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# Visceral Pericardium (Epicardial) Adipose Tissue in the Development of Cardiovascular Disease in Diabetic Patients

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## Abstract

The relationship between metabolic diseases such as T2DM and regional fat deposits, particularly epicardial adipose tissue (EAT) and pericardial adipose tissue (PAT), play an important role in the development of cardiovascular diseases (CVD). Both EAT and PAT are a subset of visceral adipose tissue (VAT) associated with T2DM. They are metabolically active visceral fat deposits found around the heart, that are strongly associated with CVD including coronary artery disease (CAD) and the development of cardiac arrhythmias, predominantly due to the secretion of pro-inflammatory mediators and cytokines. In this paper, we review the emerging evidence of impact of T2DM on VAT and the specific role of EAT and PAT both as a cardiac risk marker and as a potentially active player in the development of cardiovascular pathology.

**Keywords:** Epicardial fat tissue; Diabetes mellitus; Meta-analysis; Pericardial adipose tissue; Type 2 diabetes; Cardiovascular disease

## Introduction

Diabetes mellitus (DM) is one of the most common metabolic diseases worldwide and is characterized as a metabolic disorder of carbohydrates, proteins and lipids. In recent years, the incidence of DM has gradually increased, becoming a serious public health threat. DM can be divided into type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus (T2DM), gestational diabetes mellitus (GDM) and other types of diabetes. Abnormally accumulated visceral fat is a risk factor for insulin resistance, which can reduce insulin sensitivity, increase the expression and secretion of proinflammatory cytokines in adipose tissue and promote the development of DM and cardiovascular diseases.

Epicardial fat tissue (EFT) is a visceral adipose tissue that surrounds the myocardium and pericardium. It is one of the visceral fat stores in the body. Previous studies have suggested that measurements of EFT are a substitute for visceral fat. EFT can secrete inflammatory factors, such as TNF-alpha, IL-6, adipocytokines, and leptin, via paracrine or endocrine activities to locally regulate the myocardium and coronary artery function and regulate lipid and energy homeostasis in vivo. EFT has the ability to release and uptake free fatty acids and to affect low glucose utilization, which plays an important role in metabolic syndrome and coronary artery disease. EFT can be measured by echocardiography, cardiac magnetic resonance imaging, and computed tomography (CT). Recent studies have confirmed that EFT is associated with obesity, fasting blood glucose levels, insulin resistance, and adiponectin in patients with T2DM, and an increase in EFT was observed in patients with T1DM and T2DM [1-3].

## Methodology

The aim of this presentation is to review the role of epicardial adipose tissue (EAT) in the development of cardiovascular disease in diabetic patients. Adipose tissue has long been considered as an energy store, however various studies have shown that it has very significant endocrine activity. The main visceral fat location sites that are associated with cardiometabolic risk are intra-abdominal, perivascular, intrahepatic and epicardial fat.

Epicardial fat (EAT) is true visceral fat deposited around the heart, between the myocardium and the visceral layer of the pericardium. Its embryonic origin derives from the splanchnic mesoderm. EAT and myocardium share microcirculation and there is no fascia between them. Therefore adipokines produced by EAT spread directly into the myocardium and coronary vessels by means of vasocrine and paracrine signaling.

## Discussion

EAT provides mechanical protection and acts as a source of energy to the heart. Under normal conditions this visceral fat secretes anti atherogenic and anti-inflammatory cytokines as: adiponectin and omentin. When EAT increases it´s thickness, releases pro-inflammatory and pro-atherogenic adipocytokines as resistin and tumor necrosis factor. In addition, epicardial adipose tissue secretes intravascular and infiltrating inflammatory cells, between those that highlight monocytes and macrophages, predominantly M1 type proinflammatory and mast cells. Epicardial adipose tissue also secretes uric acid. In fact, hyperuricemia triggers a cytotoxic effect on the βcells of the pancreas and favors insulin resistance, hyperinsulinemia and can also develop diabetes mechanisms by which EAT is associated with metabolic syndrome, diabetes mellitus and cardiovascular disease [4].

Today more than 425 million people around the world live with diabetes and unfortunately 50% of them don't know that they live with this life condition. It is estimated that by 2035 the estimated number of patients with diabetes mellitus will be 592 million.

Abnormal glucose metabolism in epicardial adipose tissue also contribute to the development and progression of atherosclerosis, present a decrease in glucose transporter 4 (GLUT 4) and enzyme MRNA catalase, which prevents the generation of reactive oxygen species [5]. Measuring the thickness of the epicardial fat is a simple procedure of perform, accessible, non-invasive and an objective method for risk assessment of subclinical atherosclerosis. It can be assessed by echocardiography, multidetector computed tomography (MDCT) or magnetic resonance. Transthoracic echocardiography is a reliable method for the measurement of EAT.

Body weight, age, sex and even ethnicity are factors that must be taken into consideration with regard to the determinants of epicardial fat thickness; our group found that, in a Mexican population, a thickness equal to or greater than 3 mm is associated with metabolic syndrome [6].

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Our research in the Ticoman General Hospital has shown that EAT thickness is increased when compared with non-diabetic and pre-diabetic patients. Also we have found that EAT has a better correlation than Intra-abdominal visceral fat with carotid intima-media thickness. Also EAT correlates with pressure levels, low-density lipoproteins, fasting blood glucose and serum levels of uric acid.

Diabetic patients who have developed acute myocardial infarction have been shown to have an increased thickness of epicardial fat. The thickness of the epicardial fat is significantly reduced after aerobic training and is associated with decreased adipose tissue visceral, which is an effective non-pharmacological strategy, very low calorie diets also reduce it significantly.

Statins in addition to their effects on lipids, have pleiotropic effects such as stabilizing the atheroma plaque and decreased inflammation of adipose tissue; A study showed that atorvastatin, but not the ezetimibe / simvastatin combination, decreased (10%) epicardial fat in diabetics, and in another study the same statin was superior to pravastatin in reducing epicardial adipose tissue. Analogues of GLP-1 and DPP-4 inhibitors have shown not only reduce the volume of epicardial fat, but also its inflammatory content, on the other hand, insulin detemir was shown to induce greater decreases in epicardial fat than insulin glargine.

## Conclusion

We consider that the measurement of EAT should be included in global evaluation of the diabetic patient, and that the reduction of visceral fat (included EAT) must be a goal in the management of those patient as an approach to the reduction of the inflammatory and atherogenic state and the reduction of cardiovascular disease in the diabetic patient. We believe that it's important to evaluate as well EAT in subjects at high risk for the development of diabetes and even more so in patients with a history of a cardiovascular event.

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