UNIT 7 COMPLICATIONS IN LATE PREGNANCY–I (Pregnancy Induced Hypertension and Antepartum Haemorrhage)

Structure

7.0 OBJECTIVES

After going through this unit, you should be able to:

- diagnose preeclampsia early and institute treatment;
- provide essential care in eclampsia before referring to a tertiary health care centre;
- list the complications of eclampsia;
- advise women with preeclampsia and eclampsia regarding the importance of postnatal care;
- define the terms antepartum haemorrpage, placenta praevia, abruptio placentae, vasa praevia;
- list the causes of antepartum haemorrhage;
- describe the diagnostic features of abruptio placentae, vasa praevia, placenta praevia and indeterminate bleeding; and
- discuss the management of placenta praevia, abruptio placentae, vasa praevia and indeterminate bleeding.

7.1 INTRODUCTION

In Unit 6, you learnt about the complications during first trimester of pregnancy. This unit deals with the important complications of preeclampsia, eclampsia and antepartum heamorrhage, which are important causes of maternal mortality. Though eclampsia is preventable, preeclampsia cannot be prevented but early identification of preeclampsia and prompt management help in avoiding the serious complications of eclampsia. Ante-partum haemorrhage includes mainly placenta praevia and abruptio placentae, which contributes to a large extent to the deaths caused by haemorrhage. You will learn about the diagnosis and appropriate management of these conditions at various levels of health care in this unit.

7.2 PREECLAMPSIA AND ECLAMPSIA

Reading this Section will help you understand the principles of management of preeclampsia and eclampsia. A peripheral hospital is not equipped to adequately treat eclampsia and we have made an effort to help you to decide when to refer patients to better equipped hospital.

7.2.1 Preeclampsia

Preeclampsia is the development of hypertension with proteinuria, edema or both induced by pregnancy after the 20th week of gestation and sometimes earlier in presence of hydatidiform mole.

a) **Pathophysiology**

Why do women develop preeclampsia? Exact etiology of this condition is still controversial. However the main basic problem in this condition is uteroplacental arterial insufficiency causing placental ischaemia and hypoxia. 'Vasospasm' is basic to the pathophysiology of preeclampsia. Vascular cosnstriction causes a resistance to blood flow and accounts for the development of arterial hypertension.

Women with preeclampsia have an increased vascular reactivity to angiotensin II which precedes the onset of hypertension. Moreover angiotensin II appears to have a direct action on endothelial cells causing them to contract. These factors lead to inter endothelial cell leaks through which blood constituents including platelets and fibrinogen are deposited subendothelially. The vascular changes together with local hypoxia of the surrounding tissues presumably lead to hemorrhage, necrosis and other end organ disturbances that have been observed at times with preeclampsia.

The pathology in preeclampsia is not restricted to the placenta. A number of changes occur in other systems of the body and these contribute to the clinical manifestations.

i) ***Cardiovascular Changes***

There is a virtual absence of pregnancy induced hypervolemia in preeclampsia. The causes could be:

- Generalised vasoconstriction
- Increased vascular permeability leading to less fluid intravascularly and marked excess extravascularly.

Therefore these women are extremely sensitive to even normal blood loss at delivery.

ii) ***Hematological Changes***

- Intravascular coagulation
- Thrombocytopenia
- Evidence of erythrocyte destruction in the form of hemolysis, hemoglobinuria
- Reduced antithrombin III

iii) ***Endocrine Changes***

Reduced serum angiotensin II and aldosterone.

iv) ***The Kidney***

- Diminished glomerular filtration
- Proteinuria

Microscopic picture includes:

- Glomerular capillary endothelial swelling
- Sub endothelial deposits of protein material

These together constitute capillary endothelial.

v) ***The Liver***

Serum liver enzyme elevation due to periportal haemorrhagic necrosis in the periphery of the liver lobule.

Bleeding may extend beneath hepatic capsule and form a subcapsular hematoma giving rise to epigastric pain in severe preeclampsia.

vi) ***The Brain***

Cerebral oedema, hyperemia, focal anemia, thrombosis and hemorrhage may be seen in cases of severe preeclampsia.

b) **Classification**

Pregnant women may be detected to have hypertension for the first time during pregnancy but this hypertension may develop during pregnancy, antedate the pregnancy or be aggravated during pregnancy. Hence, hypertension in pregnancy may be classified as:

i) ***Pregnancy Induced Hypertension (PIH)***

PIH is defined as hypertension that develops as a consequence of pregnancy and regresses in Postpartum. It could be:

- Hypertension without proteinuria or oedema.
- Preeclampsia with proteinuria and/or oedema
  - Mild
  - Severe
- Eclampsia—preeclampsia with convulsions.

ii) ***Pregnancy Aggravated Hypertension***

Underlying hypertension worsened by pregnancy.

- Superimposed preeclampsia
- Superimposed eclampsia

iii) ***Coincidental Hypertension***

Chronic underlying hypertension that antecedes pregnancy or persists postpartum.

c) **Diagnosis**

Main clinical features of preeclampsia are rise in blood pressure, abnormal weight gain, oedema and proteinuria.

i) ***Blood Pressure***

- It is the most dependable sign
- If blood pressure is 140/90 mmHg or more or an increase of 30 mmHg systolic or 15 mm diastolic over baseline values on at least 2 occasions 6 or more hours apart, is diagnostic of preeclampsia.

ii) ***Weight Gain***

Preeclampsia should be suspected if the weight gain is sudden and excessive i.e. more than 2 pounds in a week or 6 pounds in a month.

iii) ***Oedema***

- oedema over the hands and face that persists even after adequate rest may be associated with preeclampsia
- dependent oedema that disappears in the morning may be associated with normal pregnancy.

iv) ***Proteinuria***

- It is defined as 300 mg or more of urinary protein in 24 hours or 100 mg per dl or more in at least two random urine specimens collected 6 hours or more apart.
- Almost always it develops later than hypertension and usually later than excessive weight gain.

- It is a sign of worsening preeclampsia and if it is persistent the foetal and maternal risks are markedly increased.

Preeclampsia is not preventable but can be detected early by good antenatal care.

Women with mild preeclampsia are usually asymptomatic. Thus its early detection demands careful observation at appropriate intervals.

- Pregnant women should be examined every 2 weeks from the seventh month and weekly in the 9th month or more often if blood pressure rises.
- At these visits the woman is weighed and careful blood pressure measurements are made. Rapid weight gain any time during the latter half of pregnancy or an upward trend in the diastolic blood pressure, even while still in the normal range should be taken serious note of.

Woman with severe preeclampsia may have the following symptoms:

1) *Headache*
   - is usually frontal
   - is not relieved by ordinary analgesics
   - severe headache usuaIly precedes the first convulsion
2) *Epigastric Pain or Right Hypochondriac Pain*
   - is a symptom of severe preeclampsia
   - may be indicative of imminent convulsions
   - is due to stretching of liver capsule by oedema or hemorrhage
3) *Visual Disturbances*
   - may range from slight blurring of vision to partial or complete blindness
   - occur due to ischaemia of retina or occipital cortex

d) **Effect of PIH on Pregnancy**

Effect of PIH /Preeclampsia on mother and foetus is given below :

On Mother:

- Eclampsia
- Abruptio Placentae
- Pulmonary edema
- Cerebral haemorrhage
- Acute renal failure
- DIC (Disseminated Intravascular Coagulation)

On Foetus:

- IUGR
- Foetal distress
- Prematurity
- IUD (Intra Uterime Death)

e) **Management**

Management of preeclampsia will depend upon the severity of preeclampsia. Preeclampsia is classified into mild and severe depending upon the following clinical and laboratory features.

