PLACENTAL DELIVERY AT CEASAEREAN SECTION

There are different methods for the delivery of the placenta at caesarean section (1) placental drainage with spontaneous delivery (2) cord traction and (3) manual removal.

In placental drainage, the end of the umbilical cord is left unclamped, placental blood drained and placenta delivers spontaneously through uterine incision, this method is not widely used ***(Sharma, 1995).***

After the infant was delivered the cord was doubly clamped, then cut again above the proximal clamp to permit the placenta to drain, allowing for the free egress of the fetal blood, which was also promoted through milking of the cord blood into the pocket of the sterile drape. After no further blood

flow was observed, the placenta expelled spontaneously within three minutes after delivery, placental drainage of the fetal blood before spontaneous placental delivery significantly reduces the incidence of feto-maternal transfusion ***(Leavitt et al., 2007).***

The methods most frequently used are cord traction combined with external uterine massage or expression of the uterus and manual removal.

Cord traction involves gentle traction on the umbilical cord with external uterine massage after delivery of the baby and oxytocic has been given this method takes about three to five minutes ***(Cotter, 2001).***

Manual removal of the placenta done by the use of gloved hand of the surgeon with gentle sawing action to create a line of cleavage and separate the placenta from its placental bed, this method takes about two minutes to be done. "Since few women had spontaneous delivery of the placenta after 5

minutes, we recommend manual removal after 5 minutes, "Dr. Morales said".

In the spontaneous delivery group, surgeons waited up to 10 minutes with no systematic manual exploration of the uterine cavity. If the patient started bleeding excessively or didn't deliver the entire placenta within that time, the placenta was manually removed from the uterus.

In the manual removal group, surgeons cleaned out the placenta shortly after delivering the neonate. In the spontaneous delivery group, surgeons did not routinely clean out the uterine cavity or insert a hand, but simply checked to see if the placenta was completely delivered; if it was not, they manually removed it ***(Morales et al., 2004).***

Mechanism of placental separation:

The process of placental separation starts immediately after the delivery of the baby, when contraction and retraction of uterine muscles result

in reduction in the size of uterine muscle. Consequently, the placental bed to which the placenta is attached become smaller than the incompressible placenta, the placenta sheared off and blood vessels supplying the denuded placental bed are compressed by continued contraction and retraction of uterine muscle to reduce the bleeding so the separation of placenta results from disproportion between the incompressible placenta and the reduced size of the underlying implantation site ***(Cunningham et al., 2005).***

Separation of amniochorion

The greatest decrease in the surface area of the cavity of the uterus immediately causes fetal membranes and deciduas to be thrown into innumerable folds that increase the thickness of the layer from less than 1mm to 3-4 mm.

The membranes remain in situ until separation of placenta is nearly completed then they are peeled off the uterine wall partly by traction by the separated

placenta. Separation takes place along the plane of the stratum sponiosum and extends beyond the placental area so as to detach almost the whole thickness of the deciduas parietalis, remains of decidua capsularis ***(Cunningham et al., 2001).***

Hemostasis at the placental site:

Near term 600 ml/min of blood flows through the intervillous space. With separation of the placenta, there is also separation of many uterine arteries and veins that carry blood to and from the placenta. Usually, hemostasis in absence of surgical ligation depends on. Intrinsic vasospasm and formation of blood clots at the placental implantation site. Contraction and retraction of the myometrium to compress the vessels and obliterate their lumens ***(Cunningham et al., 2005).***

In the pregnancy, plasma fibrinogen levels are 300-600 mg/dL with activation of coagulation and these high levels protect against clinically significant

hypofibrogenemia and promote clinical coagulation at placental site after delivery ***(Cunningham et al., 2001).***

Complication of Placental Failure to Deliver

1- Retained placenta: retained separated placenta which either retained placental fragment or succinturate lobe presented by secondary postpartum haemorrhage.

2- Retained unseparated placenta as placenta accrete, percreta and increta. Placenta accreta is defined as abnormal adherence, either in whole or in part of placenta the afterbirth to the underlying uterine wall. Placenta accreta and other pathological placentations (such as increta, percreta) are rare complications of pregnancy with potential life threatening and fertility threatening consequences ***(Cunningham et al., 2005)***.

