Literature review

Navigating myeloproliferative neoplasms during pregnancy: from preconception to postpartum

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Introduction

The classical Philadelphia chromosome-negative myeloproliferative neoplasm (MPN) is a heterogeneous group of clonal hematopoietic stem cell disorders that include polycythemia vera (PV), essential thrombocythemia (ET), and primary myelofibrosis (PMF).[1] These disorders are characterized by one of the shared driver mutations, *JAK2*, calreticulin (*CALR*), and *MPL*, which result in consecutive activation of the JAK-STAT signaling pathway, eventually leading to abnormal hematopoietic cell proliferation and excessive cytokine production.[2] Although MPNs have traditionally been considered diseases of older adults–given a median age at diagnosis in the 60s–their occurrence in adolescents and young adults is increasingly recognized, with 10-20% of cases diagnosed before the age of 40.[3] Among this younger population, ET is the most common subtype and displays a female predominance (65-70%),[4,5] highlighting the clinical importance of reproductive health and pregnancy considerations. Additionally, the trend toward advanced maternal age further contributes to the growing number of pregnant patients with MPN.[6,7]

Pregnancy in women with MPN poses unique challenges (Figure 1). Thrombosis and bleeding are common complications that contribute to significant morbidity and mortality in MPNs.[8,9] Consequently, the prevention of thrombotic events is considered one of the primary goals in the management of MPN and therapeutic options are considered based on an individual's thrombotic and bleeding risk profile.[10,11] Pregnancy by itself is a prothrombotic state, driven by estrogen-mediated changes in coagulation and fibrinolysis.[12] The risk of venous thromboembolism (VTE) is increased approximately 4- to 5-fold during pregnancy, and up to 20-fold in the postpartum period, compared to nonpregnant women.[13] When compounded by MPN, this baseline risk escalates further, presenting considerable challenges to fetal and maternal outcomes.[14] In women of childbearing age

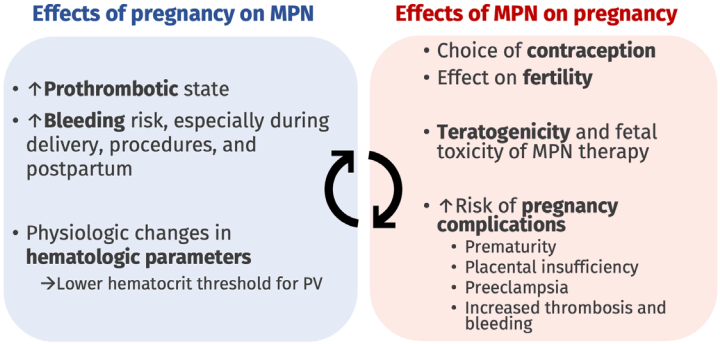

Figure 1 The interaction of myeloproliferative neoplasm (MPN) and pregnancy. Hct, hematocrit; PV, polycythemia vera. Adapted from.[26]

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presenting with unusual thrombotic events–particularly in atypical sites such as the splanchnic or cerebral veins–a complete blood count (CBC) should be reviewed closely for features suggestive of an underlying MPN, including erythrocytosis, leukocytosis, or thrombocytosis. Importantly, initial clinical presentation leading to a diagnosis of a MPN in women of reproductive age may differ substantially from the classic presentations seen in older populations. In some cases, diagnosis may follow a venous or arterial thrombotic event, while in others it may be triggered by recurrent pregnancy loss, abnormal uterine bleeding, or other atypical bleeding symptoms. In many women, the diagnosis may even arise incidentally through routine blood work. These diverse presentations are not well captured in current studies but are clinically important, as each scenario may require a tailored diagnostic and management approach, particularly in the context of pregnancy or fertility planning.

The peripartum period carries the highest risk of bleeding, particularly due to procedure-related factors. Moreover, concerns regarding teratogenicity and fetal toxicity from MPN-directed therapies add complexity to pregnancy planning and management. Similarly, pregnancy itself can influence the course of MPNs. The perinatal period often involves invasive procedures, which heightens the potential for bleeding complications. Additionally, the physiological changes in blood counts during pregnancy can affect hematologic management, such as adjusting the hematocrit threshold for optimal control in PV. These challenges underscore the need for multidisciplinary care to optimize outcomes for both mother and fetus.