Abnormality		Mild	Severe
a) **Clinical**			
1)	Diastolic blood pressure	< 100 mmHg	> 100 mmHg
2)	Headache	Absent	Present
3)	Visual disturbance	Absent	Present
4)	Epigastric pain	Absent	present
5)	Oliguria	Absent	Present
b) **Biochemical**			
1)	Proteinuria	Trace to 1+	2+ or more
2)	Serum creatinine	Normal	Elevated
3)	Thrombocytopenia	Absent	Present
4)	Hyperbilirubinemia	Absent	Present
5)	Liver enzyme elevation	Minimal	Marked
6)	Fetal growth retardation	Absent	Present
7)	Pulmonary oedema	Absent	Present

All the above features need not be present to classify preeclampsia as severe. The following examples will illustrate this:

1) A woman with BP of 140/90 with .severe headache would be classified as severe preeclampsia although the blood pressure is in the range of mild preeclampsia.

2) A diastolic blood pressure of more than 110 mmHg irrespective of the presence or absence of other clinical or biochemical parameters is classified as severe preeclampsia.

i) ***Role of Home Management in Mild PIH***

Women with BP ranging from 130/90 to 150/90 mmHg can be managed at home if they are not willing for admission a hospital. They should be counselled that any time BP may rise and they may develop threatening eclampsia. Hence, if they develop headache, visual disturbances such as blurring vision, double vision or blindness, has epigastric pain or oliguria, they should immediately report to the FRU.

At home women with mild PIH should rest in left lateral position and their daily BP is to be checked. If BP monitoring is not possible, they may be called to the health centre after 2-3 days. If there is no improvement or it has gone up, she may be admitted in PHC for BP monitoring and to ensure bed rest. If BP becomes less or remains the same, they may be kept in PHC. Maternal and foetal conditions are monitored daily. They require referral to a FRU if BP goes up, they develop proteinuria or they have completed 37 weeks of gestation.

ii) ***Management in Hospital***

Management of preeclampsia may be discussed under the following headings:

- General management
- Specific management
  - Drug treatment
  - Termination of pregnancy

Management of Mild Preeclampsia

Patients with mild preeclampsia need not be admitted and can be managed at home as described earlier.

Management of Severe Preeclampsia

A patient of severe preeclampsia or with mild preeclampsia who do not respond to out patient treatment needs hospitalization.

a) *General Management*

The general management includes:

1) An appropriate history and general physical examination followed by daily search for the development of signs and symptoms such as headache, visual disturbances, epigastric pain and rapid weight gain.

2) Blood pressure reading every 4 hours except between midnight and morning.

3) Abdominal girth and fundal height record.

4) Weight measurement at admission and every 2 days thereafter.

5) Bed rest throughout much of the day.

6) Diet with adequate protein and calories.

7) Sodium and fluid intake should be neither limited nor forced.

8) Urine test for protein at admission and subsequently at least every 2 days.

9) Measurements of plasma creatinine, uric acid, hematocrit, platelets and serum liver enzymes.

10) Serial sonography.

b) *Drug Treatment of Pregnancy Induced Hypertension*

Arbitrarily the blood pressure above which medication is required has been taken as 170/110 mmHg. If in any 24 hour period two measurements reach the threshold, the treatment is started. Drug treatment can be divided into:

— Acute control of hypertension

— Longer term control of hypertension

l *Acute Control*

A sudden rise in blood pressure requires a smooth and sustained reduction. The following drugs may be used.

i) **Nifedipine** can be given orally or sublinguially. Headache and tachycardia are the side effects.

ii) **Hydralazine** may be given by intravenous/intramuscular route. Its onset of action is in 20-30 minutes. Side effects are headache, tachycardia, fetal distress.

iii) **Labetalol** is a combined alpha and beta adrenergic blocking agent. It lowers blood pressures smoothly and rapidly without causing headache.

l *Longer Term Control*

i) **Methyldopa** given in a loading dose of 500-1000 mg followed by 250-750 mg four times a day can control the blood pressure within 6-12 hours. Tiredness and postural hypotension may occur. Its safety in pregnancy has been well established.

ii) **Beta blockers** include atenolol, oxprenolol and labetalol. They have the advantage of causing fewer subjective side-effects but their safety in pregnancy is not sufficiently established.

c) *Termination of Pregnancy*

The cure for preeclampsia is delivery. In mild controlled preeclampsia pregnancy may be continued till 37 weeks when the foetus has a better chance of survival. Uncontrolled or severe preeclampsia should preferably be managed in a bigger hospital with facilities for intensive maternal and fetal care. These women require immediate termination of pregnancy under the care of a specialist gynaecologist.

Check Your Progress 1

1) The basic pathophysiology in preeclampsia is .....

2) Which organ systems of the body manifest changes of preeclampsia?

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3) What are the hematological changes in preeclampsia?

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4) How is hypertension in pregnancy classified?

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5) List the main clinical features of the preeclampsia.

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6) Name the drugs used for acute control of hypertension.

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7) Name the drugs used for long term control of hypertension.

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8) The cure for preeclampsia is .....

7.2.2 Eclampsia

You have just read about preeclampsia. If a pregnant woman with preeclampsia is not treated properly or despite treatment continues to worsen, this may lead to eclampsia. Eclampsia is characterised by generalized tonic clonic convulsions in addition to other features of preeclampsia.

Eclampsia is regarded with particular concern due to its associated high maternal and fetal mortality. Hence we shall now discuss the pathophysiology diagnosis, management and complications of this condition.

a) **Pathophysiology**

What causes the convulsions of eclampsia?

The cause of convulsion is not exactly known. Non specific electro encephalographic abnormalities can usually be demonstrated for some time after eclamptic convulsions. The most common findings on computed tomography are hypodense cortical areas that correspond to petechial hemorrhage and infarction sites. The principal post mortem cerebral lesions are oedema, hyperemia, focal anemia, thrombosis and haemorrhage.

Why all women with preeclampsia do not have convulsions?

This is difficult to explain, perhaps the brain like the liver and kidney may be more involved ~ in some women than in others and the extent of ischaemia and petechial sub cortical lesions further altered by an inherent seizure threshold, influences the incidence of eclampsia.

b) **Diagnosis**

Eclampsia characterised by convulsions may occur in the antenatal period (antepartum), during labour (intrapartum) or after delivery (postpartum). Eclampsia is most common in the last trimester and becomes increasingly more frequent as term approaches. Nearly all cases of post partum eclampsia develop within 24 hours of delivery. Almost without , exception eclampsia is preceded by warning features of hypertension, proteinuria, headache; visual disturbance and epigastric or right upper quardrant pain.

The convulsion consists of four stages:

i) **Premonitory stage:** It begins with facial twitchings and onset of unconsciousness and lasts for about 30 seconds.

ii) **Tonic stage:** The whole body becomes rigid in a generalized muscular contraction. This phase lasts for 15 to 20 seconds. Respiration ceases and cyanosis appears.

iii) **Clonic stage:** All the voluntary muscles undergo alternate contraction and relaxation starting from the jaw. The tongue may be bitten and the woman may be thrown out of the bed. Blood tinged froth may exude from the mouth. This state lasts for one minute.

iv) **Stage of coma:** Gradually the movements become less frequent and the woman lies motionless.

Throughout the seizure the woman is apneic. The breathing is resumed after a long deep inhalation. Coma follows a convulsion and may last for a few minutes or persist from on convulsion to another.

After a convulsion the respiratory rate become fast. There may be cyanosis and fever which is a poor prognostic sign.

Proteinuria and oedema are almost always present. Oliguria and haemoglobinuria may also be seen.

c) **Differential Diagnosis**

The dictum is ***'Until other causes are excluded all pregnant women with convulsions should be considered to be eclamptics'.***

The other causes of convulsion in pregnancy include epilepsy, encephalitis, meningitis, cerebral tumour, ruptural cerebral aneurysm and even hysteria. These should be borne in mind whenever convulsion or coma develop during pregnancy.

d) **Management**

It should be emphasised that a patient of eclampsia requires intensive care in a specialised obstetric unit. The patient should be referred to the tertiary centre at the earliest after giving preliminary first aid. A medical personnel should accompany the patient to the referral centre. The patient must be sedated before moving her to the hospital. Anyone of

the available drugs may be used—diazepam 10 mg IV, largactil 50 mg and phenargan 25 mg or morphine 15 mg or pethidine 50 mg and phenargan 25 mg. If an obstetric flying squad is available it is the ideal one to shift the patient.

