

急性冠脉综合征并发心源性休克患者血清PTEN、Cyr61水平表达与预后的相关性研究

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摘要: **目的** 分析急性冠脉综合征(ACS)并发心源性休克(CS)患者血清磷酸酶及张力蛋白同源物(PTEN)、半胱氨酸富含蛋白61(Cyr61)水平与疾病危险程度及预后的相关性。**方法** 选取2023年1月~2025年5月上海交通大学医学院附属第九人民医院收治的ACS并发CS患者203例(ACS并发CS患者组),另选取同期未并发CS的ACS患者203例(单纯ACS组),采用酶联免疫吸附法检测血清PTEN、Cyr61水平。根据危险程度将ACS并发CS患者分为低危ACS并发CS组(77例)、中危ACS并发CS组(72例)、高危ACS并发CS组(54例),Spearman相关分析ACS并发CS患者血清PTEN、Cyr61水平与危险程度的相关性。根据30天预后将ACS并发CS患者分为死亡组(77例)和存活组(126例),分析ACS并发CS患者预后的因素及血清PTEN、Cyr61水平的预测效能。**结果** ACS并发CS组血清PTEN(4.98 ± 1.60 ng/ml)、Cyr61(396.46 ± 126.65 pg/ml)水平高于单纯ACS组(3.09 ± 0.87 ng/ml、 254.50 ± 83.89 pg/ml),差异有统计学意义($t=14.790$ 、 13.315 , 均 $P<0.001$)。血清PTEN、Cyr61水平分别在低危ACS并发CS组(3.58 ± 1.09 ng/ml、 284.00 ± 86.02 pg/ml)、中危ACS并发CS组(5.71 ± 1.31 ng/ml、 411.31 ± 63.84 pg/ml)、高危ACS并发CS组(5.99 ± 1.08 ng/ml、 537.03 ± 79.09 pg/ml)依次升高($t=4.306\sim 17.125$, 均 $P<0.001$)。ACS并发CS患者血清PTEN、Cyr61水平与危险程度呈正相关($r_s=0.682$ 、 0.661 , 均 $P<0.001$)。203例ACS并发CS患者30天死亡率为37.93%(77/203)。主动脉内球囊反搏-休克Ⅱ积分升高(OR=1.469, 95%CI: 1.174~1.839, $P<0.001$)、乳酸升高(OR=1.171, 95%CI: 1.003~1.367, $P=0.046$)、氨基末端脑钠肽前体升高(OR=1.012, 95%CI: 1.002~1.023, $P=0.019$)、PTEN升高(OR=2.604, 95%CI: 1.766~3.839, $P<0.001$)、Cyr61升高(OR=1.012, 95%CI: 1.007~1.018, $P<0.001$)是ACS并发CS患者死亡的独立危险因素。血清PTEN、Cyr61水平及二者联合预测ACS并发CS患者死亡的受试者操作特征(ROC)曲线下面积分别为0.811、0.803、0.910,敏感度分别为83.12%、81.82%、81.82%,特异度分别为65.08%、63.49%、83.33%,二者联合预测效能优于血清PTEN、Cyr61水平单独预测($Z=3.928$ 、 4.329 , 均 $P<0.001$)。**结论** ACS并发CS患者血清PTEN、Cyr61水平升高,与危险程度加重及死亡风险增加相关,二者联合对ACS并发CS患者预后的预测效能较高。

关键词: 急性冠脉综合征;心源性休克;磷酸酶及张力蛋白同源物;半胱氨酸富含蛋白61

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A Study on the Correlation between Serum PTEN and Cyr61 Levels and Prognosis in Patients with Acute Coronary Syndrome Complicated by Cardiogenic Shock

