Jaundice in pregnancy

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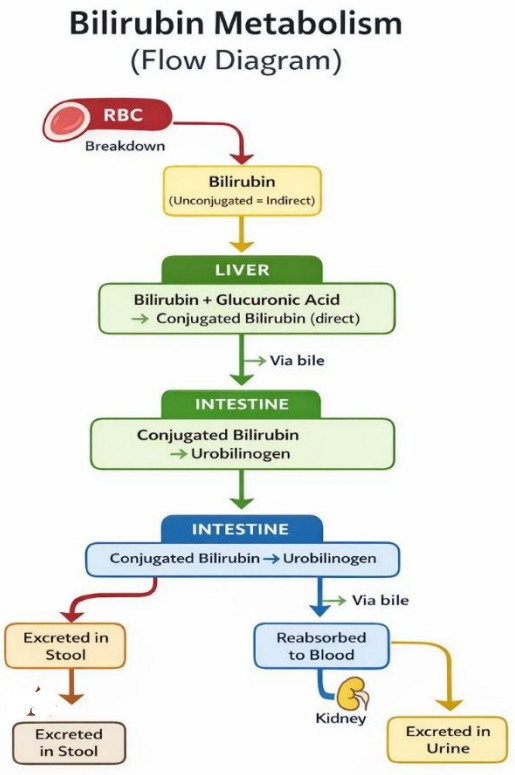


Learning objectives

1. Define jaundice in pregnancy.
2. List the main causes of jaundice during pregnancy.
3. Recognize the common clinical features.
4. Identify maternal and fetal complications.
5. Outline the basic principles of management.

Introduction

Jaundice during pregnancy, medically referred to as icterus, is a clinical condition characterized by the yellowish discoloration of the skin, sclera and mucous membranes. This pigmentation is caused by hyperbilirubinemia, an excess of bilirubin in the blood.



Physiological changes in the liver during pregnancy

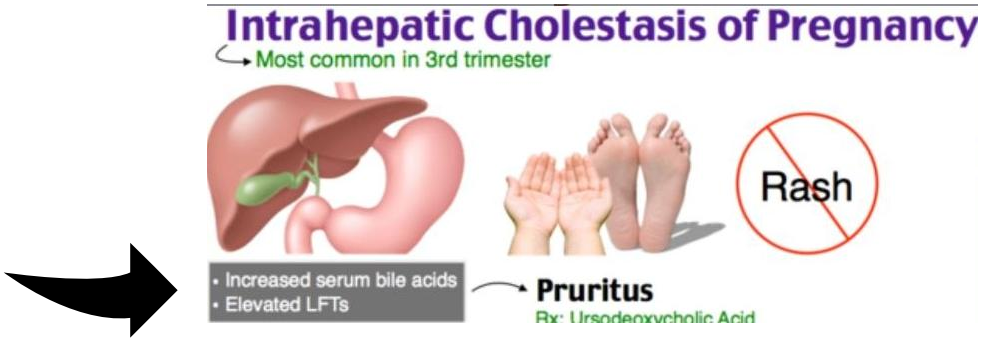
Increased:	Decreased:	No change:
• Blood volume and cardiac ouput rise by 35%–50% • Alkaline phosphatase levels rise threefold or fourfold due to placental production • Clotting factor changes create a hypercoagulable state • **Decreased:** • Gallbladder contractility • Hemoglobin • Uric acid levels • Albumin, total protein, and antithrombin III concentrations	• Gallbladder contractility • Hemoglobin • Uric acid levels • Albumin, total protein, and antithrombin III concentrations	• Liver aminotransferase levels (AST, ALT, GGT) • Bilirubin level • Prothrombin time