Definition:

Placenta accreta occurs where chorioic villi attach to the myometrium without normal intervening deciduas basalis; with the placental bed being partially or completely affected ***(Adler et al., 2014).***

A maternal mortality of 7% has been quoted previously for this condition ***(O'Brien et al., 1996).***

Incidence:

The incidence of placenta accreta has increased 10-fold over the last fifteen years, which reflects the increase in the rate of Caesarean Sections (CS) ***(ACOG, 2012).***

As the cesarean section rate and artificial abortion rates are rising, the placenta accreta rate is also increasing ***(Ac and Jm, 2013)***.

This incidence now occurs with a frequency of 1:533–1:2500 deliveries**.** This incidence varies from 1:540 in Thailand to 1:93,000 in the United States; the high incidence reported in Thailand may be

related to the increase prevalence of trophoblastic disease in Asia ***(ACOG, 2012).***

It has been suggested that the rarest form, placenta percreta, represent 5% to 7% of all abnormal placentations ***(Morken and Henriksen, 2001).***

Variants of Placenta Accreta:

The 3 variants of placenta accreta are classified by the extent of myometrial villus infiltration. Placenta accreta is typified by chorionic infiltration into the myometrium and percreta by infiltration through the entire myometrium to breach the serosa and beyond placenta incerta.

The most severe manifestations of this process result in placenta increta, when chorionic villi invade in to myometrium, and placenta percreta, when chorionic villi invade to or through the uterine serosa ***(Oyelese and Smulian, 2006)***.

The pathogenesis of placenta accrete has been well-characterized microscopically (e.g., poor decidualization with intramyometrial infiltration of the villous tissues) and macroscopically (e.g., prominent uteroplacental neo-vascularization in the region of interest) ***(Tseng et al., 2004).***

Aetiology of Placenta Accreta:

It related to the damage of the decidua basalis, which allows placental invasion into the myometrium. The barrier function of decidua is absent and the invasive trophoblasts may invade the myometrium ***(Morken and Henriksen, 2001).***

Risk Factor for Placenta Accreta:

Placenta accreta is becoming more common because of number of factors ***(Eshkoli et al., 2013)***, which include:

1- Placenta praevia with or without previous uterine surgery.

2- Previous myomectomy.

3- Previous caesarean section.

4- Submucosal fibroid.

5- Maternal age more than 35 years

6- Previous trophoblastic disease ***(Eshkoli et al., 2013).***

The relation between placenta praevia and placenta accreta:

Some studies reported that the risk of placenta accreta increased to 39% to 40% for those who had 2 caesarean sections ***(Khan et al., 2004).*** About 75% of placenta percreta cases are associated with placenta praevia.

Clinical Presentation:

Placenta accreta classically presents with retained placenta and hemorrhage and a life threatening acute uterine inversion and massive PPH during CS but seldom in a primigravida**.** The association between uterine inversion and placenta accreta is unclear, however, strong traction on the umbilical cord with fundal placenta, excessive

fundal pressure, relaxed uterus, short umbilical cord, uterine anomalies and antepartum use of magnesium sulphate are known associated factors ***(Hostetler et al., 2000).***

Uterine inversion and retained placenta accreta can both be fatal complications ***(Agarwal et al., 2005).***

Placenta percreta is associated with a maternal mortality reportedly as high as 10% and significant maternal morbidity, including massive haemorrhage, disseminated intravascular coagulation, hysterectomy, bladder and ureteric trauma, acute respiratory distress syndrome and acute tubular necrosis ***(Bennett et al., 2003).***

The average blood loss at delivery is reported to be 3000–5500 mL, which leads to significant postoperative morbidity and death ***(Leung et al., 2007).***

A recent retrospective study found an average blood loss of 3630 -2216 mL for placenta increta and 12,140-8343 mL for placenta percreta ***(Sumigama et al., 2007).***

Antenatal diagnosis of placenta accreta:

Ultrasound and MRI have been described to diagnose placenta accreta. However, prenatal diagnosis of placenta accreta remains a challenge. Most of placenta accreta was diagnosed in the third stage of labor ***(Wang et al., 2015 and Bauwens et al., 2014)***. One prospective population-based study of women diagnosed with placenta accreta found that only less than 50 % (66/133) were suspected antenatally. Antenatal diagnosis of placenta accreta reduced the ratio of haemorrhage and blood transfusion with statistical significance. So, antenatal diagnosis is very important for placenta accreta ***(Fitzpatrick et al., 2014)***.

