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单核细胞计数、单核细胞/高密度脂蛋白比值对维持性血液透析患者腹主动脉钙化的预测价值

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[摘　要]　**目的：**探讨单核细胞计数、单核细胞/高密度脂蛋白比值(monocyte to high density lipoprotein ratio，MHR)与维持性血液透析(maintenance hemodialysis，MHD)患者血管钙化的关系。**方法：**本研究为回顾性研究，纳入2018年10月至2019年10月在徐州医科大学附属医院血液净化中心行规律血液透析治疗的77例患者，使用腹部侧位平片检测腹主动脉钙化(abdominal aortic calcification，AAC)情况，并以此为依据分成腹主动脉钙化组及腹主动脉非钙化组，腹主动脉钙化组根据钙化评分，又分为腹主动脉钙化轻中度组和重度组，采用Spearman相关分析、ROC曲线分析探讨MHR与腹主动脉钙化及腹主动脉钙化严重程度的关系。**结果：**共纳入MHD患者77例，其中发生腹主动脉钙化患者39例，轻中度钙化患者28例，重度钙化患者11例；腹主动脉钙化组与腹主动脉非钙化组患者在年龄、单核细胞计数、血清白蛋白、MHR与甲状旁腺激素(parathyroid hormone，PTH)，合并高血压、合并心血管疾病(cardiovascular diseases，CVD)、使用活性维生素D制剂的差异均有统计学意义($P<0.05$)，其中单核细胞计数、MHR与腹主动脉钙化呈正相关($r=0.389$，$r=0.377$，$P<0.05$)，轻中度腹主动脉钙化组的MHR值低于重度腹主动脉钙化组(0.40±0.18 vs 0.62±0.31，$P<0.05$)，MHR与腹主动脉钙化严重程度有相关性($r=0.359$，$P=0.025$)。单核细胞计数预测腹主动脉钙化的ROC曲线下面积为0.725($P=0.001$)，敏感性64.1%，特异性73.7%，MHR预测腹主动脉钙化的ROC曲线下面积为0.718($P=0.001$)，敏感性46.2%，特异性89.5%。**结论：**维持性血液透析患者的腹主动脉钙化发生率较高，单核细胞计数、MHR与腹主动脉钙化相关，且MHR与腹主动脉钙化严重程度相关，可用于以预测腹主动脉钙化的发生。

[关键词]　血液透析；单核细胞计数；单核细胞/高密度脂蛋白比值；血管钙化

Predictive value of monocytes count and monocyte to high-density lipoprotein ratio for abdominal aortic calcification in maintenance hemodialysis patients

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Abstract　**Objective:** To explore the association of monocyte count, monocyte to high-density lipoprotein ratio (MHR)

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and vascular calcification in patients with maintenance hemodialysis (MHD). **Methods:** This study is a retrospective study. A total of 77 patients who underwent regular hemodialysis at the Blood Purification Center of the Affiliated Hospital of Xuzhou Medical University from October 2018 to October 2019 were determined abdominal aortic calcification (AAC) using abdominal plain radiographs. According to the results of this examination, patients were divided into an abdominal aortic calcification group and an abdominal aortic non-calcified group, then abdominal aortic calcification group was divided into abdominal aortic calcification mild and moderate group and severe group according to calcification score. The relationship between MHR and abdominal aortic calcification and the severity of abdominal aortic calcification was studied by Spearman correlation analysis and ROC curve analysis. **Results:** A total of 77 MHD patients were included in this study, including 39 cases of abdominal aortic calcification, 28 cases of mild to moderate calcification, 11 cases of severe calcification. In terms of age, monocyte count, serum albumin, MHR, parathyroid hormone (PTH), hypertension, cardiovascular disease, and the use of active vitamin D preparations was statistically significant (P<0.05). Monocytes and MHR were positively correlated with abdominal aortic calcification (r=0.389, r=0.377, P<0.05). The MHR value in the mild to moderate abdominal aortic calcification group was lower than that in the severe abdominal aortic calcification group (0.40±0.18 vs 0.62±0.31, P<0.05), and MHR was correlated with the severity of abdominal aortic calcification (r=0.359, P=0.025). The area under ROC curve for monocytes was 0.725 in abdominal aortic calcification (P=0.001), sensitivity was 64.1%, and specificity was 73.7%. The area under ROC curve for MHR was 0.718 in abdominal aortic calcification (P=0.001), sensitivity was 46.2%, and specificity was 89.5%. **Conclusion:** The incidence of abdominal aortic calcification is higher in patients with maintenance hemodialysis. Monocytes and MHR are associated with abdominal aortic calcification and MHR is related to the severity of abdominal aortic calcification, which can predict the occurrence of abdominal aortic calcification.

