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Case Report

Pregnancy in a patient with myelofibrosis secondary to ileocecal tuberculosis: a case report

Sreelakshmi K.*, Sajala Vimalraj, Smitha Sreenivas K., Rameshan C. K.

Department of Obstetrics and Gynecology, Government Medical College, Kozhikode, Kerala, India

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***Correspondence:**
Dr. Sreelakshmi K.,
E-mail: sreelak648@gmail.com

ABSTRACT

Myelofibrosis (MF) is a chronic myeloproliferative neoplasm that may occur de novo or secondarily due to infections, autoimmune disorders, or malignancies. Tuberculosis (TB)-induced MF is uncommon but clinically relevant in TB-endemic regions, as it represents a reversible, non-clonal process that can mimic primary MF and lead to unnecessary cytotoxic therapy if not correctly identified. We report the case of a 24-year-old woman who initially presented with pancytopenia, splenomegaly, and constitutional symptoms. Bone marrow examination revealed grade 2 fibrosis, and further evaluation indicated ileocecal TB, leading to a diagnosis of secondary MF due to TB. She was initiated on anti-tubercular therapy (ATT), resulting in significant clinical and hematologic improvement. The patient subsequently conceived spontaneously and later presented at 36 weeks of gestation with mild anemia, severe thrombocytopenia, and a history of allergic reactions to platelet transfusions. When she was admitted at term with critically low platelet counts necessitating transfusion, perioperative management became particularly challenging and required a coordinated multidisciplinary approach involving hematology, transfusion medicine, obstetrics, neonatology, and critical care. Her pregnancy was managed with strict antenatal, intraoperative, and postpartum monitoring. She remained clinically stable and did not require cytotoxic or disease-modifying therapy, and both maternal and neonatal outcomes were favorable. This case highlights the importance of differentiating reactive MF from clonal myeloproliferative neoplasms, especially in TB-endemic settings. TB-induced MF is reversible with appropriate antimicrobial therapy, and successful pregnancy outcomes are achievable with individualized, coordinated obstetric and hematological care. Diagnostic vigilance is essential to avoid overtreatment and to ensure optimal outcomes for women of reproductive age presenting with marrow fibrosis secondary to TB.

Keywords: Myelofibrosis, Pregnancy, Ileocecal tuberculosis, Myeloproliferative neoplasm, High-risk pregnancy

INTRODUCTION

Myelofibrosis (MF) is a chronic myeloproliferative neoplasm characterized by clonal proliferation of hematopoietic stem cells, leading to progressive bone marrow fibrosis, extramedullary hematopoiesis and splenomegaly.[1-3] It may present as primary MF or evolve secondarily from other myeloproliferative neoplasms such as polycythemia vera or essential thrombocythemia, driven largely by mutations in JAK2, CALR or MPL that activate the JAK-STAT signalling pathway.[1,4] In addition to clonal causes, MF may also occur as a reactive process due to infections, autoimmune diseases or malignancies. TB is an uncommon but important cause of secondary MF in TB-endemic regions, where chronic granulomatous inflammation and cytokine-mediated fibroblast activation can result in reversible marrow fibrosis.[5-8] Clinical differentiation between reactive and primary MF is essential, as reactive forms improve with targeted treatment of the underlying condition and do not require cytotoxic agents or JAK inhibitors.[4] Pregnancy in women with MF is rare, and available literature is limited,

particularly concerning MF secondary to infectious etiologies such as TB. Physiological hematological changes of pregnancy may complicate management, and risks such as anemia, thrombocytopenia, placental insufficiency and postpartum hemorrhage require careful monitoring.[9]

The objective of this case report is to describe the successful pregnancy outcome in a woman with MF secondary to ileocecal TB, who was in remission and presented at term with severe thrombocytopenia and an inability to receive platelet transfusion due to a history of allergic reactions, managed successfully through a critical multidisciplinary approach.

