Management of thrombocytopaenia in pregnancy

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1. Scope and background

This guideline aims to describe the diagnostic approach to investigating thrombocytopaenia found in pregnancy, followed by a brief discussion on managing specific causes of thrombocytopaenia. This provides evidence-based information to health professionals to formulate a rational care pathway.

A platelet count of less than 150×10^9/L is defined as thrombocytopenia. Maternal thrombocytopaenia is in most cases mild and has no adverse outcome for both mother and fetus. Rarely a platelet count may be the presenting feature of a significant disorder with life threatening complications. Therefore management of thrombocytopaenia during pregnancy is challenging in both diagnostic as well as management of delivery.

2. Summary of key recommendations

2.1 Initial assessment

A platelet count below 150×10^9/L should warrant assessment for thrombocytopaenia during pregnancy. Errors during blood collection and automated haematology analysis may yield falsely low values. Hence low platelet counts should be reconfirmed with a repeat Full Blood Count (FBC) and a request for a manual platelet count.

2.2 Diagnosis of specific causes for thrombocytopaenia

A multidisciplinary approach with the haematologist and the obstetrician is required for optimal care.

If thrombocytopenia is confirmed, careful history, examination and laboratory workup is essential for the diagnosis.

A blood picture examination is vital to find the cause for thrombocytopenia. Microangiopathic hemolytic anaemia (MAHA) in the blood picture, which is a hemolytic process with red cell fragmentation and thrombocytopenia, can be associated with severe Preeclampsia(PE), HELLP syndrome, TTP (Thrombotic Thrombocytopaenic Purpura), aHUS (atypical Haemolytic Uraemic Syndrome), AFLP(Acute Fatty Liver in Pregnancy) and Disseminated Intravascular Coagulation (DIC).

To differentiate between above conditions apart from a good clinical assessment, serum creatinine, lactate dehydrogenase (LDH), Prothrombin Time (PT), Activated Partial Thromboplastin Time (APPT), liver function tests (bilirubin direct/ indirect, albumin, total protein, transferases, and alkaline phosphatase) and ultrasound scan of abdomen are required.

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Gestational Thrombocytopaenia (GT) is the most common reason for low platelets in pregnancy. It is a diagnosis of exclusion. GT commonly develops in the latter half of the pregnancy, and the platelet count is usually above 70×10^9/L. The diagnosis of GT is less likely if the platelet count falls below 70×10^9/L.

Incidence of Immune-Thrombocytopaenic Purpura (ITP) is approximately in 1/1000-1/10 000 pregnancies. It is the commonest cause of a low platelet count presenting in the first and second trimesters.

PE is the most common cause of thrombocytopenia associated with MAHA presenting in the late second or the third trimester of pregnancy. Infrequently, it may appear during the first week postpartum.

HELLP syndrome may be a variant of PE characterized by more severe thrombocytopenia, more fulminant MAHA and profoundly elevated liver function tests.

Even though it is rare, microangiopathies such as TTP, aHUS and AFLP should be carefully looked into when the woman presents with acute clinical features.

Patients with Antiphospholipid Syndrome (APLS) and Systemic Lupus Erythematosus (SLE) may also present with thrombocytopenia.

Antinuclear Antibodies (ANA), thyroid function test, antiphospholipid antibodies and viral screening should be considered if clinically indicated.

2.3 Management of GT

Antenatal platelet count should be monitored every 2 to 4 weeks.

No special management is required.

When the platelet count is less than 100×10^9/L, the woman should be referred to an anaesthetist prior to delivery.

GT is not associated with neonatal thrombocytopenia.

2.4 Management of ITP in pregnancy

In ITP, a multidisciplinary approach involving the obstetrician, haematologist, anaesthetist, transfusion physician and neonatologist are required for optimal care.

FBC should be monitored at 2-4 weeks intervals or more frequently if indicated.

If the platelet count is less than 30×10^9/L or bleeding manifestations are present, first-line therapy is oral corticosteroids, and if a rapid platelet increment is required as in impending delivery or significant bleeding, intravenous immunoglobulin (IVIg) should be administered.

Treatment to increase the platelet count for delivery is initiated by 36 weeks or earlier if early delivery is planned.

Delivery should be planned in a setting where 24 hours blood bank facilities and ICU care are available.

The obstetric team should liaise with the haematologist, the transfusion physician and the anaesthetist when planning delivery.

Platelets count of at least 50×10^9 /L should be obtained for safe delivery.

