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论著

CXCL10及其受体参与子宫内膜异位症发病的免疫机制初探

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摘要 目的： 通过研究趋化因子配体10（CXCL10）及其受体（CXCR3）的表达，探讨其参与子宫内膜异位症（EM）发病的免疫机制。方法： 分别运用ELISA法及化学发光分析法检测EM患者未经手术治疗组（76例）、经手术治疗组（10例）及正常体检人员组（76例）血清标本中CXCL10及癌胚抗原CA125浓度；分离并体外活化EM患者组（10例）及正常体检人员组（10例）外周血单个核细胞（PBMC），ELISA法检测活化后PBMC培养上清液中CXCL10的分泌表达水平、流式细胞术检测活化PBMC的表面分化抗原3（CD3）及CXCR3表达、RT-PCR检测CXCR3基因亚群（CXCR3A及CXCR3B）的表达；对结果进行统计分析。结果：血清CXCL10水平，EM患者未经手术治疗组、经手术治疗组及正常体检人员组间比较均具有显著差异（P＜0.05）；EM组与正常体检人员组比较：活化PBMC培养上清液中CXCL10的表达水平无显著差异（P>0.05）、CD3+/CXCR3+PBMC细胞数无显著差异（P>0.05）、EM组高转录表达CXCR3B亚群而正常对照组表达CXCR3A亚群。结论： EM患者的血清CXCL10缺陷表达可能是参与EM发病的免疫机制之一；EM患者PBMC对细胞活化信号具有有效的免疫反应性，但活化后的PBMC细胞表面高表达的是CXCR3B亚群、而非具有趋化效应的CXCR3A亚群，推测EM通过此种免疫逃逸机制而发病。

关键词 子宫内膜异位症 CXCL10 CXCR3 CXCR3B

分类号 R711.71

Immunologic mechanism of CXCL10 and its receptor involved in endometriosis

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Abstract

<FONT face=Verdana>AIM: To investigate the immunologic mechanism of CXC chemokine ligand 10(CXCL10) and its receptor CXC chemokine receptor 3(CXCR3) involved in the process of endometriosis (EM). METHODS: Serum samples were collected from 3 groups: EM patients without operation (n=76), EM patients with operation (n=10) and the normal control persons (n=76). CXCL10 and CA125 concentrations were detected by means of ELISA and chemiluminometry. Cell surface antigens on the activated PBMC-CD3 and CXCR3, as well as CXCR3 subgene-CXCR3A and CXCR3B were tested by flow cytometry (FC) and RT-PCR when PBMC was separated from women with EM (n=10) and without EM (n=10), and then activated. RESULTS: Serum CXCL10 concentrations between three groups were significanly different (P<0.05). Compared to normal control group, although the supernatant CXCL10 concentration and CD3+/CXCR3+PBMC number in EM group has no significant difference (P>0.05), highly expressed CXCR3B in EM group rather than CXCR3A was observed. CONCLUSION: CXCL10 in women with EM is low, indicating that it plays a vital role in the process of EM and immune system of the women with EM is defected and impaired. The immunoreactivity of PBMC from both EM patients and normal person is same to activated signal, but the productions are different: PBMC in EM group mainly express CXCR3B but PBMC in normal person mainly express CXCR3A after activation, which may be one of the immune mechanisms that EM escapes from immunological lethal effect of the infected host.</FONT>

Key words Endometriosis CXCL10 CXCR3A CXCR3B

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