CASE REPORT

A 24-year-old primigravida at 36 weeks of gestation was referred to our tertiary care centre for further management of severe thrombocytopenia in the background of MF secondary to ileocecal TB. Her past medical history dated back two years, when she initially presented to a local hospital at 22 years of age with complaints of abdominal distension and vomiting of two weeks' duration, accompanied by dull aching abdominal pain, anorexia, significant unintentional weight loss, dyspnea on exertion, recurrent minor infections and palpitations. There was no significant family history of hematologic or autoimmune disorders. Clinical examination at that time revealed marked pallor, palpable cervical lymphadenopathy and moderate splenomegaly.

Initial laboratory investigations demonstrated pancytopenia with hemoglobin 6.6 g/dl, total leukocyte count 3450/µL and platelet count 63,000/µl. Peripheral smear showed microcytic hypochromic anemia with anisopoikilocytosis, leukopenia and thrombocytopenia. Iron studies revealed severe iron deficiency with low serum ferritin and reduced transferrin saturation. Given her constitutional symptoms and splenomegaly, further imaging was performed.

Contrast-enhanced CT of the abdomen showed hepatomegaly, splenomegaly and irregular enhancing thickening of the proximal ascending colon and cecum along with multiple enlarged lymph nodes, findings strongly suggestive of ileocecal TB.

Bone marrow examination revealed a hypercellular marrow with striking megakaryocytic proliferation and Grade 3 reticulin fibrosis, raising the differential diagnosis of primary versus secondary MF.

Bone marrow biopsy showing features of grade 3 reticulin fibrosis.

Mutation analysis for JAK2 V617F, CALR and MPL was negative. Colonoscopy showed ulceration and nodularity of the cecum and ileocecal valve, compatible with the ulcerohypertrophic variant of ileocecal TB.

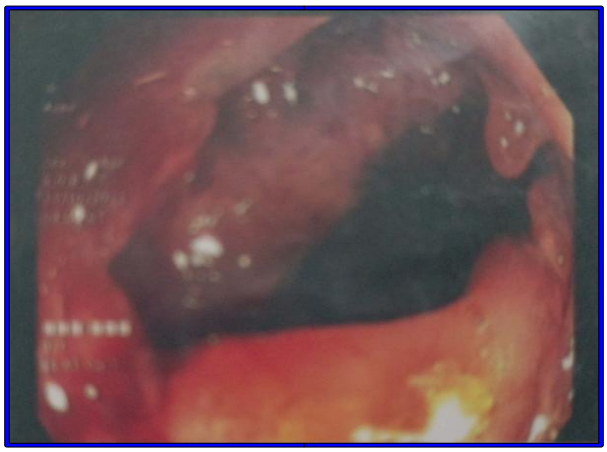

Figure 2: Initial colonoscopy showing ulcerated ileocecal mucosa.

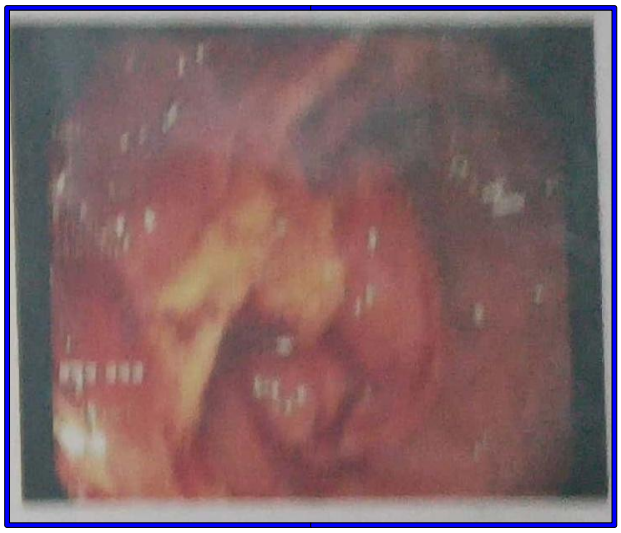

Figure 3: Initial colonoscopy showing nodularity and inflammation.