If platelet count of less than 50×10^9/L, platelet concentrate should be available on-site for transfusion if necessary.

Caesarean delivery is reserved for obstetric indications only.

At a platelet count of $\geq 80\times10^9$/L, in the absence of other hemostatic abnormalities, regional anaesthesia can be performed.

IgG antibodies in ITP are known to cross the placenta, causing thrombocytopenia in the fetus and neonate. The occurrence of intracranial haemorrhage (ICH) is a major neonatal concern. Measures should be taken to avoid traumatic delivery to the baby and the mother during delivery. Scalp electrodes, fetal blood sampling, vacuum and difficult forceps delivery should be avoided. If instrumental delivery is indicated, forceps is the choice.

Prophylactic measures should be taken to prevent Postpartum Haemorrhage (PPH), which includes active management of the third stage of labour, oxytocin infusion and intravenous tranexamic acid.

Pregnant women who are on long term steroids should have regular blood sugar monitoring with PPBS and blood pressure monitoring.

Non-Steroidal Anti-Inflammatory drugs (NSAIDs) should be avoided for postpartum or postoperative analgesia in women with thrombocytopenia due to increased hemorrhagic risk.

2.5 Management of thrombocytopaenia due to PE, HELLP syndrome and AFLP

Urgent delivery should be arranged as it is the mainstay of treatment.

Maternal corticosteroids should be administered considering fetal maturity to reduce fetal respiratory morbidity.

If DIC present, supportive care with FFP, platelet and cryoprecipitate should be administered with advice from the haematologist.

2.6 Management of thrombotic thrombocytopaenic purpura/atypical hemolytic uraemic syndrome

Despite the diagnostic challenge, plasma exchange (plasmapheresis) needs to be commenced as soon as TTP/aHUS is suspected. Management requires a multidisciplinary approach with the transfusion physician, the obstetrician and the haematologist. Plasma transfusions should be given if there is any delay in plasmapheresis.

In TTP, plasmapheresis should continue daily until the platelet count is maintained in the normal range (>150×10^9/L) for a minimum of 2 days.

Platelet transfusions are contraindicated as they are known to precipitate or exacerbate thrombosis.

3. Introduction

Thrombocytopenia is a common haematological condition affecting 7-10% of the pregnant population[1]. It occurs four times more frequently in pregnancy than in non-pregnant women and is the second leading cause of blood disorders in pregnancy after anaemia[2]. Thrombocytopaenia is defined as a platelet count of less than 150×10^9/L.

GT accounts for 70-80% of all cases of thrombocytopaenia in pregnancy. Hypertensive disorders explain approximately 20% of thrombocytopenia, and immune thrombocytopenia accounts for about 3-4%. Other etiologies such as TTP and HUS are considered rare in pregnancy but carry high morbidity and mortality for both the mother and fetus[3].

4. Recommendations and discussion

4.1 Initial assessment

A platelet count below 150×10^9/L should warrant assessment for thrombocytopaenia during pregnancy. Errors during blood collection and automated haematology analysis may yield falsely low values. Hence low platelet counts should be reconfirmed with a repeat FBC and a request for a manual platelet count. All new patients presenting with thrombocytopenia need reconfirmation of the low platelet number with a repeat FBC and a manual platelet count. The repeat FBCsample should be taken from a direct, uncomplicated venipuncture and added into an EDTA tube and mixed well. This will prevent minute clot formation in the sample leading to erroneously low platelet values. Assessing the manual platelet count will exclude any errors in automated platelet analysis.

If large platelet aggregates are detected in the blood smear taken from an EDTA sample with thrombocytopenia reported in the automated FBC results, it is considered as EDTA induced pseudo thrombocytopenia. If it is necessary to obtain the accurate platelet number, blood should be collected into acitrated tube and sent to the laboratory for analysis within 15 minutes of collection. As the platelets can undergo deterioration in a citrated sample, immediate analysis is vital, and the laboratory should be informed of the procedure before collecting blood from the patient to a citrated sample. When thrombocytopenia is confirmed, careful history, examination, and laboratory workup are needed to arrive at a diagnosis.

History should include.

