Clin Investig Arterioscler. 2018;30(3):118–119

Sociedad Española de Arteriosclerosis

CLÍNICA E INVESTIGACIÓN EN ARTERIOSCLEROSIS

www.elsevier.es/arterio

EDITORIAL

Epicardial fat and cardiovascular disease☆

Grasa epicárdica y enfermedad cardiovascular

Vicente Lahera

Department of Physiology, Faculty of Medicine, Universidad Complutense de Madrid, Spain

Obesity and excess adipose tissue have been linked to cardiovascular disease.[1,2] Many studies have shown that visceral fat is linked to the development of atherosclerotic disease through indirect mechanisms of metabolic, inflammatory and oxidative origin as well as risk factors linked to obesity, such as hypertension, dyslipidaemia, diabetes and metabolic syndrome.[3,4]

Epicardial fat is a type of ectopic visceral adipose tissue located around the heart and along the main coronary branches. This fat is metabolically active and produces many inflammatory factors which may affect cardiac function and coronary circulation.[5] Various studies using echocardiography, computed tomography or magnetic resonance imaging to measure epicardial fat have shown a relationship between the amount of epicardial fat and decreased coronary perfusion. This may suggest a correlation with the development of coronary atherosclerosis.[6] Prospective studies have shown a relationship between pericardial fat and the onset of cardiovascular episodes. This supports the notion that pericardial fat seems to be involved in the pathogenesis and progression of atherosclerotic coronary artery disease.[6] Despite this evidence, no clear direct correlation between epicardial fat and the onset of atherothrombotic events has been shown.

The relationship between pericardial fat and coronary artery disease has mainly been attributed to paracrine mechanisms of the pericardial adipose tissue on the coronary arteries. However, it must be borne in mind that increased pericardial fat occurs within the context of increased visceral fact, which is mainly responsible for associated poor general metabolic regulation.[7] Epicardial fat has been shown to be closely correlated with body mass index, waist circumference, visceral fat, insulin resistance and metabolic syndrome.[7] This means that a generalised metabolic disorder could significantly contribute to the development of thrombotic events. It is obvious that pericardial fat has deleterious effects which contribute to coronary atherosclerosis. However, this may simply be a matter of a paraphenomenon indicating other cardiovascular risk factors linked to obesity. Therefore, total visceral fat may be a confounding factor for showing a relationship between pericardial fat and the development of thrombotic coronary events.

A study by Basurto Acevedo et al.[8] was based on various pieces of evidence indicating that patients with coronary artery disease have decreased fibrinolytic activity and increased epicardial adipose tissue thickness.[6,7] The study sought to determine the link between epicardial fat and fibrinolytic activity by measuring PAI-1 levels in women aged 45–60 years. The study results showed a correlation between serum PAI-1 levels and epicardial adipose tissue thickness measured by transthoracic echocardiography. A

DOI of original article:
https://doi.org/10.1016/j.arteri.2018.05.001

☆ Please cite this article as: Lahera V. Grasa epicárdica y enfermedad cardiovascular. Clin Invest Arterioscler. 2018;30:118–119.

E-mail address: vlahera@med.ucm.es

multivariate regression analysis indicated that epicardial fat independently predicts PAI-1 levels. In addition, epicardial fat pad thickness was correlated with glucose and triglyceride levels as well as insulin resistance, body mass index, visceral adipose tissue content and total body fat. This supports the notion that pericardial fat is linked to visceral fat and several metabolic disorders which may foster the onset of thrombotic events.

In summary, the results of a study by Basurto Acevedo et al.[8] and others support the idea that obesity and epicardial adipose tissue accumulation represent a significant risk factor for the development of cardiovascular disease and its complications. A better grasp of the biology, aetiology and associated risk of pericardial fat will contribute to better understanding the complex relationship between obesity and cardiovascular disease. Genetic studies may aid in elucidating the mechanisms, pathophysiological role and causes of epicardial fat accumulation. Indeed, some of these studies have shown a specific locus for ectopic fat not linked to visceral fat.[9] Specific studies simultaneously addressing different forms and appearances of cardiac adipose tissue will shed light on the independent or synergistic role of epicardial and visceral fat.

References

1. Mahabadi AA, Massaro JM, Rosito GA, Levy D, Murabito JM, Wolf PA, et al. Association of pericardial fat, intrathoracic fat, and visceral abdominal fat with cardiovascular disease burden: the Framingham Heart Study. Eur Heart J. 2009;30:850–6.
2. Berg AH, Scherer PE. Adipose tissue, inflammation, and cardiovascular disease. Circ Res. 2005;96:939–49.
3. Carey VJ, Walters EE, Colditz GA, Solomon CG, Willett WC, Rosner BA, et al. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. Nurs Study Am J Epidemiol. 1997;145:614–9.
4. Pou KM, Massaro JM, Hoffmann U, Vasan RS, Maurovich-Horvat P, Larson MG, et al. Visceral and subcutaneous adipose tissue volumes are cross-sectionally related to markers of inflammation and oxidative stress: the Framingham Heart Study. Circulation. 2007;116:1234–44.
6. Cheng KH, Chu CS, Lee KT, Lin TH, Hsieh CC, Chiu CC, et al. Adipocytokines and proinflammatory mediators from abdominal and epicardial adipose tissue in patients with coronary artery disease. Int J Obes. 2008;32:268–74.
5. Mazurek T, Zhang L, Zalewski A, Mannion JD, Diehl JT, Arafat H, et al. Human epicardial adipose tissue is a source of inflammatory mediators. Circulation. 2003;108:2460–6.
7. Fernández-Muñoz MJ, Basurto Acevedo L, Córdoba Pérez N, Vázquez Martínez AL, Tepach Gutiérrez N, Vega García S, et al. La grasa epicárdica se relaciona con la visceral, el síndrome metabólico y la resistencia a la insulina en mujeres menopáusicas. Rev Esp Cardiol. 2014;67:436–41.
8. Basurto Acevedo L, Barrera Hernández S, Fernández Muñoz MJ, Saucedo García RP, Rodríguez Luna AK, Martínez Murillo C. El incremento de la grasa epicárdica en mujeres se asocia a riesgo trombótico. Clin Investig Arterioscler. 2018;424:1–6.
9. Chu AY, Deng X, VAFisher, Drong A, Zhang Y, Feitosa MF, et al. Multiethnic genome-wide meta-analysis of ectopic fat depots identifies loci associated with adipocyte development and differentiation. Nat Genet. 2016;49(1):125–46.