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Platelets And Parturition - A Case Series on Thrombocytopenia in Pregnancy

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Abstract:

Background: *Thrombocytopenia is a frequent hematological abnormality in pregnancy, affecting 7–10% of women. While gestational thrombocytopenia is usually benign, pathological causes such as immune thrombocytopenic purpura (ITP), inherited platelet function disorders, and thrombocytopenia associated with autoimmune disease carry significant maternal and perinatal risks.*

Objective: *To present and analyze four cases of thrombocytopenic disorders in pregnancy, highlighting the spectrum of etiologies, management challenges, and maternal–neonatal outcomes.*

Case Presentation: *This series describes four pregnant women with distinct thrombocytopenic disorders: Bernard–Soulier syndrome, chronic ITP, refractory ITP post-splenectomy, and ITP associated with autoimmune disease (APLA positivity). Clinical presentations included severe thrombocytopenia, bleeding diathesis, superimposed preeclampsia with placental abruption, and preterm premature rupture of membranes. Management strategies were individualized, incorporating corticosteroids, intravenous immunoglobulin, immunosuppressants, and platelet transfusions, with multidisciplinary input from hematology, obstetrics, anesthesiology, and rheumatology. Obstetric outcomes varied, with both cesarean and vaginal deliveries undertaken based on clinical indications. Maternal complications included postpartum hemorrhage, abruptio placentae, and fluctuating platelet counts postpartum. Neonatal outcomes ranged from neonatal immune thrombocytopenia requiring IVIg to extreme prematurity.*

Conclusion: *Thrombocytopenia in pregnancy requires accurate etiological diagnosis and a multidisciplinary, guideline-directed approach. Corticosteroids and IVIg remain cornerstone therapies in ITP, while platelet transfusion is vital in inherited platelet disorders. Delivery planning should be obstetric-driven with optimization of platelet counts. Neonatal monitoring is essential, particularly for infants of ITP mothers, to detect and manage neonatal thrombocytopenia. Early diagnosis, proactive peripartum care, and coordinated follow-up optimize maternal and neonatal outcomes even in complex, high-risk cases.*

Keywords: *Thrombocytopenia in pregnancy, Immune thrombocytopenic purpura, Bernard–Soulier syndrome, Refractory ITP, Autoimmune thrombocytopenia, Platelet transfusion, Neonatal immune thrombocytopenia*

INTRODUCTION:

Thrombocytopenia is one of the most common hematological abnormalities encountered in pregnancy, affecting up to 7–10% of all pregnancies. While the majority are attributable to benign gestational thrombocytopenia, pathological causes such as immune thrombocytopenic purpura (ITP), inherited platelet function disorders (e.g., Bernard-Soulier syndrome), and secondary thrombocytopenia associated with autoimmune disease represent high-risk entities. These conditions predispose to maternal complications, including antepartum and postpartum hemorrhage, and adverse perinatal outcomes such as intrauterine growth restriction, preterm birth, and neonatal thrombocytopenia. Management is complex, requiring a multidisciplinary approach involving obstetricians, hematologists, anesthesiologists, and when indicated, rheumatologists and dermatologists. Therapeutic strategies typically include corticosteroids, intravenous immunoglobulin (IVIg), immunosuppressants, and platelet transfusions, each tailored to disease severity, platelet count, and proximity to delivery. This case series presents four pregnant women with diverse

thrombocytopenic disorders, including Bernard-Soulier syndrome, chronic ITP, refractory ITP, and ITP associated with autoimmune disease and highlights the challenges of individualized peripartum management, maternal complications, and neonatal outcomes.

Case series description :

Case 1

A 31-year-old primigravida at 37 weeks + 4 days of gestation presented with a breech presentation. She was a known case of Bernard Soulier syndrome for the past 10 years and had a significant history of menorrhagia, recurrent epistaxis, and gingival bleeding. At the time of admission, her platelet count was 30,000/cumm, and peripheral smear revealed giant platelets without granules. She also had a background history of hypothyroidism and moderate anemia, which had been corrected antenatally. She was on oral Wysolone 40 mg once daily during pregnancy. In view of her thrombocytopenia and anticipated need for surgical intervention, she was optimized with four units of single donor platelet (SDP) transfusion preoperatively, in consultation with hematology and anesthesia. An elective lower segment cesarean section was performed for breech presentation. A live female infant was delivered with an Apgar score of 8/10 and 9/10 at 1 and 5 minutes respectively, and a birth weight of 2.75 kg. The immediate intraoperative and postpartum course was uneventful. However, on postoperative day 0, repeat platelet count dropped to 9000/cumm with persistence of giant platelets, raising the suspicion of Grey platelet syndrome. One unit of SDP was transfused, following which platelets improved to 16,000/cumm. On postoperative day 1, the platelet count rose to 38,000/cumm, and on day 2, it was 28,000/cumm, with persistence of giant platelets. The patient was discharged on a tapering dose of oral steroids, with hematology follow-up arranged.