Treatment of eclampsia consists of:

- General care of the unconscious
- Control of convulsions
- Blood pressure control
- Delivery after control of convulsion

General Care

a) The patient should be nursed in a lateral position on a railed cot in an isolated room.

b) After the patient is sedated IV line is started. Total fluid intake should not exceed 2 litres in 24 hours.

c) The air passage is to be cleared of mucus at frequent intervals with a suction catheter.

d) A gag is placed between the teeth to prevent tongue bite.

e) Oxygen by mask should be given.

f) Antibiotics like penicillin should be given to prevent infection.

g) Bladder should be catheterised.

h) Blood pressure, pulse and respiration to be recorded 1/2 hourly.

i) Intake-output record to be maintained.

Control of Convulsions

Various regimes have been used to treat convulsion in eclampsia. Two of them are described below:

1) *Magnesium Sulphate*

This is the recommended regime by the Govt. of India and is commonly practiced initial dose consists of 4 gm of magnesium sulphate given intravenously as a 20% solution at a rate not to exceed 1 gm/min. This should be followed by 10 gm of 50% solution (5 gm in each buttock) injected deeply. If convulsions persist after 15 minutes, 2 gm more may be given intravenously as a 20% solution at a rate not to exceed 1 gm/min.

Every 4 hours thereafter 5 gm of 50% solution may be injected deep intramuscularly but only after ensuring that:

a) the patellar reflex is present

b) respiration is not depressed

c) urine output in the previous 4 hours exceeds 100 ml.

Magnesium sulphate is discontinued 24 hours after delivery.

2) *Lytic Cocktail Regime*

This regime is safe and simple to regulate.

Initially 25 mg chlorpromazine and 100 mg pethidine in 20 ml of 5% dextrose are given intravenously along with 50 mg chlorpromazine and 25 mg phenargan intramuscularly. Subsequently 25 mg phenargan and 50 mg chlorpromazine are given alternatively at 4 hourly interval upto 24 hours following the last fit. Intravenous 500 ml of 10% dextrose with 100 mg pethidine is started from the beginning at a drip rate of 20-30 drops per minute. Not more than 2 litres of dextrose and 300 mg pethidine are given in 24 hours. The pethidine drip should be continued up to 24 hours following the last fit.

3) *Phenytoin Sodium (Dilantian Sodium)*

This is tried in certain centres. More reports are awaited for its widespread use.

Blood Pressure Control

If the blood pressure remains more than 150/100 mmHg anithypertensives should be given. Any of the following drugs may be given.

a) Hydralazine 5 mg I/v slowly repeated in 20 min if no response is seen.

b) Nifedepine 5 mg sublingual may be given repeated after half an hour if required. Sudden hypotension is an adverse effect which should be looked for.

Delivery after Control of Convulsion

In eclampsia, labour often starts spontaneously or can be induced successfully even if the women is remote from term. Serious morbidity is less common during puerperium in women delivered vaginally. Hence vaginal delivery is attempted and is quite often successful. The women with eclampsia lack normal pregnancy hypervolemia and are thus much less tolerant of blood loss than a normotensive pregnant woman.

e) **Complications**

Many organ system are affected in patients of eclampsia since it is a multi system disorder. The complications include the following:

i) **Aspiration pneumonia** follows inhalation of gastric contents if vomiting accompanies convulsion and airway is not adequately maintained.

ii) **Pulmonary oedema** may be the result of the combination of severe hypertension and vigorous intravenous fluid administration. If not treated promptly, it is associated with a poor prognosis.

iii) **Hemiplegia** may be caused by cerebral haemorrhage.

iv) **Coma** or altered sensorium may follow seizures. It may be caused by cerebral oedema or ruptured vessels and requires supportive management.

v) **Blindness** may be due to retinal detachment or occipital lobe ischaemia. In either case the prognosis for return of normal vision is good and usually complete within a week.

vi) **In Psychosis** the patient may become violent following seizures. The state may last up to two weeks and has a good prognosis.

vii) **Renal failure** requires supportive treatment and the renal function usually returns to normal.

viii) Sudden **death** may occur along with a convulsion or shortly thereafter usually as a result of massive cerebral haemorrhage. Pulmonary oedema is another important cause of maternal death in eclampsia. .

7.2.3 Post Partum Care

After delivery, there is usually rapid improvement of blood pressure and proteinuria. However, the patient should be closely monitored for at least 48 hours. One must remember that convulsions may occur upto 48 hours after delivery.

Apart from the routine post partum care the following regime should be followed:

During Hospital Stay

i) Blood pressure should be checked every two hours.

ii) Antihypertensives should be given if diastolic BP is ≥ 100 mmHg.

iii) If patient appears restless she should be sedated.

iv) Anti convulsant regimes if started before delivery should be continued for at least 24 hours after delivery.

v) The dose of antihypertensive should be reduced according to BP records.

vi) Patient should be kept in the hospital till BP is brought down to safe levels and proteinuria disappears.

After Discharge from Hospital

i) The patient is reevaluated 2 weeks later. Most often hypertension induced by pregnancy dissipates spontaneously during the first 2 weeks post partum. If hypertension persists a beta blocker or nifedipne may be prescribed.

ii) Hypertension persisting 6 weeks after delivery usually signifies chronic vascular disease, often essential hypertension. Basic investigations like urine protein, microscopic examination, blood urea, serum creatinine, chest X-ray and ultrasound of kidneys should be done. The patient should be followed closely to ascertain the cause of hypertension and whether the antihypertensives should be stopped or continued. It may be better to refer the patient to a specialist for further evaluation.

Contraception

The patient should be advised to avoid pregnancy for at least one year. Intrauterine contraceptive devices are an effective method of contraception. Double barrier (condom and spermicide) are an alternative. Oral contraceptives are not contraindicated if the BP comes back to normal after delivery though they should be avoided during lactation.

Check Your Progress 2

1) What are the warning symptoms that precede eclampsia?

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2) What are the stages of an eclamptic convulsion?

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3) What are the prerequisites before repeating a dose of magnesium sulphate?

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4) List the complications of eclampsia.

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7.3 ANTEPARTUM HAEMORRHAGE (APH)

Antepartum haemorrhage is defined as haemorrhage from the genital tract after 28 weeks of pregnancy (bleeding befor 28 weeks of pregnancy is called abortion).

Causes

Bleeding from the genital tract after 28 weeks of pregnancy can occur because of the following conditions:

- Placenta praevia
- Abruptio placentae
- Vasa praevia

- Extra placental incidental causes
- Indeterminate causes

Incidence of antepartum haemorrhage among hospital deliveries varies from 2-3%. In 2/3 of the cases the causes is either placenta praevia or abruptio placentae. Vasa praevia and extra placental incidental causes like cervical and vaginal lesions are rare. In about 1/3 of the cases no cause can be found and these form the unclassified or indeterminate bleeding (Fig. 7.1).

