Diuretic Resistance Management in a Patient with Type I Cardiorenal Syndrome

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Diuretic Resistance Management in a Patient with Type I Cardiorenal Syndrome: A Case Report

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Abstract

Diuretic resistance is commonly found as a problem in acute heart failure (AHF). A further understanding of diuretic response could lead to improved personal approaches for treating patients with AHF. A 48 yo male suffered shortness of breath with a history of hypertension and DM. The patient was diagnosed as Ischemic Cardiomyopathy with Type I Cardiorenal Syndrome. The patient was given a 40 mg continued by 80 mg intravenous furosemide and low dose dobutamine pump. As the patient had zero urine production, a 160 mg intravenous furosemide followed by 15 mg/hr. After high-dose furosemide was given, the urine production was increased and the patient showed improved signs and symptoms. Deteriorating kidney function and bad response to diuretics is a principal clinical problem in AHF. Some treatment strategies include a combination of diuretic therapy, an increased dose of intravenous loop diuretics, and ultrafiltration. However, this patient gave good respond only to high doses of loop diuretics.

Keyword: *diuretic resistance, acute heart failure, type I cardiorenal syndrome, loop diuretics*

Introduction

Acute heart failure (AHF) is one of the main causes of hospitalization which is associated with high mortality, morbidity, and rehospitalization.[1] Excessive fluid retention is related to most symptoms associated with AHF. As the treatment of choice for excessive fluid retention in AHF, loop diuretic is usually given in up to 90% of patients who are hospitalized for AHF.[2]

Bad response to diuretic therapy is often found in hospitalized patients due to AHF.[3,4] Poor decongestion is often associated with impaired symptoms relieve, higher risk of worsening heart failure during hospitalization, increased post-discharge mortality, and a threefold higher rate of rehospitalization compared to patients who have better decongestion.[5]

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This case report will discuss the management of diuretic resistance in patients with Ischemic Cardiomyopathy and Type I Cardiorenal Syndrome with type II Diabetes Mellitus, anemia, hypoalbuminemia, left pleural effusion, and suspicion of relapsing pulmonary tuberculosis.

Case Illustrations

A 48-year-old Asian man came with complaints shortness of breath since 20 days before and getting worse in the last 2 days. It was felt even at rest and getting worse when lying on the bed or during activity. Swollen legs had been felt for 7 months. In addition, patients also complained of coughing and fever for 3 days.

Previous medical history of the patients was diabetes mellitus for 5 years under insulin treatment, uncontrolled hypertension, and history of pulmonary tuberculosis, and a heavy smoker.

When being received in the ward, the patient was weak, GCS 456, BP 120/80 mmHg, pulse 104 bpm,

respiratory rate 28 x/min, 99% peripheral oxygen saturation with 8 liters per minute oxygen mask.

His physical examination showed anemia, dyspnea, increased jugular venous pressure, bilateral basal rales, ascites, and bilateral leg swelling. ECG showed sinus tachycardia 104 beats per minute, normal frontal and horizontal axis, left ventricular hypertrophy. Chest X-ray showed that the left heart border was difficult to evaluate due to left pleural effusion, however, the impression showed cardiomegaly and pulmonary congestion. Echocardiography displayed LV dilatation (LVIDd 5.9 cm), impaired systolic function (EF by TEICH 30%, by Biplane 26%), LV diastolic function showed restrictive filling, regional LV wall motion showed akinesia of anterior (BMA) and hypokinesia in other segments, eccentric LVH. Hemodynamic parameters obtained PCWP 22.15 mmHg, SVR 2424.24 dynes.sec/cm5, LVCO 2.75 L/min, LVCI 1.64 L/min. m2, RAP 15 mmHg. Initial laboratory examination found anemia (HB: 9.5), leucocytosis (WBC: 10.480), impaired renal function (SC: 5.17; BUN: 73), elevated transaminases (AST: 77, ALT: 52), hypokalemia (K: 3.7), hypoalbuminemia (Alb: 2.95), proteinuria (3+), glucosuria (1+), leucocyturia (4+).

His diagnosis was Ischemic Cardiomyopathy with type I Cardiorenal Syndrome, Type II Diabetes Mellitus, anemia, hypoalbuminemia, left pleural effusion.

