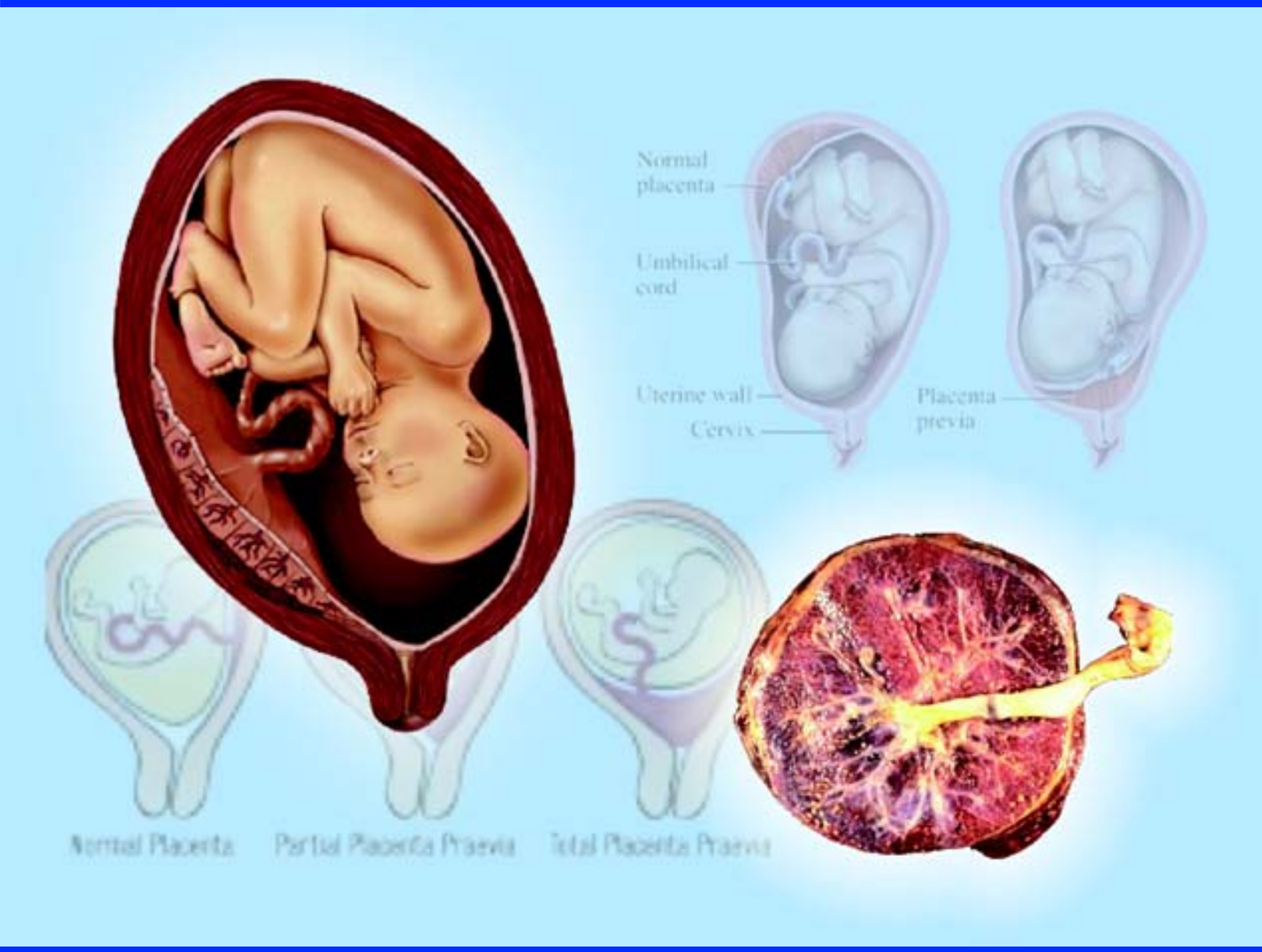
FOGSI FOCUS

January 2010



Antepartum Heamorrhage

Editor (s):
Prof. (Dr) Sheela V. Mane
Dr. B.S. Susheela Rani

The Federation of Obstetrics & Gynecological Societies of India

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FOGSI FOCUS EDITORIAL

Prof. Dr. Sheela V. Mane
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The entity 'Antepartum hemorrhage' is responsible for majority of the maternal deaths either directly through the torrential bleeding it causes or indirectly through making the woman vulnerable to life threatening complications. Management of major degree Placenta Previa is a challenge especially if the placenta is morbidly adherent or invading the bladder. Similarly, once placental abruption begins, it can progress rapidly and kill both mother and baby. Prompt delivery is the only way to save these lives. In this edition of FOGSI FOCUS we present chapters on Placenta Previa & Placental abruption with emphasis on guidelines for management of these life-threatening emergency situations.

We would like to acknowledge the encouraging response given to this edition of FOGSI FOCUS 2010. Our contributors have done great justice to the chapters bringing to our readers the current update on *Antepartum Haemorrhage* with the prevailing practices in India.

We hope that this volume measures up to the expectations of our readers and we look forward to serving them with equal enthusiasm in the future.

Destination Safe Motherhood
Dare to Dream
निरोगी – निर्भय – निश्चित नारी
MMR - From 300 to 30 in 3 yrs

President's message 2009

Women continue to die from pregnancy related causes at an alarming rate. Maternal mortality was first called a neglected epidemic in 1958.

The UN Millenium Development Goals call for a 75% reduction in maternal mortality in 2015, which will only be realized when priority setting, funding, and program implementation can create conditions for appropriate human resources, infra-structure, and patient education for high-quality obstetric care.

No longer a “neglected epidemic”, widespread knowledge of the high rates of maternal morbidity and mortality, and a 20+ year period of maternal mortality rate programs paradoxically highlight the fact that maternal mortality is reducing. Increased awareness may be one major outcome of the Safe Motherhood movement.

I congratulate the Safe Motherhood Committee for bringing to its readers current update on Antepartum Haemorrhage with the currently prevailing practices.

We have conducted more than 100CME and workshops on “Saving Lives” all over India.

The devotion of team of FOGSI can go a long way in helping its Members to understand the need of the hour and thereby achieve the goals of reduction in maternal mortality as soon as possible.

President message 2010

Pune

December 13, 2009

Dear friends,

At the outset I wish you all a very prosperous and a healthy new year and may all our wishes come true. I have been bestowed upon the honor of the President of FOGSI and it is my desire to make our organization the largest organization in the world. The maternal mortality of India is a continuous nagging issue in the minds of all of us and therefore through FOGSI we are constantly taking efforts to try and contribute towards helping to reduce this rate. This FOGSI Focus is one such endeavor to help all of us update our knowledge. I congratulate Dr. Sheela Mane, Chairperson Safe Motherhood Committee of FOGSI for this.

This year I have undertaken standardization, data collection and clinical research as the main plans of FOGSI. We also need to formulate detailed policies regarding clinical and ethical issues through our organization and we need to make our organization a role model for other such organizations in the country to emulate. This requires great vision collective hard work and perseverance through advocacy of right goals.

As the President of FOGSI and I bring in 2010 with **'Reaching The Unreached'** as the philosophy this year .This means reaching out with knowledge, reaching far and wide to all the members and also beyond the frontiers of the country and to reach the unreached goals .There are focused international conferences planned viz the International Conference on Contraception, February at Jaipur , the Satellite Conference, July at Bhubaneswar ,the World congress on Clinicopathothysiology of Pregnancy, August in Pune , the WHO conference In New Delhi , the Young Talent conference for Postgraduates and the Medicolegal regional conference . We also have planned about six various kinds of workshops to be conducted all over the country and over 25 research projects for you all to participate in. Please look out for the dates of the same.

As you are aware the National Eclampsia Registry was initiated through the ICOG-FOGSI in August 2008 and has received a great response from the members .It needs to be taken forward with more intense efforts as today we are in the possession of a strong data base . We will soon now be able to fill up the information online and that I feel is a new feather in the cap of FOGSI

I urge all FOGSI members to actively participate in all these projects which have been planned exclusively for you. Data collection and standardization are the two important aspects which will be stressed upon in all these projects.

Warm regards

Dr. Sanjay Gupte

President FOGSI 2010

Dr Duru Shah
Chairman, ICOG

Motherhood - a high price to pay

Every year about 515,000 women die from causes related to pregnancy and childbirth, a rate of over 1400 maternal deaths each day, and a little short of one death every minute, somewhere in the world of which 360 occur in India. Women who die are in the prime of their lives! As mothers, their deaths have a major health and social impact on their families. Moreover maternal death is only the tip of the iceberg of maternal morbidity and chronic suffering. For every maternal death which occurs, there are many women who survive serious illness with continued suffering and incapacity, such as with vesicovaginal and rectovaginal fistulae which cause a continuous leak of urine and stool. Only 1% of these deaths take place in developed countries, whilst 99% occur in the less developed countries. Whilst 10 women per 100,000 lose their lives during childbirth in USA, 1800 die in Ethiopia. In India, the average maternal mortality rate is 300 per 100,000 births. Maternal mortality is not a one time risk – it is a recurrent risk every time a woman gets pregnant. The cumulative risk with every pregnancy is 1 in 16 for a woman in Africa, 1 in 110 for a woman in Asia and 1 in 2500 for a woman in the developed countries such as USA, Canada, etc. Unsafe abortion is another important cause of mortality due to pregnancy. 200 women die every single day globally due to unsafe abortion. Annual mortality is 78000 with the major share being in the developing countries. 13% of unsafe abortion leads to maternal death.

A woman from the rural area goes through the journey of pregnancy and childbirth burdened with a heavy luggage of social injustice which she has accumulated as a girl, not considered to be her brother's equal, as a woman denied control of her fertility, and as a pregnant mother whose health is considered secondary to the

All pregnant women face some unpredictable and often unpreventable risks during pregnancy and childbirth. The majority of maternal deaths are caused by **severe bleeding (25%)** infection (15%) eclampsia / convulsions due to high blood pressure (12%) obstructed

labor (8%) and unsafe abortion (13%). It is estimated that 15% of pregnant women will require medical care to avoid death or disability from life threatening complications. To deal with such complications a facility should be able to provide Essential Obstetric Care which includes surgical procedures, anesthesia, blood transfusion, management of high blood pressure and convulsions and special care of the newborn. The WHO estimates that 99% of births in the developed countries are attended by skilled birth attendants whilst only 34% of them do so in the less developed countries. Antenatal care is availed by only 62% of our women as compared to 95% in developing countries and 20% in underdeveloped countries.

It is not possible for every woman to be delivered by a skilled birth attendant; hence human resources have to be developed. Health personnel such as primary health care physicians and nurses should be trained by the specialists so that they can take care of normal labor recognize when labor is not normal and transfer in good time to a facility were adequate help would be available.

The failure to address the avoidable causes of maternal mortality is a form of discrimination against women. The neglected tragedy of maternal mortality is not just a health problem, it is a violation of women's human rights! When over a half million women are dying every year with not a mention in global news it can be probably explained by the fact that it is **women** who are dying! If so many numbers were affected in a military conflict, there would be an international intervention! The crash of an aeroplane that costs over 350 civilian lives creates international headlines but with four times the same number dying every single day, how come we don't even hear a whisper?

I am happy to note that Dr. Sheela Mane, the Chairman of the Safe Motherhood Committee of FOGSI has focused on this subject of Antepartum hemorrhage. This is the need of the hour. I am confident that this issue will go a long way in improving and minimizing the complications of this dreaded problem which causes maternal death.

2nd Confidential FOGSI Survey of Maternal Mortality in India

Prof P K Sekharan
Vice-President FOGSI (2010)

Dear Colleague

Kindly spare a few minutes from your busy schedule and complete the appended datasheet summarising trends in maternal mortality* in your institution. The previous sample survey conducted in 2004-2005 allowed us to collect epidemiological and demographic information of great relevance to formulating public health policies in maternal and child health.

I am aware of the challenges in collecting this data and also appreciate that all the data may not be readily available. In teaching hospitals and district hospitals the delivery register should contain all the relevant clinical details which may be collected by postgraduate students and nurses under supervision. It is equally important to have data from smaller hospitals as well as from private institutions, especially in areas where a significant proportion of deliveries take place in the private sector. I would also encourage institutions to adapt the questionnaire to collect relevant data in the future.

The form may be completed electronically and emailed to drsekharanpk@hotmail.com. Alternatively a paper copy may be sent to: Dr P K Sekharan, Dwaraka, Near SBI Officers' Colony, Calicut - 673017, Kerala. You are welcome to clarify any queries by email or by phone (mobile: 9447156954).

Kindly send in your responses by end of March 2010.

In sincere appreciation of your co-operation and contribution to this survey.

* Please send the number of deliveries in your institution even if there are no maternal deaths.

Questionnaire

Name of the Institution (will be anonymised):

District: State:

Name and address of contributor:

	2007	2008	2009
Total no. of Deliveries			
Total no. of Caesarean Section			
Total no. of Maternal deaths			
Total no. of death due to PPH			
(a) Atonic			
(b) Traumatic			
Total no. of death due to APH			
(a) Placenta Praevia			
(b) Abruption			
Total no. of PIH			
No. of deaths due to PIH			
Total no. of Eclampsia			
No. of deaths due to Eclampsia			
No. of deaths due to Rupture Uterus			
No. of deaths following ectopic pregnancy			
No. of deaths following unsafe abortion			
Total no. of deaths due to sepsis			
(a) Pelvic			
(b) Extra-pelvic			
(c) Post operative			
Total no. of deaths due to medical illness			
(a) No. of deaths due to anaemia			
(b)			
(c) No. of deaths due to heart disease			
(d) No. of deaths due to viral hepatitis			
(e) No. of deaths associated with HIV/AIDS			
(f) No. of deaths due to thromboembolism			
(g) No. of deaths due to amniotic fluid embolism			
Others (specify_____)			
Total No. Perinatal deaths			

Index

1. Placenta previa, types and etiology

Dr. Sheela V. Mane
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Placenta Previa

Common causes of antepartum hemorrhage

Obstetric causes	Non-obstetric causes
Bloody show	Cervical cancer or dysplasia
Placenta previa	Cervicitis
Abruptio placenta	Cervical polyp
Vasa previa	Cervical erosion
Coagulation defects	Vaginitis
Uterine rupture	Vaginal laceration

Introduction:
APH: Bleeding occurring beyond 20 weeks of gestation prior to the onset of labour. Incidence 0.5%

Placenta previa & Abruption constitute 50% of APH.
Incidence of Placenta previa 0.5%, 20% of which are complete.

By definition, Placenta previa is described placenta that is inserted at least in part in the lower uterine segment. Williams definition: The placenta instead of becoming implanted in the body of the uterus well away from the cervical internal OS is located over or very near the internal OS.

Women with placenta previa may present with,

1. No bleeding
2. Recurrent episodes of bleeding where there is increased blood transfusion rate + increased incidence of post partum hysterectomy

Diagnosis is by Ultrasound scan. If transabdominal scan is uncertain or equivocal, transvaginal scan is done. Transvaginal scan is the gold standard for diagnosis of placenta previa. Transperineal & Translabial techniques are also used as diagnostic tools.

Adherent placenta is diagnosed by absent subplacental sonolucent space which is seen as hypoechoic RP zone. Colour Doppler is 87.5% diagnostic . MRI can also be done to diagnose the same.

Classification of Placenta Previa

Williams

1. Total placenta previa: Cervical OS is covered completely by placenta.
2. Partial placenta previa: OS is partially covered by placenta.
3. Marginal placenta previa: The placenta is implanted in the lower segment such that the placental edge does not actually reach the internal OS but is in close proximity of it.

Studd

1. The placenta is sited within lower uterine segment but the leading edge does not encroach on the internal cervical OS [Minimum of 20 mm]
2. The leading placental edge reaches but does not cover the internal cervical OS.
3. The placenta partially or asymmetrically covers the internal cervical OS.
4. The placenta wholly covers internal cervical OS.

Degree of placenta previa depends in a large measure on the cervical dilatation at the time of examination. This implies changing relations as cervix dilates.

Eg: A low lying placenta at 2 cms dilatation may become partial placenta previa at 8 cms because the dilated cervix has uncovered the placenta increasing the grade with dilatation

Conversely: Total placenta previa before dilatation may becomes partial as cervix dilates beyond the edge of placenta.

Etiology:

1. Multiparty – (Bakins et al)
2. Large placenta like in foetal erythyoblastosis and multiple gestation (No consistent evidence)
3. Defective vascularization of the decidua as a result of inflammatory or atrophic changes.
4. It is positively linked with maternal age 35 or more or age 20 years & less
5. Previous caesarean section – (Mittal et al)
   Anteriorly sited placenta previa is reported to be increased with prior caesarean section due to mechanical contribution of uterine scar influencing placental implantation. Rate of placenta previa increases 2 fold after 2 previous lower segment caesarean sections.
6. Uterine curettage or surgical abortion increases the risk factor by almost 2 fold.
7. Cigarette smoking is associated with 3-6 fold increase risk of placenta previa – (William et al)
8. Cocaine use is associated with an increased incidence by a factor of 2.4
9. Increased incidence in blacks.

References:

1. Williams Obstetrics
2. Progress in OBG 17th edition Jhon Studd, 13th chapter by Richard N. Brown
3. Selected Topics in Obstetrics and Gynaecology-4 by Shrish N Daftary & Shyam V.Desai, Antepartum Haemorrhage.

2. Clinical presentation of placenta praevia

Dr. Ambuja

Former superintendent, Institute of obstetrics & Gynaecology,
Former HOD, Dept. of Ob and Gyn,
Usmania Medical college, Hyderabad.

The placenta is partly or completely inserted into the lower uterine segment leading to Painless, causeless and recurrent bleeding.

1. The most common presentation is unprovoked painless vaginal bleeding or bleeding after sexual intercourse. Revealed hemorrhage is the only symptom .Without scanning one in six cases present for the first time in labor. Usually bleeding occurs after 20 weeks of pregnancy, near the end of second trimester or after. General condition is commensurate with the bleeding and the patient is seldom in shock at the first presentation as the initial episodes are usually mild and ceases spontaneously only to recur.

