Jaundice in pregnancy

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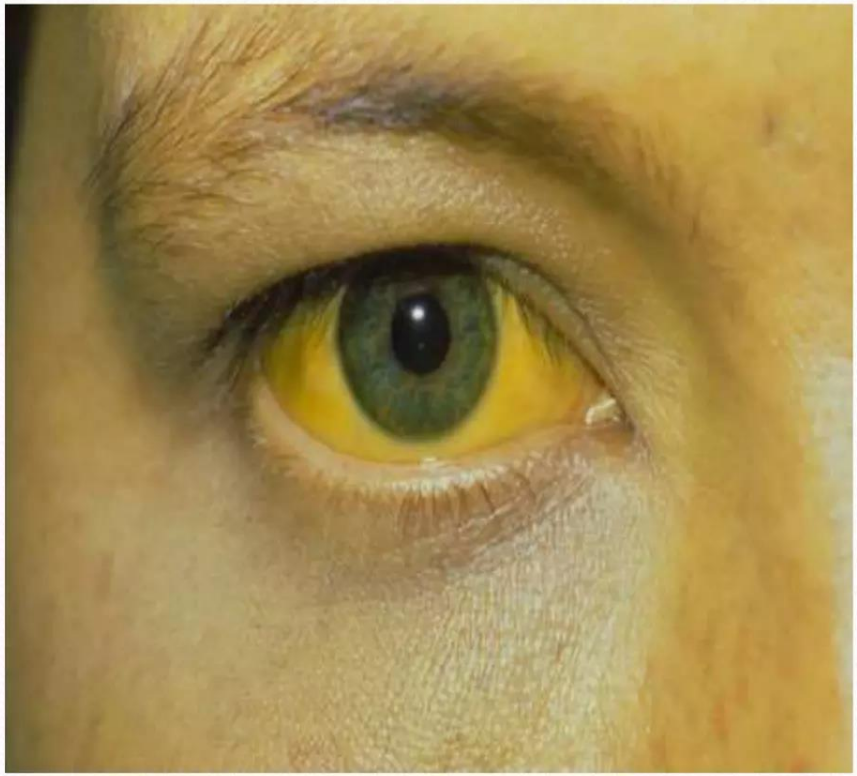
4th year
2023-2024

- Yellow discoloration of skin, conjunctiva, sclera and mucosa associated with rise in serum bilirubin above 2mg/dl

(normal 0.2-01mg/dl)