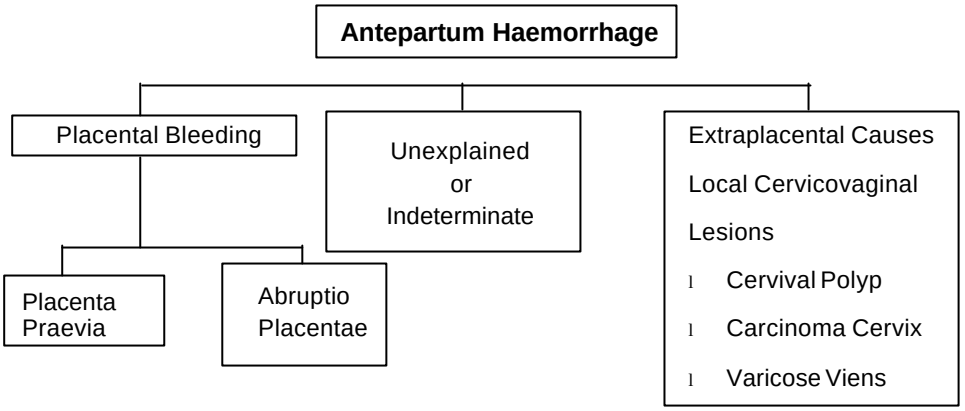

Fig. 7.1: Causes of Antepartum Haemorrhage

7.3.1 Placenta Praevia

Lower uterine segment has been defined in different ways:

Anatomical Definition: It is that part of the uterus which lies below the level at which the peritoneum on the anterior surface of the uterus ceases to be intimately applied to the uterus.

Metric Definition: It is that portion of the uterus which, towards term, lies within three inches (7.5 cm.) from the cervical os. It represents the distance over which the uterine cavity can be explored by the examining finger passed through the cervix during vaginal examination.

Physiological Definition: It is that part of the uterus which passively stretches in labour and takes hardly any active contractile part in the expulsion of foetus (Donald I).

a) **Incidence**

Placenta praevia occurs in about 1 in 200 deliveries. The frequency with which the zygote implants in lower part of the uterus is much higher but many of these pregnancies end in abortions. In many others the placenta migrates and comes to lie in the upper uterine segment. Only in few cases where it is implanted over the os, it persists as placenta praevia.

b) **Aetiology and Associated Conditions**

Aetiology

Placenta praevia is caused by implantation of the blastocyst at a site low in the uterine cavity but what causes this low implantation is not known. There are certain factors which predispose to placenta praevia and these are:

Multiparity: Multiparity somehow predisposes to placenta praevia. The incidence of placenta praevia is much higher, about 5% in grand multipara as compared to 0.2% in nulliparous.

Advanced Maternal Age: Placenta praevia is 2-3 times more common in women after 35 years as compared to women less than 20 years.

Placental Size: Incidence of placenta praevia is higher in twin pregnancy, presumably because of large placental size.

Uterine Scars and Pathology: Placenta praevia is found more commonly in cases with previous caesarean section, previous dilation and curettage, myomectomy and endometritis.

Placental Pathology: Marginal or velamentous cord insertions, succinturate lobes, bipartite placenta and fenestrated placenta are more commonly found in placenta praevia.

Smoking: Smoking at any time increases the risk of developing placenta praevia. Compensatory placental enlargement due to carbon mono oxide hypoxaemia could be the cause.

Associated Conditions

Abnormal Placentation: Placenta accreta and percreta may be associated with placenta praevia specially if there is a caesarean scar. The decidua is poorly developed in the lower uterine segment and therefore the placenta may get morbidly adherent to the uterus and give rise to third stage complications. .

Malpresentations: In placenta praevia the bulk of the placenta in the lower uterine segment may prevent the engagement of foetal head and predispose to malpresentation. Therefore, if high floating headlbreech presentation, oblique or transverse lie is present, one should suspect the possibility of placenta praevia.

Congenital Anomalies: Incidence of congenital anomalies is higher in placenta praevia. The reason is not known.

c) **Classification**

There are four types of placenta praevia depending on the degree of extension of placenta into the lower uterine segment (Fig. 7.2) .

Fig. 7.2: Types of placenta praevia

Type I (Low lying placenta): Placenta encroaches on the lower uterine segment (it lies within 5 cm of the internal os) but does not extend to the internal os.

Type II (Marginal placenta praevia): Placenta reaches the intern,all os but does not cover it.

Type III (Partial placenta praevia): Placenta covers the os but ceases to do so as the cervix dilates.

Type IV (Total placenta praevia): Placenta covers the internal os even when the cervix is dilated.

d) **Clinical Features**

Symptoms

The only symptom of placenta praevia is vaginal bleeding. The bleeding is sudden in onset, painless, apparently causeless and recurrent. The bleeding may vary from being slight to profuse. The first bout of bleeding may not be alarming and it usually but not always ceases spontaneously, only to recur later. Bleeding in some cases may not occur till the onset of labour, then it may be profuse.

Signs

The general condition of the patient is proportionate to the amount of blood loss. If the bleeding has been profuse, all the signs of shock may be present. These include pallor, cold and clammy skin, dyspnoea, restlessness, agitation, syncope, anxiety, confusion, falling blood pressure, tachycardia and oliguria or anuria.On abdominal examination size of the uterus corresponds to period of gestation. Uterus is relaxed, soft and without any areas of tenderness. The foetal parts are palpable, presenting part is high up, foetus may present in oblique or transverse lie. Foetal heart sounds are present unless there is major degree of placental separation, hypovolaemic shock or cord accident.

e) **Diagnosis and Differential Diagnosis**

Diagnosis

The antepartum diagnosis of placenta praevia is confirmed by transabdominal and endovaginal ultrasound.

Vaginal Examination must not be done as it can provoke further separation of placenta with torrential bleeding. Vaginal examination is done only in the operation theatre prior to termination of pregnancy and is discussed in the management. Speculum examination after bleeding has stopped for 48 hour to exclude local cause is also discussed in management.

The accuracy of transabdominal ultrasound in the diagnosis of placenta praevia is excellent with false positive and false negative rates of 7 and 8% respectively. This accuracy is even higher when a vaginal probe is used. Both transabdominal and endovaginal ultrasound are safe techniques with minimal or no risk for mother and foetus.

Differential Diagnosis

Placenta praevia has to be distinguished from other causes of antepartum haemorrhage specially the abruptio placentae. The differentiating features of placenta praevia and abruptio placentae are:

	Criteria	Placenta Praevia	Abruptio Placentae
l	Nature of bleeding	— Painless, causeless and recurrent	— Painful, often localised to start with and later becomes generalised, attributed to toxaemia or trauma and is continuous
		— Bleeding is always revealed	— Bleeding is revealed, concealed or usually mixed
l	General condition and anaemia	Proportionate to blood loss	Out of proportion to the visible blood loss in concealed variety
l	Features of toxaemia	Not relevant	Present in one-third of cases
l	Height of uterus	Proportionate to gestational age	May be disproportionately enlarged in concealed type
l	Feel of uterus	Soft and relaxed	May be tense tender and rigid
l	Malpresentation	Common. The head is high floating	Unrelated, the head may be engaged
l	FHS, foetal heart sound	Usually present	Usually absent, specially in concealed type
l	Localisation of placenta	Placenta in lower segment	Placenta in upper segment
l	Vaginal examination	Placenta felt in lower segment	Placenta is not felt in lower segment

Local lesions in cervix and vagina can be ruled out by doing a per speculum examination but the placenta praevia may coexist with them.

f) **Management**

There are two principles of management of antepartum haemorrhage:

1) All women with antepartum haemorrhage must be evaluated in a hospital capable of managing massive haemorrhage and with all the facilities for operative delivery.

2) A vaginal or rectal examination must not be performed till all preparations for immediate caesarean section and blood transfusion are available.

All cases of antepartum haemorrhage should be regarded as due to placenta praevia unless proved otherwise.

Management at Home

A woman who starts bleeding at home must be instructed to remain in bed and if she is still bleeding when first seen, 15 mg of morphine should be given intra muscularly and intravenous fluids should be started. A quick assessment of blood loss is made. Pulse, blood pressure is recorded and abdomen is palpated gently mainly to exclude any area of uterine tenderness or hardness suggestive of abruptio placentae. Fundal height is marked to detect any increase later on. F.H.S. is auscultated. Vulva is examined for any bleeding and no vaginal examination is done. **All patients of A.P.H. must be admitted to hospital.** This is important because the initial bleeding may be small and may cease spontaneously but it can recur at any time and may be life threatening.