Some abortions however may result from such an abnormal location of the developing placenta. Placenta implanted near the internal os, not over the os does not bleed until the onset of labor, then it may vary from slight to profuse and clinically may mimic placental abruption.

Bleeding may continue after delivery because lower uterine segment contracts poorly compared with the body. Bleeding also may result from laceration in the friable cervix and lower uterine segment , especially fallowing manual removal of placenta of a some what adherent placenta. Placenta previa carries risk to the mother from massive obstetric haemorrhage, complications of surgery and anaesthesia, air embolism and postpartum sepsis.

2. on palpation uterus is felt to be relaxed and non tender.

The fetal parts are well felt. Fetal heart sounds are heard normally.

Fetal mal presentations and mal positions are seen in 30% of patients. Generally presenting part is high up and floating.

Firm clinical diagnosis of placenta previa is difficult.

Avoid vaginal examination as this may cause catastrophic bleeding.

Ultrasound examination is safe and generally reliable although false negative scans occour in 7% of cases. The latter are more common when the placenta is posterior, bladder is too full, the fetal head obscures the lower edge of the placenta, or the sonographer is inexperienced.The simplest, most precise and safest method of placental localization is by transabdominal sonography.Trans vaginal ultrasound has substantially improved the accuracy .

3. Pregnancy is at increased risk for a poor outcome like preterm labor, intra uterine growth restriction ,congenital malformation, malpresentation, fetal anaemaia and cord complications.. Leung and co (2001) found 62% risk of delivery within one week of bleeding when associated with contractions and a 13% risk even in the absence of contractions. Increased total fetal loss including abortions and prenatal deaths was 32 percent (Lipitz &colleagues 1991). During labor there is increased incidence of fetal bradycardia and late decelerations in posterior marginal placenta previa . With each uterine

contraction as the presenting part presses over the placenta blood supply is depleted.

The perinatal mortality rate was reported as 100% at less than 27 weeks, 19.7% between 33 and 37 weeks, and 2.6% after 36 weeks but is likely to be les with current management.

4. Incidence of placenta praevia is increased in multiparty and multiple gestation.(Ananth&co2003-40%higher incvidence) There is three fold increase in praevia with prior caesarean delivery. (Miller 1996) The incidence increased with number of previous caesarean deliveries.1.9% with previous 2 C S and 4.1 % with previous 3 C S .

So careful evaluation even with spotting is mandatory.

5.Smoking increases the risk twofold. (Williams & co 1991, Ananth 2003a).It is due to carbon monoxide hypoxemia leading to defective decidual vascularisation, inflammation leading to abnormal implantation.

6. Coagulation defects are rare because thromboplastins liberated by separation of placenta escape through the cervical canal and are not forced into maternal circulation.

3. Imaging Modalities in *Antepartum Haemorrhage*

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Imaging modalities to diagnose Placenta Previa:

Diagnosis of antepartum hemorrhage is first based on clinical suspicion. Clinically differentiation is possible between placenta previa and abruption. In patients with previous scar on uterus with antepartum hemorrhage the aim is to differentiate simple placenta previa from advanced forms like accreta or percreta. USG is used to confirm or diagnose asymptomatic patients with placenta previa or where differentiation is difficult between abruption placenta and placenta previa. MRI is used only to differentiate different degrees of myometrial invasion. USG with colour doppler can also be used to differentiate different degrees of myometrial invasion.

Various imaging modalities used to diagnose placenta previa includes:

- ❏ Ultrasound
- ❏ Magnetic resonance imaging

Placenta previa may be associated with placenta accreta or one of its more advanced forms, placenta increta or percreta. Such abnormally firm attachment of the placenta might be anticipated because of poorly developed decidua in the lower uterine segment. Almost 7 percent of 514 cases of previa reported by Frederiksen and collaborators had an associated abnormal placental attachment[1].

Ultrasonography:

Diagnostic ultrasound scanning is safe, accurate and noninvasive**,** and is the diagnostic method of choice. Routine placental localization is considered part of the anomaly scan at 20 to 22 weeks gestation. Unfortunately the earlier the scan is performed, the more likely the placenta is to be found in the lower pole of the uterus. For example, approximately 28% of placenta in women who undergo transabdominal scanning before 24 weeks are low, but by 24 weeks this number drops to 18%, and only 3% are low lying by term[2].

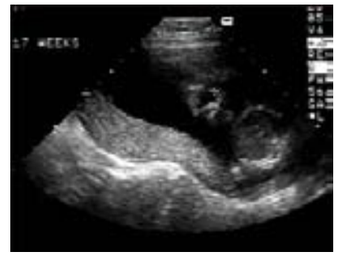

Fig 1: Second trimester scan showing posterior placenta reaching to the lower segment

Fig 1 shows second trimester scan showing posterior placenta following the curve of uterus and reaching to the lower segment. A false negative scan for a low placenta is found in as many as 7% of patients at 20 weeks[3]. These results are more common when the placenta is posterior, the bladder is overfilled, the fetal head obscures the margin of the placenta, or the operator doesn't scan the lateral uterine wall[4]. Comeau and associates and Ruparelia and Chapman[5,6] showed that the more advanced the pregnancy, the more accurate the diagnosis of placenta previa based on scanning findings.

Various types of ultrasound scanning which can be used include **Transabdominal, Transvaginal and Transperineal scanning.** When a transvaginal ultrasound scan is performed for placental localization and the distance between the lower edge of the placenta and the internal cervical os is measured, the persistence of a low lying placenta at later gestation is higher. Taipale and colleagues[7] observed that if a placenta overlapped the internal os by at least 25 mm at 18 to 23 weeks, the positive predictive value for placenta previa at delivery was 40%, with a sensitivity of 80%.

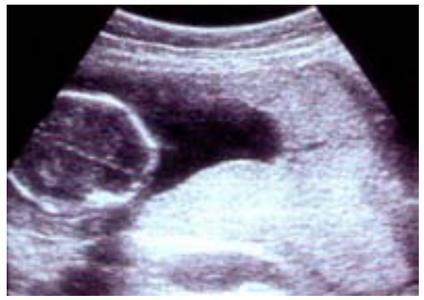

Fig 2 : Transabdominal ultrasound showing complete placenta previa

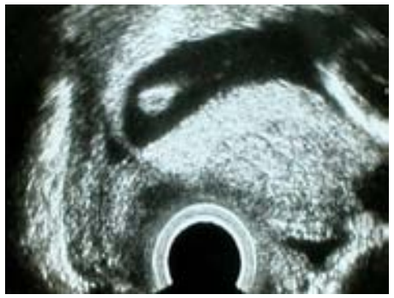

Fig 3: Transvaginal ultrasound showing complete placenta previa

Fig 2 and 3 shows placenta previa. Transvaginal ultrasound is more accurate than transabdominal in diagnosing placenta previa. If the internal cervical os can be visualized and if no placental tissue overlies it, major degree placenta previa is excluded. An attempt must be made to identify the inferior-most aspect of the placenta and to determine the distance between it and the internal os.

When the fetal head obscures a posteriorly positioned placenta or when the inferior placental margin is not visualized with transabdominal imaging, a transvaginal or transperineal approach is nearly always adequate in revealing its position. Two conditions misdiagnosed as placenta previa includes over distended bladder and myometrial contractions. Overdistention of the maternal urinary bladder places pressure on the anterior aspect of the lower uterine segment, compressing it against the posterior wall and causing the cervix to appear elongated. Thus, a normal placenta may appear to overlie the internal os. The cervix should be no longer than 3-3.5 cm during the third trimester. If the cervical length exceeds 3.5 cm or if a falsely elongated cervix is suspected, further imaging should be performed after the patient empties her bladder.

During a myometrial contraction, 2 situations that mimic placenta previa may occur**: First**, the wall of the uterus may thicken and imitate placental tissue. **Second**, the lower uterine segment may shorten and bring the inferior edge of the placenta into contact with the internal cervical os, creating a condition that mimics placenta previa. To avoid this pitfall, a contraction should be suspected if the myometrium is thicker than 1.5 cm. Findings from repeat imaging performed after 30 minutes should be sufficient to exclude this condition. With a qualified operator, sonography is more than 95% accurate. Transvaginal evaluation of the placenta has a 1% false-positive rate and a 2% false-negative rate.

Transabdominal sonography is the test of choice to confirm placenta previa. When the internal cervical os cannot be visualized or when the results are inconclusive, **transperineal or transvaginal sonography is recommended** as an adjunct. The overall accuracy of ultrasonography in the evaluation of placenta previa has been reported to be 93-98%. Transperineal studies have a negative predictive value of nearly 100% for this diagnosis. No increased risk of hemorrhage has been associated with transvaginal or transperineal sonography in this clinical setting.

The American College of Radiology (ACR) Appropriateness Criteria recommend against the use of the terms like total, partial and marginal previa in radiologic reports, because they are "vague and difficult to quantify." Instead, the ACR suggests describing the relationship between the placenta and internal cervical os.

M.R.I:

Usually, the placenta is relatively homogeneous. Its signal intensity on T1-weighted spin-echo images is low and slightly higher than that of the myometrium. On T2-weighted spin-echo images, placental tissue has high signal intensity, and it is clearly distinguishable from the adjacent fetus, uterus, and cervix. By increasing the T2 weighting, the placenta signal increases in intensity, facilitating the accurate location of the placental site. Sagittal images best demonstrate the placental position in relation to the internal cervical os. Detail of the internal architecture of the placenta may be visible. The intercotyledonary septa from the chorionic plate and venous lakes in the placental bed may be identified. The MRI appearance of the placenta is not homogenous, and within its substance are areas of increased signal intensity.

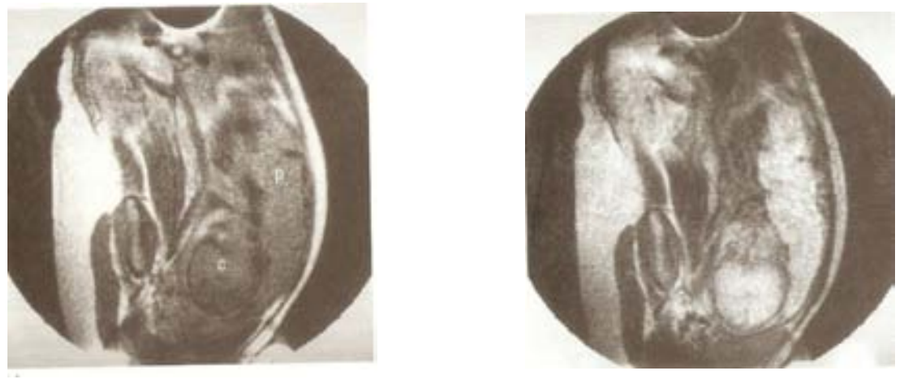

Fig 4: The placenta anatomical detail at 36 weeks gestation

Fig 4 shows the placenta anatomical detail at 36 weeks gestation on T2 weighted image.

Occasionally, endometrial veins may be seen at the margins of the placenta. Normal physiologic placental calcifications, which occur during late pregnancy, usually are not seen on MRI. The placenta in the first trimester of pregnancy occupies a far greater proportion of the internal surface area of the uterine cavity. The signal intensity of a T2 weighted pulse sequence is comparable to that of the placenta later in the pregnancy, but possesses a more uniform and high signal. The diagnosis of placenta previa is established on finding that placental tissue covers all or part of the internal cervical os. MRI is a technique which doesn't have the same operator dependence as ultrasound and also does not require the bladder to be distended. As the placenta and cervix are easily recognizable with MRI, the placental site may be accurately determined, as may the relationship between its lower edge and the internal os of the cervix.

Imaging modalities to diagnose placental invasion:

Placenta accreta is a condition in which all or part of the placenta is adherent to the uterine wall because of myometrial invasion by chorionic villi.

Accreta: chorionic villi are in contact with the myometrium, rather than being contained within the decidua. (80%)

Increta: extensive villous invasion in to the myometrium (15%)

Percreta: villous invasion extends to or through the serosal covering of the uterus (5%)

Prevalence of placenta accreta has raised considerably 1 in 25,000 deliveries8 a rise attributed to the rise in cesarean section rates. All three grades of placenta accreta are most commonly found in patients with history of uterine surgery, principally cesarean section, with the probability of placenta previa being accreta rising as high as 67% after four cesarean sections9. Placenta accreta is associated with placenta previa and advanced maternal age. This close association of both previous cesarean section and placenta previa means that placenta accreta rarely complicates vaginal delivery. In the series reported by Miller and collegues10 55 of the 62 cases were associated with placenta previa and all were delivered by cesarean section. The rate of placenta accreta was therefore only 1 in 22,000 in absence of placenta previa.

The diagnosis of placenta accreta can be made using colour flow Doppler ultrasound as early as the second trimester. Typical features of a placenta accreta include the following[11]

- The normal hypoechoic boundary between the placenta and the urinary bladder of serosa is lost.
- The placenta appears to be contiguous with the bladder wall
- Sonolucent spaces are visible within the placenta, adjacent to the uterine wall
- Color Doppler reveals persistent blood flow between the basal placenta and the myometrium[12]

A thickness of myometrial involvement greater than 1mm, accompanied by the presence of large placental lakes, can predict myometrial invasion with a sensitivity of 100% and specificity of 72%[13]. Chou and coworkers also described successful use of three dimensional color lower Doppler imaging for diagnosis of placenta accreta14

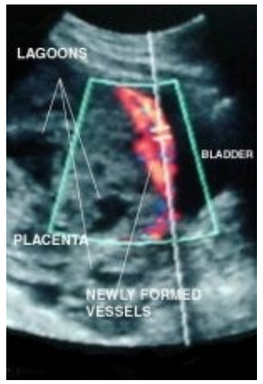

Fig 6: image of anterior placenta percreta
Above image shows colour doppler showing anterior placenta percreta. Doppler show newly formed vessels between uterus and the bladder. Multiple lacunae appear in the placental bed.

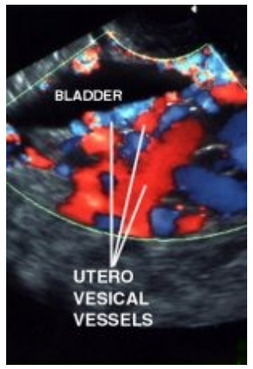

Fig 7: Placenta percreta by ultrasound

Above figure shows massive anterior placenta percreta. US Doppler shows the multiple layers of newly formed vessel between uterus and the bladder. The uterine segment and the vesical muscle are completely replaced by vessels.

The criteria for suspecting placental invasion are well known and may be summarized as follows:

1. Loss of retroplacental hypoechoic myometrial zone.
2. Presence of numerous vascular lacunae within placental substance.
3. Thinning or disruption of the linear hyperechoic boundary echo, representing the uterine serosa and its interface with the posterior wall of the bladder.
4. Focal nodular projections beyond the expected plane of the uterine margin.

Magnetic resonance imaging may be useful in the presence of a posterior placenta and for assessing deep myometrial, parametrial, and bladder involvement, or when the ultrasound findings are equivocal15. Maldjian16 and coworkers used MRI to diagnose placenta accreta, but Lam17 and colleagues reported that its sensitivity was only 38%. Baxi and associates found that elevated D dimers may predict significant blood loss and morbidity in women with placenta accreta, perhaps reflecting trophoblastic invasion in to myometrium and adjacent tissues.

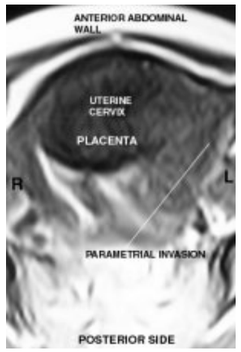

Fig 8: The placenta percreta invading the lateral wall of the uterus and additionally the parametrium.

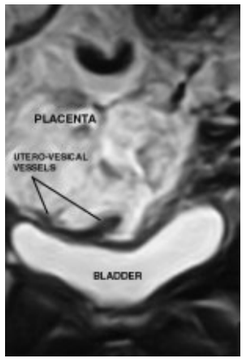

Fig 9: vascular invasion clearly to the vesical muscle in a case of placenta percreta

Imaging modalities to diagnose placental abruption:

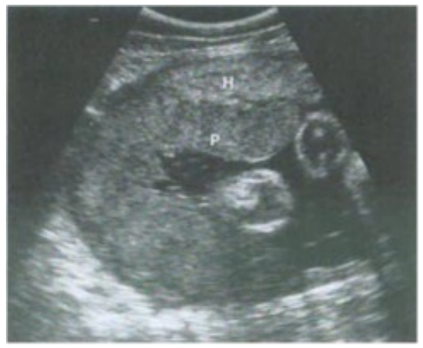

Fig 10: transabdominal ultrasound showing small retroplacental clot

The diagnosis of abruption is usually made on clinical grounds, but ultrasonography is helpful in some cases e.g., when there is a large retroplacental haematoma, although this is uncommon in severe cases. The symptoms and signs are diagnostic in moderate to severe cases. In mild forms, the diagnosis may not be obvious until after delivery; when a retroplacental clot is identified. Vaginal bleeding occurs in more than 70 – 80% of cases with abruption[18].

The quality and sensitivity of ultrasonography in detecting placental abruptions has improved significantly; however, it is not a sensitive modality for this purpose—findings are positive in only 25% of cases confirmed at delivery and the negative predictive value is low at around 50%.

Placental abruption shows as a retroplacental clot on an ultrasound image, but not all abruptions are ultrasonographically detectable.

In the acute phase, a hemorrhage is generally hyperechoic, or even isoechoic, compared with the placenta; a hemorrhage does not become hypoechoic for nearly a week.

Conclusion:

Diagnosis in patients presenting with ante partum hemorrhage involves clinical suspicion and imaging modalities. Abruption and previa can be suspected based on the clinical presentation. Placenta abruption is an obstetric emergency where the diagnosis is made clinically and further treatment is carried on. Ultrasonography is very less sensitive in diagnosis of abruption as mentioned above. Placenta previa a common cause of antepartum hemorrhage requires imaging modality to confirm the diagnosis as well assess degree of myometrial invasion if there is history suggestive of scar on the uterus. Colour Doppler as well as magnetic resonance imaging can be used to diagnoses degree of myometrial invasion. The management of patients with simple placenta previa and those with myometrial invasion differs significantly.

The role of MR imaging is less certain; although it may be invaluable in cases of posterior placenta previa with invasion, its lack of portability and expense restrict its overall usefulness.

