A DISSERTATION ON

PREVALENCE PATTERN PRECIPITATING FACTORS AND PRESENTING FEATURES OF CONGESTIVE HEART FAILURE

M.D. Degree

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CONTENTS

INTRODUCTION

Heart failure is the end stage of all diseases of the heart and is a major cause of morbidity and mortality. Since 1970s the treatment of CHF has been transformed, resulting in major benefit to patients. This advance has been the consequence of better understanding of the pathophysiology, investigations, the introduction of newer drugs and cardiac transplantation. The traditional treatment of heart failure with digoxin and diuretics has been replaced by diuretics, ACE inhibitors and drugs directed against the origins of heart failure such as aspirin and lipid lowering drugs. Newer objectives are optimisation of the quality of life, avoidance of hospital admission prevention of progression of damage to the myocardium and prolongation of life.

So, it becomes important to conduct clinical and paraclinical studies to know about the status, precipitating factors and complications of the disease. Only with a reliable study changes in the modality of approach in controlling, diagnosing and treating the disease can be done. Here, an attempt has been made to study on selected aspects of congestive heart failure.

AIMS AND OBJECTIVES

1. To find out the prevalence of congestive heart failure among the patients admitted in the hospital.
2. To analyse their presenting features & precipitating factors
3. To correlate the clinical findings with the underlying etiology
4. To study outcome of these cases during their hospital stay.

REVIEW OF LITERATURE

History :

Heart failure, angina and the pulse were known in the ancient Egyptian and early Greek civilizations. (Dallas 1993, Horine 1941). Hippocrates described cardiac cachexia, most vividly reports of the benefits of foxglove exists in Roman literature. (Moore, 1985) Hering used nitrate in 1853 to treat heart failure, the first use of a vasodilator. Bruton later in 1867 described the use of amyl nitrate to treat angina. ACE inhibitors were shown to be of benefit in terms of mortality in patients with heart failure for the first time in 1987. The large trail of digoxin, showing no effect on overall mortality, was reported in 1996.

Definitions of heart failure

1. A Pathophysiological state in which an abnormality of cardiac function is responsible for the failure of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues (Braunwald 1994)
2. Congestive heart failure represents a complex clinical syndrome, characterized by abnormalities of left ventricular function and Neurohormonal regulation, which are accompanied by effort intolerance, fluid retention and reduced longevity. (Packer 1988).
3. Symptoms of heart failure, objective evidence of cardiac dysfunction and response to treatment directed towards the heart failure (Task force of the European society of cardiology 1995)

EPIDEMIOLOGY:

Heart failure is a relatively common disorder. It is estimated that 4.6 million persons in the United States are being treated for heart failure, with 550,000 new cases diagnosed each year. (Dallas – 1999, Massie – 1977)

The prevalence of heart failure increases dramatically with age, occurring in 1 to 2 percent of persons aged 50 to 59 and up to 10 percent of individuals older than the age of 75. (Ho et al 1993)

Approximately 80 percent of all heart failure admissions occur in patients older than 65, as a result, heart failure is the leading discharge diagnosis in persons 65 years or older in the United States (Rich 1999)

Despite a steady decline in the incidence of coronary artery disease and stroke, both the incidence and prevalence of heart failure continue to rise.

Between 1985 and 1995 the number of heart failure hospitalizations increased by 5 percent and 8,70,000 hospital discharges for heart failure occurred in 1996. (Haldeman – 1999)

In the United States approximately 45,000 deaths each year are primarily caused by heart failure and heart failure is listed as a contributing cause in 260,000 deaths (Dallas 1999)

In smaller mid western areas, recent assessment has suggested a prevalence as high as 6% of population. The same prevalence of 6-7 % also has been observed in Urban population. (Senni et al – 1999)

The overall prevalence of heart failure is 3-20 per 1000 population although this exceeds 100 per 1000 in those aged 65 yrs and over. The annual incidence of heart failure is 1-5 per 1000 and the relative incidence doubles for each decade of life after the age of 45 yrs. (Davis Hobbs, Lip – 2003). The overall incidence is likely to increase in the future because of both an aging population and therapeutic advances in the management of acute myocardial infarction leading to improved survival in patients with impaired cardiac function. (Cowie et al – 1999).

Three major factors such as age, race and gender influence the prevalence and outcome in patients with heart failure.

Age :

The most important factor is age. The prevalence of CHF is less than 1% in patients <50 yrs of age regardless of gender. At older than 50 years of age however, the prevalence increases to approximately 5% for patients between 50 and 70 years of age, nearly 10% for all patients over 70 yrs of age and perhaps as high as 15% for patients over the age of 80.

The impact of age alone leads to a significant continued increase in the prevalence of heart failure as recent statistics have suggested that there will be a near doubling of patients over 65 years of age by the year 2030. (Dallas 2001).

Race :

The second major influencing factor is race. There has been a higher prevalence of heart failure in blacks compared to whites.

More importantly black patients develop heart failure at a younger age than white patients. (Bourassa et al 1993).

Gender :

The third major factor influencing the heart failure is gender. The national heart and nutrition education survey (NHANES) from 1988-1994 estimated that there is an increased prevalence of congestive heart failure in men aged 70 years and younger. In contrast, women aged older than 70 years have a higher prevalence. This differences may be in part owing to the increased average life expectancy in women. (Anderson 2001). The highest prevalence of CHF is in black men followed by black women.

Data derived from the National centre of Health Statistics report that total life expectancy for the United States Population is 76.7 years. Life expectancy is 73.8 years for men and 79.5 years for Women (2000 estimate). While the overall incidence of congestive heart failure is probably equal between the two genders, there are several unique features that may influence the prevalence of congestive heart failure in women, including a higher average ejection fraction for an equivalent amount of symptoms than men at all ages. This may reflect a higher prevalence of hypertension in women with advancing age and almost certainly a higher prevalence of primary diastolic rather than systolic dysfunction. (Gheorghiade, Bonow 1998)

The Framingham data shows an age adjusted annual incidence of heart failure of 0.14% in women and 0.23 % in men. Survival in the women is generally better than in the men.

AETIOLOGY:

The relative importance of aetiological factors in heart failure is dependent on the nature of the population being studied, as coronary artery disease and hypertension are common causes of heart failure in western countries, whereas valvular heart diseases and nutritional cardiac diseases are more common in the developing world. (Zannad – 1999)

<u>The common causes of heart failure include :</u>

1. coronary artery disease-MI, ischaemia
2. hypertension
3. cardiomyopathy

4. valvular and congenital heart diseases
5. arrhythmias

    -tachycardias

    -bradycardia(CHB, sick sinus syndrome)

6. alcohol and drugs

    - alcohol
    - cardiac depressant drugs (beta blockers and calcium channel blockers)

7. high output failure

    - anaemia, thyrotoxicosis, av fistulas, paget's disease

8. pericardial diseases
9. primary right heart failure

    Pulmonary hypertension eg. pulmonary embolism, corpulmonale

    Tricuspid incompetence

Coronary artery disease and its risk factors:

Coronary artery disease is the commonest cause of heart failure. In the studies of left ventricular dysfunction (SOLVD) Coronary artery disease accounted for almost 75% of the cases of chronic heart failure.

(Khadra Saleem, Rand 1998) Coronary artery disease and hypertension (either alone or in combination) were implicated as the cause in over 70% of cases of heart failure in the Framingham study. (Ho – 1993)

Coronary risk factors, such as smoking and diabetes are also risk markers of the development of heart failure. Smoking is an independent and strong risk factor

for the development of heart failure in men, although the findings in women are less consistent.

In Framingham heart study, diabetes and left ventricular hypertrophy were the most significant risk markers for the development of heart failure. Body weight and high ratio of total cholesterol concentration to high density lipoprotein cholesterol concentration are also independent risk factors for heart failure. Clearly these risk factors may increase the risk of heart failure through their effects on coronary artery disease, although diabetes alone may induce important structural and functional changes in the myocardium, which further increase the risk of heart failure.

Hypertension :

Hypertension has been associated with an increased risk of heart failure in several epidemiological studies. In the Framingham heart study, hypertension was reported as the cause of heart failure either alone or in association with other factors, in over 70% of cases on the basis of non invasive assessment. (Ho-1993). However hypertension is probably a more common cause of heart failure in selected patient groups, including females and black populations. Hypertension predisposes to the development of heart failure via a number of pathological mechanisms, including left ventricular hypertrophy. Left ventricular hypertrophy is associated with left ventricular systolic and diastolic dysfunction and an increased risk of myocardial infarction and it predisposes to both atrial and ventricular arrhythmias. Electrocardiograpic left ventricular hypertrophy is strongly correlated with the development of heart failure, as it is associated with a 14 fold increase in the risk of heart failure in those aged 65 years or under.

Valvular heart disease :

Rheumatic heart disease may have declined in certain parts of the world, but it still remains an important cause of heart failure in India and other developing nations. In the Framingham heart study, rheumatic heart disease accounted for heart failure in 2% of men and 3% women, although the overall incidence of valvular heart disease has been steadily decreasing in the Framingham cohort over the past 30 years (HO – 1993).

MR and AS are the most common causes of heart failure, secondary to valvular disease. MR and AR lead to volume overload, in contrast with AS which leads to pressure overload. The progression of heart failure in patients with valvular heart disease is dependent on the nature and extent of the valvular disease. In aortic stenosis heart failure develops at a relatively late stage and without, valve replacement, it is associated with a poor prognosis. In contrast, patients with chronic mitral or aortic regurgitation generally decline in a slower and more progressive manner. (Teerlink, et al 1991).

Cardiomyopathies :

Cardiomyopathies are defined as the disease of the heart muscle that are not secondary to coronary heart disease, hypertension or others. As primary disease of heart muscle, cardiomyopathies are less common causes of heart failure, but awareness of their existence is necessary to make a diagnosis. Cardiomyopathies are separated into four functional categories dilated, hypertrophic, restrictive and obliterative. These groups can include rare specific heart muscle diseases such as

hemochromatosis in which cardiac involvement occurs as part of a systemic disorder. Dilated cardiomyopathy is a more common cause of heart failure than hypertrophic and restrictive cardiomyopathies, obliterative cardiomyopathy is essentially limited to developing countries. (Oakley 1997).

Arrhythmias :

Cardiac arrhythmias are more common in patients with heart failure and associated structural heart diseases, including hypertensive patients with left ventricular hypertrophy.

