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Contents

Thrice Monthly Volume 11 Number 13 May 6, 2023

OPINION REVIEW

- 2855 Long-term implications of fetal growth restriction
D'Agostin M, Di Sipio Morgia C, Vento G, Nobile S

REVIEW

- 2864 Appraisal of gastric stump carcinoma and current state of affairs
Shukla A, Kalayarasan R, Gnanasekaran S, Pottakkat B
- 2874 Burden of severe infections due to carbapenem-resistant pathogens in intensive care unit
Pace MC, Corrente A, Passavanti MB, Sansone P, Petrou S, Leone S, Fiore M

MINIREVIEWS

- 2890 Individualized diabetes care: Lessons from the real-world experience
Khor XY, Pappachan JM, Jeeyavudeen MS
- 2903 Clinical management of dural defects: A review
Dong RP, Zhang Q, Yang LL, Cheng XL, Zhao JW
- 2916 Potential impact of music interventions in managing diabetic conditions
Eseadi C, Amedu AN
- 2925 Implications of obesity and adiposopathy on respiratory infections; focus on emerging challenges
Lempesis IG, Georgakopoulou VE

ORIGINAL ARTICLE

Case Control Study

- 2934 Association of C-reactive protein and complement factor H gene polymorphisms with risk of lupus nephritis in Chinese population
Li QY, Lv JM, Liu XL, Li HY, Yu F

Retrospective Study

- 2945 Comparison of the application value of transvaginal ultrasound and transabdominal ultrasound in the diagnosis of ectopic pregnancy
Hu HJ, Sun J, Feng R, Yu L

Observational Study

- 2956 Assessment of knowledge, cultural beliefs, and behavior regarding medication safety among residents in Harbin, China
Liu XT, Wang N, Zhu LQ, Wu YB

SYSTEMATIC REVIEWS

- 2966 Palliative oral care in terminal cancer patients: Integrated review
Silva ARP, Bodanezi AV, Chrun ES, Lisboa ML, de Camargo AR, Munhoz EA

META-ANALYSIS

- 2981 Effect of preoperative inspiratory muscle training on postoperative outcomes in patients undergoing cardiac surgery: A systematic review and meta-analysis
Wang J, Wang YQ, Shi J, Yu PM, Guo YQ
- 2992 Efficacy and safety of intravenous tranexamic acid in total shoulder arthroplasty: A meta-analysis
Deng HM

CASE REPORT

- 3002 Awake laparoscopic cholecystectomy: A case report and review of literature
Mazzone C, Sofia M, Sarv  I, Litrico G, Di Stefano AML, La Greca G, Latteri S
- 3010 Bilateral malignant glaucoma with bullous keratopathy: A case report
Ma YB, Dang YL
- 3017 Finger compartment syndrome due to a high-pressure washer injury: A case report
Choi JH, Choi SY, Hwang JH, Kim KS, Lee SY
- 3022 Primary dedifferentiated chondrosarcoma of the lung with a 4-year history of breast cancer: A case report
Wen H, Gong FJ, Xi JM
- 3029 Importance of proper ventilator support and pulmonary rehabilitation in obese patients with heart failure: Two case reports
Lim EH, Park SH, Won YH
- 3038 Multiple flexor tendon ruptures due to osteochondroma of the hamate: A case report
Kwon TY, Lee YK
- 3045 Fractional flow reserve measured *via* left internal mammary artery after coronary artery bypass grafting: Two case reports
Zhang LY, Gan YR, Wang YZ, Xie DX, Kou ZK, Kou XQ, Zhang YL, Li B, Mao R, Liang TX, Xie J, Jin JJ, Yang JM
- 3052 Uterine artery embolization combined with percutaneous microwave ablation for the treatment of prolapsed uterine submucosal leiomyoma: A case report
Zhang HL, Yu SY, Cao CW, Zhu JE, Li JX, Sun LP, Xu HX
- 3062 Metachronous urothelial carcinoma in the renal pelvis, bladder, and urethra: A case report
Zhang JQ, Duan Y, Wang K, Zhang XL, Jiang KH
- 3070 Unusual phenomenon-“polyp” arising from a diverticulum: A case report
Liew JJJ, Lim WS, Koh FH

