· 临床研究 ·

健康成人颈动脉斑块与肾功能增龄性下降的相关性

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【摘 要】 **目的** 探讨健康成人颈动脉斑块(CP)与肾功能增龄性下降的相关性。**方法** 回顾性分析 2016 年 1 月至 2019 年 1 月在复旦大学附属华山医院老年科就诊及体检中心体检的 449 例受试者的临床资料,收集基线时 CP、肾功能、血压、血糖、血脂等临床指标,并定期随访 CP、肾功能,随访至 2023 年 6 月结束。根据入组时的年龄将患者分为年轻组(<60 岁,n=286)和老年组(≥60 岁,n=163);根据入组时是否存在 CP,将受试者分为 CP 组(n=153)和非 CP 组(n=296);根据基线、随访后 CP 的变化情况将患者分为:组 1(入组、随访后均没有斑块,n=244),组 2(入组时没有,随访后新发斑块,n=52),组 3(入组时有斑块,随访后斑块消失,n=26)和组 4(入组、随访后均有斑块,n=127)。采用 SPSS 26.0 统计软件进行数据分析。根据数据类型,分别采用 t 检验、单因素方差分析、Mann-Whitney U 检验或 χ^2 检验进行组间比较。采用 logistic 回归模型分析 CP 的影响因素。**结果** 与年轻组相比,老年组的 CP 发生率增加(53.4%和 25.2%);收缩压(SBP)、胆固醇(CHO)、甘油三酯(TG)、低密度脂蛋白胆固醇(LDL-C)、尿酸(UA)及糖化血红蛋白(HbA1c)水平增高;血红蛋白(Hb)、基线肾小球滤过率(eGFR)水平降低,差异均有统计学意义(P<0.05)。与入组时无 CP 组相比,CP 组的年龄、SBP、CHO、TG、LDL-C 及 UA 水平增加;高密度脂蛋白胆固醇(HDL-C)及基线 eGFR 水平降低,差异均有统计学意义(P<0.05)。随访结束后,共有 52 例(11.6%)受试者新发 CP,26 例(5.6%)受试者 CP 消失。与组 1 相比,组 2 的基线 eGFR 水平降低,eGFR 年均下降值增高;年龄、SBP、CHO、TG、LDL-C、UA、HbA1c 水平增高,差异均有统计学意义(P<0.05)。与组 3 相比,组 4 的基线 eGFR 水平降低,eGFR 年均下降值增高;年龄、SBP、CHO、LDL-C、UA 水平也增高,差异均有统计学意义(P<0.05)。多因素 logistic 回归分析结果提示,年龄(OR=5.098,95%CI 1.125~8.718;P<0.001)、SBP(OR=1.986,95%CI 1.019~3.289;P<0.001)、CHO(OR=1.804,95%CI 1.109~4.597;P=0.018)、LDL-C(OR=1.814,95%CI 1.010~1.964;P=0.031)、基线 eGFR(OR=0.974,95%CI 0.907~0.985;P<0.001)及 eGFR 年均下降值(OR=1.228,95%CI 1.014~1.572;P=0.016)都是 CP 存在的独立危险因素。**结论** 健康成人的肾功能存在增龄性下降,eGFR 水平及其下降速率是 CP 存在的独立危险因素。增龄过程中,积极管理血压、血脂以外,定期对肾功能进行监测,有助于预防动脉粥样硬化的发生与发展。

【关键词】 颈动脉斑块;肾小球滤过率;增龄
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Correlation of carotid plaque and age-related decline of renal function in healthy adults

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【**Abstract**】 **Objective** To investigate the correlation between carotid plaque (CP) and age-related decline of renal function in healthy individuals. **Methods** A retrospective cohort study was conducted on 449 subjects who visited Department of Geriatrics or took physical examination in Physical Examination Center in Huashan Hospital Affiliated to Fudan University from January 2016 to January 2019. Their clinical data, including CP, renal function, blood pressure, and blood glucose and lipids were collected at baseline, and CP and renal function were detected regularly during the follow-up, which was completed in June 2023. According to their age at baseline, they were divided into a young group (<60 years, n=286) and an elderly group (≥60 years, n=163). Subjects were divided into CP group (n=153) and non-CP group (n=296) according to whether CP was present at the time of enrollment. Based on the changes in CP at baseline and follow-up, they were assigned into four groups: Group 1 (without CP at baseline or follow-up, n=244), Group 2 (without CP at baseline but newly developed at follow-up, n=52), Group 3 (with CP at baseline but disappeared CP at follow-up, n=26) and Group 4 (with CP both at baseline and follow-up, n=127). SPSS statistics 26.0 was used for data analysis. Depending on data