In the Hillingdon heart failure study 30% of patients presented for the first time with heart failure had atrial fibrillation and over 60% of patients admitted urgently with atrial fibrillation to a Glasgow hospital had echocardiographic evidence of impaired left ventricular function. (Stevenson, 1995).

Alcohol and drugs:

Alcohol has a direct toxic effect on the heart which may lead to acute heart failure or heart failure as a result of arrhythmias commonly atrial fibrillation. Excessive chronic alcohol consumption also leads to dilated cardiomyopathy.

Alcohol is the identifiable cause of heart failure in 2-3% of cases. Chemotherapeutic agents(doxorubicin) and antiviral drugs(zidovudine) have been implicated in heart failure, through direct toxic effects on the myocardium. (Maki et al – 1998)

Endocrine causes :

High output heart failure is most often seen in patients with anemia and thyrotoxicosis. Myxedema may present with heart failure as a result of myocardial involvement or secondary to pericardial effusion.

Corpulmonale :

Corpulmonale is defined as enlargement of the right ventricle secondary to abnormalities of the lungs, thorax, pulmonary ventilation or circulation. It sometimes leads to right ventricular failure, with an elevation of transmural right ventricular end diastolic pressure. (Rich et al 2005).

Pericardial Diseases :

Pericardial diseases like tuberculosis, CRF and malignant involvement may also cause congestive heart failure.

Nutritional Causes :

Vitamin deficiency like wet beriberi, iron deficiency anaemia and deficiency of hemopoeitic factors leading to heart failure, continue to produce a problem especially in developing countries like India.

PATHOPHYSIOLOGY

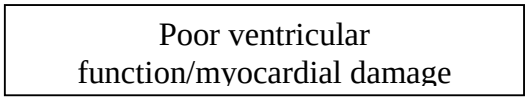
Heart failure is a multisystem disorder which is characterized by abnormalities of cardiac, skeletal muscle and renal function ; stimulation of the sympathetic nervous system; and a complex pattern of neurohormonal changes.

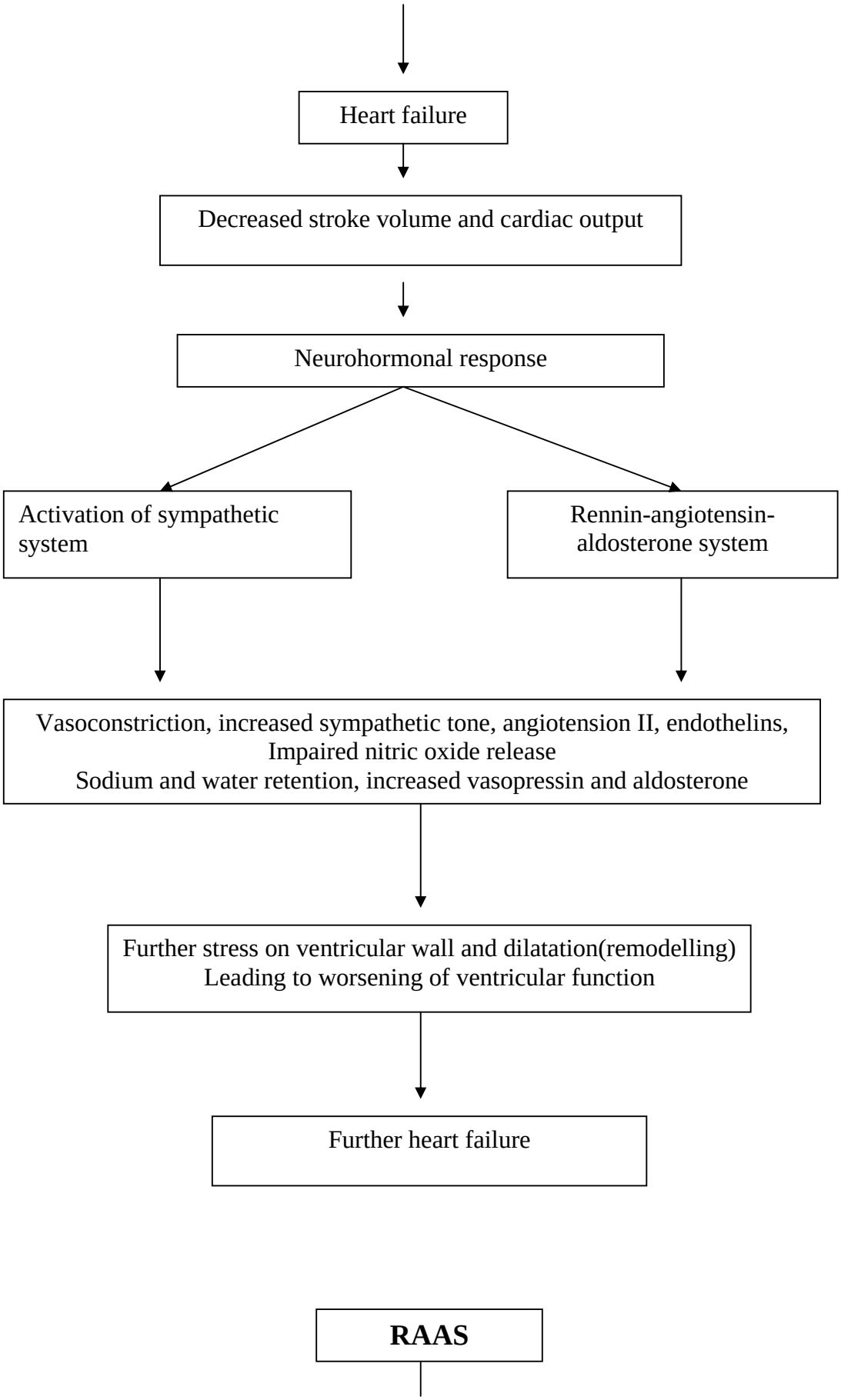
Myocardial systolic dysfunction :

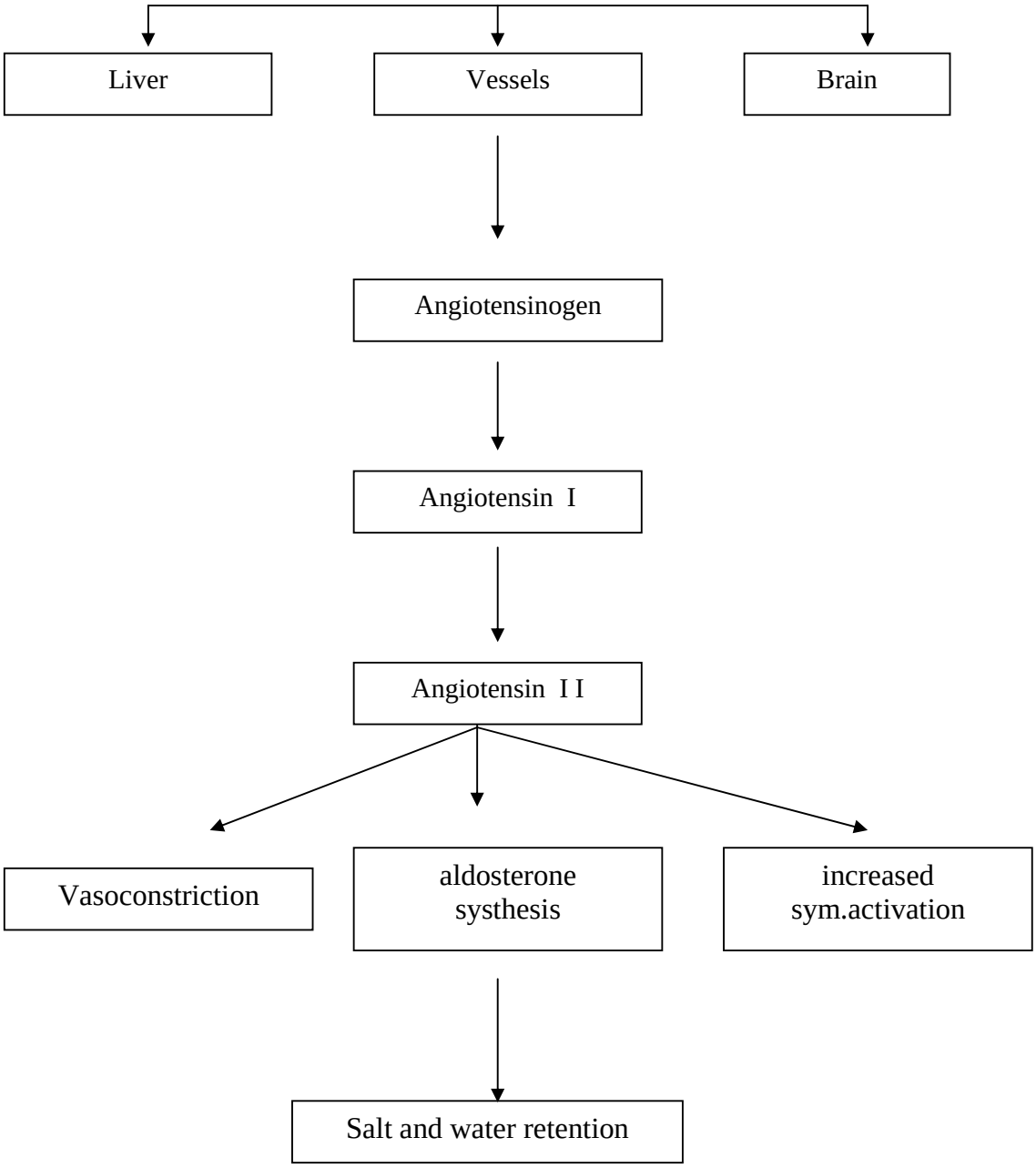
The primary abnormality in non valvular heart failure is an impairment in the left ventricular function, leading to a fall in cardiac output. The fall in cardiac output leads to activation of several neurohormonal compensatory mechanisms aimed at improving the mechanical environment of the heart.

