International Journal of Research in Medical Sciences
Panda S et al. Int J Res Med Sci. 2022 Dec;10(12):xxx-xxx
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20222988

Original Research Article

Clinical dimensions of hypokinetic non-dilated cardiomyopathy in terms of severity and hospital outcome

Sibaram Panda[1]*, Sunil K. Sharma[1], Mayadhar Panda[2]

[1]Department of Cardiology, VIMSAR, Burla, Odisha, India
[2]Department of Community Medicine, SJMC, Puri, Odisha, India

Received: 20 October 2022
Accepted: 04 November 2022

***Correspondence:**
Dr. Sibaram Panda,
E-mail: drsibaram@gmail.com

ABSTRACT

Background: Hypokinetic non-dilated cardiomyopathy [HNDC/DCM (ND-H)] is a recently proposed (by ESC, 2016) subtype of dilated cardiomyopathy (DCM), which is characterized by the absence of left ventricular (LV) dilatation despite of global LV systolic dysfunction. Knowledge regarding clinical severity and outcomes of patients with DCM (ND-H) is very limited. Objective of the study was to evaluate clinical severity and hospital outcome of patients with HNDC [DCM (ND-H)].
Methods: Total 1248 admitted patients with primary DCM were finalized as study participants considering inclusion and exclusion criteria. The study participants were categorized into two groups depending on presence or absence of LV dilatation. 411 (32.9%) patients without any LV dilatation included in group A [HNDC/DCM (ND-H) group] and 837 (67.1%) patients with LV dilatation included in group B [DCM (D-H) group]. Data with respect to clinical, electrocardiographic, echocardiographic findings and disease outcome of patients compared statistically between the two groups.
Conclusions: HNDC [DCM (ND-H)] is a subclinical subtype, which represents 1/3rd population of DCM. Apart from absence of cardiomegaly, typical clinical signs, electrocardiographic abnormalities, from which we can suspect heart disease, were less prevalent in patients with DCM (ND-H). Therefore, patient most often miss the diagnosis till the advance stage. Non cardiac co-morbidities along with late diagnosis can be important contributing factors for adverse clinical outcomes in patients with DCM (ND-H) comparable to the DCM (D-H) counterpart.

Keywords: DCM, DCM (ND-H), DCM (D-H), MR, S3

INTRODUCTION

Dilated cardiomyopathy (DCM) is the commonest prototype of cardiomyopathy in which systolic dysfunction is typically associated with LV dilatation. However, there is a subtype of DCM that does not meet the standard criteria of LV dilatation in spite of global LV systolic dysfunction. About three decades ago, for the first time Andre Keren presented an excellent insight about the subtype of DCM while studying end stage heart failure patients.[1] Unfortunately the study didn't get the recognition as it deserve. But gradually over a period of time, numerous studies have been attempted to emphasize the same subtype to evaluate clinical dimension, however uniformity has not been maintained among them while defining the disease subtype due to use of different terminologies, inclusion criteria and cut off values for EF, LVEDD.[1-6] Therefore for early diagnosis and prevention of progression of the disease subtype, in 2016 European society of cardiology (ESC) proposed a new terminology HNDC [DCM (ND-H)],which is defined as left ventricular or biventricular global dysfunction (EF<45%) without dilation not explained by abnormal loading condition or CAD.[7] Prevalence of patients with HNDC is 0.9-1.9% among population as per a recent study.[8] As newer studies completely based on ESC criteria are very few in number

enrolling very small study populations, so knowledge about clinical profile, severity and outcomes of patients with HNDC is very limited. Therefore, evaluation of clinical severity and hospital outcome of patients with HNDC is highly essential in this regard.

