UNIT 9 COMPLICATIONS IN LATE PREGNANCY-III

Structure

9.0 OBJECTIVES

After reading this unit, you should be able to:

- describe the importance of blood grouping and Rh tying during pregnancy;
- provide immuniprophylexis against Rh-isoimmunization;
- enumerate the organism causing intrauterine infection with the sequel;
- manage appropriately pregnancy and labour in women with intrauterine infections;
- list the causes of pain abdomen during pregnancy; and
- diagnose the cause of pain abdomen during pregnancy and manage them.

9.1 INTRODUCTION

This unit deals with miscellaneous topics on Rh incompatibility, congenital infection and pain abdomen during pregnancy. Immunoprophylaxis with “anti D” immunizlobalin has greatly reduced Rh-isoimmunization in pregnant women. In section on intrauterine infection, you will read about various types of infection and the resulting complications. Pain in abdomen is a common problem for which pregnant women seek care. In majority of cases, it is due to pregnancy related or incidental physiological causes and you can manage them. Life threatening conditions also may present with pain abdomen and may be due to obstetric, gynaecological, surgical or medical causes. You should be able to diagnose them and refer to FRU/CHC/District Hospital after primary care.

9.2 RH-INCOMPATIBILITY

In this section, you will be reading about presentation of haemolytic disease of newborn caused by Rhesus incompatibility. Rhesus incompatibility is the commonst cause of haemolytic disease. ABO incompatibility can also cause haemolytic disease but it is rare.

9.2.1 Classification of Rhesus factor and isoimmunisation

In every individual, Rhesus genes are carried on two chromosomes one of which is passed on to the succeeding generation. There are six main Rhesus genes, three carried in each chromosome. Of the six, 3 are dominant genes and 3 are recessive genes. The 3 dominant genes are C, D and E and the recessive genes are c, d and e. each chromosome has a locus for C, D and E which may be occupied by either dominant or recessive gene of the particular type.

In Rh-incompatibility, the dominant D gene is the most important. The individual possessing D gene is one or both chromosome is known as Rh+ve. When dominant D gene is absent in both chromosomes and its place is occupied by its allele d genes, the person is known as Rh-negative. Genotype means the mixture of these three pairs of genes on the two chromosomes. Many combinations are possible to illustrate the point, the following example is given:

CDe/cDE …… Homozygous Rhesus Position

CDE/cde …… Heterozygous Rhesus positive

CDE/cde…….. Rhesus negative

When a Rh negative women marry a Rh positive man, the offspring will be Rh positive if the man is homozygous positive and can either be Rh-negative or Rh positive if he is heterozygous. If the man is also Rh negative, the offspring will be Rh negative and there will be no incompatibility. This is made clear in the following diagram.

Figure showing the possible Rh blood group of children.

In India, prevalence of Rh D antigen varies from 90 to 95%, lower in South India compared to North India. It is important *for* you to remember that CDE antigen other than D have low immunogenicity and not very important except on few occasions. Therefore, all the pregnant women should be routinely screened for D (Rh) antigen on erythrocytes.

The problem of isoimmunization occurs because an Rh negative individual (mother) can produce antibodies when exposed to antigen from fetus (paternally ilherited antigen). You must know that 0.25 ml of fetal blood is enough to cause Rh isoimmunization. The interval between the maternal exposure to antigen and its al1tibody response is usually many weeks. Hence, in primigravida, fetus may not be affected. in antibodies produced by the mother in turn cross the placenta, enter the fetal circulatioll resulting in fetal haemolysis and its consequences.

The mother is exposed to antigen if the fetus is Rh +ve at the time of separation of the placenta after the birth of the baby. Some amount of fetal blood enters into maternal circulation. The mother is exposed to antigen if the fetus is Rh +ve mother will produce antibodies to Rh antigen which in subsequent pregnancies enter the fetal circulation and lead to haemolysis of fetus (if it is Rh positive). It is also possible that the fetal red cells can enter into maternal circulation during pregnancy if there is separation of placenta (e.g. Abruptio placenta). There are other situations when there is likelihood of Rh isoimmunization due to transfusion with Rh positive blood, abortion, MTP, amnillocenteric and external version.

