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Foreword for Guideline

Pregnancy presents unique physiological changes that can significantly influence the course and management of liver diseases in pregnancy. The intersection of hepatology and obstetrics requires evidence based, multidisciplinary approaches to ensure the health and safety of both mother and fetus. Recognizing the increasing need for clarity and standardization in this area, this guideline on liver diseases in pregnancy has been developed to support health care professionals to deliver informed, compassionate and effective care.

This document brings together current evidence, expert consensus and clinical experience to address common and rare liver conditions encountered during pregnancy including preexisting liver diseases, pregnancy specific hepatic disorders, and management of acute liver failure. Special attention has been given to diagnostic challenges, safe pharmacological interventions and multidisciplinary care strategies

I am grateful to team SOGP and HEPNET for their joint effort to come up with this comprehensive document

Special thanks to Professor Rizwana Chaudhri and Mr. Syed Aftab for publishing this special supplement.

I hope that these guidelines will serve as a valuable resource for clinicians, educators for promoting best practices and improving outcomes for pregnant patients with liver diseases.

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Introduction

Pregnancy is a physiological state that can affect the liver, it may lead to a spectrum of liver diseases that pose risks to both maternal and fetal health.[1] In countries like Pakistan which are amongst WHO's Lower- & Middle-Income Countries (LMIC's) list where access to healthcare may be limited, these conditions can have particularly severe consequences. The Society of Obstetricians and Gynecologists of Pakistan (SOGP) and the Hepatology Expert Network (HEPNET) have collaborated to develop a comprehensive position statement on Diagnosis & Management of Liver Diseases during Pregnancy to address the gaps in context of local practice in Pakistan.

Pregnancy presents a unique diagnostic challenge in clinical practice. The physiological adaptations inherent to gestation sometimes mimic or exacerbate liver disease presentation, demanding meticulous clinical evaluation. Hormonal shifts, hemodynamic alterations, and changes in biochemical markers can mask underlying pathology or create misleading impressions. For instance, elevated estrogen levels can manifest palmar erythema & spider angiomas in cirrhosis. Similarly, hemodynamic changes may worsen pre-existing portal hypertension, while normal pregnancy-induced fluctuations in serum albumin, alkaline phosphatase, and bile acids necessitate careful interpretation.[2] Although pregnancy is a procoagulant state, any deviation from expected ranges for AST, ALT, total bilirubin, or INR warrants immediate and thorough investigation to rule out new or previously undiagnosed liver disease. This document aims to provide clarity and guidance on the diagnosis and management of liver-related problems in pregnancy, ensuring optimal outcomes for both, the mother and child.

Liver diseases during pregnancy include conditions unique to gestation as well as pre-existing acute or chronic diseases of liver that may be associated with pregnancy. Regardless of their origin, these conditions pose significant risks for poor fetal & maternal outcomes.[1] "Pregnancy-specific liver conditions" include hepatic infarction and rupture, acute fatty liver of pregnancy (AFLP), liver complications linked with preeclampsia (including HELLP syndrome: "hemolysis, elevated liver enzymes, and decreased platelet count"), hyperemesis gravidarum (HG), and intrahepatic cholestasis of pregnancy (ICP).[3-5] Global estimates for these conditions are 3% of all pregnancies worldwide, with prevalence varying based on country and racial or ethnic background.[5]

Preconception counseling should be offered to pregnant ladies with chronic liver disease (CLD) to optimize their clinical outcomes. This could offer understanding regarding how pregnancy might influence their condition and the risks that come with it. Medications used in CLD management can be continued safely during pregnancy to prevent clinical decline; however, certain drugs may require discontinuation or replacement, which should be discussed during preconception counseling. This position statement aims to address existing gaps in clinical practice by aligning local needs with the best international practices, offering practical guidance for healthcare providers. We strongly emphasize the necessity of preconception counseling and risk stratification for women with liver disease to ensure appropriate clinical care throughout the perinatal period. Furthermore, these high-risk pregnancies should be referred to tertiary care centers and managed by multidisciplinary teams to provide the comprehensive care required for optimal outcomes.

