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抗 HP 治疗对急性冠脉综合征合并 HP 感染患者血清炎性标记物及再发心肌缺血事件的影响 *

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摘要 目的:探讨抗幽门螺杆菌(HP)治疗对急性冠脉综合征(ACS)合并 HP 感染患者炎性标记物及再发心肌缺血事件的影响。**方法:**选取 2016 年 10 月到 2018 年 10 月期间我院收治的 ACS 患者 90 例,随机分为对照组(n=45,常规治疗)和观察组(n=45,常规治疗+抗 HP 治疗)。观察两组患者的临床疗效,比较 HP 根除率、血清炎性标记物[C 反应蛋白(CRP)、白细胞介素-6(IL-6)、可溶性细胞间粘附分子-1(sICAM-1)]水平,记录再发心肌缺血事件和不良反应发生情况。**结果:**观察组的总有效率和 HP 根除率均高于对照组($P<0.05$)。两组患者经治疗后血清 CRP、IL-6、sICAM-1 水平均较治疗前有所改善($P<0.05$),且观察组的改善效果优于对照组($P<0.05$)。观察组患者再发心肌缺血事件的总发生率低于对照组($P<0.05$)。两组患者的不良反应发生率比较无明显差异($P>0.05$)。**结论:**ACS 合并 HP 感染患者采取抗 HP 治疗可更好地缓解患者体内的炎症反应,降低再发心肌缺血事件发生率的同时还不会增加不良反应发生风险。

关键词:急性冠脉综合征;幽门螺杆菌;感染;炎性标记物;心肌缺血事件

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Effects of Anti-HP Therapy on Serum Inflammatory Markers and Recurrent Myocardial Ischemia in Patients with Acute Coronary Syndrome Combined with HP Infection*

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ABSTRACT Objective: To investigate the effect of anti-*Helicobacter pylori* (HP) therapy on inflammatory markers and recurrent myocardial ischemia in patients with acute coronary syndrome (ACS) combined with HP infection. **Methods:** 90 patients with ACS who were treated in our hospital from October 2016 to October 2018 were selected, which were randomly divided into the control group (n=45, conventional therapy) and the observation group (n=45, conventional therapy combined anti-HP therapy). The clinical effect of the two groups was observed, HP eradication rate, serum inflammatory markers levels [C reactive protein(CRP), interleukin-6(IL-6), soluble intercellular adhesion molecule-1 (sICAM-1)] were compared between the two groups, the incidence of myocardial ischemia and adverse reactions were recorded. **Results:** The total effective rate and HP eradication rate of the observation group was higher than that of the control group ($P<0.05$). The levels of serum CRP, IL-6 and sICAM-1 the two groups were improved after treatment ($P<0.05$), and the improvement effect of the observation group is better than that of the control group($P<0.05$). The total incidence of recurrent myocardial ischemic events in the observation group was lower than that in the control group ($P<0.05$). There was no significant difference in the incidence of adverse reactions between the two groups ($P>0.05$). **Conclusion:** Anti-HP therapy for patients with ACS and HP infection can better alleviate the inflammatory reaction in the body, reduce the incidence of recurrent myocardial ischemia events without increasing the risk of adverse reactions.

Key words: Acute coronary syndrome; *Helicobacter pylori*; Infection; Inflammatory markers; Myocardial ischemic events

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前言

急性冠脉综合征(Acute coronary syndrome, ACS)是冠心病的一种严重类型,其病理基础为动脉粥样硬化斑块破裂或侵袭导致血栓形成,患者主要表现为胸痛、胸闷等症状,并且可引发心律失常、心力衰竭等严重并发症,对患者的生命健康构成巨大的威胁^[1,2]。幽门螺杆菌(*Helicobacter pylori*, HP)是一种螺旋形、微厌氧的细菌,众所周知,HP感染是消化性溃疡^[3]、慢性胃炎^[4]、胃癌^[5]等疾病的重要危险因素,然而已有相关研究证实^[6,7],HP感染是诱发或加重动脉粥样硬化的重要原因,并且对动脉粥样硬化斑块的稳定性产生不利的影响,目前HP感染与心血管疾病的相关性已成为临床研究的重点。由于HP感染对心血管疾病患者病情进展存在一定的影响,因此临床治疗ACS合并HP感染患者时,抗HP治疗逐渐受到重视^[8],然而抗HP治疗对ACS合并HP感染患者炎性标记物及再发心肌缺血事件的影响报道尚不多见,鉴于此,本研究对ACS合并HP感染患者予以抗HP治疗,取得了较为满意的效果,报道如下。

