

A journey in structural heart failure

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EDITORIAL



A journey in structural heart failure

Cardiovascular disease (CVD) is the leading cause of death in Western countries [1, 2]. Structural heart disease refers to conditions that affect the heart valves, walls, chambers, or muscles, with dyspnoea being the common cardinal symptom [3–5]. Heart failure (HF) with preserved ejection fraction (HFpEF) represents nearly half of all HF cases. We have already addressed the difficult question of clinical diagnosis of HFpEF [6, 7]. Wang et al. showed that HFA-PEFF and H2FPEF scores can be used to effectively rule out or confirm HFpEF [7], with the caveat that patients without a high risk score might require interventional catheterisation or stress testing [8]. Growth differentiation factor 15 (GDF-15) is a member of the transforming growth factor- β cytokine superfamily with immunosuppressive, anti-apoptotic, and anti-inflammatory properties [9]. In this issue, Wang et al. reported the high diagnostic value of GDF-15 in patients with HFpEF (Figure 1) [10].

Inflammation has been recognised as a major pathophysiological contributor to HF. Periodontal disease is one of the most prevalent systemic inflammatory diseases worldwide [11]. In their systematic review and meta-analysis, Leelaviwat et al. showed that periodontal disease is associated with an increased risk of HF. Better oral health could therefore constitute an easily modifiable risk factor to reduce the risk of HF, although larger studies are needed to validate this result (Figure 2) [12].

Coronary artery disease (CAD) is a major contributor to cardiovascular disease and HF [4]. Management of CAD is now better guided by the physiological significance of coronary artery stenosis, which can be assessed during invasive coronary angiography by measuring fractional flow reserve (FFR) during adenosine-induced hyperaemia, which can however cause some discomfort to patients [13, 14]. The instantaneous wave-free ratio (iFR), which is a type of non-hyperemic pressure ratio (NHPR), is an innovative test that does not involve the additional administration of drugs [15]. In their meta-analysis, comprising 1,084 patients with 1,312 lesions, Luo et al. showed that resting full-cycle ratio (RFR), a new non-congestive resting index, has good diagnostic accuracy in detecting coronary ischaemic lesions and may be an effective alternative to FFR in the future [16]. Treatment of CAD is aimed at alleviating angina symptoms and preventing myocardial infarction or premature death. Coronary artery bypass graft (CABG) provides an effective treatment option for patients with CAD. However, patients undergoing CPB may develop systemic inflammatory responses and organ dysfunction, resulting in dismal postoperative outcomes [17]. In their animal study, Zhang et al. showed that overexpression of hypoxia-inducible factor-1 α (HIF-1 α) can improve

myocardial injury in cardiopulmonary bypass rats *via* the miR-124-3p/NR4A1 axis [18].

Low-density lipoprotein cholesterol (LDL-C) is a well-established risk factor for cardiovascular disease development [19]. The study by Jiang et al. sheds light on the complex relationship between LDL-C levels and all-cause mortality in patients with idiopathic dilated cardiomyopathy (iDCM) [20, 21]. Indeed, in their retrospective study concerning 1058 patients with iDCM, the authors showed that lower LDL-C level was associated with an increased risk of all-cause mortality. The correlation between mortality and LDL-C level was stronger in patients with worse heart function (Figure 3) [21]. The exact reasons for this

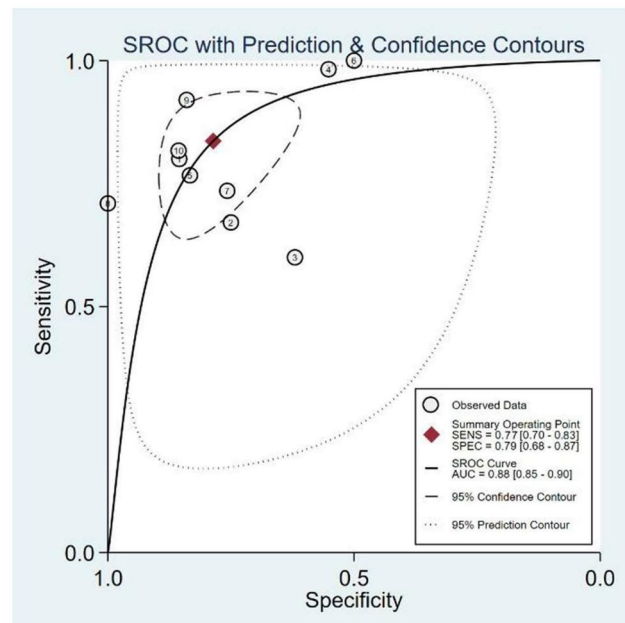


Figure 1. SROC plot of GDF-15 for HFpEF diagnosis (from reference [10]).



Figure 2. An early stage of periodontitis demonstrates bleeding and puffy gum (from reference [12]).

