·临床研究·

凝血酶原活动度与冠状动脉慢血流患者预后的相关性分析

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【摘 要】 **目的** 筛查预测冠状动脉慢血流(CSFP)患者出院后再发不良心血管事件的有效分子标志物。**方法** 通过前瞻性观察研究分析了2013年1月至2014年12月在解放军总医院心内科住院治疗的行冠状动脉造影(CAG)确诊为单纯CSFP患者共54例,出院后随访发生心血管不良事件患者10例(不良事件组),未发生不良事件44例(无不良事件组),对比分析两组患者一般实验室指标和心脏超声指标。对单因素分析有统计学意义的数据及预测CSFP预后的实验室指标纳入Cox回归分析模型;将最为显著的独立危险因素纳入ROC曲线分析;并采用Kaplan-Meiers生存分析比较不同危险因素患者的不良心血管事件的发生率。**结果** 不良事件组甘油三酯(TG)、载脂蛋白B(ApoB)、肌酸激酶MB同工酶(CK-MB)、凝血酶原活动度(PTA)显著高于无不良事件组($P<0.05$)。Cox回归模型分析显示PTA增高是患者预后不良的独立危险因素($HR=1.193$, 95% *CI* 为1.036~1.374, $P<0.05$)。采用Kaplan-Meiers生存率分析,高PTA患者的总体不良事件发生率明显高于低PTA患者($P<0.05$),血浆PTA预测患者出院后随访期间不良心血管事件的ROC曲线相关性好(曲线下面积0.717, $P=0.033$)。**结论** PTA可作为预测CSF患者的长期不良事件发生率的检测指标。

【关键词】 凝血酶原活动度;冠状动脉慢血流;危险因素;预后
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Correlation analysis of prothrombin activity with prognosis of patients with coronary slow flow phenomenon

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【**Abstract**】 **Objective** To explore the effective laboratory markers for predicting adverse cardiovascular events in the patients with coronary slow flow phenomenon (CSFP). **Methods** A prospective study was carried out on 54 patients who underwent coronary angiography (CAG) and diagnosed with simple CSFP in our department from January 2013 to December 2014. According to the incidence of adverse cardiovascular events during the follow-up, they were divided into adverse events group ($n=10$) and adverse events-free group ($n=44$). The laboratory indicators and echocardiographic parameters were compared and analyzed between the 2 groups. Statistical data from univariate analysis and laboratory indicators predicting CSFP prognosis were included in the Cox regression model. The most significant independent risk factors were included in the receiver operating characteristic (ROC) curve analysis. Kaplan-Meiers survival analysis was used to compare the incidence of adverse cardiovascular events among the patients with different risk factors. **Results** The serum contents of triglycerides (TG), apolipoproteins B (ApoB), creatine kinase MB isoenzyme (CK-MB), and plasma prothrombin activity (PTA) were significantly higher in the adverse events group than the adverse events-free group ($HR=1.193$, 95% *CI* 1.036 – 1.374, $P<0.05$). Cox regression analysis showed that increased PTA was an independent risk factor for poor prognosis ($P<0.05$). Kaplan-Meiers survival analysis also indicated that those with higher PTA had higher incidence of adverse events than those with low PTA ($P<0.05$). The plasma PTA showed good correlation between the ROC curve and adverse cardiovascular events during the follow-up period after discharge (area under the curve = 0.717, $P=0.033$). **Conclusion** PTA can be used as an indicator in the prediction of long term adverse events in CSFP patients.

