



COURSE GUIDE

NSC 505: MATERNAL AND CHILD HEALTH NURSING III

COURSE CODE: NSC 505

COURSE TITLE: Maternal and Child Health III

COURSE UNITS: 5 Credit units

YEAR: 5

SEMESTER: 1st Semester

PRE-REQUISITE COURSES: All courses in the BNSC degree programme in the year 1 to 4

CON-CURRENT COURSES: NSC 501, NSC 503, NSC 509, NSC 511, NSC 513

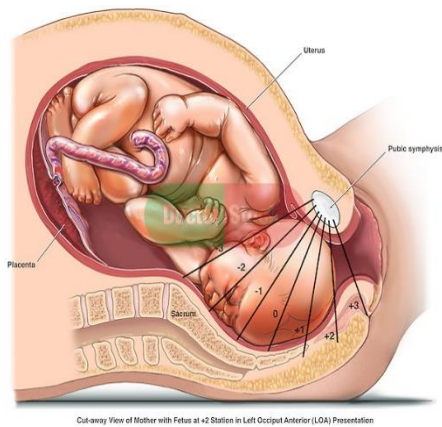
SESSION: 2019/2020 **COURSE WEBSITE:** www.noun.edu.ng

COURSE WRITERS

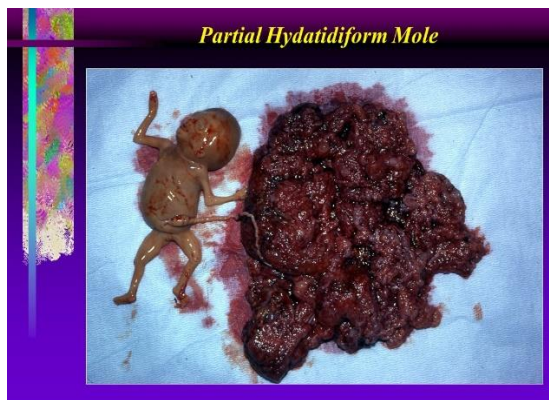
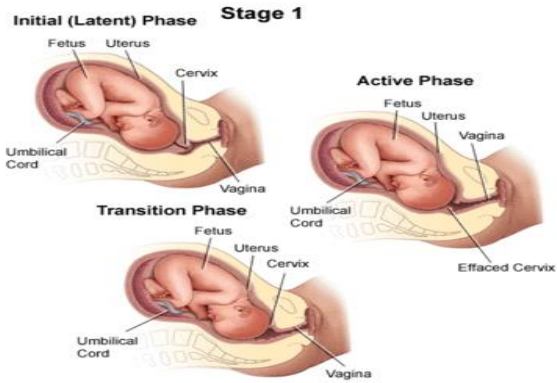
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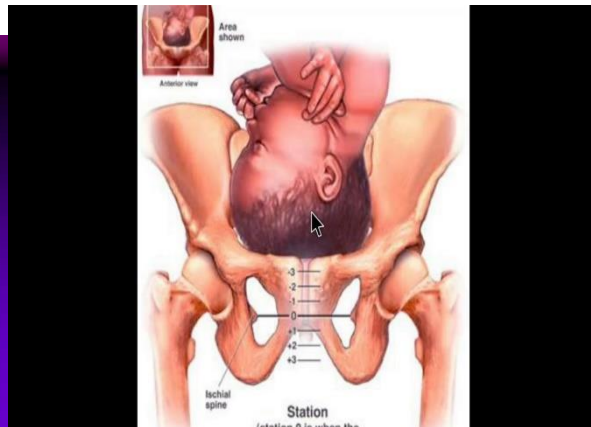
COURSE COORDINATOR: Dr E.M Joseph-Shehu



Cut-away View of Mother with Fetus at +2 Station in Left Occipital Anterior (LOA) Presentation



Partial Hydatidiform Mole



Source: <https://www.google.com/url?sa=i&source=images...>

General Introduction

Hello, we are happy to have you doing NSC505 – Maternal and Child Health Nursing III. You must have taken courses in Maternal Child Health Nursing in your basic School of Nursing/School of Midwifery. This course will expand your scope of knowledge and update you with current trends in the management of women with complications in pregnancy, labour, puerperium and postpartum.

Maternal and Child Health Nursing III is a five (5) credits unit course for the students in the Bachelor of Nursing Science degree programme. The course is made up of 5 modules with 12 study units. It is important that you register for the course at the beginning of the Semester. Doing the course will help improve your competence for evidence based midwifery practice, in caring for women and neonate.

This Course Guide will give you essential information about the course to help you plan to do well in the course. It is important that you read, master and utilize the information in the course guide.

Course Overview

Maternal and Child Health Nursing (MCHN) III is the last series of three courses in a nursing specialty, Maternal and Child Health Nursing and Midwifery Practice. This course focuses on abnormal midwifery. The course updates your knowledge on complications related to pregnancy, labour, puerperium, postpartum and neonatal life. It also gives overview on infertility and assisted reproduction. Wherever necessary, relevant examples in form of illustrations and illustrative diagrams are given for better understanding of the subject of discourse. Maternal and Child Health Nursing (MCHN) focuses on the delivery of professional quality health care that recognises, focuses on and helps in adaptation to the physical and psychosocial needs of the child-bearing woman, the family, and the newly born offspring. A significant observation about MCHN is that the care involves purposeful, sustained interactions between the nurse and her client(s).

Course Aim

The aim of this course is to update your knowledge and skills in management of common conditions related to abnormal midwifery.

Course Objectives

At the completion of this course, you will be able to:

At the completion of this course, you will be able to:

Apply the knowledge, skills and attitude acquired in this course in caring for women and neonates with complications associated with pregnancy, labour, puerperium, postpartum and neonatal life

Doing the Course

The course will be delivered adopting the blended learning mode, 70% of online but interactive sessions and 30% of face-to-face during laboratory sessions. You are expected

to register for this course online before you can have access to all the materials and have access to the class sessions online. You will have hard and soft copies of course materials, you will also have online interactive sessions, face-to-face sessions with instructors during practical sessions in the laboratory. The interactive online activities will be available to you on the course link on the Website of NOUN. There are activities and assignments online for every unit every week. It is important that you visit the course sites weekly and do all assignments to meet deadlines and to contribute to the issues that would be raised for everyone's contribution. You will be expected to read every module along with all assigned readings to prepare you to have meaningful contributions to all sessions and to complete all activities.

Course Requirements and Expectations

Course Mode – Blended

70% online class sessions; 30% practical of face-to-face working with preceptors. To participate in online sessions, you will need to register for the course as indicated by the School of Health Science Website.

NUMBER AND PLACES OF MEETING (ONLINE, FACETO-FACE, LABORATORY PRACTICALS)

The details of these will be provided to you at the time of commencement of this course

Discussion Forum

There will be an online discussion forum and topics for discussion will be available for your contributions. It is mandatory that you participate in every discussion every week. Your participation links you, your face, your ideas and views to that of every member of the class and earns you some mark.

Course Evaluation

This will be done through group review, written assessment of learning during clinical posting; teacher-learner joint review of clinical posting. Students evaluation: The students will be assessed and evaluated based on the following criteria.

In-Course Examination:

In line with the university's regulation, in-course examination will come up in the middle of the semester. These would come in form of three compulsory Tutor Marked Assignment (TMA's) and three (3). Group Assignments, projects and case studies will constitute 10% of the total mark for the course

Final Examination: The final written examination will come up at the end of the semester comprising essay and objective questions covering all the contents covered in the course. The final examination will amount to 60% of the total grade for the course.

Grading Criteria

The total of 100% for this course shall be made up as follows:

Continuous Assessment - 30%

End of Course Examination - 70% 100%

GRADING SCALE

A = 70-100

B = 60 - 69

C= 50 - 59

F = < 49

GRADE POLICY

A= 70% and above

B= 60-69

C= 50-59.

Equipment and Software Needed to Assess the Course

Students will be expected to have the following tools:

1. A computer (laptop or desktop or a Tablet)
2. Internet access, preferably broadband rather than dial-up access
3. MS Office software – Word PROCESSOR, Powerpoint, Spreadsheet
4. Browser – Preferably Internet Explorer, Moxilla Firefox, Goggle Chrome
5. Adobe Acrobat Reader 8

Sites of Practical

As would be specified at the time of registration for the course.



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Module 1 Complication associated with pregnancy

Complication associated with pregnancy are abnormal conditions in pregnancy that are significance in midwifery practice as they are the most common causes of maternal death. Such conditions are haemorrhage, embolism, hypertensive disease, and infection. Therefore, caring for women with any abnormal condition in pregnancy requires the nurse/midwife to seek for more knowledge to be able to render the appropriate care. This module deals with some causes of bleeding before and after 24th week of pregnancy such as ectopic pregnancy, abortion, antepartum haemorrhage, placenta praevia, placenta abruption and other conditions like pre-eclampsia, eclampsia, hyperemesis gravidarum and prolonged or post-term pregnancy

Module Objectives

At the end of this module you should be able to:

- i. Describe bleeding in pregnancy
- ii. Discuss other complications associated with pregnancy

Unit 1 Bleeding before 24th week pregnancy

CONTENTS

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
- 3.1 Ectopic pregnancy
- 3.2 Abortion
- 3.3 Gestational trophoblastic disease (GTD)
- 4.0 Conclusion
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1.0 INTRODUCTION

Any form of vaginal bleeding in early pregnancy is worrisome to the woman and her partner as it is abnormal until it proves otherwise. It became of great concerns when there is a history of previous pregnancy loss. Therefore, any bleeding in early pregnancy calls for investigation. Pregnant women must be informed to report without delay the slightest vaginal bleeding. The midwife in most cases usually the first point of contact and there is a need for her to render all the necessary advice and support to the woman and her family accordingly. The midwife should be aware of the local policies pertaining to her employment and how to guide the woman. In all cases of bleeding in pregnancy, history should be obtained to establish the amount and colour of the bleeding, when it occurred and whether there was any associated pain. The causes of vaginal bleeding in early pregnancy are implantation bleeding, cervical ectropion/erosion, cervical cancer, cervical polyps, ectopic pregnancy, abortion etc. Some of these causes of vaginal bleeding in early pregnancy can occasionally lead to life-threatening situations and others of less consequence for the continuance of pregnancy. In this unit, we will discuss ectopic pregnancy and abortion

2.0 OBJECTIVES

At the end of this unit you should be able to:

- Describe common causes of bleeding before the 24th week of pregnancy
- Describe ectopic pregnancy and its management
- Explain the different types of abortion
- Describe the management of different types of abortion

3.0 MAIN CONTENT

3.1 Bleeding before 24th week of pregnancy

Bleeding before 24th week of pregnancy is often referred to as bleeding in early pregnancy. They are conditions that caused vaginal bleeding in a pregnant woman before the 24th week of pregnancy. The incidence is high in pregnancy due to increased vascularity of the genital organs

3.1.1 Causes of bleeding before 24th week of pregnancy

There are two groups namely: extra gestational or incidental cause of bleeding and gestational causes

The causes of extra gestational or incidental are:

Cervical erosion: this produce slight blood-stained discharge often mixed with mucus. This seldom requires treatment during pregnancy

Cervical polyps: A small vascular pedunculated growths on the cervix. It consists of squamous or columnar epithelia cell over a core of connective tissue rich with blood supply. If its surface becomes ulcerated, it bleeds freely on touch or after intercourse.

Carcinoma of the cervix: this is a serious condition but it very rare in pregnancy. The incident is high among multiparous patients. The discharge may be mucous, blood-stained and foul-smelling. A history of metrorrhagia prior to pregnancy should arouse suspicion and calls for further investigation. Prompt and adequate care is necessary because pregnancy accelerates the growth of malignant cells.

Any trauma or laceration in the genital tract will also cause bleeding

The gestational causes are:

- Rupture ectopic pregnancy
- Abortion
- Hydatidiform mole
- Implantation or decidual bleeding

3.2 Ectopic pregnancy

3.2.1 Introduction

An ectopic pregnancy is a gestation that implants outside the uterine cavity, often within the fallopian tube. However, implantation can also occur within the abdominal cavity (for instance on the large intestine or in the Pouch of Douglas), the ovary or in the cervical

canal. Despite recent advances in earlier detection, it continues to represent a serious hazard to women's health and their future reproductive potential. Within Vitro fertilization (IVF) and other assisted reproductive technologies (ARTs), the risk for ectopic pregnancy increases substantially, and the location of those ectopic implantation's changes (Figure 1-1).

3.2.2 Incidence of ectopic pregnancy

Despite the mortality rates for ectopic pregnancy have dropped due to early diagnosis, this condition still causes 4% to 6% of maternal deaths in the United States and is the most common cause of maternal mortality in the first trimester. In general, ectopic pregnancy is estimated to occur in 1 of every 80 spontaneously conceived pregnancies. More than 95% of ectopic pregnancies implant in various anatomic segments of the fallopian tube, including the ampullary (75% to 80%), isthmic (12%), infundibular and fimbrial (6% to 11%), and interstitial (2%). Other, less common sites of ectopic implantation are the ovary, uterine cervix, and a rudimentary uterine horn. Importantly, the risk for heterotropic implantations (one intrauterine and one ectopic) may rise to 1 in 100 with IVF. In the past two decades, there was a significant increase in diagnosed ectopic pregnancy rates because of the following:

1. Improved technology, which has allowed for earlier and more complete recognition of early ectopic pregnancies, including ectopic pregnancies that would previously have gone undetected
2. Rising incidence of acute and chronic salpingitis, especially related to Chlamydia trachomatis
3. Increasing number of tubal surgeries, such as tubal ligation and tubal reconstruction
4. Increase in ARTs, especially IVF

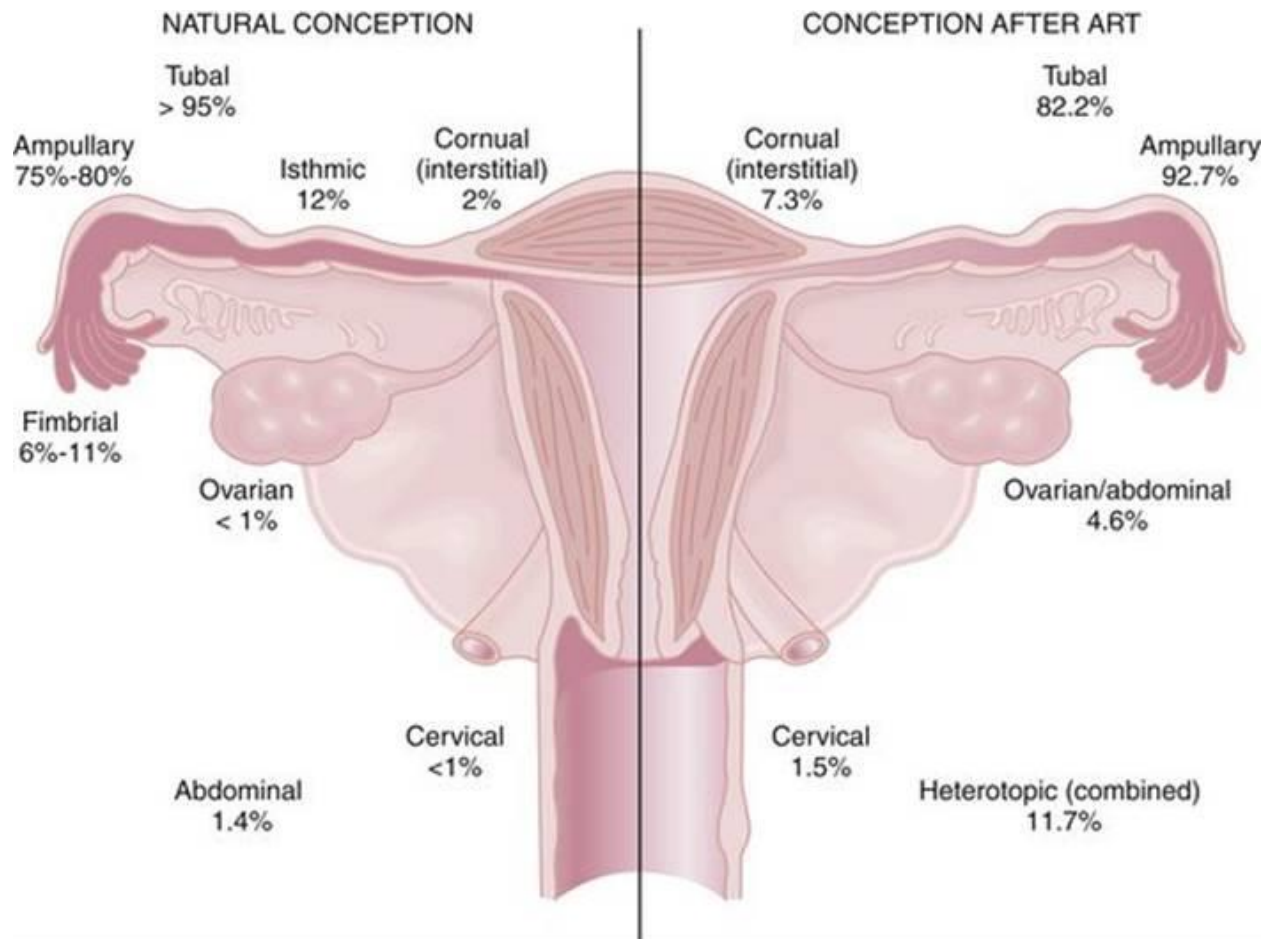


Figure 1-1: Possible locations of ectopic pregnancy with spontaneous conception vs pregnancies that result from assisted reproductive technologies (ART) such as in-vitro fertilization (IVF). Source: *Source: Ectopic Pregnancy – Doctorlib.info*

Etiology of ectopic pregnancy

The etiology of ectopic pregnancy is not always clear.

Risk factors for ectopic pregnancy

- History of tubal infection
- Cigarette smoking (increased relative risk, 1.26)
- History of tubal sterilization within the past 1 to 2 years (higher incidence if cauterization was used)
- History of tubal reconstructive surgery
- contraceptive pill use
- Pregnancy with current intrauterine device

- Infertility due to tubal factors
- Use of assisted reproductive technology
- Previous pelvic surgery
- Hormonal factors e.g. depot medroxyprogesterone acetate, or emergency contraceptive pill use
- Previous ectopic pregnancy
- Previous spontaneous abortion
- Alterations in tubal physiology
- Assisted reproductive technology
- Uterine curettage

3.2.3 Pathophysiology of ectopic pregnancy

Any abnormality in tubal morphology or function may lead to ectopic pregnancy. In normal pregnancy, the egg is fertilized in the fallopian tube and the embryo is transported into the uterus. It is believed that the most important cause of ectopic pregnancy is damage to the tubal mucosa, which could obstruct embryo transport due to scarring. The other possibility is that a small defect in the mucosa attracts implantation in the fallopian tube. The mucosal damage may be caused by infection or surgical trauma. However, evidence of tubal damage is lacking in many cases of ectopic pregnancy. In these women, the cause of ectopic pregnancy may be dysfunction of tubal smooth muscle activity. In general, oestrogens stimulate tubal myoelectrical activity while progesterone has an inhibitory effect. An altered oestrogen/progesterone ratio may affect tubal motility in different ways. Abnormally high oestrogen levels may cause tubal spasm, which could block transport of the embryo towards the uterine cavity. This may be an explanation for increased rates of ectopic following ovarian hyperstimulation and post-coital oral contraception. Conversely, pharmacological doses of progesterone in women using progesterone-only contraception could cause complete tubal relaxation leading to retention of the fertilized egg within the tube. Embryonic abnormalities have also been studied in an attempt to explain the occurrence of ectopic in the absence of tubal

pathology and although the majority of tubal pregnancies are non - viable, the incidence of chromosomal defects is no higher than in samples obtained from intrauterine pregnancies.

3.2.4 Clinical manifestations

- History of amenorrhoea of about 6-8weeks duration
- Brown vaginal discharge, which starts soon after the missed menstrual period. Although, about 10 – 20% of ectopic pregnancies present without bleeding
- Abdominal pain - Some women may complain of period-like pain or upper abdominal discomfort. The pain is usually caused by tubal miscarriage and bleeding through the fimbrial end of the tube into the peritoneal cavity.
- Severe rupture sometimes presents with nausea, vomiting and diarrhoea, which may resemble a gastrointestinal disorder.
- Transient feeling of faintness or dizziness
- Typical signs of haemorrhagic shock, such as pallor, tachycardia, hypotension, skin is cold and clammy and oliguria.

3.2.5 Diagnosis

Ultrasound scan

Serum human chorionic gonadotrophin (hCG) - Abnormally slow rise in serum hCG has also been used to diagnose ectopic pregnancy. In normal early pregnancy, the hCG doubling time is 1- 4 days before 5 weeks' gestation and 2.4 days from then until 7 weeks' gestation.

3.2.6 Management of ectopic pregnancy

Ectopic pregnancy may be managed surgically, medically or expectantly. All cases of suspected or diagnosed ectopic pregnancy should be referred to the hospital. In the clinic, the patient is made to lie down, treated for shock and transfer to the hospital as quickly as

possible. Management is tailored to individual patients, based on their presentation and on the severity of their condition, suitability of treatment options and patient preference. Figure 2 demonstrates a suggested diagnosis and management pathway.

Medical management

Medical treatment is useful for patients with an unruptured tubal ectopic pregnancy who are haemodynamically stable and have minimal symptoms and a low volume of free intraperitoneal fluid on ultrasound scan.

Drug of choice – methotrexate, it is very successful for small stable ectopic pregnancies.

