Letters to the Editor

Epicardial fat and liver disease; the contribution of cardio autonomic nervous system function

To the Editors:

We read with great interest, the recent article by Petta et al. [1]. The authors show a positive relationship between higher epicardial fat thickness and the severity of liver fibrosis in patients with non-alcoholic fatty liver disease (NAFLD), which may indicate a possible pathogenic role of ectopic fat depots in whole body organ damage. They also stated that morphological and functional cardiac alterations were more pronounced according to the severity of fibrosis. In our letter, we would like to highlight another possible mechanism between epicardial fat and liver disease; the contribution of cardio autonomic nervous system function (CAF).

In the literature, Chiang et al. evaluated the association between cardiovascular autonomic functions and time-to-death (TTD) in patients with terminal hepatocellular carcinoma. They concluded that the inclusion of HRV measurement in the prognostic models may improve accuracy in TTD prediction and, hence, facilitate medical decision making in hospital care [2]. Liu et al. investigated the influence of NAFLD on autonomic changes and they clearly showed that NAFLD is associated with autonomic neural modulation of cardiac function in the sympathetic direction. The former association was independent of conventional cardiovascular risk factors and serum biomarkers (insulin resistance and leptin). They recommended further risk stratification of autonomic dysfunction by the HRV parameters in patients with NAFLD [3]. The results of these previous studies, indicate that impairment in autonomic nervous system function may play a role in the development of NAFLD.

Several studies show a close relation between epicardial fat and cardiac autonomic function [4–6]. It was shown that the cardiac ganglionated plexus in the epicardial fat integrates the autonomic innervation between the extrinsic and intrinsic cardiac autonomic nervous system and affects atrial electrophysiology and pathophysiology [4]. Carnevali et al. concluded that epicardial fat deficiency in mice leads to an imbalance of the autonomic neural modulation of cardiac function in the sympathetic direction and to a potentially proarrhythmic remodeling of electrical and structural properties of the heart [5]. Additionally, Balcioglu et al. showed that sympathovagal imbalance, detected by heart rate variability and turbulence parameters, is related to epicardial fat thickness. As sympathovagal imbalance is a predictor of arrhythmic events, epicardial fat may play an important arrhythmogenic role [6].

As a consequence, we believe that the impairment of the cardiac autonomic nervous system function may be helpful in explaining the results of the article by Petta et al. [1]. We hope that the above-mentioned items would add to the value of the well-written article of Petta et al. on the relationship between epicardial fat and NAFLD.

Conflict of interest

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References


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