- 3076** Idiopathic steno-occlusive disease with bilateral internal carotid artery occlusion: A Case Report
Hamed SA, Yousef HA
- 3086** Solitary acral persistent papular mucinosis nodule: A case report and summary of eight Korean cases
Park YJ, Shin HY, Choi WK, Lee AY, Lee SH, Hong JS
- 3092** Eosinophilic fasciitis difficult to differentiate from scleroderma: A case report
Lan TY, Wang ZH, Kong WP, Wang JP, Zhang N, Jin DE, Luo J, Tao QW, Yan ZR
- 3099** Misdiagnosis of scalp angiosarcoma: A case report
Yan ZH, li ZL, Chen XW, Lian YW, Liu LX, Duan HY
- 3105** Discrepancy among microsatellite instability detection methodologies in non-colorectal cancer: Report of 3 cases
Şenocak Taşçı E, Yıldız İ, Erdamar S, Özer L

ABOUT COVER

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The primary aim of *World Journal of Clinical Cases* (WJCC, *World J Clin Cases*) is to provide scholars and readers from various fields of clinical medicine with a platform to publish high-quality clinical research articles and communicate their research findings online.

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Importance of proper ventilator support and pulmonary rehabilitation in obese patients with heart failure: Two case reports

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Abstract

BACKGROUND

The optimal treatment for heart failure (HF) is a combination of appropriate medications. Controlling the disease using only medical therapy is difficult in patients with HF, severe hypercapnia, and desaturation. These patients should first receive ventilator support followed by pulmonary rehabilitation (PR).

CASE SUMMARY

We report two cases in which arterial blood gas (ABG) improved and PR was possible with appropriate ventilator support. Two patients with extreme obesity complaining of worsening dyspnea—a 47-year-old woman and a 36-year-old man both diagnosed with HF—were hospitalized because of severe hypercapnia and hypoxia. Despite proper medical treatment, hypercapnia and desaturation resolved in neither case, and both patients were transferred to the rehabilitation department for PR. At the time of the first consultation, the patients were bedridden because of dyspnea. Oxygen demand was successfully reduced once non-invasive ventilation was initiated. As the patients' dyspnea gradually improved to the point where they could be weaned off the ventilator during the daytime, they started engaging in functional training and aerobic exercise. After 4 mo of follow-up, both patients were able to perform activities of daily living and maintain their lower body weight and normalized ABG levels.

CONCLUSION

Symptoms of patients with obesity and HF may improve once ABG levels are normalized through ventilator support and implementation of PR.

Key Words: Noninvasive ventilation; Heart failure; Obesity; Rehabilitation; Dyspnea; Case report

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Core Tip: We describe two patients with heart failure (HF) and obesity who experienced respiratory failure, including hypercapnia and hypoxia. Neither patient demonstrated a significant response to pharmacological management; however, in both cases, symptoms improved with noninvasive ventilation, and they were able to return to their daily life. These findings suggest that in patients with obesity and HF who developed pulmonary hypertension and cor pulmonale may need to be treated for obesity hypoventilation and sleep apnea. The symptoms of these comorbidities may improve when arterial blood gas levels are normalized with appropriate ventilator support and pulmonary rehabilitation.

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INTRODUCTION

Heart failure (HF) is a clinical syndrome characterized by shortness of breath, extreme fatigue, limb, and ankle swelling that are often accompanied by signs, including respiratory distress, gallop rhythm, and pulmonary edema. HF is most commonly caused by a structural or functional abnormality of the heart, resulting in increased intracardiac pressure and/or insufficient cardiac output at rest or during exercise [1]. HF may be classified as either acute or chronic depending on the time and speed of occurrence [2]. Acute HF refers to the rapid or gradual onset of symptoms and/or signs of HF that are sufficiently severe for the patient to seek urgent medical attention, leading to unplanned hospital admissions or emergency department visits [3,4]. Acute HF has a 1-year mortality rate of 20%–30% and an additional risk of hospitalization [5].

Pharmacological management is considered the optimal treatment for patients with acute HF [1]. However, in patients with HF and severe hypercapnia and desaturation, controlling the disease with medication alone is difficult. Appropriate ventilator support followed by pulmonary rehabilitation (PR) should be considered in such patients. Herein, we describe two patients with HF whose symptoms improved after arterial blood gas (ABG) levels normalized with the aid of noninvasive ventilation (NIV) without intubation, which was administered after medication and oxygen supply treatment proved ineffective due to severe hypercapnia.

CASE PRESENTATION

Chief complaints

Case 1: A 47-year-old woman with extreme obesity was admitted to the emergency department because of worsening dyspnea.

