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The Prevalence of Chest Infection in Patients with Heart Failure in Al-Wahadah Teaching Hospital, Dhamar, Yemen

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ABSTRACT
Background: Heart failure is a common clinical manifestation of most organic heart diseases that progress to the end stage. Patients with heart failure are often accompanied by secondary conditions such as pulmonary circulatory congestion and pulmonary edema, which can lead to dyspnea, gas exchange disorders, and other consequences, creating certain conditions for pathogens to invade and colonize the lungs. Therefore, patients with heart failure may have higher risk of pulmonary infection.
Aim: This study aimed to assess the prevalence of chest infection among patients with heart failure and to determine the characteristics of the patients with heart failure who had chest infection and the potential risk factors.
Methods: A retrospective, descriptive cross-sectional study was carried out at internal medicine department, Al-Wahdah Teaching Hospital, Dhamar, Yemen over six months (June – December 2022). A total of 100 patients were enrolled in our study. A semi-structured questionnaire was designed and used for data collection.
Results: A total of 100 heart failure patients including 53 (53%) males, and 47 (47%) females were successfully enrolled in this study. The majority of participated patients were of age group 40 – 60 years (55%), and were of rural residence (83%). Chest infection was documented in approximately two-thirds of included patients (63%). Chest infection was documented in majority of patients who were smokers, Qat chewer, shamma intakes, being obese, had cardiomyopathy, as well as those patients who had dyspnea of NYHA class III & IV, orthopnea, PND, productive cough, high sputum amount, whitish sputum color, peripheral chest pain, fatigue, sweating, fever and high WBC count, such associations were significant statistically.
Conclusion: This study revealed that, chest infection in heart failure patients was common with overall prevalence of (63%) among included patients this study was. Based on this, an early prevention and intervention measures should be taken to reduce pulmonary infections in patients with heart failure.

KEY WORD: Chest infection, heart failure, Yemen

CHAPTER I

1.1. Introduction

Heart failure is the leading cause of hospital admission among adults older than 65 years in western countries [1, 2]. The risk of death considerably increased over a prolonged period after a heart failure hospitalization [3]. Several factors have been identified that may precipitate an episode of acute decompensated heart failure (ADHF) leading to hospital admission (i.e. arrhythmia, myocardial ischaemia, infection, uncontrolled hypertension, inadequate preadmission treatment and non-adherence to medications or diet) [4–6]. In large registries, respiratory infection was one of the most prevalent factors precipitating ADHF occurring in 15% to 29% of cases [4,7,8] and was independently associated with in hospital mortality in some analyses [4,7, 8].

In the United States, community-acquired pneumonia affects >5 million adults and is associated with approximately 1.5 million hospital admissions and up to 100,000 deaths annually [9–11]. In the United Kingdom, pneumonia accounts for more hospital admissions and bed-days than any other lung condition, and worldwide, pneumonia is a significant cause of morbidity and mortality, especially in the elderly [12]. Indeed, the aging of many populations is thought to be one reason why hospitalizations for pneumonia have increased by up to 50% in Western countries over the past 2 decades and survival from pneumonia has changed little in half a century [13,14]. Although usually considered to be an acute event, there is evidence that pneumonia is associated with long-term cardiovascular sequelae, particularly acute coronary events, and the same may be true for heart failure (HF), although this has not been investigated fully [9].

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Patients with HF thought to be at 2-fold higher risk of pneumonia than age- and sex-matched individuals in the population, and survival from pneumonia is lower in patients with HF than in those without. Conversely, pneumonia increases the risk of worsening HF and often considered a factor in decompensating leading to hospitalization [14–17]. The most common cause of pneumonia is infection with the bacterium Streptococcus pneumonia [18, 19]. Two pneumococcal vaccines are available and recommended in patients with HF. Influenza also causes pneumonia, and vaccination against influenza is widely available and inexpensive [20]. However, uptake of each of these vaccines is poor and may even be declining [21]. This deficiency in care may be particularly problematic for patients with HF and preserved ejection fraction (HFpEF), who have fewer other treatment options, compared with patients with HF and reduced ejection fraction (HFrEF) [22–24].

Diagnosing pneumonia is often challenging in patients admitted for ADHF as dyspnea and rales are cardinal symptoms for both heart failure and pneumonia [25, 26]. Clinically diagnosing pneumonia becomes even more challenging in elderly patients because respiratory and non-respiratory symptoms less often reported in this subset of patients [27]. Based on current guidelines, the presence of infiltrates demonstrated by chest radiograph or another lung imaging modality is mandatory for the diagnosis of community-acquired pneumonia [28–30]. Moreover, infections other than pneumonia may precipitate ADHF. Together with leukocyte count, C-reactive protein (CRP) is the most commonly used laboratory marker for inflammation and infection. However, it is not specific for bacterial infections and is also elevated in systemic inflammatory states such as ADHF [31].

1.2. Research Justification

1. To the best of researchers knowledge, there is no regional literature about this subject and our study is the first study in Yemen at all, and the data on the frequency of chest infection among Yemeni patients with heart failure are lacking to date.
2. The search is gaining importance over the results of which will be reached and the possibility of dissemination and utilization.

1.3. Objectives

1. To determine the prevalence of chest infection among patients with heart failure.
2. To determine the characteristics of patients with heart failure who had chest infection.
3. To identify potential risk factors for development of chest infection among patients with heart failure.

CHAPTER II
LITERATURE REVIEW

2.1 Congestive heart failure

2.1.1 Introduction

Heart failure is a common disease, affecting approximately 5 million people in the United States, and it occurs predominately in the elderly, with almost 80% of cases occurring in patients over the age of 65 [32]. The magnitude of the problem can not be precisely assessed, because reliable population-based data on the prevalence, incidence, and prognosis are lacking. Nevertheless, several studies have found that CHF is associated with a 2-year mortality rate of approximately 45–50%, which approaches that of many malignancies [33] Moreover, from a societal perspective, caring for patients with CHF accounts for 2–3% of the federal health-care budget. The estimated direct and indirect cost of CHF in the United States in 2005 was $27.9 billion [32].

There are two mechanisms of reduced cardiac output and heart failure: systolic dysfunction and diastolic dysfunction. The most common causes of systolic dysfunction (defined by a left-ventricular ejection fraction of 50%) are ischemic heart disease, idiopathic dilated cardiomyopathy, hypertension, and valvular heart disease. Diastolic dysfunction (defined as dysfunction of left-ventricular filling with preserved systolic function) may occur in up to 40–50% of patients with heart failure, it is more prevalent in women, and it increases in frequency with each decade of life. Diastolic dysfunction can occur in many of the same conditions that lead to systolic dysfunction. The most common causes are hypertension, ischemic heart disease, hypertrophic cardiomyopathy, and restrictive cardiomyopathy. Many patients who have symptoms suggestive of heart failure (shortness of breath, peripheral edema, and paroxysmal nocturnal dyspnea) but also have preserved left ventricular function may not have diastolic dysfunction; instead, their symptoms are caused by other etiologies, such as lung disease, obesity, or occult coronary ischemia [34]

2.1.2 Definition

Congestive heart failure (CHF) is a complex clinical syndrome that can result from any functional or structural cardiac disorder that impairs the ventricle's ability to fill with or eject blood. Since there is no definitive diagnostic test for heart failure, it

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remains a clinical diagnosis that is largely based on a careful history and physical examination and supported by ancillary tests such as chest radiograph, electrocardiogram, and echocardiography [32].

2.1.3 Pathophysiology of Congestive Heart Failure

The syndrome of CHF arises as a consequence of an abnormality in cardiac structure, function, rhythm, or conduction. In developed countries, ventricular dysfunction accounts for the majority of cases and results mainly from myocardial infarction (systolic dysfunction), hypertension (diastolic and systolic dysfunction), or in many cases both. Degenerative valve disease, idiopathic cardiomyopathy, and alcoholic cardiomyopathy are also major causes of heart failure. Heart failure often occurs in elderly patients who have multiple comorbid conditions (eg, angina, hypertension, diabetes, and chronic lung disease). Some common comorbidities such as renal dysfunction are multifactorial (decreased perfusion or volume depletion from overdiuresis), whereas others (eg, anemia, depression, disorders of breathing, and cachexia) are poorly understood [35].

CHF indicates not only an inability of the heart to maintain adequate oxygen delivery; it is also a systemic response attempting to compensate for the inadequacy. The determinants of cardiac output include heart rate and stroke volume. The stroke volume is further determined by the preload (the volume that enters the left ventricle), contractility, and afterload (the impedance of the flow from the left ventricle). These variables are important in under-standing the pathophysiologic consequences of heart failure and the potential treatments. Furthermore, an appreciation of cardiopulmonary interactions is important in our understanding of heart failure. In the simplest terms, the heart can be viewed as a dynamic pump. It is not only dependent on its inherent properties, but also on what is pumped in and what it must pump against. The preload characterizes the volume that the pump is given to send forward, the contractility characterizes the pump, and the afterload determines what the heart must work against.

