

Review Article

Primary Myelofibrosis: Current Concepts in Molecular Pathogenesis, Precision Diagnosis, and Emerging Therapeutic Strategies

Francisca Obianuju Ndubuisi¹, Silas Anayo Ufelle¹ and Alphonsus Ogbonna Ogbuabor ^{2*}

¹Department of Medical Laboratory Sciences, Faculty of Health Science and Technology, University of Nigeria.

²Department of Medical Laboratory Science, Faculty of Allied Health Sciences, Enugu State University of Science and Technology, Agbani, Nigeria.

*Corresponding author: ogbuaborao@yahoo.com

Article Info

Keywords: Primary myelofibrosis, Epidemiology, Pathogenesis, Diagnosis, Prognosis, Treatment.

Received: 16.09.2026;

Accepted: 21.10.2026;

Published: 30.10.2026



© 2026 by the author's. The terms and conditions of the Creative Commons Attribution (CC BY) license apply to this open access article.

Abstract

Primary myelofibrosis (PMF) is a clonal myeloproliferative neoplasm characterized by bone marrow fibrosis, extramedullary hematopoiesis, and progressive cytopenias. Advances in molecular biology, diagnostic criteria, prognostic modeling, and targeted therapies have significantly improved the understanding of management of PMF. This review focused on the molecular pathogenesis, diagnostic criteria, prognostic scoring systems, therapeutic strategies, and precision medicine approach in PMF. A comprehensive literature search on molecular pathogenesis, clinical trials, prognostic models, and emerging therapies in PMF. Aberrant activation of the JAK-STAT pathway due to driver mutations (JAK2, CALR, MPL) and additional high-risk mutations promotes uncontrolled megakaryopoiesis, chronic inflammation, and fibrosis. The WHO 2022 and ICC 2022 criteria refined diagnostic accuracy through integrated morphological and molecular approaches. Molecular-based prognostic models such as MPSS70 + v2.0 and GIPSS provide superior risk stratification. JAK inhibitors (ruxotinib, fedratinib, pancritinib, mpmelotinib) remain central in symptom control and spleen reduction. Emerging therapies including telomerase inhibitors, BET inhibitors, and precision combinations show promising early results. Hematopoietic stem cell transplantation remains the only curative approach for eligible patients. Precision medicine integrating multi-omics, AI, and real-world data is transforming PMF management. In recent times, PMF management has evolved toward molecularly guided, risk-adapted, and patient-centered care. The integration of advanced diagnostics, novel therapeutics, and precision approaches offers hope for improved survival and quality of life.

1. Introduction

Primary myelofibrosis (PMF) is a chronic Philadelphia chromosome-negative myeloproliferative neoplasm (MPN) characterized by clonal hematopoietic stem-cell proliferation, progressive bone marrow fibrosis, ineffective hematopoiesis, splenomegaly, constitutional symptoms, and an increased risk of acute myeloid leukemia (AML) transformation [1]. Among the classic MPNs, PMF generally exhibits an aggressive clinical course, substantial symptom burden, and adverse survival outcomes [2]. PMF arises from somatic mutations in hematopoietic stem cells, predominantly involving JAK2, CALR, or MPL. These mutations activate JAK-STAT signaling, promoting abnormal myeloid

proliferation, inflammation, and bone marrow remodeling [3, 4]. Additional mutations involving ASXL1, EZH2, SRSF2, IDH1, IDH2, and U2AF1 contribute to disease progression, leukemic transformation, and adverse outcomes [4, 5]. Advances in molecular understanding have transformed PMF from a fibrosis-defined disorder into a complex clonal disease involving genetic alterations, inflammatory signaling, immune dysregulation, and stromal remodeling. The 2022 World Health Organization classification and International Consensus Classification distinguish prefibrotic from overt fibrotic PMF, emphasizing integrated morphological, molecular, clinical, and laboratory assessments for accurate diagnosis [5, 6]. Despite advances in supportive care, PMF causes substantial morbidity, including cytopenias, splenomegaly, thrombohemorrhagic complications, and possible leukemic transformation [7, 8]. JAK inhibitors improve symptoms and quality of life but rarely eradicate the malignant clone. Allogeneic hematopoietic stem-cell transplantation (HSCT) remains the only established curative treatment, although eligibility is limited by age, comorbidities, disease risk, and treatment-related complications [9–11]. Emerging sequencing, artificial intelligence, and multi-omics technologies offer opportunities for precision risk stratification and individualized treatment [12, 13]. This review examines PMF epidemiology, pathogenesis, clinical manifestations, diagnosis, prognosis, treatment, and emerging innovations, highlighting research gaps and future directions toward effective disease modification and curative therapies.

2. Epidemiology

Primary myelofibrosis is a rare but clinically significant MPN characterized by progressive bone marrow fibrosis, ineffective hematopoiesis, and variable clinical outcomes. Despite its rarity, PMF imposes substantial clinical, psychosocial, and economic burdens, with significant implications for survival and quality of life [14]. PMF has an estimated annual incidence of approximately 0.4–1.5 cases per 100,000 individuals, although reported rates vary by population, diagnostic criteria, and registry coverage [15, 16]. Improved molecular diagnostics, disease recognition, and supportive management have influenced reported epidemiological patterns. Revised diagnostic frameworks, including the 2022 WHO classification and International Consensus Classification, have improved differentiation between prefibrotic and overt fibrotic disease, supporting more consistent epidemiological reporting [5, 6]. PMF predominantly affects older adults, with a median diagnostic age commonly reported around 65–70 years. Its incidence increases with age, potentially reflecting the accumulation of somatic mutations and age-associated clonal hematopoiesis [16, 17]. Age-related comorbidities also influence treatment selection and eligibility for transplantation [9]. Geographical disparities remain substantial. Although higher reported rates occur in regions with established cancer registries and advanced diagnostic services, PMF may be underdiagnosed in Africa, South Asia, and parts of Latin America because of limited access to bone marrow histopathology, molecular testing for JAK2, CALR, and MPL, and specialist care. Available international estimates should therefore be interpreted cautiously when comparing populations with different diagnostic capacities. Further population-based studies and broader genomic investigations are needed to clarify geographical differences in disease incidence, mutation profiles, and clinical outcomes [14, 17, 18]. Clinically, PMF causes fatigue, anemia, splenomegaly, bone pain, and constitutional symptoms, often necessitating transfusions, hospitalization, and prolonged supportive care [19]. Prognosis varies considerably according to clinical and molecular risk. Patients with low-risk disease may survive for many years, whereas those with very-high-risk disease may have substantially shorter survival. Transformation to AML represents a particularly serious complication [7, 20]. Treatment costs further intensify this burden, particularly where targeted therapies and transplantation are inaccessible. Standardized surveillance, broader molecular profiling, and equitable access to diagnostic and therapeutic services are therefore essential to establish PMF's true global burden and improve patient outcomes.

