REVIEW ARTICLE

BENEFITS OF HIGH-FLOW NASAL OXYGEN THERAPY IN ACUTE HEART FAILURE

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Acute heart failure (AHF) is a life-threatening condition. The majority of patients come to the emergency room (ER) with pulmonary congestion and respiratory failure. Therefore, oxygen therapy is an essential modality in AHF apart from diuretics and improving hemodynamic. Non-invasive ventilation (NIV) is often required to reduce hypercapnia, acidosis, and respiratory rate in AHF. However, it is more invasive than conventional oxygen therapy. NIV is also less comfortable for patients and has limited use in the ER. The high-flow nasal oxygen (HFNO) is increasing in popularity during the COVID-19 pandemic. HFNO can deliver high flow oxygen with a constant fraction in patients with respiratory failure. Studies have reported the benefits of HFNO in acute heart failure. Therefore, HFNO may benefit and may be used as an alternative to acute heart failure patients who cannot tolerate NIV.

Keywords: Acute heart failure, High-flow nasal oxygen, Non-invasive ventilator, Oxygen therapy

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INTRODUCTION

Acute heart failure is the leading cause of patient morbidity and mortality. Shortness of breath is a cardinal symptom most often found in acute heart failure patients admitted to hospital.[1] Cardiogenic pulmonary edema is a severe complication of acute heart failure and is the second most common cause of respiratory failure after pneumonia. Initial therapy for acute heart failure includes diuretics, vasodilators, and oxygen supplementation with a non-invasive ventilator (NIV) or mechanical ventilator.[2] In patients with respiratory failure, conventional oxygen therapy is often ineffective. It is necessary to use continuous airway positive pressure (CPAP) or non-invasive positive pressure ventilation (NIPPV) to reduce hypercapnia, acidosis, and respiratory rate. This therapy can reduce the endotracheal intubation rate and the need for a ventilator, but it is more invasive than conventional oxygen therapy, is less comfortable for patients, and has limited use in the ER.[3]

In recent years high-flow nasal oxygen (HFNO) has been safe and valuable in various clinical conditions.[4] The popularity of HFNO is reaching its peak amid the COVID-19 pandemic, where there is an increasing need for ICUs and ventilators globally. HFNO is increasingly being used to prevent acute hypoxemic respiratory failure patients from falling on a ventilator.[5] HFNO is a less invasive oxygen therapy than NIPPV. HFNO can flow up to 60 liters of air per minute and is closer to the inspiratory flow requirements of respiratory failure patients than conventional oxygen therapy, which only flows a maximum of 15 liters per minute. HFNO also has various physiological benefits compared to conventional oxygen therapy.[6] There are no international guidelines that recommend HFNO as the first choice for cardiogenic pulmonary edema. However, studies on its benefits have been reported.[3] This literature review was created to inform physicians about the benefits of HFNO therapy in patients with acute heart failure.

Etiology of Acute Heart Failure: About two-thirds of patients with acute heart failure have previously been diagnosed as chronic heart failure patients. On initial arrival in the ER, the patient showed worsening heart function. Myocardial ischemia, atrial fibrillation (AF), hypertension, infection, poor medication adherence, and excessive salt or alcohol consumption are all things that can cause acute worsening of chronic heart failure. In addition, about 50% of cases are associated with a decreased left ventricular ejection fraction. Cases associated with normal ejection fraction include patients with rapid AF or valve disease.[7] Various etiologies and risk factors for acute heart failure are presented in Table 1 and Table 2.