REFERENCES:

1. Frederiksen MC, Glassenberg R, Stika CS: placenta previa: a 22 year analysis. Am J Ostet Gynecol 180:1432, 1999
2. Chapman MG, Furness ET et al: Significance of the location of placenta site in early pregnancy. BJOG 1989; 86:846-848
3. McClure N, Dornan JC: early identification of placenta previa. BJOG 1990;97:959-961
4. Laing FC: Placenta previa avoiding false negative diagnosis. J Clin Ultrasound 1981;9:109-113
5. Comeau J, Shaw L et al Early placenta parevia and delivery outcome. Obstet Gynecol 1983; 61: 577 – 580
6. Ruparelia BA, Chapman MG et al Early low lying placenta ultrasonic assessment, progress and outcome. Eur J Obstet Gynecol Reprod Biol 1985;20:209-213
7. Taipale P, Hiiesmaa V, et al Transvaginal ultrasonography at 18 – 23 weeks in predicting placenta previa at delivery. Ultrasound Obstet Gynecol 1998;12:422-425
8. Bider D, Dulitzky M, Goldenberg m et al Intraumbilical vein injection of prostaglandin F2alpha in RP. Eur J Obstet Gynecol Reprod Biol 1996;64:59-61
9. Zaki ZMS, Bahar AM, et al, Risk factors and morbidity in patients with placenta previa accretacompared to placenta previa non accreta. Acta Obstet Gynecol Scand 1998;77:391-394
10. Miller DA, Chollet JA, et al Clinical risk factors for placenta previa/placenta accreta. Am J Obstet Gynecol 1997;177:210-214
11. Herman A Complicated third stage of albour. Time to switch on the scanner (editorial) Ultrasound Obstet Gynecol 2000;15:89-95
12. Krapp M, Baschat A Gray scale and color doppler sonography in the third stage of labor for early detection of failed placental separation. Ultrasound Obstet Gynecol 2000;15:138-142.
13. Twickler DM, Lucas MJ, et al Color flow mapping for myometrial invasion in women with a prior cesarean delivery. J Matern Fetal MEd 2000;9:330-335
14. Chou MM, Tsng JJ et al Three dimensional color power doppler imaging in the assessment of uteroplacental neovascularization in placenta previa increata/percreta Am J Obstet Gynecol 285:1257,2001
15. Levine D, Hulka CA, Ludmir e t al: Placenta accreta: Evaluation with color doppler US, power Doppler US and MR imaging Radiology 1997;205:773-776
16. Maldjian C, Adam et al MRI apppearance of placenta percreta and placenta accreta. Magn Reson Imaging 17:965,1999
17. Lam G, Kuller J et al, Use of magnetic resonance imaging and ultrasound in the antentatl diagnosis of placenta accreta.J Soc Gynecol Invest 9:37,2002
18. Knuppel AR, Drukker JE et al Bleeding in late pregnancy: Antepartum bleeding. High Risk Pregnancy: A Team Approach. Philadelphia, Saunders,1986

4. Maternal and perinatal outcome in placenta previa

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Placenta previa, an important cause of antepartum hemorrhage. It is estimated to occur in 2.8/1000 singleton pregnancies and 3.9/1000 twin pregnancies and represents a significant clinical problem[1]

Perinatal Mortality- Perinatal mortality rates are three to four times higher than in normal pregnancies.[2] Preterm delivery is a major cause of perinatal death even with expectant management of placenta previa. Using U.S. linked birth and infant death data Salihu et al [3] found that the neonatal mortality rate was threefold higher in pregnancies complicated by placenta previa primarily because of increased preterm birth. In another large series, Ananth and associates[2] reported a comparably increased risk of neonatal death even for those fetuses delivered at term. Some of this risk appears related to fetal growth restriction and limited prenatal care. Although some investigators suggested earlier that congenital malformations are increased with previa, Crane and co-workers[4] were the first to confirm this. Their study controlled for maternal age and for reasons that are unclear, fetal anomalies were found to be increased 2.5-fold. Other causes of fetal death may be due to intrauterine asphyxia or birth injury.

Prematurity – This accounts for 60% of perinatal deaths in placenta previa. The Cochrane meta-analysis found that cerclage decreased the risk of premature birth before 34 weeks (relative risk = 0.45; 95% confidence interval, 0.23 to 0.87); however, it is recommended that additional studies of cerclage be performed before this clinical practice is introduced.[5]

Fetal hemorrhage- although all the blood loss from placental accident is maternal, some fetal blood loss is possible, particularly if the substance of placenta is traumatised. Hemorrhage due to tearing of placenta occurs with vaginal manipulations, especially upon entry to uterine cavity at caesarian section. About half of caesarean babies lose some blood. Fetal blood loss is directly proportionate to the time lapse between lacerating the cotyledons and clamping the cord. Bleeding from vasa previa is the only cause for pure fetal blood loss, but fortunately it is rare.

Growth restriction-It is uncertain if there is associated fetal growth restriction with a previa. Brar and colleagues (1988) reported that the incidence was nearly 20 percent. Conversely, Crane and co-workers (1999)[4] and many recent studies found no increased incidence of fetal growth restriction after controlling for gestational age.

Long term sequel- Long term follow up of infant delivered of women with placenta previa at all gestation shows normal growth and psychomotor development but a small increase in neurological abnormalities.[6]

Maternal morbidity and mortality-

Placenta previa was associated with 30% maternal mortality in a series reported in 1962.[7] Maternal mortality has been drastically reduced with the improvement in obstetric care. The major cause of death in placenta previa reported is hemorrhagic shock and a few cases of thromboembolism.

Antepartum hemorrhage- Bleeding is more likely to commence early in pregnancy if previa is of major degree. Menon et al(1966) reported the first onset of bleeding prior to 32 weeks in 47.9% of cases of type III & IV as against 33.3 % in type I &II for the same period of pregnancy. Earlier in the pregnancy the manifestation, the worse is the prognosis. Initial bleeding episode (warning hemorrhage) is rarely associated with maternal mortality.

Postpartum hemorrhage- PPH is common following delivery of placenta. It is due to uterine atony, which is again due to limited ability of lower segment to contract down and stop bleeding. Associated accreta may further complicate the situation. The relative risk of placenta accreta in the presence of placenta previa is 1:2065. The risk of placenta acreta in the presence of placenta previa increases dramatically with number of previous CS, with a 25% risk for one prior CS, and more than 40% for two prior CS.[8] Placenta accreta is a significant condition with high potential for hysterectomy, and a maternal death rate reported at 7%. Secondary PPH may result due to infection and sub involution. A low lying placenta predisposes for post partum infection.

DIC- Bleeding may be compounded by prolongation of bleeding time and low platelet count, secondary to DIC occurring as a sequel to massive hemorrhage. More commonly it is due to dilutional coagulopathy from blood loss with crystalloid replacement or transfusion of stored blood which is deficient in coagulation factors.

Placental abruption- Placenta previa is also at greater risk of placental abruption.

Maternal morbidity- it is difficult to quantify but remains high. Prolonged hospitalization, blood transfusion, operative delivery presents relatively minor risks compare to classical caesarean section, Caesarian hysterectomy. Morbidity associated with placenta previa is increased in women with prior caesarian section, recurrent hemorrhage, Placenta acreta.

Factors affecting maternal and perinatal outcome- the maternal mortality for placenta previa is nil in most developed countries. Unfortunately it is still 1-2% in most developing countries. This is mainly due to delayed diagnosis, inadequate blood transfusion facility, delay in transfer of the patient to health care facilities. In the developed country the abnormal position of placenta is diagnosed by routine use of sonography and allows time to make adequate arrangement for deciding the time, place, and mode of delivery. The routine use of ultrasound is not available in all the centers in the developing countries, especially in rural areas & hence a greater chance of mortality.

Conclusion

Although occurrence of placenta previa, premature labor, placental separation, cord accidents, uncontrollable hemorrhage cannot be avoided , both the perinatal and maternal mortality and morbidity rate can greatly be reduced if ideal obstetric and newborn care is given. Accurate and early diagnosis by transvaginal or trans abdominal sonography, aggressive expectant management, use of caesarean section, availability of blood and blood products, expert anaesthesia and sophisticated neonatal care facilities are important in the management to avoid adverse maternal and fetal outcome.

References :

1 Ananth CV, Demissie K, Smulian JC, Vintzileos AM. 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 2003;188: 275–81.

2 Ananth CV, Smulian JC, Vintzileos AM. The effect of placenta previa on neonatal mortality: a population-based study in the United States, 1989 through 1997. Am J Obstet Gynecol 2003;188:1299–304

3 Salihu HM, Li Q, Rouse DJ, et al: Placenta previa: Neonatal death after live births in the United States. Am J Obstet Gynecol 188:1305, 2003 [PMID: 12748503]

4 Crane JMG, Van Den Hof MC, Dodds L, et al: Neonatal outcomes with placenta previa. Obstet Gynecol 93:541, 1999 [PMID: 10214830]

5 Neilson JP. Interventions for suspected placenta previa. Cochrane Database Syst Rev 2003;(2):CD001998.

6 Naeye RL Placenta previa – predisposing factor and effect on fetus and surviving infants. Obstet Gynecol 1978;52:521-525)

7 Macafee CH, Miller WG M aternal and fetal mortality in placenta previa. L Obstet Gynecol Br Common Wealth 1962;69:203-212

8 Miller DA, Chollet JA, Goodwin TM. Clinical risk factors for placenta praevia -placenta accreta. Am J Obstet Gynecol 1997;177:210–4.

9 Crane JM, Van den Hof MC, Dodds L, Armson BA, Liston R. Maternal complications with placenta previa. Am J Perinatol. 2000;17:101–5

10 Droste S, Kell K Expectent management of placenta previsa cost benefit analysis of outpatient treatment Am J Obstet Gynecol 170;1254-57,1994

11 JOAN M. G. CRANE, MD, Neonatal Outcomes with Placenta Previa Obstetrics & Gynecolo VOL. 93, NO. 4, APRIL 1999

12 S. R. Singhal, N. & S. Nanda : Maternal And Perinatal Outcome In Antepartum Hemorrhage: A Study At A Tertiary Care Referral Institute . *The Internet Journal of Gynecology and Obstetrics.* 2008 Volume 9 Number 2, ISSN: 1528-8439S,

5. Management of Placenta previa

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Early diagnosis & Expectant management

1. *Confirm diagnosis, exclude placenta accreta*

With the availability of ultrasound imaging for antenatal assessment, the diagnosis of placenta previa is possible *before* any vaginal bleeding occurs. This puts the caregiver on alert for a possible bleed at any time. Improved imaging technology now enables us to identify abnormal placental invasion too. Color Flow Doppler ultrasound should be performed in women with placenta previa who are at a particularly increased risk of placenta accreta (eg. those with an anterior placenta previa with previous caesarean delivery). Where this is not possible locally, the woman should be managed as if they have placenta accreta until proven otherwise.

2. *Exclude fetal bleeding due to vasa previa in a low lying placenta*

Clinically, the presenting part is high and mobile, malpositions are common. Unless the blood loss is major, there is no fetal compromise. When there is evidence of fetal distress or demise following a minor vaginal bleed, it is important to exclude fetal exsanguination from a bleeding vasa previa by doing a Kleihaur test on the discharged blood.

3. *Admit and evaluate*

Women with major degrees of placenta previa with a history of vaginal bleeding, however small, should be admitted and managed as inpatients from 34 weeks of gestation. This is because each subsequent episode of bleeding is going to be bigger than the preceding one. Asymptomatic women, having never bled, require careful counseling before contemplating outpatient care. Any home-based care requires close proximity with the hospital and the constant presence of a companion.

3. *Obtain informed consent for expectant management*

Once a diagnosis of placenta previa is made and confirmed, the condition has to be explained to the pregnant woman and her immediate family, and their understanding and co-operation sought for efficient management. While placenta previa Grades I and II may be able to deliver vaginally, the need for Cesarean delivery at any point of time must be explained. A placental edge less than 2 cm from the internal os is likely to need delivery by cesarean section, especially if it is posterior or thick.

The idea of expectant management is to reach a gestational age that will maximize the likelihood of fetal maturity and minimize the risk of hemorrhage that may result from the normal onset of

uterine contractions. Successful expectant management culminates in a planned cesarean section on completion of 37 weeks. Delaying delivery beyond 37 weeks of gestation does not benefit the fetus, and increases the risks for the mother. Placental site bleeding during a cesarean section for placenta previa can be troublesome, and therefore, it is important that an experienced Obstetrician is on the scene.

During expectant management,

1. **Ongoing counseling of the woman and her family:** The woman must be advised to report to hospital immediately in the event of any vaginal bleeding, however small.
2. **Quick correction of anemia:** This may require packed cell transfusion. Consider IV iron therapy or auto transfusion in those who may decline blood transfusion.
3. **Arrange for adequate blood back-up:** This may be organized with a blood bank or with cross-matched donors who will be available at any time until delivery, for an emergency.
4. **Antenatal steroids:** Two doses of Betamethasone 12 mg IM, 24 hours apart, to be given for RDS prophylaxis in the newborn because of the high possibility of preterm delivery.
5. **Antepartum Anti – D prophylaxis** may be given to those women who are Rh negative, and have presented with bleeding.
6. **Plan delivery in a tertiary center:** This ensures availability of trained personnel, OT facility, anesthesia services, NICU facility and blood transfusion services round the clock.
7. **Hospitalization Vs ambulatory care:** In patients who remain stable for a period of days after an initial episode of bleeding, the need for continued hospitalization is controversial. In selected patients, outpatient management is reasonable following the first episode of bleeding. If bleeding recurs, prolonged hospitalization may be necessary.
8. **Threatened preterm labor:** This may be controlled with a tocolytic agent to buy time for steroiding and planning safe delivery.
9. **Sign out:** Expectant management should be called off when bleeding continues / becomes severe or at 37 weeks.

From the patient's side, it pays to follow certain precautions: these may not be supported by strong evidence, but it is safer to err on the side of caution!

1. "Pelvic rest" until a follow-up ultrasound indicates that the placenta is no longer covering the cervix. This specifically means no intercourse, no orgasms, no vaginal douches and no pelvic examination by any caregiver.
2. Avoidance of all strenuous activities, such as running or lifting more than approximately 20 lb (9.1 kg) is strongly advised. While walking for about 30 minutes a day may be safe, strenuous exercise must certainly be avoided.
3. To remain close to a hospital that can provide emergency care for both the mother and a sick or premature infant. It is advised to be as homebound and stress free as possible in the last trimester; and staying horizontal rather than vertical has also been found to be beneficial.
4. Having a phone or a companion nearby at all times is useful.
5. Most importantly, patients are strongly advised to **reach hospital if there is any vaginal bleed, however small.** They are counseled to be prepared for hospitalization / delivery at any time.

Delivery

Vaginal delivery is permissible if the placenta praevia is minor and the head is engaged. All other cases of placenta praevia with a viable foetus need to be delivered by caesarean section. In remote areas where there are no facilities for caesarean section or transfer, foetus is not viable and the patient is bleeding actively, placenta is compressed by a foetal part ***if*** it is accessible at the cervix. In a cephalic presentation, the head is pulled down by its scalp with Willet/ Tenaculum forceps[1] and in a breech presentation the foetus is pulled down by a leg. This helps in minimising blood loss and facilitating delivery.

Caesarean Section

Caesarean Section is performed,

1. Electively at the completion of 37weeks in all patients who are managed conservatively
2. Any time in a patient with uncontrollable haemorrhage.

Prerequisites:

1. Ultrasound examination to show the margins of the placenta, the extent of its invasion(accrete/increta/percreta)
2. Matched compatible blood – a minimum of 4 units of packed cells or whole blood if packed cells are unavailable. Blood should be available in the OT for immediate use and not just reserved.
3. A senior obstetrician and anaesthetist, well equipped OT and geared up personnel are very important. For this reason, all these patients must be operated in centres where intensive care is available unless the patient presents as an emergency and cannot be transferred.
4. An informed consent. Patient and her relatives should be explained about the risks involved in a Caesarean section for placenta praevia. The possibility of blood transfusions, hysterectomy if bleeding is unmanageable and the risks thereof should be explained. Risks to the baby like prematurity, difficult extraction, exsanguination and the necessity for a NICU care should be explained.

Anaesthesia: Earlier, general anaesthesia was preferred to regional anaesthesia because the peripheral dilatation associated with regional anaesthesia was thought to be detrimental. However, several studies have shown that regional anaesthesia is safe and epidural is superior to general anaesthesia.

Problems that can be encountered at Caesarean section. These problems can be compounded by foetal malpresentations.

1. Poorly formed lower segment since many of these cases are delivered preterm

   This problem can be compounded by a high presenting part. Incision can be extended in J, U or inverted T fashion. In some cases a classical incision may be required.
2. Large vessels in the line of incision. These can be ligated individually before incising the uterus.
3. Placenta at the site of incision. This is possible in an anterior placenta praevia. Once the uterine incision is made and placenta encountered, the operator's fingers are insinuated between the placenta and the uterine muscle to reach the nearest edge of the placenta. Membranes are ruptured at this site and the foetus delivered[2]. Blood loss from the separated placental tissue is inevitable and can exsanguinate the foetus if there is a delay in delivery. The other alternative is to cut through the placenta and quickly deliver the foetus. Again, delay in delivery can cause troublesome haemorrhage and exsanguination of the foetus.