METHODS

An analytical cross sectional study was conducted in the department of cardiology, VIMSAR, Burla, during the period August 2019 to August 2022. As per departmental protocol, total of 8569 patients with suspected heart failure had been undergone echocardiography in the cardiology department during the above-mentioned study period, out of which 2877 numbers of patients found to have echocardiographic feature of dilated cardiomyopathy (DCM) [i.e. EF<45 % with global hypokinesia]. Total 1629 patients with secondary DCM (e.g., arrhythmic cardiomyopathy, post-partum cardiomyopathy, alcoholic cardiomyopathy, hypertensive cardiomyopathy and ischemic cardiomyopathy and drug induced cardiomyopathy etc.) were excluded from the study. Finally, 1248 patients with primary DCM were enrolled as study participants. The enrolled study participants were categorised into two groups basing on presence or absence of LV dilatation on Echocardiography. LV dilatation defined as left ventricular end diastolic diameter (LVEDD) >2SD from normal, according to adult normogram corrected by age and gender.[7] Patients without LV dilatation included in group A [HNDC/DCM (ND-H) group] and patients with LV dilatation included in group B (DCM (D-H) group). Data were collected in prescribed format after obtaining informed written consent from all the study subjects about baseline characteristics of patients like age, sex, risk factors like type-2 DM, smoking, obesity, family history, CKD. Family history of disease was elicited strictly based on proposed ESC criteria.[7] Clinical symptoms (dyspnea, fatigability, syncope, and chest pain) and signs (raised JVP, canon wave, oedema, cyanosis, hepatomegaly, crepitation, rhonchi, S3, S4, any murmuror abnormal heart sounds) duly noted in every patient. Electrocardiography (ECG) abnormalities (LVH, RVH, LAE, RAE, LBBB, RBBB), echocardiographic findings (EF, MR with grading, TR with grading, right atrial pressure, pericardial effusion, LV clot) were recorded. Study findings of the two groups were analysed and appropriate statistical tests were applied to test the significance.

Statistical analysis

Categorical data were analysed using statistical tests e.g., proportions, percentages, Chi-square test (X^2) and fishers extract test. Quantitative data were analysed with mean, standard deviation and un-paired t-test was applied to compare the means between the two groups. P value less than 0.05 was considered significant and p value less than 0.01 was taken as highly significant (HS) and P value more than 0.05 considered as non-significant (NS).