Case 2: A 36-year-old man with extreme obesity and chronic HF was admitted to the cardiology outpatient department because of worsening dyspnea.

History of present illness

Case 1: The patient visited the emergency room due to worsening dyspnea that had started 1 wk earlier, and systemic edema had worsened during the last 3 d. She was diagnosed with HF and admitted to the Department of Cardiology. Edema management was initiated because respiratory failure was suspected owing to the deterioration of her pulmonary edema. However, despite medical treatment, her hypercapnia and desaturation could not be reversed, and the patient was referred to the rehabilitation department for PR.

Case 2: The patient visited the emergency room because of worsening dyspnea, which had begun 2 wk earlier, and systemic edema. He was diagnosed with HF and admitted to the Department of Cardiology. He was alert at the time of hospitalization; however, he suddenly lost consciousness and was moved to the intensive care unit (ICU), where he was intubated and treated with mechanical ventilation. Therefore, edema management was initiated. However, despite medical treatment, an attempt to wean

him off the ventilator failed, his hypercapnia could not be reversed, and he was referred to our department for PR.

History of past illness

Case 1: The patient had a history of diabetes mellitus, hypertension (HTN), and chronic kidney disease (Table 1). She was also diagnosed with asthma, chronic HF, and pulmonary HTN within the previous year.

Case 2: The patient had a history of diabetes mellitus and HTN and had been diagnosed with HF approximately 6 mo prior to admission (Table 1).

Personal and family history

Case 1: The patient had no remarkable family history.

Case 2: The patient had no remarkable family history.

Physical examination

Case 1: On admission, the patient weighed 130 kg [body mass index (BMI): 48.63 kg/m²]. She was alert at the time of the first consultation, although her oxygen demand was high (15 L/min *via* an oxygen mask), and she was bedridden owing to dyspnea (Table 2).

Case 2: On admission, the patient's weight was 167.1 kg (BMI: 56.48 kg/m²). The patient was alert at the time of the first consultation. He had undergone extubation 2 days earlier. His oxygen demand was high (10 L/min *via* a T-piece), and he was bedridden because of dyspnea (Table 2).

Laboratory examinations

Case 1: When the patient arrived at the emergency room, the ABG analysis (ABGA) results indicated severe hypercapnia: pH 7.307; pCO₂ 97.1 mmHg; pO₂ 73.3 mmHg; and SaO₂ 93.2%. At the time of the consultation, her ABG levels still indicated hypercapnia: pH, 7.354; pCO₂ 96.7 mmHg; pO₂ 63.8 mmHg; and SaO₂ 88.6% (Table 2).

Case 2: On ICU admission, his ABGA results indicated severe hypercapnia and hypoxemia: pH 7.148; pCO₂ 110 mmHg; pO₂ 79 mmHg; and SaO₂ 91.7% (Table 2). At the time of the consultation, the patient's ABG levels still indicated hypercapnia: pH 7.351; pCO₂ 74.9 mmHg; pO₂ 103 mmHg, and SaO₂ 97.5% (Table 2).

Imaging examinations

Case 1: Upon arrival at the emergency room, a chest radiograph indicated cardiomegaly (Figure 1A), and chest computed tomography (CT) revealed mosaic attenuation in both lungs and mild pericardial and pleural effusion. Transthoracic echocardiography (TTE) revealed a D-shaped left ventricle (LV) with normal LV systolic function (ejection fraction, 56%) and right ventricle (RV) dysfunction with severe tricuspid regurgitation due to coaptation failure, severe resting pulmonary HTN, RV dilatation (44 mm), right atrial enlargement, and a dilated main pulmonary artery (33 mm). These findings indicated resting pulmonary HTN deterioration compared to the TTE results that the patient had received 3 mo earlier (severe tricuspid regurgitation with moderate resting pulmonary HTN, RV dilatation (43 mm), right atrial enlargement).

