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Predictors of coronary slow flow in stable coronary artery disease

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ABSTRACT

Background: Although several studies have shown that endothelial dysfunction and inflammation may play roles in coronary slow flow phenomenon (CSFP), its etiology and pathogenesis still unclear and based on this observation our aim was detection of clinical and laboratory predictors of primary CSFP judged by TIMI frame count in coronary angiography (CA) for stable coronary artery disease patients (SCAD).

Methods: Case control study included 120 patients with SCAD who underwent CA. They were classified into two groups: PCSF group and normal coronary flow group. All patients subjected to CBC, platelet count, leucocytic count (total and differential), platelet-to lymphocyte ratio (PLR), neutrophil-to lymphocyte ratio (NLR), uric acid, albumin, Hs.CRP levels and CA with TIMI frame count.

Results: Incidence of diabetic mellitus and smoking were significantly higher in PCSF compared to control ($P = 0.01$), also we found PLR, NLR, uric acid and Hs-CRP levels were significantly higher in PCSF compared to control ($P < 0.0001$). The sensitivity of PLR $\geq$ 150, NLR $\geq$ 2, albumin level $\leq$ 3.5 g/dl, uric acid $\geq$ 6 mg/dl and Hs.CRP $\geq$ 6 mg/L for prediction of CSFP were (83.3%, 90.0%, 50.0%, 76.7% and 83.3% respectively) while the specificity were (86.7%, 90.0%, 53.3%, 83.3% and 86.7%) respectively. The multivariate analysis showed NLR $\geq$ 2 as the only independent predictor of PSCF.

Conclusion: PCSF was common in smokers and diabetic patients and this phenomenon is associated with high PLR, NLR, Serum uric acid and Hs.CRP. The only independent predictor of CSFP was neutrophil-to lymphocyte ratio (NLR).

1. Introduction

The coronary slow flow phenomenon (CSFP) is an angiographic clinical entity, characterized by delayed distal vessel opacification in absence of significant epicardial coronary stenosis. CSFP has direct clinical implications, because it is linked to myocardial ischemia, life-threatening arrhythmias, sudden cardiac death, and recurrent acute coronary syndromes. However, in our clinical practice we underestimate the consequences of CSFP due to many factors like unclear mechanism, relative rarity and difficulties in conducting clinically randomized trials to evaluate treatment options.[1]

Primary CSFP should be differentiated from secondary CSFP which defined as delay in the contrast progression in the case of coronary angioplasty and/or stenting for acute STEMI, or other causes such as coronary artery ectasia, coronary artery spasm, valvular heart disease, or connective tissue disorders.[2]

The coronary slow flow phenomenon was reported in 1–7% of patients underwent coronary angiography for suspected coronary heart disease. Although several studies showed that endothelial dysfunction, microvascular disease, inflammation and increased platelet activation may play a role in CSFP, the etiology and pathogenesis of this condition is still unclear.[3]

Several clinical and laboratory conditions were found as predictors of coronary slow flow like platelet-to lymphocyte ratio (PLR) in which increased activation of platelets has a marked effect in atherosclerosis initiation and progression.[4] High serum uric acid, C-reactive protein and low serum albumin levels were known as predictors and independent risk factors for cardiovascular events, coronary heart disease and coronary slow flow.[5]

Previous studies have suggested that a combination of morphological and functional abnormalities in small vessels and epicardial coronary arteries may explain the pathogenesis of CSFP. CSFP frequently associated with multiple vascular abnormalities and this feature probably require special consideration.[6]

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1.1. Objective

To find predictors of CSFP based on clinical data and simple laboratory tests judged by calculation of TIMI Frame count in coronary angiography (CA) for patients with stable coronary artery disease (SCAD).

2. Patients and methods

2.1. Patients

Case control study conducted in Zagazig University Hospital during the period from December 2014 to December 2015. This study included 120 patients with stable coronary artery disease (chronic stable angina) who underwent coronary angiography. They were classified into two groups: Primary coronary slow flow (PCSF) group (60 patients) and normal coronary flow (Control) group (60 patients). Patients with significant valvular heart disease, left ventricular systolic dysfunction (EF < 40%), old myocardial infarction, hypothyroidism, hyperthyroidism, hepatic insufficiency failure, renal impairment and previous coronary revascularization were excluded.