【**Key words**】 prothrombin activity; coronary slow flow phenomenon; risk factors; prognosis
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冠状动脉慢血流现象(coronary slow flow phenomenon,CSFP)指除溶栓治疗后、冠状动脉成形术后、冠状动脉痉挛、冠状动脉扩张、心肌病及瓣膜病等因素外,冠状动脉造影(coronary angiography,CAG)中没有发现冠状动脉存在明显病变,却发生血流灌注延迟的现象[1]。CSFP患者通常表现为胸痛反复发作,严重影响了生活质量[2]。随着CAG技术的广泛开展和应用,CSFP诊断率日趋提高,但其具体机制目前尚不清楚,有相当比例的CSFP患者预后并不理想,心绞痛反复发作,再住院率高,影响了生活质量,给患者家庭及社会造成了沉重的负担。因此,寻找CSFP预后相关的分子标志物十分重要。本研究旨在寻找与CSFP患者预后相关的简单实用的预测指标。

1 对象与方法

1.1 研究对象

入选2013年1月至2014年12月在解放军总医院心内科住院,有明显胸闷症状,伴有心电图缺血改变,且CAG未见明显狭窄,除外冠状动脉夹层、痉挛,证实为单纯CSFP的患者54例,其中男43例,女11例。纳入标准:(1)左室射血分数 > 55%;(2)CAG无冠状动脉狭窄或扩张、冠状动脉痉挛;(3)入院检查肌钙蛋白I(troponin I, cTnI) < 0.01;(4)CSFP定义为对比剂在≥3个心动周期内不能到达血管末端;(5)全部患者均自愿参加本研究。排除标准:(1)急性心肌梗死或既往有陈旧性心肌梗死病史;(2)既往有冠状动脉介入(percutaneous coronary intervention,PCI)治疗病史;(3)既往有心肌病、瓣膜病、高血压心脏病、先天性心脏病史;(4)肝肾功能不全者[天门冬氨酸氨基转移酶(aspartate aminotransferase, AST)、丙氨酸氨基转移酶(alanine aminotransferase, ALT) > 2倍正常值上限],血肌酐(creatinine, SCr)≥2 mg/dl;(5)恶性肿瘤、合并感染、免疫性疾病患者;(6)有脑血管意外病史;(7)难以控制的高血压病、糖尿病、继发性高血压、外周血管病;(8)患有器质性心脏病(如心瓣膜病、扩张型心肌病、肥厚型心肌病、慢性肺源性心脏病等)及严重心功能不全[美国心脏病学会(NYHA)分级≥Ⅲ级]。

1.2 方法

详细记录患者的一般临床资料,包括姓名、性别、年龄、吸烟史、饮酒史等资料,在入院或急诊手术当天进行实验室化验及心脏超声检查并记录,包括血常规、血生化、凝血,并在进行PCI术时记录冠状动脉狭窄情况以及心肌梗死溶栓(thrombolysis in myocardial infarction,TIMI)血流情况。于患者出院后进行定期电话或门诊随访,中位随访时间为848 d(369.5~1239.2 d),随访主要不良心血管事件(major adverse cardiovascular events,MACE,包括急性心肌梗死、心源性休克、PCI、冠状动脉搭桥和卒中)和二级终点(包括再住院、再发心绞痛、血栓、出血、心力衰竭和心律失常)。出院后发生不良事件患者10例(不良事件组),男性7例,女性3例;未发生不良事件患者44例(无不良事件组),男性36例,女性8例。

1.3 统计学处理

采用SPSS 17.0软件对数据进行统计分析,计量资料呈正态分布的采用均数±标准差($\bar{x} \pm s$)表示,组间比较采用t检验;呈偏态分布的采用四分位数表示,组间比较采用秩和检验;计数资料采用率和相对数表示,组间比较采用χ^2检验;对单因素分析有统计学意义的指标及预测CSFP预后的实验室指标纳入Cox回归分析模型,研究与CSFP预后相关的危险因素;将最为显著的独立危险因素纳入ROC曲线分析;并采用Kaplan-Meiers生存分析比较不同危险因素患者的不良心血管事件的发生率。$P < 0.05$为差异有统计学意义。

