Congestive heart failure as an indication for continuous renal replacement therapy

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Congestive heart failure as an indication for continuous renal replacement therapy. Continuous venovenous hemofiltration (CVVH) is the most widely used renal replacement therapy for the treatment of critically ill patients with acute renal failure on the intensive care unit. Whether or not congestive heart failure is an indication for CVVH is controversial and needs to be discussed. Therefore, we present a patient with congestive heart failure who was treated successfully with CVVH.

While continuous venovenous hemofiltration (CVVH) is the most often used renal replacement therapy for critically ill patients with acute renal failure, its use for those with severe congestive heart failure remains a topic for discussion. After the initial cardiogenic shock, acute renal failure and impaired renal perfusion often occur, and if renal function is not swiftly restored then multi-organ damage and death can result. These decompensated patients are usually resistant to diuretics and have a massive fluid overload; their prognosis is poor. This article examines one case in light of the current literature, and discusses therapeutic options for the management of congestive heart failure patients who are in critical care.

We report the case of a 40-year-old male who was admitted to the intensive care unit because of severe congestive heart failure (CHF). The patient suffered from dilative cardiomyopathy as a consequence of viral myocarditis eight years earlier. On admission, the patient presented with severe hypotension and dyspnea, peripheral edema, and liver and renal function impairment. The relevant laboratory values were as follows: aspartate aminotransferase (AST) 410 U/liter (<17 U/liter), alanine aminotransferase (ALT) 260 U/liter (<23 U/liter), bilirubin 5.4 mg/dl (<1.1 mg/dl), alkaline phosphatase (ALP) 244 U/liter (<180 U/liter), serum creatinine 2.7 mg/dl (1.2 mg/dl), sodium 135 mmol/liter (135 to 145 mmol/liter), potassium 5.9 mmol/liter (3.5 to 5.0 mmol/liter), and blood urea nitrogen 55 mg/dl (<28 mg/dl). The mean arterial pressure was 80 mm Hg, heart rate was 90 beats per minute, and the central venous pressure was 24 mm Hg. Initial treatment with catecholamines and high doses of furosemide (1000 mg in 6 hr) in order to stimulate renal fluid excretion was unsuccessful; therefore, the patient was treated with continuous venovenous hemofiltration (CVVH) for 10 hours (Fig. 1). A lactate-buffered solution (45 mmol/liter) was applied at a filtration rate of 1000 ml/hr and a substitution rate of 750 ml/hr, resulting in a negative fluid balance of 250 ml/hr. In order to reduce the preload, a maximal ultrafiltration volume of 2,500 ml was reached. Immediately after the beginning of CVVH therapy, the patient’s diuresis returned. The amount of catecholamines administered was reduced significantly, and symptoms of CHF such as dyspnea and edema improved. The central venous pressure (CVP) fell to a minimum of 19 mm Hg. The mean arterial pressure increased to 120 mm Hg, and the heart rate was unchanged. The patient developed a sufficient diuresis under further diuretic therapy, and renal function returned to normal (creatinine 1.1 mg/dl) after four days. The hemodynamic situation of the patient under a therapy with dobutamine in a low dose intravenous (15 mg/hr) and angiotensin-1 receptor blocker (Lorzaar®) 25 mg/day orally was stable, but because of the severe reduction of cardiac function (cardiac index 1.7 liter/min/m²) by dilative cardiomyopathy, the patient underwent a successful heart transplantation two months later.

This case report presents a patient with acute decompensation of CHF by cardiomyopathy. The patient had acute renal failure in the beginning of onset, and suffered from liver congestion and pulmonary edema. He needed catecholamines to stabilize the hemodynamic situation, but did not respond to large doses of diuretics. Early use of CVVH led to an increase in urinary output, raised the mean arterial pressure, and relieved the symptoms. Thus, CVVH led to an improvement of cardiac function.

Key words: acute renal failure, heart failure, continuous venovenous hemofiltration, renal replacement therapy.
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as well as renal function. CVVH therapy led to a stabilization of the patient and prevented further deterioration of organ function.

**CONTINUOUS VENOVENOUS HEMOFILTRATION IN SEVERE CONGESTIVE HEART FAILURE**

In cardiogenic shock after massive myocardial infarction or low-output syndrome caused by cardiomyopathy, acute renal failure and loss of diuresis caused by renal perfusion impairment is frequent. This leads to fluid and electrolyte imbalance and the accumulation of toxic metabolites, which also further suppresses cardiac function. These factors contribute to marked cardiovascular instability. In these patients, the performance of intermittent hemodialysis is not recommended because of severely compromised hemodynamics. Therefore, CVVH is administered in order to remove excess body fluid, to correct metabolic acidosis, and to eliminate uremic metabolites. As a result, an improvement of the cardiovascular situation is observed. Therefore, CVVH in patients with cardiogenic shock and acute renal failure is the first choice for renal replacement therapy [1].

Whether or not CVVH is indicated even in an earlier state of cardiac dysfunction, when renal failure is impending and multiorgan dysfunction caused by low cardiac output might be prevented, is of specific interest.

Congestive heart failure is characterized by low cardiac output and leads to sodium and water retention [2], neuroendocrine activation, and constriction of the peripheral circulation. Therapeutic options include pharmacological therapies, the use of mechanical devices, and heart transplantation [3]. The type of treatment applied to a patient depends on the symptoms, the cause for heart failure, and the time of progression. There are some patients who appear to be in a decompensated state with peripheral edema, pleural effusion, and edema of the lung, and they do not respond to diuretic therapy. Thus, they are in a state of decompensated heart failure with a high sodium and water retention as a neurohumoral response to low cardiac output. When these patients show no improvement under therapy with catecholamines in high doses and a maximum of diuretic therapy, hemofiltration might be beneficial.