Table 1. Etiology and risk factors for acute heart failure.[7]

Reference	Population	Population characteristic	The main cause	%
(Fonarow et al., 2008)[8]	48.612	- Mean age 73 years - Percentage of women 52% -Mean LVEF 39%	Multiple Infection of airway Ischemia Arrhythmia	61 12 12 14
(Roguin et al., 2000)[9]	150	-Mean age 75 years - Percentage of women 40% - Moderate to a severe decline in left ventricular function in 67% of the population	Myocardial ischemia Arrhythmia Hypertension Infection Anemia Drug abuse Excess salt consumption	51 31 29 18 13 8 8

Table 2. Circumstances are causing acute heart failure.[10]

Conditions that cause rapid heart failure	Conditions that cause heart failure that is not too fast
• Severe tachyarrhythmias or bradycardia disorders • Acute coronary syndrome • Mechanical complications in acute coronary syndromes (intraventricular septal rupture, acute mitral regurgitation, right heart failure) • Acute pulmonary embolism • Hypertensive crisis • Aortic dissection • Cardiac tamponade • Perioperative and surgical problems • Peripartum cardiomyopathy	• Infections (including infective endocarditis) • Acute exacerbations of COPD / asthma •Anemia • Renal dysfunction • Non-compliance with treatment Iatrogenic causes (corticosteroid drugs, NSAIDs) •Arrhythmias, bradycardia, and conduction disturbances that do not cause sudden changes in pulse rate • Uncontrolled hypertension • Hyper and hypothyroidism • Use of drugs and alcohol

Pathophysiology of Acute Heart Failure: Various cardiovascular or non-cardiovascular conditions result in acute heart failure through a single or a combination of pathogenesis mechanisms.[11] The main sign of acute heart failure is congestion. Congestion is characterized by weight gain, peripheral edema, jugular vein distension, liver enlargement, hepatojugular reflux or ascites, and pulmonary congestion, characterized by shortness of breath and rhonchi.[11] The two main mechanisms that cause congestion are fluid retention and fluid redistribution. In fluid retention, cardiac dysfunction causes a decrease in cardiac output (CO). Conversely, neurohormonal compensatory mechanisms will cause the release of aldosterone and vasopressin-arginine. The mechanism results in sodium and fluid retention in the kidneys. As a result, it causes congestion in the lungs and peripheral blood vessels. On the other hand, fluid redistribution occurs due to venous vasoconstriction. Venous vasoconstriction causes increased venous return and preload, whereas arterial constriction increases afterload. Both make left ventricular pressure increase, pulmonary capillary pressure increase, and pulmonary congestion occurs.[11]

In the presence of cardiac dysfunction, several neurohormonal pathways, including the sympathetic nervous system, the renin-angiotensin-aldosterone system, and the arginine-vasopressin system, are activated to counteract the effects of heart failure to deliver oxygen to the periphery. Neurohormonal activation causes impaired sodium regulation and fluid accumulation. Tissue edema results from transudation from capillaries to interstitials due to excess drainage capacity of the lymphatic system.[12]

Management of Acute Heart Failure: The initial management of acute heart failure should consist of 3 parts: triage, diagnosis, and initial therapy. Because acute heart failure is a life-threatening condition, current guidelines for managing acute heart failure recommend that initial diagnosis and therapy be carried out as soon as possible, especially 30-60 minutes from when the patient arrives at the hospital. Acute heart failure is a life-threatening pharmacological condition, so the process of diagnosing and administering pharmacological and non-pharmacological management must be carried out simultaneously and as early as possible. Patients with acute heart failure accompanied by hemodynamic disturbances (cardiogenic shock) or respiratory failure should receive circulatory support management and ventilatory assistance. The leading cause or precipitate causes the patient to fall in a condition of acute heart failure and administration of treatment according to that cause to prevent further deterioration.[10]

Recommended Oxygen Therapy in Acute Heart Failure: In managing acute heart failure, apart from making the patient euvolemic with nitrates and loop diuretics, oxygen therapy is also an essential component in its management, especially in patients with hypoxemic respiratory failure. Oxygen can be given from conventional methods such as nasal oxygen, simple masks, non-rebreathing masks to NIV via CPAP or BiPAP, depending on the degree of severity.[13] Oxygenation is given to patients with hypoxaemic conditions or respiratory distress with peripheral oxygen saturation (SpO_2) of less than 90% or a partial arterial oxygen pressure (PaO_2) of less than 60 mmHg (< 8.0 kPa). It is recommended to start oxygen therapy with a fraction of 40-60% and titrated until a SpO_2 reached above 90%, but caution should be exercised in patients with CO_2 retention.[10]