3. Pathogenesis

Primary myelofibrosis is a clonal hematopoietic stem-cell neoplasm characterized by progressive bone marrow fibrosis, ineffective hematopoiesis, aberrant megakaryocyte proliferation, and disruption of the bone marrow microenvironment. Its pathogenesis involves interacting genetic, epigenetic, inflammatory, and stromal abnormalities that drive disease initiation, progression, and, in some patients, transformation to AML [3, 4].

3.1. Clonal Evolution and Driver Mutations

The disease originates from acquired somatic mutations in hematopoietic stem cells, conferring proliferative and survival advantages. The principal driver mutations affect JAK2, CALR, and MPL, resulting in constitutive activation of the JAK–STAT pathway [3, 4, 21, 22]. JAK2 V617F occurs in approximately 55–65% of patients and promotes growth factor-independent hematopoietic proliferation. CALR mutations, present in approximately 20–30%, produce abnormal calreticulin proteins that activate the thrombopoietin receptor, sustaining JAK–STAT signaling [23–25]. MPL mutations, identified in a smaller proportion of patients, directly activate thrombopoietin receptor signaling and promote abnormal megakaryopoiesis [26, 27]. Approximately 10% of patients lack these three principal driver mutations and are classified as having triple-negative PMF. This group is biologically heterogeneous, and its prognosis depends on the broader clinical, cytogenetic, and molecular profile [28].

3.2. Additional Molecular Abnormalities

Mutations affecting epigenetic regulation and RNA splicing contribute to clonal evolution and disease heterogeneity. ASXL1 mutations disrupt chromatin regulation and are associated with shortened survival and increased leukemic transformation risk [29]. EZH2 mutations impair histone methylation and transcriptional regulation, contributing to disease progression [30]. Mutations in IDH1 and IDH2 can generate the oncometabolite 2-hydroxyglutarate, promoting epigenetic dysregulation. Meanwhile, SRSF2 and U2AF1 mutations disrupt RNA processing and are associated with adverse disease features, including reduced survival and severe anemia [31, 32]. These abnormalities help explain the substantial biological heterogeneity observed among patients with PMF [33, 34]. Evolving classification frameworks have increasingly integrated these molecular findings with morphology and clinical features [35].

3.3. Megakaryocyte Dysfunction and Bone Marrow Fibrosis

A central feature of PMF is the abnormal proliferation and maturation of megakaryocytes, which display atypical morphology and clustering. These cells release profibrotic and proinflammatory mediators, particularly transforming growth factor-beta (TGF- β) and platelet-derived growth factor (PDGF). These mediators activate bone marrow stromal cells and fibroblasts, stimulating excessive deposition of reticulin and collagen fibers [36, 37]. Importantly, the fibrosis is predominantly a reactive response to the malignant hematopoietic clone rather than an independent clonal proliferation of fibroblasts. Changes in the marrow niche further sustain abnormal hematopoiesis and disease progression [36].

3.4. Inflammation, Ineffective Hematopoiesis, and Disease Progression

Chronic inflammatory signaling sustains abnormal hematopoiesis, stromal activation, and angiogenesis, reinforcing a self-perpetuating cycle of marrow injury [38]. Progressive fibrosis disrupts the hematopoietic niche, leading to ineffective blood-cell production, cytopenias, and the release of immature blood cells into peripheral circulation.

As marrow function deteriorates, hematopoiesis shifts to extramedullary sites, particularly the spleen and liver, causing organ enlargement. Additional genetic abnormalities may promote clonal evolution and leukemic transformation [4, 20]. Molecular prognostic systems, including MIPSS70+ Version 2.0 and GIPSS, integrate adverse mutations with clinical and cytogenetic variables to improve risk stratification and guide management [20, 39].

4. Clinical Manifestations

The clinical presentation of PMF is heterogeneous, reflecting progressive bone marrow fibrosis, clonal hematopoiesis, chronic inflammation, and extramedullary hematopoiesis. While some patients remain asymptomatic and are diagnosed incidentally, others develop debilitating constitutional symptoms, severe cytopenias, massive splenomegaly, or life-threatening thrombohemorrhagic complications [7, 11]. Constitutional symptoms arise largely from persistent inflammatory cytokine production. Common symptoms include fatigue, weakness, unintended weight loss, fever, night sweats, pruritus, anorexia, and early satiety [38]. Fatigue is particularly debilitating, reflecting anemia, inflammation, and increased metabolic demand. Symptom assessment using validated instruments, such as the Myeloproliferative Neoplasm Symptom Assessment Form, supports clinical monitoring and treatment evaluation [40, 41]. Splenomegaly is a hallmark manifestation resulting from extramedullary hematopoiesis as marrow function deteriorates. Enlargement may cause abdominal discomfort, early satiety, weight loss, splenic infarction, and hypersplenism, further aggravating cytopenias. Hepatomegaly may also develop through hepatic extramedullary hematopoiesis, occasionally contributing to portal hypertension and abnormal liver function [42]. Hematological abnormalities include anemia, leukocytosis, leukopenia, thrombocytosis, and thrombocytopenia. Anemia results from ineffective erythropoiesis, marrow fibrosis, inflammatory suppression, hypersplenism, nutritional deficiencies, and treatment effects. It causes fatigue, pallor, dyspnea, dizziness, and reduced exercise tolerance, sometimes requiring transfusion support. Leukocytosis and thrombocytosis may characterize earlier disease, whereas leukopenia and thrombocytopenia can emerge with progression, indicating reduced marrow reserve and increased bleeding risk [7, 20]. Extramedullary hematopoiesis predominantly affects the spleen and liver but may involve lymph nodes, lungs, kidneys, skin, gastrointestinal tissues, and paraspinal regions. Depending on location, it can cause pulmonary hypertension, respiratory impairment, or spinal cord compression, occasionally requiring radiotherapy or other interventions [42]. Finally, PMF predisposes patients to thrombotic and hemorrhagic complications through abnormal platelet function, endothelial dysfunction, inflammation, and coagulation disturbances. Arterial and venous events include stroke, myocardial infarction, deep vein thrombosis, and pulmonary embolism. Bleeding may result from thrombocytopenia, platelet dysfunction, or acquired von Willebrand syndrome. These complications substantially increase morbidity and mortality, making individualized risk assessment essential for treatment planning and transplantation decisions [43].

