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Evaluating the Effectiveness of Emergency Ventilator Treatment for Severe Acute Left Ventricular Heart Failure

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Abstract: *Objective:* To evaluate the efficacy of emergency ventilator therapy in severe acute left heart failure. *Methods:* A total of 75 patients with severe acute left ventricular heart failure who were admitted to the hospital from July 2020 to July 2023 were randomly divided into two groups. Group A received additional emergency ventilator treatment, and group B received conventional treatment. The efficacy was compared. *Results:* The curative effect of patients with severe acute left heart failure in group A was higher than that in group B ($P < 0.05$); all blood gas indicators in group A were better than those in group B ($P < 0.05$); all vital signs indicators in group A were better than those in group B ($P < 0.05$); group A was more satisfied with the treatment of severe acute left ventricular heart failure than group B ($P < 0.05$). *Conclusion:* Patients with severe acute left heart failure who receive emergency ventilator treatment can stabilize vital signs, improve blood oxygen supply, and enhance curative effect.

Keywords: Severe heart failure; Acute left heart failure; Emergency treatment; Ventilator treatment

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1. Introduction

Under the influence of various heart diseases, the patient's myocardial contractions can be weakened. At this time, the cardiac output is low, which can induce acute left heart failure, leading to myocardial ischemia, hypoxia, and dyspnea, with a high mortality rate. Severe acute left-sided heart failure accounts for a high proportion of cardiovascular diseases. After the onset, it can reduce the patient's cardiac output and increase the end-diastolic pressure of the heart. If not treated in time, it can block the pulmonary venous return and cause vascular fluid to enter the alveolar cavity and interpulmonary space. It induces dyspnea, and some patients are complicated by symptoms of wet rales in the lungs and pink frothy sputum. Diuretics, cardiotonic drugs, and other drugs are primarily used in clinical practice to treat left heart failure symptomatically. However, they are ineffective in patients with acute heart failure [1]. With the rapid development of ventilator technology, ventilator therapy is gradually used in emergency rescue of critically ill patients. This study reports a total of 75 patients treated with severe acute left heart failure from July 2020 to July 2023 to explore the therapeutic value of emergency ventilators.

2. General information and methods

2.1. General information

Seventy-five patients diagnosed with severe acute left ventricular heart failure from July 2020 to July 2023 were randomly divided into two groups. There was no difference in the patients with left heart failure data in groups A and B ($P > 0.05$), as shown in **Table 1**.

Table 1. Baseline data analysis table

Group	No.	Gender		Age (years)		The course of disease (h)	
		Male	Female	Range	Mean	Range	Means
Group A	38	20 (52.63)	18 (47.37)	50–75	56.84 ± 2.11	1–6	4.18 ± 0.85
Group B	37	21 (56.76)	17 (45.95)	51–76	56.87 ± 2.13	1–7	4.21 ± 0.87
x^2 / t		0.0530		0.0613		0.1511	
P		0.8180		0.9513		0.8804	

2.2. Inclusion and exclusion standards

Inclusion criteria included: (1) The organs and tissues are in a low perfusion state; (2) Symptoms of systemic circulation congestion and pulmonary congestion occur; (3) Informed consent; and (4) Mental normality.

Exclusion criteria included: (1) Patients with blood lesions; (2) Patients with cancer; (3) Patients with abnormal immune function; and (4) Patients who cannot tolerate ventilator treatment.

2.3. Treatment methods

Group A received additional ventilator treatment: The ventilator was used to complete the treatment, the mask was pressed in two directions to trigger the flow, the inspiratory pressure and expiratory pressure were adjusted to 8–15 cm H_2O and 2–6 cm H_2O, respectively, and at the same time the respiratory frequency and oxygen flow were adjusted to 16–20 times/min and 5–10 L/min, respectively. During ventilator treatment, vital signs were monitored and ventilator use was suspended until the symptoms of dyspnea were relieved.

