

Thrombocytopenia in Pregnancy

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Background: Thrombocytopenia, defined as a platelet count of less than 150,000/ μ L, has been more commonly diagnosed in pregnant women in the last 20 years because platelet counts are included with the automated blood cell counters. Evaluation and treatment of this condition can be expensive and invasive and can result in an adverse outcome.

Methods: MEDLINE was searched from 1980 to present using the key words. "thrombocytopenia," "pregnancy," and "platelet." Case reports were excluded from literature review.

Results and Conclusions: Thrombocytopenia is the second most common hematologic abnormality during pregnancy and is usually a benign condition. Some patients, however, will have chronic medical disorders or pregnancy-induced conditions that require further evaluation and therapy. Even with its wide differential diagnosis, the cause of thrombocytopenia during pregnancy can usually be determined with a thorough history, physical examination, and directed laboratory studies. The challenge to the clinician is to weigh the risks of maternal and fetal bleeding complications against the benefits of diagnostic tests and interventions. (J Am Board Fam Pract 2002;15:290–7.)

Thrombocytopenia is classically defined as a platelet count of less than 150,000/ μ L.^{1,2} Counts from 100,000 to 150,000/ μ L are considered mildly depressed, 50,000 to 100,000/ μ L are moderately depressed, and less than 50,000/ μ L are severely depressed.³ Use of automated blood counters in routine prenatal screening has resulted in an increased diagnosis. Thrombocytopenia is second only to anemia as the most common hematologic abnormality during pregnancy.⁴ Some causes are unique to pregnancy, whereas others can be serious medical conditions that have been previously undiagnosed.

The increased recognition of maternal and fetal thrombocytopenia has resulted in controversies regarding management. In fact, screening at all for thrombocytopenia is an issue, because the reason for this common condition usually is benign. Performing studies on both the mother and the fetus can cause considerable morbidity and mortality, and the mode of delivery, eg, cesarean section

rather than vaginal delivery, has not improved outcome. Health care providers must weigh the benefits of interventions in relation to cost and morbidity against the risk of maternal and fetal bleeding complications.

The overall incidence of thrombocytopenia in pregnancy is 8%, but when patients with obstetric or medical conditions are excluded, the incidence drops to 5.1%.⁴ The most common cause is gestational thrombocytopenia (Table 1),¹ which accounts for almost three fourths of all cases. Hypertensive disorders account for 21%.¹ Thrombocytopenia occurs more commonly in patients with eclampsia (30%) than in patients with both mild and severe forms of preeclampsia (15%–18%).⁴ Of the patients who have severe preeclampsia, 4% to 12% will manifest criteria for HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet counts).⁴ Immune-mediated thrombocytopenia, including idiopathic thrombocytopenia purpura and neonatal alloimmune thrombocytopenia, is responsible for 4.1% of cases,¹ which is a proportionally small number. These conditions, however, can cause considerable morbidity and mortality and must be managed closely. Other, less common causes include rheumatologic disease (eg, systemic lupus erythematosus), disseminated intravascular coagulation, thrombotic thrombocytopenia purpura, fatty liver, antiphospholipid syndrome, hu-

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Table 1. Cause of Thrombocytopenia During Pregnancy.

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|---|
| Gestational thrombocytopenia |
| Pregnancy-induced hypertension |
| HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome |
| Spurious thrombocytopenia |
| Human immunodeficiency virus (HIV) infection |
| Immune thrombocytopenic purpura |
| Systemic lupus erythematosus |
| Antiphospholipid syndrome |
| Hypersplenism |
| Disseminated intravascular coagulation |
| Thrombotic thrombocytopenic purpura |
| Hemolytic uremic syndrome |
| Congenital thrombocytopenia |
| Medication |
| Folate deficiency |
| Primary bone marrow dysfunction: |
| Leukemia |
| Aplastic anemia |

man immunodeficiency virus (HIV) infection, and medications (Table 2).¹

Methods

MEDLINE was searched from 1980 to the present, using the key words “thrombocytopenia,” “pregnancy,” and “platelet.” Case reports were excluded from the literature review.

