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ETIOLOGIC AND DEMOGRAPHIC PROFILE OF HEART FAILURE PATIENTS IN NORTHERN INDIA

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ABSTRACT

Background: Heart failure is a major public health problem. Incidence of heart failure increases with age and is higher in men.[2] Incidence of heart failure among persons aged > 45 yrs is 7.2/1000 in men and 4.7/1000 in women.[3] Coronary heart disease , hypertension and diabetes , singly or in combination , predominate as etiologies for heart failure in developed world whereas in developing world Rheumatic heart disease continues to be leading cause of heart failure.[11] Heart failure is highly lethal, with 5 yrs survival rate of 25 % in men and 38 % in women.[2,4] Bad Functional class and ischemic etiology are the most important prognostic factors for heart failure.[5,6,7,8] In addition shorter 6-minute walk test distance , anaemia , renal dysfunction are other bad prognostic factors.
Material & Methods: This study was prospective cohort study, conducted in the department of cardiology Sher-i-Kashmir institute of Medical Sciences , Soura , Srinagar. Total of 350 patients were studied and 240 patients were included in the study. Framingham‘s criteria were used to identify heart failure patients. All those patients whose dyspnea was not clearly secondary to CHF were excluded . All relevant data of patients were recorded including past history , present history in detail , physical examination , baseline blood investigations like haemoglobin , total leucocyte count , creatinine , uric acid , LFT ; and electrocardiogragh and chest X-ray. Transthoracic echocardiography was performed in all patients. Selected patients were regularly followed in ward and outcome was noted. Those who survived there NYHA class and 6 minute corridor walk test was noted at time of discharge.
Results: Total of 240 patients were studied, 56.7% were male. Majority were in 60-69 years age group comprising 31.7 %. Among 240 patients, 32 patients died during hospital stay (overall mortality of 13.5 %) comprising of 20 males (14.7 %) and 12 females (11.5 %) ,without statistically significant gender difference (p=0.723). Among studied parameters following had a statistically significant relation with adverse outcome: low blood pressure and tachy- cardia at admission, worse NYHA class , pulmonary edema, low hemoglobin , raised creatinine and uric acid (> 7 mg/dl) and depressed ejection fraction [< 34.0 %].
Conclusion: Poor Functional class and ischemic etiology are the most important prognostic factors for heart failure. Shorter 6-minute walk test distance , anaemia , renal dysfunction are other bad prognostic factors. These factors can help identify poor prognosis patients and help prioritise level of care in our overburdened health care system.
Key Words: Heart failure, etiology, prognostic factors.

INTRODUCTION

Heart failure is a complex Clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. The clinical features of heart failure are dyspnea and fatigue which limits the exercise tolerance ; and causes fluid retention which may lead to pulmonary congestion and peripheral edema. Both impair functional capacity and lead to frequent hospitalizations, resulting in poor quality of life and a reduced life expectancy1.

Heart failure is a major health problem in developing nations2. Heart failure is affecting approximately 1.3 to 4.6 million Indians3,4. Incidence of heart failure rises with age and is higher in men5. The annual age adjusted incidence of heart failure in persons aged 45 yrs or more is 7.2/1000 in men and 4.7/1000 in women , and age adjusted prevalence of heart failure is 24/1000 in males and 25/1000 in females5.

Heart failure carries a poor prognosis, with a median survival of 1.7 yrs in males and 3.2 yrs in females5,6. Survival is better in females than in males and mortality increases with advancing age6. In United States mortality related to heart failure is estimated at 20.2 deaths per lac of population7.

In the Framingham study , coronary heart disease , hypertension and diabetes predominate as etiologies for heart failure8. About 40 % of heart failure in the Framingham study was attributable to a combination of coronary heart disease and hypertension; and 19 % of heart failure in males and 7 % in females were due to coronary artery disease in isolation , so that more than 50 % of the cardiac failure had coronary heart disease as a major contributing cause. Around 40 % of heart failure was attributable to hypertension. Hypertension only or in combination with coronary artery disease was responsible for about 60-70 % of the cardiac failure8. Around 10 % of heart failure are due to cardiomyopathies in the general population.

