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ACEI 及无创呼吸机辅助治疗心衰合并 OSAHS 的疗效及对血清 BNP 水平的影响 *

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摘要 目的：探讨血管紧张素转换酶抑制剂(Angiotensin-converting enzyme inhibitor，ACEI)联合无创呼吸机辅助治疗心衰合并阻塞性睡眠呼吸暂停低通气综合征(Obstructive sleep apnea hypopnea syndrome，OSAHS)疗效及对血清脑钠素(Brain natriuretic peptide，BNP)水平的影响。**方法：**抽取我院自 2013 年 1 月到 2019 年 4 月收治的 98 例心衰合并 OSAHS 患者，根据治疗方法分为对照组(49 例，ACEI 常规治疗)与实验组(49 例，ACEI 联合无创呼吸机辅助治疗)，比较两组患者治疗前后红细胞生成素(Erythropoietin，EPO)、血红蛋白(hemoglobin，Hb)、红细胞计数(red blood cell，RBC)、平均红细胞蛋白含量(Mean corpuscular protein content，MCH)、血细胞比容(Hematocrit，HCT)、平均红细胞体积(Mean red blood cell volume，MCV)、夜间平均最低血氧合度(Lowest oxygen saturation，$LSaO_2$)、睡眠呼吸暂停低通气指数(apnea hypopnea index，AHI)、血清脑钠素(Brain natriuretic peptide，BNP)水平、中心收缩压(systolic pressure，SP)及中心舒张压(diastolic pressure，DP)的变化。**结果：**治疗后，两组患者 EPO、Hb、RBC、MCH、HCT、MCV 水平均明显低于治疗前，且实验组患者的以上指标水平均显著低于对照组(P<0.05)。两组治疗后 $LSaO_2$ 高于明显高于治疗前，AHI 水平低于治疗前，且实验组 $LSaO_2$ 高于对照组，AHI 水平低于对照组(均 P<0.05)。治疗后两组患者 BNP、SP 和 DP 明显低于治疗前(P<0.05)，且实验组患者的以上指标水平低于对照组(P<0.05)。实验组患者的术后并发症发生率明显低于对照组(P<0.05)。**结论：**ACEI 及无创呼吸机辅助治疗心衰合并 OSAHS 可改善患者的睡眠紊乱、睡眠呼吸障碍、心衰及高血压，具有临床推广应用的价值。

关键词：血管紧张素转换酶抑制剂；无创呼吸机；心衰；心衰合并阻塞性睡眠呼吸暂停低通气综合征；脑钠素；临床效果

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Efficacy of ACEI and Non-invasive Ventilator in the Treatment of Heart Failure Combined with OSAHS and Its Effect on the Serum BNP Levels*

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ABSTRACT Objective: To investigate the efficacy of angiotensin-converting enzyme inhibitor (ACEI) and non-invasive ventilator in the treatment of heart failure with obstructive sleep apnea hypopnea syndrome (OSAHS) and its effect on BNP. **Methods:** 98 patients with heart failure and OSAHS from January 2013 to April 2019 were enrolled. According to the treatment, they were divided into control group (49 cases, conventional treatment with ACEI) and experimental group (49 cases, ACEI combined with non-invasive breathing). Machine-assisted treatment), comparing EPO, Hb, RBC, MCH, HCT, MCV, $LSaO_2$, AHI, serum BNP, SP, DP before and after treatment in the two groups. **Results:** After treatment, the levels of EPO, Hb, RBC, MCH, HCT and MCV in the two groups were significantly lower than those before treatment, and the above indicators in the experimental group were significantly lower than those in the control group (P<0.05). The $LSaO_2$ levels in the two groups were significantly higher than those before treatment, and the AHI level was lower than that before the treatment, and the $LSaO_2$ in the experimental group was higher than that in the control group, and the AHI level in the experimental group was lower than that in the control group (both P<0.05). After treatment, the BNP, SP and DP in the two groups were significantly lower than those before treatment (P<0.05), and the above indexes in the experimental group were lower than that in the control group (P<0.05). The incidence of postoperative complications in the experimental group was significantly lower than that in the control group (P<0.05). **Conclusion:** ACEI and non-invasive ventilator assisted heart failure combined with OSAHS can improve the patient's sleep disorder, sleep-disordered breathing, heart failure and high blood pressure, and has the value of clinical application.