The preload is often expressed as the end-diastolic pressure/volume of the left ventricle and is clinically assessed by measuring the right atrial pressure. However, the preload is not only dependent on intravascular volume; it is also influenced by any restriction to ventricular filling. Since the heart resides in the thoracic cavity, an increased positive pleural pressure (as seen with dynamic hyperinflation in chronic obstructive pulmonary disease or asthma) can reduce right-atrial pressure (which equals central venous pressure minus pleural pressure) and thus reduce ventricular filling. The cardiac pump is a muscle and will respond to the volume it is given with a determined output. If volume increases, so will the amount pumped out in a normal physiologic state, to a determined plateau; this relationship is described by the Frank-Starling law [36].

A concept that is often poorly understood is the diastolic function of the heart. Diastolic function is determined by 2 factors: the elasticity or distensibility of the left ventricle, which is a passive phenomenon, and the process of myocardial relaxation, which is an active process that requires metabolic energy [37]. Relaxation of the myocardium occurs in early diastole, and the “untwisting” of the left ventricle is an active process that produces a suction effect that augments left-ventricular filling. Loss of normal leftventricular distensibility or relaxation by either structural changes (eg, left-ventricular hypertrophy) or functional changes (eg, ischemia) impairs ventricular filling (preload). The exercise intolerance seen with diastolic dysfunction largely results from the impairment of ventricular filling, which elevates left-atrial pressure and pulmonary venous pressure and causes pulmonary congestion [38]. Additionally, inadequate cardiac output during exercise results in poor perfusion of skeletal muscles, especially the leg muscles and the accessory muscles of respiration [39].

The second variable of stroke volume is cardiac contractility, which represents the muscular pumping of the heart and is commonly expressed as the ejection fraction. Based on autonomic input, the heart will respond to the same preload with different stroke volumes, depending on inherent characteristics of the heart. A heart with normal systolic function will maintain an ejection fraction of over 50–55%. A previous myocardial infarction may result in nonfunctioning myocardium that will impair contractility. A recent concept is that ischemic myocardial tissue can be nonfunctioning (hibernating) but revitalized by surgical or medical therapy directed at ischemic heart disease [40]. Other depressants of myocardial systolic function include pharmacologic agents (calcium-channel blockers), hypoxemia, and severe acidosis.

The final determinant of stroke volume is afterload. In basic terms, afterload is the load that the pump has to work against, which is usually clinically estimated by the mean arterial pressure. The normal cardiac output is relatively insensitive to afterload up to 140 mm Hg. However, the afterload represents not only the vascular resistance but also the wall tension and intrathoracic pressure that the myocardium must work against. Together, these 3 variables are impaired in the patient with CHF [36].

The failing heart in CHF can be best evaluated with the above variables considered together. If cardiac output falls, either the heart rate or stroke volume must change in order to maintain perfusion. If stroke volume cannot be maintained, then heart rate

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must increase to maintain cardiac output. However, the pathophysiology behind CHF includes not only a structural abnormality; it also includes the cardiovascular response to poor perfusion with the activation of the neuro-humoral system [41]. Activation of the renin-angiotensin system attempts to increase preload by stimulating retention of salt and water, increasing vasoconstriction (and, thus, afterload), and augmenting cardiac contractility. Initially, this response will suffice, but prolonged activation results in loss of myocytes and maladaptive changes in the surviving myocytes and the extracellular matrix. The stressed myocardium undergoes remodeling and dilation in response to the insult [42]. This process also has detrimental effects on the functioning of the lungs, kidneys, muscles, blood vessels, and probably other organs. Remodeling also results in additional cardiac decompensation from complications, including mitral regurgitation from valvular annulus stretching, and cardiac arrhythmias from atrial remodeling [41].

The respiratory care provider often becomes involved with the CHF patient as the elevated end-diastolic pressure leads to pulmonary edema and dyspnea. Patients' presentation can greatly differ, depending on the chronicity of the disease. For instance, most patients experience dyspnea when pulmonary-artery occlusion pressure exceeds 25 mm Hg. However, the patient with longstanding CHF can tolerate filling pressure up to 40 mm Hg [43]. The lung provides multiple mechanisms to avoid the consequences of pulmonary edema. Initially, as pressure increases, pulmonary capillaries are recruited and increase capacitance to deal with the added volume [44]. As pressure continues to increase, volume can be diverted from the alveoli to the interstitium. At this point, by action of pressure gradients, fluid will form in the interlobular septae and the perihilar region. As noted above, chronic heart failure is associated with increased venous capacitance and lymphatic drainage of the lung. As a result, crackles are often absent, even in the setting of elevated pulmonary capillary pressure. Continued sodium retention preferentially results in peripheral edema and, ultimately, in the development of pleural effusions [45]. With acute decompensation, the pulmonarycapillary membrane may succumb to increased pressure, with shearing of the capillary and release of fluid, protein, and occasionally red blood cells into the alveoli [46]. The lungs' response will include cough, to expel the fluid in the alveoli. The long-term response to elevated pulmonary venous pressure includes interstitial fibrosis with thickening of the alveolar membrane [43]. Thus, severe, chronic heart failure can result in interstitial fibrosis and a restrictive lung disease.

2.1.4 Evaluation of the Patient With Congestive Heart Failure

The approach to the patient with suspected heart failure includes a history and physical examination, chest radiograph, and a series of diagnostic tests to assess both the acuity and severity. History alone is insufficient to make the diagnosis of heart failure, but often provides clues to the cause (myocardial infarction or uncontrolled hypertension), the precipitating event (noncompliance with diet or medications), and the severity. The symptoms of heart failure can be related to either the reduction of cardiac output (fatigue, weakness) or to excess fluid retention (dyspnea, orthopnea, and "cardiac wheezing"). With progression to right-heart failure there may be hepatic congestion (with right upper-quadrant discomfort), early satiety, anorexia, and discomfort with bending. Fluid retention also results in peripheral edema and occasionally in increasing abdominal girth secondary to ascites. Absence of dyspnea on exertion essentially rules out heart failure due to leftventricular dysfunction [47]. Pulmonary congestion (with crackles and wheezing) is predominant in acute or subacute disease. Although commonly taught as an absolute finding in CHF, crackles are present in only 20% of patients with chronic CHF. In addition, lower-extremity edema presents in about 25% of patients younger than 70 years of age [48].

The most reliable indicator of volume overload is the presence of elevated jugular venous pulsation (estimated by distention of the jugular veins with the patient sitting at 45°), which correlates with elevated pulmonary-artery occlusion pressure 80% of the time.18 Ventricular enlargement can be estimated by precordial palpation, and an apical pulsation displaced laterally to the midclavicular line is usually indicative of left-ventricular enlargement. In patients with dyspnea, a chest radiograph is a useful first test for differentiating patients with heart failure from patients with primary pulmonary disease. Radiographic findings suggestive of heart failure include cardiomegaly (cardiac-to-thoracic ratio above 50%), cephalization of blood vessels, increased interstitial markings, and pleural effusions [49].

Clinical classification of patients with chronic CHF was described by New York Heart Association (NYHA) classification system, which separates patients based on the limitations they experience in performing certain activities (Table: 2.1). An NYHA class I patient has almost no evidence of limitation in daily performance of activities, whereas an NYHA class IV patient experiences severe symptoms even at rest [50].

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Table 2.1: New York Heart Association Classification of Congestive Heart Failure

Stage/Degree	Symptoms and Activity-Limitations
I – None	No symptoms from ordinary activities
I – None	Comfortable at rest or during mild exertion
III – Moderate	Symptomatic with any activity
IV – Severe	Symptomatic at rest. Confined to bed or chair

A constellation of characteristic signs, symptoms, and radiographic findings, as described above, often lead to the diagnosis of CHF (Table: 2.2). Patients with previous evidence of heart disease, diabetes mellitus, hypertension, or documented coronary-artery disease are at increased risk for CHF, and one should always consider CHF in the differential diagnosis of acute respiratory failure in these patients [51]. Until recently, beyond direct functional assessments of cardiac contractility, there were no laboratory tests to assist in the diagnosis of heart failure. Brain natriuretic peptide is one of a family of neurohormones that is produced by the ventricles in response to increased pressure and volume load. It works to counteract the effect of the renin-angiotensin system by providing vasodilation, natriuresis, diuresis, and decreased smooth-muscle proliferation. It has become a valuable tool to assist in determining the etiology, prognosis, and maintenance therapy for patients with CHF [52]. Plasma concentrations of brain natriuretic peptide are helpful in distinguishing dyspnea from cardiac causes (either left-ventricular dysfunction or cor pulmonale) versus from pulmonary causes (chronic obstructive pulmonary disease, asthma, or pneumonia). A cut-off value of 100 pg/mL diagnoses heart failure with a sensitivity of over 90% and a predictive accuracy of 83% [53]. Reducing the cut-off point to 50 pg/mL increased the sensitivity to 97%, and a value below 50 pg/mL virtually rules out heart failure as a cause of dyspnea. Most patients with heart failure have values of over 400 pg/mL, but caution is necessary because pulmonary emboli and other causes of cor pulmonale are associated with high values of brain natriuretic peptide [53].