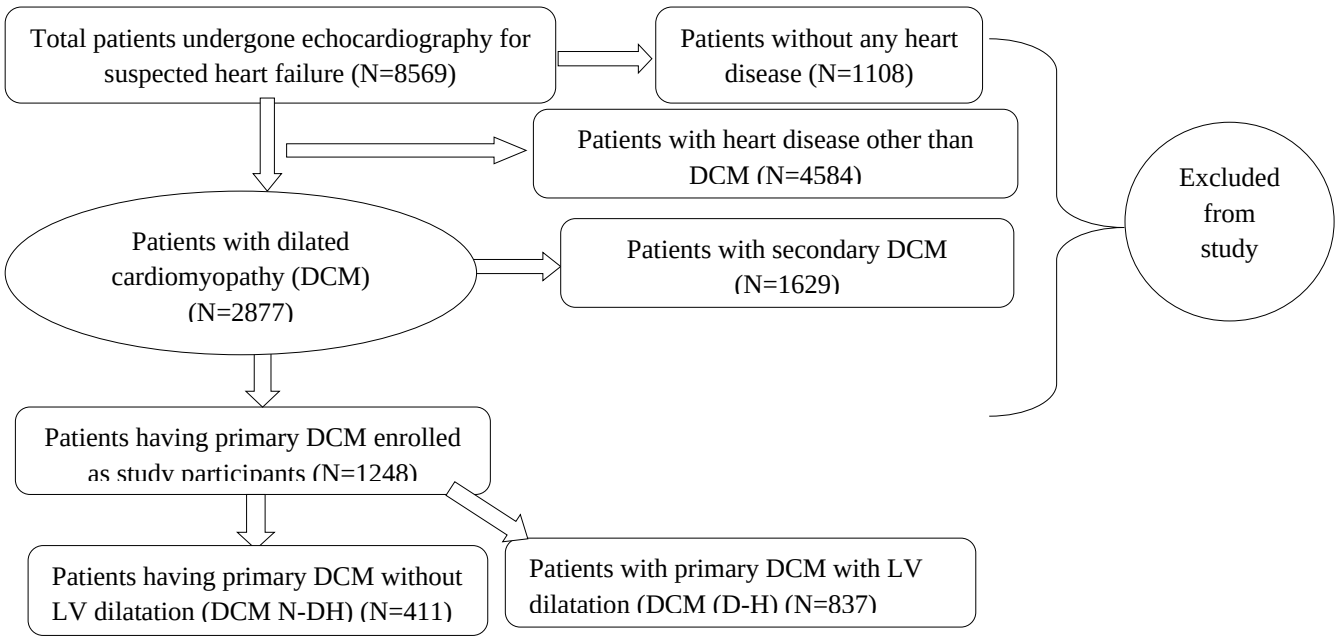

Figure 1: Selection of study subjects.

RESULTS

Out of 1248 patients, 411 (32.9 %) patients found to have DCM (ND-H) and 837 (67.1%) patients found to have DCM (D-H) (Figure 2). With respect to the baseline characteristics and comorbidities, as evidenced from Table 1 that, mean age of the patients with DCM (ND-H) was 49.2±8.8 years as compared to 53.2±7.6 years in patient

with DCM (D-H) and the difference in means between the groups was found statistically highly significant with p value <0.001. Male sex involvement with DCM (ND-H) was less in proportion i.e., 47% in comparison to 78% with DCM (D-H) group, which was found statistically highly significant. On the other hand, positive family history was associated significantly in higher number of cases with DCM (ND-H) as compared to DCM (D-H) patients (Table 1). While studying the associated co-morbid conditions, diabetes, obesity and CKD was found in 112 (29.6 %), 115 (27.9 %) and 73 (17.7 %) patients respectively in DCM (ND-H) group as compared to 164 (19.5%), 172 (20.6%) and 93 (11.1%) patients with DCM (D-H) respectively and the difference between the groups was found to be statistically significant (Table 1).

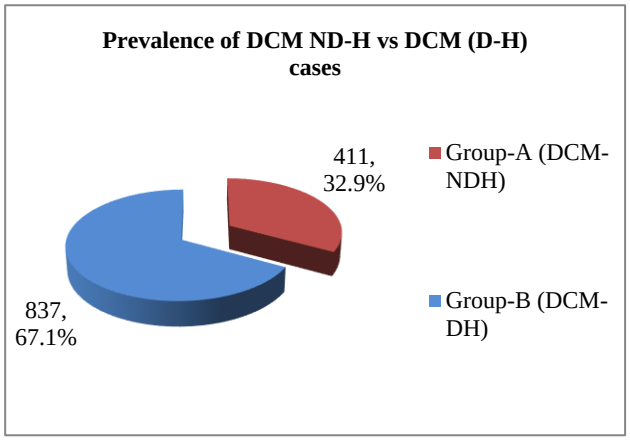

Figure 2: Prevalence of DCM (ND-H) versus DCM (D-H) cases.

While comparing clinical feature of two groups (table-2), S4 found in 182 (44.2%) patients with DCM (ND-H) as compared to 267 (31.8%) patients with DCM (D-H), which was found highly significant statistically with p value <0.001, whereas significant difference in proportion was found with respect to S3 i.e., 393 (46.7%) patients with DCM (D-H) as compared to 154 (37.4%) with DCM (ND-H) with p value=0.0015, that suggests S3 found in comparatively higher proportion of cases with DCM (D-H) as compared to DCM (ND-H).

While studying electrocardiographic features of two groups (table-3), we found LVH, LBBB, LAE in 95 (23.1%), 95 (23.1%), 54 (13.1%) patients with DCM (ND-H) as compared to 293 (35%), 309 (36.9%) and 187 (22.3%) patients with DCM (D-H) respectively and the above ECG changes found in proportionately higher number of cases with DCM (D-H) as compared to DCM (ND-H) which was also found statistically significant, whereas no significant difference was noticed between the two groups in terms of the findings of RVH, RAE.

