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早发冠心病患者血清锌 a2 糖蛋白、单核细胞趋化蛋白 -1 水平与血脂的关系及其影响因素分析 *

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摘要 目的:分析早发冠心病(PCAD)患者血清锌 a2 糖蛋白(ZAG)、单核细胞趋化蛋白 -1(MCP-1)水平与血脂的关系及其影响因素。**方法:**收集 2017 年 1 月 -2019 年 12 月在我院经冠状动脉造影确诊的冠心病患者 184 例,其中 PCAD 患者 86 例(PCAD 组),非 PCAD 患者 98 例(NPCAD 组),再选取同期男性 <55 岁,女性 <65 岁健康体检者 86 例作为对照组。收集所有研究对象的基线资料并检测空腹血糖(FBG)、总胆固醇(TC)、低密度脂蛋白(LDL)、甘油三酯(TG)、高密度脂蛋白(HDL)、血清 ZAG、MCP-1 水平,采用 Pearson 相关性分析 ZAG、MCP-1 与血脂相关性。多因素 Logistic 回归分析 PCAD 的影响因素。**结果:**PCAD 组、NPCAD 组糖尿病史、高血压病史、冠心病家族史比例、体质指数(BMI)、FPG、TG、MCP-1 高于对照组,HDL、ZAG 水平低于对照组($P<0.05$),PCAD 组年龄、HDL、ZAG 水平低于 NPCAD 组,冠心病家族史、吸烟史比例高于 NPCAD 组($P<0.05$)。Pearson 相关性分析结果表明,PCAD 患者 ZAG 与 HDL 呈正相关,MCP-1 与 HDL 呈负相关($P<0.05$);多因素 Logistic 回归分析显示,高血压、吸烟史、冠心病家族史、HDL、TG、ZAG、MCP-1 是 PCAD 的独立危险因素。**结论:**PCAD 患者 MCP-1 水平升高、ZAG、HDL 水平降低,MCP-1、ZAG 与 HDL 密切相关,且是 PCAD 的独立危险因素。

关键词:早发冠心病;锌 a2 糖蛋白;单核细胞趋化蛋白 -1;血脂;危险因素

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Relationship between Serum Zinc a2 Glycoprotein and Monocyte Chemotactic Protein-1 Levels and Blood Lipids in Patients with Premature Coronary Artery Disease and Analysis of Influencing Factors*

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ABSTRACT Objective: To analyze the relationship between serum zinc a2 glycoprotein (ZAG), monocyte chemotactic protein-1 (MCP-1) levels and blood lipids and their influencing factors in patients with premature coronary artery disease (PCAD). **Methods:** 84 patients with coronary heart disease diagnosed by coronary angiography in our hospital from January 2017 to December 2019 were collected, including 86 patients with PCAD (PCAD group) and 98 patients with non PCAD patients (NPCAD group). Then, 86 healthy patients with male <55 years old and female <65 years old during the same period were selected as the control group. Baseline data of all subjects were collected and levels of fasting blood glucose(FBG), total cholesterol(TC), triglyceride(TG), low-density lipoprotein(LDL), high-density lipoprotein (HDL), serum ZAG, MCP-1 were measured. Pearson correlation was used to analyze the correlation between ZAG, MCP-1 and blood lipid. Multivariate Logistic regression was used to analyze the influencing factors of PCAD. **Results:** Diabetes history, hypertension history, family history of coronary heart disease, body mass index (BMI), FPG, TG, MCP-1 in the PCAD group, NPCAD group were higher than control group, the levels of HDL, ZAG were lower than control group ($P<0.05$). The age and levels of HDL, ZAG levels in the PCAD group were lower than the NPCAD group, the proportion of family history of coronary heart disease and smoking history was higher than the NPCAD group($P<0.05$). Pearson correlation analysis results showed that ZAG was positively correlated with HDL, and MCP-1 was negatively correlated with HDL ($P<0.05$). Multivariate Logistic regression analysis showed that hypertension, smoking history, family history of coronary heart disease, HDL, TG, ZAG and MCP-1 were independent risk factors for PCAD. **Conclusions:** In patients with PCAD, MCP-1 levels increase, ZAG, HDL levels decrease, MCP-1, ZAG and HDL are closely related, and they are independent risk factors for PCAD.

