Introduction

Placenta accreta is a placenta that implants too deeply and too firmly into the uterine wall. Similarly, placenta increta and percreta are placentae that imbed themselves even more deeply, into the uterine muscle or through the entire thickness of the uterine wall, sometimes extending into nearby structures such as the bladder ***(ACOG, 2002).***

Placenta accreta is potentially life threatening complication of pregnancy and it is one of the major causes of massive obstetric hemorrhage. With the increasing rate of cesarean delivery, the incidence of both placenta previa and placenta accreta is steadily increasing in frequency. It is not surprising then that there will be an increased rate of placenta previa accreta. In some recent series, placenta accreta has emerged as the major indication for peripartum hysterectomy accounting for 40–60% of cases. In view of this association, a patient with placenta previa and previous cesarean section is at high risk of having placenta previa accreta and is exposed to all the complication of placenta previa accreta, namely; massive postpartum hemorrhage and its resultant coagulopathy, cesarean hysterectomy and death. It would therefore be useful if antenatal diagnosis of placenta previa accreta can be made so that the necessary precaution and management can be instituted for the affected patient ***(Japaraj et al., 2007).***

Placenta accreta is classified into three types: (i) placental villi attachment to the myometrium (in the narrow sense); (ii) actual invasion of myometrium, when it is termed placenta

increta; and (iii) penetration through the myometrium in placenta percreta. The last two are more serious conditions and often require extensive life-saving surgical interventions because of uncontrollable bleeding at deliveries. As a consequence of placental invasion to adjacent organs in cases of placenta percreta, reconstruction of the bladder may also be necessary and maternal morbidity is high. For this reason, several surgical techniques such as prior embolization of the uterine arteries and balloon occlusion of hypogastric arteries during the operation have been employed to avoid massive bleeding. The condition is frequently diagnosed only after attempted manual removal of the placenta fails, so precise preoperative diagnosis is needed to allow appropriate preparation ***(Sumigama et al., 2007).***

From a clinical standpoint, the early antepartum recognition of placenta percreta with the bladder involvement is extremely important because patients are able to receive appropriate counseling with regard to the potential risk of uterine rupture with fatal hemoperitoneum or life-threatening hemorrhage in late pregnancy as well as appropriate surgical strategies. Early antepartum identification of placenta accreta/increta/percreta offers opportunity for life-threatening pregnancy termination ***(Chou et al., 2000).***

The first-trimester diagnosis should therefore be pursued aggressively in any woman with a placenta previa and an at-risk history of previous uterine surgery ***(Chou et al., 2003).***

The diagnosis of placenta previa accreta can be made with the use of ultrasonography. Gray scale ultrasonography, color Doppler and three dimensional color Doppler imaging have all

been described with varying specifically and sensitivity ***(Japaraj et al., 2007).***

Transvaginal sonographic findings of intraplacental lacunae in patients with placenta previa totalis and a history of Cesarean section are useful in the prediction of adherent placenta and may have a role in the prediction of clinical outcome ***(Yang et al., 2006)***

MRI offers a non-invasive method of diagnosis, with excellent tissue contrast and multiplanar capabilities and no apparent adverse effects on the fetus ***(De Friend et al., 2000).***

MRI may specifically be indicated in instances of placental implantation abnormalities in atypical locations not immediately accessible to evaluation with a high frequency transducer, such as posterior or fundal locations as would occur secondary to prior myomectomies in these areas ***(Maldjian et al., 1999).***

Aim of the Work

The aim of this study is to assess the value of using ultrasound, color Doppler versus MRI in prenatal diagnosis of placenta accreta.

Anatomy of normal Placenta

PLACENTAL DEVELOPMENT AND ANATOMY:

