Available online on 15.03.2024 at ijmspr.com

International Journal of Medical Sciences and Pharma Research

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An Observational Study on Guideline Mediated Prescribing Pattern in Patients with Acute Decompensated Heart Failure

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Article Info:

Article History:

Received 01 January 2024
Reviewed 03 February 2024
Accepted 24 February 2024
Published 15 March 2024

Cite this article as:

Rama Rao T, Sravya G, Akshaya D, Kaushik T, An Observational Study on Guideline Mediated Prescribing Pattern in Patients with Acute Decompensated Heart Failure, International Journal of Medical Sciences & Pharma Research, 2024; 10(1):13-21

DOI: http://dx.doi.org/10.22270/ijmspr.v10i1.84

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Abstract

Introduction: Studies on prescribing patterns are useful investigative instruments for figuring out how drugs function in society. Examining the prescription pattern is a crucial part of gauging the ability of medical professionals to choose a medication that will benefit patients to the fullest. The current study goals were to evaluate, using accepted guidelines, the general medicine department's practice of prescribing in accordance with guidelines in a tertiary care hospital and to assess the inclusion of drugs from essential drug list patients with acute decompensated heart failure.

Methodology: Hospital based prospective case observational study was conducted for a period of six months after obtaining the permission from institutional ethical committee. The data was collected and analysed according to the inclusion and exclusion criteria and necessary statistical testing like chi square test and pearson correlation were performed.

Results and Discussion: Based on the study, the data of total 100 patients (n=100) were evaluated. Gender distribution of data shows high prevalence rate in males. The most commonly high susceptibility of disease age was found in 40-60 in males and females. The maximum number of patients were hospitalized for 6-7 days, hypertension was the most commonly found precipitating factor. Diuretics were prescribed maximum in the prescription. Out of 650 drugs the maximum number of drugs fall under the category of Anti-hypertensives. Most drugs are given through oral route. The American heart association (AHA) guidelines were assessed for drug prescription using IBM SPSS Statistics. It was depicted that most of the prescriptions were in compliance with the guidelines.

Conclusion: The results of our study conclude that prescribing practices were satisfactory according to the American college of cardiology guidelines.

Keywords: Prescribing pattern, Guidelines, Acute heart failure, Anti-hypertensives.

INTRODUCTION:

Recent years have seen the successful addition of more recent heart failure (HF) medications to more established ones, greatly increasing the arsenal of treatments available to lower the morbidity and mortality linked to HF. Patients with heart failure and a reduced ejection fraction (HFrEF) should be especially aware of this. The guidelines for guideline-directed medical therapy (GDMT) in both American and European societies have been informed by high-quality, randomized evidence.

There is much room for improvement in the way evidence-based therapies are implemented sequentially, patient-provider relationships, and the healthcare system as a whole. The slow uptake of GDMT can be attributed to a number of factors, including clinician competency, therapeutic inertia, low healthcare literacy, worries about adverse events, inadequate access to multidisciplinary resources, uneven insurance coverage, and unpredictable out-of-pocket costs.

As of 2022, the most proposed modern pharmacologic therapies consist of a comprehensive four drug regimen consisting of beta-blockers, sodium-glucose cotransporter-2 inhibitors (SGLT2i), mineralocorticoid receptor antagonists (MRA), and angiotensin receptor-neprilysin inhibitors(ARNI) [1].

One method for evaluating the prescription, dispensing, and distribution of medications is to use prescription pattern monitoring studies (PPMS). The main aim of PPMS is to facilitate rational use of medicines (RUM). Medicines are an essential component of healthcare, and without accessibility, modern healthcare would not be possible. In addition to promoting health and saving lives, they also stop diseases and epidemics. Every individual has a basic right to have access to medications. But in order to provide the greatest benefit, they must be sensible, safe, effective, and economical. Drug utilization studies called prescription pattern monitoring studies (PPMS) primarily concentrate on the prescription, dispensing, and administration of medications. They encourage the responsible use of drugs under observation and work to prevent their abuse or misuse. Along with collaborating and building relationships with other important organizations to achieve a rational use of drugs, PPMS also provides guidance and support to prescribers, dispensers, and the general public regarding the appropriate use of drugs[2].

AHF(Acute Heart Failure), ADHF(Acute Decompensated Heart Failure), AHF syndrome, and hospitalized HF are some of the

terms used to describe the clinical presentation of symptoms and signs of congestion and poor organ perfusion caused by HF that require urgent, usually intravenous, therapy. Dyspnea, exercise intolerance, palpitations, presyncope, peripheral edema, abdominal bloating, early satiety, and fatigue are signs and symptoms of left or right (and frequently both) increased ventricular filling pressures caused by compensatory mechanisms being overwhelmed by subacute or acute dysfunction in ADHF .Ascites, peripheral edema, elevated jugular venous distension, abdominojugular or hepatojugular reflux, third heart sound, and worsening mitral or tricuspid regurgitation murmurs are among the physical exam findings that include pulmonary crackles in the presence of pulmonary edema and pleural effusions. Furthermore, decreased organ perfusion in the context of decreased cardiac output may exacerbate end-organ damage. The augmentation of chronotropy and inotropy through increased ventricular pressures and neurohormonal compensatory mechanisms can lead to tachycardia, arrhythmias, myocardial strain, and ischemia.

