Research Progress on the Etiology and Pregnancy Outcomes of Thrombocytopenia During Pregnancy

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Abstract: Thrombocytopenia during pregnancy is a common hematological abnormality with complex and diverse etiologies, involving physiological changes during pregnancy, immune factors, genetic factors, and other underlying diseases. Based on etiology, thrombocytopenia during pregnancy can be classified into pregnancy-specific and non-pregnancy-specific categories. The impact of thrombocytopenia caused by different etiologies on pregnancy outcomes varies. This article reviews the common etiologies of thrombocytopenia during pregnancy and related research progress on pregnancy outcomes, aiming to provide a theoretical basis for early clinical identification, diagnosis, and appropriate intervention to improve maternal and fetal prognosis.

***Keywords*:** thrombocytopenia during pregnancy, preeclampsia, immune thrombocytopenia, pathogenesis, pregnancy outcomes

1. Background

The incidence of thrombocytopenia during pregnancy is 7%-12%, which is four times higher than in non-pregnant women, making it the second most common hematological abnormality during pregnancy after anemia[1]. Pregnancy-specific causes of thrombocytopenia include gestational thrombocytopenia (GT), acute fatty liver of pregnancy (AFLP), preeclampsia, and HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count syndrome). Non-pregnancy-specific causes include primary immune thrombocytopenia (ITP), thrombotic thrombocytopenic purpura (TTP), systemic lupus erythematosus (SLE), antiphospholipid syndrome (APS), hematological malignancies, genetic factors, and drug-induced thrombocytopenia. These are relatively rare and are referred to as secondary immune thrombocytopenia, requiring comprehensive evaluation to avoid misdiagnosis[2]. Thrombocytopenia during pregnancy not only affects maternal health, such as increasing the risk of bleeding during delivery, but also adversely impacts fetal growth and pregnancy outcomes, including fetal growth restriction (FGR), intracranial hemorrhage, and neonatal thrombocytopenia. This article details the common causes of thrombocytopenia during pregnancy and their effects on pregnancy outcomes, aiming to raise awareness of this condition and improve pregnancy outcomes.

2. Etiological classification

2.1 Pregnancy-Specific causes

2.1.1 Gestational thrombocytopenia (GT)

GT is the most common cause of thrombocytopenia during pregnancy, accounting for about 80% of cases and 5%-8% of all pregnancies[3]. It is also known as gestational incidental thrombocytopenia (GIT)[4]. GT is a diagnosis of exclusion and lacks specific diagnostic criteria. Platelet counts typically recover 1-2 weeks postpartum. A platelet count below 100×109/L in the mid-to-late stages of pregnancy, without other hematological or clinical abnormalities, may suggest GT[5]. The mechanism of GT is often attributed to hemodilution, as blood volume increases significantly during pregnancy, peaking at 32-34 weeks[6]. Platelet counts are usually above 70×109/L, and no bleeding symptoms are observed, so excessive testing and intervention are generally unnecessary[3].

2.1.2 Hypertensive disorders of pregnancy (HDP)

HDP, including preeclampsia, eclampsia, and HELLP syndrome, is the second most common cause of thrombocytopenia during pregnancy, with an incidence of 15%-22%. Preeclampsia is characterized by high blood pressure, proteinuria, and edema. The mechanism of thrombocytopenia in HDP may involve vascular spasms, increased thromboxane synthesis, platelet aggregation, and accelerated platelet clearance[6]. Severe preeclampsia and HELLP syndrome are among the most

dangerous obstetric complications, often requiring early termination of pregnancy, which is a major cause of cesarean sections and preterm births[7]. Severe preeclampsia (SPE) and HELLP syndrome are among the most dangerous obstetric complications, often requiring early termination of pregnancy, which is a major cause of cesarean sections and preterm births. These conditions typically occur earlier in pregnancy, affecting placental function and leading to intrauterine growth restriction (IUGR) and intrauterine fetal death, posing significant risks to both maternal and fetal lives. In early pregnancy, if there are signs of preeclampsia or risk factors for preeclampsia, low-dose aspirin can be administered until 36 weeks to prevent the onset of preeclampsia[8]. Some studies suggest that low-dose aspirin only reduces the incidence of preeclampsia before 34 weeks of gestation[9]. After 34 weeks, the incidence of preeclampsia is positively correlated with maternal body mass index (BMI)[10]. Therefore, controlling diet and weight during pregnancy is crucial. In addition to low-dose aspirin, supplementation with vitamin D and calcium (≥1 g/day) may also have protective effects[11]. In cases of HELLP syndrome, if the condition is stable, expectant management may be considered. For pregnancies at earlier gestational ages, corticosteroids can be administered to promote fetal lung maturation. After fetal lung maturation, termination of pregnancy should be considered.

