

## 维持性血液透析患者血清sICAM-1、sVCAM-1水平和SOD活性及其与冠状动脉钙化的关系

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**[摘要]** **目的:** 分析维持性血液透析(MHD)患者血清可溶性细胞间黏附分子1(sICAM-1)和可溶性血管细胞黏附分子1(sVCAM-1)水平及超氧化物歧化酶(SOD)活性, 探讨其与冠状动脉钙化(CAC)的关系。**方法:** 回顾性分析102例MHD患者(MHD组)的临床资料, 另招募同期接受健康体检的74名志愿者(健康体检组)。MHD组患者经多层螺旋CT(MSCT)检查行CAC分数(CACs)评定, 并将其分为无钙化组、轻度钙化组、中度钙化组和重度钙化组。比较2组研究对象一般资料和血清sICAM-1、sVCAM-1水平及SOD活性, 分析不同钙化程度组患者血清中钙(Ca)、磷(P)、甲状旁腺激素(PTH)、sICAM-1和sVCAM-1水平及SOD活性。采用Pearson相关分析法分析MHD患者血清sICAM-1和sVCAM-1水平及SOD活性与CACs的相关性。**结果:** 与健康体检组比较, MHD组患者血清sICAM-1和sVCAM-1水平均明显升高( $P<0.01$ ), SOD活性明显降低( $P<0.01$ )。与无钙化组比较, 轻度、中度和重度钙化组患者血清PTH、sICAM-1和sVCAM-1水平均明显升高( $P<0.05$ ), SOD活性均明显降低( $P<0.05$ ); 中度和重度钙化组患者血清P水平均明显升高( $P<0.05$ )。与轻度钙化组比较, 中度和重度钙化组患者血清P、PTH、sICAM-1和sVCAM-1水平均明显升高( $P<0.05$ ), SOD活性均明显降低( $P<0.05$ )。与中度钙化组比较, 重度钙化组患者血清sICAM-1、sVCAM-1和P水平均明显升高( $P<0.05$ ), SOD活性明显降低( $P<0.05$ )。MHD患者血清SOD活性与CACs呈负相关关系( $r=-0.484, P<0.01$ ), sICAM-1和sVCAM-1水平与CACs呈正相关关系( $r=0.441, P<0.01$ ;  $r=0.561, P<0.01$ )。**结论:** MHD患者血清sICAM-1和sVCAM-1水平及SOD活性异常, 并且随着SOD活性降低和sICAM-1及sVCAM-1水平升高, MHD患者的CAC程度加重。

**[关键词]** 维持性血液透析; 超氧化物歧化酶; 可溶性细胞间黏附分子1; 可溶性血管细胞黏附分子1; 冠状动脉钙化

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## Levels of sICAM-1 and sVCAM-1 and activity of SOD in serum and their relationships with coronary artery calcification in patients with maintenance hemodialysis