Management in Hospital

On admission an overall assessment of the patient is made. History is taken and complete examination except vaginal examination is done. Blood samples are taken for haemoglobin, grouping and cross matching and coagulation studies. Routine urine examination for albumin sugar is done. Intravenous line is established with atleast 18 gauze needle. Further management of placenta praevia will depend on the amount of bleeding, maturity of foetus and whether patient is in labour.

In cases with preterm live foetus, with minimal bleeding expectant line of treatment is given. Active management of patient is done in cases where bleeding is excessive or it continues, or where foetus is mature or patient is in labour.

Expectant Treatment

Expectant line of treatment is always at the hospital. Absolute bed rest is given for at least 5 days after the bleeding stops. Careful monitoring of pulse, blood pressure, fundal height, foetal heart and vaginal bleeding is done. Rh -ve woman should be given Anti-D immunoglobulin. Ultra sound examinations is done to localise placenta and to exclude any foetal malformation. When bleeding stops perspeculation examination is done to exclude incidental causes. Anti anaemic treatment is given and patient is kept in hospital, under observation.

Aim of expectant treatment is to continue pregnancy till 38 weeks i.e till the foetus is clinically viable. Active management is termination of pregnancy which is described below.

Active Management

Active management is indicated in following conditions:

1) In eases who have been on expectant treatment and reach 38 weeks of pregnancy.

2) In cases who are admitted with severe bleeding.

3) Where bleeding recurs and continues with expectant management.

4) Where on admission pregnancy is more than 38 weeks.

5) In patients who are in labour.

6) Where foetus is dead or congenitally malformed.

In cases who have no bleeding or minimal bleeding active management includes examination of patient in operation theatre with all preparations for an immediate caesarean section and blood transfusion. A gentle per speculum examination is done, if not already done, to exclude local incidental causes in the cervix and the vagina like polyps, varicose veins etc. A vaginal examination is carefully done, first through the fornices and if no bogginess is felt, then finger is introduced through the os to feel for placenta. If no placenta is felt, the finger is swept above the os in a concentric fashion till whole of the lower segment is explored. If no placenta is felt or it is type I or type II anterior, rupture of membranes is done and patient is monitored for vaginal delivery. Oxytocin drip, if not contraindicated is given.

In type II posterior, type III, type IV placenta praevia or in cases where bleeding continues following rupture of membranes or foetal distress is detected caesarean section is done. In cases where bleeding is excessive or there are associated factors like malpresentation a caesarean section is indicated without a prior per vaginal examination.

In cases with hypovolaemic snock two intravenous infusions with large caliber should be established to allow rapid transfusion of blood and fluids but caesarean must not be deferred, unless, patient has already stopped bleeding when restorative measures may be carried out before starting caesarean section.

Lower segment caesarean section is preferred. In cases where placenta is implanted anteriorly, the placenta is sepatated manually to reach the margin, membranes are ruptured and baby delivered. Alternatively one may cut through the placenta and deliver the baby. In both these conditions the cord must be clamped promptly to prevent further foetal blood loss. New born must be monitored carefully and it may require blood transfusion.

Management of Third Stage

Post partum haemorrhage is common in cases of placenta praevia. Even a small amount of blood loss in third stage may lead to a shock in an already exsanguinated patient. The effects of bleeding are more marked in placenta praevia but post partum haemorrhage is most likely to occur because of following reasons:

1) Placental site is in lower uterine segment which does not contract and retract as efficiently as the upper segment.

2) Placenta is larger and thinner and may not separate completely.

3) Placental site is larger.

4) There may be morbid adhesions of placenta.

Blood should, therefore, be always available and prophylactic measures must be adopted to minimise the third stage bleeding. High dose syntocinon infusion and methergin soon after the second stage is sufficient in most cases.

Serious post partum haemorrhage should be managed with carboprost, manual uterine compression and uterine packing. If these fail, internal iliac artery ligation or hysterectomy may be required specially in cases of placenta accreta. Before hysterectomy, haemostatic sutures in the placental bed may be tried. In case the general condition of the patient is not good hysterectomy must not be delayed.

Scheme of management of placenta praevia is given in Fig. 7.3.

g) **Complications**

Placenta praevia is one of the most common complications of pregnancy. Maternal and foetal mortality and morbidity can be very high unless timely proper management is given to the patient.

Maternal Complications

Severe antepartum haemorrhage, shock and death may result from placenta praevia. Deat may also occur as a result of intra partum and post partum haemorrhage, operative trauma or infection. Placenta accreta is a serious complication and may add to the mortality and morbidity of placenta praevia.

Foetal Complications

Prematurity accounts for 60% of the perinatal mortality in placenta praevia. Intrauterine asphyxia, birth injury, congenital anomaly and foetal haemorrhage are the other causes of foetal death in placenta praevia.

h) **Prognosis**

Maternal

With good management it has been shown that maternal mortality can be brought down to zero. This is achieved because of the availability of blood, timely caesarean section, safe anaesthesia and antibiotics. Good antenatal care with elimination of anaemia and routine diagnosis of placenta praevia on ultrasound also contributes to lowered mortality.

