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ANALYZING GUIDELINE - DRIVEN PRESCRIBING PATTERNS IN ACUTE DECOMPENSATED HEART FAILURE: AN OVERVIEW

Pharmaceutical Science

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ABSTRACT

Acute decompensated heart failure is a major global health burden that requires ongoing treatment strategy optimization. The results of several studies on guideline directed prescribing practices in the treatment of acute decompensated heart failure are compiled in this thorough review. The aim is to present a comprehensive review of the present state of affairs, pinpoint similarities, and emphasize differences in implementation. The review encompasses a wide range of studies, including randomized controlled trials, observational studies, and meta-analyses, spanning various geographical regions and patient populations. A systematic analysis of these studies reveals the prevailing adherence to established guidelines in disease management, emphasizing the importance of evidence-based practices. Using advised pharmacotherapies like beta-blockers, diuretics, Angiotensin Converting Enzyme Inhibitors, and angiotensin receptor blockers—is one of the main themes this review explores. Furthermore, the introduction of new drugs, like sodium-glucose cotransporter-2 inhibitors and angiotensin receptor-neprilysin inhibitors, is studied to comprehend how their role in treatment is changing. Additionally, the effect of following guidelines on clinical outcomes—such as death, readmissions to hospitals, and alleviation of symptoms—is evaluated. This review attempts to give a thorough overview of the current situation of guideline-directed prescribing and to provide guidance for future directions in patient care optimization by combining findings from various studies.

KEYWORDS

Acute decompensated heart failure, Guidelines, Overview, Drugs.

INTRODUCTION:

Acute Decompensated Heart Failure (ADHF) is characterized by the sudden or gradual onset of symptoms and/or signs of heart failure (HF), prompting the patient to seek urgent medical attention, resulting in unplanned hospitalization or an emergency department visit. This condition necessitates the initiation or intensification of treatment. ADHF can manifest as clinical deterioration in individuals with a prior HF diagnosis, termed acute decompensated HF, or 'de novo' in patients without a history of HF. Both scenarios may represent a new onset or exacerbation of pre-existing HF, requiring timely and comprehensive medical intervention to address the acute and potentially life-threatening nature of the condition.[1]

Even though subclinical congestion can appear for days or even weeks before an acute event and is the main cause of heart failure relapses, only 17–35% of rehospitalizations are attributable to HF exacerbations; the majority of HF admissions are related to non-cardiovascular (CV) conditions like sepsis, pulmonary disease, renal disorders, and arrhythmias.

International guidelines from the European Society of Cardiology and the American College of Cardiology emphasize how important it is to continue taking heart failure (HF) medications, such as beta-blockers (BBs), angiotensin-receptor blockers (ARBs), ACE inhibitors (ACEI), and mineralocorticoid receptor antagonists (MRAs), both after discharge and during acute HF episodes. With a class I recommendation, these medications are essential to the treatment of chronic heart failure. Even with these drugs' established advantages, doctors' compliance with prescribing them can be erratic, especially in the wake of acute decompensated heart failure. The Get With the Guidelines-Heart Failure (GWTG-HF) registry data showed varying rates of continuation; the most common time for MRAs to be stopped was during hospitalization. Younger age and use of medications prior to admission were predictors of post-discharge medication adherence.

This review's primary goal is to provide an overview of the medical treatment options available to patients with AHF (American College of Cardiology/American Heart Association (ACC/AHA)), with a focus on the variations among treatment approaches from various studies. Primarily this review stresses the usage of guidelines for the purpose of identifying issues in general public from various studies and literature which can be helpful in improving outcomes and treatment performance with targeted therapy.[2] Pathophysiology of Acute decompensated heart failure is shown in Fig. 1.