1 资料与方法

1.1 一般资料

选取我院2016年10月到2018年10月期间收治的ACS患者90例,纳入标准:(1)所有患者均通过冠脉造影、心电图、心肌酶学诊断为ACS^[9];(2)所有患者均进行了14C尿素呼气实验,且结果为阳性,即合并有HP感染;(3)患者及其家属对本次研究均知情同意;(4)美国纽约心脏病学会(New York Heart Association, NYHA)心功能分级^[10]I-II级。排除标准:(1)近期有肺部感染、尿路感染、上呼吸道感染等感染病史者;(2)器官功能衰竭、严重营养不良、严重肝肾功能不全、严重心衰者;(3)合并有消化系统疾病者;(4)近期有使用抗HP感染、消炎药、调脂药物、激素类药物者;(5)拟近期进行介入治疗者;(6)对本次研究药物过敏者;(7)合并神经疾病及精神疾病者。将90例ACS患者随机分为对照组和观察组,两组均为45例。其中对照组男性27例,女性18例,年龄42-71岁,平均年龄(58.26±6.48)岁,NYHA分级:I级8例,II级37例,基础疾病:高血压16例,糖尿病11例。观察组男性25例,女性20例,年龄40-73岁,平均年龄(59.37±6.54)岁,NYHA分级:I级7例,II级38例,基础疾病:高血压17例,糖尿病10例。两组患者的一般资料比较无差异($P>0.05$)。

1.2 治疗方法

根据《急性冠脉综合征急诊快速诊疗指南》^[9]和临床实际情况对所有患者进行抗血小板、抗凝、抗缺血、他汀类药物等常规治疗。阿司匹林肠溶片(湖南恒生制药股份有限公司,50 mg/片,国药准字H43021971),口服,前3d服用300 mg,后续100 mg/d,1次/d;硫酸氢氯吡格雷片(深圳信立泰药业股份有限公司,75 mg/片,国药准字H20120035),口服,负荷剂量为300 mg,而后75 mg/d,2次/d;低分子肝素钙(深圳赛保尔生物药业有限公司,1.0 mL:5000AXaIU,国药准字H20060190),皮下注射,8000IU/次,2次/d;硝酸甘油注射液(广州白云山明兴制药有限公司,1 mL:5 mg,国药准字H44020569),用250 mL 5%葡萄糖注射液或氯化钠注射液稀释后静脉滴注,开始剂量为

15 mL/min,后续视患者血压情况调整,12 h/次;阿托伐他汀钙片(辉瑞制药有限公司,10 mg/片,国药准字H20051407),20 mg/d,睡前口服,均持续服用/注射1个月。观察组患者在此基础上进行抗HP治疗,治疗方案为克拉霉素三联方案,阿莫西林胶囊(西安利君制药有限责任公司,0.25 g/粒,国药准字H61023107),口服,500 mg/次,2次/d,克拉霉素缓释片(江苏恒瑞医药股份有限公司,0.5 g/片,国药准字:20031041),口服,250 mg/次,2次/d,奥美拉唑肠溶胶囊(悦康药业集团股份有限公司,20 mg/粒,国药准字H20056577),口服,20 mg/次,1次/d,抗HP治疗共持续7d。

1.3 观察指标

1.3.1 疗效观察 治疗后1个月根据患者的心电图、临床症状和相关指标的检测结果进行疗效判定^[11],显效:患者心电图恢复正常或ST段恢复50%,患者的各项临床症状消失,各项检查指标在正常范围内或接近正常范围;有效:患者心电图基本恢复,ST段恢复30%-50%,患者的各项临床症状明显改善,1周内胸痛未发作,各项检查指标接近正常范围;无效:患者心电图、ST段、各项临床症状无明显改善,1周内胸痛再次发作,各项检查指标无明显改善。总有效率=显效率+有效率。