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type, student's *t* test, one-way analysis of variance, Mann-Whitney *U* test or *Chi*-square test was used for intergroup comparison. Logistic regression model was employed to identify the influencing factors for CP. **Results** The elderly group had significantly higher incidence of CP (53.4% *vs* 25.2%), increased systolic blood pressure (SBP), and elevated levels of cholesterol (CHO), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), uric acid (UA) and glycosylated hemoglobin A1c (HbA1c), but lower hemoglobin (Hb) level and decreased estimated glomerular filtration rate at baseline (baseline-eGFR), when compared with the young group ($P<0.05$). Advanced age, increased SBP, higher levels of CHO, TG, LDL-C and UA, but lower high-density lipoprotein-cholesterol (HDL-C) level and baseline-eGFR were observed in the participants with CP at baseline than those without CP ($P<0.05$). At the end of follow-up, 52 participants developed new plaques (11.6%) and 26 had plaque disappeared (5.6%). Compared with Group 1, baseline-eGFR was lower, its annual decline was increased, older age, higher SBP, and elevated CHO, TG, LDL-C, UA and HbA1c levels were observed in Group 2 ($P<0.05$). Similarly, lower baseline-eGFR level, obvious increase of annual decline of eGFR, older age, higher SBP, and raised CHO, LDL-C and UA were found in Group 4 when compared with Group 3 ($P<0.05$). Multivariate logistic regression analysis showed that age ($OR=5.098$, 95%*CI* 1.125-8.718; $P<0.001$), SBP ($OR=1.986$, 95%*CI* 1.019-3.289; $P<0.001$), CHO ($OR=1.804$, 95%*CI* 1.109-4.597; $P=0.018$), LDL-C ($OR=1.814$, 95%*CI* 1.010-1.964; $P=0.031$), baseline-eGFR ($OR=0.974$, 95%*CI* 0.907-0.985; $P<0.001$) and the annual decline of eGFR ($OR=1.228$, 95%*CI* 1.014-1.572; $P=0.016$) were independent risk factors for CP. **Conclusion** There is an age-related decline in renal function in healthy adults, and eGFR and its annual decline are independent risk factor for CP. In the process of aging, besides active control for blood pressure and blood lipids, regular monitoring of renal function is helpful to prevent the occurrence and development of atherosclerosis.

【Key words】 carotid plaque; glomerular filtration rate; aging

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颈动脉粥样硬化(carotid atherosclerosis, CAS)是广泛性动脉粥样硬化的重要指标,而颈动脉斑块(carotid plaque, CP)被认为是动脉粥样硬化和心血管事件的替代标志物[1]。CP在反映CAS的程度和识别亚临床血管疾病方面具有较高的敏感性,是发生心血管疾病(cardiovascular disease, CVD)的重要危险因素[2]。

与CAS相关的传统危险因素包括年龄、男性、吸烟、血脂异常、高血压和糖尿病等。近年来,慢性肾脏疾病(chronic kidney disease, CKD)也被认为是CVD发展的另一个促进因素[3],但这些研究大多是在CKD人群中进行[3,4]。伴随着增龄,肾功能存在一定的生理性下降,但这种肾功能的增龄性下降是否也是动脉粥样硬化的独立危险因素仍存在争议[5]。因此,本研究将探讨健康人群中CP的发生、发展情况及危险因素,尤其关注肾功能增龄性下降与CP之间的相关性。