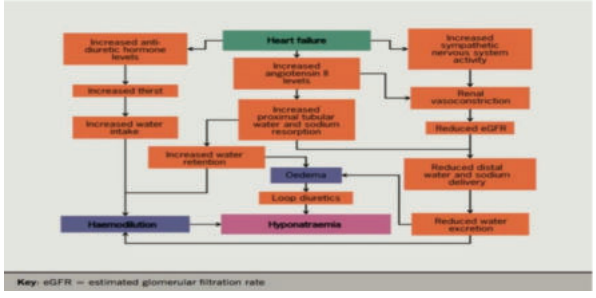

Figure 1: *Pathophysiology of Acute decompensated heart failure.[3]*

Precipitating Factors For Adhf: [4-5]

The absolute factors that lead to precipitation of ADHF are pathogenic afflictions caused by bacteria/virus/fungal and parasites in patients, acute coronary syndrome, myocardial infarction, anemia, uncontrolled or untreated hypertension, renal impairment and non compliance are connected to the morbidity and mortality and their prevalence. Infections such as chronic obstructive pulmonary disease which is most commonly seen since elevated pressure on lungs causes hypoxia and increases ventricular afterload. Furthermore, viral infections can also have adverse effect on myocardial function and lead to myocarditis others include flu symptoms. Uncontrolled BP has its fair share in inadequate muscular responsiveness and resting ability of heart. Dietary incompliances, such as excessive intake of saturated fats, sodium, and low consumption of fruits and vegetables, can negatively impact heart health. The habits increase the risk of ADHF and is crucial in maintaining cardiovascular health. Anemia, which is deficiency of red blood cells or hemoglobin ultimately strains the heart and makes heart harder to pump to deliver appropriate oxygen to the tissues and leading to precipitation of heart failure.

These precipitating factors worsen the heart failure and leading to conditions such as fluid accumulation in lungs, cardiogenic shock, and right sided heart failure.[6]

Since inflammatory response is seen in ADHF a study was conducted by Yuji Nishimoto et al., to study the C-reactive protein at discharge

and mortality in hospitalized patients and concluded that a high C-reactive protein (CRP) level (>10mg/L) at discharge from acute decompensated heart failure (ADHF) hospitalization was associated with an increased adjusted risk of all-cause death at 1 year. The elevated CRP levels reflected a prolonged inflammatory response despite ADHF treatment. The excess mortality risk persisted across various patient subgroups, irrespective of left ventricular ejection fraction or other clinical factors. Notably, there was no significant excess risk for heart failure hospitalizations in the high CRP group. These findings suggest that CRP at discharge serves as a simple prognostic predictor for 1-year mortality in ADHF patients, reflecting underlying inflammation.[7]

Clinical Approach In The Treatment Of ADHF:

The management of Acute Heart Failure (AHF) involves a personalized, 7-item stepwise approach considering the patient's clinical profile, pathophysiology, precipitants, underlying cardiac pathology, comorbidities, iatrogenic harms, patient preferences, and ethical considerations. AHF patients often present with a 'warm-wet' or 'wet-cold' profile, indicative of congestion and/or hypoperfusion. Further categorization based on fluid distribution and systemic hypoperfusion guides therapeutic decisions. Identifying high-risk patients with unstable vital signs is crucial, and early interventions involving drugs, supplemental oxygen, ventilatory support, and temporary mechanical circulatory support aim to restore perfusion status and limit end-organ damage. The dynamic nature of AHF underscores the importance of timesensitive interventions for improved outcomes, emphasizing positive pressure ventilation initiation, prompt diuretic administration, and, if clinically suspected, determining the presence and severity of cardiogenic shock. Early differentiation guides appropriate management strategies, optimizing the chances of recovery.[1]

DIURETICS:

Effective fluid management is crucial in acute decompensated heart failure (ADHF), where fluid overload is common. Swift initiation of diuretic therapy is vital, associated with improved outcomes. Diuresis reduces intravascular volume, lowering central venous and pulmonary pressures, alleviating pulmonary edema, and enhancing cardiac output. Despite diuretics' efficacy in relieving congestive symptoms, their aggressive use, especially in refractory cases, poses challenges. Complications include electrolyte imbalances, arrhythmias, hypotension, and renal dysfunction, impacting prognosis. Higher diuretic doses may be linked to worse outcomes, although causation is complex. Observational evidence supports diuretics as a mainstay for ADHF, emphasizing their role in symptom relief despite potential adverse effects.

