IMJM

ISSN 1409-6366 UDC 61 Vol · 21 (1) · 2016

Original scientific paper

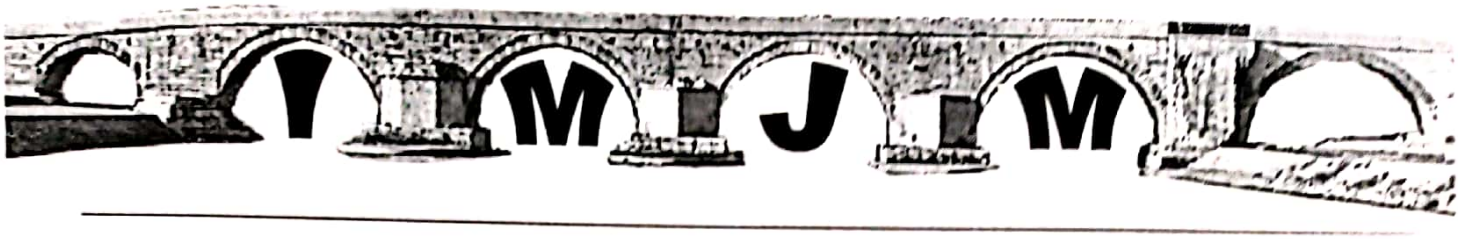
Review

Case report

Brief communications

Editorial Board

Medical Journal



ISSN 1409-6366 UDC 61 Vol · 21 (1) · 2016

Revistë Shkencore Nderkombëtare e Shoqatës së Mjekëve Shqiptarë të Maqedonisë
International Journal of Medical Sciences of the Association of the Albanian Doctors from Macedonia

Botues/ Publisher: **SHMSHM / AAMD**
Tel. i Kryeredaktorit / Contact: **+389 (0)31 25 044**
Zhiro llogaria / drawing account: **200-000031528193**
Numri tatimor / tax number: **4028999123208**
Adresa e Redaksisë-Editorial Board Address: **50 Divizija, No 6, 1000 Shkup**
e-mail: **medicus.shmshm@gmail.com**

Shtypur në
Shtypshkronjen "Pruf Print", Shkup

Medicus shtypet në tirazh: 600 ekzemplarë
Revista shpërndahet falas

Printed in:
Print House "Pruf Print", Skopje

The Jounal Medicus is printed and distributed free of charge with a circulation of 600 copies.

ANGINA PECTORIS WITH SLOW CORONARY FLOW PHENOMENON: A CASE REPORT

ANGINA PECTORIS СО БАВЕН КОРОНАРЕН ПРОТОК -ПРИКАЗ НА СЛУЧАЈ

Otljanska M., Boshev M.

University Clinic of Cardiology, Medical Faculty, University "Sts. Cyril and Methodius"-Skopje, Macedonia

Corresponding author: e-mail: magdalenaotljanska@gmail.com

Medicus 2015, Vol. 21 (1): 92-96

ABSTRACT

Objectives and background: Coronary slow flow phenomenon (CSFP) was identified as an exclusive clinical entity in 1972 [1] where the distal opacification of the coronary artery is delayed on angiography in the absence of significant coronary artery disease. It is a frequent finding, typically observed in patients presenting with acute coronary syndromes. Although it is well known to interventional cardiologists, but the pathogenic mechanisms remain unclear. The clinical implications are significant, with over 80% of patients experiencing recurrent chest pain, resulting in considerable impairment in quality of life.

Methods, procedures and results: We present a clinical case of 54 years old female patient complaining of recurrent chest pain. Physical examination showed non-specific signs. She had a history of high blood pressure, dislipidemia more than 5 years, and a genetic history of coronary artery disease. At the beginning she was treated with antihypertensive therapy with ACE inhibitors and beta blokers. We performed blood laboratory, ECG, echocardiography (ECHO), 24h ambulatory blood pressure monitoring (ABPM), coronary exercise stress test, coronary angiography and myocardial perfusion scintigraphy (MPS) after two years of hospitalisation. After coronary angiography, the diagnosis of slow coronary flow of LAD was confirmed. She was followed up two years after and treated with antihypertensive and antihyperlipemic therapy-statins and dipiridamol.

