Thrombocytopenia: Gestational, Idiopathic, and Preeclampsia

160

Joshua Zevy Hamburger and Yaakov Beilin

Gestational Thrombocytopenia (GT)

Incidence

GT occurs in about 5% of pregnancies accounting for 74% of thrombocytopenia in pregnancy [1].

Presentation

Platelet counts typically range from 70 to 150,000 mm^{-3} in GT and remain relatively stable. However, some patients may develop lower platelet counts. Patients are almost always asymptomatic and will not describe any history of easy bleeding or bruising. GT generally occurs in the third trimester, and the platelet count returns to baseline after the pregnancy. There should be no history of thrombocytopenia in the non-gravid state, and the fetus does not typically suffer from platelet deficiencies [2].

Diagnosis

The diagnosis of GT is one of exclusions in the setting of mildly low platelets and no bleeding history. There are no specific tests to make the definitive diagnosis of GT [3].

Clinical Implications

While platelet counts may drop significantly from baseline, GT is not associated with platelet dysfunction. There are no studies that have determined the lowest absolute number that is safe for neuraxial anesthesia, though many practitioners will place a neuraxial anesthetic with platelet counts of 70,000 mm^{-3} with some proceeding at counts above 50,000 but only in stable asymptomatic disease [4].

Consultation

It is important to see these patients in antenatal consultation. Frequently, a hematologic consult is warranted for diagnosis and to guide management. The risks, benefits, and alternatives should be discussed with the patient. In general, most patients with GT will have a higher manual count and will be able to enjoy regional analgesia and anesthesia.

Idiopathic Thrombocytopenic Purpura (ITP)

Incidence

ITP accounts for 4% of thrombocytopenia in pregnancy [1].

Presentation

1. ITP generally presents earlier in pregnancy than GT and is often discovered before or between pregnancies.
2. Although platelet counts may drop below 50,000 mm^{-3}, ITP is most often a relatively stable disease process. That is, the platelets appear to function well. However, precipitous drops, while rare, have been attributed to ITP [3].

J. Z. Hamburger, M.D. (✉)
Department of Anesthesiology, Perioperative and Pain Medicine, Icahn School of Medicine at Mount Sinai, New York, NY, USA
e-mail: Joshua.Hamburger@MountSinai.org

Y. Beilin, M.D.
Department of Anesthesiology, Perioperative and Pain Medicine, Icahn School of Medicine at Mount Sinai, New York, NY, USA

Department of Obstetrics, Gynecology and Reproductive Science, Icahn School of Medicine at Mount Sinai, New York, NY, USA

S. K. W. Mankowitz (ed.), *Consults in Obstetric Anesthesiology*, https://doi.org/10.1007/978-3-319-59680-8_160

3. One critical difference between ITP and GT is the higher likelihood of having a positive bleeding history before, after, or during pregnancy in parturient with ITP.
4. In addition, the fetus may also have a decreased platelet count.

Diagnosis

A detailed history, physical, blood smear analysis, and the exclusion of other causes of low platelets, specifically preeclampsia and liver disease, is required to make the diagnosis of ITP. There is no specific lab test to confirm the diagnosis, and routine tests for platelet antibodies are not recommended due to poor sensitivity [5].

Management

Predelivery

1. Treatment includes corticosteroids and intravenous infusion of gamma globulin (IVIG), both of which take time to have an effect, particularly corticosteroids.
2. While therapies are tailored to individual patients, many will wait until the platelet count falls below 50,000 mm^{-3} before initiating treatment in the third trimester. IVIG is sometimes reserved for those with counts less than 10,000 mm^{-3} or those with counts below 30,000 mm^{-3} with active bleeding.
3. Splenectomy is a last resort that is appropriate even in pregnancy if counts do not increase above 10,000 mm^{-3} despite other therapies.
4. For delivery, platelet transfusions are indicated for platelet counts less than 30,000 mm^{-3} but should be considered if the patient is bleeding [6].

Anesthetic Management

There are no studies to date which dictate the lowest safe platelet count for neuraxial anesthesia [4]. However, in our practice, we utilize neuraxial anesthesia in most patients with ITP with a stable platelet count between 50 and 75,000 mm^{-3} and no bleeding history. However, we will not transfuse platelets in order to proceed with neuraxial anesthesia.

Pre-Eclampsia (PEC) and Hemolysis, Elevated Liver Enzymes, and Low Platelet Count (HELLP) Syndrome Are Another Common Cause of Thrombocytopenia in Pregnancy

Incidence

1. Preeclampsia (PEC) complicates 4% of all pregnancies in the United States, though the rate seems to be increasing [7]. HELLP complicates 0.5–0.9% of pregnancies [1].
2. Combined with HELLP syndrome, PEC is responsible for 24% of thrombocytopenia during pregnancy [1].

Presentation

For a full discussion of the presentation, please see the section on PEC. HELLP syndrome is characterized by the triad of hemolysis, elevated liver enzymes, and low platelets.

