

Hemoglobin E Disease Identified During Evaluation of Persistent Microcytosis and Thrombocytosis: A Case Report

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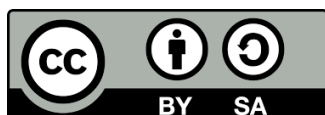
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Abstract

Hemoglobin E (HbE) disease may present with marked microcytosis and mild anemia, mimicking iron deficiency and complicating the diagnostic process when thrombocytosis coexists. This case report aimed to describe the diagnostic evaluation and clinical implications of suspected homozygous HbE disease in a woman of reproductive age. A descriptive review of the medical records of a 28-year-old woman referred following pregnancy loss was conducted. Data included serial complete blood counts, iron studies, peripheral blood smear findings, hemoglobin electrophoresis, JAK2 mutation testing, and abdominal ultrasonography. Hemoglobin levels increased from 9.9 to 11.8 g/dL, whereas mean corpuscular volume remained low at 59.4–63.2 fL, and platelet counts ranged from 530 to 709 × 10³/μL. Hemoglobin analysis revealed an HbE fraction of 93.4% with no detectable HbA, a pattern consistent with homozygous HbE disease. Iron studies did not indicate severe iron deficiency. JAK2 mutation testing yielded negative results, and abdominal ultrasonography revealed no hepatosplenomegaly. Thrombocytosis was considered potentially reactive; however, its underlying cause remained undetermined, and a clonal hematologic disorder could not be excluded. Molecular confirmation of the HbE genotype was unavailable. This case highlights the diagnostic value of hemoglobin analysis in patients with persistent microcytosis and emphasizes the importance of partner screening and genetic counseling. The available clinical and laboratory findings do not establish a direct causal relationship between HbE disease and the preceding pregnancy loss.



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INTRODUCTION

Hemoglobin E (HbE) disease is an inherited hemoglobin disorder caused by a structural variant of the β -globin chain that also exhibits mild β -thalassemic characteristics due to reduced HbE synthesis. It is particularly prevalent in Asian populations, especially in Southeast Asia, where HbE and thalassemia variants occur at relatively high frequencies and may interact with other hemoglobin abnormalities, resulting in a broad spectrum of clinical manifestations (Cao & Galanello, 2010; Fucharoen & Weatherall, 2012; Taher et al., 2018). In its homozygous form, HbE disease is generally clinically mild but may manifest as persistent microcytosis, hypochromia, mild anemia, and characteristic erythrocyte morphological abnormalities, including target cells (Fucharoen & Weatherall, 2012; Munkongdee et al., 2020).

The diagnostic challenge of HbE disease arises from its clinical and hematologic overlap with more common causes of microcytic anemia, particularly iron deficiency anemia. Patients

may exhibit a disproportionately low mean corpuscular volume despite having only mild anemia, a pattern that may be misinterpreted when hemoglobinopathy testing is not performed (Kiran et al., 2016; Munkongdee et al., 2020). Hemoglobin electrophoresis or capillary electrophoresis plays a central role in establishing the diagnosis. In the present case, hemoglobin electrophoresis was performed on March 27, 2026. Homozygous HbE disease typically demonstrates a predominance of HbE, an absence of HbA, and a relatively low HbF fraction (Kiran et al., 2016; Munkongdee et al., 2020; Prasing & Pornprasert, 2014). This distinction is clinically important because unnecessary or prolonged iron supplementation may occur when persistent microcytosis is attributed solely to iron deficiency. Although the available iron parameters did not indicate severe iron depletion, concomitant iron deficiency could not be completely excluded, particularly given the patient's history of iron supplementation and the potential influence of inflammation on serum ferritin concentrations (Dolai et al., 2026; Foord et al., 2026; Godfrey et al., 2026).

Thrombocytosis may further complicate the diagnostic evaluation of microcytic anemia. In clinical practice, elevated platelet counts are more frequently reactive than clonal and may be associated with iron deficiency, inflammation, tissue injury, infection, hemolysis, or other secondary conditions (Bleeker & Hogan, 2011; Rokkam et al., 2026). However, persistent thrombocytosis warrants consideration of essential thrombocythemia and other myeloproliferative neoplasms, particularly when platelet counts remain elevated on serial examinations (Almanaseer et al., 2024; Bleeker & Hogan, 2011). Therefore, a comprehensive diagnostic approach integrating serial complete blood counts, peripheral blood smear findings, iron studies, assessment of inflammatory conditions, abdominal imaging, and molecular testing, including JAK2 mutation analysis, is essential to minimize the risk of premature diagnostic conclusions. A negative JAK2 mutation test alone does not exclude an underlying myeloproliferative neoplasm.