Table 2.2: Modified Framingham Criteria for the Diagnosis of Chronic Heart Failure

Major Criteria	Minor Criteria
▪ Neck-vein distention	▪ Bilateral ankle edema
▪ Orthopnea or paroxysmal nocturnal dyspnea	▪ Night cough
▪ Crackles (10 cm above base of lung)	▪ Dyspnea on exertion
▪ Cardiomegaly on chest radiograph	▪ Hepatomegaly
▪ S3 gallop	▪ Pleural effusion
▪ Central venous pressure 12 mm Hg	▪ Tachycardia (120 beats/min)
▪ Left ventricular dysfunction on echocardiogram	
▪ Weight loss 4.5 kg in response to CHF treatment	
▪ Acute pulmonary edema	

2.1.5 Therapy for Congestive Heart Failure

i. Nonpharmacologic Therapy

Patients with heart failure can benefit from attention to exercise, diet, and nutrition. [54, 55] Restriction of activity promotes physical deconditioning, so physical activity should be encouraged. However, limitation of activity is appropriate during acute heart failure exacerbations and in patients with suspected myocarditis. Most patients should not participate in heavy labor or exhaustive sports.

A meta-analysis showed that aerobic exercise training, particularly over the long term, can reverse left ventricular remodeling in clinically stable heart failure patients, whereas strength training had no effect on remodeling [56].

Because nonadherence to diet and medication can have rapid and profound adverse effects on patients' clinical status, close observation and follow-up are important aspects of care [54, 57]. Patient education and close supervision, including surveillance by the patient and family, can improve adherence. These measures also facilitate early detection of weight gain or slightly worsened symptoms, which often occur several days before major clinical episodes that require emergency care or hospitalization. Patients can then alert their clinicians, who may be able to prevent such episodes through prompt intervention [54].

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Dietary sodium restriction to 2-3 g/day is recommended. Fluid restriction to 2 L/day is recommended for patients with evidence of hyponatremia (Na < 130 mEq/dL) and for those whose fluid status is difficult to control despite sodium restriction and the use of high-dose diuretics. Caloric supplementation is recommended for patients with evidence of cardiac cachexia [54].

An analysis of concentrations of plasma eicosapentaenoic acid (EPA), a long-chain omega-3 fatty acid, in the Cardiovascular Health Study identified plasma phospholipid EPA concentration as being inversely related to incident congestive heart failure [58]. These results support additional studies on the potential benefits of omega-3 fatty acids for primary prevention of heart failure.

The GISSI-HF (Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico) trial, which included nearly 7000 patients with systolic heart failure (any LV ejection fraction) who received either 1 g of omega-3 polyunsaturated fatty acids (PUFAs) or placebo daily, demonstrated that the PUFA regimen had a small but significant reduction in both all-cause mortality and all-cause mortality/hospitalization for cardiovascular causes [59].

ii. Pharmacologic Therapy

Pharmacologic therapy for heart failure (HF) includes but is not limited to the following [54, 55, 57, 60]:

a. **Diuretics** (to reduce edema by reduction of blood volume and venous pressures) and salt restriction (to reduce fluid retention) in patients with current or previous heart failure symptoms and reduced left ventricular (LV) ejection fraction (EF) for symptomatic relief
b. **Angiotensin-converting enzyme inhibitors (ACEIs)** for neurohormonal modification, vasodilatation, improvement in LVEF, and survival benefit
c. **Angiotensin receptor blockers (ARBs)** for neurohormonal modification, vasodilatation, improvement in LVEF, and survival benefit
d. **Hydralazine and nitrates** to improve symptoms, ventricular function, exercise capacity, and survival in patients who cannot tolerate an ACEI/ARB or as an add-on therapy to ACEI/ARB and beta-blockers in the black population for survival benefit
e. **Beta-adrenergic blockers** for neurohormonal modification, improvement in symptoms and LVEF, survival benefit, arrhythmia prevention, and control of ventricular rate
f. **Aldosterone antagonists,** as an adjunct to other drugs for additive diuresis, heart failure symptom control, improved heart rate variability, decreased ventricular arrhythmias, reduction in cardiac workload, improved LVEF, and increase in survival
g. **Digoxin,** which can lead to a small increase in cardiac output, improvement in heart failure symptoms, and decreased rate of heart failure hospitalizations
h. **Anticoagulants** to decrease the risk of thromboembolism
i. **Inotropic agents** to restore organ perfusion and reduce congestion
j. **Soluble guanylate cyclase (sGC)** stimulators to augment smooth muscle relaxation and vasodilation
k. **Selective sodium-glucose cotransporter-2 (SGLT2)** inhibitors to reduce the risk of cardiovascular death

A. 2022 ACC/AHA/HFSA Guidelines

As noted earlier, the updated American College of Cardiology, American Heart Association, and Heart Failure Society of America (ACC/AHA/HFSA) guidelines include four core foundational medication classes (SGLT2Is, beta blockers, mineralocorticoid receptor antagonists [MRAs], and renin-angiotensin system [RAF] inhibitors) in guideline-directed medical therapy (GDMT) for HF with reduced EF (HFrEF). [61, 62, 63, 64]

I. HFrEF

- Renin-angiotensin system inhibition with ACEI or ARB or ARNI (class 1 recommendations)
  - Patients with HFrEF and New York Heart Association (NYHA) class II-III symptoms: Use ARNI for reduction of morbidity/mortality
  - Patients with previous/current symptoms of chronic HFrEF: When ARNI is not feasible, ACEI is of benefit for reduction of morbidity/mortality
  - Patients with previous/current symptoms of chronic HFrEF and intolerant of ACEI due to cough or angioedema and when ARNI is infeasible: Use ARB for reduction of morbidity/mortality

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- Patients with chronic symptomatic HFrEF NYHA class II/III tolerant of ACEI/ARB: Replace with an ARNI for further reduction of morbidity/mortality
- Beta blockers (class 1 recommendation)
  - Patients with HFrEF, with previous/current symptoms: Use one of three beta blockers with proven mortality reduction (eg, bisoprolol, carvedilol, sustained-release metoprolol succinate) to lower mortality and hospitalizations.
- MRAs (class 1 recommendation)
  - Patients with HFrEF and NYHA class II-IV symptoms: Use an MRA (spironolactone or eplerenone) to reduce morbidity/mortality, if the estimated glomerular filtration rate is >30 mL/min/1.73 m2 and serum potassium is < 5.0 mEq/L. Minimize the risk of hyperkalemia and renal insufficiency by closely monitoring potassium levels, renal function, and diuretic dosing at initiation and thereafter.
- SGLT2Is (class 1 recommendation)
  - Patients with symptomatic chronic HFrEF: Irrespective of the presence of type 2 diabetes, use SGLT2Is for reduction of hospitalization for HF and cardiovascular mortality.

II. HF with mildly reduced EF (HRmrEF)

- Patients with HFmrEF: SGLT2Is can provide benefit in reducing HF hospitalizations and cardiovascular morbidity (class 2a).
- Patients with previous/current symptomatic HFmrEF (LVEF: 41-49%) taking evidence-based beta blockers for HFrEF: Consider ARNIs, ACEIs or ARBs, and MRAs to lower the risk of HF hospitalizations and cardiovascular mortality (especially among patients with LVEF on the lower end) (class 2b).

III. HF with preserved EF (HFpEF)

- (New) Patients with HFpEF: SGLT2Is can provide benefit in reducing HF hospitalizations and cardiovascular morbidity (class 2a).
- (New) In select patients with HFpEF: Consider MRAs or ARNIs to lower hospitalizations (especially among patients with LVEF on the lower end) (class 2b)
- (Renewed) Patients with HFpEF and hypertension: To prevent morbidity, titrate medication to achieve blood pressure goals as laid out in pubished clinical practice guidelines (class 1).
- (Renewed) Patients with HFpEF and atrial fibrillation (AF): Management of AF can be useful for symptomatic improvement (class 2a).
- (Renewed) Selected patients with HFpEF: Consider ARBs to lower hospitalizations (especially among patients with LVEF on the lower end) (class 2b).
- (Renewed) In patients with HFpEF, there is no benefit to the routine use of nitrates or phosphodiesterase-5 (PDE5) inhibitors to increase activity or quality of life (class 3).

IV. HF with improved EF (HFimpEF)

- Patients with posttreatment HFimpEF: Continue guideline-directed medical therapy (GDMT) to prevent relapse of HF and LV dysfunction, even in those who may become asymptomatic (class 1).

2.2 Pneumonia

Community-acquired pneumonia (CAP) is one of the most common causes of hospital admissions and death worldwide. The incidence and mortality of CAP are associated with the presence of comorbidities and increasing age [65].

Community-acquired pneumonia is the main cause of infectious disease related mortality worldwide and is responsible for approximately 1 million hospital admissions with a great impact on health care resources. Its incidence and mortality are related to the increase in age and the presence of comorbidities [66]. Given that the population is aging, it is expected that CAP will continue increasing as an outstanding public health problem [67].

Community-acquired pneumonia epidemiology can show differences according to geographical areas, health care setting, and study population. According to a report by the National Center for Health Statistics in 2014, both influenza and pneumonia were the eighth cause of mortality in the USA. In addition, in the USA in 2013, CAP incidence reported in adults

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>65 years ranged from 63 cases to 164.3 cases per 10,000 in adults >80 years [68]. For the same period in 2013, CAP incidence ranged from 76 to 140 cases per 10,000 adults in patients >65 years in Europe [69]. In developed countries, older age is the main risk factor for CAP. In a population-based surveillance study by Jain et al. performed in five hospitals in Chicago and Nashville, USA from January 2010 through June 2012, it was found that the incidence of hospitalized CAP increases with older age. They reported an overall annual incidence of pneumonia of 24.8 cases per 10,000 adults. By age groups, those adults between 65 and 79 years showed a rate of 63.0 cases per 10,000 adults, whereas the group with ≥80 years showed the highest rate, with 164.3 cases per 10,000 adults [68].