Conclusion: Coronary slow flow phenomenon is not an infrequent angiographic finding and contributes to morbidity. This phenomenon should be considered a separate clinical entity with peculiar characteristics, pathogenic mechanisms, and defined diagnostic criteria.

Keywords: coronary slow flow phenomenon, coronary artery, chest pain.

INTRODUCTION

Although a number of formal definitions have been proposed, the CSFP essentially consists of a delay in the progression of the contrast injected into the coronary arteries during coronary angiography [1, 2]. This condition, which may affect one or all coronaries, was originally described by Tambe *et al.* in 1972 [5]. Since then it has been accepted as an independent clinical entity, which is called "CSFP", "coronary slow flow syndrome" "syndrome Y", or "primary" coronary slow flow [6-9]. Importantly, "primary" CSFP should be distinguished from the delay in the contrast progression in the context of coronary reperfusion therapy such as angioplasty or stenting for acute myocardial infarction, or other "secondary" causes of coronary slow flow [8-10], coronary ectasia or spasm, ventricular dysfunction, valvular heart disease and connective tissue disorders. Incidence of coronary slow-flow is reported to be 1-7% of all coronary angiograms.

Clinically, this phenomenon occurs most commonly in young men and smokers, and patient admitted with acute coronary syndrome [12]. The clinical course is complicated, with over 80% of patients experiencing recurrent chest pain, most occurring at rest, necessitating readmission to the coronary care unit in almost 20% of affected patients [12]. Most importantly, coronary slow flow has been described to be associated with life-threatening arrhythmias and sudden cardiac death [3, 4], probably due to increased QTc dispersion in these patients.

Further, Yilmaz *et al.* [14] recently delineated the clinical and laboratory features of CSFP compared to the control subjects without CSFP. Metabolic syndrome was more frequent in CSFP in the presence of higher total cholesterol, low-density lipoprotein-cholesterol, fasting glucose and body mass index levels. These data are in line with the observations that insulin resistant states [15] and impaired glucose tolerance [16] correlate with CSFP occurrence. These data suggest that a common underlying pathophysiologic mechanism of the metabolic syndrome and CSFP may be endothelial dysfunction.

Diagnosis and evaluation of CSFP in coronary angiographic studies was initially described subjectively by visual judgement [5]. A semi-quantitative assessment of coronary blood flow is the thrombolysis in myocardial infarction (TIMI) flow grade classification, which reflects the speed and completeness of the passage of the injected contrast through the coronary tree [17,19]. Although this widely used method of grading coronary flow has been a valuable tool for comparison of flow data in clinical trials, variability in the visual assessment may limit the broad clinical applicability. In contrast, as an objective, quantitative index of coronary flow, corrected TIMI frame count (CTFC) facilitates the standardization of TIMI flow grades and flow assessment. It represents the number of cine-frames required for contrast to first reach standard distal coronary landmarks [18]. Currently, by using CTFC as a quantitative index of coronary flow, coronary angiography is the only tool for the diagnosis and assessment of CSFP. Despite good prognosis of CSFP patients, the subsequent progress is frequently characterized by remitting, relapsing anginal episodes resulting in considerable impairment in quality of life. Unfortunately, currently available anti-anginal agents are of limited clinical value. It was shown that dipyridamole and mibefradil, which both influence functional obstruction in arteries <200 μm, normalized CTFC but nitroglycerine, which dilates arteries with diameters >200 μm, did not [20,21]. Importantly, statins appear beneficial for patients with CSFP, likely in part due to their anti-inflammatory properties [22-24]. More recently, several studies demonstrated that nebivolol can both improve endothelial function and markedly ameliorate symptoms, thereby improving quality of life in patients with CSFP [25-27]. Besides its beta-receptor blocking activity, nebivolol can cause endothelium-dependent vasodilatation through increased nitric oxide release [25].

CLINICAL PRESENTATION

A 54-year-old female was admitted to the cardiology department with a history of exertional angina for one year duration and past medical history of hyperlipidemia and hypertension. Chest pain was described as a pressure-like sensation in the middle of her chest. Right upon admission vital signs were stable. Her blood pressure was 150/90 mmHg, heart rate was 94/min, and heart sounds were normal. Systematic examination showed no further significant symptoms. There was a family history of coronary artery disease, her father died from myocardial infarction. She was not a smoker. The resting 12-lead ECG was unremarkable. (Fig.1). We performed ABPM (Fig.2) and results showed that blood pressure was not well controlled.

