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Editorial: Interactions between NAFLD and cardiac conduction, structure and function: recent advances and treatments

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KEYWORDS

NAFLD, MAFLD, atrial fibrillation, atrial remodeling, left ventricular diastolic dysfunction, monocyte-to-high-density lipoprotein ratio (MHR), heart failure, mortality

Editorial on the Research Topic

Interactions between NAFLD and cardiac conduction, structure and function: recent advances and treatments

Non-alcoholic fatty liver disease (NAFLD) represents the most common liver disease globally (1), and its prevalence has been rising along with the obesity and type 2 diabetes epidemic (2). According to a recent systematic review, the global prevalence of NAFLD increased from 25.3% in 1990–2006 to 38.0% in 2016–2019 (3). Consequently, NAFLD is becoming a major public healthcare challenge (4). It causes a deterioration of quality of life, which increases with disease progression and is worsened by comorbidities (5, 6), representing the leading cause of liver-related morbidity and mortality (7). NAFLD does not only affect the liver but was shown to be a multisystem disease with increased risk of type 2 diabetes, dyslipidaemia, metabolic syndrome, hypertension, kidney and cardiovascular diseases (8). Increasing literature data suggests that subjects with NAFLD show myocardial functional and structural changes, leading to cardiac remodeling and increased risk of heart failure (HF) (9). In particular, different studies showed associations between NAFLD and left ventricular and diastolic dysfunction (9, 10), right ventricular dysfunction (11) and left ventricular hypertrophy (12). Moreover, recent data showed that NAFLD is associated with an increased risk of incident atrial fibrillation (AF) (13) and subjects with higher hepatic fibrosis index were associated with increased AF risk (14).

Mechanisms that link NAFLD to cardiac functional and structural changes, apart from the common risk factors, have yet to be explored. In NAFLD, insulin resistance, inflammation and oxidative stress may lead to cardiac insulin resistance and cardiac fibrosis, determining an alteration in cardiac structure (15). Moreover, the increased myocardial fatty acid oxidation, which is less efficient than glucose metabolism, may contribute to the development of heart failure (16).

This Research Topic aimed to provide insight into several aspects of the connections between NAFLD cardiac conduction, structure and function, presenting novelties on the potential pathological mechanism at the basis of these relationships. Overall, four original articles and two meta-analyses have been published.

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