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**ABSTRACT Objective:** To analyze the levels of serum soluble intercellular adhesion molecule-1 (sICAM-1), soluble vascular cell adhesion molecule-1 (sVCAM-1), and superoxide dismutase (SOD) activity in the patients with maintenance hemodialysis (MHD), and to discuss their relationships with coronary artery calcification (CAC). **Methods:** The clinical materials from 102 MHD patients (MHD group) were retrospectively analyzed. Additionally, 74 volunteers underwent routine health examination at the same time (health examination group) were selected. The CAC scores (CACs) of the patients in MHD group were detected by multi-slice computed tomography (MSCT), and the patients were categorized into non-calcification group, mild calcification group, moderate calcification group, and severe calcification group. The general data and serum levels of sICAM-1, sVCAM-1, and SOD activities of the subjects in two groups were compared. The levels of calcium (Ca), phosphorus (P), parathyroid hormone (PTH), sICAM-1, sVCAM-1, and SOD activities in serum of the patients with different degrees of calcification were analyzed. Pearson's correlation analysis was used to analyze the correlations between the levels of sICAM-1, sVCAM-1, and SOD activity in serum of the MHD patients and CACs. **Results:** Compared with health examination group, the levels of sICAM-1 and sVCAM-1 in serum of the patients in MHD group were significantly increased ( $P < 0.01$ ), and the SOD activity was significantly decreased ( $P < 0.01$ ). Compared with non-calcification group, the levels of PTH, sICAM-1, and sVCAM-1 in serum of the MHD patients in mild, moderate, and severe calcification groups were significantly increased ( $P < 0.05$ ), and the SOD activities were significantly decreased ( $P < 0.05$ ); the levels of P in serum of the MHD patients in moderate and severe calcification groups were significantly increased ( $P < 0.05$ ). Compared with mild calcification group, the levels of P, PTH, sICAM-1, and sVCAM-1 in serum of the MHD patients in moderate and severe calcification groups were significantly increased ( $P < 0.05$ ), and the SOD activities significantly decreased ( $P < 0.05$ ). Compared with moderate calcification group, the levels of sICAM-1, sVCAM-1, and P in serum of the MHD patients in severe calcification group were significantly increased ( $P < 0.05$ ), and the SOD activity was significantly decreased ( $P < 0.05$ ). The SOD activity in serum of the MHD patients was negatively correlated with CACs ( $r = -0.484$ ,  $P < 0.01$ ), while the levels of sICAM-1 and sVCAM-1 were positively correlated with CACs ( $r = 0.441$ ,  $P < 0.01$ ;  $r = 0.561$ ,  $P < 0.01$ ). **Conclusion:** The levels of sICAM-1 and sVCAM-1 and activity of SOD in serum of the MHD patients are abnormal. With the decreasing of the SOD activity and increasing of the levels of sICAM-1 and sVCAM-1, the degree of CAC in the MHD patients is aggravated.

**KEYWORDS** Maintenance hemodialysis; Superoxide dismutase; Soluble intercellular adhesion molecule-1; Soluble vascular cell adhesion molecule-1; Coronary artery calcification

目前, 维持性血液透析(maintenance hemodialysis, MHD)患者数量呈逐年增加的趋势, 至2019年底, 全国共有63.3万例MHD患者<sup>[1]</sup>。随着医疗技术的发展, MHD患者的生存状况有了明显改善, 但透析患者死亡率仍是正常人群的6.5~7.9倍<sup>[2]</sup>。研究<sup>[3]</sup>显示: 血液透析患者的5年生存率仅为39.8%~60.2%。其中, 约有50%的患者因心血管疾病(cardiovascular disease, CVD)而死亡<sup>[4-5]</sup>。冠状动脉钙化(coronary artery calcification, CAC)是心血管事件发生及导致患者死亡的主要危险因素, 且MHD患者CAC的患病率为80%<sup>[6-7]</sup>。因此, 如何有效预测和评估CAC的程度, 已成为临床急需解决的难题之一。既往研究<sup>[8]</sup>发现: 冠心病患者血清超氧化物歧化酶(superoxide dismutase, SOD)活性降低与冠状动脉病变的发生发展有密切关联。CAC与可溶性细胞间黏附分子1(soluble intercellular adhesion molecule-1, sICAM-1)和可溶性血管细胞黏附分子1(soluble vascular cell adhesion molecule-1, sVCAM-1)等黏附因子有关<sup>[9-10]</sup>。但目前关于MHD患者血清sICAM-1和sVCAM-1水平及SOD活性与CAC之间相关性的研究较少。本研究通过回顾性分析MHD患者血清sICAM-1和sVCAM-1水平及SOD活性, 探讨其与CAC的关系, 为MHD患者的治疗提供参考。

