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Emerging Pathological Events in Liver Diseases of Pregnancy

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ABSTRACT

Hepatic dysfunction in a gestational woman can arises from pregnancy-related factors, illnesses that occur concurrently with gestation, or previously existing hepatic disorders which is aggravated by pregnancy. Abnormal liver test findings occur in 3% to 5% of pregnancies due to a variety of probable causes, with clinical effects ranging from self-limiting to swiftly deadly. Abnormal liver tests in pregnant individuals are caused by four major conditions: (i) Physiological changes in pregnancy, (ii) Newly acquired Liver disease, (iii) Pre-existing Liver disease, and (iv) Pregnancy-related Liver disease. Physiologic changes in pregnant women lead to abnormal liver function tests. Hepatic disorders may result in severe illness and death in both mothers as well as fetuses. Quick identification of the disease is crucial, as in critical situations immediate delivery is vital for the mother and developing infant survival. This review focuses on liver problems that are specific to pregnancy. Hyperemesis gravidarum (HG), which appears within the first 12 weeks of pregnancy, is connected to liver damage. Intrahepatic Cholestasis of Pregnancy (ICP) is the most prominent all across the middle and the end term of pregnancy. After birth, pruritus and the related biochemical indications of cholestasis resolve. Both pre-eclampsia and HELLP (elevated liver enzymes, hemolysis, and low platelets) are severe conditions that arise in the last term of pregnancy. Acute fatty liver during pregnancy (AFLP) is a potentially dangerous, uncommon condition that can affect pregnancy's third trimester. This review summarizes the etiologies, pathogenesis, identification, and treatment of hepatic disorders during pregnancy.

1. Introduction

Throughout pregnancy, typical physiological and hormonal profiles change. Furthermore, alterations in the biochemical profile of the liver are normal to some extent in pregnancy. However, liver diseases can complicate pregnancy in up to 3 percent of cases. Although severe liver disease is uncommon it can cause increased morbidity and death (Leventhal *et al.*, 2015). Liver illnesses pose a particular clinical challenge during pregnancy since they can harm the pregnant mother as well as the fetus (Ma *et al.*, 2019). Every year, approximately 2M morbidity occurs as a result of hepatic damage, 1 million because of cirrhosis problems, and 1 million because of viral hepatitis and hepatocellular cancer (Asrani *et al.*, 2019). About three per cent of pregnancies are affected by various hepatic diseases every year. Early detection and specific treatment can improve both maternal and fetal outcomes (Bacq and Yannick 2011). During pregnancy, three forms of liver disease must be distinguished. One form is pregnancy-related liver illness such as Hyperemesis gravidarum (HG), Intrahepatic cholestasis of pregnancy (ICP), etc. which can strike at a certain time during pregnancy. The second type is Non-pregnancy-related hepatic disorder, like hepatitis caused by a virus or a medication, which may strike at any time. The third category includes women with previously existing hepatic illnesses. Clinicians must be familiar with Pre-eclampsia, HELLP (Haemolysis, Elevated liver enzymes, and Low platelets), ICP etc. disease in order to respond quickly and correctly in each of the above situations, especially when last-minute parturition is expected (Mikolasevic *et al.*, 2018).

Types of liver disease during pregnancy
(Mikolasevic *et al.*, 2018)

Disease	Symptoms	Time of appearance	Histology
Hyperemesis gravidarum	Nausea, vomiting, weight loss	First trimester	hepatocyte necrosis, bile plugs, steatosis
HELLP	Hypertension, vomiting, oedema	Third trimester	Fibrin deposits, necrosis, and periportal haemorrhage

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Intra hepatic cholestasis of pregnancy (ICP)	Jaundice, fatigue, abdominal pain.	Second and third trimester.	No inflammation, centrilobular cholestasis
Acute fatty liver of pregnancy (AFLP)	Nausea, diarrhea, tiredness, and hepatitis	Third trimester	Micro vesicular fat, necrosis, bile plugs, steatosis
Preeclampsia-induced liver damage, Eclampsia induced liver damage	Hypertension, proteinuria, headache, coma	Second and third trimester	Periportal haemorrhage, necrosis, fibrin deposits, and microvesicular fat

