Non-ST-Elevation Acute Myocardial Infarction and Sustained Slow Ventricular Tachycardia due to Coronary Slow Flow Phenomenon: a Case Report

Baiq Gerisa Rahmi Faharani*, Anggoro Budi Hartopo, NaharTaufiq

Department of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada – Dr. Sardjito Hospital, Yogyakarta, Indonesia

* Corresponding author:
Baiq Gerisa Rahmi Faharani MD, -email: gerisafaharani@gmail.com
Department of Cardiology and Vascular Medicine, Faculty of Medicine , Public Health and Nursing, Universitas Gadjah Mada – Dr. Sardjito Hospital, Radiopoetro Building 2nd Floor, Jalan Farmako Sekip Utara, Yogyakarta, Indonesia 55281

Manuscript submitted: April 26, 2019; Revised and accepted: June 21, 2019

ABSTRACT

Coronary slow flow is a phenomenon that is found in coronary angiography.It is a rare case and identified by the corrected TIMI frame count. The presence of a slow flow in the coronary arteries is associated with life-threatening arrhythmias, sudden death, and acute coronary syndrome. We aim to report a coronary slow flow phenomenon present with non-ST-elevation acute myocardial infarction and sustained slow ventricular tachycardia in 66-year-old male patient. Brief heparinisation and continued by oral acetosal, ticagrelor, bisoprolol and atorvastatin therapy successfully diminish the symptom.

Keywords: coronary slow flow phenomenon;ventricular tachycardia; non-ST-elevation acute myocardial infarction

INTISARI

Aliran lambat koroner adalah fenomena yang ditemukan dalam pemeriksaan angiografi koroner. Fenomena ini jarang dijumpai dan diidentifikasi melalui jumlah frame TIMI terkoreksi. Munculnya fenomena aliran lambat di arteri koroner berhubungan dengan aritmia yang mengancam jiwa, kematian mendadak, dan sindrom koroner akut. Kami bermaksud melaporkan fenomena aliran lambat koroner yang bermanifes sebagai infark miokard akut non-ST-elevasi dan takikardia ventrikel lambat yang menetap pada pasien pria berusia 66 tahun. Pemberian heparinisasi dalam waktu singkat dan dilanjutkan dengan obat oral asetosal, ticagrelor, bisoprolol, dan atorvastatin berhasil mengurangi gejala.

INTRODUCTION

The coronary slow flow phenomenon (CSFP) is an angiographic finding typified by delayed opacification to the distal vasculature in the absence of obstructive epicardial coronary artery disease (CAD).[1] It is a rare phenomenon with the incidence around 1-7% among coronary angiograms.[1] The CSFP patients were mostly young males and have risk factors of smoking, hypertension and dyslipidaemia.[2] They often present with acute chest pain and ECG changes. An ischaemic origin in those with the CSFP suggest that T wave changes in ECG may be an indication of microvascular dysfunction.[3]

The significance of CSFP in patients with angina is unclear and therefore an effective treatment is unknown. Recently CSFP has been found to be associated with increased QTc dispersions, which may be an independent risk factor for ventricular arrhythmias and sudden cardiac death.[2]

Here we report the CSFP in male patient with clinical presentation of non-ST-elevation acute myocardial infarction (NSTEMI) and sustained slow ventricular tachycardia (VT).

CASE REPORT

A 66-year-old man came to the emergency unitof Dr. Sardjito Hospital, Yogyakarta with chief complaint of chest pain. Ten hours before coming to the emergency unit, he suffered from chest pain radiating to the right and left arms, accompanied with diaphoresis and abdominal dyscomfort. He tried to assuage the chest pain by taking analgetic remedy, however the pain persisted. The patient visited nearby hospital and was diagnosed as non-sustained VT. He was referred to the emergency unit of Dr. Sardjito Hospital. At the emergency unit, the chest pain was still presented but without diaphoresis, abdominal discomfort and shortness of breath. The patient had history of bronchial asthma and hypertension.

On physical examination, the patient was fully conscious, with blood pressure 177/107 mmHg, pulse rate 72 x/minute (regular), respiratory rate 20 x/minute, and axillae temperature 36.7℃. The JVP was 5+2 cmH_2O. At chest examination was unremarkable. Lung and heart examination were also normal. Abdominal and extremity examination were normal. An ECG examination from district hospital showed the sinus rhythm, right axis deviation and bigemini ventricle extrasystole (Figure 1). The QTc dispersion from the ECG was 120 msec (Figure 2).

