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CLINICAL PROFILE AND PRESCRIPTION PATTERN OF DILATED CARDIOMYOPATHY IN TERTIARY CARE HOSPITAL

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ABSTRACT

Dilated Cardiomyopathy is defined as disorder of the heart muscle where the ventricles are dilated and is associated with varying degree of systolic pump failure. Dilated Cardiomyopathy is the most common variety and accounts for 25% of all patients presenting with heart failure. In the present world the number of persons suffering from cardiovascular complications is increasing. This may be due to change in lifestyle and stress related factors. The aim of this study was to observe clinical profile of patients with Dilated Cardiomyopathy and to assess the prescribing pattern in Dilated Cardiomyopathy attending a tertiary care hospital. This was a non experimental, prospective, study conducted in the inpatient department of medicine Era's Lucknow Medical College and Hospital for a period of 10 months. 100 patients who were diagnosed with Dilated Cardiomyopathy who satisfied the inclusion and exclusion were included. Specially designed standardized data collection form was used. From this study we identified that the majority of the patients were male who more than 60 years of age. Dyspnea was the most common symptom associated with Dilated Cardiomyopathy. Majority of the patients have cardiothoracic ratio in the range of 50-60% and Left Ventricular Ejection Fraction in the range of 20-29%. Sinus tachycardia is the most common electrocardiographic profile. Ischaemic Cardiomyopathy was the most common variety which was observed. Among the various classes of drugs diuretics was the most commonly prescribed drug. Among this Furosemide were the most frequently used drug.

INTRODUCTION

Dilated Cardiomyopathy is a heart muscle disorder defined by the presence of a Dilated and poorly functioning left ventricle. In North America and Europe, symptomatic dilated Cardiomyopathy has an incidence of 20 per 100,000 and a prevalence of 38 per 100,000. Men is affected more than twice as often as women. Dilated Cardiomyopathy is a disease of the heart muscle in which the heart chambers become enlarged or dilated. The heart muscle is weakened, making it more difficult for blood to flow from the body and lungs into the heart and for blood to be pumped from the heart to the rest of the body. The left ventricle is affected most commonly, although the right ventricle can also be affected. The dilatation often becomes severe and the heart may become quite enlarged. As the function of the left (and/or right) ventricle worsens, signs and symptoms of heart failure may develop. Dilated Cardiomyopathy is one of the leading conditions that cause a decrease in cardiac output and it is one of the most important causes of heart failure. Dilated Cardiomyopathy is characterized by cardiac enlargement and systolic dysfunction of one or both the ventricles. The main features are increasing systolic and diastolic ventricle volume with decreasing Left Ventricular Ejection Fraction below 40%. In many patients ventricular dilatation and reduced ejection fraction could be detected many years before the development of Cardiac failure. Dilated Cardiomyopathy represents the final common pathway produced by variety of toxic, metabolic, immunological, familial, and infectious mechanism damaging the heart muscle. Multiple alterations in organ and cellular physiology contribute to heart failure. The various mechanism leading to ventricular dilation in Dilated Cardiomyopathy include myocyte dysfunction and loss (apoptosis) interstitial changes, remodeling, abnormalities of contractile proteins, mitochondrial dysfunction, abnormal sarcoplasmic reticulum function, membrane channel defect, abnormal myocardial receptor function, neurohormonal changes. In many cases the cardiac dysfunction begins with a primary insult to the cardiomyocyte like toxin (eg.alcohol) ischemia, (acute myocardial infarction) myocarditis etc. Dilated Cardiomyopathy is associated with complex remodeling of one or both ventricles, resulting in a change of ventricle shape and the architecture of the myocardium fibers. The ventricular shape changes from an elliptical to a more spherical form. In addition patient with DCM may have a stiffer myocardial wall, caused by increased myocardial mass and alteration in the extracellular collagen network. The structural ventricular remodeling process associated with Dilated Cardiomyopathy including chamber enlargement and alteration in shape. The various causes of dilated Cardiomyopathy are collagen vascular disease, drugs like alcohol,

sympathomimetic, anthracyclines, and doxorubicin. Growth hormone deficiency, hypothyroidism, hypocalcaemia, diabetes mellitus and various nutritional deficiencies just like selenium, carnitine, and thiamine.