Aims And Objectives

- To determine etiology of heart failure in northern India.
- To determine demographic profile of heart failure patients in north India.

MATERIALS AND METHODS

This study was conducted in the department of cardiology of Sher-i-Kashmir institute of Medical Sciences , Soura , Srinagar over a period of 3 years. Total of 350 patients were studied , who presented to the department of cardiology with heart failure. The symptoms used to identify heart failure patients were orthopnea, Paroxysmal nocturnal dyspnea, increase in weight, pedal oedema, cough or wheezing9. All those patients whose dyspnea was not clearly secondary to Heart failure (trauma, COPD, pericardial effusion) were excluded. Framingham‘s criteria were used to identify heart failure patients. After exclusion 240 patients were included in the study.

Once the patients was selected for study, consent was taken and same time all relevant data of patients were recorded including demographic parameters, past history, present history in detail, physical examination, baseline blood investigations like haemoglobin, total leucocyte count, creatinine, uric acid, LFT; and electrocardiogragh and chest X-ray. In ECG heart rate , rhythm , IVCD were noted. On echocardiography we examined systolic as well as diastolic function. Important parameters which were noted are LVEDD (left ventricular end-diastolic dimension), LVESD (LV end-systolic dimension), EF (ejection fraction), EDV (end-diastolic volume), ESV (end-systolic volume) and mitral inflow velocity (ratio of E/A , deceleration time). We defined LV systolic dysfunction in our study by EF ≤ 40 % ; and for diastolic dysfunction we divided it into 4 grades : Grade I dysfunction (impaired relaxation) – ratio of E/A < 1 and deceleration time > 240 milliseconds , Grade II dysfunction – ratio of E/A > 1 and deceleration time > 160 milliseconds , Grade III dysfunction (restrictive pattern which is reversible) – ratio of E/A > 1.5 and deceleration time of 160 milliseconds ; and Grade IV dysfunction (restrictive pattern which is irreversible) – ratio of E/A > 1.5 and deceleration time of < 160 milliseconds.

Selected Patients were regularly followed in ward ; outcome was noted (how many died and how many survived) in all 240 selected patients. Those who survived there NYHA class and 6 minute corridor walk test was noted at time of discharge.

Statistical Analysis

Data was described as mean±SD and percentage. The intergroup compa- rison was made by students t – test , mann-whitney U test, and chi-square test . Besides bivariate correlation by EtwartSpareman was done. P-valuof < 0.05 was considered significant and software used was SPSS 11.5, Minitab and MS excel0.

RESULTS AND DISCUSSION

Table 1 showing age and sex distribution of HF patients.8. [Table 1]

Table 2 showing gender wise distribution of comorbities (hypertension and diabeties) and etiological diagnosis of HF. [IHD=Ischemic heart disease, AF=Atrial fibrillation related HF,DCM=Dilated Cardiomyopathy,RHD=Rheumatic heart disease,HTCVD=Hypertensive cardiovascular disease and PCM=Peripartum cardiomyopathy]. [Table 2]

Table 3 showing baseline characteristics of total 240 patients with gender distribution; their values were expressed as mean±SD (for pulse and blood pressure) and in number and percentage for rest of variables with their P – value

[SBP=systolic blood pressure and DBP=diastolic blood pressure]. [Table 3]

Table 4 showing chest x-ray, ECG and Echo findings at time of presentation with gender distribution; Ejection fraction (EF) were expressed as mean±SD and rest of variables were expressed in percentages. [Table 4]

Table 5 Showing biochemical profile of patients in relation to gender distribution. [Table 5]