5. Diagnosis

Diagnosing PMF requires an integrated assessment of clinical features, blood counts, peripheral blood morphology, bone marrow histopathology, cytogenetics, and molecular testing. Because PMF overlaps with other MPNs, myelodysplastic syndromes, and reactive marrow disorders, accurate diagnosis depends on combining these findings rather than relying on a single test [5–7]. Anemia is common and typically normocytic and normochromic, becoming more severe as disease progresses. Leukocytosis and thrombocytosis may occur early, whereas leukopenia and thrombocytopenia often indicate advanced disease and poorer prognosis [7, 44]. The blood smear characteristically reveals a leukoerythroblastic picture, tear-drop erythrocytes (dacrocytes), anisopoikilocytosis, immature granulocytes, nucleated red blood cells, and occasionally circulating blasts. These findings support the diagnosis and may indicate disease progression or leukemic transformation [7]. Bone marrow aspiration and trephine biopsy are essential for distinguishing prefibrotic from overt fibrotic PMF. Aspiration may produce a dry tap because of extensive fibrosis. Histopathology typically demonstrates atypical megakaryocytic proliferation, abnormal clustering, irregular hyperchromatic nuclei, and progressive reticulin or collagen deposition. Reticulin grading, alongside assessment of cellularity, osteosclerosis, and hematopoietic architecture, establishes disease stage and supports classification under WHO and ICC criteria [5, 6, 45]. Conventional karyotyping identifies chromosomal abnormalities, including deletions of 20q and 13q, trisomies 8 and 9, and complex karyotypes. Monosomy 7, chromosome 17 abnormalities, and complex karyotypes are associated with adverse outcomes and increased leukemic transformation risk [20, 39]. Molecular testing detects the principal driver mutations: JAK2, CALR, and MPL. Patients lacking all three are classified as triple-negative PMF. Additional mutations involving ASXL1, EZH2, SRSF2, IDH1, IDH2, U2AF1, RUNX1, and TP53 provide prognostic information and can inform treatment decisions [4, 17, 29, 31]. Next-generation sequencing enables simultaneous detection of multiple mutations, low-frequency subclones, and clonal evolution, strengthening diagnosis, risk stratification, and individualized management [4, 17]. Combined with clinical, morphological, and cytogenetic findings, genomic profiling advances precision medicine in PMF.

5.1. WHO Diagnostic Criteria

The diagnosis of PMF is established using integrated diagnostic criteria that incorporate bone marrow morphology, molecular genetics, clinical findings, and laboratory investigations. Contemporary classifications distinguish prefibrotic PMF from overt fibrotic PMF, recognizing these as different stages within the same disease spectrum [5, 6]. Major diagnostic criteria include characteristic megakaryocytic proliferation and atypia, accompanied by either minimal fibrosis in prefibrotic disease or significant reticulin or collagen fibrosis in overt PMF. Patients must also meet exclusion criteria for other myeloid neoplasms, including chronic myeloid leukemia, polycythemia vera, essential thrombocythemia, and relevant myelodysplastic or myeloproliferative disorders. Identification of a driver mutation involving JAK2, CALR, or MPL, or another clonal molecular abnormality, supports the diagnosis [5, 6]. Minor diagnostic criteria include anemia not explained by another condition, leukocytosis, palpable splenomegaly, elevated serum lactate dehydrogenase, and leukoerythroblastosis. The required combination of major and minor criteria depends on the classification system and whether prefibrotic or overt fibrotic PMF is being evaluated [5, 6]. Secondary causes of bone marrow fibrosis must also be excluded. The incorporation of molecular testing into contemporary diagnostic criteria represents a major advance over earlier classification systems and reflects the increasing importance of genomic medicine in hematological diagnosis.

6. Risk Stratification

Modern risk-stratification systems are indispensable components of PMF management because they directly influence therapeutic decision-making. Patients classified as low or intermediate-1 risk are frequently managed with observation, symptom-directed therapy, or JAK inhibitors, depending on symptom burden and disease progression. Individuals with intermediate-2 or high-risk disease are generally evaluated for allogeneic HSCT, which remains the only established curative treatment [10, 20]. Prognostic systems include the International Prognostic Scoring System (IPSS), Dynamic International Prognostic Scoring System (DIPSS), DIPSS Plus, and molecularly informed models such as MIPSS70+ Version 2.0 and GIPSS. These models incorporate varying combinations of clinical characteristics, blood counts, transfusion requirements, cytogenetic findings, and adverse mutations [20, 39, 46–50].

Contemporary prognostic models facilitate:

1. Selection of candidates for transplantation.
2. Identification of patients requiring intensive monitoring.
3. Stratification of participants in clinical trials.
4. Individualized counseling regarding prognosis.
5. Optimization of targeted therapeutic strategies.

As molecular diagnostics evolve, future prognostic systems may incorporate artificial intelligence, machine learning, measurable residual disease assessment, transcriptomics, epigenomics, and other multi-omics technologies to improve prognostic accuracy and personalize patient management [12, 51].