Keywords maintenance hemodialysis; monocyte count; monocyte to high density lipoprotein ratio; vascular calcification

近年来，维持性血液透析患者(maintenance hemodialysis，MHD)发生血管钙化受到广泛关注[1]。血管钙化在慢性肾脏病患者(chronic kidney disease，CKD)中很常见[2]，其发病机制主要与血管平滑肌细胞(vascular smooth muscle cells，VSMC)的表型转化、矿物质代谢紊乱等密切相关[3]。氧化应激在CKD诱发的血管钙化早期发病机制中具有重要作用[4]，且炎症状态和氧化应激增加可促使VSMCs钙化[5]，血管钙化一旦出现，目前尚无有效治疗手段，重点在于早期发现、早期预防，因此能早期预测血管钙化发生的指标一直是研究热点。单核细胞/高密度脂蛋白比值(monocyte to high density lipoprotein ratio，MHR)作为近年来新发现的一个炎症标志物，与动脉粥样硬化相关，可早期预测心血管疾病(cardiovascular diseases，CVD)的发生[6]。国内外研究尚无MHR与MHD患者血管钙化的关系等方面的研究。MHR作为一个与动脉粥样硬化明确相关的炎症指标，本研究拟探讨MHR与MHD患者血管钙化的关系。

1 对象与方法

1.1 对象

回顾性分析2018年10月至2019年10月在徐州医科大学附属医院血液净化中心行规律血液透析治疗的77例患者，纳入标准：年龄≥18周岁；维持性血液透析≥6个月；均规律行血液透析治疗，3次/周，4 h/次，使用自体动静脉瘘和碳酸氢盐透析液，血流量为200~300 mL/min，透析流量为500 mL/min。排除标准：自身免疫性疾病；结缔组织疾病；急、慢性感染者；使用过腹膜透析；肝功能异常；肿瘤性疾病；严重血液系统疾病；甲状腺功能异常；服用免疫抑制剂。

1.2 观察指标

1.2.1 患者一般资料

记录年龄、性别、透析龄、体重指数(BMI)、收缩压、舒张压、是否有合并症(高血压、糖尿病、脑梗死、CVD等)，BMI=体重(kg)/身高的平

方(m^2)。

1.2.2 实验室检查

采集纳入患者透析前血液，统一送至徐州医科大学附属医院检验科测量，测量数据包括血红蛋白、单核细胞计数、血清白蛋白、血尿酸、总胆固醇、三酰甘油、高密度脂蛋白、脂蛋白(a)、甲状旁腺激素(parathyroid hormone，PTH)等。

1.2.3 腹主动脉钙化检测

纳入的患者均行腹部X线检查，钙化诊断标准：当腹主动脉发生钙化时，腰椎旁可见纵向线条样或条形高密度影[7]，并计算腹主动脉钙化评分(abdominal aortic calcification score，AACs)。钙化评分标准：依据Kauppila评分，血管无钙化为0分，血管壁钙化≤1/3为1分，1/3~2/3为2分，>2/3为3分，患者AACs为0~24分[8]。根据患者AACs分组，≤4分为无或轻度钙化组，5~15分为中度钙化组，AACs≥16分为重度钙化组。钙化评分由2位影像科副主任及以上医师经专业培训后进行盲法阅片和评分，评分结果不一致时重复阅片，取得一致意见后再次评分。

1.2.4 MHR测量与分析

根据腹部侧位平片结果分为腹主动脉钙化组与腹主动脉非钙化组，分别计算两组的MHR，比较两组患者MHR，采用Spearman相关分析探讨MHR与腹主动脉钙化的关系。腹主动脉钙化组根据钙化评分分为腹主动脉钙化轻中度组与重度组，采用Spearman相关分析探讨MHR与腹主动脉钙化严重程度的关系。采用ROC曲线分析探讨MHR预测腹主动脉钙化的能力，并进一步分析腹主动脉钙化的影响因素。

1.3 统计学处理

采用SPSS 25.0统计学软件进行数据分析，计量资料中符合正态分布的用均数±标准差($\bar{x}\pm s$)表示，组间比较采用t检验；符合偏态分布的用中位数(四分位数)表示，组间比较采用Mann-Whitney U检验。计数资料用例(%)表示，组间比较采用χ^2检验。采用Pearson相关分析及Spearman相关分析探讨患者各影响因素与血管钙化的相关性，ROC曲线分析各影响因素的预测价值，P<0.05为差异有统计学意义。