2 结 果

2.1 两组一般临床资料、实验室指标、超声指标情况比较

不良事件组TG、ApoB、CK-MB、PTA均显著高于无不良事件组,差异具有统计学意义($P < 0.05$;表1)。

2.2 CSFP患者预后的危险因素分析

以发生不良心血管事件为随访观测终点事件,将具有统计学意义的TG、ApoB、CK-MB、PTA纳入Cox回归模型分析显示,PTA($HR = 1.193$, 95% CI 1.036~1.374)增高是患者预后不良的独立危险因素($P = 0.014$;表2)。

2.3 PTA对CSFP患者预后发生不良事件的诊断价值

将血浆PTA纳入ROC曲线图,患者出院后随访期间是否发生不良心血管事件为金标准,以PTA对CSF患者预后发生不良事件的预测灵敏度为纵坐标,1-特异度为横坐标,绘制ROC曲线。其曲线下面积为0.717(95% CI 0.521~0.913, $P = 0.033$)。当PTA为97.5%时,具有最佳预测效能,此时灵敏度为90%,特异度为45.5%(图1)。

表1 两组不同预后的CSF患者一般临床资料和化验指标比较

Table 1 Comparison of clinical data and laboratory test indices between two groups with different prognosis in CSF patients

Item	Adverse events group($n=10$)	Adverse events-free group($n=44$)	P value
Age(years,$\bar{x}\pm s$)	53.2±11.7	52.9±10.0	0.941
HR(beats/min,$\bar{x}\pm s$)	75.8±12.7	73.4±11.9	0.577
SBP[mmHg,$M(Q_1,Q_3)$]	128.5(118.8,144.0)	126.5(117.3,142.5)	0.798
DBP(mmHg,$\bar{x}\pm s$)	78.9±9.8	79.1±12.0	0.967
BMI(kg/m^2,$\bar{x}\pm s$)	27.4±3.6	26.1±4.0	0.325
Male[n(%)]	7(70.0)	36(81.8)	0.408
Alcohol drinking[n(%)]	4(40.0)	15(34.1)	0.595
Smoking[n(%)]	3(30.0)	19(43.2)	0.728
Hypertension[n(%)]	4(40.0)	21(47.7)	0.736
Diabetes mellitus[n(%)]	1(10.0)	7(15.9)	1.000
Hyperlipidemia[n(%)]	2(20.0)	7(15.9)	0.667
Biochemical index			
TC(mmol/L,$\bar{x}\pm s$)	4.5±0.9	4.4±0.9	0.764
TG[mmol/L,$M(Q_1,Q_3)$]	2.40(1.80,3.17)	1.55(0.98,2.40)	0.041
LDL-C[mmol/L,$M(Q_1,Q_3)$]	2.840(2.220,3.155)	2.610(2.190,3.020)	0.498
HDL-C[mmol/L,$M(Q_1,Q_3)$]	1.060(0.925,1.145)	0.970(0.895,1.140)	0.767
Apo A1(g/L,$\bar{x}\pm s$)	1.32±0.12	1.19±0.19	0.113
Apo B(g/L,$\bar{x}\pm s$)	1.02±1.03	0.85±0.17	0.027
SCr(μmol/L,$\bar{x}\pm s$)	72.6±15.5	75.7±13.3	0.534
BUN[mmol/L,$M(Q_1,Q_3)$]	4.7(4.0,6.8)	5.4(4.1,7.2)	0.798
BUA(μmol/L,$\bar{x}\pm s$)	363.5±63.8	366.8±98.9	0.921
ALT[U/L,$M(Q_1,Q_3)$]	29.2(22.5,36.7)	20.3(13.7,37.3)	0.114
AST(U/L,$\bar{x}\pm s$)	21.7±6.0	21.6±13.9	0.983
γ-GT[U/L,$M(Q_1,Q_3)$]	41.7(28.7,63.1)	25.6(17.9,57.9)	0.162
TBIL[μmol/L,$M(Q_1,Q_3)$]	12.2(9.1,15.9)	11.7(8.8,14.0)	0.603
