Left Ventricular Systolic Dysfunction After Myocardial Infarction

Miyokard Enfarktüsü Sonrası Sol Ventriküler Sistolik Disfonksiyon

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ABSTRACT

The left ventricular systolic dysfunction is one of the most common complications after myocardial infarction. The damage of the large area of the cardiac muscle leads to a significant decrease in cardiac contractility. Our study presents a very unusual case of the left ventricular systolic dysfunction after myocardial infarction. A 76-year-old female was hospitalized at Chapidze Emergency Cardiology Center (Tbilisi, Georgia) with the diagnoses of ischemic heart disease and acute subendocardial myocardial infarction. Due to cardiac muscle ischemia, sinus bradycardia and diastole prolongation was revealed, which increased left ventricular end-diastolic volume and eventually caused left ventricular overload. It seemed to be a main predictor of developing compensatory ventricular premature contractions, followed by ventricular fibrillations. This argument was later supported by the stable medical condition reached by transcutaneous cardiac pacing. According to this result, the decision was made to implant a dual-chamber cardioverter - defibrillator as a non-standard treatment option, which turned out to be very successful. According to recent data, implanting permanent dual-chamber cardioverter-defibrillators as a preventive step for ventricular fibrillation has not been reported yet.

Key Words: Myocardial infarction, ischemic heart disease, ventricular fibrillation.

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ÖZET

Sol ventrikül sistolik disfonksiyonu, miyokard enfarktüsünden sonra en sık görülen komplikasyonlardan biridir. Kalp kasının geniş bölgesinin hasarı, kalp kasılmasında önemli bir azalmaya yol açar. Çalışmamız, miyokard infarktüsü sonrası çok nadir görülen bir sol ventrikül sistolik disfonksiyonu olgusunu sunulmaktadır. 76 yaşında bir kadın, iskemik kalp hastalığı ve akut subendokardiyal miyokard enfarktüsü tanısıyla Chapidze Acil Kardiyoloji Merkezi'nde (Tiflis, Gürcistan) hastaneye kaldırıldı. Kalp kası iskemisine bağlı olarak sol ventrikül diyastol sonu hacmi artıran ve sonunda sol ventrikül aşırı yüklenmesine neden olan sinüs bradikardisi ve diyastol uzaması ortaya çıktı. Bu, kompansatuar ventriküler erken kasılmalar ve ardından ventriküler fibrilasyonların gelişmesinin ana belirleyicisi gibi görünüyordu. Bu argüman daha sonra transkutan kardiyak pacing ile ulaşılan stabil tıbbi durumla desteklendi. Bu sonuca göre, standart olmayan bir tedavi seçeneği olarak çift odacıklı bir kardiyoverter - defibrilatör yerleştirilmesine karar verildi ve bu çok başarılı oldu. Son verilere göre, ventriküler fibrilasyon için önleyici bir adım olarak kalıcı çift odacıklı kardiyoverter defibrilatörlerin implante edilmesi henüz rapor edilmemiştir.

Anahtar Sözcükler: Miyokardiyal enfarktüs, iskemik kalp hastalığı, ventriküler fibrilasyon.

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INTRODUCTION

The left ventricular systolic dysfunction is one of the most common and severe complications after myocardial infarction. The damage of the large area of the cardiac muscle leads to a significant decrease in cardiac contractile function (1). Delay of intervention can promote these conditions. The disease significantly increases the risk of sudden death and patients often develop heart failure (2). An appropriate therapy can reduce the progression of the disease and lower the risk of morbidity and mortality (3,4). Accordingly, it is essential to organize a special patient care structure, which will provide early detection and suitable management of patients with left ventricular systolic dysfunction after myocardial infarction (5). The most common complications of systolic dysfunction are cardiogenic shock, life-threatening arrhythmias and organic failure (6,7,8). Sudden cardiac death due to systolic dysfunction and heart failure may be caused by recurrent myocardial infarction or life-threatening arrhythmias (9). Despite the fact that the main cause of the Heart failure is systolic dysfunction due to myocardial infarction, there can also be seen several factors, responsible for prognosis and outcomes, for example: papillary muscle dysfunction due to myocardial infarction, mitral valve regurgitation, arrhythmias, etc. The outcome of such case is mostly unfavorable (10,11).

