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ORIGINAL ARTICLE

Serum visfatin and omentin levels in slow coronary flow

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KEYWORDS
Slow coronary flow;
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Abstract
Objective: The adipocytokines visfatin and omentin have a direct effect on inflammation and endothelial injury. The expression of visfatin is closely associated with the expression of proinflammatory cytokines. Omentin has an anti-inflammatory effect and is inversely associated with coronary artery disease (CAD). The slow coronary flow phenomenon is an angiographic finding characterized by delayed distal vessel opacification in the absence of significant epicardial coronary disease. The pathophysiology of SCF has not been clearly identified, although multiple abnormalities including endothelial dysfunction, atherothrombosis and inflammation have been reported. However, the relationship between visfatin, omentin and SCF is still unknown. In this study, we aimed to investigate the relationship of these adipocytokines with SCF.
Methods: The study included slow coronary flow (n=45) and normal coronary flow (n=55) subjects, according to the corrected TIMI frame count, who underwent angiography in the catheterization laboratory of Duzce University. Statistical analyses were performed with SPSS version 12.
Results: Visfatin levels were significantly higher in patients with SCF than in controls (p<0.001). Plasma omentin levels were lower in the SCF group than in controls, although without statistical significance. Visfatin, gender and platelet count were significant predictors of SCF in multivariate logistic regression analysis (OR 0.748, 95% CI 0.632–0.886, p=0.01; OR 30.016, 95% CI 4.355–206.8, p=0.01; OR1.028, 95% CI 1.006–1.050, p=0.011, respectively).
Conclusion: Adipocytokines such as visfatin and omentin may play a role in the pathogenesis of coronary slow flow.

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PALAVRAS-CHAVE
Fluxo coronário lento;
Visfatina;
Omentin

Níveis séricos de visfatina e omentin no fluxo coronário lento

Resumo
Objetivo: A visfatina e o omentin são adipocitocinas e têm um efeito direto sobre a inflamação e a lesão endotelial. A expressão da visfatina está intimamente associada com a expressão de citoquinas pró-inflamatórias. O omentin tem efeito anti-inflamatório e está inversamente associado com a doença coronária (DC). O fenómeno do fluxo coronário lento (FCL) é um achado angiográfico caracterizada por atraso de opacificação distal na ausência de doença coronária epicárdica significativa. A fisiopatologia do FCL não está claramente identificada, apesar de terem sido relatadas várias alterações, incluindo disfunção endotelial, aterotrombose e inflamação. No entanto, a relação entre visfatina, o omentin e a FCL ainda é desconhecida. Neste estudo, procurou-se investigar essas relações das adipocitocinas com o FCL.
Métodos: No estudo foram incluídos indivíduos com fluxo coronário lento (n = 45) e fluxo coronário normal (n = 55) de acordo com a contagem corrigida de quadros TIMI, que necessitavam de angiografia no laboratório de cateterismo da Universidade de Duzce. As análises estatísticas foram realizadas com o programa estatístico SPSS 12.
Resultados: Os níveis de visfatina foram significativamente mais altos em pacientes com FCL do que nos do grupo controle (p < 0,001). Os níveis plasmáticos de omentin foram menores no grupo FCL do que nos controles, embora sem significado estatístico. Numa análise de regressão logística multivariada a visfatina, o género e a contagem de plaquetas foram definidos como preditores significativos da FCL (respetivamente, OR 0,748, IC 95% 0,632-0,886, p = 0,01; OR 30,016, 95% CI 4,355-206,8, p = 0,01 e OR 1.028, IC 95% 1,006-1,050, p = 0,011).
Conclusão: As adipocitocinas, por exemplo a visfatina e o omentin, podem desempenhar um papel na patogénese do fluxo coronário lento.

Introduction

The slow coronary flow (SCF) phenomenon was identified as a discrete clinical entity in 1972[1] in which distal opacification of the coronary artery is delayed on angiography in the absence of significant coronary artery disease. Another feature of SCF is its frequent occurrence in association with more widespread vascular abnormalities. SCF is a frequent finding in patients presenting with acute coronary syndrome, usually unstable angina.

Although some underlying etiologies such as abnormally high microvascular resistance and widespread atherosclerosis of the coronary arteries have been proposed, the exact pathophysiological mechanism of this phenomenon remains unclear. In some patients, diffuse atherosclerosis or diffuse calcifications have been identified on intravascular ultrasound.[2–4] Other factors that may cause SCF are abnormalities in the coronary microcirculation, microvascular endothelial dysfunction and inflammation.[5]

Inflammation is controlled by various hormones and cytokines. Adipose tissue secretes a variety of adipocytokines, including leptin, adiponectin, visfatin, TNF-α, IL-6 and omentin, and it is considered an endocrine organ due to its effects on metabolism in several organs and systems.[6]

The inflammatory cytokine visfatin plays a key role in delayed neutrophil apoptosis in sepsis. It is highly enriched in the visceral fat of both humans and mice, and its plasma levels increase during the development of obesity,[7] as well as being elevated in patients with type 2 diabetes, suggesting that measurement of plasma visfatin can be a useful tool for understanding metabolic diseases.[8,9]

Omentin is a recently identified adipokine that is selectively expressed in visceral adipose tissue.[10,11] Recent studies have shown that omentin levels are negatively correlated with acute coronary syndrome and stable angina pectoris.[12] Plasma omentin levels correlate negatively with systolic blood pressure, hemoglobin A1C, body mass index (BMI) and total cholesterol levels, and positively with HDL cholesterol.[13]

The relationship between plasma levels of these adipocytokines and SCF has not been investigated. We aimed to investigate the relations of visfatin and omentin plasma levels with SCF.

Methods

Forty-five consecutive patients who had undergone coronary angiography in Duzce University School of Medicine Cardiology Clinic between June 2012 and January 2013 and were found to have slow coronary flow were included in the study. Fifty-five consecutive patients with completely normal coronary arteries were recruited as the control group. The indications for coronary angiography were stable angina and positive treadmill test. Patients with a history of congestive heart failure, CAD including spasm, plaque, or ectasia, valvular heart disease, hyperthyroidism, chronic obstructive pulmonary disease and patients with acute coronary syndrome were excluded from the study. Those with a BMI