Table 1: Age and Gender Distribution of the Heart Failure Patients

Age (yr)	Male		Female		Total		p value
	n	%	n	%	n	%	
< 40	8	5.9	16	15.4	24	10.0	0.223 (NS)
40 to 49	16	11.8	16	15.4	32	13.3	
50 to 59	36	26.5	28	26.9	64	26.7	
60 to 69	52	38.2	24	23.1	76	31.7	
≥ 70	24	17.6	20	19.2	44	18.3	
Total	136	56.7	104	43.3	240	100.0	
mean ± SD	58.9 ± 10.8 (35, 75)		55.0 ± 13.8 (28, 76)		57.2 ± 12.2 (28, 76)		

Table 2: Etiological Diagnosis of the Heart Failure Patients

		Male		Female		Total		p value
		n	%	n	%	n	%	
Comorbidity	Hypertension	72	52.9	72	69.2	144	60.0	0.202 (NS)
	Diabetes Mellitus	20	14.7	16	15.4	36	15.0	0.942 (NS)
Diagnosis	IHD	36	26.5	24	23.1	60	25.0	0.514 (NS)
	AF	4	2.9	4	3.8	8	3.3	
	DCM	28	20.6	12	11.5	40	16.7	
	RHD	24	17.6	28	26.9	52	21.7	
	HTCVD	44	32.4	28	26.9	72	30.0	
	PCM	0	0.0	8	7.7	8	3.3	

Table 3: Clinical profile of the Heart Failure Patients

Characteristics		Male		Female		Total		p value
		n	%	n	%	n	%	
NYHA(Admission)	II	40	29.4	24	23.1	64	26.7	0.605 (NS)
	III	64	47.1	52	50.0	116	48.3	
	IV	32	23.5	28	26.9	60	25.0	
Orthopnea	Present	128	94.1	96	92.3	224	93.3	0.782 (NS)
PND	Present	84	61.8	56	53.8	140	58.3	0.541 (NS)
S3		96	70.6	68	65.4	164	68.3	0.670
JVP		100	73.5	80	76.9	180	75.0	0.765
Crepts		132	97.1	92	88.5	224	93.3	0.190
Pedal Edema		60	44.1	56	53.8	116	48.3	0.459
SBP(mmHg)		112.6 ± 18.9 (80, 150)		113.5 ± 20.4 (84, 156)		113.0 ± 19.4 (80, 156)		0.874
DBP(mmHg)		70.2 ± 9.7 (60, 90)		71.5 ± 13.1 (50, 94)		70.8 ± 11.2 (50, 94)		0.659
Pulse (beats/min)		90.3 ± 10.2 (80, 130)		90.8 ± 11.8 (76, 136)		90.5 ± 10.9 (76, 136)		0.847

Table 4: Patient Characteristics

Characteristic		Male		Female		Total		p value
		n	%	n	%	n	%	
CXR:CTR	≤ 0.5	16	11.8	12	11.5	28	11.7	0.979 (NS)
	> .5	120	88.2	92	88.5	212	88.3	
CXR:Pleural Effusion	Nil	92	67.6	76	73.1	168	70	0.554 (NS)
	Right side	20	14.7	20	19.2	40	16.7	
	Left Side	8	5.9	0	0	8	3.3	
	Bilateral	16	11.8	8	7.7	24	10	
CXR:Pulmonary Edema	Absent	92	67.6	72	69.2	164	68.3	0.897 (NS)
	Present	44	32.4	32	30.8	76	31.7	
Rhythm	Atrial Fibrilation	24	17.6	20	19.2	44	18.3	0.940 (NS)
	Atrial Flutter	4	2.9	0	0	4	1.7	
	Sinus	108	79.4	84	80.8	192	80	
IVCD	Nil	80	58.8	72	69.2	152	63.3	0.330 (NS)
	LBBB	40	29.4	28	26.9	68	28.3	
	LAHB	12	8.8	4	3.8	16	6.7	
	RBBB	4	2.9	0	0	4	1.7	
Diastolic Dysfunction	0	80	58.8	56	53.8	136	56.7	0.907 (NS)
	I	8	5.9	12	11.5	20	8.3	
	II	16	11.8	16	15.4	32	13.3	
	III	32	23.5	20	19.2	52	21.7	
EF(%)		38.6 ± 8.2 (22, 55)		41.0 ± 8.7 (24, 60)		39.7 ± 8.4 (22, 60)		0.289 (NS)