1 对象与方法

1.1 研究对象

回顾性分析2016年1月至2019年1月于复旦大学附属华山医院老年医学科就诊及体检中心体检的成年人的临床资料。纳入标准:(1)年龄≥30岁;(2)肾小球滤过率(glomerular filtration rate, GFR)≥60 ml/(min·1.73 m^2);(3)既往无呼吸、循环、消化、神经、内分泌、风湿免疫、泌尿系统疾病,无恶性肿瘤及慢性感染病史;(4)未使用任何降脂药物。排除标准:(1)伴有其他系统慢性疾病或恶性肿瘤、慢性传染病;(2)伴有精神类疾病或意识障碍;(3)任何原因无法配合随访。最终纳入449例符合标准的受试者,收集基线及随访期间的相关临床指标,随访至2023年6月结束。根据入组时的年龄分为年轻组(<60岁,286例)和老年组(≥60岁,163例);根据入组时是否存在CP,将受试者分为CP组($n=153$)和非CP组($n=296$);根据随访后斑块的变化情况,将受试者分为四组:组1(入组和随访后均没有斑块,$n=244$),组2(入组时没有,随访后新发斑块,$n=52$),组3(入组时有斑块,随访后斑块消失,$n=26$)和组4(入组时和随访后均有斑块,$n=127$)。本研究经复旦大学华山医院医学伦理委员会批准(RIN S20-024),并按照《赫尔辛基宣言》及其后续修正案中的伦理原则进行,受试者或家属对研究内容知情并签署知情同意书。

1.2 方法

1.2.1 一般资料与相关实验室指标 收集受试者的体质量指数(body mass index, BMI)、收缩压(systolic blood pressure, SBP)、舒张压(diastolic blood pressure, DBP)、血红蛋白(hemoglobin, Hb)、白蛋白(albumin, Alb)、空腹血糖(fasting blood sugar, FBG)、胆固醇(cholesterol, CHO)、甘油三酯(triglyceride, TG)、低密度脂蛋白胆固醇(low-density lipoprotein cholesterol, LDL-C)、高密度脂蛋白胆固醇(high-density lipoprotein cholesterol, HDL-C)、尿酸(uric acid, UA)、血肌酐

(serum creatinine, SCr)和糖化血红蛋白(glycosylated hemoglobin A1c, HbA1c)水平资料。每年定期随访血肌酐,使用慢性肾脏疾病流行病学协作(chronic kidney disease-epidemiology collaboration equation, CKD-EPI)方程来估计肾小球滤过率(estimated glomerular filtration rate, eGFR)[6],并根据每年的 eGFR 计算 eGFR 年均下降值。eGFR 年均下降值 = (基线时 eGFR−随访末次 eGFR)/随访年限。

1.2.2 颈动脉斑块测定 通过超声检测仪(Aplio500,东芝医疗系统,东京,日本)对颈动脉斑块进行测定。颈动脉内中膜厚度(carotid-intimedia thickness, c-IMT)测量为动脉壁的内膜管腔线和中膜外膜边界之间的距离。颈动脉斑块被定义为侵犯动脉腔内至少 0.5 mm 或周围 c-IMT 值的 50% 的病灶结构,或显示 c-IMT>1.5 mm[2][7]。每次检查均由 2~3 名固定的经验丰富的超声医师进行。

1.3 统计学处理

采用 SPSS 26.0 统计软件进行数据分析。符合正态分布的计量资料以均数±标准差($\bar{x}\pm s$)表示,组间比较采用 t 检验;不符合正态分布的计量资料采用中位数(四分位数间距)[$M(Q_1, Q_3)$]表示,组间比较采用 Mann-Whitney U 检验。计数资料以例数(百分率)表示,组间比较采用 χ^2 检验或 Fisher 精确检验。采用多因素 logistic 回归模型分析影响颈动脉斑块的危险因素。所有数据分析均采用双尾检验,$P<0.05$ 为差异有统计学意义。

2 结 果

2.1 年轻组与老年组基线资料比较

研究共纳入 449 例受试者,年龄(58.01±10.91)岁,其中男性 309 例(68.8%)。与年轻组相比,老年组的 CP 发生率增高;SBP、CHO、TG、LDL-C、UA 及 HbA1c 水平增高;Hb、基线 eGFR 水平降低,差异均有统计学意义($P<0.05$;表 1)。

2.2 颈动脉斑块组与无颈动脉斑块组基线资料比较

449 例受试者中,153 例入组时存在 CP(34.1%)。与无 CP 组比较,CP 组的年龄、SBP、UA、CHO、TG、LDL-C 的水平均升高,HDL-C 水平和基线 eGFR 降低,差异均有统计学意义($P<0.05$;表 2)。