Latent jaundice:1-2mg/dl

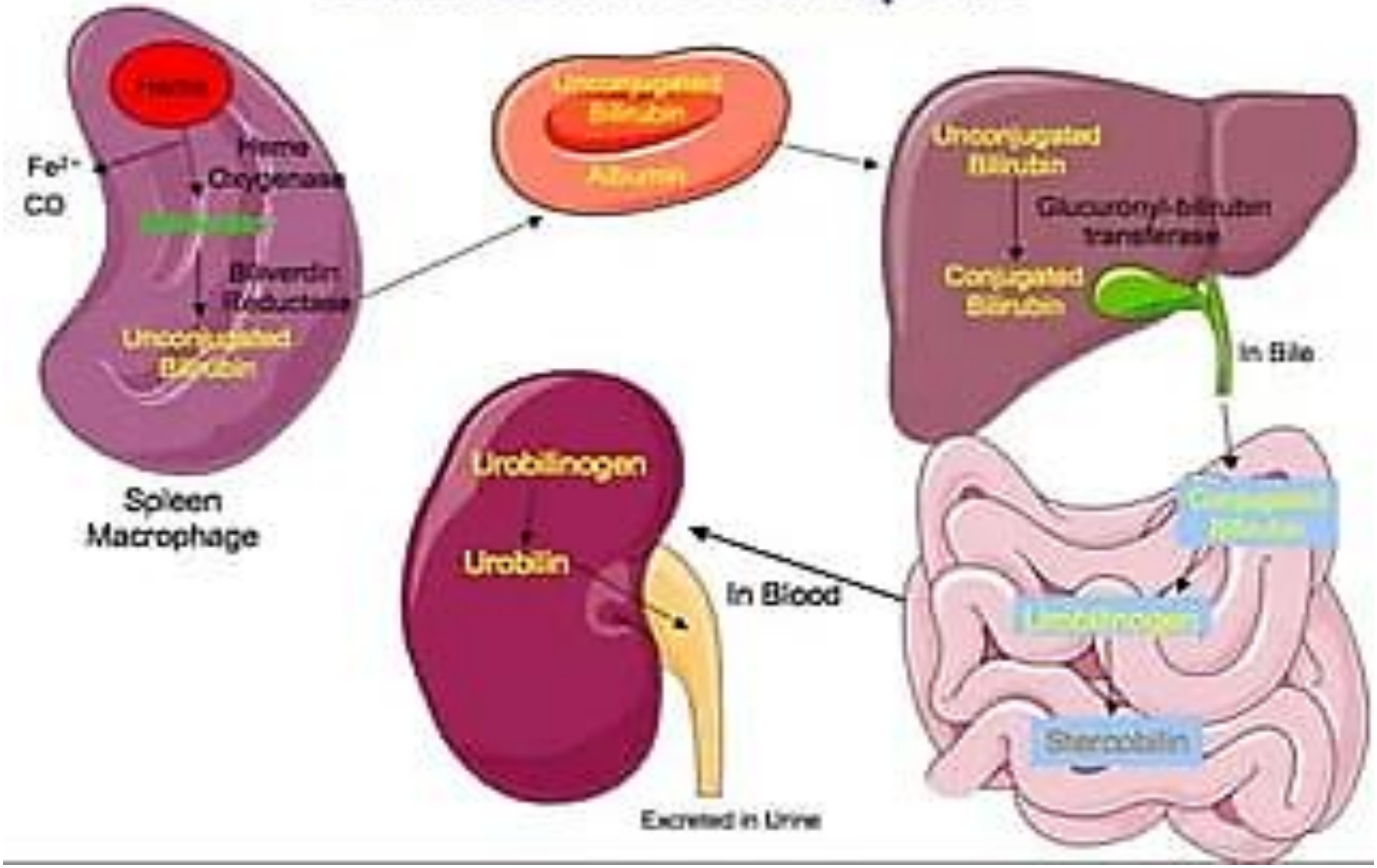


Physiological changes in liver during pregnancy

- Liver is not palpable during pregnancy.
- Sr. protein decrease by 20%. Bilirubin also decrease
- In 3rd trimester ALP level increase up to 2-4 times the normal. It returns to normal in 2-3 month of post delivery.
- ALT and AST level normal, but increase during labour and normalize in 1-2 days postpartum.

- 5’ nucleotides is significantly raised but GGT slightly decrease.
- Increase sr. triglyceride and VLDL. Cholesterol increases up to 2 times the normal.
- 10-15% normal pregnant may have bilirubin level of over 1mg% d/t delayed excretion of bilirubin that may leads to increase incidence of purities in pregnancy.

Bilirubin Transport



Causes of jaundice in pregnancy

A. ***causes not specific to pregnancy:***
like acute and chronic viral hepatitis, autoimmune liver disease, hemolytic anaemia

B. ***causes specific to pregnancy:*** like obstetric cholestasis, acute fatty liver of pregnancy, severe preeclampsia with HELLP syndrome, severe hyperemesis gravidarum leading to impaired liver Function.