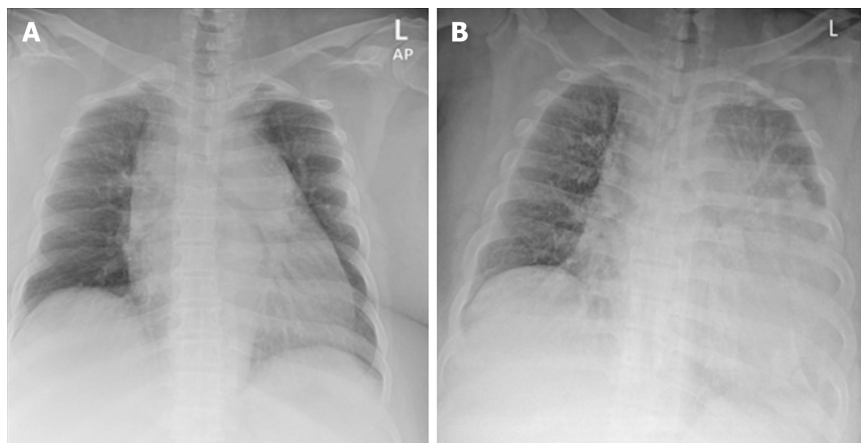
Case 2: On ICU admission, chest radiography indicated cardiomegaly, pericardial effusion, and pulmonary interstitial edema with bilateral pleural effusion (Figure 1B). TTE revealed concentric LV hypertrophy, global hypokinesia with mild LV systolic dysfunction (ejection fraction, 47%), left atrial enlargement (48 mm), RV dysfunction with resting pulmonary HTN, diastolic dysfunction (grade 1), and mild pericardial effusion without hemodynamic significance. These findings indicated an aggravation of LV ejection fraction (57% → 47%), newly developed RV dysfunction with mild resting pulmonary HTN, and a decrease in LV end-diastolic pressure (15 → 11) compared to the TTE results that the patient had received 7 mo earlier. Chest CT revealed patchy consolidation with decreasing lung volume in the dependent portion of both lungs, an increase in heart size, and mild pericardial effusion. Therefore, the patient was diagnosed with aspiration pneumonia and cardiomegaly with a small pericardial effusion.

FINAL DIAGNOSIS

Both patients were diagnosed with HF and respiratory failure.

Table 1 Demographic data		
	Case 1	Case 2
Age	47	36
Gender	F	M
Height (cm)	163.5	172
Weight (kg)	130	167.1
Body mass index (kg/m ²)	48.93	56.48
Past medical history		
Hypertension	O	O
Diabetes mellitus	O	O
Heart failure	O	O
Pulmonary hypertension	O	O
Cor pulmonale	O	×
Chronic kidney disease	O	×
Bronchial asthma	O	×

F: Female; M: Male.



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Figure 1 Chest X-ray at admission. A: Patient 1, cardiomegaly; B: Patient 2, cardiomegaly, pericardial effusion, and pulmonary interstitial edema with pleural effusion on both sides.

TREATMENT

Case 1

NIV was initiated, and oxygen demand was gradually reduced (O₂ 1–2 L). As the patient’s dyspnea and hypercapnia/hypoxia gradually improved to the point where she could be weaned off the ventilator during the day, she began engaging in aerobic exercise and functional training. In the early stages, evaluation using a pulmonary function test was not possible because of the patient’s severe dyspnea. When her status improved with the ventilation treatment, the pulmonary function test was finally performed and yielded a forced vital capacity (FVC) of 2.27 L (75% of predicted maximum) and a forced expiratory volume in the first second (FEV1) of 1.71 L (66% of predicted maximum), resulting to an FEV1/FVC of 75%.

Case 2

We changed the patient’s treatment from mechanical ventilation to NIV because we expected long-term ventilator use after extubation. NIV was started immediately, and his oxygen demand was gradually reduced. At the time of discharge from the ICU, he was still bedridden because of dyspnea and required oxygen at a rate of 5 L/min (administered *via* a nasal prong) during the day; NIV was continued during