MATERIAL AND METHODS

An observational, prospective was carried out in 100 patients diagnosed to have Dilated Cardiomyopathy visiting Era's Lucknow Medical College and Hospital during the study period of 10 months. The diagnosis of Dilated Cardiomyopathy was made from the Chest x ray, Echocardiography and through Electrocardiogram. A standardized data collection form was prepared and necessary data was obtained from patients and their care givers. The data collection form provided the information regarding the demography which includes age, sex, and family history. Patient written consent were obtained from each patient prior to the interview.

RESULT

Table no.1 showing effect of age and sex on distribution of DCMP

| Age Group | Male(n) | Female(n) | Total(n) |
|-----------|---------|-----------|----------|
| 20-39 | 12 | 10 | 22 |
| 40-59 | 18 | 17 | 35 |
| >60 | 33 | 10 | 43 |

Figure No.1

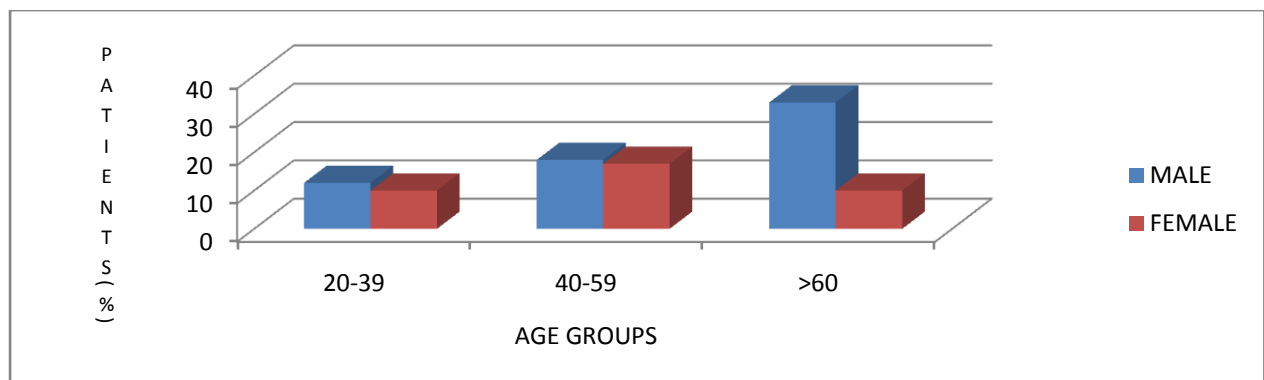


Figure No1. shows the demographic profile of patients with Dilated Cardiomyopathy. Majority of the cases are above the age of 60 years. Among males the majority of cases are above the age of 60 years whereas in females there is a clustering of cases among the young and middle aged population.

Table No.2 showing the symptom profile of the patients

| SYMPTOMS | (n) | (%) |
|--------------------|-----|-----|
| Dyspnea | 96 | 96% |
| Easy Fatiguability | 84 | 84% |
| PND | 63 | 63% |
| Cough | 59 | 59% |
| Orthopnea | 54 | 54% |
| Abdominal Pain | 33 | 33% |
| Chest Pain | 49 | 49% |
| Misc | 24 | 24% |
| Pedal Edema | 48 | 48% |