1- Abnormal levels of biochemical markers like alpha-feto protein (AFP) and creatine kinase (CK)

have been linked with morbid adherence of the placenta. ***O'Brien et al., (1996)*** reported an association of elevated maternal alpha-feto protein with the extent of myometrial extrauterine invasion.

In the absence of fetal anomalies, unexplained elevated maternal serum AFP may suggest the presence of placenta percreta. AFP may be more useful than CK in this respect, as there are reports of cases of placenta percreta in which the CK level was normal ***(Singh et al., 2002).***

2- Placenta accreta is diagnosed ideally in the antenatal period by either sonographic or magnetic resonance imaging techniques. Several studies have demonstrated the usefulness of ultrasonography in making this diagnosis, particularly at 20 week gestation ***(Bauwens et al., 2014).***

Prenatal Ultrasound reported sensitivity of 94% and specificity of 79% for placenta accreta, but offer no more than provisional diagnostic probability

statement ***(Armstrong et al., 2004).*** Moreover, because 45% of placenta accreta cases were not detected by ultrasound, it is important to consider avoiding manual removal of placenta if there were intraoperative signs of accreta ***(Armstrong et al., 2004).***

The visualization of placental lacunae had the highest sensitivity to detect placenta accreta (78.6%), followed by obliteration of clear space (57%) and the interruption of the posterior bladder-uterine wall interface (21.4%) ***(Comstock et al., 2004).***

Management of placenta accreta:

There is debate over the ideal therapeutic approach or management of placenta accreta. The generally held opinion is that the placenta accrete should be treated by Caesarean hysterectomy with placenta left in situ, without attempts at removal of the placenta ***(ACOG, 2012).***

If clinically or sonographically the patient is suspected antenatally to be at risk of placenta accreta, appropriate management options should be considered, such as attempted conservative management or hysterectomy and counseling provided about potential sequelae ***(ACOG, 2012***).

Conservative management of abnormally invasive placentation can be effective and fertility can be preserved. It should be only considered in highly selected cases when blood loss is minimal and there is wish for fertility preservation ***(ACOG, 2012*****).**

There is no standard conservative treatment option for placenta accreta. Conservative treatment includes uterine artery embolization, methotrexate, mifepristone and hysteroscopic resection. Uterine artery catheterization can reduce risk of massive hemorrhage in the management of placenta accreta ***(Gustavo et al., 2015)***. Methotrexate has been used to promote the necrosis and resorption of placenta.

However, methotrexate has some serious side effects, women unable to breastfeed and a maternal death related to the use of methotrexate has been reported ***(Sentilhes et al., 2010***). Royal College of Obstetricians and Gynaecologists noted that methotrexate has little benefit in enhancing placental resorption ***(RCOG, 2011).*** Mifepristone has been successfully used in placenta accreta without any complication ***(Sentilhes et al., 2010***).

Leaving the placenta in situ after delivery can delay placental expulsion or resorption for weeks or even months. Resection of persistent retained placenta tissue by hysteroscopy can shorten this period and reduce the risk for infection and bleeding ***(Legendre et al., 2014)***. One large series on the conservative treatment of placenta accreta includes uterotonic drugs (oxytocin or sulprostone or both), prophylactic antibiotic therapy, methotrexate, pelvic arterial embolization. The success rate of this study

was 78.5 % (131/167) and the remaining 36 (22%) women had hysterectomy ***(Sentilhes et al., 2010***).

There is a study to describe the use of a staged procedure that involved femoral artery catheterization, classic caesarean section delivery, and uterine and placental embolization before hysterectomy for placenta accreta, it associated with decreased maternal morbidity ***(Tobias et al., 2010).***

In a recent review of management of placenta percreta, the authors included 36 women who were treated conservatively. In this group, 58% of the women underwent a subsequent hysterectomy. In addition, 44% suffered postoperative haemorrhage and 25% developed infections, compared with only 12% who developed postoperative haemorrhage and 12% who developed infections in those who had primary hysterectomy or local resection ***(Clausen et al., 2014).***

Placenta Praevia:

Placenta praevia refers to the presence of placental tissue overlying or proximate to the internal cervical os. Bleeding, which ranges from spotting to hemorrhagic, is the main complication.

Four placental configurations have been defined:

Complete placenta praevia — The placenta completely covers the internal os (Fig. 3). A central placenta praevia occurs when the internal os is approximately equidistant from the anterior and posterior placental edges. 20 to 30 percent of placenta praevias are central.