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Abstract: **Objective** To analyze the correlation between serum phosphatase and tensin homolog (PTEN), cysteine-rich protein 61 (Cyr61) levels and disease severity and prognosis in patients with acute coronary syndrome (ACS) complicated by cardiogenic shock (CS). **Methods** From January 2023 to May 2025, 203 ACS patients with CS (ACS with CS group) and 203 ACS patients without CS (ACS alone group) were enrolled from the Ninth People's Hospital Affiliated to Shanghai Jiaotong University School of Medicine. Serum PTEN and Cyr61 levels were detected by enzyme-linked immunosorbent assay. Patients with ACS combined with CS were further divided into low-risk (77 cases), medium-risk (72 cases) and high-risk (54 cases) groups according to disease severity. Spearman correlation analysis was used to analyze the correlation between serum PTEN and Cyr61 levels and disease severity in patients with ACS combined with CS. Based on 30-day prognosis, ACS patients with CS were divided into non-survivors group (77 cases) and survivors group (126 cases). Prognostic factors of ACS patients with CS and the predictive efficacy of serum PTEN and Cyr61 levels were analyzed. **Results** Serum PTEN (4.98 ± 1.60 ng/ml) and Cyr61 (396.46 ± 126.65 pg/ml) levels in ACS combined with CS group were higher than those in ACS group (3.09 ± 0.87 ng/ml, 254.50 ± 83.89 pg/ml), the differences were statistically significant ($t=14.790$, 13.315 , both $P<0.001$). Serum PTEN and Cyr61

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levels increased progressively across the low-risk (3.58 ± 1.09 ng/ml, 284.00 ± 86.02 pg/ml), medium-risk (5.71 ± 1.31 ng/ml, 411.31 ± 63.84 pg/ml) and high-risk (5.99 ± 1.08 ng/ml, 537.03 ± 79.09 pg/ml) ACS with CS groups ($t=4.306\sim 17.125$, all $P < 0.001$). Serum PTEN and Cyr61 levels were positively correlated with disease severity ($r_s=0.682, 0.661$, both $P < 0.001$). The 30-day mortality rate of 203 ACS patients with CS was 37.93% (77/203). Elevated intra-aortic balloon pump in cardiogenic-shock II score (OR=1.469, 95%CI: 1.174~1.839, $P < 0.001$), lactic acid (OR=1.171, 95%CI: 1.003~1.367, $P=0.046$), N-terminal pro-brain natriuretic peptide (OR=1.012, 95%CI: 1.002~1.023, $P=0.019$), PTEN (OR = 2.604, 95%CI: 1.766~3.839, $P < 0.001$), and Cyr61 (OR=1.012, 95%CI: 1.007~1.018, $P < 0.001$) were independent risk factors for death in ACS patients with CS. The area under the receiver operating characteristic (ROC) curve of serum PTEN, Cyr61 levels and their combination for predicting death in ACS patients with CS were 0.811, 0.803 and 0.910, respectively, with sensitivity of 83.12%, 81.82% and 81.82%, respectively, and specificity of 65.08%, 63.49% and 83.33%. The combined prediction efficiency was better than that of serum PTEN and Cyr61 levels alone ($Z=3.928, 4.329$, both $P < 0.001$). **Conclusions** Elevated serum PTEN and Cyr61 in patients with ACS complicated with CS are related to increased disease severity and higher death risk. The combination of the two has a higher predictive value for prognosis of patients with ACS complicated with CS.

Keywords: acute coronary syndrome; cardiogenic shock; phosphatase and tensin homolog; cysteine-rich protein 61

急性冠状动脉综合征(acute coronary syndrome, ACS)是冠状动脉粥样硬化引起冠脉急性狭窄或闭塞的一类临床综合征,近年来ACS发病率逐年上升,其相关死亡率居高不下^[1-2]。心源性休克(cardiogenic shock, CS)是一种以心输出量明显下降、组织器官灌注不足及全身微循环功能障碍为特征的严重临床综合征,其中绝大多数由ACS引起^[3]。ACS并发CS患者病死率高达50%,尽管近年来相关治疗措施取得显著进步,但预后仍不理想^[4-5]。因此早期识别ACS并发CS患者预后并开展干预至关重要。第10染色体同源缺失性磷酸酶及张力蛋白同源物(phosphatase and tensin homolog, PTEN)是一种抑癌基因,能通过调控多条信号通路抑制心肌细胞增殖和促进细胞凋亡^[6]。LING等^[7]报道,血清外泌体PTEN水平升高有助于ACS诊断。半胱氨酸富含蛋白61(cysteine-rich protein 61, Cyr61)是一种细胞外基质相关蛋白,参与心肌细胞增殖和细胞凋亡过程^[8]。LI等^[9]报道,血清外泌体Cyr61水平升高有助于ACS诊断。但关于血清PTEN、Cyr61在ACS并发CS患者中的表达及与预后的相关性尚缺乏报道,鉴于此本研究拟分析如下,以期改善ACS并发CS患者预后提供更多的依据。