Table 2 Comparison of admission and discharge results

	Patient 1				Patient 2			
	Adm	RM consult	Discharge	F/U (4 mo)	Adm	RM consult	Discharge	F/U (4 mo)
Height (cm)	163.5	163.5	163.5	163.5	172	172	172	172
Weight (kg)	130	105.5	98.2	88	167.1	138.4	135.7	138
BMI	48.63	39.47	36.73	32.92	56.48	46.78	45.87	46.65
Noninvasive ventilation								
Mode			iVAPS	iVAPS			PCV	PCV
Setting			Target Va 8	Target Va 8			IPAP 19	IPAP 19
			PS 2-14	PS 2-14			EPAP 5	EPAP 5
			EPAP 4-10	EPAP 4-10				
O ₂			4 L/min	4 L/min			4 L/min	4 L/min
Apply time			9 pm-7 am	9 pm-7 am			10 pm-6 am	10 pm-6 am
Daytime O ₂	15 L/min	15 L/min	1.5L/min	None	10 L/min	10 L/min	None	None
(reserve mask)								
ABG test ¹								
pH	↓7.307	7.354	7.39	-	↓7.148	7.351	7.406	-
PCO ₂ (mmHg)	↑97.1	↑96.7	↑51.7	-	↑110	↑74.9	37.7	-
PO ₂ (mmHg)	↓73.3	↓63.8	↓72.3	-	↓79	103	97.3	-
SaO ₂ (%)	93.2	88.6	94.3	-	91.7	97.5	98	-
Pulmonary function test								
PCF (L/min)	NT	NT	260	-	NT	340	370	510
FVC (L)	NT	NT	2.27	3.21	NT	NT	4.72 (102%)	4.64 (101%)
			-0.75	-0.95				
FEV ₁ (L)	NT	NT	1.71	2.39	NT	NT	3.46 (101%)	2.76
			-0.66	-0.93				-0.81
FEV ₁ /FVC (%)	NT	NT	75	75	NT	NT	73	59
PEF (%)	NT	NT	72	-	NT	NT	84	56
6MWT	NT	NT	234 m	376 m	NT	389 m	480 m	549 m
AHI ²	Not done						78.1/h	

¹Measured in the morning (reflecting respiration values during sleep).

²Measured value use of polysomnography.

RM: Rehabilitation medicine; F/U: Follow up; BMI: Body mass index; NIV: Non-invasive ventilation; ABGA: Arterial blood gas analysis; PFT: Pulmonary function test; NT: Not testable; PCF: Peak cough flow; FVC: Forced vital capacity; FEV₁: Forced expiratory volume in the first second; PEF: Peak expiratory flow; 6MWT: 6-minute walking test; AHI: Apnea/hypopnea index.

the night.

OUTCOME AND FOLLOW-UP

Case 1

At the time of discharge, the patient could move around with the aid of a walking device and only needed oxygen at a rate of 1.5 L/min during the day; ventilation continued, but only during the night. On the day of discharge, she performed a 6-minute walking test (6MWT), which yielded a 6MWT distance of 234 m; her weight at that point was 98.2 kg (BMI: 36.73 kg/m²). The ABGA results indicated

hypercapnia, although her levels (pH 7.390; pCO₂ 51.7 mmHg; pO₂ 72.3 mmHg; and SaO₂ 94.3%) improved compared to those at the last assessment. The patient was discharged and prescribed home ventilation and O₂ therapy.

The patient visited the outpatient department. She still relied on NIV during the night but did not need O₂ supply during the day. Her O₂ saturation in room air was $\geq 93\%$. The patient reported a subjective improvement in dyspnea, and the pulmonary function test also indicated improvements, with an FVC of 3.21 L (95% of predicted maximum), an FEV₁ of 2.39 L (93% of the predicted maximum), and an FEV₁/FVC ratio of 75%. The 6MWT distance measured at this visit was 376 m, and her weight was 88 kg (BMI: 32.92 kg/m²). After 4 mo of follow-up, the patient returned to work while performing activities of daily living independently, maintaining her body weight with aerobic exercises, and relying on NIV only during the night.

Case 2

As the patient's dyspnea gradually improved, he started engaging in aerobic exercises. In the early stages, a pulmonary function test could not be performed because of severe dyspnea. When his respiratory function had improved, he finally underwent the test, which yielded an FVC of 4.72 L (102% of predicted maximum), an FEV₁ of 3.46 L (101% of predicted maximum), and thus an FEV₁/FVC of 73%. The patient expressed an interest in active rehabilitation treatment and was therefore transferred to our department. At the time of transfer to the rehabilitation department, he did not rely on additional oxygen during the day and used NIV only at night.

Polysomnography was performed because we observed desaturation during sleep, and the use of NIV was maintained during sleep because of the patient's severe obstructive sleep apnea (AHI 78.1/h). He engaged in aerobic exercises on an ergometer and treadmill, and gait training using a walking device. He also exercised to strengthen his muscles as his lower extremities were weak after he had been bedridden for approximately a month. When he was discharged from the hospital, the ABGA results and 6MWT demonstrated improvements, and the patient could move around independently using a walking device. The 6MWT yielded a distance of 480 m, and his weight was 135.7 kg (BMI: 45.87 kg/m²), indicating a reduction compared to his weight at admission (167.1 kg). When the patient was discharged, his ABGA results were within the normal range as follows: pH, 7.406; pCO₂ 37.7 mmHg; pO₂ 97.3 mmHg; and SaO₂ 98.0%.

