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Congestive Heart Failure: Treatment of Symptoms or Causes

Daiyuan Wang[1,2*], Jing Wang[1]

[1]Russellville Cardiology Consultant, Russellville, AR, USA
[2]Department of Cardiology, St. Peter's Hospital, Albany, NY, USA
Email: *daiyuanwang123@gmail.com

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Abstract

This paper is based on the author's 20+ years of experience treating patients with congestive heart failure (CHF) as a cardiologist. In the 20+ years, 64 patients were treated, including both with reduced and preserved left ventricular function. Most patients had a 4 - 5 days hospitalization in their first admission with one readmission (1.6%) over seven years. This paper will help us understand the physiology and pathophysiology of congestive heart failure, especially how to use beta blockers and diuretics. It will shorten the length of hospitalization and lower the readmission rate and cost of CHF treatment. This paper will help us to open another research direction for CHF.

Keywords

Congestive Heart Failure, Beta-Blocker, Diuretics

1. Introduction

Congestive heart failure (CHF) is a medical condition that causes significant morbidity and mortality. By 2030, US heart failure costs are expected to be at least 70 billion per year, with 75% - 80% of the direct costs for heart failure attributable to inpatient hospital stays [1]. Patients with heart failure requiring inpatient admission are a highly vulnerable population and have a poor prognosis, with nearly 1 in 4 heart failure (HF) patients readmitted within 30 days of discharge, and approximately half are readmitted within six months [2]-[4]. Improved treatment of CHF is essential for improving the quality of the patient's life and lowering the health care cost. Effective treatment of CHF starts with a better understanding of the physiology of the cardiovascular system and the pathophysiology of CHF.

The treatment for CHF exacerbation has usually focused on correcting fluid status since patients often present with shortness of breath, pulmonary edema, and peripheral edema, suggesting fluid overload. Diuresis has been the initial treatment [5] to correct fluid overload to euvolemia. However, in everyday practice, this treatment is not effective and is associated with prolonged hospitalization with significant complications and readmission. It raises a possibility that fluid overload is the symptom. Instead, it is a consequence of the dysfunction of the cardiovascular system since one of its essential functions is fluid metabolism. The treatment should focus on the restoration of function of the cardiovascular system. Diuresis should be a tool to correct the function of the system. It is important to realize that inappropriate diuresis will further disturb the system, leading to difficulty or failure in treatment, extended hospital stays, and potential complications such as acute renal insufficiency. Inappropriate outpatient diuretic use may also set the stage for recurrent CHF exacerbation and readmission.

CHF patients are divided into two groups: reduced and preserved left ventricular systolic function. Each group may contribute to 50% of CHF admission. It suggests LV systolic function may not directly cause CHF exacerbation since patients with normal LV systolic function have CHF symptoms, and those with severely decreased LV systolic function may not have any symptoms of CHF exacerbation. The patients with CHF exacerbation in both groups may have a similar mechanism, which is diastolic dysfunction. They share a similar treatment strategy with some minor differences, which will be the center of this paper.

2. Method

This paper is based on retrospective and observational data. It is not planned research. Therefore, there is no control group or organized statistics. The author did not intend to publish the paper and only kept a simple case count for professional interests. The author did not have access to the patient's medical records when he planned to write this manuscript due to changing jobs.

Every patient and their family had complete and extensive education in fluid management, diuretics use, and other guideline-recommended medical treatments. It started with the inpatients' first encounter, followed by every clinical follow-up until they fully understood and followed the instructions. It usually needs 4 - 5 time encounters. They had continuous and regular clinic follow-ups based on their needs.

3. Results

In the author's more than 20 years of practice, 64 patients were treated, including 51 (80%) with reduced left ventricular function with left ventricular ejection fraction (LVEF) 15% - 30% and 13 (20%) with preserved left ventricular function. Most patients had a 4 - 5 days hospital stay with their first hospitalization. One patient was readmitted once (1.6%). In 51 patients with reduced LVEF,

LVEF fully recovered in 45 patients (88%), and LVEF partially recovered to 30% - 35% in 6 cases (12%). Every patient had lived a normal daily life. They had no dyspnea on moderate daily physical activities consistent with their ages. No patient needs diuretics daily as outpatients. A few of them required diuretics occasionally based on their daily weight. Two cases were transferred to the tertiary center due to cardiogenic shock and severely decreased LV systolic function, requiring device support at their first presentation. My office did not follow them. They are not in the 61 cases reported. All the patients were on guideline-recommended medical treatments such as ACE, spironolactone, and entrosto, with priority given to beta-blockers. The following two cases illustrate the management of CHF in the author's practice in the last 20 years.