At the emergency room, the patient was administered with 40 mg intravenous furosemide injection. After one hour, the patient had zero urine production, so the patient was administered with 80 mg intravenous furosemide and dobutamine 3 mcg / KgBB / minute intravenous pump. After one hour of evaluation again, urine still had not come out. When in the room, the patient was given an injection of high-dose furosemide 160 mg intravenously and continued with furosemide pump 15 mg/hour. In addition, the patient was also given ramipril 1x5 mg, spironolactone 1x25 mg, acetylsalicylic acid 1x100mg, isosorbide dinitrate 3x5 mg, and potassium supplementation. After high-dose furosemide was administered, lots of urine were produced. Shortness of breath and congestive signs began to decrease then.

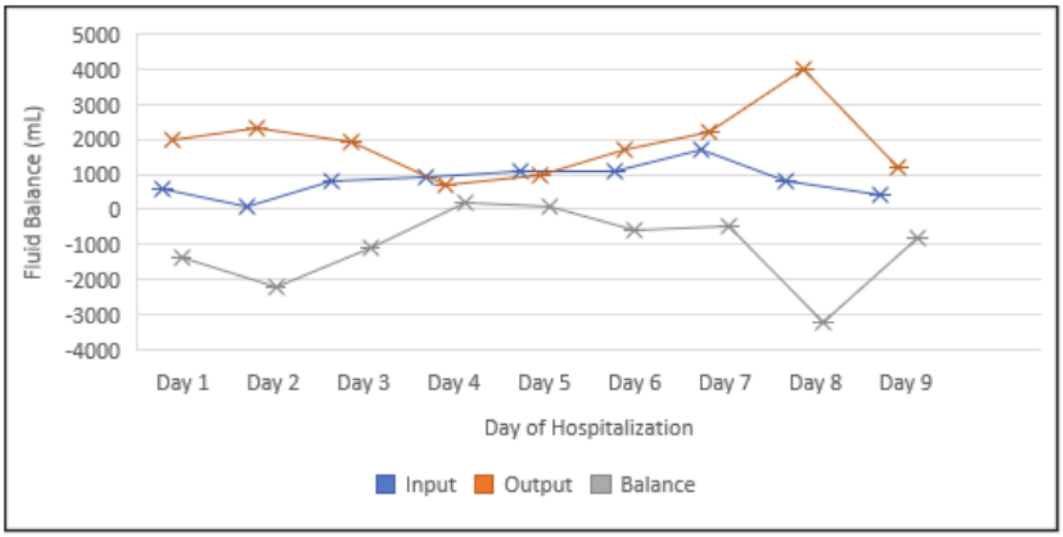

Figure 1. Fluid Balance Monitoring Each Day of Hospitalization

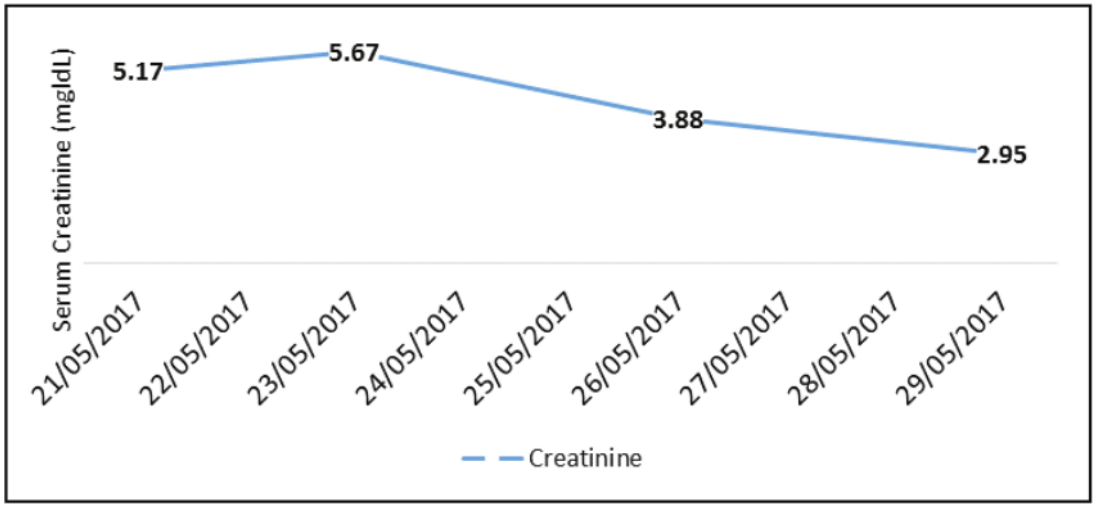

Figure 2 Serial Monitoring of Kidney Function

On the third day of treatment, the dose of furosemide was lowered and the dobutamine pump was stopped. On the sixth day, low-dose bisoprolol therapy was started. On serial examination of kidney function, there was a tendency for a decrease in serum creatinine and improvement in kidney function. After ten days of treatment, patients could be discharged with 2x40 mg oral furosemide, ramipril 5 mg, bisoprolol 2.5 mg, acetylsalicylic acid 100 mg, and isosorbide dinitrate 3x5 mg. The patient was planned to have an evaluation at cardiology outpatient clinic, internal medicine outpatient clinic, and pulmonary outpatient clinic for a workup on suspicion of relapsed pulmonary tuberculosis

Discussion

Mechanism of water and salt retention

The regulation of total salt and body fluid in the normal range is under atrial-renal reflex control, renin-angiotensin aldosterone system (RAAS) and sympathetic nervous system (SNS) which are activated by low renal blood flow. Under normal circumstances, RAAS acts as a protection against underperfusion. In conditions of heart failure, salt and water retention caused by hemodynamics and angiotensin reabsorptive effects cause further congestion. Therefore, RAAS has a vital role in edema in heart failure.[6]