Because of their effectiveness and tolerability, loop diuretic agents are the first-line therapy for acute decompensated heart failure (ADHF). High doses of them can be taken without risk of ototoxicity, an uncommon diuretic side effect. Intravenous administration is preferred for improved bioavailability in hospitalized ADHF patients. Individualized dosages are used, starting with intravenous doses of torsemide (10–20 mg), bumetanide (1 mg), or furosemide (40 mg). Given its potential to inhibit the aldosterone cascade and its distinct sympathetic nervous system actions, trometemide may present special advantages. Furosemide, however, continues to be an essential and reasonably priced diuretic for HF patients until clear clinical superiority is established. This is especially true when taking concurrent medication effects into account.[8] Mechanism of Diuretics is shown in Figure 2.

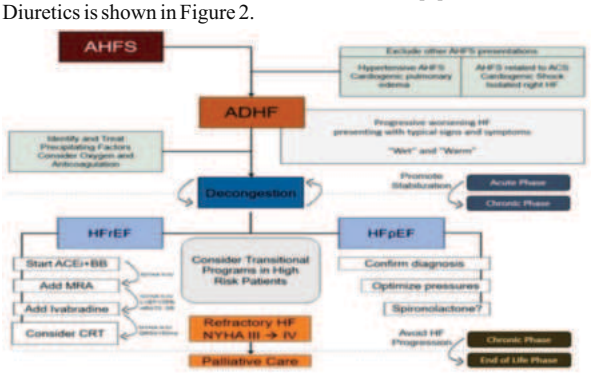

Figure 2: *Mechanism of Diuretics.[9]*

VASODILATORS:

In order to treat acute decompensated heart failure (ADHF), one must comprehend congestion. Equilibrium is maintained in chronic heart failure by compensatory mechanisms. Disturbances cause tissue and intravascular congestion by increasing fluid pressure through vasoconstriction. Dyspnea, edema, and exhaustion are symptoms of decompensation that lead to multiorgan dysfunction. Fast blood volume influx promotes subendocardial ischemia by exacerbating myocardial dysfunction. The diuretic-focused treatments that are currently available try to remove fluid without restoring proper distribution, which may lower plasma osmolality. While high-dose diuretics reduce symptoms, their overall prognosis might not improve. Taking care of the fluid distribution in organ tissues may provide a more thorough method of managing ADHF. Vasodilators like nitroglycerin can improve blood distribution in acute decompensated heart failure (ADHF), but their effectiveness in clinical outcomes falls short due to inappropriate usage. Administering vasodilators promptly to non-hypotensive ADHF patients can preserve cardiomyocytes, enhance recovery, and mitigate symptoms. Correcting the ischemic cascade early is vital for long-term prognosis optimization.[10]

BETA BLOCKERS:

In-hospital mortality risk significantly lowers when used beta blockers, irrespective of ischemic etiology and left ventricular ejection fraction at admission. Previous studies showed varied outcomes for beta-blocker use in ADHF, with potential benefits in reducing sympathetic activation, suppressing atrial fibrillation, and prophylactic effects on sudden cardiac death. Unexpectedly, beta-blocker use was associated with lower noncardiovascular mortality, possibly linked to the mitigation of catecholamine excess during infection. While limitations exist, emphasizing the importance of early beta-blocker administration in ADHF emerged as a key finding.[11]

ACE inhibitors/ARB:

