

Abstract: P20

Inhibition of nitric oxide (NO) synthesis antagonises the oestrogen-induced increase in coronary blood

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1. Objective

Oestrogen receptors have been found in coronary arterial endothelial and vascular smooth muscle cells. Therefore, the present study was designed to determine if oestradiol-17 β and conjugated oestrogens can increase coronary blood flow and if so whether the changes are mediated by nitric oxide (NO).

2. Study design

Five oophorectomised non-pregnant sheep were chronically instrumented to measure blood pressure, heart rate, cardiac output, left circumflex coronary blood flow and central venous pressure. Animals received oestradiol-17 β or conjugated oestrogens (1.0 μ g/kg) and cardiovascular responses were followed for 135 min.

3. Results

Oestradiol-17 β (1.0 μ g/kg) increased the left circumflex (coronary) blood flow (28 \pm 3%), cardiac output (15 \pm 1%) and heart rate (13 \pm 3%). Coronary and systemic vascular resistance decreased by 23 \pm 5% and 12 \pm 2%, respiratory blood pressure did not change significantly. Conjugated oestrogens showed similar reactions. Administration of the nitric oxide synthetase inhibitor L-nitroarginine methylester (L-NAME) had no effect on basal coronary blood flow, but completely reversed oestradiol-17 β -induced increases in coronary blood flow.

4. Conclusions

These results demonstrate that oestrogen increases coronary blood flow in the non-pregnant sheep and that L-NAME, an inhibitor of nitric oxide, is able to reverse the oestrogen-induced flow changes.

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Abstract: P21

Endometrial monitoring in postmenopausal patients with breast cancer who are treated with tamoxifen: report of 207 cases

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1. Objective

To study the result of endometrial assessment in patients who were treated with tamoxifen.

2. Methods

Clinical and ultrasonographical data from 207 consecutive patients, who had been referred for endometrial assessment, were prospectively collected. The proportion of patients with endometrial pathology was studied, based on ultrasonographical or histopathological findings.

3. Results

An endometrial malignancy was histologically confirmed in 7 patients (3.4%): 4 well-differentiated and 1 moderately differentiated endometrioid adenocarcinomas, 1 serous papillary carcinoma and 1 metastatic breast cancer. The mean uterine volume and double endometrial thickness were 74 ml and 9 mm, respectively in all patients without endometrial malignancy; 88 ml and 17 mm in 41 patients with benign polyps; and 171 ml and 17 mm in patients with endometrial malignancy. Benign endometrial polyps were removed in 41 patients (19.8%). During a follow-up period ranging from 3 to 56 months none of the patients has clinically presented with endometrial cancer. No mortality from endometrial cancer occurred after a median follow-up of 24 months.

4. Conclusion

A high proportion of patients who are treated with tamoxifen have benign or malignant endometrial pathology. Ultrasonography is a reliable tool to detect endometrial pathology. However, a large randomised trial assessing the impact of endometrial monitoring on the mortality from endometrial cancer is needed to prove that endometrial monitoring improves survival in patients on tamoxifen.

Abstract: P22

Apoptosis and anti-apoptosis in oestrogen-receptor negative endometrial cancer cells in response to anastrozole, 4-hydroxytamoxifen and medroxyprogesterone acetate

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1. Purpose

Several studies have addressed the potential of anti-oestrogens and aromatase inhibitors to induce apoptosis in breast cancer cells *in vitro*. To our knowledge, the effect on endometrial cancer cells has so far not been evaluated. Our aim was to determine if anastrozole (ANAST) and 4-hydroxytamoxifen (4-OHT) had any effects on proliferating oestrogen receptor (ER)-negative endometrial cancer cell lines, in terms of apoptosis and the cell cycle distribution.

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