2.3 四组患者临床资料比较

根据受试者入组和随访时 CP 的变化情况将受试者分为四组。与组 1 比较,组 2、组 4 受试者基线 eGFR 水平降低,eGFR 年均下降值增高;年龄、SBP、CHO、TG、LDL-C、UA、HbA1c 水平增高;组 3 受试者 HbA1c 水平升高。与组 2 比较,组 3 受试者基线 eGFR 水平增高,eGFR 年均下降值降低;年龄、CHO、UA 水平降低,HDL 水平增高;组 4 受试者 eGFR 年均下降值降低。与组 3 比较,组 4 受试者基线 eGFR 水平降低,eGFR 年均下降值增高;年龄、SBP、DBP、CHO、LDL-C 及 UA 水平增高,差异均有统计学意义($P<0.05$;表 3)。

2.4 颈动脉斑块危险因素的多因素 logistic 回归分析

以随访后受试者是否存在 CP 为因变量,纳入单因素分析中有意义的变量及临床上可能影响结局的变量,包括基线年龄、BMI、SBP、DBP、Hb、Alb、CHO、TG、LDL-C、HDL-C、UA、FBG、HbA1c、基线 eGFR 及 eGFR 年均下降值为自变量,进行多因素 logistic 回归分析。结果提示:年龄、SBP、CHO、LDL-C 和基线 eGFR、eGFR 年均下降值均是 CP 存在的独立危险因素($P<0.05$;表 4)。

表 1 年轻组与老年组基线资料比较

Table 1 Comparison of baseline data between young group and elderly group

Item	Young group(n=286)	Elderly group(n=163)	P value
Male[n(%)]	203(71.0)	106(65.0)	<0.001
Physical examination($\bar{x}\pm s$)			
BMI(kg/m^2)	24.21±4.53	24.02±3.46	0.471
SBP(mmHg)	130.23±13.29	138.25±15.36	<0.001
DBP(mmHg)	78.25±9.43	80.57±10.65	0.162
Laboratory examination[$M(Q_1, Q_3)$]			
Hb(g/L)	152.01(145.12,161.24)	146.15(136.35,157.67)	0.032
Alb(g/L)	47.46(46.29,49.13)	46.24(44.09,47.36)	0.371
CHO(mmol/L)	4.91(4.32,5.48)	5.21(4.33,5.84)	0.034
TG(mmol/L)	1.44(0.94,2.24)	1.61(0.89,2.31)	<0.001
HDL-C(mmol/L)	1.50(1.31,1.90)	1.42(1.21,1.92)	0.391
LDL-C(mmol/L)	2.91(2.43,3.58)	3.22(21.5,3.92)	0.005
UA(μmol/L)	0.37(0.31,0.42)	0.47(0.30,0.52)	<0.001
FBG(mmol/L)	5.41(5.06,6.82)	5.52(5.17,6.92)	0.282
HbA1c(%)	5.71(5.31,6.54)	5.94(5.71,6.82)	0.018
Baseline-eGFR[ml/(min·1.73 m^2), $M(Q_1, Q_3)$]	95.94(80.62,101.87)	87.46(74.82,96.47)	<0.001
Carotid plaque[n(%)]	72(25.2)	81(53.4)	<0.001

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; Hb: hemoglobin; Alb: albumin; CHO: cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; UA: uric acid; FBG: fasting blood glucose; HbA1c: glycosylated hemoglobin A1c; eGFR: estimated glomerular filtration rate. 1 mmHg=0.133 kPa.