Major Causes

Gestational Thrombocytopenia

Gestational thrombocytopenia is a diagnosis of exclusion, and the following five characteristics make it more likely: (1) the degree of thrombocytopenia is usually mild to moderate, usually remaining greater than 70,000 μL (however, the lower level has never been established); (2) patients are asymptomatic with no history of bleeding; (3) there is no preconception history of thrombocytopenia; (4) an early gestation or preconception platelet count is normal; and (5) the platelet count returns to normal within 2 to 12 weeks postpartum.⁵ The early gestation or preconception platelet count becomes extremely important when differentiating this disorder from idiopathic thrombocytopenia purpura, with which it is commonly confused. Women with idiopathic thrombocytopenia purpura often have a

Table 2. Medications Associated with Thrombocytopenia.

| Class | Medication |
|-----------------|---|
| Antibiotics | Ampicillin Penicillin Rifampin |
| Diuretics | Thiazides Furosemide |
| Analgesics | Aspirin Acetaminophen Indocin |
| Anticonvulsants | Phenytoin Valproic Acid Carbamazepine |
| Miscellaneous | Methyldopa Heparin Digitalis Ranitidine Cimetidine Procainamide Gold compounds Cis-platinum Cyclosporin |

history of bleeding complications and have thrombocytopenia on a prepregnancy platelet count.

The cause of gestational thrombocytopenia is unclear, although it might be secondary to an accelerated platelet consumption and the increased plasma volume associated with pregnancy.⁵ Antiplatelet antibodies have been detected in the serum, but this finding does not differentiate it from idiopathic thrombocytopenia purpura, and the presence of antiplatelet antibodies is not specific for gestational thrombocytopenia.

Gestational thrombocytopenia essentially poses no risk to either the mother or fetus-neonate. A prospective cohort study by Burrows and Kelton⁶ of 756 women with a diagnosis of gestational thrombocytopenia showed that none of the mothers and only 1 infant, which had congenital bone marrow dysfunction diagnosed later, had any bleeding complication. In another study by Nagey et al⁷ of 730 pregnancies with platelet counts of less than 150,000/ μL , no neonate had a platelet count of less than 100,000 μL , and no bleeding complications were observed. Thus, it appears that mildly to moderately depressed platelet counts from gestational thrombocytopenia are not associated with any adverse effects to either the fetus, neonate, or mother, and no management is necessary other than periodic monitoring.

Pregnancy-Induced Hypertension

The thrombocytopenia in pregnancy-induced hypertension is moderate, and the platelets rarely drop below 20,000/ μ L.⁵ Hemorrhage is an uncommon event unless the patient develops disseminated intravascular coagulation, but thrombocytopenia can be a sign of worsening hypertensive disease. A slowly decreasing platelet count can be noted before the clinical manifestations of pregnancy-induced hypertension. When accompanied by microangiopathic hemolytic anemia, hemolysis, elevated liver enzymes, and low platelets, HELLP syndrome is diagnosed.

The causes of thrombocytopenia from pregnancy-induced hypertension and HELLP syndrome are unknown. One explanation is that it might be related to abnormal vascular tone with resultant accelerated platelet destruction, platelet activation, and coagulation defects.⁵ The increased levels of platelet-associated immunoglobulin G (IgG) that have been detected in patients with pregnancy-induced hypertension have been shown to be nonspecific and do not necessarily imply an immunologic basis for the thrombocytopenia.⁵ HELLP syndrome appears to be initiated by microvascular damage that results in platelet activation. Degranulation of the platelets is followed by vasospasm and further endothelial damage. The only known therapy for this cycle is delivery of the fetus. Patients with disseminated intravascular coagulation are at risk of catastrophic bleeding complications secondary to rapid consumption of platelets and coagulation factors.

Preterm neonates of mothers with pregnancy-induced hypertension are at risk for neonatal thrombocytopenia and resulting bleeding complications, such as excessive bleeding from blood draws or circumcision, mucosal bleeds in the gastrointestinal tract, cephalohematomas, subgaleal bleeding, and intracranial hemorrhages. Full-term infants have no specific risk when considering bleeding complications but are at an increased risk for intrauterine growth retardation.⁵

Immune Thrombocytopenic Purpura

Immune thrombocytopenic purpura is an immune-mediated process resulting in platelet destruction. It has an acute form, usually affecting children with viral infections, which is self-limited. The chronic form, which predominately affects women in their

second to third decades of life, accounts for 3% of all cases of thrombocytopenia during pregnancy.^{1,6}

Immune thrombocytopenic purpura is an autoimmune disorder in which patients produce IgG antiplatelet antibodies to their own platelet membrane glycoproteins. Increased platelet destruction occurs in the reticuloendothelial system, primarily the spleen. The rate of platelet loss is greater than production, and thrombocytopenia ensues.