- recent history of fever (to exclude viral infections such as dengue fever)
- the presence of severe headaches and other neurological manifestations (seen in PE and TTP)
- past history of thrombocytopenia (favouring ITP)
- symptomatic anaemia and recurrent infections (bone marrow failure/haematological malignancy)
- past history of pregnancy-associated thrombocytopenia
- history of connective tissue disorders (SLE and APLS)

- hypothyroidism
- liver disease
- drug history
- past and family history of bleeding disorders (rare inherited bleeding disorders such as type IIB Von Willebrand disease).

On examination, it is uncommon to detect bleeding manifestations unless the platelet count is significantly low. It is vital to check the blood pressure (PE, HELLP syndrome), abdominal tenderness (PET, HELLP syndrome, AFLP), anaemia, lymphadenopathy, hepatosplenomegaly (haematological malignancy) and neurological manifestations (severe PE, TTP).

Reduction of serum platelet counts is arbitrarily considered mild if the count is $<150\times10^9$/L, moderate at 50-100×10^9/L and severe at $<50\times10^9$/L.

4.2 Diagnosis of specific causes for thrombocytopaenia

A multidisciplinary approach with the haematologist and the obstetrician is required for optimal care.

If thrombocytopenia is confirmed, careful history, examination and laboratory workup is essential for the diagnosis.

A blood picture examination is vital to find the cause for thrombocytopenia. MAHA in the blood picture, a hemolytic process with red cell fragmentation and thrombocytopenia, can be associated with severe PE, HELLP syndrome, TTP, aHUS, AFLP and DIC[4].

To differentiate between above conditions apart from a good clinical assessment, serum creatinine, lactate dehydrogenase (LDH), Prothrombin Time (PT), Activated Partial Thromboplastin Time (APPT), liver function tests (bilirubin direct/ indirect, albumin, total protein, transferases, and alkaline phosphatase) and ultrasound scan abdomen are required.

GT is the most common reason for low platelets in pregnancy[5]. It is a diagnosis of exclusion. GT commonly develops in the latter half of the pregnancy, and the platelet count is usually above 70×10^9/L. The diagnosis of GT is less likely if the platelet count falls below 70×10^9/L.

The incidence of ITP is approximately in 1/1000-1/10 000 pregnancies. It is the commonest cause of a low platelet count presenting in the first and second trimesters[6].

PE is the most common cause of thrombocytopenia associated with MAHA presenting in the late second or third trimester of pregnancy. Infrequently, it may appear during the first week postpartum[7].

HELLP syndrome may be a variant of PE characterized by more severe thrombocytopenia, more fulminant MAHA and profoundly elevated liver function tests5. Even though it is rare, microangiopathies such as TTP, aHUS and AFLP should be carefully looked into when the woman presents with acute clinical features.

Patients with APLS and SLE may also present with thrombocytopenia.

ANA, thyroid function test, antiphospholipid antibodies and viral screening should be considered if clinically indicated.

GT is a condition with mild to moderate platelet drop and is a diagnosis of exclusion. The platelet count in GT is usually above 70×10^9/L. The patient is asymptomatic, and thrombocytopenia is commonly detected in the second half of pregnancy. The platelet count spontaneously reverts to normal within the first two months of postpartum but can recur in subsequent pregnancies.

The incidence of ITP is approximately in 1/1000-1/10 000 pregnancies. It is the commonest cause of a low platelet count presenting in the first and second trimesters[6]. Despite improved understanding of the pathophysiology, there is no specific diagnostic test, and, like GT, it is a diagnosis of exclusion. The presence of other autoimmune phenomena or a low platelet count during pre-pregnancy can help to diagnose.

Thrombocytopaenia associated with hypertensive disorders is the most frequent causes in the late second trimester onwards8.Therefore PE screening should be carried out to rule out hypertensive variants (HELLP, AFLP).

HELLP syndrome, which affects 0.6% of pregnant women, is a severe variant of pre-eclampsia. However, in 15-20% of cases of HELLP syndrome, neither hypertension nor proteinuria is present[9].

AFLP occurs in 1 in 5000 to 10 000 pregnancies and is more common with multiple gestations than in singletons. Up to 75% of women present with nausea or vomiting, and 50% have abdominal pain or signs and symptoms similar to PE. Although it is often difficult to differentiate HELLP from AFLP, evidence of hepatic insufficiency, including hypoglycemia, DIC, or encephalopathy, is seen more often in AFLP[5].