4. Bleeding from the placental bed. Because of the poor contractile nature of the lower segment, bleeding can be troublesome and difficult to arrest. The following methods are useful in reducing the blood loss.

   a) Bimanual compression, intravenous oxytocics

   b) Intramyometrial injections of Prostaglanding F2á or dilute Vasopressin(5units in 20ml saline)

   c) Over sewing the bleeding points with figure of eight sutures.

   d) Compression sutures. B Lynch brace suture allows continuous vertical compression of the vascular system. The sequence of suturing is quite difficult to remember in an emergency without access to the original description because any one individual is likely to perform this procedure infrequently. Hayman's modification of B Lynch suture is as effective but simpler to perform. It involves 2 or more independent vertical anteroposterior sutures with Chromic No2 chromic catgut which are tied at the fundus. In situations where the upper segment is well contracted and the bleeding is limited to the placental site horizontal or square sutures approximating the anterior and posterior wall of the uterus in the cervico-isthmic region help in reducing blood loss. However, care must be taken to allow drainage of lochia by avoiding complete obliteration of the cervical canal.

   e) Intrauterine tamponade[3]. If the intrauterine pressure is more than the uterine artery pressure, blood flow to uterus can be stopped. This can be achieved by tightly packing the uterine cavity with ribbon guaze. Intrauterine tamponade with a balloon is the other alternative. Amongst the various balloons available like Sengstaken Blakemore, Rusch, Bakri, condom tamponade has gained popularity in the Indian set up because of its easy availability. A condom tied to one end of a catheter is inserted through the uterine incision and brought out of the vagina through the cervical canal. The uterine incision is closed and the condom is filled with 400-500 ml of normal saline. This causes compression of the uterus. After ensuring that there is no further oozing of blood from the site of incision, abdomen is closed. Triple antibiotic coverage, continuous oxytocics and removal of the condom catheter after 24-48hours helps in preventing a hysterectomy.

   f) Devascularization procedures[4]:

      i. Bilateral uterine artery ligation. A simple, safe procedure, the ascending branch of the uterine arteries is ligated.

      ii. Bilateral utero-ovarian artery ligation. This and the uterine artery ligation are useful for atonic PPH. However when the bleeding is from the lower segment, they may not be very effective.

      iii. Bilateral Internal iliac artery ligation. The anterior division of the internal iliac artery is ligated. This can be prophylactic or therapeutic. The operator should be conversant with the pelvic vascular anatomy.

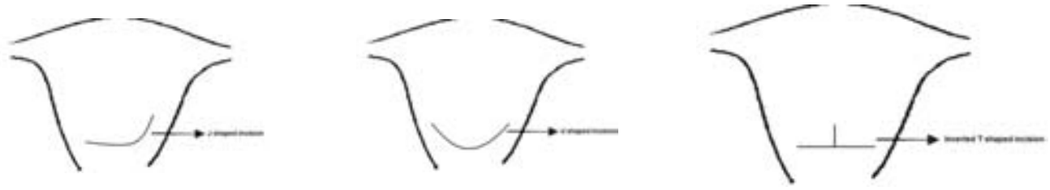
      iv. Preoperative placement of vascular catheters in the internal iliac arteries by interventional radiologist. Embolisation can be done in the event of uncontrollable haemorrhage[5].

   g) Hysterectomy. In Placenta praevia, bleeding is from the cervical branch of the uterine artery. A total hysterectomy is required to control haemorrhage and is done as the last resort.

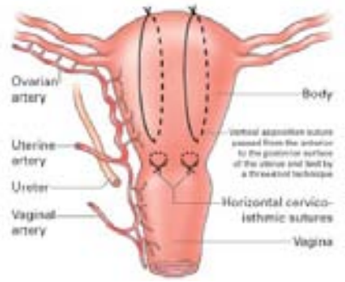
Placenta praevia accrete

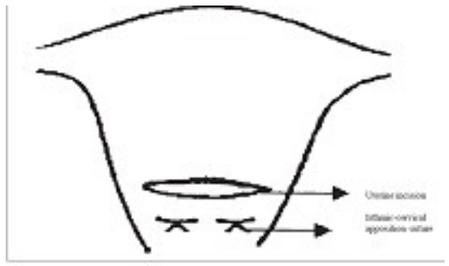
More common among women who have had previous caesarean section, the diagnosis of this condition can now be made before delivery with modern imaging techniques[6]. Indicators of Placenta accreta on Ultrasound examination include a distance less than 1mm between the uterine serosal bladder interface and the retroplacental vessles and presence of large intraplacental lakes. The problems associated with delivery of placenta depends on the site of implantation, depth of myometrial invasion and the area of the placenta that is adherent. In focal placenta accreta where one or more cotyledon is adherent, forceful separation of the adherent placenta causes bleeding. When a larger area is adherent, attempts at removal cause profuse bleeding. Ligation/angiographic embolisation of the uterine arteries may be required to control haemorrhage. Most of these cases are managed by doing a total hysterectomy. The ACOG committee opinion states that "If the clinician is extremely confident in the diagnosis, it may be prudent to complete the delivery of the infant and proceed with hysterectomy while the placenta remains attached." In a totally adherent placenta with no active bleeding, there are some who advise preservation of the uterus[7] The placenta is left undisturbed and the abdomen is closed. Methotrexate is given postoperatively. The adherent placenta is expelled spontaneously a few days later or may get resorbed in 6months. Haemorrhage and sepsis are two major complications of this management.

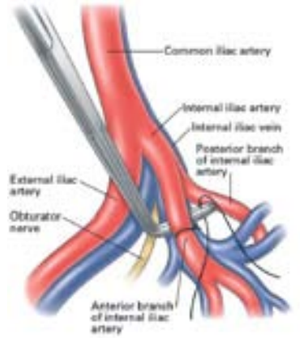
Delivery in placenta praevia is fraught with problems. Anticipated planning & tactful handling can ensure successful delivery with minimum morbidity to the mother & foetus.



Condom tamponade







References:

1. Willet JA. The treatment of Placenta previa by continius weight traction Proc R Soc med 1925; 18:90-94
2. Ward CR: Avoiding an incision through the anterior previa at cesarean delivery. Obstet Gynecol 2003 Sep; 102(3): 552-4
3. *D. Danso and P. W. Reginald: Internal uterine tamponade Ch 28, 263-267*
4. Baskett TF, Equipment tray for PPH, Ch 21, 179-182
5. Ornan D, White R, Pollak J, Tal M: Pelvic embolization for intractable postpartum hemorrhage: long-term follow-up and implications for fertility. Obstet Gynecol 2003 Nov; 102(5 Pt 1): 904-10
6. Comstock CH, Love JJ, Bronsteen RA, et al: Sonographic detection of placenta accreta in the second and third trimesters of pregnancy. Am J Obstet Gynecol 2004 Apr; 190(4): 1135-40
7. CourbiereB, Bretelle F, Porcu G, Gamerre M, Blanc B. Conservative treatment of placenta accreta J Gynecol Obstet Biol Reprod 2003;32:549-554

6. Adherent placenta - Placenta Previa

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Introduction - Adherent placenta is one of the concerning situations which has a great potential to affect the health and life of both the patient and the obstetrician if not managed well.

Incidence- Its incidence is increasing day by day probably due to rise in the rate of caesarian section. ACOG estimated in 2002 that placenta accreta complicates around 1 in 2500 pregnancy, a tenfold rise in last 50 years.

Presentation - Defect in decidua basalis, [partial or total absence] and imperfect development of fibrinoid layer [Nitabuch membrane] allows abnormal invasion of placental bed by placental villi, during placentation.

Adherent placenta - Depending upon the degree of invagination the adherence is classified and the management differs.

A. Placenta Accreta- The placental villi are adherent to myometrium.

1. Total placenta accreta-Totally adherent
2. Partial placenta accreta- Partial adherence- Few or several cotyledons of placenta,
3. Focal placenta accreta- Single cotyledon is adherent
4. Adherent succenturate lobe of placenta,

The placenta is adherent to the myometrium, but plane of separation is present. It is narrow and difficult to approach, but it may be possible to separate.

B. Placenta Increta- The placental villi actually invade the myometrium

C. Placenta Percreta- The placental villi actually invade the uterine wall and perforate the uterine serosa. It may encroach the bladder and/or adjoining tissue as well.

Management- Management depends upon degree of severity; gestational age, parity and maternal condition. There is nothing we can do to revert accreta position to non adherent position of placenta. Prediction of degree of severity, and counseling are the only modalities available to us to decide the plan of management.

Predictors- High risk group-

Placenta Previa,
Previous cesarean section,
Advanced maternal age,
Multiparity,
Previous uterine curettage
H / O - MRP in last pregnancy

Investigations - 1. To diagnose possibility of adherent placenta.

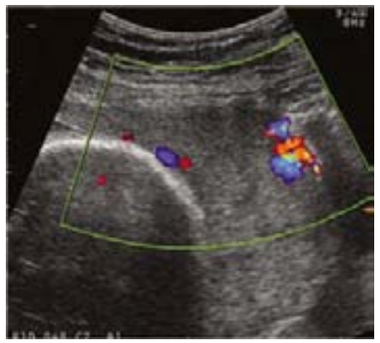
2. To improve the general condition of the patient.
3. To diagnose co-conditions, important for management of pregnancy.

Diagnosis -

It is very difficult to diagnose antenatally, hence high degree of suspicion is important.

- Imaging modalities only can help in some cases. Ultrasonography and color Doppler are better compared to MRI.

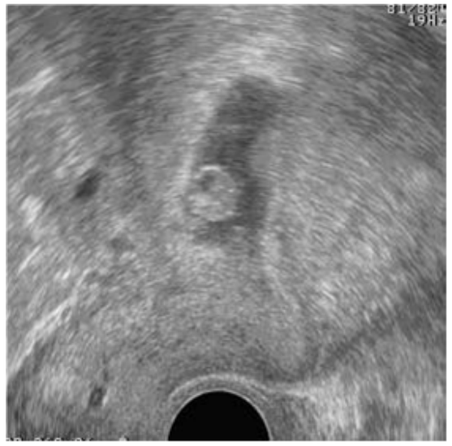
Grey scale sonographic findings-



1. Absence of normal hypoechogenic retroplacental myometrial zone.
2. Placental lacunae- presence and degree decide frequency of placenta accreta,

Fig. Sonographic image at 26 weeks' gestation revealing anterior placenta previa with large dilated blood vessels in the anterior uterine wall, which is suggestive of placenta accreta.
J Ultrasound Med 2005; 24:1569-1573

3. Invasion of myometrium- Typical hyperechogenic picture of myometrium is disturbed.
4. Thinning or disruption of the hyper-echogenic uterine serosa-
5. Thinning or disruption of the hyper-echogenic uterine serosa-bladder interface.



Longitudinal section of the uterus showing the gestational sac implanted in the anterior uterine wall. The trophoblast is herniating toward the left adnexa through the gap in the myometrium.
J Ultrasound Med 2005; 24:1569–1573

6. Herniation of gestation sac through uterine scar -The presence of focal mass-like elevations or extensions of placental tissue beyond the uterine serosa,
7. Increased lower segment vascularity, gives clue to possibility of placenta previa.

Transvaginal sonography is better to diagnose placenta previa.

- Biochemistry - 1. Raised Serum alpha fetoprotein.
  2. Raised free beta-human chorionic gonadotropin.

Biochemical tests per se are not recommended to diagnose the adherence, it is a co-finding done to diagnose Down's syndrome.

Supportive-

- To improve the general condition of the patient-

  Nutrition, correction of anaemia, counseling for physical activity, hospital admission or staying near hospital, blood donor identification, treatment of focus of infection if any.

- To diagnose co-conditions, important for management of pregnancy- . Placenta previa, multifetal pregnancy, Rh isoimmunisation, PIH, D.M, Infections like HIV, Syphilis, T.B. etc.

Specific-

Sequele of adherence -

- It is possible that focal adherence may go unnoticed. Histopathological examination, if done, reveals fibers of myometrium.
- A placental cotyledon may get separated with little force, with slightly excessive bleeding
- A placental cotyledon may remain attached and present later as delayed PPH
- A placental cotyledon may remain attached and present latter as ?Placental mole.
- Inversion of uterus [Fundal Placenta]
- Partial separation- Severe PPH
- No excessive bleeding, but no plane of separation found.
- Delayed post partum bleeding.

Management-

The problems associated with delivery of the placenta and subsequent developments vary appreciably, depending upon the site of implantation, depth of the myometrial penetration, and number of cotyledons involved. Management also depends on patient's desire to preserve her fertility and the possibility of conservation of the uterus.

Significant facts in the management in Placenta previa are:

- Condition of the patient, facilities available, expertise of the operating staff, time at hand, are important factors for taking the decision.
- First trimester detection of placenta percreta- Counseling for possible complications, possibility of hysterectomy should be discussed.

- Second trimester- All patients of placenta previa should be followed at 32- 34 weeks, in case there is no bleed. Transvaginal sonography gives better results.
- Third trimester- Confirmation of placenta previa, placenta percreta if possible.
- Planned C-section in major placenta previa, placenta accreta, placenta increta, placenta percreta, if diagnosed antenatally.
- Standard recommendations include a cesarean hysterectomy if risk factors and imaging findings are highly suggestive of this diagnosis.

Conservative management-

a. 800 mcg of Misoprostol dissolved in 30 ml of Normal saline injected through 10F catheter into Umbilical vein provides instant separation of placental bed and hence can act as an intermediary to reduce the need of MRP.[Pipingas method]

b. Manual Removal of placenta [MRP] - Three pulls are given to placenta, if it is not delivered, think about adherence. Palpate for the placental edge, if plane is found, then only proceed for MRP. With action like separating two papers, try to create space between placental disc and myometrial surface. Care should be taken not to dig into the uterine wall.

Complications- Manual removal of placenta is performed in 1-3% of cases, and whilst a well established and relatively safe procedure, it is not without complications, which include infection, hemorrhage, uterine rupture, and occasional maternal death.

c. Internal iliac artery ligation- Prophylactic, in case a decision of leaving the placenta in situ is taken or for stopping the excessive intraoperative hemorrhage, specially in major placenta previa.

d. Internal Iliac artery embolisation in similar cases as above, where facilities are present.

e. A conservative approach whereby the placenta is left in place may however, be proposed in selected cases, if the woman wishes to preserve her fertility. This strategy implies a rigorous follow-up until complete resorption of the placenta. Methotrexate, is tried to enhance the placental resorption, Some authors have left the placenta in situ without giving any agent for resorption, and the results are good. Nevertheless, in case of major hemorrhage, hysterectomy should be carried out without delay to prevent major maternal complications or even maternal death.

References :

1. Benirscke K, Kaufmann P[eds] Pathology of Human Placenta, 5 th ed, New York, Springer, ...2000,p554
2. Zelop C M, Harlow B L, Fringoletto FD Jr. Et al Emergency Peripartum hysterectomy, AmJ, Obst et gynecol 168,1443, 1993.
3. America n College of Obstetricians and Gynecologists Placenta Accreta Committee opinion, No. 266, January 2002.
4. Fox H, Placenta Accreta, 1945 1969,Obstet Gynecol surv 27;475, 1972.
5. Zaki Z M, Bahar A M, Ali M E, et al, Risk factors in placenta previa accr eta as compared to placenta previa non accreta, Acta Obstet Gynecol Scand, 77;391,1998.
6. Hardardottir H, Borgida A F, Sanders M M, et al, Histologic myometrial fibers adherent to the placenta, Impact of method of method of placental removal; Am J Obstet Gynecol,174; 358,1996.
7. HungT H,Shau W Y, Hsieh C C, et al, Risk factors for placenta accrete, Obstet Gynecol, 93;545, 1999.
8. Berchuck A and Sokol R J, Previous cesarian section , Placenta increta, and uterine rupture in second trimester abortion, Am J, Obstet Gynecol, 145;766, 1983.
9. Liang H S, Jeng C J, Sheen T C, et al, First trimester uterine rupture from a placenta percreta, J reprod medicine, 48;474,2003.
10. Lam G, Kuller J, Mc Mohan m, Use of MRI and ultrasound in diagnosis of placenta in women with a prior caesarean delivery, J Matern Fetal Medicine, 9;330, 2000.
11. Twikler DM, Lucas MJ, Balis AB, et al, Color flow mapping for myometrial invasion in women with a prior caesarean delivery, J Matern Fetal Medicine, 9; 330, 2000.
12. Chou MM, Tseng JJ, Ho Es et al, Three dimensional colour power Doppler imaging in the assessment of uteroplacental neovascularisation in placenta increta / percreta. Am J Obstet Gynecol 185; 1257, 2001
13. Maldjian C, Adam R, Pelosi M, et al, MRI appearace of placenta percreta and placenta ac crete, Magn Reson Imaging, 17;965, 1999
14. Baxi LV, Liwanpo LI, Fink DJ, D dimer as a predictor of morbidity in patients with ultrasonographic evidence of placenta previa acreta, abstract 424, J Soc Gynecol Investig 11;215A, 2004
15. Karam AK, Bristow RE, Bienstock J, et al, argon beam coagulation facilitates management of placenta percreta with bladder invasion, Obstet Gynecol, 102; 555, 2003

1. Abruptio Placenta

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Introduction & Aetiology

Abruptio placenta is one of the causes of ante partum haemorrhage. It is known by several names such as accidental haemorrhage, ablation placenta and premature separation of placenta. In Latin, abruption placenta means “rending asunder of the placenta”. This denotes a sudden accident, a clinical characteristic of most of the cases. The term accidental haemorrhage was first introduced by Rigby in 1776.The incidence is up to 1.5% in pregnancies overall and 0.3% in pregnancies at term. Till date the exact aetiology has not been understood. Hence it can neither be predicted nor prevented. High degree of suspicion is needed for prompt diagnosis and timely treatment. Since it is fraught with the danger to both the mother and foetus it is an obstetrician's nightmare.Placental abruption is defined as 'complete or partial separation of normally situated placenta prior to delivery'. It can present anytime after the 20th week of gestation till term either as an ante partum or an intra partum event. Incidence in India varies anywhere between 1:50 to 1:500. The wide variation is because of different modes of presentation and inaccurate documentation. It may be an asymptomatic case where the diagnosis is done by the presence of a retro-placental clot post partum (4.5%) or a classical case presenting with sudden collapse of the pregnant woman with either overt or covert bleeding or foetal compromise.Even though the exact aetiology is not understood, the event begins with the formation of a retroplacental clot. What triggers the clot formation is a matter of speculation. Egley and Cafalio proposed that, uterine spasm followed by relaxation leads to venous engorgement which may further lead to the rupture of arteries resulting in bleeding in the basal layer of the decidua. The bleeding may also be from the feto placental vessels. The collected blood forms a clot and remains behind the placenta unseen (concealed) or the blood can dissect between the decidua & foetal membranes and present as bleeding per vagina (revealed).It can also disrupt the membranes, enter the amniotic sac and present as port wine coloured amniotic fluid. In severe cases it can extravasate into the myometrium, reach the serosa and bleed into the peritoneal cavity causing “couvelaire”uterus or uteroplacental apoplexy.

