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LETTER TO THE EDITOR

Kill two birds with one stone: Hapatologist's approach to metabolic dysfunction-associated steatotic liver disease and heart failure

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Abstract

Heart failure (HF) is a major global public health concern, and one of the less commonly known risk factors for HF development is metabolic dysfunction-associated steatotic liver disease (MASLD), as they share a similar pathophysiological background. In this article, we evaluated a recently published review article by Arriola-Montenegro *et al*. This article briefly summarizes the common pathophysiology of HF and MASLD development and evaluates the available therapeutic options to treat both conditions. Clinical practice guidelines highlight the importance of initiating and titrating guideline-directed medication therapy (GDMT) for patients with HF with reduced ejection fraction. GDMT is comprised of the four pillars currently proposed in most clinical practice guidelines, namely angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), angiotensin receptor-neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, and sodium-glucose co-transporter 2 inhibitors (SGLT-2i). Given the similarity of pathophysiology and risk factors, recent studies for GDMT regarding ACEIs, ARBs, mineralocorticoid receptor antagonists, and SGLT-2i have shown beneficial effects on MASLD. Nonetheless, other medications for both conditions and novel therapies require more robust data and well-designed clinical studies to demonstrate their efficacies in both conditions.

Key Words: Metabolic dysfunction-associated steatotic liver disease; Heart failure; Heart failure with reduced ejection fraction; Non-pharmacological; Pharmacological; Surgical intervention

Core Tip: Due to common risk factors and underlying pathophysiologic mechanisms, there is a significant association between heart failure with reduced ejection fraction and metabolic dysfunction-associated steatotic liver disease. This article will explore the current pharmacological and non-pharmacological interventions.

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TO THE EDITOR

Worldwide, heart failure (HF) represents a significant clinical, economic, and public health concern. Globally, 64.3 million people suffered from HF in 2017, and it is estimated to cost $69.8 billion in the United States in 2030[1]. HF is also considered to be most prevalent amongst adults aged greater than 60 years old[2]. Ischemia, tachyarrhythmias, infiltrative conditions, cardiac toxin exposure, substance use, and structural conditions such as valvular heart disease are common risk factors for HF development[3,4]. Treatment of HF remains multimodal with management of the underlying etiology in addition to utilization of Guideline Directed Medical Therapy (GDMT). At the core of GDMT remains four pillars consisting of different medication classes which include angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), angiotensin receptor-neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, and sodium-glucose co-transporter 2 inhibitors (SGLT-2i)[5]. Interestingly, metabolic dysfunction-associated steatotic liver disease (MASLD), formally known as non-alcoholic fatty liver disease (NAFLD), is an emerging risk factor for HF as it may share a similar pathophysiological mechanisms[3,6]. MASLD is a fatty infiltration of the liver without hepatocellular inflammation due to metabolic risk factors. Whereas metabolic dysfunction-associated steatohepatitis (MASH) is defined as the presence of adipose tissue leading to lipotoxicity and inflammatory damage to hepatocytes[7]. A recently published review article by Arriola-Montenegro *et al*[3], evaluated therapies to target both HF with reduced ejection fraction (HFrEF) and MASLD[3]. HFrEF and MASLD represent two prevalent comorbidities sharing similar pathophysiological mechanisms. Moreover, emerging epidemiological investigations substantiate a robust and independent correlation between MASLD and HF, with an approximate prevalence of HF among MASLD patients being 6.4%[8]. The pathophysiological relationship between MASLD and HFrEF involves substances that contribute towards further dysregulation such as adipokines and proinflammatory cytokines including leptin. Leptin has been associated with profibrotic activity while working at the level of the liver. Meanwhile, leptin may also be associated with endothelial dysfunction as well as cardiac hypertrophy. Other notable mediators include tumor necrosis factor-α and interleukin (IL)-6, both which confer to hepatocyte damage. Additionally, another mediator IL-33 has been noted to be released in the setting of hepatocyte injury and shown to potentiate further fibrosis. Meanwhile, the release of IL-33 by the heart has been associated as a reaction towards myocardial fiber stretching[9].

MASLD and cardiovascular disease (CVD) both possess similar risk factors (*i.e.*, sedentary lifestyle, smoking, physiological stress, and sleep deprivation). Furthermore, the presence and accumulation of visceral and ectopic fat acts as a further stimulus towards inflammatory pathway activation and release of toxic metabolites further contributing to each pathologies. CVD is known to be prevalent in patients with MASH, particularly in those with severe liver disease, remaining a leading cause of mortality. Respectfully, CVD risk factors should be proactively managed in this population [10].

THERAPIES

Non-pharmacological therapies

Primary therapeutic interventions for MASLD consist of lifestyle modifications, which include dietary alterations, increased physical activity, and weight management. Typical recommendations for patients in the hepatology clinic include trialing the Mediterranean diet, 150 minutes of moderate to high-intensity aerobic exercise with strength training, and goal weight reduction of 7%-10% of body weight[11]. These lifestyle modifications alter adipose tissue distribution and improve the risks of developing cardiovascular comorbidities[12]. When optimal results are not achieved with lifestyle modifications, bariatric surgery should be considered in obese patients with associated comorbidities. Bariatric surgery has been shown to have the potential for long-term improvement or even resolution of MASLD, both clinically and histologically. This also mitigates CVD risk among obese patients by improving glucose tolerance and lipid profiles [13]. Moreover, recent meta-analyses have revealed a decreased incidence of HF and myocardial infarction following bariatric surgery[14].