Pregnancy-Specific Liver Diseases

Table I: Characteristics and Management of Pregnancy-Specific Liver Disorders.

Feature	Hyperemesis Gravidarum	Intrahepatic Cholestasis	HELLP Syndrome	Acute Fatty Liver of Pregnancy
Timing in Pregnancy	First trimester	Second/third trimester	Third trimester	Third trimester
Key Symptoms	Severe nausea, vomiting, dehydration, weight loss	Itching, jaundice	Abdominal pain, vomiting, high blood pressure, proteinuria, headaches	Abdominal pain, vomiting, jaundice, confusion
AST/ALT Levels	1–5× ULN	1–8× ULN	2–30× ULN	5–15× ULN
Bilirubin Levels	Normal	1–6× ULN	1.5–6× ULN	6–8× ULN
Bile Acids	Normal	>10 µmol/L	Normal	Normal
Platelet Count	Normal	Normal	Low to very low	Low
Hemolysis	No	No	Yes	Yes
LDH Levels	Normal	Normal	Elevated	Elevated
Fibrinogen Levels	Normal	Normal	Normal	Very low
Hypoglycemia	No	No	No	Yes
Uric Acid Levels	Normal	Normal	Elevated	Elevated
Creatinine Levels	Elevated	Normal	Normal to elevated	Elevated to very high
Management	Rehydration, anti-nausea medications	Ursodeoxycholic acid, delivery at 37 weeks	Delivery, platelet transfusion, blood pressure control	Delivery, intensive care
Differential Diagnoses	Hepatitis, gallbladder disease, peptic ulcers, pancreatitis	Gallstones, viral hepatitis, autoimmune liver disease	HUS, TTP, lupus flare, septic shock	HUS, TTP, drug toxicity, hepatitis, lupus flare
Complications	Premature birth, poor fetal growth, Wernicke's encephalopathy	Premature birth, fetal distress, stillbirth	Liver damage, rupture, DIC, pulmonary edema, brain hemorrhage	Liver failure, DIC, kidney failure, severe bleeding

ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, DIC: Disseminated intravascular coagulation, HELLP: Hemolysis, elevated liver enzymes, low platelets, HUS: Hemolytic uremic syndrome, LDH: Lactate dehydrogenase, SLE: Systemic lupus erythematosus, TTP: Thrombotic thrombocytopenic purpura, UDCA: Ursodeoxycholic acid, ULN: Upper limit of normal

Hyperemesis Gravidarum (HG)

HG presents severe nausea & vomiting during early pregnancy, consequently leading to dehydration and loss of weight. It affects 0.3–10.8% of pregnant ladies & crucial to differentiate HG from the milder form of nausea and vomiting experienced by approximately 50% of pregnancies, which is temporary and does not result in malnutrition or weight loss.[6] HG is characterized by excessive & persistent nausea and vomiting during pregnancy (before 16 weeks). It may be associated with molar pregnancies, multifetal pregnancies, hypoadrenalism, hyperthyroidism, cerebral malignancies, and gastrointestinal obstructions. HG can lead to considerable maternal morbidity. Medical complications associated with HG include thromboembolism, thiamine deficiency (Wernicke's encephalopathy), gastrointestinal bleeding and life-threatening arrhythmias due to electrolyte imbalances such as hypokalemia & hyponatremia[7] Additionally, HG is linked to elevated rates of suicidal thoughts[8], and about 7% of women affected by the condition may choose to terminate a desired pregnancy.[9] While some studies indicate a higher incidence of intra-uterine growth restriction (IUGR), this finding is not universally observed.[10] There is also evidence suggesting an increased risk of delays in neuronal development and autistic disorder in children born to mothers who experienced HG.[11]

Diagnosis

HG presents persistent excessive vomiting along with a weight loss of more than 5% of body weight, dehydration & electrolyte imbalance during the first trimester.[7]

Management[7, 12-14]

⇒ Lifestyle: Advise patients to consume small, frequent meals and avoid foods or smells that trigger symptoms.