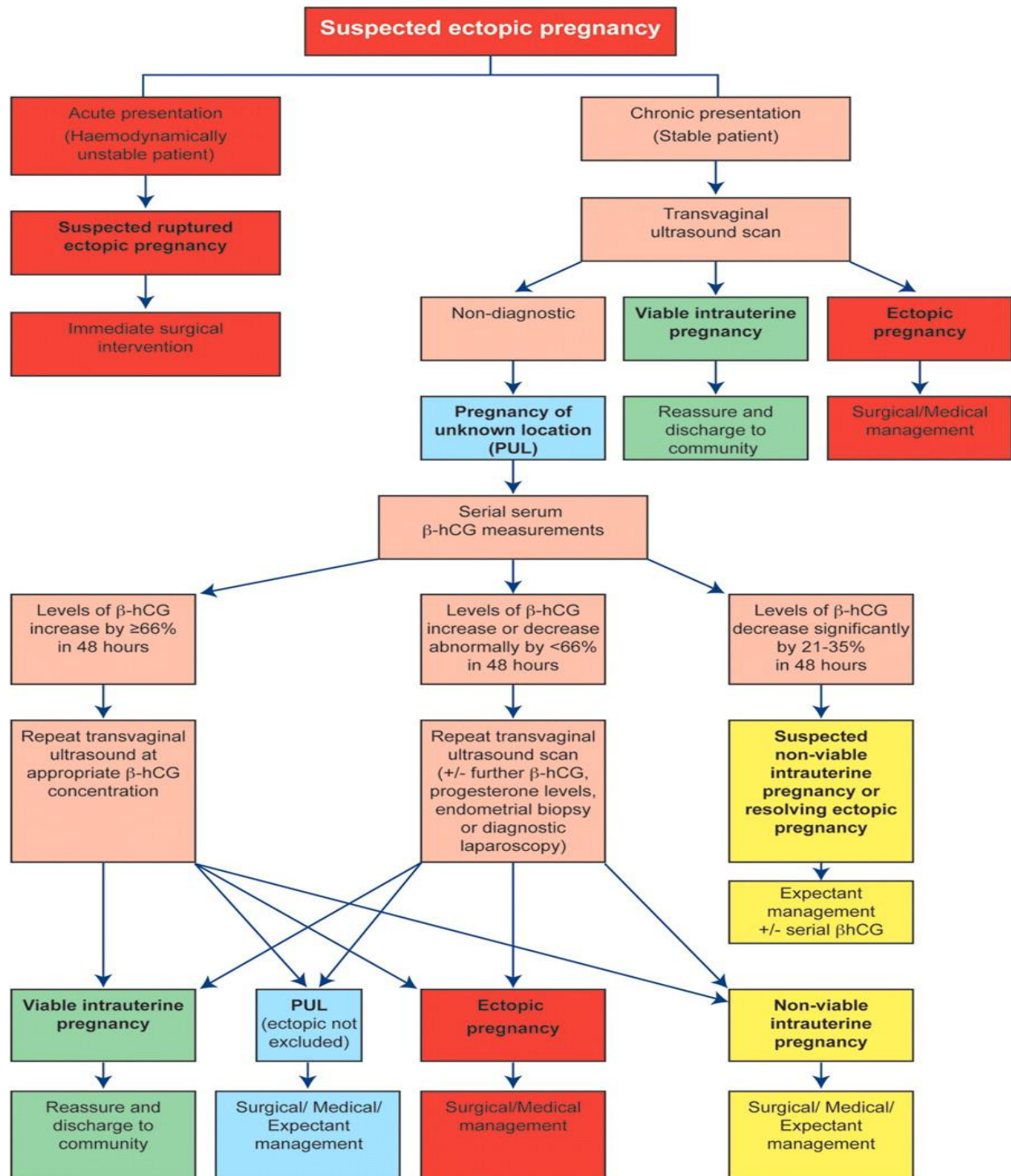


Figure 1-2: Recommended diagnostic and management approach for suspected ectopic pregnancy. It is important to highlight that the figure of 66% is used as a practical guide only and that all cases of pregnancy of unknown location should be considered as a potential ectopic pregnancy until assessment proves otherwise or management is

complete. β -hCG, beta-human chorionic gonadotrophin. Source: Diagnosis and management of ectopic pregnancy. BMJ Sexual & Reproductive health; 37(4)

Dosage - A dose of 50 mg/m of the calculated body surface area (calculated using height and weight measurements) is administered in a single-dose protocol.

Approximately 14–20% of patients receiving single-dose treatment will require a repeat dose. usually decided on following a fall of the β -hCG concentration of less than 15% from Day 4 to 7 after treatment.

Route of administration - Intramuscular

Pharmacological action - Methotrexate is a folic acid antagonist that targets rapidly dividing cells and arrests mitosis. It prevents the proliferation of cytotrophoblast cells, reducing cell viability and β -hCG secretion and thus progesterone support for the pregnancy. This facilitates the resolution of the ectopic pregnancy and tissue remodelling.

Side-effect

abdominal discomfort 1–3 days after treatment and abdominal bloating;

significant hepatotoxicity;

bone marrow toxicity;

alopecia is extremely rare with ectopic pregnancy treatment regimens.

Inclusion criteria for medical management of ectopic pregnancy with methotrexate

Patient characteristics

- Would prefer medical option
- Willing to attend follow-up for up to 6 weeks
- Willing to abstain from alcohol for 7 days following the treatment
- Not breastfeeding or willing to stop

Clinical features

- Haemodynamically stable
- Minimal abdominal pain

Ultrasound scan findings

- No fetal heart activity or clear yolk sac in adnexal mass
- Small amount of free fluid
- Unlikely to be early intrauterine pregnancy failure

Serum beta-human chorionic gonadotrophin (β -hCG) concentrations

- Usually <3000 IU/l (Although limits of <5000 IU/l are used in some units and earlier studies; treatment success rates are higher when this more commonly used lower limit applies.)

Medical history

- No active peptic ulcer disease
- No severe medical conditions including renal disease, hepatic disease, severe anaemia, leucopenia or thrombocytopenia

Should not be on concurrent medication

- Non-steroidal anti-inflammatory agents (NSAIDs), aspirin, penicillin, sulphonamides, trimethoprim, tetracyclines, diuretics, phenytoin, antimalarials, ciclosporin, retinoids, probenecid, folic acid, hypoglycaemics, live vaccines, nephrotoxic or hepatotoxic drugs

Special consideration

- Obtain a baseline full blood count, renal and liver function tests for the patient

- Patients require careful monitoring to ensure complete resolution of the ectopic gestation using serial assessment of β -hCG levels every 4–7 days (protocols vary between units) until the β -hCG level is <5 IU/l.
- Both staff and patient should be aware of potential treatment failure. Hence, observe a close treatment surveillance as rupture still remains a possibility during treatment.
- Failure of single-dose medical management is associated with initial serum β -hCG concentrations >5000 IU/l, a moderate or large amount of free fluid on ultrasound, the presence of fetal cardiac activity and a pretreatment increase in serum β -hCG of $>50\%$ over a 48-hour period.

Expectant management

This is reported to be most useful when the initial β -hCG is <1000 IU/l. Some ectopic pregnancies resolve spontaneously through either regression or tubal abortion, without causing harm to the patient. Expectant management is a conservative strategy consisting of observation and assessment of whether the ectopic pregnancy is continuing to resolve spontaneously and successfully without intervention. A suitable candidate for expectant management must have an ectopic pregnancy with no evidence of rupture, be clinically stable and asymptomatic, and have consistently declining β -hCG concentrations. A low serum progesterone is also a possible marker of suitability for the expectant approach. Follow-up should be between one and three times weekly with β -hCG measurement and ultrasonography as required. A rapidly declining β -hCG level also appears to predict a favourable outcome. Success rates between 47% and 82% are reported, depending on the patient's initial status. The importance of compliance with follow-up and ease of access to the hospital should be emphasised. If β -hCG levels remain static or decline sub-optimally, consideration should be given to reverting to surgical or medical management.

Surgical management

If the ectopic pregnancy is causing heavy bleeding, emergency surgery might be required to save the woman. A surgery is performed through an abdominal incision (laparotomy). In some cases, the fallopian tube can be repaired or removed (salpingectomy) depends on the state of the ruptured tube.

- Preparation is made for intravenous infusion and blood collection for the estimation of haemoglobin and packed cell volume, as well as grouping and cross-matching. Administer prescribed analgesic e.g. morphine (15mg)
- Administer blood transfusion as soon as it's available
- Prepare the patient for salpingectomy of the affected side
- The postoperative medical and nursing care of the patient is important and is the same as after any major obstetric or gynecological operation

3.2.7 Outcome of tubal pregnancy

- Tubal mole
- Tubal abortion – more common in ampulla
- Tubal rupture – more common in isthmus

3.3 Abortion

Abortion can be defined as termination of pregnancy prior to 24 weeks of gestation. According to World Health Organisation, it is bleeding or expulsion of the fetus before 20th week of gestation or a fetus born weighing less than 500g. It is the most common cause of bleeding in early pregnancy. It can spontaneous or induced.

3.3.1 Spontaneous abortion

The term miscarriage is used to describe a spontaneous pregnancy loss in preference to the term of abortion which is associated with the deliberate ending of a pregnancy. A miscarriage is seen as the loss of the products of conception prior to the completion of 24 weeks of gestation, with an early pregnancy loss being one that occurs before the 12th completed week of pregnancy.

Incidence

An incidence of recognized spontaneous abortion of 15% to 25% is commonly cited, with approximately 80% occurring during the first 12 weeks (First - trimester miscarriage) of pregnancy which accounts for the majority. The overall rate is 20%.

Second - trimester miscarriages are less common, accounting for 1 – 4% of all miscarriages. While some second - trimester miscarriages can be explained as first - trimester losses where the diagnosis is made in the second trimester, nevertheless it seems likely that the causes are different.

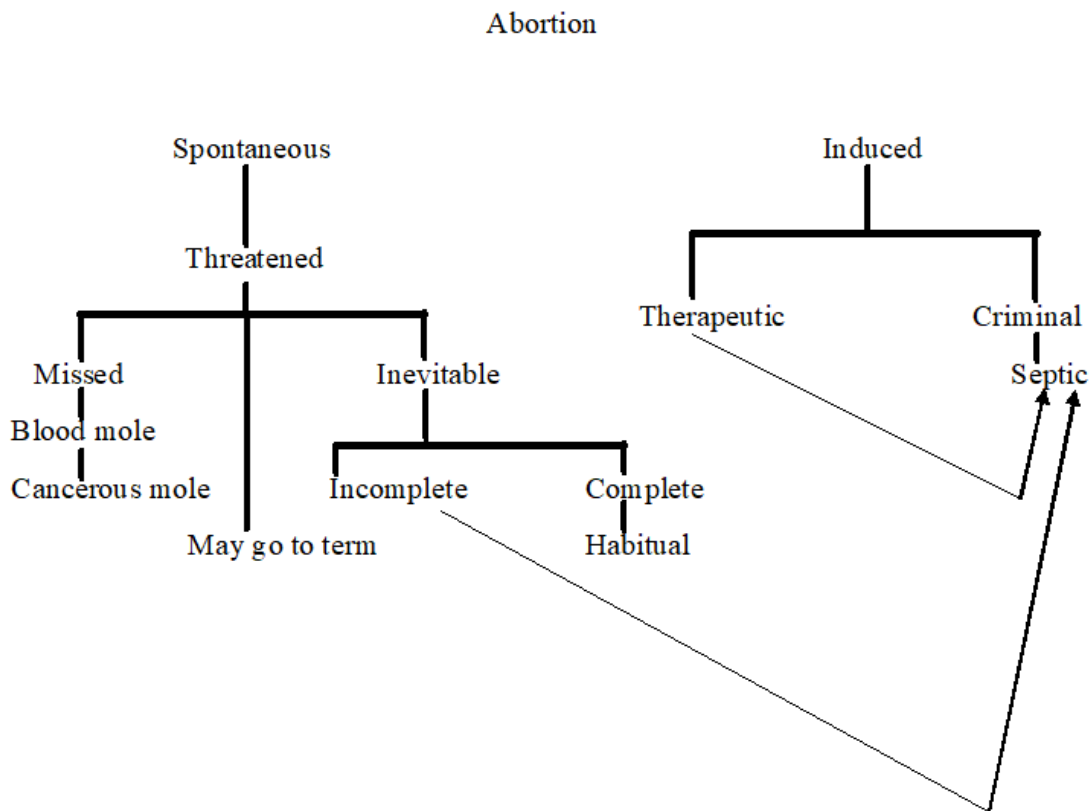


Figure 1-3: Classification of abortion adapted from Ojo & Briggs (1992) textbook for midwives in the tropics; 2nd edition

Etiology

Chromosomal abnormalities: Approximately 50-70% of early spontaneous abortions are attributed to chromosomal abnormalities, most of which are trisomy. Compared with first-trimester abortions, second trimester abortions are less likely to be caused by chromosomal abnormalities and more likely to be caused by maternal systemic disease, abnormal placentation, or other anatomic considerations. Also, it is likely that abnormal implantation has a role to play in some cases of spontaneous abortion and this is an area of current research. The following chromosomal abnormalities associated with miscarriage are Trisomies: 68%, mainly trisomy 16, 21 and 22. Triploidy: 17.1%.

Monosomy: 9.8% (XO, Turner's syndrome).

Infectious Factors

Infections are an uncommon cause of early spontaneous abortion. Chlamydia trachomatis and Listeria monocytogenes, varicella, rubella and other viral illnesses have been associated with spontaneous abortion. Serological evidence supports a role for Mycoplasma hominis and Urea plasma urea lyticum in abortions. Finally, abortion is independently associated with serological evidence of syphilis, human immunodeficiency virus (HIV)-1 infection, and with vaginal colonization with group B streptococci.

Endocrine factors

Thyroid autoantibodies are associated with an increased incidence of spontaneous abortion, even in the absence of clinical hypothyroidism. In women with type 1 diabetes, the degree of metabolic control in early pregnancy is associated with an increased risk of spontaneous abortion and major congenital malformation.

Environmental factors

The abortion risk increases in a linear fashion with the number of cigarettes smoked per day. Both spontaneous abortion and fetal anomalies may result from frequent, high doses of alcohol use during the first 8 weeks of pregnancy. Radiation administered at

therapeutic doses to treat cancer may be an abortifacient. It is important to note that exposure to less than 5 rads does not increase the risk for miscarriage.

Immunologic factors

There are a number of genetic disorders of blood coagulation that may increase the risk of both arterial and venous thrombosis. Some of the better-studied thrombophilias are caused by mutations of the gene for factor V Leiden, prothrombin G20210A mutation, antithrombin III, proteins C and S, and methylene tetrahydrofolate reductase (hyperhomocysteinemia). These are most commonly associated with recurrent miscarriage.

Uterine Factors

Large and multiple uterine leiomyomas are common, and they may cause miscarriage. In most instances, their location is more important than their size, with submucous leiomyomata playing a more significant role than others, presumably because of their effect on implantation. In utero exposure to diethylstilbestrol (DES) has been associated with abnormally shaped uteri as well as cervical incompetence and spontaneous abortion. Intrauterine synechiae (Asherman syndrome), a condition that is caused by uterine curettage with subsequent destruction and scarring of the endometrium, may also be a cause of spontaneous abortion

Classification and Differential Diagnosis of Spontaneous Abortions

Because the differential diagnosis of bleeding in the first trimester of pregnancy includes a wide range of possibilities, such as ectopic pregnancy, hydatidiform mole, cervical polyps, cervicitis, and neoplasm, the patient should be examined whenever there is bleeding in early pregnancy. Laboratory investigations and ultrasonography examination such as serial quantitative serum β -hCG, progesterone levels and transvaginal sonography are used to ascertain if there is an intrauterine live fetus.

Management of spontaneous abortion

Management of spontaneous abortion is individualized based on sonographic report of the patient. The management options are either any of these three - expectant, medical, or surgical management except there is serious bleeding or infection with an incomplete abortion. However, each of the option has its own risks and benefits for example, the first two are associated with unpredictable bleeding, and some women will undergo unscheduled curettage. Also, the success of any method depends on whether the woman has an incomplete or missed abortion.

Risk and benefits of the three options of spontaneous abortion treatment

Expectant management: this is when the products of conception are to be passed spontaneously and, in many cases, it is the appropriate management. However, women should be aware that this can take several weeks. Women adopting this option should be given full information regarding the probable sequence of events and be provided with contact details for further advice, with the option of admission to hospital if required. It is important that women are educated to actively observe for signs of infection and know what to do if they suspect this. Incomplete abortion has failure rates as high as 50 percent with this treatment option

Medical management: Medical management of miscarriages includes a variety of regimes involving the use of prostaglandins, such as misoprostol, and may include the use of an antiprogesterone such as mifepristone for a missed miscarriage, or progesterone alone for an incomplete miscarriage. It has varying failure rates of 13 to 96 percent, depending on the gestation and size of the gestational sac. However, complications include abdominal pain and bleeding, overall the medical management of miscarriage reduces both the number of hospital admissions and the time women spend in hospital.

Surgical management: The surgical method where the uterine cavity is evacuated of the retained products of conception (ERPC) prior to 14 weeks' gestation is suitable for women who do not want to be managed expectantly and who are not suitable for medical management. Under either a general or local anaesthetic the cervix is dilated, and a

suction curettage is used to empty the uterus. The use of prostaglandins prior to surgery makes the cervix easier to dilate, thus reducing the risk of cervical damage. With the surgical management, there is a quick resolution and is 95 to 100 percent successful. It is invasive and not necessary for all women. The main complications are perforation of the uterus, tears to the cervix and haemorrhage.

Clinical classification of spontaneous abortion

- Threatened Abortion
- Inevitable Abortion
- Incomplete Abortion
- Complete Abortion
- Missed Abortion

3.3.2 Threatened abortion

Vaginal bleeding during the first 20 weeks of pregnancy, whether the bleeding is associated with uterine contraction or not. It can be distinguished from implantation bleeding which is usually bright red colour and stops quickly.

Signs and Symptoms

- Slight bleeding
- Os is closed and not effacement
- Slight uterine contraction
- Slight abdominal discomfort
- There may be low-midline clearly rhythmic cramps
- persistent low backache with pelvic pressure or dull and mid-line suprapubic discomfort
- On speculum examinations, cervix is closed and membranes intact

Treatment

1. Admission in the hospital
2. Reassure client

3. Assess general condition – history, vital signs etc.
4. Routine Observation bid or 4hrly
5. No Vaginal Examination and enema
6. Save all discharges – Pads, soiled clothing, linens etc.

Blood Test: Grouping and Cross matching, Hb, Rh factor, Human placenta lactogen level – helps to determine prognosis as low level indicate that pregnancy will terminate (inevitable abortion)

Drugs

- Valium 5mg TDS
- Amylobarbitone sodium (sodium Amytal) 200mg nocte Pethidine
- 50-100mg to relief pain of uterine contractions,
- Morphine 15mg.
- Speculum examination to rule out bleeding from local lesion.
- Monitor fetal condition – FH by sonicaid/Dipltone
- Do pregnancy test.
- Allow up and about after bleeding has stopped for 3 days
- Nutritious diet and personal hygiene

Prognosis: 70-80% - continue with pregnancy. Prognosis is better if bleeding becomes brownish from bright red-only about 10% will result in abortion, while initial brown blood becomes red 66% will abort. If accompanied with severe uterine contraction there is increased possibility of abortion.

Advice on Discharge

Rest, less activities, no lifting, or coitus for 2-3 weeks, she should report any case of bleeding.

3.3.3 Inevitable abortion

Inevitable abortion is when bleeding is accompanied with uterine contractions, gross rupture of the membranes along with cervical dilatation. It is impossible for the pregnancy to continue. It may end up complete or incomplete.

Signs and Symptoms

- Slight or severe vaginal bleeding
- Increase contraction of the Uterus – Pain
- Dilatation of the cervix
- Membranes may or may not be ruptured, it may bulge through the cervix or in the vagina
- Shock may be present
- Product may protrude through the cervical Os or in the vagina

Treatment

- Treat as threatened abortion until doctor's arrival.
- If bleeding is severe, give 0.5mg ergometrine or 1ml syntometrine, keep all blood loss for inspection.
- Give analgesics – Pethidine 100mg or Morphine 15mg.
- Oxytocin drip is given or prostaglandin E2 if it is after 16 weeks.

3.3.4 Complete Abortion

When the entire products of conception are passed, abortion is considered complete and is usually accompany regression of all the signs and symptoms of pregnancy. There is usually a history of heavy bleeding, cramping, and passage of tissue or a fetus. During examination, the cervical os is closed. Patients are encouraged to bring in passed tissue, which may be a complete gestation, blood clots, or a decidual cast. If an expelled complete gestational sac is not identified, sonography is performed to differentiate a complete abortion from threatened abortion or ectopic pregnancy.

3.3.5 Incomplete Abortion

Bleeding that follows partial or complete placental separation and dilation of the cervical os is termed incomplete abortion. The fetus and the placenta may remain entirely within

the uterus or partially extrude through the dilated os. The entire product of conception frequently expelled prior to 10th week but after then they are delivered separately.

Signs and symptoms

- Bleeding may be profuse
- Pain may or may not be present
- Os is partly closed –cervix patulous,
- There is sub-involution.

Treatment

Management options of incomplete abortion include curettage, medical abortion, or expectant management in clinically stable women. With surgical therapy, additional cervical dilatation may be necessary before suction curettage. In some cases where retained placental tissue simply lies loosely within the cervical canal and can be easily extracted with ring forceps. Whatever treatment option closely observe the patient vital signs. Observe for shock and resuscitate if necessary, treat for anaemia if present and administer antibiotic coverage.

3.3.6 Missed Abortion

This term is applied when the fetus is dead and is retained with its placenta in the uterus. Death usually occurs before 8 weeks though the mother may not know. Ultrasound may diagnose it even before the woman notices it.

Treatment

1. Some obstetrician will prefer to leave it as spontaneous expulsion will take place (expectant management): this may cause anxiety and distress to the mother.
2. Administer prostaglandins, such as misoprostol, and an antiprogestone such as mifepristone (Medical management)
3. Manual Vacuum aspiration of the content may be performed
4. Blood coagulation disorder may develop if up to 6-8 weeks
5. Plasma fibrinogen estimate weekly
6. If several weeks have elapsed between death and expulsion of the conceptus give fresh compatible blood.

3.3.7 Septic Abortion

Septic abortion is a common complication of either complete or incomplete abortion due to ascending infection. Patients may present with sepsis, shock, haemorrhage, and possibly renal failure. It rarely occurs as a complication of a legal abortion but is more commonly associated with criminal abortions, that is, those done illegally, under unsterile conditions, by persons who may have little or no knowledge of medicine or anatomy.

Signs & Symptoms

- Signs of Miscarriage
- Feeling unwell
- Lower abdominal pain
- Headache
- Vomiting
- Pyrexia
- Rapid pulse
- Offensive vaginal discharge
- Hypotension
- Profound leukocytosis
- Anaemia
- Generalized septicemia with peritonitis

Treatment

Broad-spectrum parenteral antibiotics, intravenous fluid therapy, and prompt evacuation of the uterus are indicated. A careful evaluation for trauma, including perforation of the uterus, vagina, or intraabdominal structures, should also be carried out.

3.3.8 Blood Mole

Occasionally mixed abortion may progress to blood mole. This is a smooth brownish red mass which contains the fetus and the placenta, and it is completely surrounded by the capsular deciduas. The mole usually forms before 12th week and it is retained in the

uterus for a period of months. Later the fluid is extracted from the blood and the fleshy, firm hard mass that is remaining is known as a Carneous Mole. On histological investigation, the fetus may be found in the centre of the mass.

Treatment

Management is the same as missed abortion.

3.3.9 Habitual Abortion or Recurrent Miscarriage

Abortion is said to be habitual or recurrent miscarriage if it has occurred spontaneously for at least three or more consecutive occasions. Following a history of three or more miscarriages, a referral is usually made to a specialist where appropriate and accurate information and support can be given as the risk of further abortion with subsequent pregnancies is high.

Incidence

Occurrence is about 1% of all pregnancies and in the early weeks of pregnancy if pregnancy continues till mid-trimester there is risk of threatened abortion or premature labour.

Causes

Most time unknown

Occurs more with incompetent cervix

Genetic factor which can be identified through karyotyping of the fetal tissue, as well as both parents.

Local causes such as fibroid, displacement of the uterus

Medical condition such as diabetes mellitus, nephritis, and tuberculosis.

Treatment

- Early booking
- No coitus,
- Hospitalization may be imperative for complete bed rest
- Shirodker stitches – (cervical cerclage) at about 14th –16th week
- Ventolin tablets 2-4mg bid or daily

- Give low dose aspirin and heparin if lupus anticoagulant and anticardiolipin antibodies are present.
- Give other treatment based on the cause(s) of the miscarriage identified.

3.3.10 Induced abortion

The term induced abortion is defined as the medical or surgical termination of pregnancy before the time of fetal viability. There are four statutory grounds for termination of pregnancy-

- (a) that the pregnancy has not exceeded its twenty-fourth week and that the continuance of the pregnancy would involve risk, greater than if the pregnancy were terminated, of injury to the physical or mental health of the pregnant woman or any existing children of her family; or
- (b) that the termination is necessary to prevent grave permanent injury to the physical or mental health of the pregnant woman; or
- (c) that the continuance of the pregnancy would involve risk to the life of the pregnant woman, greater than if the pregnancy were terminated; or
- (d) that there is a substantial risk that if the child were born it would suffer from such physical or mental abnormalities as to be seriously handicapped.