## 1 资料与方法

**1.1 一般资料** 回顾性分析2019年10月—2021年12月于四川省人民医院肾脏内科就诊的102例MHD患者临床资料, 主要包括年龄、透析年限、原发病类型、糖尿病病史、高血压病史、血钙(calcium, Ca)、血磷(phosphorus, P)及甲状旁腺素(parathyroid hormone, PTH)水平。纳入标准: ①规律血液透析>6个月, 每周透析次数为3次, 透析时间为4h; ②年龄≥18周岁; ③接受多层螺旋CT(multislice helical CT, MSCT)检查; ④CT扫描资料及临床病历资料完整患者。排除标准: ①严重肝脏疾病患者; ②有明显感染者; ③活动性结核患者; ④恶性肿瘤患者; ⑤具有先天性心脏病、冠状动脉搭桥手术史和冠状动脉植入支架史及怀孕者; ⑥临床资料丢失或缺损者。将102例MHD患者纳入MHD组。纳入同期在四川省人民医院体检中心接受健康体检的74名志愿者作为健康体检组。本研究经过四川省人民医院医学伦理委

员会同意(伦理审批号: QYYKJ-2023-37)。

**1.2 MSCT观察MHD患者CAC程度** 使用德国西门子公司生产的双源CT(型号: SOMOATOM Force CT)对所有MHD患者进行MSCT扫描, 并使用Agatston钙化积分计算方法计算CAC分数(CAC score, CACs), 根据CACs评估MHD患者CAC程度: ≤10分为无钙化组( $n=33$ ); 11~100分为轻度钙化组( $n=23$ ); 101~400分为中度钙化组( $n=22$ ); >400分为重度钙化组( $n=24$ )。

**1.3 2组研究对象血清sICAM-1和sVCAM-1水平及SOD活性检测** 健康体检组研究对象体检当日和MHD组患者1周内行第2次透析治疗当日透析前, 分别抽取2组研究对象3mL外周静脉血, 注入一次性真空管内, 3000 $\text{r}\cdot\text{min}^{-1}$ 离心10min, 离心半径为10cm, 分离上层血清, 将血清置于Eppendorf管中, 置于-80℃低温保存, 避免反复冻融。采用酶联免疫吸附试验(enzyme linked immunosorbent assay, ELISA)试剂盒检测2组研究对象血清sICAM-1和sVCAM-1水平, 全自动血液分析仪检测血清SOD活性, 试剂盒均由深圳达科为生物技术有限公司提供, 按试剂盒说明书进行操作。

**1.4 统计学分析** 采用SPSS 26.0统计软件进行统计学分析。2组研究对象临床资料中的原发病类型、糖尿病病史和高血压病史为计数资料, 以例数表示, 2组间比较采用 $\chi^2$ 检验。研究对象年龄, 透析年限, 血清Ca、P、PTH、sICAM-1和sVCAM-1水平及SOD活性为计量资料, 均符合正态分布, 以 $\bar{x}\pm s$ 表示, 多组间样本均数比较采用单因素方差分析, 组间样本均数两两比较采用SNK- $q$ 检验, 2组间样本均数比较采用两独立样本 $t$ 检验。采用Pearson相关分析法分析MHD患者血清sICAM-1和sVCAM-1水平及SOD活性与CACs的相关性, 并绘制散点图。以 $P<0.05$ 为差异有统计学意义。

## 2 结果

**2.1 2组研究对象一般资料** MHD组共102例患者, 其中男性59例(57.8%), 女性43例(42.2%), 年龄42~82岁, 平均年龄(62.71±10.36)岁, 平均透析龄(59.73±24.73)个月, 原发病类型包括慢性肾小球肾炎19例(18.62%)、高血压肾病40例(39.22%)、糖尿病肾病31例(30.39%)和其他12例(11.76%), 并发高血压87例(85.29%), 并发糖尿病69例(67.65%)。健康体检组共有74名研究对象, 其中男性42名

(56.8%), 女性 32 名 (43.2%), 年龄 40~83 岁, 平均年龄 (63.31 ± 11.35) 岁。2 组研究对象年龄和性别比较差异均无统计学意义 ( $t = -0.364$ ,  $P = 0.716$  和  $\chi^2 = 0.021$ ,  $P = 0.886$ )。