Pharmacological therapies

Several evidence-based pharmacologic interventions for HFrEF have shown a beneficial effect on MASLD therapies. As the backbones of GDMT for HFrEF, ACEIs and ARBs have potential beneficial effects on MASLD treatment[15]. ACEIs

and ARBs are both known to have mortality benefits in hospitalized patients with HFrEF, advanced kidney disease, and for MASLD treatment. ACEIs, and ARBs also inhibit Angiotensin II, a key contributor to abnormal lipid metabolism[16]. This in turn, decreases lipid accumulation in the liver and diminishes the risk of fibrosis. SGLT-2i, which are also used for type 2 diabetes treatment, may inhibit the development of MASLD and improve histological features of hepatic steatosis or steatohepatitis[17]. The possible mechanism of SGLT-2i in MALSD management is weight loss and reduction of visceral fat by inhibition of de novo hepatic lipogenesis[18]. A recent meta-analysis demonstrated SGLT-2i induced a significant decrease in liver enzymes involving serum alanine, aspartate aminotransferases, gamma glutamyl transferases, in addition to decreasing liver steatosis[19]. Another meta-analysis also evaluated the efficacy of SGLT-2i to significantly decrease serum alanine aminotransferase, gamma-glutamyl transferase levels and liver fat content on imaging techniques compared to placebo/reference therapy[20]. Additionally, the risk of cardiovascular death or hospitalization amongst patients with HFrEF regardless of type 2 diabetes mellitus (T2DM) status has been shown to be reduced with SGLT-2is. SGLT-2i seems to be a promising pharmacological option for both MASLD and HFrEF. Patients with NAFLD and HFrEF also have potential for favorable outcomes with the use of mineralocorticoid receptor antagonists (MRAs) such as spironolactone and eplerenone. In a mouse model, eplerenone effectively improved insulin resistance and MALSD-associated histological change by acting on Kupper cells and macrophages[21]. Another mouse model study demonstrated spironolactone not only improved accumulation of triglycerides within the liver but also suppressed the expression of proinflammatory, gluconeogenic, and lipogenic enzymes[20]. Additionally, spironolactone and vitamin E combination therapy may convey improvement with insulin resistance within an *in vivo* study[21]. These results indicate that MRAs may be pivotal treatments for both MASLD and HFrEF. For novel medications, resmetirom is an oral, liver-directed, thyroid hormone receptor beta-selective agonist and is currently the most recent and only United States Food and Drug Administration (FDA) approved treatment of MASH with liver fibrosis (F2 and F3). MAESTRO-NASH trial, a phase 3 trial, involving adults with biopsy-confirmed NASH and a fibrosis stage of F1B, F2, or F3 [stages range from F0 (no fibrosis) to F4 (cirrhosis)] demonstrated that both the 80-mg and the 100 mg dose of resmetirom were superior to placebo with respect to NASH resolution and improvement in liver fibrosis by at least one stage[22]. As this is a recent FDA-approved medication in as of March 2024, long-term data is currently lacking, in addition to an unknown effect on HF risk modification. Glucagon-like peptide 1 (GLP-1) receptor agonists promote weight loss by improving hyperglycemia and delaying gastric emptying[23]. These provide an appealing therapeutic choice amongst patients with MASLD, particularly those with obesity and diabetes mellitus. Although observations regarding utilization of GLP-1 receptor agonists in those with MASLD suggest benefit, while randomized trials infer an absence of benefit in HF related outcomes in addition to uncertainty involving safety amongst those with HFpEF[24]. Tirzepatide, a dual glucose-dependent insulinotropic polypeptide, and a GLP-1 receptor, is a novel medication for the treatment of T2DM and has provided encouraging results amongst ongoing clinical trials, for T2DM in addition to improving body weight and steatosis[25]. Currently, there is no available data to support the evidence of tirzepatide in patients with HFrEF, but a clinical trial assessing the efficacy and safety of tirzepatide in patients with HF with preserved ejection fraction and obesity is being undertaken (ClinicalTrials.gov ID: NCT04847557). Although many practitioners are leery of using statins in patients with liver disease, statins have been reported as safe for patients with MASLD, including those with advanced liver disease, and are also associated with a clear reduction in cardiovascular morbidity and mortality. For the management of dyslipidemia in MASLD, moderate- to high-intensity statins should be the preferred agents based on lipid associated risk level and atherosclerosis atherosclerotic cardiovascular disease risk score[11]. Regrettably, there is currently a lack of data elucidating favorable effects of sacubitril/valsartan, beta-blockers, hydralazine, isosorbide nitrates, ivabradine, or digoxin, on MASLD.

CONCLUSIONS

Numerous recent studies have revealed a strong correlation between HF, particularly the HFrEF subtype, and MASLD. Various pathophysiological mechanisms have been proposed, most of which revolve around common factors contributing to systemic inflammation. To the present time, a variety of pharmacologic and non-pharmacologic treatments have been explored in patients simultaneously managing HFrEF and MASLD. Specific pharmacologic therapies such as diet, ACEIs, ARBs, MRAs, SGLT-2i inhibitors, and bariatric surgery have been implicated to be effective. Yet, there continues to be an absence of solid data and well-designed clinical trials regarding several other pharmacologic therapies and innovative treatments which may be potentially beneficial for patients with these conditions.

FOOTNOTES

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