Diagnosis

1. There are no specific tests to determine which patients with PEC will develop clinically significant thrombocytopenia [8]. Therefore, it is mandatory to check a platelet count once PEC is suspected.
2. HELLP syndrome is diagnosed by the presence of features of the triad. More specifically, hemolysis can be detected by blood smear analysis or total bilirubin >1.2 mg/d or LDH > 600 U/l. Aspartate aminotransferase (AST) >70 U/l or double the laboratory normal value is used to evaluated liver function tests, and a platelet count below 100,000 mm^{-3} determines thrombocytopenia [9, 10].
3. However, occasionally, common findings are not present.
   (a) Up to 15% of HELLP syndrome occurs without hypertension or proteinuria [9]. In the absence of such findings, HELLP may be coined atypical PEC. Thrombocytopenia is typically present.
4. While delivery is presented as the definitive cure, up to 30% of cases of thrombocytopenia from PEC occur after delivery [11].

Clinical Implications

1. One of the challenges in managing HELLP syndrome is that patients may appear well despite the imminent progression of severe disease [12]. While proteinuria and hypertension may occur, HELLP syndrome often presents with right upper quadrant or epigastric pain, nausea and vomiting, headache, or fatigue [10], making the distinction between labor and serious disease difficult to distinguish. Beyond the triad, HELLP syndrome is also strongly associated with disseminated intravascular coagulopathy, placental abruptions, renal failure, pulmonary edema and effusions, cerebral edema, retinal detachment, liver hematomas, laryngeal edema, and death [10].
2. Anesthetic management
   (a) It is commonly believed that patients with PEC/HELLP pose a greater challenge for airway management based on anatomic and physiologic changes to their airways [13]. Therefore, most would prefer a neuraxial approach if at all possible.

(b) PEC can be a very dynamic process with precipitous drops in platelet counts. This may occur over just a few hours. To date, there are no specific tests to determine which patients will have such a course. Therefore, a platelet count and coagulation studies should be evaluated in all patients with elevated blood pressure, as defined at 140/90, before initiation of neuraxial analgesia. Both the absolute number and the trend of past counts should be taken into consideration prior to neuraxial placement.

(c) The platelet count should be periodically rechecked if there is a delay between the first result and neuraxial placement or before removal of the epidural catheters, especially in patients who progress to PEC with severe features or HELLP syndrome.

(d) Blood products should be available for parturient with HELLP syndrome as hemorrhage is a distinct possibility [14].

(e) For additional information on anesthetic management, see the chapter "Thrombocytopenia: An Introduction."

References

1. Burrows RF, Kelton JG. Fetal thrombocytopenia and its relation to maternal thrombocytopenia. N Engl J Med. 1993;329(20):1463–6.
2. Palta A, Dhiman P. Thrombocytopenia in pregnancy. J Obstet Gynaecol. 2016;36(2):146–52.
3. Shehata N, Burrows R, Kelton JG. Gestational thrombocytopenia. Clin Obstet Gynecol. 1999;42(2):327–34.
4. Katz D, Beilin Y. Disorders of coagulation in pregnancy. Br J Anaesth. 2015;115(Suppl 2):ii75–88.
5. Neunert C, Lim W, Crowther M, Cohen A, Solberg L, Crowther MA. The American Society of Hematology 2011 evidence-based practice guidelines for immune thrombocytopenia. Blood. 2011;117(16):4190–207.
6. George JN, Woolf SH, Raskob GE, Wasser JS, Aledort LM, Ballem PJ, et al. Idiopathic thrombocytopenic purpura: a practice guideline developed by explicit methods for the American Society of Hematology. Blood. 1996;88(1):3–40.
7. Ananth CV, Keyes KM, Wapner RJ. Pre-eclampsia rates in the United States, 1980-2010: age-period-cohort analysis. BMJ. 2013;347:f6564.
8. American College of Obstetricians and Gynecologists, Task Force on Hypertension in Pregnancy. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' task force on hypertension in pregnancy. Obstet Gynecol. 2013;122(5):1122–31.
9. Sibai BM. The HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets): much ado about nothing? Am J Obstet Gynecol. 1990;162(2):311–6.
10. Sibai BM, Ramadan MK, Usta I, Salama M, Mercer BM, Friedman SA. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). Am J Obstet Gynecol. 1993;169(4):1000–6.
11. Kirkpatrick CA. The HELLP syndrome. Acta Clin Belg. 2010;65(2):91–7.
12. Weinstein L. It has been a great ride: the history of HELLP syndrome. Am J Obstet Gynecol. 2005;193(3):860.
13. Izci B, Riha RL, Martin SE, Vennelle M, Liston WA, Dundas KC, et al. The upper airway in pregnancy and pre-eclampsia. Am J Respir Crit Care Med. 2003;167(2):137–40.
14. del-Rio-Vellosillo M, Garcia-Medina JJ. Anesthetic considerations in HELLP syndrome. Acta Anaesthesiol Scand. 2016;60(2):144–57.