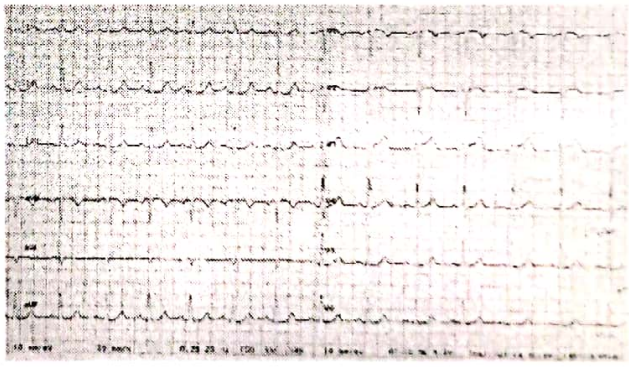


Fig.1 ECG on admission

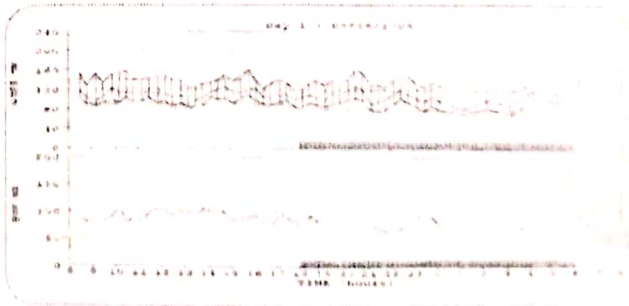


Fig.2. ABPM

Transthoracic echocardiographic evaluation showed a normal left ventricular function and no regional wall motion abnormality. The patient underwent a

treadmill exercise stress test using the Bruce protocol. He experienced typical angina at 6 min of exercise, with nearly a 2 mm ST segment depression in V4-V6 derivations and coronary test was interpreted as a positive (Fig 3).

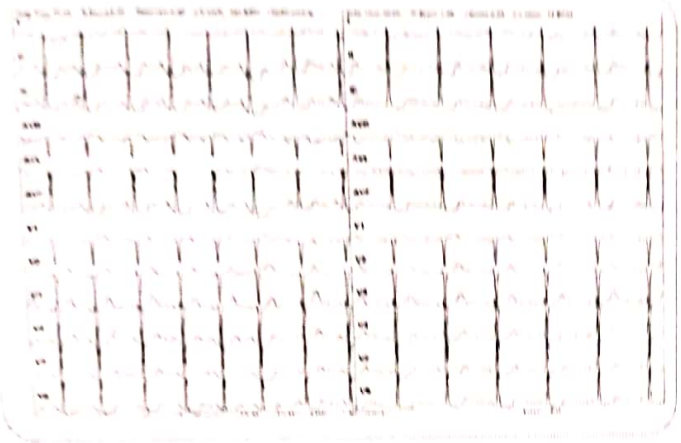


Fig. 3 ECG changes (V4-V6) during exersice coronary test

Coronary angiography was performed for suspected coronary artery disease. During left heart catheterization, systemic arterial pressure was normal and there was no gradient across the aortic valve. A left ventricular angiogram obtained in the right anterior oblique view revealed no regional wall motion abnormality. The Thrombolysis in Myocardial Infarction (TIMI) frame-count method was used to evaluate the degree of the slow antegrade filling. The corrected TIMI frame counts were observed to be 41 frames for the left anterior descending coronary artery (LAD) (Fig 4, 5). Coronary angiography revealed no stenosis of the right coronary artery (RCA) and left circumflex coronary artery (LCx). The angiogram showed normal coronary arteries without evidence of coronary vasospasm or an existing myocardial bridge. Slow flow, however, was noted in the left anterior descending artery (LAD). After 4 days of hospitalization patient was discharged from the hospital with advise for regular use of antihypertensive therapy, statins, dipiridamol and regular controls.

