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Perspective

The Link between Adipokines and Frailty in Heart Failure

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DESCRIPTION

Heart Failure (HF) is a complex clinical syndrome characterized by the heart's inability to pump sufficient blood to meet the body's demands. It affects millions worldwide and is associated with significant morbidity and mortality. One emerging aspect of HF management is understanding the role of adipokines–signaling molecules secreted by adipose tissue–in the development and progression of frailty, a state of increased vulnerability to stressors due to decreased physiological reserves. This article explores the association between adipokines and frailty in HF, illuminate on potential mechanisms and clinical implications.

Adipokines in heart failure

Adipose tissue, once regarded as a passive energy reservoir, is now recognized as an active endocrine organ producing various bioactive substances, including adipokines. These molecules exert diverse effects on metabolism, inflammation, and cardiovascular function, thereby influencing the pathophysiology of HF. Among the adipokines implicated in HF are adiponectin, leptin, resistin, and visfatin, each with unique roles in modulating cardiovascular and metabolic homeostasis.

Frailty in heart failure

Frailty is increasingly recognized as a crucial determinant of outcomes in HF. It manifests as decreased physiological reserve and resilience, predisposing individuals to adverse events such as hospitalization, disability, and mortality. Frailty in HF is multifactorial, involving biological, psychosocial, and environmental factors. Common manifestations include weakness, exhaustion, slow gait speed, unintentional weight loss, and low physical activity.

The association between adipokines and frailty

Emerging evidence suggests a bidirectional relationship between adipokines and frailty in HF. Adipokines contribute to the pathogenesis of frailty by promoting inflammation, oxidative stress, insulin resistance, and endothelial dysfunction, all of which are implicated in the development of frailty phenotypes. Conversely, frailty-related alterations in body composition, physical activity, and metabolic function may influence adipokine secretion and signaling, creating a feedback loop that exacerbates HF progression.

Potential mechanisms

Several mechanisms may underlie the association between adipokines and frailty in HF. Adiponectin, for instance, has anti-inflammatory and cardioprotective effects but is paradoxically elevated in frail individuals with HF, possibly reflecting adipose tissue dysfunction or impaired clearance. Leptin, known for its role in appetite regulation and energy balance, may contribute to frailty by promoting inflammation and insulin resistance. Resistin and visfatin, although less studied in the context of frailty, have been implicated in cardiovascular disease and metabolic dysfunction, suggesting potential links with frailty pathways.

Clinical implications

Understanding the interplay between adipokines and frailty has important clinical implications for HF management. Targeting adipokine dysregulation through lifestyle interventions, pharmacotherapy, or novel therapies may mitigate frailty and improve outcomes in HF patients. Additionally, incorporating frailty assessment into routine clinical practice can help identify high-risk individuals who may benefit from personalized interventions aimed at preserving functional independence and quality of life.

CONCLUSION

The association between adipokines and frailty represents a novel paradigm in HF research, highlighting the intricate interplay between adipose tissue dysfunction, inflammation, and functional decline. Further studies are needed to elucidate the causal pathways linking adipokines to frailty and to identify potential therapeutic targets for intervention. By addressing adipokine dysregulation and frailty in HF, clinicians can optimize patient care and enhance outcomes in this vulnerable population.

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