TTP is an acute life-threatening disorder associated with thrombocytopenia, MAHA and microvascular thrombosis. It results from a deficiency of the enzymeADAMTS13, required to cleave secreted ultra-large von Willebrand factor molecules (ULVWF). An inherited deficiency or acquired reduction of ADAMTS13 due to IgG autoantibodies to ADAMTS13 leads to persistence of ULVWF molecules resulting in abnormal platelet aggregation and microvascular thrombosis. Pregnancy is an important precipitant of acute TTP, accounting for approximately 5-10% of all cases of TTP in women[4]. TTP classically consists of a pentad of thrombocytopenia, MAHA, neurological signs, renal impairment and fever. However, TTP commonly presents without the full spectrum of the pentad. Laboratory features indicating a diagnosis of TTP are MAHA with many schistocytes in the blood picture, increased Lactate dehydrogenase (LDH), which is often out of proportion to the degree of haemolysis due to associated tissue ischemia, normal PT/APTT and possibly elevated serum creatinine level[10].

aHUS is a rare MAHA associated with pregnancy. The majority of cases occur during the postpartum period. The patient has MAHA, thrombocytopenia and severe renal impairment. The outcome is severe, with two-thirds of cases developing end-stage renal failure within one month[4].

4.3 Management of GT

Antenatal platelet count should be monitored every 2 to 4 weeks.

No special management is required.

When the platelet count is less than 100×10^9/L, the woman should be referred to an anaesthetist prior to delivery.

GT is not associated with neonatal thrombocytopenia. GT does not require treatment except periodic monitoring of platelet count. The thrombocytopenia resolves spontaneously. If the thrombocytopenia persists beyond 6 to 8 weeks, the patient should undergo further haematological investigations.

4.4 Management of ITP in pregnancy

In ITP, a multidisciplinary approach involving the obstetrician, haematologist, anaesthetist, transfusion physician and neonatologist, is required for optimal care.

FBC should be monitored at 2-4 weeks intervals or more frequently if indicated.

If the platelet count is less than 30×10^9/L or bleeding manifestations are present, first-line therapy is oral corticosteroids, and if a rapid platelet increment is required as in impending delivery or significant bleeding, IVIg should be given.

Treatment to increase the platelet count for delivery is initiated by 36 weeks or earlier if early delivery is planned.

Delivery should be planned in a setting where 24 hours blood bank facilities and ICU care are available.

The obstetric team should liaise with the haematologist, the transfusion physician and the anaesthetist when planning delivery.

Platelets count of at least 50×10^9 /L should be obtained for safe delivery.

If platelet count of less than 50×10^9/L, platelet concentrate should be available on-site for transfusion if necessary.

Caesarean delivery is reserved for obstetric indications only.

At a platelet count $\geq 80\times10^9$/L, regional anaesthesia can be performed in the absence of other hemostatic abnormalities.

IgG antibodies in ITP are known to cross the placenta, causing thrombocytopenia in the fetus and neonate. The occurrence of intracranial haemorrhage (ICH) is a major neonatal concern. Measures should be taken to avoid traumatic delivery to the baby and the mother

during delivery. Scalp electrodes, fetal blood sampling, vacuum and difficult forceps delivery should be avoided. If instrumental delivery is indicated, forceps is the choice.

Prophylactic measures should be taken to prevent Postpartum Haemorrhage (PPH), which includes active management of the third stage of labour, oxytocin infusion and intravenous tranexamic acid.

Pregnant women who are on long term steroids should have regular blood sugar monitoring with PPBS and blood pressure monitoring.

Non-Steroidal Anti-Inflammatory drugs (NSAIDs) should be avoided for postpartum or postoperative analgesia in women with thrombocytopenia due to increased hemorrhagic risk.

In ITP, a multidisciplinary approach involving the obstetrician, haematologist, transfusion physician, anaesthetist and neonatologist, is required for optimal care.

Women with no bleeding manifestations and platelet counts above 30×10^9/L do not require any treatment until 36 weeks gestation[9].

If the platelet count is $<30\times10^9$/L or bleeding manifestations are present, first-line therapy is oral corticosteroids 0.25-1mg/kg daily (dose to be adjusted to achieve a safe platelet count) or if a rapid platelet increment is required as in impending delivery or significant bleeding, IVIg 1g/kg[9].

IVIg has a relatively rapid therapeutic response (within 1-3 days). Prednisolone shows a therapeutic response within 2-14 days[11].

Current recommendations aim for a platelet count of $\geq 50\times10^9$/L prior to labour and delivery as the risk of cesarean delivery is present with everylabour[9].

