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论著

肠淋巴途径在二次打击致大鼠MODS的发病学作用

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摘要 目的：探讨淋巴途径在二次打击致大鼠MODS的发病学作用。 方法： 结扎肠系膜淋巴管致肠淋巴液断流，以二次打击方法复制MODS模型。Wistar大鼠45只均分为结扎组、未结扎组、假手术组3组，术前及创伤后24 h取血，制备肾、肝、肺、心、肠组织10%匀浆，检测TNFα、NO、MDA、SOD等指标。 结果： 成功复制了MODS大鼠模型。二次打击后，未结扎组大鼠血清TNFα、NO2-/NO3-、NOS、iNOS、MDA均显著高于实验前及假手术组，SOD显著下降（P<0.01，P<0.05）；结扎组大鼠血清NO2-/NO3-、NOS、MDA高于假手术组（P<0.01），TNFα、NO2-/NO3-、iNOS、MDA显著低于未结扎组，SOD显著高于未结扎组（P<0.01）。未结扎组肠匀浆TNFα、NO2-/NO3-、NOS、iNOS、MDA，肾匀浆NO2-/NO3-、NOS、MDA，肝匀浆NO2-/NO3-、MDA及肺、心匀浆NO2-/NO3-均显著高于假手术组，肠匀浆SOD显著低于假手术组（P<0.01，P<0.05）；结扎组肾匀浆NO2-/NO3-、MDA及肝匀浆MDA显著高于假手术组（P<0.01，P<0.05）。结扎组肠、肾、肝匀浆NO2-/NO3-显著低于未结扎组，肠、心匀浆SOD显著高于未结扎组（P<0.01）。 结论： 肠系膜淋巴管结扎阻断了二次打击所致内毒素经淋巴流的移位，抑制TNFα释放，使iNOS生成减少，NO形成降低，减少自由基释放与SOD消耗，MODS的淋巴机制值得重视。

关键词 多器官功能衰竭； 淋巴系统； 结扎术； 一氧化氮； 肿瘤坏死因子； 自由基

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Role of intestinal lymphatic pathway in MODS pathogenesis by two-hit in rats

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Abstract

<FONT face=Verdana>AIM: To explore the role of intestinal lymphatic pathway in MODS pathogenesis by two-hit in rats. METHODS: Mesenteric lymph was diverted by ligating mesenteric lymph duct, and the MODS model was established by two-hit method. 45 Wistar rats were divided into three groups: mesenteric lymph duct ligation group, non-ligation group and sham group. All rats facilitated blood withdrawal for serum sample at pre-experiment and after 24 h. Then organs including kidney, liver, lung, heart and intestine were collected for preparing homogenate. The tumor necrosis factor-alpha (TNFα), nitric oxide (NO), malondialdehyde (MDA) and superoxide dismutase (SOD) were determined. RESULTS: It showed that the MODS model was established successfully. After two-hit, the TNFα, NO2-/NO3-, NOS, iNOS and MDA in serum of non-ligation group were significantly increased than that in pre-experiment and sham group, and SOD was significantly lower (P<0.01, P<0.05). NO2-/NO3-, NOS, iNOS and MDA in serum of ligation group were significantly increased than that in sham group (P<0.01), but TNFα, NO2-/NO3-, iNOS and MDA were obviously lower compared with non-ligation group and the SOD was significantly increased (P<0.01). Compared with sham group, the TNFα, NO2-/NO3-, NOS, iNOS and MDA in intestinal homogenate, NO2-/NO3-, NOS and MDA in renal homogenate, NO2-/NO3- and MDA in hepatic homogenate, and NO2-/NO3- in lung and heart homogenate in non-ligation group were significantly increased, SOD in intestinal homogenate was significantly lower (P<0.01, P<0.05), the NO2-/NO3-, MDA in renal homogenate and MDA in hepatic homogenate in ligation group were significantly increased (P<0.01, P<0.05). The NO2-/NO3- in intestinal, renal and hepatic homogenate of ligation group were significantly lower and SOD in intestinal and heart homogenate of ligation group were significantly increased than that in non-ligation group (P<0.01). CONCLUSIONS: The ligation of mesenteric lymph duct blocks the enterogenous

displacement of endotoxin, inhibits the release of TNFα, reduces the production of iNOS and the synthesis of NO, reduced the releasing of free radical and consuming of SOD. Lymphatic mechanism may play a role in the development of MODS.</FONT>

Key words Multiple organ failure Lymphatic system Ligation Nitric oxide Tumor necrosis factor Free radicals

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