Angiotensin-converting enzyme inhibitors (ACEI) and angiotensin II receptor blockers (ARB) are commonly prescribed medications for heart failure with reduced ejection fraction (HFrEF). They work by inhibiting the renin-angiotensin-aldosterone system, reducing the strain on the heart. Continuation or initiation of ACEI/ARB therapy in eligible patients has been associated with improved outcomes, including lower mortality and reduced readmissions. These medications are integral to guideline-directed medical therapy for HFrEF, aiming to optimize patient outcomes and prevent disease progression. Discontinuation of ACEI/ARB has been linked to higher mortality rates, emphasizing the importance of adherence to these medications in the management of heart failure.[12]

NON-Pharmacological Treatment:

Assisted ventilation: In patients with ADHF and cardiogenic pulmonary edema, oxygen supplementation and non-invasive ventilation methods, including continuous positive airway pressure(CPAP) and non-invasive intermittent positive pressure ventilation(NIPPV). However, standard oxygen therapy shows significant impact than non-invasive ventilation and demonstrated notable improvements in reducing dyspnea, heart rate, and hypercapnia.

Ultrafiltration:

Patients with acute heart failure (AHF) who are resistant to high diuretic doses may find that ultrafiltration (UF), which efficiently reduces excess fluid and salt, is a promising intervention. The UNLOAD trial showed improved weight loss, decreased 90-day readmission rates, and demonstrated the superiority of UF over conventional diuretic therapy. But UF may have negative effects as well; worsening renal function and an increase in adverse events were found in the later CARRESSHF trial when compared to standard therapy. The early termination of the AVOID-HF trial revealed that UF did not clearly outperform adjustable diuretic treatment. Although UF is recognized by ESC guidelines in cases of diuretic resistance, its precise function is still unknown and may only apply in cases of severe and unresponsive fluid retention.[12]

Observations From Other Similar Studies:

From the comparative study on usage of ACA/ACH guidelines in USA and Japan showed that the initial choice of treatment was diuretics mainly furosemide while the dosages differ in both USA and Japan whereas USA uses 20 to 40mg once or twice daily and 40-80mg daily in Japan. Usage of thiazide diuretics was prominent in USA rather than

Japan. Vasodilators such as nitroglycerin, isosorbide dinitrate, sodium nitroprusside and nesiritide are in use in USA and Japan and there is no notable difference in their usage and are not considered standard therapy. Dobutamine and milrinone are used for inotropic therapy in both the countries but due to its lack of promising effect on quality of life in USA other alternatives were used. In contrary, dobutamine was preferred in Japan. [13]

From the study conducted by Eric J. Velazquez to study the Angiotensin-Neprilysin Inhibition in acute decompensated heart failure. Enrolled subjects were given with enalapril and sacubitril valsartan in equal numbers and examined the outcome and reported that NT-proBNP levels markedly decreased in patients receiving sacubitril valsartan than in patients received enalapril. The study revealed a notable reduction inthe concentration of high-sensitivity cardiac troponin T, a biomarker associated with myocardial injury and indicative of cardiac structural and functional anomalies, leading to a poorer prognosis in heart failure patients.

5 NT-proBNP, serving as a biomarker for neurohormonal activation, hemodynamic stress, and subsequent cardiovascular events, also demonstrated noteworthy changes. Interestingly, there were no statistically significant differences observed in the incidence rates of renal dysfunction, hyperkalemia, and symptomatic hypotension between the groups treated with enalapril and sacubitril–valsartan. Recommended dose of sacubitril(49mg) and valsartan(51mg) and enalapril(2-5mg) twice daily based on guidelines. Moreover, an analysis of exploratory clinical outcomes indicated that the in-hospital initiation of sacubitril–valsartan therapy was associated with a lower rate of rehospitalization for heart failure at 8 weeks compared to enalapril therapy. These findings suggest potential benefits and improved outcomes with sacubitril–valsartan in the management of heart failure. [14]

