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2019冠状病毒病患者临床特征对重症化的预测作用

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[摘 要] 目的：2019冠状病毒病(coronavirus disease 2019，COVID-19)疫情暴发以来，已在中国和其他许多国家迅速传播，病例数量的急剧增加引起了人们的普遍恐慌，已成为目前全球主要的公共卫生问题。重症患者多在发病1周后出现呼吸困难和/或低氧血症，少数危重症患者不仅可能迅速发展为急性呼吸窘迫综合征，还可能引起凝血功能障碍，以及心、肝、肾等多器官功能衰竭甚至死亡。本研究通过分析COVID-19患者临床特征对重症化的预测作用，旨在为临床医师监测患者临床特征相关变化，阻止疾病进展，提高该病的医疗救治水平提供参考依据。**方法：**收集2020年1月17日至3月14日在长沙市公共卫生救治中心集中收治的208例入院时为轻型和普通型的成人COVID-19患者的临床资料，其中男105例，女103例，年龄19~84(中位年龄44)岁。按照国家卫生健康委员会办公厅和国家中医药管理局办公室印发的《新型冠状病毒肺炎诊疗方案(试行第七版)》作为诊断和分型标准。根据住院期间是否进展至重症，将患者分为轻症组(*n*=183)和重症转化组(*n*=25)，对患者的年龄、基础疾病、入院时血常规、凝血功能、血生化、氧合指数等临床特征进行比较和分析，其中实验室检查包括：白细胞(white blood cell，WBC)、淋巴细胞(lymphocytes，LYM)、中性粒细胞(neutrophil，NEU)、血红蛋白(hemoglobin，Hb)、血小板(platelet，PLT)、凝血酶原时间(prothrombin time，PT)、血浆纤维蛋白原(plasma fibrinogen，Fib)、活化部分凝血酶原时间(activated partial prothrombin time，APTT)、凝血酶时间(thrombin time，TT)、*D*-二聚体(*D*-dimer)、总胆红素(total bilirubin，TBIL)、白蛋白(albumin，ALB)、谷丙转氨酶(alanine aminotransferase，ALT)、谷草转氨酶(aspartate aminotransferase，AST)、血尿素氮(blood urea nitrogen，BUN)、血肌酐(serum creatinine，Cr)、肌酸激酶(creatine kinase，CK)、肌酸激酶同工酶-MB(creatine kinase isoenzyme-MB，CK-MB)、乳酸脱氢酶(lactate dehydrogenase，LDH)、C反应蛋白(C-reactive protein，CRP)及动脉血氧分压。计算氧合指数(partial pressure of oxygen in arterial blood/fractional concentration of inspiratory oxygen，PaO_2/FiO_2)。对有统计学意义的变量进行logistic回归分析。**结果：**重症转化组较轻症组患者合并的基础疾病更多(*P*<0.05)；从疾病分布来看，重症转化组合并高血压病更多(*P*<0.05)。重症转化组较轻症组PT延长，Fib，ALT，AST，CK，LDH及CRP水平显著升高(*P*<0.05或*P*<0.001)，LYM，ALB及PaO_2/FiO_2显著降低(*P*<0.05或*P*<0.001)。以差异有统计学意义的临床特征，即合并高血压病，LYM，PT，Fib，ALB，ALT，AST，CK，LDH及CRP为自变量，以是否重症化为因变量，进行logistic回归分析，结果显示合并高血压病、LYM降低、PT延长、CK升高是影响COVID-19患者发生重症化的独立危险因素(*P*<0.05)。**结论：**轻症COVID-19患者的重症化可能与合并高血压病、LYM降低、PT延长、CK升高有关，针对有这些临床特征的轻症患者及早进行干预，可能有效阻止疾病进展至重症化，提高疾病的治愈率。

[关键词] 2019冠状病毒病；肺炎；临床特征；重症化；预测

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Predictive role of clinical features in patients with coronavirus disease 2019 for severe disease

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ABSTRACT

Objective: Since the outbreak of coronavirus disease 2019 (COVID-19), it has spread rapidly in China and many other countries. The rapid increase in the number of cases has caused widespread panic among people and has become the main public health problem in the world. Severe patients often have difficult breathing and/or hypoxemia after 1 week of onset. A few critically ill patients may not only rapidly develop into acute respiratory distress syndrome, but also may cause coagulopathy, as well as multiple organs failure (such as heart, liver and kidney) or even death. This article is to analyze the predictive role of clinical features in patients with COVID-19 for severe disease, so as to help doctor monitor the severity-related features, restrain the disease progress, and provide a reference for improvement of medical treatment.