Fortunately, erythroblastosis fetalis occurs in very few pregnancies because of ABO incompatibility between mother and fetus. If the mother and fetus are ABO incompatible, the fetall red cells are destroyed immediately and no sensitisation for Rh D antigen occurs. If they are ABO compatible, the fetal red cells persist in maternal circulation and sensitisation occurs resulting in antibody formation.

Check Your Progress I

1) Who is called an Rh +ve individual?

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2) Give 4 clinical signs that suggest that suggest hydrops fetalis.

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3) What is Anti-D ?

4) Anti-D must be given to RH negative mother with in..... hour of delivery?

5) Klehauer Betke test is use for detecting

6) Give three conditions when Anti 'D' must be administered to mother ?

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9.2.2 Pathological Changes in Haemolytic Disease of the New Born

You are already aware of the basic concept of isoimmunization. But, it is important to kIl0W the pathologic changes in the organ system of the new born in the haemolytic disease of the new born.In a D-positive fetus, maternal att.tibodies (IgG) are both adsorbed to D-positive erythrocytes and exist unbound in fetal serum. The absorbed.antibodies act as haemolysins leading to an accelerated rate of red cell destruction. Maternal antibodies detectable in fetal serum at birth gradually disappear from infants circulation over a period of I to 4 months, to some extent influenced by exchange tratlsfusion.

a) Immune Hydrops

You must realise that hall mark ofR11 isoimmunization is haemolysis in fetus leading to hydrops fetalis or hyperbilirubinemia itl neonatal period. The affecte.d fetus shows severe atlaemia due to haemolysis leaditlg to hydrops fetalis. Marked tissue edema will be seen in the affected fetus as well as effusion into the serus cavities (Hydrops fetalis).

The placenta is markedly enlarged boggy, with prominent cotyledons. Excessive extra medullary hemetopoiesis results in massive hepatospleenomegaly. Histological examination of liver shows fatty degenerative parenchymal Chatlges as well as deposition of

haemosiderin and engorgement of hepatic canaliculi with bile. There may be cardiomegaly atld pulmonary haemorrhage. The ascitis along with hepatospleenomegaly may result in severe dystocia. The fetus with hydrops may die in utero from profound atlaemia and circulatory failure. The live born of hydropic infant appears pale, edematos alld limp at birth; often requiring resuscitation. Dyspnoea and circulatory failure are conllnon.

b) Hyperbilirubinemia

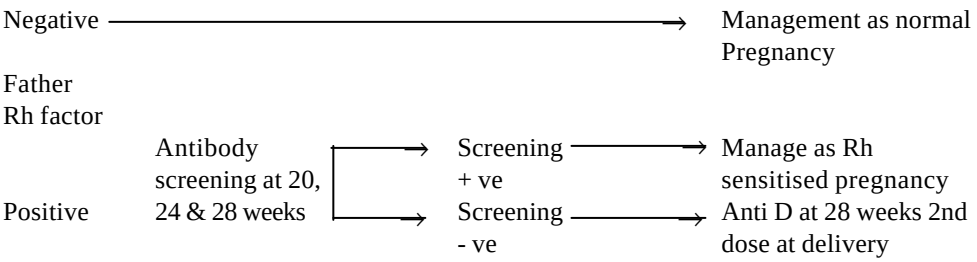
As you Call understand the less severely affected infallts appear well at birth and become jaundiced within *few* hours. The fetus does not develop jaundice in utero because unconjugated bilirubin resulting from fetaillaemolysis freely cross.es placenta and ellters maternal circulation where it is disposed by maternal liver. But at birth, newborn especially pre~ature liver is not mature enough to metabolise high levels *of* un conjugated bilirubin resulting in hyperbilirubinaemia. Theunconjugated bilirubin crosses the blood brain barrieralld enters central nervous system resulting in degeneration of. the basal ganglia alld hYPPocalnpus. This complication occurs more often in premature, hypoxic, acidotic infants. Surviving infants show spasticity, muscular in co-ordination alld varying degree of mental retardation. Tllere is a positive correlation between serum .unconjugated bilirubin and Kernicterus above 18-20 mg/dl, although Kernicterus Call develop at lower concentrations.