Induced abortion is classified as therapeutic and elective or voluntary abortion.

3.3.11 Therapeutic abortion

This is an abortion conducted for medical and surgical reason to save either the life of the mother or to prevent the child from being born with congenital abnormality.

Indication for therapeutic abortion

- The most common indication currently is to prevent birth of a fetus with a significant anatomical, metabolic, or mental deformity.
- Persistent cardiac decompensation especially with fixed pulmonary hypertension; advanced hypertensive vascular disease
- Conditions like diabetes and malignancy.
- In cases of rape or incest.

3.3.12 Elective or voluntary abortion

The interruption of pregnancy before viability at the request of the woman, but not for medical reasons, is usually termed elective or voluntary abortion. Regardless of terminology, these are stigmatized in many countries. Most abortions done today are elective, and thus, it is one of the most commonly performed medical procedures. It contributed greatly to maternal mortality rate and there are a lot of issues on legality and controversies surrounding it. For elective or voluntary abortion, there is a need to emphasize the need to provide standard-of-care counselling and timely referral if providers have individual beliefs that preclude pregnancy termination. There are three basic choices available to a woman considering an abortion: the first choice is to continued pregnancy with its risks and parental responsibilities; secondly, continued pregnancy with arranged adoption; and thirdly, terminate the pregnancy with its risks. A knowledgeable and compassionate counsellor should objectively describe and provide information regarding these choices so that a woman or couple can make an informed decision.

Techniques for abortion

Abortion procedures does not require hospitalization except there is a serious maternal medical disorder. However, there should be availability of equipment for cardiopulmonary resuscitation in the clinic and transportation for immediate transfer to a hospital if it requires. The methods used for terminating the pregnancy will depend on the gestational age. **First-trimester abortions** can be performed either medically or surgically:

Surgical: Dilatation and curettage, Vacuum aspiration, Menstrual aspiration.

Medical: Prostaglandins E2, F2 α , E1, and analogues Vaginal insertion, Parenteral injection, Oral ingestion Sublingual Antiprogesteroes—RU-486 (mifepristone) and epostane, Methotrexate—intramuscular and oral Various combinations of the above

Terminations in later pregnancy are carried out medically, using a regime of drugs to prepare and dilate the cervix. The actual regime used may vary across healthcare providers. The cervix is initially prepared using mifepristone, which is a progesterone

antagonist. This is given orally and is followed 36–48 hours later by vaginal and/or oral prostaglandins, such as misoprostol. The woman may return home in between the administration of the two drugs and should be provided with clear information about what to expect, the contact details of a named healthcare professional and the reassurance that admission to hospital can be at any time. During the termination, analgesia appropriate to her needs should be available. A termination of pregnancy should not result in the live birth of the fetus. To this effect, should the procedure take place after 21 weeks and 6 days gestation, feticide may be performed prior to the commencement of the termination process. This involves an injection of potassium chloride being injected into the fetal heart to prevent the fetus being born alive. Where nurses and midwives have a conscientious objection to termination of pregnancy, they have the right to refuse to be involved in such procedures. However, they cannot refuse to give life-saving care to a woman and must always be non-judgmental in any care and contact that they provide. As with other pregnancy losses those women who undergo a termination of pregnancy and are Rhesus negative will require anti-D immunoglobulin as recommended by national and local guidelines.

3.4 Gestational trophoblastic disease (GTD)

This is a condition in which there is an abnormal placental development leading to gross malformation of the trophoblast. The chorionic villi proliferate and become vesicles which looks like a bunch of English grape. It classified into a complete hydatidiform mole or a partial mole. and there is no viable fetus.

In the **complete** hydatidiform mole, there is no evidence of embryo, cord or membrane while in the **incomplete or partial** hydatidiform mole there is an evidence of embryo, fetus or amniotic sac.

Risk factors

- Age: risk is higher in teenager and women over 45 years
- Previous history of molar pregnancy
- Those with blood type Group A

Signs & Symptom:

These vary according to the type of mole.

- Exaggerated pregnancy symptoms by 6 – 8 weeks.
- Bleeding or blood-stained vaginal discharge after a period of amenorrhea.
- Slight pink or brownish discharge,
- Passage of vesicles per vagina,
- Anaemia,
- High chorionic gonadotrophic hormone (CGTH) level,
- Pre-eclampsia in early pregnancy,
- On palpation – uterus larger than date feels doughy or elastic, no fetal parts, no fetal height can be mapped, no fetal movement.

Diagnosis

- Ultrasound
- Increase CGTH

Treatment

- Remove all the trophoblastic tissues by terminating the pregnancy
- Follow up-to 2 years until CGTH is negative
- Give psychological support.

3.5 Post Abortion Care (PAC)

This is an approach for reducing morbidity and mortality from incompetent and unsafe abortion and resulting complications and for improving women's sexual and reproductive health lives.

Elements of PAC:

There are 5 elements of PAC which are:

- Treatment of incomplete and unsafe abortion and abortion-related complications that are potentially life-threatening
- Counselling to identify and respond to women's emotional and
- Physical health needs and other concerns

- Contraceptives and family planning services to
 - Help women prevent unwanted pregnancy
 - Encourage the practice of birth spacing
 - Reproductive and other health services that are:
 - Provided on-site
 - Provide via referrals to other facilities in providers' networks
- Community and service provider partnerships to:
 - Prevent unwanted pregnancies and unsafe abortion
 - Mobilize resources for timely care for complications from abortion
 - Ensures health services reflect and meet community expectations and needs

Principles that Support Patients' Rights in PAC Setting

- Having empathy and respect for patients
- Maintaining positive interaction and communication with patients
- Respecting privacy and confidentiality

Roles of the Nurse/Midwife in PAC

- The nurse/midwife is the general overseer or manager of the totality of Manual vacuum Aspiration (MVA) services within the facility
- The nurse/midwife has the responsibility of ensuring that the facilities and the necessary equipment's are always available at the MVA room. Portable water should be made available.
- She should ensure proper cleaning and setting of trolley.
- She must also ensure completeness of the items on both shelves of the trolley
- Pre and post-procedure care of the patients is an important responsibility of the nurse/midwife.
- Her role in the actual MVA procedure depends on whether she is permitted to carry out the procedure or to assist the doctor during a procedure. In whichever situation, she must have a good grip of the procedure.

- She must possess a proper understanding of cleaning and sterilization/or disinfecting of equipment used during the procedure and disposal of wastes, aspirates and sharp instruments in order to prevent infection especially HIV/AIDS and Hepatitis B virus
- She is responsible for keeping record of details of the procedure.

3.6 Manual Vacuum Aspiration (MVA)

This is a procedure carried out to evacuate uterine contents in incomplete abortion.

Indications

- Threatened or imminent abortion, Inevitable abortion, Incomplete abortion
- Infected abortion, Missed abortion, Hydatidiform mole
- Retained placental products

Advantages

- Requires only slight dilatation and scrapes gently
- Lower risk of complications, Lower cost of services, Can be used in low resource setting, Decreased need for hospitalization, is a day case.
- The procedure is usually carried out by trained health personnel.

(Refer: Health worker roles in providing safe abortion care and post-abortion contraception by WHO)

4.0 CONCLUSION

In module 1, unit 1, you have learned some major causes of bleeding in early pregnancy. You have learned how to recognize each of such conditions and their management. The concept of post-abortion care and the role of the nurse/midwife have been clearly described in this unit

5.0 SUMMARY

In this unit, you have learnt about the following:

- I. Causes of bleeding before 24th week of pregnancy
- II. Post-abortion care

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Describe the different types of abortion and their management

Describe the possible locations for ectopic pregnancy

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Unit 2: Bleeding after 24th week pregnancy

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
 - 3.1 Antepartum haemorrhage
 - 3.2 Placental praevia
 - 3.3 Placental abruption
 - 3.4 Vasa praevia
- 4.0 Conclusion
- 5.0 Summary
- 6.0 Online discussion and assignment
- 7.0 References/further reading

1.0 INTRODUCTION

Bleeding after 24 weeks of pregnancy is of great concerns for the woman and her family as it is associated with fear of losing the foetus among others. It is associated with increased in perinatal and maternal morbidity and mortality. Therefore, the knowledge of topics treated in this unit will equip you to be able to recognise these conditions and offer appropriate care. In this unit, you will learn how to nurse a woman with antepartum haemorrhage, placenta praevia and placenta abruption

2.0 OBJECTIVES

At the end of this unit you should be able to:

- Describe antepartum haemorrhage
- Describe placenta praevia and its management
- Discuss placenta abruption and its management
- Describe the management of vasa praevia

3.0 MAIN CONTENT

3.1 Antepartum haemorrhage (APH)

Antepartum haemorrhage is defined as bleeding from the vagina after the 24th week of pregnancy before the onset of labour. It is critical for the well-being of both the mother and the fetus that the patient who presents with third trimester bleeding be evaluated and managed emergently. The incidence of antepartum haemorrhage is 3 per cent. It is estimated that 1 per cent is attributable to placenta praevia, 1 per cent to placental abruption and the remaining 1 per cent is from other causes. Placental causes are obviously the most worrying, as potentially the mother's and/or fetus' life is in danger. The factors that cause antepartum haemorrhage may be present before 20 weeks, but the distinction between a threatened miscarriage and an antepartum haemorrhage is based on whether the fetus is considered potentially viable.

Causes of antepartum haemorrhage

1. Placental causes
2. Non-Placental or incidental causes

Placental causes

- i. Placental Praevia: Unavoidable haemorrhage
- ii. Abruption Placenta – Accidental haemorrhage
- iii. Vasa praevia

Non-Placental causes:

Incidental causes – Bleeding from other lesion of the genital tract e.g. Cervical causes – cervical erosion, cervical laceration, Polyps, cervical carcinoma.

Vaginal causes – laceration, vaginitis, ruptured varicose veins of the vulva.

Effect of APH on the mother

A small amount of bleeding will not physically affect the woman (unless she is already severely anaemic) but it is likely to cause her anxiety. In cases of heavier bleeding, this may be accompanied by medical shock and blood clotting disorders. The midwife will be

aware that the woman can die or be left with permanent morbidity if bleeding in pregnancy is not dealt with promptly and effectively.

Effect of APH on the foetus

Fetal mortality and morbidity are increased as a result of severe vaginal bleeding in pregnancy. Stillbirth or neonatal death may occur. Premature placental separation and consequent hypoxia may result in severe neurological damage in the baby.

Management of APH

- **History**
 - How much bleeding?
 - Triggering factors (e.g. postcoital bleed).
 - Associated with pain or contractions?
 - Is the baby moving?
 - Last cervical smear (date/normal or abnormal)?
- **Examination**
 - Pulse, blood pressure.
 - Is the uterus soft or tender and firm?
 - Fetal heart auscultation/CTG.
 - Speculum vaginal examination, with particular importance placed on visualizing the cervix (having established that placenta is not a praevia, preferably using a portable ultrasound machine).
- **Investigations**
 - Ultrasound (fetal size, presentation, amniotic fluid, placental position and morphology).
 - Depending on the degree of bleeding:
 - Conduct full blood count and clotting
 - Give specific management, if praevia/abruption suspected

3.2 Placenta praevia

Placenta praevia is a condition whereby the placenta is partially or wholly implanted in the lower uterine segment. The lower uterine segment grows and stretches progressively

after the 12th week of pregnancy. In later weeks this may cause the placenta to separate and severe bleeding can occur. The amount of bleeding is not usually associated with any particular type of activity and commonly occurs when the woman is resting. The low placental location allows all of the lost blood to escape unimpeded and a retroplacental clot is not formed. For this reason, pain is not a feature of placenta praevia. Some women with this condition have a history of a small repeated blood loss at intervals throughout pregnancy whereas others may have a sudden single episode of vaginal bleeding after the 20th week. However, severe haemorrhage occurs most frequently after the 34th week of pregnancy. The degree of placenta praevia does not necessarily correspond to the amount of bleeding. A type 4 placenta praevia may never bleed before the onset of spontaneous labour or elective caesarean section in late pregnancy or conversely, some women with placenta praevia type 1 may experience relatively heavy bleeding from early in their pregnancy.

Degrees of placenta praevia

Type 1 placenta praevia: this is also called low lying placenta. The majority of the placenta is in the upper uterine segment. Blood loss is usually mild and the mother and fetus remain in good condition. Vaginal birth is possible.

Type 2 placenta praevia: The placenta is partially located in the lower segment near the internal cervical os (marginal placenta praevia). Blood loss is usually moderate, although the conditions of the mother and fetus can vary. Fetal hypoxia is more likely to be present than maternal shock. Vaginal birth is possible, particularly if the placenta is anterior.

Type 3 placenta praevia: The placenta is located over the internal cervical os but not centrally and it is also called partial placenta praevia. Bleeding is likely to be severe, particularly when the lower segment stretches, and the cervix begins to efface and dilate in late pregnancy. Vaginal birth is inappropriate because the placenta precedes the fetus.

Type 4 placenta praevia: The placenta is located centrally over the internal cervical os (complete placenta praevia) and torrential haemorrhage is very likely. Caesarean section is essential to save the lives of the woman and fetus.

Incidence

Placenta praevia affects 2.8 per 1000 of singleton pregnancies and 3.9 per 1000 of twin pregnancies. There is a higher incidence of placenta praevia among women with increasing age and parity, in women who smoke and those who have had a previous caesarean section. Furthermore, it is known that there is also an increased risk of recurrence where there has been a placenta praevia in a previous pregnancy.

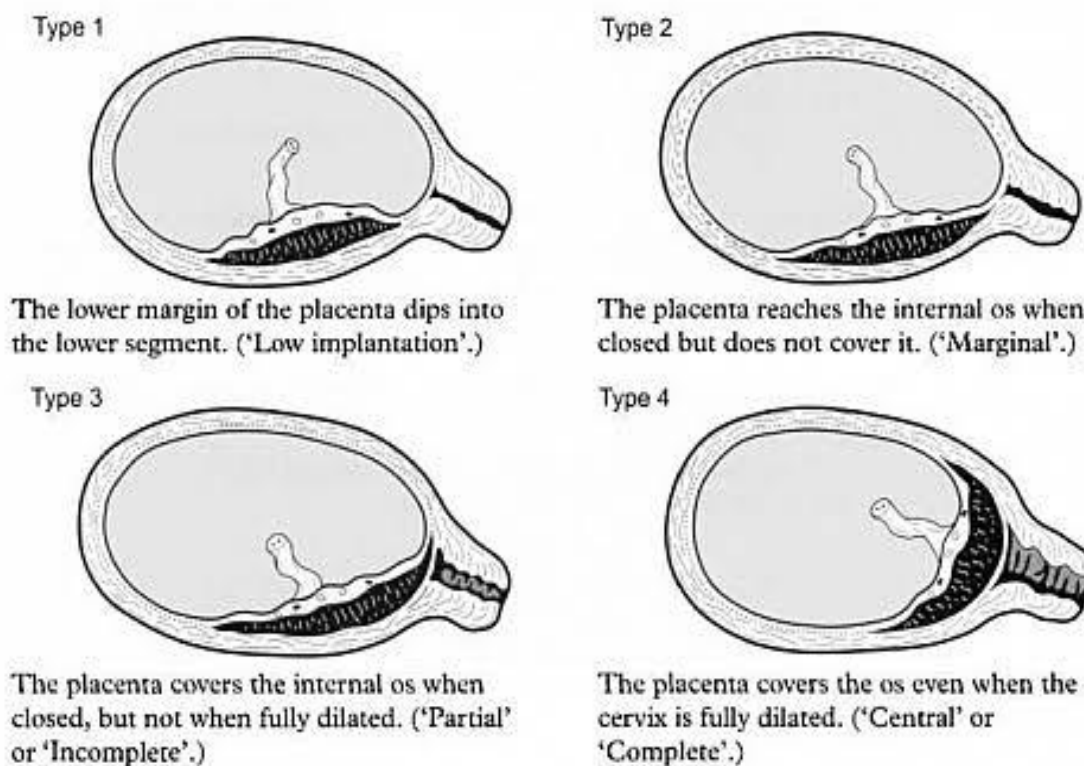


Figure 2-1: Types and positions of placenta praevia: Source: Placent Praevia. JolScroll

Causes of placenta praevia;

- Grande multiparity; Previous C/S,

- Multiple Pregnancy, Previous Placenta Praevia
- Certain fetal abnormalities.
- Age – older mother are more at risk than younger ones.
- Abnormal Placenta – Bipartita and succenturiate placentae

Diagnosis

- Commonly it manifest 34-38wk though sometimes earlier.
- On general examination, the woman may be clinically normal.
- On Abdominal Examination:
 - Difficulty in identifying the fetal part on palpation.
 - Fundal height may be normal
 - Uterus is normal in consistency, no tenderness or tension
 - Fetal Heart rate may also be normal – depending on the severity of bleeding.
 - Presentation may be abnormal e.g. Breech.
 - High head, oblique or Transverse lie or unstable
- On vulva Inspection Slight or severe bleeding
- Fetal movement – Excessive or slow or normal.
- Rapid respiration, Pulse – Signs of shock in the mother

Management of placenta praevia

Immediate re-localization of the placenta using ultrasonic scanning is a definitive aid to diagnosis, and as well as confirming the existence of placenta praevia it will establish its degree. Relying on an early pregnancy scan at 20 weeks of pregnancy is not very useful when vaginal bleeding starts in later pregnancy, as the placenta tends to migrate up the uterine wall as the uterus grows in a developing pregnancy. Further management decisions will depend on: the amount of bleeding, the condition of the woman and fetus, the location of the placenta, the stage of the pregnancy.

Conservative management

This is appropriate if bleeding is slight and the woman and fetus are well. The woman will be kept in hospital at rest until bleeding has stopped. A speculum examination will have ruled out incidental causes. Further bleeding is almost inevitable if the placenta encroaches into the lower segment; therefore, it is usual for the woman to remain in, or close to hospital for the rest of her pregnancy. A visit to the special care baby unit/neonatal intensive care unit and contact with the neonatal team may also help to prepare the woman and her family for the possibility of pre-term birth. A decision will be made with the woman about how and when the birth will be managed. If there is no further severe bleeding, vaginal birth is highly likely if the placental location allows. The midwife should be aware that, even if vaginal birth is achieved, there remains a danger of postpartum haemorrhage because the placenta has been situated in the lower segment where there are fewer oblique muscle fibres and the action of the living ligatures is less effective.

Immediate management of life-threatening bleeding

Severe vaginal bleeding will necessitate immediate birth of the baby by caesarean section regardless of the location of the placenta. This should take place in a maternity unit with facilities for the appropriate care of the newborn, especially if the baby is preterm. During the assessment and preparation for theatre, the woman will be extremely anxious, and the midwife must comfort and encourage her, sharing information with her as much as possible. The partner will also need to be supported, whether he is in the operating theatre or waits outside. If the placenta is situated anteriorly in the uterus, this may complicate the surgical approach as it underlies the site of the normal incision. In major degrees of placenta praevia (types 3 and 4) caesarean section is required even if the fetus has died in utero. Such management aims to prevent torrential haemorrhage and possible maternal death.

Complications of placenta praevia

- Maternal shock, resulting from blood loss and hypovolaemia.

- Anaesthetic and surgical complications, which are more common in women with major degrees of placenta praevia and in those for whom preparation for surgery has been suboptimal.
- Placenta accreta, in up to 15% of women with placenta praevia.
- Air embolism, an occasional occurrence when the sinuses in the placental bed have been broken.
- Postpartum haemorrhage: occasionally uncontrolled haemorrhage will continue, despite the administration of uterotonic drugs at the birth, even following the best efforts to control it, and a ligation of the internal iliac artery. A caesarean hysterectomy may be required to save the woman's life.
- Maternal death is rare in the developed world.
- Fetal hypoxia and its sequelae due to placental separation.
- Fetal death, depending on gestation and amount of blood loss.

3.3 Placenta abruption

Bleeding is due to premature separation of a normally situated placenta occurring after 22 weeks of pregnancy. It is sometimes referred to as accidental bleeding, it is about 2% of all pregnancies. It may occur at any stage of pregnancy or during labour and it is severe enough to result in fetal death occurs in 1 per 500 deliveries. The risk for recurrent abruption is 10% after one abruption and 25% after two.

Causes

The aetiology of haemorrhage is not always clear in 40% cases but it is often associated with:

- Maternal hypertension
- Placental abruption in a prior pregnancy
- Trauma
- Polyhydramnios with rapid decompression
- Premature rupture of membranes
- Short umbilical cord

- Tobacco use
- Folate deficiency

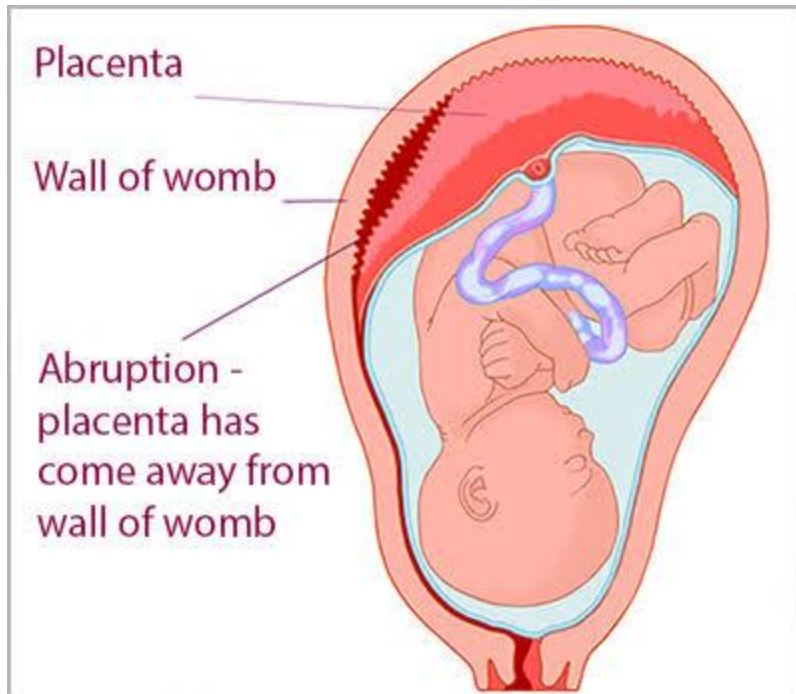


Figure 2-2: Placenta abruption. Source: Figure 2-2: Placenta abruption. Source: www.tommys.org

Pathophysiology

Placental separation is initiated by haemorrhage into the decidua basalis with formation of a decidual hematoma. The resulting separation of the decidua from the basal plate predisposes to further separation and bleeding as well as to compression and destruction of placental tissue. The inciting cause of placental separation is unknown. It may be due to an inherent weakness or anomaly in the spiral arterioles. Blood may either dissect upward toward the fundus, resulting in a concealed hemorrhage, or extend downward toward the cervix, resulting in an external or revealed hemorrhage.

Types of placenta abruption

Revealed haemorrhage

In this case blood escapes from the vagina it is the commonest type. Bleeding may become severe from slight. It may be accompanied by abdominal pain and tenderness, delivery of the baby should be accomplished within a few hours (6 hours) to avoid coagulation failure developing. Bleeding is proportional to the amount of visible vaginal blood loss.

2. Mixed or combined hemorrhage.

This haemorrhage is primarily concealed then later becomes revealed with little vaginal bleeding. A degree of shock is exhibited which is usually severe compared with the vaginal bleeding. It is usually associated with blood coagulation disorders.

