Rapid effects of estrogen on intracellular Ca2+ regulation in junctional myometrium through the menstrual cycle in uteri with and without adenomyosis

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Introduction: The etiology of adenomyosis (ADS) is largely unknown. Dysfunctional contractility of the junctional zone (JZ) might be associated with the etiology of ADS. 17?-estradiol (E2) may regulate the contractility of JZ across the menstrual cycle by altering intracellular [Ca2+]

Materials /Patients and methods? 43 patients were recruited during February to November of 2014. 23 women who underwent hysterectomy for ADS were enrolled as the case group, including 10 samples collected during the secretory phase (SP) and 13 collected during proliferative phase (PP) of the menstrual cycle. The control group consisted of 20 patients with cervical intraepithelial neoplasia (CIN) III, including 7 samples at the SP, and 13 at the PP. Fresh junctional myometrium was obtained and processed for cell dispersion. We investigated membrane estrogen receptor-? (ER?) expression using Western blotting in JZ smooth muscle cells (JZSMCs). We also investigated the rapid effects of E2 on intracellular [Ca2+]i using laser scanning confocal microscopy.

Results: The membrane ER? expression in JZSMCs was comparably high, showing no cyclical change in ADS. E2 rapidly induced an increase in [Ca2+]i in JZSMCs in both groups. The [Ca2+]i flux was statistically different across the menstrual cycle in the control group, but not in the ADS. Also, the elevation of [Ca2+]i induced by E2 in the ADS was statistically greater than that in the controls, regardless of cycle phase. When pretreated with ER? antagonist ICI182, 780, the increase in [Ca2+]i was reduced in both groups showing no statistical differences. Filtered E-6-BSA removing free estradiol also induced [Ca2+]i flux. Removal of extracellular Ca2+ did not alter the effect of E2, but phospholipase C inhibitor U73122 and 2-aminooethoxydiphenyl borate, inhibitor of the inositol-1,4,5,-trisphosphate-gated intracellular Ca2+ channel, significantly reduced the estradiol-induced [Ca2+]i flux. E2 was unable to induce [Ca2+]i flux in thapsigargin-depleted cells. These results indicate that estradiol mediates [Ca2+]i flux in JZSMCs through ER? activating the phospholipase C pathway.

Conclusion: The rapid effects of E2 on [Ca2+]i flux in JZSMCs from ADS patients were different from those in controls. The abnormal intracellular [Ca2+]i response to E2 could account for the aberrant JZ peristalsis of ADS.

Mots clefs : Adenomyosis, junctional zone, contraction,estrogen, [Ca2+]i flux
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