Activation of the sympathetic system, for example tries to maintain cardiac output with an increase in heart rate, increased myocardial contractility and peripheral vasoconstriction (Increased catecholamines). Activation of Renin angiotensin aldosterone system also results in vasoconstriction and increase in blood volume, with retention of salt and water. Concentration of vasopressin and natriuretic peptides increase. Furthermore there may be progressive cardiac dilatation or alterations in cardiac structure (remodelling) or both. (Borgeon, Burnett 1997)

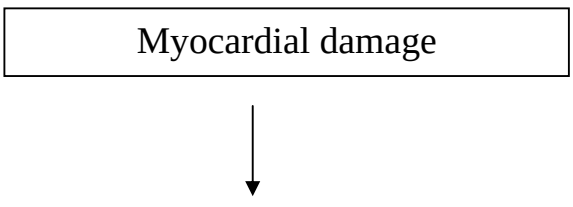
Neurohormonal activation:

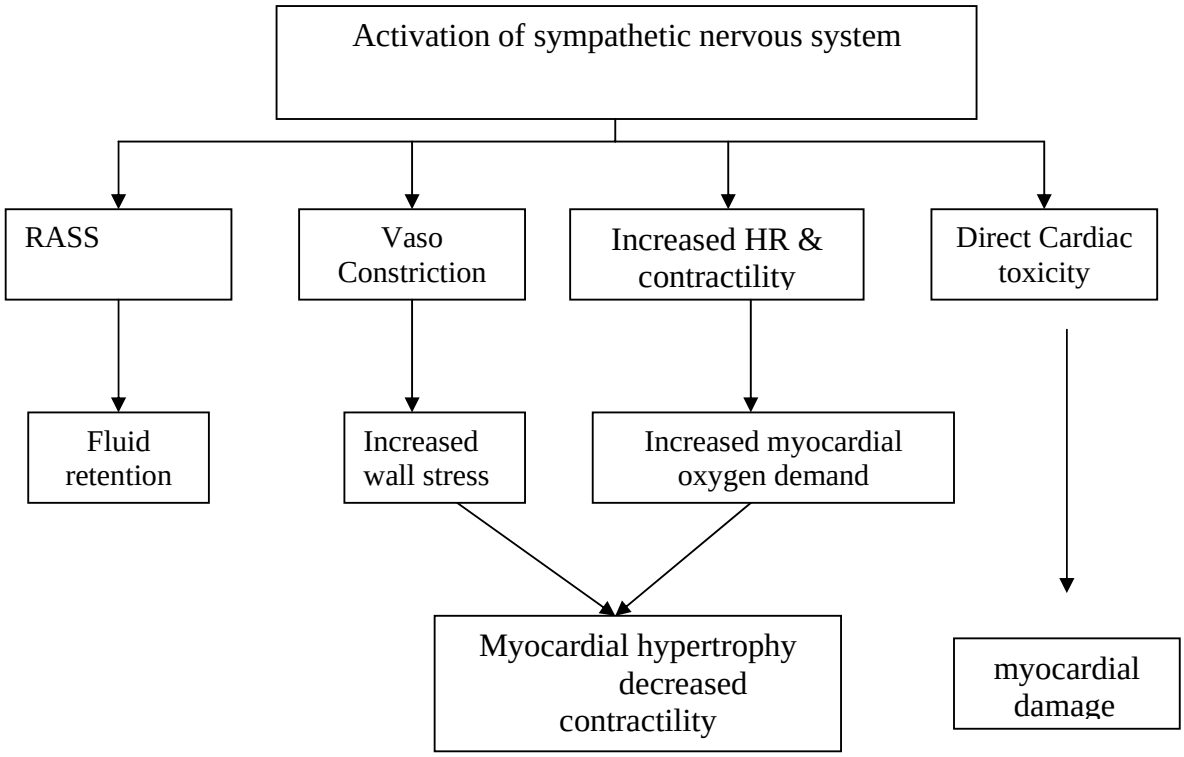



Heart failure
Decreased stroke volume and cardiac output
Neurohormonal response
Activation of sympathetic system
Rennin-angiotensin-aldosterone system
Vasoconstriction, increased sympathetic tone, angiotension II, endothelins,
Impaired nitric oxide release
Sodium and water retention, increased vasopressin and aldosterone
Further stress on ventricular wall and dilatation(remodelling)
Leading to worsening of ventricular function
Further heart failure
RAAS



Effects of Sympathetic nervous system on heart failure :





RAAS :

Stimulation of the RAAS leads to increased concentration of renin, angiotension II and aldosterone. Angiotension II is a potent vasoconstrictor of the renal and systemic circulation where it stimulates release of noradrenaline from sympathetic nerve terminals which inhibits vagal tone and promotes the release of aldosterone. This leads to the retention of sodium and water. In addition, angiotension II has an important effects on cardiac myocytes and may contribute to the endothelial dysfunction that is observed in chronic heart failure. (Francis et al, 1990)

Sympathetic nervous system :

The sympathetic nervous system is activated in heart failure via low and high pressure baroreceptors, as an early compensatory mechanism which provides

inotropic support and maintains cardiac output. In the long term, the ability of the myocardium to respond to chronic high concentration of catecholamines is attenuated by a down regulation in beta receptors. This may be associated with baroreceptor dysfunction and a further increase in sympathetic activity. (Schoffer et al, 1987).

Natriuretic peptide:

There are 3 natriuretic peptides of similar structure and these exert a wide range of effects on the heart, kidneys and central nervous system.

Atrial natriuretic peptides is released from the atria in response to stretch, leading to natriuresis and vasodilatation. In humans, brain natriuretic peptide (BNP) is also released from the heart (mainly from the ventricles) and its actions are similar to those of ANP. C-type natriuretic peptide is limited to the vascular endothelium and central nervous system and has only limited effects on natriuresis and vasodilatation. (Van cheng et al, 2001 & Moe et al, 1993.)

Vasopressin:

Vasopressin concentration is also increased in severe chronic heart failure. High concentrations of the hormone are particularly common in patients receiving diuretic treatment and this may contribute to the development of hyponatremia. (Francis et al, 1990 & Goldsmith, 1986).

Endothelin :

Endothelin is secreted by vascular endothelial cells and is a potent vasoonstrictor on the renal vasculature, promoting the retention of sodium and water. Endothelin concentration is also correlated with indices of severity such as the

pulmonary artery capillary wedge pressure need for admission to hospital and death. (Tsutamoto et al, 1995)

In view of the vasoconstriction properties of endothelin interest has developed in endothelin receptor antagonist as cardio vascular protective agents which inhibit endothelin mediated vascular and myocardial remodelling. (Yanagisawa et al, 1988). .

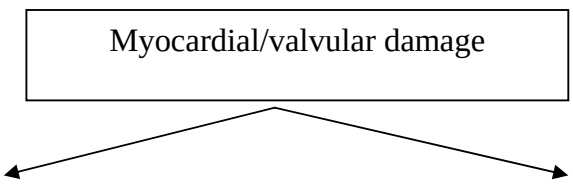
Patterns of neurohormonal activation and prognosis :

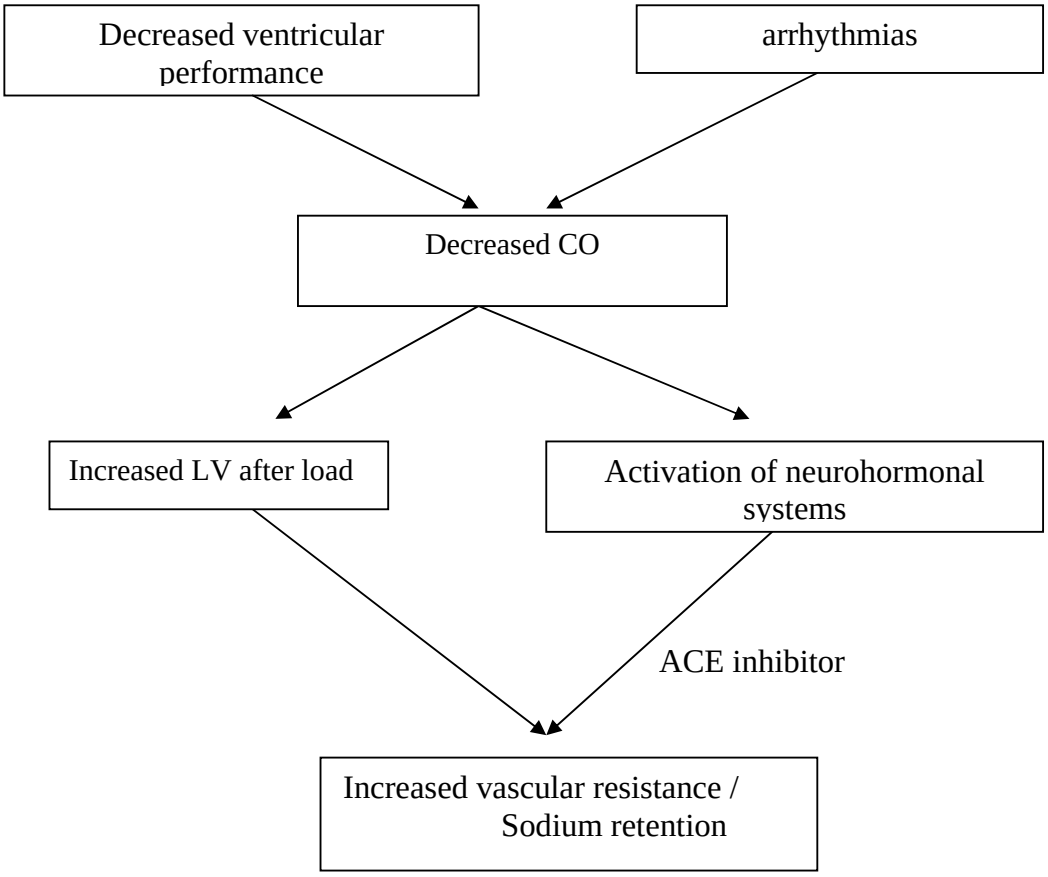
Asymptomatic LV dysfunction :

Plasma norepinephrine concentrations increase early in the development of left ventricular dysfunction and plasma renin activity usually increases in patients receiving diuretic treatment. Norepinephrine concentration in asymptomatic LV dysfunction is a strong and independent predictor of the development of symptomatic heart failure and long term mortality.

