Similar Characteristics of Endometrial and Endometriotic Epithelial Cells

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Abstract

Epithelial-mesenchymal transition (EMT) is characterized by the loss of epithelial and acquisition of mesenchymal cell characteristics. Our aim was to assess the epithelial phenotype in the pathogenesis of endometriosis with epithelial and mesenchymal markers. We used 2 structural (keratin-18, -19 [K18, K19]), 1 membrane-associated (mucin-1 [MUC1]), and 2 mesenchymal proteins (vimentin; zinc finger E-box-binding homeobox 1, [ZEB1]) to compare epithelial and mesenchymal characteristics in eutopic endometrium with the 3 endometriotic entities, peritoneal, ovarian, and deep infiltrating endometriosis (DIE). Quantitation showed no differences for K18, K19, and MUC1 between endometrium with and without endometriosis. Also, K18 was not different between endometrium and endometriotic lesions. In contrast, K19 and MUC1 were modestly but significantly decreased in the endometriotic lesions compared to endometrium. However, the maintained expression of epithelial markers in all investigated tissues, regardless of the pathological condition, clearly indicates no loss of the epithelial phenotype. This is further supported by the reduced presence of epithelial vimentin in endometriotic lesions which is in contrast to an increase in stromal vimentin in ectopic endometrium, especially in ovarian endometriosis. The ZEB1 increase in endometriotic lesions, especially in DIE, on the other hand suggests a role of partial EMT in the development of endometriotic lesions, possibly connected with the gain of invasive capabilities or stemness. Taken together, although we found some hints for at least a partial EMT, we did not observe a severe loss of the epithelial cell phenotype. Thus, we propose that EMT is not a main factor in the pathogenesis of endometriosis.

Keywords: endometriosis; endometrium; epithelial marker; epithelial-mesenchymal transition; mesenchymal marker.