As depicted in Table 4, mean LVEF in DCM (ND-H) group is higher i.e. 33.7±8.1 as compared to 30.7±7.6 in DCM (D-H) group with p value <0.001. RV dysfunction found in 182 (44.2%) patients with DCM (ND-H) as compared to 394 (47%) patients with DCM (D-H) without any statistical significance. MR found in 279 (67.8%) patients with DCM (ND-H) as compared to 609 (72.7%) patients with DCM (D-H) without any significant difference, however statistically significant number of cases with DCM (D-H) were associated with severe MR i.e., 115 (13.7%) patients with DCM (D-H) as compared to 32 (7.7%) patients with DCM (ND-H) with p value=0.002.

Regarding disease outcome of hospitalised patients, mean duration of hospital stay among patients with DCM (ND-H) and DCM (D-H) was 5.8±2.1 days and 6.1±1.9 days respectively and found statistically non-significant (p value=0.0261) (Table 5). Out of 411 patients with DCM (ND-H), 58 (14.1%) patients required ventilation (non-invasive/invasive) due to frank pulmonary oedema, 34 (8.2%) patients developed cardiogenic shock, and 33 (8.0%) patients died. Whereas out of 837 patients in group DCM (D-H), 142 (16.9%) patients required ventilation due to frank pulmonary oedema, 96 (11.4%) patients were on inotrope due to cardiogenic shock, 81 (9.6%) patients died. No significant difference in terms of disease outcomes was noticed among the two groups as shown in Table 5.

Table 1: Comparative analysis of baseline characteristics and comorbidities between DCM (ND-H) and DCM (D-H).

Baseline characteristics and comorbidities	Group-A (%) [DCM (ND-H)]	Group-B (%) [DCM (D-H)]	P value
Mean age in years	49.2±8.8	53.4±7.6	0.001(HS)
Sex			
Male	193 (47)	652 (78)	<0.0001(HS)
Female	218 (53)	185 (22)	
Positive family history	93 (22.6)	139 (16.6)	0.01(S)
H/O smoking	66 (16)	151 (18)	0.38 (NS)
Dyslipidaemia	86 (20.9)	182 (21.7)	0.74 (NS)
Diabetes	112 (29.6)	164 (19.5)	0.002 (HS)
Obesity	115 (27.9)	172 (20.6)	0.003 (HS)
CKD	73 (17.7)	9 (11.1)	0.001(HS)

Table 2: Comparison of clinical features between DCM (ND-H) and DCM (D-H).

S. no.	Clinical feature	Group-A (DCM (ND-H))	Group-B (DCM (D-H))	Significance
		No. (%)	No. (%)	P value
1	Oedema	116 (40.3)	376 (44.9)	0.12 (NS)
2	Raised JVP	152 (36.9)	343 (40.9)	0.175 (NS)
3	MR	279 (67.8)	609 (72.7)	0.73(NS)
4	S3	154 (37.4)	393 (46.9)	0.0015(S)
5	S4	182 (44.2)	267 (31.8)	0.001(HS)
6	Crepitation	172 (41.8)	368 (43.9)	0.239 (NS)

Table 3: Comparative analysis of electrocardiographic findings between DCM (ND-H) and DCM (D-H).

S. no.	ECG abnormality	Group-A [DCM (ND-H)]	Group-A [DCM (D-H)]	P value
		No. (%)	No. (%)	
1	Axis			
	LAD	33 (8.02)	150 (17.9)	0.001 (HS)
	RAD	23 (5.5)	69 (8.24)	*0*.092 (NS)
2	Bundle			
	LBBB	95 (23.1)	309 (36.9)	<0.001 (HS)
	RBBB	15 (3.65)	51 (6.09)	0.69 (NS)
3	Atrial enlargement			
	LAE	54 (13.1)	187 (22.3)	<0.001 (HS)
	RAE	22 (5.35)	67 (8.0)	0.08 (NS)
4	Hypertrophy			
	RVH	23 (5.6)	71 (8.4)	0.69 (NS)
	LVH	95 (23.1)	293 (35.0)	<0.001 (HS)
	BOTH	17 (4.13)	76 (9.0)	0.001 (HS)

Table 4: Comparison of echocardiographic features between DCM (ND-H) and DCM (D-H).