表2 颈动脉斑块组与非颈动脉斑块组基线资料比较

Table 2 Comparison of baseline data between non-CP group and CP group

Item	Non-CP group(n=296)	CP group(n=153)	P value
Male[n(%)]	227(76.7)	108(70.6)	0.292
Age(years, $\bar{x}\pm s$)	54.34±3.56	62.09±4.21	<0.001
Physical examinationr($\bar{x}\pm s$)			
BMI(kg/m^2)	24.14±3.45	24.26±4.05	0.671
SBP(mmHg)	130.43±14.35	137.26±15.65	<0.001
DBP(mmHg)	80.46±9.72	81.35±10.08	0.391
Laboratory examination[$M(Q_1, Q_3)$]			
Hb(g/L)	147.21(142.04,160.25)	148.26(137.19,159.24)	0.114
Alb(g/L)	46.12(45.09,49.14)	46.45(45.16,48.67)	0.281
UA(μmol/L)	0.35(0.29,0.39)	0.46(0.31,0.51)	0.006
CHO(mmol/L)	4.66(4.14,5.35)	5.21(4.19,5.95)	0.013
TG(mmol/L)	1.42(0.94,2.13)	1.62(0.89,2.33)	0.016
HDL-C(mmol/L)	1.47(1.03,1.85)	1.36(1.04,1.95)	0.018
LDL-C(mmol/L)	2.65(2.29,3.36)	3.15(2.46,3.97)	0.004
FBG(mmol/L)	5.41(5.05,6.21)	5.53(5.11,6.23)	0.460
HbA1c(%)	5.52(5.15,6.42)	5.66(5.22,6.81)	0.361
Baseline-eGFR[ml/(min·1.73 m^2), $M(Q_1, Q_3)$]	95.74(81.57,100.61)	89.81(74.46,101.88)	<0.001

BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; Hb: hemoglobin; Alb: albumin; UA: uric acid; CHO: cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; FBG: fasting blood glucose; HbA1c: glycosylated hemoglobin A1c; eGFR: estimated glomerular filtration rate. 1 mmHg=0.133 kPa.

表3 四组受试者临床资料比较

Table 3 Comparison of clinical data between four groups

Item	Group 1(n=244)	Group 2(n=52)	Group 3(n=26)	Group 4(n=127)
Male[n(%)]	178(73.0)	44(84.6)	22(84.6)	94(74.0)
Age(years, $\bar{x}\pm s$)	58.03±3.67	62.25±6.57*	59.35±4.58$^\#$	63.14±4.26$^{*\triangle}$
Physical examination($\bar{x}\pm s$)				
BMI(kg/m^2)	23.91±3.65	23.82±4.10	24.63±3.78	24.08±4.36
SBP(mmHg)	130.24±13.25	134.09±14.08*	132.21±14.16	136.27±15.67$^{*\triangle}$
DBP(mmHg)	80.24±9.51	80.04±10.67	78.19±10.09	82.36±10.36$^\triangle$
Laboratory examination[$M(Q_1, Q_3)$]				
Hb(g/L)	150.01(142.23,159.78)	150.75(148.57,161.05)	147.93(142.15,155.03)	147.75(137.06,159.43)
Alb(g/L)	47.14(44.75,49.10)	46.35(43.67,48.05)	45.11(44.05,46.84)	46.04(44.68,48.25)
UA(μmol/L)	0.36(0.30,0.41)	0.41(0.31,0.50)*	0.35(0.31,0.43)$^\#$	0.42(0.30,0.51)$^{*\triangle}$
CHO(mmol/L)	4.92(4.35,5.48)	5.13(4.22,5.87)*	4.99(4.48,6.51)$^\#$	5.19(4.26,5.95)$^{*\triangle}$
TG(mmol/L)	1.47(0.94,2.22)	1.62(0.99,2.40)*	1.52(0.83,2.36)	1.56(0.89,2.31)*
HDL-C(mmol/L)	1.51(1.15,1.92)	1.43(1.06,1.90)	1.53(1.32,2.02)$^\#$	1.48(1.23,1.95)
LDL-C(mmol/L)	2.99(2.47,3.49)	3.15(2.41,3.43)*	3.09(2.59,3.86)	3.20(2.43,3.91)$^{*\triangle}$
FBG(mmol/L)	5.42(5.01,6.14)	5.43(5.15,6.22)	5.42(4.61,5.63)	5.53(5.12,6.24)
HbA1c(%)	5.52(5.70,6.41)	5.81(5.54,6.55)*	5.70(5.61,6.12)*	5.81(5.62,6.41)*
Baseline-eGFR[ml/(min·1.73m^2), $M(Q_1, Q_3)$]	94.56(87.62,102.82)	86.45(81.57,100.61)*	91.96(74.46,101.88)$^\#$	84.84(72.82,98.78)$^{*\triangle}$
Annual decline of eGFR[ml/(min·1.73m^2), $M(Q_1, Q_3)$]	0.91(0.54,2.12)	2.51(1.56,4.21)*	1.02(0.48,3.51)$^\#$	1.25(0.76,2.81)$^{*\#\triangle}$

BMI: body mass index; SBP: systolic blood pressure; DBP:diastolic blood pressure; Hb: hemoglobin; Alb: albumin; UA: uric acid; CHO: cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; FBG: fasting blood glucose; HbA1c: glycosylated hemoglobin A1c; eGFR: estimated glomerular filtration rate. 1 mmHg=0.133 kPa. Compared with group 1, $^*P<0.05$; Compared with group 2, $^\#P<0.05$; compared with group 3, $^\triangle P<0.05$.

