

CHAPTER 23

PCOS AND ENDOMETRIAL RECEPTIVITY

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How does endometrium receptivity change in PCOS?

Although endometrial receptivity of some patients having PCOS might not be altered during the window of implantation the underlying mechanism of PCOS associated subfertility remain elusive. While defective ovulation is a clear cause of subfertility in PCOS women arising data suggest that failed receptivity may also contribute to subfertility. Indeed, defects in biomarkers of endometrial receptivity that contribute to subfertility have been linked to a wide range of benign gynecological disorders including PCOS. Increasing evidence showed dysregulated expression of endometrial receptivity molecules in PCOS. Decline in the expressions of some receptivity markers such as integrin, HOXA-10 and IGFBP-1 have been noted in PCOS women. We, therefore, proposed that PCOS associated subfertility is not exclusively due to defective folliculogenesis but also the result of defective receptivity. As supportive, It has been reported that defect in receptivity might further complicate achieving pregnancy in PCOS. Although resumption of ovulation in anovulatory PCOS cases is easy with gonadotropin or clomiphene citrate implantation rates remain lower than we expect. Moreover, high miscarriage rates among PCOS subjects further support the possibility of receptivity defect.

The impact of PCOS related metabolic parameters on receptivity markers have been investigated comprehensively. Concordantly, high levels of serum androgens might cause failed fertility. Actually, PCOS women with hyperandrogenism have low HOXA-10 and β 3-integrin expression suggesting high androgens may have detrimental impact on endometrium. Increase in circulating androgens might antagonize the expression of estrogen-dependent receptivity genes that might lead to decline in HOXA-10 mRNA expression. Moreover, high serum androgen levels may directly inhibit the endometrial HOXA-10 mRNA expression. Therefore treatment aimed to reduce androgen levels may improve receptivity in PCOS. Moreover, increased expression of the steroid receptor coactivators may emphasizes the increased action of estrogen on endometrium in PCOS women. In contrast to negative impact of androgens on receptivity genes, high serum androgen levels may increase the number of preantral and antral follicles.

References

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