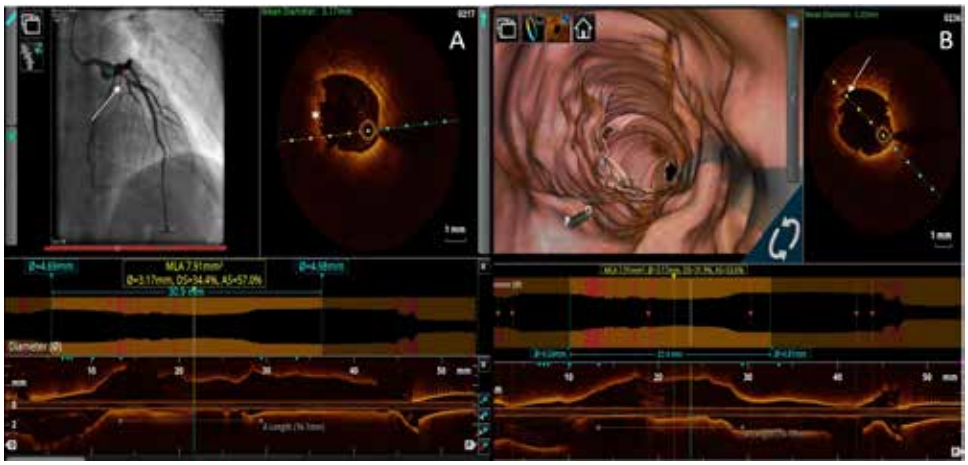

Figure 3A. Arrow pointing to the erosion site on co-registration. Asterisk pointing to the erosion; **3B** arrow pointing to the white thrombi

plaque erosion was 5.0mm, while the proximal reference diameter was 5.5mm. A 57% stenosis was registered in the area of the plaque erosion, with minimal lumen area (MLA) of 7.91mm^2. According to the OCT findings, it was decided to proceed with the medical treatment (Figure 3A and 3B).

Echocardiography showed hypertrophic myocardial left heart chamber of normal endocavital dimensions, without wall motion abnormalities, and of regular systole and diastole functions. 2D strain registered decreased myocardial deformation in medio-basal anterior and medio-basal anterolateral segments. A 24-hour ECG was performed and it registered sinus rhythm. There were no rhythm disturbances.

Laboratory registered slightly elevated values of thyroid-stimulating hormone, while fT3 and fT4 were referential. Endocrinologist's opinion was that there is no need for a treatment. In the further hospital stay, the patient was stable.

The patient was discharged on the fifth day of the hospitalization with an administered therapy that included Aspirin, Ticagrelor, Bisoprolol, Zofenopril, Rosuvastatin in 40mg dosage and a proton pump inhibitor. After six weeks, the Rosuvastatin dosage was lowered to 20mg