The sympathetic nervous system has a contribution to long-term regulation of blood pressure and intravascular volume. In congestive heart failure, the initial phase is triggered by reflexes to provide an inotropic effect and improve cardiac output. However, the excess activity of SNS can increase cardiomyocyte cell death, while catecholamines can cause myocardial hypertrophy. In addition, aggressive diuretics administration can lead to further neurohormonal activation and trigger systemic and renal vasoconstriction, which causes worsening of renal function. As a result, c

Pathophysiology of deteriorating kidney function in acute heart failure is multifactorial. In many cases, impaired renal perfusion causes a decrease in kidney function. This may be due to hypovolemia (declined preload), excessive neurohormonal vasoconstriction (increased afterload), and hypotension with low output syndrome. This can also be triggered by diuretic resistance and kidney toxicity caused by certain combination of drugs, for example, nonsteroidal antiinflammatory drugs (NSAIDs), cyclosporine, ACE inhibitors, angiotensin receptor blockers (ARBs), and contrast agents. Congestive veins are another trigger factor. Patients with heart failure and excess fluid will make a combination of high central venous pressure with low systemic pressure. It can cause a decrease in renal perfusion pressure thus causing a significant reduction in

kidney blood flow and urine production.[7]

In this patient, kidney function was decreased. When the patient arrived, SC was 5,17. After improving general conditions and adequate diuresis, there was a tendency to improve kidney function to SC 2.95 a day before the patient was discharged. In addition, a history of diabetes mellitus that had been known for 5 years with signs of existing chronicity (anemia, hypoalbuminemia, proteinuria), underlined the suspicion of diabetic nephropathy. The condition of acute heart failure caused the deterioration of kidney function. So, clinically, type I cardiorenal syndrome was found in this patient.