2 结果

2.1 MHD患者的临床基线资料

77例MHD患者中，男49例(63.64%)，女28例(36.36%)，年龄(52.12±14.43)岁，透析龄为6~156个月；有64例(83.12%)合并高血压，24例(31.17%)合并糖尿病，14例(18.18%)合并CVD，13例(33.47%)合并脑梗死。根据腹部侧位平片钙化情况分为腹主动脉钙化组(n=39)及腹主动脉非钙化组(n=38)，其中腹主动脉钙化组再分为轻中度腹主动脉钙化组(28例，36.36%)与重度腹主动脉钙化组(11例，14.29%)。

2.2 腹主动脉钙化组与腹主动脉非钙化组生化及临床资料

两组患者间年龄、单核细胞计数、血清白蛋白、PTH的差异均有统计学意义(P<0.05)。其中腹主动脉钙化组的MHR为0.46±0.24，腹主动脉非钙化组患者的MHR为0.30±0.15，差异有统计学意义(P=0.001)。两组在透析龄、性别、BMI等方面的差异无统计学意义(表1)。

2.3 合并症与用药情况

腹主动脉钙化组患者合并高血压、CVD、脑梗死，活性维生素D制剂使用比例显著高于腹主动脉非钙化组，差异有统计学意义(均P<0.05)，两组糖尿病的患病比例，使用钙剂的差异无统计学意义(P>0.05，表2)。

2.4 腹主动脉钙化与其影响因素的相关性

腹主动脉钙化与年龄、单核细胞计数、MHR、合并CVD、合并脑梗死呈正相关(均P<0.05)，与PTH呈负相关(P<0.05)，与血清白蛋白、合并高血压、合并糖尿病无相关性(P>0.05，表3)。

2.5 MHR与腹主动脉钙化严重程度

轻中度腹主动脉钙化组与重度腹主动脉钙化组患者MHR值分别为0.40±0.18，0.62±0.31，差异有统计学意义(P=0.009，P<0.05)。采用Spearman双变量相关分析发现：MHR与腹主动脉钙化严重程度有相关性(r=0.359，P=0.025)。

表1 腹主动脉钙化组与腹主动脉非钙化组的临床资料比较

Table 1 Comparison of clinical characteristics between abdominal aortic calcification group and abdominal aortic non-calcified group

项目	腹主动脉钙化组(n=39)	腹主动脉非钙化组(n=38)	P
年龄/岁	58.54 ± 11.10	45.53 ± 14.60	<0.001
透析龄/月	36 (12，60)	24 (12，60)	0.800
性别(男)/[例(%)]	26 (66.7)	23 (60.53)	0.575
BMI/($kg·m^{-2}$)	23.34 ± 2.99	22.85 ± 4.04	0.555
收缩压/mmHg	148.77 ± 31.22	142.63 ± 21.06	0.316
舒张压/mmHg	82.62 ± 14.91	83.71 ± 9.82	0.705
平均动脉压/mmHg	104.67 ± 19.28	103.35 ± 12.02	0.721
血红蛋白/($g·L^{-1}$)	103.44 ± 25.3	102.00 ± 22.65	0.794
单核细胞计数/($\times 10^{9}·L^{-1}$)	0.43 ± 0.15	0.31 ± 0.13	<0.001
血尿酸/($\mu mol·L^{-1}$)	349.62 ± 96.27	371.26 ± 101.02	0.339
肾小球滤过率/[($mL·min^{-1}·1.73\ m^{-2}$)]	6.58 (5.20，9.88)	5.76 (4.71，8.33)	0.099
白蛋白/($g·L^{-1}$)	40.63 ± 5.96	43.28 ± 5.53	0.047
空腹血糖/($mmol·L^{-1}$)	5.40 (4.54，7.08)	4.84 (4.45，5.58)	0.201
血钙/($mmol·L^{-1}$)	2.31 ± 0.24	2.23 ± 0.29	0.227
血磷/($mmol·L^{-1}$)	1.69 ± 0.32	1.72 ± 0.53	0.706
钙磷乘积/($mmol^{2}·L^{-2}$)	3.92 ± 0.94	3.89 ± 1.39	0.928
总胆固醇/($mmol·L^{-1}$)	4.19 ± 1.07	3.93 ± 0.96	0.274
三酰甘油/($mmol·L^{-1}$)	1.09 (0.76，1.63)	1.18 (0.83，1.59)	0.639
高密度脂蛋白/($mmol·L^{-1}$)	1.02 ± 0.32	1.12 ± 0.38	0.190
低密度脂蛋白/($mmol·L^{-1}$)	2.48 ± 0.78	2.16 ± 0.74	0.068
脂蛋白a/($mg·L^{-1}$)	316 (228，673)	405 (249，619)	0.603
PTH/($pg·mL^{-1}$)	165.10 (65.60，279.80)	377.30 (214.65，599.58)	0.002
MHR	0.46 ± 0.24	0.30 ± 0.15	0.001

平均动脉压=舒张压+1/3脉压。

Mean arterial pressure = diastolic blood pressure + 1/3 pulse pressure.

