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沙库巴曲缬沙坦对急性前壁心肌梗死急诊 PCI 术后 合并心功能不全患者近期预后的影响 *

陈春望^{1,2} 钱文浩^{3Δ} 丁浩² 周浩² 王万虹²

(1 徐州医科大学 江苏 徐州 221004; 2 徐州医科大学附属医院宿迁医院心血管内科 江苏 宿迁 223800;

3 徐州医科大学附属医院心血管内科 江苏 徐州 221000)

摘要 目的: 研究沙库巴曲缬沙坦对急性前壁心肌梗死急诊经皮冠状动脉介入治疗 (primary percutaneous coronary intervention, PCI) 术后合并心功能不全患者近期预后的影响。**方法:** 选择 2018 年 1 月~2019 年 1 月在我院就诊并入院接受治疗的 52 例急性前壁心肌梗死急诊 PCI 术后合并心功能不全患者, 采用随机数字表法将其分为两组, 每组各 26 例。观察组在常规治疗的基础上使用沙库巴曲缬沙坦, 对照组在常规治疗基础上应用依那普利治疗。分别比较两组患者入组后、出院前 1 d 及随访时血浆肌钙蛋白 (I cardiac troponin I, cTnI)、肌酸激酶同工酶 (Creatine kinase-MB, CK-MB)、超敏 C 反应蛋白 (High-sensitivity creatine protein, hs-CRP)、纤维蛋白原 (Fibrinogen, FIB)、肿瘤坏死因子- α (Tumor necrosis factor, TNF- α)、肌酐 (Creatine, CREA)、N 末端脑钠肽前体 (N-terminal-pro-brain-natriuretic-peptide, NT-proBNP)、左室舒张末期前后径 (Left ventricular end-diastolic diameter, LVEDD)、左室射血分数 (Left ventricular ejection fraction, LVEF)、左室质量指数 (Left ventricular mass index, LVMI)、美国堪萨斯城心肌病患者生存质量表 (Kansas City Cardiomyopathy Questionnaire, KCCQ) 评分、6 min 步行试验 (6MWT)、临床事件及不良反应的发生情况。**结果:** ① 出院前 1 d, 两组患者的血清 cTnI、hs-CRP、NT-proBNP、CK-MB、FIB、TNF- α 、CREA 水平均比治疗前明显下降 ($P < 0.05$); 出院 1 周时, 观察组的血清 cTnI、hs-CRP、NT-proBNP、CK-MB 水平与对照组比较无统计学差异 ($P > 0.05$); 出院 2 周、1 个月时, 观察组的血清 cTnI、hs-CRP、NT-proBNP、CK-MB 水平均明显低于对照组 ($P < 0.05$)。治疗后的各个随访时间点, 两组患者的血清 FIB、TNF- α 、CREA 水平均比治疗前明显降低 ($P < 0.05$), 但组间差异无统计学意义 ($P > 0.05$)。② 两组在出院前 1 d LVEF、LVEDD、LVMI 水平比较差异无统计学意义 ($P > 0.05$)。出院后 1、3、6 个月时, 观察组以上指标的改善幅度显著优于对照组 ($P < 0.05$)。两组患者在接受治疗前收缩压 (SBP) 和舒张压 (DBP) 无统计学差异 ($P > 0.05$), 治疗后上述指标均显著下降 ($P < 0.05$)。但两组在随访过程中各个时间点的收缩压和舒张压无统计学差异 ($P > 0.05$)。③ 两组患者治疗后 KCCQ 生存质量评分较治疗前显著升高 ($P < 0.05$), 且治疗 3 个月和 6 个月时, 观察组的 KCCQ 生存质量评分显著高于对照组 ($P < 0.05$)。观察组临床事件及不良反应的发生率均显著低于对照组 ($P < 0.05$)。**结论:** 沙库巴曲缬沙坦和依那普利用于急性心肌梗死 PCI 术后合并心功能不全患者的疗效相当, 但沙库巴曲缬沙坦治疗的患者近期预后优于依那普利。

关键词: 急性心肌梗死; 经皮冠状动脉介入治疗; 心功能不全

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Effect of Shakuba Trivalsartan on the Short-term Prognosis of Patients with Acute Anterior Wall Myocardial Infarction Complicated with Cardiac Insufficiency after Emergency PCI*