Methods: The clinical data of 208 patients with COVID-19 who were isolated and treated in Changsha Public Health Treatment Center from January 17, 2020 to March 14, 2020 were collected. All patients were the mild and ordinary adult patients on admission, including 105 males and 103 females from 19 to 84 (median age 44) years old. According to the “Program for the diagnosis and treatment of novel coronavirus (COVID-19) infected pneumonia (Trial version 7)” issued by the General Office of National Health Committee and Office of State Administration of Traditional Chinese Medicine as the diagnostic and typing criteria. According to progression from mild to severe disease during hospitalization, the patients were divided into a mild group (n=183) and a severe transformation group (n=25). The clinical features such as age, underlying disease, blood routine, coagulation function, blood biochemistry, oxygenation index, and so on were analyzed. Among them, laboratory tests included white blood cell (WBC), lymphocytes (LYM), neutrophil (NEU), hemoglobin (Hb), platelet (PLT), prothrombin time (PT), plasma fibrinogen (Fib), activated partial prothrombin time (APTT), thrombin time (TT), *D*-dimer, total bilirubin (TBIL), albumin (ALB), alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood urea nitrogen (BUN), serum creatinine (Cr), creatine kinase (CK), creatine kinase isoenzyme-MB (CK-MB), lactate dehydrogenase (LDH), C-reactive protein (CRP), and oxygen partial pressure in arterial blood. Partial pressure of oxygen in arterial blood/fractional concentration of inspiratory oxygen (PaO_2/FiO_2) was calculated. The variables with statistical significance were analyzed by logistic regression analysis.

Results: Patients in the severe transformation group had more combined underlying diseases than those in the mild group (P<0.05). From the perspective of disease distribution, patients in the severe transformation group had more combined hypertension

(*P*<0.05). In the severe transformation group, PT was significantly longer, the levels of Fib, ALT, AST, CK, LDH, and CRP were significantly higher than those in the mild group (*P*< 0.05 or *P*<0.001), while LYM, ALB, and PaO_2/FiO_2 were significantly lower than those in the mild group (*P*<0.05 or *P*<0.001). Logistic regression analysis was performed on clinical features with statistically significant differences. Combined with hypertension, LYM, PT, Fib, ALB, ALT, AST, CK, LDH, and CRP as independent variables, and having severe disease or not was the dependent variable. The results show that combined hypertension, decreased LYM, longer PT, and increased CK level were independent risk factors that affected the severity of COVID-19 (*P*<0.05).

Conclusion: The patients with mild COVID-19 who are apt to develop severe diseases may be related to combined hypertension, decreased LYM, and longer PT, and increased CK level. For the mild patients with these clinical features, early intervention may effectively prevent the progression to severe diseases.

KEY WORDS coronavirus disease 2019; pneumonia; clinical feature; severity; prediction

2019 冠状病毒病(coronavirus disease 2019，COVID-19)疫情暴发以来，已在中国和其他许多国家迅速传播，病例数量的急剧增加引起了人们的普遍恐慌，已成为目前全球主要的公共卫生问题[1-2]。国家卫生健康委员会明确COVID-19为乙类传染病，按甲类传染病管理[3]。湖南省卫生健康委员会明确长沙市公共卫生救治中心(长沙市第一医院北院)为省级、市级定点收治医院。COVID-19以飞沫和接触为主要传播途径[4]。COVID-19有多种临床表现，大多数患者感染后症状以发热、干咳、乏力为主要表现，重症患者多在发病1周后出现呼吸困难和/或低氧血症，少数危重症患者不仅可能迅速发展为急性呼吸窘迫综合征(acute respiratory distress syndrome，ARDS)，还可能引起凝血功能障碍，以及心、肝、肾、胃肠道等多器官功能衰竭甚至死亡[4-6]。本研究通过探讨COVID-19患者的临床特征对重症化的预测作用，旨在为临床医师监测患者临床特征相关变化，及时通过早期干预有效阻止疾病进展至重症，提高疾病治愈率，为COVID-19的医疗救治水平的提升提供一定的参考依据。