Key words: ACEI; Non-invasive ventilator; Heart failure; OSAHS; BNP; Clinical effect

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前言

心力衰竭(Heart failure)是由于心脏功能发生障碍,不能将回心血量充分排出,导致动脉系统血液灌注不足,引起心脏循环障碍[1]。临床上,心力衰竭并不是独立存在的疾病,其症候群表现出多样性,极易出现其他并发症[2],阻塞性睡眠呼吸暂停低通气综合征 (obstructive sleep apnea hypopnea syndrome,OSAHS)是其中的一种并发症,临床表现有夜间睡眠打鼾伴呼吸暂停和白天嗜睡[3,4]。由于患者睡眠期间容易出现低通气及呼吸暂停,而引发低氧血症、高碳酸血症、高红细胞生成素,最终导致多器官功能障碍[5]。研究表明,血管紧张素转换酶抑制剂(ACEI)可显著降低 OSAHS 合并高血压患者的睡眠呼吸暂停低通气指数(AHI),增加睡眠时最低血氧饱和度($LSaO_2$),对 OSAHS 合并高血压患者的睡眠呼吸障碍有改善作用。但常因 ACEI 作用时程不同、对无症状左室功能不全治疗与否、以及对猝死和心律失常的影响等方面的不确定因素,使 ACEI 的疗效争议颇大[6]。此外,无创呼吸机对慢性阻塞性肺病(COPD)合并 OSAHS 的治疗效果显著,但对其在心衰合并 OSAHS 中的治疗作用却鲜少报道。本研究通过分析我院收治的 98 例心衰合并 OSAHS 患者,探讨了 ACEI 及无创呼吸机辅助治疗心衰合并 OSAHS 疗效及对血清 BNP 水平的影响,现报道如下。

1 资料与方法

1.1 一般资料

选择 2013 年 1 月到 2019 年 4 月我院收治的 98 例心衰合并 OSAHS 患者,纳入标准:符合《中国心力衰竭诊断和治疗指南 2014》与《阻塞性睡眠呼吸暂停通气综合征诊治指南》中的诊断标准[7,8];签订知情同意书。排除标准:严重心肝肾功能不全者;先天性心脏病者;脑卒中病史;精神病者。根据治疗方法将患者分为对照组与实验组,对照组:49 例,男性 30 例,女性 19 例;年龄(41-76)岁,平均年龄(54.42± 1.45)岁;心力衰竭病程(3-7)年,平均病程(4.34± 1.21)年;睡眠呼吸暂停低通气指数:中度 35 例,重度 14 例。实验组:49 例,男性 31 例,女性 18 例;年龄(40-78)岁,平均年龄(54.23± 1.56)岁;心力衰竭病程(3-9)年,平均病程(4.56± 1.12)年;睡眠呼吸暂停低通气指数:中度 32 例,重度 17 例。两组的基本资料比较差异无统计学意义(P>0.05),具有可比性。

1.2 治疗方法

对照组:给予 ACEI 治疗,心衰不稳定期采用利尿剂、硝酸酯等药物,心衰稳定后加用 ACEI 治疗,连续治疗 12 w。实验组:在对照组基础之上给予无创呼吸机辅助治疗,采用全自动 CPAP 呼吸机, 经人工呼吸机压力滴定后行无创呼吸机治疗,根据患者实际情况选择适合的压力参数,压力为 8-18 cm H20,每晚呼吸机治疗时间超过 5 h,连续治疗 12 w。

1.3 观察指标

观察及比较两组治疗前后红细胞生成素(Erythropoietin,EPO)、血红蛋白(hemoglobin,Hb)、红细胞计数(red blood cell,RBC)、平均红细胞蛋白含量(Mean corpuscular protein content,MCH)、血细胞比容(Hematocrit,HCT)、平均红细胞体积(Mean red blood cell volume,MCV)、夜间平均最低血氧合度(Lowest oxygen saturation,$LSaO_2$)、睡眠呼吸暂停低通气指数(apnea hypopnea index,AHI)、脑钠素(Brain natriuretic peptide,BNP)、中心收缩压(systolic pressure,SP)及中心舒张压(diastolic pressure,DP)。