Recognition of HbE disease is particularly important in women of reproductive age. Hemoglobinopathy screening and genetic counseling are recommended before or during pregnancy because identifying individuals who carry or are affected by hemoglobin variants facilitates timely partner screening, reproductive risk assessment, and counseling regarding potential fetal hemoglobin disorders (Gavillet et al., 2026). In this context, the identification of HbE disease during the diagnostic evaluation following early pregnancy loss has broader clinical implications beyond anemia management, particularly for future pregnancy planning and family screening. This case describes a woman of reproductive age with newly identified HbE disease who presented with persistent hypochromic microcytic anemia and thrombocytosis following a previous anembryonic pregnancy (blighted ovum). It emphasizes the diagnostic importance of hemoglobin electrophoresis and partner screening in patients with unexplained microcytosis while recognizing that the available evidence does not establish a causal association between HbE disease and the preceding pregnancy loss.

METHOD

This case report employed a descriptive approach to examine the diagnostic process and clinical characteristics of a patient with hemoglobin E (HbE) disease identified during the evaluation of persistent microcytic anemia and thrombocytosis. The patient was a 28-year-old

woman referred to Wangaya Hospital for anemia and suspected thrombocytosis following an anembryonic pregnancy (blighted ovum) during her first pregnancy. Clinical data were retrospectively obtained from medical records, including medical history, physical examination findings, and laboratory investigations.

The diagnostic evaluation included serial complete blood counts, iron studies, peripheral blood smear examination, hemoglobin electrophoresis, JAK2 mutation testing, and abdominal ultrasonography. These investigations were reviewed to characterize the microcytic anemia, assess iron status, identify hemoglobin abnormalities, and investigate potential causes of thrombocytosis.

Clinical and laboratory findings were analyzed descriptively to characterize the diagnostic pattern and assess relevant differential diagnoses. Hemoglobin electrophoresis findings provided the primary evidence supporting a pattern consistent with homozygous HbE disease, while additional investigations informed the assessment of alternative causes of anemia and thrombocytosis. Molecular analysis of the β -globin (HBB) gene was not performed; therefore, the suspected homozygous HbE genotype could not be definitively confirmed.

RESULTS AND DISCUSSION

Case Illustration

A 28-year-old woman, KS, was referred to RSUD Wangaya with anemia and suspected thrombocytosis following a previous diagnosis of blighted ovum managed with uterine curettage by an obstetrician. The patient had been in her first pregnancy at 9 weeks of gestation when she developed vaginal bleeding. At the time of referral, active vaginal bleeding was no longer present, although she was informed that there was still uterine wall thickening and suspected inflammation. She had no history of diabetes mellitus, systemic lupus erythematosus, joint pain, malar rash, or hair loss. A family history of thalassemia or other hematologic disorders was not clearly established. She had previously received oral iron supplementation for one month before the serum iron test results were available. Physical examination showed bilateral conjunctival pallor, while cardiac and pulmonary examinations were within normal limits. No hepatosplenomegaly was found.

Serial complete blood count examinations showed persistent mild hypochromic microcytic anemia with thrombocytosis. On November 4, 2025, the hemoglobin level was 9.9 g/dL, with an MCV of 59.4 fL, MCH of 19.9 pg, and platelet count of $530 \times 10^3/\mu\text{L}$. Subsequent examinations showed hemoglobin levels of 10.8 g/dL on November 14, 2025, 11.4 g/dL on January 1, 2026, and 11.8 g/dL on April 10, 2026, with persistently low MCV values ranging from 60.7 to 63.2 fL, MCH values ranging from 19.5 to 20.5 pg, and platelet counts ranging from 569 to $709 \times 10^3/\mu\text{L}$. Iron studies performed on November 14, 2025, showed a serum iron level of 74 $\mu\text{g/dL}$, TIBC of 293 $\mu\text{g/dL}$, and ferritin level of 45 ng/mL. The calculated transferrin saturation was approximately 25.3%. Hemoglobin analysis performed on 27 March 2026 demonstrated a predominant HbE fraction of 93.4%, HbA₂ of 4.6%, a low fraction in the Hb F/variant zone (1.1%), and a fraction in the Hb D zone (0.9%), with HbA reported as 0%. The overall hemoglobin pattern was consistent with homozygous HbE disease.