1.3.2 HP根除情况 治疗后1个月所有患者均进行14C尿素呼气实验,在空腹状态下口服检查胶囊,25 min后进行吹气,直至二氧化碳集气卡内指示剂为黄色时停止,测量结果为阴性则认为HP被根除,计算两组患者的HP根除率。

1.3.3 血清炎性标记物检测 在治疗前和治疗后1个月抽取患者的空腹静脉血5 mL,3000 r/min离心10 min,离心半径8 cm,分离上清后采用免疫透射比浊法测定血清C反应蛋白(C reactive protein, CRP)水平,采用酶联免疫吸附法测定白细胞介素-6(Interleukin-6, IL-6)、可溶性细胞间粘附分子-1(Soluble intercellular adhesion molecule -1, sICAM-1)水平(试剂盒均购自武汉博士德生物有限公司),严格遵从试剂盒说明书进行相关操作。

1.3.4 再发心肌缺血事件和不良反应 所有患者均通过电话、门诊等方式进行随访,对所有患者的再发心肌缺血事件(急性心肌梗死、心绞痛、心源性猝死、心力衰竭等)进行统计,记录患者治疗期间出现的不良反应,随访终点时间为随访时长到达12个月或患者死亡。

1.4 统计学方法

采用SPSS23.0进行统计分析,计数资料以%表示,行卡方检验,计量资料以($\bar{x} \pm s$)表示,行t检验。 $P<0.05$ 为差异有统计学意义。

2 结果

2.1 两组患者临床疗效的比较

治疗后1个月,观察组的临床总有效率为95.56%(43/45),高于对照组的82.22%(37/45)($P<0.05$)。见表1

2.2 两组患者HP根除情况对比

治疗后1个月,观察组中有42例患者14C尿素呼气实验结果为阴性,HP根除率为93.33%(42/45),对照组中有18例患者14C尿素呼气实验结果为阴性,HP根除率为40.00%(18/45),观察组的HP根除率明显高于对照组,差异有统计学

意义($\chi^2=28.876, P=0.000$)。

2.3 两组患者血清炎性标记物对比

治疗前两组患者血清中 CRP、IL-6、sICAM-1 的水平比较

无明显差异 ($P>0.05$), 治疗后两组患者血清中 CRP、IL-6、sICAM-1 的水平均明显降低 ($P<0.05$), 且观察组明显低于对照组 ($P<0.05$), 见表 2。

表 1 两组患者临床疗效的比较 [n(%)]

Table 1 Comparison of clinical efficacy between the two groups [n(%)]

Groups	n	Markedly effective	Effective	Invalid	Total effective
Control group	45	15(33.33)	22(48.89)	8(17.78)	37(82.22)
Observation group	45	19(42.22)	24(53.33)	2(4.44)	43(95.56)
χ^2					4.057
P					0.044

表 2 两组患者血清炎性标记物对比($\bar{x} \pm s$)

Table 2 Comparison of serum inflammatory markers of patients in two groups($\bar{x} \pm s$)

Groups	n	Time	CRP(mg/L)	IL-6(ng/L)	sICAM-1(μ g/L)
Control group	45	Before treatment	16.44 $\pm$ 3.32	43.71 $\pm$ 11.53	513.28 $\pm$ 51.32
		After treatment	7.89 $\pm$ 2.81 ^a	36.34 $\pm$ 6.35 ^a	453.51 $\pm$ 39.37 ^a
Observation group	45	Before treatment	16.87 $\pm$ 3.29	44.41 $\pm$ 11.32	522.11 $\pm$ 53.62
		After treatment	5.26 $\pm$ 1.41 ^{ab}	30.11 $\pm$ 5.39 ^{ab}	416.10 $\pm$ 32.49 ^{ab}

Notes: Compared with before treatment, ^a $P<0.05$, compared with the control group, ^b $P<0.05$.

2.4 两组患者再发心肌缺血事件对比

观察组患者再发心肌缺血事件的总发生率低于对照组

($P<0.05$), 见表 3。

表 3 两组患者再发心肌缺血事件对比[n(%)]

Table 3 Comparison of recurrent myocardial ischemia events of patients in two groups [n(%)]

Groups	n	Acute myocardial infarction	Angina pectoris	Sudden cardiac death	Heart failure	Total incidence rate
Control group	45	1(2.22)	8(17.78)	1(2.22)	1(2.22)	11(24.44)
Observation group	45	1(2.22)	3(22.22)	0(0.00)	0(0.00)	4(8.89)
χ^2						3.920
P						0.048

2.5 两组患者不良反应对比

治疗期间对照组出现 1 例口干、1 例轻微头晕, 不良反应发生率为 4.44%(2/45), 观察组出现 1 例口干、1 例轻微头晕、1 例恶心, 不良反应发生率为 6.67%(3/45)。两组患者的不良反应发生率比较无差异($\chi^2=0.212, P=0.645$)。