In developing countries, few data can be found about population-level pneumonia incidence. Using hospital data, pneumonia is one of the most frequent causes of hospitalization in adults. In developed countries, the main burden of hospitalized patients with CAP is in older patients with comorbidity, whereas in many developing countries, the main burden of hospitalized patients with CAP is among adults in the working age [65]. The World Health Organization reported that the average number of deaths related to CAP was approximately 700,000 deaths per year in developing countries [65]. Moreover, the contracting risk of CAP is strongly linked to the prevalence of the disease in the environment, which is the case of populations with poor access to primary health care services [66].

2.2.1 Risk Factors for CAP

Prompt identification of patients at risk for severe CAP is important to pneumonia prevention and management. The etiology has been related to age and variations in less representative pathogens [65].

The patient's age and comorbidities play an important role in determining the risk and disease severity of pneumonia. Therefore, patients with other diseases, such as diabetes, cancer, chronic heart failure, chronic obstructive pulmonary disease (COPD), Alzheimer, coronary artery disease, cystic fibrosis, renal insufficiency/dialysis, diabetes mellitus (DM), malignancy, chronic neurological disease, or chronic liver disease, have a higher incidence of pneumonia. In addition to comorbidities, toxic habits, such as smoking or alcoholism, have been also reported as risk factors for CAP. In the elderly (≥60 years), the risk increases in the presence of asthma, alcoholism, or immunosuppression [70]. Other important factors are male sex and the development of acute respiratory failure (ARF), severe sepsis, and bacteremia [71].

With regard to mortality, the study conducted between 2011 and 2013 by Tokgoz Akyil et al examined the reasons and factors that underlie the patients' lower survival. According to their study, the risk factors that must be considered include advanced age, male sex, black race, pneumonia associated with medical care, and chronic comorbid diseases [72]. In general, 82% of patients were diagnosed with at least one of the following diseases: asthma, COPD, coronary heart disease, chronic kidney disease, congestive heart failure, malignancy, and DM, among others. During follow-up, it was observed that malignancy, COPD, cardiovascular diseases, and neurodegenerative disorders increased mortality [65].

2.2.2 Causative Microorganisms

Knowledge of the most common causes of CAP is important to initial empirical antibiotic prescription. S. pneumoniae caused >90% of cases of pneumonia in adults globally [73]. On the other hand, atypical pneumonia is due to fastidious organisms, such as Mycoplasma pneumoniae, Legionella pneumophila, Coxiella burnetii, Chlamydophila pneumoniae, and Chlamydia psittaci [74], representing up to 22% of all cases. In immunocompromised patients with CAP, Enterobacteriaceae spp., Pseudomonas aeruginosa, methicillin-resistant Staphylococcus aureus, and extended-spectrum beta-lactamase positive are more frequent [75]. The study by Gunduz et al. [76] conducted in Turkey between 2009 and 2013 observed that the causative bacteria isolated most fre-quently in patients with CAP are S. pneumoniae, P. aeruginosa,Escherichia coli, Haemophilus influenzae, S. aureus, Klebsiella pneumoniae, Streptococcus spp. and Moraxella catarrhalis. An other study conducted between 2002 and 2009 highlighted that 0.5% to 10% of cases of CAP are attributed to Legionella, being the most common specie L. pneumophila [77].

A more useful approach in clinical practice is to classify organisms and episodes based on the degree of severity. Scores, such as CURB-65 (Confusion, Urea nitrogen, Respiratory rate, Blood pressure) or CAP-PIRO (predisposition, insult, response, and organ dysfunction of community-acquired pneumonia), help to stratify severity, being ARF and shock the most important cause of ICU admission [71, 78, 79].

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As mentioned above, S. pneumoniae is the main bacterial agent that causes CAP. This is consistent with studies conducted in the last 10 years in Europe, Asia, and the USA, showing that the most common bacteria worldwide are S. pneumoniae, P. aeruginosa, S. aureus, and H. influenzae [80, 81].

2.2.3 Diagnosis

Pneumonia is the main cause of sepsis. As a consequence, personalized medicine is an important approach in current management strategies. A detailed review on biomarkers and molecular diagnostic tests is not included in the purposes of this review. We refer the interested reader to the recent ESCMID position paper Towards a personalized medicine approach in sepsis [82].

An important part of the diagnosis of CAP is a thorough evaluation of the patient's condition. Before making a diagnosis, the patient's history must be acquired, and physical examination and microbiological tests, such as Gram stain and blood cultures, must be per-formed. It is very important to follow the proposed guidelines of the national and international clinical practice guidelines for the correct microbiological diagnosis of pneumonia [70]. According to these guidelines for severe CAP, it is recommended to perform blood cultures, culture and sputum staining, and urinary antigen test for Legionella and Pneumococcus. For example, sputum culture and urinary antigen test for L. pneumophila and S. pneumoniae are used for outpatients with failure of antibiotic therapy; sputum and blood culture are used for hospitalized patients with positive urinary antigen test for Pneumococcus and cavitary infiltrates; sputum, blood culture, and urinary antigen test for L. pneumophila and S. pneumoniae; tracheal aspirate or bronchoalveolar lavage culture and viral studies also need to be performed and are used for severe CAP admitted to the ICU; urinary antigen test for Legionella serogroup 1; and influenza test during influenza season is used for epidemiological factor or specific risk factors suggesting pathogen. The low performance, the long period of response, and the previous antibiotic exposure are the main problems of these diagnostic methods [83].

2.3 Pneumonia and heart failure

In elderly patients with heart failure, the left ventricular myocardial contractility is weakened and the ejection ability is reduced, which often leads to pulmonary hypertension, pulmonary edema, and pulmonary congestion.[84] Besides, in elderly patients, various functions of the body are reduced, bronchial glands and mucosal atrophy, and airway barrier function is weakened [85,86]. Decreased alveolar elasticity and decreased mobility of the mucocilia in the trachea, all of which lead to a decline in immune function and a decrease in cough reflex sensitivity in the elderly, which makes the elderly heart failure more likely to merge with lung infection [87,88]. Inflammatory factors stimulate pulmonary small blood vessels during lung infection, causing vasculitis, resulting in thickening of the vessel wall and narrowing of the official cavity [89]. Meanwhile, inflammatory exudation increases the pressure in the alveoli, of which lead to varying degrees of pulmonary hypertension and increase the afterload of ventricular contraction [90]. However, elderly patients often lack specific symptoms and signs due to the decline of the body's stress ability, which brings difficulties to clinical diagnosis [91]. Meanwhile, patients with heart failure often have different degrees of pulmonary edema [92]. It is difficult to diagnose the lungs through clinical lung auscultation [93]. Therefore, it is difficult to determine whether it is complicated with lung infection. In the process of clinical treatment, doctors also rely on clinical experience to use antibiotics, which is prone to bacterial resistance [94]. Therefore, early identification of risk factors for pulmonary infection is of great significance.

Elderly heart failure combined with pulmonary infection is a common condition in clinical settings [95]. The severity and prognosis of heart failure and lung infection are also closely related [96]. For patients with pulmonary infection, clinicians generally adopt empirical antibiotic treatment, but it is often accompanied by the overuse of antibiotics and the production of drug-resistant bacteria, and it brings unnecessary economic burden to patients, so understand that the distribution of pathogenic bacteria in patients with pulmonary infection is of great significance for guiding the choice of clinical treatment strategies [97]. The pulmonary infections of patients with heart failure are mainly Staphylococcus aureus, Klebsiella pneumoniae, and Pseudomonas aeruginosa, which are consistent with the results of previous related studies [98,99]. suggesting that the sputum of patients with heart failure and lung infection needs to be cultured. Therefore, the use of antibiotics in the treatment of elderly patients with heart failure and pulmonary infection should mainly inhibit Gram-negative bacteria. And we should use medicines reasonably according to the specific pathogenic bacteria infection to effectively control pulmonary infection.

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Elderly patients with heart failure complicated with lung infection should take relevant preventive measures. Strictly monitor the sputum and bacteria culture of patients during treatment, and instruct them to eat more foods with high calories, high protein, and high vitamins, instruct patients who stay in bed for rest to properly raise the head of the bed and encourage their family members to help them get out of bed [100,101]. Besides, back pat, expectoration, turn over times is beneficial to the prevention of pulmonary infection [102]. And patients should be carried out oral care regularly, rinse mouth with 0.9% sodium chloride or 0.3% soda after meals to inhibit the growth of fungi [103]. Patients with a history of smoking are advised to quit smoking, and patients with diabetes should strengthen blood sugar management and correct their wrong lifestyle [104,105]. Those measures may be effective to the prevention of pulmonary infection in patients with heart failure [105].

CHAPTER III
METHODS AND MATERIALS

3.1. Study Design

A retrospective, descriptive cross-sectional study was conducted to assess the prevalence of chest infection among patients who had heart failure in Internal Medicine Department, Al-Wahdah Teaching Hospital over six months (June – December 2022).