9.2.3 Management of Rh-ve non-immunised pregnant woman

You must know that Rh -ve isoimmunised women are to be referred to specialised centres for more investigations and management. This section will deal with management of Rh -ve non-immunised pregnant women.

You must have appreciated by now the need for routine screening of all pregnant women for the blood group and Rh typing at 1st prenatal visit. It is important to note that the chances of Rh isoimmunization depends on Rh blood type of the father. If the father is Rh negative, there are no chances of Rh isoimmunization. If the father is Rh positive, the baby has the chance of 50% being Rh positive (Heterozygos) and 100% being Rh positive (Homozygos). Hence if the woman is Rh -ve, hushand's blood grouping is done. If he is Rh +ve, wherever possible, his Rh genotyping is done to know if he is homozygous or heterozygous Rh +ve. Rh sensitisation occurs as mentioned earlier in about 1% of pregnancies are. We are already aware that the chances of isoimmunization due to excessive feto maternal haemorrhage.

Once the pregnant Woman is identified as Rh negative, with an Rh +ve husband, the next logical step is to screen for allti-RIl aluibodies to rule out sensitization which is done by indirect Coomb's test. Indirect Coomb's test detects maternal antibodies (i.e., IgG; incomplete antibodies) by Coomb's serum, which contains anti IgG immunoglobulul. The IgG antibodies (Anti Rh antibodies) cannot agglutinate red blood cells themselves at 37 degree centigrade but they do so in presence of albumin or Coomb's serum. Indirect Coomb's test is done at I st prenatal visit and if negative repeated at 20, 24 and 28 weeks of gestation. If any time the indirect Coomb's test is positive it is managedas Rh isoimmunized pregnancy. If indirect Coomb's test is negative by 28 weeks, anti-D prophylaxis given and indirect Coomb's test is discontinued. In cases where excesive feto maternal haemorrhage is suspected (e.g. abruptio placenta, caesarean section, manual removal of placenta), Kleihauer-Betke's test is used to quantify the amount offetal erythrocytes in maternal circulation and amount of anti D required is calculated. The approach to a Rh negative non-immunized pregnant women can be schematically represented as follows:



The effective maternal prophylaxis against Rh isoimmunization with anti D was developed by Finn and associates and Freda and co-workers 1963. Anti D immunoglobulin (lgG) when given to mother, coats the fetal erytnfocytes in maternal circulation leading to enhanced

destruction offetal erythrocytes in maternal reticulo endothelial system alld prevents exposure of anti D antigen sites to maternal immune system.

Each dose of anti D contain's 300 microgram of anti D which neutralises about 15 ml of fetal erythrocytes (30 ml of cord blood). Anti D (300 microgram) is given routinely to all Rh negative, non-sensitised women within 72 hours of delivery and it prevents Rh isoimmunizatioil in 98%of the cases. Routi ne antenatal administration of anti D given at 28 weeks along with the second dose at immediate post, partum period (within 72 hours) brings down the Rh sensitisation to less than 0.07%. Late antenatal administration of anti D at 28 weeks is not routinely practiced in our country. It is recommended that the need and the amount of Anti D to be given in the immediate post partum period be judged by quantifying feto maternal haemorrhage. About 2% of Rh negative women undergoing spontaneous abortion, 5% of those undergoing medical termination of pregnancy and 6% of those undergoing amniocentesis have a chance of Rh isoimmunization. Hence, anti D prophylaxis is routinely given to all Rh negative women undergoing spontaneous abortion, medical ten.nination of pregnancy and amniocentesis.