In a randomized, placebo-controlled trial involving heart failure patients with reduced left ventricular ejection fraction by John J.V. McMurray et al., dapagliflozin (10mg) once daily demonstrated significant cardiovascular benefits. The primary composite outcome, encompassing worsening heart failure (hospitalization or urgent visit with intravenous therapy) or death from cardiovascular causes, was markedly lower in the dapagliflozin group compared to the placebo group. Each component of the composite outcome, as well as total hospitalizations for heart failure and cardiovascular deaths, exhibited reduced incidence with dapagliflozin. Notably, patients receiving dapagliflozin reported fewer heart failure symptoms, measured by the Kansas City Cardiomyopathy Questionnaire. Remarkably, dapagliflozin's efficacy extended beyond patients with type 2 diabetes, proving equally effective in those without diabetes. The observed benefits were consistent across various subgroups, although there was a potential variation in treatment benefit among patients in different NYHA functional classes. Adverse effects were infrequent, with minimal occurrences of major hypoglycemia and diabetic ketoacidosis, primarily in patients with diabetes. While the trial had specific criteria, limited representation of certain demographics, and low baseline use of sacubitril–valsartan, the findings suggest dapagliflozin's potential as a therapeutic option for heart failure patients with reduced ejection fraction, irrespective of diabetes status.[15]

From the studies conducted by Bertram Pitt et al., and Essraa Bayoumi et al., on the usage of spironolactone(12.5-25mg) once daily for ADHF with preserved ejection fraction reported that the TOPCAT trial, which had sufficient power to assess its primary composite outcome in heart failure patients with preserved ejection fraction, found no evidence to support the hypothesis that adding spironolactone to current treatment would reduce the risk of cardiovascular death, aborted cardiac arrest, or heart failure hospitalization. The study discovered that taking spironolactone did not significantly change the length of time until a patient's first hospitalization or death from any cause. Only the rate of hospitalization for heart failure demonstrated a decrease in the spironolactone group, despite the fact that the primary outcome components were vital. Based on the eligibility stratum, subgroup analyses showed a significant treatment interaction that highlighted regional differences and unexpected event rate discrepancies. Limitations, however, such as adverse effects and stopping the medication, highlight how difficult it is to treat heart failure with preserved ejection fraction when using spironolactone. Spironolactone did not show a significant overall improvement in the composite primary endpoint in the former and latter suggested reliable, nevertheless, clinical efficacy of aldosterone antagonist in patients with ADHF. [16-17]

Polypharmacy is the practice of a person taking several medications at the same time, usually four or more. It is typical of people with long-term illnesses and can result in more complications, possible drug interactions, and elevated risks. In particular for older adults, careful administration is essential to guaranteeing the safety and efficacy of medications.The real-world study conducted by Neiko Ozasa et al., in 16,052 patients regarding polypharmacy and clinical outcomes with heart failure (HF) revealed that at hospital discharge, the median number of medications was 8, with 81.5% receiving over 5 medications. Patients on 12 or more medications had a significantly higher adjusted risk for death or hospitalization in the first year postdischarge. This study highlighted the complex medical and social backgrounds of older HF patients, often burdened with multiple comorbidities and polypharmacy. Dementia, social isolation, and unemployment were prevalent, complicating HF management. These findings underscore the importance of judicious prescribing, individualized medication reviews, and potential interventions like outpatient clinics specializing in polypharmacy to optimize outcomes in this vulnerable population.[18]

Similarly , a study by Ozan Unlu et al., on polypharmacy in older adults hospitalized for heart failure reported high prevalence of polypharmacy, with over half taking at least 10 medications. The prevalence of polypharmacy increased over time, emphasizing the urgent need for strategies to manage medication burden effectively. Most medications were non-cardiovascular, reflecting the significant comorbidity burden in this population. The study suggests reconsideration of a polypharmacy cutoff of 10 medications for risk assessment in HF patients, emphasizing the importance of addressing non-cardiovascular medications to optimize guidelinedirected medical therapy (GDMT) and improve outcomes in HF. Strategies to reduce unnecessary medications and prioritize GDMT warrant further investigation.[19]