2.2. Methods

All patients were subjected to the followings:

1) Detailed medical history and clinical examination.
2) Resting 12 lead-ECG.
3) Laboratory investigation included complete blood picture, mean platelet count, total and differential leucocytic count and to detect (platelet-to lymphocyte ratio & neutrophil-to-lymphocyte ratio), serum uric acid level, serum albumin level and high sensitivity C reactive protein (Hs-CRP) level.
4) Coronary angiography, coronary blood flow was measured quantitatively using the TIMI frame count which was derived from the number of cine-frames (rate 30 f/sec) recorded from the first frame being the one in which >70% of the arterial lumen is filled with dye and the last frame the one in which dye first appears in the landmark. The last frames used for the LAD, LCX and RCA were those in which the dye first entered the mustache segment, the most distal obtuse marginal bifurcation and first branch of the posterolateral artery, respectively. The TIMI frame count of the LAD artery was corrected by dividing the final count by 1.7, LAD corrected frame count (CTFC) to compensate for the LAD length compared to LCX and RCA (Fig. 1).[7]

The first counting frame (frame 1) is the image where the contrast advances and fills at least 70% of the diameter of the arterial ostium. The last frame (final frame) is the image where the contrast begins to fill the final landmark.[7]

Figs. 2 and 3 showed TIMI frame count for one of our index study cases.

2.3. Statistical analysis

SPSS 20 (SPSS Science, Chicago, IL, USA) for Windows was used for statistical analysis. Continuous variables are presented as mean ± standard deviation (SD), and categorical variables as percentages. Comparison of categorical and continuous variables between the two groups was performed using chi-square test and independent sample *t* test, respectively. Linear regression analysis was used to test univariate relations. A *P* value < 0.05 was considered statistically significant.

3. Results

We found no significnat difference as regrad age and gender between both groups, however the prevalence of diabetis mellitus and smoking in the PCSF group were significantly higher compared to control group (53.4% vs 26.7%, *P* = 0.03 & 50.0% vs 20.0% *P* = 0.01) respectively (Table 1).

We found statistically significant diference as regard platelets-to lymphocytes ratio (PLR), neutrophils-to lymphocytes ratio (NLR), serum uric acid level and Hs.CRP between PCSF and control group (244.01 ± 91.62 vs 111.93 ± 37.96), (4.70 ± 2.16 vs 1.44 ± 0.77), (7.37 ± 1.74 vs 5.01 ± 1.40) and (7.32 ± 2.08 vs 4.17 ± 1.81) respectively (*P* < 0.0001). There was no statistically significant difference between both groups as regard the serum albumin level (Table 2).

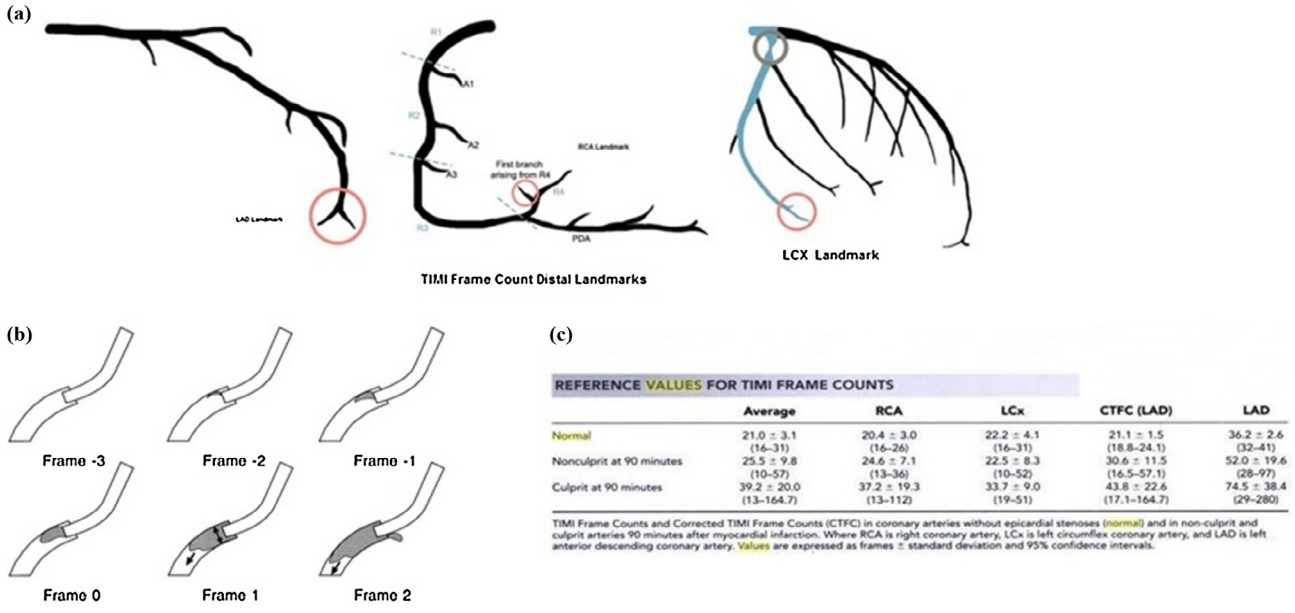

Fig. 1. TIMI frame count for normal and stenotic coronary arteries distal landmark (1a), 1st frame counting point (1b) and normal reference values (1c).