This review aims to summarize current understanding of MPNs in pregnancy, with an emphasis on risk stratification, management approaches, and the importance of individualized, multidisciplinary care to optimize maternal and fetal outcomes.

MPNs and pregnancy: How common and what are the consequences?

A common concern among women of childbearing age with MPN is the potential impact on fertility and pregnancy outcomes. While current evidence is limited, emerging studies offer valuable insights. The incidence of MPN-related pregnancies is estimated to be 3-12 per 100,000 pregnancies.[6,15] In a population-based study that compared the childbirth rate of 1,141 women with MPN to 4,565 age-matched controls, women with MPN had a 22% lower likelihood of successfully undergoing childbirth. Interestingly, while childbirth rates were significantly reduced in women with PV and PMF, those with ET were comparable to controls. Women with MPN also had fewer children on average (1.82 vs. 2.01) and a higher prevalence of previous stillbirths at diagnosis.[16] Additionally, a systematic review and meta-analysis of 22 observational studies reported an overall live birth rate of 71.3%, with rates of 71.1% for ET and 66.7% in PV.[17] These figures are slightly lower than the approximate 80% live birth rate observed in the general healthy population.[17] It remains unclear whether MPN, particularly PV and PMF, have a direct biological impact on fertility, or if women with MPN chose not to conceive due to the challenges associated with the diagnosis and its treatment. Notably, recurrent miscarriage can sometimes be the first clinical clue leading to the diagnosis of an underlying MPN, given that spontaneous miscarriage rate in MPN range from 25 to 50% in MPN, which is higher than the general population.[18] However, data suggest that with optimized care, typically consisting of low-dose aspirin, anticoagulation, and cytoreductive therapy (interferon) in high-risk patients, successful pregnancy is achievable for most women with MPN.

Pregnancy Outcomes and Risk Stratification in MPN

Women with MPNs are at increased risk for pregnancy complications such as thrombosis, recurrent pregnancy loss, preterm delivery, placental insufficiency,

preeclampsia and post-partum hemorrhage.[14] In a population-based study comparing 342 MPN pregnancies with control pregnancies (matched for age, calendar year, and parity), MPN pregnancies were associated with a significantly lower rate of live birth rate of 71% vs. 80% with a hazard ratio of 0.78 (95%CI: 0.68-0.90), increased preterm birth (14% vs. 4%), low birth weight (< 2,500 g, 10% vs. 4%), and cesarean section (32% vs. 17%).[6] Preeclampsia was identified as one of the most common obstetric complications in patients with MPN, with a pooled incidence of 3.1% reported in a systematic review and meta-analysis.[17]

Thrombotic complications were consistently noted to be more frequent in MPN pregnancies compared to the general population. A meta-analysis of 21 retrospective studies, encompassing 756 pregnancies in women with ET, reported VTE rates of 1.3% during the antepartum period and 1.8% during the postpartum period.[19] Notably, the risk was higher in pregnancies where low-molecular-weight heparin (LMWH) was not administered, with VTE rates increasing to 2.5% antepartum and 4.4% postpartum,[19] supporting the use of LMWH prophylaxis in ET during the postpartum period. A more recent meta-analysis that included all MPN subtypes reported a pooled VTE incidence of 1.5%.[17] Additionally, one study found a thrombosis rate of 1% in MPN pregnancies compared to 0% in controls.[6] A series of 129 PV pregnancy reported a higher thrombosis rate of 3.1%.[20] These findings underscore the elevated thrombotic risk in MPN pregnancies, particularly in PV, and support the consideration of prophylactic anticoagulation, especially in the postpartum period.