Group B received conventional treatment, giving high-flow oxygen, diuretics, cardiotonic agents, vasodilators, and corrected electrolyte imbalance.

2.4. Observation indicators

The observation indicators in this study included:

(1) Efficacy: If the respiratory rate and heart rate were average, there was no lung murmur, and the symptoms of heart failure disappeared, it was marked as markedly effective; if the respiratory rate and heart rate were improved, the lung murmurs were reduced, and the symptoms of heart failure were relieved, it was marked as effective; if the respiratory rate and heart rate were abnormal, and the lung murmur was abnormal, it was marked as ineffective.

(2) Blood gas indicators: Arterial blood oxygen partial pressure (PaO_2), blood oxygen saturation (SaO_2), and arterial blood carbon dioxide partial pressure ($PaCO_2$) were detected.

(3) Vital signs: Heart rate, systolic blood pressure, respiratory rate, left ejection fraction, and other indicators were detected.

(4) Treatment satisfaction: Evaluated according to the self-made Severe Acute Left Heart Failure Treatment Satisfaction Scale.

2.5. Statistical research

The data of patients with left heart failure were processed with SPSS 21.0. The count data of patients with left heart failure were recorded in % (x^2 test), and the measurement data of patients with left heart failure were recorded in mean ± standard deviation (*t*-test). There is a statistical difference when $P < 0.05$.

3. Results

3.1. Curative effect on left ventricular heart failure

The curative effect of patients with left ventricular heart failure in group A was 97.37%, which was higher than in group B, 83.78% ($P < 0.05$), as shown in **Table 2**.

Table 2. Comparison of left ventricular heart failure [*n* (%)]

Group	Markedly effective	Effective	Ineffective	Total effective rate
Group A (*n* = 38)	14 (36.84)	23 (60.53)	1 (2.63)	97.37
Group B (*n* = 37)	15 (40.54)	16 (43.24)	6 (16.22)	83.78
x^2	-	-	-	4.0882
P	-	-	-	0.0432

3.2. Blood gas indicators of left ventricular heart failure

Table 3 shows that after treatment, blood gas indicators such as PaO_2, SaO_2, and $PaCO_2$ in group A were better than those in group B ($P < 0.05$).

Table 3. Analysis table of blood gas indicators in patients with left ventricular heart failure (mean ± SD)

Group	PaO_2 (mmHg)		SaO_2 (%)		$PaCO_2$ (mmHg)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Group A (*n* = 38)	54.82 ± 2.41	80.02 ± 3.85	79.88 ± 2.25	94.88 ± 4.25	71.25 ± 4.36	54.28 ± 2.48
Group B (*n* = 37)	54.79 ± 2.39	71.69 ± 3.42	79.91 ± 2.19	88.47 ± 3.69	71.27 ± 4.38	62.36 ± 3.63
t	0.0541	9.8968	0.0585	6.9668	0.0198	11.2820
P	0.9570	0.0000	0.9535	0.0000	0.9842	0.0000

3.3. Vital signs indicators of left ventricular heart failure

After treatment, the heart rate, systolic blood pressure, respiratory frequency, left ejection fraction, and other indicators of group A were better than those of group B ($P < 0.05$), as shown in **Table 4**.

Table 4. Analysis table of vital signs indicators in patients with left ventricular heart failure (mean ± SD)

Group	Heart rate (beats/min)		Systolic blood pressure (mmHg)		Respiration rate (times/min)		Left ejection fraction (%)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Group A (*n* = 38)	150.36 ± 6.28	110.25 ± 3.84	170.85 ± 8.15	140.61 ± 3.25	45.81 ± 6.28	30.28 ± 3.25	35.81 ± 2.51	45.81 ± 3.21
Group B (*n* = 37)	150.38 ± 6.31	140.86 ± 5.19	170.81 ± 8.17	152.69 ± 5.19	45.79 ± 6.31	59.81 ± 4.41	35.82 ± 2.49	41.69 ± 3.08
t	0.0138	29.0897	0.0212	12.1154	0.0138	33.0739	0.0173	5.6692
P	0.9891	0.0000	0.9831	0.0000	0.9891	0.0000	0.9862	0.0000

3.4. Treatment satisfaction

Table 5 shows that the left ventricular heart failure treatment satisfaction rate in group A was 97.37%, which was higher than in group B, 83.78% ($P < 0.05$).