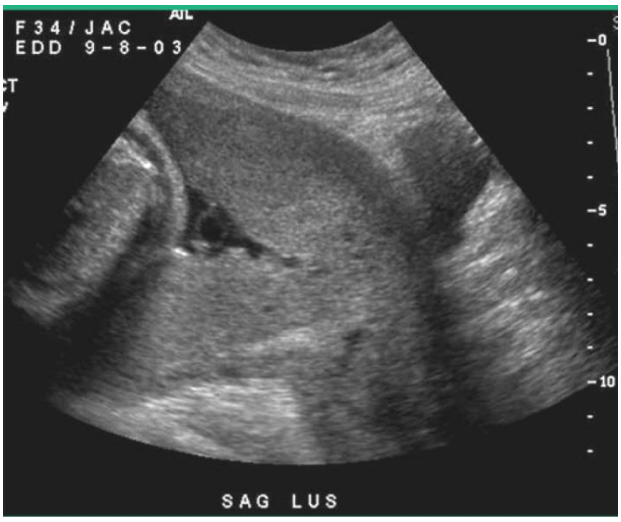

Figure (1): Transabdominal study shows the placenta completely covering the cervix. The placenta completely covers the internal os. A

central placenta praevia occurs when the internal os is approximately equidistant from the anterior and posterior placental edges. Twenty to 30 percent of placenta praevias are central.

Partial placenta praevia — The placental edge partially covers the internal cervical os, which must be partly dilated for this to occur.

Marginal placenta praevia — The placenta is adjacent to the internal os, but does not cover it (Fig. 4).

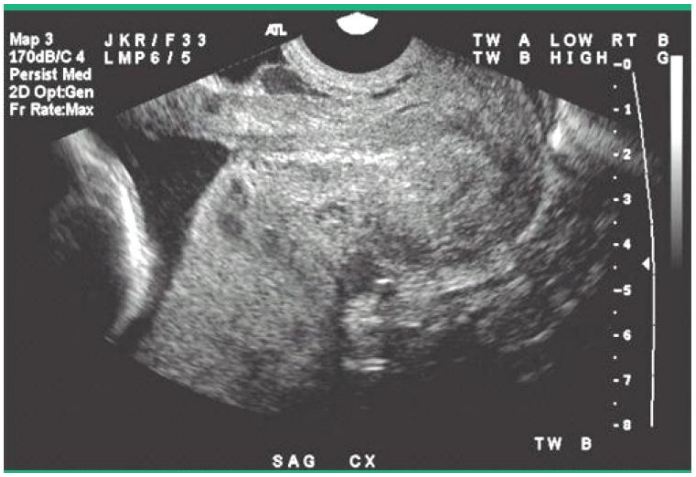

Figure (2): Transvaginal study shows a posterior placenta with the tip of the placenta on the internal os. The placenta is adjacent to the internal os, but does not cover it.

Low-lying placenta — This term is used in several ways: (1) to describe an apparent placenta praevia in the second trimester, (2) to describe a

placenta that lies in the lower uterine segment, but the exact relationship of the placenta to the os has not been determined, or (3) to describe a placental edge that lies within 2 to 3 cm of the internal os. Low lying placentas are also associated with an increased risk of bleeding, and possibly other adverse perinatal outcomes, although less than with true placenta praevias ***(Predanic et al., 2005).***

A recent classification of placenta praevia has been described by Royal College of Obstetricians and Gynaecologists by ultrasound imaging according to what is relevant clinically: if the placenta lies over the internal cervical os, it is considered a major praevia; if the leading edge of the placenta is in the lower uterine segment but not covering the cervical os, minor or partial praevia exists (***(RCOG, 2011)***.

Incidence, pathogenesis, and risk factors — Placenta praevia complicates approximately 4 per

1000 pregnancies that are over 20 weeks of gestation ***(Faiz et al., 2003).***

Risk factors can be grouped* according *to the pathogenetic mechanism involved:

(1) Endometrial scarring in the upper segment of the uterus may promote either initial trophoblastic nidation into the relatively unscarred lower uterine segment or unidirectional growth toward the unscarred lower uterine segment ***(Faiz and Ananth, 2003)***. Risk factors associated with endometrial scarring include:

- Increasing number of prior cesarean deliveries ***(Downes et al., 2015)***.
- Increasing parity (the incidence of placenta praevia is 0.2 percent in nulliparas versus up to 5 percent in grand multiparaious ***(Lavery, 1990).***
- Increasing maternal age (incidence is 0.03 percent in nulliparous women aged 20 to 29

years versus 0.25 percent in nulliparous women ≥40 years of age).

