1. Objective

To study the clinical relevance of (sub)endometrial cysts in patients being treated with tamoxifen.

2. Materials and methods

The prospectively collected ultrasonographic data from 200 consecutive patients without endometrial malignancy were compared with the findings in 7 consecutive patients with endometrial malignancy, who had been referred for endometrial assessment. The proportion of patients with endometrial cysts was studied in both groups, based on ultrasonographic or histopathological findings.

3. Results

In 49% of all patients who are treated with tamoxifen (sub)endometrial cysts were found at ultrasonography. At histology these cysts are cystically dilated endometrial glands, often in the context of endometrial cystic atrophy or adenomyosis. Only 1 of the 7 patients with endometrial malignancy had endometrial (14%) and 1 had subendometrial (14%) cysts, whereas 29 (71%) of the 41 patients with benign endometrial polyps had anecheoic endometrial cysts. All patients with endometrial malignancy had hyperechoic or inhomogeneous endometrium and 1 patient had ascites.

4. Conclusion

The presence of (sub)endometrial cysts is not a suspicious sign in postmenopausal patients with breast cancer who are treated with tamoxifen. It is a typical finding in patients on tamoxifen, but it may hamper an accurate ultrasonographic endometrial assessment. Sonohysterography is helpful in difficult cases.

Abstract: P26

Differences in oestrogen receptor α variant messenger RNAs between normal human breast tissue and primary breast carcinomas

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1. Introduction

The presence of high levels of human oestrogen receptor alpha (hERα) is an important predictor of a favourable response of breast cancer patients to tamoxifen therapy. However, approximately 40% of all hERα-positive tumours do not respond to tamoxifen therapy. hERα variant mRNAs have been described extensively for breast cancer patients and are hypothesised to contribute to tamoxifen resistance. However, some studies have shown that the presence of hERα variants is not limited to malignant breast tissue.

2. Objective

In this study, we evaluated the differences in prevalence and functional activity of hERα between normal breast tissue and primary breast carcinoma using a functional assay in yeast (hERα-FASAY).

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3. Materials and methods

We examined 14 specimens of normal breast tissue that originated from cosmetic breast surgery (mean age: 35 years; standard deviation (S.D.) 11 years); 7 specimens of normal breast tissue that originated from primary tumour containing breast mastectomy specimens (mean age: 57 years; S.D. 14 years) and 41 specimens of primary breast carcinoma (mean tumour per cent is 89%; S.D. 18%) (mean age 64 years; S.D. 6 years) for the presence of wild-type and variant hERα.

4. Results

Firstly, we found that the presence of wild-type hERα, relative to the total amount of hERα present, differs markedly ($P < 0.0001$) between normal breast tissue (median: 85% wild-type hERα; S.D. 5%) and breast tumours (median: 74% wild-type hERα; S.D. 17%). Secondly, the hERα variants with altered function that are present in normal breast tissue are mainly one-exon deleted splicing variants (median: 100%; S.D. 11%), whereas in breast tumours only half of all variants lacks just one single exon (median: 50%; S.D. 22%) ($P < 0.0001$). Interestingly, despite age effect and the presence or absence of cancer, there are no apparent differences between both groups of normal tissue.

5. Conclusions

Thus, this indicates that the higher amount and different molecular structure of the variants detected in breast cancer are specific for tumour tissue and not for the complete breast with malignant disease. Furthermore, our results suggest that hERα-dependent oestrogen responsiveness of breast tissue will change during tumour outgrowth, indicating that specific hERα variants may play a role in breast cancer development or progression.

Abstract: P27

The modulation of oestrogen receptor-alpha activity by melatonin in MCF-7 human breast cancer cells

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1. Introduction

We have previously demonstrated that the pineal hormone, melatonin, can suppress oestrogen receptor-α (ERα) gene transcription and repress the mitogenic oestrogen-response pathway in human breast tumour cells.

2. Objective

To address the relationship between the oestrogen response pathway and the growth-inhibition observed in response to melatonin. To investigate the cell signalling pathway(s) by which melatonin, via its mt1 G-protein coupled receptor, modulates ERα activity, we examined the effect of melatonin on intracellular cyclic adenosine monophosphate (cAMP) levels in MCF-7 cells.

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