

# Navigating Pregnancy with Portal Hypertension and Varices: A Case of High-Risk Harmony

Dr. Geethanjali M<sup>1</sup>, Dr. Nidhi Sharma<sup>2</sup>

<sup>1</sup>Postgraduate, Department of Obstetrics and Gynaecology, Saveetha Medical College, Saveetha University, Chennai, Tamilnadu, India- 602105

<sup>2</sup>HOD and Professor, Department of Obstetrics and Gynaecology, Saveetha Medical College, Saveetha University, Chennai, Tamilnadu, India-602105

\*Corresponding Author

Dr. Geethanjali M

Article History

Received: 08.08.2025

Revised: 15.09.2025

Accepted: 24.10.2025

Published: 05.11.2025

## Abstract:

Thrombocytopenia, which means a low platelet count, is rare in pregnancy and can lead to serious problems for both the mother and baby. We describe the case of a young pregnant woman who had very low platelets and was later found to have chronic liver disease with portal hypertension and swollen veins in her food pipe (esophageal varices). She received medicines and blood transfusions, but her bleeding could not be controlled. To save her life, the pregnancy had to be ended. This case shows how difficult it can be to manage pregnant women with blood and liver problems and stresses the importance of regular check-ups and teamwork between doctors for safe outcomes.

**Keywords:** Thrombocytopenia, portal hypertension, pregnancy, liver disease, esophageal varices.

## INTRODUCTION

Thrombocytopenia, characterized by a platelet count below 150,000/ $\mu$ L, is the second most frequently encountered hematological disorder during pregnancy (1). Thrombocytopenia in pregnancy occurs either due to obstetric conditions (like gestational thrombocytopenia, pre-eclampsia/eclampsia) or secondary to systemic disorders (like thrombocytopenic thrombotic purpura, immune thrombocytopenia) (2). Overall, about 75% of cases are due to gestational thrombocytopenia, 15-20% are secondary to hypertensive disorders; 3-4% are due to an immune process, and the remaining 1-2% are made up of rare constitutional thrombocytopenia, infections and malignancies (3).

Even though thrombocytopenia is a common abnormality in pregnancy, it seldom leads to life-threatening complications by itself. The management of thrombocytopenia focuses on the underlying cause. Platelet transfusion is usually not required to achieve a particular goal and is only for bleeding patients (1). The condition is often benign, especially in cases of gestational thrombocytopenia, which usually does not result in severe maternal bleeding (3,4). However, distinguishing between benign and potentially dangerous causes of thrombocytopenia is vital, as conditions like preeclampsia and immune thrombocytopenic purpura can pose significant risks to maternal and fetal well-being (2,5).

Portal hypertension, a severe complication of chronic liver disease, is a rare occurrence during pregnancy.

The precise mechanisms linking pregnancy and liver disease remain elusive, although hormonal fluctuations, immune system dysregulation, and altered hemodynamics are considered potential contributors (1,6). Pregnancy was once considered uncommon in women with liver scarring (cirrhosis). However, the increasing prevalence of liver disease, particularly among young women, is changing this. There's been a notable rise in the number of young women developing cirrhosis, with rates reaching 46.9 per 100,000 (7). As a consequence, it is becoming more frequent for women with cirrhosis to become pregnant (8,9). Pregnancy poses significant risks for both mother and child when liver disease, specifically cirrhosis, is present. Women with cirrhosis are more likely to experience worsening liver function due to increased pressure in the portal vein. As for the unborn child, there's a higher chance of premature birth, low birth weight, and potential harm from medications taken during pregnancy (7).

The concurrent presence of thrombocytopenia and portal hypertension in pregnancy poses significant diagnostic and therapeutic challenges. Effective management requires a comprehensive approach to optimize maternal and fetal outcomes. Early identification, thorough evaluation, and timely intervention are crucial to mitigate the risks associated with these potentially severe complications (4-6). By presenting this case, we aim to expand the limited knowledge of this rare clinical scenario, thereby improving our understanding of associated risks and potential management strategies.

## RESULTS AND OBSERVATIONS:

An 18-year-old female, gravida 2 para 1 living 1 (G2P1L1), presented at 15 weeks gestation with a history of thrombocytopenia and chronic liver disease with portal hypertension. She was referred from a primary health center for thrombocytopenia, with a platelet count of 72,000 cells/cumm.