PATHOPHYSIOLOGY OF ADHF:

The degree of systolic and diastolic heart failure, the relative contribution of the right and left ventricles, the arterial and venous blood flow, the neurohormonal and inflammatory response state, and concurrent contributing influences are some of the variables that affect the pleiotropic pathophysiology of ADHF.

INTRAVASCULAR CONGESTION: Intravascular congestion is directly linked to the most prevalent symptoms and indicators of ADHF. This congestion can arise from a gradual build-up of fluid through interrelated mechanisms, such as sodium retention from renal failure, dietary mismanagement, or noncompliance with medication regimens, or from elevated left ventricular filling pressures that lead to increased central and pulmonary venous congestion. When assessing the causes of congestion, concurrent or subsequent valvular disease—particularly mitral regurgitation—should be taken into account as another contributing factor. Retrograde blood flow from mitral regurgitation raises pulmonary pressures and congestion directly. Vasodilators and the start of diuresis are two therapies that target these pathophysiologic mechanisms in an effort to improve the clinical signs and symptoms of intravascular congestion.

VENOUS AND ARTERIAL VASOCONSTRICTION: A combination of preexisting pulmonary hypertension, increased left ventricular filling pressures, and hypoventilation-related pulmonary artery vasoconstriction frequently results in increased pulmonary arterial pressures, which are also commonly observed in ADHF patients. The central venous system may experience a significant volume redistribution as a result of increases in peripheral and splanchnic venous vasoconstriction. Significant and quick increases in central venous pressure are brought on by this redistribution and direct central venous vasoconstriction, which may deteriorate right ventricular and renal function.

NEUROHORMONAL SIGNALING AND CIRCULATING BIOMARKERS: The kidneys secrete the enzyme renin, which tells angiotensinogen to activate angiotensin I. Vascular endothelium releases ACE (angiotensin-converting enzyme), which cleaves angiotensin I into active angiotensin II. Systemic vascular resistance is raised as a result of angiotensin II's signaling of vasoconstriction, which occurs both directly on vascular endothelium and through the secretion of vasopressin and norepinephrine. Additionally, it triggers the release of aldosterone from the adrenal glands and activates renal sodium transporters to boost glomerular sodium reabsorption. Aldosterone increases glomerular sodium and water reabsorption by acting directly on the kidneys. Signs of low renal perfusion or increased activation of the sympathetic nervous system trigger the renin-angiotensin-aldosterone system. The overall activation causes a paradoxical worsening of ADHF through increased volume retention and vasoconstriction.

Myocardial cells release BNP (B-type natriuretic peptides) in response to ventricular chamber dilatation brought on by an increase in volume or pressure. This signaling process causes vasodilation, a decrease in renin activity, and consequent diuresis. The ventricles produce BNP in an inactive form called preproBNP, which is subsequently broken down into active BNP and inactive N-terminal-proBNP by enzymatic cleavage to proBNP. In the event of atrial dilation, atrial myocardial cells discharge atrial natriuretic peptide (ANP), which is also signaled by sympathetic activity through β-adrenergic stimulation. Natriuretic peptide receptor A, or NPR-A, is bound by both BNP and ANP. It is a protein that is highly expressed in the kidney and vascular endothelium. Since natriuretic peptides are a marker of elevated risk for cardiovascular death and rehospitalization in patients admitted with ADHF, they can be used to identify clinical events that may be modifiable in the enrolled patient population. It has also been demonstrated that the shift in troponin during the initial stages of admission is predictive, indicating the potential for therapies to be able to improve intermediate-term clinical outcomes by focusing on the acute myocardial injury in ADHF.

ROLE OF INFLAMMATION: It is known that several cytokines, such as TNF (tumor necrosis factor), TGF-β (transforming growth factor-β), and IL(1 and 6), play a part in the the development of HF. There is reason for increased bacterial or endotoxin translocation in ADHF, which may be related to gut edema or relative hypoperfusion, even though the triggers of these inflammatory cytokine cascades may be secondary to the neurohormonal activation and oxidative stresses associated with ADHF. These results raise the possibility that anti-inflammatory signaling therapies could be useful in the management of ADHF.[3]

CAUSES OF ADHF: Dietary or pharmacological noncompliance, Systemic infection, Myocardial ischemia, Acute or worsening valvular insufficiency, Supraventricular tachycardias, Uncontrolled hypertension, Alcohol consumption, Cocaine, amphetamines, excessive bronchodilator use, sleep disordered breathing, Hyperthyroidism and hypothyroidism, Anemia, Pulmonary embolism, Peripartum cardiomyopathy are some of the important causes of decompensated heart failure.