CHEN Chun-wang^{1,2}, QIAN Wen-hao^{3Δ}, DING Hao², ZHOU Hao², WANG Wan-hong²

(1 Xuzhou Medical University, Xuzhou, Jiangsu, 2210004, China;

2 Department of Cardiology, Suqian Hospital Affiliated to Xuzhou Medical University, Suqian, Jiangsu, 223800, China;

3 Department of Cardiology, Affiliated Hospital of Xuzhou Medical University, Xuzhou, Jiangsu, 221000, China)

ABSTRACT Objective: To study the Shakurba valsartan on acute anterior wall myocardial infarction emergency percutaneous coronary intervention postoperative combined effect of the recent outcomes for patients with cardiac insufficiency. **Methods:** From January 2018 to January 2019 in our hospital 52 patients was admitted to hospital as the research object, using the random number table method divided the patients into observation group and control group, 26 cases each group, observation group on the basis of conventional treatment using Shakurba valsartan, and the control group on the basis of conventional treatment of enalapril application. Respectively in the patient group, 1 day before discharge and follow-up after plasma troponin detection cardiac troponin I (cTnI), creatine kinase isoenzyme (CK-MB), hypersensitive c-reactive protein (hs-CRP), fibrinogen (FIB), tumor necrosis factor alpha (TNF- α), CREA, n-terminal-pro-brain-natriuretic-peptide (nt-ProBNP), left ventricular end-diastolic diameter (LVEDD), left ventricular ejection fraction (LVEF)

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作者简介: 陈春望 (1978-), 男, 硕士, 副主任医师, 研究方向: 冠心病介入治疗, 电话: 13485091325, E-mail: Chenchunwang1978@163.com

Δ 通讯作者: 钱文浩 (1967-), 男, 硕士, 主任医师, 研究方向: 冠心病及先心病的介入治疗,

电话: 13485091325, E-mail: Chenchunwang1978@163.com

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and left ventricular quality index (LVMI) were measured by color doppler ultrasound. KCCQ scale score, 6-minute walk test (6MWT); The incidence of clinical events and adverse reactions was calculated. **Results:** (1) 1 day before discharge, cTnI, hs-crp, nt-probnp, ck-mB, FIB, TNF- α , CREA of the two groups were significantly lower than before treatment, the difference was statistically significant ($P<0.05$). At 1 week after discharge, the levels of cTnI, hs-crp, nt-probnp and ck-mB in the observation group were not significantly different from those in the control group ($P>0.05$). At 2 weeks and 1 months after discharge, the levels of cTnI, hs-CRP, nt-Probnp and CK-MB in the observation group were significantly lower than those in the control group ($P<0.05$). FIB, TNF- α and CREA in the two groups were significantly lower at each time point after treatment than before treatment, with statistically significant differences ($P<0.05$), but no significant differences between the two groups ($P>0.05$). (2) There was no significant difference in LVEF, LVEDD, and LVMI levels between the two groups at 1 day before discharge ($P>0.05$). At 1, 3, and 6 months after discharge, the improvement of the above indicators was significantly better than that of the observation group. Control group ($P<0.05$). There was no significant difference in SB-Pand DBPbetween the two groups ($P>0.05$). After treatment, the above indexes were significantly decreased ($P<0.05$). However, there was no significant difference in systolic and diastolic blood pressure between the two groups at each time point during follow-up ($P>0.05$). (3) The KCCQ score of the two groups was significantly higher after treatment than before treatment, and the difference was statistically significant ($P<0.05$). At 3 months and 6 months after treatment, the observation group was significantly higher than the control group, and the difference was statistically significant ($P<0.05$). The incidence of clinical events and adverse reactions in the observation group was lower than that in the control group ($P<0.05$). **Conclusion:** For patients with acute myocardial infarction combined with cardiac insufficiency after PCI, the efficacy of shakurba valsartan and enalapril is comparable, but the short-term prognosis of shakurba valsartan is better than that of enalapril.