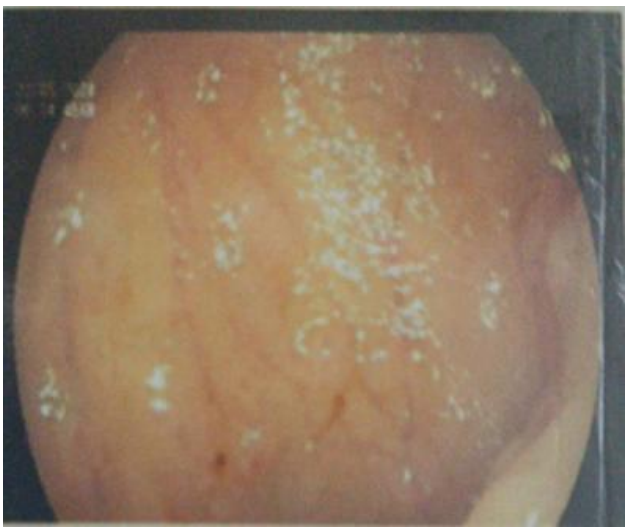

Figure 4: Post treatment colonoscopy showing healed mucosa.

Taken together, these findings favoured a diagnosis of secondary MF due to TB. The patient was commenced on standard ATT. During this period, she received packed red cell and platelet transfusions for symptomatic anemia and thrombocytopenia, during which she developed allergic transfusion reactions to platelet trasfusion manifesting as erythema and respiratory discomfort and hence further transfusions were withheld. Nevertheless, she completed the full course of ATT, and interval colonoscopy demonstrated good mucosal healing with only mild residual nodularity.

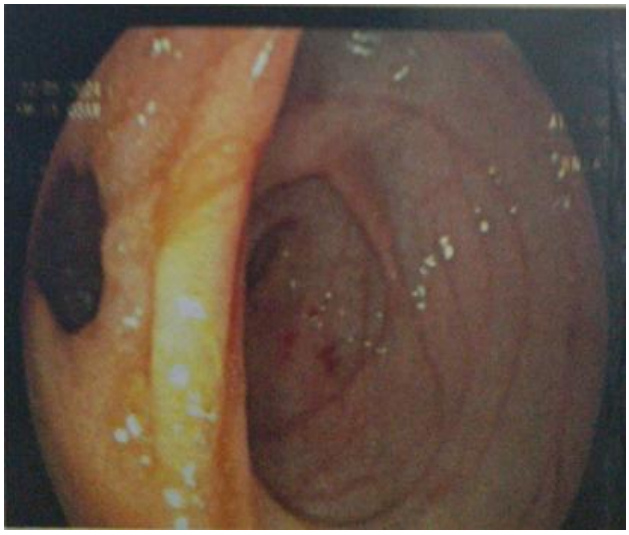

Figure 5: Post-treatment colonoscopy showing resolution of lesions.

Her hematological parameters improved partially, with hemoglobin rising to 9.9 g/dl and platelets to 48,000/µl, consistent with prior reports that TB-associated MF is a reversible, non-clonal process responding well to ATT. She was subsequently lost to follow-up after clinical stabilization.

The patient conceived spontaneously the following year. Early pregnancy proceeded uneventfully, with regular antenatal visits and normal fetal growth parameters. She had persistent moderate anemia and thrombocytopenia, but no bleeding manifestations. As pregnancy advanced, however, her platelet counts progressively declined. At 36 weeks, she was started on prednisolone at a peripheral centre and referred to us with platelet count 22,000/µl and hemoglobin 10.8 g/dl and inability to give platelet transfusions in view of prior history of allergic reaction to transfusion.

