

doi: 10.13241/j.cnki.pmb.2014.25.035

急诊慢性阻塞性肺疾病急性加重期患者预后相关因素分析

谈晓侠 姬润美 陈建玲 白文洁

(西安医学院第二附属医院急诊科 陕西 西安 710038)

摘要 目的:探讨急诊收治的慢性阻塞性肺疾病急性加重期患者的预后相关因素。方法:选取我院2008年12月-2013年12月急诊收治的慢性阻塞性肺疾病急性加重期患者94例,根据预后结果分为死亡组17例及存活组77例,回顾分析两组患者的相关资料。结果:存活组较死亡组BMI、血肌酐值、清蛋白、pH值、PaCO₂、FT₃差异具有统计学意义。死亡组较存活组在APACHE II, CCS, 有创通气率及合并肺心病率上差异均具有统计学意义。结果显示合并肺心病率、APACHE II评分、肌酐、清蛋白为影响慢性阻塞性肺疾病急性加重期患者预后的独立因素。结论:急诊收治的慢性阻塞性肺疾病急性加重期患者如合并肺心病率,APACHE II评分较高,血肌酐较高,清蛋白较低,这些因素提示我们患者预后较差,需及早进行相关治疗。

关键词:慢性阻塞性肺疾病;急性加重期;急诊;预后分析

中图分类号:R563 文献标识码:A 文章编号:1673-6273(2014)25-4940-03

Prognostic Factors Analysis of Acute Exacerbation Patients with Emergency Chronic Obstructive Pulmonary Disease

TAN Xiao-xia, JI Run-mei, CHEN Jian-ling, BAI Wen, WU Jie

(Department of Emergency, The Second Affiliated Hospital of Xi'an Medical University, Xi'an, Shaanxi, 710038, China)

ABSTRACT Objective: To investigate the prognostic factors of chronic obstructive pulmonary disease in acute exacerbation in emergency department. **Methods:** 94 patients with the acute exacerbation chronic obstructive pulmonary disease who were treated in our hospital from Dec. 2008 to Dec. 2013 were selected. According to the prognostic results, the objectives were divided into the death group(17 cases) and the survived group (77 cases), then the related information of two groups were retrospectively analyzed. **Results:** Compared with death group, the BMI, the serum creatinine values, the albumin, pH, PaCO₂ and FT₃ of patients in the survived group were significantly different ($P<0.05$). The complications of pulmonary heart disease rate, APACHE II score, creatinine and albumin were the independent factors of acute exacerbation chronic obstructive pulmonary disease. **Conclusion:** The patients with the acute exacerbation chronic obstructive pulmonary disease who were treated in the emergency department showed a high incidence of heart disease, APACHE II score and creatinine and low albumin. The risk factors prompt a poor prognosis for patients that should be treated early.

Key words: Chronic obstructive pulmonary disease; Acute exacerbation; Emergency; Prognostic analysis

Chinese Library Classification: R563 **Document code:** A

Article ID: 1673-6273(2014)25-4940-03

前言

慢性阻塞性肺疾病(Chronic Obstructive Pulmonary Disease,COPD)是呼吸系统最常见的疾病之一,其死亡率已居世界疾病死亡率第四位,未来趋势亦不容乐观^[1]。慢性阻塞性肺疾病急性加重期(Acute Exacerbation Of Chronic Obstructive Pulmonary Disease, AECOPD)患者短期内病情急剧恶化,医疗费用高,为患者及其家属带来巨大的经济压力^[2]。近年来,国内外已有多个研究报道AECOPD的预后影响因素分析,但因患者个体差异,样本量不一及统计方法不同等原因造成结果不一致^[3,4,6,13,14],且国内大多研究针对于住院治疗的AECOPD患者,关于急诊AECOPD患者的研究较少^[3,4],但AECOPD患者入院时常于急诊就诊,因此,研究急诊AECOPD患者的预后因素就