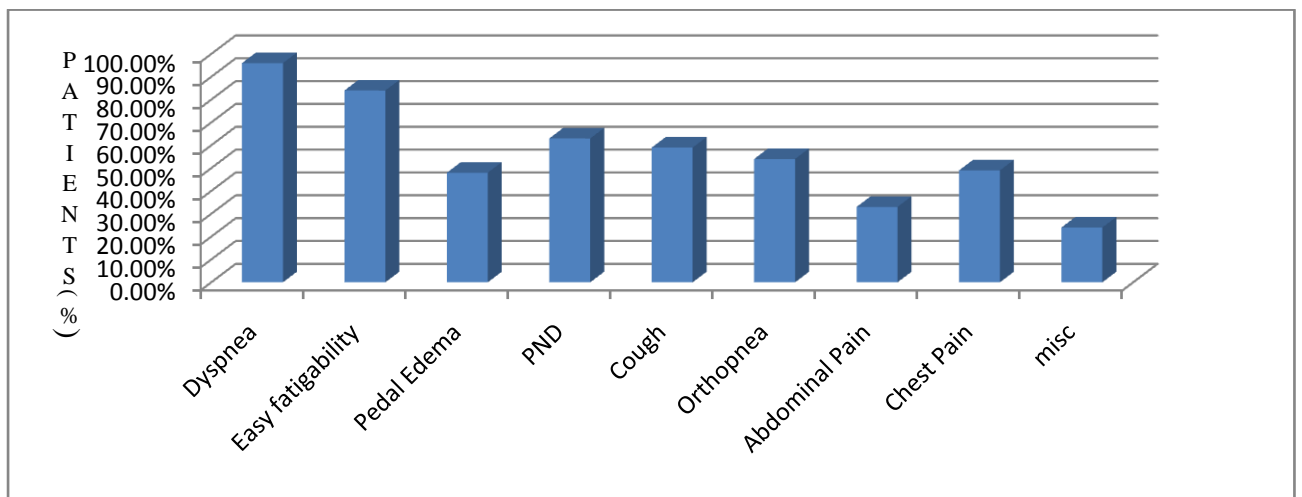
Figure No.2

Figure no 2. Shows the symptom profile of patients with Dilated Cardiomyopathy. Most of the patients are presented with Dyspnea. Easy fatigability is seen in (84%) of the patients constituting the second most common symptoms followed by Pedal edema. History of cough are seen in (59%), PND (63%), Orthopnea (54%), Abdominal pain (33%), Chest pain (49%) and misc symptoms are present in (24%)

Table no.3 showing Etiological Distribution

| Type of DCMP | (n) | (%) |
|--------------|-----|-----|
| Ischaemic | 43 | 43% |
| Idiopathic | 13 | 13% |
| Diabetic | 25 | 25% |
| Alcoholic | 12 | 12% |
| Misc | 7 | 7% |

Figure No.3

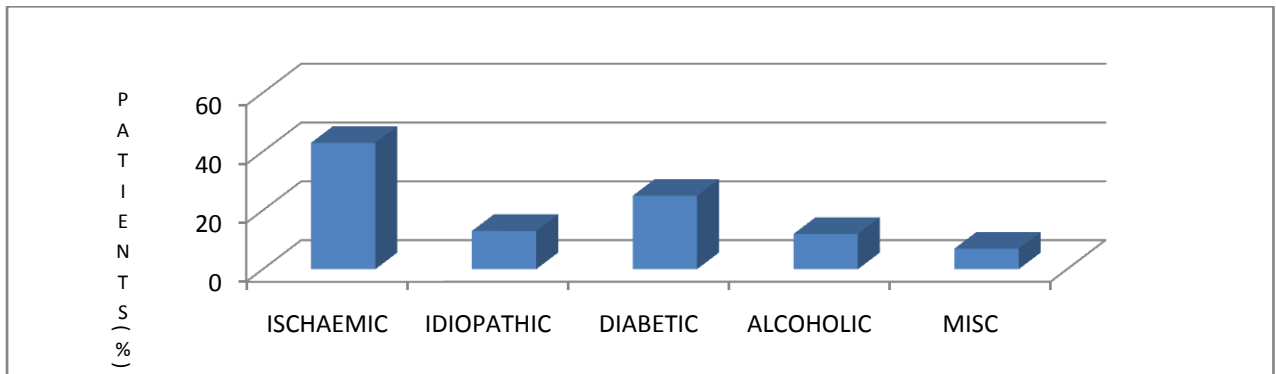


Figure No 3. Shows the etiological distribution of Dilated Cardiomyopathy. Most of the patients are presented with Ischaemic variety constituting (43%) followed by diabetic (25%), idiopathic (13%) alcoholic (12%) and misc (7%)