3 讨论

ACS 包括了不稳定性心绞痛、非 ST 段抬高型心肌梗死、ST 段抬高型心肌梗死, 是严重的心血管疾病^[12,13], ACS 患者起病急、病情变化快, 若未得到及时有效的治疗患者的死亡率较高^[14]。绝大部分 ACS 患者是由于动脉粥样硬化斑块不稳定所导致的, 当斑块发生破裂或侵袭时可继发血栓, 进而导致冠状动脉供血不足, 心肌出现缺血缺氧^[15], 影响患者预后。大量研究显示^[16-18], 斑块的稳定性与机体内的炎症反应程度密切相关, 斑块的纤维帽是维持斑块稳定性的关键因素之一, 不稳定斑块的

纤维帽较薄, 而炎症反应正是导致斑块纤维帽变薄的主要原因。巨噬细胞、肥大细胞、泡沫细胞等炎症细胞可促进基质金属蛋白酶的表达, 基质金属蛋白酶可水解细胞外基质, 进而导致斑块纤维帽变薄, 并且部分炎症因子还可以直接对平滑肌细胞的凋亡起到促进作用, 平滑肌细胞是斑块纤维帽主要成分, 因此炎症反应可以直接或间接的导致斑块纤维帽变薄, 进而影响斑块的稳定性^[19,21]。由此可见, 在治疗 ACS 时控制患者体内的炎症反应尤为重要。

在本次研究中, 观察组的总有效率高于对照组, 这说明抗 HP 治疗能有效提高 ACS 合并 HP 感染患者的临床疗效。近年来, 不断有研究显示, HP 感染与心血管疾病的发生、发展有关, 徐晶等人的研究显示^[22], HP 感染会影响机体炎症反应及脂质代谢, 进而促进颈动脉粥样硬化的发展, Lai CY 等人的研究结果显示^[23], HP 感染会增加患上 ACS 的风险。目前对于 HP 感染影响心血管疾病进展的具体作用机制尚无一定论, 但已明确

HP 的促炎作用是主要机制之一,HP 定植在胃黏膜上皮细胞中后,可引起局部慢性炎症^[24]。HP 的代谢产物中有众多的促炎因子,如 HP 分泌的细胞毒素相关基因 A (Cytotoxin-associated gene A, CagA) 蛋白可促进肿瘤坏死因子分泌^[25],黄煌等人的研究也证实 CagA 可促进原发性不孕症患者血清促炎因子水平升高^[26];此外 HP 产生的空泡毒素基因 (vacuolating cytotoxin gene A, vacA) 也是一种促炎因子,可促进肥大细胞分泌肿瘤坏死因子、促进巨噬细胞分泌 IL-6,进而增加体内的炎症反应^[27]。

本研究选用的是美国胃肠病学院推荐的抗 HP 一线治疗方案,结果显示观察组的 HP 根除率明显高于对照组,表明该治疗方案能可有效根除 HP。CRP、IL-6 和 sICAM-1 均是常见的炎症标记物,CRP 在机体发生炎症反应时迅速升高,IL-6 是一种前炎症细胞因子,通过与其受体结合来诱导炎症反应,sICAM-1 可促进单核细胞黏附到血管内皮细胞,加重局部炎症反应^[28,29]。本研究结果显示,治疗后观察组患者血清中 CRP、IL-6、sICAM-1 的水平明显低于对照组,这说明抗 HP 治疗能有效降低 ACS 合并 HP 感染患者血清中的炎症标记物的水平,这主要是因为 HP 感染具有一定的促炎作用,对机体内的 HP 进行清除后,体内的炎症反应程度减轻^[30],因此根除 HP 能有效缓解炎症反应。防止心肌缺血事件再次发生是临床治疗 ACS 的重点,本研究结果显示,观察组患者再发心肌缺血事件的总发生率低于对照组,这提示抗 HP 治疗能有效降低 ACS 合并 HP 感染患者再发心肌缺血事件的发生风险,这可能与抗 HP 治疗有更好的临床疗效有关。两组患者治疗期间均未出现严重的不良反应,且两组患者的不良反应发生率比较差异无统计学意义,说明抗 HP 治疗安全性好。

综上所述,抗 HP 治疗可控制 ACS 合并 HP 感染患者的炎症反应,并可降低再发心肌缺血事件的发生率,且用药安全性较好。

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