3. Concealed haemorrhage

This is a serious condition with high maternal and fetal mortality. It account for 55% of maternal death. It is associated with severe bleeding but no vaginal bleeding occurs but large retro-placental clot forms behind the placenta – maternal surface. Mother shows signs and symptoms of hypovolaemic shock, uterine enlargement and severe pain.

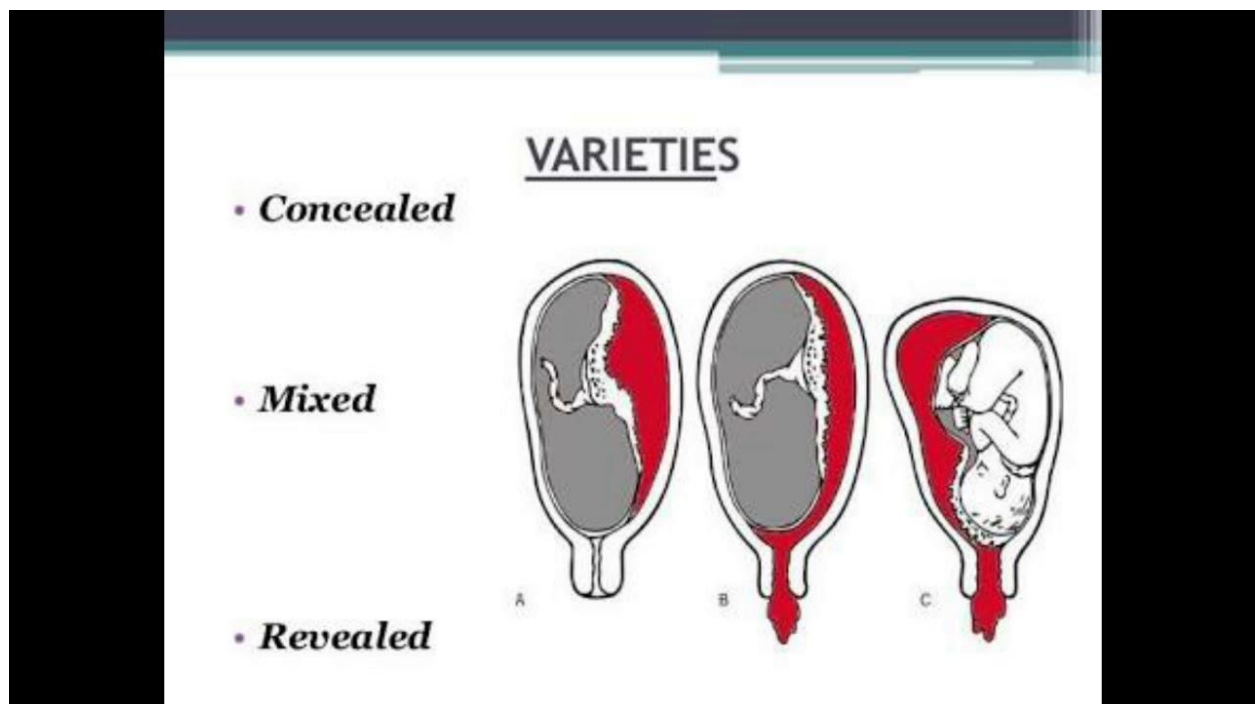


Figure 2-3: Types of placenta abruption. Source: Slideshare – Abruption placenta

Signs and Symptoms

- The most common finding is vaginal bleeding (66%)
- Abdominal pain
- Uterine tenderness
- Fetal distress
- Uterine hyperactivity and increased uterine tone
- Fetal demise
- History of pregnancy-induced hypertension
- Headache
- Nausea and vomiting
- Epigastric pain, following road traffic accident or trauma.

Diagnosis

The diagnosis of placental abruption is made clinically.

Ultrasonography may detect only 2% of abruptions. Because placental abruption may coexist with a placenta praevia, the reason for doing an initial ultrasonic examination is to exclude the praevia.

Management of placenta abruption

- Maternal hemodynamic and fetal monitoring
- Serial evaluation of the hematocrit and coagulation profile, and delivery.
- Intensive monitoring of both the mother and the fetus is essential because the rapid deterioration of the condition of either one can occur.
- Blood products for replacement should always be available, and a large-bore (16- to 18-gauge)
- Intravenous line must be secured.
- Red blood cells should be given liberally if indicated.
- In the setting of placental abruption, the use of tocolytics or uterine relaxants is not advised.

- Uterine tone must be maintained to control bleeding following delivery, or at least to control the bleeding sufficiently to allow a safe hysterectomy to be performed, if necessary.
- Low Protein diet, low sodium and potassium, Estimate blood urea, Potassium for 3 days.
- Accurate fluid balanced chart.
- Report signs of Oliguria (less than 500mls daily).

Complication

- Disseminated intravascular coagulation (DIC) – moderate & severe
- Post-partum haemorrhage due to convelaire uterus or DIC.
- Renal failure – hypovolaemia, poor kidney perfusion.
- Pituitary necrosis resulting from prolonged and severe hypotension – shock.
- Increased mortality for the infants – 50-80%

3.4 Vasa praevia

Vasa praevia is a very rare condition where one of the branches of the fetal umbilical vessels lies in the membranes and across the cervical os. It is a condition in which fetal blood vessels cross or run near the internal opening of the uterus. These vessels are at risk of rupture when the supporting membranes rupture, as they are unsupported by the umbilical cord or placental tissue. Rupture of the membranes over the cervical os may cause a tear in the vessels which will result in the rapid exsanguination of the fetus.

Incidence of vasa praevia

Vasa praevia is uncommon in the general population with a prevalence ranging between 1 in 1200 and 1 in 5000 pregnancies, although the condition may have been under-reported. The fetal mortality rate in this situation is at least 60% despite urgent caesarean delivery. However, improved survival rates of over 95% have been reported where the diagnosis has been made antenatally by ultrasound followed by planned caesarean section

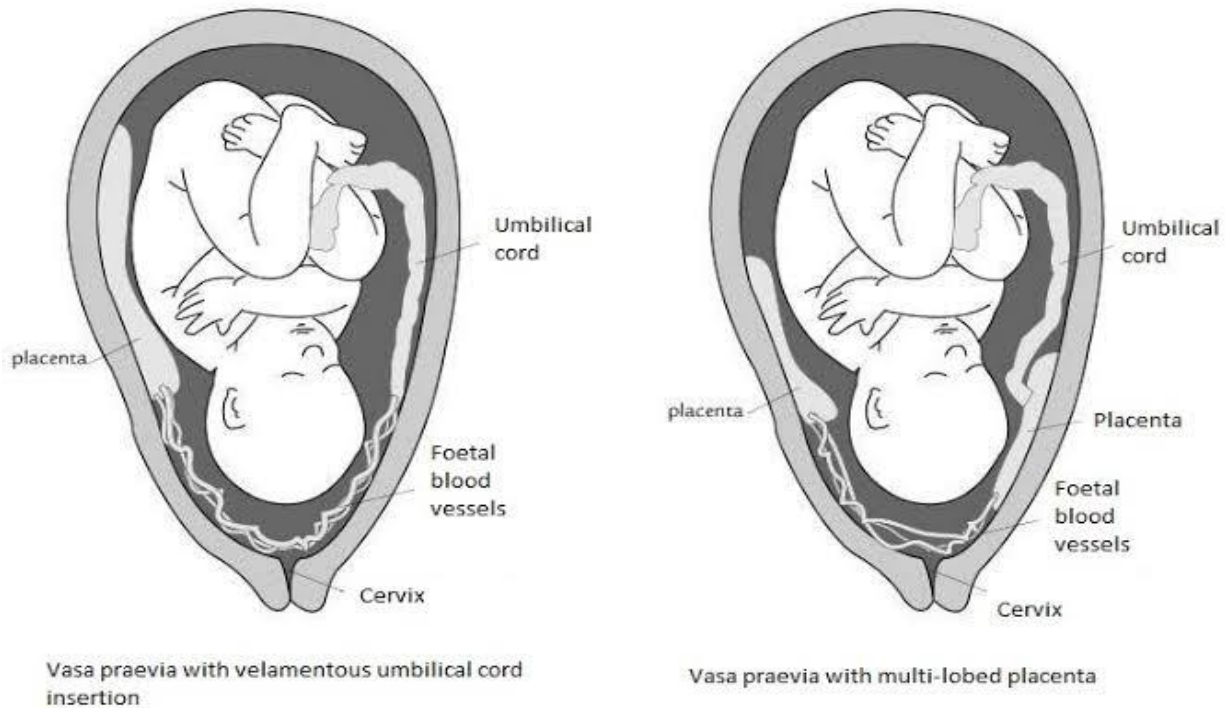


Figure 2-4: causes of vasa praevia: Source: Vasa praevia - Wikipedia

Causes of vasa praevia

- Velamentous cord insertion: this is an umbilical cord abnormality, where there is a membranous insertion of the cord and the vessels course through the membranes to resulting in vessels that are unprotected leading to the placenta. This is classified as type I
- Bilobed placenta or succenturiate lobe of the placenta: this is where the placenta is in two pieces and the vessels in the membrane connect the main placental mass and the separate lobe. This is classified as type II

Diagnosis and management

Vasa praevia is diagnosed with colour Doppler ultrasound or prenatal ultrasound scan. Delivery of a pregnancy that is complicated by vasa previa should occur by cesarean birth at a center that is capable of providing immediate neonatal blood transfusion if needed.

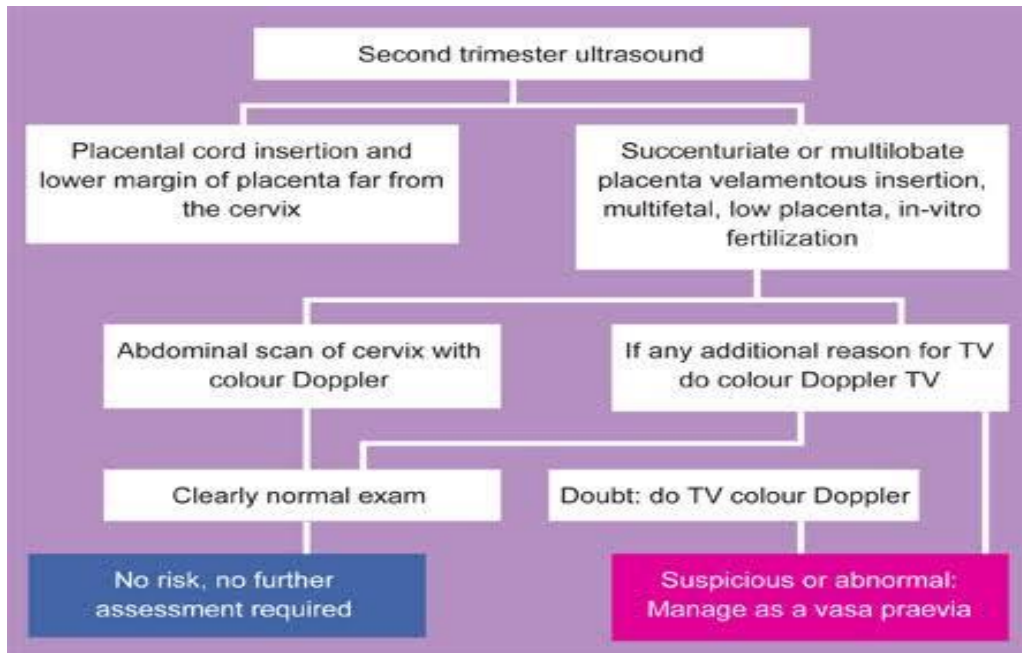


Figure 2-5: Diagnosis and management of vasa praevia. Source: Vasa Praevia raising awareness

4.0 CONCLUSION

In this unit, you have learned major causes of bleeding in late pregnancy. You have learned how to recognize each of such conditions and their management.

5.0 SUMMARY

In this unit, you have learnt about the following:

- I. Antepartum haemorrhage
- II. Placenta praevia
- III. Placenta abruption
- IV. Vasa praevia

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Describe the different types of placenta praevia and their management

Describe the management of placenta abruption

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Unit 3 Hypertensive disorder in pregnancy

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
 - 3.1 Hypertension in pregnancy
 - 3.2 Chronic hypertension
 - 3.3 Gestational hypertension
 - 3.4 Pre-eclampsia and Eclampsia
- 4.0 Conclusion
- 5.0 Summary
- 6.0 Online discussion and assignment
- 7.0 References/further reading

1.0 INTRODUCTION

Hypertensive disorders remain the commonest complication of pregnancy in the developed world and are consistently one of the three main causes of maternal death. The incidence varies substantially in different countries and is influenced by a number of factors like- parity, ethnic group and dietary intake. Hypertensive disorder may influence the outcome of pregnancy and the progress of the disease may be influenced by the pregnancy. In its mildest form hypertension alone arising in late pregnancy appears to be of minimal risk to mother or child. In its most severe form, the condition is associated with placental abruption, convulsions, proteinuria, severe hypertension and oedema and may result in cerebral haemorrhage, renal and hepatic failure as well as disseminated intravascular coagulopathy (DIC). This may lead to fetal and maternal death. This unit examines pregnancy-induced hypertension, pre-eclampsia and eclampsia.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Define and classify hypertensive disease in pregnancy, including preeclampsia, eclampsia,
- Discuss the pathophysiology and management of pregnancy-induced hypertension
- Describe pre-eclampsia and eclampsia
- Discuss the management of pre-eclampsia and eclampsia
- Discuss the effect of hypertensive disorders on the mothers and her foetus

3.0 MAIN CONTENTS

3.1 Hypertension in pregnancy

Hypertensive disorders occur in approximately 12% to 22% of pregnancies and cause substantial perinatal morbidity and mortality for both mother and fetus. Hypertensive disease is directly responsible for approximately 20% of maternal deaths in the United States. The exact cause of hypertension in pregnancy remains unknown. Classification of hypertension in pregnancy include chronic hypertension, gestational hypertension, preeclampsia, eclampsia and

3.1.1 Pathophysiology of hypertension in pregnancy

Hypertension in pregnancy affects the mother and newborn to varying degrees. The predominant pathophysiologic finding in preeclampsia and gestational hypertension is maternal vasospasm. Several potential causes for maternal vasospasm have been postulated as follows:

1. **Vascular changes:** Instead of noting the physiologic trophoblast-mediated vascular changes in the uterine vessels (decreased musculature in the spiral arterioles leads to the development of a low-resistance, low pressure, high-flow system), inadequate maternal vascular response is seen in cases of preeclampsia and/or intrauterine fetal growth restriction. Endothelial damage is also noted within the vessels.

2. **Hemostatic changes:** Increased platelet activation with increased consumption in the microvasculature is noted during the course of preeclampsia. Endothelial fibronectin levels are increased and antithrombin III and α 2-antiplasmin levels are decreased,

reflecting endothelial damage. Low antithrombin III levels are permissive for microthrombi development. Endothelial damage is then thought to promote further vasospasm.

3. **Changes in prostanoids:** Prostacyclin (PGI₂) and thromboxane (TXA₂) are increased during pregnancy, with the balance in favour of PGI₂. In patients who develop preeclampsia, the balance shifts to favour TXA₂. Again, PGI₂ functions to promote vasodilatation and decrease platelet aggregation, and TXA₂ promotes vasoconstriction and platelet aggregation. Because of this imbalance, vessel constriction occurs.

4. **Changes in endothelium-derived factors:** Nitric oxide, a potent vasodilator, is decreased in patients with preeclampsia and may explain the evolution of vasoconstriction in these patients.

5. **Lipid peroxide, free radicals, and antioxidant release:** Lipid peroxides and free radicals have been implicated in vascular injury and are increased in pregnancies complicated by preeclampsia. Decreased antioxidant levels are also noted.

These five mechanisms above are thought to contribute to the following common pathophysiologic changes seen in patients with preeclampsia:

1. **Cardiovascular effects:** Elevated blood pressure is seen as the result of potential vasoconstriction as well as an increase in cardiac output.

2. **Hematologic effects:** Plasma volume contraction may develop, with risk of rapid onset hypovolemic shock, if haemorrhage occurs. Plasma volume contraction is reflected in increased hematocrit values. Thrombocytopenia/disseminated intravascular coagulation may also develop from microangiopathic hemolytic anemia. Involvement of the liver may lead to hepatocellular dysfunction and further evolution of coagulopathy. Third spacing of fluid may be noted, because of increased blood pressure and decreased plasma oncotic pressure. DIC

3. **Renal effects:** Decreased glomerular filtration rate (increasing serum creatinine) and proteinuria (urine protein levels greater than 300 mg per 24 hours) develop secondary to atherosclerotic-like changes in the renal vessels (glomerular endotheliosis). Uric acid filtration is decreased; therefore, elevated maternal serum uric acid levels may be an indication of evolving disease.

4. **Neurologic effects:** Hyperreflexia/hypersensitivity may develop. In severe cases, grand mal (eclamptic) seizures may develop.

5. **Pulmonary effects:** Pulmonary edema may occur and can be related to decreased colloid oncotic pressure, pulmonary capillary leak, left heart failure, iatrogenic fluid overload, or a combination of these factors.

6. **Fetal effects:** Decreased intermittent placental perfusion secondary to vasospasm is thought to be responsible for the increased incidence of intrauterine growth restriction, oligohydramnios, and increased perinatal mortality of infants born to mothers with preeclampsia. An increased incidence of placental abruption is also seen. With the stress of uterine contractions during labour, the placenta may be unable to adequately oxygenate the fetus. This may result in signs of intrapartum uteroplacental insufficiency. Specifically, a non-reassuring fetal-heart-rate pattern may necessitate cesarean delivery. Presumably because of vasospastic changes, placental size and function are decreased. The results are progressive fetal hypoxia and malnutrition, as well as an increase in the incidence of intrauterine growth restriction and oligohydramnios.

3.2 Chronic hypertension

Chronic hypertension is defined as hypertension present before the 20th week of pregnancy or hypertension present before pregnancy. A major risk with chronic hypertension is the development of preeclampsia or eclampsia later in the pregnancy, which is relatively common and difficult to diagnose. The acute onset of proteinuria and worsening hypertension in women with chronic hypertension is suggestive of

superimposed preeclampsia. The categories of hypertension in pregnancy and the blood pressure (BP) criteria used to define each are as follows:

Mild hypertension: Systolic pressure of ≥ 140 – 180 mm Hg or diastolic pressure of ≥ 90 – 100 mm Hg or both

Severe hypertension: Systolic pressure of ≥ 180 mm Hg or diastolic pressure of ≥ 100 mmHg.

Management of chronic hypertension

Closely monitoring maternal blood pressure and observe her for superimposition of preeclampsia or eclampsia and following the fetus for appropriate growth and fetal wellbeing. Antihypertensive medication in women with chronic hypertension is generally not given unless the systolic blood pressure is 150 to 160 mm Hg or the diastolic blood pressure 100 to 110mmHg. In such case, you continue **antihypertensive medications** with the exception of angiotensin-converting enzyme (ACE) inhibitors. These drugs have been associated with progressive and irreversible renal injury and possibly other structural anomalies in the fetus. It was formerly taught that diuretics were contraindicated during pregnancy, but diuretic therapy is no longer discontinued, and indeed is usually continued, in the patient who already has been on such therapy before becoming pregnant.

Fetal testing (serial ultrasound examinations for fetal growth with or without fetal non-stress testing) should be initiated after 32 weeks' gestation.

Complications of chronic hypertension

- Superimposed preeclampsia
- Intrauterine fetal growth restriction (IUGR)
- Placental abruption
- Stillbirth.

3.3 Gestational hypertension

Hypertension that develops after 20 weeks of gestation in the absence of proteinuria and returns to normal postpartum is termed gestational hypertension. Gestational hypertension develops in 5% to 10% of pregnancies that proceed beyond the first trimester, with a 30% incidence in multiple gestations, regardless of parity. Maternal morbidity is directly related to the severity and duration of hypertension. Approximately 25% of women with gestational hypertension develop superimposed preeclampsia or eclampsia. It is often difficult to distinguish between preeclampsia and gestational hypertension when a patient is seen late in pregnancy with an elevated blood pressure level. In such cases, it is always wise to assume that the findings represent preeclampsia and treat accordingly.

Diagnosis of gestational hypertension

Persistent elevation of BP \geq 140/90 mmHg in the third trimester without evidence of preeclampsia.

Causes of gestational hypertension

- It probably represents an exaggerated physiologic response of the maternal cardiovascular system to pregnancy.
- Rarely associated with adverse maternal or fetal outcome.

3.4 Preeclampsia and eclampsia

Preeclampsia is the development of hypertension with proteinuria and edema after 20 weeks of gestation. This condition can occur earlier in the presence of gestational trophoblastic disease.

Eclampsia

Eclampsia is the additional presence of convulsions (grandmal seizures) in a woman with preeclampsia that is not explained by a neurologic disorder. Eclampsia occurs in 0.5% to 4% of patients with preeclampsia.

Risk factors for preeclampsia

- Nulliparity
- Multifetal gestation
- Maternal age over 35 years
- Preeclampsia in a previous pregnancy
- Chronic hypertension
- Pregestational diabetes
- Vascular and connective tissue disorders
- Nephropathy
- Antiphospholipid syndrome
- Obesity
- African-American race

Criteria for diagnosis of preeclampsia

- Blood pressure of ≥ 140 mmHg systolic or ≥ 90 mmHg diastolic that occurs after 20 weeks of gestation in a woman with previously normal blood pressure
- Proteinuria, defined as urinary excretion of 0.3 g protein or higher in a 24-hour urine specimen

Severe preeclampsia

Severe preeclampsia is an indication for delivery, regardless of gestational age or maturity.

Clinical manifestations of severe preeclampsia

- Blood pressure ≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic on two occasions at least 6 hours apart while the patient is on bed rest
- Marked proteinuria (generally ≥ 5 g per 24-hour urine collection, or 3 + or more on two dipstick of random urine samples collected at least 4 hours apart)
- Oliguria < 500 mL in 24 hours

- Cerebral or visual disturbances such as headache and scotomata (“spots” before the eyes) Pulmonary edema or cyanosis
- Epigastric or right-upper-quadrant pain (probably caused by subcapsular hepatic haemorrhage or stretching of Glisson capsule)
- Evidence of hepatic dysfunction
- Thrombocytopenia
- Intrauterine fetal growth restriction (IUGR)

Observations and investigations

Maternal

1. Blood pressure should be measured every 15–20 minutes (initially using a mercury sphygmomanometer to exclude cases in which automated machines underestimate pressure)
2. Oxygen saturation should be monitored continuously
3. Urine output measured hourly
4. Urea and electrolytes, full blood count, liver function tests and coagulation screen at least every 24 hours and more often as clinically indicated.

Fetal

1. Ultrasound biophysical assessment (Fetal maturity and estimate of fetal size if not known.)
2. Continuous cardiotocography

Management of preeclampsia and eclampsia

1. Magnesium sulphate reduces the risk of eclampsia by around half in women with preeclampsia before delivery or presenting within 24 hours of giving birth. Intravenous access should be established as part of the admission protocol, ideally using the cannula used for obtaining initial blood samples, and 4g of magnesium sulphate given over 5–10 minutes. This should be followed by a maintenance infusion of 1g per hour. This should be continued until 24 hours after the last fit or if the deep tendon reflexes are absent (check hourly) or the respiratory rate is 14 per minute or less. Repeat fits may be treated by using further boluses of magnesium sulphate or diazepam.