⇒ Pharmacotherapy

- First-Line Treatment: Doxylamine & pyridoxine (Vitamin B6) as combination pill is recommended as the initial pharmacologic therapy for symptoms like nausea and vomiting.
- Alternative Antiemetics: If symptoms persist, other medications such as metoclopramide or ondansetron may be considered.

⇒ In severe cases, hospitalization is recommended, IV fluid hydration, electrolyte correction and total parenteral nutrition may be considered.

Intrahepatic Cholestasis of Pregnancy (ICP)

ICP remains the most prevalent liver condition associated with pregnancy. It affects about 0.7% of pregnancies in European descent women while in Asian women is twice more frequent.[15] ICP is also more frequent in women with multiple pregnancies.[16, 17] and in those who have undergone in vitro fertilization.[18] Additionally, maternal chronic HCV infection is associated with a higher risk of ICP.[19]

Women with ICP may present with pruritus around 32 to 34 weeks of gestation, some may present as early as the first trimester. The etiology of ICP is complex, involving hormonal, genetic and environmental factors. Several medications, including anabolic steroids, contraceptive steroids, PPIs, amoxicillin, thiazolidinediones, bosentan, and psychotropic drugs, have been implicated in worsening or aggravating intrahepatic cholestasis of pregnancy. These drugs often interfere with bile acid transport or metabolism, leading to cholestasis. Clinicians should exercise caution when prescribing these medications to pregnant women and monitor for signs of ICP.[20-23]

Diagnosis

⇒ Any pregnant mother presenting with pruritus and abnormal LFT with normal liver ultrasound in the 2nd & 3rd trimester, a diagnosis as ICP should be considered.[12]

⇒ Key laboratory finding for diagnosis is elevated serum bile acids (SBA) >10 µmol/L

⇒ ICP patients should be classified based on SBA level as Mild ICP (10-39 umol/L), Moderate ICP (40-99 umol/L) or Severe ICP (>100umol/L).[24]

⇒ A single peak level is sufficient to define severity.[12]

Management

⇒ In resource limited settings where SBA levels are not available ICP may be diagnosed clinically with pruritis and cholestatic LFT and may be started with UDCA.

⇒ Treatment with Ursodeoxycholic acid (10-15mg/kg/day) may be initiated empirically based on pruritus & abnormal LFT and/or serum bile acid (SBA).[12, 13, 25] It relieves pruritus but no effect on fetal outcome.[26-28]

⇒ Additional symptomatic therapy for pruritus may be used to relieve patient discomfort.

⇒ Fetal health monitoring includes a weekly biophysical profile.[24]

⇒ Early delivery should be considered between 35th to 36th weeks in cases of severe ICP while in cases of moderate ICP, it may be in the 38th and in mild cases, 39-40 weeks to improve fetal outcomes.[14, 24]

⇒ Vaginal delivery should be the aim, however the decision of the caesarian section should be based on pure obstetric reasons.

HELLP (Hemolysis, Elevated Liver Enzymes. Low platelets) Syndrome

HELLP is usually present in the second or third trimester of pregnancy, but some may present after delivery.[29] It is regarded as severe presentation of hypertensive disorders linked with pregnancy (Pre-eclampsia & Eclampsia). These are major contributors to global maternal and perinatal deaths, where preeclampsia and eclampsia complicate 2% to 8% of pregnancies, they are responsible for 25% of severe maternal complications, including fatalities.[30,31,32] HELLP syndrome was first identified by Weinstein in 1982 as a condition characterized by "thrombocytopenia, elevated liver enzymes, and hemolysis".[33]

Additionally, certain comorbidities that increases its risk include preexisting vascular or kidney diseases, autoimmune disorders (such as lupus), chronic hypertension, pregestational or gestational diabetes, obesity, intrahepatic cholestasis of pregnancy (ICP), and antiphospholipid antibody syndrome(34). The risk factors for HELLP syndrome largely overlap with those of pre-eclampsia, age above 35-years at the time of conception is highest risk(29).