Viral hepatitis

Hepatitis A

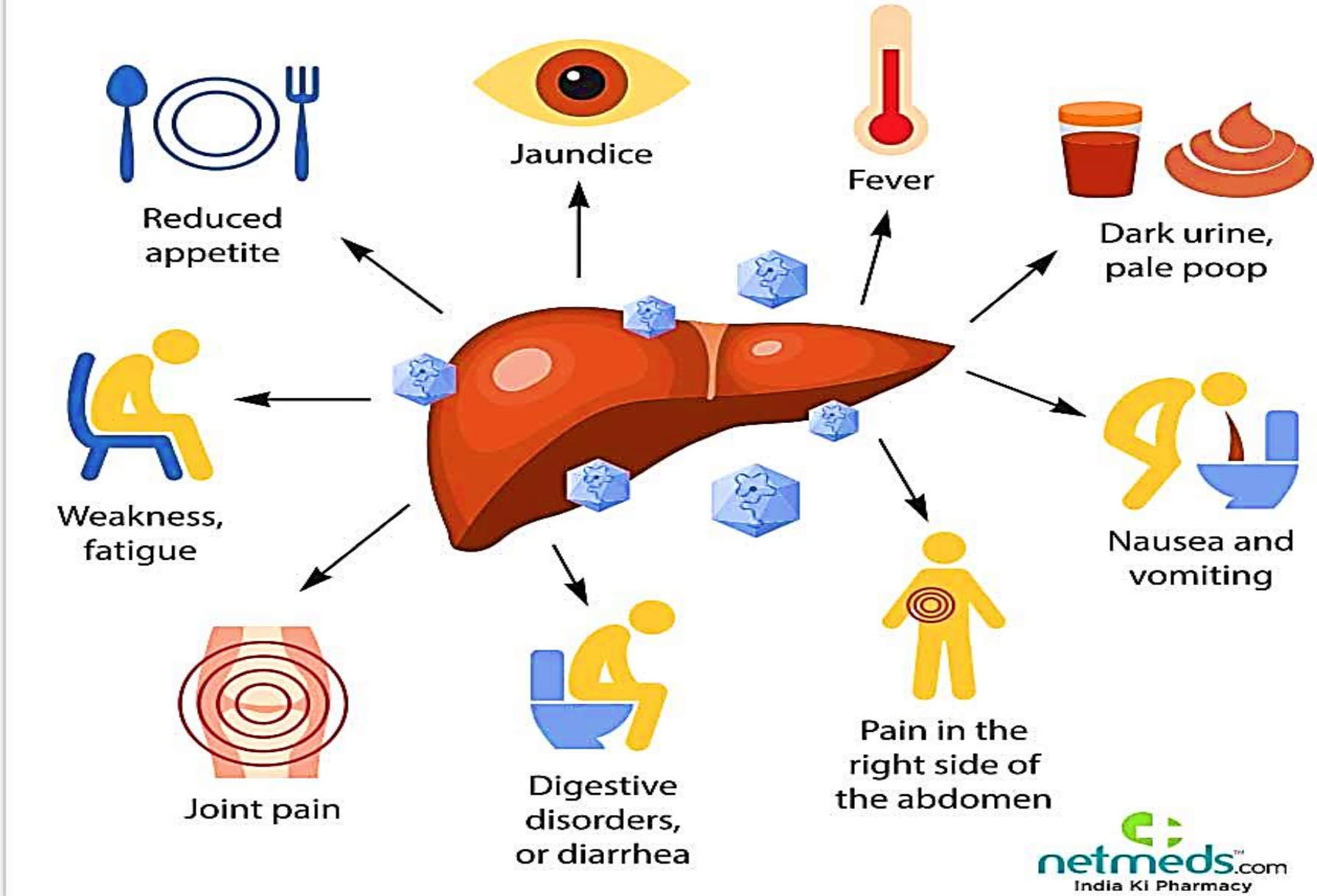
Aetiology: transmitted most commonly by fecal-oral route by either person to person contact or ingestion of contaminated food or water.