2.1.3 Acute fatty liver of pregnancy (AFLP)

AFLP is a rare but severe condition occurring in late pregnancy, primarily in primiparas around 35 weeks. It is associated with metabolic disturbances leading to fat accumulation in the liver[12]. The clinical manifestations are predominantly gastrointestinal symptoms such as nausea, vomiting, and fatigue. As the disease gradually progresses, it can involve multiple organs, leading to hypoglycemia, liver and kidney failure, coagulation abnormalities, hepatic encephalopathy, etc. The reason why AFLP causes thrombocytopenia remains unclear. The pathogenesis of AFLP is thought to be related to the physiological increase in free fatty acids during pregnancy (likely to support fetal growth), combined with maternal deficiency of 3-hydroxyacyl-CoA dehydrogenase or, less commonly, other enzymes involved in mitochondrial fatty acid metabolism. This results in the accumulation of intermediates in the metabolic pathway, leading to fat storage within the liver[13], thereby causing the development of fatty liver.

2.2 Non-Pregnancy-Specific causes

2.2.1 Immune thrombocytopenia (ITP)

Immune thrombocytopenia (ITP) is an acquired autoimmune disorder characterized by autoantibodies targeting platelet surface glycoproteins, which stimulate increased clearance of circulating platelets, leading to a reduction in platelet count[14]. This diagnosis is exclusionary, as there are no specific diagnostic markers[15]. Pregnancy-related immune-mediated thrombocytopenia can be classified into two main categories: maternal ITP and neonatal alloimmune thrombocytopenia (NAIT). The pathogenesis primarily involves Fcγ segment-mediated binding of antibody-coated platelets to Fcγ receptors on antigen-presenting cells, ultimately leading to enhanced platelet destruction in the spleen and liver. Maternal IgG autoantibodies can also cross the placental barrier into fetal circulation, where they destroy neonatal platelets, subsequently causing NAIT. Based on the timing of onset, pregnancy-associated ITP includes both pre-existing ITP in pregnant patients and ITP developing after conception. According to disease duration, pregnancy-related ITP can be categorized as newly diagnosed ITP (duration <3 months), persistent ITP (duration 3-12 months), or chronic ITP (duration >12 months). Clinical management should be tailored based on platelet count, gestational age, and specific clinical issues. Treatment is required when platelet counts fall below 20×109/L, even in the absence of bleeding manifestations. During mid to late pregnancy, patients with platelet counts above 20×109/L and no clinical symptoms generally do not require treatment but need regular monitoring. In the perioperative period or in cases of uncontrollable clinical bleeding, aggressive platelet-enhancing therapy should be administered even if the platelet count is above 30×109/L[16]. Glucocorticoids serve as first-line treatment for immune-mediated thrombocytopenia such as ITP, functioning by suppressing autoimmune reactions, reducing platelet destruction, and elevating platelet counts. Commonly used agents like prednisone require monitoring for adverse effects including elevated blood glucose, blood pressure fluctuations, and osteoporosis, with dose adjustments based on platelet response[17]. For glucocorticoid-intolerant patients, high-dose intravenous immunoglobulin (IVIG) provides rapid platelet count elevation and is often used in urgent situations such as pre-delivery thrombocytopenia with bleeding risk or steroid intolerance, despite its high cost[18]. Other therapeutic options include recombinant human thrombopoietin (rhTPO) as a second-line treatment for rapid platelet count elevation in pregnancy-associated ITP patients. Combination therapies (e.g., glucocorticoids with mycophenolate mofetil) demonstrate efficacy in over 90% of patients without increased side effects[19]. Medications such as azathioprine, cyclosporine A, danazol, and vinca alkaloids are rarely used in pregnant ITP patients due to potential teratogenic risks. Platelet transfusions are reserved for emergencies or preoperative preparation rather than therapeutic use[20]. Studies indicate that eltrombopag combined with cyclosporine can effectively increase platelet counts and improve clinical

symptoms in refractory ITP patients with favorable safety profiles[21]. Splenectomy may be considered when pharmacological therapies prove ineffective.