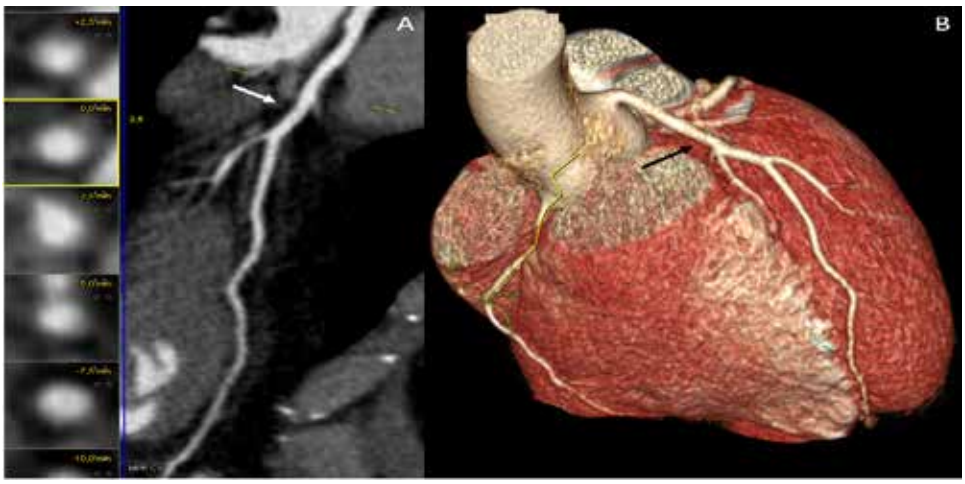

Figure 4A. Arrow pointing to the stenosis of 25% in prior erosion site; **4B**. 3D reconstruction with an arrow pointing to the prior erosion site

due to LDL levels which persisted in the treatment range, during the follow-up. After 6 months period, stress echocardiography registered normal functional reserve of the myocardium while 2D strain showed complete improvement in kinetics. On a one-year-follow-up visit Ticagrelor was excluded from the therapy. Treadmill test was normal without signs of ischemia on 12METs.
CT coronarography after two-year period, Calcium score was 0 and there was a mild 25% stenosis in the area of prior plaque erosion (Figure 4A and 4B).

Discussion

Patients with MINOCA, in general, have a better prognosis when compared with the patients with an obstructive coronary disease and acute coronary event. Still, the mortality rate of the patients with MINOCA is higher when compared with the healthy population, in both long- and short-term follow-up [1]. Considering, a thorough and detailed approach is required in both diagnostics and treatment of the patients with MINOCA. Today, a number of invasive and non-invasive diagnostic techniques are available. These methods enable an adequate diagnostic of MINOCA etiology and as a result a proper treatment of the patients. Invasive diagnostic methods should be applied when there a suspicion of coronary etiology of MINOCA.
Intracoronary application of Ergonovine and Acetylcholine is recommended when provocative tests are needed due to a doubt on underlying coronary or microvascular vasospasm[1,4].
If the coronary angiogram suspects the presence of abnormalities of coronary artery, it is recommended to perform intracoronary imaging, such as intravascular ultrasound (IVUS), or OCT[1,5].
In the presented case, coronary angiography registered non-significant lesion in the proximal part of the LAD, with TIMI 3 flow. Non-invasive diagnostics, such as, ECG, Echocardiography, and cardio specific enzymes implied MINOCA as a working diagnosis. Considering angiographic findings with a lesion in the proximal part of the LAD, and according to the guidelines, intracoronary imaging was performed registering plaque erosion.

Current OCT studies have shown that the most common cause of acute coronary syndrome is plaque rupture in the 2/3 of the cases, plaque erosion is registered in 1/3, whereas calcium node is registered in 1/20 cases[6]. Thrombosis mechanism is different in plaque rupture and plaque erosion. In the first case, with plaque rupture, there is a typical vulnerable plaque with thin fibrose cap and lipid or necrotic core beneath. Opposite to this, there is a plaque erosion which has intact fibrose cap and less inflammation and lipids inside the vessel wall. The usual cause of thrombosis is apoptosis or denudation of endothelial cells, which leads to a malformation of white platelet rich thrombus[7].
As the atherosclerotic process is different in these two entities, the question is if they should be treated in the same way. Respectively, is there always a need for stent implantation in patients with plaque erosion.
Hu et al. used OCT to analyze the influence of plaque morphology on vessel healing and stent endothelization. Even though it was a rather small study of 43 patients, they concluded that in the patients with plaque erosion the vessel healing and neointimal coverage of the stent struts are delayed, when compared to the patients with the plaque rupture[8].
In their analyses, Prati et al. showed that OCT can identify non-obstructive lesions with plaque erosion, that can be treated with medications, dual antiplatelet therapy without stent implantation with a satisfactory clinical outcome[9].
Erosion study shows that plaque erosion is frequent in 25% of acute coronary events. In this research, after achieving anterograde flow by thrombus aspiration, OCT was used to confirm plaque morphology. In patients who were clinically stable after achieving anterograde flow, but who had registered plaque erosion on OCT as a cause of ACS, and angiographically registered stenosis less than 70% with TIMI 3 flow, stent was not implanted. A conversative treatment was conducted with potent antiplatelet therapy, in the first place. OCT follow-up registered that after a one-month-period there is a significant thrombus regression [10]. Shin et al. used OCT to assess morphological characteristics of vasospastic angina (VSA). They noticed that plaque erosion is registered in around 25%