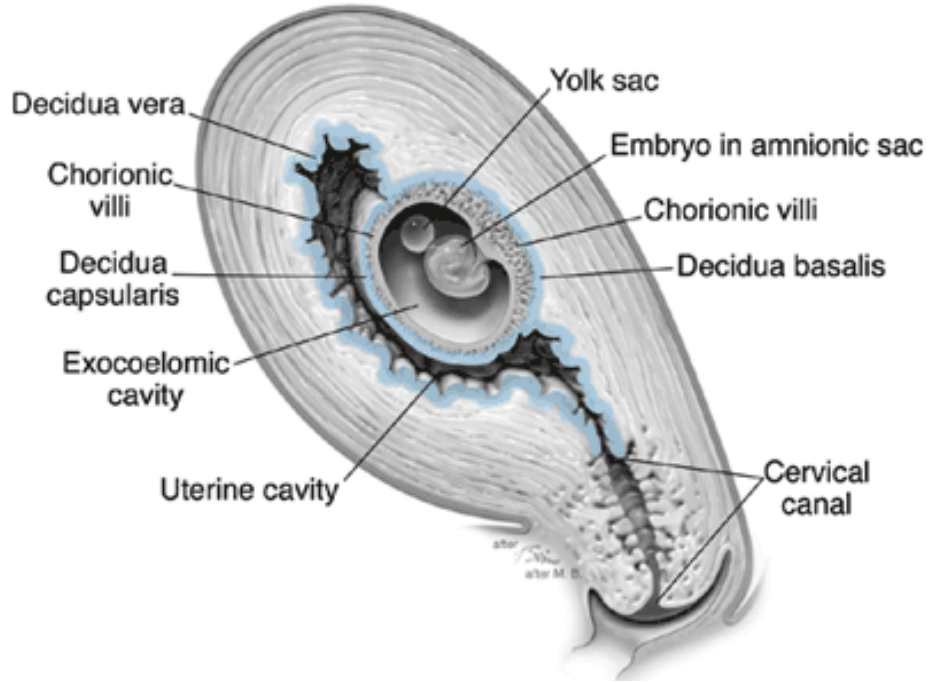
The Decidua of the Endometrium

The decidua is a specialized, highly modified endometrium of pregnancy and is a function of hemochorial placentation. Decidualization, the transformation of secretory endometrium to decidua, is dependent on the action of estrogen and progesterone and factors secreted by the implanting blastocyst during trophoblast invasion ***(Lala and colleagues, 2002).***

Decidual Structure

The decidua of pregnancy is composed of three parts based on its anatomical location. The portion of the decidua directly beneath the site of blastocyst implantation is modified by trophoblast invasion and becomes the decidua basalis. That portion overlying the enlarging blastocyst, and initially separating it from the rest of the uterine cavity, is the decidua capsularis **(Fig. 1)**. The remainder of the uterus is lined by decidua parietalis, sometimes called the decidua vera at the point in development when decidua capsularis and decidua parietalis are joined. ***(Damjanov, 1985).***

Figure (1): Decidualized endometrium covers the early embryo. Three



portions of the decidua (basalis, capsularis, and vera or parietalis) also are illustrated ***(Quoted from Gary et al., 2007)***.

The decidua parietalis and the decidua basalis, like the secretory endometrium, each are composed of three layers: a surface, or compact zone (zona compacta); a middle portion, or spongy zone (zona spongiosa), with remnants of glands and numerous small blood vessels; and a basal zone (zona basalis). The zona compacta and spongiosa together form the functional zone (zona functionalis). The basal zone remains after delivery and gives rise to new endometrium ***(Gary et al., 2007).***

Decidual Blood Supply

This supply is changed as a consequence of implantation. The blood supply to the decidua capsularis is lost as the embryo-

fetus grows and expands into the uterine cavity. The blood supply to the decidua parietalis through the spiral arteries persists, as in the endometrium during the luteal phase of the cycle. The spiral arteries in the decidua parietalis retain a smooth muscle wall and endothelium and thereby remain responsive to vasoactive agents that act on the smooth muscle or the endothelial cells of these vessels ***(Damjanov, 1985).***

The spiral arterial system supplying the decidua basalis directly beneath the implanting blastocyst, and ultimately the intervillous space surrounding the syncytiotrophoblast of the placenta, is altered remarkably. These spiral arterioles and arteries are invaded by the cytotrophoblasts, and during this process the walls of the vessels in the basalis are destroyed, leaving only a shell without smooth muscle or endothelial cells. As a consequence, these vascular conduits of maternal blood—which become the uteroplacental vessels—are not responsive to vasoactive agents. By contrast, the fetal chorionic vessels, which transport blood between the placenta and the fetus, contain smooth muscle and do respond to vasoactive agents ***(Gary et al., 2007).***

Decidual Histology

The decidua is composed of a variety of cell types, which varies with the stage of gestation. The primary cellular components of the decidua are the true decidual cells that differentiated from the endometrial stromal cells and numerous bone marrow–derived cells. The compact layer of the decidua consists of large, closely packed, epithelioid, polygonal, lightly

staining cells with round nuclei. Most of these are a particular type of natural killer lymphocyte and are referred to as endometrial large granular lymphocytes (LGLs), in which a special and unusual phenotype has been defined ***(Loke and King, 1995).***