For spinal anaesthesia, the British Committee for Haematology and Anaesthetic Guideline standards recommends a threshold of $>80\times10$[12,13]. An anaesthetic consultation in the third trimester to discuss options for delivery is required.

While platelet transfusion alone is generally not effective in ITP, if an adequate platelet count has not been achieved and delivery is emergent, or if there is bleeding, platelet transfusion in conjunction with IVIg can be considered[9].

After delivery, close monitoring of the neonate is required as 21% to 28% will develop thrombocytopenia presumably from passive transfer of maternal auto-antibodies (IgG) against platelet antigens[13]. Less than 1% of neonates develop intracranial hemorrhage[14]. Risk for thrombocytopenia is increased if siblings had thrombocytopenia at delivery. Maternal platelet count during pregnancy does not impact the risk of thrombocytopenia in the neonate[15]. The mode of delivery is determined by the obstetric indications, with avoidance of procedures associated with an increased haemorrhagic risk to the fetus, such as fetal scalp electrode/fetal blood sampling and operative vaginal delivery[14]. A cord blood sample should be taken to check neonatal platelet count. Intramuscular injection of vitamin K should not be given if the platelet count is not available, but intravenous or subcutaneous vitamin K can be administered.

4.5 Management of thrombocytopaenia due to Pre-eclampsia/HELLP/AFLP

Urgent delivery should be arranged as it is the mainstay of treatment.

Maternal corticosteroids should be administered considering fetal maturity to reduce fetal respiratory morbidity.

If DIC present, supportive care with FFP, platelet and cryoprecipitate should be administered with advice from the haematologist.

PET affects 4% of all first pregnancies[16]. Thrombocytopenia is the commonest abnormality, occurring in up to 50% of women with pre-eclampsia. HELLP syndrome is a serious complication specific to pregnancy characterized by haemolysis, elevated liver enzymes, and low platelets. It occurs in about 0.5-0.9% of pregnancies and 10-20% of cases with severe pre-eclampsia[17]. As delivery is the definitive mode of treatment for maternal concerns, steroid should be administered for fetal lung maturity. Supportive care with the correction of clotting derangement following delivery should be arranged. Careful observation is needed to detect DIC as a complication in 20% of women with HELLP syndrome[18]. AFLP treatment consists of supportive management and resuscitation of the mother and prompt delivery of the fetus, irrespective of the gestational age.

4.6 Management of thrombotic thrombocytopaenic purpura and atypical haemolytic uraemic syndrome

Despite the diagnostic challenge, plasma exchange (plasmapheresis) needs to be commenced as soon as TTP/aHUS is suspected. Management requires a multidisciplinary approach with the transfusion physician, the obstetrician and the haematologist. Plasma transfusions should be given if there is any delay in plasmapheresis.

In TTP, plasmapheresis should continue daily until the platelet count is maintained in the normal range ($>150\times10^9$/L)for a minimum of 2 days.

Platelet transfusions are contraindicated as they are known to precipitate or exacerbate thrombosis.

Plasmapheresis is the first-line therapy in TTP and aHUS. Plasmapheresis removes substances promoting platelet-aggregation and is successful with TTP but is less successful with HUS. Plasma infusion should be considered if there is any delay in plasmapheresis.

Clinical governance

According to the national recommendation, all pregnant women should have a FBC at booking and repeated at 26 to 28 weeks of gestation. Haemoglobin and platelet count should be recorded in maternity notes.