1 材料与方法

1.1 研究对象 选取2023年1月~2025年5月上海交通大学医学院附属第九人民医院收治的ACS并发CS患者203例(ACS并发CS患者组),年龄33~86(63.55 ± 11.73)岁;女97例、男106例;体质量指数(BMI) $19.83 \sim 30.45$ (25.11 ± 2.11)kg/m²。根据主动脉内球囊反搏-休克II(intra-aortic balloon pump in cardiogenic shock II, IABP-SHOCK II)积分将ACS并发CS患者分为低危ACS并发CS组(0~2分, 77例)、中危ACS并发CS组(3~4分, 72例)、高危ACS并发CS组(5~9分, 54例)^[10]。根据ACS并发CS患者入院

后30天内预后分为死亡组(77例)与存活组(126例)。另选取同期未并发CS的ACS患者203例(单纯ACS组),年龄27~82(63.18 ± 10.22)岁;女94例、男109例;BMI $18.72 \sim 30.17$ (25.04 ± 2.08)kg/m²。两组年龄、性别和BMI比较,差异无统计学意义(均 $P > 0.05$)。纳入标准:①有完整的临床资料;②年龄>18岁;③初诊为ACS并发CS,符合相关诊断标准^[11]。排除标准:①并发心力衰竭、先天性心脏病、心脏瓣膜病等其他心脏疾病者;②恶性肿瘤患者;③心脏手术史或血运重建史者;④其他心肌病变或心脏结构病变、心包疾病心律失常等原因引起的CS;⑤血液、自身免疫系统疾病或近1个月内使用免疫抑制剂。本研究经医院伦理委员会批准(SH9H-2022-T56-1),患者或家属自愿签署知情同意书。

1.2 仪器与试剂 PTEN(货号:EH1661)、Cyr61(货号:EH0108)试剂盒(武汉菲恩生物科技有限公司);全自动酶标仪(型号:Multiskan™ FC)(赛默飞世尔科技公司)。

1.3 方法

1.3.1 血清PTEN、Cyr61水平检测:采集所有患者入院时4ml肘静脉血,4℃条件下3 000r/min离心20min(半径10cm)提取血清,采用酶联免疫吸附法检测PTEN、Cyr61水平,具体操作严格按照试剂说明进行。

1.3.2 资料收集:收集ACS并发CS患者资料,包括性别、年龄、BMI、ACS类型、心率、血压、平均动脉压、呼吸频率、体温、血氧饱和度、基础疾病、IABP-SHOCK II积分、白蛋白、血红蛋白、白细胞计数、血小板计数、总胆红素、血肌酐、C反应蛋白、乳酸、心肌肌钙蛋白I、氨基末端脑钠肽前体(N-terminal pro-B-type natriuretic peptide, NT-proBNP)数据和治疗资料。

1.4 统计学分析 选用SPSS28.0软件。计数资料以 $n(\%)$ 表示,采用 χ^2 检验;经K-S检验正态分布的计量资料以均数 $\pm$ 标准差($\bar{x} \pm s$)表示,采用 t 检验或趋势

*F*检验,组间两两比较邦弗伦尼校正($P<0.017$);非正态分布的计量资料以 $M(P_{25}, P_{75})$ 表示,采用*U*检验;Spearman相关分析ACS并发CS患者血清PTEN、Cyr61水平与危险程度的相关性;多因素Logistic回归(输入法)和受试者操作特征(receiver operating characteristic, ROC)曲线分析ACS并发CS患者预后的因素及血清PTEN、Cyr61水平的预测效能,Delong检验比