The patient visited the outpatient department. He still relied on NIV at night and breathing normal room air during the day. He reported subjective improvement in his dyspnea. The pulmonary function test results were similar to those at the last assessment, and yielded an FVC of 4.64 L (101% of predicted maximum), an FEV₁ of 2.76 L (81% of the predicted maximum), and an FEV₁/FVC of 59%. His 6MWT distance improved compared to the previous test (510 m), and his weight was similar at 138 kg (BMI: 46.65 kg/m²). After 4 mo of follow-up, the patient returned to work while performing activities of daily living independently, maintaining his body weight with aerobic exercises, and relying on NIV only during the night.

DISCUSSION

These cases demonstrate that appropriate ventilator application and PR in patients with obesity and HF complaining of dyspnea caused by severe hypercapnia can improve symptoms and help patients return to daily life. Because dyspnea is a major barrier for patients with HF in performing activities of daily living, controlling its symptoms is especially important. HF causes complications, such as arrhythmia, thromboembolism, respiratory muscle weakness, and pulmonary HTN[6]. Obesity can also occur because of reduced physical activity[7] and is associated with mortality and various complications, including diabetes mellitus, heart problems, dementia, and cancer[8]. Therefore, treating HF is important and generally involves pharmacological management, such as diuretic therapy, in acute HF. Furthermore, whether the patient's HF is caused by hypoventilation needs to be considered.

Previous studies have demonstrated that NIV is more effective than conventional oxygen therapy, improves dyspnea, and decreases intubation rates for acute cardiogenic pulmonary edema and acute HF associated with pulmonary disease[9,10]. By contrast, large randomized trials have reported that NIV application does not lead to a reduction in intubation rates; however, this observation might have been attributed to the relatively low intubation rates in the study patient population[11]. Additionally, NIV support is recommended as adjuvant therapy in patients with acute cardiogenic pulmonary edema with severe dyspnea or when medication treatment is ineffective because it has been proven to improve dyspnea and metabolic abnormalities in a faster and safer way than standard oxygen therapy[11,12]. Our two patients who did not significantly benefit from pharmacological management demonstrated symptom improvement and were able to return to their daily lives with the aid of appropriate NIV.

Obesity may occur in response to decreased physical activity in patients with HF or may cause HF by contributing to cardiac hemodynamics, endothelial dysfunction, insulin resistance, vascular changes, and metabolic disorders, including cardiac lipotoxicity[13]. Therefore, obesity should be carefully monitored in patients with HF. Previous studies have also reported independent associations between

obesity and pulmonary HTN and between obesity and mortality in the presence of pulmonary HTN [14]. Obesity, insulin resistance, and sleep apnea cause pulmonary HTN, which impairs endothelial function [15]. Moreover, patients with obesity hypoventilation syndrome may experience daytime hypoventilation, chronic hypoxemia, polycythemia, pulmonary HTN, and cor pulmonale [16], which increases the likelihood that these patients will require invasive mechanical ventilation or ICU admission [17].

Dyspnea exacerbation can contribute to functional disabilities that degrade the quality of life of patients with HF, progress over time, and are associated with poor prognosis [18]. Exercise-based cardiac rehabilitation (CR) for all patients with HF has been proven to be safe and effective in improving heart and body functions, reducing readmission rates, and improving quality of life [19,20]. Chronic HF management guidelines in some countries specifically list exercise-based CR as a category I recommendation [21,22]. The two patients presented here were bedridden when rehabilitation was initiated, were able to improve their dyspnea symptoms through NIV until they were able to engage in gait training, and improved further with the aid of breathing education and aerobic exercises until they could resume performing activities of daily living. These two cases demonstrate that respiratory assistance should be prioritized at the start of rehabilitation to facilitate training thereafter.

CONCLUSION

Patients with obesity and HF who develop pulmonary HTN and cor pulmonale need to be assessed and potentially treated for obesity hypoventilation and sleep apnea, in addition to receiving medication for HF. The two reported cases suggest that the symptoms of these comorbidities may improve once ABG levels are normalized through appropriate ventilator support and PR.

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