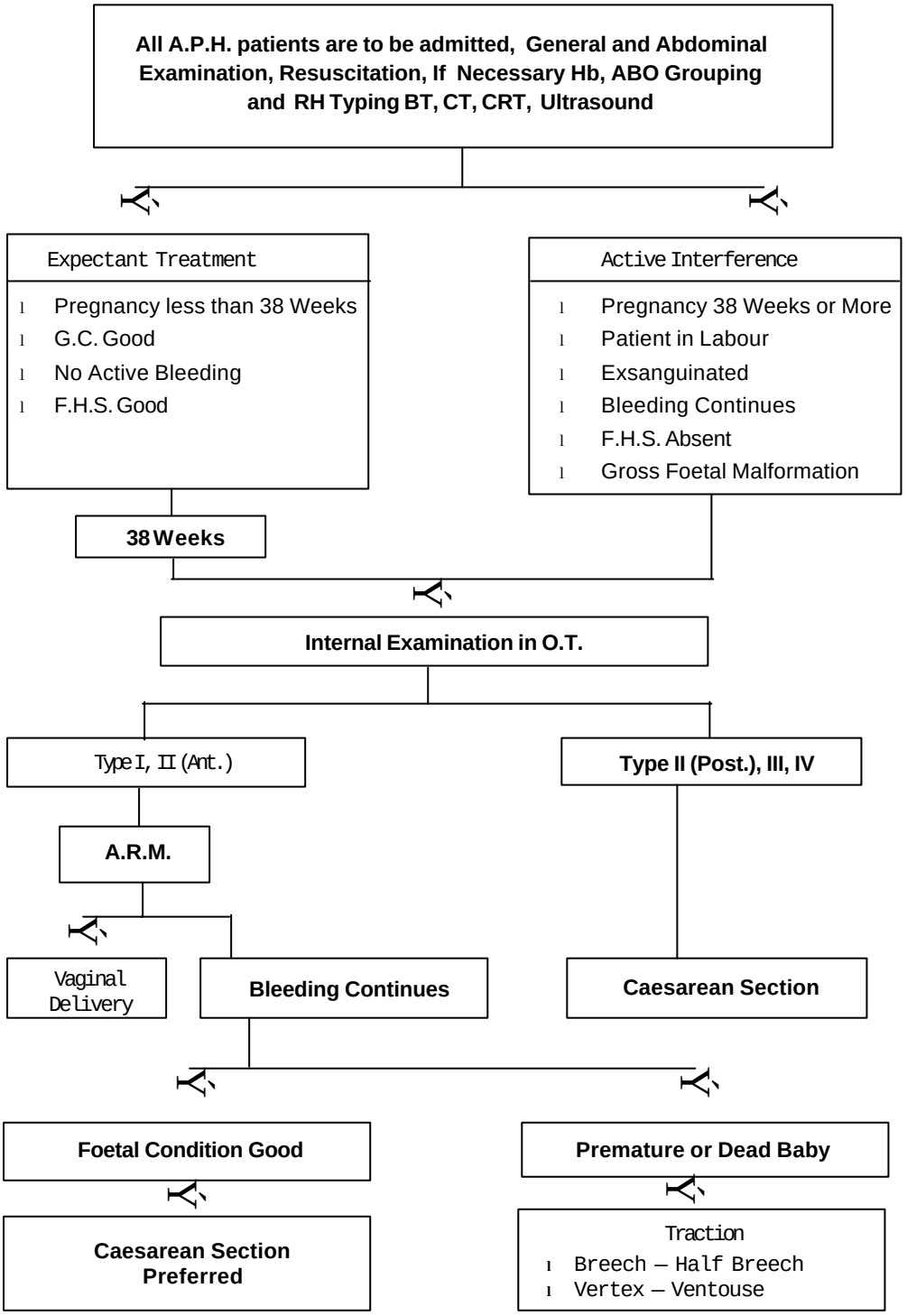

Fig 7.3: Scheme of management of placenta praevia

Foetal

The perinatal mortality inspite of the expectant management and available resources still remains high. In most of the centres it ranges from 10-20% .

Check Your Progress 3

1) List the diagnostic features of placenta praevia.

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2) In which cases of placenta praevia vaginal examination is not indicated even in the operation theatre?

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3) Give two basic principles of managemenf of placenta praevia.

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4) In which cases of placenta praevia expectant management should not be given?

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5) Why is post partum haemorrhage more likely to occur in cases of placenta praevia?

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7.3.2 Abruptio Placentae

Abruptio plecentae is also known as ablatio placenta and accidental-heamorrhage. Abruptio placentae is the premature separation of a normally situated placenta and accounts for 30% of cases of ante partum haemorrhage.It occurs in about 1 % of deliveries, the severe form occurring in about 1 in 500 deliveries.

a) **Aetiology**

The exact cause of separation of normally situated placenta is often difficult to ascertain. Following are some of the conditions which lead to abruptio placentae:

- External trauma—fall or blow on abdomen or external cephalic version may lead to placental separation.
- Acute decompression of polyhydramnios—sudden diminution of surface area of uterus where placenta is attached results in placental separation.
- Preterm rupture of membranes may lead to abruptio placentae. Hypertension has been found to be the most commonly associatecd condition with placental separation. In severe abruption pregnancy induced or chronic hypertension was found in 50% of the cases. In milder form of abruption the incidence of hypertension is not high.
- Other predisposing factors include advanced maternal age multiparity, uterine leomyoma, smoking and preyious history of abruption. Folic acid deficiency though implicated has not proved to be the cause of abruptio. In many cases, no cause is found.

b) **Varieties — Concealed and Revealed**

Two principal forms of the premature separation of placenta may be recognised depending on whether the haemorrhage is concealed or revealed (Fig. 7.4).

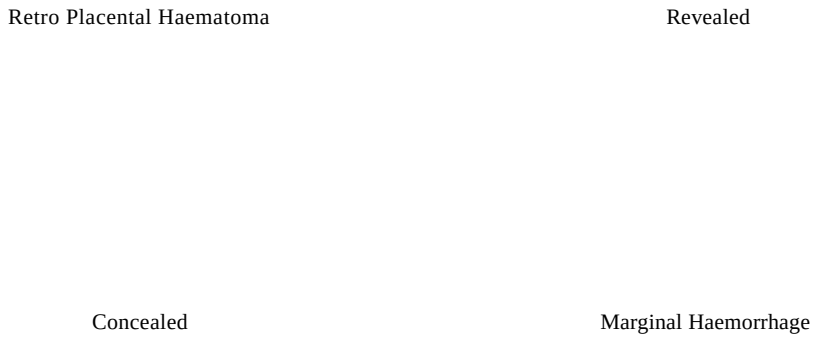

Fig 7.4: Varities of abruptio placentae

Concealed Form

This is a severe form of abruptio placentae seen in about 20% of the cases. Following the separation of the placenta, the blood does not escape out but is retained behind the placenta within the uterine cavity. Foetal death is common and coagulation disorders and other maternal complications are also more common.

Revealed Form

The blood from the separated placenta drains out through the cervix. The placental detachment is usually incomplete and complications are fewer and less severe. The revealed form of abruptio placentae is more common and is found in approx. 80% of the cases.

c) **Clinical Features**

Vaginal bleeding is the predominant feature and is found in about 80% of the cases. Uterine tenderness and rigidity is present in most of these cases. Back pain and uterine contractions may be observed in some cases. Foetal distress is found in about 50% of the cases and foetal death occurs in about 15% of the cases. In severe forms of concealed haemorrhage no vaginal bleeding may be observed but the uterus is found to be tender and hard and foetal heart is usually absent. There may be a clinically significant amount of disseminated intra vascular coagulation associated with depletion of fibrinogen as well as other clotting factors. The patient may develop a haemorrhagic diathesis leading to active bleeding from all the sites. Hypovolaemic shock and renal failure may develop.

Small separation of placenta may present as APH and may not produce any signs. It may be diagnosed only after delivery:

Ultra sound rarely helps in diagnosing abruptio placentae because many a times it may not reveal a retroplacental clot. Ultra sound may be helpful in excluding placenta praevia in milder forms of abruptio where clinical signs of tense and tender uterus are not present.

Clinical Classifications

Depending upon the degree of abruption and its clinical effects the cases are graded as follows:

Grade I

Clinical features suggestive of placental separation are absent, the diagnosis is made retrospectively. Retro placental clot volume is about 150 ml. Foetus is usually not at risk.

Grade II

In this grade antepartum haemorrhage is accompanied by classic features of abruptio placentae and the foetus is alive. Retro placental clot is about 500 mI. The uterus is tense and tender and foetal heart abnormalities are present.

Grade III

Along with the features of grade II there is foetal death. It has associated maternal shock, coagulation failure or renal failure.

d) **Pathology**

placental abruption is initiated by haemorrhage into the decidua basalis. As the decidual haematoma expands, it separates and compresses the placenta. The blood then separates the membranes and escapes giving rise to revealed type I abruptio placentae. In early stages there may not be any clinical features, only on inspection of the placenta after delivery a circumscribed depression with dark clotted blood is found. Recently abrupted placenta may however show no evidence of separation.

In concealed variety, the placental margins or the membranes remain adherent to the uterine wall and blood keeps collecting behind the placenta. Blood may enter the amniotic cavity after breaking through the membranes. In some cases the head which is closely applied to the cervix may prevent the blood to escape.

Couvelair Uterus (Utero Placental Apoplexy)

In the more severe form of placental abruption widespread extravasation of blood occurs into the uterine musculature and beneath the serosa giving rise to couvelair uterus which appears ecchymotic, purplish and hard and can be seen only on laparotomy. These myometrial haematomas do not interfere with uterine contractions and do not warrant hysterectomy.

e) **Management**

Management of abruptio placentae depends on the severity of the case and on the condition of the mother and the foetus.

Management of Severe Abruptio Placentae with Foetal Death

If the abruption of placeta is severe enough to cause foetal death the average intrapartum blood loss has been shown to be about 2500 ml These patients, therefore, require prompt and adequate transfusion of fluids and blood. Two intravenous lines should be established to allow rapid administration of fluids and blood. Haemotocrit and coagulation studies should be done. Indwelling urinary catheter should be put and vital signs monitored. Haematocrit should be maintained at 30% or more, it sustains the oxygen carrying capacity of the patient. Urinary output of atleast 30 ml per hour signifies effective intravascular volume and acute tubular or cortical necrosis, the most common cause of maternal mortality in abruptio placentae is avoided. Central venous pressure may be monitored by putting a catheter in internal jugular vein and this should be maintained at 10cm of water. This helps in infusing correct volume of fluids. Lung bases also must be auscultated for signs of overload along with other clinical signs.