Table 5: Blood investigation in relation to gender distribution

	Male	Female	Total	p value
Creatinine (mg/dl)	1.5 ± 0.7 (0.5, 3)	1.6 ± 0.7 (0.7, 3.1)	1.6 ± 0.7 (0.5, 3.1)	0.619
Hemoglobin (g/dl)	10.2 ± 2.0 (6.9, 14)	10.1 ± 1.5 (8, 13)	10.2 ± 1.8 (6.9, 14)	0.705
CXR:CTR	0.6 ± 0.1 (0.5, 0.75)	0.6 ± 0.1 (0.5, 0.7)	0.6 ± 0.1 (0.5, 0.75)	0.416
Sodium(mmol/l)	133.0 ± 10.1 (116, 155)	132.8 ± 7.3 (116, 145)	132.9 ± 8.9 (116, 155)	0.922
Uric Acid (mg/dl)	5.9 ± 2.8 (2.8, 13.5)	6.2 ± 2.9 (2.7, 13.4)	6.0 ± 2.8 (2.7, 13.5)	0.759

Table 6: Patient Characteristics and blood investigations in relation with Outcome

	Survived	Died	Total	p value
Age (yr)	56.0 ±12.2 (28,76)	64.6 ±10.1 (45,75)	57.2 ±12.2 (28,76)	0.064 (NS)
SBP(mmHg)	116.6 ±18.2 (84,156)	89.5 ±5.4 (80,96)	113.0 ±19.4 (80,156)	0.000 (Sig)
DBP(mmHg)	72.6 ±10.9 (50,94)	59.0 ±3.7 (50,62)	70.8 ±11.2 (50,94)	0.001 (Sig)
Pulse (beats/min)	88.7 ±8.7 (76,130)	102.3 ±16.2 (88,136)	90.5 ±10.9 (76,136)	0.001 (Sig)
Hemoglobin (g/dl)	10.4 ±1.8 (7.8,14)	8.9 ±1.4 (6.9,11.6)	10.2 ±1.8 (6.9,14)	0.027 (Sig)
Sodium(mmol/l)	133.4 ±8.7 (116,155)	129.9 ±10.5 (118,147)	132.9 ±8.9 (116,155)	0.306 (NS)
Creatinine	1.4 ±0.6 (0.5,3)	2.5 ±0.5 (1.7,3.1)	1.6 ±0.7 (0.5,3.1)	0.000 (Sig)
Uric Acid (mg/dl)	5.3 ±2.1 (2.7, 13.4)	10.8 ±2.2 (7.8,13.5)	6.0 ±2.8 (2.7,13.5)	0.000 (Sig)

Table 7: Clinical profile in relation with Outcome

		Survived		Died		p value
		n	%	n	%	
Age (yr)	< 40	24	100	0	0	0.097 (NS)
	40 to 49	28	87.5	4	12.5	
	50 to 59	60	93.8	4	6.3	
	60 to 69	64	84.2	12	15.8	
	≥ 70	32	72.7	12	27.3	
Gender	Male	116	85.3	20	14.7	0.723 (NS)
	Female	92	88.5	12	11.5	
Hypertension	Absent	84	91.3	8	8.7	0.409 (NS)
	Present	124	83.8	24	16.2	
Diabetes Mellitus	Absent	180	88.2	24	11.8	0.399 (NS)
	Present	28	77.8	8	22.2	
NYHA(Admission)	II	64	100	0	0	0.007 (Sig)
	III	104	89.7	12	10.3	
	IV	40	66.7	20	33.3	
Diagnosis	IHD	44	73.3	16	26.7	0.024 (Sig)
	AF	4	50	4	50	
	DCM	32	80	8	20	
	RHD	52	100	0	0	
	HTCVD	68	94.4	4	5.6	
	PCM	8	100	0	0	
S3	Absent	72	94.7	4	5.3	0.214 (NS)
	Present	136	82.9	28	17.1	
JVP	Absent	60	100	0	0	0.082 (NS)
	Present	148	82.2	32	17.8	
Crepts	Absent	16	100	0	0	0.421 (NS)
	Present	192	85.7	32	14.3	
Pulse (beats/min)	Normal	188	92.2	16	7.8	0.003 (Sig)
	Tachycardia	20	55.6	16	44.4	

