cultural, ethnical and geographical differences. Cardiac embolic stroke is the leading pathology. Early detection and proper treatment will definitely reduce the socio economic burden. Echocardiography is the simplest non invasive tool for assessment.

Conclusion/observation: Heart is a vital organ, which actively pumps the blood to all vital organs continuously. One of the important sources for CVA is intra cardiac thrombus in general and LV in particular. TTE is an excellent non invasive tool for assessment of gross appearance of thrombus and associated cardiac conditions. Predisposing factors are subendocardial injury, wall motion abnormality and hyper coagulable state of blood. All these factors can reasonably evaluated by Echo. Knowledge of presence of various non thrombotic conditions, normal variants and ultrasonic artifacts are essential to evaluate any differential diagnosis. Echo also helps in monitoring the size of the thrombus with respect to efficiency of medications provided and rule out its presence before any cath intervention. This helps in removal of thrombus along with associated surgical intervention (as in Valve repair). Contrast Echocardiography, TEE and Cardiac MRI are other tests easily available tools for assessment.

Myocardial deformation in hypertensive patients: An analysis using speckle tracking echocardiography

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Introduction: Speckle tracking echocardiography is an important contributing factor to the systolic function of the left ventricle in health and disease. Evaluation of left ventricular (LV) strain using speckle tracking is a sensitive technique used to assess cardiac performance and can be a better index of systolic function than ejection fraction (EF) in hypertensive patients. We postulate that changes in the myocardial strain, as reflected by longitudinal, radial and circumferential strain, may contribute to LV systolic function in hypertensive patients.

Methods: This study evaluated LV myocardial strain in 123 hypertensive patients – 41 with EF <50% (HTLEF group) and 41 with EF \geq 50% (HTNEF group) with heart failure and 41 with EF >50% and no heart failure (HHD). Subjects were consecutively recruited from cardiac and general medicine and nephrology Clinics from January 2013 to December 2014. Inclusion criteria were: documented prior diagnosis of hypertension (measurements on 3 separate occasions where systolic BP was \geq 140 mmHg or diastolic BP was \geq 90 mmHg taken over a period of 2 months at the Hypertension Clinic), documented HF using Framingham Study criteria, sinus rhythm and normal coronary angiography. Myocardial strain imaging was acquired using 2D transthoracic imaging and analysed off line using QLab software (Phillips).

Results: Longitudinal (LS), circumferential (CS) and radial (RS) strain were progressively lower in HTNEF and HTLEF as compared to HHD patients [-11.4 + 0.2 vs. -8.7 + 1.2 vs. -13.5 + 1.33, p < 0.0001 (LS); -11.5 + 0.3 vs. -9.0 + 1.4 vs. -15.2 + 1.69, p < 0.0001(CS); 42.2]2.4 vs. 29.0]1.3 vs. 57.8]4.5, p < 0.0001 (RS)], respectively. Furthermore, in HHD patients, radial and circumferential strain was preserved as compared to HTNEF group. Longitudinal, radial and circumferential strain correlated with ejection fraction [r = 0.56 (LS); r = 0.64 (CS); r = -0.66 (RS), p < 0.0001]; left ventricular mass index [r = 0.62 (LS); r = 0.53 (CS); r = -0.55 (RS), p < 0.0001] and left ventricular sphericity index at end diastole [r = -0.75 (LS); r = -0.70 (CS); r = 0.78 (RS), p < 0.0001] in hypertensive patients.

Conclusion: LV longitudinal strain, which is predominantly governed by the subendocardial region, is the most vulnerable

component of LV myocardial deformation in hypertension and therefore most sensitive to the presence of myocardial disease. In hypertensive patients, abnormalities in baseline myocardial deformation are identified in patients with altered left ventricular geometry. Myocardial strain varies depending on the left ventricle's degree of remodeling and systolic function. Myocardial deformation indices may play a role in reflecting the mechanisms linking altered left ventricular geometry with progression to decompensated left ventricular systolic function.

Cardiac amyloidosis, Congo Red negative: Diagnostic error or a disease begging to be diagnosed?



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Introduction: Restrictive Cardiomyopathy (RCM) is a well known entity characterized by progressive diastolic and subsequently systolic biventricular dysfunction. Of the specific causes of RCM, infiltrative disorders are easily recognizable and have specific characteristics. Amyloidosis tops the list among infiltrative disorders presenting as RCM.

We report a series of 7 patients with amyloidotic and non amyloidotic infiltrative cardiomyopathies, their presentation, diagnosis and implications in the management.

Case series report: Our series includes 7 patients (5 patients were males and 2 were females, with age of 46–58 years). There was no history of hypertension or diabetes. Only one patient had known multiple myeloma and presented subsequently with heart failure and RCM – AL Amyloidosis. 4 patients were diagnosed elsewhere to have non ischemic dilated cardiomyopathy and 2 other patients were diagnosed with hypertrophic cardiomyopathy.

On evaluation, they were diagnosed to be having RCM with possible infiltrative etiology. All had low voltage ECG complexes despite severe Biventricular hypertrophy on the echocardiogram. Echocardiography revealed biventricular hypertrophy, speckled hyperechoic myocardium, thickened inter atrial septum, restrictive pattern of diastolic dysfunction, varying degrees of mitral and tricuspid regurgitation and pulmonary arterial hypertension. Four of them also had severe LV systolic dysfunction with EF of 20-35%. Three patients had reduced global longitudinal strain with "apical sparing" and relative preservation of radial and circumferential strain, a feature in favor of amyloidosis. Free light chains (kappa or lambda) were elevated in all. Serum immunoelectrophoresis revealed monoclonal gammopathy in four. Urine Bence Jones protein was elevated in two. All but one had Congo Red positivity on bone marrow; the remaining patient was Congo Red negative even on endomyocardial biopsy. Non amyloidotic light chain deposition disease was diagnosed by exclusion (reports of electron microscopy and immunohistochemistry are awaited). 6 of them had multisystem involvement (renal dysfunction and proteinuria, hepatic dysfunction).

Five were initiated on chemotherapy with advice for heart transplantation together with Bone marrow transplantation and chemotherapy (Bortezomib regimens). Four died in 6 months from the time of presentation and 3 are on follow up with results of electron microscopy and immunohistochemistry awaited in one.

Conclusion: Cardiac amyloidosis is an underappreciated entity. After our diagnosis of first case of non amyloidotic light chain deposition disease, we have been more systematic in analysing patients diagnosed with RCM. Establishing an etiological diagnosis is very important for initiating appropriate therapy including specific pharmacological measures & has an impact on organ transplantation advice and prognostication.

