Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Metabolism Clinical and Experimental 117 (2021) 154722

Contents lists available at ScienceDirect

Metabolism Clinical and Experimental

journal homepage: www.metabolismjournal.com

Correspondence

COVID-19 letter to the editor: Epicardial fat inflammation as possible enhancer in COVID-19?

To the editor:

We have read with interest the report by Kajetan Grodecki et al. in which epicardial adipose tissue (EAT) volume and radiological attenuation associated with the quantitative burden of coronavirus disease 2019 (COVID-19) and an increasing EAT volume or attenuation independently predict clinical deterioration or death [1]. This article may provide a method for risk stratification of COVID-19 patients, which has great clinical significance.

We currently know that inflammation plays a major role in the development and progression of COVID-19. EAT, the metabolically active visceral fat, is considered as a novel marker of inflammation. The imbalance between anti- and pro-inflammatory adipokine secretion from EAT take part in the cytokine storm in critically ill COVID-19 patients [2]. However, caution is still needed in accepting the independent prognostic role of EAT volume or attenuation. C-reactive protein (CRP), serum lactate dehydrogenase (LDH), neutrophil-to-lymphocyte ratio (NLR), lymphocyte-to-CRP ratio (LCR), and platelet-to-lymphocyte ratio (PLR) were all inflammatory markers, which are easily accessible through daily-performed laboratory tests. Abrishami et al. identified serum LDH as a single biomarker to independently predict COVID-19-related death [3], while Cheng et al. showed NLR to be an effective predictor of disease progression in COVID-19 [4]. In addition, exclusion criteria should be set more clearly. EAT is a direct target of medications modulating adipose tissue, such as renin-angiotensin-aldosterone system inhibitors and lipid lowering agents. The volume of EAT may be also affected by a long history of anti-inflammatory treatment. Whether patients with this history should be excluded from the trial need reconsidered.

Although clinically very relevant, it remains difficult to elucidate the mechanisms between EAT and COVID-19 regarding adverse outcomes. Many COVID-19 patients end with cardiovascular events. Angiotensin-converting enzyme 2 (ACE2) is recognized as the entry ligand receptor of severe acute respiratory syndrome coronavirus-2. Based on experimental evidence, ACE2 and inflammatory cytokines, tumor necrosis factor-α and interleukin-6, were demonstrated to be expressed at higher levels in EAT in heart explants removed from obese patients [5]. In addition, Bois et al. reported lower ACE2 endothelial expression in COVID-19 cases versus influenza A/B or non-virally mediated deaths [6]. This reduction in ACE2 was associated with EAT inflammation, partly due to the virus taking advantage of more ACE2-binding sites for internalization of the virus into adipocytes and then triggering an augmented inflammatory signaling cascade. Bois et al. also showed that a high proportion of COVID-19 deaths were accompanied with cardiac amyloidosis [6], which may be an additional risk factor for severe clinical condition.

It is of great significance to find out a specific biomarker to predict adverse outcomes in patients with COVID-19. EAT play a role in COVID-19 patients and probably become a clinically measurable and modifiable therapeutic target, but more studies are needed to explore this intriguing relationship.

Declaration of competing interest

The authors declare no conflict of interest.

References

[1] Grodecki K, Lin A, Razipour A, Cadet S, McElhinney PA, Chan C, et al. Epicardial adipose tissue is associated with extent of pneumonia and adverse outcomes in patients with COVID-19. Metabolism. 2021;115:154436.
[2] Malavazos AE, Goldberger JJ, Iacobellis G. Does epicardial fat contribute to COVID-19 myocardial inflammation? Eur Heart J. 2020;41:2333.
[3] Abrishami A, Eslami V, Baharvand Z, Khalili N, Saghamanesh S, Zarei E, et al. Epicardial adipose tissue, inflammatory biomarkers and COVID-19: is there a possible relationship? Int Immunopharmacol. 2021;90:107174.
[4] Cheng B, Hu J, Zuo X, Chen J, Li X, Chen Y, et al. Predictors of progression from moderate to severe coronavirus disease 2019: a retrospective cohort. Clin Microbiol Infect. 2020;26:1400–5.
[5] Kim I, Han S. Epicardial adipose tissue: fuel for COVID-19-induced cardiac injury? Eur Heart J. 2020;41:2334–5.
[6] Bois MC, Boire NA, Layman AJ, Aubry M, Alexander MP, Roden AC, et al. COVID-19–associated nonocclusive fibrin microthrombi in the heart. Circulation. 2021;143:230–43.

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23 January 2021

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https://doi.org/10.1016/j.metabol.2021.154722