Table no. 4 shows CTR range Distribution

| CTR Range | (n) | (%) |
|-----------|-----|-----|
| 50-60% | 47 | 47% |
| 60-70% | 40 | 40% |
| >70% | 13 | 13% |

FigureNo.4

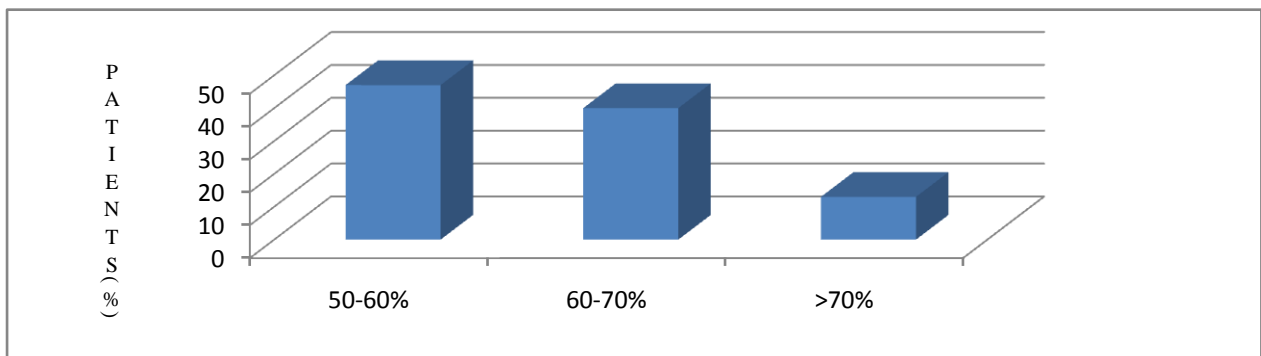


Figure No.4 shows the chest radiographic profile of patients.(47%) of patients had cardiothoracic ratio in the range of 50-60%, followed by 40% of patients in the range of 60-70% and(13%)of patients are having CTR greater than 70%

Table No.5 showing Echocardiographic Profile

| LVEF range (%) | (n) | (%) |
|----------------|-----|-----|
| 40-45% | 17 | 17% |
| 30-39% | 36 | 36% |
| 20-29% | 40 | 40% |
| <20 | 7 | 7% |

Figure No.5

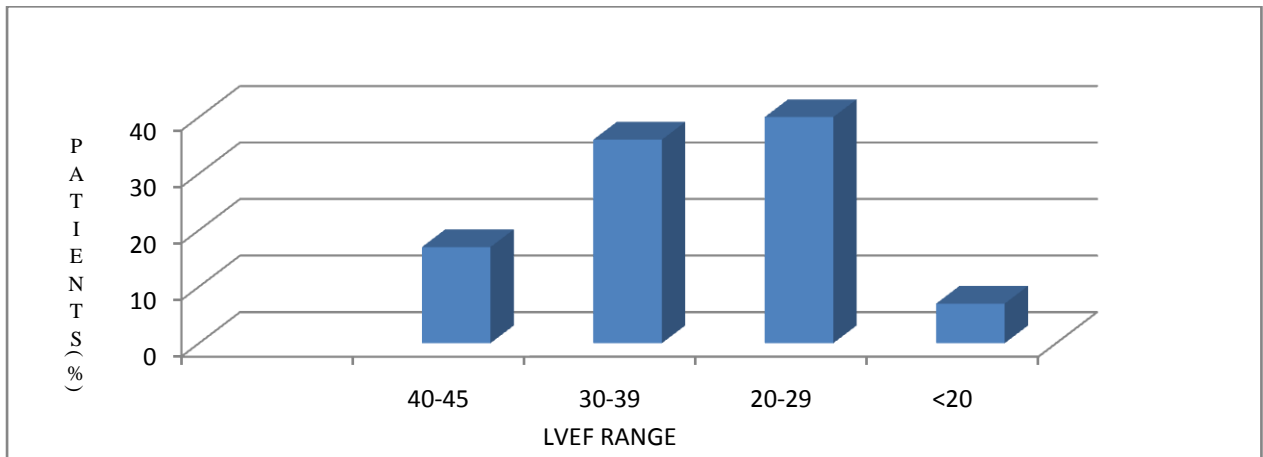


Figure No 5: shows the echocardiographic profile of patients with Dilated Cardiomyopathy.(40% of patients have LVEF in the range of 20-29%,(36%)of patients in the range of 30%-39%,(17%) in the range 40-45%,and(7%)of patients in the range of less than 20%.