7. Current Therapeutic Strategies

Management of PMF has advanced through targeted therapies and transplantation, although most pharmacological treatments do not eradicate the malignant stem-cell clone. Treatment is individualized according to symptoms, cytopenias, molecular risk, age, and transplant eligibility, aiming to improve quality of life, control splenomegaly, prevent complications, and prolong survival [9, 20]. Contemporary management integrates supportive care, targeted pharmacotherapy, and allogeneic HSCT [8, 10, 52]. Supportive care remains essential throughout the disease course [8]. Red blood cell transfusions address symptomatic anemia, although prolonged transfusion dependence may cause iron overload requiring monitoring and, in selected patients, chelation therapy. Nutritional support, vaccination, infection prevention, cardiovascular risk management, and psychological care further improve patient well-being [40]. Anemia management is individualized according to its severity and underlying mechanisms, including ineffective erythropoiesis, inflammation, marrow fibrosis, and hypersplenism. Erythropoiesis-stimulating agents may benefit selected patients with low endogenous erythropoietin levels. Danazol, corticosteroids, thalidomide, and lenalidomide-based regimens may also improve anemia, although adverse effects limit their use. Momelotinib, which inhibits JAK1/JAK2 and activin receptor type I, can improve anemia, splenomegaly, and constitutional symptoms. Other anemia-directed agents, including luspatercept, are under investigation or may be considered in selected clinical settings [53, 54]. JAK inhibitors have transformed PMF treatment by suppressing aberrant JAK–STAT signaling, reducing inflammation, shrinking the spleen, and alleviating constitutional symptoms [55]. Ruxolitinib is widely used for symptomatic intermediate- or high-risk disease [56, 57], while fedratinib provides an option for patients with advanced disease, including some previously treated with ruxolitinib [58, 59]. Pacritinib is particularly valuable for patients with severe thrombocytopenia [60]. Momelotinib offers an additional advantage in anemic patients by targeting pathways involved in hepcidin regulation and iron availability for erythropoiesis [53, 54, 61–63]. These agents enable treatment selection based on individual clinical characteristics and tolerability. Allogeneic HSCT remains the only established curative treatment, offering the potential for disease eradication through replacement of malignant hematopoietic cells with donor-derived stem cells. However, substantial treatment-related risks necessitate careful selection, particularly among intermediate-2- and high-risk patients, younger individuals with adverse molecular features, and patients with progressive disease despite medical therapy [10, 11, 64].

8. Future Therapeutic Directions

Despite remarkable advances in targeted therapy, important therapeutic challenges remain. Current JAK inhibitors primarily provide symptomatic benefit rather than reliably eradicating the malignant clone. Consequently, clinical research increasingly focuses on combination therapies that simultaneously target malignant stem cells, inflammatory pathways, bone marrow fibrosis, and the surrounding microenvironment [65]. Novel therapeutic strategies under investigation include:

- BET inhibitors: Targeting epigenetic mechanisms involved in abnormal gene expression.
- BCL-2/BCL-xL inhibitors: Promoting apoptosis of malignant hematopoietic cells.
- Telomerase inhibitors: Targeting mechanisms involved in clonal cell survival.
- TGF- β pathway inhibitors: Potentially reducing profibrotic signaling and marrow remodeling.
- Epigenetic therapies: Targeting abnormal DNA methylation, chromatin regulation, and transcription.
- Immunotherapy: Modulating immune responses against malignant cells.
- Gene-editing technologies: Exploring targeted correction or modification of hematopoietic stem cells.
- Personalized combination regimens: Integrating molecular profiling, disease risk, treatment response, and tolerability.

Several investigational agents and combination strategies are being evaluated in clinical trials, including BET-inhibitor combinations, apoptosis-directed regimens, and telomerase inhibition, although their long-term efficacy, safety, and ability to modify disease progression remain to be established [65–70]. The combination of JAK inhibitors with agents targeting apoptosis, epigenetic regulation, or the bone marrow microenvironment may offer opportunities to improve treatment depth and durability. Artificial intelligence and multi-omics approaches may further improve patient selection, identify predictive biomarkers, and guide individualized treatment strategies [12, 51]. Nevertheless, these technologies require rigorous validation in diverse patient populations before routine clinical implementation [71–75].

9. Conclusion

Primary myelofibrosis is a biologically heterogeneous myeloproliferative neoplasm characterized by clonal hematopoiesis, progressive bone marrow fibrosis, chronic inflammation, ineffective hematopoiesis, and a variable risk of leukemic transformation. Advances in molecular genetics have improved understanding of disease mechanisms, while integrated morphological, clinical, cytogenetic, and molecular assessments have strengthened diagnosis and prognostic classification. Contemporary risk-stratification systems support individualized treatment decisions, including the use of JAK inhibitors, supportive therapies, and allogeneic hematopoietic stem-cell transplantation. Although targeted therapies have substantially improved symptom control, splenic responses, and quality of life, their capacity to eradicate the malignant clone remains limited. Future progress will depend on developing disease-modifying combination therapies, improving anemia management, targeting the bone marrow microenvironment, and integrating genomic and multi-omics technologies into precision medicine. Equitable access to molecular diagnostics, effective treatments, and transplantation services is particularly important in resource-limited settings. Evidence from long-term treatment follow-up, mutation studies, and contemporary diagnostic and risk-stratification research also underscores the need to integrate clinical, molecular, and longitudinal data when refining individualized prognosis [71–75]. Ultimately, collaborative research and inclusive clinical trials are essential to improve survival and advance curative strategies.

Article Information

Consent for Publication: All the authors have reviewed and approved this version of the manuscript for publication.

Data Availability : Data sharing is not applicable to this article as no new data were created or analyzed in this study.

Conflict of Interest: The authors declare that they have no conflict of interest.

Funding: No external funding was received for this study.

Author Contributions: F.O.N. - Conceptualization, Methodology, Investigation, Data Curation, Formal Analysis, Resources, Writing – Original Draft, Writing – Review & Editing, Project Administration; S.A.U. - Conceptualization, Methodology, Data Curation, Writing – Review & Editing, Supervision; A.O.O. - Conceptualization, Methodology, Data Curation, Formal Analysis, Writing – Original Draft, Writing – Review & Editing.