Oxygen therapy can be given conventionally, but positive pressure ventilation (CPAP/BiPAP) can be given if hypoxemia still occurs. If the positive pressure ventilation does not improve, then transfer to the ICU for ventilator installation. A high dose of oxygen is recommended in patients with saturation below 90% and a PaO_2 below 60 mmHg.[10] Giving oxygen therapy to patients with heart failure without hypoxemia should not be routinely given because it can cause hyperoxemia conditions.[14] In patients with acute pulmonary edema, the use of NIV can improve respiratory distress, shortness of breath, respiratory rate, and acidosis. Despite the many benefits of NIV, its use is often inconvenient for patients, causing necrosis and thickening of the facial skin, conjunctivitis due to air leaking into the eyes, and the risk of aspiration in patients who vomit while using NIV. When compared with HFNO, the NIV had more facial skin damage and erythema.[13]

High-Flow Nasal Oxygen Therapy: Traditionally, nasal oxygen therapy was delivered at a low flow rate through nasal oxygen. However, new nasal oxygen has emerged in recent years to deliver high-flow, moisturized oxygen to patients. The device is high-flow nasal oxygen (HFNO). HFNO can deliver oxygen at flow rates of up to 60 liters per minute. This device consists of oxygen connected to a humidifier and delivered through particular nasal oxygen. HFNO was initially used for preterm neonates and pediatric patients, but now HFNO has become one of the first-line therapies for patients with acute respiratory distress.[15]

In the era of the COVID-19 pandemic, the popularity of HFNO is increasing. HFNO becomes a hypoxemia therapy amidst the limitations of ICUs and ventilators.[16] The primary clinical manifestation of severe COVID-19 patients is hypoxemia accompanied by heavy breathing. Although NIV is known to have advantages in the presence of PEEP in patients with hypercapnia such as COPD, its efficiency is still low to reduce heavy breathing in severe COVID-19 patients. This occurs because the patient requires high inspiratory pressure, which causes air leakage in the NIV mask. The increased respiratory drive has also occurred in COVID-19 patients, requiring high airflow to meet the patient's respiratory needs.[17] The survival sepsis campaign guidelines (SSC) recommend HFNO as a better therapy than conventional oxygen in hypoxemia COVID-19 patients.[18]

Physiological Effects of High-Flow Nasal Oxygen Therapy: Various effects and advantages of HFNO have been reported. HFNO is more comfortable and tolerable to patients. Administration of conventional oxygen therapy delivers oxygen that is not humidified, drying the upper airway, affecting mucociliary clearance, creating atelectasis, and reducing patient comfort. In HFNO, the air has been pre-warmed so that it generally makes the patient comfortable, reduces tightness, and does not make the upper airway dry.[15]

The peak inspiratory flow in patients with acute respiratory failure averages 30-40 liters per minute and can exceed 60 liters per minute in patients with severe sepsis. This airflow requirement is higher than standard oxygen therapy flow. Standard oxygen in patients with severe acute respiratory failure will cause the inhaled airflow to mix with room air so that the inhaled oxygen fraction will not reach 70%. In NIV, the oxygen fraction given can reach 100% if there is no air leak. However, the increase in airflow, higher in NIV, can cause leakage in face masks. HFNO can deliver high fraction oxygen and high flow rates of up to 60 liters per minute. It will meet the peak inspiratory flow requirements in respiratory failure patients.[19] HFNO provides a constant high airflow so that there is no gas dilution, decreasing the oxygen fraction. The oxygen fraction in HFNO can also be adjusted from 21% to 100%.[2]

Patients with acute respiratory distress usually have increased mucus secretion and require extra effort to spit it out. The efforts made by the patient will cause fatigue of the respiratory muscles, especially if the mucus is thick and cannot be coughed up. This is often seen in patients with NIV.[15] In the provision of standard oxygen and NIV that flowed is dry and cold air. This air can cause mucociliary disorders, inflammation, and mucus retention.[13] The water content of the mucus will affect the thickness of the sputum. Physiological humidification is essential in providing good respiratory epithelial function through sodium absorption and chloride secretion. Inspiratory air warmed to a temperature of 37° Celsius promotes adequate mucosal function and improves mucus secretion. HFNO is an oxygen therapy device that provides warmed air. Some tools have user-adjustable temperature settings.[15]