Early in pregnancy, the spongy layer of the decidua consists of large distended glands, often exhibiting marked hyperplasia and separated by minimal stroma. At first, the glands are lined by typical cylindrical uterine epithelium. They have abundant secretory activity that contributes to the nourishment of the blastocyst. As pregnancy progresses, the epithelium gradually becomes cuboidal or even flattened, later degenerating and sloughing to a greater extent into the lumens of the glands. Later in pregnancy the glandular elements of the decidua largely disappear ***(Vince and Johnson, 2000).***

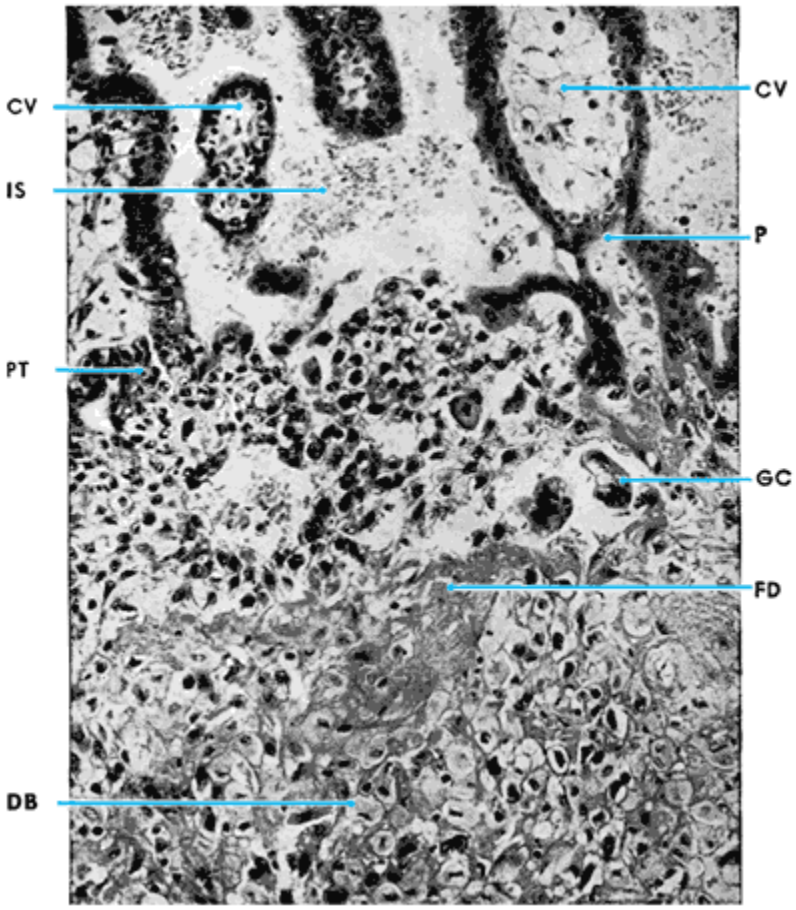


Figure (2): Section through junction of chorion and decidua basalis at fourth month of gestation. (CV = chorionic villi; DB = decidua basalis; FD = fibrinoid degeneration (Nitabuch layer); GC = giant cell; IS = intervillous space containing maternal blood; PT = proliferating trophoblast) ***(Quoted from Gary et al., 2007).***

Where invading trophoblasts meet the decidua, there is a zone of fibrinoid degeneration, the Nitabuch layer **(Fig. 2)**. Whenever the decidua is defective, as in placenta accreta, the Nitabuch layer is usually absent. There is also a more superficial, but inconsistent deposition of fibrin—Rohr stria—at the bottom of the intervillous space and surrounding the anchoring villi ***(Vince and Johnson, 2000).***

Implantation and Formation of the Placenta and Fetal Membranes

The development of the human placenta is as uniquely intriguing as the embryology of the fetus. During its brief intrauterine existence, the fetus is dependent on the placenta for pulmonary, hepatic, and renal functions. The placenta accomplishes these functions through its unique anatomical association with the mother. The placenta links the mother and fetus by indirect interaction with the maternal blood that spurts out of the uteroplacental vessels. This blood bathes the outer syncytiotrophoblast, allowing exchange of gases and nutrients with fetal capillary blood within the connective tissue at the villous core. Fetal and maternal blood are not mixed in this hemochorial type of placenta ***(Gary et al., 2007).***

Fertilization and Implantation

Ovum Fertilization and Zygote Cleavage

The union of egg and sperm at fertilization represents one of the most important processes in biology. Ovulation frees the secondary oocyte and the adhering cells of the cumulus oophorus from the ovary. Transport of the oocyte through the fallopian tube toward the uterus is accomplished by directional movement of ciliary action as well as peristalsis. Fertilization occurs in the fallopian tube. The steps involved to achieve fertilization are highly complex and have been the topic of much research ***(Primakoff and Myles, 2002).***