In severe untreated chronic heart failure, concentrations of renin, angiotension II, aldosterone, noradrenaline and ANP are all increased. Plasma concentrations of various neuroendocrine markers correlate with both the severity of heart failure and the long term prognosis. Patients with chronic heart failure and raised plasma noradrenaline concentrations do also have a worse prognosis. (Esler 1997)

Effect of ACE inhibitor in CHF: (Yusuf et al 2000)





Diastolic dysfunction:

Diastolic heart failure refers to the clinical syndrome of HF with a preserved left ventricular ejection fraction (0.50 or more) in the absence of major valvular disease. The pathophysiology of diastolic heart failure characterized by a low cardiac output that results typically from a ventricle that has thick walls but a small cavity (increased LV mass). The left ventricular cavity is stiff. It relaxes slowly in early diastole and offers greater resistance to filling in late diastole, so that diastolic pressure is elevated. The low cardiac output manifests as fatigue, while the higher end diastolic pressure is transmitted backwards through the valveless pulmonary veins to the pulmonary capillaries, resulting in exertional dysponea. (Vasan, Levy 2000). Mechanisms contributing to abnormal left ventricular diastolic properties include, stiff arteries, hypertension, ischaemia, diabetes and intrinsic myocardial

changes with or without associated hypertrophy. (Kitzman et al 2002). The prognosis of diastolic heart failure is generally better than that of systolic HF.

Diastolic heart failure is common in clinical practice. The diagnosis of diastolic heart failure may be considered in patients with HF who have a normal LV ejection fraction (0.50 or more). Prevention of diastolic heart failure can be achieved through better control of hypertension and other cardiac vascular risk factors in the community. (Ibrahim 2003).

The pathophysiological abnormalities trigger neurohormonal activation as happens in systolic heart failure. Symptoms may be unmasked by exercise, because unlike normal people, patients with diastolic heart failure are unable to augment their stroke volume by increasing their left ventricular diastolic volume.

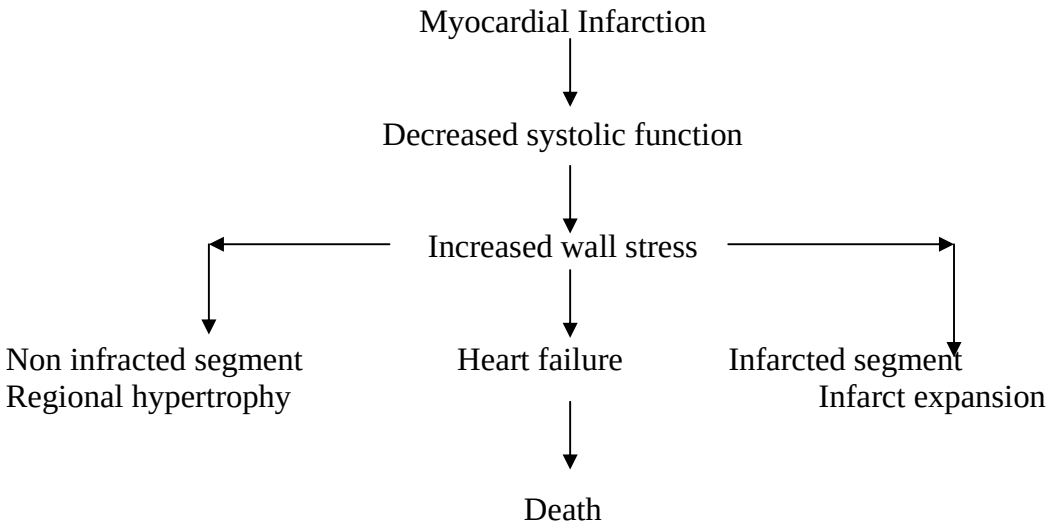
Myocardial dysfunction due to remodelling, hibernation and stunning:

After extensive myocardial infarction, cardiac contractility is frequently impaired and neurohormonal activation leads to regional eccentric and concentric hypertrophy of the non-infarcted segment, with the expansion of the infract zone. This is known as ventricular remodelling. Particular risk factors for this development of progressive ventricular dilatation after a myocardial infarction include a large infarct, anterior infarctions, occlusion of the artery related to the infarct and hypertension.

Myocardial dysfunction may also occur in response to stunning (post ischaemic dysfunction) which describes delayed recovery of myocardial function, despite restoration of coronary blood flow, in the absence of irreversible damage. This is in contrast to ‘hibernating’ myocardium, which describes persistent myocardial dysfunction at rest, secondary to reduced myocardial perfusion although

cardiac myocytes remain viable and myocardial contraction may improve with revascularization. (Rahimtoola 1989)

Process of ventricular remodeling



Types of heart failure :

Forward failure :

Forward failure developes due to marked reduction of left ventricular output and cardiogenic shock occurs. (Mechenzie J. 1913)

Backward failure :

Small transient inequality of output between the two ventricles resulting in acute pulmonary edema.

Right sided heart failure :

Due to stagnation of blood in the right side of heart and lungs right heart failure signs develop.

Left sided heart failure :

Due to poor cardiac output in aortic stenotic lesion or massive MI the left sided heart failure develops.

Acute heart failure Vs chronic heart failure :

The clinical manifestations of HF depends mainly on the rate at which the syndrome develops and specifically whether sufficient time has elapsed for compensatory mechanisms to become operative and for fluid to accumulate in the interstitial space.

Low output Vs high output failure :

Heart failure occurring in low cardiac output at rest or in milder cases during exertion characterizes most forms of cardiovascular diseases.

High output heart failure occurs in variety of high output states like thyrotoxicosis, paget's disease, AV fistulas, anaemia and beriberi. (Stevenson 1989).

Systolic Vs diastolic HF :

Implicit in the physiological definition of heart failure (inability to pump an adequate volume of blood and or to do so only from an abnormally elevated filling pressure) is that heart failure can be caused by an abnormality in systolic function leading to a defect in expulsion of blood leading to systolic heart failure or by an abnormality in diastolic function leading to a defect in ventricular filling (diastolic failure). This may be due to slowed or incomplete ventricular relaxation, which may be transient as occurs in ischaemia, or sustained, as occurs in concentric myocardial hypertrophy or restrictive cardiomyopathy secondary to infiltrative conditions like amyloidosis.

The principal clinical manifestations of systolic failure result from an inadequate cardiac output and secondary salt and water retention – forward heart failure. (Gaasch 1994)

Whereas the major consequence of diastolic HF is related to the elevation of ventricular filling pressure and the high venous pressure upstream to the ventricular cavity, causing pulmonary and systemic congestion (backward failure). (Vasan 1995).

Clinical features and complications :

Patients with HF present with a variety of symptoms, most of which are nonspecific. The common symptoms of congesitive HF include fatigue, dyspnoea, pedal edema and exercise intolerance or symptoms that relate to the underlying cause.

Symptoms and signs in heart failure :

Symptoms

Dyspnoea

Orthopnoea

Palpitation

Chest pain

PND

Reduced exercise tolerance, lethargy, fatigue

Nocturnal cough

Wheeze

Ankle swelling

Anorexia

Signs

Cachexia and muscular wasting

Tachycardia

Pulsus alternans

Increased JVP

Displaced apex beat

RV heave

Crepitations or wheeze

3rd heart sound

Oedema

Hepatomegaly, ascites

Symptoms :

Dyspnoea

Exertional breathlessness is a frequent presenting symptom in heart failure, although it is a common symptom in patients with pulmonary disease. Dyspnoea is therefore moderately sensitive but poorly specific for the presence of HF.

Orthopnoea is a more specific symptom. PND results from increased LV filling pressure and therefore has a greater sensitivity and predictive value. (Manning 1995).

Fatigue and lethargy

Fatigue and lethargy in CHF are due to impaired muscle blood flow and deficient endothelial function. (Manchini 1994)

Oedema

Swelling of ankles and feet is another common presenting feature. Heart failure may manifest as oedema, right hypochondrial pain (liver congestion), abdominal distension(ascites) and loss of appetite(due to bowel congestion). An increase in weight may be associated with fluid retention although cardiac cachexia and weight loss are important markers of disease severity. (Milne 1985)

Physical signs :

Physical examination has serious limitations as many patients particularly those with less severe heart failure, have few abnormal signs. In addition, some physical signs are difficult to interpret, and if present, may occasionally be related to causes other than HF.

Oedema and a tachycardia, for example are too insensitive to have any useful predictive value and although pulmonary crepitations may have a high diagnostic specificity. Increased JVP has a high specificity in diagnosing HF in patients who are known to have cardiac disease.

Displaced apex beat in patients with myocardial infarction and 3rd heart sound have a relatively high specificity.

Precipitating factors of heart failure : (Ghali 1988)

1. iatrogenic cause – NSAID's and steroids
2. arrhythmias, especially atrial fibrillation
3. infection especially pneumonia
4. acute myocardial infarction

5. angina pectoris or recurrent myocardial ischaemia
6. anaemia
7. alcohol excess
8. poor drug compliance especially in antihypertensive treatment.
9. thyroid disorders – thyrotoxicosis
10. pulmonary embolism
11. pregnancy
12. Excess water intake
13. Exertion
14. Excess salt consumption due to prepared dishes

European society of cardiology guidelines for diagnosis of heart failure :

Essential features :

Symptoms of heart failure (eg breathlessness, fatigue, swelling)
and objective evidence of cardiac dysfunction at rest.

Nonessential features :

Response to treatment directed towards heart failure (in cases where the diagnosis is in doubt)

Complications of heart failure:

1. **Arrhythmias**

Atrial fibrillation:

AF that occurs with severe left ventricular dysfunction following myocardial infarction is associated with a poor prognosis. In addition patients with heart

failure and atrial fibrillation are at particularly high risk of stroke and other thromboembolic complications. (Skinbane 1997).

Ventricular arrhythmias

Malignant ventricular arrhythmias are common in end stage HF, sustained monomorphic VT occurs in upto 10% of patients with advanced heart failure.

2. Stroke and thromboembolism:

CHF predisposes to stroke and thromboembolism with an overall estimated annual incidence of approximately 2%. Factors contributing to the increased thromboembolic risk in patients with HF include low cardiac output regional wall motion abnormality and associated atrial fibrillation. (Zuccala 1997)

Prognostic factors in CHF:

Most long term(>10 yrs of followup) longitudinal studies of HF , including the Framingham heart study(1971) were performed before the widespread use of ACE inhibitors. In the Framingham study overall survival at 8 years for all NYHA classes was 30%. The prognosis in patients whose LV dysfunction is asymptomatic is better than that in those whose LV dysfunction is symptomatic

The prognosis in patients with CHF is dependent on severity, age and sex with a poorer prognosis in male patients. In addition numerous prognostic indices are associated with an adverse prognosis, including NYHA class, LV ejection fraction and neurohormonal status.

