Effective Treatment of Congestive Heart Failure Using Adaptive Servo-ventilation in an End-stage Renal Disease Patient on Hemodialysis

Naoki Aizawa¹, Kazufumi Nagahama¹, Kaoru Goya¹, Shoichiro Yamazato¹, Hidekazu Ikemiyagi¹, Katsuhiko Ohshiro¹, Tomoko Shinzato¹, Yasushi Higashiuesato¹, Tetsuya Ishiki¹, Takanori Yasu³, Kunitoshi Iseki³ and Yusuke Ohya¹

Abstract

A 61-year-old man who was being treated with hemodialysis (HD) for end-stage renal disease presented with symptoms of severe congestive heart failure (CHF). Removing excess intravascular fluid during HD was difficult due to the patient’s chronic hypotension induced by severe left ventricular (LV) dysfunction. The application of adaptive servo-ventilation (ASV) increased the patient’s cardiac output and blood pressure during HD, thus resulting in the effective removal of excess intravascular fluid. Therefore, ASV may be effective for treating CHF in HD patients with LV dysfunction and chronic hypotension.

Key words: adaptive servo-ventilation, heart failure, hemodialysis, end-stage renal disease (ESRD)

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Introduction

Congestive heart failure (CHF) is highly prevalent among patients with end-stage renal disease (ESRD) treated with hemodialysis (HD) (1). Periodic increases in the intravascular volume between HD sessions raise the risk of CHF. Moreover, removing excess intravascular fluid during HD is often difficult in patients with left ventricular (LV) dysfunction and chronic hypotension. Therefore, physicians often encounter difficulties when providing standard therapies for heart failure (HF) using beta-blockers and/or renin-angiotensin inhibitors in such cases. Adaptive servo-ventilation (ASV) is a new technique for providing positive airway pressure in which the pressure support level continuously varies by monitoring the patient’s breathing pattern. We herein report, to the best of our knowledge, the first clinical experience of using ASV to treat CHF in an ESRD patient on HD.

Case Report

A 61-year-old man with ESRD on HD was admitted to our hospital due to worsening dyspnea. He had been under treatment with maintenance HD for 33 years due to chronic glomerulonephritis. Three years before admission, he underwent coronary artery bypass grafting for coronary artery disease, mitral annuloplasty for mitral regurgitation (MR) and pacemaker implantation for bradycardic atrial fibrillation. However, he continued to suffer postoperatively from LV dysfunction and chronic hypotension, and removing excess intravascular fluid during HD was difficult due to chronic hypotension induced by LV dysfunction, which prevented treatment with renin-angiotensin inhibitors and beta-blockers. On admission, he presented with severe symptoms of HF [New York Heart Association (NYHA) functional class IV] and hypotension (84/42 mm Hg). An electrocardiogram revealed atrial flutter (58 beats/min), while chest ra-
diography showed a cardiothoracic ratio (CTR) of 55% with bilateral pulmonary congestion (Fig. 1A). Meanwhile, echocardiography disclosed severe LV dysfunction (LV ejection fraction, 26%) with severe MR (grade 3) due to leaflet tethering caused by LV dilation (Fig. 2A). The pre-HD serum N-terminal pro-brain natriuretic peptide (NT-pro BNP) level was high (18,691 pg/mL). In addition, polysomnography showed no evidence of sleep-disordered breathing (apnea-hypopnea index, 4.6/h), while right heart catheterization performed one day after HD revealed a low cardiac index (1.9 L/min/m²) and high pulmonary capillary wedge pressure (PCWP) (17 mmHg). After obtaining the patient’s informed consent, we applied ASV (Autoset CS®, ResMed, Sydney, Australia) with an end-expiratory pressure of 5 cmH₂O and inspiratory pressure support of 3-10 cmH₂O in order to examine the acute hemodynamic effects. Twenty minutes after the initiation of ASV, the patient’s cardiac output increased from 2.97 L/min to 3.16 L/min (Fig. 3). In contrast, no significant changes were noted in his heart rate or level of systemic vascular resistance (Table). Therefore, we initiated treatment with ASV for HF during the day, as ASV causes insomnia.

