Longterm Effect of Hypertension on LV Function

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Hypertension

- Hypertension is a prevalent and well-recognized cardiovascular risk factor
- It is the leading cause of cardiovascular disease, stroke, and death.
- It affects a substantial proportion of the population worldwide and remains underdiagnosed and undertreated.
- It tends to cluster with other cardiovascular risk factors, such as obesity and smoking, making it difficult to identify independent effects of blood pressure on the structure and function of heart.

Hypertension and the myocardium

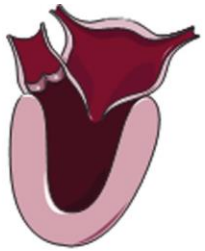
- Long-standing high blood pressure leads to left ventricular hypertrophy and diastolic dysfunction that cause an increase in myocardial rigidity, which renders the myocardium less compliant to changes in the preload, afterload, and sympathetic tone

Hypertension and the myocardium

- High BP increases the left ventricular (LV) afterload and peripheral vascular resistance and prolonged exposure to an increased load leads to pressure- and volume mediated LV structural remodeling

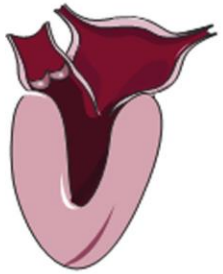
Staging of Hypertensive Heart Disease

Degree I



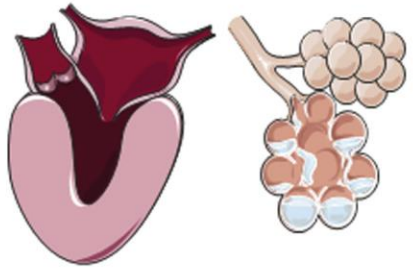
- LV diastolic dysfunction
- No LV hypertrophy

Degree II



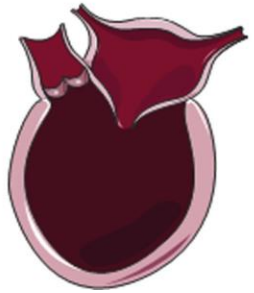
- LV diastolic dysfunction and
- LV hypertrophy

Degree III



- Clinical heart failure with
- Preserved LV ejection fraction

Degree IV



- Eccentric LV hypertrophy
- Reduced LV ejection fraction

Diastolic Dysfunction

- Diastolic dysfunction is one of the first changes observed in a heart that has been exposed to an increased load
- Diastolic dysfunction increases the LV filling pressure and left atrial (LA) volume, which, in turn, increase the pulmonary artery pressure

Left Ventricular Hypertrophy

- Ventricular hypertrophy is an initial compensatory mechanism in response to the chronic pressure overload that preserves the cardiac output and delays cardiac failure
- Chronic HTN,
- involves both pressure and volume overloads, and leads to both concentric or eccentric hypertrophy

Heart Failure

- The pathway from LV hypertrophy to overt HF is complex and unclear.
- Most patients with concentric hypertrophy develop HFpEF, but despite the absence of a history of myocardial infarction, some can progress to HFrEF