3.2. Study Area & Setting

This study was carried out at Internal Medicine Department, Al-Wahdah Teaching Hospital, Ma'aber City, Dhamar Governorate, Yemen.

3.3. Study Size

A total of 100 patients whose data were met the study's inclusion criteria, were available during study period, and were successfully enrolled in our study.

3.4. Study Population

All patients who were diagnosed with heart failure and chest infection, and were admitted to internal medicine department in Al-Wahdah Teaching Hospital during the study's period.

3.5. Data Collection

Data were collected by a structured questionnaire, which was designed basing on similar previous studies, and was divided into the following sections:

1) Demographic data of study population who had been admitted with diagnosis of heart failure and chest infection will be registered in separated sheet.
2) Patient`s clinical data will be documented on a sheet – focusing on symptoms and clinical emanations and the duration of the disease.
3) Patient`s investigation will be documented on the sheet included laboratory and radiological tests.

3.6. Statistical Analysis

Data processing, statistical analysis, and graph drawing were conducted using a Statistical Package for Social Sciences software SPSS (version 26.0).

3.7. Ethical Consideration

This study was introduced and approved by Thamar University Ethics Committee TUMEC (No: 2300-). Data formula have no name and the data was extracted from all the data formula in general.

CHAPTER IV
RESULTS

4.1. Socio-Demographic Characteristics

A total of 100 patients were included in our study including 53 (53%%) males, and 47 (47%) females. The majority 55 (55%) were of age group 40 – 60 years. The majority of the included patients were from rural residence 83 (83%). The highest percentage of the respondents were housewives 34 (34%), followed by farmer 39 (29%). One-quarter 25 (25%) of included patients

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were smokers, the mean duration of smoking was 28.84 years (SD 13.24), more than half of the participants were Qat chewers 59 (59%), while only 13% were have habit of Shamma intakes (Table: 4.1).

Table 4.1: Socio-demographic characteristics among study's participants (n = 100)

Socio-Demographic Characteristics		Frequency	Percent
Age	< 14 years	1	1%
	15 to 40 years	5	5%
	40 to 60 years	55	55%
	More than 60 years	39	39%
Sex	Female	47	47%
	Male	53	53%
Residence	Rural	83	83%
	Urban	17	17%
Occupation	Builder	1	1%
	Driver	1	1%
	Farmer	29	29%
	Housewife	34	34%
	Manual worker	14	14%
	Nil	15	15%
	Solider	3	3%
	Student	2	2%
	Teacher	1	1%
Smoking	Yes	25	25%
	No	75	75%
Smoking duration	Mean (years) 28.84 SD (years) 13.24		
Qat chewing	Yes	59	59%
	No	41	41%
Shamma	Yes	13	13%
	No	87	87%

4.2. Risk factors

Regarding the risk factors and comorbidities among studied patients, more than half of patients were had hypertension 57 (57), the mean duration of hypertension was 7.43 years (SD 6.44), more than half of hypertensive patients weren't under regular treatment for hypertension 52 (52%). Around one-third of the patients were diabetic, with mean duration of 7.22 years (SD = 5.57), the majority of them were under treatment with oral hypoglycemic drugs 28 (90.3%), and about one-quarter of them weren't treated regularly for their diabetes state 8 (25.8%). Around one-quarter of included patients were obese 26 (26%). Less than one-fifth were had coronary artery disease 18 (18%), for a mean duration of 4.82 years (SD = 2.97). Only one patient was had congenital heart disease which was of non-cyanotic category. Rheumatic heart disease was found in 11 (11%) patients, for a mean duration of 9.91 years (SD = 7.89). One-fifth of patients were had cardiomyopathy 20 (20%) for a mean duration of 4.20 years (SD = 2.52). Ten percent of patients had arrhythmia. The minority of patients were had COPD and Asthma (5% and 2% respectively). History of stroke was found in 8 (8%) patients, which was ischemic type in the majority of them 6 (75%). The minority of patients were anemic 7 (7%) for a mean duration of 2 years (SD = 2.97). History of thyroid disease was found in two patients (2%) for a mean duration of 2 years (SD = 1.41) (Table: 4.2).

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Table 4.2: Risk factors and comorbidities among study's participants (n = 100)

Risk Factors		Frequency	Percent
Hypertension	Yes	57	57%
	No	43	43%
Duration of hypertension	Mean (years) 7.43 SD (years) 6.44		
Hypertension treatment	Regular	27	47%
	Irregular	30	52%
Diabetes mellitus	Yes	31	31%
	No	69	69%
Duration if diabetes mellitus	Mean (years) 7.22 SD (years) 5.57		
Diabetes treatment	Regular	23	74%
	Irregular	8	25.8%
Type of diabetes treatment	Insulin	3	9.7%
	Oral	28	90.3%
Obesity	Yes	26	26%
	No	74	74%
Coronary artery disease	Yes	18	18%
	No	82	82%
Duration of coronary artery disease	Mean (years) 4.82 SD (years) 2.97		
Congenital heart disease	Yes	1	1%
	No	99	99%
Type of congenital heart disease	Cyanotic	0	0%
	Non-cyanotic	1	100%
Rheumatic heart disease	Yes	11	11%
	No	89	89%
Duration of RHD	Mean (years) 9.91 SD (years) 7.89		
Cardiomyopathy	Yes	20	20%
	No	80	80%
Duration of cardiomyopathy	Mean (years) 4.20 SD (years) 2.52		
Arrhythmia	Yes	10	10%
	No	90	90%
COPD	Yes	5	5%
	No	95	95%
Duration of COPD	Mean (years) 7.20 SD (years) 4.76		
Asthma	Yes	2	2%
	No	98	98%
Duration of asthma	Mean (years) 3.50 SD (years) 2.12		
Stroke	Yes	8	8%
	No	92	92%
Type of stroke	Hemorrhagic	2	25%
	Ischemic	6	75%
Anemia	Yes	7	7%
	No	93	93%
Duration of anemia	Mean (years) 2.00 SD (years) 1.52		
Thyroid disease	Yes	2	2%
	No	98	98%
Duration of thyroid disease	Mean (years) 2.00 SD (years) 1.41		

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4.3. Present history and physical examination

Regarding the heart failure duration among included patients, the majority of patients 37 (37%) were had HF for a duration between 1 – 3 years, followed by 17 (17%) patients for a duration between 7 – 10 year, while only 7 patients had the HF for a duration more than 10 years (figure: 4.1).

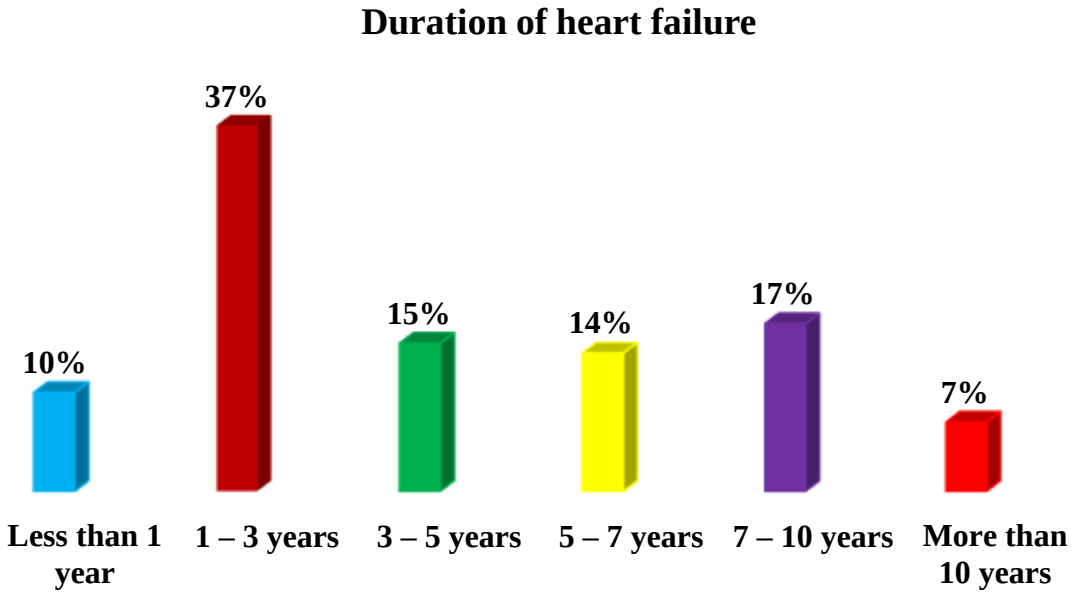

Figure 4.1: Duration of heart failure among studied patients (n = 100)

The majority of included patients were diagnosed with chest infection 63 (63%) (figure: 4.2), it was the second attack in the highest proportion of included patients 24 (38.1%), first attack in 22 (34.9%) patients, while the highest frequency rate of chest infection was 6 times and found in only two patients (3.2%). The duration of the current chest infection was 12.03 +/- 9.99 days. (Table: 4.3).