After fertilization in the fallopian tube, the mature ovum becomes a zygote—a diploid cell with 46 chromosomes—that then undergoes cleavage into blastomeres **(Fig. 3)**. As the blastomeres continue to divide, a solid mulberry-like ball of cells, referred to as the morula, is produced **(Fig. 3 D).** The morula enters the uterine cavity about 3 days after fertilization. The gradual accumulation of fluid between the cells of the morula results in the formation of the early blastocyst ***(Gary et al., 2007).***

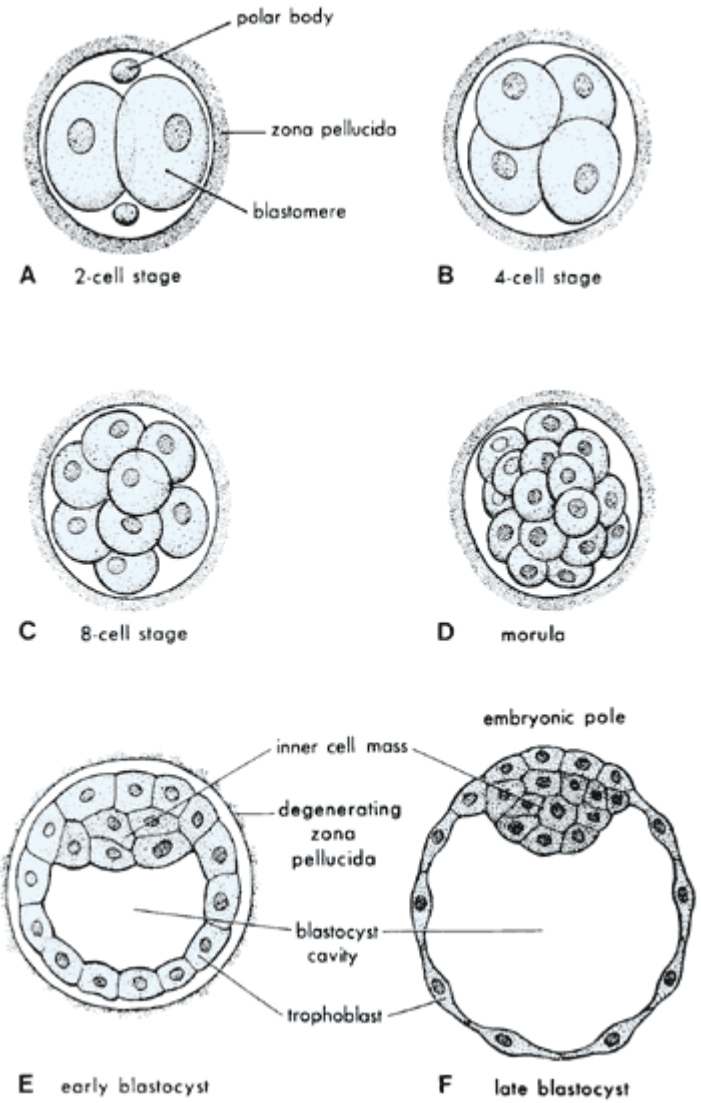


Figure (3): Cleavage of the zygote and formation of the blastocyst. A through D show various stages of cleavage. The period of the morula begins at the 12- to 16-cell stage and ends when the blastocyst forms, which occurs when there are 50 to 60 blastomeres present. E and F are sections of blastocysts. The zona pellucida has disappeared by the late blastocyst stage (5 days). The polar bodies shown in A are small, nonfunctional cells that soon degenerate ***(Quoted from Gary et al., 2007).***

In a 58-cell blastocyst, the outer cells, called the trophectoderm, can be distinguished from the inner cell mass that forms the embryo **(Fig. 3 E)** ***(Hertig, 1962).***

The 107-cell blastocyst is found to be no larger than the earlier cleavage stages, despite the accumulated fluid **(Fig. 3 F).** It measured 0.155 mm in diameter, which is similar to the size of the initial postfertilization zygote. At this stage the eight formative, or embryo-producing, cells are surrounded by 99 trophoblastic cells. It is at this stage that the blastocyst is released from the zona pellucida ***(O'Sullivan et al., 2002).***

Implantation of the Blastocyst

Implantation of the embryo into the wall of the uterus is a common feature of all mammals and in humans occurs six or seven days after fertilization. The ability of the blastocyst to adhere to the epithelium is mediated by cell surface receptors at the implantation site that interact with receptors on the blastocyst ***(Carson, 2002)***.