The patient has a history of conception with an intrauterine contraceptive device (IUCD) in situ, which was removed at 8 weeks gestation. She perceives fetal movements well and has no history of bleeding manifestations, vaginal bleeding, or leaking. Additionally, there is no history of burning micturition, white discharge per vagina (PV), fever, abdominal pain, or any imminent signs and symptoms.

The patient experienced thrombocytopenia in her previous pregnancy two years ago. She has no known history of diabetes mellitus (DM), hypertension (HTN), cardiac disease, asthma, epilepsy, or tuberculosis (TB). The patient has no history of surgical procedures. She follows a mixed diet, has regular sleep and appetite, and has normal bowel and bladder habits. She has been married for three years and has no history of consanguineous marriage.

### Obstetric History:

- G1 (First Pregnancy): Two years ago, she delivered a boy weighing 2.6 kg via normal vaginal delivery (NVD). The baby was appropriate for gestational age (AGA), alive, and healthy.
- G2 (Current Pregnancy):
  1. First Trimester: The pregnancy was spontaneous with the last menstrual period (LMP) unknown and confirmed by a urine pregnancy test (UPT) at 2 months gestation. A dating scan was performed and showed a corresponding gestational age, and the nuchal translucency (NT) scan was normal at 1.3 mm. The patient has been taking Tab. Folic Acid 5 mg once daily regularly. There is no history of excessive vomiting, fever with rash, abdominal pain, bleeding/spotting PV, burning micturition, radiation exposure, or teratogenic drug intake.
  2. Second Trimester: The patient has been taking Tab. Iron and Tab. Calcium regularly. There is no history of abdominal pain, bleeding, or leaking PV, and no imminent signs or symptoms.

**Menstrual History:** The LMP is unknown. The patient had her menarche at 13 years, with regular menstrual cycles of 3-4 days every 30-day cycle, using 2-3 pads per day with moderate flow, and no history of clots or dysmenorrhea.

**On Arrival:** The patient was conscious, oriented, and afebrile. Her vital signs were stable: pulse 80 bpm, blood pressure 110/70 mmHg, respiratory rate 20/min, oxygen saturation 98%, and temperature 97°F.

### Systemic Examination:

- Cardiovascular System: S1 and S2 heart sounds were present.
- Respiratory System: Normal vesicular breath sounds were noted.
- Central Nervous System: No focal neurological deficits, Glasgow Coma Scale score of 15/15.
- Abdomen Examination: No tenderness or organomegaly was noted.

### Baseline and Radiological Investigations:

| CATEGORY                            | FINDING                                                                                                                                                                                                                                      |
|-------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 1. Hematological Parameters         | <ul style="list-style-type: none"><li>● Thrombocytopenia (platelet count 60,000 cells/<math>\mu</math>L)</li></ul>                                                                                                                           |
| 2. Liver Function                   | <ul style="list-style-type: none"><li>● Mildly elevated total and direct bilirubin levels<ul style="list-style-type: none"><li>● Decreased albumin (2.8 g/dL)</li></ul></li><li>● Mildly elevated gamma-glutamyl transferase (GGT)</li></ul> |
| 3. Coagulation Profile              | <ul style="list-style-type: none"><li>● Mildly prolonged prothrombin time (PT) and activated partial thromboplastin time (APTT)<ul style="list-style-type: none"><li>● Elevated D-dimer</li></ul></li></ul>                                  |
| 4. Abdominal ultrasound             | <ul style="list-style-type: none"><li>● Splenomegaly (20 cm)</li><li>● Increased renal cortical echogenicity</li></ul>                                                                                                                       |
| 5. Portal vein Doppler              | <ul style="list-style-type: none"><li>● Features suggestive of chronic liver disease with portal hypertension</li></ul>                                                                                                                      |
| 6. Upper gastrointestinal endoscopy | <ul style="list-style-type: none"><li>● Grade II-III esophageal varices</li></ul>                                                                                                                                                            |
| 7. Urine Analysis                   | <ul style="list-style-type: none"><li>● Elevated urine protein-to-creatinine ratio (0.107)</li></ul>                                                                                                                                         |