Recent advances have produced antigen capture assays for platelet glycoproteins that help provide a definitive diagnosis of immune thrombocytopenic purpura in approximately two thirds of cases.⁸ Immune thrombocytopenic purpura has traditionally been a diagnosis of exclusion, however, and these studies are usually not necessary if a complete history, physical examination, and early platelet count are obtained. Many women already have a history of immune thrombocytopenic purpura before becoming pregnant, and the early gestation platelet count will help differentiate this disorder from gestational thrombocytopenia. If this count is less than 100,000/ μ L, immune thrombocytopenic purpura is likely,⁵ whereas the platelets in a patient with gestational thrombocytopenia are usually not affected until the second or third trimester. Five characteristics of immune thrombocytopenic purpura make the diagnosis likely: (1) moderate thrombocytopenia (50,000–100,000/ μ L), (2) a preconception or early gestation platelet count that is less than 100,000/ μ L, (3) normal to increased megakaryocyte levels as determined by bone marrow biopsy, (4) exclusion of other systemic disorder or use of drugs that might be associated with decreasing platelet counts (Table 2), and (5) an absence of splenomegaly.^{5,8}

Pregnancy does not seem to worsen the course of immune thrombocytopenic purpura, but cases of severe thrombocytopenia can cause serious morbidity and mortality to the mother and fetus. The mother is at risk for spontaneous hemorrhage, particularly if the platelet count drops to less than 20,000/ μ L. The maternal IgG will cross the placenta and could cause fetal thrombocytopenia. This condition is manifested by purpura, ecchymosis, melena, and even intracranial hemorrhage in the neonatal period. A retrospective case series showed that 12% to 15% of infants born to women with immune thrombocytopenic purpura develop platelet counts of less than 50,000/ μ L, with 3% having

major bleeding complications and less than 1% having intraventricular hemorrhage.^{9,10}

Neonatal Alloimmune Thrombocytopenia

Neonatal alloimmune thrombocytopenia results from maternal alloimmunization to fetal platelet antigens. The exact process of sensitization is unknown, but it is theorized that the maternal immune system recognizes as foreign the fetal platelets that cross the placental barrier.¹¹ The maternal immune system produces IgG antiplatelet antibodies to the fetal platelet antigens, particularly PLA1.¹¹ PLA1 is inherited from the father and is recognized as foreign by the mother's immune system.¹¹ The IgG antiplatelet antibodies then cross the placental barrier and cause platelet destruction.¹¹ It is the platelet equivalent of red cell hemolytic (Rh) disease of the newborn. It affects approximately 1 in 1,000 to 2,000^{4,5,11} live births and causes serious morbidity and mortality.

One difference between Rh disease and neonatal alloimmune thrombocytopenia is the occurrence in the first pregnancy. Fifty percent of neonatal alloimmune thrombocytopenia cases are discovered in the first live-born infant,¹¹ and subsequent pregnancies are affected in similar to increasing severity. Most cases are diagnosed after delivery. The maternal history and pregnancy are unremarkable, and the platelet count is often normal.

Neonates manifest evidence of severe thrombocytopenia either at delivery or during the first few hours of life. Petechia or ecchymosis appear over the fetal presenting part, their platelet count is severely depressed, and they have considerable bleeding when circumcised or when blood is drawn. The most serious complication, intracranial hemorrhage, occurs in 10% to 20% of all infants affected⁵ and occurs in utero 25% to 50% of the time.¹² Ultrasound findings of fetal bleeding complications can be used to differentiate neonatal alloimmune thrombocytopenia from immune thrombocytopenic purpura, which occurs during the neonatal period and is associated with maternal thrombocytopenia. Findings on a sonogram can include intracranial hemorrhage, porencephalic cysts, hydrocephalus, and fetal hydrops.⁵

Workup for Maternal Thrombocytopenia

A detailed medical history (to include family history, sexual history, and drug use) and physical

examination (Figure 1) help to narrow the focus when determining the cause of maternal thrombocytopenia. Questions focusing on the cardinal signs and symptoms of pregnancy-induced hypertension are helpful. Patients with immune thrombocytopenic purpura have a history of easy bruising, gingival bleeding (often while brushing their teeth), and menometrorrhagia. Patients should also be questioned about possible systemic diseases associated with thrombocytopenia, such as a history consistent with systemic lupus erythematosus, antiphospholipid syndrome, or possible HIV exposure. Also, certain medications can cause a depressed platelet count (Table 2).⁴ Signs of petechia, purpura, anemia, splenomegaly, and joint arthralgias found during the physical examination help aid the diagnosis.

