Aberrant Telomerase Expression in the Endometrium of Infertile Women with Deep Endometriosis

Fernanda A. Mafra, a Denise M. Christofolini, a Viviane Cavalcanti, a Fabia L. Vilarino, a Gustavo M. Andrê, a Patricia Kato, b Bianca Bianco, a and Caio P. Barbosa a

aIdeia Fértil Institute, Human Reproduction and Genetics Center, Faculdade de Medicina do ABC, Santo André/SP, Brazil
bTechnical Support, Applied Biosystems, São Paulo/SP, Brazil

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Background and Aims. Considering the complex cellular and molecular mechanisms involved in endometriosis formation and progression and the similarities concerning the association of endometriosis with tumorigenesis and metastasis, we hypothesized a possible relationship between telomerase and the development/progression of endometriosis. The present study aimed to evaluate the expression of telomerase in the endometrium and peritoneal endometriotic lesions from women with endometriosis and controls.

Methods. A case-control study was performed comprising 25 infertile women with endometriosis and 44 fertile women without endometriosis as controls. Samples of endometrium and endometriotic peritoneal lesions of the same patient were harvested in the late luteal phase of the cycle. The expression of hTERT and GAPDH genes was measured by mRNA using qRT-PCR based on TaqMan methodology. Student t test was used to compare the values between the groups; \( p \leq 0.05 \) was accepted as statistically significant.

Results. The mean expression of hTERT in the endometriosis group was significantly high when compared to the control group (1.24 \( \pm \) 4.67 vs. 0.31 \( \pm \) 1.10, \( p = 0.026 \)). When the expression of hTERT was compared in relation to disease stage, the group of moderate/severe endometriosis showed increased expression in relation to control group (2.59 \( \pm \) 7.35 vs. 0.31 \( \pm \) 1.10, \( p = 0.026 \)). Regarding endometriotic peritoneal lesions, only one (1/25) expressed hTERT mRNA. This patient had deep endometriosis.

Conclusions. There was an association between the expression of telomerase (hTERT mRNA) and the genesis and progression of endometriosis. © 2014 IMSS. Published by Elsevier Inc.

Key Words: Endometriosis, Endometrium, Infertility, Telomerase.

Introduction

Endometriosis is a common, benign, chronic estrogen-dependent gynecological disease characterized by the implantation, growth and development of endometrial tissue in extra-uterine sites that often results in dysmenorrhea, pelvic pain and infertility (1). It affects 3–10% of women in their reproductive years and 20–50% of women with infertility (2).

The human endometrium is a tissue with high cell turnover and marked cyclical remodeling under the influence of ovarian steroid hormones. Disturbances of this intricate hormonal regulation cause functional deficiencies and may result in conditions such as endometrial cancer, endometriosis and infertility (3).

The association of endometriosis with cancer is unclear. Although it is not neoplastic, certain processes characterizing metastasis and carcinogenesis are also seen in endometriosis, such as cell motility, adhesion, invasion, immunological factors, angiogenesis and metaplasia (4). Moreover, evidence exists that endometriotic lesions present genetic changes similar to certain malignancies (4,5).