论著

卵巢切除大鼠降钙素基因相关肽、乙酰胆碱和去甲肾上腺素血管效应的 变化

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摘要 目的 研究雌性大鼠卵巢切除和雌激素替代治疗4个月后,血管活性物质对离体动脉作用的改变。方法 采用双侧卵巢切除的雌性大鼠,分为假手术组、去卵巢组(8周龄切除双侧卵巢)和雌激素替代组(苯甲雌二醇40 μg, sc, 每日1次,卵巢切除后14 d开始给药至切除后4个月)。于给药结束时取升主动脉、胸主动脉及尾动脉,进行离体血管功能实验,观察对降钙素基因相关肽(CGRP)、乙酰胆碱(ACh)及去甲肾上腺素(NE)的反应。结果 卵巢切除4个月使CGRP引起的大鼠升主动脉舒张最大效应明显降低,而对胸主动脉无影响;同时ACh对胸主动脉的舒张效应曲线明显右移,而对升主动脉无影响。雌激素替代治疗使上述变化反转。去卵巢组的各类动脉对NE 的反应性无变化,而雌激素替代组的升主动脉和胸主动脉对NE引起的最大收缩效应明显降低。在尾动脉,去卵巢组和雌激素替代组的CGRP和NE反应性均无改变。结论 内源性雌激素可以通过多种途径调节血管反应性,发挥其心血管保护作用。本研究进一步提示对绝经期妇女进行激素替代治疗的重要性。

关键词 <u>卵巢切除术</u> <u>降钙素基因相关肽</u> <u>乙酰胆碱</u> <u>去甲肾上腺素</u> <u>动脉</u>

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Alteration of calcitonin gene related peptide, acetylcholine, and norepinephrine induced vascular responses in ovariectomized rats

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Abstract

AIM To determine the role of intrinsic estrogen in the modulation of vascular responses to various agents. **METHODS** Ovariectomized rats with or without estrogen (β-estrodiol-3-benzoate 40 μg, sc, once daily, 14 d after bilaterally ovariectomy at 8 weeks age for 4 months) replacement were raised for four months. Vascular responses to calcitonin generelated peptide (CGRP), acetylcholine, and norepinephrine were tested on three different arteries (ascending aorta, thoracic aorta, and caudal artery) by functional experiments. **RESULTS** Four months after ovariectomy, the maximal relaxation induced by CGRP was significantly lowered in ascending aorta but not in thoracic aorta. The EC₅₀ value for acetylcholine-induced relaxation shifted to the right in thoracic aorta but not in ascending aorta. Estrogen substitution reversed these effects. As for the response to norepinephrine, no difference was found after ovariectomy, but estrogen replacement lowered the maximal response to norepinephrine. Ovariectomy did not influence the response to CGRP and nore pinephrine in caudal artery. **CONCLUSION** Endogenous estrogen has protective effects on vasoresponsiveness through different mechanisms. This study gives further support to the hormone replacement therapy for postmenopausal women.

Key words ovariectomy calcitonin gene-related peptide acetylcholine norepinephrine arteries

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