Bleeding complications are another significant concern in MPN pregnancies. A trend toward increased bleeding risk was observed in one study, with rates of 14% in MPN pregnancies compared to 9% in controls.[6] In PV pregnancies, bleeding was the most frequently reported complication, occurring in 15% of cases.[15] A meta-analysis reported pooled incidences of postpartum hemorrhage at 1.5% and other bleeding events at 1.1%.[17] The variability in thrombosis and bleeding rates across studies may reflect differing criteria for defining thrombosis and bleeding endpoints, emphasizing the need for standardized outcome measures in this patient population.

Ideally, patients with predictive factors for pregnancy-related complications should be stratified and managed accordingly. However, the identification of reliable predictive factors remains an area requiring further investigation. Current thrombotic risk stratification models for MPN, such as components of the IPSET score –which includes age > 60, history of thrombosis, and presence of *JAK2* mutation– are limited in their ability to predict pregnancy-related complications. While there was a trend toward increased fetal loss in the high-risk group (44%) compared to the very low/low-risk group (25%), this difference did not reach statistical significance ($p = 0.23$), likely due to the small sample size.[21] In a study evaluating 121 pregnancies in women with ET, a history of prior pregnancy loss was significantly associated with subsequent pregnancy complications, whereas factors such as *JAK2 V617F* and *CALR* mutation status, maternal age, and pre-pregnancy blood counts showed no significant association.[22] Additionally, diabetes mellitus was associated with fetal loss and preeclampsia in ET pregnancies.[21] The impact of the *JAK2 V617F* mutation on pregnancy outcomes has been inconclusive, with earlier studies suggesting a correlation between the mutation and pregnancy complications[23,24] while more recent studies found no significant association.[17,21,22] Overall, the lack of consistent, reliable predictors highlights the need for individualized assessment and further research to guide risk stratification in MPN pregnancies.

Management of MPN during Pregnancy

With the absence of randomized controlled trials specific to this population, the management of MPN during pregnancy is predominantly guided by expert opinions, observational data, and extrapolations from management strategies used in other high-risk pregnancies.[25,26] We propose a step-wise management approach for MPN-related pregnancy as follows (Figure 2):