Table II: Complications of HELLP syndrome
Complications of HELLP syndrome. (35–37)

Maternal	Neonatal
Acute liver failure	Low Birth Weight
Acute renal failure	Birth Asphyxia
Disseminated Intravascular Coagulation (DIC)	Stillbirths
Abruptio Placentae	Neonatal Deaths
Eclampsia	
Pulmonary Edema	
Subcapsular Liver Hematoma	
Hepatic Rupture	

Diagnosis

The diagnostic criteria(7) include:

Hemolysis: An abnormal peripheral blood smear suggestive of microangiopathic hemolysis and elevated bilirubin levels, or a drop-in hemoglobin without any significant hemorrhage.

Elevated Liver Enzymes: A 2x increase in serum aspartate aminotransferase (AST) or alanine aminotransferase (ALT) levels.

Low Platelet Count: Platelet count less than 100,000 per microliter.

Management[7, 12-14]

⇒ Definitive management is expediting delivery of fetus as per obstetric guidelines once coagulopathy and hypertension of the mother has been corrected.

⇒ Control of hypertension, for patients with severe hypertension (systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg), urgent treatment in a high dependency unit is advised.

- Preferred antihypertensive agents include oral methyldopa, labetalol or nifedipine.
- Intravenous therapy options are hydralazine or labetalol, may be used where required.

⇒ Seizure prophylaxis should be administered to all patients (IV magnesium sulfate).

⇒ Corticosteroids are used for lung maturity of fetus only; no benefits are seen in maternal outcomes.

⇒ A platelet transfusion should be taken into account for pregnant women whose platelet count is less than 100x109/L since this is linked to an elevated risk of aberrant coagulation and negative maternal consequences related to preeclampsia. Additionally, in cases of ongoing bleeding or when invasive procedures are anticipated.

⇒ In cases of hepatic rupture who are bleeding or ALF a prompt referral to a facility with liver transplant should be considered.

⇒ Prophylaxis with 150 mg aspirin daily from 16 to 36 weeks' gestation is recommended In pregnant women with prior history of preeclampsia/HELLP syndrome if there are no contraindications.

Acute Fatty Liver of Pregnancy (AFLP)

Sheehan was the first to describe AFLP clinically as "jaundice with rapidly progressive liver failure within the third trimester".[38] Liver biopsy may show hepatocyte ballooning, micro vesicular steatosis, and minimal hepatic necrosis or inflammation.[39]

AFLP though rare yet potentially fatal liver disorder. Pathophysiology of AFLP remains elusive, a complex interplay of genetic, metabolic, and hormonal factors is likely contributing in it, It is thought to be a disorder of fatty acid oxidation. One of the primary contributing factors is a deficiency in the long-chain enzyme 3-hydroxyacyl-CoA dehydrogenase (LCHAD), which is responsible for the breakdown of long-chain fatty acids. During pregnancy, plasma triglyceride and free fatty acid levels increase, it may cause fatty acid metabolites accumulation in the fetus and mother. This may cause maternal hepatocytes mitochondrial dysfunction and acute liver failure.

The clinical presentation of AFLP can be variable, with minor symptoms ranging from nausea, vomiting, and abdominal pain to more severe ones like jaundice, coagulopathy and encephalopathy. If untreated, this condition can progress rapidly can be life-threatening for mothers and fetus. This may lead to maternal mortality even after the delivery, as some patients may have delayed complications.

Diagnosis

The diagnosis is based on if ≥6 of the above Swansea criteria are present, in the absence of other plausible explanation (e.g., viral hepatitis, pre-eclampsia, HELLP syndrome). as listed in table III.

Management[7, 12-14]

⇒ High risk AFLP patients are managed by a multidisciplinary team, preferably in ICU with level 2 or 3 care, those who score >7 on the 'Swansea criteria', presenting with encephalopathy, serum lactate more than 2.8 mg/dl or a model for end-stage liver disease (MELD) score ≥ 30.

⇒ Supportive management includes hydration through I/V fluids, glucose, and blood products if required.

