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Research Article

HEART FAILURE REGISTRY IN YEMENI HOSPITALS

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Abstract

Background: Heart failure is Inability of Heart to maintain adequate cardiac output to meet the demands of body, its end stage of all diseases that affecting heart and it is a major health problem in our society. It measuring major challenges in its diagnosis and management. **Aim:** This study aimed to registry of Heart Failure at Hospitals of Dhamar City, Yemen, measuring major risk factors can precipitate this condition and guideline treatment their relation to improvement and death. **Patients and Methods:** All patients in the term of heart failure admitted to Dhamar Governorate major hospitals between 15th February 2019 to 15th July 2019. All patients subjected to clinical evaluation, baseline ECG, results of laboratory investigations, treatment strategies. **Results:** Out of 120 patients registered with mean age (58.45 years old) we were found that qat chewing was a major risk factor in 81%, hypertension52%, smoking 41% and ischaemic heart diseases33.3%, Diabetes mellitus 33.3%. On clinical evaluation, dyspnea (NYHA class IV) was most common presenting symptoms in 69.5 %, patients whose received guideline treatment was 49.2%, 63.2% of them had improved the rest of them still not control their co morbidity or present of risk factors especially chewing qat. **Conclusion:** Heart failure registry is intending to provide unique insight into demographic, etiology, clinical presentation and outcomes of heart failure in Dhamar city, this registry will help the local clinician to identify the deficiency in the management of heart failure as well as provide the platform to implement evidence based preventive and treatment strategies. also this registry substantially improve our understanding and care of heart failure, improved cases they were stop qat and treated under guideline.

Keywords: Heart failure, Registry, Dhamar Governorate, Yemen.

INTRODUCTION

Background

Heart failure is a complex syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the heart to function as a pump to support a physiological circulation. Worldwide, the incidence of heart failure is variable but increases with advancing age. For example, in Scotland, the prevalence of heart failure is high at 7.1 in 1000, increasing with age to 90.1 in 1000 among people over 85years. In the UK, overall incidence is about 2 in 1000. Approximately 26 million people worldwide have heart failure. The prognosis of heart failure has improved over the past 10 years with evidence-based therapy, but the mortality rate remains high and approximately 50% of patients are dead at 5years. Heart failure accounts for 5% of admissions to hospital medical wards. The cost of managing heart failure in the UK exceeds £1 billion per year. Coronary artery disease is the most common cause of heart failure in Western countries [1].

Study Justification

Although heart failure is popular in Dhamar governorate, Yemen there are no previous researches about heart failure, so this research will provide information to the researchers and the readers about malpractice, and risk factor related to heart failure disease.

Objectives

1. To register the Heart Failure at Hospitals ofDhamar City, Yemen.
2. This study will assessed the treatment strategies for the following endpoints:

    a. Percent of Heart Failure patients who will receive guideline.
    b. Percent of Heart Failure patients who will receive CRT.
    c. Percent of Heart Failure patients who will receive ICD.
    d. Percent of Heart Failure patients who will undergo coronary angiography.

3. Episodes of hypotension; defined as SBP < 100 mmHg.
4. Incidence of bleeding.
5. Adverse clinical outcome; defined as the composite of:

    a. Cardiogenic shock.
    b. Mortality.
    c. Arrhythmias.
    d. Stroke (whether ischemic or hemorrhagic).

REVIEW OF LITERATURE

Heart failure is a complex syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the heart to function as a pump to support a physiological circulation [1]. Heart failure develops when the heart cannot maintain adequate output, or can do so only at the expense of elevated ventricular filling pressure [2]. In mild to moderate forms of heart failure, symptoms occur only when the metabolic demand increases during exercise or some other form of stress, in severe heart failure, symptoms may be present at rest. In clinical practice, heart failure may be diagnosed when a patient with significant heart disease develops the signs or symptoms of a low cardiac output, pulmonary congestion or systemic venous congestion at rest or on exercise.

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Worldwide, the incidence of heart failure is variable but increases with advancing age. The prognosis of heart failure has improved over the past 10 Years with evidence-based therapy, but the mortality rate remains high and approximately 50% of patients are dead at 5years. Heart failure accounts for 5% of admissions to hospital medical wards [1].

Pathophysiology

When the heart fails, considerable changes affect the heart and peripheral vascular system in response to the hemodynamic changes associated with heart failure; these physiological changes are compensatory and maintain cardiac output and peripheral perfusion. However, as heart failure progresses, these mechanisms are overwhelmed and become pathophysiological. The development of pathological peripheral vasoconstriction and sodium retention in heart failure by activation of the reninangiotensin-aldosterone system entails a loss of beneficial compensatory mechanisms and represents cardiac decompensation. Factors involved are venous return, outflow resistance, contractility of the myocardium, and salt and water retention.

Venous Return (Preload): In the intact heart, myocardial failure leads to a reduction of the volume of blood ejected with each heartbeat, and an increase in the volume of blood remaining after systole. This increased diastolic volume stretches the myocardial fibers and, as Starling's law of the heart would suggest, myocardial contraction is restored. However, the failing myocardium results in depression of the ventricular function curve (cardiac output plotted against the ventricular diastolic volume). Mild myocardial depression is not associated with a reduction in cardiac output because it is maintained by an increase in venous pressure (and hence diastolic volume). However, the proportion of blood ejected with each heartbeat (ejection fraction) is reduced early in heart failure. Sinus tachycardia also ensures that any reduction of stroke volume is compensated for by the increase in heart rate; cardiac output (stroke volume heart rate) is therefore maintained. When there is more severe myocardial dysfunction, cardiac output can be maintained only by a large increase in venous pressure and/or marked sinus tachycardia. The increased venous pressure contributes to the development of dyspnea, owing to the accumulation of interstitial and alveolar fluid, and to the occurrence of hepatic enlargement, ascites and dependent oedema due to increased systemic venous pressure. However, the cardiac output at rest may not be much depressed, but myocardial and haemodynamic reserve is so compromised that a normal increase in cardiac output cannot be produced by exercise. In very severe heart failure, the cardiac output at rest is depressed, despite high venous pressures. The inadequate cardiac output is redistributed to maintain perfusion of vital organs, such as the heart, brain and kidneys, at the expense of the skin and muscle.