较曲线下面积(AUC);检验水准 $\alpha=0.05$, $P<0.05$ 为差异具有统计学意义。

2 结果

2.1 ACS并发CS组与单纯ACS组血清PTEN、Cyr61水平比较 见表1。ACS并发CS组血清PTEN、Cyr61水平高于单纯ACS组,差异有统计学意义(均 $P<0.001$)。

表 1 ACS 并发 CS 组与单纯 ACS 组血清 PTEN、Cyr61 水平比较 ($\bar{x} \pm s$)

项目	ACS 并发 CS 组 ($n=203$)	单纯 ACS 组 ($n=203$)	<i>t</i>	<i>P</i>
PTEN (ng/ml)	4.98 ± 1.60	3.09 ± 0.87	14.790	< 0.001
Cyr61 (pg/ml)	396.46 ± 126.65	254.50 ± 83.89	13.315	< 0.001

2.2 不同危险程度ACS并发CS患者血清PTEN、Cyr61水平比较 见表2。中危ACS并发CS组、高危ACS并发CS组血清PTEN、Cyr61水平高于低危ACS并发CS组($t=10.302$ 、 10.414 ; 13.735 、 17.125),且高危ACS并发CS组血清PTEN、Cyr61水平高于中危

ACS并发CS组($t=4.306$ 、 9.574),差异具有统计学意义(均 $P<0.001$)。Spearman相关分析显示,ACS并发CS患者血清PTEN、Cyr61水平与危险程度呈正相关($r_s=0.682$ 、 0.661 ,均 $P<0.001$)。

表 2 不同危险程度 ACS 并发 CS 患者血清 PTEN、Cyr61 水平比较 ($\bar{x} \pm s$)

项目	低危 ACS 并发 CS 组 ($n=77$)	中危 ACS 并发 CS 组 ($n=72$)	高危 ACS 并发 CS 组 ($n=54$)	<i>F</i>	<i>P</i>
PTEN (ng/ml)	3.58 ± 1.09	5.71 ± 1.31	5.99 ± 1.08	134.746	< 0.001
Cyr61 (pg/ml)	284.00 ± 86.02	411.31 ± 63.84	537.03 ± 79.09	343.488	< 0.001

2.3 不同预后ACS并发CS患者临床资料比较 见表3。203例ACS并发CS患者30天死亡率为37.93%(77/203)。死亡组IABP-SHOCK II积分、乳酸、心肌肌

钙蛋白I、NT-proBNP、C反应蛋白、PTEN、Cyr61水平均高于存活组,差异具有统计学意义(均 $P<0.05$)。

表 3 不同预后 ACS 并发 CS 患者临床资料比较 [$n (\%)$, $\bar{x} \pm s$, $M (P_{25}, P_{75})$]

项目	死亡组 ($n=77$)	存活组 ($n=126$)	$\chi^2/t/U$	<i>P</i>
男性	45 (58.44)	61 (48.41)	1.927	0.165
年龄 (岁)	65.58 ± 11.09	62.31 ± 11.98	1.944	0.053
体质量指数 (kg/m^2)	24.89 ± 2.03	25.25 ± 2.15	-1.185	0.237
ACS 类型	急性心肌梗死	48 (38.10)	3.735	0.053
	不稳定型心绞痛	78 (61.90)		
心率 (次 /min)	96.00 (81.50, 113.00)	90.50 (76.75, 106.25)	4 374.500	0.241
收缩压 (mmHg)	102.77 ± 19.27	103.37 ± 20.04	-0.210	0.834
舒张压 (mmHg)	68.00 (58.50, 76.00)	66.50 (59.00, 78.25)	4 698.000	0.706
平均动脉压 (mmHg)	77.13 ± 14.52	79.13 ± 16.57	-0.876	0.382
呼吸频率 (次 /min)	19.00 (17.50, 22.00)	20.50 (18.00, 23.00)	4 196.500	0.106
体温 ($^{\circ}C$)	36.64 ± 0.57	36.53 ± 0.56	1.316	0.190
血氧饱和度 ($\%$)	96.00 (95.00, 97.00)	96.00 (95.00, 97.00)	4 423.000	0.286
基础疾病	高血压	36 (38.57)	3.619	0.057
	糖尿病	34 (26.98)	3.176	0.075