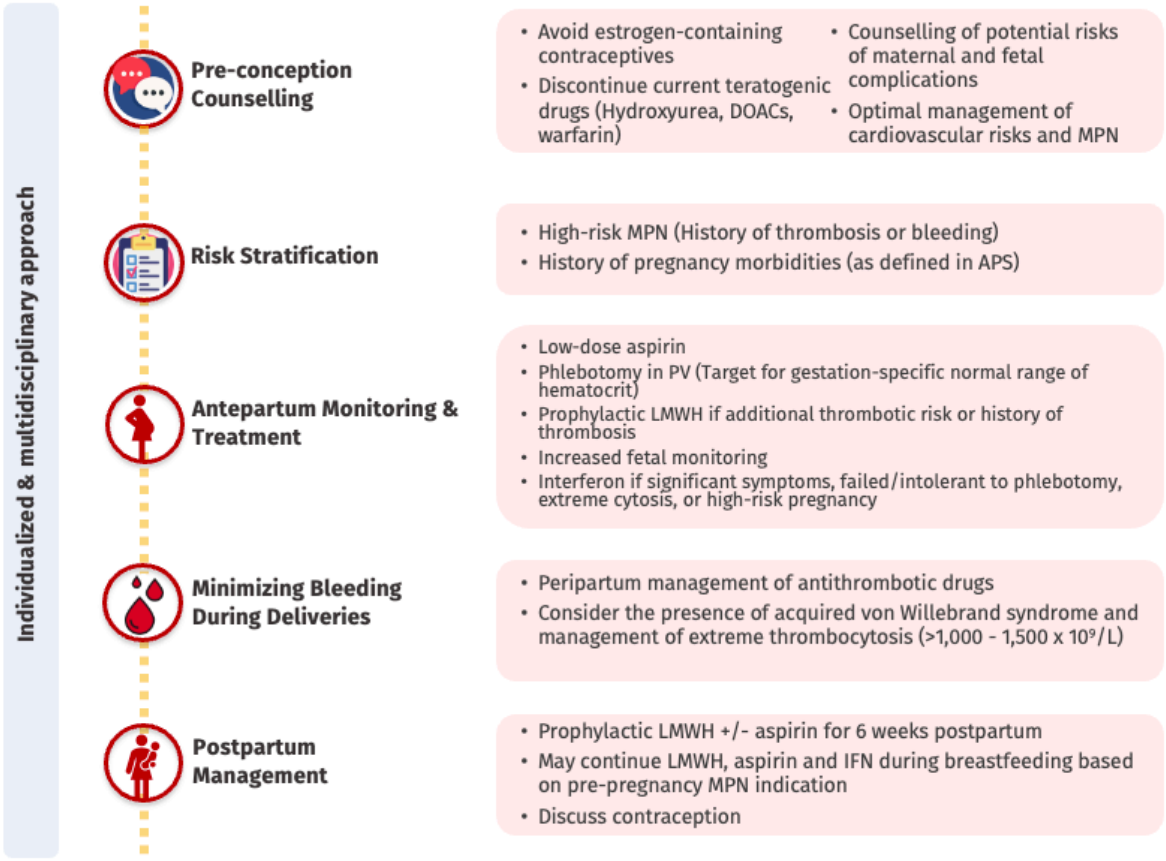

Figure 2 Framework for the management of myeloproliferative neoplasm in pregnancy. MF, myelofibrosis; DOACs, direct oral anticoagulants; MPN, myeloproliferative neoplasm; APS, antiphospholipid syndrome; PV, polycythemia vera; LMWH, low-molecular-weight heparin; IFN, interferon. Adapted from.[26]

Preconception Counseling

Discussions about pregnancy should be an integral part of the routine follow-up for women with MPN who are of reproductive age. For a small subset of patients, such as those with high-risk overt myelofibrosis characterized by severe cytopenia and significant symptoms requiring intensive treatment, pregnancy may need to be discouraged or deferred. In these cases, appropriate contraception methods should be recommended to ensure effective family planning and discussion about further therapy such as allogeneic stem cell transplantation considered.

For patients desiring pregnancy, comprehensive counseling is essential to address the potential risks of maternal and fetal complications. This includes discussing the possibility of thrombotic and bleeding events, as well as other pregnancy-related complications associated with MPN. Achieving optimal control of MPN and other comorbidities for maternal and fetal complications such as diabetes or essential hypertension before conception is crucial. For patients already on cytoreductive therapies, teratogenic agents should be replaced with pregnancy-safe alternatives. Due to its potential teratogenic effects, hydroxyurea should be discontinued at least 3 months before conception and the complete blood counts monitored.[27] If subsequent cytoreductive therapy is required, dose-adjusted interferon can safely be used in pregnancy. Anagrelide and *JAK2* inhibitors such as ruxolitinib are also not recommended or approved for use in pregnancy. The indication for anticoagulation with warfarin or DOACs should be reviewed and if continued switched to LMWH either at preventive or treatment dose to minimize fetal risk. An early interdisciplinary approach is critical, involving close collaboration between experienced obstetricians, hematologists, and, when necessary, other specialists.

Table 1 Features of high-risk MPN pregnancy.

High-risk MPN	● History of thrombosis ● History of bleeding
History of pregnancy morbidity	● Recurrent unexplained early pregnancy loss (gestation age < 10 weeks) ● Fetal loss (early: 10-16 weeks; late: 16-34 weeks) ● Preeclampsia ● Placental insufficiency (intra-uterine growth restriction, still-birth, placental abruption, or oligohydramnios) ● Prematurity (gestation age < 34 weeks)

MPN, myeloproliferative neoplasm

Consultation with a high-risk maternal-fetal medicine specialist for maternal and fetal monitoring is recommended in all pregnant patients with MPN.