AI Use Disclosure: No generative artificial intelligence tools were used in the preparation of this manuscript.

References

- [1] M. Mahmud, S. Vasireddy, K. Gowin, and A. Amaraneni. Myeloproliferative neoplasms: contemporary review and molecular landscape. *Int J Mol Sci*, 24(24):17383, 2023.
- [2] J. Baumeister, N. Chatain, A. M. Sofias, T. Lammers, and S. Koschmieder. Progression of myeloproliferative neoplasms (MPN): diagnostic and therapeutic perspectives. *Cells*, 10(12):3551, 2021.
- [3] W. Vainchenker and R. Kralovics. Genetic basis of myeloproliferative neoplasms and JAK-STAT pathway activation. *Nat Rev Cancer*, 23(6):385–402, 2023.
- [4] T. Verma, N. Papadantonakis, D. Peker Barclift, and L. Zhang. Molecular genetic profile of myelofibrosis: implications in the diagnosis, prognosis, and treatment advancements. *Cancers (Basel)*, 16(3):514, 2024.
- [5] D. A. Arber, A. Orazi, R. P. Hasserjian, M. J. Borowitz, K. R. Calvo, H. M. Kvasnicka, S. A. Wang, A. Bagg, T. Barbui, S. Branford, et al. International Consensus Classification of Myeloid Neoplasms and Acute Leukemias: integrating morphologic, clinical, and genomic data. *Blood*, 140(11):1200–28, 2022. URL <https://doi.org/10.1182/blood.2022015850>.
- [6] J. Thiele, H. M. Kvasnicka, A. Orazi, U. Gianelli, N. Gangat, A. M. Vannucchi, T. Barbui, D. A. Arber, and A. Tefferi. The International Consensus Classification of myeloid neoplasms and acute leukemias: myeloproliferative neoplasms. *Am J Hematol*, 98(1):166–79, 2023. URL <https://doi.org/10.1002/ajh.26821>.

- [7] A. Tefferi and A. Pardanani. Primary myelofibrosis: 2023 update on diagnosis, risk stratification, and management. *Am J Hematol.*, 98(5):801–21, 2023.
- [8] D. P. McLornan, B. Psaila, J. Ewing, A. Innes, S. Arami, J. Brady, N. M. Butt, C. Cargo, N. C. P. Cross, et al. The management of myelofibrosis: a British Society for Haematology guideline. *Br J Haematol.*, 204(1):136–150, 2024. URL <https://doi.org/10.1111/bjh.19186>.
- [9] C. N. Harrison, A. M. Vannucchi, J. J. Kiladjian, H. K. Al-Ali, H. Gisslinger, L. Knoops, F. Cervantes, M. M. Jones, K. Sun, M. McQuitty, V. Stalbovskaya, P. Gopalakrishna, and T. Barbui. Long-term findings from COMFORT-II, a phase 3 study of ruxolitinib vs best available therapy for myelofibrosis. *Leukemia.*, 30(8):1701–7. URL <https://doi.org/10.1038/leu.2016.148>.
- [10] N. Kröger, A. Bacigalupo, T. Barbui, M. Ditschkowski, N. Gagelmann, M. Griesshammer, V. Gupta, N. Hamad, C. Harrison, J. C. Hernandez-Boluda, et al. Indication and management of allogeneic haematopoietic stem-cell transplantation in myelofibrosis: updated recommendations by the EBMT/ELN International Working Group. *Lancet Haematol.*, 11(1):e62–e74, 2024. URL [https://doi.org/10.1016/s2352-3026\(23\)00305-8](https://doi.org/10.1016/s2352-3026(23)00305-8).
- [11] J. Mascarenhas and P. Bose. Precision medicine and future therapeutic directions in myelofibrosis. *Nat Rev Clin Oncol.*, 21(4):245–60, 2024.
- [12] A. Golestan, M. Zareinejad, and A. Ramezani. Comprehensive biomarker profiles in hematological malignancies: improving diagnosis, prognosis, and treatment. *Biomark Med.*, 19(6):223–38, 2025.