Patients with foetal death should be delivered vaginally. Amniotomy should be done as soon as possible and oxytocin drip started. Careful monitoring of vital signs, coagulation profile and urinary output is done. Oxytocin drip and uterine massage is usually sufficient to prevent post partum haemorrhage.

Ceaserean section may be required if there is malpresentation, disproportion or some contraindication to vaginal delivery. Coagulation disorder, if present, must be treated before doing caesarean section.

In severe abruption acute disseminated intravascular coagulation may occur leading to fall in fibrinogen levels below 150 mg per dl. and drop in platelet count along with prolongation of partial thromboplastin time (P. T. T.) and prothrombin time (P. T.). The blood does not clot and all venepuncture start oozing blood.

Normal Values for D.I.C. Profile

Test	Normal Results
B.T.	1-3 min
C.T.	3-7 min
Fibrinogen	150 to 600 mg/dl
PT	11 to 16 seconds
PTT	22 to 37 seconds
Platelet count	120,000 to 350,000/cu.mm
FDP (Fibrin Degradation Products)	< 10 micrograrn/dl

Bedside Investigations

In absence of facilities for these tests a simple bed side clot observation test is invaluable in managing these cases. A venous blood sample is drawn and is placed in a clean dry test tube. It is observed for clot formation and clot lysis. Failure of clot formation within 5-10 minutes or dissolution of a firm clot when the tube is gently shaken at the end of an hour suggests clotting deficiency due to lack of fibrinogen and platelets. Bleeding time is also determined at bedside. The venepuncture site would be oozing if bleeding time is prolonged.

Management of Coagulopathy

Treatment of coagulopathy will depend on the amount of bleeding and anticipated route of delivery. Fresh whole blood is best for treating clotting deficiency and replacing blood loss. Cryoprecipitates can be given as they contain all necessary coagulation factors and are free of hepatitis B virus. 10 -20 units can be given. Fibrinogen deficiency usually corrects itself but if required fresh frozen plasma or fibrinogen can be transfused. Platelet transfusion in a bleeding patient is indicated if count is below 40,000 per cubic mm. and in patients with counts of 20,000 per cubic mm. even if there is no abnormal bleeding. Coagulation defect has to be treated if caesarean section or episiotomy carried out.

Management of Renal Failure

Acute renal failure is rare with lesser degrees of placental abruption but is seen in cases when there is delayed or incomplete treatment of hypovolaemia. The possibility of renal cortical or tubular necrosis must be considered if oliguria persists after an adequate volume has been restored. An attempt should be made to improve renal circulation and promote diuresis by increasing fluid volume (under close monitoring). If renal failure persists dialysis is indicated.

Management of Abruptio Placentae with Live Foetus

There are two sub-groups:

1) Those with Hypertonic Uterus

2) Those with Soft Uterus

If the foetus is alive and the uterus is rigid, the abruption is large but is less than 50%. . chances of foetal distress are high and therefore patient should be prepared for immediate caesarean section unless there is maternal shock or pre viable foetus. Blood coagulation studies are done and blood arranged before taking the patient for caesarean section. If the uterus is soft and abruptio is present, the pregnancy should be terminated by induction of labour with low rupture of membranes and oxytocin. If uterus becomes rigid and foetal distress develops caesarean section should be done. In very mild cases of abruptio placentae where foetus is pretenm, expectant treatment can be given under very close supervision. Ultrasound is done for placental localisation.

Remember that complications are more when abruption and delivery interval is more. Hence the aim is to terminate pregnancy and deliver the foetus without delay.

Management of abnormal placemtae is summarized in Fig. 7.5.

f) **Prognosis**

Maternal: Maternal mortality in abruptio placentae ranges from 0.5 to 5%. Haemorrhage D.I.C., cardiac and renal failure are responsible for high mortality. A high degree of suspicion, early diagnosis and definitive therapy results in lowered maternal mortality.

Foetal: Foetal mortality ranges from 50 to 80%. Live born infants have a high morbidity due to hypoxia, birth trauma and prematurity.

Indications for caesareans section in abruptio placenta are:

1) When foetus is live.
2) When maternal complication sets in they are corrected and caesarean done.
3) Inspite of adequate blood transfusions, bleeding continues and patient's BP continues to fall or does not come up.
4) Inspite of ARM and syntocnion drip, delivery does not take place in 12-16 hours.

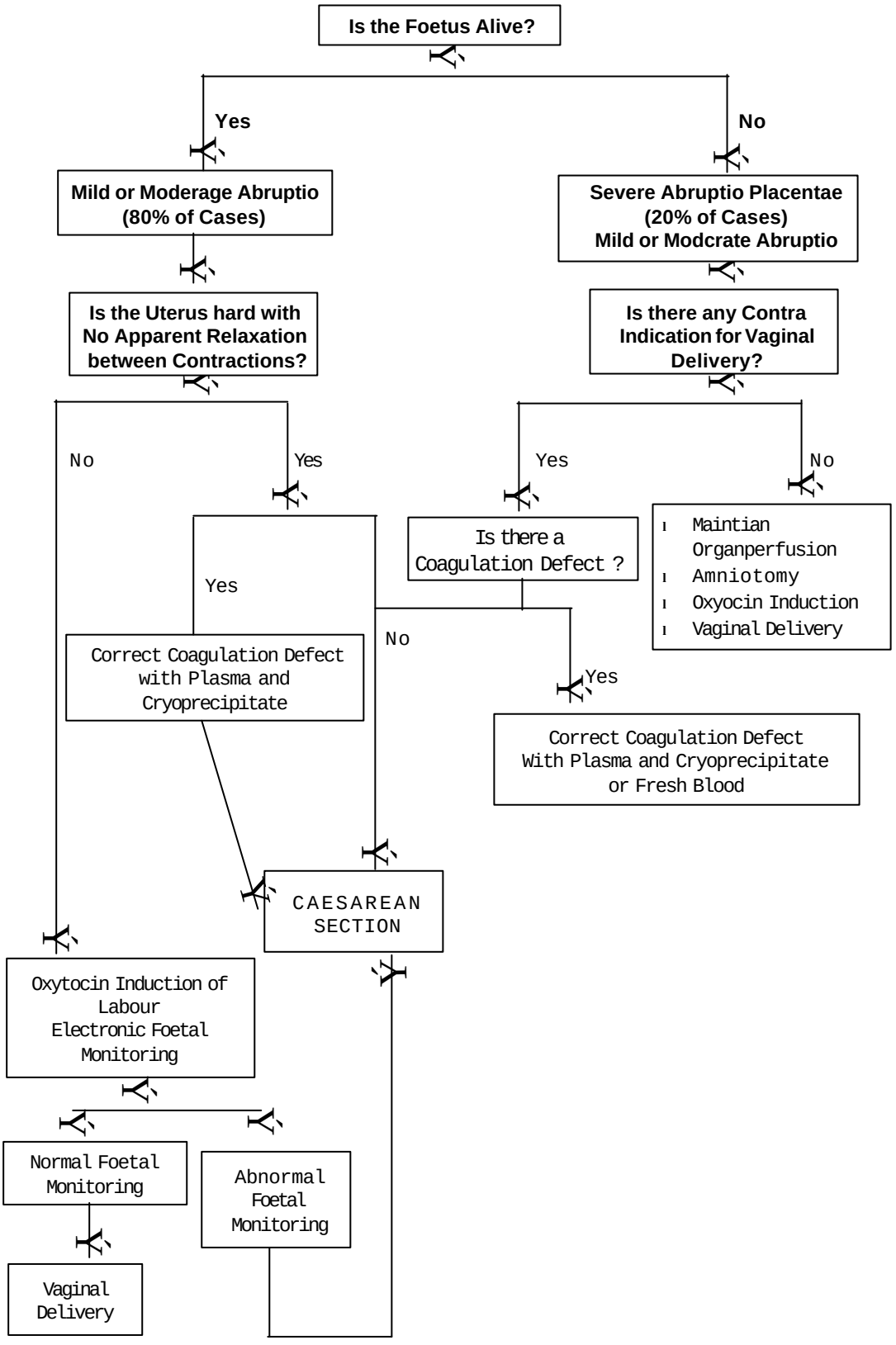

Fig. 7.5: Management of abruptio placentae

7.3.3 Other Causes of APH

Besides placenta praevia and abruptio placentae, antepartum haemorrhage is caused by incidental causes, marginal placenta and vasa praevia.

