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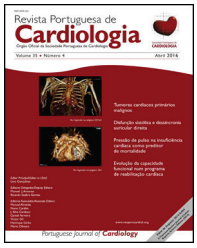

EDITORIAL COMMENT

Epicardial adipose tissue: An important therapeutic target

Tecido adiposo epicárdico: um alvo terapêutico importante

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Cardiovascular disease is the leading cause of death in Portugal, accounting for one third of all deaths.[1] The rising prevalence of cardiovascular risk factors such as obesity and type 2 diabetes is concerning, in Portugal as elsewhere in the world.[1]

In the modern era of obesity and diabetes pandemics, adipose tissue is in the spotlight. Epicardial adipose tissue (EAT) is the visceral fat depot of the heart[2] and is of interest for cardiovascular diagnostics and therapeutics.

EAT can be measured with standard echocardiography, as proposed and validated by Iacobellis et al.[3,4] Echocardiographic EAT is a linear measurement at a single location and therefore may not reflect the variability of fat thickness or total EAT volume as measured by computed tomography.

Of note, EAT has been shown to correlate with waist circumference and intra-abdominal and intracardiac fat, and to serve as a good surrogate marker of visceral adiposity.[4–6]

Impaired myocardial performance as assessed echocardiographically by left ventricular global longitudinal strain[7] was recently associated with increased myocardial fat content and enhanced EAT volume. EAT penetrates inside the human myocardium, affecting its biological behavior, which supports the concept that EAT and cardiac function are closely connected.[8]

Metformin, which was developed from galegine, an antidiabetic compound derived from *Galega officinalis*, is the first-line therapy to treat type 2 diabetic patients.

In this issue of the *Journal*, Ziyrek et al.[9] add interesting findings to the growing literature on cardiac adiposity and heart disease. The authors demonstrated that metformin monotherapy significantly decreases EAT thickness and body mass index (BMI) after three months of therapy in newly diagnosed type 2 diabetes patients.

We read Ziyrek et al.'s study with interest, as it unveils new roles for metformin beyond its use as a first-line therapy for diabetes. The authors did not assess cardiovascular outcomes and no other visceral fat depots were measured in this study. Further work is needed to study and correlate the beneficial effects of metformin on EAT with possible positive cardiovascular outcomes in type 2 diabetic patients.

In addition, diabetic patients in Ziyrek et al.'s study were uncomplicated, and the results may not be applicable to diabetic patients with macrovascular complications. However, their results may warrant further studies in complicated and/or poorly controlled diabetic subjects.

Inflammation is the real culprit when it comes to triggering cardiovascular disease. This prospective observational study should have measured inflammatory markers, suggesting a mechanism of action and highlighting the importance of metformin in this context.

The main finding of the study was that metformin significantly reduces BMI and EAT thickness in newly diagnosed type 2 diabetic patients, reinforcing the use of metformin as a first line therapy for type 2 diabetes. Nevertheless,

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further research is needed to determine whether type 2 diabetic patients with macrovascular disease treated with metformin can achieve similar reductions in EAT thickness and volume. Also, modifications of the EAT transcriptome may open new avenues of treatment for cardiometabolic diseases. Indeed, EAT may serve as a therapeutic target for medications modulating adipose tissue in patients with cardiovascular disease.[10]

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Conflicts of interest

The author has no conflicts of interest to declare.

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