Case #1 is a 62-year-old gentleman who presented with 2-week progressive exertional dyspnea and lower extremity edema. He used to exercise on a treadmill for 45 minutes regularly. On presentation, he could not walk 20 yards without shortness of breath. He has a history of hypertension. A physical exam revealed bilateral crackles and edema. Chest X-ray was consistent with pulmonary edema. An echocardiogram revealed normal LV systolic function with EF 60% - 65% and mild LV diastolic dysfunction with no significant valvular disease. His blood chemistry, CBC, and cardiac panels were normal. EKG showed NSR. His blood pressure was 150/85 mmHg, and his heart rate was 80 beats per minute. Furosemide 20 mg IVP daily started. The patient had 3 L urine on the first day and 1 L on the second day. Furosemide increased to 40 mg daily without an increase in urine output, and on the fourth day, urine output decreased to 500 cc with a darker color. Dyspnea was worse, and edema increased. The patient developed orthopnea and PND. His creatinine increased from 1.1 to 1.7. A cardiology consult was requested. Under the care of cardiology service, Furosemide was discontinued, and 250 cc NS was administered over 30 minutes. In 2 hours, the patient produced 350 cc urine. 50 mg Lopressor was given. Shortness of breath and orthopnea was mildly improved. Heart rate is 80 beats per minute, and blood pressure is 140/80. On day 5, fluid restriction 1.2 L/day, Lopressor 75 mg bid started. On day 6, his heart rate was 65 bpm, and his blood pressure was 124/75. He produced 1.5 L urine. Orthopnea and PND resolved. He was able to walk in the hallway without dyspnea. Lower extremity edema mildly decreased. On day 7, the patient was discharged home with metoprolol 75 mg PO bid and fluid restriction of 1.2 L/day with a 2-week follow-up. He still had edema, but it was better. Upon his return to the clinic for a 2-week follow-up, he reported that edema resolved at the end of the first week after the discharge. He resumed exercising on the treadmill and reached his previous level in a week. In 4-week, 3-month, 6-month, and 12-month follow-ups, his condition remained stable, and he was allowed fluid 2 L/day and lopressor 75 mg po bid without diuretics. In this case, fluid overload and diastolic dysfunction of the left ventricle triggered the CHF exacerbation. Over-diuresis shrank intravascular space, which resulted in an increased level of sympathetic activity. It caused an increase in

heart rate and blood pressure, which worsened LV diastolic dysfunction. It also causes vessel constriction, leading to decreased renal perfusion and reduced urine output. It was why the diuretics lost their effect. A quick volume expansion with bolus fluid challenge and beta-blocker restored cardiovascular function by decreasing heart rate and blood pressure. It resulted in the resolution of the symptoms and improved renal perfusion with increased urine output.

Case 2: An 82-year-old male with a history of CAD, 3 V CABG 15 years ago, CHF with LVEF 20% - 25%. He was on Lopressor 50 mg po bid, Lasix 120mg bid, lisinopril 10 mg daily. He presented with a 2-month history of progressive DOE, lower extremity edema, and 30-pound weight gain. Lasix dose has been increased from 40 mg PO daily to 80 mg PO to 120 mg and then bid with progressive worsening symptoms. The patient was admitted for CHF exacerbation and aggressive diuresis. On admission, His heart rate was 90 beats per minute, his blood pressure was 156/90, and his oxygen saturation was 85% in room air; his chemistry panel showed creatinine 2.9 (1.5 2 months ago) and moderate pulmonary edema on Chest X-ray. Echocardiogram reported LVEF 20% - 25% and mild LV diastolic dysfunction. During the physical exam, he was distressed and unable to complete a sentence without shortness of breath, bilateral crackles, 2+ lower extremity pitting edema, and inability to ambulate without significant dyspnea. The primary physician requested a nephrology consult, which recommended furosemide infusion at 5 mg per hour. The patient and family were informed that he would have a fair chance of needing hemodialysis due to his compromised renal function with creatinine 2.9 and advanced CHF. Hospice service was involved in the case due to his condition. It was his third admission in 12 months. Each time, his condition has been worse than the previous one. After a discussion of the case with inpatient cardiology, his treatment plan was changed: discontinue the diuretics, increase Lopressor from 50 mg bid to 100 mg bid, fluid restriction 1.5 L/day, and low sodium diet. Entrosto was discontinued. On day 2, he reported mild improvement in PND, orthopnea, and dyspnea. He produced 1000 cc of urine over 12 hours. His heart rate was 71 beats per minute, and his blood pressure was 136/70 mmHg. Chest X-ray showed improvement of CHF. 40 mg Furosemide IVP was given in the morning with a urine output of 1500 cc over 24 hours. Lopressor increased to 150 mg bid. His symptoms improved significantly. On day 3, the treatment of day two was repeated. He produced 2000 urine, and he continued to improve. On day 4, he reported the resolution of his symptoms with 2000 urine. He was able to walk for more than 100 yards without dyspnea. On day 5, he remained stable with 1200 fluid restriction, a low sodium diet, and 150 mg Lopressor bid. His heart rate was 62 beats per minute, and his blood pressure was 118/70. He received no lasix. He still had lower extremity edema, although it had improved when compared with that on admission. His creatinine was 2.1. He was discharged with fluid restriction of 1.2 L/day, low sodium diet, weight-based diuretics use, and Lopressor 150 mg bid, and He returned to the CHF clinic in 2 weeks. He reported no CHF symptoms