Anti-D prophylaxis may not be protective against Rh isoimmul1ization when amount of fetal erythrocytes in maternal circulation exceed 15 (Feto maternal haemorrhage exceeds 30 ml). Excessive feto maternal haemorrhage (happens in Abruptio placenta, manual removal of placenta, manual removal of placenta, Caesarean section etc. In such cases it is necessary to quantify the exact amount of fetal erythrocytes in maternal circulation which is done by using Kleihauer-Betke test (Acid elusion test). The principle behind Kleihauer-Betke test is that when treated with acid the maternal haemoglobin will be driven out of maternal erythrocytes, giving them 'ghost' cell appearance, which does not happen with fetal erythrocytes. Fetal erythrocytes identified by Kleihauer-Betke test can be quantified and dose of anti D can be calculated.

Pregnancy in non-immunised women is allowed to go to term - fo spontaneous labour. They have to be induced if they go beyond 40 weeks. After delivery, cord is clamped immediately after birth to the fetomaternal transmission cord. Blood is collected and sent for blood group (ABO, Rh), coumb's test, Hb and serum bilirukin. Anti D (300 ug) is given I/m as early as possible within 72 hours if baby's blood group is Rh +ve more about anti D therapy is given in the following paragraph.

9.3 MATERNAL INFECTION AFFECTING FOETUS AND NEONATE

Intra uterine infection and vertical transmission to foetus (congenital infection) can occur at any time during pregnancy. Severity of infection depends on their virulence of the organism, route of infection, gestational age at infection and susceptibility of foetus. These infections result in increased perinatal mortality and morbidity and occasionally delayed symptoms and signs. Infection can be viral, protozoal, bacterial or fungal.

9.3.1 Viral Infections

You must know that besides the foectus being affected, the pyraxia caused by any viral infection can cause abortion, preterm labour or start term labour pains. Viral infection can cause congenital defects, abortions, preterm births, IUGR, still births, early or late neonatal complications or even late complications due to perisistence of the virus in various organs. Important viral infections affecting the foetus is given below:

i) Rubella

ii) Cytomegalovirus (CMV)

iii) Herpes Simplex Virus (HSV)

iv) Human Immunodeficiency Virus (HIV)

v) Varicella Zoster (Chickenpox Virus)

vi) Hepatitis B VAs mentioned earlier in about 1 % of pregnancies areirus (HBV)

vii) Human Papilloma Virus (HPV)

1. Rubella

It is a RNA Virus. After an infection, the immunity acquired is long. Infection may be silent or clinically evident. The clinical features are faecial risk, postauricular lymphadenopathy, flu like symptoms, arthralgia or arthritis of small joints. The incubation period is 2-3 weeks. Ig M antibodies appear almost with onset of symptoms and tests for one month.

If infection occurs in early pregnancy, the risk of foetal infection is very high. The pregnancy may end in abortion or the foetus may be born with congenital rubella syndrome i.e. catarect, deafness and congenital heart disease. Psychomotor retardaction, foetal and neonatal growth retardation and hepatosplenomegarly may be present. Presence of IgM suggests current or recent past infection. Ig G persists life long. Mid-trimester infection in mother may cause psychomotor defects after birth like spasticity and mental retardation.

You must have received by now that with maternal infection in first trimester, MTP should be offered. MMR vaccination at 9 months age the girl child is protected from future infection. When not sure of MMR vaccination, school girl in their teens may be vaccinated routinely against rubella. Those who have no antibiotics may be given vaccination but should avoid pregnancy for 3 months.

If no antibodies are found during pregnancy, the mother should protect herself from exposure to rubella infection and get vaccinated in the postpartum period.

2. Cytomegalovirus (CMV)

CMV infection causing intrauterine infection is an important infectious cause of mental retardation and congenital deafness. It is a DNA virus once infected, the virus persist in the body and cause recurrent infection. The virus is extracted through saliva, urine, cervical sacretionsl and semen. Many women are asyomptomatic during primary and recurrent infections. Primary maternal infection may present as severe fatigue, malaria, lymphadeno pathy and hepatosplenomagcli. During primary maternal infection, foetus effected by vertical transmission through transplacental route. Infection can be acquired during vaginal delivery by contact with maternal secretionand during breast feeding through breast milk.