- [13] D. P. McLornan, A. L. Godfrey, A. Green, R. Frewin, S. Arami, J. Brady, N. M. Butt, C. Cargo, J. Ewing, S. Francis, et al. Diagnosis and evaluation of prognosis of myelofibrosis: a British Society for Haematology guideline. *Br J Haematol.*, 204(1):127–135, 2024. URL <https://doi.org/10.1111/bjh.19164>.
- [14] M. Griesshammer, H. K. Al-Ali, G. M. Baerlocher, K. Döhner, S. Koschmieder, N. Kröger, P. E. Petrides, D. Wolf, and F. H. Heidel. Primary myelofibrosis (PMF)—the German ONKOPEDIA guideline 2025. *Int J Cancer.*, 159(7):1642–1662, 2026. URL <https://doi.org/10.1002/ijc.70596>.
- [15] F. Cervantes, A. Pereira, and J. C. Hernández-Boluda. Epidemiology and clinical burden of primary myelofibrosis. *Leuk Res.*, 118:106879, 2022.
- [16] L. Masarova, S. Verstovsek, and P. Bose. Contemporary perspectives on primary myelofibrosis epidemiology and outcomes. *Lancet Haematol.*, 11(3):e221–e233, 2024.
- [17] A. Tefferi and P. Guglielmelli. Molecular genetics and pathogenesis of primary myelofibrosis. *Nat Rev Dis Primers*, 10(1):12, 2024.
- [18] M. Griesshammer, H. K. Al-Ali, J. N. Eckardt, M. Fiegl, J. Göthert, K. Jentsch-Ullrich, S. Koschmieder, H. M. Kvasnicka, A. Reiter, B. Schmidt, and F. H. Heidel. How I diagnose and treat patients in the pre-fibrotic phase of primary myelofibrosis: practical approaches of a German expert panel discussion in 2024. *Ann Hematol.*, 104:295–306, 2025. URL <https://doi.org/10.1007/s00277-025-06191-7>.
- [19] R. A. Mesa, S. Verstovsek, and V. Gupta. Quality of life and symptom burden in myelofibrosis. *J Clin Oncol.*, 41(14):2684–93, 2023.
- [20] A. Tefferi, P. Guglielmelli, and A. M. Vannucchi. Contemporary prognostic models in primary myelofibrosis. *Am J Hematol.*, 99(2):201–15, 2024.
- [21] C. James, V. Ugo, J. P. Le Couédic, J. Staerk, F. Delhommeau, C. Lacout, L. Garçon, H. Raslova, R. Berger, A. Bennaceur-Griscelli, J. L. Villeval, S. N. Constantinescu, N. Casadevall, and W. Vainchenker. A unique clonal JAK2 mutation leading to constitutive signalling causes polycythaemia vera. *Nature.*, 434(7037):1144–1148, 2005. URL <https://doi.org/10.1038/nature03546>.
- [22] R. Kralovics, F. Passamonti, A. S. Buser, S. S. Teo, R. Tiedt, J. R. Passweg, A. Tichelli, M. Cazzola, and R. C. Skoda. A gain-of-function mutation of JAK2 in myeloproliferative disorders. *N Engl J Med.*, 352(17):1779–1790, 2005. URL <https://doi.org/10.1056/NEJMoa051113>.
- [23] V. Bellani, B. Mora, A. Iurlo, and F. Passamonti. CALR-mutated myeloproliferative neoplasms. *Leuk Lymphoma.*, 66(7):1198–210, 2025.
- [24] T. Klampfl, H. Gisslinger, A. S. Harutyunyan, H. Nivarti, E. Rumi, J. D. Milosevic, Nicole C.C. Them, T. Berg, B. Gisslinger, D. Pietra, et al. Somatic mutations of calreticulin in myeloproliferative neoplasms. *N Engl J Med.*, 369(25):2379–2390, 2013. URL <https://doi.org/10.1056/NEJMoa1311347>.
- [25] J. Nangalia, C. E. Massie, E. J. Baxter, F. L. Nice, G. Gundem, D. C. Wedge, E. Avezov, J. Li, K. Kollmann, D.G. Kent, et al. Somatic CALR mutations in myeloproliferative neoplasms with nonmutated JAK2. *N Engl J Med.*, 369(25):2391–2405, 2013. URL <https://doi.org/10.1056/NEJMoa1312542>.
- [26] S. Zhang, M. S. Ashaq, M. Xu, M. Guo, L. Wang, Z. Li, Y. Wang, Y. Huang, Y. Li, and B. Zhao. Thrombopoietin receptor MPL in hematopoiesis and myeloproliferative neoplasms: physiological roles and pathogenic mechanisms. *Stem Cell Rev Rep*, 22(5):2252–2265, 2026. URL <https://doi.org/10.1007/s12015-026-11136-2>.