HFNO can also reduce the metabolic cost of breathing. Metabolic cost is the energy the body needs to warm the incoming air when breathing. The air will be warmed by the body to prevent bronchoconstriction due to cold effects, especially in tachypnoea and acute respiratory failure patients. HFNO will reduce breathing and metabolic cost because the circulated air has been warmed by the tool.[15]

HFNO is an open system, while NIV is a closed system.[2] HFNO can exert positive pressure on the upper airway, but the pressure varies (the pressure is higher when the mouth is closed). When the mouth is closed, the HFNO will provide an inspiratory pressure of 3 cm H_2O, whereas if the mouth is open, it is less than 2 cm H_2O at a flow of 60 liters per minute.[19] This will increase the washout of the nasopharyngeal dead space. The ability to remove CO_2 in the upper airway is an excess of HFNO due to high airflow. Various studies have shown HFNO to have the same PEEP effect as nasal CPAP.[15] However, clinical studies show that the lower effect of PEEP HFNO only increases PaO_2 without changing the PaO_2/FiO_2 ratio, whereas NIV increases both. This is because the presence of PEEP increases alveolar recruitment, which increases the PaO_2/FiO_2 ratio.[19] PEEP causes the fluid in the alveoli to be pushed to the perivascular so that the surface of the alveoli can diffuse oxygen more.[2]

Use of the High-Flow Nasal Oxygen: Clinical practical guidelines issued by the European Society of Intensive Care Medicine state that administering HFNO in hypoxemic respiratory failure is a strong recommendation.[6]

HFNO therapy has many benefits, but doctors must remain vigilant about patient responses such as respiratory rate and respiratory rate. If the patient's condition deteriorates, it is better to consider the use of a ventilator. Monitoring is vital in using HFNO so that patients who fail do not delay the use of ventilators and increase patient mortality. Intubation should be performed in patients with failed HFNO who meet at least 2 of the criteria below:[17]

1. Respiratory rate> 40 times per minute
2. There is no sign of a change in breathing pattern
3. Increased breath secretion
4. Respiratory acidosis (pH < 7.35)
5. Decrease in $SpO_2 < 90\%$ for more than 5 minutes

Benefits of High-Flow Nasal Oxygen in Acute Heart Failure: There are no international guidelines yet to recommend HFNO as the first choice in cardiogenic pulmonary edema, but few studies on its benefits have been reported.[3] Several studies are comparing NIV and HFNO ingestion in acute heart failure patients. A study by Carratala et al. reported that five acute heart failure patients who did not improve with NIV and switched to HFNO improved their condition after 24 hours, in which the SpO_2 increased from 85% to 99%.[20] Another study by Yang et al. showed no difference between efficacy and intubation rates between the two groups, but HFNO was more comfortable and well-tolerated by patients. Haywood et al. study also stated that in patients with acute heart failure, HFNO was not inferior to NIPPV.[21]

Table 3. Comparison of HFNO and NIV in acute heart failure patients.[13]

	HFNO	NIV
Advantage	(I) The patient is more comfortable and can tolerate (II) There is no distraction when the patient eats and drinks (III) Avoiding complications related to intubation (IV) Easy to use and monitor (V) Lowering metabolic costs	(I) Can provide high PEEP (II) Provide bilevel ventilation for type 2 respiratory failure patients (III) Avoiding complications related to intubation (IV) There is already much evidence on its use in acute heart failure
Disadvantage	(I) Little evidence of benefit when high PEEP is required (II) Useful only in type 1 respiratory failure, evidence is limited to type 2 respiratory failure (III) Studies on acute heart failure are few	(I) Oxygenation is disturbed when the patient eats or drinks because of removing the NIV mask (II) The patient is uncomfortable (III) Complications on the face such as erythema, skin necrosis, and risk of aspiration if the patient vomits (IV) Patient non-compliance leads to treatment failure