2. **Hypertensive therapy:** Hydralazine, 5mg over 15 minutes and repeated to a maximum cumulative dose of 20mg, is the approach of choice. A labetalol infusion also has a role as a second-line agent.

3. **Fluid balance:** Fluid overload can readily occur and pulmonary oedema rapidly develop. Standard fluid regimes should be used and monitored. A CVP line may be required to assess fluid balance and aid management.

4. **Delivery:** Delivery is the only effective treatment for preeclampsia and is recommended:

i. If the condition is one of severe pre-eclampsia, then the timing of delivery will depend on the rate of deterioration of the mother's condition and the maturity of the pregnancy.

ii. If an eclamptic fit has occurred, then, if the baby is alive and viable, delivery should be expedited, often by caesarean section. If the cervix is favourable, then induction of labour still has a role, particularly in parous women.

5. Depending on the coagulation status, consideration should be given to prophylaxis of DVT and even during the assessment period compression stockings should be provided.

Prognosis

Preeclampsia and its complications always resolve after delivery (with the exception of cerebrovascular accident). Diuresis (> 4 L/day) is the most accurate clinical indicator of resolution. Fetal prognosis is dependent largely on gestational age at delivery and problems related to prematurity.

4.0 CONCLUSION

Hypertensive disorder may influence the outcome of pregnancy and the progress of the disease may be influenced by the pregnancy. Pre-eclampsia is largely a disease of signs rather than symptoms. Headache, visual disturbance and abdominal pain may indicate progression towards eclampsia.

5.0 SUMMARY

In this unit, you have learnt hypertension in pregnancy and its pathophysiology, the mechanisms that contribute to the pathophysiologic changes seen in patients with preeclampsia, definition and classification of chronic hypertension, management of

chronic hypertension, gestational hypertension and its management, preeclampsia and eclampsia

6.0 ONLINE DISCUSSION AND ASSIGNMENT

- Explain the pathophysiology of hypertension in pregnancy
- Explain the mechanisms that contribute to the pathophysiologic changes seen in patients with preeclampsia
- Describe the management of preeclampsia and eclampsia

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Module 2 Complication associated with labour

Complications associated with labour also refer to as abnormal labour and they are often characterized by the abnormal progression of labour. Abnormal labour is the leading indication for primary cesarean delivery in the United States. Despite the high prevalence of labour disorders, considerable variability exists in the diagnosis, management, and criteria for abnormal labour that requires intervention. In this module you will learn: intrapartum complication such as cord prolapse, maternal distress, foetal distress, shoulder dystocia and brachial plexus injury, obstetric shock, prolonged labour; obstructed labour; inverted uterus/retroverted uterus; malposition and malpresentation

Module Objectives

At the end of this module you should be able to:

- i. Describe some common intrapartum complications
- ii. Discuss the management of some common complications associated with pregnancy

Unit 1 Intrapartum complications

CONTENTS

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
 - 3.1 Cord prolapse
 - 3.2 Maternal and Foetal distress
 - 3.3 Prolong labour
 - 3.4 Obstetric shock

- 4.0 Conclusion
- 5.0 Summary
- 6.0 Online discussion and assignment
- 7.0 References/further reading

1.0 INTRODUCTION

Intrapartum complications discussed in this unit are conditions that require urgent attention as they contribute to maternal and neonatal mortality rate. Because recognition of these conditions and the early commencement of emergency measures and management may determine the outcome for the mother or the fetus. The nurse/midwife should remain calm and attempt to keep the woman and her partner fully informed to obtain her consent and cooperation for procedures that may be required.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Discuss cord prolapse and its management
- Describe maternal and foetal distress
- Discuss the management of maternal and foetal distress
- Discuss the causes and management of obstetric shock
- Discuss shoulder dystocia

3.0 MAIN CONTENTS

3.1 Cord prolapse

A cord presentation is defined as the presence of umbilical cord below the fetal presenting part when the membranes are intact. Occult presentation means that the cord is lying alongside the presenting part but will not be palpable on vaginal examination. It is a particularly dangerous condition and may be a cause of unexpected fetal distress. Cord prolapse is the presence of the cord below the presenting part when the membranes are ruptured. Once the cord is out of the uterus, and especially when out of the vagina, the

fetal blood supply is obstructed, either because of the drop in temperature, and spasm of the vessels, or compression between the pelvic brim and the presenting part. If delivery is not expedited, fetal death is likely.

Incidence

It has an incidence of 1:500 deliveries and occurs when the fetal presenting part does not fit well into the maternal pelvis, giving 'space' for the cord to prolapse when the membranes rupture.

Risk factors for cord prolapse

Maternal causes:

- Pelvic tumours such as fibroids in the lower segment
- Narrow pelvis
- Multiparity

Fetal causes:

- Prematurity
- Malpresentation, e.g. breech and transverse lie
- Multiple pregnancy
- Polyhydramnios
- Placenta praevia
- Large baby

Diagnosis of cord prolapse

- Cord will be felt pulsating during vaginal examination with the membranes intact.
- The presence of prolapse may not be recognised until cord appears at the vulva
- Cord may be palpated on vaginal examination done to assess the progress of the labour or because of the sudden onset of acute fetal distress. It is essential to make a vaginal examination as soon as the membranes rupture in all patients who display an ill-fitting or non-engaged presenting part.

- Abnormal fetal heart rate pattern discovered during auscultation may suggest cord prolapse, as compression of the umbilical vein between the presenting part and the pelvis, reduces or stops the flow of oxygenated blood to the fetus, causing deep variable decelerations, then bradycardia if the situation is not relieved.

Management of cord prolapse

1. Determine the presence or absence of cord pulsation and fetal heart sounds. If the fetus is dead the labour may be left to proceed normally (if no other complication is present).
2. If the fetus is still alive, the caesarean section must be carried out as soon as possible unless vaginal delivery by forceps or breech extraction is likely to be straightforward.
3. While arrangements are being made for operation, the cord should be pushed back into the vagina and kept up with a gauze pack or by hand.
4. An attempt is made to prevent compression of the cord between the presenting part and the pelvis by getting the mother to adopt a suitable position. The foot of the bed should be raised.
5. Handling of the cord should be minimised as far as possible.
6. Prolapse of the cord, although potentially fatal for the child, carries little risk for the mother unless proper precautions are neglected for the sake of saving time. However great the need for haste, the mother must be properly prepared for operation and appropriate blood products available.
7. Presentation of the cord, when discovered by vaginal examination is an indication for caesarian section but, as the membranes are intact, there is no immediate danger for the fetus, and more time is available.

Prognosis

Not good as it carries 50% mortality

3.2 Maternal and Fetal Distress

3.2.1 Maternal distress

Maternal distress is referring to maternal exhaustion and it associated with complications associated with abnormal labour such as prolonged labour, Starvation and Prolonged

dehydration. Maternal distress should be anticipated in trial of labour, induction of labour, malposition and malpresentation

Signs and symptoms

1. Increase pulse rate (90 -120) or more.
2. Rise in temperature (37.20C) or more).
3. Increase Respiration (24 beats) or more.
4. Signs of dehydration – furred tongue, dry skin, presence of acetone in breath and urine.
5. Distension of the bowel with gas.
6. Vomiting may occur.
7. Restlessness, weakness, sweating. Patient looks ill, worried & anxious.

Management:

1. Adequate rest, sedation, hydration and avoidance of prolonged labour are preventive measures against maternal distress.
2. Administer 20mls 50% dextrose, followed by 10% Dextrose intravenously to correct dehydration & ketosis.
3. Termination of labour through Caesarean Section if in 1st stage. Episiotomy in second stage of labour.

3.2.2 Fetal Distress

Fetal distress is referring to fetal hypoxia in-utero, and it occurs when there is interference with the supply of oxygen to the fetus. Conditions that can predispose to fetal Distress are:

1. Maternal conditions: - Pre-eclampsia, Eclampsia, severe hypertension, chronic nephritis, chronic pyelonephritis, Diabetes: These conditions may lead to placenta insufficiency.
2. Severe Anaemia in pregnancy.
3. Abnormal uterine Actions e.g. hypertonic type
4. Prolonged labour.
5. APH due to premature separation of placenta.
6. Prolapse of the cord or presentation which compression.

7. True knots in the umbilical cord.
8. Prematurity
9. Post maturity – degeneration of the placenta
10. Congenital fetal abnormalities.

Diagnosis:

1. Tachycardia: Increase of 20 beats/minute in foetal heart rate is an early sign of foetal distress. A rate of over 160 beats/minute or more should cause concern.
2. Bradycardia: decrease of 20 beats/minute fetal heart rate is a significant sign of foetal distress. The heart rate may become progressively slow until a rate of 100 beats/minute or slower. Heart rate of 80 beats/minute or below may result in foetal death
3. Irregular heart rate follow the slow heart rate
4. Passage of meconium – cephalic presentation
5. Fetal blood sampling.

Management

Prophylaxis:

1. Good screening of all pregnant women.
2. Complicated case should have Hospital birth
3. All women with high head should be on bed.
4. Frequent observation of foetal heart in susceptible cases.
 - a. Inform doctor and inform the woman to lie on one side.
 - b. Stop oxytocic drug if any.
 - c. Give oxygen therapy to the mother.
 - d. Immediate delivery – (Cesarean section, Episiotomy, Forceps)
 - e. Notify Paediatrician.
 - f. Get resuscitation tray ready.

Complication of Fetal Distress

1. Asphyxia

2. Stillbirth

3. Mental retardation

3.3 Prolonged labour

Prolonged labour means poor progress in labour. During the first stage of labour, cervical dilatation of less than 2cm in 4 hours (primigravida) and cervical dilatation of less than 2cm in 4 hours (multigravida) or a slowing in progress are termed prolonged labour. A delay in delivery of > 2h (primip) or > 1 h (multip) from active pushing during the second stage is also a prolonged labour. Prolonged labour during third stage is failure to deliver the placenta after > 30 min (active management), > 1 h (physiological).

Causes of prolonged labour

First/second stage

- ‘Powers’: Frequency and duration of contractions.
- ‘Passenger’: Fetal size, malpresentation, malpositioning.
- ‘Passages’: Cephalo pelvic disproportion (CPD).

Third stage

Failure of the placenta to detach from the placental bed.

Risk Factors

First/second stage

- See the above causes. Also: primigravidity,
- Maternal age
- Big baby
- Short stature
- Obesity
- Induction of labour,
- Epidural (delay in second stage)
- Cervical surgery
- Pelvic trauma
- Fetal malformations.

Third stage

- Previous retained placenta
- Previous injury to the uterus
- Pre-term delivery
- Induction of labour,
- Multiparity

History: Assess risk factors as above, review partogram.

Examination

First/second stage

General examination: Maternal exhaustion, dehydration, blood-stained urine (sign of obstructed labour).

Abdomen Examination: Fetal size, lie, presentation, engagement.

Vaginal examination: Cervical dilatation, station, presentation, position, presence of membranes, signs of obstruction (caput/moulding), assess pelvic capacity.

Third stage Failure of placental delivery with controlled cord traction.

Investigations Bloods: FBC, G&S (preparation for delivery/theatre).

CTG Fetal wellbeing (first/second stage).

Management

First stage

Fluid rehydration, adequate pain relief, ARM if appropriate, augmentation of labour with oxytocin infusion (caution in multiples). Caesarean section for malpresentation. Consider FBS/ delivery if signs of fetal distress.

Second stage

Consider oxytocin. If fully dilated, < 1/5 palpable abdominally and vertex at/below spines: instrumental delivery. Otherwise Caesarean section.

Third stage

Administer oxytocin, if still retained after 30 minutes needs manual removal of placenta (often in theatre).

Complications

First/second stage

Maternal: Risks of instrumental delivery or Caesarean section, PPH, uterine rupture, fistula formation (rare in UK).

Foetal: Fetal distress, complications of instrumental delivery, " risk shoulder dystocia.

Third stage PPH, infection.

Prognosis

First/second stage While many cases respond to intervention, prolonged labour still a

3.4 Obstetric Shock

Shock is collapse which is mostly due to circulatory failure. Shock in obstetric does not differ from surgical shock.

Causes:

In most cases, shock in obstetrics are associated with

1. Haemorrhage (especially carried by Trauma)
2. Prolonged or severe anaesthesia
3. Severe pains associated with manual removal of placenta. a. Difficult labour, forceful dilatation of the cervix, difficult instrumental delivery, internal version, Rupture of uterus.

Inversion of the uterus. Concealed accidental haemorrhage, Pulmonary embolism.

4. Amniotic fluid embolism – Intravascular coagulation.
5. Reaction to blood transfusion of incompatible blood.
6. Severe infection (clostridia or gm-ve enteric bacteria).
7. Very rarely – Air embolism.
8. It may be purely neurogenic and due to fear.
9. Sudden reduction in intra-abdominal pressure following the delivery of twins.

In most cases, shock is caused by more than one factor – hemorrhage and trauma and prolonged anaesthesia.

Signs and symptoms

1. Rapid and thready pulse - 90 beats & above
2. B/P of below 90 systolic call for alarm

3. Increased pallor of the skin, Cold sweat, cyanosis, Subnormal temperature
4. Deep and sighing respiration. Restlessness, Patient may complain of thirst or faintness. May lose consciousness.

Management:

1. Call Doctor at the first sign of rising pulse rate.
2. Urgent resuscitative treatment
3. Principle of treatment.
 1. The administration of fluids – collapse is due to circulatory failure so increase blood volume – using ABO group and Rhesus type. Plasma may be used. Saline or Dextrose may be set up temporarily.
 2. Raise foot of bed – to maintain circulation to the vital organs.
 3. Oxygen by mask at the rate of 1 –2 litres/minute
 4. Rest – Morphine to relief pain.
 5. Keep in a quiet and undisturbed as possible.
 6. Cortisone or adrenaline are sometimes affected in adrenal failure but not in other cases as it may cause severe vasoconstriction and decrease venous return further.
 7. Avoid warm – cold skin constricts the arterioles in the skin directing the little blood to the heart and brain. Warming the skin may contradict this compensatory mechanism.
 8. Stimulant such as coramine (2mls) may be given intramuscularly.

4.0 CONCLUSION

Recognition of any deviation from normal labour and the early commencement of emergency measures and management may determine the outcome for the mother or the fetus.

5.0 SUMMARY

In this unit you have learnt:

- Description of cord presentation and cord prolapse
- Incidence and risk factors for cord prolapse
- Diagnosis and management of cord prolapse
- Description and management of maternal and foetal distress

- Prolong labour and its management
- Obstetric shock and its management

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Explain the management of obstetric shock

Discuss the management of maternal and foetal distress

7.0 REFERENCES AND OTHER READING MATERIALS

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Unit 2 Malposition and malpresentation

CONTENTS

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
 - 3.1 Occipitoposterior position
 - 3.2 Face presentation
 - 3.3 Brow presentation
 - 3.4 shoulder presentation
 - 3.5 Unstable lie
 - 3.6 Compound presentation
 - 3.7 Breech presentation
- 4.0 Conclusion
- 5.0 Summary
- 6.0 Online discussion and assignment
- 7.0 References/further reading

1.0 INTRODUCTION

Progress in labour depend on the three variables: the powers, that is, efficiency of uterine contraction, the passenger: that is, the fetus with respect to its size, presentation and positions and the passages: that is uterus, cervix and the bony pelvis. Abnormalities in one or more of these factors can slow down the progress of labour. In this unit, we are going to examine these abnormal presentations and their effect on the course of labour.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Outline the types of abnormal presentation and positions.
- Identify the causes of abnormal presentation and positions.
- Make accurate diagnosis of abnormal presentation and positions.
- Manage the conditions effectively.
- Describe the possible outcomes.

3.0 MAIN CONTENTS

3.1 Occipitoposterior positions

Occipito posterior positions (OPP) are the most common type of malposition of the occiput and occur in approximately 10–30% of labours but only around 5% of birth. Women can be reassured that internal rotation to anterior positions can be expected in the majority of cases. It can either be right occipito-posterior position (long rotation) or persistent OPP (the occiput points to either right or left sacro-iliac joint) results from a failure of internal rotation or malrotation prior to birth. In OPP the vertex is presenting, but the occiput lies in the posterior rather than the anterior part of the pelvis. Therefore, the fetal head is deflexed and larger diameters of the fetal skull present at the maternal pelvis.

Causes of OPP

The direct cause is often unknown, but it may be associated with an abnormally shaped pelvis. Such as android pelvis in which the forepelvis is narrow and the occiput tends to occupy the roomier hindpelvis. The oval shape of the anthropoid pelvis, with its narrow transverse diameter, favours a direct OPP position.

Diagnosis of Right Occipito-Posterior antenatally

Abdominal Examination

On inspection, there is a saucer-shaped depression at or immediately below the umbilicus, the high head with the depression above it looks rather like a full bladder.

On palpation: the head is high, feels unduly large; this is due to the larger circumference of the deflexed head. The occiput and sinciput are on the same level. The back is difficult to palpate because it is placed well out on the right side. Limbs are felt on both sides of the midline.

On auscultation: the fetal heartbeat will be located in the right flank, somewhat muffled as the muscles there are thick. It may also be heard in the midline near the umbilicus or slightly to the left.

Diagnosis during labour

Posterior position should be suspected where there is no disproportion and a vertex presentation is held up at the brim in spite of good uterine action.

On vaginal examination: locating the anterior fontanelle to the left anterior is diagnostic of an R.O.P. The sagittal suture will be in the right oblique diameter of the pelvis... The large caput may make identification of sutures and fontanelles difficult

Outcome of Labour

Long internal rotation of the head commonly takes place and the baby is born normally.

Short internal rotation of the head takes place and the baby is born face to pubes.

Deep transverse arrest of the head occurs in the pelvis which has projecting ischial spines that inhibit forward rotation of the head.

Labour may be prolonged because larger diameters of the skull present, the deflexed head does not dilate the cervix effectively.

The necessity for interference is greater. Epidural analgesia may be used for backache.

Rotation of the head may have to be assisted manually or by forceps; application of forceps is frequently required because of delay in the second stage, or on account of fetal or maternal distress.

The fetal mortality and morbidity rates are higher because of intracranial injury and hypoxia.

Summary of Clinical Features

The head descends slowly, even when there are good contractions.

The uterine contractions are sometimes weak.

Dilatation of the cervix is retarded.

The membranes usually rupture early.

Backache

Difficulty in micturition is common.

The urge to bear down at the end of the first stage is especially great, probably because the occiput is pressing on the rectum.

Nursing care

Although only 10 per cent of these patients will have a prolonged or difficult labour, such a possibility should be anticipated in every case so that further complications can be averted. Additional nursing care, including observation of the maternal and fetal conditions

3.2 Persistent occipito posterior position short-rotation

The occiput point to the sacro-iliac joint, left or right. In this condition, the occiput fails to rotate forwards. Instead, the sinciput takes the lead reaching the pelvic floor first and rotate forwards. The occiput goes into the hollow of the sacrum and the baby is born facing the pubic bone – face to pubis

Diagnosis

Head is slow to engage

Fetal heart sound is heard in the flank or midline above the umbilicus

Delayed second stage

Large caput succedaneum

The pinna of the ear is pointing to the maternal sacrum, is indicative of posterior position

Excessive bulging of the anus and the perineum due to the biparietal diameter descending the perineum instead of the bi-tempora

At birth, the sinciput appear first under the symphysis pubis

Management

You should allow the sinciput to engage as far as the root of the nose, and then maintain flexion by restraining it from escaping.

Allow the occiput to sweep the perineum and be born. Then grasp the head and extend it and bring the head down under the symphysis pubis because of the large diameter it may be necessary you give episiotomy.

Complication

Third or fourth degree tear

Intracranial haemorrhage

Excessive moulding

Obstructed labour

Cerebral haemorrhage and chronic hypoxia which may accompany prolonged labour

3.3 Face presentation

When the attitude of the head is one of complete extension, the occiput of the fetus will be in contact with its spine and the face will present. The incidence is about $\leq 1: 500$ and the majority develop during labour from vertex presentations with the occiput posterior;

this is termed secondary face presentation. Less commonly, the face presents before labour; this is termed primary face presentation. There are six positions in a face presentation and the denominator is the mentum. The positions are:

Right mentoposterior

Left mentoposterior

Right mentolateral

Left mentolateral.

Right mentoanterior

Left mentoanterior

Causes

Pendulous abdomen

Tumour of the fetal neck(rare)

Anencephaly

Polyhydramnios

Occipito posterior position

Contracted pelvis

Anterior obliquity of the uterus

Diagnosis

Antenatal diagnosis is rare since face presentation develops during labour in the majority of cases. A cephalic presentation in a known anencephalic fetus may be presumed to be a face presentation.

During labour

Abdominal palpation

The occiput feels prominent, with a groove between the head and back

The limbs may be palpated on the side opposite to the occiput and the fetal heart is best heard through the fetal chest on the same side as the limbs.

In a mentoposterior position, the fetal heart is difficult to hear because the fetal chest is in contact with the maternal spine

Vaginal examination

On vaginal examination, the chin orbital ridges, malar bone, bridge of the nose, eyes may be felt

Possible course and outcomes of labour

Prolong labour

With good uterine contractions, descent and rotation of the head occur and labour progresses to a spontaneous birth in **Mentoanterior positions**

In **Mentoposterior positions**, if the head is completely extended so that the mentum reaches the pelvic floor first, and the contractions are effective, the mentum will rotate forwards and the position becomes anterior.

Persistent mentoposterior position: In this case, the mechanism of labour is not possible due to large diameter of 13.8 descending the perineum. usually caesarean section is the mode of delivery.

Reversal of face presentation: A face presentation in a persistent mentoposterior position may, in some cases, be manipulated to an occipitoanterior position using bimanual pressure. This method was developed to reduce the likelihood of an operative birth for those women who refused caesarean section. A tocolytic drug, such as terbutaline is used to relax the uterus, the fetal head is disengaged using upward

transvaginal pressure. The fetal head is then flexed with bimanual pressure under ultrasound guidance to achieve an occipitoanterior

Complication

Obstructed labour

Cord prolapse

Facial bruising

Cerebral haemorrhage

Excessive perineal laceration

Increase maternal morbidity

3.4 Brow presentation

In the brow presentation, the fetal head is partially extended with the frontal bone, which is bounded by the anterior fontanelle and the orbital ridges, lying at the pelvic brim. This presentation is rare, with an incidence of approximately 1 in 1000 births.

Causes

These are the same as for a secondary face presentation

Diagnosis

Brow presentation is not usually detected before the onset of labour.

- A depression is felt between the fetal head and the back
- Presentation part is high
- Head is unduly large
- Cephalopelvic disproportion (CPD) may be present. The presenting part is high and may be difficult to reach.
- The anterior fontanelle may be felt on one side of the maternal pelvis and the orbital ridges
- Baby has large caput succedaneum

Management

The doctor must be informed immediately this presentation is suspected. This is because vaginal birth is extremely rare and obstructed labour usually results. Although, it is possible that a woman with a large pelvis and a small baby may give birth vaginally. However, there may not be a favourable outcome.

Complications

These are the same as in a face presentation, except that obstructed labour requiring caesarean section is the probable rather than a possible outcome.

3.5 Shoulder presentation/transverse lie

The term is applied when the fetus lies with its long axis across that of the mother. In transverse lie, the shoulder usually presents. This is a very serious complication in obstetrics and the ratio is 1:300 cases near term. The incidence is greater in multiparae than in primigravidae. The breech is usually slightly higher on one side than on the other side. Back may be in anterior or posterior.

