

DISTINCT ARTERIAL AND CARDIAC REMODELLING PATTERNS IN HYPERTENSIVE HEART FAILURE WITH A PRESERVED EJECTION FRACTION

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ABSTRACT

BACKGROUND AND OBJECTIVES

Hypertension (HTN) affects approximately 30% of Africans and is a major contributor to heart failure with preserved ejection fraction (HFpEF), typically in older adults due to arterial stiffness. However, HFpEF has been observed in younger black South African populations. Given the frequent presence of left ventricular hypertrophy (LVH) in hypertensive individuals, this study aimed to determine whether hypertensive patients with HFpEF exhibit distinct clinical, haemodynamic, and cardiac structural characteristics compared to those with HTN alone.

METHODS

A community-based case–control study included 125 HFpEF cases and 510 controls. Of these, 95 hypertensive HFpEF cases and 256 hypertensive controls were analysed. Clinically stable adults (>18 years) were enrolled. Assessments included echocardiographic, anthropometric, haemodynamic, and pulse wave velocity (PWV) measurements.

RESULTS

The mean age was approximately 58 years, with approximately 80% of participants overweight or obese. Compared to controls, HFpEF participants had lower central diastolic

blood pressure (DBPc) ($p=0.0295$) and central pulse pressure (PPc) ($p=0.0352$), but significantly higher PWV (10.9 ± 4.8 vs 6.9 ± 3.9 m/s, $p<0.0001$), indicating increased arterial stiffness. Concentric LVH predominated in HFpEF (73%), whereas eccentric LVH was more common in controls (25%). Peripheral blood pressure was associated with the ratio of early (E) to late (A) ventricular filling velocities (E/A), while DBPp, PPp, and E/A correlated with antihypertensive treatment. HFpEF was further associated with reduced renal function, higher heart rate, and elevated inflammatory markers (C-reactive protein and galectin-3).

CONCLUSION

Hypertensive HFpEF is characterised by increased arterial stiffness, concentric LVH, impaired renal function, elevated heart rate, and heightened inflammation. These findings support a role for adverse arterial–ventricular coupling and inflammation-driven concentric remodelling in HFpEF.