a) **Local Lesions of Cervix**

These include cervicitis, cervical erosion, polyps, varicose veins in vagina or cervix and cancer cervix. Per speculum examination will reveal these but they may coexist with other causes of antepartum haemorrhage which should, therefore, be excluded.

b) **Vasa Praevia**

This is a rare condition occurring in one in two thousand to three thousand deliveries. Vaginal blood tested for foetal haemoglobin in cases of APH showing foetal distress may reveal this condition. Caesarean section is indicated if foetal bleeding is detected.

c) **In Determinate Bleeding**

Many cases of ante partum haemorrhage have no evidence of placenta praevia or abruption and speculum examination is negative. Perinatal mortality is high in this group.

The cause of bleeding in these cases is not known. It can be marginal separation of placenta or excessive show. The bleeding is usually slight. The management of these patients is expectant in pregnancy less than 38 weeks. At 38 weeks, vaginal examination is done in operation theatre and if placenta is not felt, low rupture of membranes is done, oxytocin drip is given and close foetal monitoring is required.

Check Your Progress 4

1) What are the varieties of abruptio placentae?

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2) List the features of abruptio placentae which distinguish it from placenta praevia.

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3) What are the two basic principles in management of abruptio placentae?

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4) What is couvelair uterus?

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5) In which condition of abruptio placentae is vaginal delivery indicated'?

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7.4 LET US SUM UP

Reading this unit will make you realise how you could prevent the serious complication of eclampsia by diagnosing preeclampsia early and managing the patient properly.

Essential features of preeclampsia include hypertension, oedema and proteinuria. In eclampsia convulsions occur apart from these features. Early diagnosis of this condition can be made by good antenatal care.

Placenta praevia accounts for one-third cases of APH and occurs in about one in two hundred delivery. Bleeding in placenta praevia is painless, causeless and recurrent. General condition of the patient corresponds to the amount of blood loss, Uterus is soft, non tender,

presenting part may be high up, malpresentations are common. Diagnosis is confirmed by ultrasound or vaginal examination in the operation theatre prior to termination of pregnancy Expectant treatment is given if bleeding is slight or it stops, pregnancy is less than 38 week foetus is live and there are no congenital malformations. Termination of pregnancy is indicated if bleeding is excessive and continues, pregnancy is more than 38 weeks, foetus dead or malformed.

Vaginal delivery is allowed in placenta praevia type I and II anterior. Caesarean section is done in type II posterior, type III and type IV placenta praevia or if bleeding is excessive if there are associated indications for caesarean sections. Maternal complications are ante partum, intra partum and post partum haemorrhage, shock, retained placenta and puepereal sepsis. Perinatal mortality ranges from 10-25% and is due to prematurity, asphyxia, birth injury, haemorrhage and congenital malformations.

Abruptio placentae is the separation of normally situated placenta and occurs in about 1% of deliveries. Bleeding in abruptio placentae is associated with pain. Uterus is tense and tender, foetal parts are not easily made out and foetal heart may be absent. In concealed variety of abruptio placentae there is no external bleeding but patient may be in shock with all the other signs of abruptio placentae. Severe haemorrhage, shock, coagulation failure and renal failure may be present in severe cases. Prompt and adequate administration of fluid and blood, early delivery with careful monitoring of vital signs, urinary output and coagulation profile are life saving. Vaginal, delivery is preferred with careful foetal monitoring in mild cases with relaxed, uterus and in cases with dead foetus. Long uneffaced cervix is not an indication for doing caesarean section. In most cases of-abruptio placentae cervix dilates rapidly after amniotomy and oxytocin induction. Caesarean section is indicated where immediate delivery is not imminent, foetus is alive, uterus is hard and in cases with associated conditions requiring caesarean section. Coagulation disorder, if present, must be corrected before surgical procedure. Maternal mortality ranges from 0.5 to 5% and foetal mortality from 50 to 80%.

7.5 KEY WORDS

Amniotomy : Rupture of membranes

ARM : Artificial Rupture of Membranes

Succinturate lobe : Lobe of placenta away from the placental margin with communicating blood vessels.

Velamentous placenta : The cord is attached to the membrances and vessels traverse in the membranes before entering the placenta.

7.6 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) vasospasm.

2)
- Placentae
- Cardiovascular system
- Hematological system
- Endocrine system
- Kidney
- Liver
- Brain

3)
- Intravascular coagulation
- Thrombocytopenia
- Hemolysis
- Reduced antithrombin III

4) l Pregnancy induced hypertension
   l Pregnancy aggravated hypertension
   l Coincidental hypertension
5) l High blood pressure
   l Excessive weight gain
   l Oedema
   l Proteinuria
6) l Nifedipine
   l Hydralazine
   l Labetalol
7) l Methyldopa
   l Betablockers
8) delivery.

Check Your Progress 2

1) l Headache
   l Visual disturbances
   l Epigastric or right upper quadrant pain
2) l Premonitory stage
   l Tonic stage
   l Clonic stage
   l Stage of coma
3) l Patellar reflex is present
   l Respiration is not depressed
   l Urine output in previous 4 hours is 100 ml
4) l Aspiration pneumonia
   l Pulmonary oedema
   l Hemiplegia
   l Coma
   l Blindness.

Check Your Progress 3

1) l Bleeding is painless, causeless and recurrent.
   l Uterus is relaxed and non tender.
   l Foetal parts are easily palpable.
   l Presenting part is high up and malpresentation may be present.
   l On ultra sound, placenta is located in the lower uterine segement
2) l Patients in exsanginated state.
   l Diagnosed cases of major degrees of placenta praevia.
   l Associated complicating factors which are self indications for caesarean section-example-contracted pelvis.
3) l All patients must be hospitalised.
   l No vaginal examinations to be done till facilities of caesarean section and anaesthesia are available in O.T.

4) l In cases who are admitted with severe bleeding.
   l In cases where bleeding recurs or continues with expectant management.
   l In cases who reach 38 weeks of pregnancy with expectant managment.
   l Where on admission pregnancy is more than 38 week
   l In patients who are in labour
   l Where foetus is dead or congenitally malformed.

5) l Placental site is in lower Uterine segment which does efficiently as the upper segment.
   l Placenta is larger and thinner and may not separate completely
   l Placental site is larger.
   l There may be morbid adhesions of placenta.

Check Your Progress 4

1) l Concealed
   l Revealed

2) l Bleeding is associated with pain.
   l Uterus is tense and tender.
   l Foetal heart is usually absent.
   l On ultrasound placenta is not low.

3) l To correct hypovolaemia with fluids and blood transfusion.
   l To deliver the patient, preferably, by vaginal route.

4) In the more severe form of placental abruption widespread extravasation of blood occurs into the uterine musculature and beneath the serosa giving rise to couvelair uterus which appears ecchymotic, purplish and hard and can be seen only on laparotomy. These myometrial haematomas do not interfere with and do not warrant hysterectomy.

5) l Mild abruptio placentae where uterus is relaxed, but close maternal and foetal monitoring is required.
   l When placental separation is mild to extensive but the foetus is dead

7.7 FURTHER READINGS

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