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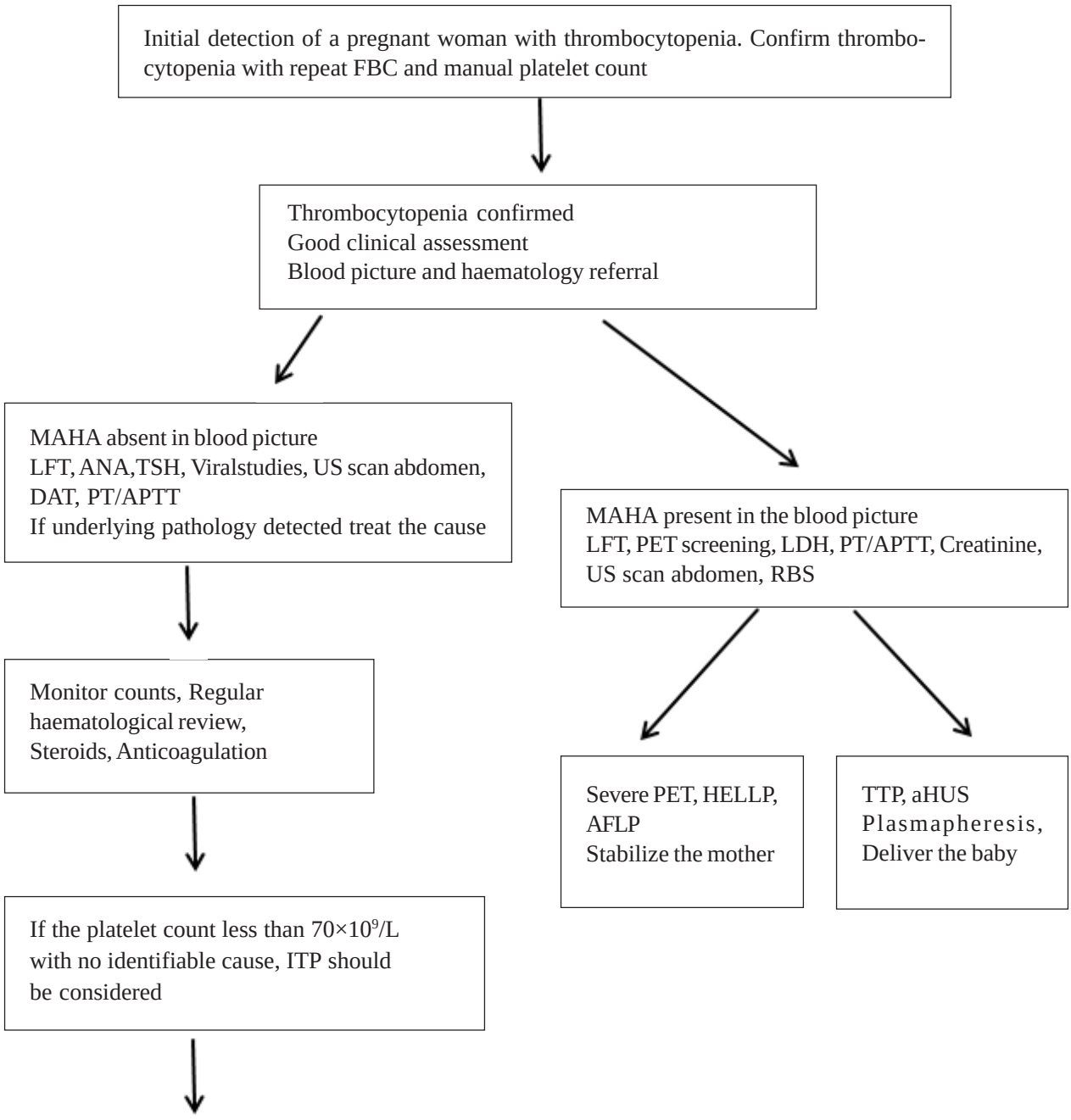
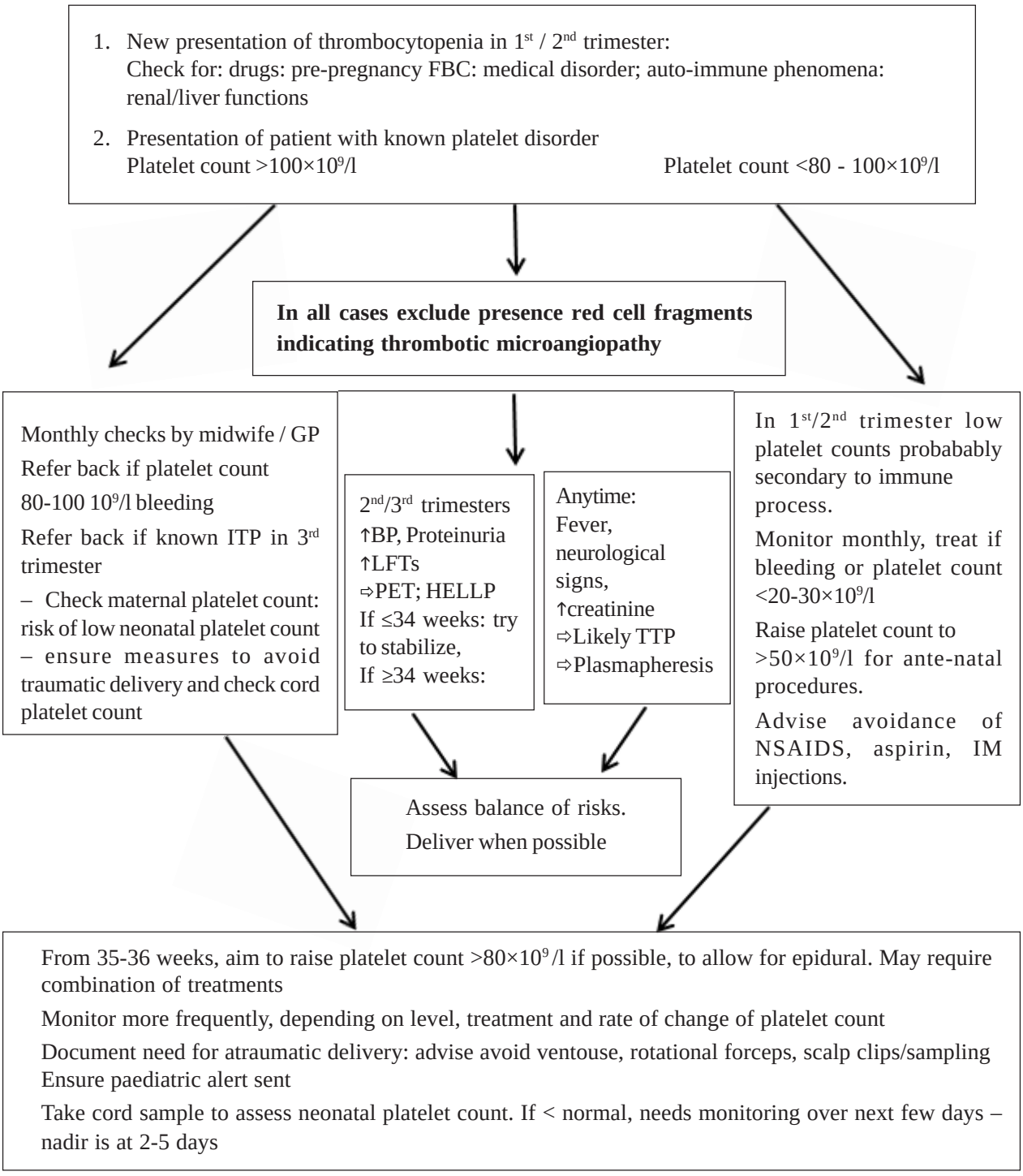
Appendix