Outflow Resistance (Afterload): Outflow resistance (afterload) is the load or resistance against which the ventricle contracts. It is formed by:

- Pulmonary and systemic resistance
- Physical characteristics of the vessel walls
- The volume of blood that is ejected. An increase in afterload decreases the cardiac output. This results in a further increase of end-diastolic volume and dilatation

Of the ventricle itself, which further exacerbates the problem of afterload. This is expressed by Laplace's law: the tension of the myocardium (T) is proportional to the intraventricular pressure (P) multiplied by the radius of the ventricular chamber (R) - that is, T=PR.

Myocardial Contractility (Inotropic State): The state of the myocardium also influences performance. The sympathetic nervous system is activated in heart failure via baroreceptors as an early compensatory mechanism, which provides inotropic support and maintains cardiac output. Chronic sympathetic activation, however, has deleterious effects by further increasing neurohormonal activation and myocyte apoptosis. This is compensated by a down regulation ofBeta-receptors. Increased contractility (positive inotropism) can result from increased sympathetic drive, and this is a normal part of the Frank-Starling relationship. Conversely, myocardial depressants (e.g. hypoxia) decrease myocardial contractility (negative inotropism).

Neurohormonal and Sympathetic System Activation:

Salt and Water Retention

The increase in venous pressure that occurs when the ventricles fail leads to retention of salt and water and their accumulation in the interstitium, producing many of the physical signs of heart failure. Reduced cardiac output also leads to diminished renal perfusion, activating the renin-angiotensin system and enhancing salt and water retention, which further increases venous pressure. The retention of sodium is, in part, compensated by the action of circulating atrial natriuretic peptides and antidiuretic hormone. The interaction of haemodynamic and neurohumoral factors in the progression of heart failure remains unclear. Increased ventricular wall stress promotes ventricular dilatation and further worsens contractile efficiency. In addition, prolonged activation of the sympathetic nervous and renin-angiotensin-aldosterone systems exerts direct toxic effects on myocardial cells.

Myocardial Remodeling in Heart Failure: Left ventricular remodeling is a process of progressive alteration of ventricular size, shape and function owing to the influence of mechanical, neurohumoral and possibly genetic factors in several clinical conditions, including myocardial infarction, cardiomyopathy, hypertension and valvular heart disease. Its hallmarks include hypertrophy, loss of myocytes and increased interstitial fibrosis. Remodelling continues for months after the initial insult, and the eventual change in the shape of the ventricle becomes responsible for significant impairment of overall function of the heart as a pump. In cardiomyopathy, the process of progressive ventricular dilatation or hypertrophy takes place without ischaemic myocardial injury or infarction.

Changes in Myocardial Gene Expression: Haemodynamic overload of the ventricle stimulates changes in cardiac contractile protein gene expression. The overall effect is to increase protein synthesis, but many proteins also switch to fetal and neonatal isoforms. Human myosin is composed of a pair of heavy chains and two pairs of light chains. Myosin heavy chains (MHCs) exist in two isoforms, a and p, that have different contractile properties and ATPase activity; acx-MHC predominates in the atria and PP-MHC in the ventricles. In

animal models, pressure overload results in a shift from acx-to PP-MHC in the atria, in parallel with Atrial size.
This results in a reduction in atrial contractility but lower energy demands. This shift is less significant in the human ventricle, as the PP-MHC isoform already predominates. Other genes affected in heart failure include those encoding Na+; K+-ATPase, Ca2

+-ATPase and Beta1adrenoceptors.

Abnormal Calcium Homeostasis: Calcium ion flux within myocytes plays a pivotal role in the regulation of contractile function. Excitation of the myocyte cell membrane causes the rapid entry of calcium into myocytes from the extracellular space via calcium channels. This triggers the release of intracellular calcium from the sarcoplasmic reticulum and initiates contraction. Relaxation results from the uptake and storage of calcium by the sarcoplasmic reticulum~, controlled by changes in nitric oxide. In heart failure, there is a prolongation of the calcium current in association with prolongation of contraction and relaxation.

Apoptosis: Apoptosis (or 'programmed cell death') of myocytes has been demonstrated in animal models of ischaemic reperfusion, rapid ventricular pacing, mechanical stretch and pressure overload. Apoptosis is associated with irreversible congestive heart failure, and the spiral of ventricular dysfunction, characteristic of heart failure, results from the initiation of apoptosis by cytokines, free radicals and other triggers.

Natriuretic Peptides (ANP, BNP and C-type):

- Atrial natriuretic peptide (/tNP) is released from atrial myocytes in response to stretch. ANP induces diuresis, natriuresis, vasodilatation and suppression of the reninangiotensin system. Levels of circulating ANP are increased in congestive cardiac failure and correlate with functional class, prognosis and haemodynamic state.
- Brain natriuretic peptide (BNP) (so called because it was first discovered in the brain) is predominantly secreted by the ventricles in response to increased myocardial wall stress.
- N-terminal (NT)-proBNP is an inactive protein that is cleaved from proBNP to release BNP. Both BNP and NT-proBNP are increased in patients with heart failure, and levels correlate with ventricular wall stress and the severity of heart failure.
- BNP and NT-proBNP are good predictors of cardiovascular events and mortality, and there is increasing interest in monitoring levels to help guide heart failure therapy.
- C-type peptide, which is limited to vascular endothelium and the central nervous system, has similar effects to those of ANP and BNP.

Endothelial Function in Heart Failure: The endothelium has a central role in the regulation of vasomotor tone. In patients with heart failure, endothelium-dependent vasodilatation in peripheral blood vessels is impaired and may be one Mechanism of exercise limitation. The cause of abnormal endothelial responsiveness relates to abnormal release of both nitric oxide and vasoconstrictor substances, such as endothelin (ET). The activity of nitric oxide, a potent vasodilator, is blunted in heart failure. ET secretion from a variety of tissues is stimulated by many factors, including hypoxia, catecholamines and angiotensin II. The plasma concentration of ET is elevated in patients with heart failure, and levels correlate with the severity of haemodynamic disturbance. The major source of circulating ET in heart failure is the pulmonary vascular bed. ET has many actions that potentially contribute to the pathophysiology of heart failure: vasoconstriction, sympathetic stimulation, renin-angiotensin system activation and left ventricular hypertrophy. Acute intravenous administration of ET antagonists improves haemodynamic abnormalities in patients with congestive cardiac failure, and oral ET antagonists are being developed. Plasma concentrations of some cytokines, in particular tumour necrosis factor (TNF), are increased in patients with heart failure.

