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An Overview of Medical Management of Acute Decompensated Heart Failure

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

ADHF is a heterogeneous clinical syndrome that usually leads to hospitalization due to a combination of interconnected renal dysfunction, cardiac dysfunction, and vascular compliance. Hospitalizations from ADHF are linked to increased morbidity and mortality, with about half of the

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patients on readmission within six months and short-term cardiac mortality. Importantly, the overall long-term outcome is still poor, combining rates of cardiovascular death, hospitalization for heart failure, myocardial infarction, and stroke. Managing these patients remain a challenge, with an emphasis on end-organ perfusion (coronary and renal), primarily volume control and reduction of vascular resistance.

Keywords: Digoxin; afterload; funny current inhibitor; furosemide; pitting edema.

1. INTRODUCTION

ADHF is a group of symptoms which include dyspnea, progressive weakness and leg swelling that occur from a weakened heart. This can lead to scheduled or unplanned medical attention [1]. The primary target of managing ADHF patients is to discover and treat the cause of decompensation [2]. Based on the 2017 AHA data, about 6 million Americans at 20 years and beyond are affected by heart failure [3]. With a fair improvement of patients with acute MI, heart failure keeps on to become the main health challenge in the United States. From the data provided by AHA in 2012-2030, there is more than 40% rise in heart failure; this results in about 7 million people at age 18 years and above living with heart failure in America [3]. The pathophysiology of HF lies on the factors involving the level of cardiovascular disorder, abnormalities with both ventricles, etc [4]. The decompensated HF begins when the compensated heart failure has gained stability between the pre and afterloads. Additionally, lungs injuries, and organ impairments can cause HF [5].

2. CAUSES OF ADHF

Medication nonadherence; NSAIDs which leads to sodium retention; hypertensive emergency; acute endocarditis leading to severe reflux; acute dilated cardiomyopathy; cardiac tamponades; advanced HF (eg. paget's disease, thyrotoxicosis, beriberi, sepsis) [6].

3. MEDICAL MANAGEMENT

The main objectives of treatment for heart failure are to record good prognosis, decrease symptoms, and ultimately decrease morbidity and mortality. Additionally, it also includes: reducing the duration of hospital stay and frequency of returning to the hospital for admission, prevent end-organ injury, and to adequately manage co-morbid conditions that may worsen the outcome [7]. The 2010 HFSA (heart failure society of america) guidelines, the updated 2013 ACA/AHA (american college of cardiology/american heart association) guidelines, and the 2008 ESC (european society of cardiology) guidelines, all provide different levels of evidence for the management of HF patients. This reports management of HF cases will be under two categories; In-hospital and out-patient care [8,9,7]. In-hospital management: it is so recommended to admit the patient to the telemetry bed or the intensive care unit (ICU) and provide care based on the - oxygen levels (PaO2 less than 60% or SaO2 less than 90%), and noninvasive positive pressure ventilation (NIPPV) given in cases with respiratory distress to support ventilation. Several pharmacological agents can be administered depending on presenting signs/symptoms and on the contributing factors: Diuretics: loop diuretics, thiazides, and potassium-sparing diuretics for years have been the mainstay for managing HF cases. Loop diuretics inhibit NA-K-2CL cotransporter on the luminal membrane of the ascending limb of the loop of henle to reduce renal reabsorption of sodium chloride. Examples are furosemide, torsemide, bumetanide, and ethacrynic acid as an option in cases of sulfa allergy [10,11]. Diuretics reduce volume overload in HF. ACEIs/ or ARBs/angiotensin-neprilysin receptor. Blockers: this category works by suppressing the renin-angiotensin-aldosterone system (RAAS) and is considered the gold standard to modulate RAAS in HF patients for more than two decades [12]. ACEIs block the conversion of angiotensin one to angiotensin two. However, it potentiates bradykinin accumulation which promotes both positive and negative effects. ARBs are the alternatives to ACEIs in patients that cannot tolerate ACEIs. ARBs specifically block the angiotensin ii receptors and do not decrease the breakdown of bradykinin online ACEIs [13]. A newer therapy class, the angiotensin receptor neprilysin inhibitor (ARNI), was brought about by adding a neprilysin inhibitor to angiotensin receptor inhibition [14]. Being an endopeptidase, neprilysin breaks down various endogenous vasoactive peptides [11]. These effects lead to appreciable hypotensive outcomes and are massively beneficial in comparison with ACEIs or