Table 8: Chest X-Ray, ECG and ECHO in relation with Outcome

		Survived		Died		p value
		n	%	n	%	
CXR:CTR	≤ 0.5	24	85.7	4	14.3	0.938 (NS)
	> .5	184	86.8	28	13.2	
CXR:Pleural Effusion	Absent	144	87.8	20	12.2	0.706 (NS)
	Present	64	84.2	12	15.8	
CXR:Pulmonary Edema	Absent	160	97.6	4	2.4	0.000 (Sig)
	Present	48	63.2	28	36.8	
Rhythm	Atrial Fibrilation	32	72.7	12	27.3	0.033 (Sig)
	Atrial Flutter	0	0	4	100	
	Sinus	176	91.7	16	8.3	
IVCD	Nil	140	92.1	12	7.9	0.125 (NS)
	LBBB	52	76.5	16	23.5	
	LAHB	12	75	4	25	
	RBBB	4	100	0	0	
Diastolic Dysfunction	0	112	82.4	24	17.6	0.245 (NS)
	I	16	80	4	20	
	II	32	100	0	0	
	III	48	92.3	4	7.7	
EF(%)		40.5 ±8.2 (22,60)		34.0 ±8.3 (24,50)		0.040 (Sig)

DISCUSSION

This study has assessed the etiology and demographic profile of HF patients admitted to a tertiary medical centre in northern india. It has also assessed the factors associated with poor prognosis and has compared the factors and aetiologies between males and females.

In our study 56.7% were male and 43.3% female, relatively young with overall mean age of 57.2±12.2 yrs, 58.9±10.8 yrs for males and 55.0±13.8 yrs for females. [Table 1] These finding were in contrast to what was reported from the Europe (71.3 ±12.7 yrs).[10,11] It is known that in non-western countries , cardiovascular diseases including HF tend to occur a decade or two earlier than do in western countries. This is attributable to both earlier occurrence of cardiovascular events.[12] Majority of cases in our study were in age group of 60-69 yrs. However only 18.3 % of patients were ≥ 70 yrs , unlike studies from West,[8,13,14] possibly due to low life expectancy in our part of world15. Mean age of patients who died was 64.6±10.1 yrs and for those survived 56±12.2 yrs statistically insignificant. [Table 7] This male predominance is similar to that seen in the Framingham heart study8 possibly due to higher rate of hypertension and coronary artery disease.[8,13,14] Females predominated the males (F:M ratio – 1.3:1) under the age of 50 yrs.

In our study out of 240 HF patients 32 died (13.3%) comprising of 20 male (14.7 %) and 12 female patients (11.5 %), without any statistically significant gender difference (Table 6 and 7). With increase in age mortality increased in our series being 25.0 % in ≥70 yrs and 7.14% in ≤ 60 yrs.

Among the 240 patients, 144 (60 %) were hypertensive with 72 males and 72 females (Table 2). This observation was consistent with Framingham heart study8. Further 36 (15 %) patients were diabetic with 20 males and 16 females. When these comorbities were analyzed in relation to outcome, among hypertensive patients 24 died (16.2 %) and in normotensives only 8 died (8.7 %) but it was not statistically significant. Among diabetics there were 8 deaths (22.2 %) whereas in non-diabetics there were 24 deaths (11.8 %) but it was not significant.