Hypotension, Renal dysfunction, Older age, Male gender, Ischemic heart failure etiology, Previous heart failure hospitalizations, Respiratory rate on admission 30/min, Anemia, acute or chronic Hyponatremia, Elevated troponin T or I, Elevated pre-discharge B-type natriuretic peptide level, Left ventricular ejection fraction 40%, Comorbid conditions: Chronic obstructive pulmonary disease, Dementia, Cerebrovascular or peripheral vascular disease, Hepatic cirrhosis, Malignancy are some of the possible predictors of adverse outcome during hospitalization of acute decompensated heart failure.[4]

Common factors precipitating HF Hospitalization with Acute Decompensated Heart Failure:

Acute Coronary Syndrome
Uncontrolled hypertension
Atrial Fibrillation and other arrhythmias
Additional cardiac disease (eg, endocarditis)
Acute infections (eg, pneumonia, urinary tract infections)
Non adherance with medication regimen or dietary intake

Anemia
Hyper – or Hypothyroidism
Medications that increase sodium retention (eg, NSAID) [5]

DIAGNOSIS:

Initiating appropriate treatment for acute decompensated heart failure requires a prompt diagnosis. Failing to do so results in higher treatment costs, a longer hospital stay, and a greater need for mechanical ventilation support. Sadly, there is frequently overlap between the signs and symptoms of acute decompensated heart failure and other common medical conditions, especially chronic obstructive pulmonary disease. Furthermore, no single finding is ideal for diagnosis due to the heterogeneous nature of acute decompensated heart failure.

Chest radiographs showing interstitial edema and pulmonary venous congestion roughly twelve times increase the risk of acute decompensated heart failure. Patients with stable, compensated heart failure frequently have chronically elevated levels of B-type natriuretic peptide, making it challenging to interpret an elevated level in them. The features of assays for N-terminal B-type natriuretic peptide are comparable to those of assays for B-type natriuretic peptide, with the exception that the absolute values are approximately six times higher. By assessing left ventricular systolic function, diastolic functional class, valvular function, left atrial filling pressures, right ventricular systolic pressure, and inferior vena caval pressure, transthoracic echocardiography can be used to diagnose acute decompensated heart failure. Patients suspected of having acute decompensated heart failure should consider electrocardiography, complete blood counts, basic metabolic panels, and potentially thyroid function tests, renal function tests, measurement of electrolyte levels, glucose levels, transaminase levels, prothrombin time, troponin level, D-dimer level and arterial blood gas pressure and urinalysis.[6]

PHARMACOLOGICAL THERAPY FOR ADHF:

THE MAIN GOALS OF THERAPY INCLUDE: -

The main objective is to quickly relieve symptoms. Due to high ventricular filling pressures, the majority of patients exhibit volume overload (congestion) symptoms at presentation. Every endeavor ought to be developed to enhance end-organ perfusion as well. Arrhythmias should be managed, especially if they have the potential to worsen heart failure. Examples of these include flutter or rapid atrial fibrillation.

Along with specific information about medications and their role in treatment, this is an opportunity for patient and family education regarding the symptoms, management, and signs of heart failure. And lastly, being admitted to the hospital offers yet another chance to think about alternative treatment options, such as cardiac resynchronization therapy for people who maintain normal sinus rhythm but show signs of left ventricular desynchrony, coronary revascularization for patients with underlying ischemic heart disease, or investigational drugs for patients with refractory symptoms.

DIURETICS:

The fastest and most efficient way to treat congestion symptoms that require hospitalization for heart failure is through intravenous loop diuretic therapy. Doubling the starting dosage, adding a thiazide diuretic, or titrating with an MRA that has diuretic effects in addition to cardiovascular benefits can all be necessary to achieve effective diuresis. Prior to discharge, one of the main objectives of treatment is to eliminate the congestion's symptoms. Carefully measuring fluid intake and output, vital signs, standing body weight at the same time every day, and observing clinical signs and symptoms of congestion and hypoperfusion are all part of the monitoring process for heart failure treatment. Serum electrolyte, urea nitrogen, and creatinine concentrations are among the daily laboratory tests performed during active medication adjustment. Because increased volume status and vasoconstriction can contribute to elevated filling pressures, decongestion frequently necessitates not only diuresis but also modification of other guidelines-directed therapies[5,7].

DIURETIC RESISTANCE:

In ADHF, even with dose increases, diuretics may not be able to effectively control water and salt retention. An ADHF sub-population at high risk of morbidity and death is captured by the concept of diuretic resistance. This gradual decline in loop diuretic efficacy is caused by a number of mechanisms. Since loop diuretics are "threshold drugs," a sufficient dosage is required to produce a desired therapeutic outcome. A common reason for a lack of diuretic response is inadequate dosing, as indicated by the shift of the dose-response curve in HF.