**2.2 2 组研究对象血清 sICAM-1 和 sVCAM-1 水平及 SOD 活性** 与健康体检组比较, MHD 组患者血清 sICAM-1 和 sVCAM-1 水平均明显升高 ( $P < 0.01$ ), SOD 活性明显降低 ( $P < 0.01$ )。见表 1。在 MHD 患者中, 与无钙化组比较, 轻度、中度和重度钙化组患者血清 PTH、sICAM-1 及 sVCAM-1

水平均明显升高 ( $P < 0.05$ ), SOD 活性均明显降低 ( $P < 0.05$ ); 中度和重度钙化组患者血清 P 水平均明显升高 ( $P < 0.05$ )。与轻度钙化组比较, 中度和重度钙化组患者血清 P、PTH、sICAM-1 及 sVCAM-1 水平均明显升高 ( $P < 0.05$ ), SOD 活性均明显降低 ( $P < 0.05$ )。与中度钙化组比较, 重度钙化组患者血清 sICAM-1、sVCAM-1 和 P 水平均明显升高 ( $P < 0.05$ ), SOD 活性明显降低 ( $P < 0.05$ )。见表 2。

表 1 2 组研究对象血清 sICAM-1 和 sVCAM-1 水平及 SOD 活性

Tab. 1 Levels of sICAM-1 and sVCAM-1 and activities of SOD in serum of subjects in two groups ( $\bar{x} \pm s$ )

Group	n	Level of sICAM-1 [ $\rho_B / (\mu\text{g} \cdot \text{L}^{-1})$ ]	Level of sVCAM-1 [ $\rho_B / (\mu\text{g} \cdot \text{L}^{-1})$ ]	Activity of SOD [ $\lambda_B / (\text{IU} \cdot \text{L}^{-1})$ ]
Health examination	74	285.63 ± 31.65	3 018.84 ± 154.18	114.83 ± 20.95
MHD	102	432.11 ± 74.12	6 698.43 ± 206.74	79.86 ± 10.34
t		15.967	129.204	-14.595
P		<0.01	<0.01	<0.01

表 2 不同钙化程度组 MHD 患者血清 P、Ca、PTH、sICAM-1 和 sVCAM-1 水平及 SOD 活性

Tab. 2 Levels of P, Ca, PTH, sICAM-1, and sVCAM-1 and activities of SOD in serum of MHD patients in different degrees of calcification groups ( $\bar{x} \pm s$ )

Group	n	Level of P [ $c_B / (\text{mmol} \cdot \text{L}^{-1})$ ]	Level of Ca [ $c_B / (\text{mmol} \cdot \text{L}^{-1})$ ]	Level of PTH [ $\rho_B / (\text{ng} \cdot \text{L}^{-1})$ ]	Activity of SOD [ $\lambda_B / (\text{IU} \cdot \text{L}^{-1})$ ]	Level of sICAM-1 [ $\rho_B / (\mu\text{g} \cdot \text{L}^{-1})$ ]	Level of sVCAM-1 [ $\rho_B / (\mu\text{g} \cdot \text{L}^{-1})$ ]
Non-calcification	33	1.68 ± 0.08	2.20 ± 0.10	176.88 ± 10.51	94.52 ± 7.21	316.87 ± 61.36	5 456.95 ± 168.73
Mild calcification	23	1.68 ± 0.09	2.21 ± 0.12	346.57 ± 15.10*	83.49 ± 6.14*	409.63 ± 63.42*	6 534.79 ± 170.67*
Moderate calcification	22	2.06 ± 0.09* <sup>△</sup>	2.22 ± 0.07	473.23 ± 11.55* <sup>△</sup>	72.25 ± 5.98* <sup>△</sup>	467.39 ± 70.09* <sup>△</sup>	7 295.52 ± 179.63* <sup>△</sup>
Severe calcification	24	2.30 ± 0.17* <sup>△</sup> #	2.23 ± 0.82	475.50 ± 12.12* <sup>△</sup>	63.22 ± 4.51* <sup>△</sup> #	579.74 ± 79.85* <sup>△</sup> #	8 014.96 ± 183.27* <sup>△</sup> #

\* $P < 0.05$  compared with non-calcification group; <sup>△</sup> $P < 0.05$  compared with mild calcification group; # $P < 0.05$  compared with moderate calcification group.