An ECG examination on the emergency room at our hospital showed a sinus rhythm, right axis deviation, with periodic slow VT (Figure 3). The laboratory examination results were: haemoglobin level 17 g/dL, haematocrit 52.6%, platelet count 279,103 /µL, leukocyte count 10,6000 /µl, albumin level 5.02 g / dL, GOT level 158 U/L,GPT level 10 U/L, glucose level 95 mg/dL, urea nitrogen level 12 mg/dL, creatinine level 1.3 mg/dL, sodium level 137 mmol/L, potassium level 4.62 mmol/L, chloride level 101 mmol/L, calcium level 2.1 mmol/L, and magnesium level 2.28 mmol/L. Cardiac enzyme showed increasing level of CKMB (226 U/L) and hs-troponin I (38.455 ng/L).

The patient was assessed with NSTEMI Killip I with sustained slow VT (a very high risk). The patient was administered bolus and continuous infusion of unfractionated heparin (UFH), ticagrelor 180 mg and acetosal 320 mg. He was performed an immediate invasive strategy by coronary angiography, and the result showed: left main (LM) was normal; left anterior descendent (LAD) was ecstatic in the proximal part and 30% stenosis in the middle part with slow flow; left circumflex artery (LCx) was 50% stenosis in the proximal part, thrombus in the distal part with slow flow; right coronary artery (RCA) was 30% stenosis in the middle part with slow flow (Figure 4).

No coronary intervention was performed to the patient. Because of high burden thrombus, optimal medical therapy with antithrombotic was decided. The patient was transferred to Intensive Cardiac Care Unit (ICCU) and received 600 U/hour of UFH, and oral treatment of acetosal 80 mg q.i.d., ticagrelor 90 mg b.i.d., atorvastatin 40 mg q.i.d., captopril 25 mg t.i.d. and bisoprolol 2.5 mg q.i.d.. The transthoracic echocardiography examination showedglobal LV systolic function (ejection fraction) was normal, i.e. 54%, segmental wall motion abnormalities were present, i.e. hypokinetic basal-mid inferoseptalwall, inferolateral basal-mid wall and inferior basal-mid wall.

DISCUSSION

We report a 66-year-old man with coronary angiography diagnosis of CAD non-significant with slow flow presenting with NSTEMI and sustained slow VT.In this case, by coronary angiography it can be detectedthe slowdown of contrast flow in all coronary arteries. As one example wasof the LAD, the number of frames from osteal to distal LAD was 65 with the corrected TIMI frame count(CTFC) of 38.

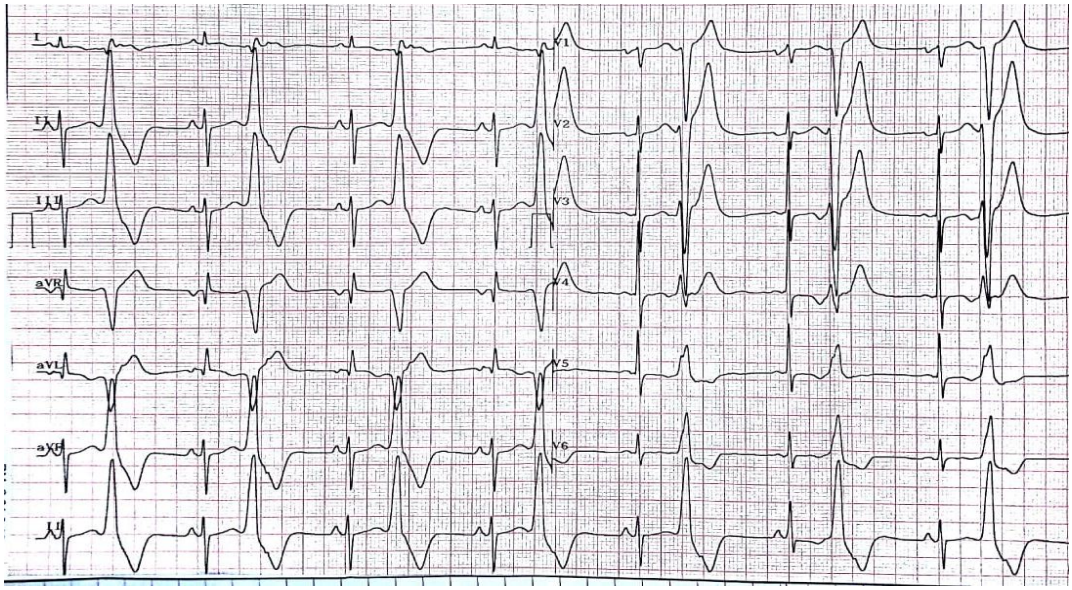


Figure 1.ECG examination in previous hospital showed sinus rhythm, right axis deviation, and bigemini pattern ventricle extrasystole.