1 资料与方法

1.1 一般资料

收集2020年1月17日至3月14日在长沙市公共卫生救治中心集中收治的208例COVID-19确诊患者的临床资料，其中男105例，女103例，年龄19~84(中位年龄44)岁。本研究通过长沙市第一医院医学伦理委员会审批。

1.2 分型标准

按照国家卫生健康委员会办公厅和国家中医药管理局办公室印发的《新型冠状病毒肺炎诊疗方案(试行第七版)》[3]作为诊断和分型标准。轻型：临床症状轻微，影像学未见肺炎表现。普通型：具有发热、呼吸道等症状，影像学可见肺炎表现。重型(成人符合下列任何一条)：出现气促，呼吸频率≥30 min^{-1}；在静息状态下，指氧饱和度≤93%；氧合指数(partial pressure of oxygen in arterial blood/fractional concentration of inspiratory oxygen，PaO_2/FiO_2)≤300 mmHg(1 mmHg=0.133 kPa)。肺部影像学显示24~48 h内病灶明显进展>50%者。入院时均为轻型和普通型的成人患者，根据住院期间是否进展至重症，将患者分为轻症组(183例)和重症转化组(25例)。

1.3 资料收集

收集包括患者的一般情况、入院时实验室检查等资料。其中实验室检查包括：白细胞(white blood cell，WBC)、淋巴细胞(lymphocytes，LYM)、中性粒细胞(neutrophil，NEU)、血红蛋白(hemoglobin，Hb)、血小板(platelet，PLT)、凝血酶原时间(prothrombin time，PT)、血浆纤维蛋白原(plasma fibrinogen，Fib)、活化部分凝血酶原时间(activated partial prothrombin time，APTT)、凝血酶时间(thrombin time，TT)、*D*-二聚体(*D*-dimer)、总胆红素(total bilirubin，TBIL)、白蛋白(albumin，ALB)、谷丙转氨酶(alanine aminotransferase，ALT)、谷草转氨酶(aspartate aminotransferase，AST)、血尿素氮(blood urea nitrogen，BUN)、血肌酐(serum creatinine，Cr)、肌酸激酶(creatine kinase，CK)、肌

酸激酶同工酶-MB(creatine kinase isoenzyme-MB，CK-MB)、乳酸脱氢酶(lactate dehydrogenase，LDH)、C反应蛋白(C-reactive protein，CRP)、动脉血氧分压。计算PaO_2/FiO_2。

1.4 统计学处理

采用SPSS 17.0软件进行统计分析，计数资料采用例数和率表示，样本比较用χ^2检验。正态分布的计量资料采用均数±标准差($\bar{x}\pm s$)表示，比较用t检验；非正态资料采用中位数(四分位间距)表示，比较用非参数统计中的Mann-Whitney U检验。对差异有统计学意义的指标用多因素logistic回归分析，以P<0.05为差异有统计学意义。

2 结　果

2.1 一般情况

在入院时为轻型和普通型的患者中，重症转化组较轻症组合并基础疾病[高血压病、糖尿病、冠心病、脑血管疾病、慢性阻塞性肺疾病(chronic obstructive pulmonary disease，COPD)、慢性肝病、慢性肾病、自身免疫性疾病、恶性肿瘤]者显著增多(P=0.015)；其中重症转化组患者合并高血压病较轻症组更多(P=0.001)。两组性别，年龄以及合并糖尿病、冠心病、脑血管疾病、慢性肝病、慢性肾病、自身免疫性疾病、恶性肿瘤、COPD比较，差异无统计学意义(P>0.05，表1)。

表1　两组间一般情况比较

Table 1　Comparison of general conditions between the 2 groups

组别	n	男/[例(%)]	女/[例(%)]	年龄*/岁	有基础疾病/[例(%)]	高血压病/[例(%)]	糖尿病/[例(%)]
轻症组	183	88(48.09)	95(51.91)	42.00(21.00)	77(42.08)	22(12.02)	12(6.56)
重症转化组	25	17(68.00)	8(32.00)	53.00(27.50)	17(68.00)	10(40.00)	2(8.00)
χ^2/z		3.489		−1.609	5.968	11.164	0.070
P		0.062		0.108	0.015	0.001	0.792