On admission, the patient was clinically stable with mild pallor, no lymphadenopathy and persistent splenomegaly. Cardiotocography showed a reactive fetal heart pattern. Ultrasound revealed a normally growing fetus with an estimated fetal weight of 2403 g, adequate liquor and normal Doppler indices, with no evidence of placental insufficiency. In view of severe thrombocytopenia, hematology consultation was sought, and pulse methylprednisolone therapy was administered for three days, followed by oral prednisolone. Intravenous immunoglobulin (IVIG) was planned 24 hours prior to delivery to improve her platelet count. Infectious disease assessment revealed no evidence of TB reactivation.

A multidisciplinary team-comprising obstetricians, anesthesiologists, hematologists, transfusion medicine specialists and neonatologists-concluded that delivery by caesarean section would be the safest obstetric choice due to her worsening platelet trend and potential risk of intrapartum hemorrhage. A single-donor platelet (SDP) transfusion was planned under steroid and antihistamine cover due to her prior transfusion reactions. Washed, leukoreduced blood components were arranged in advance.

However, upon initiating SDP transfusion, the patient developed acute respiratory difficulty, facial flushing and chest discomfort, prompting immediate discontinuation. Maternal vitals showed tachycardia and mild hypotension, and fetal heart rate decelerations to 90 bpm were noted. Given the fetal distress and inability to proceed with platelet transfusion, an emergency lower-segment caesarean section under general anesthesia was performed. A live male neonate weighing 2071 g was delivered at 36+ weeks.

Intraoperatively, the uterus was atonic with brisk bleeding. Multiple uterotonics were administered, followed by bilateral uterine artery ligation, modified B-Lynch suturing and bilateral utero-ovarian vessel ligation to achieve hemostasis.[10] An intraperitoneal drain was placed. Postoperatively, the patient remained hemodynamically stable. Further evaluation to determine the cause of recurrent transfusion reactions revealed markedly decreased IgA and low IgE levels, confirming an underlying immunoglobulin deficiency predisposing her to allergic transfusion reactions.[17]

She received prophylactic antibiotics, tranexamic acid and meticulous postoperative monitoring. The abdominal drain was removed on postoperative day three once output was minimal. She did not require further transfusion support. Early ambulation and hydration were encouraged. Thromboprophylaxis was withheld due to persistent thrombocytopenia. Both mother and baby were discharged in stable condition on postoperative day ten.

DISCUSSION

This case highlights the unique challenges encountered when managing pregnancy in a woman with MF secondary to TB, a condition rarely described in the literature. The patient initially presented with severe pancytopenia and grade 3 marrow fibrosis that closely mimicked primary MF, but her subsequent improvement following ATT confirmed the reactive, reversible nature of TB-induced marrow fibrosis, consistent with previously reported cases of reversible MF following treatment of

disseminated TB.[7] Sharma and Mohan have similarly noted that chronic granulomatous inflammation in extrapulmonary TB can lead to hematologic abnormalities including marrow fibrosis, which often regresses with appropriate therapy.[6]

Pregnancy after resolution of reactive MF is uncommon, and little data exist regarding maternal and fetal outcomes. Physiological hemodilution, increased plasma volume and relative immunomodulation of pregnancy can further complicate hematologic stability in women with underlying marrow disease. Published literature on myeloproliferative neoplasms in pregnancy indicates increased risks of fetal growth restriction, preterm delivery and postpartum hemorrhage due to disordered hematopoiesis and thrombocytopenia findings that correlate with the concerns encountered in our patient.[11]

The need for cesarean section in the presence of profound thrombocytopenia posed significant perioperative challenges, particularly because of her history of severe allergic reactions to platelet transfusions. Her subsequent diagnosis of IgA and IgE deficiency provides a plausible explanation, as these conditions are known to predispose patients to allergic and anaphylactoid reactions to blood products, necessitating careful use of washed components and premedication in peripartum transfusion planning.[12]