Antidiuretic Hormone (Vasopressin): Antidiuretic hormone (ADH) is raised in severe chronic heart failure, particularly in patients on diuretic treatment. A high ADH concentration precipitates hyponatraemia, which is an ominous prognostic indicator [1].

Classification of heart failure

Table 2.1. Classification of heart failure

New York Heart Association (NYHA)classification of heart failure	
Class	Features
Class I	No limitation. Normal physical exercise does not cause fatigue**,** dyspnea or palpitations
Class II	Mild limitation. Comfortable at rest but normal physical activity produces fatigue, dyspnea or palpitations
Class Ill	Marked limitation. Comfortable at rest but gentle physical activity produces marked symptoms of heart failure
Class IV	Symptoms of heart failure occur at rest and are exacerbated by any physical activity

Types of Heart Failure

Left Heart Failure: This is characterized by a reduction in left ventricular output and an increase in left atrial and pulmonary venous pressure. If left heart failure occurs suddenly – for example, as the result of an acute MI – the rapid increase in left atrial pressure causes pulmonary oedema. If the rise in atrial pressure is more gradual, as occurs with mitral stenosis, there is reflex pulmonary vasoconstriction, which protects the patient from pulmonary oedema. However, the resulting increase in pulmonary vascular resistance causes pulmonary hypertension, which in turn impairs right ventricular function.

Right Heart Failure: This is characterized by a reduction in right ventricular output and an increase in right atrial and systemic venous pressure. The most common causes are chronic lung disease, pulmonary embolism and pulmonary valvular stenosis. The term 'cor pulmonale' is used to describe right heart failure that is secondary to chronic lung disease.

Biventricular Heart Failure: In biventricular failure, both sides of the heart are affected. This may occur because the disease process, such as dilated cardiomyopathy or ischaemic heart disease, affects both ventricles or because disease of the left heart leads to chronic elevation of the left atrial pressure, pulmonary hypertension and right heart failure [2].

Causes of Heart Failure

There are many causes of heart failure that can present suddenly, with acute heart failure (AHF), or more insidiously, with chronic heart failure (CHF).

Left ventricular systolic dysfunction (LVSD) or heart failure and a reduced (R) ejection fraction (HFREF) is commonly caused by ischaemic heart disease but can also occur with valvular heart disease and hypertension.

Diastolic heart failure is a syndrome consisting of symptoms and signs of heart failure with preserved (PJ left ventricular ejection fraction (HFPEF) >45-50%. There is increased stiffness in the ventricular wall and decreased left ventricular compliance, leading to impairment of diastolic ventricular filling and hence decreased cardiac output. Echocardiography may demonstrate an increase in left ventricular wall thickness, increased left atrial size and abnormal left ventricular relaxation with normal or near-normal left ventricular volume. Diastolic heart failure is more common in elderly hypertensive patients but may occur with primary cardiomyopathies (hypertrophic, restrictive, and infiltrative).

Right ventricular systolic dysfunction (RVSD) may be secondary to chronic LVSD but can occur with primary and secondary pulmonary hypertension, right ventricular infarction, arrhythmogenic right ventricular cardiomyopathy and adult congenital heart disease

Main Causes:

- lschaemic heart disease (35-40%).
- Cardiomyopathy (dilated) (30-34%).
- Hypertension (15-20%).

Other Causes:

- Cardiomyopathy (undilated): hypertrophic, restrictive (amyloidosis, sarcoidosis).
- Valvular heart disease (mitral, aortic, tricuspid).
- Congenital heart disease (ASD, VSD).
- Alcohol and drugs (chemotherapy - trastuzumab, imatinib).
- Hyperdynamic circulation (anaemia, thyrotoxicosis, haemochromatosis, Paget's disease).
- Right heart failure (right ventricular infarct, pulmonary hypertension, pulmonary embolism, COPD).
- Tricuspid incompetence.
- Arrhythmias (atrial fibrillation, bradycardia (complete heart block, sick sinus syndrome).
- Pericardial disease (constrictive pericarditis, pericardia! effusion).
- Infections (Chagas' disease), e.g. myocarditis.

Clinical Features of Heart Failure

***Symptoms*:**

- Exertional Dyspnea.
- Orthopnea.
- Paroxysmal Nocturnal Dyspnea.
- Fatigue.

***Signs*:**

- Tachycardia.
- Elevated Jugular Venous Pressure.
- Cardiomegaly.
- Third and Fourth Heart Sounds.
- Bi-basal Crackles.
- Pleural Effusion.
- Peripheral Ankle Oedema.
- Ascites.
- Tender Hepatomegaly.