Key words: Acute Myocardial Infarction; Percutaneous Coronary Intervention; cardiac insufficiency

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前言

急性心肌梗死(Acute myocardial infarction, AMI)是一种急性缺血缺氧导致的疾病^[1], 临床表现一般为剧烈而持久的胸骨后疼痛, 如得不到及时而有效的医治, 患者极易并发休克、心力衰竭、心功能不全等, 其中心功不全是心肌梗死最为常见的并发症, 病死率较高^[2-4]。研究表明 AMI 患者肾素 - 血管紧张素 - 醛固酮系统(Rennin-angiotensin-aldosterone system, RAAS)激活过度, 使心室发生重构^[5]。血管紧张素转换酶抑制剂(Angiotension converting enzyme inhibitor, ACEI) 和醛固酮拮抗剂能抑制此系统, 降低 AMI 合并心功能不全的病死率和病残率^[6]。但还有部分 AMI 患者急诊 PCI 术后出现心功能恶化。

研究表明 ACEI 类药物仅能阻断部分血管紧张素受体, 对于其他途径产生的血管紧张素 II 则不能阻断^[7]。血管紧张素 II 受体拮抗剂(Angiotensin II type 1 receptorblockers, ARBs)作用于血管紧张素 II 受体, 阻断血管紧张素 II 受体的不利作用, 更好的实现阻断 RAAS 系统, 理论上比 ACEI 类药物更适用于急性心肌梗死 PCI 术后心功能不全患者, 改善患者的心脏功能, 提高生存率。目前有关 ARBs 和 ACEI 用于急性前壁心肌梗死急诊 PCI 术后合并心功能不全的比较研究较少, 本研究选取了 52 例急性前壁心肌梗死急诊 PCI 术后合并心功能不全的患者, 拟对沙库巴曲缬沙坦与依那普利进行比较, 目前 ARBs 用于此类疾病的疗效及患者的近期预后情况。

1 资料与方法

1.1 临床资料

选取 2018 年 1 月至 2019 年 1 月于我院就诊并接受治疗的急性前壁心肌梗死急诊 PCI 术后合并心功能不全的 52 例患者, 发病 14 d 以内入住医院。采用随机数字表法将患者分为两

组, 每组各 26 例。其中, 对照组男 14 例, 女 12 例, 年龄 41~70 岁, 平均年龄(55.7 ± 9.7)岁; 观察组男 15 例, 女 11 例, 年龄 40~73 岁, 平均年龄(56.3 ± 10.1)岁。两组一般资料比较差异均无统计学意义($P>0.05$), 具有可比性。

1.2 入选及排除标准

入选标准: (1) 临床诊断符合 WHO 关于急性前壁心肌梗死诊断新标准; (2) 接受 PCI 术后伴有心功能不全, 心功能 Killip 分级为 II~IV 级; (3) 持续性胸痛时间 <12 h, 且患者发病 14 d 以内入住医院; (3) 心脏生化标志物(cTnI)水平升高超过参考值 99% 值; (4) 病变位于前降支近段或近中段且血管直径 >3 mm。排除标准: (1) 非急性前壁心肌梗死; (2) 心源性休克或存在其他相关因素, 不宜对患者应用扩张血管的药物; (3) 存在严重肝肾功能不全者; (4) 有自身免疫性、感染性、出血性疾病、严重创伤、合并肿瘤者; (5) 对造影剂过敏者; (7) 患者或家属拒绝参与本研究。

1.3 研究方法

两组患者均给予常规治疗, 包括扩张冠状动脉、调节血脂、抗凝治疗等, 对照组患者在此基础上联合应用依那普利(扬子江药业集团江苏制药股份有限公司, 国药准字 H32026568), 口服, 剂量 5 mg, 口服 1 次 /d, 连续用药 6 个月。观察组在常规治疗的基础上联合使用沙库巴曲缬沙坦, (Novartis Pharma Stein AG, 国药准字 H20170344), 初始给药剂量为每次 50 mg, 每天 2 次, 然后每 1-2 周视患者情况增加 50 mg, 最高给药剂量为每次 200 mg, 连续用药 6 个月。在出院后 1、3、6 个月时行门诊随访, 同时根据患者情况适当调整药物剂量。

1.4 观察指标

1.4.1 血液及生化指标 分别在入组时、出院前 1 d、出院后 1 周、2 周、1 个月检查血浆 cTnI、CK-MB、hs-CRP、FIB、TNF- α 、CREA、NT-proBNP 水平。

1.4.2 心室重构效果 分别在出院前 1 d, 出院后 1 个月, 3 个月, 6 个月检查心脏彩超, 记录两组患者的左室舒张末期前后径(LVEDD)、左室射血分数(LVEF)及左室质量指数(LVMI), 并进行组间比较。

