



## Early Ultrafiltration Therapy in a Patient with Decompensated Heart Failure and Acute Pulmonary Edema in the Setting of Diuretic Resistance

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Congestive heart failure is a major mortality cause in developed countries. In patients with decompensated heart failure who are resistant to diuretic treatment options are limited. Application of ultrafiltration therapy is a reasonable alternative in the setting of decompensated heart failure with diuretic resistance. We report a 74 years old woman with acute decompensated heart failure who did not respond to diuretic and was treated with ultrafiltration therapy with a successful outcome.

**Key Words:** Congestive heart failure, Diuretic resistance, Ultrafiltration

### **Kalp Yetmezliğine Bağlı Akut Akciğer Ödemine Giren Hastada Ortaya Çıkan Diüretik Rezistansının Erken Ultrafiltrasyon İle Tedavisi**

Kongestif kalp yetmezliği gelişmiş ülkelerde başta gelen ölüm sebepleri arasındadır. Diüretik rezistansı olan dekompanse kalp yetmezlikli hastalarda tedavi seçenekleri sınırlıdır. Diüretik rezistansı gelişen dekompanse kalp yetmezliği olgularında ultrafiltrasyon terapisi bir seçenek olarak karşımıza çıkmaktadır. Olgu sunumumuzda diüretik rezistansı gelişmiş olan akut dekompanse kalp yetmezliğindeki 74 yaşındaki bayan hastanın ultrafiltrasyon ile tedavisi ve sonuçlarını sunduk.

**Anahtar Kelimeler:** Konjestif kalp yetmezliği, Diüretik rezistansı, Ultrafiltrasyon

Congestive heart failure is a major mortality cause in developed countries. Medical therapy can alleviate the associated symptoms and increase survival rate.<sup>1</sup>

Despite medical treatment mortality occurs due to progression of underlying heart disease eventually. As ventricular dysfunction progresses decreased cardiac output and renal perfusion lead to activation of the renin angiotensin aldosterone system (RAAS), with efferent arteriolar vasoconstriction secondary to angiotensin II production, peripheral vasoconstriction, and aldosterone secretion, resulting in sodium and water retention.

If diuretic resistance develops in a patient under inotropic treatment eventually morbidity will increase dramatically. In patients with decompensated heart failure who are resistant to diuretics treatment options are limited. There is no agreement in management of these patients. Application of ultrafiltration therapy is a reasonable alternative in the setting of decompensated heart failure with diuretic resistance.<sup>2-5</sup>

### **PATIENT**

A 74 year old female patient with a known history of heart failure presented to the emergency service with severe dyspnea, and pretibial edema. She was taking 160 mg furosemide/day orally for more than three months due to worsening congestive symptoms. Blood pressure 70/40 mmHg, heart rate 135/min, respiratory rate 30/min. She was severely orthopneic, her respiratory sound were generally decreased. Liver was palpable 6 cm below the costal margin. In PA telecardiography there was cardiomegaly and bilateral consolidation in the lower zones. The findings

in her initial echocardiography performed in emergency service were as follows: Left ventricular end systolic diameter (LVESD): 6,9 mm, Left ventricular end diastolic diameter (LVEDD): 7,8 mm, and ejection fraction (EF) was calculated as %15 .

Initial laboratory values were: Urea 50 mg/dl, creatinin 0.9 mg/ml, sodium 138 mEq/l, potassium 4.3 mEq/l. Blood gas values: pH: 7.56, CO<sub>2</sub> :19, BE:-2.4, HCO<sub>3</sub>:19. Patient was admitted to the intensive unit. Dobutamin 10 µg/kg/hour and dopamine 3 µg/kg/hour was started. Sufficient response to diuretic treatment was not observed (120mg iv bolus followed by a infusion at a rate of 20mg/hour).

6 hours after her admission her dyspnea progressively increased and acute pulmonary edema developed soon followed by respiratory failure and cardiac arrest. She was intubated and cardiopulmonary resuscitation was performed for 30 minutes until she was hemodynamically stable.