- Increasing number of prior curettages for spontaneous or induced abortions ***(Faiz and Ananth, 2003).***

(2) The need for increased placental surface area to compensate for a reduction in uteroplacental oxygen or nutrient delivery is another potential cause of placenta praevia. Risk factors associated with a need for increased uteroplacental transport of oxygen and nutrients include:

- Maternal smoking ***(Meyer and Tonascia, 1977).***
- Residence at higher altitudes
- Multiple gestation (3.9 and 2.8 praevias per 1000 live twin and singleton births, respectively) ***(Ananth et al., 2003).***

(3) Early gestational age is a risk factor for placenta praevia because placental migration away from the cervical os occurs as pregnancy progresses.

(4) Studies have consistently reported a preponderance of births of male infants in women with placenta praevia ***(Ananth et al., 2003 and Wen et al., 2000).***

A meta-analysis noted a 14 percent excess of placenta praevia when women were carrying a viable male fetus as compared to a viable female fetus (the worldwide normal sex ratio is 106 males to 100 females) ***(Demissie et al., 1999).*** One group postulated that male embryos are more likely to result from fertilization late in the menstrual cycle, and this may result in a delay in the development and implantation of the blastocyst, such that implantation occurs in the lower uterine segment ***(MacGillivary, et al., 1986).***

(5) An association between placenta praevia and maternal race has been observed. A large population-based cohort study reported that the rate of placenta praevia in white, black, and other races was 3.3, 3.0, and 4.5 per 1000 births,

respectively. Asian women had an excess risk compared with white women ***(Yang et al., 2008)***.

Clinical Manifestations:

The characteristic clinical presentation of placenta praevia is painless vaginal bleeding after 20 weeks of gestation; this occurs in 70 to 80 percent of patients. An additional 10 to 20 percent of women present with uterine contractions associated with bleeding, while fewer than 10 percent are incidentally detected by ultrasound examination and remain asymptomatic ***(Silver et al., 1984).***

Approximately one-third of affected pregnancies experience an initial bleeding episode prior to 30 weeks of gestation; this group is more likely to require blood transfusions and is at greater risk of preterm delivery and perinatal mortality than women whose bleeding begins later in gestation ***(McShane et al., 1985).***

Placenta praevia and accreta:

(Clark et al.1985) observed an increased incidence of placenta praevia after caesarean section from 0.26% in women with a normal uterus to 0.65% after 1 and up to 10% after 4 or more caesarean sections. Some studies reported that the risk of placenta accreta increased to 39% to 40% for those who had had 2 caesarean sections ***(Khan et al., 2004).*** About 75% of placenta percreta cases are associated with placenta praevia.

Malpresentation — The large volume of placenta in the lower portion of the uterine cavity may predispose the fetus to assume a noncephalic presentation ***(Sheiner et al., 2001).***

Intrauterine growth restriction — An increased risk of intrauterine growth restriction (up to 16 percent) has been reported by several ***(Brenner et al., 1978; Newton et al., 1984 and Ananth et al., 2001).*** But not all investigators ***(Shane et al., 1985; Comeau et al., 1983 and Crane et al., 1999).***

However, a population-based study of women with placenta praevia reported a small increase in the prevalence of infants with birth weight at least 25 percent below the range expected for gestational age (OR 1.37, 95% CI 1.25-1.50) ***(Ananth et al., 2003).***

Amniotic fluid embolism — A large population-based cohort study reported a strong association between placental pathology, such as placenta praevia, and amniotic fluid embolism ***(Abenhaim et al., 2002).***

Differential diagnosis

Third trimester bleeding complicates 3 to 4 percent of pregnancies. The differential diagnosis includes abruptio placentae (31 percent), and a variety of other causes (47 percent), such as decidual or cervical bleeding associated with labor or neoplasms ***(Hibbard and Jeffcoate, 1966).*** Sometimes a cause cannot be identified.