From the maternal standpoint, abruptio Placenta can produce haemorrhage of varying severity with the subsequent serious sequelae of coagulopathy, renal failure and even death. From the foetal point of view, placental separation leads to varying degrees of loss of surface area for exchange of gases and nutrients, thus causing severe morbidity and even mortality.

The triad of sudden onset of abdominal pain, bleeding per vagina, tense and tender uterus constitutes the main diagnostic criteria. In early stages it can be totally asymptomatic and go unnoticed. In late stages the patient can present in a state of severe shock with either acute foetal distress or intrauterine death. Since the presenting symptoms are variable, other causes of A.P.H. like placenta praevia, vasa praevia and non obstetric causes of vaginal bleeding should be ruled out. The treatment is individualized depending on the gestational age and severity of bleeding.

In spite of its aetiology being enigmatic, numerous factors have been incriminated in the causation of abruption placentae.They are:

1. **Hypertension:** Pregnancy induced hypertension is associated with 2.1-4% incidence where as in chronic hypertension its association is seen in 1.8-3%.

2. Maternal age: Advanced age of more than 35 years and less than 20 years is a risk factor.

3. Parity: It is more common in multiparous women.

4. **Maternal trauma:** Reported incidence varies between 1.5-9.4%. Abdominal trauma sustained due to road traffic accidents is one of the leading causes. Though the seat belts offer protection, the lower belt should extend across the pelvis and not across the mid abdomen where the foetus is located. Even domestic violence and abuse is a contributory factor. External cephalic version can also cause trauma and rarely cause abruption.

5. Anaemia: Folic acid deficiency has been implicated to be a cause.

6. Smoking or significant tobacco usage: a prospective study has shown that the risk of abruption increases by 40%of each year of smoking prior to pregnancy.

7. Drug abuse: Cocaine use in pregnancy causes hypertension. The increased levels of Catecholamines due to cocaine are thought to be responsible for vasospasm in uterine blood vessels which may trigger the process of separation. This theory is not yet proven. The incidence is dose dependent and ranges between 13-35%.

8. Abruptio placenta in previous pregnancy: Pritchard and coworkers noted a recurrent rate of severe abruption in 1 in 8 pregnancies.

9. Invasive procedures: amniocentesis may cause retroplacental bleeding due to needle puncture and trigger the process of bleeding, clot formation and ultimately separation of Placenta.

10. Short labour: uterine tachysystole (more than 5 contractions per 10 minutes) causes abruption.

11. Malformation of uterus or presence of retroplacental fibromyoma.

12. Short cord.

13. Over distended uterus: In polyhydramnios, sudden decompression caused by Premature rupture of membranes separates the placenta. In multiple pregnancies it may occur after the delivery of the first twin.

14. Low lying placenta: the bleeding which starts in low lying placenta may extend behind the normally situated portion and cause further separation. So placenta praevia and abruptio placenta can rarely coexist.

15. Preterm premature rupture of membranes of more than 24 hours duration has been associated with increased risk. Chorioamnionitis could be the cause.

16. Idiopathic: The probable reasons could be abnormalities of uterine blood vessels and decidua.

17. Genetic predisposition: Studies have shown that it is more common in African Americans than Latin Americans.

18. Thrombophilia: A number of inherited or acquired thrombophilias have been described which also cause abruptio placentae and infarction. Most of these are single gene mutations that include genes for factor 5 Leiden, Prothrombin, Methylenetetrahydrofolatereductase, Protein S and C, and Antithrombin 3. The acquired Antiphospholipid autoantibobies including Lupus anticoagulant are associated with abruption.

19. Unexplained elevation of alphafetoprotein is associated with abruption placenta in 5% of cases

When we keep all these possibilities in mind, the unexpected can be expected. Prompt and timely treatment ensures safety to both mother and the foetus. Maternal and foetal morbidity and mortality can thus be reduced to a bare minimum.

2. Pathophysiology of Abruptio Placentae

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Abruptio placentae forms one of the major causes of maternal mortality and perinatal mortality and morbidity. 10% of abruption are associated with clinically significant coagulopathies.

Abruption is characterised by retroplacental haemorrhage due to bleeding in basal layer of decidua. The precise initiating event or cause of such bleed is still elusive.

The vessels could be pathologically altered and become prone to rupture.

There may be some inherent weakness & fragility in these vessels.

There could be vascular malformations leading on to rupture.

The underlying event in many cases of abruption is thought to be vasospasm of abnormal maternal arterioles. Some cases may result from venous haemorrhage into the areas of decidua that have become necrotic secondary to thrombosis.

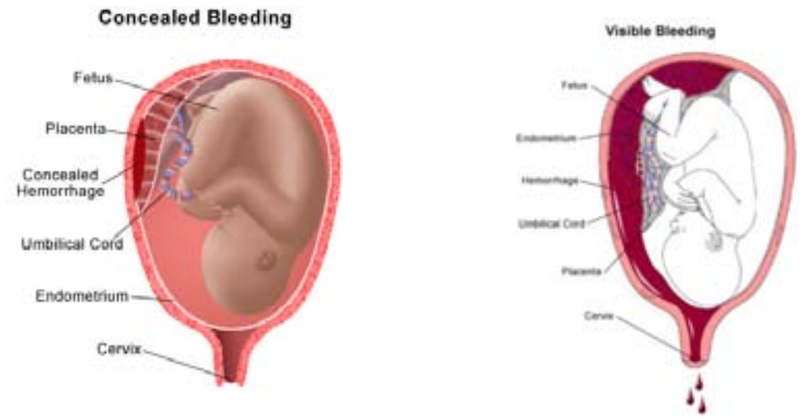
Retroplacental abruption: This results from rupture of spiral arterioles and is a “high pressure “ bleed. It is associated with hypertension and vascular diseases.

Marginal abruption: This results from tears of marginal veins and represents “low pressure bleed” It is associated with cigarette smoking.

Evidence of pre existing pathology in women with abruption includes poor trophoblastic invasion, inadequate remodelling of uterine circulation as reflected by abnormal uterine artery doppler flow and well established associations among Pre eclampsia, IUGR & abruption, all of which may be regarded as primary placental disorder.

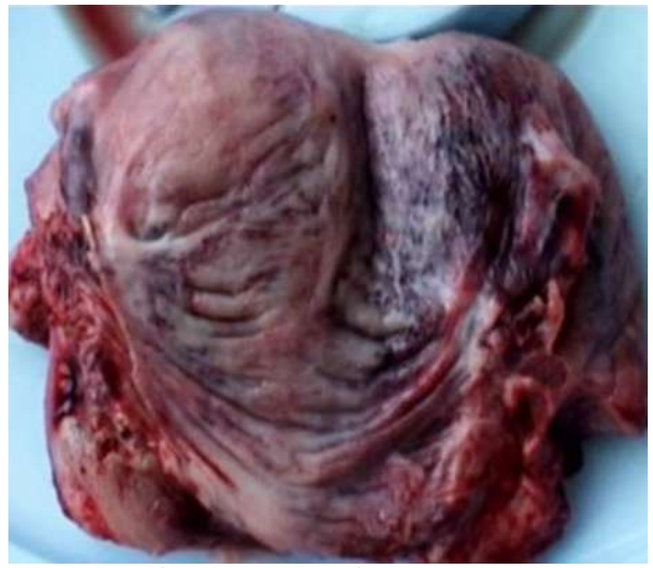
Abruption may also occur secondary to acute shearing forces affecting the placental decidual interface , such as sudden decompression of an overdistended uterus, that occurs in membrane rupture in polyhydramnios , multiple pregnancy,and rarely due to trauma.

In abruption the health risks of fetus and mother are tied to both the quantity of the placenta that is damaged as well as haemorrhage.



Course of bleeding:-

- Most of the times, it progresses since the uterus still distended with the products of conception is unable to contract sufficiently to compress the torn vessels that supply the placental site.
- The blood may find its way out per vaginum by dissecting between decidua and fetal mambranes.
- Or may find its way into the amniotic sac by disrupting the membranes giving rise to portwine coloured amniotic fluid.
- Or it may accumulate as retroplacental clot.
- Or extravasate into the myometriium.



- Occasionally it may be self llimiting with no further progression.

COUVELAIRE UTERUS;

In concealed abruption, blood does not decompress by drainage through cervix and pressure at placental bed increases. The adjacent myometrium is then unable to contract around the torn vessels to stop them from bleeding .This blood may extravasate into the myometrium and reach the serosa,resulting in purplish, copper coloured, echymotic uterus.(Couvelaire uterus) .As there is no limitation by any fascia it can extend into the peritoneal cavity. Finding of couvelaire uterus at caserean section is not an indication for hysterectomy unless complicated by atonicity.

DIC:

The inciting event is tissue thromboplastin release into maternal circulation with subsequent microvascular coagulation. The maternal fibrinolytic system is then activated with critical depletion of platelets ,fibrinogen and other clotting factors. Consumptive coagulopathy also can occur at the site of large RP clot. In either case, the final result is inadequate hemostasis & an even greater maternal blood loss.

Fetal pathology:

Fetal hypoxia & even fetal death can occur as placental disruption renders the involved placental surface inadequate for gas & metabolic exchange. Additionally disrupted maternal and fetal vascular channels may communicate, resulting in a potentially catastrophic fetal blood loss into maternal circulation, maternal Rh sensitisation or even fatal amniotic fluid embolism.

Folic acid, Homocysteine, metheonine and abruption:
Serum homocysteine decreases during pregnancy. This is in association with the physiological fall in albumin as well as with folic acid supplementation. Hyperhomocystenemia has been found to be associated with placental abruption or infarction.

A combined heterozygosity for MTHFR mutation C677T & A1298C may represent a genetic marker for abruption.

Prediction of Abruption:

1. Those with early pregnancy bleeding who have subchorionic hematoma visible on USG are at increased risk for abruption
2. Women with PAPP A below 5th percentile at the time of Ist trimester screening have increased risk of abruption.
3. Uterine artery doppler velocity at 11-14 weeks as a screening of IUGR & Pre eclampsia-pulsatality index higher than 95th percentile or PAPP A lower than 10th percentile predicted 43% of pregnancy with subsequent abruption.
4. Uterine artery doppler –Persistent notching of waveform after 24 weeks has been associated with increased risk for abruption.
5. Women with otherwise unexplained elevation of >2 MOM of alpha feto protein on 2nd trimester screening also show increased risk of abruption.
6. Obstetric complications like Pre eclampsia, fetal growth restriction and placental abruption are associated with inadequate placental perfusion. AGT (Angiotensinogen) Thr 235 mutation is associated with abnormal remodelling of the uterine spiral arteries & thereby increase the risk of placental abruption. AGT mutant allele frequency in placental abruption (0.637) was found to be significantly higher than in control group(0.377) ($p<0.001$)

References:

1. William's Obstetrics 22nd edition
2. Current diagnosis & treatment obstetrics & Gynaecology 10th edition ALAN h.Decherney et al
3. Obstetrics and Gynaec emergencies diagnosis & management
4. European journal of obstetrics and gynaecology 2001

3. Diagnosis, Differential diagnosis and Grading

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An Abruptio placenta is an obstetric emergency presenting as antepartum haemorrhage (APH), usually in the third trimester. The diagnosis of abruptio placentae is mainly clinical. Laboratory investigations help in assessing the severity and management of the condition. Depending on the type of bleeding, abruption can be revealed (75-80%), concealed (15-20%) or mixed (5-10%). Three types are described: 1) retroplacental (between placenta & myometrium) 2) marginal or subchorionic (between placenta & membranes) where the edge of placenta is separated and 3) preplacental (between placenta & amniotic fluid) which is usually of no clinical significance1.

Clinical features: History of hypertension in pregnancy, abdominal trauma/external cephalic version or sudden decompression as in multiple pregnancy and polyhydramnios may be present. Vaginal bleeding in third trimester (80%) with abdominal pain (65%) or back ache (in cases of posterior placenta) are the most common symptoms. The bleeding is dark and liquid. In concealed variety (in 20%) there may be only pain without bleeding. The pain does not get easily relieved by drugs. At the time of presentation 50% are in labour.

Pallor is usually present. There may be hypertension which may be masked if the patient has excess bleeding. Features of shock may be present. The clinical features can vary and may not reflect the extent of placental separation. Uterine tone is increased and there may be more frequent or long lasting contractions making the feel of fetal parts difficult. Uterine tenderness is also common. There may be increased uterine height and abdominal girth in concealed haemorrhage. There may be fetal distress or death in moderate/severe cases. Fetal death is seen in 30-40% cases. In all idiopathic preterm labors abruption should be ruled out.

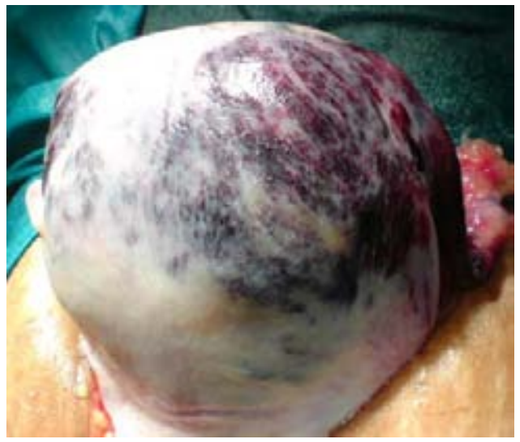

Fig – 1: Couvelaire uterus at cesarean section

An uncommon finding seen at cesarean section is "couvelaire uterus" (also known as uteroplacental apoplexy). It is a classic finding which is due to penetration of blood into the layers of myometrium. There may also be free blood in the peritoneal cavity. This may be associated with non responsiveness to oxytocin. However, couvelaire uterus per se does not warrant hysterectomy. About 10% of patients may have clinical coagulopathy. In abruption with dead fetus, 30-40% may show coagulation abnormalities due to consumption coagulopathy.

Renal failure, seen in severe abruption can be due to uncorrected hypotension and pre-existing preeclampsia. This is due to acute tubular and cortical necrosis. In the majority it is reversible.

Differential Diagnosis: The common conditions include placenta previa, preterm labor, and red degeneration of fibroid, rupture uterus, acute polyhydramnios, chorioamnionitis and appendicitis. However, it may be remembered that small amounts of retroplacental haemorrhages are quite common and are not diagnosed until examination of placenta after delivery.

Role of Ultrasound: USG is not very useful in the diagnosis of abruption with sensitivity of 20 to 50%. Careful examination may reveal a retroplacental clot which initially is 'hyperechoic' or 'isoechoic' with the surrounding myometrium making it more difficult to diagnose. Increased heterogenous thickness of placenta more than 5 cm should make one suspect this condition2. By 48 hrs the clot may be seen as hypoechoic area behind the placenta indicating blood clot. By 2 weeks it becomes sonolucent.

Differential diagnosis includes fibroid uterus where the Doppler will show blood flow in the periphery but a clot does not show active flow by color Doppler. Normal vascular complex of uterine vessels, decidua and myometrium may wrongly be interpreted as blood clot. However, this is usually less than 2cm in thickness and shows blood flow on color Doppler. Acute revealed bleeding does not show any abnormality on USG. The important use of USG is to rule out placenta previa in all cases of APH besides assessing fetal wellbeing in doubtful cases. Negative finding on ultrasound does not rule out abruption.

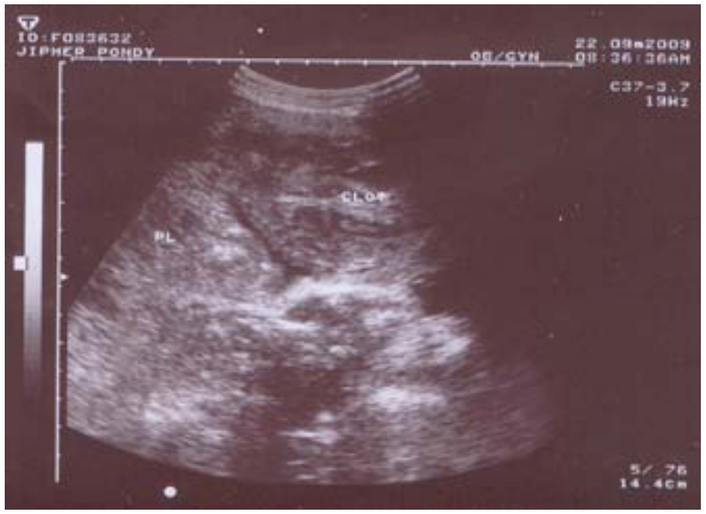

Fig-2: USG - Retroplacental clots

MRI imaging is not practical in an emergency. Biochemical markers like elevated maternal serum alpha fetoprotein (MSAFP) was observed in abruption but not studied extensively. Decreased levels of ?-hCG and inhibin as markers need to be studied.

Lab Investigations: Complete hemogram (Hb and hematocrit), coagulation profile and renal function tests will help in assessing the severity and management. Bedsides clotting time, 'clot retraction test' can be done hourly especially where round-the-clock lab facilities are not available. Normal clotting time is usually 6 min. Prolonged clotting time and clot lysis on shaking the test tube within 30 min indicate coagulation defect with fibrinogen level <150mg/dl. Other tests include fibrinogen level, platelet count, FDP levels, prothrombin time (PT) and partial thromboplastin time (PTT). Blood grouping and cross matching are done and adequate blood is arranged. In Rh negative mothers Kleihauer-Betke test should be done to assess the extent of feto-maternal haemorrhage.

Continuous fetal heart rate monitoring is done. CTG may show increased resting tone. Fetal bradycardia, loss of variability, persistent late or variable decelerations and sinusoidal pattern indicate fetal compromise.

Grading: Various types of grading are in vogue (e.g. Page). Knab (1978)3 graded abruption as mild (57%), moderate (27%) and severe(16%). The clinical grading suggested by Sher and Statland (1985)4 is of prognostic importance.

Grade I: Asymptomatic. Diagnosed after delivery by finding retroplcental clot on placenta.

Grade II: Classic signs of abruption present but without maternal or fetal distress.