Modification in liver characteristics during pregnancy

Physiological changes occur in the pregnant woman to promote foetal growth and development. The levels of estrogen and progesterone in the blood rise gradually during pregnancy, peaking in the last term (Akinloye *et al*., 2013). These sex hormones have an impact on liver metabolic, synthetic, and excretory functions. During gestation, cholesterol levels increase by 53% on average, with two-thirds of the rise occurring in the middle term (Herrera *et al*., 2014). The concentration of triglycerides rises thrice in pregnancy. The majority of this rise occurred during the third trimester of pregnancy. Within 12 to 24 hours of delivery, cholesterol levels drop by 15% (Soma-Pillay *et al*., 2016). Plasma volume rises gradually throughout normal pregnancy (Rodger *et al*., 2015), so that the serum albumin content decreases, whereas alkaline phosphatase activity increases as placental production increases. In general, aminotransferase (aspartate aminotransferase and alanine aminotransferase), bilirubin, and gamma-glutamyl transpeptidase quantities remain normal throughout pregnancies and should be researched more. The liver seems normal or near normal under light microscopy (Soma-Pillay *et al*., 2016).

Disease-associated with liver damage during pregnancy

1. Preeclampsia-induced liver damage

Pre-eclampsia is a pregnancy-specific condition characterized by extensive endothelial dysfunction and vasospasm that generally manifests around twenty weeks of gestation. It is defined as having systolic blood pressure greater than or equal to 140 mm Hg or diastolic blood pressure of 90 mm Hg or higher (Kee-Hak *et al*., 2017). When there is evidence of end-organ damage, such as pulmonary edema, renal insufficiency, or hepatocellular damage, severe preeclampsia occurs. The presence of grand mal seizures in addition to preeclampsia symptoms is the distinguishing hallmark of eclampsia (Ma *et al*., 2019). It is a leading cause of death around the world. Even in developed countries, perinatal mortality is increasing by fivefold (Jeyabalan and Arun 2013). Blood supply to organs other than the placenta is diminished in women with preeclampsia. Reduced perfusion with secondary necrosis and hemorrhage can be seen in the liver (Chaiworapongsa *et al*., 2014). This condition causes intra-uterine restriction, early delivery, poor birth weight, and perinatal death. Higher neonatal morbidity along with mortality rates are depressing as some of it is due to premature birth, which was done to save the fetus and mother from deteriorating further (Townsend *et al*., 2016). In the event of preeclampsia, platelet levels in the blood are reduced, a condition known as thrombocytopenia, which leads to an increase in liver enzymes, indicating a liver problem (Sasamori *et al*., 2020). Preeclampsia-induced liver damage is a condition that occurs only during pregnancy and occurs most commonly in the last term. HELLP is the term given to this disorder. HELLP disorder shows signs of hemolysis, low blood platelet levels, and high hepatic functions (Gauer *et al*., 2012). The HELLP syndrome appears in approximately 70% of casesprior to birth; 10% appear before twenty-seven weeks and 20% appear after thirty-seven weeks of pregnancy. In women who had proteinuria and hypertension previously, the HELLP disorder frequently occurs in the two days after delivery (Folk and Diane 2018).