DBIL(μmol/L,$\bar{x}\pm s$)	3.6±1.3	3.5±1.7	0.909
CK[U/L,$M(Q_1,Q_3)$]	93.5(77.0,119.5)	73.6(53.1,103.0)	0.087
CK-MB[ng/ml,$M(Q_1,Q_3)$]	12.5(2.1,17.5)	1.7(1.3,11.5)	0.021
cTnT[ng/ml,$M(Q_1,Q_3)$]	0.003(0.003,0.005)	0.005(0.003,0.008)	0.115
Potassium(mmol/L,$\bar{x}\pm s$)	4.0±0.3	3.8±0.3	0.063
Sodium[mmol/L,$M(Q_1,Q_3)$]	142.7(139.7,143.6)	141.7(140.6,143.5)	0.874
Chloride[mmol/L,$M(Q_1,Q_3)$]	103.7(101.5,106.6)	105.4(104.2,107.2)	0.159
Calcium[mmol/L,$M(Q_1,Q_3)$]	2.24(2.15,2.31)	2.27(2.22,2.32)	0.314
Phosphorus inorganic(mmol/L,$\bar{x}\pm s$)	1.1±0.1	1.1±0.2	0.517
Magnesium(mmol/L,$\bar{x}\pm s$)	0.92±0.09	0.91±0.08	0.612
Glucose[mmol/L,$M(Q_1,Q_3)$]	5.4(5.0,6.0)	5.2(4.8,6.1)	0.400
BNP[pg/ml,$M(Q_1,Q_3)$]	90.5(6.1,222.2)	39.6(20.0,80.1)	0.835
Glycosylated hemoglobin[%,$M(Q_1,Q_3)$]	5.9(5.0,6.3)	6.9(5.5,6.1)	0.679
Hcy[μmol/L,$M(Q_1,Q_3)$]	11.8(9.3,16.8)	14.3(12.1,20.2)	0.168
CRP[mg/L,$M(Q_1,Q_3)$]	0.31(0.24,0.34)	0.30(0.10,0.35)	0.609
Hematologic index($\bar{x}\pm s$)			
Hemoglobin(g/L)	148.8±13.2	148.1±12.7	0.871
Red blood cell count($\times10^{12}$/L)	4.90±0.45	4.80±0.42	0.454
White blood cell count($\times10^9$/L)	6.5±1.2	6.6±1.6	0.873
Neutrophil(%)	0.56±0.05	0.59±0.08	0.305
RDW(%)	12.70±0.50	12.80±0.46	0.672
PLT(fl)	230.1±81.9	222.5±51.8	0.715
Coagulative function			
D-dimer[μg/ml,$M(Q_1,Q_3)$]	0.26(0.19,0.31)	0.24(0.19,0.43)	0.633
TT(s,$\bar{x}\pm s$)	16.5±1.8	16.5±1.0	0.981
APTT(s,$\bar{x}\pm s$)	36.0±2.8	37.0±5.1	0.534
PT[s,$M(Q_1,Q_3)$]	12.7(12.2,13.0)	13.0(12.6,13.6)	0.068
PTA[%,$M(Q_1,Q_3)$]	109.0(100.3,114.5)	99.0(93.0,108.0)	0.035
Fg(g/L,$\bar{x}\pm s$)	3.00±0.47	3.00±0.59	0.967

续表

Item	Adverse events group($n=10$)	Adverse events-free group($n=44$)	P value
Echocardiogram			
LVEDD(mm, $\bar{x}\pm s$)	45.9±5.6	45.3±4.1	0.710
IVST[mm, $M(Q_1,Q_3)$]	31.0(27.0,33.5)	30.5(28.3,33.8)	0.924
LVPw[mm, $M(Q_1,Q_3)$]	10.0(9.0,11.0)	10.0(9.0,11.0)	0.784
LVEF[%, $M(Q_1,Q_3)$]	61.0(56.5,62.0)	60.0(57.0,62.0)	0.942