Primary infection may produce great CMV disease in the body. In this, there is hepatosplenomagali, thromlocytopenia with petechiar and purpura; hepatitis with ichems, pneumonitis and chrogetinitis. Abnormal neurological findings may be microcapheli, aplesia of various parts of brain and mocriphthelmia. IUGR is common. Intracraniel calcifications may be seen, late requate of CMV infection is deafness, mental retardation and visual defects. In recurrent infection, because of protection by maternal antibodies, chances of baby being affected is less and sequale is also less severe.

3. Herpes Simplex Virus (HSV)

It is a DNA virus and like CMV has the ability to persist throughout life, with recurrent infection. Though HSV-II affect genital tract and HSV II affects genital tract and HSV I orsphareyeal areas, due to orogenital contact and contact by fingers to mouth and face, neonatal infection can occur with both HSV II and I, but infection with HSV II is more common. Adult infection may be asymptomatic or present with genital lesion. The lesions are seen in labia and other parts of vulva. Blister like lesions with interior pains are present. Other non-specific genital tract lesions are cervicities, leucorrhoea and pelvic pain.

Dysuria and/or haematuria may be present. Shedding of virus occur between one week and 3 months of appearance of lesions.

Primary infection cause abortion due to toxamia or foetal infections. Transplacental infection is not common. Primary infection after 20 weeks of gestation is anocited with inhered preterm delivery. Intrapartum infection occurs while delivering through birth passage on contact with maternal secretions containing virus. Again infection is more with primary infection. Neonatal infection can occur by contact with infected care given. Neonatal infection is localized to skin, mouth and eyes. Babies with disseminated infection usually have symptoms of lethargy, irritant and appear between 9-11 days. This is followed by seigures, coemplopathing, heart failure, liver involvement and death.

Acyclovir is the treatment for primary or recurrent infection. It can be given during pregnancy safely though not yet fully approved. If ……. Lesions are present at the time of labour, caesarean delivery is advised, if no lesions are present, vaginal delivery is allowed.

You must have realized by now that CMV and HSV cause recurrent infection.

4. Varicella Zoster (Chickenpox) Virus

It is a DNA virus, once infected, it give life long imunity. When infection occurs in a pregnant women, it causes significant maternal and foetal morbidity and mortality. The foetus is infected through transplacental route. You already know about presentation of chickenpox hence not described here. Infection is severe in adults as compared to children. When it affects pregnant mothers, it becomes serious with development of pnemonia, which occurs 2-3 days after cutaneous lesions appear. They may develop adult respiratory distress syndrome (ARDs). A rare complication is maternal encephalitis. Preterm labour congenital and preterm delivery and herpes zoster are more common.

Congenital infection is diagnosed when the symptoms develop before 10^{th} day after birth or when born with developmental abnormalities such as varicella embryopathy. When mother has infection in later third trimester and new born develop varicella, the infection is severe with pneumonitis, hepatitis and dineminated intravascular coagulation it is seen that belries born 5 days or more after maternal disease develops are protected and are also those babies developing neonatal varicella between birth and 5 days of age. Mortality is more in those babies who develop rash between 5-10 days after birth. After 10 days of birth, neonatal infection that develop is mild.

Women who are exposed to varicella infection can be protected by giving varialla zoster immunoglobulin (VZIG) in a single dose (IM ng) within 4 days of exposure. Varicella appear, watch for complications. Those who develop complications are admitted in the hospital and treated with acyclovir. Women with varicella in late 3^{rd} trimester should be monitored for preterm labour and given together if they have excessive uterine contraction to delay labour by at least 5 days after the onset of maternal lesions so that foetus is protected by maternal antibodies.

Hepatitis B: You may refer to Block 1, Unit 4, Section on Joundice.

HIV: Again you may refer to Block 5, Unit 22 on RTI and STJ.

Parvovirus B_{19}: Parvovirus B_{19} is a DNA virus. It may be arymptomated or Infection cause arthrelgia in adults and may have a role in development of rheumatoid arthritis. It may also cause haemolytic anaemies. This virus is important to maternal care providing since infected mothers transmit to foetus causing non immuno hydrops foetalis. Foetal loss is higher when infection occurs before 20 weeks of gestation.