Ultrasonography

Transabdominal — The sonographic diagnosis of placenta praevia requires the identification of echogenic placental tissue overlying or proximate to the internal cervical os (a distance greater than 2 cm from the os excludes the diagnosis of praevia, although the placenta may be low lying). Transverse views at and above the internal cervical os should facilitate an accurate diagnosis.

Transvaginal — Transvaginal sonography is the preferred technique for diagnosis of placenta praevia because it generally provides a clearer image of the relationship of the edge of the placenta to the internal cervical os than transabdominal ultrasound ***(Smith et al., 1997).*** The procedure can be performed safely since the optimal position of the vaginal probe for visualization of the internal os is 2 to 3 cm away from the cervix and the angle between the cervix and vaginal probe is sufficient to prevent

the probe from inadvertently slipping into the cervical canal ***(Timor-Tritsch and Yunis, 1993).***

Translabial (transperineal) ultrasound imaging is an alternative technique that provides excellent images of the cervix and placenta ***(Dawson et al., 1996).***

Persistence after second trimester diagnosis — One to six percent of pregnant women display sonographic evidence of a placenta praevia between 10 and 20 weeks of gestation ***(Oyelese and Smulian, 2006).***

Women with placenta praevia at 15 to 19 weeks, 20 to 23 weeks, 24 to 27 weeks, 28 to 31 weeks, and 32 to 35 weeks had a praevia that persisted until delivery in 12, 34, 49, 62, and 73 percent of cases, respectively ***(Dashe et al., 2002).***

Magnetic resonance imaging — Utilizing MRI enhances the detection of posterior placenta praevia since the fetal calvarium does not obscure the

placental position**.** This modality is suited to the assessment of placental-cervical relationships owing to the differing magnetic resonance characteristics of the two tissues. We believe MRI usually does not contribute substantially to the diagnosis of placenta praevia, but can be helpful in the setting of accreta and percreta ***(Warshak et al., 2006).***

Abruptio placenta:

In abruptio placenta, postpartum uterine atony and Couvelaire's uterus can occur. Disseminated intravascular coagulation seems to result from a massive release of thromboplastin into the circulation, causing intravascular formation of fibrin, consumption of coagulation factors, and subsequent activation of the fibrinolytic system ***(Arias, 1993)***

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Use of ecbolics:

Careful consideration should be given to the timing of administration and to the type of oxytocic drug used. It is customary to give intravenous (IV) or intramuscular (IM) ecbolics like oxytocin 10-20 international unit (IU), ergometrine 0.2-0.4 µg, or syntometrine with crowning of the head or at delivery of the anterior shoulder. The practice of prophylactic administration of parenteral oxytocics in the active management of the third stage of labor has led to a 30% to 40% reduction in the incidence of postpartum hemorrhage ***(Chong et al., 2001).***

REFERENCES

Abenhaim HA, Azoulay L, Kramer MS and Leduc L (2008): Incidence and risk factors of amniotic fluid embolisms: a population-based study on 3 million births in the United States. *Am J Obstet Gynecol*; 199:49.

Ac W and Jm A (2013): Placenta accreta, increta, and percreta [J]. *Obstet Gynecol Clin North Am*; 40(1):137–54.

Adler E, Madankumar R, Rosner M, et al. (2014): Increased placental trophoblast inclusions in placenta accreta [J]. *Placenta*; 35(12):1075–8.

Agarwal S, Minocha B and Dewan R (2005): Uterine inversion and concomitant perforation following manual removal of placenta. *Int J Gynaecol Obstet*; 88:144–145.

American College of Obstetricians and Gynecologists: ACOG Committee on Obstetric Practice (2012): Committee opinion no. 529: placenta accreta. *Obstet Gynecol*; 120(1):207–211**.**

Ananth CV, Smulian JC and Vintzileos AM (2003): The effect of placenta previa on neonatal mortality: a population-based study in the United States, 1989 through 1997. *Am J Obstet Gynecol* 188:1299.

Ananth CV, Demissie K, Smulian JC and Vintzileos AM (2003): Placenta previa in singleton and twin births in the United States, 1989 through 1998: A comparison of risk factor profiles and associated conditions. *Am J Obstet Gynecol* 188:275.

Arias F (1993): Postpartum complications in practical guide to high risk pregnancy and

delivery Published by *Mosby- year Book Inc 2 nd ed. Eng.* P. 433-51.

Armstrong CA, Harding S and Dickinson JE (2004): Clinical aspects and conservative management of placenta accreta. *Obstet and Gynaecol*.