Our goal is to present an uncommon case of the left ventricular systolic dysfunction after myocardial infarction that happened at Chapidze Cardiology Emergency Center (Tbilisi, Georgia).

CASE REPORT

Anamnesis of a 76 - year-old Female with Arterial Hypertension III (ESC II class & NYHA II), Diabetes Mellitus type 2, without any treatment. BMI – 35 kg/m^2. The patient was hospitalized at our center, diagnosed with Acute heart failure (Killip II). 5 days earlier she experienced a sharp, long-term (7-8 hours) chest pain (angina pectoris). She had not called an ambulance. She was diagnosed with ischemic heart disease and acute myocardial infarction (NSTEMI) (hs-cTnI - 0.211ng/ml, Normal range – 0.014ng/ml) at our center. Echocardiography revealed a pattern of ischemic cardiomyopathy (LVD-5cm, LVS-4.4cm, anteroseptal wall hypokinesis, EF-25-28%, left and right-sided hydrothorax).

Performed Coronary Angiography revealed: RCA-distally <30%, LAD proximally-90%, mid->50%, IDB-90%, CX-mid-90%, DM-90% (Figure 1, Figure 2, Figure 3).

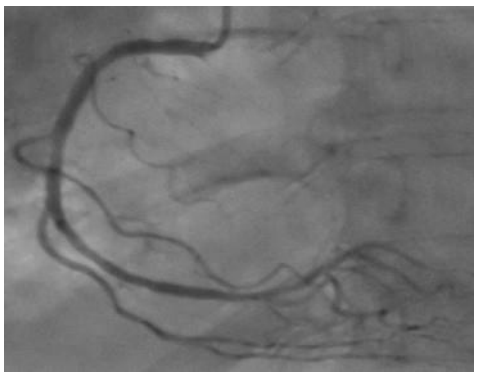

Figure 1. Coronary angiography imaging, right coronary artery

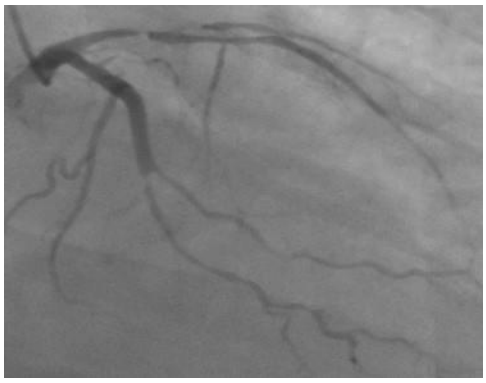

Figure 2. Coronary angiography imaging, circumflex artery

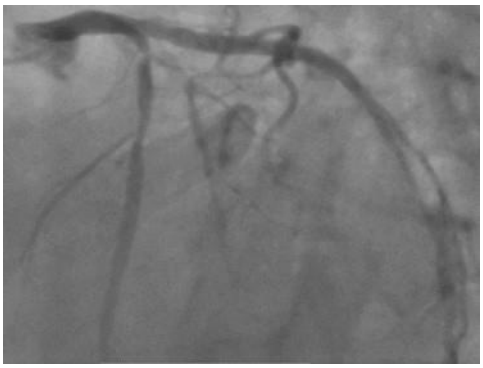

Figure 3. Coronary angiography imaging, left anterior descending artery

Percutaneous coronary intervention of the left anterior descending artery was recommended. (LAD PCI). The patient developed sudden ventricular fibrillation before starting coronary stent placement procedure. The complete AV block was registered after immediate defibrillation. After that a temporary pacemaker was implanted. And soon the sinus rhythm was restored (HR-80-90'). Then stent was inserted into the proximal and middle segments of the left anterior descending artery (LAD) (Figure 4 (a) (b).