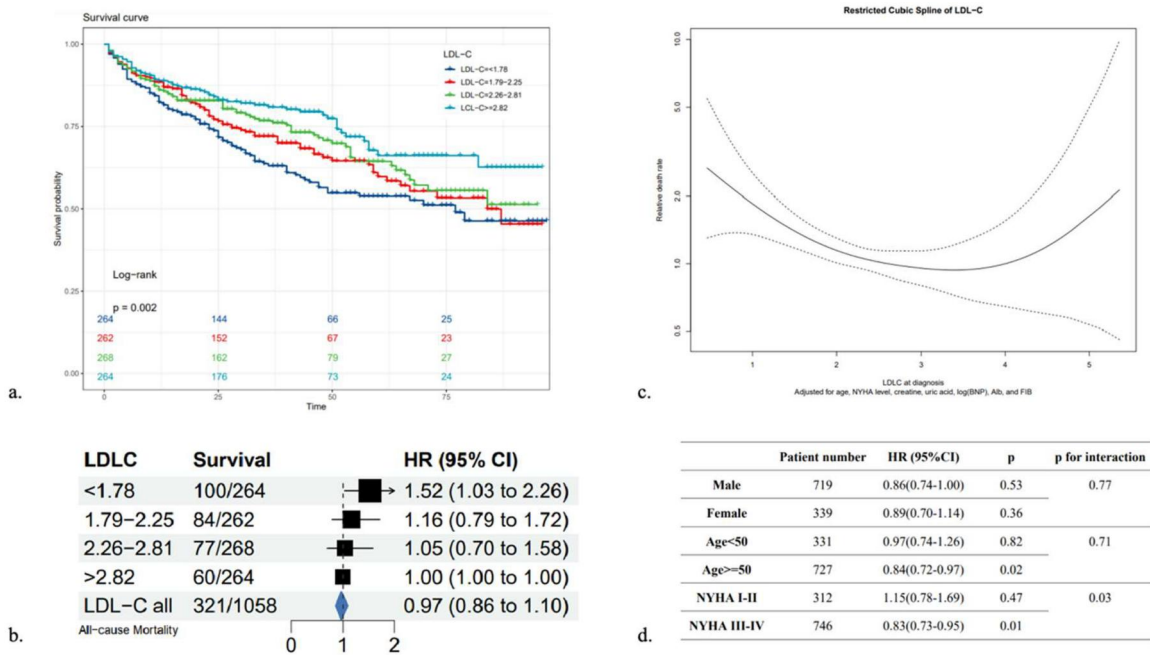


Figure 3. (a) Kaplan–Meier estimates for cumulative survival in patients of different LDL-C levels. (b) Multivariable adjusted hazard ratios for all-cause mortality according to levels of LDL-C on a continuous scale. (c) Forest plot of multivariable-adjusted hazard ratios for all-cause mortality. (d) Interaction and stratified analysis according to potential interaction factors. LDL-C: low-density lipoprotein cholesterol (from reference [21]).

paradoxical observation as referred to as the ‘lipoprotein paradox’ still remain enigmatic.

The goal of therapy for chronic HF is to improve symptom management and quality of life, decrease hospitalisations, and decrease overall mortality associated with this disease. The core foundational medication classes for HFrEF includes a renin-angiotensin system inhibitor (such as an angiotensin receptor neprilysin inhibitor (ARNI), angiotensin-converting enzyme (ACE) inhibitor, or angiotensin II receptor blockers (ARB)), a beta-blocker, a mineralcorticoid receptor antagonist (MRA) and a sodium-glucose co-transporter 2 Inhibitor (SGLT2i) [3, 22–24]. In their article, El-Hefny et al. presented novel findings on the predictive value and difference in serum aldosterone level between right side HF, HFrEF and HFpEF, and compared the efficacy and safety of adding MRA for treatment of right HF versus left HF. A total of 151 patients were studied. As expected, aldosterone levels were significantly higher in HF patients than in controls, although levels were lower in right HF than left HF. In the HFrEF group, there was a significant decrease in LV end diastolic dimension and a significant increase in LVEF after treatment. In the HFpEF group, there was a significant decrease in E/A and E/e’ after MRA treatment [25]. In acute pulmonary embolism (APE), echocardiographic markers of right ventricular (RV) dysfunction (d) or RV pressure overload (PO) have been used for risk stratification of APE [26, 27]. Ballas et al. showed that non-conventional echocardiographic indices of $RV_{d/PO}$ were correlated with biochemical and clinical prognostic markers and *RV free wall longitudinal strain (FWLS)* was

by far the most frequent index of $RV_{d/PO}$ in patients with APE. These findings suggest that conventional echocardiographic markers of $RV_{d/PO}$ underestimate the true incidence of RVd in patients with APE [28].

In this issue of Acta Cardiologica, several focus images and cases highlighting interesting clinical conditions have also been reported [29–38].

Disclosure statement

No potential conflict of interest was reported by the author(s).

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