## Management:

Under subarachnoid block (SAB) and general anesthesia (GA), the patient was positioned in lithotomy, and the surgical site was painted and draped. The bladder was drained using a metal catheter. During the per speculum (P/S) examination, profuse bleeding through the os was observed. The per vaginal (P/V) examination revealed an open os with products of conception felt through the os, including the cord and placenta at the level of the os and fetal parts in the lower part. Injection Oxytocin 5 units IV infusion was administered. Products of conception were removed using sponge-holding forceps. Wedge resection of the fetus was performed, and the fetus was removed in parts. Suction and evacuation were carried out under USG guidance, and retained products were removed. A check curettage was performed, and no undue bleeding was noted through the os. Tablet Misoprostol 400 mcg was placed per vaginally. The bladder was catheterized, and clear urine was drained. Intraoperatively, the patient received 4 RDP, 1 PRBC, and 1 FFP transfusion.

The patient improved symptomatically during her hospital stay and requested discharge. She was discharged with appropriate advice for follow-up and management.

### ETIOLOGY (2,10,11)

Thrombocytopenia in pregnancy can arise from various etiologies, each with distinct pathophysiology and presentation. Below is a summary of potential causes:

1. Gestational Thrombocytopenia
2. Immune Thrombocytopenia (ITP)
3. Thrombotic Microangiopathy (TMA): TMA in pregnancy can be classified into pregnancy-specific and non-pregnancy-specific TMA, which differ in their management.

#### o Pregnancy-Specific TMA:

- Pre-eclampsia (PEC)
- Hemolysis, Elevated Liver Enzymes, Low Platelets (HELLP) Syndrome

- Acute Fatty Liver of Pregnancy (AFLP)

#### o Non-Pregnancy-Specific TMA:

- Thrombotic Thrombocytopenic Purpura (TTP)
- Atypical Hemolytic-Uremic Syndrome (HUS)
- 4. Disseminated Intravascular Coagulation (DIC)
- 5. Hereditary Thrombocytopenia (HT): Further classified based on platelet size, genetic defect, and inheritance pattern (e.g., WAS gene, HOXA11 gene, MYH9 disorders)

#### 6. Others:

##### o Bone Marrow Failure Syndromes:

- Aplastic Anemia
- Myelodysplastic Syndrome (MDS)
- Myeloproliferative Neoplasms (MPN)
- Leukemia/Lymphoma
- Marrow Infiltrative Disorders

##### o Paroxysmal Nocturnal Hemoglobinuria (PNH)

##### o Drug-Induced Thrombocytopenia

##### o Type IIB von Willebrand Disease (VWD)

##### o Heparin-induced thrombocytopenia (HIT)

The etiology of thrombocytopenia can also be categorized based on the trimester and platelet count:

#### First Trimester:

- Immune Thrombocytopenia (ITP) (most common)
- Hereditary Thrombocytopenia (HT)
- Others (as listed above)
- Thrombotic Microangiopathy (TMA)

#### Second Trimester:

- Platelet count greater than 100,000/ $\mu$ L:
- 1. Gestational Thrombocytopenia
- 2. ITP

#### 3. Pre-eclampsia/HELLP

#### 4. HT/Others/TMA

- Platelet count under 100,000/ $\mu$ L:

#### 1. ITP (most common)

#### 2. HT

#### 3. Pre-eclampsia/HELLP

#### 4. Others

#### 5. Gestational Thrombocytopenia

#### 6. TMA

#### Third Trimester:

- Platelet count more than 100,000/ $\mu$ L:

#### 1. Gestational Thrombocytopenia (most common)

#### 2. Pre-eclampsia/HELLP (second most common)

#### 3. Others/TMA/ITP/HT

- Platelet count less than 50,000/ $\mu$ L:

#### 1. Pre-eclampsia/HELLP (most common)

#### 2. ITP

#### 3. Others

#### 4. TMA

#### 5. Gestational Thrombocytopenia/HT

## DISCUSSION

Given the patient's presentation and the diagnostic findings, the thrombocytopenia in this case can be attributed to chronic liver disease with portal hypertension, exacerbated by pregnancy. The differential diagnosis includes gestational thrombocytopenia, immune thrombocytopenia, and potential thrombotic microangiopathy.