⇒ INR correction in liver failure remains debatable not only for its questionable effectiveness but for increased risk of volume overload and paradoxical bleeding.[30] Nevertheless, if INR is more than 1.5, correction is only indicated if any invasive or surgical intervention is planned, or patient is actively bleeding.

- For correction of INR of more than 1.5, 10 ml/kg FFP transfusion is recommended. It may be repeated within 6 hours if clinically indicated.[40]

Table III: The Swansea Criteria.

The Swansea Criteria for Acute Fatty Liver of Pregnancy (AFLP)	
Clinical Features (at least 6 required)	**Laboratory Features**
Vomiting	Elevated bilirubin (>0.8 mg/dL or >14 µmol/L)
Abdominal pain	Hypoglycemia (<72 mg/dL or <4 mmol/L)
Polydipsia/Polyuria	Elevated uric acid (>5.7 mg/dL or >340 µmol/L)
Encephalopathy	Leukocytosis (>11 x 10^9/L)
Elevated bilirubin (clinical jaundice)	Elevated transaminases (AST or ALT >42 U/L)
Hypoglycemia (symptoms or documented)	Elevated ammonia (>47 µmol/L)
Ascites or bright liver on ultrasound	Renal impairment (creatinine >1.7 mg/dL or >150 µmol/L)
Leukocytosis (elevated white blood cell count)	Coagulopathy:
Elevated transaminases (AST or ALT)	Prolonged PT (>14 seconds)
Elevated ammonia	Low fibrinogen (<300 mg/dL or <3 g/L)
Renal impairment (elevated creatinine or oliguria)	Microvesicular steatosis on liver biopsy (if performed)
Coagulopathy (prolonged PT or aPTT, low fibrinogen)	**Imaging Features:**
Microvesicular steatosis on liver biopsy (if performed)	Bright liver on ultrasound (increased echogenicity suggestive of fatty infiltration)
	Ascites (detected on ultrasound)

⇒ Plasma exchange or N-acetylcysteine (NAC) may be considered in the treatment AFLP if advised by hepatologist. Dose of NAC IV infusion 150mg/kg in 200 ml 5% DW over 1 hour, followed by 50mg/kg in 500ml 5% DW over 4 hours, then 100mg/kg in 1000ml 5% DW daily for next 3 days (Oral NAC 1st dose of 140mg/kg followed by 70 mg/kg every 4 hours for 17 doses)

⇒ Expedited delivery as early as possible, whatever the age of gestation, in a tertiary care setting.

⇒ Vaginal delivery should be the aim; however, the decision of the caesarian section should be based on pure obstetric reasons.

⇒ In cases failing to respond to expedited delivery, prompt referral to a liver transplant unit should be considered.

Pregnancy Non-specific Liver Diseases

For the purpose of simplicity & clarity only viral hepatitis & cirrhosis is discussed here as rest are uncommon in clinical practice.

Viral Hepatitis

The high global prevalence of viral hepatitis is significant impact on pregnant women and leading to considerable maternal and perinatal abnormalities and deaths. Hepatitis A and E, primarily transmitted via the fecal-oral route & responsible for most of the epidemic outbreaks. During pregnancy, usually present as acute hepatitis. While hepatitis A generally resolves without complications during pregnancy, not only is the prevalence of HEV high it is also linked with higher morbidity & maternal mortality (around 20%).[41]

Hepatitis B, C and D are usually chronic infections found equally in both pregnant and non-pregnant women. Screening for hepatitis C during pregnancy and its management remain uncertain; however, the advent of direct-acting antiviral (DAA) medications may alter this landscape if their safety during pregnancy is confirmed.[12] Hepatitis D virus (HDV) is an incomplete virus that depends on hepatitis B virus (HBV) for its infection; thus, controlling HBV is crucial for managing HDV. HBV and HCV screening during Ist antenatal visit is now the standard of care.

Hepatitis A

Incidence of hepatitis A in pregnancy is 1; 1000.It is acquired through fecal-oral route. Disease is usually mild, no specific treatment is required, vertical transmission is rare, vaccination is safe in pregnancy.