Mechanism of diuretic retention

A bad response to diuretics is a principal clinical problem in AHF in which it has various mechanisms. There are several mechanisms of diuretics. First, diuretics are given orally must first be absorbed in the intestine. If the patient has gastrointestinal edema or intestinal hypoperfusion, oral diuretics absorption is impaired. In this condition, intravenous administration should be given. In patients with kidney impairment or heart failure, higher diuretic doses are needed to obtain a similar effect, and an increase in diuretic doses will be less effective by time.[8]

Second, most diuretics are bound to albumin. Hypoalbuminemia, which is commonly found in heart failure, interferes with the removal and separation of active furosemide and converts it to inactive forms. In addition, albumin lost in the tubules can bind furosemide and prevent it from working on ion channel transporters. Giving furosemide and albumin together improves decongestion in patients with liver or kidney disease, but there is no available evidence in patients with heart failure.[9,10]

Third, patients with heart failure and chronic kidney failure have increased organic acids level which competitively inhibits organic anion transport and reduces the availability of diuretics at their work point. High level of blood urea nitrogen also reflects kidney function which actively works to maintain sodium and water. Thus, impaired absorption, decreased renal blood flow, azotemia, hypoalbuminemia, and proteinuria can affect the effectiveness of diuretics.[11]

At the beginning of diuretic treatment, natriuretic effect produces the desired negative sodium balance. The homeostatic response will occur due to decreased extracellular volume. Mediated by activation of RAAS and the SNS, it will cause increased tubular sodium retention. This condition works as a suitable response that inhibits excessive volume reduction during advanced diuretic administration. However, in patients with heart failure,it can cause rapid and large sodium reabsorption which contributes to diuretic resistance.[12]Moreover, continuous channeling of sodium or diuretics causes distal tubular cells hypertrophy, which passes through the proximal effect of loop diuretics causing increased sodium retention. Other mechanisms that result in reduced response to diuretics, such as decreased renal blood flow due tostenosis of the renal artery or drug interactions, must also be taken into consideration when giving loop diuretics.[8]

In this patient, acute heart failure was present, which causes decreased blood flow to the kidneys. In addition, this patient also had azotemia, hypoalbuminemia, and proteinuria so that they could cause a decrease in active diuretics in the tubular lumen and result in decreased effectiveness of the diuretics given. This would cause diuretic resistance.

Evaluation of diuretic response

Diuretic resistance is the failure to decongestants even with adequate diuretic doses and increased by more than 80 mg per day.[13] Some researchers have tried to quantify the diuretic response by combining decongestive effects and diuretic doses. In present study, a diuretic response was defined as a decrease in body weight from entry into the 4th day of treatment / 40 mg of furosemide (or equivalent). A bad diuretic response independently predicts repeated rehospitalization and increases the mortality of heart failure patients.[3]

In this patient, at the time of arrival, the injection of furosemide 40 mg was administered, followed by furosemide 80 mg intravenously. But for two hours of observation, urine production did not come out. The administration of dobutamine continuous pump at low dose also could not help. For this reason, the patient was concluded as resistant to diuretics.

Management of diuretic resistance

Noncompliance of the patients

If diuretic resistance occurs, the possibility of noncompliance with salt restriction or drug use should be excluded. Postdiureticsodium retention can fully compensate for lost sodium during periods where diuretics reach effective tubular concentrations if sodium intake > 100 mmol/day.[8] Compliance to diuretics can be assessed by measuring the amount of diuretics in the urine.[14]

The use of NSAIDs is an important cause of poor decongestion. This drug interferes with prostaglandin synthesis by inhibiting cyclooxygenase and thus opposing response to loop diuretics. Consumption of NSAIDs is associated with an increased risk of hospitalization due to heart failure in patients with a history of previous heart failure.[15]

Adjustment of Diuretic Dose

Increased dosage is considered an effective strategy as it can compensate for pharmacokinetic and pharmacodynamic shifts in loop diuretics that occur in patients with acute heart failure. As patients show different levels of kidney damage, an increase in dosage is needed to provide the appropriate amount of diuretics at the urinary tract. Giving more frequent diuretics (2-3 times/day) will overcome the effects of postdiuretic sodium retention by limiting drug-free intervals.[16]

Intravenous Injection or Continuous Infusion on Loop Diuretics

Disruption of loop diuretics absorption in patients with heart failure will cause reduction and slowdown in peak concentration in urine, eventhough its absolute bioavailability does not change significantly compared to healthy persons. Moderately increased dose or switching into intravenous injection can overcome this problem[17]. Several studies have compared the efficacy of intermittent loop diuretics injection with continuous infusion in advanced heart failure patients. The same daily dose will cause higher urinary and electrolyte volume excretion if being administered as a continuous infusion. Maximum furosemide concentration on plasma is significantly lower and this results in reduced ototoxicity.[14]