1.4.3 生活质量 分别在入组时、出院前 1 d、出院后 1 个月、3 个月、6 个月采用生活质量量表 (Kansas city cardiomyopathy questionnaire, KCCQ), 此量表由 23 个问题组成, 包括躯体受限得分、自我评价得分、社会功能得分、自我认知得分、临床得分、心理得分、临床症状得分等 8 个方面。KCCQ 量表得分越高表示健康状态越好。6 min 步行试验(6MWT)检查, 并进行组间比较。

1.4.4 心脏不良事件发生率 记录并比较两组患者在治疗及随访期间心脏不良事件的发生率, 包括: 死亡、非致死性心肌梗死、缺血性卒中、心源性再入院、心衰再入院。

1.4.5 不良反应发生率 记录在治疗及随访期间出现低血压、血管神经性水肿、肾功能不全、高钾血症等发何时能情况。

1.5 统计学方法

采用 SPSS19.0 软件进行数据分析, 计量资料以($\bar{x} \pm s$)表示, 组间比较采用 t 检验; 计数资料以[例(%)]表示, 组间比较采用 χ^2 检验, 以 $P < 0.05$ 为差异具有统计学意义。

2 结果

2.1 两组不同时点血生化指标的比较

出院前 1 d, 两组患者的血清 cTnI、hs-CRP、NT-proBNP、CK-MB、FIB、TNF- α 、CREA 均比治疗前明显下降($P < 0.05$); 出院 1 周时, 观察组的血清 cTnI、hs-CRP、NT-proBNP、CK-MB 水平与对照组比较无统计学差异($P > 0.05$); 出院 2 周、1 个月时, 观察组的 cTnI、hs-CRP、NT-proBNP、CK-MB 水平均明显低于对照组($P < 0.05$); 经治疗后的各个随访时间点, 两组患者的 FIB、TNF- α 、CREA 均比治疗前明显降低($P < 0.05$), 但两组各个随访时间点 FIB、TNF- α 、CREA 水平差异无统计学意义($P > 0.05$)。

表 1 两组患者治疗前后血液及生化指标比较(n=40)

Table 1 Comparison of the blood and Biochemical Indices before and after Treatment between Two Groups of Patients(n=26)

Groups	cTn I /(mg/L)	hs-CRP/(mg/L)	NT-proBNP/(pg/L)	CK-MB/(u/L)	FIB/(g/L)	TNF- α (pg/mL)	CREA/(mg/L)
Control group							
Before treatment	5.25 $\pm$ 1.41	8.18 $\pm$ 1.67	4322.74 $\pm$ 511.19	91.53 $\pm$ 16.92	4.47 $\pm$ 1.53	12.15 $\pm$ 2.17	23.32 $\pm$ 2.24
At 1 day before discharge	5.04 $\pm$ 1.26 [#]	7.94 $\pm$ 1.53 [#]	3997.55 $\pm$ 479.66 [#]	85.66 $\pm$ 14.72 [#]	4.32 $\pm$ 1.23 [#]	11.29 $\pm$ 1.79 [#]	20.07 $\pm$ 2.57 [#]
At 1 week after discharge	4.22 $\pm$ 1.32 [#]	6.67 $\pm$ 1.43 [#]	3557.37 $\pm$ 372.74 [#]	74.61 $\pm$ 13.85 [#]	4.08 $\pm$ 1.17 [#]	9.48 $\pm$ 1.67 [#]	18.33 $\pm$ 2.16 [#]
At 2 weeks after discharge	2.37 $\pm$ 1.24 [#]	5.53 $\pm$ 1.37 [#]	2274.34 $\pm$ 336.34 [#]	51.27 $\pm$ 11.74 [#]	3.27 $\pm$ 1.20 [#]	9.11 $\pm$ 1.76 [#]	17.44 $\pm$ 1.99 [#]
At 1 month after discharge	1.22 $\pm$ 0.24 [#]	4.56 $\pm$ 1.54 [#]	1279.27 $\pm$ 337.25 [#]	32.33 $\pm$ 10.81 [#]	3.01 $\pm$ 0.74 [#]	5.89 $\pm$ 2.35 [#]	15.48 $\pm$ 2.54 [#]
Observation group							
Before treatment	5.21 $\pm$ 1.24	8.13 $\pm$ 1.26	4326.25 $\pm$ 513.22	92.42 $\pm$ 17.53	4.51 $\pm$ 1.47	13.27 $\pm$ 2.28	25.61 $\pm$ 1.97
At 1 day before discharge	5.01 $\pm$ 1.21 [#]	7.85 $\pm$ 1.52 [#]	3974.52 $\pm$ 476.79 [#]	84.61 $\pm$ 13.46 [#]	4.33 $\pm$ 1.13 [#]	10.17 $\pm$ 2.24 [#]	21.04 $\pm$ 2.26 [#]
At 1 week after discharge	4.21 $\pm$ 1.33 [#]	6.66 $\pm$ 1.27 [#]	3567.29 $\pm$ 378.46 [#]	73.62 $\pm$ 13.84 [#]	4.07 $\pm$ 1.27 [#]	9.47 $\pm$ 1.77 [#]	18.47 $\pm$ 1.39 [#]
At 2 weeks after discharge	1.29 $\pm$ 1.22 ^{#*}	5.47 $\pm$ 1.36 ^{#*}	1479.67 $\pm$ 317.21 ^{#*}	51.22 $\pm$ 11.78 ^{#*}	3.26 $\pm$ 1.21 [#]	8.87 $\pm$ 1.81 [#]	17.57 $\pm$ 1.96 [#]
At 1 month after discharge	1.05 $\pm$ 0.24 ^{#*}	2.45 $\pm$ 1.26 ^{#*}	647.17 $\pm$ 213.38 ^{#*}	30.16 $\pm$ 9.88 ^{#*}	3.02 $\pm$ 0.77 [#]	5.77 $\pm$ 2.15 [#]	15.36 $\pm$ 2.27 [#]