Most of the patients have LVSD more than 5cm and LVEDD greater than 6cm.Mitral Regurgitation is the most common physical signs.

Table no.6 showing electrocardiographic profile

| ECG | (n) | (%) |
|-------------------|-----|-----|
| Sinus Tachycardia | 45 | 45% |
| ST- T Changes | 14 | 14% |
| Both | 21 | 21% |
| PSVT | 15 | 15% |
| Heart Block | 5 | 5% |

Figure No.6

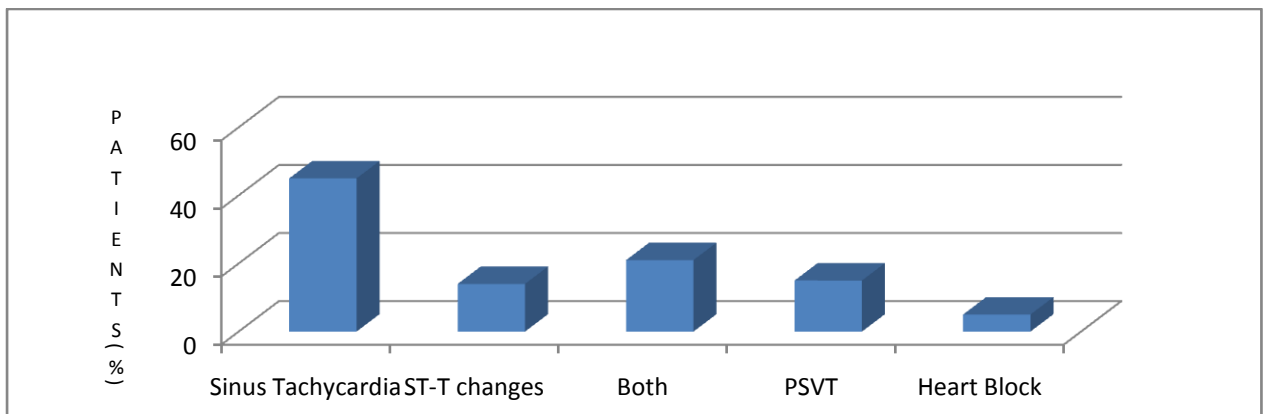
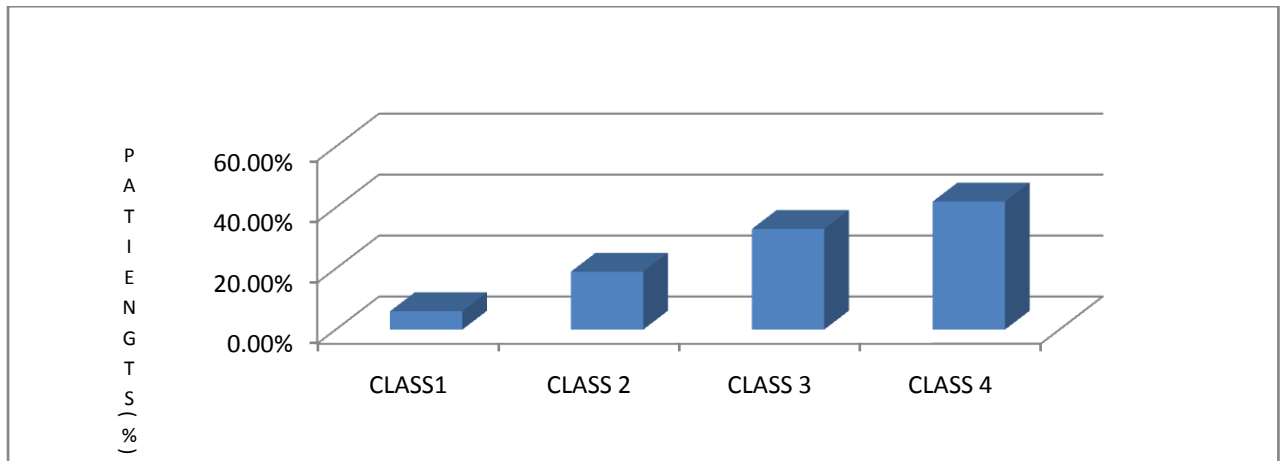