清晨空腹抽取 3 mL 静脉血,采血 3 h 内进行离心处理,转速 3000 转 /min,离心半径为 17 cm。分离血清后,置于 -20 ℃冰箱内保存。采用酶联免疫吸附法检测 EPO, 血常规检测 Hb、RBC、MCH、HCT、MCV,采用美国 emdla 多导睡眠检测仪检测 $LSaO_2$、AHI,采用酶联免疫吸附法检测 BNP,采用中心动脉压检测仪检测 SP、DP。试剂盒均购买于武汉默沙克生物科技有限公司,严格按照说明书操作。

1.4 统计学方法

本次研究数据采用 SPSS19.0 软件进行统计分析,EPO、Hb、RBC、MCH、HCT、MCV、$LSaO_2$、AHI、BNP、SP、DP 水平采用均数表示计量,用独立样本 t 检验,治疗前后比较采用配对样本 t 检验。以 P<0.05 时表示差异具有统计学意义。

2 结果

2.1 两组治疗前后 EPO 及血常规指标水平的比较

治疗前, 两组的 EPO、Hb、RBC、MCH、HCT、MCV 水平比较差异均无统计学意义(P>0.05);治疗后,两组 EPO、Hb、RBC、MCH、HCT、MCV 水平明显低于治疗前(P<0.05),且实验组以上指标水平均低于对照组(P<0.05)。详见表 1 所示。

表 1 两组患者治疗前后 EPO 及血常规指标水平比较($\bar{x}\pm s$)

Table 1 Comparison of EPO and blood routine index levels before and after treatment between two groups of patients($\bar{x}\pm s$)

Groups	EPO(U/L)		Hb(g/L)		RBC(× 10^{12})		MCH(pg)		HCT(%)		MCV(fl)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Control group (n=49)	34.12± 2.12	30.23± 2.12	143.34± 13.45	139.34± 11.25	4.76± 1.12	4.56± 1.02	34.56± 3.23	32.23± 3.12	0.45± 0.03	0.40± 0.02	86.78± 8.56	83.23± 7.34
Experimental group (n=49)	34.23± 1.34	25.45± 2.16	143.67± 12.24	130.56± 10.21	4.78± 1.23	4.12± 1.03	34.67± 3.12	29.23± 3.11	0.46± 0.02	0.35± 0.15	86.45± 8.12	79.23± 7.12
t	1.156	6.478	2.178	7.866	1.262	8.564	2.112	9.441	1.454	8.565	1.232	9.445
P	0.145	0.012	0.532	0.032	0.521	0.026	0.232	0.012	0.544	0.022	0.343	0.003

2.2 两组患者治疗前后 $LSaO_2$、AHI 水平的比较

治疗前，两组患者的 $LSaO_2$、AHI 水平比较差异均无统计学意义(P>0.05)；治疗后，两组患者 $LSaO_2$ 水平明显高于治疗前(P<0.05)，AHI 水平低于治疗前 (P<0.05)，且实验组患者的 $LSaO_2$ 高于对照组(P<0.05)，AHI 水平低于对照组(P<0.05)。详见表 2 所示。

表 2 两组患者治疗前后 $LSaO_2$、AHI 水平的比较($\bar{x}\pm s$)

Table 2 Comparison of the $LSaO_2$ and AHI levels between the two groups before and after treatment($\bar{x}\pm s$)

Groups	$LSaO_2$(%)		AHI(次 /h)	
	Before treatment	After treatment	Before treatment	After treatment
Control group (n=49)	43.34± 5.67	55.34± 5.23	29.56± 9.34	25.45± 8.34
Experimental group (n=49)	43.67± 5.78	78.56± 4.23	29.34± 9.12	23.34± 7.67
t	1.054	11.966	2.343	8.655
P	0.532	0.001	0.521	0.032