组别	冠心病/[例(%)]	脑血管疾病/[例(%)]	COPD/[例(%)]	慢性肝病/[例(%)]	慢性肾病/[例(%)]	自身免疫性疾病/[例(%)]	恶性肿瘤/[例(%)]
轻症组	4(2.19)	5(2.73)	0(0.00)	7(3.83)	0(0.00)	3(1.64)	2(1.09)
重症转化组	1(4.00)	1(4.00)	1(4.00)	2(8.00)	1(4.00)	1(4.00)	0(0.00)
χ^2/z	0.265	0.115	1.371	0.192	1.371	0.520	0.515
P	0.607	0.735	0.242	0.661	0.242	0.471	0.473

*中位数(四分位间距)

2.2 实验室指标

在入院时为轻型和普通型的患者中，重症转化组较轻症组PT延长，Fib，ALT，AST，CK，LDH及CRP水平显著升高，LYM，ALB及PaO_2/FiO_2显著降低，差异有统计学意义(P<0.05或P<0.001)，两组WBC，NEU，Hb，PLT，APTT，TT，D-dimer，TBIL，BUN，Cr及CK-MB比较，差异无统计学意义(均P>0.05，表2)。

2.3 重症化logistic回归分析

以差异有统计学意义的临床特征，即合并高血压病，LYM，PT，Fib，ALB，ALT，AST，CK，LDH及CRP为自变量，以是否重症化为因变量，进行logistic回归分析。结果显示：合并高血压病、LYM降低、PT延长、CK升高是影响COVID-19患者发生重症化的独立危险因素(P<0.05或P<0.001，表3)。

表2　两组间实验室指标比较

Table 2　Comparison of laboratory indicators between the 2 groups

组别	n	血常规				
		WBC*/($\times10^9\cdot L^{-1}$)	LYM*/($\times10^9\cdot L^{-1}$)	NEU*/($\times10^9\cdot L^{-1}$)	Hb/($g\cdot L^{-1}$)	PLT*/($\times10^9\cdot L^{-1}$)
轻症组	183	4.49(2.04)	1.20(0.71)	2.80(1.40)	130.16±16.52	170.00(88.00)
重症转化组	25	4.51(2.01)	0.76(0.44)	2.93(1.64)	132.44±19.51	158.00(57.00)
t/z		−1.568	−4.292	−0.331	−0.633	−1.394
P		0.117	<0.001	0.740	0.527	0.163

表2(续)

组别	凝血功能				
	PT*/s	Fib*/(g·L^{-1})	APTT*/s	TT*/s	*D*-dimer*/(μg·mL^{-1})
轻症组	11.70(1.10)	3.49(1.14)	32.40(4.50)	13.90(1.70)	0.25(0.39)
重症转化组	12.70(1.10)	4.19(1.43)	32.70(4.80)	14.00(1.35)	0.28(0.37)
t/*z*	−4.193	−4.348	−0.997	−0.447	−0.243
P	<0.001	<0.001	0.319	0.655	0.808

组别	肝功能				肾功能	
	TBIL*/(μmol·L^{-1})	ALB/(g·L^{-1})	ALT*/(U·L^{-1})	AST*/(U·L^{-1})	BUN*/(mmol·L^{-1})	Cr*/(μmol·L^{-1})
轻症组	10.92(7.04)	38.72±4.07	19.10(12.46)	23.27(9.38)	4.18(1.73)	51.11(24.20)
重症转化组	9.97(9.17)	36.24±3.91	28.41(15.46)	29.05(8.88)	4.29(1.97)	53.43(20.12)
t/*z*	−0.955	2.868	−2.551	−4.072	−0.267	−0.143
P	0.340	0.005	0.011	<0.001	0.789	0.886

组别	生化指标			炎症指标	氧合指数
	CK*/(U·L^{-1})	CK-MB*/(U·L^{-1})	LDH*/(U·L^{-1})	CRP*/(mg·L^{-1})	PaO_2/FiO_2*
轻症组	68.20(64.50)	9.57(6.20)	155.10(64.50)	12.10(20.74)	438.57(175.24)
重症转化组	90.30(341.00)	11.20(8.15)	195.40(48.75)	27.74(24.02)	397.14(162.30)
t/*z*	−2.947	−1.247	−3.288	−3.870	−1.989
P	0.003	0.212	0.001	<0.001	0.047

*中位数(四分位间距)