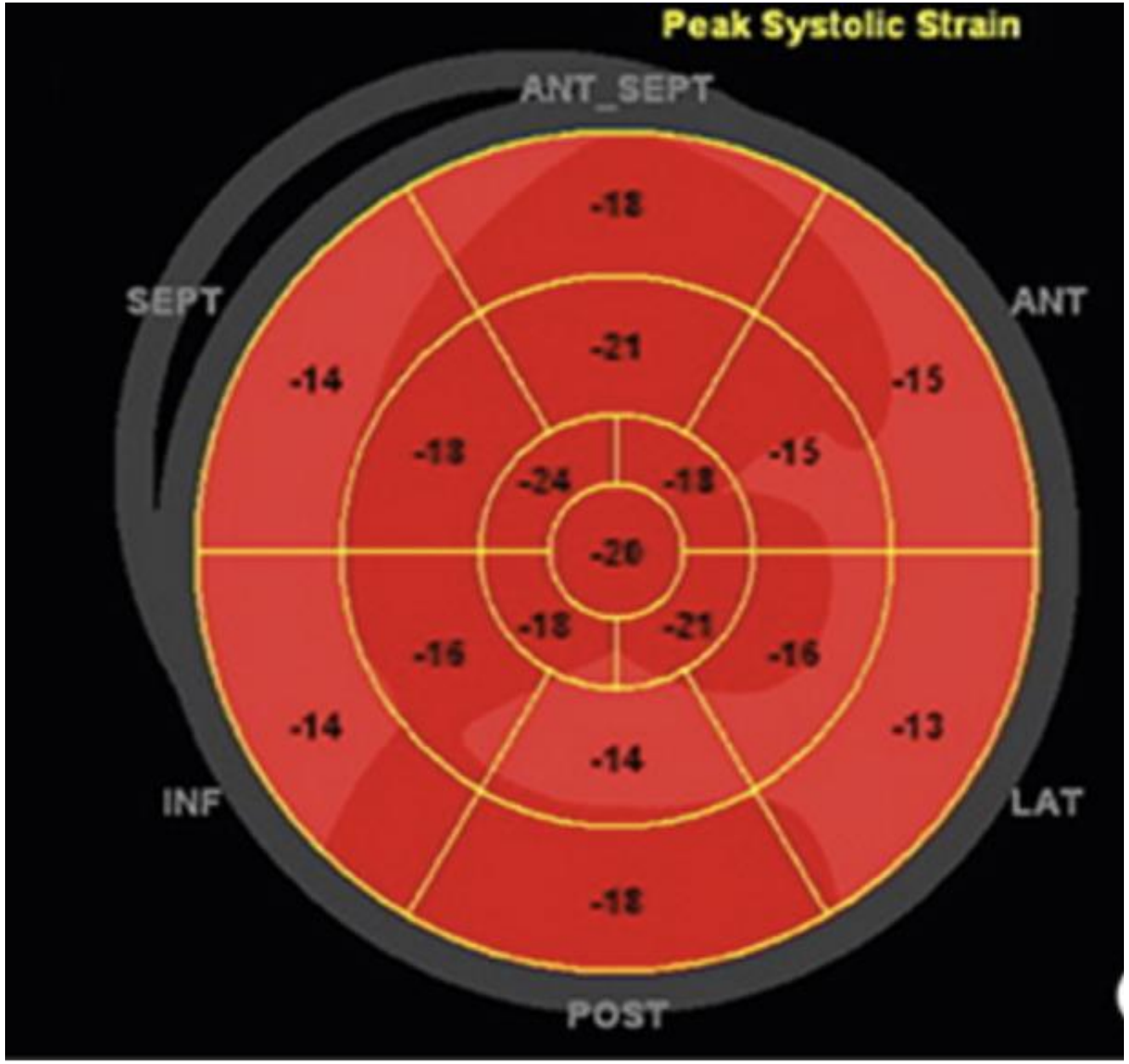
HF with Preserved Ejection Fration

- The development of HFpEF seems to be associated with changes in the extracellular matrix that cause progressive fibrosis of the myocardium and, subsequently, an increase in LV stiffness

Hypertension and LV longitudinal Function

- Patients with hypertension and normal EF were demonstrated to have reduced LV systolic longitudinal function.
- Longitudinal strain is more sensitive than tissue Doppler imaging to assess this dysfunction.
- LV systolic strain is early reduced during systemic hypertension and seems to be an index of global LV remodeling

Bull's Eye of Hypertensive Heart Disease using Speckle Tracking Echo



Pulmonary edema and Hypertension

- Patients with long-standing HTN are more sensitive to changes in pressure, volume, and sympathetic tone .
- The reduced compliance of the ventricle and systemic vasculature in patients with hypertensive HF results in abnormal ventricular-vascular interactions

Pulmonary edema and Hypertension

- The premature return of aortic pulse waves increases the resistance to the ventricular outflow, which, in turn, impedes the pulmonary venous flow towards the heart
- Consequently, small changes in the preload, afterload, or sympathetic tone can further increase the LV filling pressure, thereby disrupting the pulmonary capillary blood-gas barrier, which leads to flash pulmonary edema

HF with Reduced Ejection Fraction

- A subset of patients with longstanding hypertension progresses to develop systolic dysfunction and clinical HFrEF.
- Unlike those who develop HFpEF, these patients appear to develop disproportionate myocyte loss rather than hypertrophy.
- Myocyte death leads to increased wall stress and the shift toward a dilated cardiomyopathy

HF with Reduced Ejection Fraction

- there is a paucity of evidence of a direct progression from HFpEF to HFrEF in these patients, defining how hypertension causes systolic heart failure is more complex.
- A study suggests the idea of a “second hit” leading to accelerated myocyte dysfunction in the background of hypertensive remodeling.

HF with Reduced Ejection Fraction

- Such a second hit may occur from myocardial infarction, medications (eg, anthracyclines), toxins (eg, alcohol or cocaine), or genetic polymorphisms
- Ischemia is by far the most common insult.
- In the Framingham cohort, 42% of hypertensive heart failure patients had preceding myocardial infarction. In addition, LVH and hypertension are potent risk factors for coronary artery disease, creating a synergistic risk profile.

HF with Reduced Ejection Fraction

- One study proposes myocardial infarction as an obligate step in the incidence of systolic heart failure. This likely applies to many but not all patients.
- Decreased renin-angiotensin-aldosterone activity and accelerated collagen breakdown have also been linked to dilated hypertrophy in hypertensive patients.

HF with Reduced Ejection Fraction

- In patients with end-stage systolic heart failure owing to hypertension, blood pressure may paradoxically be low.
- the term “decapitated hypertension” is used to refer to the lower mean blood pressures frequently seen in hypertensive heart failure patients with reduced EF.

HF with Reduced Ejection Fraction

- This finding of low blood pressure may confound the diagnosis of hypertensive heart disease in patients with HFrEF.
- Elevation in blood pressure after initiation of goal-directed medical therapy may be a clue to underlying hypertensive heart disease.

HF with Reduced Ejection Fraction

- Similar to HFpEF, once HFrEF develops in patients with hypertensive heart disease, the prognosis becomes markedly worse.
- The estimated 4-year survival for a patient with symptomatic New York Heart Association (NYHA) class II–III heart failure is only 60%
- Survival is markedly worse for those with NYHA class IV symptoms


Thank You!
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