The "braking phenomenon," which occurs when long-term loop diuretic administration reduces the natriuretic response, is another mechanism for diuretic resistance. Relative or absolute contraction of the extracellular fluid volume causes this phenomenon, which lowers the amount of solute delivered to the proximal tubule through mechanisms mediated by the sympathetic nervous system and the RAAS.

WAYS TO OVERCOME DIURETIC RESISTANCE: As a useful addition to loop diuretics, thiazide diuretics can be used. Usually, thiazides are administered orally as a single dose one hour prior to loop diuretic dosage. While this approach can be useful in overcoming diuretic resistance, it is important to closely monitor serum electrolytes and fluid status. Thiazides and loop diuretics together can cause severe volume depletion or electrolyte imbalances, such as hypokalemia and hypomagnesemia, which increases the risk of arrhythmias.[8]

POSITIVE INOTROPIC AGENTS: Currently, the only positive inotropic drugs are those that raise intracellular cyclic adenosine monophosphate (cAMP), authorized for the management of sudden cardiac failure. By stimulating β-adrenergic receptors, β -agonists activate adenylate cyclase, which subsequently catalyzes the conversion of adenosine triphosphate to cAMP. Some of them include dopamine, epinephrine, norepinephrine, phenylephrine, dobutamine, and isoproterenol.

VASODILATORS: As was previously mentioned, vasoconstriction, elevated systemic vascular resistance, and fluid retention are the outcomes of activating the sympathetic nervous system, rennin - angiotensin system, and other mediators. The main mechanism of action of vasodilators is usually described as arterial or venous. By acting as impedance-reducing agents, arterial vasodilators lower afterload and raise cardiac output reflexively. By increasing venous capacitance, venodilators reduce preload in patients with high cardiac filling pressures by alleviating symptoms of pulmonary congestion. In decompensated heart failure, nitroprusside, nitroglycerin, and nesiritide are the most often prescribed intravenous vasodilators.[9]

ULTRAFILTRATION IN THE MANAGEMENT OF ADHF:

In response to a transmembrane pressure gradient, ultrafiltration enables the extracorporeal removal of plasma water from whole blood across a semipermeable membrane. With volume removal rates as high as 500 ml/h, ultrafiltration provides a mechanism for the relatively quick and controlled treatment of volume overload. Since the urine produced by loop diuretics is hypotonic while the ultrafiltrate is isotonic, UF may be preferable to loop diuretics in that it removes more sodium (and less potassium) for the same volume loss.[8]

POST DISCHARGE PATIENT MONITORING TO IMPROVE OUTCOMES:

In high-risk groups, post discharge monitoring has been investigated using a range of devices that measure physiological parameters (such as blood pressure and weight), gather data regarding changes in symptoms, or use biomarkers to help guide subsequent therapy. Most hospitalized ADHF patients exhibit congestion signs and symptoms, and data from early device studies suggested that these patients experience increases in filling pressures in the time frame prior to the clinical episode.

Transitioning from the hospital to an outpatient setting, such as home or an extended care facility, has been recognized as a potential area of focus for enhancing results and lowering avoidable readmission rates.

The discharge plan's three main components are: supplying vital information regarding the patient's prescriptions at the time of discharge; (2) instructing the patient in identifying the indicators of worsening heart failure (and what to do if they do); and (3) scheduling a follow-up appointment with a physician as soon as possible after being released from the hospital.[10]

The main aim and objective of the study is to evaluate the usage of guideline directed medical therapy in patients with Acute De-compensated Heart Failure.

OBJECTIVES:

To analyse the guideline directed prescription pattern in patients with ADHF.

To promote effective medical care in case of irrational prescribing which refers to prescribing that fails to follow good standards oftreatment and prevent the irrational use of medications.

To know the precipitating factors of ADHF.

To evaluate the number of medications given, dosage form, dose and frequency.

METHODOLOGY

A Prospective observational study was carried out in an inpatient department of general medicine in a tertiary care hospital. The duration of the study was 6 months i.e., from July 2023 to December 2023. Hundred patients were enrolled in the study.

INCLUSION CRITERIA:

1. Subjects diagnosed with Acute Decompensated Heart Failure
2. Subjects in age 20 years and above.
3. Subjects with previous history of heart failure.
4. Subjects of either gender.
5. Subjects with and without comorbidities.

