Antepartum Haemorrhage

Antepartum haemorrhage (APH) is defined as bleeding from the genital tract in pregnancy before the onset of labour at gestation from 20-24 weeks.

Aetiology:

The causes of APH can be divided into three main groups:
1-Placenta praevia.
2-Placental abruption.
3- Others:
-Marginal placental bleeding.
-Show.
-Friable cervical ectropion/cervical trauma.
- Local infection of the cervix/vagina.
- Genital tract tumours.
- Varicosities.
- Vasa praevia.
Placenta praevia and abruption together account for 50% of bleeding and represent the greatest threat to the fetus and mother.

PLACENTA PRAEVIA:

Placenta praevia is defined as a placenta partially or wholly situated in the lower uterine segment. It is graded in two ways, as either grades 1-4 or minor/ major.

1-Grade 1: the placental edge is in the lower segment but does not reach the internal os.

2-Grade 2: the placental edge reaches but does not cover the internal os.

These grades represent a minor degree of placenta praevia.

3-Grade 3: the placenta covers the internal os and is asymmetrically situated.

4-Grade 4: the placenta covers the internal os and is centrally situated.

These grades represent a major placenta praevia.

Diagnosis:

1-placenta praevia usually present with painless bleeding (though 10% will have concurrent abruption).

2-Often a small bleed will precede a much larger one (though this is not always the case).

3-The presenting part is usually high, being prevented from engaging by the placenta lying in the lower segment.

4-The fetal condition generally remains good until the maternal blood loss cause compromise.

It's difficult to diagnose placenta praevia until the lower segment begins to form about 28 weeks, however, a low lying placenta can cause bleeding from the second trimester. Many cases are now detected on routine ultrasound at 18-23 weeks.

Prognosis:

The chief causes of death in cases of placenta praevia are haemorrhage and shock. The amount of bleeding will be least with a type 1 but progressively greater with the more central types. Both antepartum and postpartum haemorrhage may occur. Some hazards arise from CS.

Treatment:

Following an episode of painless blood loss, the woman must be admitted to hospital as soon as possible. With more sever bleeding an intravenous infusion is set up before transferring the woman urgently to hospital. It is most important that no vaginal examination is made.

In all cases with sever bleeding immediate active treatment is required but in most common type of case seen today the woman admitted when the bleeding has only been slight and there is time for investigation, it is usually possible to make a diagnosis by clinical and ultrasound examination. The chief cause of fetal mortality used to be prematurity, but if bleeding was only slight and the fetus was some weeks premature,

then the risk of keeping the woman in bed while the fetus grew was justifiable, provided that the mother was in a hospital where treatment by blood transfusion and operation was available immediately should sever bleeding occurs. In few cases the diagnosis is still uncertain after ultrasound examination and in these few exceptional cases when the pregnancy is near term it may be justifiable to make a pelvic examination, provided that it is done in the operating theatre with the woman anaesthetized and ready for CS. A finger passed gently through the cervix. If a placenta of type 2,3or 4 is encountered or if moderate or severe bleeding is precipitated then CS is performed. If the placenta is of type 1 and particularly if situated anteriorly, low rupture of the membranes is done which allow the presenting part to descend and compress the lower margin of the placenta and so control bleeding.

PLACENTAL ABRUPTION:

This is defined as bleeding following premature separation of a normally sited placenta. It concealed in approximately one-third of cases (i.e. no blood loss is seen per vagina) and revealed in two-thirds of cases.

Risk factors for placental abruption:

1-hypertension.
2-smoking.
3-trauma to the abdomen.
4-crack cocaine usage.
5-anticoagulant therapy.
6-polyhydramnios.
7-low socio-economic group.
8-intrauterine growth restriction.

Clinical presentation:

The classical presentation is that of abdominal pain, vaginal bleeding and uterine contractions. The vaginal bleeding is usually dark and non-clotting, however, as the bleeding may concealed, its absence does not preclude the diagnosis. If blood loss is significant, there may be sign of hypovolaemic shock, with increased pulse rate, hypotension and signs of peripheral vasoconstriction. Abdominal palpation reveals a tender uterus that is often described as being (woody hard). The uterus may be

larger than gestation suggests and the fetus is often difficult to palpate. Depending on the size of the abruption, and the area of placental separation, the fetus may be dead, in distress or unaffected. Vaginal examination may reveal blood or cervical dilatation if the abruption has precipitated labour.

Diagnosis:

This is usually made on clinical ground. Where abruption has not been severe, the diagnosis may only be made by inspection of the placenta after the third stage of labour is complete. Ultrasound can be helpful in some cases, demonstrating retro-placental clot and excluding placenta praevia. The differential diagnosis of placental abruption can broadly divided into two groups: other causes of vaginal bleeding and other causes of abdominal pain in pregnancy.

Effect on the mother:

1-Hypovolaemic shock: There is a tendency to underestimate the amount of blood loss due to some haemorrhage being concealed behind the placenta and within the uterine wall. In addition some patients will have been hypertensive prior to abruption, masking the hypotensive effect of blood loss.

2-Disseminated intravascular coagulation: DIC is always a secondary phenomenon following a trigger to generalized activation of coagulation systems. Consumption of fibrin, clotting factors and platelets occurs, resulting in continued bleeding and further depletion of these factors. The triggers known to precipitate DIC include tissue thromboplastin release, endothelial damage to small vessel and pro-coagulant phospholipids production secondary to intravascular coagulation.

3-Acute renal failure: This is a consequence of poor renal perfusion, secondary to hypovolemia, hypotension and DIC.

4-Feto-maternal haemorrhage: This can lead to sensitization of the mother to fetal blood group antigens.

5-Maternal mortality: Placental abruption is a significant cause of maternal death, usually as a consequence of the complication listed above.

6-Recurrence: After a single episode of abruption, the recurrence rate is approximately 10 %, increasing to 25% after two episodes.

Effect on the fetus:

1-Perinatal mortality: abruption is a significant cause of fetal and neonatal loss.

2- Intrauterine growth restriction: where abruption is chronic or recurrent, the area of the placenta available for nutrient and waste exchange between the fetus and the mother is reduced.

Management:

A large abruption is an obstetric emergency as it is life threatening for both the mother and fetus. When fetal compromise is confirmed, the aim should be to deliver the fetus by CS. At very low gestation, a vaginal delivery should be the aim. Labour is often quick and although prostaglandin can be used, they are rarely needed.

If the fetus is already dead, a vaginal delivery should be the expectation. IF abruption is small, the fetus uncompromised and the mother well, a conservative approach may be utilized.

Vasa praevia:

Vasa praevia, rupture of vessels on the fetal side of the placenta. It is fetal blood that is lost. Risk factors include placenta praevia and multiple pregnancy. The vessels are usually in a velamentous insertion of the cord. In these cases the fetal vessels cross the cervix and they may rupture at spontaneous rupture of membranes or be damaged at artificial rupture of membranes. Relatively slight bleeding is seen but can cause acute fetal exsanguinations and death, major fetal heart rate changes soon become apparent. Fetal tachycardia develops, followed by deep deceleration. The best solution is high index of suspicion and rapid CS.