Causes

Maternal:

1. Contracted pelvis – prevent engagement (rare)
2. Tumours – Fibroids (rare)
3. Grande multiparity – lax uterine and abdomen muscles
4. Abnormal uterus – Bicornuate\ subseptate uterus

Fetal:

1. Multiple pregnancy – Twins.
2. Prematurity – Large amniotic volume & small fetus
3. Macerated fetus.
4. Placenta praevia – Hydrocephalus & gross abnormality
5. Polyhydramnios, Anterior obliquity of the uterus

Positions

1. Dorsal Anterior – the fetus lies with the back to the front of the mother. Head could be to the left or right.
2. Dorsal Posterior – the fetus lies with the back to the back of the mother.

Diagnosis

During pregnancy

On inspection – Abdomen looks broad fundal height is lower than normal

On Palpation

1. No presentation part either at the pelvis or in the fundus.
2. Fundal height lower than gestational age.
3. Head is felt on one side and the breech at the other side

On Auscultation

Fetal heart sound is head below the umbilicus

Ultrasound: May be used to confirm the lie & presentation.

In Labour

When membranes rupture the uterus appears more irregular, the uterus mould round the fetus making palpation to be difficult – shoulder may be wedged into the pelvic brim.

There may be prolapse of the arm, foot, cord or both foot and arm.

On Vaginal Examinations

Presenting part is very high – cord may prolapse – soft irregular mass may be felt.

Ribs may be felt, arm prolapse may occur. Spontaneous delivery becomes impossible except in macerated fetus when it is born doubled – up.

No mechanism for shoulder presentation. Placenta praevia must first be excluded before performing vaginal examination.

Management

In pregnancy:

Adequate prenatal care to diagnose the case antenatally. Causes should be investigated by Doctor. The position can be rectified or appropriate management is arranged prior to labour e.g. in case of contracted pelvis and placenta praevia elective C/S is done at term. If no contraindication external cephalic version is done at 34th week to longitudinal lie.

In Labour:

Inform Doctor immediately. In early labour and membranes intact external cephalic version could be done, followed by immediate rupture of membranes and close observation to ensure a longitudinal lie. In late labour with ruptured membranes, internal cephalic version is performed under general anaesthesia and the baby delivered by breech extraction. Caesarean section is the method of choice in cases of:

1. Failed external cephalic version.
2. When membranes are gone
3. Cord prolapse
4. Prolonged labour.

Immediate C/S is done whether fetus is alive or dead.

Danger

1. Infection.
2. Stillbirth
3. Ruptured uterus
4. Obstructed labour
5. Cord prolapse and arm prolapse.
6. Early rupture of membranes leading to oedematous vulva

3.6 Unstable lie

The lie is defined as unstable when after 36 weeks' gestation, instead of remaining longitudinal, it varies from one examination to another between longitudinal and oblique or transverse.

Causes

- Any condition in late pregnancy that increases the mobility of the fetus e.g. Polyhydramnios and Laxed uterine muscle as in grande multiparity
- Any condition that prevents the fetal head from entering the pelvic brim such as contracted pelvis, placenta praevia

Management

1. Admit the woman to the hospital at 37th – 38th week of pregnancy till she delivers to avoid

- a. Unsupervised onset of labour with transverse lie.
 - b. To receive essential and expert supervision – investigation to detect the cause – rule out placenta praevia.
2. Further attempts are made to correct the lie by external cephalic version.
 3. At the 38th week or when labour starts membranes may be ruptured after making the lie longitudinal bearing in mind the risk of cord prolapse.
 4. Intravenous oxytocin drip is set up taking appropriate precaution, especially in multiparous patients.
 5. Vigilant supervision is important in labour to see that the longitudinal lie is maintained throughout labour by thorough abdominal examination at the onset of labour and at frequent intervals. Fetal heart sound should be checked frequently for possible cord prolapse.
 6. The bladder should be emptied 2hrly to aid descent of the presenting part.
 7. Bowels should be emptied so as to facilitate and preserve longitudinal lie.
 8. If the correction of the lie fails at term caesarean section is done. Labour is considered trial.

Complication

- Same as transverse lie if labour commences in any lie other than longitudinal.

3.7 Compound Presentation

When a hand or foot lies alongside the head the presentation is said to be compound.

Common with small pelvis or roomy pelvis – it maybe head, hand, foot

Causes

1. Small fetus
2. Very roomy pelvis

Diagnosis

1. Compound presentation diagnosis is usually made on vaginal examination or
2. Seen at the vulva during labour.

Management

First Stage:

Seek medical aid.

An attempt could be made to push the arm upwards over the baby's face.

Second Stage

Midwife should hold the hand back pushing it over the baby's face.

Occasionally caesarean section may be necessary where there is average pelvis and average size baby.

3.8 Breech presentation

Breech presentation is a malpresentation that occurs in about 2.5% of deliveries at term and more frequently in the early third and second trimesters. In addition to prematurity, other conditions associated with breech presentation include multiple pregnancy, polyhydramnios, hydrocephaly, anencephaly, aneuploidy, uterine anomalies, and uterine tumors.

Positions of Breech

1. Left Sacro-Anterior; LSA
2. Right Sacro-Anterior; RSA
3. Left Sacro-Posterior; LSP
4. Right Sacro-Posterior; RSP
5. Left Sacro-Lateral; LSL
6. Right Sacro-Lateral; RSL

Types of breech presentation

Complete or full breech: In this type, the fetus lies in attitude of complete flexion, the thighs and legs are both flexed and the head is well flexed on the chest. The presenting part is therefore bulky consisting of buttocks external genitalia and both feet.

Franck Breech/extended Breech: In this type, the breech presents with the thighs flexed and the legs extended on the fetal abdomen - common type (70%)

Footling Breech: In this type, both legs or one leg is fully extended but partially extended at the hip so that the foot lies lowest in the birth canal. It is rare (25%) type of breech presentation

4. **Knee presentation:** The knee is flexed while the thigh is extended at the hip. In

frank Breech, the buttocks fit the cervix accurately and cord prolapse is uncommon but may not in other types. The hands may be extended above the head during labour

Causes

1. mostly unknown
2. conditions that favour breech presentation include:
 - a. contracted (brim) pelvis
 - b. placenta praevia
 - c. pelvic tumors – fibroids
 - d. grand multiparity, polyhydramnios
 - e. abnormal uterus – bicornate uterus

Fetal causes

1. prematurity
2. fetal abnormalities – anencephaly and hydrocephaly
3. multiple pregnancy –twins
4. intrauterine death.

Diagnosis

Palpation

1. Longitudinal lie.
2. Firm lower pole.
3. Limbs to one side.
4. Hardhead at fundus. (Head may not be palpable at fundus because it is under the ribs — always confirm by pelvic examination or ultrasound scan.)

Auscultation: The fetal heart (FH) is best heard above the umbilicus.

Vaginal examination:

- No head in the pelvis. Soft buttocks felt and hard irregular sacrum. Feet may be in pelvis as leading part.
- Ultrasound Differentiation of head and breech is not always easy but the head will be readily detected by a scan.

Dangers

(a) **Antenatal**

As with other malpresentation, there is an increased risk of premature rupture of the membranes and cord prolapse. This applies least to the extended breech which is a well-fitting presenting part.

Delivery

- The main danger in breech delivery is the speed with which the head descends through the pelvis. Rapid compression and decompression can cause intracranial injury.
- Conversely, undue delay in the delivery will lead to asphyxia due to cord compression, at least from the time of delivery of the shoulders.
- Traumatic injuries may occur if intervention in the delivery is required.
 - Erb's Palsy – due to damage to the brachial plexus
 - Fracture of Humerus – extended arm
 - Fracture of Femur – extended legs
- Rupture of the liver and internal organs due to increase
- The placenta separates frequently in the second stage of labour as the active uterus contracts and the fetal head is in the pelvis. Apnoea is, therefore, a danger. Manual assistance to complete delivery of the baby is essential and may be a sudden need. Episiotomy is desirable to permit sudden interference, or complete perineal tear may result.
- A large multicentre study of breech delivery at term concluded that caesarean section was the safest route of delivery for the newborn.
- In this study perinatal mortality, neonatal mortality, or serious neonatal morbidity was significantly lower for the planned caesarean section group than for the planned vaginal birth group.
- Although there was no significant difference in early morbidity between vaginal delivery and section for the mothers, there was a reduced incidence of urinary incontinence for mothers delivered abdominally at three months postnatal.

Management of Breech Presentation in Labor

- Immediately a woman is admitted in labour with breech, the doctor should be informed; all cases of breech should be delivered in the hospital.

Antenatal

As already noted, many babies present by the breech at 30 weeks. Most undergo spontaneous version by 32–34 weeks. If this does not occur the possible causes should be considered and an ultrasound scan carried out for localisation of the placenta and to exclude major fetal abnormality.

External cephalic version (ECV) in which the breech is elevated from the pelvis may be undertaken at any time from 34 weeks gestation. Technically this is easier earlier in pregnancy but spontaneous version is likely before this stage.

Technique for ECV

The mother rests supine with the upper body slightly tilted down. The presentation and placental position are confirmed by ultrasound. The fetal heart rate is checked preferably with a small strip of CTG. A tocolytic agent (oral nifedipine or IM terbutaline) is given to relax the uterus as this improves the success rate. The breech is disimpacted from the pelvic brim and shifted to the lower abdomen and the fetus is gently rotated, keeping the head flexed. The fetal heart rate should be checked during the procedure. It is essential not to use excessive force and, if there is evidence of fetal bradycardia, the fetus should be returned to the original presentation if the version is not past the halfway point and the fetus monitored with continuous CTG.

Complications of ECV

1. The placenta may be partly separated. The fetal heart must be checked on completion and the vagina examined for bleeding.
2. There may be unsuspected complications such as an abnormal uterus or short umbilical cord.
3. Excessive force may rupture the uterus.

Contra-indications of ECV

1. Pre-eclampsia (as predisposing to placental bleeding).

2. Previous scar on the uterus.
3. Multiple pregnancy or fetal abnormality.

The commonest causes of failure are too large a fetus or too little liquor, or ‘splinting’ of the fetus by extended legs. Version should not be performed in pregnancies complicated by twins, hypertension and when caesarean section is already planned.

Complications that may arise :

Extended legs. This can be delivered using the Pinead’s method.

Extended hands. This can be delivered using the Lovset’s Maneuver.

Extended head. This can be delivered using the Mauriceau-Smellie Veit’s Method

4.0 CONCLUSION

When occiput does point to any other landmark on the pelvic brim other than the anterior, the presentation and position are said to be mal. Mal position and presentations lead to delayed progress of labour. Both the mother and the baby are vulnerable to complications which may endanger their lives. Since the diagnosis may not be made until the woman comes in labour, the midwife must be vigilant and accurate in her observation of the woman in labour so that prompt intervention can help to circumvent most of the complications accompanying these conditions and the successful outcome for the mother and baby are ensured.

5.0 SUMMARY

In this unit you have learnt:

- The features of the malpresentation and malpositions
- Recognize the predisposing factors to malpresentation and malpositions
- Possible causes of these positions and presentations
- The physical landmarks to aid recognition and diagnosis of malpresentation and malpositions
- The management and the current uncertainties of malpresentation and malpositions.

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Identify one case of malpresentation and malpositions in your hospital and discuss the management and outcome of the case

7.0 REFERENCES AND OTHER READING MATERIALS

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Module 3 Complication associated with puerperium and postpartum

Puerperium refers to the 6-week period following completion of the third stage of labour, when considerable adjustments occur before return to the pre-pregnant state. During this period of physiological change, the mother is also vulnerable to psychological disturbances, which may be aggravated by adverse social circumstances.

Module Objectives

At the end of this module you should be able to:

- i. Describe the management of puerperal pyrexia and puerperal sepsis
- ii. Discuss postpartum haemorrhage
- iii. Discuss psychiatric illness presents in the puerperium.

Unit 1 Intrapartum complications

CONTENTS

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
 - 3.1 Puerperal pyrexia
 - 3.2 Puerperal sepsis
 - 3.3 Mastitis
- 4.0 Conclusion
- 5.0 Summary
- 6.0 Online discussion and assignment
- 7.0 References/further reading

1.0 INTRODUCTION

Most women continue to enjoy good health during the puerperium but infection or preexisting ill health may interfere with the patient wellbeing. In this unit, you will learn some of the problem's women encounter postnatally.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Identify the causes and management of puerperal pyrexia
- Outline the microorganism responsible for puerperal sepsis
- Discuss the pathophysiology of puerperal sepsis
- Describe mastitis and its management

3.0 MAIN CONTENTS

3.1 Puerperal pyrexia

Puerperal pyrexia is defined as a febrile condition occurring within 14-21 days of delivery. Puerperal pyrexia may be due to an infection, genital or extra-genital. Infection can occur during labour, especially if associated with prolonged rupture of the membranes. Vaginal examination in labour, even with proper care and aseptic precautions, can encourage the transfer of organisms from the vagina to uterine cavity. The most worrying organism, which can be found in the normal, healthy vagina, is the beta-haemolytic streptococcus.

Causes

1. Genital tract infection - perineum, vagina, cervix, uterus, adnexa.
2. Urinary tract infection
3. Incidental causes such as malaria, amoebic dysentery, typhoid and pneumonia.
4. Breast - breast engorgement, mastitis or breast abscess
5. Thrombotic condition such as superficial thrombophlebitis or deep vein thrombosis
6. Respiratory system — common cold, influenza, after general anaesthesia.
7. Pyrexia of unknown origin, in this case, no cause can be found for the rise in temperature

Investigation

- A complete physical examination and bacteriological examination of urine specimen, throat swab or sputum, high vaginal swab, cervical swab and in some cases blood culture.
- Blood film for malaria parasite and blood culture in case of septicaemia

Management

- A patient should be made to rest in bed. The temperature should be reduced by fanning and exposure or tepid sponging if the temperature is about 37.90C.
- Isolate or barrier nurse the patient while investigations are being carried out, the patient should be thoroughly examined for evidence of infection especially in the breast, chest, throat and the genital tract.
- The type of labour and mode of delivery of the patient are also reviewed.
- In Nigeria and other countries where malaria is endemic, antimalaria drugs such as chloroquine 800mg stat then 400mg twice daily for 3 days are given in conjunction with the specific treatment after a film have been sent to the laboratory of evidence of malaria parasites.
- Nursing care includes rest, observation and physical care

3.2 Puerperal sepsis

Puerperal sepsis is an infection of the genital tract during the first six weeks of delivery or abortion. Pyrexia in the puerperium may be due to puerperal sepsis or to extragenital causes such as pyelonephritis, mastitis and pneumonia. Puerperal sepsis still accounts for significant postpartum maternal morbidity and mortality. Patients with a puerperal genital tract infection are susceptible to the development of septic shock, pelvic thrombophlebitis, and pelvic abscess. Following a vaginal delivery, about 6% to 7% of women demonstrate febrile morbidity

Causes of puerperal sepsis

The infecting organism

The source of infection

The predisposing factors

Pathophysiology of puerperal sepsis

The pathophysiology of puerperal sepsis is closely related to the various microbial inhabitants of the vagina and cervix. The vaginal flora during gestation resembles the nonpregnant state, although there is a trend toward isolating more **Mycoplasma genitalis** and **anaerobic streptococci** in the last trimester. Potentially pathogenic organisms can be cultured from the vagina in about 80% of pregnant women. These organisms include **enterococci, hemolytic and nonhemolytic streptococci, anaerobic streptococci, enteric bacilli, pseudodiphtheria bacteria, and Neisseria species other than N. gonorrhoeae**. Excessive overgrowth of these organisms during pregnancy is inhibited by the acidity of the vagina (pH 4 to 5), primarily as a result of the production of lactic acid by the lactobacilli. The uterine cavity is normally free of bacteria during pregnancy. After parturition, the pH of the vagina changes from acidic to alkaline because of the neutralizing effect of the alkaline amniotic fluid, blood, and lochia, as well as the decreased population of lactobacilli. This change in pH favours an increased growth of aerobic organisms. About 48 hours postpartum, progressive necrosis of the endometrial and placental remnants produces a favourable intrauterine environment for the multiplication of anaerobic bacteria. About 70% of puerperal infections are caused by anaerobic organisms. Most of these are anaerobic cocci (*Peptostreptococcus*, *Peptococcus*, and *Streptococcus*), although mixed infections with *Bacteroides fragilis* are encountered in up to one-third of cases. Of the aerobic organisms, *Escherichia coli* is the most common pathogen, followed by enterococci. Puerperal infection from clostridia is rare.

After delivery, the placental site vessels are clotted off, and there is an exudation of lymph-like fluid along with massive numbers of neutrophils and other white cells to form the lochia. Vaginal microorganisms readily enter the uterine cavity and may become pathogenic at the placental site, depending on such variables as the size of the inoculum, the local pH, and the presence or absence of devitalized tissue. The latter may include tissue incorporated in the suture line of a caesarean incision. The normal body defence

mechanisms usually prevent any progressive infection, but a breakdown of these defences allows the bacteria to invade the myometrium. Further invasion into the lymphatics of the parametrium can cause lymphangitis, pelvic cellulitis, and the possibility of widespread infection from septic emboli. Endomyometritis is a potentially life-threatening condition. It commonly begins with retention of secundines (placental and amniochorionic membrane fragments) that block the normal lochia flow, allowing accumulation of intrauterine lochia, which in turn changes the local pH and acts as a culture medium for bacterial growth. Unless normal lochia flow is established, bacterial invasion progresses.

Factors Predisposing to the Development of Puerperal Genital Tract Infection

- Poor nutrition and hygiene
- Anaemia
- Premature rupture of the membranes (PROM)
- Prolonged rupture of the membranes
- Prolonged labour
- Frequent vaginal examinations during labour
- Caesarean delivery
- Forceps or vacuum delivery
- Cervical or vaginal lacerations
- Manual removal of the placenta
- Retained placental fragments or foetal membranes

Clinical features

- High temperature
- Increasing uterine tenderness on postpartum day 2 or 3.
- With the development of parametritis (pelvic cellulitis), the temperature elevation will be sustained, and signs of pelvic peritonitis may develop.

- Erratic temperature fluctuations and severe chills suggest bacteremia and dissemination of septic emboli, with the particular likelihood of spread to the lungs.
- Pelvic vein thrombophlebitis, usually on the right side of the pelvis. The clinical picture of pelvic thrombophlebitis is characterized by a persistent spiking fever for 7 to 10 days after delivery, despite antibiotic therapy.

Diagnosis

- Evaluation of a febrile postpartum patient should include a careful history and physical examination.
- Extrapelvic causes of fever, such as breast engorgement, mastitis, aspiration pneumonia, atelectasis, pyelonephritis, thrombophlebitis, or wound infection, should be excluded.
- Ultrasound scan may be helpful.
- Blood and urine culture
- Vagina and cervical swab

Management

- A febrile puerperal patient with cessation of lochia flow should undergo a pelvic examination and removal of any secundines that may be occluding the cervical os.
- The antibiotic treatment of puerperal infection usually follows two major principles:
 - First, early antibiotic treatment should be instituted to confine and then eliminate the infectious process.
 - Second, the antibiotics should provide anaerobic coverage because these organisms are involved in 70% of puerperal infections.

Antibiotics should be continued for at least 48 hours after the patient becomes afebrile. Anaerobic organisms especially require prolonged chemotherapy for elimination. Broad-spectrum antibiotics, such as ampicillin and the cephalosporins, are effective first-line drugs for mild and moderate cases of

puerperal infection. When the infection is moderate to severe, a penicillin-aminoglycoside combination has traditionally been used as first-line therapy. The major pelvic pathogen resistant to this combination is *Bacteroides fragilis*, which is usually sensitive to clindamycin. The use of clindamycin with either an aminoglycoside or ampicillin will provide the best first-line coverage.

- When pelvic thrombophlebitis or thromboembolism is suspected or clinically diagnosed, unfractionated heparin therapy should be instituted to increase the clotting time (Lee-White method) or activated prothrombin time to 2 to 3 times normal. Only 2 to 3 weeks of anticoagulant therapy are needed for uncomplicated pelvic thrombophlebitis. Patients with femoral thrombophlebitis require 4 to 6 weeks of heparin therapy followed by the administration of oral anticoagulants for a few months.
- If the patient does not respond to heparin therapy and the clinical course is one of unrelenting fever and pelvic tenderness, a diagnosis of pelvic abscess must be considered. Diagnosis is made by pelvic examination and confirmed by pelvic ultrasonography or computed tomography scan. The finding of a tender, pelvic parametrial mass suggests an abscess. Ultrasonography will confirm that the mass is fluid-filled rather than solid. The presence of a pelvic abscess requires surgical drainage.

3.3 Mastitis

Mastitis is an inflammatory condition of the breast which may or may not be accompanied by infection. Mastitis can be incorrectly diagnosed in the first few days but is usually caused by engorgement or milk stasis producing increased pressure in the alveoli due to nonremoval of milk. The pressure builds up, forcing the milk out into the surrounding tissues. Mastitis most commonly occurs in the second or third week postpartum.

Infective mastitis is caused by bacterial invasion, usually via a cracked nipple. Staphylococci or streptococci are the most common organisms and these act on the milk forced outside the alveoli into the surrounding cells.

Causes

- Most frequent is poor positioning and attachment, preventing adequate drainage of milk • Tiredness and stress
- Initial engorgement with a blocked duct and pressure from a tight-fitting bra.

Signs and symptoms

- ‘Flu-like symptoms’
- Pyrexia
- A reddened area appears around the infected breast or segment.
- If left untreated, an abscess may form.

Management

- Antibiotics
- If an abscess forms – drainage as appropriate
- The midwife’s skill of preventing stasis of milk by advice on positioning, important of emptying each breast, correct handling of the breasts and prevention of clothing restrictions will ensure good practice prevails, transmitting a positive public health message.

4.0 CONCLUSION

In the puerperium, infection may enter through one or more of these wounds: In the puerperium, the mother should be encouraged to be mobile as quickly as possible and to bath or use a bidet at least twice daily.

5.0 SUMMARY

You have learnt:

- Puerperal pyrexia – its causes and management
- Puerperal sepsis – its causes, pathophysiology, investigation and management
- Mastitis and its management

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Describe the pathophysiology of puerperal sepsis.

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Unit 2 primary and secondary postpartum haemorrhage

CONTENTS

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
 - 3.1 Postpartum haemorrhage
 - 3.2 Primary postpartum haemorrhage
 - 3.3 Secondary postpartum haemorrhage
- 4.0 Conclusion
- 5.0 Summary
- 6.0 Online discussion and assignment
- 7.0 References/further reading

1.0 INTRODUCTION

Postpartum haemorrhage is one of the complications of the third stage of labour. Third stage of labour is a delicate and dangerous period that require the full attention of the nurse/midwife and is not a period the patient should be left unattended.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Define postpartum haemorrhage
- Identify the types of postpartum haemorrhage
- Outline the causes of various types of postpartum haemorrhage
- Discuss the signs and symptoms of postpartum haemorrhage
- Describe the management of postpartum haemorrhage
- Discuss the precautionary measures to prevent or minimize postpartum haemorrhage

3.0 MAIN CONTENTS

3.1 Postpartum haemorrhage

Postpartum haemorrhage (PPH) is probably one of the most common obstetric emergencies and it is the third most common cause of death among women. It is considered to be minor if the blood loss is between 500 and 1000 mL and major if it is greater than 1000 mL. In practice, blood losses between 500 and 1000 mL are relatively common and can usually be tolerated well by the woman. Thus, it has been suggested that losses over 1000 mL should trigger emergency PPH protocols. However, it should be remembered that estimation of blood loss is notoriously inaccurate, and if a woman demonstrates evidence of cardiovascular compromise, such as tachycardia, or if there is continued bleeding, then protocols should be instituted even if estimated losses are less than 1000 mL. In common with other obstetric emergencies, PPH can often be predicted and preventative measures undertaken if significant risk factors are present.