Human Papilloma Virus

It is sexually transmitted in women. Baby gets the infection during vaginal delivery while passing through infected birth canal.

In the adults, HPV causes condylomatous lesion over vulva, vagina and cervix. Neonatal infections cause laryngeal papillomas. In children having papilloma of larynx, infection was 30% of mothers. Laryngeal papillomas are caused by HPV type 6 And 11 which also cause genital infection. Maternal infection is diagnosed by southern blot bybridization, papsmear and colposcopy.

The aim of treatment is to reduce pain and bleedings, prevent secondary infection and obstructed labour and prevent infection of neonate during delivery. Topical podophyllum and 5FU are contra indicated in pregnancy. Surgical exusion, cryosurgy and laser surgery or electroceutery is the management during pregnancy.

9.3.2 Protozoal infection

Protozoal infection of mother that can cause congenital infection are

- Toxoplasmosis—toxoplasma gondii
- Malaria—plasmodia

You have already read about malaria in Block 1, Unit 4 hence only toxoplasmosis will be dealt in this section.

Toxoplasma

As already mentioned above, it is a protozoal infection. Cat acts as host. Infection may occur from ingestion of new or partly cooked meat, direct contact with contaminated infection may be asymptomatic or produce mild symptoms, such as fatigue and lymphadenopathy. Congenital transmission of parasite may occur during acaquired acute maternal infection rebuilding in transmission of paraste may occur during acquired, acute maternal infection rebulting in abortions, still births or congenital toxoplasmosis with characteristic cerebral calafication, chorioctinitis, hydrocephali or nucrocephate. Infected asymptomatic neonate may develop mental retardation, visual impairment and other neurologic sequale late in life.

Acute toxoplasmosis is diagnosed by presence of Ig M antibodies and sabin Feldman sye test. Dye test is not commonly used in our country. Ig G antibodies persists and denotes past infection. Heating meat thoroughly during cooking prevents infection. Treatment is by revomycin during pregnancy.

9.3.3 Bacterial Infection

You will be reading about 4 bacterial infection in mother affecting the foetus and neonate. They are

- Syphilis
- Gonococci
- Chlamydia
- Group B streptococci (GBS)

Syphilis

You are already aware that syphilis is caused by a spirochete called Ireponema pallidum primary, secondary, latent and tertiary syphilis are different stages of disease progressing from the beginning of infection. It is a sexually transmitted disease. The test routines done to diagnose to disease is VDRL in both partners. Reternal primary and secondary syphilis can be diagnosed by demonstration of organism on dark ground illumination. T. Pallidum can cross the placenta and infect the foetus at any time during pregnancy. The infection can result in stillbirth, preterm labour, IUGR, foetal hydrops and congenital syphilis. Baby may have vascular bullons lesions on the skin mainly palm and soles or look healthy at birth. Symptoms and signs appear in 3-4 days after birth. Iritis, signs of meningeal irritation, rhinitis, pharyngitis generalized lymphadenopathy and splinting of arms and lep due to arthralgia may be present. Acpato splanomyali with icterms may be seen. Rash appears all over the body. A symptomatic babies may develop the disease later in life. The more recent the maternal infection, the more severe the congenital disease. Severity also depends on the gestational age of foetus at the time of infection. Syphilis is treated by bezathina pericillin G NACO guidelines for treatment of syphilis is given in Block 5, Unit 22.

Gonococci

Gonorrhoeal infection in mother is often asymptomatic. Infection is transmitted to the baby during labour and gonococcal ophthalmia develop in 3-4 days. Routine prophylaxis against ophthelmia neonatorum is not practiced any longer. Only when symptoms and signs develop, treatment is started. Mother and her partner is also treated. Refer to block 5, unit 22 for more details.