Causes of jaundice in pregnancy

UNRELATED TO PREGNANCY	PREGNANCY SPECIFIC
• **HEPATIC CAUSES** • Acute viral hepatitis • Drug induced hepatitis • Chronic hepatitis -viral (HBV, HCV) -Autoimmune hepatitis • Wilson's disease • Cirrhosis of liver • Budd-chiari syndrome **PRE-HEPATIC CAUSES** • Hemolytic anemia **POST-HEPATIC CAUSES** • Common bileduct stone/ strictures • Biliary parasitosis	• Hyperemesis gravidarum • Intrahepatic cholestasis of pregnancy • Pre-eclampsia/ eclampsia • HELLP Syndrome • Acute fatty liver of pregnancy

Intrahepatic cholestasis (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

Maternal complications	Fetal complications
1.postpartum haemorrhage related to vit. K deficiency due to malabsorption of fat 2. premature labour 3.steatorrhea	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

1. Viral hepatitis
2. autoimmune hepatitis
3. extra hepatic obstruction from gallstone
4. preeclampsia HELLP
5. acute fatty liver of pregnancy
6. sepsis
7. drug induced hepatitis

investigation

maternal	**fetal**
○ 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. ○ RBS	○ ultrasound for growth and amniotic fluid volume ○ CTG

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 45- 90%

Acute fatty liver

AFLP is a rare, potentially life-threatening, pregnancy- related disease that affects 1 in 7000 to 1 in 16 000 pregnancies.

The pathogenesis of AFLP is not completely understood. Fatty acid oxidation disorders contribute to approximately 20% of cases of AFLP.



Risk factors

1. AFLP in previous pregnancy
2. first pregnancy,
3. multiple pregnancies
4. male fetus.
5. Preeclampsia and HELLP

Clinical features

- AFLP typically manifests in the third trimester (30-38 WEEK)
- nausea and anorexia, vomiting
- abdominal pain
- polydipsia and polyuria
- jaundice, ascites, coagulation disorders, and confusion develop rapidly and can result in multi-organ failure.

investigation

- Liver function tests: levels of liver transaminases can be **markedly increased**, but it does not reflect disease severity since hepatocytes cannot release transaminases if they have already been destroyed by severe injury. high levels of bilirubin

- RFT: ↑ creatinine
- uric acid markedly elevated
- Coagulation profile: a prolonged (PT), DIC
- RBS: **hypoglycemia.**
- CBC: ↑neutrophils

Treatment

- Immediate delivery
- supportive therapy with blood transfusion, fresh frozen plasma, vitamin K, platelets.
- 50% dextrose to correct hypoglycaemia
- dialysis may be required in renal failure.

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

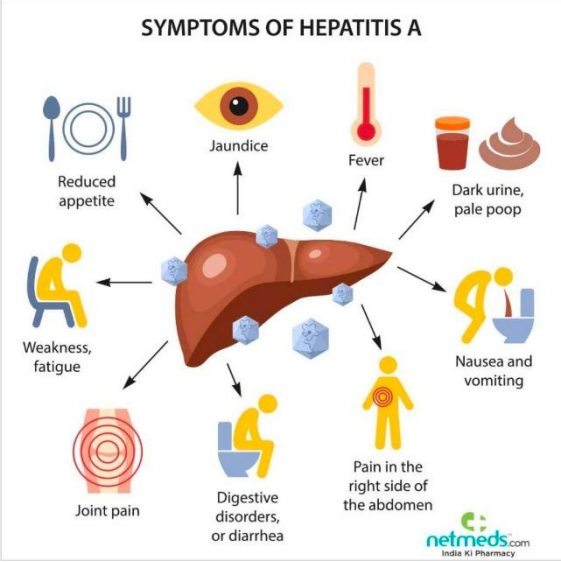
Hepatitis A

Transmission: fecal - oral person to person contact or ingestion of contaminated food or water.

Clinical features

Vary from mild nonspecific 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



Investigation

- Raised serum alanine Transaminase
- ↑bilirubin
- 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.

Nonspecific symptoms and signs include: nausea, vomiting, fatigue, photophobia, headache, right upper abdominal pain, diarrhoea 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



Haematological tests:

1.CBC: leucopoenia, and may show anaemia 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:

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

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)
- Early cord clamping.

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%.