Grade III: Severe abruption with dead fetus

a) with out coagulopathy

b) with coagulopathy

Our analysis of 70 patients (un published) shows that grade I 14%, grade II 24% and grade III 62% with 20% FDP positivity and maternal mortality of 2.2% - all due to DIC. Large retro-placental hematoma more than 60ml is associated with 50% fetal mortality and subchorionic hematoma with 10%5. More than 50% placental separation will result in acute fetal distress. Fetal growth restriction is also very common

References:

1. Kay HH. Placenta previa and abruption in Gibbs RS, Karlan BY, Haney AF and Nygard IE. (eds) Danforth's obstet gynecol, LWW, 10th ed, 2008
2. Nyberg DA, Cyr DR, Mack LA et al. Sonographic spectrum of placental abruption.AJ Roentgenol 148:161, 1987
3. Knab D. Abruptio placentae: an assessment of the time and method of delivery. Obstet gynecol 52: 625, 1978
4. Sher G and Statland BE. Abruptio placentae with coagulopathy: A rational basis of management. Clin obstet gynecol 28:15, 1985
5. Nyberg DA, Mack LA, Benedetti TJ et al. Placental abruption and placental haemorrhage: correlation of sonographic findings with fetal outcome. Radiology 164:357, 1987

4. Maternal & Foetal Outcome & Perinatal Outcome in Abruptio Placenta

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Maternal & Perinatal Outcome in Abruptio Placenta

Introduction :

Any bleeding after 22weeks of pregnancy is considered as antepartum haemorrhage (APH). It complicates 2-5% of all pregnancies and has various causes. APH is unpredictable and at any time before, during or after presentation patient's condition may deteriorate rapidly. Management must therefore aim to treat or prevent such deterioration. Management should be in a hospital with adequate facilities for transfusion, delivery by cesarean section and neonatal resuscitation & neonatal intensive care. For significant vaginal bleeding immediate transfer to hospital is recommended

MATERNAL OUTCOME :

In a retrospective study carried out over a year on 226 women admitted with a diagnosis of APH the incidence of APH was 3.01%. maternal and perinatal morbidity was very high with increased rates of anemia (100%) , caesarean section (43.80%), PPH (27.84%), blood transfusion (78.77%), puerperal pyrexia (10.61%), coagulation failure (10.61%), low birth weight (83.18%), birth asphyxia (12.5%), IUGR (12.39%). The maternal mortality was 2.21% and the Perinatal mortality was 23.70%. (Singhal et al 2008). In India the rates are very high due to associated problems like anemia, difficulties in transport in case of emergencies and restricted medical facilities.

Maternal mortality:

This is thought to be about 1% (RCOG Confidential enquiry 1991-93) It has fallen down from 8% in 1919 to under 1% in 1995. Although severe haemorrhage is usually the major cause, there are other complications leading to mortality. DIC itself may cause severe hemorrhage, renal failure & death.

Recurrence of APH :

Reported as 8-17% after one episode. It rises to 25% after two episodes. Of those with past history of APH , 30% of women fail to produce a live child. Mothers who stopped smoking had a 23% lower frequency of abruption placentae and 33% lower frequency of placenta praevia than women whom continued to smoke in pregnancy. In addition those who stopped smoking had about half as many fetal and neonatal deaths than those who continued to smoke. The effect is best seen when they stopped smoking as soon as pregnancy is diagnosed.(Richard Naeye 1980)

Hypovolemic shock:

There is tendency to underestimate blood loss in placental abruption. This is because of concealed bleeding into the myometrium which may be difficult to estimate.

Acute renal failure:

Results from hypovolemic shock &DIC.

Disseminated intravascular coagulation:

Postpartum hemorrhage:

Results from either coagulation failure or “covulaire uterus” ,where bleeding is into the myometrium impairing its ability to contract.

Feto-Maternal hemorrhage:

Leads to Rh sensitization in Rh negative patients. Hence anti-D immunoglobulin administration is important.

FETAL OUTCOME

In a small study of 23 women of severe placental abruption complicated by featl bradycardia, a decision to delivery interval of 20 minutes or less was associated with substantially reduced neonatal morbidity and mortality. (Salma KI, Walkinson S et al 2003)

Perinatal mortality:

It varies from 4.4% to 67.3% depending on neonatal facilities. Over 50% of the perinatal deaths are stillborn. Of those delivered alive ,16% mortality occurs within 4 weeks with babies weighing less than 2500gms.

Perinatal mortality is closely related to the gestational age .There is higher incidence of fetal malformations & IUGR, which contributes to the high

perinatal mortality. For babies weighing more than 2500gms,the survival is around 98%.In the presence of associated complications such as hypertensions , fetal mortality increases three fold.

Intra Uterine Growth Restriction:

Reported in 80% of infants born before 36 weeks of gestation. The placentas were growth retarded in the fatal cases which may have contributed to the infants' intra uterine growth retardation and subsequent death. Those with placental villous hyperplasia had a history of chronic maternal vaginal bleeding starting in the second trimester. This abnormality was possibly due to fetal anemia since it resembled the villous hyperplasia seen with severe erythroblastosis fetalis and the infants had excessive extra medullary erythropoiesis, an almost invariable consequence of subchronic or chronic fetal anemia. Premature separation of placenta explains the other placental abnormalities. These include necrosis of the deciduas basalis and thrombi at the margin of the placenta. (Richard Naeye 1978)

Congenital malformations:

As high as 4.4% (twice those in general population) .The rate of major malformations is increased three fold & most of them involve CNS.

Abnormal neonatal hematology:

Anemia results from significant fetal bleeding . Transient coagulopathies in newborn of women with placental abruption has been reported.

Conclusion:

Bleeding in late pregnancy is an important cause of fetal and maternal morbidity and mortality. The etiology of the various types is poorly understood. Current antepartum methods of detecting uteroplacental problems including dopplers are not effective in prenatal prediction of placental abruption. The outcome of affected births is still poor. Various management options are however available. The principles are initial assessment of the patient's condition and subsequent planned management aimed at resuscitation and prolongation of pregnancy if possible or immediate delivery either for maternal or fetal indication.

Refernces:

1 Singhal SR, Nanda SN.2008. Maternal and Perinatal outcome in APH. A Study in a tertiary care institute. The internet Journal of Gynaecolgy and Obsterics..

2. Why Mothers Die 1991-93. The First report of Confidential enquiries into maternal deaths in the UK. RCOG Press.

3. Richard Naeye 1980. Abruptio Placentae and placenta Praevia : Frequency, perinatal mortality and cigarette smoking. .Obstetrics and Gynaecology, Vol 55 No 6 701-704.

4. Salma Imran Kayani, Walkinshaw Stephen, Preston Carrol. 2003 to determine the relationship between decision to delivery interval and perinatal outcome in severe placental abruption. BJOG Vol 110,Issue 7, Pg 679-683

5. Richard Naeye 1978. Placenta Praevia : Predisposing factors and effects on the featus and the surviving infants. Obsterics & Gynecology . Vol 5 521-525.

5. Management of Abruptio Placentae

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INTRODUCTION:

Placental abruption is the premature separation, either partial or total, of a normally implanted placenta from the decidual lining of the uterus after the period of viability. The reported incidence varies from 0.49% to 1.8%[1]. Small episodes of placental abruption is more common than those diagnosed clinically. The recurrence rate is generally reported as 6% to 17% after one episode and increases to 25%[2].

The two principal forms of placental abruption are: revealed (65% to 80% of cases)and concealed (20-35% of cases). In the concealed form, haemorrhage is confined within the uterine cavity. Approximately 10%3of abruptions are associated with clinically significant coagulopathies, but 40% of placental abruptions severe enough to cause foetal death are associated with coagulopathies[4].

CLASSIFICATION

Sher[5] has categorised this entity into four grades on the basis of the clinical presentation. Grade 0, an asymptomatic and incidentally observed retroplacental clot, Grade1 where pain and uterine irritability is present without maternal or foetal compromise. In Grade2, there is foetal distress and in Grade 3 uterine tetany, maternal complications and foetal demise are present.6 The degree of anaemia probably will be considerably less than would seem to be justified by the amount of blood loss. A peripheral blood smear may show a reduced platelet count, schistocytes suggesting intravascular coagulation and fibrinogen depletion with release of fibrin split products. Failure of clot formation or dissolution of a formed clot is proof of a clotting deficiency. In determining coagulation status prothrombin time and partial thromboplastin time, platelet count, fibrinogen and FDP is of immense value.

Grading of placental abruption

Grade	Description
0	Asymptomatic patient with a small retroplacental clot
1	Vaginal bleeding, uterine tetany and tenderness, no signs of maternal shock or tenderness
2	External vaginal bleeding may be present, no signs of maternal shock; signs of foetal distress
3	Marked uterine tetany, persistent abdominal pain, maternal shock and foetal demise; coagulopathy may be evident in 30% of cases

MATERNAL AND FOETAL RISKS:

The implicated maternal and foetal risks are as categorised below:

MATERNAL	FOETAL
Maternal mortality rate ranges from 0.5% to 5 %.	Perinatal mortality varies from 4.4% to 67.3%, depending on neonatal facilities
Hypovolemic shock	Foetal growth restriction in upto 80% neonates born before 36 weeks

Acute renal failure - due to hypovolemia or disseminated intravascular coagulation	The rate of major congenital malformation is increased three fold
Disseminated Intravascular Coagulation	Abnormal neonatal hematologic findings
Postpartum haemorrhage : from coagulation failure or Couvelaire uterus	
Severe Rhesus sensitisation in Rhesus negative patients	

DIAGNOSIS:

The diagnosis is usually made on clinical grounds by assessing the vaginal bleeding, abdominal pain, uterine contractions and tenderness. Hypertension may mask true hypovolemia, but increasing abdominal girth or fundal height suggests significant concealed haemorrhage.The uterus is typically described as "woody hard" in severe placental abruption. If blood loss is significant, the patient may be in shock (tachycardia predominates; blood pressure is poorly correlated with blood volume in this condition). In severe cases complicated by disseminated intravascular coagulation, the vaginal blood is dark and does not clot. The cervix is usually dilated as 50% of the cases are in labour. If the membranes are ruptured, blood stained liquor is seen. The differential diagnosis would include other cases of vaginal bleeding and abdominal pain.

Ultrasonography is not a sensitive method of diagnosing placental abruption, but is useful in excluding coincident placenta previa, which is present in 10% of cases. Possible findings include hyperechoic foci posterior to the placenta suggestive of a collection of fresh blood or a hypoechoic area suggestive of a formed clot. Lack of findings does not provide reassurance and use of ultrasound should not be substituted for clinical judgement, especially on the face of a concerning clinical situation. Scintiphotoigraphy diagnosis with 99mTc pertechnetate may aid in the early diagnosis of abruptio7. A population based study8 concludes that first and second trimester uterine artery Doppler ultrasonography can have a role in prediction of abruption. An elevated resistance index, pulsatility index or the presence of a notch in uterine artery blood flow indicates a high risk of abruption. Another nonspecific marker of poor obstetric outcome is an elevation in the maternal serum AFP. Presence of Glu 298 Asp eNOS variant and MTHFDIR 653 Q polymorphism are genetic risk factors for severe abruption placentae.9

MANAGEMENT OPTIONS:

Management depends upon:

1) Severity

2) Associated complications

3) Condition of the mother and foetus

4) Gestational age.

It includes:

a) General measures (common to all patients with bleeding)

b) Specific measures : Immediate delivery

Expectant management

Management of complications

Immediate delivery: The need for immediate delivery depends on the severity of abruption and whether the foetus is alive or dead If the foetus is dead, maternal resuscitation and vaginal delivery is the goal.

Cases of placental abruption presenting with haemorrhage, uterine spasm or foetal distress should be treated as an acute emergency with adequate fluid and blood replacement (taking care to fluid overload) and termination of pregnancy is mandated.

Where the foetus is alive, studies done to test the relative effectiveness of caesarean section versus vaginal delivery concluded that the perinatal mortality in the vaginal delivery group was significantly greater than those delivered by caesarean section. The one minute APGAR score was significantly greater for the caesarean section group. This is attributed to the admission to delivery interval which was greater in the vaginal delivery group.

If the degree of separation appears to be limiting and continuous foetal heart tracings is reassuring vaginal delivery should be attempted with induction, amniotomy and oxytocin infusion . Amniotomy, as early as feasible, has long being championed in the management of placental abruption. The rational behind this is escape of amniotic fluid which would decrease both, the bleeding from the implantation site and reduce the entry of thromboplastin into maternal circulation. It is also proposed that it activates coagulation factors from retro-placental clots.However, in an immature foetus, its role is controversial. The intact sac is proposed to aid in cervical dilatation and labour.

Oxytocin should be used cautiously in cases with a tonically contracted uterus, as uterine rupture may be caused by an overstimulated uterus. If the uterus is extremely spastic, an internal monitor is used. The progress is assessed by observing the cervical dilatation. Uterine stimulation to effect vaginal delivery provides benefits that override the risks. Some researchers challenge the use of oxytocin on the basis that it might enhance the escape of thromboplastin into the maternal circulation and thereby initiate or enhance consumptive coagulopathy or amniotic fluid embolism syndrome. There is no evidence in support of this data14.

The exception to vaginal delivery is severe abruption with uncontrollable haemorrhage and it mandates operative delivery. Where vaginal delivery is contemplated, pudendal block anaesthesia is recommended and conduction anaesthesia should be avoided in the face of significant haemorrhage. In the volume repleted patients in early labour, a preemptive epidural should be considered. In associated coagulopathies, episiotomy is better avoided.

The indications of caesarean section are both foetal and maternal. It should be considered in the presence of foetal distress and where delivery is not imminent. Caesarean section also indicated if the foetus is in good condition, but the cervix is not favourable and in the face of progressive or severe placental separation. Maternal indications are uncontrollable haemorrhage, rapidly expanding uterus with concealed haemorrhage with or without a live foetus, uterine apoplexy or refractory uterus. Peritoneal drain and subcuticular drain is to be considered in cases of suspected DIC. Uterine atony may result due to extensive infiltration of the myometrial wall with blood and at times, hysterectomy may be necessary. B-lynch suture is quick, easy to learn and safe, providing a useful alternative to hysterectomy. If child bearing is desired, bilateral uterine artery ligation should be done. If ineffective, bilateral ligation of the hypogatric arteries may reduce the arterial pressure within the uterus to venous level and may effect hemostasis.

Expectant management of suspected placental abruption is the exception and not the rule. The goal is to prolong pregnancy with the hope of improving foetal maturity and survival. It may be considered when the mother is stable, foetus is immature (<37 weeks)and foetal heart tracing is reassuring. The patient should be observed in the labour suite for 24 - 48 hrs to ensure that further placental separation is not occurring. Continous foetal and maternal monitoring should be maintained. Odendal and co workers showed that late decceleration detected by 6hrly heart rate monitoring was the first warning of abruptio.10 Sea-saw pattern of uterine contraction, sinusoidal pattern of foetal heart rate implicate an

advanced grade of placental abruption. Administration of corticosteroids to accelerate foetal lung maturity may be a part of the plan. In mild cases, there is no evidence supporting the routine admission of such patients especially where there is no evidence of maternal or foetal compromise or uterine contractions

Use of tocolytics is controversial. Beta-mimetics can mask or blunt the patient's cardiovascular response and calcium channel blockers may further reduce the blood pressure, hence best avoided.11In this context MgSO4 is the drug of choice. Once the patient is stable the decision to manage the patient as an outpatient should be tailored and the foetus should be followed closely with CTG. Once the foetus attains maturity termination of pregnancy should be considered with individualisation of the cases. If the initial ultrasound scan shows a retroplacental clot, the clot may be monitored by serial ultrasound scans.

Management of complications:

The major complications are hemorrhagic shock, disseminated intravascular coagulation, ischemic necrosis of the distal organs (especially kidneys and brain) and postpartum haemorrhage.

Placental abruption can lead to initiation of coagulation cascade and DIC. Fresh whole blood, fresh frozen plasma, platelet concentrate, fibrinogen, cryoprecipitate should be in hand to manage these patients. Heparin has no role in abruption. In case of uncontrollable hemorhage bilateral Internal iliac artery ligation/embolisation, ballon tamponade and abdominal packing can be attempted. Hysterectomy is rarely necessary.

In persistent oliguria or anuria renal cortical or tubular necrosis is probable and fluid intake and output must be carefully monitored. Continuing impairment of renal function may require peritoneal dialysis or hemodialysis.

Foetomaternal haemorrhage during abruption can be significant. All Rhesus-negative women with abruption must undergo a Kleihauer-Betke test and receive an appropriate dose of anti D immunoglobulin within 72 hours of abruption to prevent immunisation. When management is expectant, regular monitoring in collaboration with the haematologist and blood bank is advisable.

Maternal mortality ranges from 0.5-5%. 14 High degrees of suspicion, early diagnosis and definitive therapy should reduce the rate to 0.5 - 1%. Perinatal mortality rate varies from 4.4% to 67.3%. Live born infants have a high rate of morbidity resulting from hypoxia, birth trauma and the hazards of prematurity (40-50%). Early intervention, reducing admission to delivery interval may reduce the perinatal mortality and maternal hazards.

Reference:

1. Salihu H and others: Perinatal mortality associated with abruption placenta in singletons and multiples, Am J Obstetrics Gynecol 193 (1):198-203,2005
2. Morgan K,Arulkumaran S :Antepartum haemorrhage,current obst gynaecol 13(2):81,2003.
3. Sher G .A rational basis for the management of abruption placentae .J Reprod Med 1978;21:123-9
4. Knuppel A R, Drukker J E: W B Saunders 1986.
5. F. E. Ohnofue, O A Olatunbosun, April 2004.
6. Anthony J :Major obstetrical haemorrhage:disseminated intravascular coagulation .In James D and others ,editors:High risk pregnancy: management options ed 3,Philladelphia 2006, saunders
7. Rasmussen S et al - perinatal mortality and case fatality after placental abruption Acta obst gynecol search 1996; 75:229.