2.3 两组治疗前后 BNP、SP、DP 的比较

治疗前两组患者 BNP、SP 和 DP 对比差异无显著差异(P>0.05)。治疗后，两组患者 BNP、SP 和 DP 明显低于治疗前(P<0.05)，且实验组患者的 BNP、SP 和 DP 低于对照组(P<0.05)。详见下表 3 所示。

表 3 两组治疗前后 BNP、SP、DP 比较($\bar{x}\pm s$)

Table 3 Comparison of BNP, SP and DP before and after treatment in both groups($\bar{x}\pm s$)

Groups	BNP(pg/mL)		SP(mmHg)		DP(mmHg)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Control group (n=49)	856.34± 321.45	297.56± 127.34	143.23± 12.23	130.45± 11.34	95.45± 5.34	87.45± 5.23
Experimental group (n=49)	857.67± 311.34	131.34± 112.78	141.67± 11.89	126.54± 10.23	95.67± 5.12	82.78± 5.14
t	2.232	8.655	1.896	8.454	1.906	9.322
P	0.123	0.034	0.342	0.014	0.233	0.004

3 讨论

心力衰竭是临床常见慢性疾病，而阻塞性睡眠呼吸暂停低通气综合征(OSAHS)是其常见并发症。心衰合并 OSAHS 患者由于上气道狭窄或者接近闭塞，呼吸道阻力增加，而减少气体进入，引起低氧及高碳酸血症，最终出现一系列病理生理变化[9,10]，主要表现在以下几方面[11,12]：上气道阻力增加，吸气费力，增加胸腔负压及回心血流量、前负荷。由于心脏需要更多的做功代偿低氧血症，最终会引起组织缺氧，导致心脏负担[13,14]。低氧及高碳酸血症使得心肌代谢紊乱，增加血红细胞及血粘滞度，增加循环阻力及心脏负荷。若 OSAHS 加重，将诱发心衰，从而加重病情[15,16]。因此，如何提高心力衰竭合并 OSAHS 的临床疗效是当前临床研究的热点课题。

ACEI 是一种抑制血管紧张素转化酶活性的化合物，通过降低血管紧张素Ⅱ和醛固酮减轻心脏前后负荷，降低外周血管与冠状血管阻力，并减少心肌纤维化，减缓心肌细胞凋亡等[17,18]。无创呼吸机具有无创、简单易行的优点，便于 OSAHS 患者使用。在整个吸气、呼气过程中施以压力，防止气道萎陷，增加功能残气[19,20]，并通过胸壁、迷走神经传入及反馈作用，增加上气道开放肌群，改善肺顺应性，确保气道开放，减轻呼吸道阻力，并减少气体进入导致的低氧、高碳酸血症[21,22]。EPO 是一种特异性糖蛋白，位于皮质的肾小管周围细胞分泌，多种液体、血样浓度都对其产生影响[23,24]。OSAHS 发展中，由于通气障碍而引发高碳酸血症及睡眠结构紊乱，若长期缺氧将促进 EPO 的合成及其释放[25]。通过 ACEI 及无创呼吸机可改善 EPO 水平及其血常规指标[26]。

本研究中，实验组患者的 EPO 及血常规水平改善程度均优于对照组。$LSaO_2$、AHI 为监测睡眠质量的重要指标，$LSaO_2$ 可反映患者睡眠期间的缺氧危险程度，AHI 指每小时睡眠内呼吸暂停与低通气的次数，可反映睡眠期患者呼吸情况[27,28]。本次研究中，实验组患者的 $LSaO_2$、AHI 改善程度均优于对照组，表明 ACEI 及无创呼吸机治疗可改善心衰合并 OSAHS 患者睡眠期间的呼吸障碍。BNP 是一种神经肽类激素，具有扩张血管、利尿及利钠作用，受到心脏血流动力学与神经激素的共同调节，由于左室扩张或者充盈亏的增加，增加左心室心肌细胞分泌脑钠素。通过定量检测 BNP，可及早诊断心力衰竭患者，且该水平与心衰程度密切相关。SP 与 DP 可了解人体真是血压情况[29,30]，本次数据显示实验组患者的 BNP、SP 与 DP 水平改善情况优于对照组，表明 ACEI 及无创呼吸机治疗可降低心衰合并 OSAHS 患者的心衰程度及高血压情况。