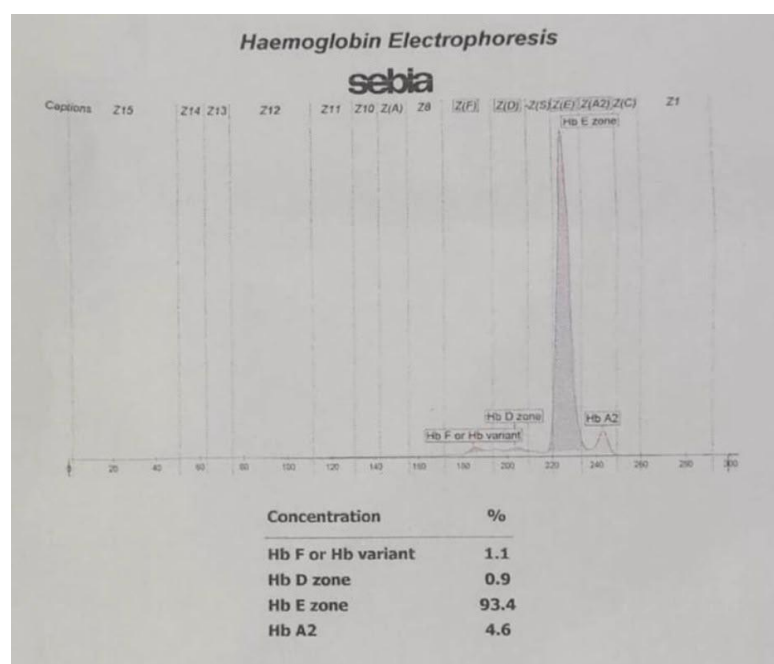


Figure 1. Hemoglobin electrophoresis demonstrating a predominant HbE fraction (93.4%), with HbA₂ of 4.6% and a fraction in HbF/variant of 1.1%, and a fraction in the HbD zone (0.9%). The overall hemoglobin pattern was consistent with homozygous HbE disease.

Peripheral blood smear examination on November 14, 2025, revealed hypochromic anisopoikilocytosis with both microcytic and normocytic erythrocytes, target cells, teardrop cells, ovalocytes, spherocytes, and positive polychromasia without normoblasts. Leukocyte morphology showed a normal overall impression, predominantly segmented neutrophils, with immature granulocytes including myelocytes, metamyelocytes, and band neutrophils, atypical lymphocytes, and no blast cells. Platelet morphology showed an increased platelet count with giant platelets and no platelet clumping. The impression was hypochromic microcytic erythrocyte morphology with thrombocytosis, with differential considerations including functional iron deficiency, essential thrombocytosis, or an inflammatory process. Further evaluation showed that JAK2 mutation was not detected.

Abdominal ultrasonography performed on November 21, 2025, showed no abnormalities in the liver, gallbladder, pancreas, spleen, kidneys, bladder, uterus, or adnexa. No para-aortic lymph node enlargement or free fluid in the abdominal and pelvic cavities was observed. Based on the clinical and laboratory findings, the patient was assessed as having HbE disease with mild hypochromic microcytic anemia, observed thrombocytosis suspected to be reactive, and a history of blighted ovum. She was treated with oral folic acid 1 mg once daily, and the husband was advised to undergo hemoglobinopathy screening and genetic counseling.

This case illustrates the diagnostic importance of evaluating persistent microcytic hypochromic anemia beyond iron deficiency, particularly in a reproductive-age woman with recent pregnancy loss. The patient showed only mild anemia, with hemoglobin gradually ranging from 9.9 to 11.8 g/dL, but the degree of microcytosis was marked and persistent, with MCV remaining around 59–63 fL and MCH around 19–20 pg. This pattern is clinically

important because hemoglobinopathy may be overlooked when microcytosis is initially interpreted as nutritional anemia, post-bleeding anemia, or anemia related to a recent obstetric event. In this patient, iron parameters did not clearly indicate severe iron depletion, while hemoglobin electrophoresis analysis demonstrated a predominant HbE fraction of 93.4%, HbA₂ of 4.6%, a low fraction in the HbF/variant zone of 1.1%, and in the Hb D zone of 0.9%, with HbA reported as 0%. The overall pattern was consistent with diagnosis of homozygous HbE disease as the underlying explanation for the persistent microcytic picture.