Bauwens J, Coulon C, Azaïs H, et al. (2014): Placenta accreta: can prenatal diagnosis be performed? Ultrasound and MRI interests. About 27 cases [J]. *Gynecol Obstet Fertil*; 42(5):306–11.

Bennett MJ and Sen RC (2003): Conservative management of placenta previa accrete report test two cases and discussion of current management options. *Aust NZ Obstet Gynecol*; 43: 249-51.

Brenner WE, Edelman DA and Hendricks CH (1978): Characteristics of patients with placenta previa and results of "expectant management". *Am J Obstet Gynecol* 132:180.

Chang YS, Su LL and Arulkumann S (2004): Current strategies for the prevention or postpartum haemorrhage in the third stage or labor. *Curr Opin Obstet Gynecol;* 16(2): 143-50.

Clausen C, Lönn L and Langhoff-Roos J (2014): Management of placenta percreta: a review of published cases. *Acta Obstet Gynecol Scand;* 93(2):138–143.

Comeau J, Shaw L, Marcell CC and Lavery JP (1983): Early placenta previa and delivery outcome. *Obstet Gynecol* 61:577.

Comstock CH, Love JJ, Bronsteen RA, Lee W, Vettraino IM, Hunag RR, et al. (2004): Sonographic detection of placenta accreta in the second and third trimesters of pregnancy. *Am J Obstet Gynecol;* 190:1135-40.

Cotter A, Ness A and Tolosa J (2001): Prophylactic oxytocin for the third stage of labour. *Cochrane Database of Systematic Reviews, Issue 4.*

Crane JM, van den Hof MC, Dodds L, et al. (1999): Neonatal outcomes with placenta previa. *Obstet Gynecol;* 93:541.

Cunningham FG, Gant NF, Leveno KJ, Gilstraf LC, Hauth JC and Wenstrom KD (2001b): Conduct of normal labor and delivery. *Williams 'obstetrics 21st Edition* published by McGraw–Hill Companies Inc; USA, P:309-29.

Cunningham FG, Leveno KJ, Hauth JC, Gilstrap LC and Wenstrom KD (2005): Normal labor and delivery. Volume (1) chapter 17 in *Williams Obstetrics 22nd edition*. Editor: P:823-35.

Dashe JS, McIntire DD, Ramus RM, et al. (2002): Persistence of placenta previa according to gestational age at ultrasound detection. *Obstet Gynecol*; 99:692.

Demissie K, Breckenridge MB, Joseph L and Rhoads GG (1999): Placenta previa: preponderance of male sex at birth. *Am J Epidemiol* 149:824.

Downes KL, Hinkle SN, Sjaarda LA, Albert PS and Grantz KL (2015): Previous prelabor or intrapartum caesarean delivery and risk of placenta previa. *Am J Obstet Gynecol; 212:669.e1*-6.

Eshkoli T, Weintraub AY, Sergienko R and Sheiner E (2013): Placenta accreta: risk factors**,** perinatal outcomes, and consequences for subsequent births. *Am J Obstet Gynecol;* 208:219.e1-7.

Faiz AS and Ananth CV (2003): Etiology and risk factors for placenta previa: an overview and meta-analysis of observational studies. *J Matern Fetal Neonatal Med* 13:175.

Fitzpatrick KE, Sellers S, Spark P, et al. (2014): The management and outcomes of placenta accreta, increta, and percreta in the UK: a population‐based descriptive study [J]. *BJOG Int J Obstet Gynaecol;* 121(1):62–71.

Gustavo I, César M, Mario G, et al. (2015): Feasibility and safety of prophylactic uterine artery catheterization and embolization in the management of placenta accreta.[J]. *J Vasc Interv Radiol Jvir;* 26:162–9.

Hibbard BM and Jeffcoate TNA (1966): Abruptio placentae. *Obstet Gynecol* 27:155.

Hostetler DR and Bosworth MF (2000): Uterine inversion – a life – threatening emergency. *J Am Board Fam Pract;* 13:120–3.

Khan AM, Dawalatly B, Khan D and Ded N (2004): An unusual presentation of placenta accrete. *J Obstet Gynecol*; 24: 180-1.

Lavery JP (1990): Placenta previa. *Clin Obstet Gynecol* 33:414.