CSF: coronary slow flow; HR: heart rate; SBP: systolic blood pressure; DBP: diastolic blood pressure; BMI: body mass index; TC: total cholesterol; TG: total triglycerides; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; Apo A1: apolipoprotein A1; Apo B: apolipoprotein B; SCr: serum creatinine; BUN: blood urea nitrogen; BUA: blood uric acid; ALT: alanine aminotransferase; AST: aspartate aminotransferase; γ-GT: γ-transglutaminase; TBIL: total bilirubin; DBIL: direct bilirubin; CK: creatine kinase; CK-MB: creatine kinase MB isoenzyme; cTnT: cardiac troponin T; BNP: brain natriuretic peptide; Hcy: homocysteine; CRP: C reactive protein; RDW: red blood cell volume distribution width; PLT: platelet; TT: thrombin time; APTT: activated partial thromboplastin time; PT: prothrombin time; PTA: prothrombin time activity; Fg: fibrinogen; LVEDD: left ventricular end-diastolic dimension; IVST: interventricular septal thickness; LVPw: left ventricular posterior wall; LVEF: left ventricular ejection fraction

表 2 随访期间不良心血管事件的 Cox 回归分析

Table 2 Cox regression analysis of the adverse cardiovascular events

Index	*B*	*SE*	*HR*	95% *CI*	*P* value
TG	0.196	0.218	1.217	0.793 – 1.866	0.369
CK-MB	–0.012	0.019	0.988	0.951 – 1.026	0.520
PTA	0.176	0.072	1.193	1.036 – 1.374	0.014
Apo B	0.125	0.076	0.882	0.760 – 1.025	0.100

TG: total triglycerides; CK-MB: creatine kinase MB isoenzyme; PTA: prothrombin time activity; Apo B: apolipoprotein B; HR: hazard ratio; CI: confidence interval

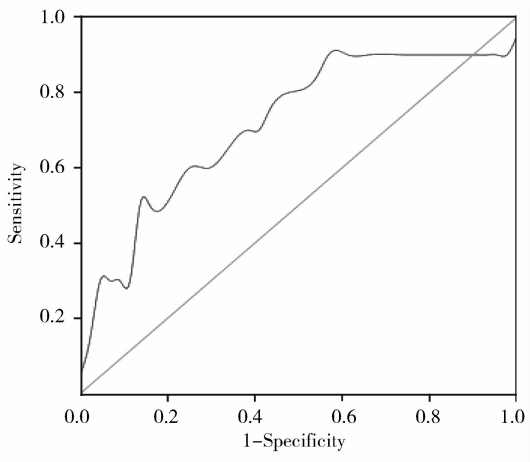


图 1 血浆 PTA 预测 CSF 患者出院后随访期间是否发生不良心血管事件的 ROC 曲线

Figure 1 ROC curve of PTA in prediction of adverse events of CSF patients during follow-up

ROC: receiver operating characteristic; PTA: prothrombin time activity; CSF: coronary slow flow

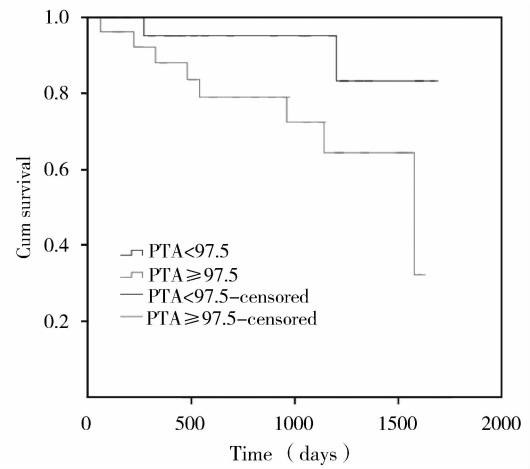


图 2 PTA 高分位组与低分位组预后比较

Figure 2 Comprison of prognosis between high PTA and low PTA

Cum: cumulative; PTA: prothrombin time activity. Cumulative survival in high PTA group is significantly lower than that in low PTA group ($P<0.05$)