Initial evaluation of thrombocytopenia in pregnancy commences with a comprehensive blood count and peripheral blood smear examination. A meticulous review of the patient's medical and family history is essential for accurate classification of the thrombocytopenic etiology. Subsequent diagnostic testing may involve assessment of hemolytic markers, liver function parameters, and serological investigations for infectious agents including hepatitis B virus, hepatitis C virus, human immunodeficiency virus, *Helicobacter pylori*, and cytomegalovirus (6).

The trimester of thrombocytopenia onset can serve as a critical clue to its underlying etiology. Gestational thrombocytopenia, characterized by a gradual platelet count decline typically occurring during the mid-second trimester, represents one such example (2). Thrombocytopenia is frequently observed in pregnant women, with an onset often occurring in the first trimester and a gradual decrease throughout gestation, reaching its nadir at delivery. This phenomenon is primarily attributed to physiologic hemodilution resulting from an expanded plasma volume, coupled with heightened platelet activation and clearance (12,13).

Gestational thrombocytopenia (GT) constitutes the most prevalent etiology of thrombocytopenia during pregnancy, with a reported incidence ranging from 5% to 11% of all pregnancies (2). Hemodilution secondary to elevated plasma volume and accelerated platelet clearance may contribute to the pathogenesis of GT. This condition is commonly observed during the mid-to late-second or third trimesters of pregnancy (6). Asymptomatic women with a stable platelet count exceeding 100,000/ $\mu$ L do not necessitate additional investigation or specific interventions beyond routine platelet count monitoring (2). Elective cesarean delivery is not indicated for gestational thrombocytopenia, and the condition does not elevate the risk of maternal or fetal bleeding complications. Platelet counts typically recover to normal levels within one to two months postpartum (6,14)

Immune thrombocytopenic purpura (ITP) represents an acquired autoimmune process characterized by a pathophysiology of accelerated platelet destruction and ineffective thrombopoiesis. It comprises 1-4% of all thrombocytopenic cases during pregnancy (2,6,12). ITP is the most frequent etiology of thrombocytopenia (<50,000/ $\mu$ L) during the first and second trimesters. Patients with a prior history suggestive of ITP or a platelet count below 80,000/ $\mu$ L warrant investigation for this condition (2,15). The International Consensus Report (ICR) on ITP stipulates that a platelet count between 20,000 and 30,000/ $\mu$ L is typically safe for pregnancy in non-bleeding patients. A platelet count greater than 50,000/ $\mu$ L is optimal for delivery. Corticosteroids and intravenous immunoglobulin (IVIG) represent the initial therapeutic approach (13,15). While corticosteroids are generally considered safe during pregnancy, they can induce significant adverse effects, particularly when administered at high doses. These potential complications encompass gestational diabetes, hypertension, mood instability, and an elevated risk of cleft palate in fetuses exposed during the first trimester (6,16,17).

Thrombotic Thrombocytopenic Purpura (TTP) is a thrombotic microangiopathy characterized by the formation of microthrombi within small vessels. This pathologic process arises from a critical deficiency of

ADAMTS13, the metalloprotease responsible for cleaving ultra-large von Willebrand factor multimers (18,19). Von Willebrand factor (vWF) production is augmented during pregnancy, with a particular increase evident in the second and third trimesters (2). Published reports indicate that 10% to 25% of all diagnosed TTP cases occur in pregnant or postpartum women (19). Characteristic manifestations of TTP encompass microangiopathic hemolytic anemia, thrombocytopenia, fever, neurologic disturbances ranging from headache to coma, and renal impairment (18,20). Upon establishing a diagnosis of TTP, therapeutic interventions should persist until normalization of platelet count and reduction of lactate dehydrogenase levels are achieved (19). A diagnosis of TTP does not necessitate pregnancy termination or delivery based solely on gestational age (19). When TTP manifests during the first or second trimester, there is an elevated risk of fetal morbidity attributed to placental ischemia. Conversely, live birth rates ranging from 75% to 90% have been reported for TTP cases developing in the third trimester (6,20).