Note: Compared with the same group before treatment, [#] $P < 0.05$; Compared with control group at the same time, * $P < 0.05$.

2.2 两组心室重构效果的比较

两组在出院前 1 d LVEF、LVEDD、LVMI 水平比较差异无统计学意义($P > 0.05$), 出院后 1、3、6 个月时, 观察组以上指标的改善幅度显著优于对照组($P < 0.05$)。两组患者在接受治疗前收缩压(SBP)和舒张压(DBP)无统计学差异($P > 0.05$), 两组在接受治疗后上述指标均显著下降($P < 0.05$)。但两组在随访过程中各个时点的收缩压和舒张压无统计学差异($P > 0.05$)。详见表 2。

2.3 两组患者治疗后不同时点 KCCQ 量表评分及 6MWT 的比较

治疗后的 1、3、6 个月, 两组患者的 KCCQ 生存质量评分均较治疗前显著升高($P < 0.05$), 且在治疗 3 个月和 6 个月时, 观察组的 KCCQ 评分显著高于对照组($P < 0.05$), 详见表 3。两组患者在出院前 1 d 和出院后 1 个月的两次检查无统计学差异($P > 0.05$); 出院后 3、6 个月, 观察组患者的 6MWT 均比对照组显著升高($P < 0.05$)。详见表 4。

2.5 两组临床事件发生情况的比较

观察组在接受治疗及随访 6 个月过程中临床事件发生率显著低于对照组($P < 0.05$), 详见表 5。

2.6 两组不良反应发生情况的比较

观察组患者不良反应发生率显著低于对照组 ($P < 0.05$), 详见表 6。

3 讨论

急性前壁性心肌梗死急诊 PCI 术后合并心功能不全患者的治疗以激活交感神经和 RAAS 系统这两个途径达到增加血容量、提高心肌收缩力和增强心脏泵血功能的目的, 从而实现心脏的正常血供^[8,9]。RAAS 系统发挥作用主要有两个途径, 即循环 RAAS 途径和组织 RAAS 途径^[10], 前者影响钠代谢和动脉的顺应性来调节动脉压, 对病理生理产生短期效应^[11], 而后者

则产生长期效应^[12],将血管紧张素 I 转换为血管紧张素 II。而 统,促进心肌细胞和血管的生长^[13]。后者与受体结合后能使得醛固酮大量分泌,激活交感神经系

表 2 两组患者治疗前后 LVEF 及血压水平的比较($\bar{x} \pm s, \%$)Table 2 Comparison of the LVEF and blood pressure before and after treatment between the two groups ($\bar{x} \pm s, \%$)