Of the 240 cases in our study, majority (48.3 %) were in the NYHA class IIIat the time of admission to hospital, the rest were equally distributed am-ong functional class II (26.7 %) and class IV (25.0 %) (Table 3 and 7). We analyzed the NYHA class at admission in relation to outcome. Among patients with NYHA class IV 20 patients died (33.3 %) and in those with NYHA class III 12 patients died where as there was no death in those with NYHA class II. This was statistically significant. Further it was seen that there is a significant relation between NYHA class at admission and at discharge. These observations were similar to those made by M R Cowie et al.[16,17]

On analysis of various clinical and laboratory parameters in relation to severity and outcome of HF patients in our study, we noticed that the prevalence of clinical parameters like JVP and S3 gallop ;and laboratory parameters like hemoglobin , creatinine, uric acid and serum sodium concentration bore a strong correlation with the severity and outcome of HF (Table 3,5,6). While only 5.3 % of patients who survived had S3 but 17.1 % of those who died had it. Similar observation was made regarding the presence of raised JVP (17.8 % of died;0 % of survived) and crepts (14.3 % of died; 0 % of survived). Similar prognostic significance of raised JVP, crepts and S3 gallop were observed by Drazner et al in their study.[18]

We also analyzed the pulse and blood pressure at admission in relation to outcome. We noticed that mean systolic and diastolic blood pressure of survivor was 116.6 ± 18.2 and 89.5 ± 5.4 mmHg respectively whereas for those who died it was 72.6 ± 10.9 and 59.0 ± 3.7 respectively (Table 3). Similarly mean pulse rate of survived and died group was 88.7±8.7 and 102.3±16.2 beats/min. These findings were consistent with observations of William T Abraham et al.[17]

We also analyzed haemoglobin, creatinine, uric acid and serum sodium in relation to severity and outcome. In our study mean haemoglobin in those who died was 8.9±1.4 g/dl whereas 10.4±1.8 g/dl in those who survived and this observation was statistically significant (Table 5,6). Similar observation were seen regarding creatinine (mean 2.5±0.5 mg/dl died and 1.4 ± 0.6 mg/dl survived) and uric acid (mean 10.8±2.2 mg/dl in died and 5.3±2.1 mg/dl in survived). Both these observations were statistically significant. These results bore resemblence to studies made by Horwich et al.[19]

Further we noted that serum sodium levels were low in those who died (129.9±10.5 mmol/l) than in survivors (133.4±8.7 mmol/l) but this was not statistically significant. This observation was in contrast to observations of Dries DL et al20 which revealed that hyponatremia was a strong predictor of inhospital mortality.

Ejection fraction (EF) was analyzed in relation to outcome and severity. Mean Ejection Fraction for those who died was 34±8.3 % and survivors it was 40.5±8.2 % which was statistically significant (Table 4,8). We also analyzed EF in relation to functional class at discharge; mean EF for NYHA class I, II and III at discharge was 43.8±7.2 , 36.9±7.3% and 34±11.1 % which was statistically significant. These observations were consistent with observations of William T Abraham.[17] Diastolic dysfunction was also analyzed in relation to outcome and functional capacity but there was no statistical significance in terms of mortality as well as functional capacity. This observation was in contrast to the observations of Senni M et al that mortality and rate of hospitalization among patients with diastolic HF is as high as patients with systolic HF.[21]

Electrocardiographic findings of rhythm and conduction abnormalities were also analyzed in relation to outcome and functional capacity at

discharge. Majority of patients were in sinus rhythm , about 18.3 % were having atrial fibrillation and 1.7 % have atrial flutter (Table 4,8). In our study 36.7 % cases had IVCD (QRS duration > 120 mS). Of these 28.3 % had LBBB, 6.7 % had LAHB and 1.7 % had RBBB. In our study rhythm disturbance had a significant relation with outcome (death or survival) as well as functional capacity at discharge where-as relation was statistically insignificant for conduction disturbances.

CONCLUSION

Identifying Poor prognostic factors in heart failure patients can help identify poor prognosis patients and help prioritise level of care in our overburdened health care system. These factors can be used to formulate algorithms to manage patients with poor prognosis in a more intense and high dependency settings.

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