EXCLUSION CRITERIA:

1. Subjects below 20 years of age.
2. Subjects from other departments of the hospital.
3. Pregnant and lactating women

RESULTS:

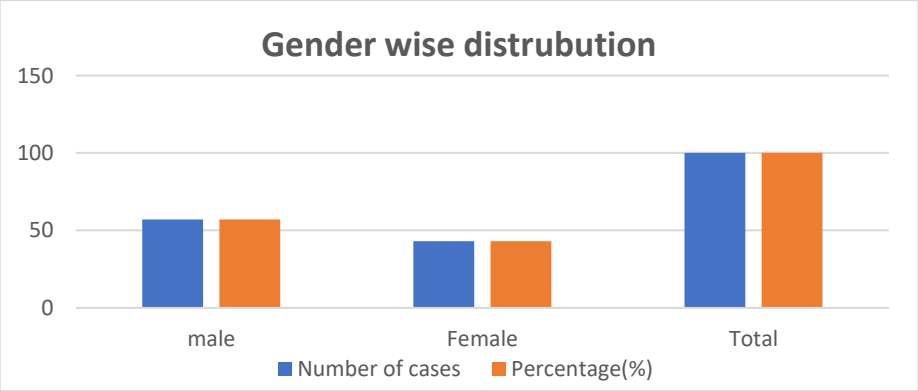

Figure 1: Gender wise distribution (n=100)

Figure 1 indicates that among 100 patients out of which 57(57%) were males and 43(43%) were females in our study.

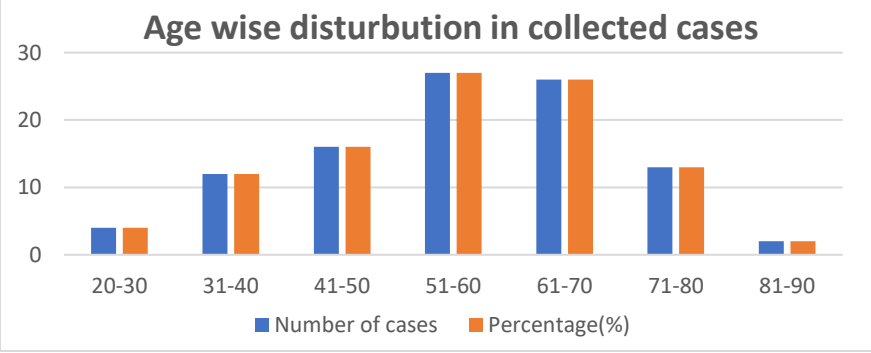

Figure 2: Age wise distribution in collected cases (n=100)

Figure 2 indicates that among 100 patients most of the patients were between age group of 51-60 (n=27; 27%) followed by 61-70 (n=26; 27%) and the least was 81-90 years (n=2 ; 2%)

Table 1: Distribution of patients according to duration of hospitalization (n=100)

S. No.	Duration of hospitalization(days)	Number of cases	Percentage(%)
1	2-3	8	8
2	4-5	15	15
3	6-7	45	45
4	>8	32	32
	Total	**100**	**100**

Table 1 indicates that out of 100 in-patients most of the patients had duration of hospitalization of 6-7days (45%) followed by >8 days (32%), and the least were 2-3 days (8%).

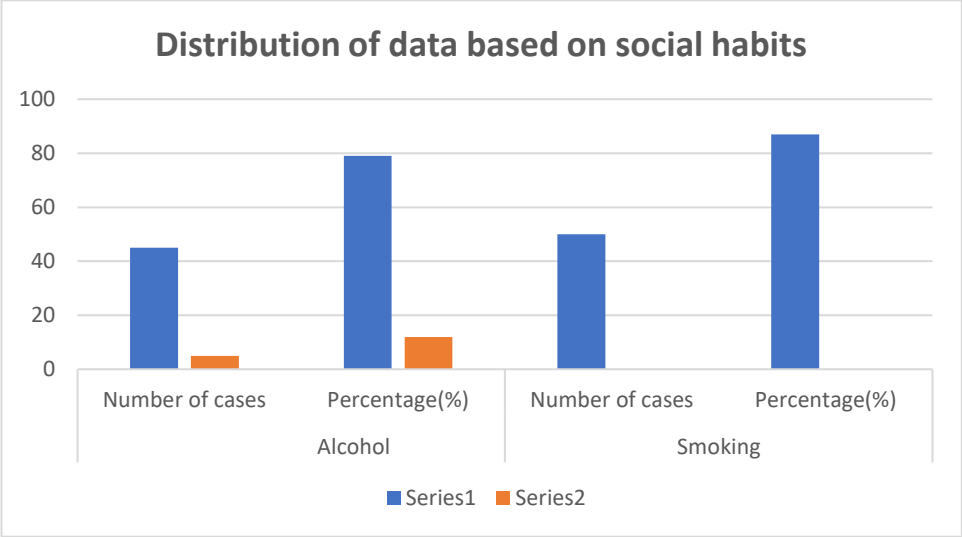

Figure 3: Distribution of data based on social habits (n=100)

Figure 3 shows men are more prone to alcohol (n=45; 79%) and smoking (n=50; 87%) than females.