Chlamydia

Chlamydia trachomatis is the organism and it is sexually transmitted. In non pregnant woman, it may be asymptomatic or causes symptoms and signs like frequency and burning mictorition, pain and enlargement of bartholis gland and mucopia rulent cervicitis. When upper genital tract is involved, it causes PID and infertility. LGV (lymphogranuloma oenerum) is another type of lesion caused by chlamydia. Infected mother during vaginal delivery may transmit infection to the bay and neonatal conjucturitis result. Pneumonia with prominent respiratory symptoms may follow in some cases. Chlamidial infection is diagnosed by Elisa test and since the test is expensive, it is not commonly done. For more, refer Unit 22 of Block 5.

Group B Streptococci (GBS)

Group B Streptococci causes severe congenital infection and also choroamnionitis, post partum endometritis, wound infection and sepsis in mother and is an important cause of intrauterine asphysia. High risk mothers likely to transmit the infection to baby are term of preterm rupture of membranes, preterm labour, intrapartum fever, prolonged induction and repeated pelvic examination. Early neonatal sepsis occurs and has a high moratlity rate. Universal screening of al pregnant mothers is not recommended in our country. Prophylactic antibiotic. In ampicillin 500 mg b his till delivery followed by oral route for 7 days) is administered to all high risk woman.

Fungal (candida Albicens)

Candidiasis is caused by c allreccans and is an infection of skins mucosa and rarely of internal organs. It is an important opportunist endogenous infection. Skin lesions are seen in folds of skin. The sites are groin, perineum, axillae and infra mammory folds where skin is macerated by perspiration. Nails may be affected when hands and feet are frequently immersed in water. Mucosal lesions are vaginitis seen frequently in pregnancy, diabetes millitis women on oral pills or steroid therapy, etc. you may refer to block 5, unit 19 and 22.

During vaginal birth, the baby may acquire infection. Oral thrush follows characterized by creamy white patches on buccal mucosa and tongue. Read more about it in problems of newborn.

Check Your Progress 2

1. List the viral infection that produce (a) life long immunity and (b) those that cause recurrent infection.
2. How can you prevent congenital rubella infection?
3. a) Parvovirus B_{19} infection in mother cause ……..

   b) Human pepilloma crus cause …….. in the neonate.
4. list the organisms producing neonatal congunctiontis.

9.4 PAIN ABDOMEN DURING PREGNANCY

You may be already aware from your past experience that pain in abdomen during pregnancy is a common symptom. Heartburn is the commonest problem. Pain could be due to obstetrical, gynaecological, medical or surgical causes. In advanced pregnancy, diagnosis or non-obstetric causes in difficult. You must remember while counseling that laparotomy for other abdominal condition is never combined with caesarean section unless there is an obstetric indication.

9.4.1 Causes

Causes can be many. Pains may be caused by physiological and pathological reasons. Some of the causes are not life threatening while other are life threatened and need urgent treatment. The causes of pain abdomen given below:

1. Pregnancy related
   - Physiological
     - Round ligament pain
     - Braxton-thick contractions
     - Severe uterine forsion (when dextrototation is aggrevted and change in position of mother relise the symptoms)
     - Miscellaneous pain due to uterine muscle stretching
   - Pathological
     - Early pregnancy

