

CHAPTER 24

ENDOMETRIOSIS AND ENDOMETRIUM

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Is implantation of embryo into uterine tubes an indicator of embryo power or sign of receptivity defect?

Attachment of an embryo on tubal mucosa is not real implantation process. Conversely, this is a sign of endometrial receptivity defect. During tubal, abdominal, or cervical implantation process apposition, attachment, and invasion steps do not take place in certain way. Only endometrium has ability to exhibit privileged immunological reaction for implanting semi-allograft fetus. Other rare but possible implantation sites of embryo such as tuba uterina, peritoneum, and cervix have not ability to manage implantation of semi-allograft embryos. If good quality oocyte was the ability to attach any site of reproductive tract all embryos from young and healthy women was implanted on the tubal mucosa. The existence of thin endometrium and small endometrial cavity may prevent the arriving of healthy blastocyst into the endometrial cavity (1,2). Hence, implantation of an embryo out of intrauterine cavity may show defect in endometrium receptivity rather than embryo power (2).

What are the types of endometrial receptivity defect?

Two types of endometrial receptivity defect has been defined. In Type I endometrial defect delayed expression of $\alpha v \beta 3$ integrin has been noted. Many cases having out of phase endometrium show Type I endometrial defect. Type II is the occult form of endometrial receptivity defect and leads to decreased implantation rates. Endometrium in the type II defect is histologically in phase and normal in appearance. However expression of the endometrial $\alpha v \beta 3$ integrin decreased (3,4).

What are the most common diseases leading receptivity defect?

Endometriosis is likely the most common cause of endometrial receptivity defect especially in cases of minimal or mild disease for which mechanical reasons do not explain the loss of fertility. Concordantly, type II endometrial receptivity

tomy in hydrosalpinx, may improve the endometrium receptivity by decreasing the expression levels of NF-kB, central inflammatory marker, during the implantation window (8,9).

What is the interval necessary for the resolution of the inflammation and improving receptivity after endometrioma resection?

The interval necessary for the resolution of the inflammation and improving receptivity after the ovarian cystectomy depends on both intensity of inflammatory reaction and degree of failed receptivity. According to our clinical studies at least 3 months period was needed for both improving endometrial inflammation and receptivity gene expressions after endometrioma cystectomy (8,9).

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