S. no.	Echo-cardiographic parameters	Group-A [DCM N-DH] N1=411	Group-B [DCM (D-H)] N2=837	P value
1	EF (%)			
	Mean ejection fraction	33.7±8.1 (mean±SD)	30.7±7.6 (mean±SD)	<0.001 (HS)
	40-45	119 (28.9)	201 (24.0)	0.6 (NS)
	30-39	154 (37.4)	292 (34.8)	0.37 (NS)
	<30	138 (33.5)	345 (41.2)	0.009 (S)
2	MR			
	Mild	118 (28.7)	199 (23.7)	0.06 (NS)
	Moderate	129 (31.3)	295 (35.2)	0.17 (NS)
	Severe	32 (7.7)	115 (13.7)	0.002 (S)
3	RV dysfunction	182 (44.2)	394 (47.0)	0.35 (NS)
4	Pericardial effusion	30 (7.2)	88 (10.5)	0.06 (NS)
5	LV clot	2 (0.48)	9 (1.07)	0.29 (NS)

Table 5: Comparison of disease outcome of patients between DCM (ND-H) and DCM (D-H).

Disease outcome	DCM (ND-H) (group-A) N1=411 (%)	DCM (D-H) (group-B) N2=837 (%)	P value
Duration of hospital stay	5.9±2.1 (mean±SD)	6.1±1.9 (mean±SD)	0.0918 (NS)
Patients required ventilation	58 (14.1)	142 (16.9)	0.2047 (NS)
Patients on inotrope	34 (8.2)	96 (11.4)	0.081 (NS)
Death	33 (8.0)	81 (9.6)	0.34 (NS)

DISCUSSION

DCM (ND-H) is a subset of dilated cardiomyopathy, which stands with the concept of LV dysfunction in the absence of LV dilatation. Prior to the proposal of DCM (ND-H), numerous older studies emphasized the same concept utilising different terminologies like; mildly dilated cardiomyopathy (MDCM), non-dilated cardiomyopathy (NDCM). NDCM was defined as LV dysfunction with normal LV dimension.[5] Whereas MDCM was defined as LV dysfunction with a normal or mildly dilated left ventricle.[6] Cut off value for LV dimension was 15% higher in MDCM in comparison to NDCM.[2] Older studies also used different cut off values for EF, like 30%, 40%, 45%, while defining above terminologies.[1-6] Most of the above studies had not utilised normogram for LV dimension, though it varies with age, sex, and body surface area. Conditions like HTN, CAD, chronic AF, which can cause cardiomyopathy, were included while defining the disease.[4,5] Whereas the new ESC criteria exclude HTN, CAD while defining DCM (ND-H) and places arrhythmic cardiomyopathy in a separate subgroup. It firmly fixes the cut off value of EF at 45% to maintain the uniformity and recommends to utilise normogram for LV end-diastolic dimension, while diagnosing the disease.[7] Current study is completely based on the ESC criteria of DCM (ND-H), to find out actual dimension of DCM (ND-H) in terms of clinical profile and hospital outcome.