表3 重症化logistic回归分析

Table 3 Logistic regression analysis of severe illness

自变量	*B*	*SE*	Wald	*P*	Exp(*B*)	95% CI
合并高血压病	1.426	0.562	6.447	0.011	4.163	1.385~12.519
LYM	−2.353	0.790	8.867	0.003	0.095	0.020~0.447
PT	0.480	0.213	5.091	0.024	1.616	1.065~2.452
CK	0.007	0.002	8.289	0.004	1.007	1.002~1.011

3 讨 论

本研究结果显示：在入院时为轻型和普通型的患者中，重症转化组比轻症组患者合并的基础疾病更多；从疾病分布来看，重症转化组合并高血压病更多。提示合并高血压病患者更易重症化。研究[7]发现：与非ICU的患者相比，ICU的重症患者合并更多基础疾病，其中合并高血压病、糖尿病、心血管疾病、脑血管疾病者更多。本研究中发现重症转化组合并高血压较轻症组更多，而糖尿病、冠心病、脑血管疾病差异无统计学意义(*P*>0.05)，可能与样本量较小有关。本研究还发现：重症转化组较轻症组PT延长，Fib，ALT，AST，CK，LDH及CRP水平显著升高，LYM，ALB及PaO_2/FiO_2显著降低。提示这些有可能作为患者重症化的预测指标。

Logistic回归分析结果显示：合并高血压病、LYM降低、PT延长、CK升高是影响COVID-19患者发生重症化的独立危险因素。多项研究[8-10]表明：严重的严重急性呼吸综合征冠状病毒2(severe acute respiratory syndrome coronavirus 2，SARS-CoV-2)可引起肺部和全身炎症反应，COVID-19重症患者常有ARDS，凝血异常，急性心脏损伤等多脏器系统受累表现。病毒可能侵入黏膜，尤其是鼻黏膜和喉黏膜，然后通过呼吸道进入肺部，可能从肺部进入外周血，引起毒血症，然后，病毒会攻击表达血管紧张素转换酶2(angiotensin converting enzyme，ACE2)的肺、心、肾、肝、胃肠道等靶器官，从而引发一系列免疫反应和体内细胞因子风暴[11-12]。COVID-19患者疾病进展为重症可能与细胞因子风暴相关，重症患者的IL-6，IL-10和TNF-α等细胞因子浓度较轻症者更高，提示细胞因子风暴与疾病严重程度相关[6,11,13]。如前所述，COVID-19通过结合ACE2进入细胞，研究[14-15]表明一些抗高血压药物(如ACE抑制剂和血管紧张素受体拮抗剂)的使用可能会增强细胞表面ACE2的表达，从而为SARS-CoV-2提供大量的“锚”用于感染细胞，但这仍然是一个有争议的问题。另一方面，也有研究[16]认为高血压患者的ACE2表达降低，当与SARS-CoV-2结合时，会降低残留的ACE2表达，导致血管紧张素II升高，从而推动ARDS的进程。淋巴细胞减少症是COVID-19重症患者的一个显著特征，超过80%的危重病患者出现淋巴细胞减少症[8]。SARS-CoV-2可能主要作用于淋巴细胞，尤其是T淋巴细胞，LYM总数的大量减少表明SARS-CoV-2消耗了许多免疫细胞，抑制了人体的细胞免疫功能[9]。研究[17]显示COVID-19患者凝血功能较正常者明显紊乱。PT是凝血功能的指标之一，58%的COVID-19患者PT延长[7]，ICU的重症患者比普通病

房患者PT延长[6]。CK主要存在于心肌和骨骼肌组织中，CK活性可用于评价心肌损伤程度，较高的CK活性与COVID-19病例严重程度和复合终点(患者被收治于ICU，机械通气或死亡)密切相关[18]。但CK并不是心肌损伤的特异性指标，COVID-19对心脏的影响仍需要进一步研究。目前COVID-19的治疗主要集中在对症和呼吸支持上。一些患者迅速进展至ARDS和脓毒症休克，最终多器官功能衰竭，因此，早期对重症化的危险因素进行识别并及时处理至关重要[9]。

综上所述，轻症COVID-19患者的重症化可能与合并高血压病、LYM降低、PT延长、CK升高有关，针对有这些临床特征的轻症患者及早进行干预，可能有效阻止疾病进展至重症化，从而提高治愈率。但因本研究涉及的地域范围不够广，样本量较少，结论存在一定局限性，需要扩充样本量，多中心、前瞻性的研究来进一步论证。同时COVID-19患者重症化的作用机制尚需要深入分析。

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