续表 3 不同预后 ACS 并发 CS 患者临床资料比较 [n (%) , $\bar{x} \pm s$, M (P₂₅, P₇₅)]

项目		死亡组 (n=77)	存活组 (n=126)	$\chi^2/t/U$	P
IABP-SHOCK II 积分 (分)		4.00 (3.00, 8.00)	3.00 (2.00, 4.00)	2 760.000	< 0.001
白蛋白 (g/L)		31.32 ± 5.88	32.37 ± 6.16	-1.192	0.235
血红蛋白 (g/L)		117.23 ± 29.13	116.73 ± 25.60	0.130	0.897
白细胞计数 (× 10 ⁹ /L)		11.94 ± 4.71	10.83 ± 3.52	1.795	0.075
血小板计数 (× 10 ⁹ /L)		192.26 ± 84.26	203.38 ± 74.97	-0.978	0.329
总胆红素 (μ mol/L)		26.65 (15.96, 38.37)	26.98 (17.38, 34.80)	4 796.500	0.893
血肌酐 (μ mol/L)		150.71 ± 83.70	130.98 ± 65.43	1.766	0.080
C 反应蛋白 (mg/L)		65.68 (41.21, 80.03)	51.58 (32.35, 69.21)	3 818.500	0.011
乳酸 (mmol/L)		4.84 (2.06, 7.99)	2.71 (1.24, 4.05)	3 010.000	< 0.001
心肌肌钙蛋白 I (μ g/L)		5.20 (1.98, 7.22)	3.52 (2.50, 5.17)	4 219.500	< 0.001
NT-proBNP (ng/L)		9 324.10 (5 011.70, 14 348.55)	5 277.25 (3 712.55, 8 092.00)	2 817.000	< 0.001
治疗资料	经皮冠状动脉介入治疗	32 (41.56)	70 (55.56)	3.746	0.053
	冠状动脉旁路移植术	3 (3.90)	8 (6.35)	0.185	0.667
	有创机械通气	41 (53.25)	50 (39.68)	3.555	0.059
	主动脉内球囊反搏	36 (46.75)	49 (38.89)	1.214	0.270
	体外膜肺氧合	8 (10.39)	4 (3.17)	3.270	0.071
	肾上腺素	49 (63.64)	63 (50.00)	3.593	0.058
	去甲肾上腺素	53 (68.83)	70 (55.56)	3.528	0.060
	β 受体阻滞剂	12 (15.58)	33 (26.19)	3.116	0.078
	抗血小板双联治疗	62 (80.52)	112 (88.89)	2.734	0.098
PTEN (ng/ml)		6.04 ± 1.35	4.32 ± 1.38	8.663	< 0.001
Cyr61 (pg/ml)		480.62 ± 112.39	345.03 ± 105.89	8.648	< 0.001

2.4 ACS 并发 CS 患者预后的多因素 Logistic 分析 以 IABP-SHOCK II 积分(原值录入)、乳酸(原值录入)、心肌肌钙蛋白 I(原值录入)、NT-proBNP(原值录入)、C 反应蛋白(原值录入)、PTEN(原值录入)、Cyr61(原值录入) 为自变量, ACS 并发 CS 患者预后(死亡/存活=1/0) 为因变量, 以输入法建立多因素 Logistic 回归模型。结果显示, IABP-SHOCK II 积分升高、乳酸升高、NT-proBNP 升高、PTEN 升高、Cyr61 升高为 ACS 并发 CS 患者死亡的独立危险因素(均 $P < 0.05$), 见表 4。

表 4 ACS 并发 CS 患者预后的多因素 Logistic 分析

变量	VIF	β	SE	Wald χ^2	P	OR	95%CI
IABP-SHOCKII 积分升高	1.121	0.385	0.115	11.285	< 0.001	1.469	1.174 ~ 1.839
乳酸升高	1.175	0.158	0.079	3.980	0.046	1.171	1.003 ~ 1.367
心肌肌钙蛋白 I 升高	1.041	-0.015	0.079	0.036	0.849	0.985	0.844 ~ 1.150
NT-proBNP 升高	1.147	0.012	0.005	5.530	0.019	1.012	1.002 ~ 1.023
C 反应蛋白升高	1.030	0.005	0.008	0.315	0.575	1.005	0.988 ~ 1.021
PTEN 升高	1.146	0.957	0.198	23.325	< 0.001	2.604	1.766 ~ 3.839
Cyr61 升高	1.156	0.012	0.003	22.786	< 0.001	1.012	1.007 ~ 1.018
常量	-	-13.985	2.012	48.328	< 0.001	< 0.001	-

