

III pattern and in them interestingly normal ECG was seen in 50 % and in another 50 % LVH with no strain was noted. QS pattern was seen in 30.4 % and left atrial abnormality in 21.6 %. TYPE IV pattern was seen in 17 patients and in which 16 patients (94.1 %) had a characteristic V4 R wave > 20 mm with deep T inversions in precordial leads. This unique pattern of electrocardiographic findings had a sensitivity of 93 % for TYPE IV HCM.

Conclusions: Both symmetrical and asymmetrical hypertrophy was noted in cases of HCM with or without obstruction. In cases with asymmetrical hypertrophy (ASH), the hypertrophy was predominantly distributed either in the upper or lower septum, lateral wall and posterior wall. The anterior ventricular septum was predominantly was the most frequently hypertrophied segment of the left ventricle seen in 78.3 %, followed by posterior part of the septum in 58.33 %, and lateral free wall in 35 %. Posterior free wall was preferentially spared and was thickened in only 5 % of the patients. Except for the classical pattern of V4 R wave of more than 20 mm with deep T inversions in precordial leads in 94.1 % of TYPE IV hypertrophy, no other singular electrocardiographic pattern seemed to be very classical for the specific distribution of hypertrophy.

Escherichia coli sepsis as a precipitant of stress cardiomyopathy: A systematic review

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Background: Stress cardiomyopathy (SC) is a novel cardiac syndrome characterized by peculiar, transient left ventricular dysfunction in the absence of significant coronary occlusion. Exacerbations of chronic medical conditions and sepsis are common precipitants that precede the “myocardial stunning.” There is evidence that inflammatory cytokines released by *Escherichia coli* and lipopolysaccharide administration cause decreased cardiac myofilament responsiveness and contractility.

Methods: The present systematic review aimed to report an updated case series of SC precipitated by *E.coli* sepsis in an attempt to summarize their demographic and clinical characteristics. A systematic search through Medline archives accessed by Pubmed (1950-2013) for all such case reports matching our inclusion criterion was performed. Overall, four such cases were eligible and we included our patient with SC with *E.coli* sepsis as the fifth report. Demographic, clinical, electrocardiographic, and echocardiographic characteristics were compared and evaluated.

Results: There was a greater male predominance with an average age of 56 years. The most common ECG abnormalities were ST segment elevation in lateral leads (40%) and anterior leads (60%). There were no fatal outcomes in the cases reported in this analysis. The average time to achieve recovery of clinical, biochemical, and echocardiographic abnormalities was 17 days.

Conclusion: In comparison to most cases of SC reported in literature due to other stressors, *E.coli* sepsis SC are younger and show a greater male predominance. Other unique characteristics of this subset of patients are ST segment elevation in lateral leads, modest increase in troponin levels, and relatively faster complete recovery. Patients in whom *E.coli* sepsis is the precipitating factor of SC have a different spectrum of clinical, biochemical, and electrocardiographic changes. A high index of clinical suspicion is essential in the diagnosis and treatment of these patients.

Effect of Ivabradine in patients with heart failure in whom beta blockers were contraindicated

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Background: Heart failure is associated with high mortality and morbidity. Tachycardia is an important risk factor for adverse outcomes. Beta-blockers are important part of heart failure management as they reduce mortality significantly. We assessed the effect of heart-rate reduction by the ivabradine on outcomes in heart failure in patients in whom beta blockers were contraindicated.

Methods: Patients were eligible for participation in this study if they had symptomatic heart failure with left-ventricular ejection fraction of 35% or lower, were in sinus rhythm with heart rate more than 75 bpm who were admitted with aggravation of symptoms, and beta blockers were contraindicated. Patients were given ivabradine titrated to a maximum of 7.5 mg twice daily or matching placebo. The primary endpoint was the composite of cardiovascular death or hospital admission for worsening heart failure.

Result: We evaluated 1500 patients of heart failure from January 2010 till June 2014. Out of 1500 patients, 800 patients were ineligible for beta-blockers in view of low blood pressure. Out of rest 700 patients, 350 patients received ivabradine and 350 patients were given placebo. 60% patients were males and rest were females. They were followed for more than two years. There was no significant difference in mortality between the two groups as 24 patients from ivabradine and 27 patients from placebo group expired (p - non significant). There was not much difference in hospital admissions due to worsening of heart failure. (82 patients from ivabradine group and 87 patients from placebo group got admitted). No significant side effects were noted with ivabradine. **Conclusion:** There was no significant difference in mortality and morbidity with ivabradine therapy in patients with heart failure in whom beta-blockers were contraindicated. Hospitalization was more or less same in both the groups.

Effects of ivabradine on left ventricular function in patients with ischemic heart failure

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Background: Ivabradine, selective I_f channel blocker, has been shown to reduce recurrent hospitalization due to worsening of heart failure. Role of this agent on left ventricular function in patients with ischemic heart failure is still unexplored.

Aims and objectives: This study was planned to assess the effects of addition of ivabradine to optimal medical therapy (OMT) in patients of ischemic heart failure with systolic dysfunction on Left ventricular function, Exercise duration, Serum Brain Natriuretic Peptide (BNP) levels and Health-related quality of life.

Methods: In this open-label, randomized, a total number of 158 patients of stable, ischemic heart failure were included and were randomly assigned into OMT group (n=80) and ivabradine group (n=78). Baseline assessments included assessment of Left ventricular dimension and Left ventricular ejection fraction (LVEF),

Exercise duration (in seconds) by exercise test and QOL score assessment by Kansas City Cardiomyopathy Questionnaire (KCCQ) and Serum BNP level. In ivabradine group, patients were started on ivabradine 5 mg in twice daily dose, in addition to OMT. Patients were followed up for 6 months. At the end of six months, LV dimensions, LV function, serum BNP levels, QOL and exercise duration were re-assessed.