表2 腹主动脉钙化组与腹主动脉非钙化组的合并症与用药情况比较

Table 2 Comparison of comorbidities and the use of drugs between abdominal aortic calcification group and abdominal aortic non-calcified group

组别	n	高血压 / [例 (%)]	糖尿病 / [例 (%)]	心血管疾病 / [例 (%)]	脑梗死 / [例 (%)]	钙剂 / [例 (%)]	活性维生素 D 制剂 /[例 (%)]
钙化组	39	36 (92.31)	16 (41.03)	12 (30.77)	11 (28.21)	8 (20.51)	18 (46.15)
非钙化组	38	28 (73.68)	8 (21.05)	2 (5.26)	2 (5.26)	4 (10.53)	8 (21.05)
P		0.029	0.059	0.004	0.007	0.227	0.020

2.6 单核细胞计数、MHR预测腹主动脉钙化能力

以单核细胞计数和MHR为检验变量，有无腹主动脉钙化为状态标量，绘制ROC曲线(图1)示：ROC曲线下面积分别为0.725和0.718，均大于0.7，差异有统计学意义(P<0.05，表4)；其中单核细胞计数在0.37×10^9/L时，敏感性和特异性之和最大(敏感性64.1%，特异性73.7%)；MHR在0.439时，敏感性和特异性之和最大(敏感性46.2%，特异性89.5%)，提示单核细胞计数、MHR预测腹主动脉钙化有较大意义。

表3 钙化与其影响因素的Spearman相关分析

Table 3 Spearman correlation analysis of calcification and its influencing factors

影响因素	相关系数 (r)	P
年龄	0.443	<0.001
血清白蛋白	−0.213	0.063
单核细胞计数	0.389	<0.001
MHR	0.377	0.001
PTH	−0.352	0.002
高血压	0.249	0.029
糖尿病	0.216	0.060
心血管疾病	0.331	0.003
脑梗死	0.306	0.007

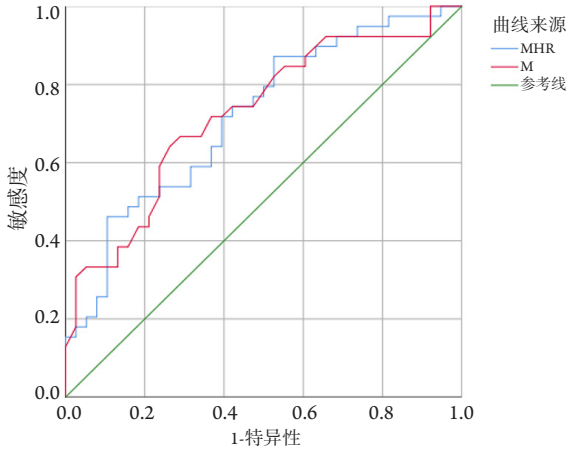

图1 单核细胞计数和MHR预测腹主动脉钙化的ROC曲线

Figure 1 ROC curve of monocyte count and MHR predicting abdominal aortic calcification

表4 单细胞计数和MHR对腹主动脉钙化的预测价值

Table 4 Predictive value of monocytes count and MHR on abdominal aortic calcification

变量	AUC	标准误	P	95%CI (L)	95%CI (U)
MHR	0.718	0.058	0.001	0.604	0.831
M	0.725	0.058	0.001	0.612	0.838