2.5 血清 PTEN、Cyr61 水平对 ACS 并发 CS 患者死亡的预测效能 见表 5 和图 1。血清 PTEN、Cyr61 联合检测对 ACS 并发 CS 患者死亡评估的 AUC 大于单

项指标检测, 差异具有统计学意义($Z=3.928$ 、 4.329 , 均 $P < 0.001$)。

表5 血清PTEN、Cyr61水平对ACS并发CS患者死亡的预测效能

项目	AUC	95%CI	P	截断值	敏感度(%)	特异度(%)	最大约登指数
PTEN	0.811	0.750 ~ 0.862	< 0.001	5.24 ng/ml	83.12	65.08	0.4820
Cyr61	0.803	0.742 ~ 0.856	< 0.001	421.17 pg/ml	81.82	63.49	0.4531
二者联合	0.910	0.862 ~ 0.945	< 0.001	-	81.82	83.33	0.6515

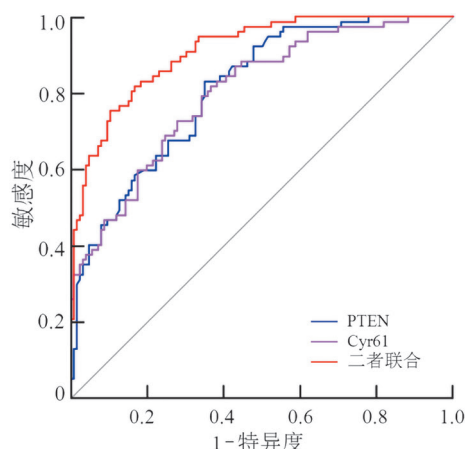


图1 血清PTEN、Cyr61水平预测ACS并发CS患者死亡的ROC曲线

3 讨论

CS是心脏泵功能急剧衰竭导致心输出量显著下降而引起组织灌注不足及多器官功能障碍的临床综合征^[10-11]。ACS因冠状动脉急性闭塞或严重狭窄导致大面积心肌缺血坏死,引起心脏泵功能急剧衰竭、心排量明显减少、低血容量、机械并发症(急性二尖瓣反流、乳头肌功能不全等)等而诱发CS,由于其进展快、危险度高,故死亡率较高^[12-13]。本研究中ACS并发CS患者30天死亡率为37.93%,这与既往报道相似^[14]。鉴于ACS并发CS患者病情进展迅速、病死率高,寻找可用于早期风险分层与预后评估的相关血液指标,对于临床实施个体化干预、改善患者预后具有重要意义。

ACS导致的急性心肌缺血缺氧可激活炎症反应、氧化应激和细胞凋亡信号通路,促使炎症因子、活性氧大量释放并引发心肌细胞损伤;持续炎症、氧化应激和细胞凋亡可进一步加重心功能障碍,导致心脏泵功能急剧衰竭,从而促进CS的发生发展^[15]。PTEN是具有脂质磷酸酶和蛋白磷酸酶双重活性的一种抑癌因子,能通过去磷酸化磷脂酰肌醇-3,4,5-三磷酸调控相关信号通路,促进炎症和细胞凋亡^[6]。PTEN在结扎左前支诱导的急性心肌梗死大鼠中高表达,能上调促炎细胞因子促进心肌细胞炎症和心肌损伤^[16]。反之,抑制PTEN表达能激活磷脂酰肌醇-3-激酶/蛋白激酶B信号通路,下调半胱天冬酶-3/9等凋亡蛋白表达,减少心肌细胞凋亡^[17]。尽管有研究指出,PTEN水平升高与ACS发生及冠状动脉狭窄程度加重有关^[7],但关于

