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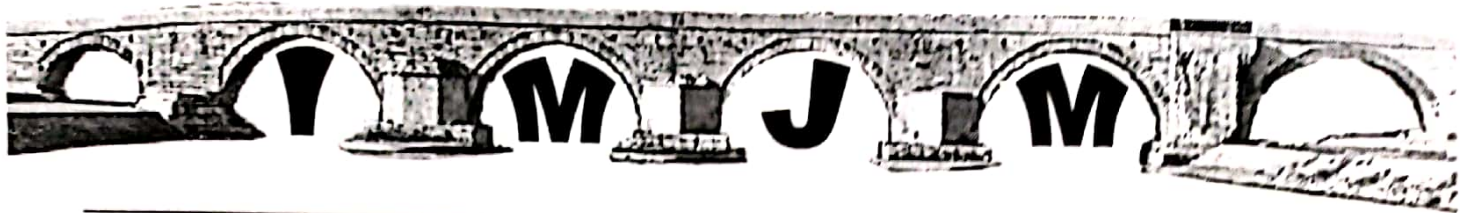
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ANGINA PECTORIS WITH SLOW CORONARY FLOW PHENOMENON: A CASE REPORT

ANGINA PECTORIS СО БАВЕИ КОРОНАРЕН ПРОТОК -ПРИКАЗ НА СЛУЧАЈ

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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, dyslipidemia 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 blockers. 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 dipyridamol.

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.

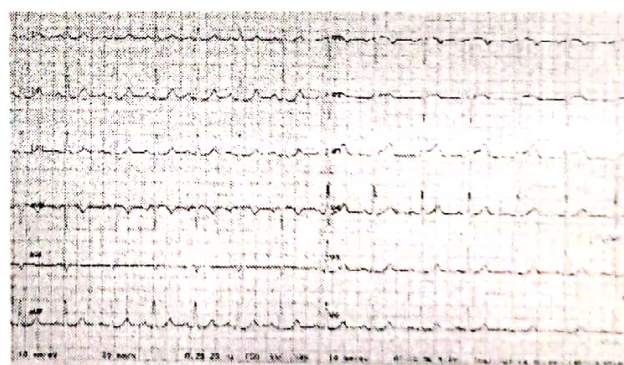


Fig.1 ECG on admission

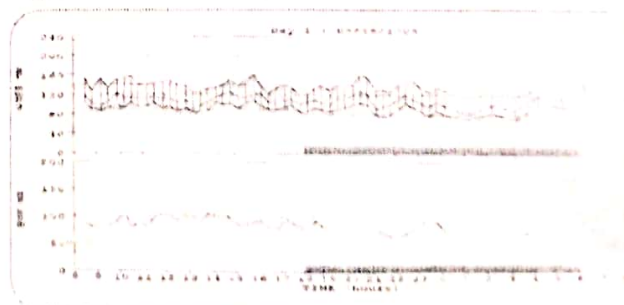


Fig.2. ABPM

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 5).

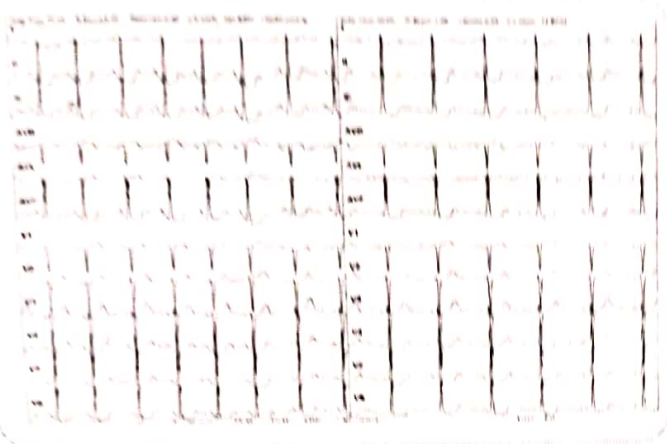


Fig. 5 ECG changes (V4-V6) during exercise coronary test

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).

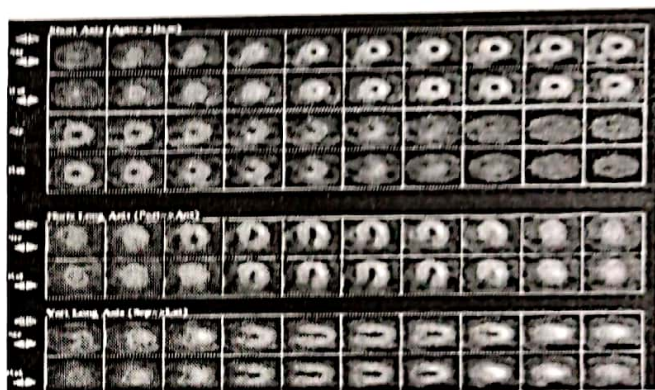


Fig.6 Segment analysis during MPS

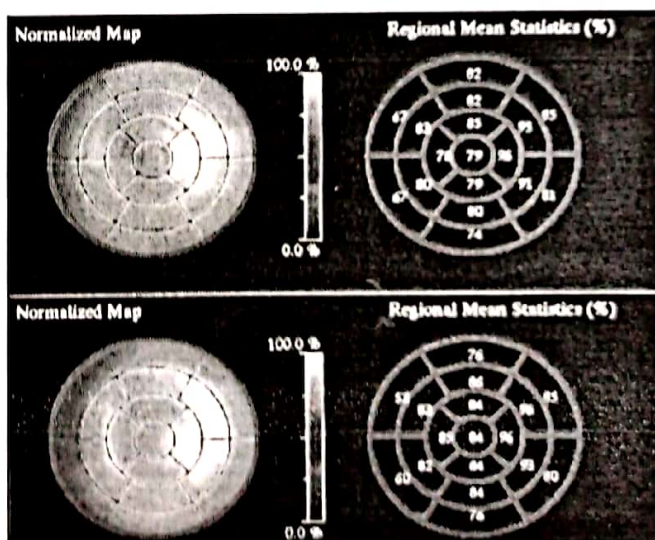


Fig.7 Regional wall abnormalities during MPS

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 dipyridamol 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.

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ANGINA PECTORIS СО БАВЕН КОРОНАРЕН ПРОТОК - ПРИКАЗ НА СЛУЧАЈ

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АБСТРАКТ

Цели: Феноменот на бавен коронарен проток е идентификуван како посебен клинички ентитет во 1972 кога дисталната опашкафикација 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 интервенитите кардиолози, патолошкиот механизам останува нерасен. Клиничките импликации се значајни, со повеќе 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 повеќе од 5 години, тешката предиспозиција за коронарна артериска болест. Направивме лабораториски испитување, ЕКГ, ехокардиографија, 24 часовен холтер за крвен притисок, коронарен стрес тест, коронарна ангиографија и миокардна перфузиона спинографија после две години од хоспитализација. Но направената коронарна ангиографија дигнозата за феномен на бавен коронарен проток на LAD е потврдена. Пациентката беше следена две години и беше поставена на антихипертензивна, антидислипидична терапија-статини и динитрокорамол.

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 карактеристични патолошките механизми и дефинирани дијагностички критериуми.

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