Clinical features

can vary from

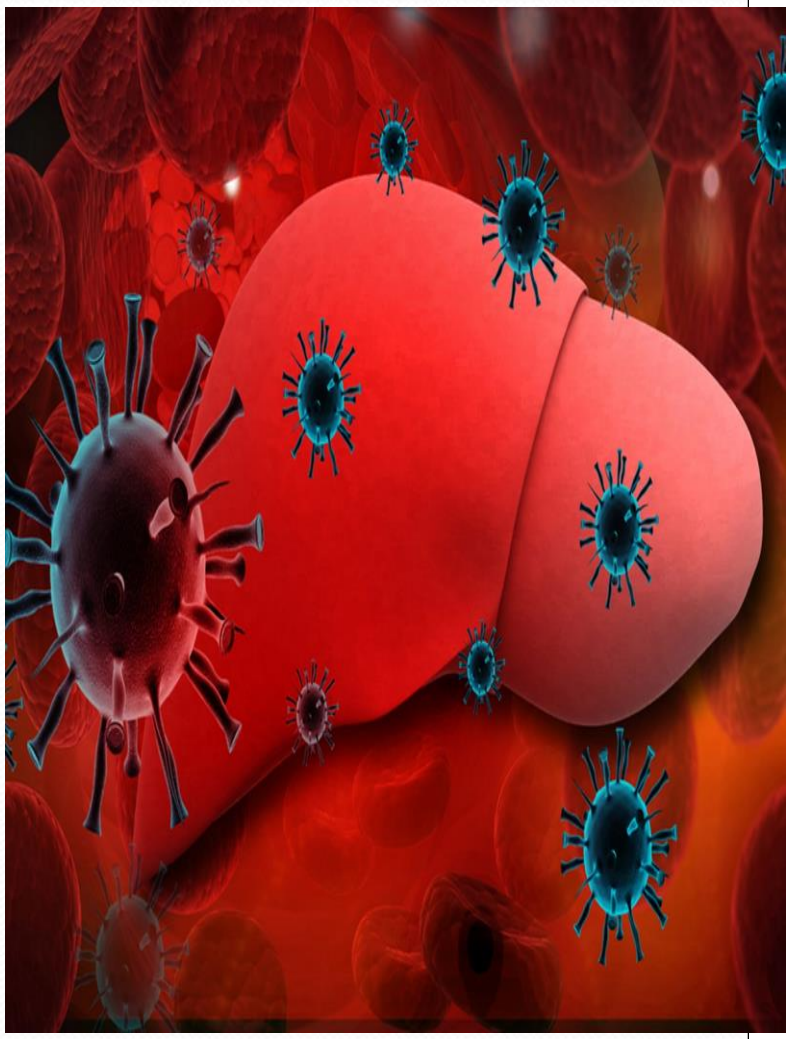
mild non specific anicteric infection to fulminant hepatic failure. Symptoms include fever, malaise, anorexia, nausea, vomiting and abdominal discomfort. Jaundice may be present with dark urine and hepatomegaly

SYMPTOMS OF HEPATITIS A



Investigations:

Raised serum alanine Transaminase, bilirubin and anti-
Hepatitis A IgM antibodies