HFNO therapy can reduce shortness of breath in patients with acute heart failure. An RCT by Makdee et al. in 128 patients with cardiogenic pulmonary edema showed HFNO significantly decreased respiratory rate, decreased breathlessness.[22] The study by Carratala et al. on HFNO saw significant improvements in comfort, oxygenation index, and respiratory rate at 1, 2, and 24 versus conventional oxygen ($p < 0.05$).[21]

HFNO therapy improves the hemodynamics of patients with acute heart failure. The effect that CPAP has on HFNO can alter the hemodynamics of patients with heart failure. A study by Roca et al. on ten heart failure patients with NYHA class III administration of HFNO with flow rates of 20 L/min and 40 L/min showed that the inferior vena cava collapse index was lower than baseline 20% and 53%. The hemodynamic status increases due to intrathoracic pressure and decreases cardiac preload. HFNO decreases ventricular afterload and improves hemodynamic status.[21]

A study conducted by Kang et al. In acute heart failure patients receiving HFNO or intubation as initial therapy after clinical deterioration showed similar results in increased oxygen saturation and clinical outcome. So maybe HFNO can be given as initial oxygen therapy in acute heart failure.[22]

Patients with severe cardiogenic pulmonary edema are frequently associated with hypercapnia. Hypercapnia causes decreased consciousness and central apnea and increase the patient's intubation rate.[23] NIV is known to have advantages in treating hypercapnia in patients.[17] A study conducted by Marjanovic et al. compared hypercapnia in cardiogenic pulmonary edema patients receiving HFNO and NIV. Baseline reduction in $PaCO_2$ was measured in the first 1 hour, wherein HFNO patients decreased 7 mmHg while in NIV 3 mm Hg. There were no differences in respiratory rate, pH, and breathwork between the two groups.[23] Of all the clinical benefits, HFNO may be provided as an alternative to heart failure patients who cannot tolerate NIV.[24]

The management of heart failure involves reducing preload and afterload to decrease pulmonary congestion. The physiological effect of NIV to help to reduce preload and afterload in decompensated heart failure is also mentioned in HFNO usage. In the presence of heart failure, increased work of breathing is poorly tolerated. Negative intrathoracic pressure increases systemic ventricular afterload, while increased diaphragmatic oxygen consumption reduces oxygen delivery. HFNO helps to decrease afterload by providing positive pressure and abating sympathetic nervous system activity by improving respiratory work. HFNO also decreases pulmonary vascular resistance (PVR) by restoring functional residual capacity (FRC) and improving alveolar oxygen tension in patients with pulmonary edema.[25] In the study of Roca et al.[4], HFNO could reduce respiratory rate and decrease inspiratory collapse of the inferior vena cava. The right atrium is a surrogate for right ventricular preload. It can be estimated from inferior vena cava collapsibility. The decrease in inspiratory collapse indicated a reduction in right ventricular preload.[13]

Table 4. Study on HFNO in cardiogenic pulmonary edema

Studi	Population	Experiment	Control	Outcome
(Ko et al., 2020)[3]	Patients suspended for pulmonary edema due to heart failure	HFNO (n=36)	Conventional oxygen therapy (n=33)	Significant differences in breath rate, SpO_2 at 30 and 60 minutes. The differences were significant in PaO_2 and SpO_2 at 30 and 60 minutes
(Makdee et al, 2017)[26]	ER patients with cardiogenic pulmonary edema	HFNO (n=63)	Conventional oxygen therapy (n=65)	Respiration rates decreased in the HFNO group at 15, 30, and 60 minutes
(Sener et al., 2020)[27]	Patients with hypertensive pulmonary edema	HFNO (n=62)	Conventional oxygen therapy (n=50)	HFNO shortens the length of stay in the ER and ICU. Increased heart rate, respiratory rate, and BGA
(Chang et al., 2020)[28]	Post-extubation heart failure patients with ejection fraction <50%	HFNO (n=58)	NIPPV (n=46)	There was no difference in treatment failure in the two groups
(Roca et al., 2016)[4]	NYHA III heart failure patients and ejection fraction <45%	HFNO (n=10)	None	HFNO is associated with the decreased inspiratory collapse of the inferior vena cava and decreased respiratory rate
(Carratala et al., 2011)[20]	Patients with acute heart failure were treated early on NIV and had refractory hypoxemia	HFNO (n=5)	None	The use of HFNO for 24 hours showed an increase in PaO_2, a degree of severity, and a decrease in respiratory rate