Table 2: Distribution based on precipitating factors (n=100)

S. No.	Precipitating factors	Frequency	Percentage (%)
1	Lower respiratory tract infections	48	18.32
2	Dilated cardiomyopathy	26	9.92
3	Drug incompliances	10	3.82
4	Biventricular failure	10	3.82
5	COPD	4	1.52
6	Thyroid	8	3.05
7	Anemia	13	4.96
8	Hypertension	59	22.53
9	Diabetes	39	14.88
10	Cardio vascular disease	7	2.67
11	Acute kidney injury	10	3.82
12	Choronary artery disease	20	7.64
13	Cardio vascular disease	7	2.67
14	Acute kidney disease	1	0.38
	Total	**262**	**100**

Table 2 demonstrates common precipitating factor as Hypertension (n=59; 22.53%) followed by lower respiratory tract infection (n=48; 18.32%), then Diabetes (n=39; 14.88%) followed by Dilated cardiomyopathy (n=26; 9.92%). The least common precipitating factor for ADHF is Acute kidney disease (n=1; 0.38%)

Table 3: Distribution based on combination of precipitating factors

S. No.	Number of precipitating factors	Number of cases	Percentage (%)
1	1-2	20	20
2	3-4	32	32
3	>5	48	48
	Total	**100**	**100**

Table 3 indicates that 48 patients have more than 5 precipitating factors (n=48, 48%)

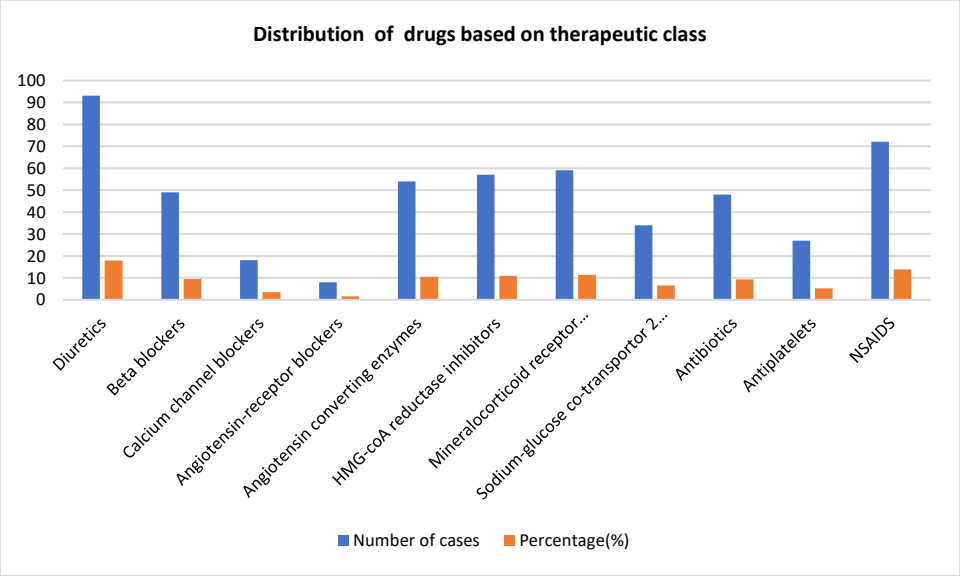

Figure 4: Distribution based on therapeutic class

Figure 4 shows drugs that are distributed based on therapeutic class, most common class of drugs were Diuretics (n=93; 17.92%) followed by NSAIDS (n=72; 13.88%), mineralocorticoid receptor antagonist (n=59; 11.36%) followed by HMG-COA reductase inhibitors (n=57; 10.98%)) and least common class of drugs are angiotensin receptor blockers (n=8; 1.55%)

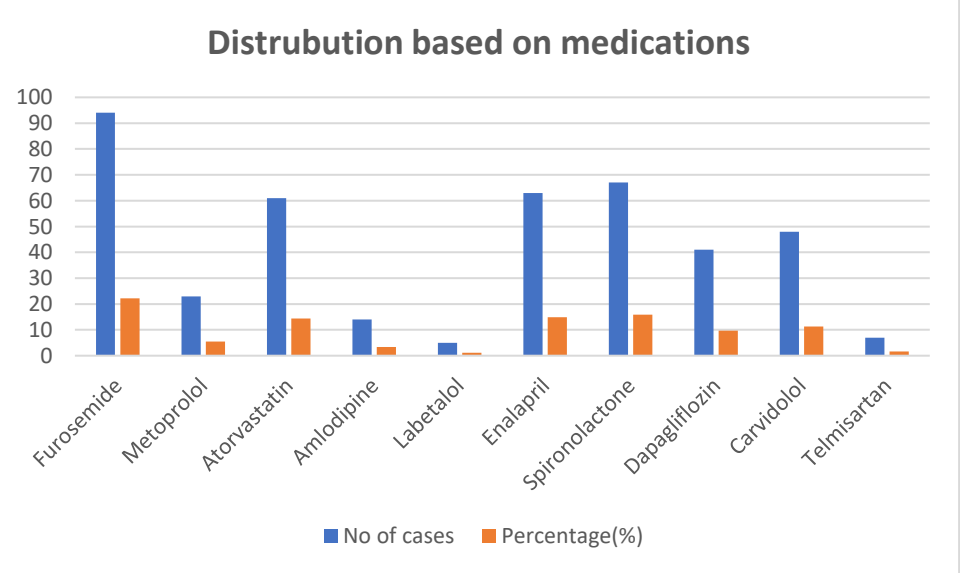

Figure 5: Distrubution based on medications

Figure 5 shows distribution of medications and the most common drug was Furosemide (n=94; 22.23%) followed by spironolactone (n=67; 15.83%), Enalapril (n=63; 14.89%) and the least common drug was Labetalol (n=5; 1.18%)