Pathogenesis:

There are many genetic variations for example glucocorticoid receptor gene (GCCR), Toll-Like receptor protein 4 (TLR4), vascular EGF genes, Fas Genes, 95 differentiation cluster (CD95) and the Leiden mutation in coagulation factor V. They all have been linked to an increased risk of HELLP in comparison to healthy women. In patients with HELLP, thrombotic microangiopathy causes microangiopathic hemolytic anemia and liver damage (Hammoud *et al*., 2014). Preeclampsia is thought to be caused by a lack of placental trophoblast penetration, which is followed by extensive maternal endothelial dysfunction. The placenta has been shown to release excessive amounts of the antiangiogenic factors soluble fms-like tyrosine kinase 1 (sFlt1) and soluble endoglin (sEng) into maternal blood, resulting in widespread endothelial dysfunction and hypertension, proteinuria, and other preeclampsia-related systemic manifestations. Furthermore, disturbance of the RAS-II (renin-aldosterone-angiotensin II axis), elevated oxidative stress, inflammation, immunological distress, and

hereditary vulnerability may all lead to the pathophysiology of preeclampsia (Ekun *et al.*, 2018).

Diagnosis

Preeclampsia is diagnosed after twenty weeks of gestation when a (pregnant) has high BP and at least one of the following symptoms: Proteinuria (an increase in protein in pregnant women's urine) indicates kidney disease, signs of renal problems such as foot edema, fatigue, weakness and reduction in platelet count, vision distortion, etc. (Magee *et al.*, 2018). Portable ultrasound is a good screening method without moving the patient. For identifying liver rupture and determining the amount of the hematoma, liver sonography or MRI offer great sensitivity. Sonography is more suitable for unstable patients (Ditisheim *et al.*, 2017). Serum AST/ALT/alkaline phosphatase levels are mildly elevated in situations of mild pre-eclampsia, and there are minor signs of disseminated intravascular coagulation with thrombocytopenia. Jaundice is uncommon, but when it occurs, it is usually terminal and hemolytic in nature, with total serum bilirubin levels rarely increasing to six mg/dL. Normal hepatic enzymes are found in twenty to thirty per cent of cases, with mild to moderate transaminase elevation (1.5-5 times more than normal). Conjugated bilirubin, albumin, and PT levels are usually normal. Hepatic involvement, which includes hepatic artery vasospasm and fibrin precipitation in the liver lobule's portal and periportal regions, might result in lobular ischemia and hepatocyte necrosis (García-Romero *et al.*, 2019).

Management

When a pregnant woman develops liver disease during pregnancy, corticosteroids are given and the patient is managed based on their clinical response. If the patient's condition worsens, delivery is preferred; if the patient's condition remains stable, the patient should be monitored in a tertiary care facility (English *et al.*, 2015).

2. Hyperemesis Gravidarum (HG)

The medical name for extreme nausea and vomiting during pregnancy is hyperemesis gravidarum. This disorder is characterized by intractable emesis throughout the gestation, resulting in electrolytes, acidic and alkaline imbalances, starvation and loss of weight that is frequently severe enough to need hospitalisation (Saha and Sumana 2019). It is rare, affecting about 0.3 percent to 2% of pregnancies. It usually occurs in embryonic stage (Ma *et al.*, 2019). It is generally associated with advanced molar gestation and multiple gestations (Zhang *et al.*, 2011). Starvation, stomach motility, hormonal variables, and psychological issues have been related to the occurrence of HG (Kamimura *et al.*, 2015). It is a complex condition in which pregnancy-induced hormonal changes, concomitant gastrointestinal dysmotility and possibly Helicobacter pylori infection all play important roles (MacGibbon and Kimber 2020).

Pathogenesis

The cause of HG-related liver damage is unknown. Overexpression of cells that generate cytokines has been linked to gestational-related hepatic disorders, like pre-eclampsia. According to another study, fatty acid buildup in the placenta as a result of poor maternal or foetal mitochondrial fatty acid oxidation, as well as a deficiency of LCHAD (a long chain of 3-hydroxy acyl-CoA dehydrogenase), all cause damage to the liver (Ahmed *et al.*, 2013).