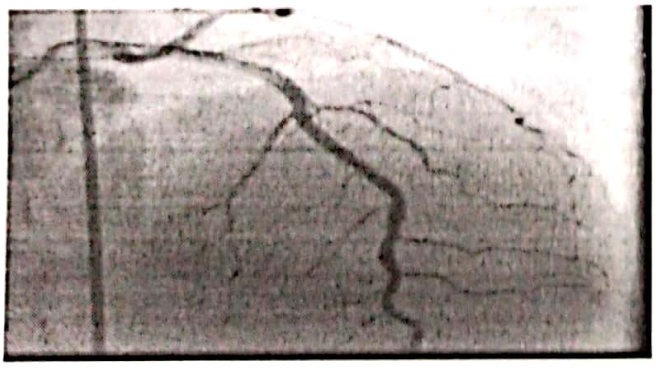


Fig.4 Coronary anatomy and flow of LAD

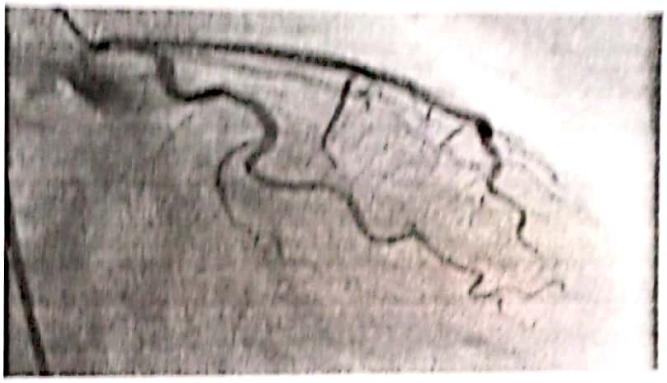


Fig.5 Coronary anatomy and flow of LAD

After two years of follow up and regular ambulatory check ups, patient continued to have occasional chest pain and we have decided to perform MPS. Myocardial perfusion scintigraphy showed reversible perfusion abnormalities of apical segment of anterior wall (6%) with good ejection fraction (Fig.6, Fig 7).

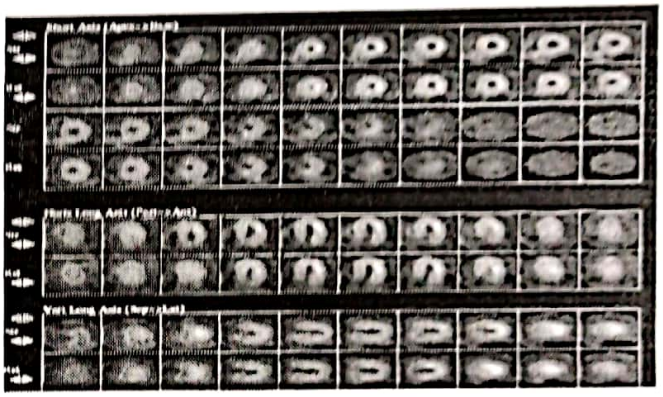


Fig .6 Segment analysis during MPS

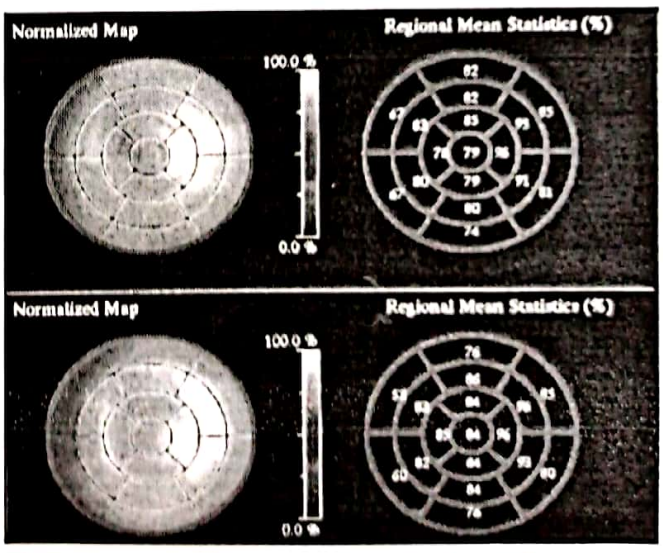


Fig.7 Regional wall abnormalities during MPS

DISCUSSION

This is a case of female patient with coronary slow flow phenomenon which is manifested with chest pain and