and was able to walk half a mile at a regular pace two times a day. His creatinine was 1.6. Entrosto started at a low dose and titrated to higher doses. On the 3-month follow-up, he resumed his regular exercise program. He was able to do light yard work. In this case, the cause for the exacerbation is not clear. It gradually developed over two months. Both excessive fluid intake and over-diuresis may contribute to it. The direct triggering factor was most likely LV diastolic dysfunction, which was corrected by the beta-blocker and appropriate diuresis. Bata-blocker decreased heart rate (maybe wall tension of the left ventricle) and increased filing time of each cardiac cycle, resulting in improved diastolic function and restoration of the cardiovascular system function. When the circulation system recovered, it could return third space fluid to the circulation system, which was more than the system could not handle due to systolic dysfunction. In this situation, diuretics would keep the system functional and improve his system. In this clinical condition, aggressive diuresis may not be an appropriate approach. A diuresis is a tool instead of a goal of the treatment. The LVEF was 20% - 25% and remained unchanged after treatment. It implies that the LV systolic function might not be the cause for the exacerbation of CHF.

4. Discussion

The Cardiovascular system comprises the heart, vascular system, and circulation volume (blood). The system maintains the circulation of the blood to perfuse the organs. It brings the nutrients to the tissue and waste to the kidney to remove them through the urine. One essential function of the system is fluid metabolism. It is this function we are interested in. Adequate circulatory volume is vital for effective circulation, renal perfusion, and regulation of electrolytes. The central nervous system regulates fluid metabolism by controlling the cardiovascular system. When the system is volume-overloaded, such as excessive fluid intake, the perfusion to the kidney will increase and allow more water to be secreted as urine. When the system is underloaded, such as when the loss of water is greater than the intake, the perfusion to the kidney will decrease or shut down, leading to a decline in urine output or anuria. This process is achieved through the response of the sympathetic nervous system to the intravascular volume status. It is a highly self-regulated system. Any disturbances to the system may cause dysfunction, leading to CHF exacerbation, most commonly as dehydration by diuretic use, which is our interest in this discussion. Any system has its operational/functional range, including the cardiovascular system. Any disturbance beyond its functional range may result in symptoms of CHF. A normal system may tolerate a broader range of disturbances and still be functional. An abnormal one may become dysfunctional under smaller disturbances. Our clinical goal is to keep each patient's cardiovascular system within its optimal range.

Exacerbation of CHF occurs when the cardiovascular system becomes dysfunctional, primarily due to volume depletion of the cardiovascular system by diuretic use. It is a common scenario. The patients have a history of CHF and

have been treated as outpatients with chronic diuretic therapy. Upon developing progressive worsening of edema and dyspnea, diuretics were often increased. They often do not respond to the treatment, and the symptoms worsen. Patients are then admitted for aggressive diuresis. However, they may initially respond to IV diuretics, but they quickly lose the response to diuretics even with the increased dose. It is due to volume depletion of the cardiovascular system. A normal cardiovascular system can tolerate a wide range of fluid status without any symptoms. An abnormal cardiovascular system has a narrower functional range and cannot handle significant depletion. The patients may develop the symptoms of CHF. Each patient has a different tolerable range. Our treatment goal is to keep the system within its operational functional capacity. This can be achieved by controlling the patient's fluid status with fluid restriction, the cautious use of diuretics, and the avoidance of tachycardia with beta-blockers. It is the restoration of the function of the cardiovascular system that should be the goal of the treatment. Diuresis, although an important tool, is not a goal of the treatment.

