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**Senior lecturer 2016-2017**

**Jaundice in pregnancy**

**Objective**

**1. Student understand the causes of jaundice in pregnancy**

**2. Student learn how to manage patient with jaundice**

Jaundice in pregnancy, whilst relatively rare, has potentially serious consequences for maternal and fetal health. It can be caused by pregnancy or occur intercurrently

# Causes of jaundice in pregnancy:

***A. causes not specific to pregnancy (occur in pregnant and not pregnant women):*** like acute and chronic viral hepatitis, autoimmune liver disease, hemolytic anaemia

***B.Causes of jaundice specific to pregnancy include:***

* [Pre-eclampsia](http://www.patient.co.uk/search.asp?searchterm=PRE+ECLAMPSIA&collections=PPsearch) associated with HELLP syndrome (= h aemolysis, e levated l iver enzymes and l ow p latelet count).
* Acute fatty liver of pregnancy.
* [Hyperemesis gravidarum](http://www.patient.co.uk/search.asp?searchterm=HYPEREMESIS+GRAVIDARUM&collections=PPsearch).
* [Intrahepatic cholestasis of pregnancy](http://www.patient.co.uk/search.asp?searchterm=INTRAHEPATIC+CHOLESTASIS+OF+PREGNANCY&collections=PPsearch).

Approximately 3-5% of pregnant women may have abnormal liver function tests.

# Viral hepatitis:

## Hepatitis A:(RNA virus )

**Aetiology**: transmitted most commonly by fecal-oral route by either person to person contact or ingestion of contaminated food or water.

**Clinical features:** can vary from mild non specific anicteric infection to fulminant hepatic failure. Symptoms include fever, malaise, anorexia, nausea, vomiting and abdominal discomfort. Jaundice may be present with dark urine and hepatomegaly.

**Investigations:** raised serum alanine transaminase, bilirubin and anti-hepatitis A IgM antibodies

**Management**: supportive, and complete recovery is the usual outcome (there are no long term fetal consequences). Administration of human serum immunoglobulin may prevent the infection or attenuate the symptoms. A vaccine is available and gives up to 10 years protection.

## Hepatitis B:

**Aetiology:** it is a blood born double strand DNA virus. The virus has three major structural antigens: surface antigen (HbsAg), core antigen (HbcAg) and envelop antigen (HbeAg). Transmission is by body secretions, and thus with sexual contact, blood transfusion, intravenous drug abuse, and perinatal transmission.

**Clinical features:** it is often asymptomatic. Non specific symptoms and signs include: nausea, vomiting, fatigue, photophobia, headache, right upper abdominal pain, diarrhea and jaundice. Physical examination is often normal, although hepatomegaly, splenomegaly and lymphadenopathy may be present.

It is usually self limiting, fulminant hepatic failure occurs in 1% of cases. Infected neonates and young children are more likely to develop chronic infection.

**Investigations:**

1.hematological tests: leucopenia, and may show anemia and thrombocytopenia

2. liver function tests: reveals highly elevated serum aminotransferases (SGOT, SGPT)and bilirubin

3. Diagnosis of infection by the presence of HbsAg

4. The presence of HbeAg indicates that the patient is highly infectious with viral shedding into the blood stream.

5. After HbeAg disappears, antibodies to e Ag appear and indicate low infectivity

6. Resolution of the disease is indicated by the disappearance of HbsAg and the appearance of surface antibodies

**Management:**

Treatment is supportive. Patient should be monitored to ensure fulminant liver disease does not develop. Serological testing should be repeated 3 months after infection to check the virus is cleared from the blood.

Long term sequelae can be the development of chronic hepatitis, cirrhosis and hepatocellular carcinoma.

## In relation to pregnancy:

***1. antepartum***: all women are routinely offered testing for hepatitis B antibodies at their booking visit, if testing positive then:

a. Determine the infectious state of the patient by serology

b. testing of the partner for hepatitis

c. testing for other sexually transmitted disease including HIV

The presence of hepatitis B does not seem to pose additional risk for the pregnancy.

***2. Intrapartum:*** keep membranes intact as long as possible, fetal scalp electrode and fetal blood sampling should be avoided. Use forceps rather than ventouse for instrumental delivery (to minimize the risk of fetal infection)

***3. Postpartum***: hepatitis B immunoglobulin to those neonates born to high-infectivity mothers (HbeAg), hepatitis B vaccine to the neonates born to low-infectivity mothers (the presence of e antibodies). There is no contraindication for breast feeding.

