Chapter 8

EPICARDIAL ADIPOSE TISSUE: FROM CELL TO BED SIDE

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Adipose tissue is a type of loose connective tissue composed mostly of adipocytes. Apart from adipocytes periadipocytes, fibroblasts, macrophages, and endothelial cells are also present in the stromal vascular fraction of adipose tissue. In recent years adipose tissue has been recognized as a major endocrine organ secreting various types of cytokines having both paracrine and endocrine effect (Kershaw & Flier, 2004)1. In the human body, localization of adipose tissue might be: beneath the skin (subcutaneous adipose tissue), around the viscera (visceral adipose tissue), in yellow bone marrow, and in breast tissue. Of all these, visceral adipose tissue (also known as organ specific fat) has the greater detrimental effect on metabolic health. Visceral adipose tissue is located in the abdominal cavity, packed between the organs. Excess visceral fat is clinically known as abdominal obesity. Besides, excess visceral fat is also known to be associated with type 2 diabetes mellitus (Montague & Rahilly, 2000)2, insulin resistance (Kern & et al, 2001)3, and inflammatory diseases (Marette, 2003)4. That' s why visceral adipose tissue attracts considerable attention. Epicardial adipose tissue (EAT) is a particular form of visceral adipose tissue which shares the same embryologic origin of omental and mesenteric fat (Talman & et al, 2014)5. Like visceral adipose tissue closely packing the abdominal organs, EAT also encases coronary arteries with no fascial barrier (figure 1). Consequently, it has been postulated that EAT may display endocrine or paracrine effects on the adjacent arterial wall to influence atherosclerotic process (Nitesh & et al, 2017)6.

The objective of this review is to explore EAT from cell to the bedside.

ANATOMICAL ASPECT:

The pericardium is composed of visceral and parietal layers. The fibrous (parietal) pericardium is a resilient sac that surrounds the heart and attaches onto the great vessels (Edwrads, 1984)7. On the other hand, the serious pericardium forms

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tioning as an immuno- mechanical barrier to the coronary arteries, and producing thermogenic effects (Bertaso & et al 2013)18. EAT also serves as a fatty acid depot during excessive exercises. The quantity of EAT increases in a state of positive energy balance, which shares similar pathophysiological mechanisms with visceral adipose tissue accumulation during the progression of metabolic syndrome. Close relationship between EAT and insulin resistance attracts attention to the effect of antidiabetic drugs regulating insulin resistance on EAT. Yagi et al mentioned that, canagliflozin treatment decreased EAT which may have effect in preventing cardiovascular events (Yagi & et al, 2017)19. Iacobellis et al showed that, liraglutide causes rapid EAT reduction (Icobellis & et al, 2017)20.

Due to the close anatomical contact of EAT and coronary arteries, which is described above, local effects of cytokines might accelerate coronary atherosclerosis through pro-inflammatory effect on endothelium (Hirata & et al, 2011)21. They may also contribute to plaque instability, that results in acute coronary syndromes (Rajsheker & et al, 2010)22. There is considerable evidence that the quantity of EAT is related to the presence and severity of coronary artery disease (Greif & et al, 2009)23. Furthermore, aside from the effects on atherosclerosis, fatty infiltration in the myocardium may also interfere with diastolic relaxation and the cardiac conduction system (Ng & et al, 2018)24.

In this brief review we tried the summarize different aspects of EAT. As visceral adipose tissue became a type of endocrine organ, it would attract more attention in future.

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