异常重要。本研究回顾分析近年来于我院急诊收治的患者相关资料,探讨分析其预后因素。

1 资料与方法

1.1 一般资料

选取我院2008年12月-2013年12月急诊收治的AECOPD患者94例,所有患者诊断均符合2007年中华医学会所制定的《慢性阻塞性肺疾病诊疗指南》中的诊断标准^[5]。排除合并严重的心力衰竭、肺栓塞、气胸、甲状腺疾病、严重的肝、肾疾病患者。其中男58例,女36例,年龄54~83岁,平均(76.2±8.9)岁。根据预后分为死亡组17例,其中男10例,女7例,年龄57~82岁,平均(76.9±7.6)岁,存活组77例,男48例,女29例,年龄59~82岁,平均(75.4±5.7)岁。

1.2 方法

回顾性分析所有患者的相关资料,其中包括①一般资料:包括性别比、年龄、体重指数(BMI)。②实验室检查:温度、心

作者简介:谈晓侠(1965-),女,专科,主治医师,研究方向:急救内科,电话:13186056735,E-mail:tanxiaoxia_6735@163.com
(收稿日期:2014-02-22 接受日期:2014-03-20)

率、血糖、白细胞计数(WBC)、血小板计数(PLT)、肌酐、尿素氮、清蛋白、PH 值、二氧化碳分压(PaCO₂)、氧分压(PO₂)、游离三碘甲状腺原氨酸(FT₃)、有创通气率,合并肺心病率。③相关量表:格拉斯哥昏迷评分(CCS)、急性生理与慢性健康状况评分Ⅱ(APACHEⅡ)。以上相关资料均采用入院 24 h 内抽取血液或 24 h 内最高值。

1.3 统计学方法

采用 SPSS 16.02 软件进行统计分析,计量资料采用($\bar{x} \pm s$)表示,组间比较采用 t 检验或 U 检验,计数资料组间比较采用

X²检验。将单因素分析结果作为自变量,是否死亡作为因变量,多因素分析采用 logistic 回归,模型筛选采用逐步回归法。P<0.05 表示差异具有统计学意义。

2 结果

2.1 一般资料及实验室检查单因素比较

两组在性别、年龄、体温、心率、血糖、WBC、PLT、尿素氮比较差异无统计学意义。存活组较死亡组 BMI、血肌酐值、清蛋白、pH 值、PaCO₂、FT₃ 差异具有统计学意义(P>0.05)。见表 1。

表 1 慢性阻塞性肺疾病急性加重期预后的单因素分析

Table 1 Single analysis of prognosis for patients with the chronic obstructive pulmonary disease in the acute exacerbation

Index	Death Group(n=17)	Survival Group(n=77)	t 或 x ²	P
Sexuality(male/female)	1.43	1.65	0.74	>0.05
Age(Years)	76.9± 7.6	75.4± 5.7	0.95	>0.05
BMI	20.5± 2.3	24.3± 4.8	5.86	<0.05
Temperature(°C)	37.1± 0.7	36.8± 0.6	0.41	>0.05
Heart Rate(次 /min)	71.4± 11.9	74.2± 12.4	1.12	>0.05
Blood Glucose(mmol/L)	9.6± 5.7	7.3± 3.1	1.21	>0.05
WBC(× 10 ⁹ /L)	9.1± 4.2	10.6± 3.9	1.46	>0.05
PLT(× 10 ⁹ /L)	106± 72	121± 87	1.20	>0.05
Creatinine(μmol/L)	101.4± 41.2	74.0± 8.5	11.27	<0.05
Urea Nitrogen(mmol/L)	7.1± 4.7	6.1± 2.5	1.36	>0.05
Albumin(mmol/L)	26.3± 1.9	32.5± 2.3	5.68	>0.05
pH Value	7.2± 0.2	7.3± 0.1	0.02	<0.05
PaCO ₂ (mmHg)	61± 26	72± 29	10.24	<0.05
FT ₃ (ng/L)	2.11± 0.35	1.72± 0.37	4.68	<0.05