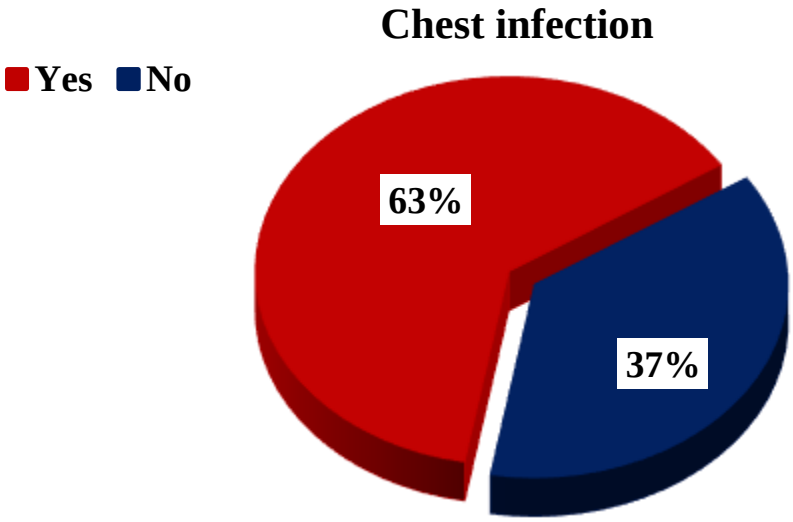

Figure 4.1: Chest infection among studied patients (n = 100)

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As shown in table 4.3, all included patients were complained of dyspnea at admission, with a duration of 8.10 +/- 7.44 days, and it was of functional class III in more than half of them 54 (54%). The majority of patients were complained of orthopnea and PND (82% and 65% respectively). About three quarters of the patients were complained of cough 75 (75%) for a duration of 9.01 +/- 8.32 days, which was productive in the majority of them 62 (82.7%), regarding sputum it was whitish in color and little in amount in the majority of the patients who had productive cough (50% and 59.7% respectively). Hemoptysis was reported in two patients (2%), which was little in amount in both of them.

Slightly more than one-third of patients were complained of chest pain 36 (36%) for a duration of 6.55 +/- 5.40 days, which was peripheral in location in more than half of them 19 (52.8%). The majority of patients were complained of lower limb edema 70 (70%) for a duration 14.55 +/- 12.31 days. The majority of patients were complained of fever, sweating and fatigue (64%, 58%, and 82% respectively), while oliguria was reported from the minority of the patients (12%).

Table 4.3: Present history among the study's participants (n = 100)

Present history		Frequency	Percent
Chest infection	Yes	63	63%
	No	37	37%
Duration of current chest infection	Mean (days) 12.03 SD (days) 9.99		
Frequency of chest infection	First attack	22	34.9%
	Two times	24	38.1%
	Three times	9	14.3%
	Four times	4	6.3%
	Five times	2	3.2%
	Six times	2	3.2%
Dyspnea	Yes	100	100%
	No	0	0%
Duration of dyspnea	Mean (days) 8.10 SD (days) 7.44		
Dyspnea class	I	4	4%
	II	23	23%
	III	54	54%
	IV	19	19%
Orthopnea	Yes	82	82%
	No	18	18%
PND	Yes	65	65%
	No	35	35%
Cough	Yes	75	75%
	No	25	25%
Duration of cough	Mean (days) 9.01 SD (days) 8.32		
Type of cough	Dry	13	17.3%
	Productive	62	82.7%
Amount of sputum	Very little	7	11.3%
	Little	37	59.7%
	Medium	15	24.2%
	Big amount	3	4.8%
Sputum colour	Frothy pinky	3	4.8%
	Greenish	2	3.2%
	Whitish	31	50%
	Yellowish	26	41.9%
Hemoptysis	Yes	2	2%

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Chest pain	No	98	98%
	Yes	36	36%
Location of chest pain	No	64	64%
	Central	17	47.2%
	Peripheral	19	52.8%
Duration of chest pain	Mean (days) 6.55 SD (days) 5.50		
Fever	Yes	64	64%
	No	36	36%
Sweating	Yes	58	58%
	No	42	42%
Fatigue	Yes	82	82%
	No	18	18%
Lower limb edema	Yes	70	70%
	No	30	30%
Duration of lower limb edema	Mean (days) 14.55 SD (days) 12.31		
Oliguria	Yes	12	12%
	No	88	88%

Regarding physical examination, the systolic blood pressure was 125.23 +/- 22.87 mmHg, and the diastolic blood pressure was 76.98 +/- 15.28 mmHg. The pulse rate was 89.7 +/- 19.93 beats per minute, the respiratory rate was 22.90 +/- 4.67 cycle per minute, and the temperature was 37.65 +/- 0.66 C°. The oxygen saturation was 90.87 +/- 5.51%. Regarding jugular venous pressure, it was elevated in slightly less than half of the participated patients 46 (46%). Basal crepitation was the most common finding by chest examination 81 (81%), followed by bilateral decreased air entry 78 (78%), while clear chest was observed in only nine patients (9%). Present of third heart sound was the commonest abnormality found by pericardial examination 22 (22%), followed by a systolic murmur in 12 (12%) patients, while accentuated heart sounds was the least common abnormality found by pericardial examination 2 (2%). (Table: 4.4)

Table 4.4: Physical examination findings among the study's participants (n = 100)

Physical examination		Frequency	Percent
Systolic blood pressure	Mean (mm Hg) 125.23 SD (mm Hg) 22.87		
Diastolic blood pressure	Mean (mm Hg) 76.98 SD (mm Hg) 15.28		
Pulse rate	Mean (beats/min) 89.70 SD (beats/min) 19.93		
Respiratory rate	Mean (cycle/min) 22.90 SD (cycle/min) 4.67		
Temperature	Mean (C°) 37.65 SD (C°) 0.66		
Oxygen saturation	Mean (%) 90.87 SD (%) 5.51		
JVP	Normal	54	54%
	Elevated	46	46%
Chest examination	Clear	9	9.0
	Crepitation	81	81%
	Wheezing	14	14%
	Decreased air entry	78	78%
	Bronchial breathing	12	12%
Pericardial examination	S1 + S2 + 0	52	52%
	Accentuated heart sound	2	2%
	Diastolic murmur	3	3%
	Muffled heart sounds	8	8%

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	S4	3	3%
	Systolic murmur	12	12%
	S3	22	22%

4.4. Investigation

As shown in table 4.5. around one third of the patients were had a high WBC counts 29 (29%), Hb level was 13.79 ± 1.96 g/dl, and HCT was 37.65 ± 8.70. Random blood sugar was 149 ± 81.81 mg/dl, C-reactive protein was 2 ± 0.78 mg/dl, ESR was 26.90 ± 24.42 mm/hour, and serum creatinine was 1.21 ± 0.64 mg/dl.

Cardiomegaly was the commonest abnormality found by chest X-ray, which was found in more than half of the patients 52 (52%), followed by the manifestations of bronchopneumonia which were found in 31 patients (31%), while the manifestations of lobar pneumonia were the least found abnormalities 12 (12%). (Table: 4.5).

Ventricular hypertrophy was the commonest abnormality detected by ECG 27 (27%), followed by atrial hypertrophy in 14 patients (14%), while the atrial flutter and left axis deviation were the least found abnormalities (1%). (Table: 4.5).

Echocardiography revealed that, the ejection fraction was 39.97 ± 11.62%. Dilated cardiac chambers was the commonest abnormality detected by echocardiography, which was found in 22 patients (22%), followed by IHD in 7 patients (7%), mitral regurgitation in 6 patients (6%). (Table: 4.5).

Table 4.5: Investigations results among studied patients (n = 100)

Investigations		Frequency	Percent
	Low	3	3%
WBC	Normal	68	68%
	High	29	29%
Hb	Mean (g/dl) 13.79 SD (g/dl) 1.96		
HCT	Mean (%) 37.65 SD (%) 8.70		
RBS	Mean (mg/d) 149.74 SD (mg/d) 81.81		
ESR	Mean (mm/hour) 26.90 SD (mm/hour) 24.42		
CRP	Mean (mg/dl) +2.00 SD (mg/dl) +0.78		
Serum creatinine	Mean (mg/dl) 1.21 SD (mg/dl) 0.64		
	Cardiomegaly	52	52%
	Lobar pneumonia	12	12%
Chest X-ray findings	Bronchopneumonia	31	31%
	Pleural effusion	18	18%
	Pulmonary congestion and pulmonary edema	16	16%
	Normal	18	18%
	Atrial fibrillation	6	6%
	Bundle branch block	7	7%
	Atrial flutter	1	1%
	Atrial hypertrophy	14	24%
	Low voltage	5	5%
	Ectopic beat	1	1%
ECG findings	Ventricular hypertrophy	27	27%
	Sinus tachycardia	2	2%
	ST elevation	7	7%
	Supraventricular tachycardia	2	2%
	Left axis deviation	1	1%
	Wide QRS complex	5	5%
	Not specified abnormalities	12	12%

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	Ejection Fraction: Mean (39.97) SD (11.62		
	AS	1	1%
	DCM	22	22%
	AR	1	1%
	TR	1	1%
Echocardiography findings	MR	6	6%
	IHD	7	7%
	RHD	5	5%
	LVH	5	5%
	Pulmonary HTN	3	3%
	MS	1	1%