- [27] Y. Pikman, B. H. Lee, T. Mercher, E. McDowell, B. L. Ebert, M. Gozo, A. Cuker, G. Wernig, S. Moore, I. Galinsky, D. J. DeAngelo, J. J. Clark, S. J. Lee, T. R. Golub, M. Wadleigh, D. G. Gilliland, and R. L. Levine. MPLW515L is a novel somatic activating mutation in myelofibrosis with myeloid metaplasia. *PLoS Med.*, 3(7):e270, 2006. URL <https://doi.org/10.1371/journal.pmed.0030270>.
- [28] S. Tharakan, J. Mascarenhas, and D. Tremblay. Understanding triple-negative myeloproliferative neoplasms: pathogenesis, clinical features, and management. *Leuk Lymphoma.*, 65(2):158–67, 2024.
- [29] Z. Wang, W. Liu, M. Wang, Y. Li, X. Wang, E. Yang, J. Ming, R. Quan, and X. Hu. Prognostic value of ASXL1 mutations in patients with primary myelofibrosis and its relationship with clinical features: a meta-analysis. *Ann Hematol.*, 100(2):465–479, 2021. URL <https://doi.org/10.1007/s00277-020-04387-7>.
- [30] S. Verma, N. Goyal, S. Goyal, P. Kaur, and S. Gupta. EZH2 dysregulation and its oncogenic role in human cancers. *Cancers (Basel)*, 17(19):3111, 2025.
- [31] J. Grabek, J. Straube, M. Bywater, and S. W. Lane. MPN: the molecular drivers of disease initiation, progression and transformation and their effect on treatment. *Cells*, 9(8):1901, 2020.
- [32] Y. Zhao, W. Cai, Y. Hua, X. Yang, and J. Zhou. The biological and clinical consequences of RNA splicing factor U2AF1 mutation in myeloid malignancies. *Cancers (Basel)*, 14(18):4406, 2022.
- [33] J. Grinfeld, J. Nangalia, E. J. Baxter, D. C. Wedge, N. Angelopoulos, R. Cantrill, A. L. Godfrey, E. Papaemmanuil, G. Gundem, et al. Classification and personalized prognosis in myeloproliferative neoplasms. *N Engl J Med.*, 379(15):1416–1430, 2018. URL <https://doi.org/10.1056/NEJMoa1716614>.
- [34] O. Abdel-Wahab and R. L. Levine. Primary myelofibrosis: update on definition, pathogenesis, and treatment. *Annu Rev Med.*, 60: 233–245, 2009. URL <https://doi.org/10.1146/annurev.med.60.041707.160528>.
- [35] D. A. Arber, A. Orazi, R. Hasserjian, J. Thiele, M. J. Borowitz, M. M. Le Beau, C. D. Bloomfield, M. Cazzola, and J. W. Vardiman. The 2016 revision to the World Health Organization classification of myeloid neoplasms and acute leukemia. *Blood.*, 127(20):2391–2405, 2016. URL <https://doi.org/10.1182/blood-2016-03-643544>.
- [36] K. Ghosh, D. K. Shome, B. Kulkarni, M. K. Ghosh, and K. Ghosh. Fibrosis and bone marrow: understanding causation and pathobiology. *J Transl Med*, 21(1):703, 2023.
- [37] Z. Ding, R. Shi, W. Hu, L. Tian, R. Sun, Y. Wu, and X. Zhang. Cancer-associated fibroblasts in hematologic malignancies: elucidating roles and spotlighting therapeutic targets. *Front Oncol*, 13:1193978, 2023. URL <https://doi.org/10.3389/fonc.2023.1193978>.
- [38] M. Chen, C. Zheng, Y. Zhang, J. He, and Z. Shi. Chronic inflammation in primary myelofibrosis: in-depth insights into pathogenesis and promising anti-inflammatory therapeutic strategies. *Mediators Inflamm.*, 2025:9967975, 2025.
- [39] A. Duminuco, A. Nardo, G. Giuffrida, S. Leotta, U. Markovic, C. Giallongo, D. Tibullo, A. Romano, F. Di Raimondo, and G. A. Palumbo. Myelofibrosis and survival prognostic models: a journey between past and future. *J Clin Med*, 12(6):2188, 2023. URL <https://doi.org/10.3390/jcm12062188>.
- [40] R. A. Mesa, S. Verstovsek, and V. Gupta. Symptom burden and quality of life in myelofibrosis. *J Clin Oncol.*, 41(14):2684–93, 2023.
- [41] R. M. Emanuel, A. C. Dueck, H. L. Geyer, J. J. Kiladjian, S. Slot, S. Zweegman, P. A. W. te Boekhorst, S. Commandeur, H. C. Schouten, F. Sackmann, et al. Myeloproliferative neoplasm (MPN) symptom assessment form total symptom score: prospective validation of an abbreviated symptom burden scoring system. *J Clin Oncol.*, 30(33):4098–4103, 2012. URL <https://doi.org/10.1200/JCO.2012.42.3863>.
- [42] I. Amanam, S. Otoukesh, V. Pullarkat, L. Ghoda, S. Forman, G. Marcucci, M. Al Malki, and H. Ali. Myelofibrosis-associated extramedullary hematopoiesis: insights into hepatic, pulmonary, and thrombotic complications. *Blood Rev.*, 75:101355, 2026. URL <https://doi.org/10.1016/j.blre.2025.101355>.
- [43] T. Barbui, A. Ghirardi, A. Carobbio, V. De Stefano, A. Rambaldi, A. Tefferi, and A. M. Vannucchi. Thrombosis in myeloproliferative neoplasms: a viewpoint on its impact on myelofibrosis, mortality, and solid tumors. *Blood Cancer J.*, 14(1):188, 2024. URL <https://doi.org/10.1038/s41408-024-01169-6>.
- [44] M. Khodier and K. Gadó. BCR: ABL1-negative myeloproliferative neoplasms: a review focused on essential thrombocythemia and polycythemia vera. *Physiol Int.*, 110(3):227–50, 2023.
- [45] J. Thiele, H. M. Kvasnicka, U. Gianelli, D. A. Arber, A. Tefferi, A. M. Vannucchi, T. Barbui, and A. Orazi. Evolution of WHO diagnostic criteria in classical myeloproliferative neoplasms compared with the International Consensus Classification. *Blood Cancer J.*, 15(1):31, 2025. URL <https://doi.org/10.1038/s41408-025-01235-7>.
- [46] F. Cervantes, B. Dupriez, A. Pereira, F. Passamonti, J. T. Reilly, E. Morra, A. M. Vannucchi, R. A. Mesa, J. L. Demory, G. Barosi, E. Rumi, and A. Tefferi. New prognostic scoring system for primary myelofibrosis based on a study of the International Working Group for Myelofibrosis Research and Treatment. *Blood.*, 113(13):2895–901, 2009. URL <https://doi.org/10.1182/blood-2008-07-170449>.