2.4 不同血浆 PTA 患者随访不良事件发生率分析

血浆 PTA≥97.5%，为高 PTA 组，共计 32 例；血浆 PTA<97.5% 为低 PTA 组，共计 22 例。采用 Kaplan-Meiers 生存率分析，高血浆 PTA 患者出院后生存率低于低血浆 PTA 患者（70.4% *vs* 92.6%）。即高血浆 PTA 患者出院后不良事件发生率高于低血浆 PTA 患者，差异有统计学意义（29.6% *vs* 7.4%；$P<0.05$；图 2）。

3 讨 论

凝血酶原是血液凝固因子之一，既往国内外研究一致认为 PTA 是判断肝细胞坏死严重程度及预后的敏感指标。当肝脏实质受到损伤，凝血因子的含量和活动度可呈不同程度的减低，常引起出血、淤血等临床表现，如维生素 K 缺乏、血循环中有抗凝物质、肝脏疾病、牙龈出血、鼻出血、青紫色淤痕等。凝血因子的含量和活动度增高，可见于先天性因子 V

增多症、口服避孕药、血栓栓塞性疾病、血液高凝状态等情况。

目前CSFP的发病机制尚不完全清楚,其发病机制可能与冠状动脉微血管内皮损伤、炎症因子、血小板功能异常以及血液黏稠度增高有关[3-6]。既往研究发现心血管疾病患者体内的凝血因子较健康人偏高[7]。血栓生成、血液的高凝状态在心血管疾病的发生和发展中起着重要的作用[8]。本研究通过Cox回归分析发现,PTA是CSFP患者预后的独立危险因素,且通过Kaplan-Meiers生存率分析发现,住院期间低PTA的患者出院后不良事件发生率低于PTA升高的患者。可能原因包括以下几个方面。(1)凝血系统的激活与血栓栓塞性疾病、心脑血管疾病及多器官功能障碍等有着密切关系,不良事件组患者常合并全身炎症反应和凝血系统的激活,两者相互影响,共同导致微循环衰竭和多器官功能衰竭,而血液高凝反应通过上调凝血物质、下调抗凝物质以及抑制纤维蛋白溶解来调节凝血系统[9,10]。(2)CSFP微循环不佳,循环末梢供血不足,心肌缺血缺氧,从而造成典型或不典型心绞痛症状发生。因此,凝血因子含量增加或活动度增高、血液处于高凝状态、增加血栓栓塞疾病发生的风险可能与CSFP患者出院后发生不良心血管事件有关。新近临床研究发现血浆D-二聚体水平与急性心肌梗死患者介入术中无复流发生密切相关[11,12]。(3)在CSFP的患者中,虽然CAG明确显示冠状动脉血流通畅,但血脂偏高、血液黏稠度过高仍是血液流速过慢的主要因素之一[13,14]。既往研究表明,TG升高1 mmol/L,男性和女性发生心血管事件的风险分别增加12%和37%[15,16]。因此,CSFP患者进行必要的血脂检测与凝血筛查并及时预防治疗,对减少不良心血管事件的发生有着重要意义。

本研究发现PTA对CSFP患者的预后存在一定的诊断价值,最佳诊断值为97.5%,当CSFP患者PTA值高于97.5%时,治疗上积极给予各种干预措施降低PTA,有助于改善患者的预后,减少心血管事件的发生。

本研究为前瞻性观察性研究,主要关注单纯CSFP患者预后生存率的危险因素分析。研究显示PTA是预测CSFP患者预后的重要标记物,PTA升高提示CSFP患者的长期预后不良。临床需对PTA较高的CSFP患者采取更为积极有效的诊疗干预,改善此类患者的预后,提高患者的生活质量。由于CSFP的整体发病率偏低,本研究的样本量有限,研究结果还需要大规模多中心的临床试验进一步观察证实。

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