Table 4: Distribution of other drugs

S. No.	Drug name	Frequency	Percentage (%)
1	Pantoprazole	72	31.72
2	Ondansetron	40	17.62
3	Augmentin	60	26.43
4	Glimepiride	10	4.40
5	Metformin	29	12.77
6	Vancomycin	1	0.44
7	Gentamycin	3	1.33
8	Monocef	3	1.33
9	Clarithromycin	7	3.08
10	Digoxin	2	0.88
	Total	**227**	**100**

Table 4 shows distribution of other drugs, most common drug was pantoprazole (n=72; 30.72%) followed by augmentin (n=60; 26.43%), ondansetron (n=40; 17.62%) and the least common drug was vancomycin (n=1; 0.44%).

Table 5: Polypharmacy

S. No.	Number of medications	Number of cases	Percentage (%)
1	0-5	11	11
2	5-10	81	81
3	10-more	8	8
	Total	**100**	**100**

Table 5 shows that most of the patients were prescribed with 5-10 drugs (n=81; 81%) followed by 0-5 drugs (n=11; 11%)

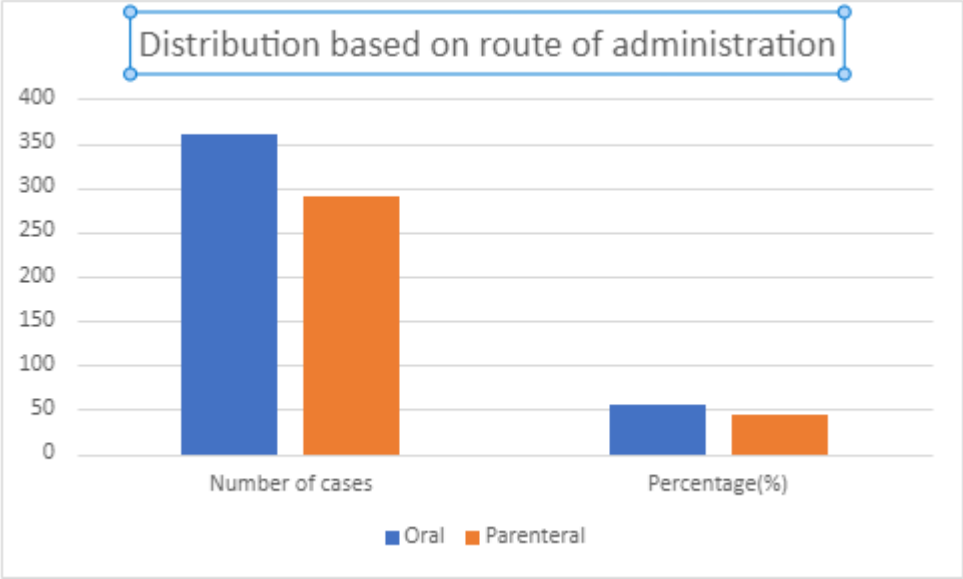

Figure 6: Distribution based on route of administration

Figure 6 shows that most of the drugs were prescribed orally (n=360; 55.38%), followed by parenteral (n=290; 44.62%).

STATISTICS:

LRTI (Lower Respiratory Tract Infections):

- **Chi-Square Test Result (p-value = 0.046):**
- The obtained p-value of 0.046 indicates a statistically significant association between age and the frequency of LRTI diseases.
- This suggests that there is evidence to reject the null hypothesis (H_0) that an increase in age doesn't lead to a higher frequency of LRTI.
- The chi-square test suggests that age might play a role in the occurrence or prevalence of LRTI diseases within our studied population.
- **Statistical Significance:**
- For LRTI, the result suggests a significant relationship between age and disease frequency.
- **Interpretation of Null Hypothesis (H0) and Alternative Hypothesis (H1):** • LRTI results suggest support for the alternative hypothesis, indicating that age might contribute to a higher frequency of LRTI.

Let's break down the interpretations based on the correlation coefficients and their significance levels:

1. **Gender:** • Notably, Gender shows very weak correlations with other variables. The correlations are all close to zero (ranging from -0.026 to 0.026), indicating an almost negligible linear relationship with Dilated cardiomyopathy(DCMP) , LRTI, Hypertension, and Diabetes. Additionally, all p-values are relatively high, indicating that these correlations are not statistically significant.