Incidence of postpartum haemorrhage

Approximately 5% of all deliveries (4% after vaginal delivery, 6–8% after caesarean section delivery).

Types of postpartum haemorrhage

Secondary postpartum haemorrhage: this is loss of 500 mL blood occurring from the genital tract between 24 hours and 12 weeks post-delivery.

Primary Postpartum Haemorrhage

Primary postpartum haemorrhage: this is haemorrhage occurring during the third stage of labour and loss of 500 mL blood from the genital tract within 24 hours of delivery.

Causes of primary postpartum haemorrhage

Uterine Atony – the uterus, although empty, fails to contract and control bleeding from the placental site. This is the commonest and potentially most dangerous cause.

Predisposing Causes

- (a) Excessive uterine distension (twins, polyhydramnios, large baby)
- (b) Multiparity (fibrosis in uterine muscle)
- (c) Prolonged labour (uterine inertia)
- (d) Labour augmented with Syntocinon.
- (e) General anaesthesia.
- (f) Placenta praevia — lower segment does not contract well enough to stop bleeding.
- (g) Placental abruption — the Couvelaire uterus may not contract. In addition, a coagulation defect may develop and fibrin degradation products (FDPs) discourage uterine contraction.

Secondary Postpartum Haemorrhage means abnormal bleeding from the genital tract from 24 hours after delivery until the completion of the puerperium.

Causes of secondary postpartum haemorrhage

1. Retained placental tissue - this inevitably leads to infection.
2. Intra-uterine infection with or without retained products.

3. Slow involution of the uterus or inadequate drainage of the lochia sometimes lead to fresh bleeding later than expected.

Consequences of postpartum haemorrhage

1. Bleeding may be very rapid, causing circulatory collapse and leading to shock and death.
2. Puerperal anaemia and morbidity.
3. (Very rarely) damage to the pituitary blood supply, leading to pituitary necrosis — Sheehan's syndrome.
4. Fear of further pregnancies. Haemorrhage is terrifying for the mother.

Management of postpartum haemorrhage

1. Measurement of blood loss: Blood spilt on bed linen and dressings is often ignored and only blood actually collected in a bowl is measured. The estimated loss is therefore invariably lower than the actual loss. The mother's response will be governed by her haemoglobin level.
2. Use of oxytocic drugs - Two are used: ergometrine 0.5 mg and oxytocin 5 units. Syntometrine is a proprietary combination of both these drugs. Ergometrine produces tonic contractions of the uterus and is also a vasoconstrictor. It may, therefore, cause elevation of the blood pressure especially if given intravenously. Its action affects the uterus for 2–3 hours. Synthetic oxytocin produces rhythmic contractions of the uterus. It is virtually free from systemic effects in therapeutic dosage and its action lasts for 20–30 minutes. In an emergency either can be given intravenously with almost immediate effect.
3. Plan of treatment - The aim is to stop the patient bleeding.
 - (a) Give an oxytocic intravenously (as above).

- (b) Rub up a contraction of the uterus to control bleeding and if the placenta is undelivered attempt removal by cord traction.
- (c) Rapid assessment of the mother's condition; set up an IV line and send blood for cross-match.
- (d) Treat the cause

- (i) If the placenta has been delivered, check for completeness. If in doubt, exploration of the uterus must be carried out.

- (ii) If the uterus appears well-contracted and bleeding continues, damage to the cervix or vagina should be suspected. Proper assessment of this will require exploration under anaesthesia. If both these causes have been excluded, uterine atony is diagnosed.

Treatment of Uterine Atony

1. Administer Prostaglandins - If the uterus continues to fail to contract in spite of the above measures, the next step is to employ the prostaglandin carboprost (Hemabate). It is given by intramuscular injection in a dosage of 250 micrograms and this may be repeated.
2. Bimanual compression of the uterus Having excluded an incomplete placenta and trauma to the genital tract by thorough exploration, the uterus is compressed between the hands to control bleeding and stimulate contraction. The fingers of one hand are pressed into the anterior fornix.
3. Uterine packing Occasionally it may still be necessary to resort to packing the uterus firmly with gauze. The packing usually remains in position for at least 12 hours. If contraction is still not obtained, hysterectomy must be carried out. By this time the patient is likely to be in a serious condition and a decision to operate, difficult as it is, must not be made too late. In cases of persistent bleeding, the presence of a clotting defect should be excluded.

Management of secondary postpartum haemorrhage

1. Ultrasound scan to detect retained products (see below).
2. Antibiotics (broad-spectrum + anti-anaerobe, e.g. metronidazole).
3. Evacuation of the uterus if products of conception are seen. This can be a treacherous condition and bleeding sometimes persists after evacuation. Occasionally packing the uterus and even hysterectomy are required.

Summary of management of postpartum haemorrhage

General Measures:

- Evaluate excessive bleeding immediately
- Assess overall patient status
- Notify other members of obstetrics team (i.e., obtain help!)
- Review clinical course for probable cause
 - Any difficulty removing placenta?
 - Were forceps used?
 - Other predisposing factors?
- Have operating room and personnel on standby
- Monitor and maintain circulation
 - Establish IV access: 2 large bore
 - Type and cross-match blood
 - Begin/increase crystalloid infusion
 - Assess for clotting or check coagulation profile

Evaluation: Perform in Rapid Succession

- Assess hemodynamic status
- Bimanual examination: assess for atony
 - May palpate for retained placental fragments
 - May palpate uterine wall for rupture
- Inspect perineum, vulva, vagina, and cervix
 - Identify lacerations, hematomas, inversions

- Recruit assistance for exposure
- You or assistant may re-inspect placenta
- Assess clotting

Targeted Interventions

Atony

- Immediate bimanual massage
- Administer uterotonics (with requisite precautions)
 - Oxytocin—IV: 10–40 units/1 L normal saline or lactated Ringer solution, continuous
 - Methylergonovine—IM: 0.2 mg IM; may repeat in 2–4 hours
 - 15-methyl PGF 2 α —IM 0.25 mg every 15 to 90 minutes for up to 8 doses
 - Dinoprostone—Suppository: vaginal or rectal; 20 mg every 2 hours
 - Misoprostol—800–1000 μ g rectally; one dose
 - Intrauterine tamponade—Bakri balloon, packing
- Operative measures
 - Uterine compression sutures
 - Sequential arterial ligation or selective arterial embolization
 - Hysterectomy

Retained placenta

- Manual removal; manage atony as above
- Ultrasound assessment/guidance to assure complete removal
- Suction curettage—ideally performed with ultrasound guidance in the operating room (OR)
- Maintain suspicion for accreta—additional intervention required

Genital tract lacerations and hematomas

- Repair lacerations immediately
- Exposure critical—get assistance, move to OR
- No blindly placed sutures

- Packing may be necessary
- Observe stable, asymptomatic hematomas

Coagulopathy

- Appropriate factor replacement
- Identify underlying cause
- Haemorrhage, infection, amniotic fluid embolism, other

Precautionary Measures to Prevent or Minimize Postpartum Haemorrhage

Before Delivery:

- Baseline haematocrit
- Blood type and screen (cross-match for very high risk)
- IV Access
- Obtain baseline coagulation studies and platelet count, if indicated
- Identify risk factors

In Delivery Room:

- Avoid excessive traction on umbilical cord
- Use forceps and vacuum judiciously
- Inspect placenta for complete removal
- Perform digital exploration of uterus (if indicated)
- Active management of the third stage
- Visualize cervix and vagina
- Remove all clots in uterus and vagina before transfer to recovery area

In Recovery Area:

- Closely observe patient for excessive bleeding
- Continue uterotonic agents
- Frequently palpate uterus with massage
- Determine vital signs frequently

4.0 CONCLUSION

Obstetric emergency like postpartum haemorrhage contributed greatly to global maternal mortality and morbidity rate. Risk factors exist to enable prediction of emergencies and preventative steps to be taken.

5.0 SUMMARY

In this unit, you have learnt:

The definition of postpartum haemorrhage

The types of postpartum haemorrhage

The causes and management of postpartum haemorrhage

The precautionary measures to prevent or minimize postpartum haemorrhage

6.0 Online discussion and assignment

Discuss your hospital protocol on management and prevention of postpartum haemorrhage

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Unit 3 Psychiatric illness presents in the puerperium.

CONTENTS

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
 - 3.1 Puerperal psychosis
 - 3.2 Postpartum depressive illness
- 4.0 Conclusion
- 5.0 Summary
- 6.0 Online discussion and assignment
- 7.0 References/further reading

1.0 INTRODUCTION

Postnatal mood disorders have an increased risk of occurrence following delivery. All women are vulnerable to postpartum mood disorders. Childbirth results in major changes to role, expectations and relationships. There are physiological, physical and neuroendocrine changes and normal increases in anxiety and instability of mood and sleep deprivation.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Describe puerperal psychosis
- Discuss the management and prevention of puerperal psychosis
- Discuss postpartum depressive illness
- Classify postnatal depressive illness
- Discuss the prevention of postnatal depressive illness
- Outline the adverse sequelae of post-natal depressive illness

3.0 MAIN CONTENTS

3.1 Puerperal psychosis

Puerperal psychosis is a very severe disorder affects between 1:500 and 1:1000 women after delivery. It rarely presents before the 3rd postpartum day (most commonly the 5th), but usually does so before 4 weeks. Approximately 50% of women with a previous bipolar illness or postpartum psychosis will become ill. This risk justifies assessment and monitoring during pregnancy and with the woman's consent prophylactic intervention following delivery.

Puerperal psychosis is characterized thus:

- Sudden onset in the early days following delivery, deteriorating on a daily basis.
- Half will present within the first postpartum week, the majority within 2 weeks and almost all within 3 months of delivery.
- Psychosis, delusions, fear and perplexity, confusion and agitation and sometimes hallucinations.

- Agitation and severe disturbance. The onset is characteristically abrupt, with a rapidly changing clinical picture.

Risk factors for puerperal psychosis

- Previous history of puerperal psychosis
- Previous history of severe non-postpartum depressive illness
- Family history (first/second-degree relative) of bipolar disorder/affective psychosis

Signs and symptoms of puerperal psychosis

- Restless agitation
- Insomnia
- Perplexity/confusion
- Fear/suspicion
- Delusions/hallucinations
- Failure to eat and drink
- Thoughts of self-harm
- Depressive symptoms (guilt, self-worthlessness, hopelessness)
- Loss of insight

Management of puerperal psychosis

The patient should be referred urgently to a psychiatrist and will usually require admission to a psychiatric unit. If possible, this should be a mother and baby unit under the supervision of a specialist perinatal mental healthcare team. These units prevent separation of the baby from its mother and this may help with bonding and the future relationship.

Treatments include:

- Acute pharmacotherapy with neuroleptics, such as chlorpromazine or haloperidol;
- Treatment of mania with lithium carbonate
- Electroconvulsive therapy (ECT) – particularly for severe depressive psychoses;

- Antidepressants (which will take 10–14 days to be effective) as a second-line treatment.

Recovery usually occurs over 4–6 weeks, although treatment with antidepressants will be needed for at least six months. These women remain at high risk of pregnancy-related and non-pregnancy-related recurrences. The risk of recurrence in a future pregnancy is approximately 1 in 2, particularly if the next pregnancy occurs within two years of the one complicated by puerperal psychosis. Women with a previous history of puerperal psychosis should be considered for prophylactic lithium, started on the first postpartum day.

Prevention of puerperal psychosis

Primary prevention: There is some evidence that starting lithium or antipsychotics soon after delivery reduces the risk of becoming ill for those with a past history. However, it is difficult to achieve a therapeutic level in cases of very early onset psychosis.

Secondary prevention: As in primary prevention, secondary prevention involves the identification of a past history of bipolar illness and postpartum psychosis and a peripartum management plan aimed at early detection and prompt intervention.

3.2 Postpartum depressive illness

Postpartum depressive illness is a non-psychotic disorder. Depression can be classified as ‘minor’ or ‘major’.

Major depression can be divided into ‘mild’, ‘moderate’ and ‘severe’ categories. It is important to distinguish postpartum depression of any degree from the postpartum ‘blues’. Between 10 and 15 percent of women will suffer from some form of depression in the first year after the delivery of their baby. At least 7 per cent will satisfy the criteria for mild major depressive illness and many more could be described as having minor depression; 3–5 per cent will suffer a severe major post-natal depressive episode. Without treatment, most women will recover spontaneously within 3–6 months; however, 1 in 10 will remain depressed at one year.

Risk factors for post-natal depressive illness

- Past history of psychiatric illness
- Depression during pregnancy
- Obstetric factors (e.g. Caesarean section/foetal or neonatal loss)
- Social isolation and deprivation
- Poor relationships
- Recent adverse life events (bereavement/illness)
- Severe post-natal 'blues'

Symptoms of severe postpartum/post-natal depressive illness•

- Early-morning awakening
- Poor appetite
- Diurnal mood variation (worse in the mornings)
- Low energy and libido
- Loss of enjoyment
- Lack of interest
- Impaired concentration
- Tearfulness
- Feelings of guilt and failure
- Anxiety
- Thoughts of self-harm/suicide
- Thoughts of harm to the baby

Clinical features

In contrast to puerperal psychosis, non-psychotic postpartum depression usually presents later in the post-natal period, most commonly around 6 weeks, with a more gradual onset. The 6-week post-natal check is an ideal opportunity to detect early postpartum non-psychotic depression, but the signs are often missed.

Severe post-natal affective disorders usually present earlier than milder forms, and in this group, biological risk factors may be more important than psychosocial factors.

Treatment options include:

- remedy of social factors;
- non-directive counselling;
- interpersonal psychotherapy;
- cognitive-behavioural therapy;
- drug therapy: If pharmacotherapy is deemed necessary, tricyclic antidepressants

The earlier the onset of the depression and the more severe it becomes, the more likely it is that formal psychiatric intervention will be needed. However, randomized trials have demonstrated the benefits of non-directive counselling from specially trained midwives and health visitors in the management of milder disorders. Even simple encouragement to join a local post-natal group may prevent social isolation and limit depression.

Women with a past history of severe post-natal depressive illness may be candidates for some form of prophylactic treatment, and the help of a specialist in perinatal mental health care should be sought before delivery.

Adverse sequelae of post-natal depressive illness***Immediate***

- Physical morbidity
- Suicide/infanticide
- Prolonged psychiatric morbidity
- Damaged social attachments to infant
- Disrupted emotional development of infant

Later

- Social/cognitive effects on the child
- Psychiatric morbidity in the child
- Marital breakdown
- Future mental health problems

Prevention of postnatal depression***Secondary prevention***

Early detection and prompt intervention will reduce the duration and severity of the illness. *Primary prevention*

There is no evidence that antenatal screening for risk factors for postnatal depression or psychosocial antenatal interventions prevents PND, however regular postnatal visiting by a health professional in an at-risk population has been shown to reduce rates of PND.

4.0 CONCLUSION

The greater part of new-onset psychiatric illness presents in the puerperium. Affective (mood) disorders account for the majority and these vary in severity from the mildest (minor depression) to moderate and severe depressive illness and, in the extreme, puerperal psychosis (a variant of manic depression or bipolar disorder).

5.0 SUMMARY

In this unit, you have learnt that a woman the management and prevention of puerperal psychosis and postpartum (non-psychotic) depressive illness

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Discuss the predisposing factors for puerperal psychosis and postpartum depressive illness

Describe the management and prevention of puerperal psychosis and postpartum

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Module 4 Complication associated with neonatal life

Certain conditions occur commonly in the neonate that can be detrimental to the growth and development of the neonate. Many of these adverse conditions, such as cold stress, are directly related to the neonatal life transition, which occurs at the time of birth. Other conditions, such as infection, may occur as a result of risk factors that occur prior to birth, at the time of birth, or in the early neonatal period. Prompt identification of adverse conditions and proper nursing care to identify specific complications and prevent further complications are required.

Module Objectives

At the end of this module you should be able to:

- i. Describe asphyxia neonatorum and hypoxia

- ii Discuss hypoglycaemia
- ii. Discuss neonatal jaundice

Unit 1 Asphyxia neonatorum and Hypoglycaemia

CONTENTS

- 1.0 Introduction
- 2.0 Objectives
- 3.0 Main contents
 - 3.1 Asphyxia neonatorum
 - 3.2 Hypoxia
 - 3.3 Reperfusion injury
 - 3.4 Hypoglycaemia
- 4.0 Conclusion
- 5.0 Summary
- 6.0 Online discussion and assignment
- 7.0 References/further reading

1.0 INTRODUCTION

In this unit, you will learn some complications that may occur at the early period of the neonatal life that can have effect on the neonate if not handle promptly.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Identify the causes and management of asphyxia neonatorum
- Discuss hypoxia and reperfusion injury
- Outline the outcomes of newborns affected with asphyxia neonatorum and hypoxia
- Describe asphyxia neonatorum, hypoxia and reperfusion injury
- Describe hypoglycaemia in neonate
- Discuss the adverse effect of hypoglycaemia in neonate

- Discuss the management of hypoglycaemia in neonate

3.0 MAIN CONTENTS

3.1 Asphyxia neonatorum

Asphyxia neonatorum occurs when a baby doesn't receive enough oxygen before, during or just after birth. The decreased or discontinued level of oxygen can occur just before, during, or after delivery. Asphyxia commonly results from a drop in maternal blood pressure or interference with blood flow to the fetal brain during delivery. This can occur as a result of inadequate circulation or perfusion, impaired respiratory effort, or inadequate ventilation after birth. Asphyxia occurs in about 4 of every 1,000 full-term births and is more common in premature births (Centers for Disease Control and Prevention [CDC], 2013b).

Causes of asphyxia

When the placenta does not provide the fetus with enough oxygen, fetal hypoxia will result. Examples of conditions that can cause asphyxia are:

- Prolonged labour
- Breech birth
- Placental abruption
- Maternal sedation in preterm infants

Signs and symptoms of low foetal oxygen states

- Meconium-stained amniotic fluid and umbilical cord
- Abnormal heart rate or rhythm
- Bradycardia
- An increased acid level in a baby's blood

At birth, symptoms may include:

- Bluish or pale skin colour
- Low heart rate
- Weak muscle tone and reflexes
- Weak cry
- Gasping or weak breathing

Diagnosis

- APGAR score between 1 to 5 after birth and score of 3 or lower for more than 5 minutes. APGAR scoring system has five factors:
 - breathing
 - pulse
 - appearance
 - response to stimulus
 - muscle tone

Each factor gets a score of 0, 1, or 2. The highest score possible is 10. A baby with a lower Apgar score after 5 minutes has a higher risk for asphyxia neonatorum. A score lower than 7 can indicate that a baby doesn't have enough oxygen.

- Blood test revealing high acid levels.

3.2 Hypoxia

Asphyxia is the most common cause of hypoxia, it is a condition in which the body is deprived of adequate oxygen, commonly occurs when the newborn fails to breathe adequately after deliver. Asphyxia, if not quickly reversed and treated, will undoubtedly lead to hypoxia and possible brain damage or death. The severity of the injury to the newborn is directly related to the severity of the asphyxia and length of time that it occurred. Cell damage occurs within minutes of the initial lack of blood flow and oxygen.

Causes of hypoxia

- Prematurity
- Cord prolapse
- Cord occlusion
- Placental infarction
- Intrauterine growth restriction
- Maternal smoking

3.3 Reperfusion Injury

There is a second stage of damage called "reperfusion injury," which occurs after the restoration of normal blood flow and reoxygenation to the brain due to the release of toxins by damaged cells. The degree of injury varies. Infants with mild or moderate

asphyxia may have a full recovery, whereas infants who had prolonged oxygen deprivation may have permanent injury to the brain, heart, lungs, bowels, kidneys, or other organs. Adverse Effects Outcomes of newborns affected with asphyxia and hypoxia vary. Premature infants are at greatest risk to experience adverse effects, which may include:

- Heart rate variations
- Respiratory distress
- Central cyanosis
- Hypotonia
- Cerebral palsy
- Developmental disabilities
- Attention deficit hyperactivity disorder
- Impaired sight
- Complete organ failure
- Death

Management

Good management during labour and delivery and the early detection of non-reassuring fetal status are the best methods of preventing asphyxia. However, some cases of asphyxia cannot be predicted or prevented. In that case, asphyxia and the subsequent prevention of prolonged hypoxia require resuscitating the newborn infant.

- The severity of the baby's symptoms and the diagnosis influences the treatment. For example, mothers may receive additional oxygen before delivery to boost a baby's oxygenation before birth. A cesarean delivery is a potential preventive measure in prolonged or difficult deliveries.
- After birth, babies with the condition may need ventilation to support their breathing. Keeping babies warm has also been shown to reduce harmful effects.
- Monitor the baby's blood pressure and fluid intake to make sure they're getting enough oxygen.

- Watch out for seizure which is a complication of asphyxia neonatorum.

3.4 Hypoglycaemia

Hypoglycaemia is a condition in which the amount of blood glucose in the blood is lower than normal. Blood glucose less than 40 mg/dL in the newborn period is considered normal. The normal range of blood glucose varies depending on the age of the baby, type of food, assay method used, and possibly the mode of delivery. Up to 14% of healthy term babies may have a blood glucose level less than 40 mg/dL during the first 3 days of life. Newborns consume fuel sources at a faster rate due to their rapid rate of breathing, loss of heat when exposed to cold, activity, and activation of muscle tone. Glucose is the main source of energy in the first 4 to 6 hours after birth and is the main source of fuel for the brain. During pregnancy, glucose is passed to the foetus from the mother through the placenta. Some of the glucose is stored as glycogen in the placenta and, later, in the foetal liver, heart, and muscles. These stores are important for supplying the newborn's brain with glucose during delivery, and for nutrition after the baby is born. Blood glucose should be checked on all babies who are small for gestational age or large for gestational age, have low temperatures, are jittery, or who had a stressful delivery.

Causes of hypoglycaemia

Inadequate maternal nutrition in pregnancy

Cold stress

- Excess insulin production by a newborn of a diabetic mother
- Incompatibility of blood types of newborn and mother
- Birth defects and congenital metabolic diseases
- Birth asphyxia
- Liver disease

Adverse Effects

- Tremors, jitteriness, irritability
- Exaggerated Moro reflex
- High-pitched cry

- Lethargy, listlessness, hypotonia
- Cyanosis, apnoea, tachypnoea
- Hypothermia, temperature instability
- Poor suck, refusal to feed
- Apnoea

Management

- Glucose screening is recommended for high-risk infants:
 - Infants whose mothers had uncontrolled gestational diabetes or diabetes mellitus
 - Large for gestational age (> 8 pounds 12 ounces or > 3,969 g)
 - Small for gestational age (< 5 pounds 12 ounces or < 2,608 g)
 - Premature (< 37 weeks gestation)
 - Low birth weight (< 2,500 g)
 - Donor twin in twin-to-twin transfusion
 - Polycythaemia (haematocrit > 70%)
 - Hypothermia
 - Stress (sepsis, respiratory distress, other)
 - Low Apgar scores (< 5 at 1 minute or < 6 at 5 minutes)
- Specific treatments for hypoglycemia are based on the infant's gestational age, overall health, and medical history. Treatment options include:
 - Early feeding of rapid-acting source of glucose, such as an early feeding of a glucose/water mixture or formula
 - During the first 4 hours of life: Any newborn glucose level less than 40 mg/dL in a baby with symptoms requires immediate IV-fluid therapy.
 - In an asymptomatic baby, with an initial glucose level of less than 25 mg/dL, an immediate feeding followed by another glucose check in an hour

is indicated. If the subsequent test is still less than 25 mg/ dL, immediate IV-fluid therapy is indicated.