Key words: Premature coronary artery disease; Zinc a2 glycoprotein; Monocyte chemoattractant protein-1; Blood fat; Risk factor

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前言

冠心病的发病率逐年增加,且呈现年轻化的趋势,其中发病年龄男性小于 55 岁,女性小于 65 岁称为早发冠心病(PCAD)^[1]。PCAD 增加不良心血管不良事件和死亡率^[2]。发展中国家 PCAD 的患病率达到 31%^[3]。早期对 PCAD 危险因素有效干预,能够降低 PCAD 发病率和减少继发性心血管疾病的发生,故寻找简单、可靠的血清学标志物对预防 PCAD 的发生或延缓疾病的进展具有重要意义。血脂代谢异常是 PCAD 的危险因素^[4]。血清锌 α₂ 糖蛋白(ZAG)是一种新型的脂肪细胞因子,可以促进脂肪分解和利用,抑制脂肪合成,减少脂肪积聚,并参与了体重、能量代谢、脂质代谢平衡的调节过程^[5]。单核细胞趋化蛋白-1(MCP-1)主要由巨噬细胞和内皮细胞产生,趋化和激活单核细胞至炎症部位,活化成为巨噬细胞,与低密度脂蛋白结合转变为巨噬细胞源性泡沫细胞,具有促进炎症应答、血管平滑肌细胞的增殖、动脉粥样硬化斑块形成的作用^[6]。本文通过分析 PCAD 患者的 ZAG、MCP-1 水平与血脂的关系及其影响因素,从而为 PCAD 的诊断及治疗方案提供依据。

1 资料与方法

1.1 研究对象

收集 2017 年 1 月-2019 年 12 月在我院经冠状动脉造影确诊的冠心病患者 184 例,再根据年龄将患者分成 PCAD 组(男性<55 岁,女性<65)和非 PCAD 组(NPCAD 组)(男≥55 岁,女≥65 岁),其中 PCAD 组 86 例,NPCAD 组 98 例。纳入标准:①符合冠心病诊断^[7];②患者意识清晰,无精神及言语障碍,可正常沟通交流;③临床资料完整;④患者知情同意,自愿参加本研究。排除标准:①风心病、先心病、心肌病、肺心病、单纯心律失常、瓣膜病等其他器质性心脏病;②肝、肾功能异常;③合

并严重感染、代谢性疾病,脑血管病变;④入院前口服降脂药物者。再选取同期男性<55 岁,女性<65 岁健康体检者 86 例,作为对照组。三组研究对象基线资料的比较见表 1。

1.2 观察指标

1.2.1 收集一般资料 记录所有研究对象性别、年龄、疾病史、吸烟等。测量所有研究对象身高、体重,计算体质质量指数(BMI)。

1.2.2 血清学指标的检测 采集研究对象晨起空腹采静脉血 5 mL,以 3000 转/min 转速,半径 20 cm,时间 20 min,离心后取血清,置于冰箱(-40℃)中保存备用,检测空腹血糖(FBG)、总胆固醇(TC)、低密度脂蛋白(LDL)、甘油三酯(TG)、高密度脂蛋白(HDL)等常规实验室指标。酶联免疫吸附法检测 ZAP、MCP-1 水平。具体操作严格按试剂盒说明书进行。BS-220 全自动生化分析仪由迈瑞公司提供。酶联免疫试剂盒购自武汉默沙克生物制剂有限公司。

1.3 统计学方法

数据应用 SPSS20.0 统计软件分析。计量资料经检验符合正态分布,以($\bar{x} \pm s$)表示,组间比较行 t 检验,多组间比较行单因素方差分析,以例或率表示计数资料,行 χ^2 检验。ZAP、MCP-1 与血脂间相关性采用 Pearson 相关性分析。PCAD 危险因素采用多因素 Logistic 回归分析。检验水准为 $\alpha=0.05$ 。

2 结果

2.1 基线资料比较

PCAD 组年龄低于 NPCAD 组,冠心病家族史、吸烟史比例高于 NPCAD 组($P<0.05$)。PCAD 组、NPCAD 组糖尿病史、高血压病史、冠心病家族史比例、BMI 高于对照组($P<0.05$)。见表 1。