need further investigations to determine its etiology. Past medical history of dislipidemia and hypertension as well as family history of coronary artery disease increase her total risk for coronary artery disease development. She was treated with antihypertensive therapy, statins and occasionally aspirin. Clinical investigations especially treadmill exercise stress test was positive without regional wall motion abnormality on transthoracic echocardiographic evaluation. Coronary angiography showed normal coronary arteries without evidence of coronary vasospasm or significant stenosis but slow flow was noted in the left anterior descending artery (LAD). The possible pathophysiological mechanism for CSFP in our case may be a small vessel dysfunction based on observations including microvascular tone dysfunction, endothelial thickening of the small vessels, and impaired endothelial release of nitric oxide (NO). Patient was treated with regular medical therapy consisting of antihypertensive medications, statins and dipiridamol and further follow up showed improvement in her exercise tolerance.

CONCLUSION

Coronary slow flow phenomenon is not an infrequent angiographic finding and it contributes to higher cardiovascular morbidity. Coronary slow flow phenomenon usually has a benign long term outcome but may be associated with relapses. Occasional ventricular arrhythmias and even sudden cardiac death have been reported. Treatment modalities for CSFP are not well established and further studies are necessary.

REFERENCES

1. Cutri N, Zeitz C, Kucia AM, et al. ST/T wave changes during acute coronary syndrome presentation in patients with the coronary slow flow phenomenon. Int J Cardiol 2011;146:457-8.
2. Horjeti B, Goda A. Acute ischemia manifestation in a patient with coronary slow flow phenomenon. J Electrocardiol 2012;45:277-9.
3. Wozakowska-Kapłon B, Niedziela J, Krzyzak P, et al. Clinical manifestations of slow coronary flow from acute coronary syndrome to serious arrhythmias. Cardiol J 2009;16:462-8.
4. Saya S, Hennebry TA, Lozano P, et al. Coronary slow flow phenomenon and risk for sudden cardiac death due to ventricular arrhythmias: a case report and review of literature. Clin Cardiol 2008;31:352-5.
5. Tambe AA, Demany MA, Zimmerman HA, et al. Angina pectoris and slow flow velocity of dye in coronary arteries--a new angiographic finding. Am Heart J 1972;84:66-71.
6. Goel PK, Gupta SK, Agarwal A, et al. Slow coronary flow: a distinct angiographic subgroup in syndrome X. Angiology 2001;52:507-14.
7. Li JJ, Wu YJ, Qin XW. Should slow coronary flow be considered as a coronary syndrome? Med Hypotheses 2006;66:953-6.
8. Leone MC, Gori T, Fineschi M. The coronary slow flow phenomenon: a new cardiac "Y" syndrome? Clin Hemorheol Microcirc 2008;39:185-90.
9. Fineschi M, Gori T. Coronary slow-flow phenomenon or syndrome Y: a microvascular angina awaiting recognition. J Am Coll Cardiol 2010;56:239-40.
10. Gori T, Fineschi M. Two Coronary "Orphan" Diseases in Search of Clinical Consideration: Coronary Syndromes X and Y.Cardiovasc Ther 2011.
11. Mangieri E, Macchiarelli G, Ciavolella M, et al. Slow coronary flow: clinical and histopathological features in patients with otherwise normal epicardial coronary arteries. Cathet Cardiovasc Diagn 1996;37:375-81.
12. Beltrame JF, Limaye SB, Horowitz JD. The coronary slow flow phenomenon--a new coronary microvascular disorder. Cardiology 2002;97:197-202.
13. Atak R, Turhan H, Sezgin AT, et al. Effects of slow coronary artery flow on QT interval duration and dispersion. Ann Noninvasive Electrocardiol 2003;8:107-11.
14. Yilmaz H, Demir I, Uyar Z. Clinical and coronary angiographic characteristics of patients with coronary slow flow. Acta Cardiol 2008;63:579-84.
15. Ozcan T, Gen R, Akbay E, et al. The correlation of thrombolysis in myocardial infarction frame count with insulin resistance in patients with slow coronary flow. Coron Artery Dis 2008;19:591-5.
16. Binak E, Gunduz H, Sahin M, et al. The relation between impaired glucose tolerance and slow coronary flow. Int J Cardiol 2006;111:142-6.
17. Chesebro JH, Knatterud G, Roberts R, et al. Thrombolysis in Myocardial Infarction (TIMI) Trial, Phase I: A comparison between intravenous tissue plasminogen activator and intravenous streptokinase. Clinical findings through hospital discharge. Circulation 1987;76:142-54.
18. Gibson CM, Cannon CP, Daley WL, et al. TIMI frame count: a quantitative method of assessing coronary artery flow. Circulation 1996;93:879-88.