Combination of Diuretics

Refractory heart failure patients will usually give respond to high-dose furosemide, either given orally or in the continuous infusion. However, in some patients, it does not overcome diuretic resistance. Some combinations with loop diuretics are practicable. In patients with heart failure, proximal diuretics should be avoided as there can be a risk of metabolic acidosis. [14] Thiazide diuretics block reabsorption only 5% -10% sodium is filtered, while loop diuretics can inhibit it up to 25%. As a result, thiazide diuretics have a weak natriuretic effect, thus it is not effective as a monotherapy in advanced heart failure. However, in the chronic condition when sodium load in the distal tubule increases, it can increase its salt carrying capacity. Combination of the loop and thiazide diuretics in congestive heart failure and diuretic resistance is a reasonable choice considering this pathophysiological mechanism.[14]

Vasopressin Antagonists

Antidiuretic hormone or vasopressin is produced in response to decreased blood volume or hyperosmolality.[18] Some studies showed that tolvaptan has strong aquaretic effects without kidney disorder in patients with AHF. When vaptans are administered in AHF patients, it can modify the renal response to water retention. However, vaptans do not affect liver and kidney remodeling well and have no effect on long-term mortality.[19]

Dopamine

Administration of low-dose dopamine (<3 µg /kg/ min) has been recommended to increase kidney blood flow, thereby improving renal function and diuresis as well. However, Research on Renal Optimization Strategies Evaluation showed that dopamine and nesiritide do not have a notable effect on urine volume, suggesting no additional benefit on diuretic therapy.[20] Despite lacking evidence, low dose dopamine is practically still often used as it is expected to stimulate a diuretic response by improving kidney function, and may be useful in patients who fail with other therapeutic agents.[5]

Hypertonic Saline

Hypertonic saline together with diuretics can improve diuresis by mobilizing extravascular fluid to intravascular. In some small studies, increased diuresis and clinical improvement were found in acute heart failure patients who were given the addition of hypertonic saline. In a large scale study (trial of SMAC-HF), consisting of 1,771 patients, there was an increase in diuresis and natriuresis, and reduced rates of rehospitalization in patients given intravenous furosemide and hypertonic saline, compared to patients with furosemide only. It shows that hypertonic salt could be a safe alternative strategy for improving diuretic responses in AHF.[21]

Ultrafiltration

Ultrafiltration is an effective method for removing fluid by filtering plasma water directly across a semipermeable membrane using a pressure gradient, which results in isoosmotic ultrafiltration compared to plasma. In two randomized controlled trials (RAPID CHF and UNLOAD) which compared diuretic and ultrafiltration therapy, higher fluid secretion was present in the ultrafiltration group.[22,23] Current knowledge regarding the effectiveness and safety of ultrafiltration in acute heart failure patients is still inconclusive, where studies are published on a small scale and reporting the relevant outcome measures is not optimal.[24]

In this patient, the therapeutic strategy used to overcome diuretic resistance is to use high-dose loop diuretics. After giving a loading dose which was raised regularly, it was followed by maintenance in the form of continuous infusion of high-dose loop diuretics as well. In addition, the patient was also given an additional 25 mg of spironolactone per day.

Conclusion

A 48-year-old man with Ischemic Cardiomyopathy was reported with type I Cardiorenal Syndrome, Type II Diabetes Mellitus, anemia, hypoalbuminemia, left pleural effusion which was resistant to initial diuretic therapy. Diuretic resistance was based on persistent congestive conditions even though furosemide diuretics had been administered with adequate doses. Conditions that cause resistance to initial diuretic therapy are decreased renal perfusion due to acute heart failure, azotemia due to diabetic nephropathy, worsening renal function, hypoalbuminemia, and proteinuria. As a decongestion effort, the patient was given a bolus of high-dose loop diuretics continued with continuous infusion. After a high-dose furosemide administration, lots of urine were produced. Shortness of breath and congestive signs began to decrease then.

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Patient' Informed Consent: Patient's informed consent hasbeen obtained.

Conflict of Interest: Nil

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