Hepatitis B

Hepatitis B is the most common & routinely screened viral hepatitis during pregnancy. It is significant public health concern with poor maternal-fetal outcomes. It can lead to chronic hepatitis B & HCC in children born to HbsAg +ve mother through mother-to-child transmission (MTCT).

Management of Hepatitis B[12, 13, 15, 42]

There are three possible scenarios:

1. Pregnant lady already on treatment for hepatitis B

a. If she is on Entecavir (ETV), it should be changed to Tenofovir Disoproxil Fumarate (TDF) or Alafenamide (TAF).

b. Tenofovir Disoproxil Fumarate (TDF) or Alafenamide (TAF), it should be continued as such.

c. After delivery, newborn should be given:

   i. Birth dose of HBV vaccination (within 12 hours of birth),

   ii. It should be followed by second dose at one month and third dose at 3-6 months of age or infants should get Pentavalent vaccine from the EPI program of Pakistan at 6, 10 and 14 weeks.

   iii. Breast feed should be encouraged to the infants born to HbsAg +ve mothers if the infant has received immunoprophylaxis at birth.

d. Hepatitis B Immunoglobulin (HBIG), if available, should be given within 12 hours of birth especially in high income settings or high HBV DNA (despite being on antiviral treatment) especially if HbeAg+.

2. Pregnant lady with unknown status of HBV infection

a. All pregnant ladies should be tested (as soon as possible) for hepatitis B by doing HBsAg by ELISA if available triple panel (HbsAg, HbsAB, HB core total).

b. If found to be HBsAg positive,

   i. Check HBV DNA PCR by Real Time PCR,

   ii. HBeAg by ELISA and ;

   iii. Serum Biochemistry for ALT.

c. If she qualifies for anti-viral treatment for her own liver status, start her on Tenofovir – Disoproxil Fumarate 300mg (TDF) or Alafenamide 25mg (TAF) and continue as per standard protocol.

d. If she does not qualify for anti-viral treatment, start her Tenofovir Disoproxil Fumarate (TDF) preferably from the second trimester of pregnancy. The treatment should continue till the time of delivery or completion of the infant HBV vaccination series), to prevent the MTCT if HBV if HBV DNA>200,000 IU/ml.

e. After delivery, newborn should be given HBV vaccination and HBIG if indicated (as outlined above).

Delivery

a. Vaginal delivery should be the aim, all precautions should be made to avoid viral transfer to staff, to avoid vertical transmission avoid instrumental delivery, internal fetal heart monitoring, scalp blood sampling, avoid episiotomy and early cord clamping.

b. Breast feed should be encouraged to the infants born to HbsAg +ve mothers if the infant has received immunoprophylaxis at birth

3. Pregnant woman is found to be HBsAg positive at the time of delivery

a. Give the newborn HBIG and HBV vaccination (as outlined above).

b. Check the mother for HBV DNA PCR by Real Time PCR, HBeAg by ELISA and serum ALT.

   i. If she qualifies for anti-viral treatment for her own health, start her on Tenofovir Disoproxil Fumarate (TDF) and continue as per standard protocol.

   ii. If she does not qualify for anti-viral treatment, start her Tenofovir Disoproxil Fumarate (TDF) for the period limited to completion of the infant HBV vaccination series), to prevent the MTCT of HBV.

   iii. If the infant has received immunoprophylaxis at birth, breast feeding should be encouraged.

Hepatitis C

The prevalence of hepatitis C in pregnancy is 1-2%. transmission is parenteral vertical, sexual and mucosal. The vertical transmission rate is 5-10%.80% of the patients will develop chronic liver disease in life.

Hepatitis C in pregnancy is associated with adverse pregnancy outcome such as preterm labour, PROM, SGA, and an increased rate of perinatal mortality. Protocols for delivery are the same as HepB. Vaccine is not available.

Hepatitis D

Mode of transmission is parenteral and 70-80% of cases progress to chronic liver disease. Fetal transmission is not reported. No specific treatment is required in pregnancy. vaccination is safe in pregnancy.