Despite the challenges posed by the COVID-19 pandemic, the number of ADHF cases continued to rise which was studied by P.B. Jayagopal et al., and reported that there was demographic shift with younger patients getting admitted. Notably, there was an increase in mortality rates among aged 41-50 in 2020, contrasting with a decrease in the >80 age group. Awareness of the importance of timely hospitalization for ADHF during pandemic was crucial but various factors stood barrier for it such as limited public transport, redirection of cardiac units to COVID wards, and patients fear of acquiring COVID in hospitals. However, incidences of comorbidities like COPD and CVA decreased during lockdown. [20] and similar observations were made by Mohammed Yousufuddin et al., and gave an insight that a decline in HF hospitalizations and a rise in 30-day mortality during the pandemic. Despite consistent treatment patterns and procedures, a higher proportion of patients were admitted to non-cardiology services. Patients during the pandemic had lower comorbidity burdens, and a decline in HF hospitalizations was seen across age groups. A sensitivity analysis supported main findings. The decline in readmissions may be due to COVID-19 fears, telemedicine adoption, and reduced triggers, while increased mortality could result from COVID-19-related factors and healthcare access disparities.[21]

Since diuretics play an important role in the treatment of ADHF to relieve congestion and maintain optimal fluid balance, an In-Hospital observation was made by Julan B. Ivey-Miranda et al., on oral diuretics after treatment for acute decompensated heart failure and reported poor correlation between weight change and net fluid balance metrics and further research is needed for a prospective, randomized controlled trail to establish the utility of oral diuretics definitively. [22] assessing and treating congestion in ADHF is very crucial as discussed by Alexandre Mebazza et al., and concluded that acute decompensated heart failure (ADHF) is characterized by water retention and venous congestion, often leading to high mortality and prolonged hospital stays. Traditional assessment methods lack consistency, resulting in residual congestion for many patients upon discharge. Recent trials, ADVOR and EMPULSE, reveal promising strategies using acetazolamide and SGLT2 inhibitors, respectively, to effectively alleviate congestion during hospitalization. However, despite advancements, a substantial number of patients still experience post-discharge congestion. A proposed contemporary approach involves

serial congestion assessments, combining intravenous furosemide-acetazolamide with oral furosemideSGLT2 inhibitors, emphasizing the need for aggressive short- and long-term management to prevent recurrences and improve outcomes in acute heart failure.[23]

While a study conducted by Sivadasanpillai Harikrishnan et al., on clinical profile and outcomes in patients admitted with ADHF and summarized that Despite improvements in guideline-directed medical therapy (GDMT) prescription, socioeconomic differentials persist in HF mortality, emphasizing the need for targeted interventions in high-risk groups. Although overall mortality rates have improved, further efforts are warranted to enhance GDMT uptake, reducing mortality and optimizing heart failure management in the Indian context.[24]

CONCLUSION:

Finally, it should be noted that acute decompensated heart failure (ADHF) is a serious and possibly fatal illness that requires immediate attention. In the context of ADHF, the value of Guideline-Directed Medical Therapy (GDMT) cannot be emphasized. According to accepted clinical guidelines, GDMT is a customized set of pharmacological and non-pharmacological interventions that have been shown to be effective in improving outcomes for people with heart failure. The importance of GDMT is found in its potential to alter the course of the disease, lower hospitalization rates, and increase survival rates in addition to its ability to relieve symptoms and improve quality of life. Following GDMT guarantees optimal blood pressure regulation, fluid retention control, and neurohormonal pathway modulation—all essential for controlling acute exacerbations and the long-term progression of heart failure. Moreover, GDMT is a comprehensive strategy that calls for cooperation between patients and healthcare professionals in order to establish a partnership that is intended to accomplish treatment objectives and lessen the effects of ADHF. Adopting and using GDMT strategies is becoming more and more important as research and clinical practices develop in the fight for improved long-term prognoses and better outcomes for patients suffering from acute decompensated heart failure. To put it simply, GDMT is a vital component of the all-encompassing care pathway that heart failure patients can take to improve their well-being, better control over their symptoms, and eventually, a better prognosis.

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