The blastocyst loosely adheres to the endometrial epithelium, a process called apposition, which most commonly occurs on the endometrium of the upper posterior wall of the uterus. ***(Gary et al., 2007).***

Successful adhesion of the blastocyst to the endometrium involves modification in the expression of cellular adhesion molecules. The integrins, one of four families of cell adhesion molecules, are cell-surface receptors that mediate the adhesion of

cells to extracellular matrix proteins ***(Lessey and Castelbaum, 2002)***.

Biology of the Trophoblast

The formation of the human placenta begins with the trophectoderm, which is the first tissue to differentiate at the morula stage of development, giving rise to a layer of trophoblast cells encircling the blastocyst. The trophoblast exhibits the most variable structure, function, and developmental pattern of all placental components. Its invasiveness provides for attachment of the blastocyst to the decidua; its role in nutrition of the conceptus is reflected in its name; and its function as an endocrine organ in human pregnancy is essential to maternal physiological adaptations and to the maintenance of pregnancy ***(Gary et al., 2007).***

Trophoblast Differentiation

By the eighth day postfertilization, after initial implantation of the blastocyst, the trophoblast has differentiated into an outer multinucleated syncytium, the primitive syncytiotrophoblast, and an inner layer of primitive mononuclear cytotrophoblasts.

After implantation is complete, the trophoblast further differentiates along two main pathways, giving rise to villous and extravillous trophoblast ***(Loke and King, 1995).***

Embryonic Development after Implantation

Early Trophoblast Invasion

After gentle erosion between epithelial cells of the surface endometrium, the invading trophoblasts burrow deeper into the

endometrium, and by the 10th day the blastocyst becomes totally encased within the endometrium. This process of erosion and invasion into the endometrium is carried out actively by the trophoblast cells. The mechanisms leading to trophoblast invasion into the endometrium are similar to the characteristics of metastasizing malignant cells. ***(Gary et al., 2007).***

One of the earliest implanting blastocysts discovered by ***Hertig and Rock (1945)*** is shown in **Figure 4**. It measured only 0.36 by 0.31 mm, and it was believed to have been in the process of penetrating the endometrium, with the thin outer wall of the blastocyst still within the uterine cavity. An implanting blastocyst at a similar stage of development, 9 days after fertilization, is shown in **Figure 5**. It appears to have been flattened in the process of penetrating the uterine epithelium.

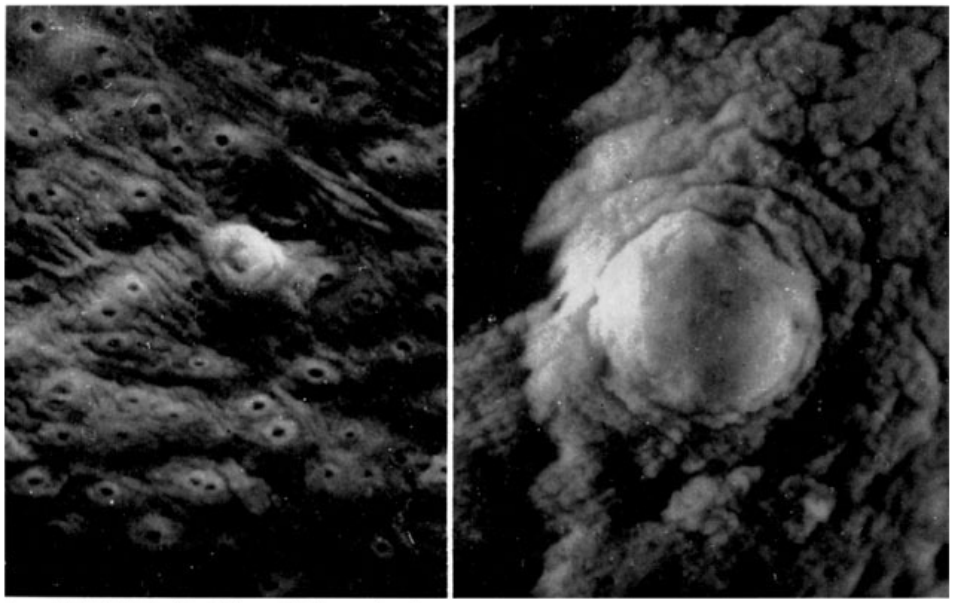


Figure (4): Low- and high-power photomicrographs of a surface view of an early implanted blastocyst obtained on day 22 of the endometrial cycle, less than 8 days after conception. The site was slightly elevated and measured 0.36 x 0.31 mm. Mouths of uterine glands appear as dark spots surrounded by halos ***(Quoted from Gary et al., 2007).***