Case 2

A 29-year-old primigravida at 36 weeks + 4 days of gestation, with a 4-year history of chronic immune thrombocytopenic purpura (ITP), was admitted for delivery. She had no significant obstetric or surgical history. At admission, her platelet count was critically low at 4000/cumm. She also had severe anemia antenatally, which was corrected before delivery. She had been on Omnacortil 20 mg daily, but in view of her low platelet counts, hematology advised escalation of therapy. She received intravenous methylprednisolone 125 mg stat, followed by two doses of IV immunoglobulin (IVIg) at 1 gm/kg body weight, 24 hours apart. Platelet monitoring showed an initial rise from 4000/cumm to 18,000/cumm, and subsequently to 46,000/cumm. Labour was induced with two doses of PGE2 gel, followed by artificial rupture of membranes and oxytocin augmentation. She progressed to a spontaneous vaginal delivery with episiotomy. A live female infant was delivered, with Apgar scores of 8/10 and 9/10 at 1 and 5 minutes respectively, and a birth weight of 2.46 kg. In the immediate postpartum period, the patient developed mild atonic postpartum hemorrhage (PPH), which was controlled medically. She also sustained cervical tears at 3 o'clock and 6 o'clock, which were sutured. Serial platelet monitoring showed values of 73,000/cumm on day 1, 64,000/cumm on day 2, and 67,000/cumm on day 3. She was discharged on tapering oral steroids with hematology follow-up.

Case 3

A 26-year-old woman, gravida 2 para 1 with one abortion, presented at 27 weeks + 5 days of gestation. She had a 2-year history of chronic ITP and was also diagnosed with chronic hypertension complicated by superimposed preeclampsia. She was APLA positive and ANA positive. Her obstetric history was significant for a 24-week medical termination of pregnancy in 2021, performed for severe uncontrolled hypertension, early-onset intrauterine growth restriction (IUGR), oligohydramnios, and thrombocytopenia, which required suction and evacuation with transfusion of 10 units of platelets. During the current pregnancy, she presented with early-onset fetal growth restriction, with bilateral uterine artery diastolic notching and umbilical artery high resistance flow on Doppler. On admission, her platelet count was 1.64 lakh/cumm. She was on hydroxychloroquine 200 mg daily and Wysolone 20 mg daily. She developed abruptio placentae in the setting

of chronic hypertension and superimposed preeclampsia, necessitating an emergency lower segment cesarean section. Intraoperatively, retroplacental clots were noted. A live female infant weighing 830 grams was delivered with Apgar scores of 4/10 and 7/10. Postoperatively, she developed hyponatremia on day 0, which was corrected. Ultrasonography revealed bilateral minimal pleural effusion, managed conservatively. She was maintained on IV hydrocortisone 50 mg every 8–12 hours in the immediate postoperative period, with repeat platelet counts showing 1 lakh/cumm with giant platelets. She was later transitioned to oral Wysolone 20 mg daily and HCQ 200 mg daily. She was discharged on tapering oral steroids with close hematology and rheumatology follow-up.

Case 4

A 26-year-old primigravida at 35 weeks + 1 day of gestation was admitted with preterm premature rupture of membranes (PPROM). She was a known case of chronic refractory ITP for the past 9 years. Her past history was notable for a subdural hemorrhage in 2015, managed medically with IVIg and methylprednisolone, and a laparoscopic splenectomy in 2015. Post-splenectomy, she had received three doses of pneumococcal vaccine and multiple platelet transfusions. She also had acne vulgaris, purpura, and foot eczema attributed to ITP. On admission, her platelet count was 45,000/cumm. She had been on Wysolone 50 mg once daily and azathioprine 100 mg daily. In view of impending delivery, azathioprine was stopped, and she was started on IV methylprednisolone 40 mg twice daily for 3 days, under hematology and rheumatology guidance. For dermatological complaints, she was prescribed liquid paraffin lotion, mometasone cream, and topical clindamycin gel. Labour was induced with two doses of PGE2 gel, following which she delivered vaginally with episiotomy. A male infant weighing 2.48 kg was delivered with Apgar scores of 8/10 and 9/10. The neonate developed neonatal immune thrombocytopenia (NITP), which was managed successfully with IVIg. In the postpartum period, her platelet counts showed significant fluctuations. On postnatal day 1, platelets were 38,000/cumm, for which she received 2 units of random donor platelets (RDP). On day 2, counts rose to 49,000/cumm, requiring another 2 units RDP transfusion. On day 3, platelets increased to 73,000/cumm, at which point IV steroids were stopped and oral steroids were initiated. By postnatal day 9, platelet count had improved significantly to 3.23 lakh/cumm. She was discharged on tapering oral Wysolone with hematology follow-up.