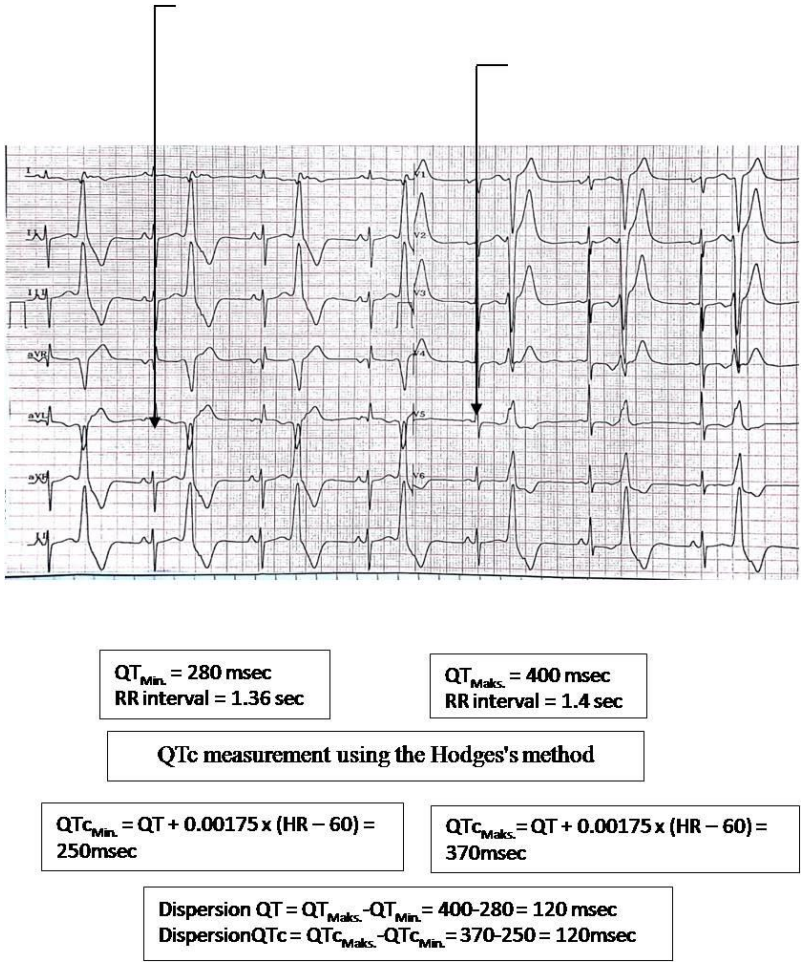


Figure 2. Twelve-Lead ECG at 50 mm/sec speed showing QT dispersion of 120 msec and QTc dispersion of 120 msec.