Current study population comprised of 1248 patients, out of which 411 (32.9%) patients found to have DCM (ND-H), whereas 837(67.1%) patients found to have DCM (D-H) (Figure 2). Mean age of the patients with DCM (ND-H) is significantly lower (i.e. 49.2±8.8 years) as compared to patients with DCM (D-H) (i.e. 51.8±7.6 years). Guoet al found that, DCM (ND-H) is more prevalent in the age group 35-45 year.[8] As Guoet al enrolled participants from general populations whereas the current study enrolled hospitalised patients comparatively at advanced stage, so DCM (ND-H) has been observed in comparatively older population in the current study as compared to study by Guoet al. In the present study, out of 411 DCM (ND-H) patients, 193 (48.8%) patients were male, 218 (51.2%) were female. Guoet al observed that, prevalence of DCM (ND-H) slightly higher in females as compared to males (2% versus 1.7%), which is concordance to the finding of present study.[8]

While studying the associated comorbidity, it was found that, diabetes, obesity, CKD in 29.6%, 27.9% and 17.7% in DCM (ND-H) patients as compared to 19.5%, 20.6% and 11.1% in DCM (D-H) patients respectively. Whereas Trimothy et al also found frequent association of comorbidities like HTN in 47%, chronic AF in 42%, diabetes in 23%, CKD in 14% in patients with non-dilated cardiomyopathy.[4] Frequent association of non-cardiac comorbidities in patients with heart failure can adversely affect disease outcomes in patients with DCM (ND-H).[9]

In current study, positive family history found in 22.6% patients with DCM (ND-H) as compared 16.6% patients with DCM (D-H) which was found to be statistically significant. Similar finding was observed in the study by Gigli et al, where positive family history associated in 23% patients with MDCM as compared to 18% patients with DCM (D-H).[6] Keren et al found positive family history in 56% patients with MDCM in CHF.[1] So from above discussion it can be concluded that, positive family history is strongly associated with DCM (ND-H)and its related groups (MDCM and NDCM). Presence of positive family history in DCM (ND-H) patients more commonly associated with laminin mutation, which can predispose patients to arrhythmia and SCD.[7,10]

While comparing electrocardiographic findings of the two groups, it was noticed that, significantly lower proportion of DCM (ND-H) cases were associated with ECG features like LVH, LBBB as compared to cases with DCM (D-H). Similar result was observed by Mombeini et al where, LBBB found in 16.1% patients with DCM (ND-H), in comparison to 43.5% in patients with DCM (D-H) with p value 0.06 though the study included limited number of patients.[11] Gigli et al also found LBBB in 24% in MDCM patients in comparison to 39% in DCM patients with p value <0.001.[6]

While studying clinical and echocardiographic features, S4 found in 182 (44.2%) patients with DCM (ND-H) as compared to 267 (31.8%) patients with DCM (D-H) which was found statistically highly significant with p value <0.001. Association of co-morbidities like diabetes, CKD, obesity can decrease ventricular compliance and which in turn cause S4 in patients with DCM (ND-H).[12] Whereas S3 found in proportionately higher number of cases i.e., 393 (46.7%) patients with DCM (D-H) as compared to 154 (37.4%) with DCM (ND-H) with p value=0.0015. Higher prevalence of severe MR, LV dysfunction may be most probable cause for higher prevalence S3 in patient with DCM (D-H).[13] Mean LVEF in DCM (ND-H) group found to be higher i.e. 33.7±8.1 as compared to 30.7±7.6 in DCM (D-H) group and the difference in means found statistically highly significant with p value <0.001. Although there is no significant difference in prevalence of MR between two groups, however severe MR found in 13.7% patients with DCM (D-H) as compared to 7.7% patients with DCM (ND-H) with p value=0.002 found significant as depicted in Table 4. Gigli et al also found moderate to severe MR in 24% patients with MDCM as compared 39 % patients with DCM (D-H) with p value <0.001.[6] While studying non-ischemic dilated cardiomyopathy with severe LV dysfunction, Gupta et al found significantly higher prevalence of MR in severely LV dilated group as compared to normal or mild to moderately LV dilated groups.[14] Higher prevalence of severe MR in patients with DCM (D-H) may be due to Mitral annular dilatation, increased inter papillary muscle distance, amplified leaflet tethering.[15] From the above discussion, it can be implied that, apart from absence of cardiomegaly (clinical/radiological feature suggestive of LV

dilation),typical clinical signs (s3, clinically detectable severe MR), electrocardiographic abnormalities (LBBB, LAE, LVH), from which we can suspect heart disease, were less commonly present in patients with DCM (ND-H). Therefore patients can miss their diagnosis at early stage and progress to late advanced stage with disastrous complications.