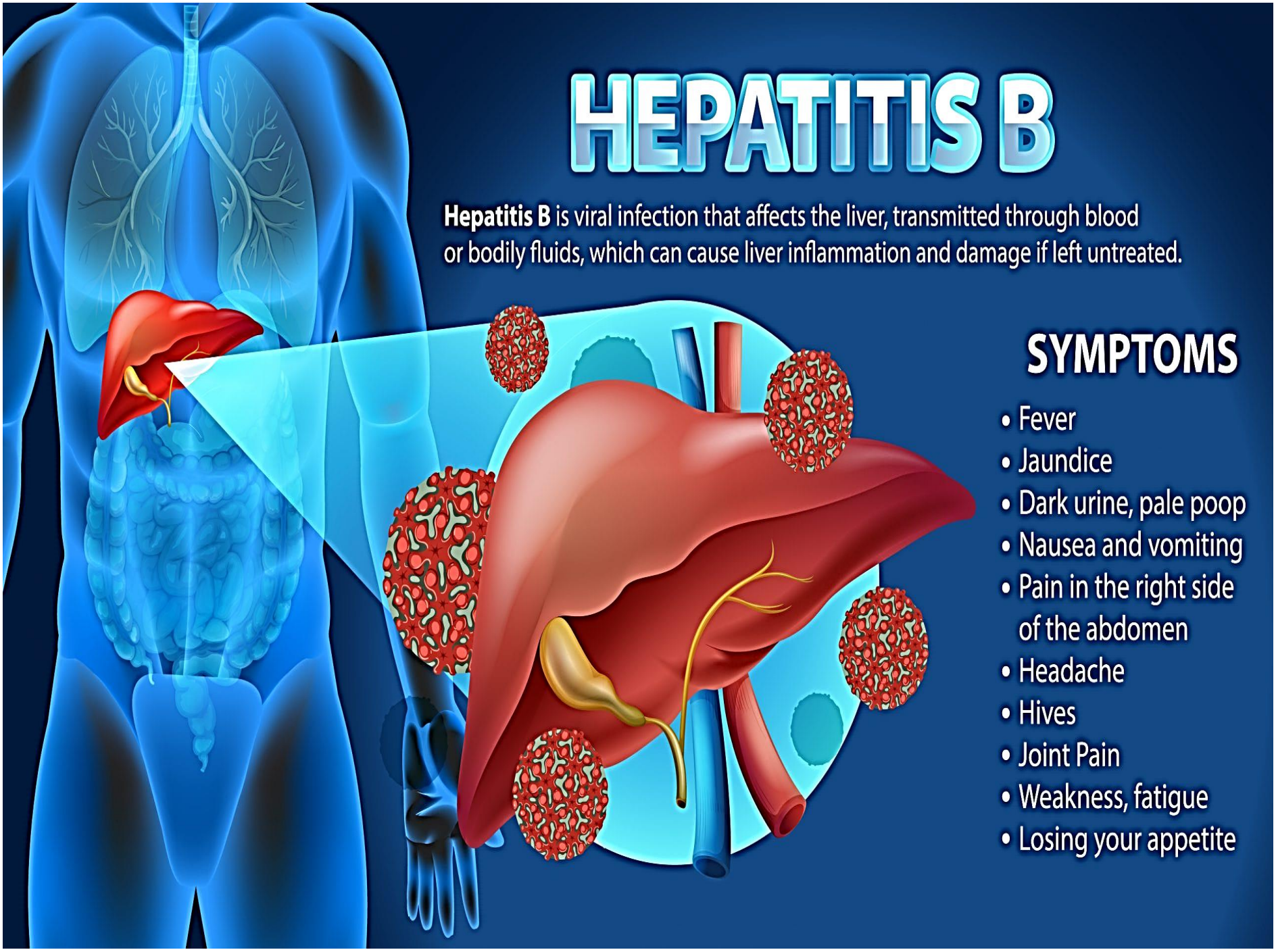


Management: supportive, and complete recovery is the usual outcome (there are ***no long term fetal consequences***). Administration of human serum immunoglobulin may prevent the infection or attenuate the symptoms. A vaccine is available and gives up to 10 years protection.