2.2 两组量表评分,有创通气率及合并肺心病率比较

死亡组较存活组在 APACHEⅡ,CCS, 有创通气率及合并肺心病率上差异均具有统计学意义(P<0.05),见表 2。

2.3 多因素分析

根据单因素分析的结果,将 P<0.05 的因素进行非条件 logistic 回归分析,结果显示合并肺心病率、APACHEⅡ 评分、肌酐、清蛋白为影响慢性阻塞性肺疾病急性加重期患者预后的独立因素(P<0.05),见表 3。

表 2 两组量表评分,有创通气率及合并肺心病率比较

Table 2 Comparison of the scores, the rate of the invasive ventilation and pulmonary heart disease of patients in the two groups

	n	APACHEⅡ	CCS	Invasive ventilation rate(%)	Pulmonary heart disease rate(%)
Death Group	17	18	8	88.2	82.4
Survival Group	77	28	14	13.0	39.0
x ^{2/u}		-4.31	-4.14	13.27	9.49
P		<0.05	<0.05	<0.05	<0.05

表 3 慢性阻塞性肺疾病急性加重期患者的预后影响多因素分析

Table 3 Multivariate analysis of risk factors for the prognosis of patients with chronic obstructive pulmonary disease in acute exacerbation

Factors	Regression coefficients	Standard error	Wald 2 Value	P Value	OR Value
Pulmonary heart disease rate	-3.426	0.651	4.341	0.018	0.851
APACHEⅡ Score	-0.167	0.045	3.427	0.023	0.113
Urea Nitrogen	-2.526	0.347	5.145	0.008	0.865
Albumin	0.507	0.034	3.352	0.027	1.135

3 讨论

AECOPD 是导致 COPD 患者急诊的主要原因之一,据美国研究报告^[6],1993 年至 2005 年每年约有 60 万人因 AECOPD 于急诊科就诊,每千人 COPD 患者因 AECOPD 急诊就诊人数约为 3.2 人,仅 2000 年约有 150 万美国人因 AECOPD 于急诊科就诊。另一项对美国 388 例 COPD 患者进行的多中心研究显示 1 年内因 AECOPD 到急诊就诊的次数达 1090 次,其中 13% 的 COPD 患者一年内就诊达 6 次以上^[7]。COPD 的急性加重在病程中可反复出现,随着每一次 AECOPD 的发作,患者的肺功能不断降低,因此,有研究显示 AECOPD 的发作次数亦可作为 COPD 病情发展的一个重要监测指标^[8]。

当 AECOPD 患者被送入急诊室时,急诊医师最关心的即是通过相关资料判断患者的预后如何,以决定下一步的治疗方案,如急诊医师判断错误,易引起相关医疗事故的发生。英国 2010 年 COPD 指南显示年龄、既往 ICU 住院史、1 秒用力呼气容积(FEV1)、既往肺功能情况、BMI 是否伴随并发症等是判断预后的重要因素^[9]。但由于个体差异上述指南不一定适用于我国急诊 AECOPD 患者。

本研究显示存活组较死亡组血肌酐值、BMI、清蛋白、pH 值、PaCO₂、FT₃ 差异具有统计学意义($P > 0.05$)。这与国内外研究基本一致。清蛋白和 BMI 反应患者就诊时的营养状况,pH 值可反应患者是否具有酸碱平衡紊乱,PaCO₂ 可反应患者是否有呼吸衰竭。Wonderling 等^[10]研究显示 BMI、FEV1、PaCO₂ 为判断 AECOPD 患者预后的重要指标。汪学琴等^[11]研究显示低蛋白血症影响 AECOPD 患者生存期的长短。Terzano 等^[12]研究显示 FT₃ 与 AECOPD 患者的预后具有密切关系,其可反映患者疾病的严重程度。本研究显示年龄不能预测急诊 AECOPD 患者的预后,但 Burney 等^[13]的研究表明年龄大的 AECOPD 患者预后较差,这可能与 Burney 等应用年龄范围作为研究有关,亦可能和患者的人种、病情等个体因素差异有关,具体仍需进一步研究。

