Isolated left ventricular noncompaction

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Background: Left ventricular noncompaction is very rare entity with prevalence rate of 0.014–1.3% in western population. There is no report from our country.
We report consecutive cases of isolated left ventricular noncompaction of SIX. We discuss historical, clinical, surface electrocardiogram, and echo-cardiographic features of this condition.
Result: One case has been linked with positive family history, wherein father and son diagnose to have left ventricular noncompaction. In this series of cases, common localization of noncompacted segments were in the apex and lateral wall of left ventricular myocardium. Severe LV systolic dysfunction (EF < 35%) seen in four cases. Grade II mitral regurgitation in four cases and two cases had pulmonary artery hypertension.

Age (years)	Sex	Clinical symptoms		Localization of non–compaction	LVF EF-%	MR	PA Pressure	Inter trabecular recesses	Cardiomegaly (chest X-ray)	ECG
		Dyspnea	Palpitation							
25	M	Class IV	No	Apex and lateral	30	Mild	Normal	8	Yes	Normal
56	M	Class II	No	Apex and lateral	36	Mild	Normal	6	Yes	Normal
39	M	Class II	No	Apex only	50	Trivial	Normal	3	No	Normal
39	M	Class II	No	Apex only	48	Trivial	50	8	No	Normal
65	F	Class IV	No	Apex, lower end of septum and lateral wall	32	Mild	Normal	10	No	Normal
74	F	Class II	No	Apex and lateral	35	Mild	57	15	Yes	Normal

Conclusion: High degree of suspicion in all cases of unexplained severe left ventricular dysfunction is necessary to identify left ventricular noncompaction.

Heart rate manipulation in dilated cardiomyopathy: Assessing the role of ivabradine

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Background: Heart rate reduction has been proven to be of benefit in patients of chronic heart failure (HF). Despite advances in management of HF, the morbidity and mortality remains high, necessitating the development of novel therapeutic strategies. Ivabradine is a selective sinus rate slowing agent which inhibits the cardiac *If current* that is responsible for the spontaneous diastolic depolarization in the sinus node. The role of ivabradine in HF secondary to dilated cardiomyopathy (DCM) merits further study.
Methods: The study included 103 patients with DCM (mean age 47.9 ± 14.8 years, NYHA class 3.2 ± 0.4, BNP 742 ± 489 pg/ml, LVEF 26.3 ± 3.6%. Patients were randomized to receive ivabradine (initiated at 2.5 mg BD titrated up to 7.5 mg BD at intervals of 2 weeks, titrated up to 7.5 mg BD, *n* = 52, *Group 1*) added to standard therapy (beta blockers, ACE inhibitors, diuretics, *n* = 51, *Group 2*). Functional class, BNP levels, and echocardiographic parameters including *LV volumes, LV end-systolic stress (LVESS), LV mass, LV work, and global LV strain* were assessed at baseline, 3 months and 6 months follow-up.
Results: Baseline demographics including age, NYHA Class, BNP levels, 6 min WT and MLWHF (Minnesota Living with HF questionnaire) scores were comparable between the two groups. There was no difference in echocardiographic systolic and diastolic parameters amongst Groups 1 and 2 at baseline, including LVEF 26.06 ± 3.5% vs 26.7 ± 3.1% ($p = 0.34$), LVESS 205 ± 44.3 vs 193 ± 5.8 ($p = 0.2$), LV mass121.7 ± 32 g vs126 ± 39 g ($p = 0.5$), LV work 531.2 ± 146 vs 469 ± 126 mmHg l/m ($p = 0.06$), and LV global strain −10.4 ± 1.3% vs −10.1 ± 1.6% ($p = 0.2$). The mean dose of ivabradine used was 12.1 mg ± 2.4 mg (range 5–15 mg); all patients tolerated ivabradine well, except for rise in serum creatinine requiring withdrawal of the drug in one patient.
3-months follow-up: *Both groups had significant improvement in mean NYHA class* (Gp 1 from 3.3 ± 0.4 to 2 ± 0, $p = 0.001$, Gp 2 from 3.2 ± 0.4 to 2 ± 0.3, $p = 0.03$), *6 min walk test* (Gp 1: 326 ± 42 to 370 ± 38 m, $p = 0.001$, Gp 2: 336 ± 76 to 364 ± 77 m, $p = 0.01$), *MLWHF questionnaire* (Gp 1: 74 ± 14 to 58 ± 11, $p = 0.001$, Gp 2: 78 ± 8 to 65 ± 10, $p = 0.001$) and *fall in BNP* (Gp 1: 760 ± 490 to 382 ± 193 pg/ml, $p = 0.001$, Gp 2: 724 ± 492 to 420 ± 289 pg/ml, $p = 0.01$). *However, the % change in 6 min WT, MLWHF and BNP was much higher in Ivabradine group* (+14%, −21% and −35% vs +9.4%, −16% and −30% respectively with corresponding *p* values of 0.06, 0.02, 0.05). While both groups demonstrated *reduction in HR* (95.6 ± 12 to 80.3 ± 7 bpm, $p = 0.001$ in ivabradine group, 94.6 ± 9 to 87.3 ± 7 bpm, $p = 0.001$ in controls), *the % change was significantly higher in the ivabradine group* (−15% vs −10.2%, $p = 0.03$). No significant reduction in systolic or diastolic BP was seen in the ivabradine group.
At 3 months, amongst those receiving ivabradine, there was significant improvement in indexed LV end-systolic (LVESVi, 86 ± 19 to 73 ± 15 ml/m^2, $p = 0.001$) and LV end-diastolic volumes (LVEDVi 117 ± 34 to 103 ± 19 ml/m^2, $p = 0.001$), MPI (0.82 ± 1.2 to 0.7 ± 0.1, $p = 0.001$) and LVEF (26 ± 3.5 to 29 ± 3.6%, $p = 0.001$). The % change in LVESVi, LVEDVi, MPI and LVEF was −15%, −15%, +11%, and +12.2%, respectively. In contrast, amongst controls there was no significant change in LVESVi (83 ± 20 to 81 ± 21 ml/m^2, $p = 0.2$), LVEDVi (113 ± 27 to 113 ± 24 ml/m^2, $p = 0.9$), MPI (0.79 ± 1 to 0.75 ± 0.1, $p = 0.06$) and LVEF (26.7 ± 3.7 to 26.7 ± 4.5%, $p = 0.9$).
6 months follow-up: At 6 months, while both group of patients had further improvement in NYHA class, 6 min WT, MLWHF score and fall in BNP, the % change in all parameters was significantly higher with ivabradine group – (6 min WT: (19% vs 11.6%, $p = 0.01$, MLWHF score 22% vs 16%, $p = 0.01$, fall in BNP 64% vs 31%, $p = 0.001$). At 6 months, the % reduction in HR was also significantly greater in those on ivabradine (−19.5% vs −7.5%, $p = 0.001$). Importantly, 96.1% achieved HR < 70/min at 6 months on therapy with ivabradine as compared to only 25.6% of those on standard treatment.