The primary maternal risks are associated with the decompensation of liver disease during pregnancy, a complication occurring in approximately 10-16% of cases. The Model for End-stage Liver Disease (MELD) has been utilized to assess this risk (7,21,22). This scoring system incorporates serum creatinine, bilirubin, and international normalized ratio (INR) values and was initially developed to predict mortality risk among cirrhotic patients undergoing surgical or invasive procedures (23). Westbrook et al. elucidated the utility of the MELD score in pregnancy. Patients with a MELD score of 6 or less experienced no complications. Conversely, all cases of decompensation occurred in women with a MELD score of 10 or greater. These findings underscore the correlation between the severity of liver disease, as quantified by the MELD score, and pregnancy outcome, rather than the underlying etiology of liver disease (7,21). Maternal mortality associated with cirrhosis ranges from 0% to 4%. Despite improvements in recent decades, this figure remains substantially elevated compared to the national maternal mortality rate of 0.01% (7,24). Elevated rates of placental abruption and postpartum hemorrhage have been documented in the literature (9,25). A comprehensive overview of potential risks is presented below (7).

#### Pathological Changes in Pregnancy and Chronic Liver Disease

- Obstetric Complications:
  - Higher risk of post-partum hemorrhage
  - Increased likelihood of pre-term delivery
  - Low birth weight
- Complications of Raised Portal Pressure:
  - Splenic artery aneurysms
  - Esophageal and pelvic varices
  - Ascites

Preterm birth can occur in up to two-thirds of cases, with the majority of infants born after 30 weeks gestation. Approximately one in five pregnancies result in delivery before 30 weeks. Prematurity is associated with higher UKELD and MELD scores at conception but is not influenced by the underlying etiology of liver disease (21). The incidence of spontaneous pregnancy loss prior to 24 weeks gestation is comparable to that of the general population (7,21). The peripartum period is associated with an increased risk of variceal hemorrhage, potentially precipitated by Valsalva maneuvers during vaginal delivery or iatrogenic injury to intra-abdominal varices during cesarean section (26,27).

A multidisciplinary approach is recommended for the management of varices in pregnancy. Second-trimester upper gastrointestinal endoscopy is indicated to assess the severity of esophageal varices. Pelvic MRI may be utilized to evaluate pelvic varices and guide obstetric planning. Vaginal delivery is generally considered safe for patients with mild varices, while cesarean delivery may be considered in cases of severe disease. However, evidence supporting specific delivery modes is limited, and individualization of care is essential (7).

In this specific case, termination of the pregnancy was deemed essential to preserve maternal life due to the patient's rapidly declining clinical status. The presence of severe thrombocytopenia significantly elevated the risk of postpartum hemorrhage, as manifested by profuse bleeding during the initial assessment. Despite aggressive medical management with uterotonic agents, hemorrhage persisted, posing a grave threat to maternal survival. Given the critical nature of the situation, surgical evacuation of the uterus was undertaken to achieve hemostasis and prevent catastrophic complications.

The second trimester and labor are periods of heightened risk for variceal bleeding (26). Acute hemorrhage necessitates immediate resuscitation with fluids, blood products, and antibiotics. While TERLIPRESSIN and OCTREOTIDE are effective in managing variceal bleeding, their potential for uterine vasoconstriction and ischemia must be carefully considered against the maternal mortality risk (28,29). Upper gastrointestinal endoscopy, optimally performed in the left lateral position, is essential for early identification and management of varices. In instances where endoscopic therapy proves unsuccessful, transjugular intrahepatic portosystemic shunt (TIPS) placement may be considered. However, it is important to acknowledge the potential risks associated with radiation exposure to the fetus, including increased susceptibility to childhood leukemia and congenital anomalies (7,30).

Our patient presented with chronic liver disease and portal hypertension, placing her at increased risk for variceal bleeding. The presence of grade II-III esophageal varices on endoscopy confirmed this risk. While the pregnancy was terminated due to severe thrombocytopenia and uncontrolled bleeding, the management guidelines highlight the importance of early endoscopic surveillance and careful consideration of delivery mode in similar cases.

Given the complexity of this case involving thrombocytopenia, portal hypertension, and a recent pregnancy termination, rigorous post-discharge monitoring is imperative. The underlying thrombocytopenia predisposes the patient to postpartum hemorrhage, necessitating vigilant observation of vital signs and blood counts. Regular assessment of liver function parameters is crucial to track disease progression and detect early signs of complications such as hepatic encephalopathy or ascites. Continued monitoring of platelet counts is vital to identify potential rebound thrombocytopenia or the development of new immune-mediated conditions. Furthermore, the surgical intervention necessitates vigilant surveillance for signs of infection. Lastly, given the significant physical and emotional trauma, comprehensive psychological support is indispensable for the patient's overall well-being.