The use of hemofiltration as an intermittent [4–7] or a continuous [8–10] therapy in patients with severe CHF has been described by several authors. In a study of arteriovenous hemofiltration, Kramer et al included patients with CHF and overhydration who were resistant to diuretic therapy [11]. Next to arteriovenous hemofiltration [6, 11], the venovenous approach is the most widely used technique [6, 8, 9]. Most studies are patient surveys [5, 6, 12] or case reports [10]. Only a few studies exist in which patients were systematically investigated [8, 13–16]. Patients with CHF were treated for a short period of time [4, 8, 9, 13, 14] or for a long period up to several months [6, 7, 12]. The effects of hemofiltration in CHF included: a relief of the patients’ symptoms [4, 6, 9], a decrease of central venous or right atrial pressure [4, 5, 9], and a lowered capillary wedge pressure [4, 5, 9], which led to an increase of urinary output [4, 13, 16]. Use of hemofiltration for a long period of time led to weight reduction, a reduced hospital stay, or reduced diuretic treatment [12]. All of these studies showed the long-lasting effect of the hemofiltration and that the patients were able to respond to pharmacological therapy again.

In patients with moderate heart failure, it was shown that a single session of ultrafiltration led to an improvement of hemodynamics, including a significant increase of the cardiac index [14]. Furthermore, compared to an infusion of furosemide, the markers reflecting neurohumoral activity returned to normal by ultrafiltration. The
removal of 1.6 liters by ultrafiltration compared to a removal of 1.6 liters by diuretics led to a sustained decrease in of the levels of norepinephrine, renin, and aldosterone [15]. One single intermittent hemofiltration therapy with the filtration of 2983 ± 1228 ml increased urinary output and sodium excretion and led to a fall in norepinephrine levels, effects that persisted for 24 to 48 hours [13]. Thus, these studies showed surprisingly that the effect of hemofiltration was superior to the effect of diuretic therapy [13–16].

The outcome of patients with heart failure grade IV according to the New York Health Association (NYHA) was poor in most of the studies, but the parameters related to poor prognosis before starting CVVH therapy were oliguria for more than 15 hours or a creatinine of more than 4.0 mg/dl [9]. The patient with the best outcome had the shortest time of oligo-anuria and had the greatest benefit from the therapy [9]. CVVH started preoperatively in patients with severe heart failure and undergoing cardiopulmonary bypass surgery resulted in an improvement of all hemodynamic variables during hemofiltration and the restoration of renal function [8]. This suggests that an early start of CVVH in CHF is necessary to improve the patient’s chance for survival.

The results of these studies show that CVVH treatment is a useful tool to interrupt further fluid retention and to re-establish a sufficient renal function. Responsiveness to diuretic therapy is restored and diuresis returns. CVVH is especially useful in patients with severely compromised cardiac function and the beginning of renal function impairment. It leads to an immediate reduction in preload, and therefore, renal perfusion is restored. CVVH may also be employed in patients with severely compromised cardiac function who are awaiting a heart transplantation in order to carefully reduce excess body fluid and stabilize the patient before the heart surgery. For this reason, CVVH was included in the guidelines of therapeutic options for the treatment in severe heart failure by the Task Force of the Working Group on Heart Failure of the European Society of Cardiology [3].

DISCUSSION

In severe CHF, hemofiltration is a helpful tool to treat the decompensated patient, who is resistant to diuretics and suffers from massive fluid overload. In the acute situation, CVVH might be the best treatment. For the chronic treatment of severe heart failure, intermittent hemofiltration (HF) is recommended for solute and water removal, but here peritoneal dialysis has been successfully applied [17]. The beneficial effects of hemofiltration include the lowering of atrial and central venous pressures, a reduction of cardiac preload, and a decrease of the capillary wedge pressure. In most patients, a relief of symptoms, a new response to diuretic therapy, and a sustained break of the vicious cycle of neurohumoral activity and water and sodium retention are achieved.

In some of the studies cited, the patients had no renal failure, which means they had oliguria but no anuria with a creatinine of less than 2.0 mg/dl. The point is that renal failure is more a question of definition and time. In reality, in most patients, prerenal azotemia is present or acute renal failure is impending because of low cardiac output and decreased renal plasma flow. However, according to current literature, it seems that an early start of water and sodium removal by hemofiltration is best [8, 9]. As in the presented case, an early start of hemofiltration prevents further multi-organ deterioration, stabilizes cardiac function, improves renal function, and, in some cases, it might prevent the intubation and mechanical ventilation of the patient. Thus, hemofiltration in CHF is indicated by fluid overload resistant to diuretics alone and not as usual by azotemia, high potassium levels, or acid-balance disorders. It is not yet clearly understood whether the beneficial effect of CVVH in CHF is a single effect of preload reduction or the effect of the reduction of toxic metabolites [8], like a myocardial depressant factor [18]. Although many CHF patients have been treated with HF, there are no controlled studies on this topic. While our reported patient showed an improvement with this therapy, the overall prognosis of these patients remains poor.

CONCLUSIONS

Hemofiltration is a therapeutic option for the management of cardiac failure and is ideal to bridge the time to transplantation. In some cases, hemofiltration may help to prolong the lifespan of patients with CHF. The important fact is that renal replacement therapy has to be started early to prevent further organ damage and to prevent further multi-organ dysfunction.

Future studies should focus the question of whether the prognosis of patients with CHF can be improved under application of continuous renal replacement therapies.

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