- Between 4 and 24 hours of life: Any glucose level less than 40 mg/dL with symptoms requires immediate IV-fluid therapy.
- If asymptomatic and a glucose level less than 35 mg/dL, initiation of oral feeding and another glucose check in 1 hour are indicated. If the subsequent test is still less than 35 mg/dL, immediate IV-fluid therapy is indicated.

4.0 CONCLUSION

Adequate monitoring of neonate will prevent hypoxia and hypoglycaemia. The brain depends on blood glucose as its main source of fuel, and severe or prolonged hypoglycaemia may result in seizures and/or serious brain injury. If the blood glucose level is below 40 mg/dL, start feeding protocol per hospital.

5.0 SUMMARY

In this unit you have learnt:

Asphyxia neonatorum, hypoxia and reperfusion injury

Neonatal

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Describe your hospital feeding protocol for neonate.

7.0 REFERENCES AND OTHER READING MATERIALS

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Unit 2 Neonatal jaundice

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- 1.0 Introduction
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1.0 INTRODUCTION

Jaundice is the most common condition that requires medical attention and hospital readmission in newborns. The yellow colouration of the skin and sclera in newborns with jaundice is the result of accumulation of unconjugated bilirubin. In most infants, unconjugated hyperbilirubinemia reflects a normal transitional phenomenon. However, in some infants, serum bilirubin levels may rise excessively, which can be cause for concern because unconjugated bilirubin is neurotoxic and can cause death in newborns and lifelong neurologic sequelae in infants who survive. For these reasons, the presence of neonatal jaundice frequently results in diagnostic evaluation.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Definition of neonatal jaundice
- Types of neonatal jaundice
- Describe the pathophysiology of neonatal jaundice
- Identify the causes of neonatal jaundice
- Discuss the management of neonatal jaundice



Neonatal Jaundice: Source: www.mummysg.com

3.0 MAIN CONTENTS

3.1 Neonatal jaundice

Jaundice refers to the yellow pigmentation of the skin and conjunctival membranes caused by excess bilirubin in the blood; it occurs when the old cells break down and haemoglobin is changed into bilirubin and removed by the liver. It is estimated that over 50% of newborns will develop some amount of jaundice during the first week of life. The build-up of bilirubin in the blood is called hyperbilirubinemia. The most common symptoms of jaundice include yellow colouring of the baby's skin, usually beginning on the face and moving down the body; poor feeding; or lethargy. The timing of the appearance of jaundice helps with the diagnosis. Bilirubin level peaks in a term baby at 3 to 5 days and in 5 to 7 days in a preterm baby. Although low levels of bilirubin are not usually a concern, large amounts can circulate to tissues in the brain and may cause seizures and brain damage. This condition is called kernicterus.

Types of jaundice

Physiologic jaundice: Occurs as a “normal” response to the baby’s limited ability to excrete bilirubin in the first days of life; occurs after 24 hours of life; most of the time resolves without treatment.

Pathologic jaundice: Jaundice may occur with the abnormal breakdown of red blood cells due to haemolytic disease of the newborn (Rh disease), polycythemia (too many red blood cells), inadequate liver function, infection, or other factors; occurs within first 24 hours of life; needs additional medical management and may be associated with infection, metabolic disorder, bleeding disorder, liver abnormality, or a defect in excretion. Neonatal hypoxia, congenital heart disease, reduced bowel motility, and intestinal obstruction are also common causes.

Breast milk jaundice: About 2% of breastfed babies develop jaundice after the first week; monitoring is needed; should resolve without treatment.

Causes of neonatal jaundice

- Prematurity
- Maternal foetal incompatibility
- Cephalhaematoma
- Sepsis
- Erthroblastosis fetalis
- Areas of bruising
- Feeding problems
- Cold stress

Investigation

Serum Blood Test

Normal values of total bilirubin are from 0.3 to 1.0 mg/dl. In a newborn, higher bilirubin is normal due to the stress of birth. Normal bilirubin in a newborn would be under 5 mg/dl, but many newborns have some kind of jaundice and bilirubin levels above 5 mg/dl. In general, a total bilirubin level above 1.9 mg/dl is considered elevated. The

normal range for total bilirubin level in the blood is 0.3 to 1.9 mg/dl. Direct, or conjugated, bilirubin normally ranges from 0 to 0.3 milligrams per deciliter.

Management

Treatment depends on many factors, including the cause of the jaundice and the level of bilirubin as well as the extent of the disease, gestational age, overall health, and medical history. Mild infant jaundice often disappears on its own within two or three weeks. For moderate or severe jaundice, your baby may need to stay longer in the newborn nursery or be readmitted to the hospital. Treatments to lower the level of bilirubin in your baby's blood may include Light therapy (phototherapy). The goal is to keep the level of bilirubin from increasing to dangerous levels. Key nursing interventions include:

- Identification of infants at risk
- Frequent nursing to maintain hydration and aid in excretion
- Maintaining thermoregulation
- Phototherapy
- Fiber optic blanket
- Exchange transfusion
- Increased breastfeeding
- Treatment of underlying conditions

Physiotherapy

Phototherapy should be instituted when the total serum bilirubin level is at or above 15 mg/dl (257 mol/l) in infants 25 to 48 hours old, 18 mg/dl (308 mol/l) in infants 49 to 72 hours old, and 20 mg/dl (342 mol/l) in infants older than 72 hours. Light treatment is the process of using light to eliminate bilirubin in the blood. Your baby's skin and blood absorb these light waves. These light waves are absorbed by your baby's skin and blood and change bilirubin into products, which can pass through their system. Phototherapy employs blue wavelengths of light to alter unconjugated bilirubin in the skin. The bilirubin is converted to less toxic water-soluble photo-isomers that are excreted in the

bile and urine without conjugation. The decision to initiate phototherapy is based on the newborn's age and total serum bilirubin level. Another bilirubin level will be checked 12–18 hours later to make sure it hasn't risen again. Babies usually need to be under phototherapy lights for around 48 hours and often longer.

The efficacy of phototherapy depends on several important factors. The ideal configuration is four special blue bulbs (F20T12/BB) placed centrally, with two daylight fluorescent tubes on either side. The power output of the lights (irradiance) is directly related to the distance between the lights and the newborn. Ideally, all lights should be 15 to 20 cm from the infant. To expose the greatest surface area, the newborn should be naked except for eyeshields. For double phototherapy, a fiber-optic pad can be placed under the newborn. This method is twice as effective as standard phototherapy.

Physiological jaundice normally clears by the time your baby is two week's old. Phototherapy will be stopped when the bilirubin level falls to a safe level, which usually takes a day or two. Phototherapy is generally very effective for newborn jaundice and has very few side effects, although your baby may develop a temporary rash or tan as a result of the treatment. Potential problems that may occur with phototherapy include burns, retinal damage, thermoregulatory instability, loose stools, dehydration, skin rash, and tanning of the skin. Because phototherapy is continuous, treatment also involves significant separation of the infant and parents.

The only contraindication to the use of phototherapy is conjugated hyperbilirubinemia, as occurs in patients with cholestasis and hepatic disease. In this setting, phototherapy may cause a dark grayish-brown discolouration of the skin (bronze baby syndrome).

Evaluation

With intensive phototherapy, the total serum bilirubin level should decline by 1 to 2 mg/dl (17 to 34 μ mol/l) within four to six hours. The bilirubin level may decline more slowly in breastfed infants (rate of 2 to 3 mg/dl/day) than in formula-fed infants.

Phototherapy usually can be discontinued when the total serum bilirubin level is below

15 mg/dL.¹ The average rebound bilirubin level after phototherapy is below 1 mg/dl. Therefore, hospital discharge of most infants does not have to be delayed to monitor for rebound elevation. If the total serum bilirubin level remains elevated after intensive phototherapy or if the initial bilirubin level is meets defined critical levels based on the infant's age, preparations should be made for exchange transfusion.

Exchange Transfusion

Exchange transfusion is the most rapid method for lowering serum bilirubin concentrations. This treatment is rarely needed when intensive phototherapy is effective. The procedure removes partially hemolyzed and antibody-coated erythrocytes and replaces them with uncoated donor red blood cells that lack the sensitizing antigen. In the presence of hemolytic disease, severe anaemia, or a rapid rise in the total serum bilirubin level (greater than 1 mg/ dl per hour in less than six hours), exchange transfusion is the recommended treatment. Exchange transfusion should be considered in a newborn with non-hemolytic jaundice if intensive phototherapy fails to lower the bilirubin level.

Complications of exchange transfusion can include air embolism, vasospasm, infarction, infection, and even death. Because of the potential seriousness of these complications, intensive phototherapy efforts should be exhausted before exchange transfusion is initiated.

4.0 CONCLUSION

Maintaining the baby's temperature and acidosis, monitoring stools for frequency encouraging early breastfeeding and adequate and frequent hydration via feedings to promote intestinal colonization and calories needed for hepatic binding proteins are imperative.

5.0 SUMMARY

In this unit you have learnt:

The definition of neonatal jaundice

The types of neonatal jaundice

Causes of neonatal jaundice

The management of neonatal jaundice

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Discuss the management of neonatal jaundice in your hospital

7.0 REFERENCES AND OTHER READING MATERIALS

Davison M.R (2014). FAST FACTS FOR THE NEONATAL NURSE A Nursing Orientation and Care Guide in a Nutshell. New York: Springer Publishing Company

Marshall J. & Raynor M. (2014). Myles Textbook for Midwives (16th ed). New York: Churchill Livingstone Elsevier

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Module 5 Infertility

Infertility is a condition that encompasses a wide spectrum of reversible and irreversible disorders, and many successful treatments are available. Today, greater numbers of men and women are seeking infertility treatment due to increased public awareness of infertility and available treatments, improvements in the availability and range of fertility treatments, improvements in physicians' ability to evaluate and diagnose infertility, and changes in social acceptance of infertility.

Module Objectives

At the end of this module you should be able to:

- i. Describe the indications and interpretation of investigations used in the assessment of the infertile couple
- ii. Discuss the principles, indications for and complications of the common methods of treatment of infertility

Unit 1 Overview of infertility

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- 1.0 Introduction
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 - 3.3 Mastitis
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1.0 INTRODUCTION

Infertility affects approximately 10-15% of reproductive-age couples. Reproductive age generally encompasses ages 15 to 44 years, although pregnancy can occur outside of this age range. Infertility is the failure of a couple to conceive after 12 months of frequent, unprotected intercourse. The probability of achieving a pregnancy in one menstrual cycle is termed fecundability. It is estimated to be 20% to 25% in healthy young couples. Similarly, fecundity is the probability of achieving a live birth in one menstrual cycle. Fecundability and fecundity both decrease over time; in other words, the probability of conceiving in a given menstrual cycle decreases as the duration of time to achieve conception increases. After 12 months without using contraception, approximately 50% of couples will conceive spontaneously within the following 36 months. If a couple does not conceive by this point, then infertility will likely persist without medical intervention.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Define infertility and discuss the causes
- Evaluation, and management of female and male infertility.
- Describe the psychosocial issues associated with infertility.

3.0 MAIN CONTENTS

3.1 Definition

Failure to conceive following at least one year of regular unprotected sexual intercourse. It is the inability to conceive after 12 months of appropriately timed intercourse without contraception in women aged < 35 years or after 6 months in women aged \geq 35 years.

It can either be:

Primary infertility which is referred to couples who have never achieved a pregnancy or secondary infertility implies at least the couples had one previous conception.

Incidence

- 10–15% of reproductive age married couples are infertile.

- The prevalence of infertility has remained constant, but the number of office visits to physicians by “infertile” couples has tripled over the past 20 years. This “infertility epidemic” has been attributed primarily to elective postponement of childbearing and increased insurance coverage of elective fertility therapy.

Causes of infertility

Female causes (50%)

Ovarian factors (20%): it could be caused by:

- Ovulatory dysfunction
- Polycystic ovarian syndrome (chronic anovulation)
- Premature ovarian insufficiency
- Hypothalamic amenorrhoea

History: Secondary amenorrhea, irregular menses.

Physical examination: Obesity, hirsutism, galactorrhea, lean body habitus (hypothalamic amenorrhea).

Screening tests: Typical confirmation of ovulation by history (predictable menstrual intervals (21–35 days), urinary kits to detect the midcycle LH surge (indicative of ovulation), recording daily basal body temperature recordings, or mid to late luteal phase serum progesterone concentration. Ovarian reserve may be assessed with day 3 FSH, estradiol, antimüllerian hormone, and/or clomiphene challenge test.

Treatment: Ovulation induction

Tubal and peritoneal factors (20%): it could be caused by:

- Pelvic adhesions
- Endometriosis
- Prior ruptured ectopic pregnancy
- Prior tubal or pelvic surgery Note: tubal obstruction can be demonstrated by either hysterosalpingogram (HSG) or during laparoscopy

History: Prior pelvic infection or ectopic pregnancy may suggest pelvic adhesive disease or tubal disease. Secondary dysmenorrhea or cyclic pelvic pain may prompt

suspicion of endometriosis. However, there are no identifiable risk factors in > 50% of patients. **Physical examination:** Retroverted fixed uterus, rectovaginal nodularity, and uterosacral nodularity are possible clinical signs of endometriosis.

Screening tests: Hysterosalpingogram (HSG) involves injection of a radio-opaque dye through the cervix into the uterus with spillage into the peritoneal cavity. It assesses tubal patency as well the contour of the uterine cavity to exclude filling defects (e.g., endometrial polyps, fibroids, synechiae). Recently, newer saline sonohysterogram methods, utilizing echogenic distending fluid (e.g. FemVue) have been utilized. Laparoscopy with tubal lavage or fertiloscopy is the “gold standard” diagnostic test because it can exclude adhesions and endometriosis.

Treatment: Tubal surgery (tuboplasty) or in vitro fertilization

Cervical factors (10%) it could be caused by:

- Cervical stenosis
- Cervicitis

History: Prior cervical surgery (cone biopsy, cautery), infection, or in utero diethylstilbestrol (DES) exposure. • Physical examination. Cervical abnormalities, lesions.

Screening tests: None is reliable. The postcoital test is a historical method to evaluate sperm–cervical mucus interaction. However, this test is no longer viewed as a standard of care given its significant diagnostic limitations.

Treatment: IUI.

Male factors (35%)

- Idiopathic (unexplained) oligospermia (most common)
- Varicocele
- **Others:** Ductal obstruction (post-infectious epididymitis, post-vasectomy) and Failure to deliver sperm to the vagina (hypospadias, impotence)

History: Testicular injury, genitourinary infection, chemotherapy or radiation exposure, genitourinary surgery, erectile or ejaculatory dysfunction, or tobacco or recreational drug use.

Physical examination: Hypospadias, varicocele, cryptorchidism (undescended testes), atrophic testicles.

Screening test: Semen analysis is the primary screening test for male infertility. Semen sample should be produced after 2–3 days of abstinence. If a single sample has abnormal parameters (e.g., concentration, motility, or morphology) it should be repeated 4 weeks later.

Treatment: Surgical correction of varicocele; intrauterine insemination or in vitro fertilization with or without intracytoplasmic sperm injection (ICSI) depending upon semen parameters.

Unexplained (idiopathic) infertility (10–15%):

History: Female patient is ovulatory and all ovarian reserve, endocrine, hysterosalpingogram, and semen analysis testing is normal.

Physical examination and screening tests are unremarkable.

Treatment: Ovulation induction and superovulation with intrauterine insemination (IUI) or in vitro fertilization

Prognosis: About 60% of couples with unexplained infertility who receive no treatment will conceive within 3–5 years.

Risk factors

Fecundability begins to slowly decline after age 28 and usually declines at a more rapid rate after age 35.

Cigarettes smoking, recreational drug use and certain occupational and environmental exposures decrease the fertility rate.

4.0 CONCLUSION

The most common causes of male and female infertility are investigated during the initial evaluation of infertility. It is important to recognize that more than one factor may be involved in a couple's infertility.

5.0 SUMMARY

In this unit you have learnt:

The definition and classification of infertility

Causes and investigation and management of infertility

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Discuss occupational and environmental exposures that decrease fertility rate in both men and women.

7.0 REFERENCES AND OTHER READING MATERIALS

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Unit 2 Assisted reproduction

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1.0 INTRODUCTION

Infertility is a major public health problem, causing significant distress to those directly involved as well as to family and friends. The population using assisted reproduction will usually include those who have prolonged infertility and, as such, is arguably not truly representative of the general population with infertility. However, outcomes in those who access IVF treatment, although under very different conditions, may give some insight into what may be occurring in natural attempts to conceive and afford help to couples in coming to terms with their infertility.

2.0 OBJECTIVES

At the end of this unit, you should be able to:

- Define assisted reproductive technology (ART)
- Describe the classification of assisted reproductive technology (ART)

- Describe the common techniques in ART

3.0 MAIN CONTENTS

3.1 Assisted reproductive technology (ART)

Assisted reproductive technology (ART) is the direct handling and manipulation of oocytes and sperm to facilitate optimal fertilization and maximize the probability of achieving pregnancy.

Classification of assisted reproductive technology (ART)

In vitro fertilization (IVF) is the paradigm of ART procedures. Other examples include gamete or zygote intrafallopian transfer (GIFT or ZIFT), but these techniques are rarely used.

Frequency: The first baby conceived by IVF was delivered in 1978. Since that historic birth, ART has undergone rapid growth. There are > 125,000 IVF cycle initiated in the USA annually.

Goal of IVF: To maximize the chance of a successful pregnancy while minimizing the risk of multiple gestations.

In vitro fertilization: IVF is usually performed in Petri dishes.

Patient selection

- IVF was originally developed for tubal factor infertility. It is now widely utilized for several infertility conditions that have not been successfully treated with other modalities.
- Female age is usually most predictive of IVF success. Most IVF programs limit IVF treatment to women aged up to 43.9 years. Although the menopause has set a natural barrier to further conception, IVF has allowed women to be pregnant in their 50s and 60s using donor egg IVF.
- A serum follicle-stimulating hormone (FSH) level > 10 mIU/mL and/ or estradiol > 80 pg/mL on day 3 of the menstrual cycle is usually indicative of diminished ovarian reserve and suboptimal responsiveness to ovarian stimulation.
- IVF with donor oocytes may be recommended for women with significantly diminished ovarian reserve regardless of age, and for those who have premature

ovarian insufficiency or been traditionally considered sterile (eg, Turner syndrome, ovarian dysgenesis).

Ovarian stimulation

- Although unstimulated (“natural cycle”) or clomiphene-stimulated IVF cycles are less costly, few oocytes are harvested, and success rates are low. These techniques are rarely used. Controlled ovarian hyperstimulation maximizes the retrieval of multiple mature oocytes.
- A typical stimulated IVF cycle (Figure 27.1) is initiated by the administration of a GnRH agonist (eg, leuprolide acetate, nafarelin acetate) in the late luteal phase of the cycle or during pretreatment with combined oral contraceptive pills. A GnRH agonist prevents premature ovulation, decreases cycle cancellation (due to premature luteinization), and increases the number of successful pregnancies per cycle.
- Follicular growth and development are achieved with daily intramuscular administration of rFSH and hMG. Once “adequate” ovarian stimulation has been achieved (at least three lead follicles > 16–18 mm diameter, a serum estradiol level ≥ 600 pg/mL), a human chorionic gonadotropin (hCG) trigger is given as a substitute for the luteinizing hormone (LH) surge to promote maturation of the oocytes in preparation for ovulation and oocyte retrieval. • Of IVF cycles 5–10% are cancelled due to inadequate follicular response.

Oocyte retrieval

- Ultrasound-guided transvaginal oocyte retrieval (Figure 27.2) is performed 35–37 hours after hCG administration.
- The number of harvested oocytes may be correlated to the number of follicles > 12 mm. Retrieved oocytes are evaluated for maturity.

Fertilization

- Semen is collected the day of oocyte retrieval. The sperm are “washed” and incubated in supplemented medium.

- Four hours after oocyte retrieval, 25,000–50,000 motile sperm are added to each dish containing a single oocyte.
- Eighteen hours after insemination, the ova are examined microscopically for evidence of fertilization (the presence of two pronuclei). Mature oocytes have a fertilization rate of 50–70%.
- Oocytes that undergo normal fertilization and become embryos are maintained in culture medium and observed for further development. After embryo transfer, supernumerary (remaining appropriate quality) embryos may be cryopreserved.

Embryo culture and transfer

The fertilized oocytes are placed in growth medium and usually examined daily until day 3 (3 days after oocyte retrieval).

Embryos are graded by an embryologist based on the number of cells (blastomeres), symmetry of blastomere growth, orientation, and degree of fragmentation.

The embryo transfer may be performed on day 3 or 5 depending on institutional guidelines. The number to be transferred depends on the number available, the age of the woman, and other health and diagnostic factors. In the UK, a maximum of two embryos are transferred except in unusual circumstances. Transcervical embryo transfer consists of loading the embryos and a small amount of medium into a flexible catheter, which is then placed through the cervix, and the contents injected with ultrasound guidance.

Luteal phase support

- Progesterone supplementation is started on the day of oocyte retrieval and continued until the 10-week estimated gestational age (EGA) of pregnancy when the placenta demonstrates autonomous progesterone production. Progesterone supplementation improves pregnancy outcome in IVF.
- A quantitative serum β hCG level may be obtained 12–14 days after transfer to assess for implantation.

Common techniques in ART

Preimplantation genetic diagnosis (PGD)

- “Embryo screening” can be performed on embryos before implantation. Specific genetic mutations or aneuploidy may be detected after analysis of a portion of each embryo.
- PGD is an alternative to prenatal diagnosis that avoids selective pregnancy termination by making it highly likely that the baby will be free of the disease under consideration (e.g. cystic fibrosis, sickle cell disease).

Intracytoplasmic sperm injection (ICSI)

- ICSI involves the direct injection of a selected single sperm into the egg. This is typically utilized in cases of male factor infertility (abnormal semen parameters), or suboptimal fertilization or failed fertilization in a prior IVF cycle.

Transfer of cryopreserved embryo(s)

The major advantage of this option includes: no need for controlled ovarian stimulation and oocyte retrieval and flexibility in scheduling the embryo transfer.

Pregnancy outcome

The live birth rate per IVF cycle initiated is dependent upon female age and infertility diagnosis. Typically, those patients aged < 35 years or undergoing donor egg IVF have the best live birth pregnancy rates.

Effect of ART on multifetal pregnancy. Transfer of multiple embryos may improve the pregnancy rate but will also increase the number of multiple pregnancies.

4.0 CONCLUSION

The preliminary assessment of the availability of eggs and sperm, together with a determination that the gametes can meet in couple seeking assisted reproduction, should provide a diagnosis for the majority of couples. A prognosis, usually favourable, should be able to be provided and, where necessary, treatment initiated within a relatively short time.

5.0 SUMMARY

In this unit you have learnt:

Definition of ART

Classification of assisted reproductive technology (ART)

Common techniques in ART

6.0 ONLINE DISCUSSION AND ASSIGNMENT

Describe the process of IVF

7.0 REFERENCES AND OTHER READING MATERIALS

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