6. Vasa Praevia

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Vasa Praevia (vasa previa) is a rare complication causing antepartum haemorrhage which is defined as "fetal vessels crossing or running in close proximity to the internal os. These vessels course within the membranes (unsupported by the umbilical cord or placental tissue) and are at risk of injury when the supporting membranes rupture." 1

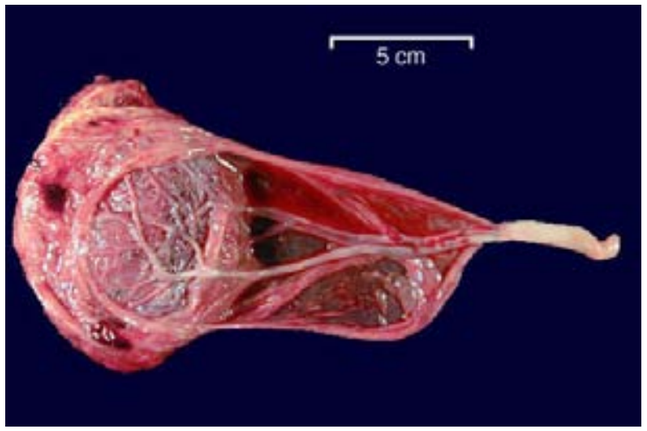
Aetiology:

Vasa praevia is present when the umbilical vessels traverse the fetal membranes over the internal cervical os for a velamentous insertion on a low lying placenta and are present below the presenting foetal part. Sometimes rarely these vessels may also be joining an accessory (succenturiate) placental lobe. This condition is rare (1 in 3000 pregnancies) but is relatively common n multiple pregnancies. Due to the absence of protecting Wharton's jelly these vessels may be easily lacerated at the time of rupture of the membranes. Alternatively these vessels may get compressed by the foetal presenting part during labour causing foetal jeopardy. If these fetal vessels rupture, the bleeding is from the foetoplacental circulation, and foetal exsanguinations will rapidly occur, leading to fetal death. The foetal mortality in vasa praevia may be as high as 75 -100%.

Diagnosis:

This is rarely confirmed before delivery but should always be suspected in all cases of antenatal haemorrhage, particularly in cases where the foetal heart rate shows compromise2. In those cases the vaginal blood should be tested for foetal cells. A simple test to distinguish foetal from maternal cells consists of two test tubes with 5 ml of tap water into which six drops of 10% KOH are put. In one test tube 3 drops of vaginal blood is put and in the other 3 drops of maternal blood is put as a control. The tube with maternal blood turns green yellowish brown after 2 minutes and if the vaginal blood contains foetal blood it remains pink in colour3.

Routine ultrasonography may also show a vessel crossing the membranes over the internal cervical os particularly with color-flow Doppler.



Transvaginal ultrasound (an ultrasound view of the cervix) in combination with color Doppler (which can show blood flowing through veins) is the most effective tool in the diagnosis of vasa previa during pregnancy and should be utilized in patients at risk, specifically those with bilobed, succenturiate-lobed, and low-lying placentas, pregnancies resulting from in vitro fertilization, and multiple pregnancy. This color Doppler technique makes it possible to see whether the veins are imbedded the placenta or are crossing the cervical opening

The diagnosis is usually confirmed after delivery on examination of the placenta and fetal membranes.

Most often the foetus is already dead when the diagnosis is made; because the blood loss (say 300ml) constitutes a major bulk of blood volume of the foetus (80-100ml/kg i.e. 300ml approx for a 3kg foetus).

Treatment

Treatment immediately with an emergency cesarean delivery is usually indicated.

When vasa previa is detected prior to labor, the baby has a much greater chance of surviving. Despite improvements in medical technology, vasa previa often remains unsuspected until fatal foetal vessel rupture occurs. When vasa previa is found, elective delivery by cesarean before labor begins can save the baby's life. Tests can be used to measure the maturity of the baby's lungs. With steroids the baby's lung maturity can be hurried along before the Caesarean section. Investigation for the source of the blood is necessary when there has been hemorrhage before or during birth, especially when associated with fetal heart irregularities. Aggressive resuscitation of the neonate is necessary where fetal vessel rupture has occurred. Without these measures, the baby is almost sure to die.

But what happens to the baby of an uneventful pregnancy with no risk factors or symptoms? Velamentous insertion - unlike other placental abnormalities, is not looked for and seldom found before delivery; but velamentous cord insertion is a definite risk factor for vasa previa. Velamentous insertion of the umbilical cord is said to have a 1:50-100 occurrence rate. Color Doppler ultrasounds should be done during all routine prenatal ultrasound exams to determine placental implantation of the cord and rule out velamentous insertion. This is not standard practice at this time. Transvaginal color Doppler ultrasounds should be done following the suspicion of any implantation abnormalities.

References

1. Yasmine Derbala, MD; Frantisek Grochal, MD; Philippe Jeanty, MD, PhD (2007). "Vasa previa". Journal of Prenatal Medicine 2007 1 (1): 2-13.
2. Dougal A,Baird CH: Vasa previa- report of three cases and review of the literature. Br J Obstet Gynaecol, 1987, 94 712 - 5.
3. Loendersloot. EW: Vasa previa, Am J Obstet Gynaecol 1979, 135, 702 - 3

7a. Etiology and pathogenesis of DIC in Abruptio Placentae

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Disseminated intravascular coagulopathy (DIC) is a thrombohemorrhagic disorder with concurrent activation of the coagulation and fibrinolytic pathways. Coagulation is always the initial event. DIC is an intermediary manifestation of disease, not a disease itself. Following are the common obstetric conditions associated with DIC.

Etiology

1) Abruptio Placentae,

2) Amniotic fluid embolism

3) Preeclampsia

4) Retained dead fetus.

Pathophysiology

Abruptio is the most common obstetric cause for acute DIC. 10-30% women with clinically significant abruption will have a gross clotting defect. The only proven treatment is stopping the triggering event. Hence one needs to know the exact etiopathogenesis of DIC.

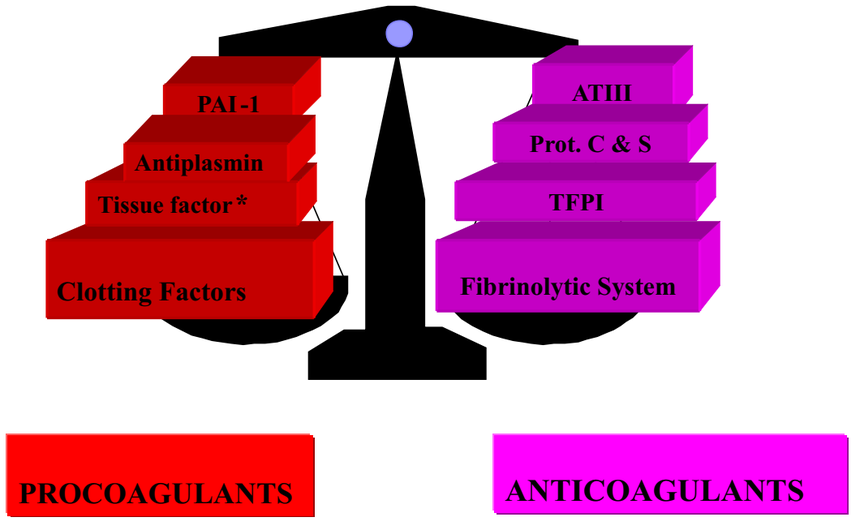
Under normal circumstances endothelial abnormalities, platelet degranulation or tissue disruption causes activation of either the intrinsic or extrinsic clotting cascade. DIC is failure of normal checks and balances resulting in systemic activation and circulation of thrombin and plasmin. The levels of plasmin and thrombin are differentially elevated depending upon the underlying etiology. If clotting process predominates secondary fibrinolysis is minimal (e.g. sepsis). If secondary fibrinolysis predominates the clinical presentation is hemorrhage as in abruption. Both thrombosis and hemorrhage can occur simultaneously. DIC may exist without being clinically apparent.

The plasmin induced breakdown products of fibrin and fibrinogen known as fibrin degradation products (FDPs), bind soluble fibrin monomer and prevent polymerization.

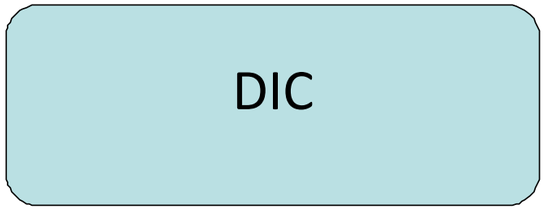
This further impairs hemostasis and hemorrhage is increased. FDPs also inhibit platelet mediated primary phase of coagulation and produce profound platelet dysfunction. Plasmin activates the complement cascade resulting in red cell and platelet lysis. Material released from RBCs propagates the DIC cycle. Once established, the cycle of thrombin induced micro vascular thrombosis (as in sepsis) and plasmin induced hemorrhage (as in abruptio) can become self- perpetuating.

Activation of the fibrinolytic system and systemic consumption of soluble components occur out of proportion to the blood loss in abruptio placentae. Decreased levels of platelet count, fibrinogen and elevated levels of fibrin degradation products correlate well with the severity of the disease. DIC occurs most often in severe degrees of abruptio where fetal demise is also evident.

The process of placental separation contributes to FDP elevation. The FDPs increase through out labor process, peak shortly after total placental separation and start declining after delivery. The elevated levels of FDPs actually inhibit myometrial contractility and thus contributing to postpartum hemorrhage. In such situations IV infusion of anti fibrinolytic agents may promptly overcome the uterine inertia.



Picture 1 : Haemostatic Balance



Picture 2 : Cascade of thrombohemorrhagic sequence

7b. Clinical features and Laboratory tests

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Abruptio placentae or placental abruption is defined as the premature separation of the normally implanted placenta leading to haemorrhage**.** It complicates approximately 1 in 200 deliveries. The reported incidence varies from 0.49% to 1.8%. The wide variation in reported incidence is believed to be caused by variations in diagnosis. Evidence of abruption in 4.5% of placentas examined routinely, suggesting that small episodes of placental abruption are more common than those diagnosed clinically (Fox). Placental abruption is concealed in 20% to 35% of cases and revealed in 65% to 80% of cases.The concealed type is more dangerous, with more severe complications. Four grades of placental abruption have been described . The most severe type (grade 3) occurs in approximately 0.2% of pregnancies.

Predisposing Factors

1. Increased age and parity
2. Preeclampsia
3. Gestational hypertension
4. Chronic hypertension
5. Preterm ruptured membranes
6. Thrombophilias
7. External trauma including aminocentesis and external cephalic version
8. Cocaine abuse
9. Acute uterine decompression as in polyhydramnios
10. Cigarette smoking
11. Folic acid deficiency
12. Short umbilical cord
13. Uterine anomalies
14. Uterine leiomyomas

Clinical features

Symptoms

1.Vaginal bleeding in revealed and mixed type
2.Severe and constant abdominal pain(more in concealed type)
3. Severe back pain
4. Loss of fetal movements.

Signs

1. Tense and tender uterus
2. Uterine hypertonus with high frequency contractions
3. Difficulty in palpating the fetal parts

4. Pallor
5. Associated hypertension
6. Uterus larger than the gestational age in concealed type
7. Preterm labour
8. Fetal distress or even absent fetal heart sounds

Types

1. **Concealed** (35%) –blood does not escape externally, but is retained between the detached placenta and the uterus
2. **Revealed** (60%) – blood escapes through the cervix
3. **Mixed** (5%)

Clinical grading (Sher and Shetland's)

Grade I - unrecognised clinically before delivery
evidence of retroplacental clots on examining the placenta
fetus usually not at risk

Grade II – classic features of abruption placentae present
but no maternal distress and the fetus is alive(fetal distress present)

Grade III – severe abruption with dead fetus
a) without coagulopathy
b) with coagulopathy

Diagnosis:

The diagnosis can usually made by clinical examination and confirmatory investigations are rarely necessary. Commonly seen after 36 weeks of gestation, placental abruption causes uterine irritation and onset of labour. Vaginal bleeding is seen in more than 60 % cases. Because labor is the most common factor precipitating placental separation, nearly 50% of patients with placental abruption are in established labor. Uterine contractions may be difficult to distinguish from the abdominal pain of abruption. The abdominal pain may be due to extravasation of blood into the myometrium. In severe cases (grade 3), the pain is sharp, severe, and sudden in onset. In addition, some patients may have nausea, anxiety, thirst, restlessness, and a feeling of faintness, whereas others report absent or reduced fetal movements.

Associated hypertension may mask true hypovolemia, but increasing abdominal girth or fundal height suggests significant concealed hemorrhage. Some patients may be in shock due to significant blood loss. The uterus is typically described as "woody hard" in severe placental abruption. In such cases, the fetus is difficult to palpate, and a continuous fetal heart rate monitor or real-time ultrasonography must be used to identify the fetal heartbeat. The fetus may be "distressed," with fetal heart rate abnormalities, or may be dead. Fetal distress occurs in grade 1 to 2 abruption, but in grade 3 abruption, fetal death is inevitable, by definition In severe cases complicated by disseminated intravascular coagulation, there may be no clotting in the vaginal blood, which is dark. The incidence of coagulopathy is 30%,and it occurs mainly in the severe forms.

Fetal status in abruption:

Separation of placenta from its bed naturally lead to compromise of the uteroplacetnal transfer of oxygen and nutrients.If more than 50% of the placenta separates, it is unlikely that the fetus survive the insult. Electronic fetal monitoring may show a variety of abnormal tracing from tachycardia, loss of variability, to variable decelerations can be found on CTG tracings. A normal tracing eventhough reassuring need not end up with a good fetal outcome, as the abruption can suudenly progress killing the fetus.

Differential diagnosis:

Conditions which produces vaginal bleeding and or abdominal pain should be considered as differential diagnosis. These include placenta previa, preterm labour, genital tract trauma, lesions of genital tract.

If the initial ultrasound scan showed a retroplacental clot, the clot may be monitored by serial ultrasound examination in the occasional case when the obstetrician decides on a conservative management of abruption.

Investigations

As soon as the patient is admitted, blood sample should be collected for investigation and for arranging several units of blood. The blood sample is send for the following investigation, and a sample retained for bedside clot observation test which rules out coagulation defects or establish its presence.

1. Haemoglobin
2. PCV
3. Grouping with Rh typing (if not done before)
4. Complete coagulation profile includes fibrinogen, fibrin degradation products, partial thromboplastin time, protrombin time, bleeding time, clotting time and platelet count.
5. Clot retraction time

6. Ultra sound – to confirm placental site, presence of retroplacental haematomas and fetal viability /joepardy

Ultrasonography is not a sensitive method of diagnosing placental abruption, but it is useful in excluding coincident placenta previa, which is present in 10% of cases. When the retroplacental clot is large, ultrasonography identifies it as hyperechogenic or isoechogenic compared with the placenta. Resolving retroplacental clots appear hyperechogenic within 1 week and sonolucent within 2 weeks. Although ultrasonography is not an accurate diagnostic tool, it is useful in monitoring cases managed expectantly. The size of the hematoma, its location and change in size over time, and fetal growth are monitored by ultrasound scan. The value of ultrasound examination is in excluding other major causes of APH such as placenta previa.

Ultrasound is also useful in determining the fetal status. Doppler studies in placental abruption has not been shown to be helpful in the diagnosis or prediction of fetal outcome.

Management depends on the severity of the case and presence of associated complications, and almost always it is by expediting delivery by appropriate route.

7c. DIC in Management & Other Therapies

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Background

Disseminated Intravascular Coagulation is defined as "An acquired syndrome characterized by the intravascular activation of coagulation with loss of localization arising from different causes. It can originate from and cause damage to the microvasculature, which if sufficiently severe, can produce organ dysfunction. [1]

Disseminated intravascular coagulation (DIC), also known as consumptive coagulopathy, is a pathological activation of coagulation mechanisms that happens in response to a variety of diseases. It leads to the formation of small blood clots inside the blood vessels throughout the body.[2] These small clots consume all the available coagulation proteins and platelets, normal coagulation is disrupted and abnormal bleeding occurs from the skin, the digestive tract, the respiratory tract IV lines and surgical wounds.

The small clots may block blood flow to organs (such as the kidneys), which may malfunction as a result. DIC can occur acutely but also on a slower, chronic basis, depending on the underlying problem. It is common in the critically ill, and may lead to multiple organ failure, and death.[3]

Epidemiology [4]

- About half of DIC cases result from complications of pregnancy. It is therefore vital for obstetricians to suspect, diagnose and provide timely treatment .
- About a third of cases result from carcinomatosis.- Cancers of lung, pancreas, prostate and stomach.

Etiology and Types of DIC

DIC can be acute or chronic in nature. The DIC that, is seen in pregnancy is usually acute in nature. It is the result of the following Pregnancy related conditions:

- Abruptio placenta/Placental abruption
- Amniotic fluid embolism
- Pre-exclampsia/eclampsia
- Retained dead fetus or placenta.

DIC is an escape of the normal mechanism of hemostasis in which intravascular coagulation becomes overwhelming to the body. Acute DIC if unrecognized and untreated may often prove fatal. In contrast ,Chronic DIC , seen more often in cancer patients presents as localized thrombotic events (eg: deep vein thromboses) and often only minor imbalances in hemostasis exist.

Massive tissue injury has also been implicated in DIC. Infection and sepsis are the most common causes of acute DIC- viral, fungal, and bacterial infections, particularly meningococcemia and other Gram-negative bacterial endotoxins have been implicated in DIC. Antibiotic therapy can also be an initiator of DIC, as it may alter intestinal flora, which is a source of vitamin K, thereby altering the coagulation process. Liver dysfunction from any cause can cause DIC. The liver synthesizes the coagulation factors and the inhibitors of coagulation. The liver also clears the activated coagulation factors and FDPs. Liver dysfunction can disrupt the normal balance of coagulation, thus leading to DIC.

Pathophysiology of DIC :

Understanding Coagulation

- Coagulation is a complex process by which blood forms clots. It is an important part of hemostasis. A platelet and fibrin-containing clot to stop bleeding and begin repair of the damaged vessel covers the damaged blood vessel wall. Disorders of coagulation can lead to an increased risk of bleeding (hemorrhage) or clotting (thrombosis).
- Under homeostatic(normal) conditions there is a balance of coagulation and fibrinolysis.
- Under homeostatic condition there is a balance between Procoagulants and anticoagulants
- The activation of the coagulation cascade irrespective of the cause yields THROMBIN, that converts fibrinogen to fibrin. The stable fibrin clot is the final end product of hemostasis.
- Activation of the fibrinolytic system generates PLASMIN which is responsible for the lysis of fibrin clots.
- The breakdown of fibrinogen and fibrin results in polypeptides called fibrin degradation products (FDPs) or fibrin split products (FSPs).