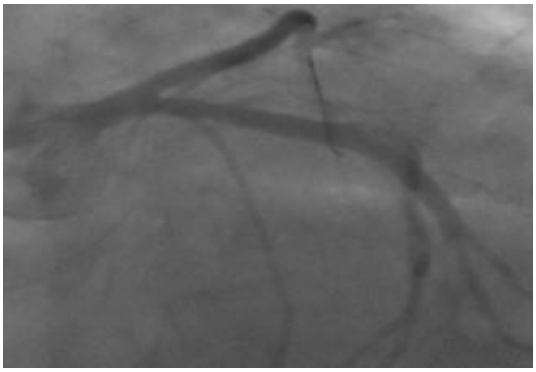

Figure 4 (a). After LAD PCI

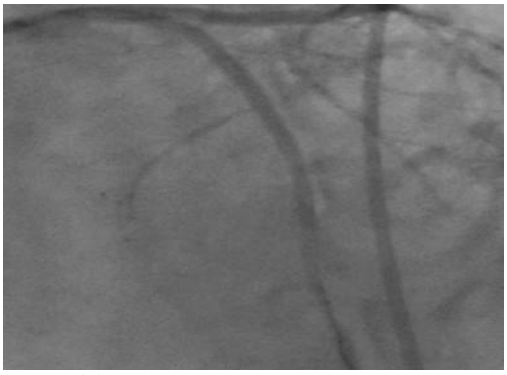

Figure 4 (b). After LAD PCI

After that procedure, the general health status of that patient improved, retrosternal pain wasn't revealed, ECG recordings showed sinus rhythm, LBBB. HR-70-80'. QRS-120msec. During the next 2 days the patient's health condition was stable, without any complications, and with the help of an appropriate medical therapy the main symptoms of heart failure became less severe. ECG recordings and Echocardiography findings didn't reveal any significant pathology. On the 3rd day after hospitalization, a temporary pacemaker was removed.

On the 4th day after hospitalization the patient's health status significantly worsened. The 3rd episode of ventricular fibrillation was registered, which was successfully treated by defibrillation. Coronary angiography was performed. The flow of blood along the stented artery was kept (TIMI III). Antiarrhythmic drug therapy was initiated. After that everyday 3 or 4 episodes of ventricular fibrillation was revealed, which led to a sharp deterioration of her condition.

On the 5th day after hospitalization ECG recordings revealed sinus bradycardia (HR-35'-40'), followed by ventricular premature contraction (VPC Bigeminy) after a few minutes and shortly followed by ventricular fibrillation. Prescribed agents that decreased heart rate were removed from the treatment scheme (Figure 5).

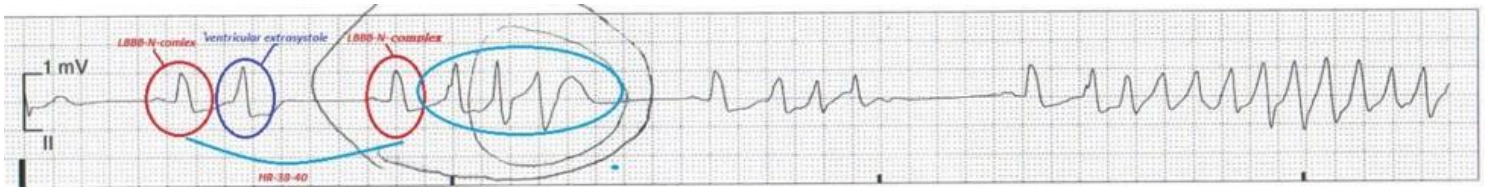

Figure 5. On the 5th day after hospitalization ECG recordings revealed ventricular fibrillation

On the 6th day after hospitalization 2 episodes of ventricular fibrillation developed in the same way, which was fixed by defibrillation. Echocardiography was done on the same day (Normal Heart Rate, LVD-5cm, LVS-4.0cm, EF- the same/unchanged). As soon as sinus bradycardia was registered, it was followed by severe left ventricular volume overload (LVD->7cm, LVS->5.5cm, DV>350ml, SV>200, EF<18%), and premature ventricular contractions with bigeminy followed by ventricular fibrillation. According to that condition, temporary cardiac electric pacing with rate 70' was initiated. As a result, the left ventricle size returned to baseline and ventricular bigeminy was fixed.

