

Idiopatic Trombocytopenia and Pregnancy

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Received: June 20, 2017; Published: July 05, 2017

Idiopathic thrombocytopenia or Immune thrombocytopenic purpura (ITP), is an immune-mediated acquired disease of adult and children. It is characterized by transient or persistent decrease of the platelet count and depending upon the degree of thrombocytopenia, increased risk of bleeding [1]. ITP is a common autoimmune disorder resulting in isolated thrombocytopenia. It can be presented as primary (isolated) or secondary (together with other conditions such as infections or some immune disorders). Characteristics of ITP are immune-mediated accelerated platelet destruction and suppressed platelet production. The etiology of ITP is not yet known, and the diagnosis continues to be one of exclusion [2].

ITP is defined as a condition where platelet count is less than 100×10^{9} /L (100,000/µL) without leukopenia or anemia. In the past, ITP was defined as a platelet count of less than 150×10^{9} /L, which is the threshold for a normal platelet count in most laboratories [3].

Thrombocytopenia affected 7 - 10% of all pregnancies, and it is the second most common hematological findings in pregnancy after anemia [3,4].

The causes of ITP specifics complicate the pregnancy, or haven't any relation with the pregnancy. Some of thrombocytopenies may occur with increased frequency during pregnancy. Some of the causes for thrombocytopenies are presented in Table 1 [5-7].

Pregnancy-specific	Pregnancy non-specific
Isolated thrombocytopenia	Isolated thrombocytopenia
Gestational thrombocytopenia	Primary ITP
	Secondary ITP
	Drug-induced thrombocytopenia
Thrombocytopenia with systematic disorders	Thrombocytopenia associated with systemic disorders
Preeclampsia/Eclampsia	• Viral infections (HCV, CMV, EBV etc.)
HELLP syndrome	• SLE
• Acute fatty liver of pregnancy	Antiphospholipid antibodies syndrome
	• Other conditions (nutritional deficiency, splenic sequestration,
	bone marrow disorders etc)

Table 1: Causes for thrombocytopenia in pregnancy [5-7].

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Thrombocytopenia affects up to 10% of all pregnancies. Pregnancy is associated with a physiological decrease of platelet count, mainly in the third trimester [3,4,8]. On the other hand, immune or idiopathic thrombocytopenia occurs on1/1,000 - 10,000 pregnancies, around for 3% of all pregnant with thrombocytopenia. ITP is an autoimmune disorder caused by development of immunoglobulin G (IgG) auto-antibodies that are directed against several platelet glycoproteins [9].

ITP is a clinical diagnosis, and the diagnostic methods aren't different in pregnant women compared to non-pregnant women. Pregnant women can have some bruising, mucosal bleeding and petechiae or they may be asymptomatic, with the severity of symptoms directly proportional to the degree of thrombocytopenia. There is no specific test that differentiates ITP from other causes of thrombocytopenia. Primary ITP is a diagnosis of exclusion. The causes of secondary thrombocytopenia such as viral infections and autoimmune disease should be ruled out clinically or with laboratory testing. However, patients with ITP usually have a prior history of ITP or other immune-mediated disorders [10].

The clinical management of the pregnant with ITP requires close consultation between the obstetrician and the haematologist. The decision to treat thrombocytopenia is determined by the patient's symptoms and the count of PLT. The goal of therapy is to prevent bleeding, and treatment is generally not required in patients with platelet counts greater than 20,000 to 30,000 x 10⁹/l if they are asymptomatic [11].

Most pregnant had mild to moderate thrombocytopenia and the pregnancies were uneventful. However, 31% required intervention to increase the PLT count. Despite remaining relatively stable through most of the pregnancy, PLT counts may decrease during the third trimester and monitoring should be more frequent. Generally, therapy late in gestation is based on the risk of maternal haemorrhage at delivery [11].

First-line therapies for ITP in pregnancy are corticosteroids and IV Immunoglobulin. A combination of the both may be effective when a patient does not respond to a single agent alone. Oral prednisone or prednisolone may be started at a low dose (10 - 20 mg/d) and adjusted to maintain a safe platelet count. Prednisone is generally safe in pregnancy, but it can increase weight gain and exacerbate hypertension and hyperglycaemia, resulting in adverse effects on pregnancy outcome. Very high doses of corticosteroids are not harmful to the fetus and may have an effect of accelerating lung maturation, but antenatal corticosteroids have not been found to have an effect or the neonatal platelet count and should not be administered to the near-term mother with this objective. The emotional effect of corticosteroids or their rapid withdrawal in the postpartum period should be carefully monitored and dosage should be tapered to avoid a rapid decrease in the platelet count after delivery. IV Immunoglobulin can be used for a rapid increase in platelet count or to maintain safe platelet counts when patients are not responsive to steroids or there is poorly tolerated side effects [3,12].

For the patients who didn't response to first-line therapy, second-line therapy may be required. Azathioprine has been safely administered during pregnancy, although immune impairment has been reported in some exposed infants. High-dose methylprednisolone may also be used in combination with IV Immunoglobulin or Azathioprine for the patient who is refractory to oral corticosteroids or IV Immunoglobulin alone or has a less than adequate response [3,12].

Anti-RhD immunoglobulin is not recommended as a first-line therapy because of concerns of acute haemolysis and anaemia. However, it has been used in refractory cases throughout pregnancy with successful outcomes. If anti-RhD (50 - 75 μ g/kg) is administered to a pregnant, the neonate should be monitored for a positive direct antiglobulin test, anaemia and jaundice as the antibody may cross the placenta [3].

Platelet transfusion alone is not helpful due to the quick destruction of transfused platelets as evidenced by a poor increment in the post-transfusion PLT count. The post-transfusion increment with platelet transfusion increases markedly after IV Immunoglobulin's infusion. In life threatening bleeding, IV Immunoglobulin followed by platelet transfusion with or without steroids may be required [13].

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ITP in the mother is not an indication for Caesarean section, and the mode of delivery in a pregnant patient with ITP is based on obstetric indications. Although there isn't universally accepted safe value of PLT count, the following can be taken as a general guideline for the intervention during pregnancy and during delivery, decreasing the risks of postpartal hemorrhage [14,15]. Safe PLT count recommended by Royal College of Obstetrics and Gynaecologist are presented in Table 2.

Intervention	Platelet Count (*10 ⁹ /l)
Antenatal, no invasive procedures planned	>20
Vaginal delivery	>40
Operative or instrumental delivery	>50
Epidural anesthesia	>60

Table 2: Safe PLT count as per RCOG [15,16].

Most neonatal hemorrhage occurs in 24 - 48 hours and is not related to trauma at the time of delivery. Determination of the fetal platelet count by periumbilical blood sampling or fetal scalp vein blood draws present a potential hemorrhagic risk to the fetus and may inaccurately predict a low platelet count. For this reason, fetal platelet count measurement is not recommended [6].

Pregnancy complicated with thrombocytopenia is a challenge to the obstetrician. The great majority of pregnant women with ITP will have a benign condition, but a minority of pregnant who have a more serious disease are at risk for serious morbidity and mortality. The diagnosis and management of ITP in pregnancy is similar to that in the nonpregnant adult patient, but the risks to the fetus must be taken into account when choosing treatment. Mode of delivery should be guided by obstetrical indications. A history of ITP or ITP in a previous pregnancy is not a contraindication to pregnancy, and majority of pregnant deliver nonthrombocytopenic or only mildly thrombocytopenic neonates.

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Citation: Emilija Jasovic-Siveska. "Idiopatic Trombocytopenia and Pregnancy". EC Gynaecology 5.1 (2017): 01-04.

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