综上所述，ACEI 及无创呼吸机治疗心力衰竭合并 OSAHS 可改善患者的睡眠紊乱、睡眠呼吸障碍、心衰及高血压，具有临床推广应用的价值。

参 考 文 献(References)

[1] Redfield M M. Heart Failure with Preserved Ejection Fraction[J]. New England Journal of Medicine, 2016, 375(19): 1868-1877

[2] Riehle C, Abel E D. Insulin Signaling and Heart Failure[J]. Circulation Research, 2016, 118(7): 1151-1169

[3] Brown D, Perry J, Allen M, et al. Mitochondrial function as a thera-

peutic target in heart failure[J]. Nature Reviews Cardiology, 2017, 14 (4): 238-250

[4] You M, Li Z, Fang L, et al. Evaluation of carotid arterial elasticity in patients with obstructive sleep apnea hypopnea syndrome by two-dimensional speckle tracking imaging[J]. Medicine, 2017, 96(51): e8817

[5] Jakovljevic D G, Yacoub M H, Schueler S, et al. Left Ventricular Assist Device as a Bridge to Recovery for Patients With Advanced Heart Failure [J]. Journal of the American College of Cardiology, 2017, 69 (15): 1924-1933

[6] Maitikuerban B, Sun X, Li Y, et al. Deletion Polymorphism of Angiotensin Converting Enzyme Gene is Associated with Left Ventricular Hypertrophy in Uighur Hypertension-Obstructive Sleep Apnea Hypopnea Syndrome (OSAHS) Patients[J]. Med Sci Monit, 2019, 25: 3390-3396

[7] Basu R, Poglitsch M, Yogasundaram H, et al. Roles of Angiotensin Peptides and Recombinant Human ACE2 in Heart Failure[J]. Journal of the American College of Cardiology, 2017, 69(7): 805-819

[8] Tsuji K, Sakata Y, Nochioka K, et al. Characterization of heart failure patients with mid-range left ventricular ejection fraction-a report from the CHART-2 Study [J]. European Journal of Heart Failure, 2017, 19 (10): 1258-1269

[9] Zhang W, Zhao Z R, Dai C F, et al. Correlation between Calpain-10 single-nucleotide polymorphisms and obstructive sleep apnea/hypopnoea syndrome with ischemic stroke in a Chinese population: A population-based study[J]. Medicine, 2017, 96(16): e6570

[10] Teerlink J R, Voors A A, Ponikowski P, et al. Serelaxin in addition to standard therapy in acute heart failure: rationale and design of the RELAX-AHF-2 study[J]. European Journal of Heart Failure, 2017, 1(6): 800-809

[11] Konstam M A, François M. Abboud. Ejection Fraction: Misunderstood and Overrated (Changing the Paradigm in Categorizing Heart Failure)[J]. Circulation, 2017, 135(8): 717-719

[12] Demissei B G, Cotter G, Prescott M F, et al. A multimarker multi-time point-based risk stratification strategy in acute heart failure: results from the RELAX-AHF trial[J]. European Journal of Heart Failure, 2017, 19(8): 1001-1010

[13] Amp L W, Wilkins. Correction to: Role of Biomarkers for the Prevention, Assessment, and Management of Heart Failure: A Scientific Statement From the American Heart Association [J]. Circulation, 2017, 136(19): e345

[14] Tampaki E C, Tampakis A, Pantos C. Letter by Tampaki et al Regarding Article, "Lower Risk of Heart Failure and Death in Patients Initiated on Sodium-Glucose Cotransporter-2 Inhibitors Versus Other Glucose-Lowering Drugs: The CVD-REAL Study (Comparative Effectiveness of Cardiovascular Outcomes in New Users of Sodium-Glucose Cotransporter-2 Inhibitors)" [J]. Circulation, 2018, 137 (9): 982-983