Mechanism of DIC

- In DIC, the processes of coagulation and fibrinolysis lose control.
- As a result there is widespread clotting with resultant bleeding.
- Mechanism of DIC is similar in all conditions irrespective of cause.
- One critical mediator of DIC is the release of tissue factor.
- TF is released in response to exposure to cytokines (particularly interleukin),
- TF is also abundant in tissues of the lungs, brain, and placenta. This helps to explain why DIC readily develops in patients with extensive trauma.

Transfusion Reactions & DIC

DIC may also be related to blood transfusions.

1 > Acute hemolytic transfusion reactions
- General endothelial injury
- caused by activated complement, cytokines, and neutrophil products
- This can potentially stimulate DIC.

2 > Massive transfusions of whole blood.
- The etiology of DIC related to massive whole blood transfusions is unknown,
- Whole blood transfusions are currently non–existent not only in the developed world but also in good hospitals in India. It is important for obstetricians to practice component thearapy and give up whole blood completely.

Signs and symptoms

- The affected person is often acutely ill with shock and widespread hemorrhage.
- Common bleeding sites are mouth, nose and venipuncture sites; extensive bruising, renal failure and gangrene follow if untreated.
- The onset of DIC in the obstetrical setting can be fulminant, as in endotoxic shock or amniotic fluid embolism.

- Diagnosis[5:] Diagnosis is usually suggested by following conditions:
- Screening Tests :

PT and PTT are screening test for DIC. Currently however, the wide availability of D-Dimer tests have reduced dependence on PT/PTT.

- *Severe cases with haemorrhage:*
  - The PT and APTT are usually prolonged
  - Fibrinogen level markedly reduced(<100mg/dl).
  - FDP- Increased.
  - D-dimer test is POSITIVE
  - There is severe thrombocytopenia-Low platelets(<20000/ul)
  - The blood film may show fragmented red blood cells (schistocytes).
- *Mild cases without bleeding:*
  - There is increased synthesis of coagulation factors and platelets.
  - PT, APTT, and platelet counts are normal.
  - Fibrin degradation products are raised.

Definitive diagnosis

- Thrombocytopenia.
- PT and PTT-Almost always elevated.
- A low fibrinogen concentration.
- Increased levels of fibrin degradation products(FDP)
- D-Dimer-Positive
- FDP and D-Dimer are specific tests for DIC.

Management of DIC

- Treat underlying cause-Ex, infections, retained products, dead foetus etc.
- Fluids- ringer lactate/ saline etc will flush our FDPs and restore normal hemostasis.
- Blood Component Therapy
- Often required for the patient experiencing DIC.
- Useful for patient who continues to bleed in spite of treatment of the underlying cause.

Use of Red cell concentrates

- Red cell concentrates – leukoreduced if the patient can afford, may be given if the patient continues to bleed or the hemoglobin drops below 8 g/dL. Volume needed varies with blood loss and can often be large-4 to 6 units. Red cell replacement is required when the blood loss exceeds >30% of total blood volume or when the Hb drops below 8g/dl.
- Advisable in severe bleeding to give 2 units of fresh frozen plasma (FFP) for every 4-6 units of red blood cells given to the patient.
- Use Of FFP And Cryoprecipitate
- The use of FFP & and Cryoprecipitate is controversial in the management of DIC.
  - FFP contains fibrinogen, which can potentially create higher levels of FDPs, which will further impair hemostasis.
  - Cryoprecipitate is even richer in fibrinogen than FFP.
  - In addition, the use of cryoprecipitate exposes the recipient to multiple donors with the associated hazards.

- Use of Platelets.
- Platelets may be given if the platelet count is < 20,000 cells/mm3 or if the patient is actively bleeding.
- Anticoagulants –Low molecular weight (LMWH) Heparins may rarely be used.

Prognosis

- Prognosis varies depending on the underlying disorder.
- The prognosis for those with DIC, regardless of cause, is often poor
- DIC is a very important contributor to Maternal and infant mortality rates in India. Proper management of DIC in abruption placenta and related conditions will help bring down MMR in India.

Points to be noted while transfusing blood components in DIC[6]

Summary of BCSH guidelines

- The diagnosis of DIC should be based on both clinical and laboratory information.
- It is important to repeat the Lab tests to monitor the dynamically changing scenario whenever DIC is suspected.
- The cornerstone of the treatment of DIC is treatment of the underlying condition.
- Transfusion of platelets or plasma (components) in patients with DIC should not primarily be based on laboratory results and should in general be reserved for patients who present with bleeding/or at high risk of bleeding (Ex Postoperative patients/planned invasive procedure or low platelets.
- In non-bleeding patients with DIC, prophylactic platelet transfusion is not indicated unless it is perceived that there is a high risk of bleeding.
- In bleeding patients with DIC and prolonged PT and APTT, administration of fresh frozen plasma may be useful.
- Plasma transfusions are to be considered not on Lab tests alone but in those with active bleeding/planned invasive procedure. There is no evidence that infusion of plasma stimulates the ongoing activation of coagulation.
- If transfusion of FFP is not possible in patients with bleeding because of fluid overload, consider using factor concentrates such as prothrombin complex concentrates. These will only partially correct the defect because they contain only selected factors, whereas in DIC there is a global deficiency of coagulation factors.
- Severe hypofibrinogenaemia(< 1g/l) that persists despite FFP replacement may be treated with lyophilized fibrinogen concentrate or cryoprecipitate. Each bag of cryoprecipitate supplies 150 mg of fibrinogen.
- Role of Heparin-In cases of DIC where thrombosis/ischemia/infarction predominates therapeutic doses of heparin (UFH in dose of 10u/Kg/hr) should be considered.
- Monitoring the APTT in these cases may be complicated and clinical observation for signs of bleeding is important. In critically ill, non-bleeding patients with DIC, prophylaxis for venous thromboembolism with prophylactic doses of heparin or low molecular weight heparin is recommended.

- Consider treating patients with severe sepsis and DIC with recombinant human activated protein C (continuous infusion, 24 ì g/kg for 4 days). This product however, should not be used in patients with low platelet counts <30000/ul or in the event of a planned invasive procedure or where is there is high risk of bleeding.
- In general, patients with DIC should not be treated with antifibrinolytic agents.
- Patients with DIC that is characterised by a primary hyperfibrinolytic state and who present with severe bleeding could be treated with lysine analogues, such as tranexamic acid (e.g. 1 g every 8 hours).

Conclusion

- DIC also called Consumptive coagulopathy is a disorder characterized by abnormal bleeding or clotting..
- DIC is caused by- Pregnancy related complications, infections and tissue injury.
- Mechanism-Release of tissue factor or tissue damage activates the intrinsic and extrinisic pathways leading to DIC.
- Diagnosis:Bleeding/Clotting tendancy+
- Prolonged PT and PTT/ Decreased Fibrinogen & Low platelets and Increased FDP + Pos D- Dimer
- Treatment of underlying cause+ Transfusion support with red cells when Hb is below 8g/dl + 2FFP/Cryo for every 4-6 PRBC +Platelets(<20,000/ul).
- Use of FFP/CRYO adds fibrinogen and is hence controversial.

References

1. The international Society on Thrombosis and Haemostasis definition of DIC
2. Churchill Livingstone Pocket Medical Dictionary 14th Edition Churchill Livingstone.
3. Davidson's Principles and Practice of Medicine 19th Edition.
4. Robbins' Pathologic Basis of Disease (6 ed.).
5. Clark, Michael; Kumar, Parveen J. (1998). *Clinical Medicine: A Textbook for Medical Students and Doctors* (4 ed.). Philadelphia: W.B. Saunders.
6. Summary of British Committee for Standardization in Hematology guidelines.

8. Complications of Placental Abruption

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Maternal mortality rate associated with abruption is 1% or more which is mainly due to the complications occurring with this condition. The table below enlists the maternal and fetal complications of placental abruption.

Table 1. Complications of placental abruption

Maternal	Fetal
Haemorrhagic shock- Antepartum & / postpartum	Intrauterine death
DIC	Fetal growth restriction
Acute renal failure	Rh isoimmunisation
Ishcaemic necrosis of distal organs like kidney, brain etc	Anemia in neonatal period
	Long term sequelae due to hypoxia

Perinatal mortality

Perinatal mortality rate (PNMR) depends on the gestational age, neonatal facilities and presence of associated complications like FGR. The CEMACH (2008) report states that placental abruption is a leading cause of stillbirths and deaths contributing to 20% of all stillbirths and 15% of neonatal deaths (1)Interestingly, it has been found that the rate of congenital malformations in pregnancies with placental abruption is increased threefold and most involve the central nervous system (2) Fetal growth restriction is associated in upto 80% of infants born before 36 weeks of gestation (3)

Haemorrhagic Shock

Blood loss in placental abruption is often underestimated due to the concealed bleeding that occurs in the retroplacental space and also because of the intravasation into the myometrium (causing what is known as the Couvelaire uterus). One has to prepared for postpartum haemorrhage due to uterine atony and DIC. It is important to promptly identify and correct the hypovolemia by infusing crystalloids, blood and blood products in order to avoid hypotension and shock. Every obstetric unit should have its own protocol customized to suit local circumstances in order to manage major obstetric haemorrhage occurring due to placental abruption and the guidelines should be followed without delay as soon as a major bleeding episode is recognized.

Disseminated intravascular coagulation (DIC)

Hypovolemia and hypoxia result in endothelial injury and the development of Systemic inflammatory response syndrome (SIRS). Vascular damage could be more severe with abruption resulting in activation of the coagulation cascade leading to early onset DIC. Placental abruption is the most common cause of severe consumptive coagulopathy. Hypofibrinogenemia, elevated levels of D-Dimers and reduction in coagulation factors occur in 30% of women. Overt hypofibrinogenemia often precedes thrombocytopenia. The mainstay of management in DIC is to manage the underlying disorder

in order to remove the initiating stimulus, to maintain circulating blood volume and to replace clotting factors and red blood cells.

Renal failure

Placental abruption is one of the common causes of renal failure, which is a rare but serious complication in pregnancy. However, three fourths of renal failure in abruption is reversible. Acute renal failure is caused by acute tubular necrosis which in turn is precipitated by hypoxia caused by hypovolemia in conjunction with DIC and its resultant ischaemia. Whereas non pregnant women who suffer an acute prerenal insult like haemorrhage,may develop transient acute tubular necrosis, the same prerenal insult in pregnancy is more likely to develop into renal cortical necrosis and permanent renal impairment. Hence prompt and vigorous fluid resuscitation cannot be overemphasized.

Couvelaire uterus

Widespread intravasation of blood into uterine musculature, beneath the uterine serosa and occasionally into the broad ligament as well as into the peritoneal cavity can be seen with severe placental abruption. Myometrial intravasation may interfere with uterine contractility and has to be managed with oxytocics. However Couvelaire uterus per se should NOT be an indication for hysterectomy.

Most often with placental abruption, prompt delivery is life saving for the mother and hopefully for the fetus. Early recognition,optimal and prompt resuscitation will remain the major factor to prevent mortality and reduce long term morbidity. Obstetricians should be aware that underlying thrombophilias could have caused the abruption. Hence liberal use of prophylactic anticoagulants and watching for signs of thromboembolism is recommended after the bleeding episode has been managed.

The chances of recurrence is 6-18% in subsequent pregnancies and this should be borne in mind when counseling regarding future pregnancies.

(This chapter deals mainly with acute placental abruption. Chronic placental abruption is unlikely to cause severe complications in the mother although it is often associated with increased perinatal morbidity due to fetal growth restriction, Rh isoimmunisation and fetal anemia.)

References

- Confidential Enquiry into Maternal and Child Health (CEMACH) Perinatal Mortality 2006: England, Wales and Northern Ireland. CEMACH: London, 2008.
- Egley C, Cefalo RC: Abruptio placenta. In Studd J (ed): Progress in Obstetrics and Gynaecology, Vol. 5. Edinburgh, Churchill Livingstone, 1985
- James DK, Steer PJ : High risk pregnancy Management options, Third edition: Bleeding in late pregnancy, Pages 1267-1268

9. Abruptio Placentae in PIH

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Abruptio placentae (ie, placental abruption) refers to premature separation of the normally located placenta after the 20th week of gestation and prior to birth.the reported incidence varies from 0.49% to 1.8%.Small episodes of abruption are more common(4.5%) than those diagnosed clinically as found by routine examination of placentas by Fox*Abruptio placentae* is commonly associated with Pregnancy induced hypertension. In 44% of cases of abruption hypertension has been found to be the itiological factor. Although this has been an observation there is no consensus on whether hypertension precedes abruption or vice versa. Naeye and colleagues found no evidence of placental abruption in patients with hypertension but Abdella and associates observed that the incidence of placental abruption in patients with preeclampsia was twice that in those without preeclampsia.In a metanalysis Ananth et al concluded that chronic hypertension was associated with a threefold increased risk of abruption compared with normotensive patients, whereas the odds ratio for patients with eclampsia was 1.73.

Pathophysiology

Placental abruption is associated with hypertensive pregnancies. These are life-threatening conditions for both the developing baby and the mother.Many theories have attempted to explain why preeclampsia arises, and have linked the syndrome to the presence of the following:

endothelial cell injury

immune rejection of the placenta

compromised placental perfusion

altered vascular reactivity

imbalance between prostacyclin and thromboxane

decreased glomerular filtration rate with retention of salt and water

decreased intravascular volume

increased central nervous system irritability

disseminated intravascular coagulation

uterine muscle stretch (ischemia)

dietary factors, including vitamin deficiency

genetic factors

All the above factors seem to contribute to the causation of abruption in PIH. Abruption is multifactorial in association. All the above factors seem to be either causative or responsible for deterioration and morbidity associated with abruption .Abruptio placentae is thought to occur as a result of degenerative changes in the arterioles in the deciduas basalis. This leads to ischemia in the decidua tissue making the vessels prone to rupture. Distended pregnant uterus prevents the smooth muscle from contracting

effectively to tamponade the bleeding vessels. A hematoma forms behind the placenta, increasing the pressure and causing further separation from the uterine wall with increased hemorrhage. Compression of the placental vessels and tissue by the hematoma can compromise circulation to the fetus. If the margins of the placenta remain intact, the bleeding may stay concealed while pressure within the hematoma continues to build. The buildup of blood causes the uterus to become irritable and tender. "Couvelaire uterus" is a phenomenon wherein the retroplacental blood may penetrate through the thickness of the wall of the uterus into the peritoneal cavity. This may occur after abruptio placentae. The hemorrhage that gets into the decidua basalis ultimately splits the decidua, and the haematoma may remain within the decidua or may extravasate into the myometrium (the muscular wall of the uterus). This may be responsible for the pain associated with abruption and tonically hard uterus .Pain Impliesw dissection of the myometrium by blood.The myometrium becomes weakened and may rupture due to the increase in intrauterine pressure associated with uterine contractions. This may lead to a life-threatening obstetrical emergency.The ruptured uterus would cause severe maternal hemorrhage and hypovolemic shock. The placenta may also be separated at its margin, allowing the blood to flow freely into the uterus. As a result of the severe bleeding, the maternal blood-clotting cascade is triggered and may lead to disseminated intravascular coagulation (DIC). Both severe maternal and fetal distress will occur. If a complete or near-complete abruption is present, fetal death is almost inevitable, unless an emergency cesarean section can be immediately performed. In addition a compromised renal function due to PIH may get further compounded and cause ischemic damage leading to acute or chronic renal failure. Severity of fetal distress correlates with the degree of placental separation. In near-complete or complete abruption, fetal death is inevitable unless an immediate cesarian delivery is performed. Abruptio placentae tends to occur early in the third trimester of pregnancy. Rasmussen et al.have reported similar findings and hypothesized that AP, preterm labor, pregnancy-induced hypertension,and intra-uterine growth restriction share a common etiologic factor or represent a clinical expression of recurrent placental dysfunction .

Prevention Treat maternal hypertension. Diagnose placental abruption at an early stage in high-risk groups (eg, maternal hypertension, maternal trauma, association with domestic violence, smoking habit, substance abuse, advanced maternal age, premature ruptured membranes, uterine fibromyomas, amniocentesis). The risk of placental abruption can be reduced by maintaining a good diet including taking folic acid, regular sleep patterns and correction of pregnancy-induced hypertension

Hemorrhagic shock, Coagulopathy, DIC, Uterine rupture, Renal failure, Ischemic necrosis of distal organs (eg, hepatic, adrenal, pituitary**)**

Fetal complications

Hypoxia,Anemia,Growth retardation,CNS anomalies,Fetal death

Medicolegal Pitfalls

- Some patients may not have the classic presentation of abruption, especially with posterior implantation.
- Consider a diagnosis of placental abruption for every patient in premature labor and PIH. Carefully monitor patients to exclude or establish this diagnosis.
- Absence of vaginal bleeding does not exclude placental abruption.
- DIC/coagulopathy may occur even if clotting factors initially are within reference ranges. Continue to monitor clotting factors.
- Normal ultrasound findings do not exclude placental abruption.

Follow –up :

Regular follow up and preconceptional counseling , nutritional corrections in subsequent pregnancy is important as Abruption is known to be recurrent.

References :

Rasmussen set al : the occurrence of placental abruption in Norway 1967-1991.Acta Obstet Gynecol Scand 1996;75:222-228

Fox H : Pathology of the Placenta. London, Saunders ,1978

Naeye RL,Harknes WL,Utts J:Abruptio Placentae and Perinatal death : A prospective study .Am J of Obstet Gynecol 1977;128:710-714

Abdella TN,Sibai BM,Hays JM,Anderson GD: Relationshipof hypertensive diseases to abruption placentae.Obstet Gynecol 1984;63:365-370

Ananth CV ,Savitz DA,Williams MA: Placental abruption and its association with hypertension and prolonged rupture of membranes: A methodological review and metanalysis. Obstet Gynecol1996;88:309-318

Rasmussen S, Lorentz M, Dalaker K. Outcome of pregnancies subsequent to placental abruptio, a risk assessment. ActaObstet Gynecol Scand 2000;79:496–501.