## Hepatitis C:

Transmitted sexually, perinatally and with intravenous drug abuse.

**Clinical features:** are non-specific. Higher risk of chronic hepatitis and hepatocellular carcinoma than hepatitis B, Cirrhosis develops in 20-40% of patients.

Management during labour is the same as for hepatitis B

## Hepatitis D:

Requires co-infection with hepatitis B virus. Mainly in intravenous drug abusers.

## Hepatitis E:

It is transmitted by fecal-oral route. During pregnancy, the risk of fulminant hepatic failure is about 15% with a mortality of 5%.

# Obstetric cholestasis:

it is an uncommon condition, occurs in 0.5-1% of pregnancies. The cause of obstetrical cholestasis is unknown, but it probably occurs in genetically susceptible women..

**Clinical features:**  it presents most commonly in the third trimester, with generalized itching (but no rash) worst on the palms and soles. Anorexia, pale stool, dark urine and steatorrhea. Jaundice is unusual.

**Obstetric complications:**

**A. Maternal:**

1. Postpartum hemorrhage related to vit. K deficiency due to malabsorption of fat

2. Premature labour

3. steatorrhea

**B. fetal complications**:

1. meconium stained liquor

2. Fetal distress (CTG abnormalities)

3. Rarely, intrauterine death, the risk increases towards and beyond term (the cause is unknown)

**Differential diagnosis:**

Viral hepatitis, autoimmune hepatitis, extra hepatic obstruction from gallstone, preeclampsia HELLP, acute fatty liver of pregnancy, sepsis and drug induced hepatitis.

**Investigations of cholestasis:**

A. maternal: liver function tests (raised transaminases),raised bile acids ( the most sensitive finding), full blood count, clotting profile, renal function, hepatitis serology, autoimmune antibodies, and liver ultrasound.

B. fetal: ultrasound for growth and amniotic fluid volume, CTG (cardiotocography) for assessment of fetal condition.

**Management:**

1. Diagnosis: clinical features and investigations with regular monitoring of liver function and fetal wellbeing

2. Symptomatic relief of pruritus with emollients and antihistamines, ursodeoxycholic acid reduces maternal itching and improves liver function in most women

3. Induction of labour at 37-38 weeks due to the risk of intrauterine death (the risk increases beyond 38 weeks)

4. vit. K should be given to the mother (10 mg orally daily) from the time of diagnosis to reduce the risk of postpartum hemorrhage.

5. Following delivery, liver function will return to normal. Symptoms may return with oral contraceptive pills. Recurrence in subsequent pregnancies exceeds 90%.

**COMPLICATION**

Maternal :( Heamrrhage , preterm labour , steatorrhea )-

Fetal :( Stiibirth , intapartum fetal distress , meconium staining of amniotic fluid) -

# Acute fatty liver of pregnancy (acute yellow atrophy of the liver)

# 

It is rare but very serious disorder occurs in 1 in 10000 pregnancies.

Aetiology

It is associated with disorder of fatty acid transport and mitochondrial oxidation

Risk are 1. First pregnancy 2. Male fetus 3. Preeclampsia 4. Multiple pregnancy 5. Obesity

**Clinical features:** it typically develops in the third trimester mean gestational age of 37 week or within a few days of a stillborn; it shares many features and probably pathophysiology with preeclampsia, may present with abdominal pain, headache, nausea and vomiting. Progressive jaundice, encephalopathy, hypoglycemia, coagulopathy and renal failure may develop.

It may result in maternal death (from encephalopathy or hemorrhage due to clotting defect) and fetal death ( due to maternal liver failure and metabolic disturbances)

Investigation

1. liver function re abnormal

Serum bilirubin increase

Increase in SGOT AND SGPT (up to 7 time )

Alkaline phosphatase increase moderately

Prothrombin time may be increasd

Clotting time increase

Hypoalbomiemia

Renal function test

1. Increase S. creatinine 2. Increase S. uric acid 3. Decrease in glucose level 4. Decease platelet count

**Management:**

1. Diagnosis: clinical features and investigations:

2. Treatment is to deliver the baby as soon as possible

3. Supportive therapy with blood transfusion, fresh frozen plasma, vitamin K, platelets, 50% dextrose to correct hypoglycemia, dialysis may be required in renal failure.

Postnatally liver function usually returns to normal and there is no long term liver dysfunction

Maternal prognosis

* Fulminant hepatic failure
* Ascites
* Acute pancreatitis
* Encephalopathy and DIC