[15] Hernández V A, Sayas C J, Benavides M P, et al. Obstructive sleep apnea-hypopnea syndrome in patients with severe chronic respiratory insufficiency[J]. Medicina Clinica, 2017, 148(10): 449-452

[16] Wang D, Gladysheva I P, Fan T H, et al. Atrial natriuretic peptide affects cardiac remodeling, function, heart failure, and survival in a mouse model of dilated cardiomyopathy [J]. Hypertension, 2017, 63 (3): 514-519

[17] Wisløff, Ulrik, Lavie C J , Rognmo, Øivind. Letter by Wisløff, et al Regarding Article, "High-Intensity Interval Training in Patients With Heart Failure With Reduced Ejection Fraction"[J]. Circulation, 2017, 136(6): 607-608

[18] Guazzi M, Naeije R. Pulmonary Hypertension in Heart Failure [J]. Journal of the American College of Cardiology, 2017, 69 (13): 1718-1734

[19] Komajda M, Isnard R, Cohen-Solal A, et al. Effect of ivabradine in patients with heart failure with preserved ejection fraction: the EDIFY randomized placebo-controlled trial [J]. European Journal of Heart Failure, 2017, 19(11): 1495-1503

[20] Obokata M, Kane G C, Reddy Y N V, et al. Role of Diastolic Stress Testing in the Evaluation for Heart Failure with Preserved Ejection Fraction: A Simultaneous Invasive-Echocardiographic Study [J]. Circulation, 2017, 136(4): 430-431

[21] O'Connor C M, Whellan D J, Fiuzat M, et al. Cardiovascular Outcomes With Minute Ventilation-Targeted Adaptive Servo-Ventilation Therapy in Heart Failure: The CAT-HF Trial[J]. Journal of the American College of Cardiology, 2017, 69(12): 1577-1587

[22] Imran T F, Shin H J, Mathenge N, et al. Meta-Analysis of the Usefulness of Plasma Galectin-3 to Predict the Risk of Mortality in Patients With Heart Failure and in the General Population [J]. American Journal of Cardiology, 2017, 119(1): 57-64

[23] Zamani P, Tan V, Soto-Calderon H, et al. Pharmacokinetics and Pharmacodynamics of Inorganic Nitrate in Heart Failure with Preserved Ejection FractionNovelty and Significance [J]. Circulation Research, 2017, 120(7): 1151-1161

[24] Kosmala W, Marwick T H. Reply: Effect of Aldosterone Antagonism on Exercise Tolerance in Heart Failure With Preserved Ejection Fraction[J]. Journal of the American College of Cardiology, 2017, 69(18): 2352-2353

[25] Wu J, Gu M, Chen S, et al. Factors related to pediatric obstructive sleep apnea-hypopnea syndrome in children with attention deficit hyperactivity disorder in different age groups [J]. Medicine, 2017, 96 (42): e8281

[26] Shen C X, Tan M, Song X L, et al. Evaluation of the predictive value of red blood cell distribution width for onset of cerebral infarction in the patients with obstructive sleep apnea hypopnea syndrome [J]. Medicine, 2017, 96(29): e7320

[27] Jin L, Li K, Li X. Distortion product otoacoustic emission together with tympanometry for assessing otitis media with effusion in children[J]. Acta oto-laryngologica, 2018, 138(8): 1-4

[28] An Z, Wang D, Yang G, et al. Role of microRNA-130a in the pathogeneses of obstructive sleep apnea hypopnea syndrome-associated pulmonary hypertension by targeting the GAX gene [J]. Medicine, 2017, 96(20): e6746

[29] Smith T A, Disler R T, Jenkins C R, et al. Perspectives on advance care planning among patients recently requiring non-invasive ventilation for acute respiratory failure: A qualitative study using thematic analysis[J]. Palliative Medicine, 2017, 31(6): 566-574

[30] Moraes I G, Kimoto K M, Fernandes M B, et al. Adjunctive Use of Noninvasive Ventilation During Exercise in Patients With Decom pensated Heart Failure[J]. American Journal of Cardiology, 2017, 119 (3): 423-427