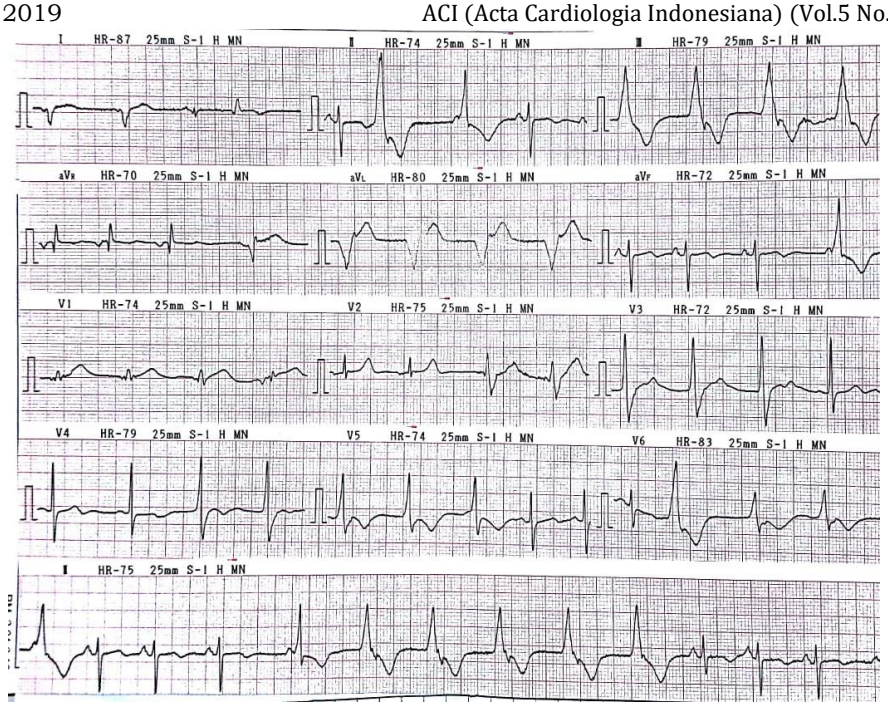


Figure 3. ECG examination in Dr. Sardjito hospital showed sinus rhythm and periodic slow ventricular tachycardia (VT).

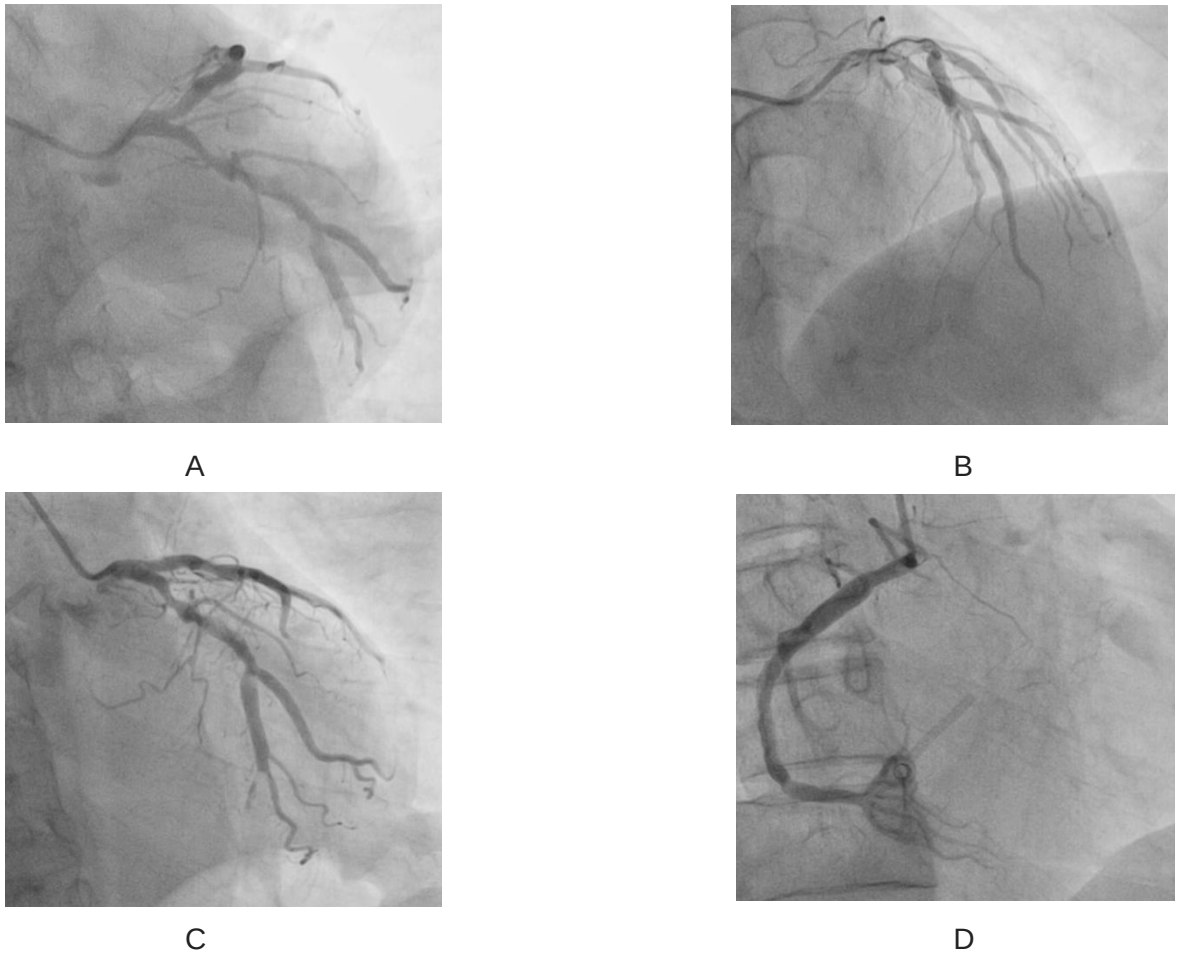
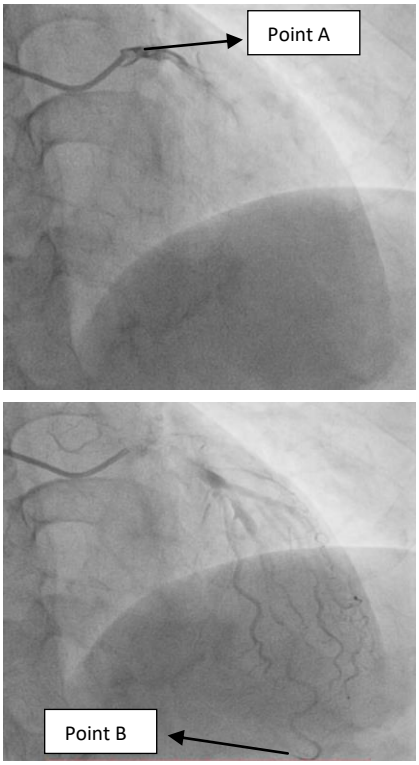