3 讨论

随着社会的发展，CKD的患病率在逐渐上升，透析逐渐成为主要治疗方式，CVD作为透析患者首要死因[9]，发病机制一直是研究热点，其中血管钙化日益引起重视。相对于普通人群，MHD患者的血管钙化发生更常见，且更严重，可作为患者独立的死亡预测因子[10]。本研究也证实：MHD患者发生血管钙化较多(39例，50.65%)。血管钙化是MHD患者CVD发生的独立危险因素，且病理机制更为复杂，其发病机制主要与VSMCs的表型转化、矿物质代谢紊乱等密切相关[3]，且非传统因素如尿毒症毒素、Klotho蛋白、胎球蛋白A、脂联素的调节等也被认为参与了MHD患者血管钙化的发生[11]。炎症与MHD患者血管钙化的发生及进展密切相关[12]。Liu等[13]认为CKD患者体内的微炎症状态通过mTORC1通道参与血管钙化的发生。一项临床研究[14]发现动脉“发炎”促进了透析儿童的血管钙化；同样有学者[15]强调慢性炎症对血管钙化的作用。以上研究表明炎症在MHD患者血管钙化的发生中有重要作用。目前检测血管钙化的方法主要有冠状动脉计算机断层扫描(computed tomography，CT)、电子束断层扫描(electron beam tomography，EBT)、腹部平片等，其中腹部侧位X线片检查简单、方便，且辐射量较低[16]。Bellasi等[17]研究发现：腹部侧位平片估算的血管钙化评分具有较好的敏感性和特异性。因此本研究选择腹部侧位平片检测腹主动脉是否发生钙化。Okuno等[18]认为腹主动脉钙化在MHD患者中普遍存在，在预测CVD病死率上有潜在价值，有可能成为预后指标。但这些影像学检查价格昂贵，在基

层医院临床应用困难，因此发现能早期预测血管钙化的发生，且简单易行、价格低廉的指标尤为重要。

MHD患者血管钙化的发生与年龄、钙磷代谢紊乱、PTH的紊乱、药物治疗、合并糖尿病、合并CVD等密切相关[19]。本研究结果显示：年龄、营养不良与腹主动脉钙化有关，但未发现高钙低磷与腹主动脉钙化有关，可能是因为选取人数较少、未考虑患者服用药物对钙磷代谢的影响等。多数研究认为PTH促进血管钙化，但本研究中腹主动脉钙化组患者的PTH水平低于非钙化组，差异有统计学意义，考虑可能与腹主动脉钙化组患者使用活性维生素D制剂、PTH水平降低有关。因为有药物干预，未来仍需扩大样本量进一步分析PTH水平与腹主动脉钙化的关系。有研究[20]认为：单核细胞衍生的巨噬细胞产生促炎细胞因子和氧化应激，促进血管平滑肌细胞发生成骨转化和矿化，血管钙化的发展与单核细胞各种亚群之间功能平衡相关。本研究也证实了这一结论：单核细胞计数与腹主动脉钙化的发生存在正相关。

Kanbay等[21]于2014年首次报道了MHR，他们对340名CKD患者进行了平均33个月的随访，首次检测了单核细胞计数与高密度脂蛋白的比值，并将其定义为MHR，首次提出MHR的升高可以成为CKD患者的心脑血管事件发生的危险因素。2019年一项随访8 159名CKD患者的横断面研究[22]证实了MHR与肾功能降低有关，并提出MHR在肾功能风险分层方面的潜在价值。MHR作为一个新的炎性指标，与C反应蛋白呈正相关[23]，与脑卒中[24]、动脉粥样硬化、高血压[25]、颈动脉内中膜厚度[26-27]及CVD的预后[28]紧密相关，但是关于MHR与MHD患者血管钙化的关系尚无报道。MHR作为一个炎性指标，我们推测MHR与MHD患者腹主动脉钙化相关。本研究发现MHR在腹主动脉钙化组和腹主动脉非钙化组存在差异，Spearman相关性分析显示MHR和腹主动脉钙化的发生呈正相关；进一步分析发现：MHR在轻中度腹主动脉钙化组与重度腹主动脉钙化组存在差异，且呈正相关，提示MHR与腹主动脉钙化的严重程度相关，并且得到MHR预测腹主动脉钙化的最佳截断值为0.439，因此推测MHR可以预测腹主动脉钙化的发生，尤其当MHR>0.439时，腹主动脉钙化发生的可能性更大，此时可以采取措施进行早期干预，延缓钙化的发展。

综上所述，本研究发现单核细胞计数、MHR与腹主动脉钙化的发生相关，且MHR与腹主动脉钙化严重程度相关，可作为腹主动脉钙化的预测指标，相较于现有的MHD患者腹主动脉钙化的预测指标，比如血清Gal-3[29]、血浆补体蛋白C3a[30]等，该指标检测方便，价格低廉，可在基层医院推广。本研究结果为探索血管钙化发生的预测指标提供了思路，但本研究为回顾性研究，选取样本量较少，可能会对一些实验结果有影响，还需要更多大样本随机对照研究来证实。

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