2.2.2 Thrombotic thrombocytopenic purpura (TTP)

Thrombotic thrombocytopenic purpura (TTP) is a life-threatening thrombotic microangiopathy characterized by microangiopathic hemolytic anemia, thrombocytopenia, and ischemic end-organ damage caused by platelet-rich microvascular thrombi. The pathophysiology of TTP is based on severe ADAMTS13 deficiency, and a diagnosis is established when ADAMTS13 activity levels are <10%[22]. TTP is further classified into hereditary TTP (also termed congenital TTP) and acquired TTP based on whether the ADAMTS13 deficiency is genetic or acquired. Hereditary TTP arises from recessive mutations in the ADAMTS13 gene and accounts for approximately 5% of TTP cases, while anti-ADAMTS13 autoantibodies are detectable in 75% of acquired TTP cases mediated by immune mechanisms. TTP can trigger severe complications such as stroke, seizures, myocardial infarction, acute kidney injury, acute pancreatitis, and pulmonary embolism, primarily due to microthrombosis in small vessels[23]. The condition involves specific proteolytic cleavage of von Willebrand factor (vWF)—a high-molecular-weight multimeric protein critical for normal hemostasis by recruiting platelets (PLT) to sites of vascular injury in high-shear blood flow. In the presence of anti-ADAMTS13 autoantibodies or low ADAMTS13 activity, high-molecular-weight vWF multimers accumulate, promoting PLT aggregation and subsequent thrombocytopenia[7], ultimately leading to thrombus formation. Early diagnosis and treatment are essential to reduce mortality. First-line therapy includes daily plasma exchange with fresh frozen plasma replacement and immunosuppression with corticosteroids. In TTP, the risk of venous and arterial thrombotic events is elevated, with most occurring after platelet count normalization[24].

2.2.3 Other causes

In addition to the aforementioned conditions, most other causes of thrombocytopenia are secondary and are collectively termed secondary thrombocytopenia, which refers to platelet reduction triggered by underlying etiologies. For example, systemic lupus erythematosus (SLE) can induce immune thrombocytopenia (ITP), a condition termed systemic lupus erythematosus–associated immune thrombocytopenia (SLE-ITP). Among ITP patients, the incidence of severe thrombocytopenia is approximately 3–20%, typically associated with the prolonged course of SLE[25]. In immune-related obstetric conditions, antiphospholipid syndrome (APS) is also common. APS is a systemic autoimmune disorder that can coexist with SLE, with approximately 30% of APS cases overlapping with SLE[26]. Clinically, APS is classified into vascular APS and obstetric APS. Vascular APS is characterized by thrombotic events in venous, arterial, or small vessels of various organs. When thrombosis progresses from superficial veins to small/medium vessels or involves multiple organs simultaneously, it is termed catastrophic APS (also known as a "thrombotic storm"). Unlike classic APS, catastrophic APS often occurs after infections, trauma, or surgery and typically manifests initially with fever, thrombocytopenia, muscle weakness, visual/cognitive disturbances, abdominal pain, renal failure, and disseminated intravascular coagulation[27]. Obstetric APS primarily presents with pregnancy complications and a lower incidence of thrombotic events.Diagnosis of APS relies on three main laboratory tests: anti-cardiolipin antibodies, anti-β2-glycoprotein I antibodies, and lupus anticoagulant testing. However, the mere presence of antiphospholipid antibodies without associated clinical symptoms does not meet diagnostic criteria for APS[28]. Low-molecular-weight heparin (LMWH) and low-dose aspirin are first-line treatments for obstetric APS complications. For refractory cases, hydroxychloroquine may be used[29]. Regular monitoring of liver/kidney function and platelet counts is essential during treatment, as LMWH may paradoxically induce thrombocytopenia (heparin-induced thrombocytopenia, HIT), likely mediated by platelet factor 4 (PF4). HIT involves IgG antibodies targeting PF4-heparin complexes, activating platelets and promoting thrombosis[31].