The laboratory evaluation begins with a complete blood count and peripheral smear. The complete blood count is done to evaluate all the blood cell lines. Visualizing the smear is essential to rule out spurious thrombocytopenia. Clumping of platelets is common and can artificially lower the total platelet count. Antiplatelet antibodies can be measured, but this test does not differentiate gestational thrombocytopenia from immune thrombocytopenic purpura, is expensive, and has poor standardization.¹² It is therefore not recommended for routine use.

The gestational age helps in differentiating between diagnoses. A mother in the first or early second trimester who has thrombocytopenia will have either immune thrombocytopenic purpura or gestational thrombocytopenia – although the latter is much more common in later gestation. In general, women with no history of thrombocytopenia, a normal early-gestation platelet count, and mild thrombocytopenia are likely to have gestational thrombocytopenia. If the platelet count is abnormal early in gestation and drops to less than 70,000/mL, immune thrombocytopenic purpura is likely. At levels of less than 50,000/ μ L, immune thrombocytopenic purpura is almost certain.⁵ If the patient develops thrombocytopenia late in the third trimester or during the postpartum period, pregnancy-induced hypertension, thrombotic thrombocytopenia purpura, hemolytic uremic syndrome, acute fatty liver, and disseminated intravascular coagulation should be entered into the differential diagnosis, and the patient should be appropriately evaluated.