DISCUSSION:

Thrombocytopenia in pregnancy represents a wide clinical spectrum, from benign gestational thrombocytopenia to serious hematologic and autoimmune disorders. Our case series highlights the complexities of diagnosis, individualized management, and perinatal outcomes in women with rare and severe thrombocytopenic disorders.

Among our patients, etiologies ranged from inherited platelet function disorder (Bernard-Soulier syndrome, Case 1) to immune-mediated causes (chronic ITP, refractory ITP, ITP with autoimmune overlap, Cases 2–4). While ITP remains the most common pathological cause of thrombocytopenia in pregnancy, inherited disorders such as Bernard-Soulier and grey platelet syndrome, though rare, pose diagnostic and management challenges. Distinguishing these entities is crucial, as treatment approaches differ — ITP responds to immunomodulation, whereas Bernard-Soulier requires platelet transfusion support.

Our cases illustrate diverse maternal complications. Postpartum hemorrhage (PPH) in Case 2, aggravated by thrombocytopenia, was successfully managed with platelet transfusion and uterotonics. Placental abruption with severe preeclampsia in Case 3 reflects the interplay of autoimmune disease (APLA positivity) and obstetric complications. Refractory thrombocytopenia despite splenectomy and medical therapy (Case 4) underscores the difficulty of managing chronic ITP unresponsive to standard treatment. Management was guided by platelet counts, bleeding symptoms, gestational age, and anticipated delivery. Corticosteroids were

the backbone of therapy, with IVIg reserved for peripartum optimization (Cases 2 and 4) or neonatal thrombocytopenia. Azathioprine and hydroxychloroquine were continued in patients with autoimmune overlap, consistent with current safety data. Platelet transfusions were strategically employed around delivery in Bernard-Soulier and refractory ITP cases.

Mode of delivery in thrombocytopenic women should be based on obstetric indications rather than platelet count alone. In our series, both vaginal and cesarean deliveries were undertaken successfully once platelet thresholds were optimized. Neuraxial anesthesia was avoided when counts were below 80,000/cumm, in keeping with international guidelines, and general anesthesia or regional blocks were used cautiously.

Neonatal outcomes varied across cases. Neonatal immune thrombocytopenia (NITP) was observed in Case 4, successfully managed with IVIg. One neonate (Case 3) was extremely preterm due to abruption, illustrating the indirect effects of maternal disease. Importantly, all neonates born to ITP mothers require serial platelet monitoring, as counts may fall postnatally, reaching a nadir by day 3–5.

Key Clinical Insights

1. Multidisciplinary approach is indispensable, involving hematology, obstetrics, anesthesia, and in select cases rheumatology and dermatology.

2. Timely optimization of platelet counts with steroids, IVIg, or transfusions reduces maternal and neonatal morbidity.

3. Postpartum vigilance is critical, as platelet counts may fluctuate dramatically after delivery.

4. Accurate diagnosis (e.g., distinguishing inherited platelet disorders from ITP) prevents mismanagement and improves outcomes.

Guidelines based approach :

Antepartum Care

According to the RCOG Green-Top Guideline No. 71 on the management of inherited bleeding disorders in pregnancy, all women with a history of thrombocytopenia or platelet function disorder should receive pre-pregnancy counselling and care in a multidisciplinary setting involving obstetricians, haematologists, anaesthetists, and neonatologists. During pregnancy, maternal platelet counts should be monitored regularly, with increased frequency as gestation advances or if there are symptomatic drops. For women with immune thrombocytopenia (ITP), treatment is generally reserved for those with platelet counts below 20–30 × 10^9/L or in the presence of active bleeding. The first-line treatment recommended is oral corticosteroids, typically prednisolone, owing to its relative safety in pregnancy. Intravenous immunoglobulin (IVIg) is also recommended when a rapid rise in platelet count is required or in cases where steroids are contraindicated or ineffective. Inherited platelet function disorders such as Bernard-Soulier syndrome necessitate proactive planning for delivery, with arrangements for platelet transfusion support, antifibrinolytic cover such as tranexamic acid, and avoidance of medications that impair platelet function. In addition, women with co-existing autoimmune or prothrombotic conditions, such as antiphospholipid antibody syndrome, should continue specific therapies like low-dose aspirin or hydroxychloroquine under close supervision.