Table 5. Comparison of satisfaction with left ventricular heart failure treatment [*n* (%)]

Group	Markedly satisfied	Satisfied	Not satisfied	Satisfaction rate
Group A ($n = 38$)	28 (73.68)	9 (23.68)	1 (2.63)	97.37
Group B ($n = 37$)	21 (56.76)	10 (27.03)	6 (16.22)	83.78
x^2	-	-	-	4.0882
P	-	-	-	0.0432

4. Discussion

Acute left heart failure can cause cardiac arrest in patients and cause arrhythmia, cardiogenic shock, and other diseases. Common pathological manifestations include left ventricular enlargement, decreased systolic blood pressure (SBP), pulmonary rales, and wheezing. Severe acute left heart failure can endanger the patient's life and requires symptomatic treatment to adjust the respiratory rate, lower blood pressure, and stabilize the heart rate. Based on the analysis of clinical practice, although conventional medication can relieve the uncomfortable symptoms of patients with left heart failure, it cannot wholly control the progression of the disease due to the impact of refractory hypoxemia in patients with heart failure. In addition, in patients with left heart failure and severe hypoxemia, cardiac function continues to be impaired, and pulmonary congestion continues to increase, which can increase capillary pressure, obstruct pulmonary ventilation, and further aggravate heart failure. Therefore, to correct hypoxemia and prevent heart failure-related complications in patients with severe acute left heart failure, some scholars recommend non-invasive ventilator-assisted treatment to increase cardiac output, correct respiratory function, and restore coronary blood perfusion. Non-invasive ventilation treatment can increase arterial blood oxygen partial pressure, slow down pulmonary blood flow, optimize cardiac function, and correct hypoxia in a short time. In addition, non-invasive ventilation can also slow down venous blood supply and reduce the body's oxygen consumption, thereby preventing and controlling hypoxemia. It can also reduce intrathoracic positive pressure and prevent related diseases [2]. However, it should be noted that during non-invasive ventilation treatment, patients should be assisted in cleaning respiratory secretions, providing nutritional support, and supplementing nutrition through nasogastric feeding and intravenous programs.

This study uses non-invasive positive pressure ventilation to treat patients with severe acute left heart failure. It can increase intrapulmonary pressure, promoting pulmonary O_2 and CO_2 exchange, thereby increasing arterial oxygen partial pressure, reducing ventricular end-diastolic myocardial pressure, and increasing lung residual capacity. The compliance of lung tissue can also improve coronary blood supply and optimize the patient's cardiac function, which is conducive to enhancing the curative effect [3]. In addition, non-invasive positive pressure ventilation does not require intubation, which is helpful for patients to restore their ability to breathe spontaneously. When patients with heart failure inhale, the pressure increases, which can prompt the patient to complete the inspiratory action, reduce expiratory pressure, increase oxygen absorption, and prevent and treat alveoli, thereby reducing uncomfortable symptoms caused by heart failure.