Other hematologic causes of thrombocytopenia include aplastic anemia, characterized by reduced hematopoietic stem cells and impaired proliferation/differentiation, leading to insufficient megakaryocyte production and subsequent thrombocytopenia. Thrombocytopenia with absent radii (TAR) syndrome is a rare inherited thrombocytopenia associated with bilateral radial aplasia. Genetically, TAR is defined by a microdeletion on chromosome 1 (encompassing the RBM8A gene) combined with a single-nucleotide polymorphism in the second RBM8A allele[32]. Recent studies highlight Helicobacter pylori infection as a contributor to thrombocytopenia. Maternal H. pylori infection may trigger platelet autoantibodies, destroying antibody-sensitized platelets in both mothers and neonates, and has been linked to childhood ITP[33]. Severe febrile illnesses, such as severe fever with thrombocytopenia syndrome (SFTS), also cause thrombocytopenia, though the mechanism remains unclear and may involve neutrophil activation and enhanced interferon signaling[34]. Viral infections (e.g., respiratory viruses, human papillomavirus) are also associated with thrombocytopenia, likely due to platelet hyperconsumption or accelerated apoptosis.

3. Pregnancy outcomes

3.1 Maternal outcomes

3.1.1 Bleeding risk

The risk of postpartum hemorrhage increases significantly when platelet counts fall below 50×109/L, especially after cesarean sections or perineal incisions. Severe cases may lead to shock and life-threatening conditions.

3.1.2 Thrombosis risk

Increased blood volume during pregnancy and postpartum blood concentration elevate the risk of thrombosis. Conditions such as APS and SLE further increase the risk of thrombosis, which may lead to complications such as cerebral infarction, myocardial infarction, and pulmonary embolism.

3.1.3 Hypertensive disorders of pregnancy

Poorly controlled HDP can lead to severe complications such as eclampsia and HELLP syndrome, increasing maternal mortality.

3.2 Fetal and neonatal outcomes

3.2.1 Fetal growth restriction (FGR)

The placenta, a vital organ interfacing between the fetus and mother, sustains fetal growth and development. However, placental function can be disrupted by conditions such as hypertensive disorders of pregnancy (HDP) and immune thrombocytopenia (ITP). In HDP, systemic small vessel vasospasm and vascular endothelial injury impair spiral artery remodeling in the uterus, compromising placental blood flow. This results in insufficient fetal nutrient supply, increasing risks of fetal growth restriction (FGR), fetal distress, and preterm birth[35].

Immune-mediated placental injury occurs when autoantibodies target placental components. For example, β2-glycoprotein I-dependent antiphospholipid antibodies bind to human trophoblasts, inducing phosphatidylserine externalization and activating the complement system via the classical pathway. This cascade causes placental damage, elevating risks of intrauterine growth restriction (IUGR) and intrauterine fetal demise[28].Additionally, placental pathologies themselves can contribute to thrombocytopenia. Conditions like placental abruption or placenta previa may lead to hemorrhage and increased platelet consumption. Placental dysfunction in HDP or immune-mediated disorders may also indirectly reduce platelet production by impairing maternal-fetal nutrient exchange or triggering systemic inflammatory responses.

3.2.2 Intracranial hemorrhage

Neonates born to mothers with severe thrombocytopenia are at higher risk of intracranial hemorrhage, which may lead to neurological sequelae or death.

3.2.3 Neonatal thrombocytopenia

Maternal autoantibodies can cross the placenta, leading to neonatal thrombocytopenia, which may manifest as skin and mucosal bleeding or purpura after birth.

4. Conclusion and future directions

The mechanisms underlying thrombocytopenia during pregnancy are complex and require further research. As a special population, pregnant women undergo significant physiological changes that contribute to these mechanisms. The application of artificial intelligence in this field shows promise but requires further validation[36]. Continued research is essential to improve maternal and fetal outcomes.

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