4.5. Chest infection V.S socio-demographic characteristics

By comparison the present of chest infection against socio-demographic characteristics data of participated patient we found that, there was no relation between patients' age, sex and residence with chest infection. The majority of patients who had history of smoking, Qat chewing and Shamma intakes were found to have chest infection, which was statistically significant with P-value of < 0.05 (82%, 72.9% and 92.3% respectively). (Table: 4.6)

Table 4.6: Chest infection V.S socio-demographic characteristics among the participated patients (n=100)

Socio-demographic characteristics		**Chest infection**				**Total**		**X^2**	**P-value**
		Yes		**No**					
		No	**%**	**No**	**%**	**No**	**%**		
Age	< 14 years	1	100.0%	0	0.0%	1	1%	9.552	0.023
	14 – 40 years	0	0.0%	5	100.0%	5	5%		
	41 – 60 years	37	67.3%	18	32.7%	55	55%		
	> 60 years	25	64.1%	14	35.9%	39	39%		
Sex	Male	34	64.2%	19	35.8%	53	53%	0.064	0.964
	Female	29	61.7%	18	38.3%	47	47%		
Residence	Rural	55	66.3%	28	33.7%	83	83%	2.233	0.135
	Urban	8	47.1%	9	52.9%	17	17%		
Occupation	Builder	1	100.0%	0	0.0%	1	1%	14.07	0.044
	Driver	0	0.0%	1	100.0%	1	1%		
	Farmer	22	75.9%	7	24.1%	29	29%		
	Housewife	21	61.8%	13	38.2%	34	34%		
	Manual worker	10	71.4%	4	28.6%	14	14%		
	Nil	5	33.3%	10	66.7%	15	15%		
	Solider	3	100.0%	0	0.0%	3	3%		
	Student	1	50.0%	1	50.0%	2	2%		
	Teacher	1	100.0%	0	0.0%	1	1%		
Smoking	Yes	22	88%	3	12%	25	25%	8.938	0.001
	No	41	54.7%	34	45.3%	75	75%		
Qat chewing	Yes	43	72.9%	16	27.1%	59	59%	6.028	0.014
	No	20	48.8%	21	51.2%	41	41%		
Shamma	Yes	12	92.3%	1	7.7%	13	13%	5.506	0.009
	No	51	58.6%	36	41.4%	87	87%		

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4.6. Chest infection V.S duration of heart failure

We compared the occurrence of chest infection with duration of heart failure we found that, chest infection was observed in all patients who had heart failure for more than ten years, but this was insignificant statistically. (Figure: 4.3).

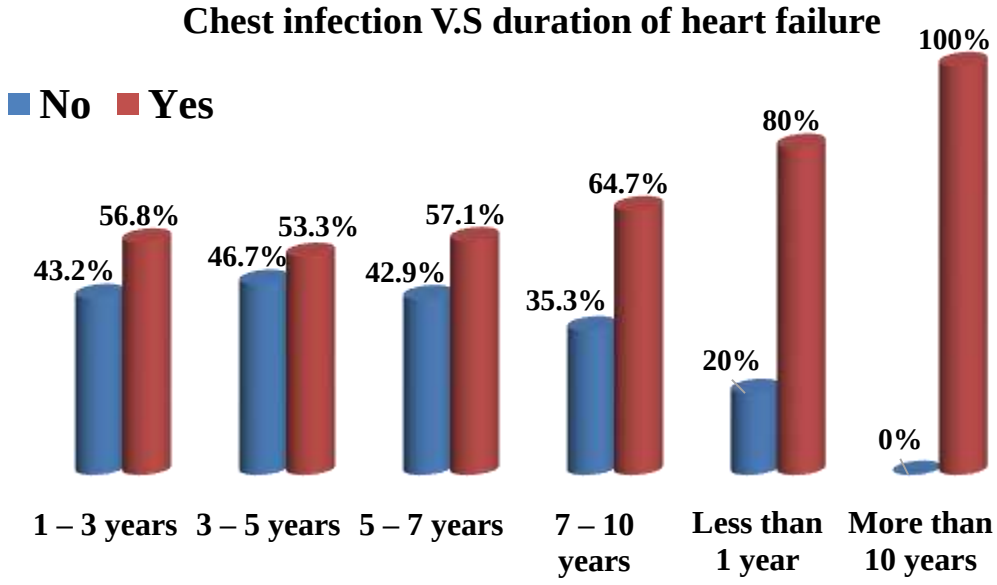

Figure 4.3: Chest infection V.S duration of heart failure among the study participants (n = 100)

4.7. Chest infection V.S risk factors and comorbidities

We compared the occurrence of chest infection with risk factors and comorbidities we found that, there was no statistically significant association between chest infection with all of the following: hypertension, diabetes mellitus, coronary artery disease, rheumatic heart disease, arrhythmia, stroke, COPD, asthma and thyroid diseases. The majority of patients who was obese and those who had cardiomyopathy (84.6% and 85% respectively) were found to have chest infection, which was statistically significant (P-value < 0.05). (Table: 4.7)

Table 4.7: Chest infection V.S risk factors and comorbidities among the study participants (n = 100)

Risk Factors		Chest infection				Total		X^2	P-value
		Yes		No					
		No	%	No	%	No	%		
Hypertension	Yes	39	68.4%	18	31.6%	57	57%	1.671	0.279
	No	24	55.8%	19	44.2%	43	43%		
Treatment of hypertension	Regular	20	74.1%	7	25.9%	27	47.4%	0.759	0.382
	Irregular	19	63.3%	11	36.7%	30	52.6%		
Diabetes mellitus	Yes	22	71.0%	9	29.0%	31	31%	1.224	0.264
	No	41	59.4%	28	40.6%	69	69%		
Treatment of diabetes mellitus	Regular	19	82.6%	4	17.4%	23	74.2%	5.862	0.015
	Irregular	3	37.5%	5	62.5%	8	25.8%		
Type of diabetes treatment	Insulin	3	100.0%	0	0%	3	9.7%	1.359	0.620
	Oral	19	67.9%	9	32.1%	28	90.3%		
Obesity	Yes	22	84.6%	4	15.4%	26	26%	7.042	0.005
	No	41	55.4%	33	44.6%	74	74%		

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Coronary Artery Disease	Yes	13	72.2%	5	27.8%	18	18%	0.801	0.371
	No	50	61.0%	32	39.0%	82	82%		
Congenital heart disease	Yes	1	100.0%	0	0%	1	1%	0.593	0.441
	No	62	62.6%	37	37.4%	99	99%		
Rheumatic heart disease	Yes	4	36.4%	7	63.6%	11	11%	3.762	0.108
	No	59	66.3%	30	33.7%	89	89%		
Cardiomyopathy	Yes	17	85%	3	15%	20	20%	5.191	0.016
	No	46	57.5%	34	42.5%	80	80%		
Arrhythmia	Yes	6	60%	4	40%	10	10%	0.043	0.836
	No	57	63.3%	33	36.7%	90	90%		
COPD	Yes	3	60%	2	40%	5	5%	0.020	0.887
	No	60	63.2%	35	36.8%	95	95%		
Asthma	Yes	2	100%	0	0%	2	2%	1.199	0.723
	No	61	62.2%	37	37.8%	98	98%		
Stroke	Yes	4	50%	4	50%	8	8%	0.235	0.628
	No	58	63.7%	34	36.3%	92	92%		
Type of stroke	Hemorrhagic	1	50%	1	50%	2	25%	0.000	1.000
	Ischemic	3	50%	3	50%	6	75%		
Anemia	Yes	6	85.7%	1	14.3%	7	7%	1.666	0.376
	No	57	61.3%	36	38.7%	93	93%		
Thyroid Disease	Yes	2	100%	0	0%	2	2%	1.199	0.274
	No	61	62.2%	37	37.8%	98	98%		

4.8. Chest infection V.S Clinical manifestations

By comparison the patients' present history with presence of chest infection we found that, the majority of patients who complained of class III (66.7%) and class IV (84.2%) were observed to had chest infection which was statistically significant P-value= 0.002. The majority of patients who had orthopnea (70.7%), PND (76.9%) was observed to had chest infection which was statistically significant P-value < 0.05. Cough was has a statistically significant association with chest infection P-value= 0.0001. Also the cough type and sputum amount were statistically significant associated with chest infection, where the majority of patients with productive cough was observed to had chest infection P-value 0.04, and the majority of patients with higher sputum amount were observed to had chest infection P-value = 0.034. Chest infection was observed among the majority of patients who had chest pain (80.6%), this association was statistically significant (P-value = 0.005). Furthermore, chest infection was had statistically significant association with location of chest pain , as the chest infection was observed in the majority of patients (94.7%) who had peripheral chest pain (P-value = 0.018). Chest infection was observed among the majority of patients with fever (85.9%), sweating (89.7%) and fatigue (74.4%), these association were statistically significant (P-value = 0.001). (Table: 4.8).