HbE/ β -thalassemia represents an important differential diagnosis because it can present with prominent microcytosis and a high proportion of HbE, but its clinical and hematological manifestations are considerably variable, ranging from mild anemia to more severe thalassemic phenotypes. The HbF and HbA levels may also vary according to the underlying β -thalassemia allele.^{16,17} In this patient, the reported absence of HbA, predominant HbE fraction, low fraction detected in the Hb F/variant zone, marked microcytosis, and relatively mild anemia were consistent with a homozygous HbE pattern. Nevertheless, molecular analysis of the β -globin genes was not performed, precluding definitive determination of the underlying genotype. Thus, based on the available hematological and hemoglobin analysis findings, the overall pattern was considered consistent with homozygous HbE disease, although molecular confirmation was not available (Brancaleoni et al., 2016; Cappellini et al., 2008; Modell & Darlison, 2008; Weatherall, 2010).

HbE disease is caused by a β -globin variant that has both structural hemoglobinopathy and thalassemia-like behavior. Homozygous HbE disease is often clinically mild, but it typically produces marked microcytosis, hypochromia, and target cells, with anemia that may be absent or mild. This fits the present case, in which the patient had mild anemia without hepatosplenomegaly, but a persistently low MCV and MCH. The peripheral blood smear also supported a hemoglobinopathy pattern, showing hypochromic anisopoikilocytosis with microcytic erythrocytes, target cells, teardrop cells, ovalocytes, spherocytes, and polychromasia. These findings highlight that the severity of red cell microcytosis may be disproportionate to the degree of anemia in HbE disease, and this discrepancy should prompt hemoglobin analysis rather than repeated empiric iron therapy alone.

The electrophoresis pattern was central to the diagnosis. In homozygous HbE disease, HbA is typically absent because both β -globin genes carry the HbE mutation, while HbE becomes the dominant hemoglobin fraction and HbF or Hb variant may be mildly elevated. In the present patient, the absence of HbA and predominance of HbE were consistent with this pattern. This distinction is important because HbE disease may resemble HbE/ β -thalassemia on initial clinical assessment, but HbE/ β -thalassemia can have a much wider and often more severe clinical spectrum. Therefore, hemoglobin electrophoresis or capillary electrophoresis is not merely confirmatory, but essential for classifying the hemoglobinopathy and guiding counseling, follow-up, and family screening. The general diagnosis of homozygous HbE disease was supported by the characteristic hemoglobin analysis pattern. The general diagnostic approach to thalassemia and hemoglobinopathies is illustrated in Figure 2.