Survival can be prolonged in chronic heart failure that results from systolic dysfunction, if angiotension converting enzyme inhibitors are given. (Consensus 1987)

Some predictors of poor outcome in chronic heart failure:

1. high NYHA functional class
2. reduced LV ejection fraction
3. low peak oxygen consumption with maximal exercise
4. third heart sound
5. increased pulmonary artery capillary wedge pressure
6. reduced cardiac index
7. diabetes mellitus
8. reduced sodium concentration
9. raised plasma catecholamine and natriuretic peptide concentrations.

Sudden death:

Sudden death may be related to ventricular arrhythmias although asystole is a common terminal event in severe heart failure. (Feldman 1988).

MATERIALS AND METHODS

Setting : The present work was carried out at the Dept. of Medicine and Cardiolgy, Govt. Rajaji Hospital and Madurai Medical College, Madurai.

Collaborating Departments	:	The work was carried out in colloaboration with Dept. of Cardiology, Govt. Rajaji Hospital and Madurai Medical College, Madurai.
Design of Study	:	Cross sectional study
Period of Study	:	May 2005 to Nov 2005
Sample size	:	196 patients
Ethical committee approval	:	The present project was approved by the ethical committee.

Inclusion criteria :

All cases of proved congestive heart failure admitted in medical wards of Govt. Rajaji Hospital, Madurai.

Exclusion Criteria :

1. All cases under anti failure treatment
2. Cases admitted in other wards apart from medical wards
3. Patients less than 12 years of age
4. Pregnant women

Consent :

Informed consent was obtained from all those who participated in the study or their relatives

Materials :

Thus a total of 196 cases who satisfied the inclusion and exclusion criteria stated above were taken up for subsequent study.

Definitions used for the study :

1. Congestive heart failure :

Heart failure is the state of any heart diseases in which despite, adequate ventricular filling, the cardiac output is decreased or in which the heart is unable to pump blood at a rate adequate for satisfying the requirements of the tissues. (Zipes et al 2004)

2. Dyspnoea :

Undue awareness of ones own breathing. Dyspnoea is a cardinal manifestation of left ventricular failure. (Zipes et al, 2004)

3. Smoking :

The subject was considered to be a smoker if he / she gave a history of tobacco smoking with in the past 20 years. Persons who had quit smoking completely before 20 years were not considered as smokers. (IARC Vol 83, 2003)

4. CAD

Those patients who are having ECHO evidence of regional wall motion abnormality are diagnosed as coronary artery disease patients.

5. RHD

Those patients who are having ECHO evidence of valvular abnormalities are diagnosed as rheumatic heart disease patients.

6. Systemic Hypertension :

A subject was considered to have systemic hypertension if he/she was already diagnosed to have systemic hypertension and was on anti hypertensive medication or if the systolic blood pressure during the hospital stay was found to be more than or equal to 140 mm Hg and / or the diastolic blood pressure was more than or equal to 90 mm Hg according to the JNC VII report. (Chobanian et al 2003)

7. Anaemia :

Anaemia may be defined as a state in which the blood Hb level is below the normal range for the patients age and sex (males < 12 gms) Non pregnant females < 11.5 gms)

8. Diabetes Melitus : (American Diabetes association 2004)

A subject was considered to have diabetes mellitus if he / she was already diagnosed to have diabetes mellitus or during the hospital stay was found to have a

- Fasting plasma glucose of $\geq$ 126 mg /dl

  or

- 2 hours PP plasma glucose $\geq$ 200 mg / dl
- Symptoms of diabetes mellitus plus

  random blood sugar $\geq$ 200 mg / dl

9. Left ventricular dysfunction : (Colucci et al 2001)

Left ventricular dysfunction was divided in to mild, moderate, severe according to ejection fractions in ECHO.

$\leq$ 29%	Severe
30-39	Moderate

40-49	Mild

10. BMI :

BMI is the convenient and reliable indicator of body fat which is the body weight in kilograms divided by square of height in meters.

>25	all abnormal
25 - 30	Over weight
> 30	obese
> 35	Markedly obese

11. Waist Hip Ratio :

It is an approximate index of intra abdominal fat mass and total body fat. High WHR (> 1.0 in men and > 0.85 in women) indicates abdominal fat accumulation. (Charney et al, 1976)

12. Cardiomyopathy :

Cardiomyopathy is a group of diseases that affect the heart muscles itself and not the result of HT or congenital or acquired valvular, coronary or pericardial abnormalities. (Zipes et al 2005)

13. Cor – pulmonale :

Corpulmonale is defined as enlargement of right ventricle secondary to abnormalities of the lungs, thorax, pulmonary ventilation or circulation. It sometimes leads to RV failure, with an elevation of transmural RV end diastolic pressure. (Zipes et al 2005)

14. Drug Intake : (NSAIDs)

Consumption of NSAIDs was considered as a precipitating factors , in those patients who developed CHF after taking NSAIDs in the recent past. All the other possible precipitating factors were excluded.

15.Infection

Infection was considered as a precipitating factor in those patients who developed CHF and presented with features of acute infection which was confirmed by clinical and laboratory means.

16. Diet :

Diet was considered as a precipitating factor in those patients who developed CHF due to intake of salt rich food like dried fish, chips and pickles, when the other factors were excluded.

17. Arrhythmias :

Arrhythmias was considered as a precipitating factor in those CHF patients who had clinical and ECG evidence of arrhythmias.

Methods :

Selected socio-demographic, clinical and laboratory data were collected from the patients and recorded in a proforma (enclosed in Appendix)

I Socio – demographic data comprised of

1. Age
2. Sex
3. History of tobacco smoking

II Clinical Data :

Clinical Examination

III Laboratory Data :

Hb

Blood sugar, Urea, Creatinine

ECG

ECHO

Hb :

Sahlis Haemoglobinometer was used for Hb estimation.

ECG

12 lead multi channel ECG was taken in all the patients.

ECHO :

Transthoracic Echo was done using ALOKO PROS 100 in all the cases. ECHO, M – MODE ECHO was done to analyse regional wall motion abnormality, presence of clot and to assess the left ventricular function. Colour Doppler evaluation was done to evaluate the presence of valvular regurgitation and also to assess the diastolic function.

Conflict of interest :

Nil

Financial Support :

Nil

Limitations :

1. There is no definite parameter to measure the serum drug level in patients with drug intake.
2. B – Natriuretic and ANP levels could not be measured.

Statistical Analysis :

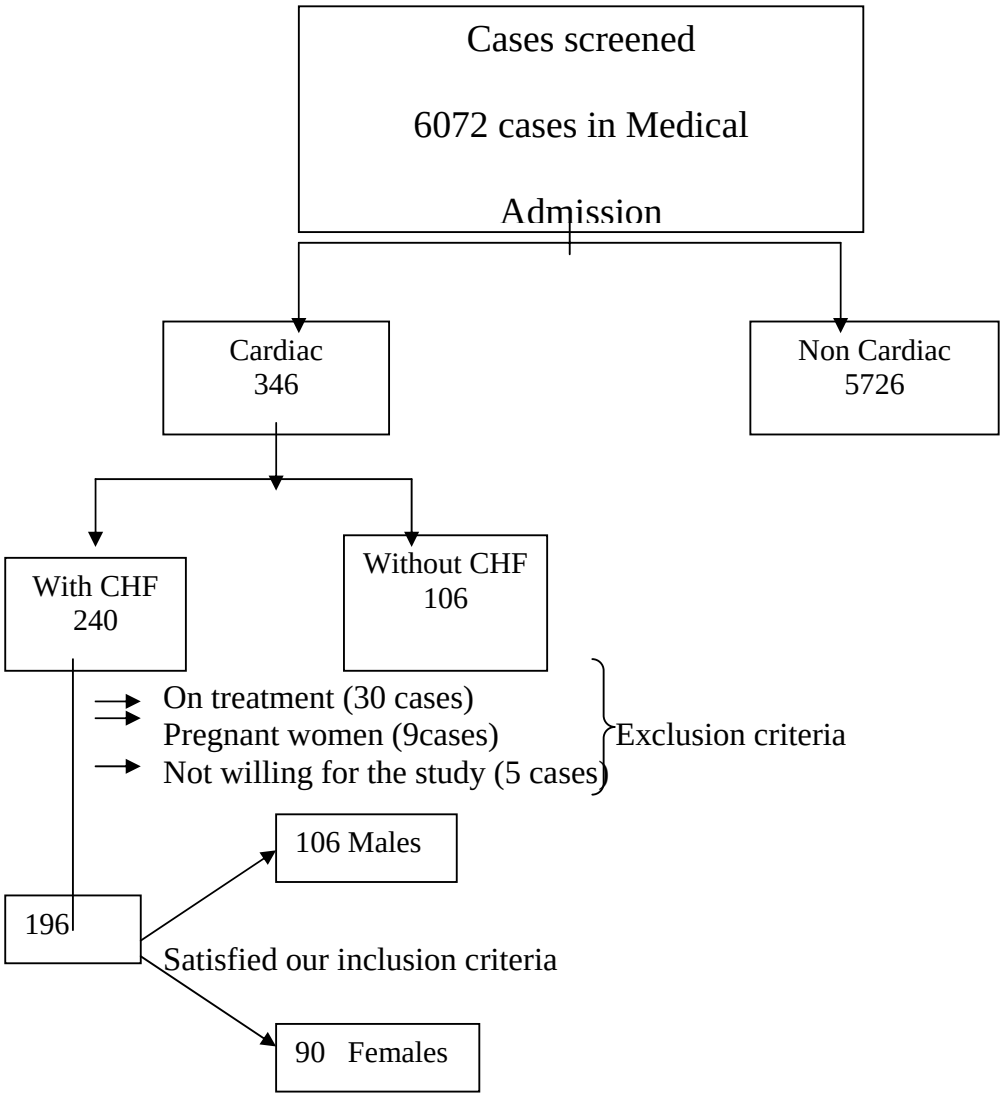
Data were entered in Microsoft Excel spread sheet and analysed utilizing the software – Epidemiological Information Package 2002 developed by the Centre for disease control and prevention, Atlanta for World Health Organization. Range, Median, Mean, Standard Deviation and 'p' values were calculated using this package. Chi-square test was done to find out the significance of relationship between the groups. Significance was considered if the 'p' value was below 0.05.

RESULTS

During the study period, 6072 patients were admitted in the medical wards. Out of them 196 satisfied the inclusion criteria. So the prevalence was 3.2 % (196/6072).

Among the 196, 7 were expired, thus case fatality rate was 3.5%. (7/196).

Disposition of Study Subjects



Distribution of cases in relation to age group and gender is produced in Table No. 1 given below.