CONCLUSION

HFNO is oxygen therapy superior to conventional oxygen therapy and has many benefits in respiratory failure. HFNO can deliver high doses of oxygen up to a maximum of 60 liters per minute with a constant oxygen fraction between 21-100%. Compared with NIV, the use of HFNO is more comfortable for the patient and well-tolerated. HFNO is beneficial in patients with acute heart failure with hypoxemia because of its ease of use, physiological benefits, and good tolerance levels. Therefore, HFNO may benefit and be used as an alternative to acute heart failure patients who cannot tolerate NIV.

AUTHORS' CONTRIBUTION

NPN, RJ, and SPH: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work.

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REFERENCES

1. Pappas L, Filippatos G. Pulmonary Congestion in Acute Heart Failure: From Hemodynamics to Lung Injury and Barrier Dysfunction. Rev Española Cardiol (English Ed). 2011;64(9):735-7.
2. Adhikari P, Bhattarai S, Gupta A, Ali E, Ali M, Riad M, et al. Physiological Effects of High-Flow Nasal Cannula Therapy and Its Use in Acute Cardiogenic Pulmonary Edema. Cureus. 2021;13(2):e13372.
3. Ko DR, Beom J, Lee HS, You JS, Chung HS, Chung SP. Benefits of High-Flow Nasal Cannula Therapy for Acute Pulmonary Edema in Patients with Heart Failure in the Emergency Department: A Prospective Multi-Center Randomized Controlled Trial. J Clin Med. 2020;9(6):1937.
4. Roca O, Hernández G, Díaz-Lobato S, Carratalá JM, Gutiérrez RM, Masclans JR. Current evidence for the effectiveness of heated and humidified high flow nasal cannula supportive therapy in adult patients with respiratory failure. Crit Care [Internet] 2016;20(1):1-13. Available from: http://dx.doi.org/10.1186/s13054-016-1263-z
5. Alshahrani MS, Alshaqaq HM, Alhumaid J, Binammar AA, AlSalem KH, Alghamdi A, et al. High-Flow nasal cannula treatment in patients with COVID-19 acute hypoxemic respiratory failure: A prospective cohort study. Saudi J Med Med Sci. 2021;9(3):215.
6. Rochwerg B, Einav S, Chaudhuri D, Mancebo J, Mauri T, Helvis Y et al. The role for high flow nasal cannula as a respiratory support strategy in adults: a clinical practice guideline. Intensive Care Med [Internet] 2020;46(12):2226-37. Available from: https://doi.org/10.1007/s00134-020-06312-y
7. Cleland J, Yassin A, Khadjooi K. CME Acute medicine. Clin Med (Northfield Il). 2010;10(1):59-64.
8. Fonarow GC, Abraham WT, Albert NM, Stough WG, Gheorghiade M, Greenberg BH, O'Connor CM et al. Factors identified as precipitating hospital admissions for heart failure and clinical outcomes: Findings from OPTIMIZE-HF. Arch Intern Med. 2008;168(8):847-54.
9. Roguin A, Behar D, Ami H Ben, Reisner S, Edelstein S, Linn S et al. Long-term prognosis of acute pulmonary oedema - An ominous outcome. Eur J Heart Fail. 2000;2(2):137-44.
10. PERKI. Pedoman Tatalaksana Gagal Jantung. In Perhimpunan Dokter Spesialis Kardiovaskular Indonesia. 2020;2:1.
11. Tubaro M, Vranckx P, Price S. Oxford Medicine Online The ESC Textbook of Intensive and Acute Cardiovascular Care (2nd ed .). 2016.
12. Arrigo M, Parissis JT, Akiyama E, Mebazaa A. Understanding acute heart failure: Pathophysiology and diagnosis. Eur Hear Journal, Suppl 2016;18:G11-8.