Intrapartum Care

The RCOG emphasizes that the mode of delivery should primarily be determined by obstetric indications and not solely by maternal platelet count. However, minimum platelet thresholds are suggested for safe delivery: a count of at least 50 × 10^9/L is recommended for vaginal birth, while a higher level of at least 80 × 10^9/L is considered appropriate if neuraxial anaesthesia (spinal or epidural) is anticipated or for caesarean section. In women with severe platelet function disorders, neuraxial anaesthesia is contraindicated due to the

high risk of neuraxial bleeding and spinal haematoma. For these women, general anaesthesia is preferred when surgical delivery is required. Intrapartum management for inherited platelet disorders should include the administration of prophylactic platelet transfusions at the onset of labour or prior to operative procedures, along with tranexamic acid to minimize postpartum haemorrhage risk. The use of instrumental delivery, particularly vacuum extraction, should be avoided to reduce the risk of neonatal intracranial haemorrhage if neonatal thrombocytopenia is suspected. Continuous maternal and fetal monitoring, a well-prepared blood bank with platelet concentrates, and a predefined plan for haemorrhage management are all mandatory components of care.

Postpartum Care

The postpartum period carries a significant risk of both maternal and neonatal complications in women with thrombocytopenia. RCOG guidelines recommend administration of tranexamic acid in women with inherited platelet disorders until lochia has substantially reduced, as this reduces the risk of secondary postpartum haemorrhage. Mothers should be monitored closely for delayed haemorrhage, postpartum thrombocytopenia relapses, or the need for repeated transfusion support. In women with ITP, corticosteroid or IVIg therapy should be continued or tapered according to clinical need, with careful follow-up to prevent rebound thrombocytopenia. Neonatal management is equally critical. Cord blood platelet counts should be obtained immediately after delivery, and neonates should undergo daily platelet monitoring until day 5, when the physiological nadir typically occurs. Neonatal immune thrombocytopenia, a well-recognized complication of maternal ITP, is managed with IVIg therapy when platelet counts fall below 30×10^9/L or if bleeding occurs. Platelet transfusions are indicated in life-threatening haemorrhage. RCOG also highlights the importance of clear discharge planning, follow-up in combined obstetric–haematology clinics, and patient education regarding the warning signs of postpartum haemorrhage or neonatal bleeding complications.

Platelet Count Thresholds

>50,000/cumm → adequate for vaginal delivery.
>80,000/cumm → preferred for cesarean delivery.
≥80,000–100,000/cumm → recommended for safe neuraxial anesthesia.

First-Line Therapy

Corticosteroids (Prednisone or equivalent, 0.5–1 mg/kg/day) are the mainstay for ITP. They are safe in pregnancy though prolonged use carries risks (GDM, hypertension, infections). IVIg (1 g/kg for 1–2 days) is effective when a rapid platelet rise is required, or in steroid-refractory cases.

Second-Line Options

Azathioprine is considered relatively safe in pregnancy and used as a steroid-sparing agent. Hydroxychloroquine is continued in autoimmune overlap (e.g., lupus, APLA). Splenectomy may be considered in refractory ITP but is ideally performed in the second trimester if unavoidable. Newer agents (e.g., thrombopoietin receptor agonists) are still under evaluation and not routinely recommended during pregnancy.

Inherited Platelet Disorders

In Bernard-Soulier syndrome and similar conditions, immunosuppression is ineffective. Platelet transfusion remains the cornerstone of peripartum management. Careful delivery planning and availability of platelet products are essential.

Neonatal Care

Neonates of thrombocytopenic mothers should undergo cord blood platelet counts at delivery. They Serial monitoring is required as counts may nadir on days 3–5 postpartum. NITP is managed with IVIg; platelet transfusions are rarely needed. Mode of delivery does not prevent NITP; thus, cesarean section is not indicated solely for maternal ITP.

CONCLUSION:

Thrombocytopenia in pregnancy represents a spectrum of disorders with varied maternal and neonatal implications. This case series emphasizes the importance of individualised, multidisciplinary management tailored to etiology, severity, and gestational timing. Corticosteroids and IVIg as cornerstone therapies in immune-mediated thrombocytopenia, with selective use of immunosuppressants and platelet transfusions. Obstetric-driven delivery planning, rather than basing mode of delivery solely on platelet counts. Proactive neonatal monitoring, particularly in infants of ITP mothers, to detect and treat NITP promptly. Despite the challenges including refractory disease, inherited disorders, and autoimmune overlap, maternal and neonatal outcomes can be optimized with timely interventions, evidence based therapy, and vigilant peripartum care. These cases highlight the need for greater awareness, early diagnosis, and guideline directed management, especially in resource limited settings where access to advanced hematology support may be restricted.

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