Diagnosis

The pregnant woman may vomit more than four times per day, get dehydrated, feel dizzy and lose 10 pounds or more (Wegrzyniak *et al.*, 2012). A full medical history should be gathered, as well as a thorough physical examination and focused laboratory tests. An ECG and a pelvic ultrasound are performed to verify pregnant viability, the no. of fetuses, (stage of pregnancy, and also to check out trophoblastic illness during pregnancy). stomach ultrasonography and entire upper GIT endoscopy might be necessary (Niemeijer *et al.*, 2014). Mild serum aminotransferase elevations are the most common, but more transaminase elevations (Alanine amino-transferase (ALT) levels 400 -1000 U/L) have been reported. Mild hyperbilirubinemia with mild jaundice is also possible. Other issues include electrolyte, water, and acid-base balance alterations, which are generally sufficiently addressed with hydration (Esposti *et al.*, 2015).

Management

Patients with HG are frequently admitted to the hospital for the replacement of IV fluids, loss of emesis, intestinal relaxation as well as possible systemic feeding . Although promethazine is the first-line treatment, additional drugs like metoclopramide, Ondansetron, and corticosteroids are employed. In serious conditions,whole systemic feeding is used with care (Ahmed *et al.*, 2013).

3. Haemolysis, Elevated Liver Enzymes, and Low Platelets (HELLP)

It's a severe type of pre-eclampsia with a characteristic laboratory triad that can be lethal. The condition affects 0.5

percent to 0.9 percent of all healthy newborns and mother, according to reports (Pascual *et al*., 2022). The emergence of HELLP disorder is a potentially fatal illness for mother as well as fetus. It usually begins in the third trimester. HELLP syndrome is characterised by dizziness, purging, and discomfort in the right side of the abdomen. Lethargy, migraine, severe pulse pressure and nephropathy are among theother symptoms (Sasamori *et al*., 2020). However, it has the potential to grow in lack of severe preeclampsia, this syndrome is more commonly recognized in patients with severe preeclampsia. Despite the fact that the cause of the disease is uncertain, histopathology in the liver has revealed intravascular fibrin deposits, which can cause hepatic sinusoidal blockage, intrahepatic vascular congestion, and increased intrahepatic pressure, ultimately leading to hepatic necrosis, intra parenchymal and sub capsular haemorrhage, capsularruptures (Dusse *et al*., 2015). If there is significant ischemia or hepatocyte infarction, liver enzymes can surge to over 1,000 U/L (Sasamori *et al*., 2020).

Pathogenesis

Certain chemicals produced by placental tissue, like NO, PGs, and endothelin, are thought to cause hypertension, platelet aggregation, and endothelial dysfunction. As a result, microangiopathic hemolytic anaemia is caused by endothelial damage and fibrin accumulation in blood vessels (Schistocytes and burr cells on smear), with activation of platelet and consumption (Abildgaard *et al*., 2013). Fibrin deposition within the hepatic sinusoids is thought to be the cause of liver dysfunction, resulting in sinusoidal obstruction, vasospasm of the hepatic vascular bed and ischemia of the liver. This can occasionally result in large hematomas, capsular tears, and i.p haemorrhage (Mikolasevic *et al*., 2018).

Diagnosis

Diagnostic indications of intrinsic hemolytic anemia, liver failure, and thrombopenia in a preeclamptic patient is used to diagnose HELLP syndrome. Early signs of HELLP disorder include a rise in lactic dehydrogenase (LDH) levels and a decrease in serum haptoglobin levels (25 mg/dL). Tests such as fibrin degradation products and antithrombin III activity can be utilised to alert the physician to the existence of a chronic disseminated coagulopathy if the platelet count is less than 55,000/L (Rimaitis *et al*., 2019).

Management

Hepatic, renal, and hematologic problems should be examined even three days after delivery. Hepatic rupture, hematoma, and decompensation all require rapid medical attention, which might entail radiological or surgical intervention, as well as liver transplantation (Sasamori *et al*., 2020). The Mississippi Protocol is strongly in favour of IV steroid, particularly dexamethasone at a high dosage combination with $MgSO_4$ and BP management (Martin Jr *et al*., 2012).