Groups	n	LVEF(%)	LVEDD(mm)	LVMI(g/m ²)	SBP(mmHg)	DBP(mmHg)
Control group	26					
Before treatment		-	-	-	134.5 $\pm$ 14.2	85.8 $\pm$ 8.4
At 1 day before discharge		47.97 $\pm$ 7.82 [#]	40.48 $\pm$ 3.37 [#]	130.25 $\pm$ 11.49 [#]	129.4 $\pm$ 11.7	84.4 $\pm$ 7.6
At 1 month after discharge		48.88 $\pm$ 7.04 [#]	38.65 $\pm$ 4.51 [#]	127.65 $\pm$ 12.46 [#]	127.6 $\pm$ 11.2	83.1 $\pm$ 7.7
At 3 month after discharge		49.71 $\pm$ 5.66 [#]	37.47 $\pm$ 3.67 [#]	127.04 $\pm$ 12.05 [#]	125.7 $\pm$ 12.8 [#]	80.6 $\pm$ 6.1
At 6 month after discharge		49.92 $\pm$ 7.63 [#]	36.92 $\pm$ 3.50 [#]	124.64 $\pm$ 12.93 [#]	121.1 $\pm$ 12.2 [#]	78.4 $\pm$ 8.1 [#]
Observation group	26					
Before treatment		-	-	-	136.7 $\pm$ 13.4	86.6 $\pm$ 9.0
At 1 day before discharge		47.87 $\pm$ 6.67 [#]	40.58 $\pm$ 3.38 [#]	130.59 $\pm$ 14.11 [#]	127.7 $\pm$ 11.9	84.7 $\pm$ 6.7
At 1 month after discharge		52.85 $\pm$ 7.11 ^{#*}	36.17 $\pm$ 3.54 ^{#*}	120.57 $\pm$ 11.17 ^{#*}	124.6 $\pm$ 12.4	84.2 $\pm$ 7.2
At 3 month after discharge		53.48 $\pm$ 7.59 ^{#*}	34.66 $\pm$ 4.01 ^{#*}	119.77 $\pm$ 13.68 ^{#*}	124.4 $\pm$ 11.7 [#]	81.1 $\pm$ 6.7
At 6 month after discharge		56.12 $\pm$ 8.43 ^{#*}	31.99 $\pm$ 3.87 ^{#*}	101.97 $\pm$ 11.92 ^{#*}	123.4 $\pm$ 12.8 [#]	79.1 $\pm$ 8.7 [#]

Note: Compared with the same group before treatment, [#] $P < 0.05$; Compared with control group at the same time, * $P < 0.05$.

表 3 两组患者治疗前后 KCCQ 量表评分比较(分)

Table 3 Comparison of the KCCQ scale scores before and after treatment between the two groups (score)

GroupS	n	Before treatment	1 day before discharge	1 month after discharge	3 month after discharge	6 month after discharge
Control group	26	43.2 $\pm$ 8.1	47.4 $\pm$ 10.2	67.7 $\pm$ 9.4	77.1 $\pm$ 10.4	80.4 $\pm$ 11.4
Observation group	26	46.1 $\pm$ 10.5	48.6 $\pm$ 9.7	69.6 $\pm$ 10.3	84.2 $\pm$ 11.7*	87.7 $\pm$ 12.2*

Note: Compared with control group, * $P < 0.05$.

表 4 两组患者治疗后 6MWT 情况比较(m)

Table 4 Comparison of the 6MWT status between the two groups after treatment(m)

GroupS	n	1 day before discharge	1 month after discharge	3 month after discharge	6 month after discharge
Control group	26	260.3 $\pm$ 24.5	286.4 $\pm$ 31.1	300.7 $\pm$ 56.9	307.5 $\pm$ 76.4
Observation group	26	266.8 $\pm$ 23.7	299.7 $\pm$ 29.7	334.6 $\pm$ 55.4*	352.1 $\pm$ 70.7*

Note: Compared with control group, * $P < 0.05$.

表 5 两组患者治疗及随访期间临床事件发生情况比较[例(%)]

Table 5 Comparison of the incidence of clinical events between the two groups during treatment and follow-up [n(%)]

GroupS	n	all-cause mortality	Nonfatal myocardial infarction	Nonfatal cardiogenic shock	Coronary artery reconstruction	Total
Control group	26	0(0)	2(7.69)	1(3.85)	6(23.08)	9(34.62)
Observation group	26	0(0)	1(3.85)	0(0)	2(7.69)	3(11.54)*

Note: Compared with control group, * $P < 0.05$.