On the following days the condition was the same. On the 8th day after hospitalization, a temporary pacemaker was removed, but antiarrhythmic drug therapy continued. Despite this, 8 episodes of ventricular fibrillation were registered that night, which was again corrected by defibrillation. Meanwhile the patient developed hemodynamic instability, requiring inotropic support.

During the next 4 days total episodes of ventricular fibrillations were 16. All the time defibrillation was done and the sinus rhythm was restored. HR-70'-80', Patient still experienced hemodynamic instability, requiring inotropic support, her neurological status was intact, Respiratory status - normal.

On the 13th day after hospitalization: normal sinus rhythm coexisted with normal heart rate and follow-up echocardiography was done. The size of the left ventricle was among normal ranges. Ejection fraction was unchanged. Later that evening the patient developed sinus bradycardia again. Once again echocardiography was repeated and revealed left ventricular volume overload with significantly reduced ejection fraction. Transcutaneous cardiac pacing was initiated (rate 70') and the size of the left ventricle returned to the normal range. An arrhythmia wasn't reported. Dosage of the inotropic medication and also diuretics were reduced. The cardiac rhythm monitor showed alteration of the sinus rhythm with pacing rhythm. Despite reducing the baseline rate of the transcutaneous pacing rhythm, over again sinus bradycardia was registered, followed by left ventricular volume overload which led to ventricular premature contractions. Because of that, we continued transcutaneous pacing over again until stabilizing the patient's medical condition.

Eventually, on the 17th day after hospitalization dual-chamber cardioverter - defibrillator was implanted. Her medical status was completely stable. No episode of arrhythmia has been registered since then. And on the 21st day the patient was discharged from our clinic with stable medical status. Nowadays, the patient is under outpatient supervision.

DISCUSSION

It is well-known that there are a lot of different factors that may cause ventricular fibrillation and there are already appropriate treatment options available in the modern world of medicine. Our case was non-standard, thus the treatment options discussed in contemporary guidelines or modern diagnostic methods were inappropriate or useless for it. All the necessary diagnostic tools and treatment management were performed to eliminate the problem. For example: Repeated coronary angiography, revascularization of other blood vessels due to its anatomical features or diameter status wasn't performed. The issue of implanted cardioverter/defibrillator implantation was consulted with a specialized team of rhythmology and was postponed, because of the frequent episodes of arrhythmia that would have led to rapid depletion of the device. Constant follow-up echocardiography and lab tests were done. The level of electrolytes was normal (in normal range). However, the recurrence of ventricular fibrillations was registered periodically.

According to the long-term follow-up and effective discussion with our team members about that case, we presented sequentially a very rare predictor of ventricular fibrillation as a case report. The cardiac muscle ischemia, sinus bradycardia and diastole prolongation, increased left ventricular end-diastolic volume and eventually caused left ventricular volume overload. It seemed to be a main predictor of developing compensatory ventricular premature contractions, followed by ventricular fibrillations. The argument is supported by the stable medical condition reached by transcutaneous cardiac pacing. According to this result, we decided to implant a dual-chamber cardioverter - defibrillator as a non-standard treatment option, which turned out to be a very successful decision.

Dual-chamber implanted cardioverter/defibrillators, programmed to optimize detection enhancements and to minimize ventricular pacing, significantly decrease inappropriate detection. The implanted cardioverter/defibrillator is highly effective in decreasing mortality due to cardiac arrhythmias in high-risk patients (12,13). However, delivery of inappropriate shocks caused by misclassification of rapidly conducted supraventricular tachycardia as ventricular tachycardia remains a substantial complication of implanted cardioverter/defibrillator therapy, affecting 8% to 40% of patients (14,15,16,17,18,19). Inappropriate shocks can lead to pain, anxiety, depression, impaired quality of life, proarrhythmia, and poor tolerance of life-saving implanted cardioverter/defibrillator therapy (20,21,22,23,24,25,26,27,28,29).

Conflict of interest

No conflict of interest was declared by the authors.

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