## CONCLUSION

In summary, this case underscores the complexities and challenges associated with managing thrombocytopenia in pregnancy, particularly when compounded by chronic liver disease and portal hypertension. Differentiating between benign and life-threatening etiologies of thrombocytopenia is crucial for optimizing maternal and fetal outcomes. This case highlights the necessity for a multidisciplinary approach, involving obstetrics, hematology, gastroenterology, and surgical teams, to ensure comprehensive care. The decision to terminate the pregnancy was critical to managing severe thrombocytopenia and preventing life-threatening hemorrhage, demonstrating the importance of timely and decisive intervention in such high-risk cases.

Post-discharge, rigorous monitoring, and follow-up are imperative to manage the patient's underlying conditions and prevent complications. This case also emphasizes the importance of psychological support in the holistic care of patients undergoing significant medical and emotional challenges. Ultimately, this report contributes to understanding thrombocytopenia in pregnancy and provides insights into effective management strategies for similar cases in the future.

## REFERENCES

1. Mangla A, Hamad H. Thrombocytopenia in Pregnancy. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2024 Aug 2]. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK547705/>
2. Cines DB, Levine LD. Thrombocytopenia in pregnancy. *Blood*. 2017 Nov 23;130(21):2271–7.
3. Myers B. Diagnosis and management of maternal thrombocytopenia in pregnancy. *Br J Haematol*. 2012 Jul;158(1):3–15.
4. Tirago D, Yemane T, Tadasa E. Magnitude of Thrombocytopenia and Associated Factors among Pregnant Women Attending the Antenatal Care Service Unit of Wachemo University Nigist Ellen Mohammed Comprehensive Specialized Hospital