While studying disease outcomes in terms of complications like pulmonary oedema, cardiogenic shock, duration of hospital stays along with mortality, no significant difference was observed among the two groups (Table 5). However, few older studies found that, patients with DCM (ND-H) related groups (NDDM, MDCM) have better clinical outcomes as compared to DCM (D-H) patients.[3-5] Those studies included a good proportion of patients with hypertension and chronic AF, which can cause cardiomyopathy. Whereas secondary cardiomyopathy (like arrhythmic cardiomyopathy, hypertensive cardiomyopathy) had been excluded in present study. As patients with cardiomyopathy either due to chronic HTN or AF, have better clinical outcome than patients with DCM (D-H), therefore above studies might have found less severe clinical outcomes in DCM (ND-H) related group of patients.[16,17] Whereas a recent study confirmed that, LV dimension has no role in prediction of clinical severity and outcome of patients with non-ischemic cardiomyopathy with LV dysfunction.[12] Frequent association of non-cardiac comorbidities in patients with heart failure have a role for higher mortality and adverse outcomes.[8] As patient with HNDC [DCM (ND-H)] had higher prevalence of comorbidities as compared to patients with DCM (D-H), therefore even if mean EF remains higher in DCM(N-DH) group, disease outcome in terms of mortality, duration of hospital stay and complications found comparable to that of DCM (D-H) counterpart. Secondly patients with DCM (ND-H) presents in late advanced stage due to subtle clinical, radiological and electrocardiographic sign, which may be a reason for severe adverse outcomes in patients with DCM (ND-H).

CONCLUSION

Burden of HNDC (DCM (ND-H)) is very high, representing about 1/3rd of the DCM population. Apart from absence of cardiomegaly, typical clinical signs (s3, clinically detectable severe MR), electrocardiographic abnormalities (LBBB, LAE, LVH), from which we can suspect heart disease, were less prevalent in patients with DCM (ND-H). Frequent association of non-cardiac comorbidities along with late diagnosis are the important contributing factors for adverse outcomes. Therefore, even if mean EF remains higher in this subtype, severity and disease outcome in terms of mortality, duration of hospital stays and complications is comparable to that of DCM (D-H) counterpart. As this subtype represents a clinically submerged population with lethal outcomes, hence need to be addressed appropriately. Therefore, the patients with subclinical features in the clinical setting of suspected heart failure should not be left as such without undergoing a screening echocardiography, so that early diagnosis can be ascertained.

ACKNOWLEDGEMENTS

Authors would like to thank patients for their adherence and kind cooperation in study and the staff and technicians of the department for performing different tests required in the study.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Keren A, Gottlieb S, Tzivoni D, Stinson EB, Popp RL. Mildly dilated congestive cardiomyopathy. Use of prospective diagnostic criteria and description of the clinical course without heart transplantation. Circulation. 1990;81:506-17.
2. Gavazzi A, De Maria R, Renosto G, Moro A, Borgia M, Caroli A, et al. The spectrum of left ventricular size in dilated cardiomyopathy: clinical correlates and prognostic implications. SPIC (Italian Multicenter Cardiomyopathy Study) Group. Am Heart J. 1993;125(2):410-22.
3. Cioffi G, Tarantini L, De Feo S, Pulignano G, Del Sindaco D, Stefenelli C, Opasich C. Dilated versus nondilated cardiomyopathy in the elderly population treated with guideline-based medical therapy for systolic chronic heart failure. J Card Fail. 2004;10(6):481-9.
4. Draper TS, Schick EC, Gaasch WH. Non-Dilated Cardiomyopathy: Changes in Left Ventricular Size and Function over Time and the Impact of Atrial Fibrillation. Cardiovascular Structure. 2009;15(6):S31.
5. Doumas A, Draper TS Jr, Schick EC, Gaasch WH. Prevalence and clinical characteristics of nondilated cardiomyopathy and the effect of atrial fibrillation. Am J Cardiol. 2010;105(6):884-7.
6. Gigli M, Stolfo D, Merlo M, Barbati G, Ramani F, Brun F, Pinamonti B, Sinagra G. Insights into mildly dilated cardiomyopathy: temporal evolution and long-term prognosis. Eur J Heart Fail. 2017;19(4):531-9.
7. Pinto YM, Elliott PM, Arbustini E, Adler Y, Anastasakis A, Böhm M, et al. Proposal for a revised definition of dilated cardiomyopathy, hypokinetic non-dilated cardiomyopathy, and its implications for clinical practice: a position statement of the ESC working group on myocardial and pericardial diseases. Eur Heart J. 2016;37(23):1850-8.
8. Guo X, Li Z, Guo L, Yu S, Li T, Zheng L, Pan G, Yang J, Sun Y, Hui R, Pletcher MJ. Prevalence of hypokinetic non-dilated cardiomyopathy in a large