4. Intrahepatic Cholestasis of Pregnancy (ICP)

It is a type of hepatic disorder characterised by a reversible cholestatic state. It's a pregnancy-related liver syndrome marked by maternal pruritus in the 3rd trimester, higher serum glycocholic acid, and an elevated risk of poor foetal result. It is also known as obstetric cholestasis, or gestational hepatitis. It usually occurs in the middle and last term of gestational (Manzotti *et al*., 2019). The first episode of idiopathic erythema with apparent hepatitis came in the prenatal period in 1883 and disappeared shortly after birth. Scandinavian and South American countries have a higher prevalence of ICP. In Europe, the United States, Canada, and Australia, the prevalence rangesfrom 0.2 - 2 percent (Huang *et al*., 2022). The major symptoms include weight loss, gastrointestinal problems, and a deficiency in fat-soluble vitamins (Mahle *et al*., 2021). ICP has a complex etiology that includes genetic, hormonal, and environmental variables. The condition commonly manifests itself during the last term of gestation, while estrogen levels are at their highest. In genetic susceptibility people, a large dosage of estrogen can lead to intrahepatic cholestasis by inhibiting sulfation and bile acid transport (Ozkan *et al*., 2015). ICP is a developmental disorder for the mother, it might have negative consequences for the fetus. In other instances, ICP caused as much as 60% of preterm deliveries. Foetal discomfort and fetal death in the uterus were also recorded at 61 percent and 1.6 percent, respectively (Ahmed *et al*., 2013).

Pathogenesis

The cause of ICP is not clearly understood. ICP is characterized by aberrant biliary transit across the canalicular membrane, which has a complex origin (Boyer and James 2013). ICP etio pathogenesis has been linked to genetic, hormonal, and environmental factors (Ozkan *et al*., 2015). Mutations in the hepatic phospholipid transporter ABCB4 (MDR3), which mediates phosphatidylcholine (lecithine) secretion into bile, are thought to account for up to 15% of all ICP cases. According to available molecular genetic studies, the bile salt export pump (BSEP), ABCB11, and the amino phospholipid transporter (FIC1), ATP8B1, are less likely to be involved in the pathophysiology of ICP (Henkel *et al*., 2019). ICP generally occurs in late pregnancy when

serum estrogen levels are at their peak; it is more frequent in twin pregnancies, which have greater oestrogen levels, and disappears after birth when sex hormone levels decline (Mutlu *et al*., 2017).

Diagnosis

The serum total bile acids level is the most important biochemical test since it may be the initial and potentially only aberrant test result, although the results change with pregnancy, making weekly checkups necessary in women with ICP. The diagnosis is confirmed by fasting plasma bile acid level of more than 10 mol/L. Aminotransferases can also be increased by 2-10 times. When a hepatic cell has been injured, ALT is gradually released into the bloodstream (Sahni *et al*., 2022). Clinicaljaundice is diagnosed in 10% to 15% of patients, and bilirubin concentration seldom increases by 100 mol/L. Women with ICP, like all cholestatic patients, have increased levels of LDL (low-density lipoprotein cholesterol) and triglycerides (Wood *et al*., 2018). A liver biopsy can detect bland cholestasis (intrahepatic cholestasis without parenchymal inflammation), however, it is seldom required (Ahmed *et al*., 2013).

Management

UDCA (Ursodeoxycholic acid) is the most common treatment in the case of Intrahepatic cholestasis of pregnancy at a dose of 500mg twice daily or fifteen mg per kg daily. Because no maternal or foetal side effects have been recorded with its usage in ICP, UDCA is safe in the third trimester. UDCA reduces the level of plasma bile acids, some enzymes, serum bilirubin and also beneficial for Erythema (Ahmed *et al*., 2013). Other drugs, such as cholestyramine and S-adenosyl-methionine, haven't worked. In women who do not react to UDCA, combining rifampicin with it improves symptoms and test abnormalities (Geenes V *et al*., 2015).