表 1 三组研究对象基线资料比较

Table 1 Comparison of baseline data of three groups of subjects

Indexes	PCAD group(n=86)	NPCAD group(n=98)	Control group(n=86)	χ^2/F	P
Age(years old)	49.25±4.72*	67.36±5.24	51.39±4.96	367.96	0.000
Male/female	55/31	52/46	44/42	0.147	0.634
Smoking history[n(%)]	45(52.33)*#	37(37.76)	30(34.83)	3.936	0.047
Diabetes history[n(%)]	16(18.60)*	33(33.67)*	9(10.46)	5.323	0.031
Hypertension history[n(%)]	31(36.05)*	61(62.24)*	13(15.12)	11.862	0.001
Family history of coronary heart disease[n(%)]	31(36.05)*#	18(18.37)*	8(9.30)	17.542	0.000
BMI(kg/m ²)	25.19±2.16*	24.67±2.28*	22.87±1.86	28.715	0.000

Note: compared with control group, * $P<0.05$; compared with NPCAD group, * $P<0.05$.

2.2 三组血清学指标比较

PCAD 组、NPCAD 组 FPG、TG、MCP-1 高于对照组,HDL、ZAG 低于对照组,且 PCAD 组 HDL、ZAG 水平低于 NPCAD 组($P<0.05$),三组在 TC、LDL 水平差异无统计学意义($P>0.05$)。见表 2。

2.3 PCAD 患者 ZAG、MCP-1 与血脂指标的相关性分析

Pearson 相关性分析结果表明,PCAD 患者 ZAG 与 HDL

呈正相关,MCP-1 与 HDL 呈负相关($P<0.05$)。见表 3。

2.4 PCAD 的多因素 Logistic 回归分析

以 PCAD 为因变量,将年龄、吸烟史、BMI、高血压史、糖尿病史、冠心病家族史、HDL、TG、ZAG、MCP-1 纳入自变量,赋值如下:年龄 1=(50 岁≤男性<55 岁,60 岁≤女性<65 岁),0=(男性<50 岁,女性<60);吸烟史:1=是,0=否;高血压病史:1=有,0=否;冠心病家族史:1=有,0=否;糖尿病史:1=有,0=否;

BMI、HDL、TG、ZAG、MCP-1 按照实际数值输入。采用多因素 Logistic 回归分析,结果显示高血压病史、吸烟史、冠心病家族史、HDL 和 ZAG 水平降低,TG 和 MCP-1 升高是 PCAD 的独立危险因素($P<0.05$)。见表 4。

表 2 三组血清学指标比较($\bar{x} \pm s$)Table 2 Comparison of serological indexes among three groups($\bar{x} \pm s$)

Indexes	PCAD group(n=86)	NPCAD group(n=98)	Control group(n=86)	F	P
FPG(mmL)	6.23± 1.32 [#]	6.76± 1.41 [#]	5.12± 0.83	42.263	0.000
TG(mmL)	2.12± 0.31 [#]	2.06± 0.34 [#]	1.21± 0.29	228.196	0.000
TC(mmL)	5.02± 1.07	5.06± 1.01	4.87± 0.83	0.939	0.392
HDL(mmL)	1.09± 0.68 ^{**}	1.23± 0.43 [#]	1.41± 0.53	7.288	0.001
LDL(mmL)	2.97± 0.72	2.94± 0.58	2.85± 0.41	0.991	0.373
MCP-1(pg/mL)	73.54± 20.11 [#]	71.24± 18.6 [#]	33.71± 14.82	134.42	0.000
ZAG(ug/mL)	7.23± 4.87 ^{**}	8.53± 4.92 [#]	8.81± 4.15	3.468	0.033

Note: compared with control group, [#] $P<0.05$; compared with NPCAD group, ^{*} $P<0.05$.

表 3 PCAD 患者 ZAG、MCP-1 与血脂指标相关性分析

Table 3 Correlation analysis of ZAG, MCP-1 and blood lipid in patients with PCAD

Indexes	ZAG		MCP-1	
	r	P	r	P
TG	0.153	0.127	-0.054	0.248
TC	0.093	0.259	-0.028	0.138
HDL	0.331	0.008	-0.433	0.003
LDL	0.162	0.086	-0.175	0.082

表 4 PCAD 的多因素 Logistic 回归分析

Table 4 Multivariate Logistic regression analysis of PCAD

Risk factors	B	SE	Wald χ^2	P	OR	95%CI
Hypertension history	1.232	0.065	2.268	0.028	3.469	2.136-4.735
Smoking history	0.085	0.017	3.166	0.004	1.286	1.083-2.115
Family history of coronary heart disease	0.286	0.003	3.287	0.003	1.345	1.123-2.347
HDL	0.376	0.329	4.567	0.000	4.237	2.213-6.782
TG	0.186	0.027	2.765	0.015	1.037	1.003-1.983
ZAG	1.123	0.484	11.286	0.000	2.956	1.257-7.129
MCP-1	0.456	0.227	8.368	0.000	3.862	2.286-6.781