Table 4.8: Chest infection V.S clinical manifestations among the study participants (n = 100)

Clinical manifestations		**Chest infection**				**Total**		**X^2**	**P-value**
		Yes		**No**					
		No	**%**	**No**	**%**	**No**	**%**		
Class of dyspnea	I	0	0%	4	100%	4	4%	13.06	0.002
	II	11	47.8%	12	52.2%	23	23%		
	III	36	66.7%	18	33.3%	54	54%		
	IV	16	84.2%	3	15.8%	19	19%		
Orthopnea	Yes	58	70.7%	24	29.3%	82	82%	11.68	0.001
	No	5	27.8%	13	72.2%	18	18%		
PND	Yes	50	76.9%	15	23.1%	65	65%	15.444	0.0001

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Cough	No	13	37.1%	22	62.9%	35	35%	21.750	0.000
	Yes	57	76.0%	18	24.0%	75	75%		
	No	6	24.0%	19	76.0%	25	25%		
Type of cough	Dry	7	53.8%	6	46.2%	13	17.3%	4.231	0.040
	Productive	50	80.6%	12	19.4%	62	82.7%		
Sputum color	Frothy pinky	3	100%	0	0%	3	4.8%	2.289	0.515
	Greenish	2	100%	0	0%	2	3.2%		
	Whitish	23	74.2%	8	25.8%	31	50%		
	Yellowish	22	84.6%	4	15.4%	26	41.9%		
Sputum amount	Very little	3	42.9%	4	57.1%	7	11.3%	8.675	0.034
	Little	30	81.1%	7	18.9%	37	59.7%		
	Medium	14	93.3%	1	6.7%	15	24.2%		
	High amount	3	100%	0	0%	3	4.8%		
Hemoptysis	Yes	2	100%	0	0%	2	2%	1.199	0.274
	No	61	62.2%	37	37.8%	98	98%		
Chest pain	Yes	29	80.6%	7	19.4%	64	36%	7.437	0.005
	No	34	53.1%	30	46.9%	37	37%		
Location of chest pain	Central	11	64.7%	6	35.3%	17	47.2%	5.166	0.018
	Peripheral	18	94.7%	1	5.3%	19	52.8%		
Fever	Yes	55	85.9%	9	14.1%	64	64%	40.126	
	No	8	22.2%	28	77.8%	36	36%		
Sweating	Yes	52	89.7%	6	10.3%	58	58%	42.092	0.000
	No	11	26.2%	31	73.8%	42	42%		
Fatigue	Yes	61	74.4%	21	25.6%	82	82%	25.355	0.000
	No	2	11.1%	16	88.9%	18	18%		
Lower limb edema	Yes	48	68.6%	22	31.4%	70%	70%	3.107	0.078
	No	15	50%	15	50%	30	30%		
Oliguria	Yes	9	75%	3	25%	12	12%	0.842	0.359
	No	54	61.4%	34	38.6%	88	88%		

4.9. Chest infection V.S JVP status and WBC count

The majority of patients with elevated JVP was observed to had chest infection, however this association was insignificant statistically P-value > 0.05. Chest infection was observed among the majority of the patients (86.2%) who had a high WBC count, which was statistically significant (P-value = 0.005). (Table: 4.9).

Table 4.9: Chest infection V.S JVP status and WBC count among the study participants (n = 100)

Clinical manifestations		**Chest infection**				**Total**		**X^2**	**P-value**
		Yes		**No**					
		No	**%**	**No**	**%**	**No**	**%**		
JVP	Normal	34	63%	20	37%	54	54%	0.001	0.993
	Elevated	29	63%	17	63%	46	46%		
WBC	Low	2	66.7%	1	33.3%	3	3%	9.669	0.005
	Normal range	36	52.9%	32	47.1%	68	68%		
	High	25	86.2%	4	13.8%	29	29%		

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CHAPTER V
DISCUSSION

Heart failure is a common clinical manifestation of most organic heart diseases that progress to the end stage [106]. It is mostly caused by the loss of ventricular pumping ability due to changes in cardiac structure and function [107]. In contemporary society with an increasingly aging population, heart failure occurs greatly [108]. The rate of heart failure shows an upward trend year by year [109]. Patients with heart failure are often accompanied by secondary conditions such as pulmonary circulatory congestion and pulmonary edema, which can lead to dyspnea, gas exchange disorders, and other consequences, creating certain conditions for pathogens to invade and colonize the lungs [110,111]. Therefore, patients with heart failure may have higher risk of pulmonary infection. Besides, pulmonary infection and insufficient oxygen uptake can increase pulmonary artery pressure, decrease the body's metabolic function, and further increase the burden on the heart [112]. Presently, the potential influencing factors of pulmonary infection in patients with heart failure remain unclear.

A total of 100 patients with heart failure were included in this study, of whom 63 had been diagnosed with pulmonary infection; the incidence of pulmonary infection in patients with heart failure was 63%. Our findings were much higher than reported in previous similar studies, where Alexander etal., reported that the incidence of pulmonary infection in their heart failure patients was 21.8% [113]. Also it was higher of reported frequencies in previously published analyses of large registries investigating precipitating factors for HF, in which the prevalence of chest infection was 15.3% in the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients With Heart Failure (OPTIMIZE-HF) [114], 28.2% in Get With The Guidelines-HF (GWTG-H) [115], and 29% in the Spanish National Registry on Heart Failure (RICA) [116].

This discrepancy between our findings and the other related studies could be attributed to that, in our study the diagnosis of chest infection wasn't based and confirmed by isolation and culturing of microorganisms, instated of the pneumonia was commonly diagnosed based on clinical features suggestive of acute lower respiratory tract infection and demonstrable infiltrates on chest radiograph. In addition, the diagnosis was often aided by elevated CRP, ESR and WBC levels, all of which attributed to exaggerate the frequency rate of chest infection as both clinical features and chest radiograph results can be found in heart failure patients without chest infection.

The current study revealed that, the advanced functional class of dyspnea (III and IV), cough, higher amount of sputum, chest pain (especially peripheral located chest pain), fever, fatigue sweating and higher WBC count and CRP level, in addition to obesity and being had cardiomyopathy, all of which were observed to has statistically significant association with chest infection among participated patients. These findings were somewhat consistent with previous related studies, which reported that age ≥ 70 years, diabetes, NYHA grade III, LVEF ≤55%, and CRP ≥10 mg/L are the independent risk factors of pulmonary infections in patients with heart failure [117]. OPTIMIZE-HF revealed that, pneumonia and respiratory processes, arrhythmia, and uncontrolled hypertension were identified as most frequently associated precipitants to HF admissions [114].

It well known that, smoking is a strong risk factors for many diseases including pneumonia and other respiratory infection, in our study the smoking history was statistically significant associated with chest infection. However, Li Shen at al., reported that no significant association between chest infection and heart failure among their patients. Beside to smoking, we also found that Qat chewing and Shamma intakes were had statistically significant association with chest infection. However, no literatures found investigated such association.

CHAPTER VI
CONCLUSIONS & RECOMMENDATIONS
6.1. Conclusion

A total of 100 patients were enrolled in our study including 53 (53%) males, and 47 (47%) females. The majority were of age group 40 – 60 years. The majority of the included patients were from rural residence. In our study, the chest infection was common, where it was found in the majority of included patients (63), and the overall prevalence of chest infection in this study was (63%). The current chest infection was the second attack in the highest proportion of included patients. Our study revealed that, the majority of patients who were smokers, Qat chewer, shamma intakes, being obese, and had cardiomyopathy were found to had chest infection which were statistically significant.

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Also the chest infection was statistically significant associated with those independent predictors; NYHA class III and IV dyspnea, orthopnea, PND, cough (particularly productive cough), high sputum amount, whitish sputum color, chest pain (particularly peripheral), fatigue, sweating, fever and high WBC count.

6.2. Recommendations

1. Tacking in consideration chest infection is common in heart failure patients, so early prevention and intervention measures should be taken to reduce pulmonary infections.
2. Patients with a history of smoking should be advised to quit smoking, which may be effective to the prevention of pulmonary infection in patients with heart failure.
3. Future studies with prospective design and more potentially associated variables in different areas and populations are needed.

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LIST OF ABBREVIATIONS

ACC/AHA/HFSA	American Heart Association, and Heart Failure Society of America
ACEI	Angiotensin-converting enzyme inhibitor
ADHF	Acute decompensated heart failure
ARB	Angiotensin receptor blocker
ARF	Acute respiratory failure
ARNi	Angiotensin receptor-neprilysin inhibitor
CAP	Community-acquired pneumonia
CBC	Complete blood cell
CHF	Congestive heart failure
COPD	Chronic obstructive pulmonary disease

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CRP	C-reactive protein
DM	Diabetes mellitus
EF	Ejection Fraction
EPA	Eicosapentaenoic acid
ESR	Erythrocytes Sedimentation Rate
GWTG-HF	Get With The Guidelines-HF
GDMT	Guideline-directed medical therapy
HFimpEF	Heart failure with improved EF
HFpEF	Heart failure with preserved EF
HFrEF	Heart failure with reduced EF
HRmrEF	Heart failure with mildly reduced EF
JVD	Jugular venous distension
MCRA	Mineralocorticoid receptor antagonists
NYHA	New York Heart Association
OPTIMIZE-HF	Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients With HF
PDE5	Phosphodiesterase-5
PUFAs	Polyunsaturated fatty acids
RAF	Renin-angiotensin system
RICA	Spanish National Registry on Heart Failure
SGC	Soluble guanylate cyclase
SGLT2	selective sodium-glucose transporter-2

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