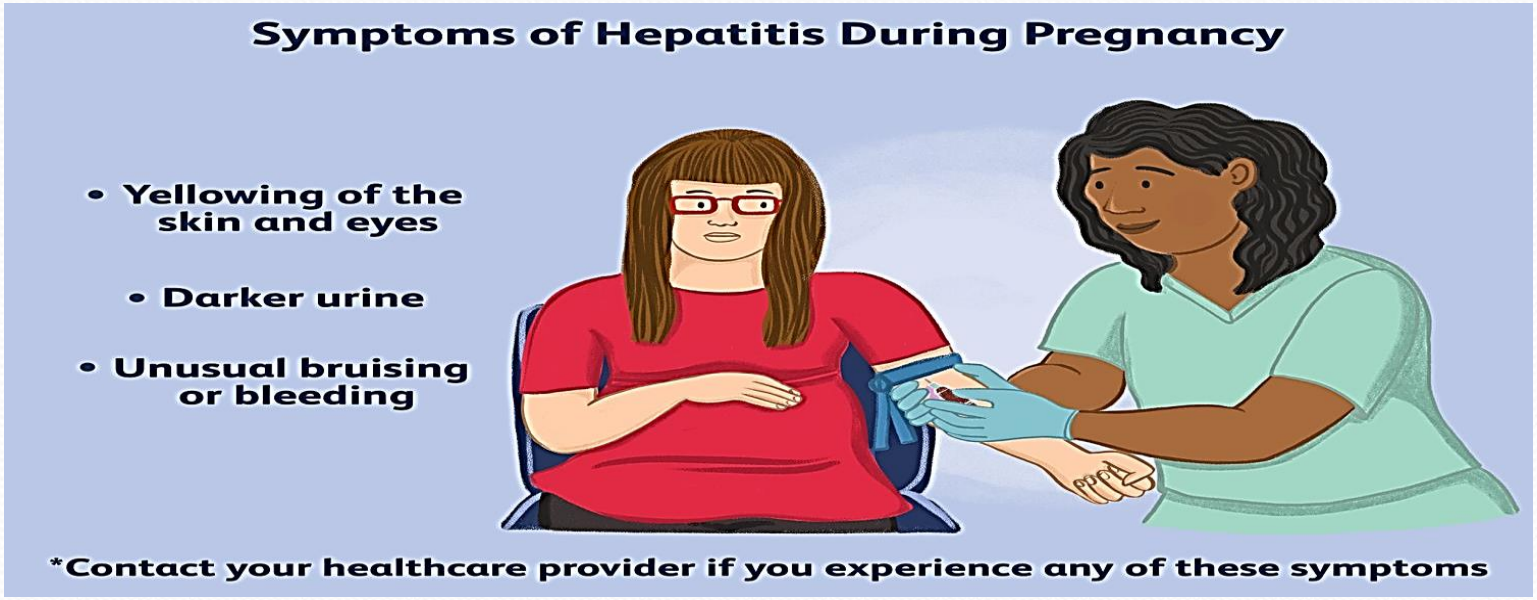
Hepatitis B:

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.


HEPATITIS B
Hepatitis B is viral infection that affects the liver, transmitted through blood or bodily fluids, which can cause liver inflammation and damage if left untreated.
SYMPTOMS
• Fever
• Jaundice
• Dark urine, pale poop
• Nausea and vomiting
• Pain in the right side of the abdomen
• Headache
• Hives
• Joint Pain
• Weakness, fatigue
• Losing your appetite

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___

Hematological tests:

1. 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:

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%

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 .

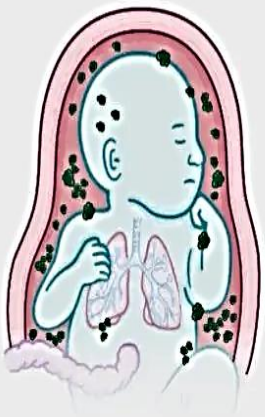
2.fetal distress (CTG abnormalities)

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

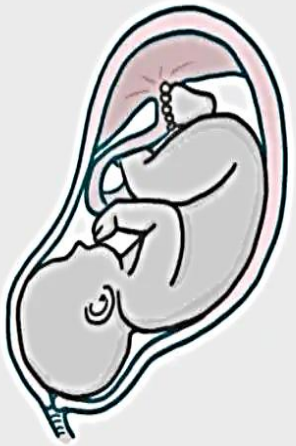
INTRAHEPATIC CHOLESTASIS of PREGNANCY (ICP)

RELATIVELY UNCOMMON DISEASE ASSOCIATED with POOR FETAL OUTCOMES

PRETERM DELIVERY



MECONIUM-STAINED AMNIOTIC FLUID



STILLBIRTH

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.