How to restore the function of the cardiovascular system: We will concentrate on the patients with exacerbation of CHF presenting to the emergency room or admitted to the hospital. Most of these patients have been on outpatient CHF treatment, especially on diuretics, due to edema and shortness of breath. It is common for an increase in outpatient diuretic doses without the desired response before presenting to the ER. Increased doses of diuretics may cause further depletion of intravascular volume, leading to an increase in sympathetic nervous activity, which leads to increases in heart rate and blood pressure and causes vessel constriction, leading to a decrease in blood perfusion to the kidney and diastolic dysfunction of the left ventricle. This result in poor response to diuretics and worsening the symptoms of CHF. However, if we take diuresis as a goal of our treatment and overuse diuretics, patients may lose response to diuretics. Their symptoms may not resolve or worsen due to further depletion of intravascular volume and a decrease in renal perfusion. An adequate intravascular volume is the vehicle to transport the fluid from the third space to the kidney. The depletion of the volume leads to the loss of this vehicle. It will lead to fluid accumulation in the third space, such as the lower extremities and the lungs. In this condition, aggressive diuresis will not help to relieve the CHF symptoms. Patients may have worsening CHF symptoms, prolonged hospital stays, require additional treatments, and even end up with dialysis. Restoration of the cardiovascular system's function should be the treatment's goal. Adjustment of the fluid status is a tool for the restoration of the function of the system. Beta-blockers are another tool to restore the function of the cardiovascular system, which we will discuss later.

Volume management: Every CHF patient should be on fluid restriction unless there are medical reasons to take more fluid, such as frequent UTI, kidney stones, or some medication requiring more fluid. Most patients with reasonable

renal function should be able to handle the daily fluid of 1.2 - 1.8 L per day without diuretics. If the intravascular space is depleted, one may need some fluid challenge to restore the function of the cardiovascular system. It does not happen often, and it should be handled with caution. Tolerance to daily fluid differs in each patient, which needs to be determined in the clinical follow-up.

Diuretics in CHF: there is no doubt that fluid status contributes to the exacerbation of CHF. Diuretics have a position in the treatment of CHF. However, diuretics should be a tool for restoring the function of the cardiovascular system without perturbing it. For example, over-diuresis may result in the shrinking of intravascular volume. This increases the activity level of the sympathetic nervous activity, leading to an increase in heart rate and blood pressure and a decrease in renal perfusion and urine output due to vessel constriction. An adequate intravascular volume is the vehicle to move the fluid from the third space to the kidney for elimination. When the fluid intake is insufficient to expand the circulation volume, the fluid will leak into the third space, resulting in third space edema such as lower extremity and pulmonary edema. These clinical signs will give clinicians a misperception that more diuretics are needed, which will cause a deterioration of the clinical condition. In the acute phase of CHF exacerbation, diuretics will help to relieve the symptoms that bring the patient to inpatient care. However, over diuresis should be avoided. Its role is to restore the function of the cardiovascular system by adjusting the intravascular volume. Removal of excessive fluid in the third space will be achieved by the cardiovascular system with restored function. Diuretics are a part of fluid management. At any given time, the use of diuretics is a disturbance to the cardiovascular system. If there is a fluid overload in the body, especially in the cardiovascular system, this disturbance will be an intervention to correct the system function.

Beta-blocker for CHF: Beta-blocker use in acute exacerbation of CHF has caused significant concern since the early 1970s when Waagstein reported Beta-blockers to treat a patient with dilated cardiomyopathy presenting to the ER with pulmonary edema and tachycardia [6]. The patient did not respond to any treatment, including diuretics and digoxin. His symptoms resolved almost immediately with a few doses of IV beta-blocker with a decrease in heart rate. The patient also started to produce urine at the same time. It took a few years to accept beta-blocker use in treating CHF with reservation due to its negative inotropic effect, especially in CHF with reduced LV function. Up to this time, there is still a concern for using beta-blockers in acute CHF exacerbation. This is based on the assumption that a decrease in the systolic function is the cause of CHF. Negative inotropic and chronotropic effects of beta-blockers will decrease the heart rate and contractility of the cardiac muscle, resulting in a decrease in cardiac output. It will result in the worsening of the CHF hemodynamic instability. However, CHF exacerbation occurs in patients with from normal to severely decreased function. Not uncommon; some patients with severely reduced LV systolic function have no symptoms of CHF. It suggests that reduced LV systolic