Table – 1 - CHF Age and Gender Distribution Chart

Age	Male	Female	Total
15-24	5	15	20
25-34	8	9	17
35-44	16	19	35
45-54	21	18	39
55-64	34	19	53
65-74	15	8	23
75-84	5	2	7
85-94	2	0	2
Total	106	90	196
Mean	43.67	52.03	48.9
S.D	16.89	14.98	16.39
Median	55	45	

In the present study median age affected by CHF among male was 55 years, among female was 45 years and in general it was 50 years.

In the present study, among 196 CHF patients , 92 patients (47%) were under the age group of 45 to 65. Among these, males were affected more, approximately 60%. The remaining patients were females.

Table – 2 - CHF gender variation

Sex	Observed	Expected
Male	106	182
Female	90	122
Total	196	304

P = 0.072

P > 0.05 Not significant

In the present study various presenting features of CHF were elicited. The details are shown in the table no. 3 below.

Table – 3 CHF - PRESENTING FEATURES and GENDER

(Overlapping with each other)

	Male	Female	Total	Proportion
Dyspnea	106	88	194	0.99
Palpitation	78	77	155	0.79
Chest pain	83	77	160	0.82
Pedal edema	101	83	184	0.94
Abdominal Pain	65	66	131	0.67
Abd. distension	49	60	109	0.56
Oliguria	83	68	151	0.77
Pulmonary rales	104	88	192	0.98
Hepatomegaly	67	62	129	0.66

Table : 4

CHF : CAD Vs Presenting features

	CAD with symptoms	CAD without symptoms
Dyspnoea	76	6
Pedal edema	66	16

Chest pain	70	12

X2 - 5.2497

P value - 0.0220

P is < 0.05 significant

In the present study, the most common presenting feature was dyspnea (99%) and the sign was pulmonary rales(98%). The second most common presenting features was pedal edema (94%).

Careful elicitation of the precipitating factors helped to identify the conditions in order to eliminate or overcome the same during therapy. During the present study various precipitating factors were identified. The details are shown in Table No. 5.

Table – 5 - CHF – Precipitating Factors

	Male	Female	Total
Diet	19	11	30
Drug	44	42	86

Anaemia	18	12	30
Infection	18	16	34
Arrhythmias	7	9	16
Total	106	90	196

Among the 196 patients, 86 patients were taking one or more drugs for the underlying disease.

Out of 196 patients 34 patients were had some form of infection. 30 patients had anaemia.

Arrthymia was a precipitating factor in 16 (M-7, F-9) out of 196 patients.

Various etiological factors were analysed in the study. The details are shown below in Table – 6

Table – 6 : CHF - Etiological factors

	Male	Female	Total
CAD	71(86.5%)	11(13.5%)	82
RHD	12(25%)	36(75%)	48
Systemic HT	12(40%)	18(60%)	30

CM	3(17%)	15(83%)	18
Corpulmonale	8(44%)	10(56%)	18
Total	106	90	196

In the present study, out of 196 CHF patients, 82 were due to CAD, 48 were due to RHD and 30 were due to systemic hypertension.

Other causes were cardio myopathy 18 and corpulmonale 18.

Out of 30 CHF patients due to Systemic hypertension, 18 were females.

In the present study waist circumference of the CHF patients were given below in table no. 7

Table No. 7 : CHF Waist circumference

Waist Circumference	Male	Female	Total
< 80 cm	38 (51%)	36(49%)	74
80 – 84.9	49 (58%)	36(42%)	85

85 – 89.9	14 (50%)	14 (50%)	28
90-94.9	4 (80%)	1 (20%)	5
95 – 99.9	1 (33%)	2 (67%)	3
> 100	0 (0%)	1(100%)	1
Total	106	90	196

Out of 196 patients 113 had waist circumference between 80-90 cms.

CHF precipitated by various factors were analysed individually, the figures are given below.

Table 8 : CAD Vs Drug Intake

Sex	CAD due to drug intake	CAD due to other precipitating factors	Total
Male	33	38	71
Female	7	4	11
Total	40	42	82

X^2 = 1.1222

P = 0.02894

P > 0.05 not significant.

Among 82 CHF patients due to CAD, 40 patients, heart failure was precipitated by NSAIDs intake.

Table No. 9 : RHD Vs Drug Intake

Sex	RHD due to drug intake	RHD due to other precipitating factors	Total
Male	5	7	12
Female	17	19	36
Total	22	26	48

X2 = 0.1119

P value = 0.738

P is > 0.05 not significant

Among 48 CHF patients due to RHD in 22 patients it was precipitated by NSAIDs intake.

Table No. 10 : Systemic hypertension Vs Drug intake

Sex	Sy.HT due to drug intake	Sy.HT due to other precipitating factors	Total
Male	2	10	12
Female	13	5	18
Total	15	15	30

X2 = 8.8889

P value = 0.0029

P < 0.05 It is significant.

Among the 30 CHF patients due to systemic HT, 15 patients failure was precipitated by drugs like NSAIDs

Table – 11 : CAD Vs Infection

Sex	CAD precipitated by Infection	CAD precipitated by Others	Total

Male	11	60	71
Female	1	10	11
Total	12	70	82

X2 = 0.3125

P value = 0.5762

P is > 0.05 not significant

In 82 patients with CHF due to CAD only 12 patients, heart failure was precipitated by an acute infection.

Table : 12 - RHD Vs Infection

Sex	RHD precipitated by Infection	RHD precipitated by Others	Total
Male	3	9	12
Female	8	28	36
Total	11	37	48

P value

X2 - 0.0393

P value - 0.8428

P is > 0.05 not significant

Among 48 CHF patients due to RHD 11 patients, heart failure was precipitated by Infection.

Table : 13

CHF- Systemic HT Vs Infection

Sex	Sys HT precipitated by infection	Sys HT precipitated by others	Total
Male	3	9	12
Female	2	16	18
Total	5	25	30

X2 - 0.999

P value - 0.3173

P is > 0.05 Not significant

Out of 30 CHF patients due to Systemic HT 5 had failure precipitated by acute infection.

Table No : 14

CHF - CAD Vs Diet

Sex	CAD precipitated by Diet	CAD precipitated by Others	Total
Male	13	58	71
Female	0	11	11
Total	13	69	82

X2 = 2.3935

P value= 0.1218

P is > 0.05 Not significant

Out of 82 CHF patients due to CAD, 13 patients heart failure were precipitated by salty foods like fried fish.

Table : 15

CHF - CAD Vs Arrythymias

Sex	CAD precipitated by Arrhythmias	CAD precipitated by Arrythymias	Total

Male	5	66	71
Female	2	9	11
Total	7	75	82

X2 - 1.5137

P value - 0.2186

P is > 0.05 Not significant

Out of 82 CHF patients, due to CAD 7 patients had heart failure precipitated by arrythymias.

Table : 16 CHF - RHD Vs Arrhythmias

Sex	RHD precipitated by Arrhythmias	RHD precipitated by Others	Total
Male	0	12	12
Female	4	32	36
Total	4	44	48

X2 - 1.4545

P value - 0.2278

P is > 0.05 Not significant

Out of 48 CHF patients due to RHD, 4 patients heart failure was precipitated by Arrythymias.

BMI was calculated in patients belonging to different categories of heart failure and the details are shown in Table No. 17.

Table No. 17 CHF - BMI

	CAD		NON CAD		
BMI	MALE	FEMALE	MALE	FEMALE	T
0-15	1	0	0	0	1

15-20	2	3	5	6	16
20-25	21	12	29	37	99
25-30	25	11	18	18	72
30-35	3	2	2	1	8
Total	52	28	54	62	196

Out of 196 CHF patients, 171 patients, BMI was between 20-30.

DISCUSSION

Heart failure has been the number one cause for death in every year during the past century, while death from CAD and stroke has been decreasing over the past decade, there has been a significant increase in the prevalence of morbidity and mortality from CHF.

Data from Framingham study suggest that the estimated prevalence of CHF ranges from 2% to 6%. In smaller mid western areas the prevalence rate is as high as 6% of population. The prevalence of CHF in the present study was 3.2%

A comparative analysis given below in Table No. 18

Table - 18

CHF comparative Analysis

Sl No.	Details of study	Prevalence	Remarks

1.	Framingham Study	2 to 6 %	Population based study
2.	Present study	3.2 %	Hospital based study

The prevalence is lower in the present study comparing to western studies.

Following causes have been identified for the low prevalence of CHF in the present study:

1. Hospital bias – highly selected hospital services
2. Referral system : Critical cases are referred from primary and secondary, care centres to the tertiary care unit.
3. Facilities : In western countries increased awareness has helped critical cases to be referred immediately to the hospital.
4. In our health care system, the medical college hospitals, that are supposed to the tertiary care centres provide primary, secondary and tertiary care services. So referrals are not uniform.
5. Complexity of cases : Variety of problems related to Infections, Toxic related and degenerative disorders, contribute to the admission. So the true prevalence gets altered.

From population studies, Sulton Gc et al observed, CHF is less than 1% for patients < 50 years and the prevalence increases to approximately 5% for patients

between 50-70 years of age, whereas it was 10% for all patients over the age of 70 years.

In the present study prevalence among the age group 45 - 54 comes around 20%, 55-64 comes to 27%, and 65-74 comes 12%

Comparing to Western data, in our study, prevalence of CHF is decreased as age advances because of the possible factors :

1. Biological factors - Longevity is low in our country.
2. Environmental factors - Patients die earlier due to other causes.
3. Socio-cultural factors - Reluctance to treat elderly patients with advanced heart failure.

Annual Incidence of Heart failure (per 1000 population in relation to gender and age)

In the Framingham heart study

Table - 19 Comparative analysis in %

	Male		Female	
Age	Framingham study	Present study	Framingham study	Present study
50-59	3	9	2	6
80-89	27	2	22	1
All ages	2.3	17	1.4	15

Males are affected more than females. NHANES 1988 – 1994 stated that there is increased prevalence of CHF in men aged 70 years.

In the Framingham study, case fatality rate was about 35-71 among 1000 CHF patients. In the present study, case fatality rate was about 36 among 1000 CHF patients.

Cowie et al stated that exertional breathlesseness was a frequent presenting symptom and pulmonary rales was the most frequent sign in CHF.

In the present study the most common presenting symptom was Breathlessness and it was observed in 99% and most of them suffered from CAD.