— Abortion including induced abortion

— Ectopic pregnancy

— Cystitis

— Retention of urine

â Late pregnancy

— Retention labour pain

— Severe preeclampsia

— Accidental haemorrhage

— Rupture uterus

— Acute hydramnios

— Acute pyelitis/pyeloneptisitis

2. Incidental causes

l Physiological

â Heart burn

â Excessive vomiting

â Constipation

l Pathological

â Gynaecological

— Red degeneration in forbroid

— Torsion of ovaries cyst

â Surgical

— Acute aprendicitis

— Acutre Cholecystitis, gall stones

— Urinary stones

— Intestinal obstruction

â Medical

— Acute pencreatitis

— Reptic ulcer

— Colitis

— Diarrhoea, dyscentry

9.4.2 Diagnosis and Management

Take a detailed history and do a thorough physical examination. Make a list of differential diagnosis from the history and clinical feelings if necessary, review the history and rule out life threatening conditions. Try to make a provisional diagnosis. Observe the patient closely. Usually the clinical picture become clear and a diagnosis can be made in a few hours. The clinical picture and diagnosis of obstretric conditions has already been dealt with in their respective units. Diagnose of red degeneration of fibroid is not difficult. When pain and tenderness is localized in nature not spreading to the other areas of uterus and there is no sign of shock. Fibroid may have been diagnosed previously before pregnancy. This can be elicited from history. It could have been diagnosed earlier in this pregnancy on clinical examination or by ultrasound. Diagnosis of torsion of ovarian cyst is difficult clinically but USG will help in diagnosis. Appropriate laboratory investigation and USG help in making the diagnosis in other conditions.

Except the physiological causes which can be treated symptomatically all other cares are referred to referral centres for investigation and treatment after instituting primary care (see unit 15).

Check Your Progress 3

1) List four causes of pregnancy related physiological causes of pain abdomen.

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2) List four life threatening obstetric conditions causing pain abdomen.

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3) Describe diagnosis of red degeneration of fibroid during pregnancy.

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9.5 LET US SUM UP

In this unit, you read about Rhesus isoimmunisation, pathology of haemolytic disease of newborn screening of pregnant woman for Rh compatibility against Rh isoimmunisation. You also read about various organisms causing intrauterine infection of foetus, the complications due to infection and how to diagnose them. Pain in abdomen is a common symptom during pregnancy. Many other obstetric, gynaecologic, surgical and medical causes of pain abdomen during pregnancy including life threatening condition presenting with pain abdomen have been described.

9.6 KEY WORDS

RNA—Ribonucleic acid

DNA—Deoxyribonucleic acid

Congenital Infection—Infection acquired during intrauterine life

Vertical Transmission—Transmission from mother to foetus

IgM antibodies—Antibodies produced during active infection

IgG antibodies—Antibodies persisting after infection

Topical Application—Local application on the affected part

9.7 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) Rh factor is rhesus antigen i.e. a type of antigen found in red bood cells of human beings identical to those found in rhesus monkeys. A person who lacks this antigen is called Rh negative.

2) 5-10%

3) Positive fetal red blood cells cross placenta and sensitise mother who in turn produce antibodies which cross placenta and cause fetal haemolysis and its consequences.

4) — Variable antigenicity

— Insufficient amount of antigen

— Variability of maternal response

— Protection due to ABO incompatibilities

— Insufficient antibody transfer across placenta.

Check Your Progress 2

1) a) Rubella, varicalla zoster

b) Cytomegalovirus, Herpes Simplex Virus

2) Giving MMR vaccination 9 months of age

– Immunizing school going girls who have no antibodies

– By protecting mothers from exposure to rubella infection and immunizing mothers who have no antibodies in the post partum period.

3) i) hydrops foctalis ii) larigngeal papillomes

4) 1) gonococcus 2) chlamydia trechomatis

Check Your Progress 3

1) i) Round ligament pain

ii) Braxton–Hicks contraction

iii) Severe uterine torsion

iv) Miscellaneous pain due to …… muscle stretching

2) i) Ectopic pregnancy

ii) Accidental haemorrhage

iii) Severe preeclampsia

iv) Rupture uterus

3) Diagnosis of red degeneration of fibroid during pregnancy

i) n/o pain in abdomen which is located in there is no sign of shock

ii) n/o diagnosed fibroid uterus

iii) Diagnosis of fibroid during ……. Antenatal examination or during USG exam

iv) Diagnosis of fibroid by USG when the women has come with pain abdomen during pregnancy.

9.8 FURTHER READING

Arias F (Indian Reprint 1997) Practical Guide to High Risk Pregnancy and Delivery, Second Edition, Harcourt Brace and Company Asia PTE Ltd.

Miller A, Govan A and Callander R (1989) Obstetrics Illustrated, 4th Edition Churchill Livingstone.

Bennet V.R., Brown L.K.(2000) Myles Textbook for Midwives, 13th Edition Churchill Livingstone.