表4 颈动脉斑块危险因素的多因素 logistic 回归分析

Table 4 Multivariate logistic regression analysis of risk factors of carotid plaque

Factor	B	SE	$Wald\ \chi^2$	OR	95%CI	P value
Age	0.795	0.154	9.871	5.098	1.125-8.718	<0.001
SBP	0.947	0.401	9.546	1.986	1.019-3.289	<0.001
CHO	0.780	0.302	7.186	1.804	1.109-4.597	0.018
LDL-C	0.813	0.524	6.794	1.814	1.010-1.964	0.031
Baseline-eGFR	0.901	0.301	9.253	0.974	0.907-0.985	<0.001
Annual decline of eGFR	0.912	0.398	9.472	1.228	1.014-1.572	0.016

SBP: systolic blood pressure; CHO: cholesterol; LDL-C: low-density lipoprotein cholesterol; eGFR: estimated glomerular filtration rate.

3 讨 论

本研究结果提示,健康成人中颈动脉斑块的发生率约为 34.1%,斑块的发生与年龄相关。伴随着增龄,肾功能存在生理性下降,但下降速率因人而异,eGFR 年均下降值为[(1.31±1.02) ml/(min · 1.73m^2)]。除了年龄、血压、血脂等传统危险因素,基线 eGFR 水平、eGFR 下降速率也是 CP 存在的独立危险因素,进一步提示肾功能的增龄性下降与颈动脉斑块存在一定的相关性。

CP 的无创评估是检测和量化 CAS 的可靠方法[6-8]。CP 的存在不仅表明了其他区域的动脉粥样硬化斑块的共存,而且还预示了斑块的总体负荷[8]。研究表明,CP 的存在和数量与心血管病的风险增加密切相关[7],具有较强的预测能力[9]。

多种因素影响 CAS,包括遗传、高血压、糖尿病、内皮功能障碍、氧化应激和炎症等,这些在 CKD 患者中经常观察到[10]。事实上,CKD 和动脉硬化及 CVD 的关系近年来获得了大量关注,但多集中在特定的 CKD 人群[11]。然而,在无 CKD 的健康人群中,eGFR 与 CAS 之间的关系研究很少,结果存在一定的争议。成年人在 30 岁以后就开始出现与增龄相关的肾功能下降[12]。Takahashi 等[13,14]认为,在健康成年人中,肾功能与早期动脉硬化之间几乎没有关联。另一项使用胱抑素 C 作为肾功能评价指标的研究表明,肾功能与 c-IMT 和 CP 显著相关[15]。本研究结果提示伴随增龄,eGFR 存在一定的增龄性下降,eGFR 的增龄性下降及其下降速率是 CP 发生、发展的另一个独立危险因素,并可能促进动脉粥样硬化及 CVD 的发生。

血压和脂质代谢异常是 CAS 的高危因素[16,17],本研究结果提示,即使在无高血压及高脂血症病史的健康成人中,LDL-C 和 SBP 水平的升高也显著增加 CP 的风险。但本研究未发现 BMI、Alb、Hb、血糖、UA 与 CP 的独立相关性,提示这些因素在正常参考范围内可能不会影响 CP 的发生、发展。

本研究存在一些局限性。首先,本研究为单中心研究,研究人群样本量较少,对证实 eGFR 下降与 CP 之间的因果关系具有挑战性。其次,本研究入组时没有检测蛋白尿,但蛋白尿是肾功能损伤及 CAS 的重要危险因素,有待于将其纳入研究。

综上所述,除了传统的血压、血脂等危险因素,肾功能的增龄性下降也是健康人群中 CP 存在的独立危险因素。增龄过程中,严格控制好血压、血脂水平以外,定期监测肾功能,包括 eGFR 及蛋白尿等,将有助于延缓肾功能的下降及 CAS 的早期防治。

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