Hepatitis E

Hepatitis E is endemic in Pakistan and seroprevalence in as high as 60% in LMICs.[43] It poses significant life-threatening risk, when it presents in third trimester, up to one fifth of the women progress to ALF.[44-47]

Diagnosis

Hepatitis E in pregnancy is diagnosed by positive anti-HEV immunoglobulin M (IgM) antibodies in the blood, and elevated liver function tests (high bilirubin, ALT, and AST).

Management

⇒ Supportive management includes rest, hydration, and a balanced diet, close fetal monitoring is required, and early delivery is considered if the situation worsens.

⇒ Expedite delivery after initial workup.

⇒ Prevention with HEV vaccine in reproductive age group should be encouraged prior to pregnancy, trials are underway for vaccination in pregnant mothers.

⇒ HEV related ALF needs urgent referral to a liver transplant center.

Hepatitis G

Transmission is parenteral. Progression to chronic disease is rare. No fetal transmission is reported and vaccine is not available.

Liver Cirrhosis

Pregnancy in cirrhotic women is rare. Cirrhosis is linked with serious liver related complications, like worsening ascites, esophageal variceal bleeding, spontaneous bacterial peritonitis, hepatic encephalopathy and hepatorenal syndrome.[48] During pregnancy, these risks are further compounded, leading to higher rates of miscarriage, preterm birth, cesarean delivery, postpartum hemorrhage, and maternal mortality.[49] There is a bidirectional relationship between cirrhosis and pregnancy: pregnancy can accentuate cirrhosis by worsening thrombocytopenia, lowering serum albumin levels, and rising portal pressure due to expanded blood volume. Conversely, cirrhosis heightens the risk of pregnancy complications, further compromising maternal and fetal health.[50,51] Preconception MELD>10 is associated with high risk of maternal hepatic decompensation while a score <6 is considered relatively safe.

Diagnosis

It is either diagnosed through clinical signs and symptoms such as ascites, esophageal and para-umbilical varices, or deranged LFTs, low albumin & prolonged INR. Imaging modalities such as ultrasound or MRIs can reveal shrunken liver with or without nodules and stiffening of the liver.

Management[12, 13, 52]

Screening, predictive scoring and pre-pregnancy counseling plays a crucial part in management and leads to improved health outcomes.

Endoscopy for esophageal varices is safe during pregnancy and hepatotoxic drugs are avoided to prevent further deterioration.

Vaginal delivery should be the aim, however the decision of the caesarian section should be based on pure obstetric reasons.

Clinical Approach to Liver Diseases in Pregnancy

Liver diseases in pregnancy can be further evaluated through clinical findings, biochemical studies or radiological findings.

Clinical Evaluation

Detailed clinical history and examination are required to diagnose and classify liver diseases in pregnancy. Nonspecific symptoms presenting clinically are abdominal pain, jaundice, nausea, and vomiting. However, coagulopathy (especially anti-thrombin activity), hypoglycemia, encephalopathy, high serum ammonia, low albumin, and renal insufficiency are

specifically associated with acute liver diseases of pregnancy.[8, 38, 48]

Laboratory Investigations

A detailed biochemical analysis of liver functions is required to establish liver injury during pregnancy, deranged prothrombin time, and raised ALT, ALP, direct and total bilirubin concentration are direct indicators of liver insufficiency. To exclude viral causes of liver trauma, serologies for hepatitis B and C can also be performed.

Imaging Modalities

Ultrasound is a preferred imaging modality during pregnancy, findings can include hepatomegaly, gall bladder wall thickening, and hepatic hypo-echogenicity. If ultrasound is inconclusive MRI is another potential modality, that can better evaluate liver diseases.

Conclusion

Optimizing maternal and fetal results during pregnancy requires early detection and focused treatment of liver disorders. This position statement encourages healthcare providers to employ a multidisciplinary approach that uses clinical and laboratory criteria to ensure accurate diagnosis and evidence-based treatment.

Further research is required on best care practices, long-term results, and advancements in diagnostic technologies for liver problems in pregnancy.

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