Figure no. 6 shows that sinus tachycardia is the most common ECG change(45%), ST-T changes(14%), PSVT(15%)And heart block in 5%

Table no.7 showing NYHA classification of Heart failure

| NYHA class | (n) | (%) |
|------------|-----|-----|
| Class 4 | 42 | 42% |
| Class 3 | 33 | 33% |
| Class 2 | 19 | 19% |
| Class 1 | 6 | 6% |

**Table no.7 showing treatment pattern in DCMF**

| Drug Name | (n) | (%) |
|----------------|-----|-----|
| Torasemide | 33 | 33% |
| Ramipril | 18 | 18% |
| Clopidogrel | 9 | 9% |
| Nifedipine | 4 | 4% |
| Atorvastatin | 36 | 36% |
| Amlodipine | 4 | 4% |
| Metoprolol | 41 | 41% |
| Calcium | 14 | 14% |
| Carvedilol | 6 | 6% |
| Furosemide | 62 | 62% |
| Enalapril | 29 | 29% |
| Digoxin | 33 | 33% |
| Valsartan | 4 | 4% |
| Heparin | 9 | 9% |
| Nicoumaline | 6 | 6% |
| Rosuvastatin | 7 | 7% |
| Aspirin | 38 | 38% |
| Diltiazem | 24 | 24% |
| Spironolactone | 78 | 78% |
| Dobutamine | 3 | 3% |