Figure 4. Coroangiography: A. Left Main (LM) : normal, B. Left Anterior Descenden (LAD) : proximal ectatic, 30% mid stenosis, slow flow,C. Left Circumflex artery (LCx) : 50% proximal stenosis, thrombus appears at distal, slow flow,D. Right Coronary Artery (RCA) : proximal ectatic, 30% mid stenosis, slow flow.



No. of frames from point A to B = 65

CTFC = 65 /1,7 = 38

Normal CTFC for LAD = 21 ± 2

Figure 5.Corrected TIMI frame count using the Gibson's method. Point A = the frame count when theostium of the vessel gets opacified. Point B = the frame count when the pre-specified most distalvessel segment gets opacified. CTFC = Corrected TIMI frame count. 1.7 is the correction factorused for the LAD due to its longer length.Corrected TIMI Frame Count in LAD which shows slow coronary flow.Number of frames from pointA to B = 65, CTFC = 65/1.7 = 38.Normal CTFC for LAD= 21±2

Table 1.Clinical criteria for suspecting microvascular angina (MVA)[2]

1. Symptoms of myocardial ischemia
   a. Effort and/or rest angina
   b. Angina equivalents (i.e. shortness of breath)
2. Absence of obstructive CAD (**<**50% diameter reduction or FFR **>**0.80) by
   a. Coronary CTA
   b. Invasive coronary angiography
3. Objective evidence of myocardial ischemia
   a. Ischemic ECG changes during an episode of chest pain
   b. Stress-induced chest pain and/or ischemic ECG changes in the presence or absence of transient/reversible abnormal myocardial perfusion and/or wall motion abnormality
4. Evidence of impaired coronary microvascular function
   a. Impaired coronary flow reserve (cut-off values depending on methodology use between **≤**2.0 and **≤**2.5)
   b. Coronary microvascular spasm, defined as reproduction of symptoms, ischemicECG shifts but no epicardial spasm during acetylcholine testing.
   c. Abnormal coronary microvascular resistance indices (e.g. IMR **>**25)
   d. Coronary slow flow phenomenon, defined as TIMI frame count **>**25.

The normal CTFC value was 21±2. It indicated that there was a slowdown in contrast flow of the LAD (Figure 5).