function may not be the direct cause of CHF exacerbation, although it may contribute to it. It also supports the opinion that CHF exacerbation is due to LV diastolic dysfunction. When the cardiovascular system is disturbed above its tolerable level, the sympathetic system will increase its activity level. It increases heart rate, which worsens diastolic dysfunction and increases left ventricular filling pressure. The heart rate has not to be faster than 100 bpm to contribute to CHF exacerbation, such as 80 beats per minute, likely due to the patient's underlying diastolic dysfunction, which has low tolerability to rapid heart rate. The slower the heart rate, the longer the diastolic feeling time, and the better the diastolic function, which is one of the essential factors in restoring and maintaining the function of the cardiovascular system. If hypotension is a concern, Cardizem may be a temporary alternative that is less likely to lower blood pressure. The beta blockers should replace the Cardizem.

Treatment of outpatients: At discharge, patients and their families should have clear instructions and education for home fluid management with fluid restriction starting at 1.2 L/day (unless they have medical reasons requiring higher fluid intake) and daily weight and weight-based PRN use of diuretics. If their renal function is reasonably fine, they usually do not need to be on daily diuretics. Excessive fluid intake with daily diuretics will disturb the intrinsic fluid balance and make the system operate less optimally. For example, when patients decrease fluid intake for one or another reason, the daily diuretics may cause dehydration and trigger exacerbation of CHF.

5. Conclusions

The cardiovascular system is highly self-regulated. The exacerbation of CHF is due to dysfunction of the system, mainly due to diastolic dysfunction. The most common factors causing the system's dysfunction are fluid overload and misuse of diuretics since they disturb the self-regulating function of the cardiovascular system. Our treatment goal is to restore the system's function in CHF exacerbation and avoid the system's dysfunction in compensated CHF. Diuresis is one of the tools to achieve those goals. Fluid restriction is another tool. Beta-blockers are the most crucial medications for restoring the cardiovascular system's function and treating CHF exacerbation. These three, fluid restriction, appropriate use of diuretics, and beta-blockers, are also essential to avoid exacerbation of CHF. One may raise concern about the negative inotropic effect of the beta-blocker. It may be a concern when the patient is bradycardic and hypotensive. However, if the patient's heart rate is 60 - 70 and systolic blood pressure is 120 - 110, it is less likely he has CHF. As we discussed in case 2, LV systolic dysfunction may not be the direct cause of CHF exacerbation.

Each clinical case is different from one another. Some cases only need some diuresis and respond to it well when they have CHF exacerbation. Those patients usually have better cardiovascular reserves and can tolerate disturbances from higher fluid overload and diuresis. Some of them do not respond to medical

treatment and require device treatment. However, a significant percentage of CHF patients are in the condition discussed in this paper, especially case 2. They will benefit from the management and treatment described. The majority of patients do not need diuretics daily. Every patient with CHF needs fluid restriction, although each patient's fluid restriction level may differ. Other causes that directly contribute to CHF, such as ischemia and valvular diseases, are outside the scope of discussion in this paper.

6. Clinical Perspectives

The study applied the knowledge of physiology and pathophysiology to everyday clinical practice, leading to a different understanding of the pathogenesis of CHF and change in the management of the patients. This treatment strategy results in shorter hospital stays, lower readmission rates, and improvement in the quality of life of the patients. It may also save the cost of CHF treatment. It is a state-of-the-art clinical trial, which is why the author did not compare the study results with those from population-based clinical trials. However, it shows a potential clinical and socioeconomic significance. It needs attention from the cardiology field and further verification of the results and studies. This paper will provide a basis for investigation for clinical verification of the results and scientific guidance to everyday medical practice.

7. Limitations of the Study

This study is a retrospective and small study, including 64 cases. The data was collected in a small community hospital, St. Mary's Regional Medical Center at 1808 W Main Street Russellville, AR 72801. It did not consider the different variants, such as age, gender, et al., due to the nature of the study. It is not intended to apply to everyday practice across the cardiology field. It is not a state-of-the-art or a clinical trial. Instead, it reminds us to apply our basic medical knowledge, which we have learned in medical school, to our everyday practice. It may help us better understand the pathogenesis of CHF with further investigation.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Abbreviation

Congestive Heart Failure: CHF

Left Ventricle: LV