血清PTEN对ACS并发CS患者的临床意义尚未可知。本研究发现,ACS并发CS患者血清PTEN水平升高,且随着危险程度加重而升高,与死亡风险增加相关。PTEN升高通过去磷酸化磷脂酰肌醇-3,4,5-三磷酸抑制磷脂酰肌醇-3-激酶/蛋白激酶B信号通路激活,促进核因子- κ B等促炎信号通路活化,增强全身炎症反应,进一步加重心肌损伤及循环功能障碍^[17-18]。同时,PTEN升高可抑制磷脂酰肌醇-3-激酶/蛋白激酶B和核因子E2相关因子2信号通路活性,削弱抗氧化防御能力,导致心脏泵功能下降,并通过循环系统波及肾脏、肝脏等多器官,促进多器官功能衰竭^[19-20]。这些病理过程均为CS典型特征,因此PTEN升高可能反映了CS患者全身炎症、内皮功能障碍及多器官损伤的综合病理状态,从而促进ACS并发CS病情进展,增加患者死亡风险。

Cyr61是被多种刺激诱导表达的一种细胞外基质相关蛋白,不仅参与促进炎症,也能发挥促增殖和抗凋亡等保护作用^[8]。Cyr61在氧化应激诱导的大鼠心肌细胞损伤模型中高表达,参与促进心肌细胞存活和抑制凋亡,下调Cyr61可促进心肌细胞凋亡^[21]。但在心肌缺血再灌注损伤中,Cyr61高表达能促进心肌炎症,加剧心肌损伤^[22];同时WINZAP等^[23]报道,血清Cyr61水平升高与ACS患者炎症水平升高及1年主要不良心脏事件风险增加有关。本研究发现,ACS并发CS患者血清Cyr61水平升高,且随着危险程度加重而升高,与死亡风险增加相关,这可能与Cyr61双向作用的综合效应有关。Cyr61升高可能反映心肌在缺血、氧化应激及高负荷状态下的应激反应,增强心肌细胞增殖和抗凋亡能力,对心肌提供一定保护作用^[24];另一方面,Cyr61同时可激活Toll样受体4/核因子- κ B及c-Jun氨基末端激酶/c-Jun信号通路,加重全身炎症反应、内皮功能障碍及多器官损伤,从而加速CS进展,提升患者死亡风险^[22,25]。因此,血清Cyr61水平升高可能既反映心肌损伤的严重程度,也参与CS的病理进程,是患者危险程度及死亡风险的重要潜在标志物。

本研究还发现,IABP-SHOCK II积分升高、乳酸升高、NT-proBNP升高为ACS并发CS患者死亡的独立危险因素,这与既往报道相似。IABP-SHOCK II积分越高提示循环衰竭和多器官功能障碍越严重;乳酸升高反映机体处于严重缺氧及能量

代谢失衡状态; NT-proBNP升高提示心室压力负荷和心功能损伤加重, 因此ACS并发CS患者死亡风险更高^[26-27]。本研究ROC曲线显示, 血清PTEN、Cyr61水平及二者联合预测ACS并发CS患者死亡的AUC分别为0.811、0.803、0.910, 二者联合预测效能优于血清PTEN、Cyr61水平单独预测。这说明血清PTEN、Cyr61水平有助于预测ACS并发CS患者预后, 且血清PTEN、Cyr61水平联合具备更高的预测效能。

综上所述, 血清PTEN、Cyr61水平升高与ACS并发CS患者危险程度加重及死亡风险增加相关, 二者联合预测ACS并发CS患者预后的效能较高。但本研究为单中心研究, 整体样本量有限, 且高危ACS并发CS组样本量相对较少, 可能影响亚组分析的统计效力并存在选择偏倚; 其次仅检测了入院时单次血清PTEN、Cyr61水平, 未动态观察其随治疗干预变化的趋势; 此外, 研究未结合影像学评估或分子机制实验进行深入验证。未来研究可通过多中心、大样本前瞻性设计, 结合多时间点动态监测及机制探讨, 以进一步提高结果的可靠性和临床可推广性。

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