The CSFP in coronary angiographic studies was initially described subjectively by visual judgment. A semi-quantitative assessment of coronary blood flow is the thrombolysis TIMI flow grade classification, which reflects the speed and completeness of the passage of the injected contrast through the coronary arteries.[4]

This broadly used technique to grade coronary flow has variability in the visual assessment which limits the clinical applicability. The CTFC is an objective quantitative index of coronary flow and assists the standardization of TIMI flow grades and flow assessment.[5]It representsthe number of cineframes required for contrast to arrive at prespecified distal coronary artery landmarks.[5] A CSFP is defined as a CTFC greater than 2 standard deviations (SD) from the normal published range,which is 21±3.5.[5]

The precise pathogenesis of CSFP is still unclear. Multifactorial involvement have been implicated in the pathogenesis, such as microvasculature functional and structural abnormality, endothelial dysfunction, chronic inflammation, occult coronary atherosclerosis and epicardial coronary artery anatomy pattern.[1] Most importantly, CSFP was associated with life-threatening arrhythmias and sudden cardiac death, probably due to increased QTc dispersion.[4]

The QTc dispersion is defined as the difference between the maximum and minimum QTc interval in a standard 12-lead ECG, which indicates the dispersion of ventricular repolarization. Increased QTc dispersion has been linkedto increased incidence of ventricular arrhythmias and associated with an adverse prognosis in a varietyof patient populations.[6] Most patients with the CSFP demonstrate the ST and T wave changes during an ACS presentation.[3]

The strong association with T wave changes raises the possibility that the T wave may be a marker of coronary microvascular dysfunction (CMD).[3]

Standardization of diagnostic criteria for ischemic symptoms due to CMD is required to further investigation of patients presenting with angina consistent with microvascular angina (MVA).[2] There isclinical criteria for suspecting MVA (Table 1).

Based on the criteria, a definitive MVA is diagnosed if all four criteria are present for a diagnosis of MVA and a suspected MVA is diagnosed if symptoms of ischemia are present (criteria-1) with no obstructive coronary artery disease (criteria-2) but only (a) objective evidence ofmyocardialischemia (criteria-3), or (b) evidence of impaired coronary microvascular function(criteria-4) alone.[2] Based on the criteria, ourcase hada definitive MVA due to CMD.

Several drugs have been tested to treat CSFP. Dipyridamole, a platelet cAMP-phosphodiesterase inhibitor, has been shown to increase the extracellular adenosine level by inhibiting its re-uptakeby erythrocytes and vascular endothelium in the coronary arteries, thus causing coronary arterial dilation.[6]

Oral dipyridamole therapy has been shown to normalize the flow in patients with CSFP.[6] Other drugs such as statins for its pleiotropic effects on the vascular function, angiotensin-converting enzyme inhibitors for its direct modulation of coronary microvascular tone, α-blockers for its ability to decrease sympathetic activity and nitric oxide potentiating β-blocker nebivolol for its effect on improving flow mediated dilatation of brachial artery and reduced chest pain.[1]

In our case, the continued medication with oral acetylsalicylic acid, ticagrelor, bisoprolol and atorvastatin therapy successfully diminish the symptom.

CONCLUSION

We report a case of 66-year-old man with coronary angiography diagnosis of CAD non-significant with slow flow presenting with NSTEMI and sustained slow VT. In this rare case, he responded wellto an antiplatelet, β-blocker and statin therapy.

REFERENCES

1. Mullasari A., Victor S.M. 2013. Coronary slow flow phenomenon. E-journal of Cardiology Practice, 11:25.
2. Ong P., Camici P.G., Beltrame J.F., Crea F., Shimokawa H., Sechtem U. 2018. International standardization of diagnostic criteria for mivrovascular angina. Int J Cardiol, 250:16-20.
3. Cutri N., Zeitz C., Kucia A.M., Beltrame J.F. 2011. ST/T wave changes during acute coronary syndrome presentation in patients with the coronary slow flow phenomenon. Int J Cardiol, 146: 457-458.
4. Wang X., Nie S.P. 2011. The coronary slow flow phenomenon: characteristics, mechanisms and implications. Cardiovasc Diagn Ther, 1:37-43.
5. Gibson C.M., Cannon C.P., Daley W.L., Dodge J.T.Jr., Alexander B.Jr., Marble S.J. et al. 1996.TIMI frame count: a quantitative method of assessing coronary artery flow. Circulation, 93:879-888.
6. Saya S., Hennebry T.A., Lozano P., Lazzara R., Schechter E. 2008. Coronary slow flow phenomenon and risk for sudden cardiac death due to ventricular arrhythmias: a case report and review of literature. Clin Cardiol, 31(8):352-325.