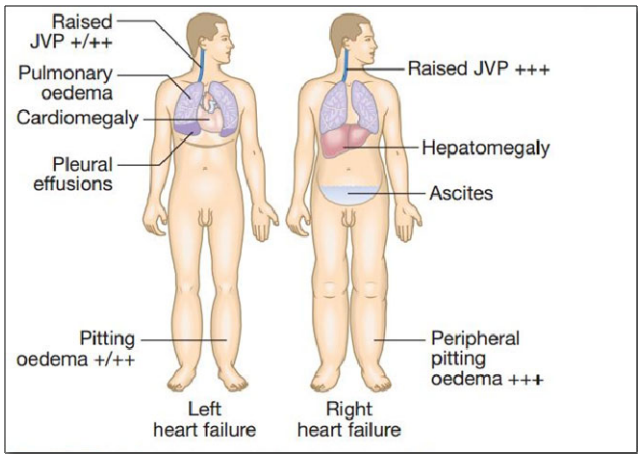

Figure 2.1 Clinical Features of Left & Right Heart Failure

Complications of Heart Failure

- Renal failure is caused by poor renal perfusion due to low cardiac output and may be exacerbated by diuretic therapy, ACE inhibitors and angiotensin receptor blockers (ARBs).
- Hypokalaemia may be the result of treatment with potassium-losing diuretics or hyperaldosteronism caused by activation of the renin–angiotensin system and impaired aldosterone metabolism due to hepatic congestion. Most of the body's potassium is intracellular and there may be substantial depletion of potassium stores, even when the plasma concentration is in the reference range.
- Hyperkalaemia may be due to the effects of drugs that promote renal resorption of potassium, in particular the combination of ACE inhibitors, ARBs and mineralocorticoid receptor antagonists. These effects are amplified if there is renal dysfunction due to low cardiac output or atherosclerotic renal vascular disease.
- Hyponatraemia is a feature of severe heart failure and is a poor prognostic sign. It may be caused by diuretic therapy, inappropriate water retention due to high vasopressin secretion, or failure of the cell membrane ion pump.
- Impaired Liver Function is caused by hepatic venous congestion and poor arterial perfusion, which frequently cause mild jaundice and abnormal liver function tests; reduced synthesis of clotting factors can make anticoagulant control difficult.
- Thromboembolism. Deep vein thrombosis and pulmonary embolism may occur due to the effects of a low cardiac output and enforced immobility. Systemic emboli occur in patients with atrial fibrillation or flutter, or with intracardiac thrombus complicating conditions such as mitral stenosis, MI or left ventricular aneurysm.
- Atrial and Ventricular Arrhythmias are very common and may be related to electrolyte changes such as hypokalaemia and hypomagnesaemia, the underlying cardiac disease, and the pro-arrhythmic effects of sympathetic activation. Atrial fibrillation occurs in approximately 20% of patients with heart failure and causes further impairment of cardiac function. Ventricular ectopic beats and runs of non-sustained ventricular tachycardia are common findings in patients with heart failure and are associated with an adverse prognosis.

- Sudden Death occurs in up to 50% of patients with heart failure and is most probably due to ventricular fibrillation [2].

Investigations in Heart Failure

- Blood tests. Full blood count, urea and electrolytes, liver biochemistry, cardiac enzymes in acute heart failure, BNP or NT-pro BNP, and thyroid function should be tested.
- Chest X-ray.
- Electrocardiogram.
- Echocardiography.
- Stress echocardiography.
- Nuclear cardiology.
- Cardiac MRI (CMR).
- Cardiac catheterization.
- Cardiac biopsy.
- Cardiopulmonary exercise testing.
- Ambulatory 24-hour ECG monitoring (Holter)[2].

Management of heart failure

Management of acute heart failure: Acute heart failure with pulmonary oedema is a medical emergency that should be treated urgently. The patient should initially be kept rested, with continuous monitoring of cardiac rhythm, BP and pulse oximetry. Intravenous opiates can be of value in distressed patients but must be used sparingly, as they may cause respiratory depression and exacerbation of hypoxaemia and hypercapnia. If these measures prove ineffective, inotropic agents such as dobutamine (2.5–10 μg/kg/min) may be required to augment cardiac output, particularly in hypotensive patients. Insertion of an intra-aortic balloon pump may be beneficial in patients with acute cardiogenic pulmonary edema and shock.

Management of chronic heart failure: The aims of treatment in chronic heart failure are to improve cardiac function by increasing contractility, optimizing preload or decreasing afterload, and controlling cardiac rate and rhythm. This can be achieved by a combination of drug treatment or non-drug treatments, as discussed below.

Education

Drug treatment

Drugs that reduce preload are appropriate in patients with high end-diastolic filling pressures and evidence of pulmonary or systemic venous congestion, whereas those that reduce afterload or increase myocardial contractility are more useful in patients with signs and symptoms of a low cardiac output.

- Diuretics
- Angiotensin-converting enzyme inhibitors ACE inhibitors
- Angiotensin receptor blockers ARBs
- Neprilysin inhibitors
- Vasodilators
- Beta-adrenoceptor blockers
- Ivabradine
- Digoxin
- Amiodarone

Non-pharmacological treatments:

- Implantable cardiac defibrillators.
- Resynchronisation devices
- Cardiac transplantation.
- Ventricular assist devices (VAD) [2].