本研究还应用相关量表来作为预测急诊 AECOPD 患者预后的指标,结果显示 APACHE II 与 CCS 评分对 AECOPD 患者的预后具有较高的预测价值。有学者^[14-17] 研究显示急诊 AECOPD 患者 APACHE II 评分大于 21 分患者死亡率较高。而 CCS 是 APACHE II 的重要组成部分,其可一定程度上反应 COPD 患者中枢神经系统障碍情况。同时本研究显示一些与疾病严重程度明显相关的指标:有创通气率及合并肺心病率两项在死亡组及存活组差异具有显著性。这与国内研究^[11]基本一致。

国内大多数研究均对 AECOPD 住院患者的预后影响因素进行探讨,本研究发现合并肺心病率、APACHE II 评分、肌酐、清蛋白为影响急诊 AECOPD 患者预后的独立因素,这表明 APACHE II 评分高,血肌酐较高,清蛋白较低,合并肺心病患者预后较差,需及早进行相关治疗。这与国外研究一致^[18-20]。这对国内急诊医师对 AECOPD 患者的诊断及预后判断具有重要意义,为后续治疗提供一定帮助。

参考文献(References)

- [1] Menezes AM, Muiño A, López-Varela MV, et al. A Population-Based Cohort Study on Chronic Obstructive Pulmonary Disease in Latin America: Methods and Preliminary Results. The PLATINO Study Phase II[J]. Arch Bronconeumol, 2014, 50(1): 10-17
- [2] Braido F, Di Marco F, Santus P, et al. COPD classification methods and informativeness on mortality: contrasting evidences [J]. Minerva Med, 2013, 11(12): 113-119
- [3] 张伟.慢性阻塞性肺疾病的急性加重患者评分对患者预后分析[J].安徽医药, 2006, 10(12): 936-937
- Zhang Wei. Acute and chronic obstructive pulmonary disease exacerbation score on the prognosis of patients analyzed [J]. Anhui Medical and Pharmaceutical Journal, 2006, 10(12): 936-937
- [4] 吕青兰.慢性心力衰竭合并慢性阻塞性肺疾病老年患者预后影响因素[J].中国老年学杂志, 2013, 33(14): 3338-3339
- Lv Qiang-lan. The prognostic factors of combined with chronic heart failure in elderly patients with chronic obstructive pulmonary disease [J]. Chinese Journal of Gerontology, 2013, 33(14): 3338-3339
- [5] 中华中医药学会.慢性阻塞性肺疾病诊疗指南[J].中国中医药现代远程教育, 2011, 09(12): 115-116
- Chinese Medical Association. Chronic obstructive pulmonary disease treatment guidelines [J]. Chinese Medicine Modern Distance Education of China, 2011, 09(12): 115-116
- [6] Tsai CL, Sobrino JA, Camargo CA Jr. National study of emergency department visits for acute exacerbation of chronic obstructive pulmonary disease, 1993-2005 [J]. Acad Emerg Med, 2008, 15 (12): 1275-1283
- [7] Mannino DM, Homa DM, Akinbami LJ, et al. Chronic obstructive pulmonary disease surveillance--United States, 1971-2000 [J]. Respir Care, 2002, 47(10): 1184-1199
- [8] 周丽荣,吴坎金,王晓晟,等.慢性阻塞性肺疾病住院患者临床评价与预后分析[J].临床肺科杂志, 2009, 14(3): 382-383
- Zhou Li-rong, Wu Kan-jin, Wang Xiao-sheng, et al. Clinical evaluation of hospitalized patients with chronic obstructive pulmonary disease and prognosis[J]. Journal of Clinical Pulmonary Medicine, 2009, 14(3): 382-383
- [9] National Clinical Guideline Centre (UK). Chronic Obstructive Pulmonary Disease: Management of Chronic Obstructive Pulmonary Disease in Adults in Primary and Secondary Care [Internet]. London: Royal College of Physicians (UK), 2010
- [10] Wonderling D, Sawyer L, Fenu E, et al. National Clinical Guideline Centre cost-effectiveness assessment for the National Institute for Health and Clinical Excellence[J]. Ann Intern Med, 2011, 154(11): 758-765
- [11] 汪学琴,程青虹,崔玉静,等.低蛋白血症对 ICU 慢性阻塞性肺疾病机械通气患者预后的影响 [J].中华急诊医学杂志, 2010, 19(3): 303-304
- Wang Xue-qin, Cheng Qing-hong, Cui Yu-jing, et al. Hypoproteinemia impact on the prognosis of patients mechanically ventilated ICU patients with chronic obstructive pulmonary disease[J]. Chinese Journal of Emergency Medicine, 2010, 19(3): 303-304
- [12] Terzano C, Romaní S, Paone G, et al. COPD and Thyroid Dysfunctions[J]. Lung, 2014, 192(1): 103-109
- [13] Burney P, Jithoo A, Kato B, et al. Chronic obstructive pulmonary disease mortality and prevalence: the associations with smoking and poverty-a BOLD analysis[J]. Thorax, 2013, 18(6): 560-565