2. **DCMP:**

•Similar to Gender, DCMP also displays very weak correlations with other variables. The correlations range from -0.108 to 0.040, again indicating a minimal linear relationship. None of these correlations are statistically significant, as indicated by the high p-values.

3. **LRTI:**

LRTI has a stronger correlation with Diabetes (-0.276**), indicating a moderate negative linear relationship. This correlation is statistically significant at the 0.01 level, as denoted by the **.

LRTI also shows very weak correlations with Gender, DCMP, and Hypertension, all of which are not statistically significant (p-values > 0.05).

4. **Hypertension and Diabetes:**

Hypertension and Diabetes display weak correlations with other variables. Both show a negligible relationship with Gender, DCMP, and LRTI, none of which are statistically significant.

In summary, based on the Pearson correlation coefficients and their significance levels in this analysis:

Gender and DCMP have very weak, non-significant correlations with other variables.

LRTI shows a moderate, statistically significant negative correlation with Diabetes.

Hypertension and Diabetes exhibit weak, non-significant correlations with the other variables in the study.

DISCUSSION:

A prospective observational study was conducted among a said population with a sample size of 100 cases were taken into consideration based on our criteria from In-patients units of Department of General Medicine, Gandhi Hospital, Secunderabad. Assessment of the precipitating factors, age, gender, other co-morbid conditions, risk factors, and standard guidelines were taken into consideration in order to know the prescribing patterns in acute decompensated heart failure.

In our study the total percentage of male patients (57%) was comparatively more than that of female patients (43%) which is similar to the study reported by **P B Jayagopal (2022)**[11].

This study was carried out with the main agenda to examine prescribing pattern, the rationality of treatment in patients with ADHF. In our study, hypertension was found to be the most commonly seen precipitating factor which is similar to the study conducted by **Rohan P.**

Christian (2014)[12]

A study conducted by **Chi Nguyen (2020)**[13] reported that Diuretics were prescribed the maximum which is similar to our study where 17.92% of diuretic drugs were prescribed, since diuretics are the drugs that increase the rate of flow of urine for the excretion of sodium and chloride ions for the treatment of various diseases including hypertension and ADHF according to the prescription pattern of diuretics in a tertiary care hospital studied by **P G Chithara(2019)**[14].

In our study age wise distribution describes that high frequency of ADHF is seen in age group between 51-70 years of age (53%) and the precipitating factors found in our study were hypertension, diabetes, CAD, AKD, AKI, anemia and others which is similar to the study conducted by **Aparna S S (2022)**[15]

According to a study conducted by **Parag Goyal (2020)**[16] Metformin and NSAID are classified as major heart failure exacerbating medications but are prescribed to few patients with ADHF inspite of alternative drugs like acetaminophen and Sodium-glucose transport-2 inhibitors.

Our study included usage of cardiovascular drugs such as ACE Inhibitors, angiotensin receptor blockers, anti-platelets, calcium channel blockers, HMG-CoA reductase inhibitors, mineralocorticoid receptor antagonists, beta blockers. The most commonly prescribed non cardiological drug was pantoprazole (31.72%) as of study conducted by **Miral Bhuchhada**

(2022) [17]**.**

According to our study out of 100 patients most of the people were admitted in the hospital for 6-7 days which is similar to the study conducted by **Sri Harsha Onteddu (2020)**[18]

CONCLUSION:

This study provides prescribing patterns of drugs and rationality in general medicine department of a tertiary care teaching hospital. It had helped to identify the rational prescribing patterns of drugs using ACC/AHA guidelines.

The study revealed that most of the drugs were prescribed from the Essential Drug List by WHO and in accordance with the class and level of recommendation of drugs based on standard guidelines.

From our study we concluded that periodic evaluation of prescribing pattern according to guidelines is required to enable prescription of suitable medication to improve therapeutic benefits and to minimize adverse effect.

Conflict of Interest: No conflict of interest was found.

Future Direction of the Study:

1) This study will act as feedback to the prescribers so as to create awareness about the rational usage of medications.

2) Provides evidence-based treatment, Clinical trials and scientific data form the foundation of GDMT. Adhering to these recommendations makes it possible to guarantee that patients obtain therapies that have been demonstrated to enhance results.

3) Standardization of Care, by giving healthcare professionals a common framework, guidelines contribute to the standardization of medical care. Having this consistency can help improve overall patient outcomes.

4) Improves patient care as GDMT seeks to offer the most proven and efficient therapies for particular ailments. Healthcare professionals can enhance patient care and possibly even improve long-term results by adhering to these recommendations.

Quality Improvement: By bringing clinical practices into compliance with the most recent evidence-based

recommendations, the application of GDMT can support efforts aimed at improving the quality of care.

Limitation of the Study:

1. Short duration of study.
2. Restricted cases from department of cardiology and ICU.
3. Study conducted in a single centre and these results may not apply to the general population.
4. Further, there is a lacuna in assessing patient care indicator and facilities indicator which is to be assessed in drug prescription pattern.

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