5. Acute Fatty Liver During Pregnancy (AFLP)

AFLP is also known as acute yellow atrophy or acute fatty metamorphosis, is a microvesicular fatty infiltration of hepatocytes (Ramanathan *et al*., 2022). AFLP is an emergency condition because it can be deadly to both the mother and the infant if it is not discovered and treated early. It is a rare situation that usually occurs itself in the 3rd term. The accurate cause of acute fatty liver in pregnancy is uncertain. It's evident that it's neither an infectious disease nor a hereditary metabolic disorder (Liu *et al*., 2017). Pain, tiredness, regurgitation, and vertigo are among the early signs of AFLP. Vertigo and regurgitation are reported by 70% of the participants and 55 percent to 80 percent report right upper quadrant or epigastric pain. An upper gastrointestinal haemorrhage caused by coagulation problems, severe renal failure, infection, pancreatitis or hypoglycaemia can all aggravate AFLP at an early stage. Multigravidas, preeclampsia, multiple gestation, and male foetus are risk factors, and it may be more common in underweight women (Mikolasevic *et al*., 2018).

Pathogenesis

The pathophysiology of AFLP is not fully cleared, but recent research suggests that it is most likely caused by mitochondrial dysfunction (Naoum *et al*., 2019). In some cases of AFLP, both the mother and the foetus have a mitochondrial fatty acid oxidation deficiency. Mutations in two mitochondrial fatty acid oxidation enzymes, mitochondrial trifunctional protein and its long-chain 3-hydroxy acyl-CoA-dehydrogenase alpha subunit(LCHAD), are considered to be intimately associated with AFLP (Mikolasevic *et al*., 2018). The clinical picture varies, with symptoms including nausea, vomiting, stomach discomfort, headache, and malaise. Acute liver failure, as well as accompanying consequences including encephalopathy, jaundice, and coagulopathy, can develop rapidly in the clinical course (Sasamori *et al*., 2020).

Diagnosis

Atypical liver values can be shown by laboratory tests, such as increased aminotransferase levels (varying from moderate to 1000 IU/L, but generally 300-500 IU/mL) and hyperbilirubinemia (commonly more than five mg/dL) (Mikolasevic *et al*., 2018). Leukocytosis (>11×10^6/L), normochromic anemia, thrombocytopenia, hypo-albuminemia, high uric acid levels, kidney dysfunction, alkalosis, hyper-ammonemia, and biochemical pancreatitis are all frequent. Proteinurea and ketonuria may be detected (Sharkey *et al*., 2010). Hypoglycemia is prevalent and is associated with a bad prognosis. Extended prothrombin time (PT) and decreased fibrinogen levels are common in severe cases and disseminated intravascular coagulation (DIC) occurs in about 10% of AFLP patients (Meng *et al*., 2016). Ascites, pleural effusions, severe pancreatitis, and respiratory and renal failure are all possible complications of AFLP (Ye *et al*., 2021). Because women with AFLP are prone to having coagulopathies as a consequence of poor synthetic liver function and/or DIC, infections, as well as vaginal bleeding or bleeding from Caesarean section wounds, are prevalent (Mikolasevic *et al*., 2018).

Management

Once a diagnosis has been made, delivery is accelerated, either with or without the use of medications. Close fatal surveillance

is recommended in those in labour. The use of ultrasound and computed tomography (CT) of the liver for the detection of this illness, as well as more aggressive care, has resulted in a considerable reduction in mortality from acute fatty liver during pregnancy over the last decade. Hepatic transplant is recommended for women who have AFLP-related fulminant hepatic failure and who's condition hasn't improved following birth and critical care (Liu *et al.*, 2017).