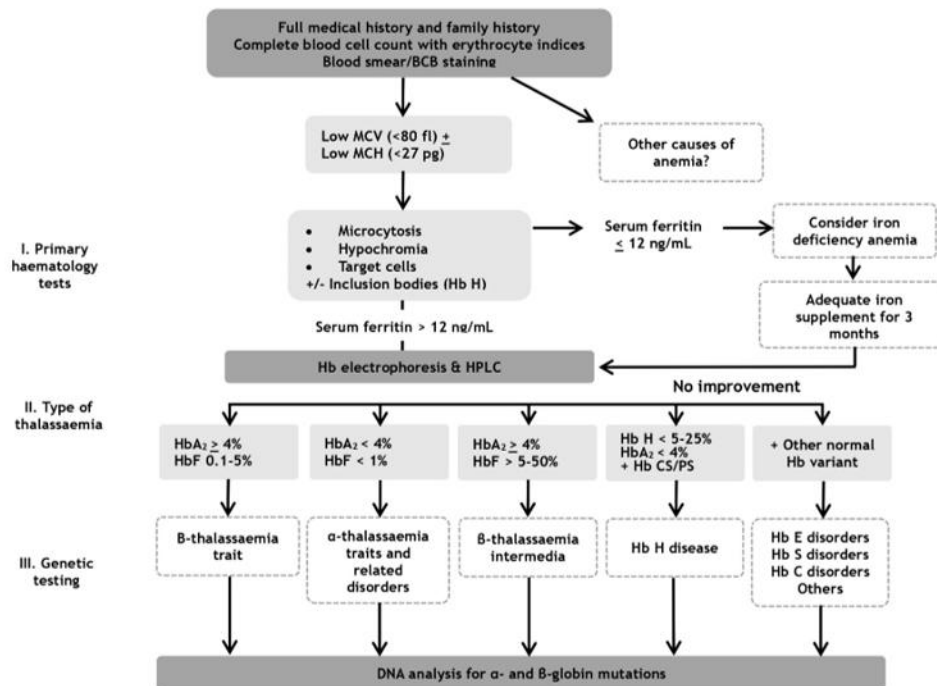


Figure 2. Diagnostic Algorithm for the Laboratory Evaluation of Thalassemia and Hemoglobinopathies. Reproduced from Brancaloni et al.

Another notable aspect of this case is the presence of persistent thrombocytosis. The patient's platelet count remained elevated across serial examinations, ranging from 530 to 709 $\times 10^3/\mu\text{L}$. In clinical practice, thrombocytosis is more frequently reactive than clonal and may occur in association with iron deficiency, inflammation, tissue injury, hemolysis, infection, or postsurgical states, although a clonal myeloproliferative process should also be considered when thrombocytosis persists. In this patient, the recent history of uterine curettage for blighted ovum and reported uterine wall inflammation provided a plausible inflammatory or reactive context. However, objective inflammatory markers were not available, and the available clinical information did not establish inflammation as the definitive cause of thrombocytosis. The temporal relationship between the curettage and the serial platelet measurements should therefore be considered when interpreting the platelet elevation.

Peripheral blood smear showed an increased platelet count with giant platelets and no platelet clumping. JAK2 mutation analysis was negative; however, a negative JAK2 result does not exclude essential thrombocythemia or another clonal myeloproliferative neoplasm. Similarly, the presence of giant platelets and absence of splenomegaly are not sufficient to distinguish reactive from clonal thrombocytosis. Therefore, the thrombocytosis in this patient was considered as suspected reactive thrombocytosis, while a clonal etiology cannot be completely excluded. If thrombocytosis persists after recovery from the recent obstetric event and secondary causes have been appropriately evaluated, further assessment for a clonal myeloproliferative neoplasm, including CALR/MPL mutation testing and bone marrow examination, may be considered according to clinical indications (Tefferi et al., 2024).

The absence of hepatosplenomegaly and the normal abdominal ultrasonography are also relevant. HbE disease is generally milder than many thalassemia syndromes, and significant

organomegaly is not always expected in uncomplicated homozygous HbE disease. In the present case, physical examination did not reveal hepatosplenomegaly, and abdominal ultrasonography showed no abnormalities in the liver, spleen, kidneys, uterus, or adnexa. This supports the interpretation that the patient's presentation was not dominated by severe chronic hemolysis, extramedullary hematopoiesis, or advanced thalassemic complications. However, the absence of organomegaly should not reduce the diagnostic significance of persistent microcytosis, because HbE disease may remain clinically subtle until detected during evaluation for anemia or reproductive planning.

The reproductive context is one of the most important clinical implications of this case. The patient was diagnosed after a first pregnancy complicated by blighted ovum and vaginal bleeding. Based strictly on the available data, HbE disease cannot be concluded as the cause of the blighted ovum, and the case should not overstate a causal relationship between the hemoglobinopathy and pregnancy loss. Nevertheless, the diagnosis has important implications for future pregnancies because hemoglobinopathies follow an inherited pattern, and fetal risk depends strongly on the partner's carrier status. If the husband carries β -thalassemia or another clinically significant hemoglobin variant, future offspring may be at risk for more severe disease, including HbE/ β -thalassemia. Therefore, the recommendation for husband screening was appropriate and represents a key learning point from this case and important components of future pregnancy planning (Gynecologists, 2017; Medicine et al., 2024).