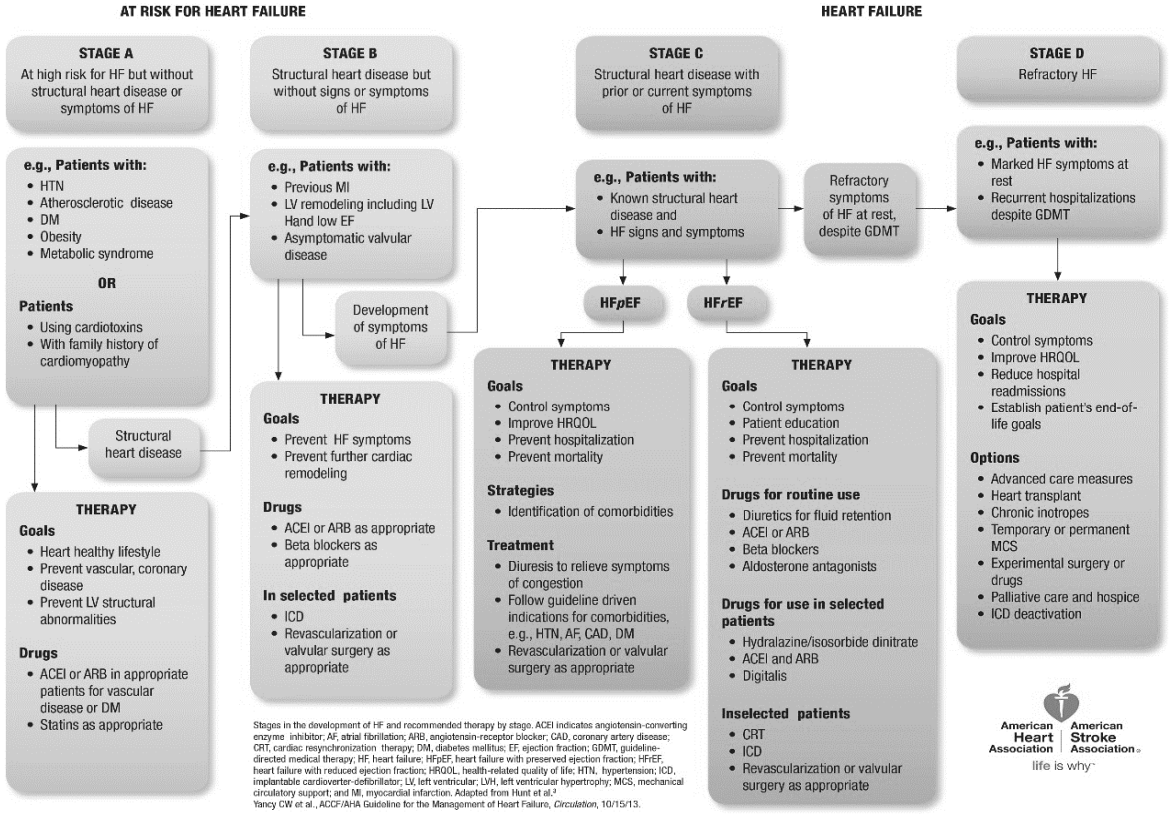

Figure 2.2. Management of heart failure

PATIENTS AND METHODS

Study Area

The present study was carried out in Dhamar governorate, Yemen which located about 100 kilometers south of the capital city of Yemen , Sana'a and its population1603000 N in 2011 . It has a total area of 7586 Km^2. The climate is cold in winter and moderate in summer.

Study Population

This study included 120 patients of Heart Failure in several Stage.

Study Design

This is a prospective study that will evaluate all the patients with Heart Failure and who are admitted at internal medicine department at Hospital from 15/ February/ 2019 to 15 / July / 2019.

Inclusion criteria for this study are

All patients with in the term of Heart Failure.

Exclusion criteria for this study are

Not exclude any cases of heart failure.

Methodology

All patients will subject to the following:

1. Clinical evaluation: It includes medical history evaluation that concentrate on following issues:

A. Personal and present history:

- Demographic Data: age, gender of the patient and body mass index.
- Chest pain, duration, severity and its functional capacity.
- Dyspnea and its functional class (NYHA class).
- Risk factors for Heart Failure.
- Qat chewing and alcohol abuser.
- Symptoms and complications during the hospital course.
- History of Ischemic Heart Disease as defined by any 1 of the following 6 criteria:

a. History of Angina.
b. Prior MI.
c. Prior Documentation of Cardiac Ischemia on exercise stress test.
d. Prior Coronary Angiographic showing stenosis > 70%.
e. Prior Coronary Revascularization (PCI or CABG).
f. an ECG with pathologic Q waves in 2 contiguous leads.

B. Drug History.
C. Family History.
D. Comorbidities.

- Diabetes Mellitus: defined as fasting blood glucose ≥126 mg/dl and HbA1C ≥6.5
- Hypertension: defined as blood pressure ≥140/90 mm Hg.
- Renal Insuffiency: defined as serum creatinine > 2mg/dl or a creatine clearance of< 60mL/min.
- History of Cerebrovascular Diseases defined as ischemic or hemorrhagic stroke or TIAs.
- Obesity (defined as BMI > 30 kg/m2).
- COPD.
- Bronchial Asthma (defined as a clinical diagnosis of asthma and use of regular inhaled steroids or B2 agonists at least once per week over the period of 1 month, any time in the last 10 y).
- Anemia.
- Limited Exercise Capacity, defined as the inability to perform most normal daily activities.

E. Clinical examination:

- The heart rate, rhythm and blood pressure will be estimated.
- Body weight, height will be measured.
- The body mass index will be calculated.
- Sings of heart failure.

2. Baseline electrocardiography at presentation, and its serial follow up (ECG).

3. Results of Labs and non-invasive testing

- Cardiac Enzymes; troponin and CKMB.
- Echocardiography (assessment of global and regional systolic functions).

4. Results of invasive testing Coronary angiography if it will be performed according to recent guideline recommendations or not.

I. Treatment Strategies

A. Medical treatment per guideline

The patients in this group will be evaluated based upon the most recent recommendation of treatment strategy for heart failure.

B. Devices

The patients in this group will be evaluated based upon the most recent recommendation of treatment strategy for HF including CRT and ICD.

In these strategies, the strategy will be evaluated and flowed during the hospital course.

Management Data

The following data will be recorded in the case report forms:

1. Type of medical therapy for HF.
2. Other drug therapy, including anticoagulation, statins antiplatelet agents, others.

An ECG will be recorded on the first presentation and during hospitalization. Additional measurements may be performed at the discretion of the attending physician according to clinical status.

Hospital Follow up

All patients will be followed up during hospital admission for the occurrence of one of the following end points: bleeding, arrhythmia, cardiogenic shock, cardiac arrest, death, stroke, or renal impairment.

Endpoints

1. This study will be assessed the treatment strategies for the following endpoints:

a. Percent of Heart Failure patients who will receive guideline.
b. Percent of Heart Failure patients who will receive CRT .
c. Percent of Heart Failure patients who will receive ICD.
d. Percent of Heart Failure patients who will undergo coronary angiography.

2. Episodes of Hypotension; defined as SBP < 100 mmHg.
3. Incidence of Bleeding.
4. Adverse Clinical Outcome; defined as the composite of:

a. Cardiogenic Shock.
b. Mortality.
c. Arrhythmias.
d. Stroke (whether ischemic or hemorrhagic).

Statistical Analysis

By SPSS version15 Continuous variables will be presented as mean + standard deviation unless otherwise mentioned and will be compared using the Students t-test. Differences between treatment groups for the categorical variables will be analyzed using the chi-square test. For the purposes of this study, P values < 0.05 will be considered significant.

RESULTS

Our study population consisted of 120 patients with Heart Failure were attended the Internal medicine department, 67 were males, and 53 were females. Most of the patients were between 24 -85 years of age and mean age was (58.45) years.