19. Tani T, Tanabe K, Kitai T, et al. Detection of severe stenosis and total occlusion in the left anterior descending coronary artery with transthoracic Doppler echocardiography in the emergency room. Echocardiography 2009;26:15-20.
20. Kurtoglu N, Akcay A, Dindar I. Usefulness of oral dipyridamole therapy for angiographic slow coronary artery flow. Am J Cardiol 2001;87:777-9.
21. Beltrame JF, Turner SP, Leslie SL, et al. The angiographic and clinical benefits of mibefradil in the coronary slow flow phenomenon. J Am Coll Cardiol 2004;44:57-62.
22. Cakmak M, Tanriverdi H, Cakmak N, et al. Simvastatin may improve myocardial perfusion abnormality in slow coronary flow. Cardiology 2008;110:39-44.
23. Li JJ, Zheng X, Li J. Statins may be beneficial for patients with slow coronary flow syndrome due to its anti-inflammatory property. Med Hypotheses 2007;69:333-7.
24. Caliskan M, Erdogan D, Gullu H, et al. Effects of atorvastatin on coronary flow reserve in patients with slow coronary flow. Clin Cardiol 2007;30:475-9.
25. Gunes Y, Tuncer M, Guntekin U, et al. Regional functions of the left ventricle in patients with coronary slow flow and the effects of nebivolol. Ther Adv Cardiovasc Dis 2009;3:441-6
26. Fragasso G. Nebivolol in patients with coronary slow flow: the right drug for the right case? Anadolu Kardiyol Derg 2009;9:296-7.
27. Albayrak S, Ordu S, Yuksel H, et al. Efficacy of nebivolol on flow-mediated dilation in patients with slow coronary flow. Int Heart J 2009;50:545-53.

ANGINA *PECTORIS* СО БАВЕН КОРОНАРЕН ПРОТОК -ПРИКАЗ НА СЛУЧАЈ

Отланска М.,Бошев М.

Универзитетска клина за кардиологија, Медицински Факултет „Св.Кирил и Методиј" Скопје, Република Македонија

Автор за кореспонденција: e-mail: magdalenaotljanska@gmail.com

АБСТРАКТ

Цели: Феноменот на бавен коронарен проток е идентификуван како посебен клинички ентитет во 1972 кога дисталната опацификација на коронарната артерија е одложена на ангиографија во отсуство на значајна коронарна артериска болест.Тоа е чест наод, типичен кај пациенти кој имаат презентација на акутен коронарен синдром. Иако овој ентитет е добро познат кај интервентите кардиолози,патолошкиот механизам останува нерасен. Клиничките имплицации се значајни,со повеке од 80% од пациентите имаат повторувачки епизоди на градни болки резултирајќи со значајно нарушување на квалитетот на живот.

Методи, процедури и резултати: Презентираме клинички случај на пациент на 54 годишна возраст, женски пол кој се жали на повторувачки епизоди на градна болка. Физикалниот преглед е без специфични знаци. Пациентката има историја на покачени вредности на крвен притисок, дислипидемија повеке од 5 години генетска предиспозиција за коронарна артериска болест. Направивме лабораториски испитување, ЕКГ ехокардиографија, 24 часовен холтер за крвен притисок, коронарен стрес тест, коронарна ангиографија и миокардна перфузиона сцинтиграфија после две години од хоспитализација. По направената коронарна ангиографија дијагнозата за феномен на бавен коронарен проток на LAD е потврдена. Пациентката беше следена две години и беше поставена на антихипертензивна, антилипемична терапија-статини и дипиридамол.

Заклучок: Феноменот на бавен коронарен проток не е редок ангиографски наод и придонесува за морбидитет. Овој феномен треба да биде сметан како посебен клинички ентитет со специфични карактеристики, патофизиолошки механизми и дефинирани дијагностички критериуми.

Клучни зборови: феномен на бавен коронарен проток, коронарна артерија, градна болка