This case emphasizes three practical lessons. First, persistent microcytosis with mild anemia should trigger consideration of hemoglobinopathy, especially when iron deficiency is not clearly severe. Second, thrombocytosis accompanying microcytic anemia should be interpreted carefully using serial blood counts, smear morphology, iron profile, inflammatory context, imaging, and molecular testing, rather than being immediately classified as a clonal platelet disorder. Third, HbE disease in a reproductive-age woman should prompt partner testing and genetic counseling, even when the patient is clinically stable. These lessons are especially relevant in Southeast Asian populations, where HbE and thalassemia variants are common and may remain undiagnosed until adulthood or pregnancy-related evaluation.^{14,15}

CONCLUSION

This case report highlights the importance of considering hemoglobinopathies in patients with persistent, marked microcytosis despite relatively mild anemia. In a 28-year-old woman, a predominant HbE fraction of 93.4%, absence of HbA, and a low HbF fraction supported a hemoglobin pattern consistent with homozygous hemoglobin E (HbE) disease, although definitive genotypic confirmation was not possible without molecular testing. Concurrent thrombocytosis was considered potentially reactive; however, its underlying cause remained undetermined, and negative JAK2 mutation testing did not exclude a clonal hematologic disorder. These findings emphasize the importance of integrating serial complete blood counts, iron studies, peripheral blood morphology, and hemoglobin analysis in the diagnostic evaluation of unexplained microcytosis. Although the available evidence did not establish a causal relationship between HbE disease and the preceding pregnancy loss, the diagnosis underscored the clinical importance of partner screening, genetic counseling, and reproductive risk assessment.

Future research should include larger prospective cohorts with molecular confirmation to differentiate homozygous HbE disease from HbE/ β -thalassemia and further characterize their hematologic profiles. Longitudinal evaluations incorporating serial platelet counts, inflammatory markers, iron status, and hemolysis parameters could help identify factors associated with concurrent thrombocytosis and its persistence. Further investigations of patients with unexplained persistent thrombocytosis should consider additional molecular testing, including CALR and MPL mutation analyses, and bone marrow examination when clinically indicated to evaluate potential clonal hematologic disorders. Research involving women of reproductive age should also investigate pregnancy outcomes and the clinical utility of partner screening and genetic counseling while accounting for other established causes of pregnancy loss. Such studies could strengthen the evidence supporting diagnostic strategies, differential diagnosis, and reproductive risk assessment beyond the limitations of this single case report.

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CONFLICT OF INTEREST

The author states that there are no conflicts of interest, whether personal, commercial, or financial, that can affect the implementation and results of this study.

AUTHOR CONTRIBUTION

Putu Niara Vinayah contributes to research design, data collection and processing, system development, result analysis, and article manuscript preparation.

GENERATIVE ARTIFICIAL INTELLIGENCE USAGE STATEMENT

In the process of compiling this article, the author uses generative artificial intelligence (AI) as a tool in language refinement, writing structure, and grammar improvement. The use of AI is carried out on a limited basis and remains under the supervision of the author. All data, analysis, interpretation, and scientific substance in this article have been checked and verified by the author. The author is solely responsible for the accuracy, authenticity, and final content of the manuscript.

REFERENCES

- Almanaseer, A., Chin-Yee, B., Ho, J., Lazo-Langner, A., Schenkel, L., & Bhai, P. (2024). An approach to the investigation of thrombocytosis: Differentiating between essential thrombocythemia and secondary thrombocytosis. *Advances in Hematology*, 2024, 3056216. <https://doi.org/10.1155/2024/3056216>
- Bleeker, J. S., & Hogan, W. J. (2011). Thrombocytosis: Diagnostic evaluation, thrombotic risk stratification, and risk-based management strategies. *Thrombosis*, 2011, 536062. <https://doi.org/10.1155/2011/536062>