Four weeks later, the patient’s blood pressure (BP) during HD increased without any associated changes in his medications, including the administration of an inotropic agent (Fig. 4). In addition, his dry weight decreased from 49.5 kg to 48.0 kg. Therefore, we gradually decreased and then discontinued the dose of the inotropic agent (docarpamine, 2,250-0 mg). Moreover, we succeeded in starting standard treatment with an angiotensin-converting enzyme inhibitor (enalapril, 1.25 mg/day) and beta-blocker (carvedilol, started at a dose of 1.25 mg/day then maintained at a dose of 5 mg/day on days without HD). Two months later, the patient continued to use ASV during the day, with a mean usage time of 3.5 hours daily. Furthermore, his NYHA functional class improved from IV to II, the NT-pro BNP level decreased from 18,691 pg/mL to 8,828 pg/mL, the CTR decreased from 55% to 50% (Fig. 1) and the MR grade de-
have high LV filling pressures (6). In the present case, ASV is suitable for ASV therapy because they tend to decrease MR (5). HD patients with LV dysfunction may be increased preload by blocking venous return, 2) ASV decreases cardiac output of a failing heart as follows: 1) ASV decreases ex-

increased from 3 to 2 (Fig. 2).

**Discussion**

We herein describe, to the best of our knowledge, the first case involving the application of ASV to treat CHF in an ESRD patient on HD. The patient exhibited both severe LV dysfunction and chronic hypotension. ASV therapy induced a rapid increase in cardiac output, as revealed on cardiac catherization. With this increase in cardiac output and BP, HD was able to remove a sufficient volume of excess intravascular fluid to permit the administration of standard medical therapies for HF.

The mechanisms underlying the effects of ASV treatment on the symptoms of HF remain to be defined in detail. An increase in cardiac output when the PCWP is high in patients with HF is considered to be a favorable effect (2-4). Furthermore, it has been suggested that the positive intrathoracic pressure produced by ASV mechanically increases the output of a failing heart as follows: 1) ASV decreases ex-

increased the PCWP. However, we do not consider that the ASV treatment increased the LV filling pressure, as the positive intrathoracic pressure provided by ASV may have raised the patient’s intracardiac pressure. Therefore, the mechanical effects of ASV may have helped to ameliorate the patient’s HF associated with a high PCWP and severe MR.

Previous studies have indicated that ASV reduces an enhanced sympathetic nerve activity (7, 8). However, the present patient experienced no significant changes in heart rate or systemic vascular resistance while under ASV therapy. Therefore, we assume that the mechanical effects of ASV observed in this case influenced other factors in addition to reducing the sympathetic nerve activity.

In conclusion, we herein reported the successful application of ASV for the treatment of CHF in an ESRD patient on HD who exhibited severe LV dysfunction and chronic hypotension. Further clinical and experimental studies are required to clarify the efficacy and mechanism of action of ASV in patients with HF.

**The authors state that they have no Conflict of Interest (COI).**

**References**

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**Table.** Changes in Hemodynamics before and during Adaptive Servo-ventilation

<table>
<thead>
<tr>
<th></th>
<th>Before ASV</th>
<th>During ASV (20min)</th>
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<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>54</td>
<td>54</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>108/54</td>
<td>120/60</td>
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<tr>
<td>Cardiac index (L/min/m²)</td>
<td>1.90</td>
<td>1.9r</td>
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<tr>
<td>PCWP (mmHg)</td>
<td>17</td>
<td>20</td>
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<tr>
<td>Systolic PAP (mmHg)</td>
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<td>RAP (mmHg)</td>
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<td>2,811</td>
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<tr>
<td>PVRI (dyne<em>s</em>cm⁻⁵*m⁻²)</td>
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<td>198</td>
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</tbody>
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