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)

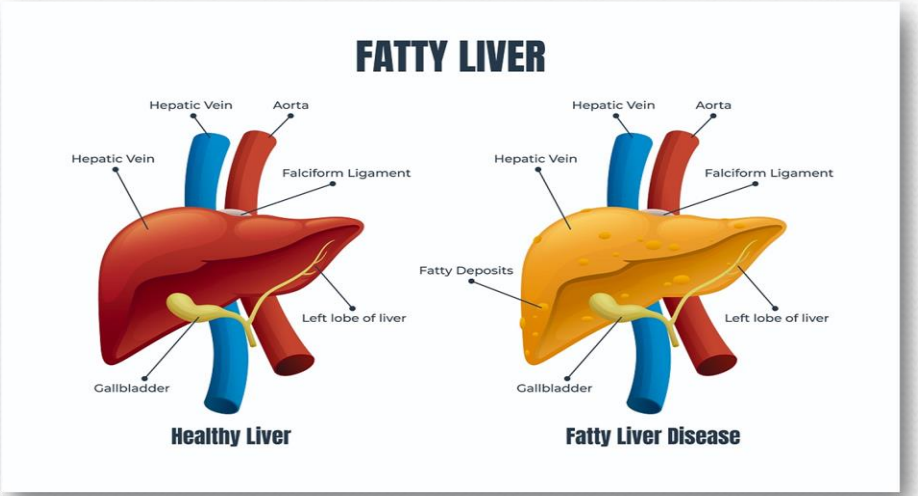
4.vit. K should be given to the mother (10 mg orally daily) from the time of diagnosis to

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%

Acute fatty liver of pregnancy

It is rare but very serious disorder
occurs in 1 in 10000 pregnancies.
AFLP is characterized by micro vesicular fatty infiltration of hepatocytes without any inflammation or necrosis.



Clinical features: it typically develops in the third trimester or within a few days of postpartum, 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)

Risk factors:

1. Primigravida
2. Male fetus
3. Preeclampsia
4. Multiple pregnancy
5. Obesity

HAPPIEST HEALTH

SYMPTOMS OF ACUTE FATTY LIVER OF PREGNANCY

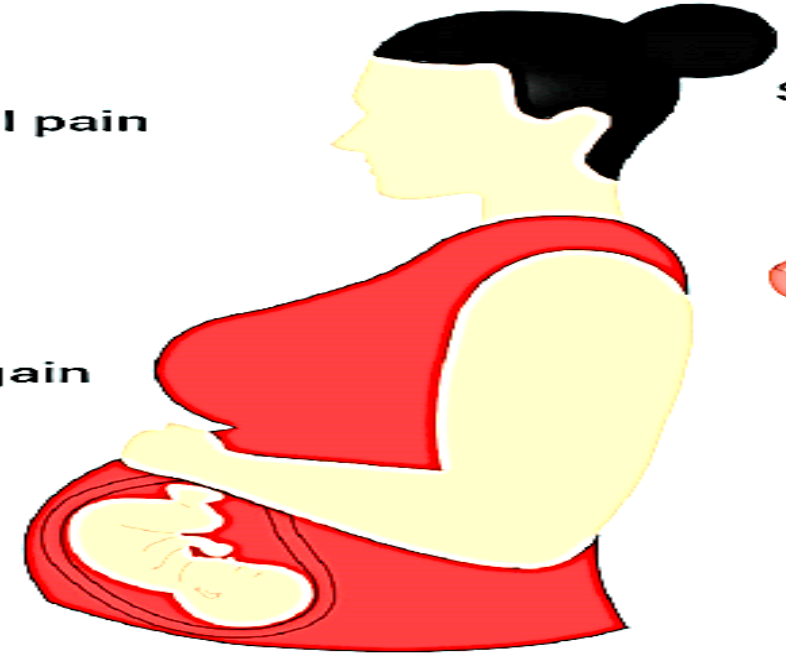
Experts state that the symptoms of this AFLP may include

Vague abdominal pain

Sudden changes in liver function

Slight weight gain

Fatigue



Arthritis

Jaundice

Nausea

Vomiting

Hallucinations

:Management

1.diagnosis: clinical features and investigations

A. liver function tests: markedly impaired

B. renal function tests: impaired and renal failure may develop

C. uric acid: markedly elevated

D.random blood sugar as hypoglycemia may develop

E.coagulation screen as DIC may develop

F.WBC may raise

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.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