(下转第 5000 页)

- autophagy in physiology and pathophysiology[J]. *Physiological reviews*, 2010, 90(4):1383-1435
- [8] Shimizu S, Kanaseki T, Mizushima N, et al. Role of Bcl-2 family proteins in a non-apoptotic programmed cell death dependent on autophagy genes[J]. *Nature cell biology*, 2004, 6(12):1221-1228
- [9] Wei Y, Pattingre S, Sinha S, et al. JNK1-mediated phosphorylation of Bcl-2 regulates starvation-induced autophagy[J]. *Molecular cell*, 2008, 30(6):678-688
- [10] Zalckvar E, Berissi H, Mizrahi L, et al. DAP-kinase-mediated phosphorylation on the BH3 domain of beclin 1 promotes dissociation of beclin 1 from Bcl-XL and induction of autophagy[J]. *EMBO reports*, 2009, 10(3):285-292
- [11] Djavaheri-Mergny M, Maiuri MC, Kroemer G. Cross talk between apoptosis and autophagy by caspase-mediated cleavage of Beclin 1 [J]. *Oncogene*, 2010, 29(12):1717-1719
- [12] Luo S, Rubinsztein DC. Apoptosis blocks Beclin 1-dependent autophagosome synthesis: an effect rescued by Bcl-xL [J]. *Cell death and differentiation*, 2010, 17(2):268-277
- [13] Yousefi S, Perozzo R, Schmid I, et al. Calpain-mediated cleavage of Atg5 switches autophagy to apoptosis[J]. *Nature cell biology*, 2006, 8 (10):1124-1132
- [14] Fesu L, Piacentini M. Transglutaminase 2: an enigmatic enzyme with diverse functions. *Trends in biochemical sciences*, 2002, 27(10): 534-539
- [15] D'Eletto M, Farrace MG, Falasca L, et al. Transglutaminase 2 is involved in autophagosome maturation[J]. *Autophagy*, 2009, 5(8):1145-1154
- [16] Mathew R, Karp CM, Beaudoin B, et al. Autophagy suppresses tumorigenesis through elimination of p62 [J]. *Cell*, 2009, 137(6): 1062-1075
- [17] Gao Z, Gammoh N, Wong PM, et al. Processing of autophagic protein LC3 by the 20S proteasome[J]. *Autophagy*, 2010, 6(1):126-137
- [18] Narendra D, Tanaka A, Suen DF, et al. Parkin is recruited selectively to impaired mitochondria and promotes their autophagy[J]. *The Journal of cell biology* 2008, 183(5):795-803
- [19] Chen Y, McMillan-Ward E, Kong J, et al. Mitochondrial electron-transport-chain inhibitors of complexes I and II induce autophagic cell death mediated by reactive oxygen species [J]. *Journal of cell science*, 2007, 120(Pt 23):4155-4166
- [20] Kiffin R, Christian C, Knecht E, et al. Activation of chaperone-mediated autophagy during oxidative stress [J]. *Molecular biology of the cell*, 2004, 15(11):4829-4840
- [21] Massey AC, Kaushik S, Sovak G, et al. Consequences of the selective blockage of chaperone-mediated autophagy [J]. *Proceedings of the National Academy of Sciences of the United States of America*, 2006, 103(15):5805-5810
- [22] Pallet N, Bouvier N, Legendre C, et al. Autophagy protects renal tubular cells against cyclosporine toxicity[J]. *Autophagy*, 2008, 4(6): 783-791
- [23] Gozuacik D, Bialik S, Raveh T, et al. DAP-kinase is a mediator of endoplasmic reticulum stress-induced caspase activation and autophagic cell death[J]. *Cell death and differentiation* 2008, 15(12):1875-1886
- [24] Lindblad D, Blomenkamp K, Teckman J. Alpha-1-antitrypsin mutant Z protein content in individual hepatocytes correlates with cell death in a mouse model[J]. *Hepatology*, 2007, 46(4):1228-1235
- [25] Donohue TM, Jr. Autophagy and ethanol-induced liver injury [J]. *World journal of gastroenterology : WJG*, 2009, 15(10):1178-1185
- [26] Singh R, Kaushik S, Wang Y, et al. Autophagy regulates lipid metabolism[J]. *Nature*, 2009, 458(7242):1131-1135
- [27] He C, Bassik MC, Moresi V, et al. Exercise-induced BCL2-regulated autophagy is required for muscle glucose homeostasis [J]. *Nature*, 2012, 481(7382):511-515
- [28] Nakai A, Yamaguchi O, Takeda T, et al. The role of autophagy in cardiomyocytes in the basal state and in response to hemodynamic stress[J]. *Nature medicine*, 2007, 13(5):619-624
- [29] Taneike M, Yamaguchi O, Nakai A, et al. Inhibition of autophagy in the heart induces age-related cardiomyopathy[J]. *Autophagy*, 2010, 6 (5):600-606
- [30] Matsui Y, Kyo S, Takagi H, et al. Molecular mechanisms and physiological significance of autophagy during myocardial ischemia and reperfusion[J]. *Autophagy*, 2008, 4(4):409-415
- [31] Zhao Y, Xue T, Yang X, et al. Autophagy plays an important role in sunitinib-mediated cell death in H9c2 cardiac muscle cells[J]. *Toxicology and applied pharmacology*, 2010, 248(1):20-27