- Hosanna, Southern Ethiopia. *Adv Hematol*. 2024;2024(1):8163447.
5. Fogerty AE, Kuter DJ. How I treat thrombocytopenia in pregnancy. *Blood*. 2024 Feb 29;143(9):747–56.
6. Park YH. Diagnosis and management of thrombocytopenia in pregnancy. *Blood Res*. 2022 Apr 30;57(Suppl 1):79–85.
7. Faulkes RE, Chauhan A, Knox E, Johnston T, Thompson F, Ferguson J. Review article: chronic liver disease and pregnancy. [cited 2024 Aug 3]; Available from: <https://onlinelibrary.wiley.com/doi/10.1111/apt.15908>
8. Hagström H, Höijer J, Marschall HU, Williamson C, Heneghan MA, Westbrook RH, et al. Outcomes of Pregnancy in Mothers With Cirrhosis: A National Population-Based Cohort Study of 1.3 Million Pregnancies. *Hepatol Commun*. 2018 Nov;2(11):1299–305.
9. Shaheen AAM, Myers RP. The outcomes of pregnancy in patients with cirrhosis: a population-based study. *Liver Int Off J Int Assoc Study Liver*. 2010 Feb;30(2):275–83.
10. Chaudhary RK, Nepal C, Khanal N, Pathak R, Giri S, Bhatt VR. Management and Outcome of Heparin-Induced Thrombocytopenia in Pregnancy: A Systematic Review. *Cardiovasc Hematol Agents Med Chem*. 2015;13(2):92–7.
11. D'Andrea G, Chetta M, Margaglione M. Inherited platelet disorders: thrombocytopenias and thrombocytopathies. *Blood Transfus*. 2009 Oct;7(4):278–92.
12. Reese JA, Peck JD, Deschamps DR, McIntosh JJ, Knudtson EJ, Terrell DR, et al. Platelet Counts during Pregnancy. *N Engl J Med*. 2018 Jul 5;379(1):32–43.
13. Provan D, Arnold DM, Bussell JB, Chong BH, Cooper N, Gernsheimer T, et al. Updated international consensus report on the investigation and management of primary immune thrombocytopenia. *Blood Adv*. 2019 Nov 26;3(22):3780–817.
14. Rottenstreich A, Israeli N, Levin G, Rottenstreich M, Elchalal U, Kalish Y. Clinical characteristics, neonatal risk and recurrence rate of gestational thrombocytopenia with platelet count  $<100 \times 10^9/L$ . *Eur J Obstet Gynecol Reprod Biol*. 2018 Dec 1;231:75–9.
15. ACOG Practice Bulletin No. 207: Thrombocytopenia in Pregnancy. *Obstet Gynecol*. 2019 Mar;133(3):e181–93.
16. Park-Wyllie L, Mazzotta P, Pastuszak A, Moretti ME, Beique L, Hunnisett L, et al. Birth defects after maternal exposure to corticosteroids: prospective cohort study and meta-analysis of epidemiological studies. *Teratology*. 2000 Dec;62(6):385–92.
17. Yildirim Y, Tinar S, Oner RS, Kaya B, Toz E. Gestational diabetes mellitus in patients receiving long-term corticosteroid therapy during pregnancy. *J Perinat Med*. 2006;34(4):280–4.
18. Mariotte E, Azoulay E, Galicier L, Rondeau E, Zouiti F, Boisseau P, et al. Epidemiology and pathophysiology of adulthood-onset thrombotic microangiopathy with severe ADAMTS13 deficiency (thrombotic thrombocytopenic purpura): a cross-sectional analysis of the French national registry for thrombotic microangiopathy. *Lancet Haematol*. 2016 May 1;3(5):e237–45.
19. Thomas MR, Robinson S, Scully MA. How we manage thrombotic microangiopathies in pregnancy. *Br J Haematol*. 2016;173(6):821–30.
20. Scully M, Thomas M, Underwood M, Watson H, Langley K, Camilleri RS, et al. Thrombotic thrombocytopenic purpura and pregnancy: presentation, management, and subsequent pregnancy outcomes. *Blood*. 2014 Jul 10;124(2):211–9.
21. Westbrook RH, Yeoman AD, O'Grady JG, Harrison PM, Devlin J, Heneghan MA. Model for end-stage liver disease score predicts outcome in cirrhotic patients during pregnancy. *Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc*. 2011 Aug;9(8):694–9.
22. Jena P, Sheela CN, Venkatachala RP, Devarbhavi H. Obstetric Outcome in Women with Chronic Liver Disease. *J Obstet Gynaecol India*. 2017 Aug;67(4):263–9.
23. Malinchoc M, Kamath PS, Gordon FD, Peine CJ, Rank J, ter Borg PC. A model to predict poor survival in patients undergoing transjugular intrahepatic portosystemic shunts. *Hepatol Baltim Md*. 2000 Apr;31(4):864–71.
24. Saving Lives, Improving Mothers' Care - Lessons learned to inform maternity care from the UK and Ireland Confidential Enquiries into Maternal Deaths and Morbidity 2014-16. [Internet]. Oxford: National Perinatal Epidemiology Unit: University of Oxford; 2018 [cited 2024 Aug 3]. Available from: <https://www.npeu.ox.ac.uk/mbrace-uk/reports/confidential-enquiry-into-maternal-deaths>
25. Aggarwal N, Sawney H, Suril V, Vasishta K, Jha M, Dhiman RK. Pregnancy and cirrhosis of the liver. *Aust N Z J Obstet Gynaecol*. 1999 Nov;39(4):503–6.
26. Tran TT, Ahn J, Reau NS. ACG Clinical Guideline: Liver Disease and Pregnancy. *Off J Am Coll Gastroenterol ACG*. 2016 Feb;111(2):176.
27. Rasheed SM, Abdel Monem AM, Abd Ellah AH, Abdel Fattah MS. Prognosis and determinants of pregnancy outcome among patients with post-hepatitis liver cirrhosis. *Int J Gynaecol Obstet Off Organ Int Fed Gynaecol Obstet*. 2013 Jun;121(3):247–51.
28. Zhou X, Tripathi D, Song T, Shao L, Han B, Zhu J, et al. Terlipressin for the treatment of acute variceal bleeding: A systematic review and meta-analysis of randomized controlled trials. *Medicine (Baltimore)*. 2018 Nov;97(48):e13437.
29. Westbrook RH, Dusheiko G, Williamson C. Pregnancy and liver disease. *J Hepatol*. 2016 Apr;64(4):933–45.
30. Ingraham CR, Padia SA, Johnson GE, Easterling TR, Liou IW, Kanal KM, et al. Transjugular Intrahepatic Portosystemic Shunt Placement During Pregnancy: A Case Series of Five Patients. *Cardiovasc Intervent Radiol*. 2015 Oct;38(5):1205–10.