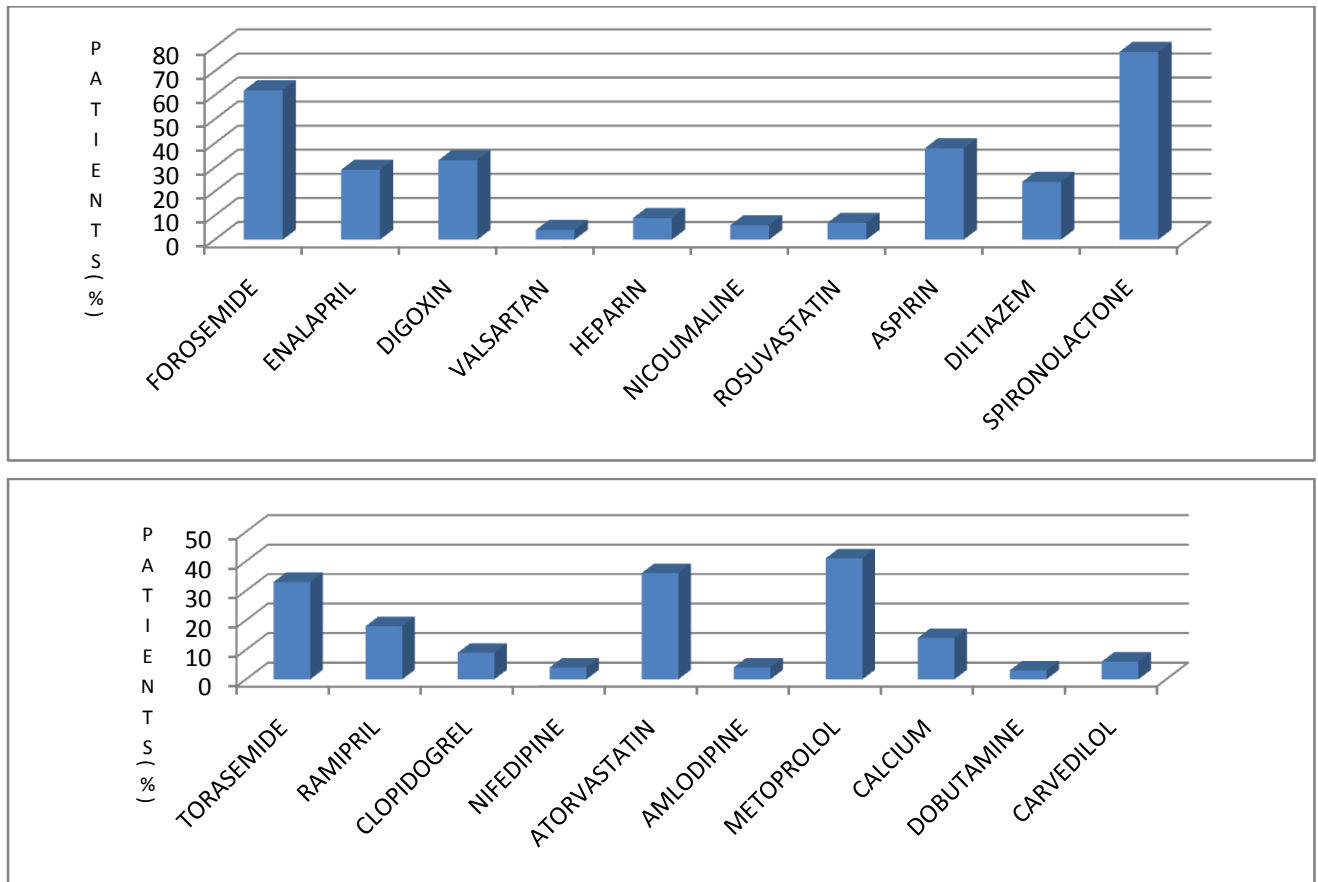
Figure No.7

Figure No.7 and 8 shows the treatment pattern and it was found out that Furosemide was given in

(62%),Enalapril(29%),Digoxin(33%),Valsartan(4%),Heparin(9%),Nicoumaline(6%),Aspirin(38%),Diltiazem(24%),Spironolactone(74%),Torasemide(33%),Ramipril(18%),Clopidogrel(9%),Nifedipine(4%),Atorvastatin(36%),Amlodipine(4%),Metoprolol(41%),Calcium(14%)Dobutamine(3%)and Cavedilol(6%).

DISCUSSION

Dilated Cardiomyopathy is the most common type of Cardiomyopathy and an important cause of CHF.Dilated Cardiomyopathy is common in elderly and middle aged population. Majority of the cases are above the age of 60 years. Among males the majority of cases are above the age of 60 years whereas in females there is a clustering of cases among the young and middle aged population.Dyspnoea is the most common symptoms associated with Dilated Cardiomyopathy.Ischaemic variety is the most common type of Cardiomyopathy. Most of the patients have cardiothoracic ratio in the range of 50-60% Biventricular failures is found to be more than LVF and RVF. The echocardiography revealed that the most common LVEF range is 20-29%. Sinus Tachycardia is the most common ECG change seen in

electrocardiography. Most of the patients belong to NYHA Class 4 and Class 3. Diuretics are the most commonly used drug class in the treatment of Dilated Cardiomyopathy. There is a symptomatic improvement in most of the patient after following the treatment regimen of Dilated Cardiomyopathy.

CONCLUSION

Understanding of the clinical profile, natural history, and triggering or exacerbating factors responsible for the increased morbidity of Dilated Cardiomyopathy can help the sufferers. In the present study most of the patients of Dilated Cardiomyopathy are managed with Diuretics. If we talk of etiological factors diabetes and alcohol intake, plays a significant role. There is direct association seen between diabetes and alcohol abuse with the risk of Dilated Cardiomyopathy. Treatment of the patients with Dilated Cardiomyopathy is still one of the major problems in cardiology. Available drugs are not able to affect the long term of prognosis. Although the drugs used commonly affect some of the clinical and physiopathological features, they can not affect the conditions supporting the pump function of the myocardium.