general Chinese population. Int J Cardiol. 2016;223:708-710.
9. Sharma A, Zhao X, Hammill BG, Hernandez AF, Fonarow GC, Felker GM, et al. Trends in Noncardiovascular Comorbidities Among Patients Hospitalized for Heart Failure: Insights From the Get With The Guidelines-Heart Failure Registry. Circ Heart Fail. 2018;11(6):e004646.
10. Verdonschot JAJ, Hazebroek MR, Wang P, Sanders-van Wijk S, Merken JJ, Adriaansen YA, et al. Clinical Phenotype and Genotype Associations With Improvement in Left Ventricular Function in Dilated Cardiomyopathy. Circ Heart Fail. 2018;11(11):e005220.
11. Mombeini H, Parsaee M, Amin A. Speckle tracking echocardiography in hypokinetic non-dilated cardiomyopathy: comparison with dilated cardiomyopathy. ESC Heart Fail. 2020;7(4):1909-16.
12. Mohammed SF, Borlaug BA, Roger VL, Mirzoyev SA, Rodeheffer RJ, Chirinos JA, Redfield MM. Comorbidity and ventricular and vascular structure and function in heart failure with preserved ejection fraction: a community-based study. Circ Heart Fail. 2012;5(6):710-9.
13. Silverman ME. The Third Heart Sound. In: Walker HK, Hall WD, Hurst JW, editors. Clinical Methods: The History, Physical, and Laboratory Examinations. 3rd ed. Boston: Butterworths; 1990.
14. Gupta A, Sharma P, Bahletal A. Left ventricular size as a predictor of outcome in patients of non-ischemicdilated cardiomyopathy with severe left ventricular systolic dysfunction. Int J Cardiol. 2016;221:310-30.
15. Al-Amri HS, Al-Moghairi AM, El Oakley RM. Surgical treatment of functional mitral regurgitation in dilated cardiomyopathy. J Saudi Heart Assoc. 2011;23(3):125-34.
16. Takarada A, Yokota Y, Kumaki T, Toh S, Seo T, Kubo M, Maehashi N, Fujitani K, Furuta Y, Fukuzaki H. [Hypertensive heart disease simulating dilated cardiomyopathy]. J Cardiogr. 1985;15(4):1015-26.
17. Takarada A, Kurogane H, Hayashi T, Fujimoto T, Yasaka Y, Fukumoto Y, Shimatani Y, Yamakawa H, Sasaki S, Matsuura A, et al. Prognostic significance of atrial fibrillation in dilated cardiomyopathy. Jpn Heart J. 1993;34(6):749-58.

Cite this article as: Panda S, Sharma SK, Panda M. Clinical dimensions of hypokinetic non-dilated cardiomyopathy in terms of severity and hospital outcome. Int J Res Med Sci 2022;10:xxx-xx.