Results: At six months, though there was significant reduction of heart rate (70.60 ± 5.06 vs 91.33 ± 8.9 , $p < 0.0001$) and improvement of QOL score ($p = 0.004$) and NYHA functional Class ($p = 0.007$) with ivabradine group compared to OMT group, ivabradine failed to show significant improvement in LVEF (35 ± 3.71 vs 33 ± 4.24 , $p = \text{NS}$), Exercise duration (320 ± 130.6 vs 311.79 ± 103.60 , $p = 0.663$) and BNP level (248.64 ± 175.70 vs 312.57 ± 222.6 , $p = 0.22$). Subgroup analysis showed significant improvement in LVEF (35.71 ± 2.98 vs 33.50 ± 3.73 , $p = 0.003$) in patients with ivabradine who achieved heart rate less than 70 ($n = 25$). No significant adverse effects on ivabradine therapy were noted at the end of six months.

Conclusions: Ivabradine when added to optimal medical therapy, in NYHA Functional Class and QoL in patients with ischemic heart failure. Improvement of Left ventricular function also occurs in presence of adequate heart rate lowering ($\text{HR} < 70/\text{min}$).

Efficacy of levosimendan compared with dobutamine in low-output heart failure

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Background: Levosimendan, a calcium channel sensitiser, improves myocardial contractility without causing an increase in myocardial oxygen demand. This study was done to compare the effects of levosimendan and dobutamine on clinical outcome in patients with low-output heart failure.

Methods: Patients were eligible for participation in this study if they had symptomatic low output heart failure. Overall 175 patients were enrolled in this study. Under continuous haemodynamic monitoring, an initial loading dose of levosimendan of 24 mcg/kg was infused over 10 min, followed by a continuous infusion of 0.1 mcg/kg/min for 24 h. Dobutamine was infused for 24 h at a dose of 5 mcg/kg/min. The primary endpoint was the proportion of patients with clinical improvement.

Results: 100 patients were given levosimendan and 75 dobutamine. The clinical improvement was achieved in 28 (28%) levosimendan-group patients and 15 (20%) in the dobutamine group $p = 0.02$. At 6 months, 20 (20%) levosimendan-group patients had died, compared with 25 (33%) in the dobutamine group $p = 0.02$.

Conclusion: In patients with severe, low-output heart failure, levosimendan improved clinical outcome more effectively than dobutamine. Lower mortality was noted in levosimendan group upto 6 months.

Correlation of clinical spectrum, echocardiographic, and angiographic patterns in patients with apical ballooning syndrome in a tertiary care centre of North India

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Background: A cardiac syndrome of "apical ballooning" is being increasingly encountered in routine cardiology practice as it usually mimics acute coronary syndrome at presentation and therefore mandates demonstration of non critical coronary artery disease on coronary angiogram for diagnostic confirmation but its feasibility is scarce in our country. We sought to assimilate clinical, echo-cardiographic and angiographic features of this syndrome at a tertiary care setting from carefully selected cases of apical ballooning and develop an algorithm which should help the emergency physician in making a simpler bedside diagnosis of this syndrome.

Methods: Patients apparently admitted with acute coronary syndrome but subsequently given the diagnosis of transient LV apical ballooning syndrome at our institution from January 2011 to June 2013 were taken prospectively.

Results: Twelve patients were enrolled, mean age was 50 ± 12 years, 10 (83%) were women. Trigger events could be identified in 9 (75%) patients (emotional stress in 3 (25%), post vocal cord surgery in 1 (8%), hemiarthroplasty in 1 (8%), cervical spine surgery in 1 (8%), cervical trauma in 1 (8%), gastrointestinal infection in 1 (8%), road side accident in 1 (8%). Presenting symptoms were; chest pain or discomfort in 3 (25%), NYHA grade III/IV dyspnoea in 9 (75%) patients. 7 (58%) patients had elevated creatine kinase MB and troponin T levels, but the levels were usually only marginally elevated. Electrocardiographic changes observed were ST-segment elevation in 3 (25%), pathological Q waves in 3 (25%), mainly in the leads V_{1-4} . ST-segment depression was found in 4 patients (30%), 3 patients (25%) exhibited T-wave inversion without ST-segment shift. 3 patients presented with cardiogenic shock and 1 patient with ventricular tachycardia. Echocardiographic parameters mean \pm SD LV end-diastolic volume was (115.9 ± 4.0 mL) mean \pm SD LV ejection fraction was ($28.2 \pm 2.5\%$). None of the patients had an E/Em ratio of more than 15. In all 12 patients, left ventricular systolic function recovered completely within three weeks. The systolic strain rate was decreased from base to apex, but the early diastolic strain rate from base to apex was marginally reduced ($+3 \pm 0.5$).

Conclusion: In patients with suspected ABS, clinical history of acute physical/emotional stress with ECG changes mimicking ischemia/infarct, echocardiographic systolic/diastolic paradox with or without contrast echocardiography is helpful in categorization of these patients.

Study of the role of ivabradine in acute heart failure

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Background: Ivabradine is a drug which acts by selectively blocking I_f current in the SA Node. It is approved for use in chronic congestive heart failure. In patients with acute decompensated systolic heart failure, tachycardia could be either a compensatory mechanism or contribute to worsening heart failure. There are situations where using a beta blocker is not an option. The present study was planned to assess the feasibility, safety and efficacy of using Ivabradine in acute heart failure.