Based on the data analysis in this study, the efficacy of group A for patients with left heart failure was 97.37%, which was higher than that of group B, 83.78% ($P < 0.05$). It is suggested that emergency ventilator treatment can improve the efficacy of severe acute left heart failure. Analysis of the reasons shows that after

ventilator-assisted treatment in patients with severe acute left heart failure, pressure support can improve pulmonary ventilation function, promote pulmonary CO_2 discharge, reduce ventilator work, and relieve respiratory muscle fatigue [4]. Furthermore, emergency ventilator treatment can also increase chest pressure and reduce cardiac load, thereby enhancing the nourishing function, increasing myocardial blood and oxygen supply, and preventing pulmonary edema. Compared with conventional emergency treatment, ventilators used in emergency treatment have the following advantages: (1) They can be directly connected to the lungs of patients with left heart failure, which can increase alveolar pressure and optimize the respiratory function of patients with left heart failure; (2) It can increase thoracic pressure, reduce venous return, and regulate cardiac load; and (3) It can reduce the oxygen consumption of respiratory muscles, promote respiratory muscle relaxation, and thereby inhibit respiratory muscle fatigue [5]. Relevant literature reports that patients with acute left heart failure receiving emergency ventilator treatment can reduce oxygen consumption, alleviate hypoxemia, and, at the same time, adjust the body's acid-base balance [6].

Another set of data showed that blood gas indicators such as PaO_2 (80.02 ± 3.85 mmHg), SaO_2 (94.88 ± 4.25%), and $PaCO_2$ (54.28 ± 2.48 mmHg) in group A were better than those in group B ($P < 0.05$). It is suggested that emergency ventilator treatment can improve blood gas indicators in patients with left heart failure. Positive pressure ventilation therapy with a ventilator can increase the intra-airway pressure, dilate the bronchi, promote the recruitment of atrophic alveoli, and optimize the body's oxygen supply. Additionally, continuous positive pressure ventilation therapy can reduce the pressure difference between capillaries and alveoli, which is beneficial to improving pulmonary edema; it can also block venous return, reduce cardiac load, and strengthen the everyday physiological work of the left ventricle. Summary analysis shows that the mechanism of ventilator treatment is as follows: reducing the work done by the heart and lung tissue during the body's respiration, restoring the body's blood oxygen supply, correcting acidosis, reducing chest pressure, relieving myocardial load, and thereby optimizing cardiac function [7].

Meanwhile, another set of data showed that group A had a heart rate of 110.25 ± 3.84 times/min, systolic blood pressure of 140.61 ± 3.25 mmHg, respiratory frequency of 30.28 ± 3.25 times/min, left ejection fraction of 45.81 ± 3.21%, and other indicators which were all better than group B ($P < 0.05$). It is suggested that emergency ventilator treatment can improve the vital signs of patients with severe left heart failure. In addition to increasing pulmonary ventilation and regulating the physiological respiratory function of respiratory muscles, the ventilator can also reduce the work of breathing and optimize respiratory function. Moreover, ventilator therapy can increase intrathoracic pressure and reduce peripheral venous return, inhibiting cardiac filling and pulmonary congestion. Ventilator therapy also positively optimizes gas exchange function, correcting hypoxia and optimizing pulmonary compliance. It can also reduce intrathoracic and interstitial pressure and prevent pulmonary edema, improving the patient's vital signs [8].

Last but not least, the final data set showed that the left heart failure treatment satisfaction in group A was 97.37%, which was higher than in group B, 83.78% ($P < 0.05$). It is suggested that emergency ventilator treatment can enhance the treatment satisfaction of patients with left heart failure. Emergency ventilators are found to be suitable for treating left heart failure patients who cannot breathe on their own or have weak breathing. It can replace the patient's respiratory function, prevent extravasation and alveolar atrophy, increase the amount of oxygen in capillaries, and enhance the body's utilization rate of oxygen supply [9]. In addition, non-invasive ventilator ventilation treatment can increase alveolar ventilation and stimulate the body to excrete carbon dioxide, thereby increasing blood return to the heart, optimizing myocardial oxygen supply, and stabilizing heart failure, increasing patient satisfaction [10].

In summary, patients with severe acute left heart failure receiving emergency ventilator treatment can

maintain stable vital signs of heart failure patients and improve their blood gas supply, which has a promotion value.

Disclosure statement

The author declares no conflict of interest.

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