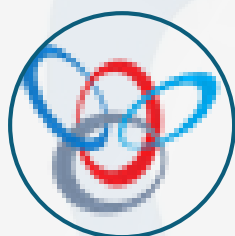


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Cervical-Endometrial Immune metabolome in Unknown genesis of Recurrent Pregnancy Loss

Abstract:

To investigate the mechanisms of endocervical cell proliferation disorders in women with RPL of unknown genesis by examining human papillomavirus (HPV) typing, and the expression levels of key molecular biological markers. A prospective observational comparative study was executed on women with RPL and healthy pregnant controls with full ethical approval. Samples were collected for HPV typing and immunocytochemical analysis to evaluate the expression of p16, Ki-67, BCL-2, and the anti-oncogenic microRNAs (miR-145 and miR-34a). The expression of mRNA for the progesterone receptor (PGR-A) was also assessed, alongside local immune status markers, including proinflammatory T-lymphocytes (Th17/Th1) and regulatory CD4⁺ Tregs. Overexpression of p16, Ki-67, and BCL-2 was observed in 52.5% of women with RPL who had an ASC-US/LSIL cytogram, with the average double expression of p16/Ki-67 being three times higher than in the healthy pregnant group. A significant decrease in PGR-A mRNA expression in the endocervix of women with RPL was noted, accompanied by a dysregulated local immune status characterized by an increased prevalence of Th17/Th1 cells and a reduction in regulatory CD4⁺ Tregs. The expression of miR-145 and miR-34a in the endocervix and endometrium of women with RPL significantly differed from the physiological pregnancy group, particularly in the context of high-risk HPV infection. The findings describe that disorders of endocervical cell proliferation in women with RPL of unknown genesis are associated with overexpression of specific molecular markers, impaired immune regulation, and altered microRNA profiles. These alterations may contribute to the pathophysiology of RPL, highlighting the need for further research into targeted interventions that could improve reproductive outcomes in affected individuals.

Keywords: recurrent pregnancy loss, miRNAa, HPV, p16/Ki-67