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IMPLANTATION

In humans, implantation is the stage ofpregnancy at which the embryo adheres to the wall of the uterus. At this stage of prenatal development, the conceptus is called a blastocyst. It is by this adhesion that the embryo receives oxygen and nutrients from the mother to be able to grow.

In humans, implantation of a fertilized ovum is most likely to occur around nine days after ovulation; however, this can range between six and 12 days.

• Stages of implantation

Implantation consists of three stages:

(a) the blastocyst contacts the implantation site of the endometrium (apposition);

(b) trophoblast cells of the blastocyst attach to the receptive endometrial epithelium (adhesion); and

(c) invasive trophoblast cells cross the endometrial epithelial basement membrane and invade the endometrial stroma (invasion).

• Apposition: Implantation begins with apposition of the blastocyst at the uterine epithelium, generally about 2-4 days after the morula enters the uterine cavity. The implantation site in the human uterus is usually in the upper and posterior wall in the midsagittal plane. Implantation is considered a pro-inflammatory reaction in which endometrial vascular permeability is markedly increased at the attachment site, mediated by Cyclooxygenase (Cox)-derived prostaglandins. Prostaglandin E2 is increased in the luminal epithelium and the underlying stroma at the both of mice and human implantation site, thus indicating its role in attachment and localized endometrial vascular permeability.

• Cell adhesion of the blastocyst trophectoderm and endometrial luminal epithelial cells of the uterus is mediated by cell adhesion molecules, including integrins, cadherins, selectins, and immunoglobulinsCell adhesion molecules are expressed on the surface of invasive trophoblast, and these molecules interact with ligands expressed by the extra-cellular matrix of the decidua in a temporal and spatial way.

•Invasion: The process of implantation allows fetal trophoblast cells to invade and migrate into the maternal decidua. By this time, the trophoblasts at the implantation site have formed masses of cytotrophoblasts and syncytiotrophoblasts. Eventually, trophoblast cells destroy the wall of the maternal spiral arteries, converting them from muscular vessels into flaccid sinusoidal sacs lined with endovascular trophoblast.

The aim of invasion is to reconstruct the maternal spiral arteries, which will maintain a high blood flow between the fetus and the mother, replacing small, high-resistance vessels with large, low-resistance vessels. The extent of trophoblastic invasion determines later placental efficiency and fetal viability in late gestation. Deficiencies in trophoblastic invasion give rise to adverse pregnancy outcomes such as intrauterine growth restriction (IUGR) and preeclampsia.