- Brancaleoni, V., Di Pierro, E., Motta, I., & Cappellini, M. D. (2016). Laboratory diagnosis of thalassemia. *International Journal of Laboratory Hematology*, 38(Suppl 1), 32–40. <https://doi.org/10.1111/ijlh.12527>
- Cao, A., & Galanello, R. (2010). Beta-thalassemia. *Genetics in Medicine*, 12(2), 61–76. <https://doi.org/10.1097/GIM.0b013e3181cd68ed>
- Cappellini, M. D., Cohen, A., Eleftheriou, A., Piga, A., Porter, J., & Taher, A. (2008). *Guidelines for the clinical management of thalassaemia* (2nd ed.). Thalassaemia International Federation.
- Dolai, T. K., Roy, S., & Panja, A. (2026). Molecular genetics and recent management strategies of HB E- β thalassemia. *Indian Journal of Hematology and Blood Transfusion*, 42(2), 344–356. <https://doi.org/10.1007/s12288-025-02074-z>
- Foord, E., Sairafi, D., Berg, M., Westerling, S., Adigun, T., Uzunel, M., Papasavva, T., & Uhlin, M. (2026). Decoding thalassemia and sickle cell disease: Advances in molecular technologies for comprehensive variant detection. *Frontiers in Genetics*, 17, 1810737. <https://doi.org/10.3389/fgene.2026.1810737>
- Fucharoen, S., & Weatherall, D. J. (2012). The hemoglobin E thalassemias. *Cold Spring Harbor Perspectives in Medicine*, 2(8), a011734. <https://doi.org/10.1101/cshperspect.a011734>
- Gavillet, M., Legardeur, H., Adam, C., Renella, R., Dashraath, P., & Baud, D. (2026). HbF-inducing gene therapies for the β -hemoglobinopathies: Implications for future pregnancies. *Blood Red Cells & Iron*, 2(1), 100047. <https://doi.org/10.1016/j.brci.2026.100047>
- Godfrey, A. L., Khan, A. A., McGregor, A., Innes, A. J., Altohami, M., Cross, N. C. P., Frewin, R., Garg, M., Green, A., Grinfeld, J., McLornan, D. P., Wilson, A. J., Harrison, C. N., Mead, A. J., & Nangalia, J. (2026). Investigation and management of thrombocytosis without JAK2, CALR or MPL mutations: A British Society for Haematology Guideline. *British Journal of Haematology*, 208(2), 471–485. <https://doi.org/10.1111/bjh.70260>
- Gynecologists, A. C. of O. and. (2017). Committee Opinion No. 691: Carrier screening for genetic conditions. *Obstetrics & Gynecology*, 129(3), e41–e55. <https://doi.org/10.1097/AOG.0000000000001952>
- Kiran, S. S., Aithal, S., & Belagavi, C. S. (2016). Hemoglobin E hemoglobinopathy in an adult from Assam with unusual presentation: A diagnostic dilemma. *Journal of Laboratory Physicians*, 8(2), 116–119. <https://doi.org/10.4103/0974-2727.180793>
- Medicine, S. for M.-F., Sinkey, R. G., Ogunbile, F. J., Kanter, J., Bean, C., & Greenberg, M. (2024). Society for Maternal-Fetal Medicine Consult Series #68: Sickle cell disease in pregnancy. *American Journal of Obstetrics and Gynecology*, 230(2), B17–B40. <https://doi.org/10.1016/j.ajog.2023.10.031>
- Modell, B., & Darlison, M. (2008). Global epidemiology of haemoglobin disorders and derived service indicators. *Bulletin of the World Health Organization*, 86(6), 480–487. <https://doi.org/10.2471/BLT.06.036673>
- Munkongdee, T., Chen, P., Winichagoon, P., Fucharoen, S., & Paiboonsukwong, K. (2020). Update in laboratory diagnosis of thalassemia. *Frontiers in Molecular Biosciences*, 7, 74. <https://doi.org/10.3389/fmolb.2020.00074>
- Prasing, W., & Pornprasert, S. (2014). Measurement of HbA₂ by capillary electrophoresis for diagnosing β -thalassemia/HbE disease in patients with low HbF. *Laboratory Medicine*, 45(3), 226–230. <https://doi.org/10.1309/LMGD96HES3DZRBZM>
- Rokkam, V. R., Killeen, R. B., & Kotagiri, R. (2026). Secondary thrombocytosis. In *StatPearls*. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK560810/>
- Taher, A. T., Weatherall, D. J., & Cappellini, M. D. (2018). Thalassaemia. *The Lancet*, 391(10116), 155–167. [https://doi.org/10.1016/S0140-6736\(17\)31822-6](https://doi.org/10.1016/S0140-6736(17)31822-6)

- Tefferi, A., Vannucchi, A. M., & Barbui, T. (2024). Essential thrombocythemia: 2024 update on diagnosis, risk stratification, and management. *American Journal of Hematology*, 99(4), 697–718. <https://doi.org/10.1002/ajh.27216>
- Weatherall, D. J. (2010). The inherited diseases of hemoglobin are an emerging global health burden. *Blood*, 115(22), 4331–4336. <https://doi.org/10.1182/blood-2010-01-251348>