**2.3 MHD 患者血清 SOD 活性、sICAM-1 和 sVCAM-1 水平与 CACs 相关性分析** MHD 患者血清 SOD 活性与 CACs 呈负相关关系 ( $r = -0.484$ ,  $P < 0.01$ ); sICAM-1 和 sVCAM-1 水平与 CACs 呈正相关关系 ( $r = 0.441$ ,  $P < 0.01$ ;  $r = 0.561$ ,  $P < 0.01$ )。见图 1~3。

### 3 讨论

MHD 患者多伴有不同程度肾性骨代谢异常综合征, 其重要的临床血清学表现为骨代谢紊乱、高钙血症和血管钙化等<sup>[11]</sup>。研究<sup>[12-13]</sup>显示: 全球 MHD 患者中 CAC 的患病率为 24%~93%, 且 CVD 是导致 MHD 患者死亡的主要原因之一。CAC 导致患 CVD 的风险增加, 而 CAC 的严重程度是血液透

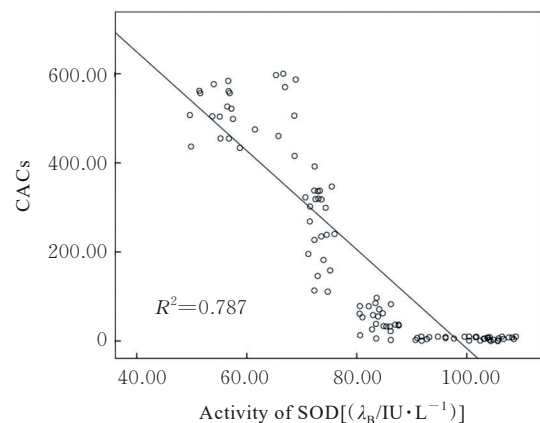


图 1 MHD 患者血清 SOD 活性与 CACs 相关性散点图

Fig. 1 Scatter plot diagram of correlation between serum activity of SOD and CACs of MHD patients

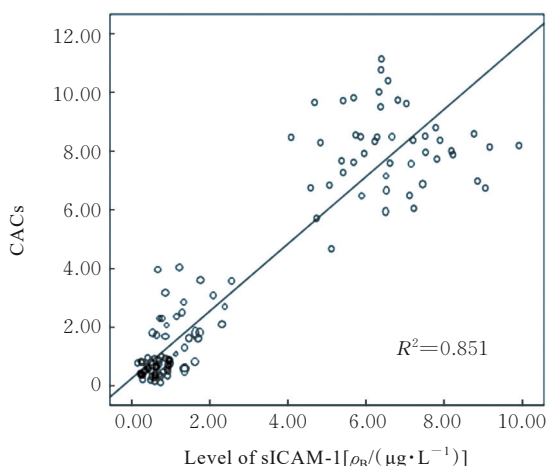


图2 MHD患者血清sICAM-1水平与CACs相关性散点图

Fig. 2 Scatter plot diagram of correlation between serum level of sICAM-1 and CACs of MHD patients