(上接第 4942 页)

- [14] Ställberg B, Janson C, Management. Morbidity and mortality of COPD during an 11-year period: an observational retrospective epidemiological register study in Sweden (PATHOS) [J]. *Prim Care Respir J*, 2013, 18(11): 762-775
- [15] Abedi H, Salimi SJ, Feizi A, et al. Effect of self-efficacy enhancement program on self-care behaviors in chronic obstructive pulmonary disease[J]. *Iran J Nurs Midwifery Res*, 2013, 18(5): 421-424
- [16] Mohan A, Sethi S. The reliability and validity of patient-reported chronic obstructive pulmonary disease exacerbations [J]. *Curr Opin Pulm Med*, 2014, 7(1): 119-125
- [17] Nath KD, Burel JG, Shankar V, et al. Clinical factors associated with the humoral immune response to influenza vaccination in chronic obstructive pulmonary disease [J]. *Int J Chron Obstruct Pulmon Dis*, 2014, 9: 51-56
- [18] Pedone C, Scarlata S, Forastiere F, et al. BODE index or geriatric multidimensional assessment for the prediction of very-long-term mortality in elderly patients with chronic obstructive pulmonary disease? A prospective cohort study [J]. *Age Ageing*, 2013, 12 (11): 253-259
- [19] Panigrahi A1, Sohani S2, Amadi C2, et al. Role of music in the management of chronic obstructive pulmonary disease (COPD): A literature review[J]. *Technol Health Care*, 2014, 7(1): 81-94
- [20] Bartlett YK, Sheeran P, Hawley MS. Effective behaviour change techniques in smoking cessation interventions for people with chronic obstructive pulmonary disease: A meta-analysis [J]. *Br J Health Psychol*, 2014, 19(1): 181-203