Risk Stratification

Risk stratification models specifically tailored for MPN pregnancies are not available. However, certain clinical factors can help identify high-risk patients (Table 1). Women with a previous history of thrombosis or bleeding should be classified as high-risk. Additionally, patients with a prior history of pregnancy complications– such as recurrent miscarriage, stillbirth, preterm birth due to placental insufficiency, or severe preeclampsia, similar to the criteria used in antiphospholipid syndrome (APS) risk assessment[28] –are also considered high-risk. These patients benefit from closer monitoring and targeted interventions, including the use of interferon therapy, to manage their condition and improve pregnancy outcomes. Currently, there is no evidence to support differential pregnancy management based on a patient's MPN driver mutation status. However, for those patients with unusual bleeding symptoms and persistent thrombocytosis, testing for acquired von Willebrand disease or abnormal platelet function –beyond aspirin-related effects– may be considered to help manage the paradox of bleeding and thrombosis in pregnant patients with MPNs.

Antepartum Monitoring and Treatment

In all MPN-pregnancies, low-dose aspirin (81-100 mg) should be recommended throughout pregnancy, as it has been shown to improve outcomes with minimal bleeding risk. Studies have demonstrated that aspirin use in MPN pregnancies was associated with a significantly higher live birth rate (unadjusted odds ratio, OR 8.6; 95%CI: 4.0-18.1),[17] a lower rate of unintentional fetal loss (45% vs. 14%),[21] a reduced risks of pregnancy complications OR 0.29; 90%CI: 0.12-0.66,[22] and a lower risk of thrombosis (0% vs. 3%).[21] The aspirin dose varied according to individual studies, with most ranged from 50 to 160 mg. A recent retrospective study reported that aspirin was used safely in 7 of 11 patients (64%) with acquired von Willebrand syndrome (aVWS), with no cases of maternal hemorrhage.[21] Despite these reassuring findings, the use of aspirin in this setting remains controversial–partly due to the variability in how aVWS is diagnosed in myeloproliferative disorders and the limited size of existing studies. Aspirin may still pose a bleeding risk, particularly in patients with extreme thrombocytosis (e.g., > 1,000-1,500 $\times 10^9$/L), where the likelihood of aVWS and platelet dysfunction is higher. Therefore, while low-dose aspirin may be considered on a case-by-case basis, especially earlier in pregnancy, it should be used cautiously and discontinued before delivery to reduce the risk of bleeding.

In patients with PV, therapeutic phlebotomy can be continued or initiated as indicated during pregnancy. Given the physiological decline in hemoglobin levels due to plasma volume expansion,[22] the target hematocrit for phlebotomy should be adjusted based on gestational age, typically ranging from 37% to 41%. Caution must to be taken against iron supplementation in this population, as it can lead to an increase in hematocrit

levels. Fetal monitoring, including placental flow scans, is recommended at gestational weeks 20, 32, and 36, with more frequent intervals if complications are suspected. Additional thrombotic risk factors, such as immobility and hyperemesis, should be monitored and managed appropriately.

Patients with high-risk features (as listed in Table 1.) may benefit from interferon and/or preventive or therapeutic dose LMWH. Treatment with interferon has been associated with increased odds of live births in MPN patients (OR 9.7; 95%CI: 2.3-41.0).[17] Although further prospective clinical trials are needed to confirm these findings, interferon is often considered in MPN patients requiring cytoreductive therapy, those with a history of thrombosis, or those with prior pregnancy complications.[29] Pegylated interferon alpha-2a has been shown to be a safe and effective alternative in pregnant women with ET.[25,30] In patients with prior history of thrombosis or a pre-existing indication for anticoagulation, a preventive dose or therapeutic weight-based dose of LMWH is indicated. However, the benefit of LMWH in preventing non-thrombotic pregnancy complications –such as recurrent miscarriage or fetal growth restriction– remains uncertain due to small studies with limited power. A prior meta-analysis found no improvement in live birth rates among MPN patients treated with heparin compared to those managed with aspirin alone or observation.[17] Similarly, a recent randomized control trial found no benefit of LMWH in improving live birth rates in patients with inherited thrombophilia and recurrent pregnancy loss.[30] Antepartum bleeding risk associated with LMWH use in MPN is approximately 4%.[19] The decision to use LMWH in patients without a history of thrombosis should be individualized, with a careful risk–benefit discussion. Identifying patient characteristics that may predict a benefit from LMWH remains an important area for future research.