Etiological workup

Diagnosis	Proportion	Pathophysiology
Gestational Thrombocytopenia	About 75%	Physiological dilution, accelerated destruction
Immune Thrombocytopenic Purpura (ITP)	About 3%	Immune destruction, suppressed production
Thrombotic Thrombocytopenic Purpura (TTP)		Peripheral consumption, microthrombi
Atypical Haemolytic Uraemic Syndrome (aHUS)		Peripheral consumption, microthrombi
Pre-eclampsia, Eclampsia, Haemolysis, Elevated liver enzymes and low platelet count syndrome (HELLP)	About 15-20%	Peripheral consumption, microthrombi
Hereditary thrombocytopenia		Bone marrow underproduction
Pseudo thrombocytopenia		Laboratory artefact
Viral infections: HIV, Epstein-Barr virus		Secondary autoimmune thrombocytopenia, Marrow suppression
Medications: heparin-induced		Immunological reaction
Leukaemia/Lymphoma		Failure of platelet production, bone marrow infiltration
Severe Vitamin B12 or Folate Deficiency		Failure of platelet production
Splenomegaly		Splenic sequestration



- Monitor counts
- Avoid NSAIDs /Aspirin
- Treatment to elevate count if bleeding or platelet less than 30×10^9/L
- Take haematology advice regarding IM injections
- Elevate platelet count to 50×10^9/L for antenatal procedures
- At 35-36wk, treat to keep platelet count above 80×10^9/L to allow epidural anaesthesia and delivery
- Document the need to avoid traumatic delivery – avoid ventouse, forceps, scalp sampling, scalp electrodes
- Inform neonatologist – paediatric alert to be sent
- Cord blood for neonatal platele count, if less than normal-monitor for thrombocytopenia, nadir 3-5 days



Safe levels of platelets for interventions

Intervention	Platelet count
Antenatal, no invasive procedures planned	>20
Vaginal delivery	>50
Operative or instrumental delivery	>50
Epidural anaesthesia	>80