Most common presenting sign was pulmonary crepitations, seen in around 98%. Pedal edema was the third most common presenting symptom. It was seen in about 94% of patients. Elevated JVP was seen in 110 of 196 patients. Tender hepatomagaly was observed in 80 out of 196 patients.

Comparative analysis of presenting symptoms is given in

Table No. 20 below.

Table - 20 Comparative Analysis

Details of study	Breath lessness	Pedal edema	JVP	Tender hepatomagaly	Pulmonary rales
Framingham Study	92%	85%	64%	42%	90%
Present study	99%	94%	72%	40%	98%

In present study, it was noted that, most of the anaemic patients presented with pedal edema and breathlessness.

Heerdinke E, et al, stated that patient who consume NSAIDs had increased risk of congestive heart failure more so in elderly patients.

An often neglected subject in the CHF is the use of drugs that are harmful in patients with heart failure. NSAIDs are notorious for provoking decompensation of previously stable heart failure.

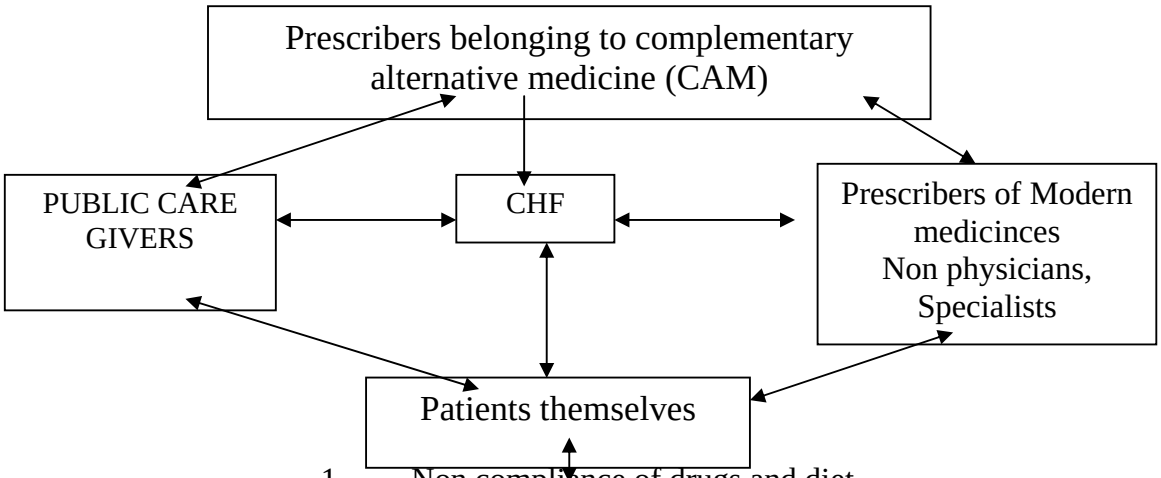
NSAIDs produce salt and water retention, Impaired renal function can be associated with life threatening hyperkalemia and increased fluid volume.

In the present study it was noted that 86 heart failure patients, out of 196 patients (44%) were precipitated by recent intake of NSAIDs. Next common precipitating factor was acute infection (17%) which was evidenced by clinical and laborotary means. Anaemia and diet contributed in 15% each. The remaining 9% of the patients it was precipitated by arrhythmias.

Regarding precipitating factors, 3 Ps are significant.

1. **Prescribers**
2. **Patients**
3. **Public**

PRECIPITATING FACTORS



1. Non compliance of drugs and diet
2. Depression
3. Frustrations
4. Economical Factors

Regarding **prescribers,** complementary alternative medicine practitioners may prescribe some native medicine or NSAIDs. That may precipitate CHF. Non physicians, or other specialists also prescribe pain killers and other drugs that can precipitate CHF.

Regarding **patients** CHF may be precipitated by

- Poor compliance of drugs and diet due to depression and frustrations
- Economical Factors

Often **public-**care givers, direct patients to nonconventional medical practices. As a result, patients consume unnecessary medicines and CHF gets precipitated.

The following remedies should be taken to reduce the prevalence of CHF. Remedial Measures are furnished in Table No. given below

Table No : 21

Educational aspects on CHF for patients – public and practitioners

Patients	Patient's Family	Public	Practitioners
Avoid	**Advice**	**Awareness through**	**Assess**
1. Excess fluid & Salt 2. Medications Such as NSAIDs	1. Drug compliance 2. Behaviour modification 3. Caring	1. TV 2. Newspapers 3. Question& answers	1.Unnecessary drugs 2. Underlying heart disease

3. Alcohol 4. Smoking	4. Periodic follow up		3. Urine for proteinuria

So, the patients should be instructed about

A - Activity reduction

B - Behaviour modification

C - Compliance of drugs

D - Diet modification

E - Evaluation of other factors

Coming to the etiological factors, Khardra et al, observed CAD as the commonest cause of heart failure. In the SOLVD study CAD accounted for almost, 75% of the cases of heart failure. CAD and HT were implicated as the cause in over 70% of cases of heart failure in the Framingham study. Recent Hillington heart failure study has identified CAD as the primary etiology in 36% of HF.

In the present study CAD is the most common etiology for CHF. It comes to around 42%. Next common etiology is RHD. which comes to around 24%, Systemic HT is the third common etiology.

In the present study, CAD and RHD are the most common etiological factors for CHF.

Etiologiocal aspects of the Heart failure is shown in Table 22 below.

Table - 22

A comparative Analysis :

Etiology Year	Present study (2005)	Teerlink et al study (1989-91)	Framingham heart study (1995)	Hillington's study (1995)
Ischemic CAD	42%	50%	53.5%	36%
Non Ischemic	58%	50%	46.5%	64%

Patients with abnormal waist circumference irrespective of age, size have elevated systolic blood pressure and diastolic blood pressure which was also a risk factor for developing coronary artery disease.

The waist circumference was low or below the standards described for men and women in 97% in the study population in contrast to Siedell et al (1998). The low waist circumference may reflect their socio-economic status.

Charney et al suggested that BMI > 30 had increased risk of CHF.

In the present study, 8% of the CHF patients had BMI < 20. 51% of the CHF patients had BMI between 20-25 and 35% of the patients between 25-30. In contrast to Charney et al, in the present study most of the CHF patients had BMI between 20-30. This may reflect the low socio-economic status of the study population.

CONCLUSION

1. The prevalence of congestive heart failure among hospitalized was 3.2 %.
2. Congestive heart failure was common among the age group of 45 – 65 years.
3. Dyspnea and pulmonary crackles were the most common presenting symptom and sign respectively.
4. Drug intake mainly Non steroidal anti inflammatory drugs, anaemia and infections were the most common precipitating factors.
5. Coronary artery disease and Rheumatic heart disease were the most common etiological factors
6. Among young individuals, female predominance was noticed and attributable to rheumatic heart disease whereas, the reverse was true in middle age and old age. The later could be due to tobacco related cardiac illness or other etiologies.
7. Waist circumference was in between 80-90 cm in most of the study population

8. The Body mass index of 171 patients out of the 196 patients included in the study was between 20-30.
9. In hospital, case fatality rate was 3.5%

SUMMARY

Congestive heart failure (CHF) a commonly encountered clinical entity was studied among the patients admitted in the medical wards over 6 months period. It was carried out, a) to find out the prevalence of congestive heart failure among the patients admitted in the hospital, b) to analyse their presenting features & precipitating factors c) to correlate the clinical findings with the underlying etiology and d) to study outcome of these cases during their hospital stay.

Among 6072 admissions 196 patients with congestive heart failure satisfied the inclusion criteria. The prevalence rate was 32.28 / 1000 population, and observed more in males but not statistically significant. The case fatality rate among the congestive heart failure patients was 3.5% (7/196).

The most common age group of affected individuals was 45 – 65 years.

Etiological factors for the congestive heart failure were in the order of Coronary artery disease. (42%), Rheumatic heart disease (24%), Systemic hypertension (15%) and others (19%)

Most common presenting symptom was breathlessness (99%) and the sign was pulmonary crackles.(98%). Other presenting features were pedal edema (94%), chest pain (82%), oliguria (77%) and tender hepatomegaly (40%). Most common precipitating factors noted were NSAIDs intake (44%) followed by infection (17%), anaemia (15%), salty diets (15%) and arrthymias (9%).

With better understanding of the pathophysiology of CHF both non pharmacological means and the available newer group of drugs made available to patients through the 3Ps **(patients, prescribers, public caregivers)** the quality of life of the unfortunate patients could be improved.

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PROFORMA

Prevalence, Pattern, Precipitating factors and Presenting Features of CCF

S.No.		I.P. No.	:
Patient's Name :		Date of Admn	:
1. Age	:	2. Sex	:
3. Occupation	:	4. Height	:
5. Body mass Index (Weight / H2 in meter)		6. Weight	:
7. Waist in cms	:	8. Hip in cms	:
9. Waist / Hip ratio	:	10. Socio-economic status	:
11. Out come	:	12. Dyspnea	:
13. Palpitations	:	14. Dyspnea Grade	:
15. Chest pain	:	16. Pedal Oedema	:
17. Sacral Oedema	:	18. Abdominal Pain	:
19. Abdominal distesion	:	20. Anorexia	:
21. Nausea, vomiting	:	22. Cyanosis	:
23. Oligurea	:	24. Pulmonary rales	:
25. Hepatomegaly	:	26. JVP	:
27. Hypertension	:	28. Smoker	:
29. Smoker	:	30. Alcoholic	:
31. Diet	:	32. Anemia	:
33. Infection	:	34. Coronary heart disease	:
35. Rheumatic heart disease	:	36. Dyslipidemia	:
37. Regular Exercise	:	38. Pregnancy	:
39. Thyrotoxicosis	:	40. Hypothyroidism	:
41. Drug Intake	:	42. NSAID	:
43. B – Blockers	:	44. Steroids	:
45. Ca – Channel blockers	:	46. Native medicine	:
47. Arrhythmias	:	48. Infective Endocarditis:	
49. Pulse rate	:	50. R – R	:
51. B.P	:	52. Hb	:
53. Echo – EF	:	54. ECG	:
55. Conclusion	:		

ABBREVIATIONS

CO	-	CARDIAC OUTPUT
HF	-	HEART FAILURE
CHF	-	CONGESTIVE HEART FAILURE
CAD	-	CORONARY HEART DISEASE
RHD	-	RHEUMATIC HEART DISEASE
BMI	-	BODY MASS INDEX
JVP	-	JUGULAR VENOUS PULSE
RAAS	-	RENIN ANGIOTENSIN ALDOSTERONE SYSTEM
NSAIDs	-	NON STEROIDAL ANTI INFLAMMATORY DRUGS