Therapeutic options commonly used in primary MF, such as thalidomide or JAK-inhibitors, are contraindicated in pregnancy because of teratogenic and antiangiogenic effects. Thalidomide embryopathy has been well documented, with limb and organ malformations resulting from exposure during early gestation.[13] Likewise, anti-VEGF therapies can impair placental vascular development and are avoided due to risks of miscarriage, intrauterine growth restriction and preeclampsia-like complications.[14] In contrast, interferon-α has been used safely in pregnant women with myeloproliferative neoplasms when cytoreduction is needed, and several reports describe favorable maternal and fetal outcomes when interferon-α is combined with aspirin and LMWH during pregnancy.[15] In our case, however, the patient did not require cytoreductive therapy because her MF was reactive and non-clonal, and her deterioration was related to thrombocytopenia rather than proliferative disease.

Overall, the successful outcome in this patient underscores the importance of distinguishing reactive from clonal MF, as management differs significantly and inappropriate cytotoxic therapy can be avoided. Once TB is effectively treated, most patients experience hematologic recovery, and pregnancy can proceed safely with multidisciplinary coordination. This case supports existing evidence that TB-associated MF is reversible and that favorable perinatal outcomes are achievable with vigilant monitoring, individualized obstetric planning and careful transfusion management.

CONCLUSION

MF secondary to TB is an uncommon but important clinical entity in TB-endemic regions, where chronic granulomatous inflammation can lead to reversible marrow fibrosis that may closely resemble primary MF. Recognition of its reactive, non-clonal nature is essential to avoid unnecessary cytotoxic therapy and to ensure timely initiation of anti-tubercular treatment. Pregnancy in women with secondary MF presents additional challenges due to persistent cytopenias, increased bleeding risk and the need to avoid teratogenic drugs, yet favorable outcomes are achievable with multidisciplinary care. In the present case, effective completion of ATT prior to conception, close antenatal monitoring, preparedness for transfusion reactions related to underlying IgE and IgA deficiency and individualized obstetric decision-making contributed to a successful maternal and neonatal outcome. This report underscores the importance of diagnostic vigilance, appropriate TB management, and coordinated perinatal care for women with MF secondary to TB.

Recommendations

Diagnosis of MF in TB-endemic regions should always include careful evaluation for infectious etiologies, particularly TB, in any patient presenting with pancytopenia, splenomegaly, or bone marrow fibrosis. Early consideration of TB as a potential cause can prevent misclassification as primary MF and thereby avoid unnecessary cytotoxic or targeted therapies. Appropriate investigations should be performed when clinically indicated. Once TB is confirmed, prompt initiation and completion of the full course of ATT is essential, as resolution of the infectious trigger often leads to reversal of reactive marrow fibrosis. Women of reproductive age should ideally complete ATT before planning pregnancy to ensure hematologic stability. Pregnancy in such patients requires coordinated multidisciplinary care involving high-risk obstetricians, hematologists, transfusion medicine specialists and infectious disease experts. Close monitoring of anemia, thrombocytopenia and fetal growth is crucial throughout pregnancy, with vigilance for complications such as fetal growth restriction, preterm labor and postpartum hemorrhage. Obstetric decisions regarding mode of delivery must account for platelet levels, bleeding risk and anesthetic considerations, with appropriate preparation for the potential need for platelet support. Intrapartum management should include strict monitoring, readiness to address hemorrhage and proactive surgical planning when thrombocytopenia is severe. Patients with IgA or IgE deficiency require special attention due to the increased risk of transfusion reactions, necessitating pre-arranged access to washed or specially prepared blood products. Postpartum care should continue to include hematologic surveillance, as delayed bleeding manifestations and late marrow recovery may occur. Invasive marrow reassessment may be deferred until after delivery unless clinically urgent.

Counseling on contraception is crucial in this population, as recurrent cytopenias and transfusion-related risks make closely spaced pregnancies hazardous; long-acting reversible contraceptive (LARC) methods may be particularly suitable to ensure safe interpregnancy intervals and minimize maternal risk.With careful individualized management, women with secondary MF due to TB can achieve safe maternal and fetal outcomes.

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