表 6 两组患者治疗及随访期间不良反应发生情况比较[例(%)]

Table 6 Comparison of the incidence of adverse reactions between the two groups during treatment and follow-up [n(%)]

GroupS	n	Hypotension	Angioneurotic edema	Renal insufficiency	Hyperkalemia	Total
Control group	26	5(19.23)	2(7.69)	1(3.85)	2(7.69)	10(38.46)
Observation group	26	1(3.85)	1(3.85)	0(0.00)	1(3.85)	3(11.54)*

Note: Compared with control group, * $P < 0.05$.

ACEI 类药物能够显著改善慢性充血性心力衰竭患者的心功能,但由于心脏组织中存大部分 AT1 是通过非 ACE 途径生成,这部分血管紧张素 II 的作用不能通过 ACEI 类药物阻断。沙库巴缬沙坦属于 ARBs 类药物,选择性作用于 AT1 受体,阻断血管紧张素 II,从而更好的阻断 RAAS 系统。Pottg rd A 等^[14]研究认为缬沙坦可通过抑制心肌肥厚和纤维化来改善心肌病的血流动力学及部分心脏功能,同时能够对血液循环产生短期效应,对局部组织产生长期效应^[15],抑制血管因子释放,降低去甲肾上腺素的释放量,增加前列腺素及缓激肽生成量,增加肾血流量,抑制分泌醛固酮,降低血压,减少心脏负荷,对于合并心功能不全的急性心肌梗死患者抗压疗效稳定、持久,可以长期应用^[16]。在本研究中,应用沙库巴缬沙坦的观察组患者 cTnI、hs-CRP、NT-proBNP、CK-MB 的水平在治疗 2 周、1 个月时均显著低于对照组,表明沙库巴缬沙坦对患者的近期疗效优于依那普利^[17]。

KCCQ 量表可用于评价 PCI 术后合并心功能不全患者的生活和预后^[18-21],本研究显示两组在出院前直到出院后 1 个月 KCCQ 评分均比入院时出现明显上升,即患者此时的生活质量得到较好的改善,患者自我感觉明显好转。但两组间这两个时点比较无统计学差异,主要是 KCCQ 量表引入了主观评价,当患者的生命体征逐渐平稳,患者的日常活动受到的影响较入院时明显减轻时,患者的自我感受均有明显好转。在出院 3 个月和 6 个月复查时,观察组的 KCCQ 得分则明显高于对照组,同时 6MWT 也显著优于对照组,说明随着治疗时间的延长,采用沙库巴缬沙坦治疗具有对急性前壁心肌梗死急诊 PCI 术后合并心功能不全患者的治疗优势。

研究显示 FIB 能促进急性心肌梗死患者的血栓形成^[22],使患者的血管平滑肌细胞增生,出现炎症反应^[23]。本研究结果显示对于急性心肌梗死 PCI 术后合并心功能不全的患者,无论观察组还是对照组的的治疗方法,都能有效降低 FIB 在血液中的含量,且不同时间点两组间无差异。TNT- α 是由单核 / 巨噬细胞分泌的一种多肽,具有多种多样的生物活性,如抗感染、抗肿瘤等^[24]。机体处于正常状态时,TNF- α 量极少,机体一旦受损,TNF- α 则大量分泌并释放,促进 C-反应蛋白及白细胞介素的生成及分泌^[25],从而介导免疫炎症反应,放大炎症信号,加剧细胞损伤^[26]。因此,TNF- α 的含量能反映机体的炎症活跃程度^[27,28]。另外,有研究指出 TNF- α 能通过调节脂溶酶的活性打破机体凝血 - 抗凝血的平衡,加速血栓生成,进而参与 AMI 过程的发生^[29,30]。本研究中,两组患者各个随访时间点 TNF- α 水平差异并不显著。

综上所述,沙库巴缬沙坦和依那普利用于急性心肌梗死 PCI 术后合并心功能不全患者的疗效相当,但沙库巴缬沙坦治疗的患者近期预后优于依那普利。但本研究选择的病例数量每组仅有 26 例,同时病例仅在本院选择可能存在选择偏倚,因此需要今后更多研究数据的支持和验证。

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