3 讨论

随着生活水平、工作压力、生活方式等不良因素的增加,冠心病患者日渐呈年轻化发展。由于 PCAD 缺少明显的临床症状,且病情发展迅猛,多表现为急性冠脉综合征,病死率较高^[8,9]。PCAD 动脉粥样硬化斑块富含脂质泡沫细胞,为软斑块,易破损、进展迅速^[10-12]。冠心病患者的血脂指标与正常人相比存在一定差异,HDL 亚型与冠心病的发生、发展,患者病变程度紧密联系,可作为冠心病患者病情程度、预测终点事件的重要指标^[13]。目前,PCAD 并没有特异性的风险预测指标,研究证实 HDL 在 PCAD 急性冠脉综合征患者外周血中明显降低,可作为该病预测指标^[14]。MCP-1 在冠心病患者中高表达,有助于监测冠心病严重程度。ZAG 水平在急性冠脉综合征中明显升高,

有可能成为冠心病危险分层有价值的参考指标。PCAD 患者中 MCP-1、ZAG 表达与 HDL 是否具有相关性,其在 PCAD 中的作用机制仍在探索中。

本研究可见 PCAD 组、NPCAD 组 FPG、TG、MCP-1 高于对照组,HDL、ZAG 低于对照组,且 PCAD 组 HDL、ZAG 水平低于 NPCAD 组,提示 PCAD 患者更容易出现血脂异常,脂质代谢紊乱更为明显。在动脉粥样硬化进展过程中,HDL 可阻止泡沫细胞生成,低 HDL 是冠心病的独立危险因素,HDL 降低,则患者发生冠心病风险增高^[15-17]。ZAG 作为一种新型脂肪动员因子,在体内多种组织中表达,与脂肪细胞的分化、成熟、分解与利用相关,通过协调多种信号转导途径参与免疫应答、炎症反应及维持代谢稳态机体的生理过程,调控机体糖脂代谢,能量平衡,抑制炎症因子表达,抗动脉粥样硬化分子的失衡,已成

为动脉粥样硬化及其相关疾病研究的热点^[18-20]。既往研究表明^[21],PCAD患者血清ZAG水平降低,ZAG水平较低的群体更有可能患有PCAD,ZAG可能是PCAD患者的潜在诊断生物标志物,并且ZAG与年龄、性别、BMI、FBG、TC、HDL、肌酐和尿素等指标联合具有更高的诊断效能。MCP-1水平在PCAD组最高,对照组最低,提示在PCAD患者中,MCP-1大量诱导巨噬细胞的渗入,炎症反应更为活跃。MCP-1通过PI3K/Akt介导的ATP结合盒转运蛋白A1、ATP结合盒转运体G1和B类I型清道夫受体表达削弱HepG2细胞中逆向胆固醇转运能力,在动脉粥样硬化的多个阶段起着至关重要的作用^[24,25]。

本研究经相关性分析后发现MCP-1与HDL呈正相关,ZAG与HDL呈负相关,这是因为二者对脂质代谢的影响机制不同,ZAG通过负调节TNF- α 来减轻炎症,加速脂解,促进增殖和减少细胞凋亡^[26,27],故推测ZAG可能通过抗炎、抗泡沫细胞形成而对PCAD发挥影响。MCP-1加速炎症细胞向内膜的迁移,加剧动脉粥样硬化。MCP-1可能会协同HDL的炎症反应,促进HDL氧化从而形成软斑块,加速血管疾病发生及病程进展,增加PCAD发生的风险^[28,29]。多因素Logistic回归分析显示年龄、吸烟史、冠心病家族史、HDL及ZAG水平降低、TG及MCP-1水平升高是PCAD的独立危险因素。PCAD的危险因素错综复杂,既往荟萃分析结果显示年轻人的糖尿病,冠心病家族史,血脂异常,吸烟和高血压是冠心病危险因素^[30],本研究结果也与之相符。

综上所述,血清MCP-1、ZAG水平与HDL具有密切的相关性,MCP-1水平的升高及ZAG水平的降低可能促进PCAD发生和发展,二者均为PCAD发生的危险因素,或可作为PCAD的早期诊断指标和治疗的靶点。

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