In many countries across Asia, including Thailand, there are important contextual limitations that affect the use of prophylactic LMWH. These include limited healthcare resources, a lower baseline risk of thrombosis, inadequate patient education, and the inability or reluctance of some patients to self-administer subcutaneous injections. Such factors may limit the applicability of Western treatment practices and raise concerns about potential overtreatment when LMWH is used routinely without individualized risk assessment. Moreover, current evidence supporting LMWH use in MPN pregnancies is largely based on small observational studies, with limited representation from Asian populations. Further research is needed to develop context-appropriate guidance for LMWH use in this setting.

Prevention of Peripartum Bleeding

The peripartum period carries the highest risk of bleeding, particularly due to procedure-related factors. Women with MPN are at an increased risk of delivering via cesarean section;[6] however, the mode of delivery should primarily be determined based on obstetric indications and complications rather than the presence of MPN alone.

For women receiving therapeutic LMWH, it is recommended to discontinue LMWH at least 24 hours prior to delivery or planned neuraxial anesthesia to reduce the risk of bleeding.[31] Similarly, low-dose aspirin should be stopped by the 37th week of gestation or at least 7 days prior to the expected onset of labor or scheduled caesarean section, particularly if neuraxial anesthesia is planned. Spinal anesthesia may be preferred over epidural in high-risk cases, as it involves a single puncture and does not require catheter placement, thereby reducing the risk of hematoma formation.[32] Platelet counts should be closely monitored in the peripartum period, particularly in patients with extreme thrombocytosis (> 1,000-1,500 $\times 10^9$/L), as these patients are at increased risk of aVWS, which can further elevate the bleeding risk. Patients receiving anticoagulation should be educated to withhold their dose if they suspect the onset of labor, experience rupture of membranes, or develop vaginal bleeding.[31]

Postpartum Management

Prophylactic LMWH is recommended during the 6-week postpartum period for women with MPN due to the elevated risk of higher VTE, estimated at 4.4%, which can be effectively mitigated with the use of LMWH.[19] However, the additional benefit of LMWH over aspirin alone during this period is not well established, and whether patients should continue aspirin, LMWH, or a combination of both in the postpartum period remains unclear, highlighting the need for further research. Aspirin and interferon can be safely continued during breastfeeding for patients with high-risk MPN, whereas hydroxyurea and DOACs should continue to be avoided. Additionally, effective contraception should be recommended to prevent unintended pregnancies.

Conclusion and Future Direction

Pregnancy in women with myeloproliferative neoplasms presents unique clinical challenges, including increased risks of miscarriage, preeclampsia, thrombotic and bleeding complications. While ET is the most common MPN in younger women, data to guide management remain limited because of limited prospective data, particularly regarding optimal risk stratification and optimal anticoagulation strategies. Current approaches emphasize individualized care that consider patient preferences, with low-dose aspirin and interferon-alpha as preferred therapies and postpartum anticoagulation playing a key role in reducing thrombotic risk. However, existing risk prediction models developed for the general MPN population are not validated in pregnancy, and the impact of genetic mutations such as JAK2, CALR, or MPL on pregnancy outcomes remains inconclusive.

Future research should focus on prospective, multicenter studies and pregnancy registries to better define maternal and fetal outcomes across MPN subtypes, including rarer entities such as PMF or MPN-unclassifiable. Additionally, the development of pregnancy-specific risk stratification models is needed to guide management. Further evidence on the role, timing, and dosing of antenatal anticoagulation in management will be critical. Finally, attention to optimal management during reproductive therapy–such as ovulation induction and assisted reproduction–is increasingly important as more women with MPN pursue pregnancy. Multidisciplinary collaboration between hematology, maternal-fetal medicine, and obstetrics remains essential to improving outcomes for both mother and child.

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