of cases with white platelet rich thrombi. It is considered that the interaction between platelets and eroded vessel wall lead to a vasospasm[11].

Dai et al. analyzed in vivo predictors of plaque erosion, concluding that it is more frequent in people younger than 50, closer to the bifurcation, without dyslipidemia regardless gender. Additionally, there is a connection between males and smoking[12].

This case presented a young male patient, smoker, with a plaque erosion in the LAD at the bifurcation area with a strong septal branch. It is assumed that plaque erosion led to a vasospasm which transitory impaired the flow through the LAD. Luckily, the patient responded well on the administered therapy. Having in mind that angiographically estimated stenosis was less than 50%, with TIMI 3 flow, as well as plaque erosion registered by OCT with MLA of 7.91mm^2, the team decided to proceed with a conversative treatment with DAPT and statin therapy.

During the follow-up period, the functional tests on ischemia showed a complete myocardial recovery. CT coronarography, two years after the event, demonstrated a complete recovery of the culprit segment in the LAD.

Conclusion

In the diagnostics and treatment of the patients with MINOCA, it is vital to use a multi-modal imaging. Plaque erosion is one of the possible causes of MINOCA, firstly because of the possibility to trigger vasospasm with consequential impaired flow. The usage of intracoronary imaging is paramount to adequately analyze the morphology in the culprit area.

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Sažetak

MINOCA uzrokovana erozijom plaka

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Uvod. *Na osnovu podataka iz kliničkih studija, prevalenca akutnog infarkta miokarda sa neobstruktivnim koronarnim arterijama (MINOCA) je oko 5%. MINOCA može nastati kao posledica različitih aterosklerotskih i neaterosklerotskih uzroka sa heterogenim patofiziološkim mehanizmima. Disrupcija plaka je česta kod pacijenata sa MINOCA i može biti posledica rupture plaka, erozije plaka i kalcijumskog nodusa. Prikazali smo slučaj muškarca od 32. godine sa MINOCA uzrokovanom erozijom plaka.*

Prikaz slučaja. *Hospitalizovan je muškarac od 32. godine zbog bola u grudima, EKG promena i povišenih vrednosti kardiospecifičnih enzima. Urgentnom koronarografijom je registrovano suženje od 30% u proksimalnom segmentu prednje descedentne koronarne arterije (LAD). Urađen je OCT kojim je registrovana erozija plaka sa belim trombotičnim masama i stenozom od 57%, kao i MLA od 7,91mm^2 . Shodno nalazu OCT, odlučeno je da se nastavi dalji medikamentni tretman. Tokom perioda praćenja, pacijent je bez tekoba. Funkcionalni testovi nakon godinu dana su bili uredni. Dve godine nakon incidenta urađenja je CT koronarografija kojom se registruje stenoza od 25% u LAD.*

Zaključak. *U dijagnostici i lečenju pacijenata sa MINOCA, multi modalni imidžing je od velikog značaja. Erozija plaka je jedan od mogućih uzroka MINOCA, pre svega jer postoji mogućnost da će pokrenuti vazospazam i narušiti protok. Upotreba intrakoronoranog imidžinga je neophodna kako bi se advekatno analizirala morfologia „kalprit" lezije.*

Ključne reči: akutni infarkt miokarda sa neobstruktivnim koronarnim arterijama, optička koherentna tomografija, erozija plaka