- Most affected age by heart failure 45- 64y 48.3%, Most affected sex male as 55.8%, Major risk factor Qat chewing 81.7%, Hypertension 52.5%, Family history 42.5%, Smoker 41.7% , Ischemic heart diseases 33.3%, Diabetes mellitus 33.3%.

Table 4.1 General characteristics of study population

Characterize	Frequency	Percentage
Age		
25-44Y	22	18.3
45-64Y	58	48.3
65-85Y	40	33.3
SEX		
Male	67	55.8
Female	53	44.2
BMI		
15-24Kg/m2	85	70.8
25-35Kg/m2	35	29.2
Ischemic Heart Disease	40	33.3
Hypertension	63	52.5
Diabetes mellitus	40	33.3
Smoker	50	41.7
Hyperlipidemia	21	17.5
Family history	51	42.5
Qat chewing	98	81.7

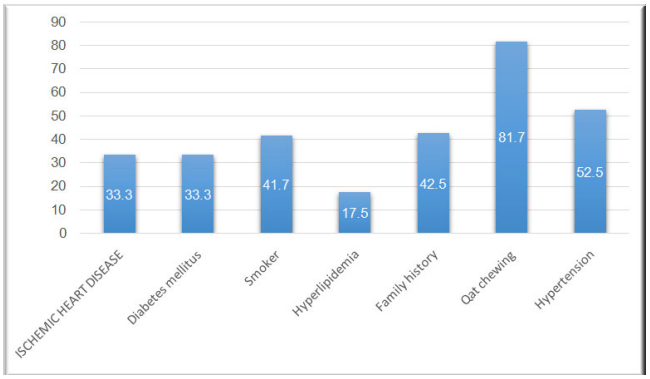

Figure 4.1. Percentage of risk factor in Heart Failure

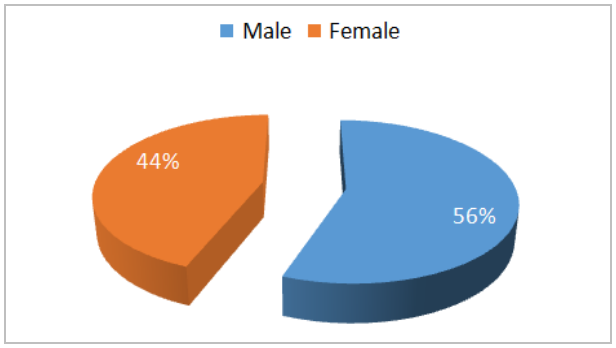

Figure 4.2. Percentage of Heart Failure male and female

- Patient reach to hospital in (NYHA Grade IV) 69.5%, with poor capacity and pulmonary edema. Chest pain not significant in cases of heart failure except when causes ACS 13.3% most of them prior MI percentage 77%. Small percentage of COPD 10.8%, asthma 6.7%, anemia 7.5% as causes of Heart Failure shows in Table 4.2.

Table 4.2 Clinical Feature of Heart Failure

Characterize	Frequency	%
Dyspnea	113	94.2
Degree of Dyspnea		
I	5	42
II	12	10.2
III	19	16.4
IV	82	69.5
Chest Pain	56	46.7
Degree of Chest Pain		
I	23	25
II	35	38
III	9	9.8
IV	25	27.2
Activity Capacity		
Good	32	26.9
Poor	87	73.1
Compensation of Heart Failure		
Decompensate	100	83.3
Compensated	20	16.7
NYHA Classification		
I	4	34
II	19	16.2
III	13	11.1
IV	81	69.2
History of Coronary Artery Disease Angina	16	13.3
History of CAD prior myocardial infarction		
Angina	20	23
Prior MI	67	77
Pulmonary edema	85	70.8
Bronchial asthma	8	6.7
COPD	13	10.8
Anemia	9	7.5

- About 15% with creatinine > 2mg/dl, Near total 85% of cases without Lipid profile, 80% without FBS, 79.2% without HbA1C, 67.5%without INR, 27.5% without echo., 46.7% Low EF, 40.8% Mitral regurage, 93.3% of cases without coronary angiography and 98.3% without PCI shows in Table 4.3 .

Table 4.3 Investigations of Heart Failure

Characterize	Frequency	%
Serum Creatinine		
<2mg/dl	100	83.3
>2mg/dl	18	15
Not found	2	1.7
Total Cholesterol		
High	11	9.2
Normal	7	5.8
Not found	102	85
Low Density Lipoprotein		
High	7	5.8
Normal	3	2.5
Not found	110	91.7
High Density Lipoprotein		
High	5	4.2
Normal	5	4.2
Low	110	91.7
Not found		
Triglyceride		
High	4	3.3
Normal	5	4.2
FBS		
High	15	12.5
Normal	9	7.5
Not found	96	80

Cont'd Table 4.3 Investigations of Heart Failure

Characterize	Frequency	%
Random Blood Sugar		
High	97	39.2
Normal	60	50
Not found	13	10.8
HemoglobineA1C		
<6	5	4.2
>6	20	16.7
Not found	95	79.2
Hemoglobin		
High	8	6.7
Normal	99	82.5
Low	13	10.8
Platelet		
High	1	8
Normal	115	95.8
Low	4	3.3
International Normalized Ratio		
High	9	7.5
Normal	30	25
Not found	81	67.5
ECG sinus		
Yes	99	82.5
No	21	17.5
ECG ST elevation		
Yes	32	26.7
No	87	72.5
ECG QRS Amplitude		
Wide	32	26.7
Narrow	88	73.3
Acute Coronary Syndrome		
STEMI	24	20

Table 4.3 Investigations of Heart FailureCont'd

Characterize	Frequency	%
NSTEMI	8	6.7
UA	8	6.7
Not found	80	66.7
Echocardiography		
Found	87	72.5
Not found	33	27.5
Ejection Fraction		
Preserved	31	25.8
Reduced	56	46.7
Mitral Regurgitation		
Present	49	40.8
Not present	39	32.5
Coronary Angiography		
Yes	8	6.7
No	112	93.3

- Most of cases not use guideline treatment of heart failure, Loop diuretic 95%, Beta-blocker 58.3%, ACEI 56.7%, Aldactone 65.8%, Aspirin80.8%, Anticoagulant 48.3% and Statin53.3%, about 1.7% of cases referred and did coronary resynchronization therapy, about 3.3% implantable defibrillator; about 2.5% received Blood transfusion shows in Table 4.4 .