REFERENCES

1. Mestroni L, Maisch B, McKenna WJ, et al. Collaborative research group of the European human and capital mobility project on familial dilated cardiomyopathy. Guidelines for the study of familial dilated cardiomyopathies. *Eur Heart J* 1999;20:93–102.
2. Ashrafian H, McKenna WJ, Watkins H. Disease pathways and novel therapeutic targets in hypertrophic cardiomyopathy. *Circ. Res.*, 109, 86–96 (2011).
3. Abdullah M, Bigras JL, McCrindle BW, Mustafa A. Dilated cardiomyopathy as a first sign of nutritional vitamin D deficiency rickets in infancy. *Can J Cardiol* 1999; 15: 699-701.
4. Csanády M, Forster T, Julesz J. Reversible impairment of myocardial function in hypoparathyroidism causing hypocalcaemia. *Br Heart J* 1990;63:58-60.
5. Cox GF, Sleeper LA, Lowe AM, Towbin JA, Colan SD, Orav EJ, *et al.* Factors associated with establishing a causal diagnosis for children with cardiomyopathy. *Paediatrics* 2006; 118:1519-31.
6. Connor TB, Rosen BL, Blaustein MP, Applefeld MM, Doyle LA. Hypocalcemia precipitating congestive heart failure. *N Engl J Med* 1982; 307: 869-972.
7. Dursun A, Aliefendiođlu D, Özkan B, Cöpkun T. Carnitinuria in rickets due to vitamin D deficiency. *Turk J Pediatr* 2000; 42: 278-280.
8. Gilor A, Groneck P, Kaiser J, Schmitz-Stolbrink A. Congestive heart failure in rickets caused by vitamin D deficiency (Abstract). *Monatsschr Kinderheilkd* 1989; 137: 108-110.
9. Hocy Genetics and clinical destiny: improving care in hypertrophic cardiomyopathy. *Circulation*, 122, 2430–2440, discussion, 2440 (2010).

10. Lohrman JA, Janzer RC, Kuntzer T, Matthieu JM, Pfend G, Goy JJ, Bogousslavsky J. Familial cardiomyopathy and distal myopathy with abnormal desmin accumulation and migration. *Neuromuscul.Disord.*, 8, 77–86 (1998).
11. Levine SN, Hypocalcemic heart failure. *Am J Med* 1985;78:1033-5.
12. Gurtoo A, Goswami R, Singh B, Rehan S, Meena HS. Hypocalcemia-induced reversible hemodynamic dysfunction. *Cardiol* 1994;43:91-3.
13. Maiya S, Sullivan I, Allgrove J, Hypocalcaemia and vitamin D deficiency: An important, but preventable, cause of life-threatening infant heart failure. *Heart* 2008;94:581-4.
14. Özkan B, Büyükavcı M, Aksoy H, Tan H, Akdağ R. Erzurum’da çocuklarda nutrisyonel rikets sıklığı. *Çocuk Sağlığı ve Hastalıkları Dergisi* 1999; 42: 389-396.
15. R. Phlogose und thrombose in geffasssystem. In: *Gesammelte Abhandlungen zur wissenschaftlichen medicine*. Frankfurt: Meidinger Sohn; 1856.
16. Ripley TL, Nutescu E. Anticoagulation in patients with heart failure and normal sinus rhythm. *Am J Health Syst Pharm* 2009;66: Virchow 134-41.
17. Root AW, Parathyroid and vitamin D-related disorders in children and adolescents. In: Sperling MA (ed). *Pediatric Endocrinology* (1st ed). Philadelphia: WB Saunders; 1996: 427-509.
18. Uysal S, Kalaycı AG, Baysal K. Cardiac functions in children with vitamin D deficiency rickets. *Pediatr Cardiol* 1999; 20: 283-286.
19. Van Dantzig JM, Delemarre BJ, Bot H, Koster RW, Visser CA. Usefulness of mitral regurgitation in protecting against left ventricular thrombus after acute myocardial infarction.