Leavitt BG and Huff DL, Bell LA and Thurnau GR (2007): Placental drainage of fetal blood at caesarean delivery and feto-maternal transfusion. *Obstetrics and Gynecology;* 110(3): 608-611.

Legendre G, Zoulovits FJ, Kinn J, et al. (2014): Conservative management of placenta accreta: hysteroscopic resection of retained tissues. *J Minim Invasive Gynecol;* 21(5):910–3.

Leung TK, Au HK, Lee CM, et al. (2007): Prophylactic transuterine embolization to reduce intraoperative blood loss for placenta percreta invading urinary bladder. *J Obstet Gynecol Res;* 33: 722-5.

McShane PM, Heyl PS and Epstein MF (1985): Maternal and perinatal morbidity resulting from placenta previa. *Obstet Gynecol;* 65:176-89.

Meyer MB and Tonascia JA (1977): Maternal smoking, pregnancy complications, and perinatal mortality. *Am J Obstet Gynecol;* 128:494.

Morales M, Boulvain M, Ceysens G, Jastrow et al. (2004): Spontaneous versus manual placental delivery during caeasarean section: a randomized controlled trial. *American Journal of Obstetrics and Gynecology;* 187 (6 pt 2): S58.

Morken MH and Henriksen H (2001): Placenta percreta-two cases and review at the literature. *Eur J Obstet Gynecol Reprod Biol;* 100: 112-5.

Newton ER, Barss V and Cetrulo CL (1984): The epidemiology and clinical history of asymptomatic midtrimester placenta previa. *Am J Obstet Gynecol* 148:743.

O'Brien JM, Barton JR and Donaldson ES (1996): The management of placenta percreta: conservative and operative strategies. *Am J Obstet Gynecol;* 175:1632-8.

Oyelese Y and Smulian JC (2006): Placenta previa, placenta accreta and vasa previa. *Obstet Gynecol;* 107:927-41.

Predanic M, Perni SC, Baergen RN, et al. (2005): A sonographic assessment of different patterns of placenta previa "migration" in the

third trimester of pregnancy. *J Ultrasound Med* 24:773.

Royal College of Obstetricians and Gynaecologists (RCOG, 2011): *Green‐top Guideline No. 27.* Placenta praevia, placenta praevia accreta and vasa praevia: diagnosis and management.

Sentilhes L, Ambroselli C, Kayem G, et al. (2010): Maternal outcome after conservative treatment of placenta accreta [J]. *Obstet Gynecol;* 115(3):526–34

Sharma JB, Sharma WA, Newman MRB and Smith RJ (1995): Evaluation of placental drainage at caesarean section as a method of placental delivery. *Journal of Obstetrics and Gynaecology;* 15:237–9.

Sheiner, E, Shoham-Vardi, I, Hallak, M, et al. (2001): Placenta previa: obstetric risk factors and pregnancy outcome. *J Matern Fetal Med* 10:414.

Silver R, Depp R, Sabbagha RE and Dooley SL (1984): Placenta previa: aggressive expectant management. *Am J Obstet Gynecol* 150:15.

Singh M, Rane A and Green E (2002): Placenta percreta: four cases in one month. *Aust N Z J Obstet Gynaecol;* 42:82-4.

Smith RS, Lauria MR, Comstock CH, et al. (1997): Transvaginal ultrasonography for all placentas that appear to be low-lying or over

the internal cervical os. *Ultrasound Obstet Gynecol* 9:22.

Sumigama S, Itakura A, Or AT, et al. (2007): Placenta previa increta, precreta in Japan a retrospective study of ultrasound findings management and clinical course. *J Obstet Gynecol Res;* 33: 606-11.

Timor-Tritsch IE and Yunis RA (1993): Confirming the safety of transvaginal sonography in patients suspected of placenta previa. *Obstet Gynecol* 81:742.

Wang Q, Liu H and Dang Q (2015): Acute trophoblastic pulmonary embolism during conservative treatment of placenta accreta: case report and review of literature. *Eur J Med Res* 20:91.

Warshak CR, Eskander R, Hull AD, et al. (2006): Accuracy of ultrasonography and magnetic resonance imaging in the diagnosis of placenta accreta. *Obstet Gynecol* 108:573.

Wen SW, Demissie K, Liu S, et al. (2000): Placenta praevia and male sex at birth: results from a population-based study. *Paediatr Perinat Epidemiol* 14:300.