Table 4.4 Management of Heart Failure

Characterize	frequency	%
Guideline	59	49.2
Loop diuretic	114	95
Thiazide	3	2.5
Beta blocker	70	58.3
Angiotensin converting enzyme inhibitors	68	56.7
Angiotensin receptors blocker	9	7.5
Aldactone	79	65.8
Aspirin	97	80.8
Anticoagulant	58	48.3
Statin	64	53.3
Need for blood transfusion	3	2.5

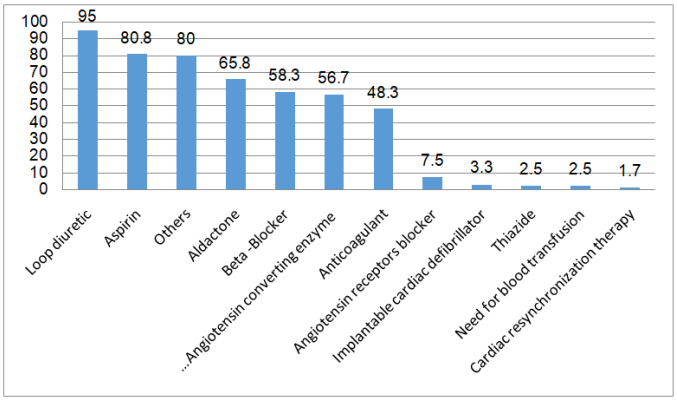

Figure 4.3 Percentage of Management of Heart Failure

- Hypotension patient bout 11.7%, Bleeding 11.7%, cardiogenic shock 2.5%, Stroke 3.3%, renal impairment 5%, about 52.5% of cases improved and 56.7% stopped Qat chewingshows in Table 4.5.

Table 4.5 Follow up of patient

Characterize	Frequency	%
Hypotension	14	11.7
Pulmonary Edema	44	36.7
Bleeding	14	11.7
Cardiogenic Shock	3	2.5
Stroke	4	3.3
Renal Failure	6	5
Death	12	10
Improvement	63	52.5
Qat chewing stop	68	56.7

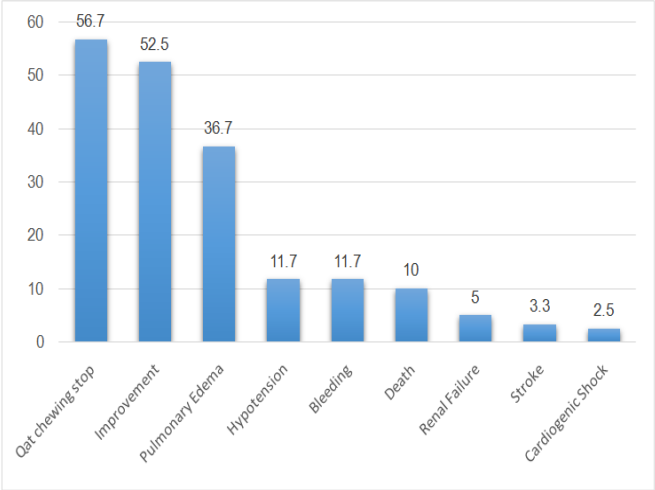

Figure 4.4. Follow up of patient

- About 52.5 %(63/120) of cases improved.79.4% stop chewing qat, 63.5%were under proper guideline treatment shows in Table 4.6.

Table 4.6. Improvement predictors

	No.	%	X2	P.V
Guideline			10.890	0.001
Found	40	63.5		
Not found	23	36.5		
Qat stop			27.828	0.000
Yes	50	79.4		
No	13	20.6		
Age			0.283	0.868
25-44	12	19		
45-64	29	46		
65-85	22	35		
Sex			1.366	0.242
Male	32	50.8		
Female	31	49.2		

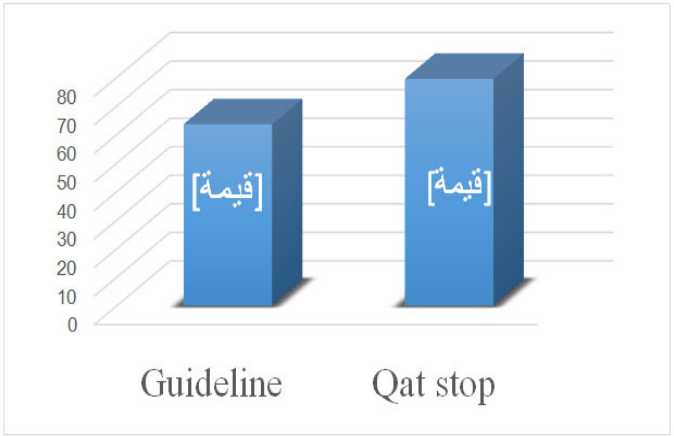

Figure 4.5 Improvement predictors

- 10 %(12/120) of case were died. Major risk factor related to death qat chewing 91.7%, HTN58.3%, IHD and smoking 41.7%, DM33.3%, Major risks factor of death was continue chewing qat 83.3% followed by not follow guideline shows in Table 4.7 .