She was followed for 12 hours with inotropic support and continuous diuretic infusion while mechanically ventilated. In order to increase the mean arterial pressure the infusion dose of inotropic agents were increased as follows: adrenaline 0.2 µg/kg/min, noradrenaline 0.5 µg/kg/dk, dobutamine 50 µg/kg/hour and dopamine 3 µg/kg/hour. Although the patient's mean arterial pressure raised to a level of 60-70 mmHg there was still no urine output. Due to concomitant presence of volume overload and oligouria refractory to diuretic treatment intermittent venous ultrafiltration was planned which would allow reduction of fluid overload without disturbing hemodynamics and electrolyte balance. Laboratory values before ultrafiltration were as follows: Urea 80 mg/dl, creatinin 1.7 mg/dl, Na 125 mEq/l and K 4.4 mEq/l. Size 11F double lumen dialysis catheter was placed through the femoral vein. High flux ultrafiltration system was used during ultrafiltration (Gambro FH 66, Polyamide 0.60 m<sup>2</sup>, high flux). Because the patient had a low blood pressure blood flow rate was held at 140 ml/min. She was heparinized during the procedure (5 IU/kg/hour).

During the ultrafiltration procedure Gambro hemodiasol solution was used for replacement for plasma that was drawn. Ultrafiltration was repeated consecutively for four days, and 3 l of fluid was drawn at every time ( a total of 12 L of fluid was drawn ). At the follow up urine output increased with the use of diuretics and therefore ultrafiltration was no longer needed and therefore stopped. At the end

of four days mean blood pressure increased to 90 mmHg, pretibial edema resolved significantly and she had a normal urine output. No significant electrolyte changes occurred after ultrafiltration also renal function remained stabile (serum creatinin values remained within normal limits). She was extubated at the eighth day of admittance and was discharged from the hospital at day 14 in good condition.

## RESULT

In our case acute pulmonary edema due to decompensated heart failure with in the setting of diuretic resistance was treated with early ultrafiltration before renal functions deteriorated and this therapy resulted in quick recovery of the patient's hemodynamic status in an effective way.

Although diuretics have a disputable role in improving survival they do improve symptoms of congestion effectively and have been a part of standard congestive heart failure therapy in all recent trials involving β blockers, angiotensin converting inhibitors and angiotensin II receptor blockers.<sup>6</sup> Diuretic resistance has been independently associated with total mortality, sudden death and pump failure death.<sup>7</sup>

In moderate to severe heart failure patients diuretic resistance occurs frequently and often becomes a clinical challenge. As in our case, the main reason responsible for this phenomenon is decreased renal perfusion due to low cardiac output which leads to decreased sodium delivery to the loop of Henle.<sup>8</sup> Because natriuretic activity of loop diuretics is related to the amount of sodium concentration in this segment of the nephron, their effectiveness is largely diminished in this clinical setting. Furthermore in the setting of acute decompensated heart failure overactivation of the RAAS may largely blunt the natriuretic function of the loop diuretics.<sup>9</sup>

Ultrafiltration removes excess fluid from the patient without causing electrolyte balance. This results in a reduction in intravascular volume and ventricular filling pressures.<sup>10</sup> Due to reduction in intravascular volume oncotic pressure increases and extravascular volume is drawn into vascular bed<sup>11</sup>. In addition, due to reduction in right atrial pressure pulmonary blood flow decreases which leads to a decrease in pulmonary venous pressure.<sup>12-13</sup>

Ultrafiltration, besides improving the outcome of pulmonary edema, increases diuretic sensitivity and as

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a result renal compensatory mechanisms begin to work effectively.

When high dose diuretics and ultrafiltration are compared in means of returning the patient into euvolemic state, ultrafiltration has the advantage of having a rapid effect of reversing pulmonary edema without effecting neurohumoral activity adversely.<sup>14</sup>

As our case demonstrates ultrafiltration gives clinicians an alternative choice for treating patients with acute decompensated heart failure with pulmonary edema not responding to diuretics under inotropic support.<sup>15</sup> Early ultrafiltration before occurrence of impairment in renal function enables quick recovery of the impaired hemodynamic system and decreases hospitalization period. We believe that in the near future ultrafiltration will be used more often as the results of ongoing trials would demonstrate that it is a predictable, safe and effective method for reducing fluid overload.

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