13. Chua MT, Kuan W Sen. The use of high-flow nasal cannula in acute decompensated heart failure: ready for prime time yet? J Emerg Crit Care Med. 2017;1(9):22.
14. Sepehrvand N, Ezekowitz JA. Oxygen Therapy in Patients With Acute Heart Failure: Friend or Foe? JACC Hear Fail. 2016;4(10):783-90.
15. Spoletini G, Alotaibi M, Blasi F, Hill NS. Heated humidified high-flow nasal oxygen in adults: Mechanisms of action and clinical implications. Chest. 2015;148(1):253-61.
16. Alshahrani MS, Alshahaq HM, Alhumaid J, Binammar AA, AlSalem KH, Alghamdi A, et al. High-flow nasal cannula for acute hypoxemic respiratory failure in patients with COCID-19 Acute Hypoxemic Respiratory Failure. Can J Anaesth. 2020;67(9):1217-48.
17. Lu X, Xu S. Therapeutic effect of high-flow nasal cannula on severe COVID-19 patients in a makeshift intensive-care unit: A case report. Medicine (Baltimore). 2020;99(21):e20393.
18. Karamouzos V, Fligou F, Gogos C, Velissaris D. High flow nasal cannula oxygen therapy in adults with COVID-19 respiratory failure. A case report. Monaldi Arch Chest Dis. 2020;90(2):337-40.
19. Frat JP, Coudroy R, Marjanovic N, Thille AW. High-flow nasal oxygen therapy and noninvasive ventilation in the management of acute hypoxemic respiratory failure. Ann Transl Med. 2017;5(14):1-8.
20. Carratalá Perales JM, Llorens P, Brouzet B, Jimenez AR, Fernandez-Canadas JM, Dalmau JC et al. High-Flow Therapy via Nasal Cannula in Acute Heart Failure. Rev Española Cardiol (English Ed). 2011;64(8):723-5.
21. LIU X, WU R, LAI L, LIN J. Clinical application of High-flow nasal cannula oxygen therapy in acute heart failure. Food Sci Technol. 2021;2061:1-8.
22. Kang MG, Kim K, Ju S, Park HW, Lee JS, Koh Js et al. Clinical efficacy of high-flow oxygen therapy through nasal cannula in patients with acute heart failure. J Thorac Dis. 2019;11(2):410-7.
23. Marjanovic N, Flacher A, Drouet L, Gouhnec AL, Said H, Vigneau JF et al. High-flow nasal cannula in early emergency department management of acute hypercapnic respiratory failure due to cardiogenic pulmonary edema. Respir Care. 2020;65(9):1241-9.
24. Masip J. Noninvasive Ventilation in Acute Heart Failure. Curr Heart Fail Rep. 2019;16(4):89–97.
25. Inata Y, Takeuchi M. Complex effects of high-flow nasal cannula therapy on hemodynamics in the pediatric patient after cardiac surgery. J Intensive Care. 2017;5(1):10-2.
26. Makdee O, Monsomboon A, Surabenjawong U, Prahruekti N, Chaisirin W, Chakorn T et al. High-Flow Nasal Cannula Versus Conventional Oxygen Therapy in Emergency Department Patients With Cardiogenic Pulmonary Edema: A Randomized Controlled Trial. Ann Emerg Med. 2017;70(4):465-72.
27. Şener K, Çaliş M, Köseoğlu Z, Sari Z, Polat M, Uzucek DM et al. Comparison of high-flow oxygen treatment and standard oxygen treatment in patients with hypertensive pulmonary edema. Anatol J Cardiol. 2020;24(4):260-6.
28. Chang CJ, Chiang LL, Chen KY, Feng PH, Su CL, Hsu HS. High-Flow Nasal Cannula versus Noninvasive Positive Pressure Ventilation in Patients with Heart Failure after Extubation: An Observational Cohort Study. Can Respir J. 2020;2020:6736475.

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