MASTER CHART ABBREVIATIONS

Sex	-	Male	- 1	Female	-	2	
others	-	Yes	- 1	No	-	2	
W/H	-	Waist Hip Ratio		Dys	-	Dyspnea	
Palp	-	Palpitation		CP	-	Chest pain	
PE	-	Pedal edema		Smok	-	Smoking	
Abd dis	-	Abdominal Distension		Alco	-	Alcohol	
DM	-	Diabetes mellitus		Hepat	-	Hepatamegaly	
Amem	-	Anaemia		Infe	-	Infections	

sl.no	age	sex	BMI	W/H	dys	palp	cp	pe	abd d	oligur	DM	smok	alcoh	diet	drug	BP	hepat	anem	infn	ECG	CAD	RHD	CM	Cor pu
1	40	2	19.5	0.9	1	1	1	1	2	1	2	2	2	2	1	2	1	2	2	2	1	2	2	2
2	46	1	20.2	0.8	1	2	2	1	2	2	2	2	2	2	2	1	1	1	2	2	2	2	2	2
3	56	2	40.7	0.8	2	2	1	1	2	1	2	2	2	2	2	2	2	2	1	2	2	1	2	2
4	42	1	18	0.9	1	1	1	2	2	1	2	1	1	1	2	1	1	2	2	2	2	2	2	2
5	40	2	27	0.9	1	2	1	1	1	1	2	2	2	2	2	2	1	2	2	1	1	2	2	2
6	40	2	23	0.9	1	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2	1	2	2
7	74	2	19	0.9	2	1	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2	1	2	2
8	35	1	19	0.9	1	1	2	1	1	1	2	1	2	2	2	2	1	2	2	1	1	2	2	2
9	40	1	22	0.9	1	1	1	1	2	2	2	1	1	1	2	2	1	2	2	2	1	2	2	2
10	19	1	22	0.9	1	1	1	1	2	2	2	2	2	2	1	2	1	2	2	2	2	1	2	2
11	55	1	27	1	1	2	2	1	1	1	2	1	1	1	2	2	2	2	2	2	1	2	2	2
12	72	1	23	0.9	1	1	1	1	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2
13	63	1	17	0.9	1	2	1	1	2	1	2	2	2	2	2	1	2	2	1	2	2	2	2	2
14	32	2	20	0.9	1	1	1	1	1	1	2	2	2	2	2	2	1	1	2	2	1	2	2	2
15	14	2	15	0.9	1	1	1	1	1	2	2	2	2	2	1	2	1	2	2	2	1	2	2	2
16	60	1	27	1	1	1	1	1	1	1	1	1	1	2	2	2	1	2	2	1	2	2	1	2
17	55	1	20	0.9	1	2	1	1	1	1	2	1	1	1	2	1	1	2	2	2	2	2	2	2
18	55	2	23	0.9	1	1	1	1	2	1	2	2	2	2	2	2	2	2	2	1	1	2	2	2
19	60	2	25	0.9	1	1	1	1	2	1	1	2	2	2	1	2	1	2	2	2	2	1	2	2
20	60	2	24	0.9	1	1	1	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
21	45	2	22	0.9	1	1	2	1	2	1	2	2	2	2	1	2	1	2	2	2	2	1	2	2
22	50	1	21	0.9	1	2	1	1	1	1	2	1	1	2	2	2	1	1	2	2	1	2	2	2
23	55	1	27	0.9	1	2	2	1	1	2	2	1	1	1	2	2	2	2	2	2	1	2	2	2
24	22	2	24	0.9	1	2	2	1	1	1	2	2	2	2	1	2	1	2	2	2	2	1	2	2

25	15	2	21	0.9	1	2	1	1	2	2	2	2	2	1	2	2	1	2	2	2	2	1	2	2
26	50	2	23	1	1	2	1	1	1	2	2	2	2	2	2	2	1	1	2	2	2	1	2	2
27	48	2	29	0.9	1	1	1	2	1	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2
28	50	2	24	0.9	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	1	2
29	48	2	20	0.9	1	1	1	2	2	1	2	2	2	2	1	2	1	2	2	2	1	2	2	2
30	55	1	22	0.9	1	1	2	1	1	1	2	1	1	1	2	1	1	2	2	2	2	2	2	2
31	48	2	22	0.9	1	1	1	1	2	2	1	2	2	2	2	2	2	1	2	2	2	1	2	2
32	36	2	23	0.9	1	1	1	1	1	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2
33	21	2	21	1	1	2	2	1	2	2	2	2	2	2	1	2	1	2	2	2	2	1	2	2
34	28	2	20	0.9	1	2	1	1	1	1	2	2	2	2	2	2	1	2	1	2	2	1	2	2
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36	60	1	25	0.9	1	1	2	2	2	1	2	2	1	2	2	2	2	1	2	2	1	2	2	2
37	56	2	22	1	1	2	2	1	1	1	2	2	2	2	1	2	1	2	2	2	2	1	2	2
38	14	2	18	1	1	1	2	1	1	1	2	2	2	2	2	2	1	2	2	1	2	1	2	2
39	54	1	20	0.9	1	2	1	1	2	2	2	1	1	2	2	2	2	2	1	2	1	2	2	2
40	58	1	24	1	1	2	2	1	1	1	1	1	1	2	2	2	2	2	1	2	1	2	2	2
41	38	2	23	1	1	1	2	1	1	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2
42	53	1	24	1	1	1	1	1	1	1	1	2	2	1	2	1	1	2	2	2	2	2	2	2
43	50	1	21	0.9	1	1	2	1	2	1	1	2	2	2	1	2	2	2	2	2	1	2	2	2
44	70	2	24	1	1	1	1	1	1	1	2	2	2	2	2	2	1	1	2	2	2	1	2	2
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46	25	1	24	0.9	1	1	2	1	2	2	2	1	1	2	1	2	1	2	2	2	1	2	2	2
47	49	1	24	1	1	1	1	1	2	1	2	1	2	2	1	2	2	2	2	2	1	2	2	2
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63	48	1	22	1	1	2	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2	1	2	2
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65	52	2	26	1	1	1	1	1	1	1	2	2	2	2	2	1	1	2	1	2	2	2	2	2
66	66	1	23	0.9	1	2	1	1	2	1	2	1	2	2	1	2	1	2	2	2	1	2	2	2
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70	60	1	25	1	1	1	1	1	2	2	2	1	1	1	2	2	1	2	2	2	1	2	2	2
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74	32	1	22	1	1	2	2	1	2	1	2	1	1	1	2	2	1	2	2	2	2	2	2	1

75	70	1	19	1	1	2	2	1	2	2	2	1	1	2	1	2	1	2	2	2	1	2	2	2
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80	35	1	24	0.9	1	1	1	1	1	1	2	1	2	2	1	2	1	2	2	2	2	1	2	2
81	65	1	20	0.9	1	1	1	1	2	1	2	1	1	2	2	2	2	2	1	2	1	2	2	2
82	45	1	21	1	1	1	1	1	2	1	2	1	1	2	2	2	2	1	2	2	2	2	1	2
83	43	1	28	1	1	2	1	1	1	2	1	1	1	2	2	1	1	2	1	2	2	2	2	2
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85	50	2	32	1	1	1	1	1	2	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2
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87	38	1	21	1	1	1	2	1	1	2	2	1	1	2	1	2	1	2	2	2	2	1	2	2
88	56	1	30	1	1	1	2	1	1	1	2	1	2	2	2	2	1	2	1	2	1	2	2	2
89	50	1	24	1	1	1	1	1	1	2	1	1	1	2	1	2	2	2	2	2	1	2	2	2
90	35	2	23	0.9	1	1	1	1	1	1	2	2	2	2	1	1	1	2	2	2	2	2	2	2
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95	28	1	22	1	1	1	1	2	1	1	2	1	2	2	1	2	1	2	2	2	1	2	2	2
96	56	1	27	1	1	1	1	1	1	1	2	1	1	2	2	2	1	2	2	1	1	2	2	2
97	65	1	23	1	1	1	1	1	2	1	2	1	1	2	1	1	2	2	2	2	2	2	2	2
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100	37	1	19	1	1	1	1	1	2	1	2	2	1	2	2	2	2	1	2	2	1	2	2	2
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105	37	1	30	0.9	1	2	1	1	1	1	2	1	1	2	2	2	1	1	2	2	2	2	1	2
106	45	1	24	1	1	1	1	1	1	2	2	1	1	2	1	2	1	2	2	2	1	2	2	2
107	31	1	28	0.9	1	1	1	1	1	1	2	1	2	2	2	2	1	1	2	2	2	1	2	2
108	75	1	22	0.9	1	1	1	1	2	1	1	1	2	2	2	2	2	2	2	2	1	2	2	2
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110	20	1	21	1	1	1	1	1	1	1	2	1	1	2	2	2	1	1	2	2	1	2	2	2
111	53	1	23	0.9	1	1	1	1	1	2	2	1	2	2	1	2	1	2	2	2	1	2	2	2
112	14	2	17	0.9	1	1	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	1	2	2
113	21	2	21	1	1	1	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	1	2
114	56	2	29	0.9	1	1	1	1	1	2	2	2	2	2	2	2	1	2	2	1	2	2	1	2
115	13	2	22	1	1	1	1	1	1	2	2	2	2	2	2	2	2	2	1	2	2	2	2	1
116	26	2	23	1	1	1	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2
117	60	2	20	0.9	1	1	2	1	1	1	1	2	2	2	1	1	1	2	2	2	2	2	2	2
118	37	2	22	1	1	1	1	1	2	1	2	2	2	1	2	2	2	2	2	2	2	2	1	2
119	57	1	27	0.9	1	1	1	1	2	1	2	1	1	1	2	2	2	2	2	2	1	2	2	2
120	56	1	26	0.9	1	2	2	1	2	1	2	1	1	2	2	2	2	1	2	2	1	2	2	2
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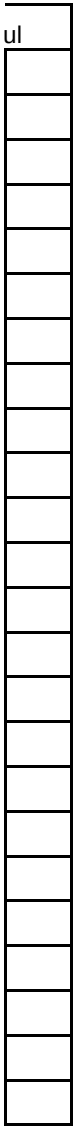
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