Simultaneously occurring liver disease in pregnancy

Acute viral hepatitis

All pregnant women should be tested for the hepatitis B virus (HBV) early in pregnancy. Vertical transmission is associated with a greater probability of chronic infection in infants born to HBsAg-positive mothers; the presence of HBV antigen and the maternal viral load are the main risk factors for vertical transmission. As a result, all neonates should receive vaccination and anti-HBs immunoglobulin within 12-24 hours of delivery (Mikolasevic *et al.*, 2018). Mothers are highly vulnerable to HEV than HAV, HBV, or HCV infection (Terrault *et al.*, 2021). The most prevalent viral cause of acute liver failure in pregnancy is HEV, which often occurs in the 2nd and 3rd trimesters (Acharya and Subrat 2013). In utero transmission of HEV to foetus may introduce additional harmful metabolites into the maternal circulation, increasing maternal morbidity and mortality (Lata and Indu 2013). HSV (herpes simplex virus) hepatitis is an infrequent disorder that primarily affects immunocompromised people or children. Gestational mothers are highly vulnerable to HSV (Schalkwijk *et al.*, 2022). Pregnant women have been shown to have HSV serotypes 1 and 2, which are caused by either primary or latent diseases. Elevated amino transferases, thrombocytopenia, leukopenia, and coagulopathy with standard serum bilirubin levels are typical laboratory findings. A liver biopsy gives definitive confirmation of HSV hepatitis, however, computed tomography, which shows several low-density regions of necrosis inside hepatocytes, may also be beneficial. If confirmed results are obtained, treatment with i.v acyclovir should not be postponed (Joshi *et al.*, 2010).

Billiary disease

Gallstones are more prevalent during gestation due to elevated cholesterol release, higher bile lithogenicity, and reduced gallbladder movement in the second and third trimesters. Gallstones or viscus biliary sludge affect about 10% of pregnant women (Lanzafame *et al.*, 2019). Regardless of pregnancy, cholecystectomy is recommended for victims with refractory biliary colic, severe cholecystitis that is not responding to conservative therapies, or severe gallstone pancreatitis. Traditional treatment, which includes bed rest, i.v fluids, and anti-biotics, is successful in higher than 80% of instances of simple acute billiary colic or acute cholecystitis, with no foetal or maternal fatality, and is recommended during the 1st and 3rd trimesters. Operation is avoided during the initial trimester to prevent the risk of anaesthesia-induced abortion, and in the 3rd trimester to reduce the risk of preterm labour (Bleeser *et al.*, 2022).

Conclusion and future perspectives

Gestation is a special state that can be influenced by a variety of events, resulting in a poor outcome for both child and mother. Although gestational liver dysfunction is rare compared to other types of liver disease, it is important clinically because it affects both mother and fetal health outcomes. Hepatic illness during pregnancy is complex and multifaceted, with widely varying causes and severity. One of the most difficult management issues in clinical medicine is the multiple organ failure that patients with hepatic disorders, and this demand extremely close collaboration between the intensivist, the hepatologist, the neurologist, and the physician. There are significant problems with nurse education and support systems in haematology, virology, biochemistry, and radiography. The focus must change from treating a patient with multiple organ failure to preventing the advancement of organ failure if the community hospitals are to realise that patients need to be referred early. The pathophysiologic basis of the ALF patient exhibits is now well understood, and newer drugs are being developed with the goal of lowering aminotransferase levels and hyperbilirubinemia levels and possibly using anti-inflammatory cytokine therapy. Researchers now have a limited grasp of the pathophysiologic underpinnings of immunological dysfunction, hemodynamic disturbance, and multiorgan failure. A deeper understanding of the molecular underpinnings of these circulatory disturbances is expected to enhance our management of these patients. In order to investigate the roles of current therapies and emerging therapeutic modalities in pertinent multicenter trials, it will be critical that we arrange ourselves in collaborative groups going forward given the relatively limited number of patients with acute liver failure. This study summarises a variety of hepatic disorders linked with pregnancy, as well as their diagnosis and treatment. If these diseases are detected earlier, appropriate treatment may minimize foetal and maternal mortality.

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