Table 4.7. Death predictor factors

	No.	%	X2	P.V
Guideline			1.337	0.247
Found	4	33.3		
Not found	8	66.7		
Qat stop			8.688	0.000
Yes	2	16.7		
No	10	83.3		
Risk Factors				
Age			3.372	0.185
45-64	8	66.7		
65-85	4	33.3		
Sex			0.034	0.854
Male	7	58.3		
Female	5	41.7		
BMI			1.008	0.315
15-24	7	58.3		
25-35	5	41.7		
IHD	5	41.7	0.417	0.519
HTN	7	58.3	0.182	0.670
DM	4	33.3	0	1
Smoker	5	41.7	0	1
Hyperlipidemia	3	25	0.519	0.471

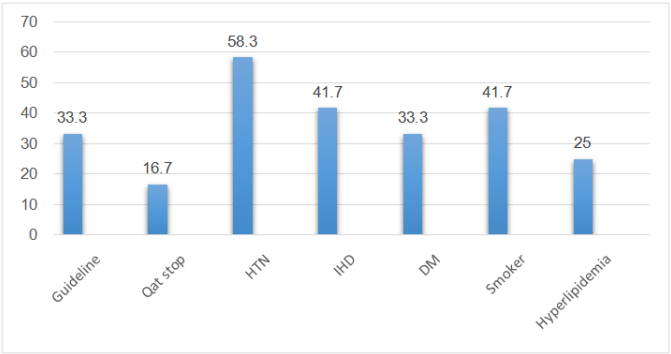

Figure 4.6. Death predictor factors

DISCUSSION

Our study about heart failure registry for 120 cases (male, female),age group 24-85years old, among duration from 15th February to 15th July 2019 in Dhamar governorate, Yemen, Several researches have been done and coming up for recommendation that heart failure still problem to people. Heart failure significantly affect more people who are habitually chewing qat and/or have other risk factor such as HTN, IHD, DM and smoking, According to our knowledge, this first study in Dhamar governorate to evaluate the prevalence of heart failure in Dhamar governorate, Yemen. Incidence of major risk factor in our study Qat chewing 81.7%, HTN 52.5%, IHD 33.3% and DM 33.3%, When comparing to study in Saudi Arabia was for acute decompensated heart failure showed the major risk factors are diabetes 64.1%, hypertension 70.6% and acute coronary syndrome[3].also study was done India reported Trivandrum heart failure registry IHD72% [4]. Qat is an important risk factor due to its toxic substance causing toxic myocarditis and causing tachycardia, increase blood pressure. The psychoactive compounds cathinone and cathine are phenylalkyleamine present in qat which are structurally related to amphetamine and are responsible for most of the effect of qat [5]. Few attempts were made to assess the cause and effect relationship of qat with cardiovascular diseases. A study conducted in

Yemen reported that the increase in blood pressure and heart rate in qat chewers coincided with raised plasma chathinone concentrations [6]. Accordingly, about 59% of qat chewers had onset of symptoms of AMI during the qat effective period and only 36.4% of non-qat chewers had a new onset of AMI symptoms [6]. A review showed that regular qat chewing was associated with elevated mean DPB [7]. According to the registry, most cases were presented with dyspnea 94.2% about 69.5% of them were grade IV, 70.8%pulmonary edema, 46.7% chest pain. About 15% with creatinine > 2mg/dl, near total 85% of cases without Lipid profile, 80% without FBS, 79.2% without HbA1C, 67.5%without INR, We were unable to include echocardiography investigation in only 72.5% of patients with 46.7% reduced EF, 40.8% Mitral regurage. Our research is unique in reporting cases of heart failure who received guidelines treatment 49.2% as loop diuretics (95 %) beta-blocker (58.3 %) ACEI (56.7 %) ARBs (7.5 %) Aldactone (65.8 %). Unfortunately form all cases we met there is no case received either CRT or ICD.

Adverse clinical outcome defined as composite of cardiogenic shock 2.5%, stroke 3.3%, mortality rate 10% was low in comparison with other studies were done in Saudi Arabia 24.3%[3], this percent is not real and does not reflect the quality of our hospitals but we find trouble when contacting with most of the patients when left hospital. Most cause of death were continuous of risk factors as qat chewing 91.7%, HTN58.3%, IHD 41.7%.most of cases were male gender58.3% are more likely to stressful life condition, about 66.7% of death were not under guideline treatment and 83.3% were continued on chewing qat especially when left hospital. Present study patient with heart failure are different and respond differently to therapy according to different schools, protocols of hospitals and ability of patients to continuous their medications and follow up, Improved cases accounting 52.5% among of these was79.4% who stop chewing qat,63.5% under guideline treatment that means qat chewing big problem and avoiding it lead to improvement. People need access to learn about major risk factor of heart failure to be avoided and responsibility in doctors who deal with patients to decrease this problem. Additionally the study result could have been affected by unmeasured confounding variables, such as socioeconomic strata, patient preferences and post hospital care.

Conclusion and Recommendations

Heart failure registry is intending to provide unique insight into demographic, etiology, clinical presentation and outcomes (both long and short term) of heart failure in Dhamar governorate. The validated data obtained from this registry will help the local clinician to identify the deficiency in the management of heart failure as well as provide the platform to implement evidence based preventive and treatment strategies, in order to developing the protocol and perform suited to our region and to identify the most important data to be captured to reduce the burden of heart failure also this registry substantially improve our understanding and care of heart failure. This study reached that most patients with Heart Failure were chewing qat which is first risk factor related with morbidity and mortality other problem with patient related to improper following guideline when treat such cases because they did not go to cardiologist at first time and treated by general practitioner and they came lately with complication ,also patient do not take medication as prescribed and not avoid risk factors.

Health professionals should also play a role in promoting the health impacts of qat and provide psychosocial support services to quit the qat chewing habit for those who are affected chronically. The global community should also work together to reduce or halt the rate of border crossing qat trade. Generally, clear policies should be designed and implemented to curb qat chewing in those countries with the most at risk populations. Improved cases they were stop qat and treated under guideline and they follow the role exactly as their doctors said. Good guidelines treatment, follow up and dealing with cases who referred to cardiologist are very important and resolve heart failure problem.

Heart failure need a nationwide primary prevention and heart failure disease management programs. By improvement the overall quality of care of the patient with heart failure, we achieved great success in optimization of pharmacological therapy. We have tried to summarize the crucial ones below:

1. Patient education about heart failure and strategies of its treatment, dietary counseling about sodium (2-3gm/day and less than 2gm in severe heart failure) and fluid restriction <2L/day, healthy life style changes (high fiber diet, regular exercise in a tolerable amount, no smoking) , understand the alarming signs and symptoms , such as shortness of breath , excessive fatigue , swelling of feet/uncle , etc.
2. Arranging follow up care with documentation of the date time and location of the follow up visit on the discharge plan as well as send for the subsequent appointment.

Specialized referral or health center. The referral to an heart failure program shown result in a decrease in the frequency of hospitalization of nearly 50% [8].

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