

The GLP-1 Revolution: Healing the Heart Through Metabolic Control

THE PROBLEM: OBESITY AS A CAUSAL DRIVER

Obese HFpEF is a Distinct Phenotype: Over 80% are overweight, showing unique metabolic & hemodynamic impairment.

PRO-INFLAMMATORY MARKERS

FIBROSIS

EPICARDIAL FAT

The "Toxic" Role of Epicardial Fat: Secretes inflammatory markers directly into myocardium, causing fibrosis.

Systemic Inflammation & Pressure

Adipose tissue drives endothelial dysfunction & compresses the heart, leading to fluid retention.

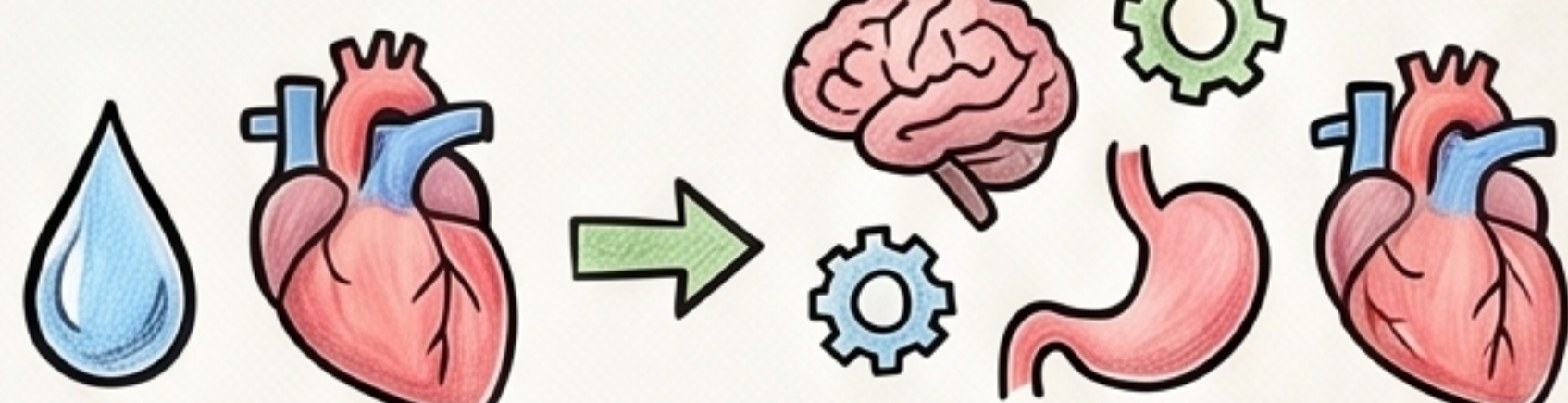
THE SOLUTION: SEMAGLUTIDE & THE STEP-HFPEF TRIAL

GLP-1 RECEPTOR AGONIST

SEMAGLUTIDE

ACTION!

Significant Symptom Improvement: Semaglutide showed marked clinical improvement in patient quality of life.



Managing Fluid

Fixing Systemic Metabolic Environment

Shifting to "Metabolic Cardiomyopathy"

STEP-HFpEF TRIAL DATA (Semaglutide 2.4mg vs. Placebo)



METRIC	SEMAGLUTIDE 2.4mg	PLACEBO
Body Weight Change	-13.3%	-2.6%
6-Minute Walk Distance	+21 meters (over placebo)	Baseline
KCCQ Symptom Score	+16 points	+8 points



GLP-1s (weight/inflammation) SGLT2is (decongestion)

Complementary Therapy: GLP-1s and SGLT2is provide a powerful, additive treatment approach.