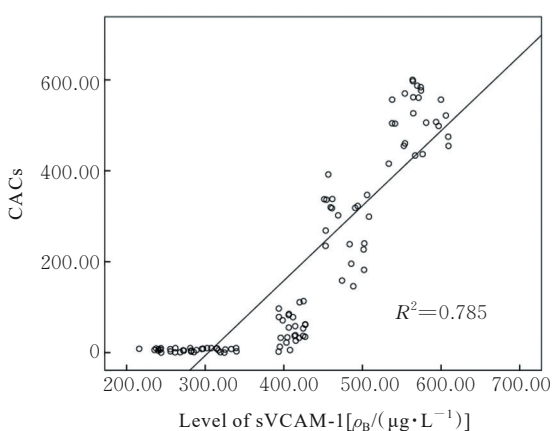


图3 MHD患者血清sVCAM-1水平与CACs相关性散点图

Fig. 3 Scatter plot diagram of correlation between serum level of sVCAM-1 and CACs of MHD patients

析患者全因死亡率的预测因素<sup>[14]</sup>。CAC属于机体退化性和被动性终末过程,但近年来研究<sup>[15-16]</sup>发现:CAC类似于骨发育及软骨形成过程,可进行治疗、预防和调节。因此,有效评估CAC程度对MHD患者具有重要意义。

钙化存在是冠状动脉粥样硬化的重要标准,而粥样斑块形成与氧化应激有密切关联,SOD是氧化应激的指标之一<sup>[17]</sup>。正常人体内的活性氧自由基有氧代谢过程功能正常,但疾病的存在破坏了体内平衡,激活抗氧化系统,会大量消耗SOD等抗氧化防御酶<sup>[18-19]</sup>。SOD可有效清除体内的脂质过氧化物,减少过氧化物对细胞的损害,从而保护血管内皮,防治冠状动脉粥样硬化<sup>[20-21]</sup>。sICAM-1是

可溶性免疫球蛋白超家族黏附分子成员之一,在正常人外周血及体液中呈低表达或无表达,当出现脂质代谢异常时,血清sICAM-1水平升高<sup>[22]</sup>。研究<sup>[23]</sup>显示:sICAM-1与冠状动脉病变程度有关。血管细胞黏附分子1(vascular cell adhesion molecule-1, VCAM-1)是免疫球蛋白超家族成员,在生理状态下呈低表达,当发生免疫反应时,受各种刺激因素影响呈高表达,且可经酶水解成为具有生物学功能的可溶性形式,即sVCAM-1<sup>[24-25]</sup>。内皮细胞表达黏附分子对炎症细胞向动脉粥样硬化斑块游走和浸润至关重要<sup>[26-27]</sup>。研究<sup>[28]</sup>表明:sVCAM-1在动脉粥样硬化病变中高表达。

本研究与李颖等<sup>[19]</sup>研究结果一致,MHD患者血清SOD活性与CAC发生发展有关,血清SOD活性降低是MHD患者CAC发生的重要因素。本研究结果显示:与健康体检组比较,MHD组患者血清SOD活性明显降低,sICAM-1和sVCAM-1水平明显升高,且MHD组患者SOD活性随CACs升高而降低,sICAM-1和sVCAM-1水平则随之升高。提示MHD患者血清sICAM-1和sVCAM-1水平及SOD活性呈异常表达。研究<sup>[30]</sup>显示:SOD活性降低可导致体内氧自由基生成增多,加重血管内皮细胞局部损伤,使得血小板易在血管内皮细胞边缘积聚,从而加速斑块形成。sICAM-1和sVCAM-1水平升高提示机体处于慢性炎症状态<sup>[31]</sup>。动脉粥样硬化是一种由细胞介导的慢性炎症过程,因此上述因子在动脉粥样硬化发生发展过程中具有重要作用<sup>[32-34]</sup>。本研究结果显示:MHD患者血清sICAM-1和sVCAM-1水平及SOD活性与CACs存在相关性。提示SOD活性降低和sICAM-1及sVCAM-1水平升高在MHD患者冠状动脉钙化发生发展过程中具有重要作用。

综上所述,MHD患者血清sICAM-1和sVCAM-1水平及SOD活性异常,并且随着SOD活性降低和sICAM-1及sVCAM-1水平升高,MHD患者冠状动脉钙化程度加重。

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