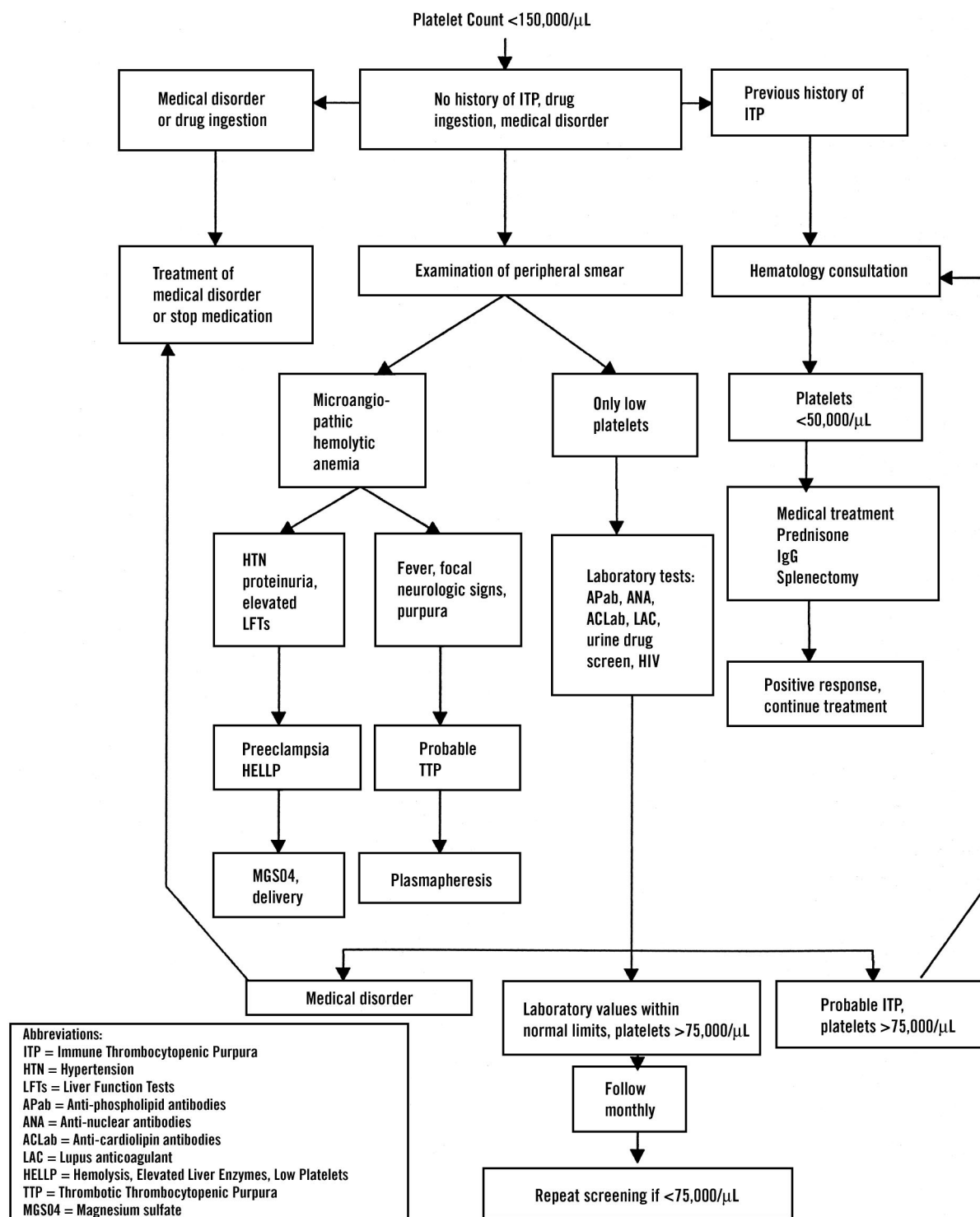


Figure 1. Evaluation of thrombocytopenia in pregnancy.

Screening for PLA1 antibodies on maternal platelets is not recommended as a routine test to rule out neonatal alloimmune thrombocytopenia.¹² Subsequent pregnancies have almost 100% risk of

the disorder recurring, however.¹¹ Strategies for screening include determination of maternal antiplatelet antibodies, zygosity of the paternal antigens, and determination of the fetal platelet anti-

gens.^{5,12} Fetal platelet antigens are fully expressed by 18 weeks' gestation, and a percutaneous umbilical artery sample can be performed at approximately 20 weeks' gestational age.¹² Fetal platelets are counted and antigen assays performed looking for PLA1. Chorionic villous sampling can increase fetal platelet sensitization and should not be performed.¹⁰

Management

Pregnancies with gestational thrombocytopenia are not at risk for fetal thrombocytopenia or maternal bleeding complications. No intervention, such as a fetal platelet count or cesarean section, is necessary.⁹ Periodic platelet counts, either once a trimester or every month, are recommended⁵ depending on the level of thrombocytopenia.

Thrombocytopenia secondary to pregnancy-induced hypertension or HELLP syndrome is treated by delivery of the fetus.¹³ The underlying pathophysiologic cause of the disorder is alleviated only after birth. In cases of severe thrombocytopenia, patients should be stabilized medically, and preterm infants should be given steroids to treat fetal lung maturity. Platelet transfusions are less effective in women with severe thrombocytopenia because of the ongoing process of platelet destruction.⁵ Two situations in which additional platelets should be given are (1) to treat severe thrombocytopenia with ongoing bleeding, and (2) to increase the platelet count to more than 50,000/ μ L for an operative delivery.⁵ The platelet count usually begins returning to normal within 72 hours of delivery.^{13,14} Therapies of uncertain benefit include plasmapheresis, steroids, and uterine curettage.⁵

Management of patients with immune thrombocytopenic purpura should be aimed at minimizing bleeding complications associated with severe thrombocytopenia. Because the platelets function normally, however, it is not necessary to maintain a normal count. Most authorities agree that 50,000/ μ L is the cutoff point for therapy in asymptomatic patients.^{5,8} Of course, patients with counts greater than 50,000/ μ L and bleeding complications require treatment. Operative deliveries, regional anesthesia, or other invasive procedures increase the risk of hemorrhage, and platelet counts of greater than 50,000/ μ L are usually recommended.^{5,8}

Initial therapy for immune thrombocytopenic purpura is prednisone, 1–2 mg/kg/d.^{4,5} This dosage

will usually increase platelet counts within 1 week. After 2 to 3 weeks of therapy, the dosage can be tapered by 10% to 20% per week to maintain adequate platelet levels.^{4,5} Two thirds of patients respond to this therapy, and one quarter will go into remission.^{4,5}

When steroids are not successful, treatment may include intravenous immunoglobulin. This therapy is also indicated for patients with platelet counts of less than 10,000/ μ L in the third trimester, for patients with platelet counts of less than 30,000/ μ L with bleeding complications, or for preoperative therapy.^{5,8} A response usually occurs within 6 to 72 hours, but the platelet count will usually decrease to pretreatment levels within 30 days.⁸

Platelet transfusions are only temporizing measures in life-threatening hemorrhages or preoperatively. The platelets transfused will also be affected by the antiplatelet antibodies and will be destroyed. If this therapy is undertaken, 6 to 10 units of platelets should be transfused, because the normal increase expected of 10,000/ μ L per unit will not be achieved.⁵