ARBs. The ARNI is fast replacing ACEI/ARB as the gold standard RAAS blocker due to its clinical advantages for HF cases [11,5]. Beta blockers (BBs): by inhibiting the actions of the sympathetic nervous system, BBs have shown improved outcomes for HF management. Subsequently, BBs decrease ischemia of the myocardium and possesses antiarrhythmic effects which help to maintain energy levels required for myocyte function by enabling glucose instead of fatty acid metabolism. As well, beta blockers inhibit renin secretion [15]. Mineralocorticoid receptor anatagonists (MRAs): this class of drugs prevent the binding of aldosterone to the mineralocorticoid receptors, mostly found in the vascular smooth muscle cells and in the myocardium. Furthermore, they demonstrate antifibrotic effects by suppressing the production of matrix metalloproteinases and other factors that contribute to myocardial remodelling [10,12,14,15]. Sodium-glucose cotransporter - 2 (SGLT-2) inhibitors: recently proven to be efficacious in treating Hf, especially HF with reduced ejection fraction (HFrEF). It increases renal glucose excretion by blocking SGLT-2, promoting osmotic diuresis and natriuresis [16]. Although it was originally manufactured as an anti-diabetic agent, it has clinical benefits for HFrEF in people living with diabetics and people who don't have diabetes [12]. Ivabradine: people benefit from the use of this drug as a result of its inhibition of the inward rectifying potassium channel (funny currents inhibitor), which slows depolarization during diastole eventually reducing the heart rate [13]. Thromboembolism risk is reduced by anticoagulants if needed. Vasodilators: such as hydralazine and nitrates combined are substantially considered to improve patient symptoms. Hydralazine is a direct arteriolar vasodilating agent whereas nitrates primarily mediate venodilation, lowering ventricular preload [14]. Oral soluble guanylate cyclase stimulators: example - vericiguat. Lately, found to enhance positive end results among HF patients at high risk. Its main mechanism of action involves boosting endogenous nitric oxide by directly binding to and stimulating the soluble guanylate cyclase [11]. Positive oral inotropes: restores perfusion and decreases congestion in HFrEF patients so as to increase cardiac output. It includes digoxin and omecamtiv mecarbil. The latter is a new drug, acting as an activator of cardiac myosin beneficially improving the left ventricular function [15,9]. In summary, the proper medical management should follow the steps below: reduce preload (preferably with loop diuretics). Administer oxygen if hypoxemia is present. Reduce afterload with ACEI if systolic blood pressure measures more than 100mmHg. Intravenous agents are recommended if hypertensive crisis is present (i.e., sodium nitroprusside). Increase inotropy if there are evidences of hypoperfusion (i.e., milrinone, dobutamine), but beware of increase in ventricular arrhythmias with inotropic agents. Digoxin reduces hospitalizations and improves clinical symptoms, but does not lower mortality rate. Consider adding digoxin when patients have symptoms despite adequate therapy with ACEI, β-blockers, and AA. NB: β-blockers are successfully used later when heart failure is compensated and stroke volume improves. They can worsen decompensated heart failure by their inotropic effects. Mineralocorticoid antagonist e.g,. spironolactone, which helps reduce morbidity and mortality. Funny current inhibitor e.g,. ivabradine which significantly increases survival rate by 120% and prolongs average survival time by 20%. Angiotensin receptor-neprilysin inhibitor, e.g,. sacubitril/valsartan. Certain agents from the above listed have been demonstrated to significantly lower morbidity and mortality, hospitalizations, and prevent cardiac remodelling. They are; beta blockers, ACEIs/ARBs, MRA, and ARNI (sacubitril/valsartan) [10-14]. SGLT-2 inhibitors also highly improve quality of life when incorporated into current standard drugs [12,14,15]. Out-patient management: [9,7] counsel has to be done to meet patients needs, patient education, promote self-care, strategies to enhance patient medication adherence, observing signs and symptoms of fluid overload, regular follow-up, access to healthcare services and assistance as required.

4. CONCLUSIONS

Acute decompensated heart failure has become a regular reason for hospital admissions, and it is associated with a tall chance of revising the hospital. Management of these patients include: diet modification, exercise, and drugs.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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