Splenectomy produces complete remission in approximately two thirds of patients, but this procedure is often difficult to perform during pregnancy and has serious fetal risks, particularly in the third trimester.⁵ Ideally, it should be performed during the first or second trimesters. It is appropriate therapy in patients who have failed both steroid and intravenous immunoglobulin therapy.^{5,8}

Therapy for immune thrombocytopenic purpura is often successful in improving the maternal platelet count but has been shown to be an unreliable indicator of the fetal platelet count.⁸ To date, there is insufficient evidence supporting maternal therapy to improve fetal outcome.⁸

Patients with immune thrombocytopenic purpura require special considerations during their prenatal care. Patients should avoid nonsteroidal anti-inflammatory agents, aspirin, and trauma.⁵ If a splenectomy is performed, they should be immunized against *Haemophilus influenzae*, pneumococcus, and meningococcus.⁸

The risk to the fetus, as previously discussed, results from maternal antiplatelet antibodies crossing the placenta and affecting the fetal platelets. Bleeding complications in a full-term fetus, particularly intracranial hemorrhage, are an uncommon but serious risk. Evaluation of the fetal platelet

count is controversial. The maternal course correlates poorly with fetal well-being. Maternal serologic findings, history of splenectomy, platelet count, and presence of antiplatelet antibodies do not provide an accurate assessment for the fetal risks of bleeding.^{5,8} Scalp sampling is often inaccurate and can produce a serious bleeding complication by itself.⁵ Cordocentesis carries a 1% to 2% risk of emergency cesarean delivery.⁵ With the low risk of intracranial hemorrhage in a full-term fetus and lack of evidence supporting an operative delivery, fetal evaluation of platelet counts is unwarranted in cases of maternal immune thrombocytopenic purpura.⁵

In the past, it has been common practice to perform cesarean sections on mothers with this disorder to lessen the risk of intracranial hemorrhage possibly caused by vaginal deliveries. It has also been postulated that fetal platelet counts of less than 50,000/ μ L place the fetus at greater risk of intracranial hemorrhage, and cesarean delivery should be considered.⁴ Cesarean delivery, however, has not proved to decrease the incidence of intraventricular hemorrhage in either situation, and the mode of delivery should not be influenced based on either finding.⁸ In a study of 474 neonates of mothers with immune thrombocytopenic purpura, 29% of vaginal and 30% of cesarean section births had a bleeding complication.¹⁵ Additionally, 4% and 3% of vaginal and cesarean births, respectively, had immune thrombocytopenic purpura.¹⁴ It is also well documented that platelet counts in neonates decrease during the first few days postpartum. This decrease could precipitate serious bleeding complications unrelated to the mode of delivery. It is therefore recommended that the mode of delivery be determined by obstetric indications.¹¹ Neonates from mothers with immune thrombocytopenic purpura do require postpartum serial monitoring of their platelet count.

Treatment of neonatal alloimmune thrombocytopenia during pregnancy focuses on preventing intracranial hemorrhage and its complications. Because of the high risk of complications during gestation, it is imperative to initiate treatment as soon as the diagnosis is made. Treatment is aimed at fetuses who test positive for PLA1 antigen or whose father is homozygous for the antigen.⁵ Intravenous immunoglobulin with steroids and fetal platelet transfusions are the standard therapy. Intravenous immunoglobulin given to the mother has

been shown to be effective, but no additional benefit is noted if given to the fetus. The platelets for fetal transfusion are maternal, which could worsen the alloimmunization, and a transfusion will need to be repeated every week as a result of their short half-life. If the count is greater than 50,000/ μ L, a trial of labor may be undertaken.^{5,10,16,17} For severe thrombocytopenia, a cesarean section is usually performed, although this has never been shown to decrease the rate of intracranial hemorrhage.⁵

Summary

Pregnancy complicated with thrombocytopenia is a challenge to the clinician. The myriad of disease processes, either pregnancy-induced disorders or preconception medical conditions, can cloud the correct diagnosis. It is important to remember the great majority of patients will have a benign condition, but a minority of patients who have a more serious disease are at risk for serious morbidity and mortality. With a thorough history, physical examination, focused laboratory evaluation, and appropriate consultation with obstetricians and hematologists, these patients uniformly have favorable outcomes and can be safely managed by family physicians.

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