- [47] F. Passamonti, F. Cervantes, A. M. Vannucchi, E. Morra, E. Rumi, A. Pereira, P. Guglielmelli, E. Pungolino, M. Caramella, M. Maffioli, C. Pascutto, M. Lazzarino, M. Cazzola, and A. Tefferi. A dynamic prognostic model to predict survival in primary myelofibrosis: a study by the IWG-MRT. *Blood.*, 115(9):1703–1708, 2010. URL <https://doi.org/10.1182/blood-2009-09-245837>.
- [48] N. Gangat, D. Caramazza, R. Vaidya, G. George, K. Begna, S. Schwager, D. V. Dyke, C. Hanson, W. Wu, A. Pardanani, F. Cervantes, F. Passamonti, and A. Tefferi. DIPSS plus: a refined dynamic international prognostic scoring system for primary myelofibrosis that incorporates prognostic information from karyotype, platelet count, and transfusion status. *J Clin Oncol.*, 29(4):392–397, 2011. URL <https://doi.org/10.1200/JCO.2010.32.2446>.
- [49] P. Guglielmelli, T. L. Lasho, G. Rotunno, M. Mudireddy, C. Mannarelli, M. Nicolosi, A. Pacilli, A. Pardanani, E. Rumi, V. Rosti, et al. MIPSS70: mutation-enhanced international prognostic score system for transplantation-age patients with primary myelofibrosis. *J Clin Oncol.*, 36(4):310–318, 2018. URL <https://doi.org/10.1200/JCO.2017.76.4886>.
- [50] A. Tefferi, P. Guglielmelli, T. L. Lasho, M. Mudireddy, N. Bartalucci, C. M. Finke, C. A. Hanson, R. P. Ketterling, K. H. Begna, N. Gangat, A. Pardanani, and A. M. Vannucchi. GIPSS: genetically inspired prognostic scoring system for primary myelofibrosis. *Leukemia*, 32(7):1631–1642, 2018. URL <https://doi.org/10.1038/s41375-018-0107-z>.
- [51] D. T. Kristensen, T. C. El-Galaly, and A. S. Roug. Automated risk stratification in myelofibrosis. *ESMO Real World Data Digit Oncol*, 10:100654, 2025. URL <https://doi.org/10.1016/j.esmow.2025.100654>.
- [52] S. Verstovsek, R. A. Mesa, J. Gotlib, R. S. Levy, V. Gupta, J. F. DiPersio, J. V. Catalano, M. W. N. Deininger, C. B. Miller, et al. Long-term treatment with ruxolitinib for patients with myelofibrosis: 5-year update from COMFORT-I. *J Hematol Oncol.*, 10:55, 2017. URL <https://doi.org/10.1186/s13045-017-0417-z>.
- [53] P. Bose. Momelotinib for the treatment of myelofibrosis. *Blood.*, 144(7):708–13, 2024.
- [54] N. Gangat and A. Tefferi. Emerging pathogenetic mechanisms and new drugs for anemia in myelofibrosis and myelodysplastic syndromes. *Am J Hematol.*, 100(Suppl 4):51–65, 2025. URL <https://doi.org/10.1002/ajh.27659>.
- [55] S. Verstovsek, J. Mascarenhas, and R. A. Mesa. JAK inhibitor therapy in myelofibrosis: current concepts and future directions. *Lancet Haematol.*, 10(11):e845–e857, 2023.
- [56] S. Verstovsek, R. A. Mesa, J. Gotlib, R. S. Levy, V. Gupta, J. F. DiPersio, J. V. Catalano, M. Deininger, C. Miller, et al. A double-blind, placebo-controlled trial of ruxolitinib for myelofibrosis. *N Engl J Med.*, 366(9):799–807, 2012. URL <https://doi.org/10.1056/NEJMoa1110557>.
- [57] C. Harrison, J. J. Kiladjan, H. K. Al-Ali, H. Gisslinger, R. Waltzman, V. Stalbovska, M. McQuitty, D. S. Hunter, R. Levy, L. Knoops, F. Cervantes, A. M. Vannucchi, T. Barbui, and G. Barosi. JAK inhibition with ruxolitinib versus best available therapy for myelofibrosis. *N Engl J Med.*, 366(9):787–798, 2012. URL <https://doi.org/10.1056/NEJMoa1110556>.
- [58] A. Duek, I. Leviatan, O. Jarchowsky Dolberg, and M. H. Ellis. Fedratinib for the treatment of myelofibrosis: a critical appraisal of clinical trial and real-world data. *Blood Cancer J.*, 15(1):6, 2025.
- [59] A. Pardanani, C. Harrison, J. E. Cortes, F. Cervantes, R. A. Mesa, D. Milligan, T. Masszi, E. Mishchenko, E. Jourdan, et al. Safety and efficacy of fedratinib in patients with primary or secondary myelofibrosis: a randomized clinical trial. *JAMA Oncol.*, 1(5):643–651, 2015. URL <https://doi.org/10.1001/jamaoncol.2015.1590>.
- [60] J. Mascarenhas, R. Hoffman, M. Talpaz, A. T. Gerds, B. Stein, V. Gupta, A. Szoke, M. Drummond, A. Pristupa, et al. Pacritinib vs best available therapy, including ruxolitinib, in patients with myelofibrosis: a randomized clinical trial. *JAMA Oncol.*, 4(5):652–659, 2018. URL <https://doi.org/10.1001/jamaoncol.2017.5818>.
- [61] P. A. Dadkhah, M. A. Karimi, M. S. G. Chahkand, F. E. Moallem, M. J. E. Kazemabad, and E. Azarm. Momelotinib in myelofibrosis and beyond: a comprehensive review of therapeutic insights in hematologic malignancies. *Discov Oncol.*, 15(1):370, 2024.
- [62] S. Verstovsek, A. T. Gerds, A. M. Vannucchi, H. K. Al-Ali, D. Lavie, A. T. Kuykendall, S. Grosicki, A. Iurlo, Y. T. Goh, et al. Momelotinib versus danazol in symptomatic patients with anaemia and myelofibrosis (MOMENTUM): results from an international, double-blind, randomised, controlled, phase 3 study. *Lancet.*, 401(10373):269–280, 2023. URL [https://doi.org/10.1016/S0140-6736\(22\)02036-0](https://doi.org/10.1016/S0140-6736(22)02036-0).
- [63] R. A. Mesa, J. J. Kiladjan, J. V. Catalano, T. Devos, M. Egyed, A. Hellmann, D. McLornan, K. Shimoda, E. F. Winton, W. Deng, R. L. Dubowy, J. D. Maltzman, F. Cervantes, and J. Gotlib. SIMPLIFY-1: a phase III randomized trial of momelotinib versus ruxolitinib in Janus kinase inhibitor-naïve patients with myelofibrosis. *J Clin Oncol.*, 35(34):3844–3850, 2017. URL <https://doi.org/10.1200/JCO.2017.73.4418>.
- [64] N. Gaggelmann, C. Wolschke, R. B. Salit, T. Schroeder, M. Ditschkowski, V. Panagiota, B. Cassinat, F. Thol, A. Badbaran, M. Robin, H. C. Reinhardt, F. Ayuk, M. Heuser, B. L. Scott, and N. Kröger. Reduced intensity hematopoietic stem cell transplantation for accelerated-phase myelofibrosis. *Blood Adv.*, 6(4):1222–1231, 2022. URL <https://doi.org/10.1182/bloodadvances.2021006827>.
- [65] A. T. Gerds, J. J. Kiladjan, and R. A. Mesa. Emerging therapeutic targets in myelofibrosis. *Leukemia.*, 37(7):1360–72, 2023.
- [66] J. Mascarenhas, M. Kremyanskaya, A. Patriarca, F. Palandri, T. Devos, F. Passamonti, R. K. Rampal, A. J. Mead, G. Hobbs, J. M. Scandura, et al. MANIFEST: pelabresib in combination with ruxolitinib for Janus kinase inhibitor treatment-naïve myelofibrosis. *J Clin Oncol.*, 41(32):4993–5004, 2023. URL <https://doi.org/10.1200/jco.22.01972>.

- [67] N. Pemmaraju, A. J. Mead, T. C. P. Somervaille, F. Palandri, S. Koschmieder, R. Delage, J. Kiladjan, C. Benton, S. K. Tantravahi, N. Lavi, et al. TRANSFORM-1 phase 3 study: efficacy and safety of navitoclax plus ruxolitinib in patients with untreated myelofibrosis. *Blood*, 148(16):2029–2042, 2026. URL <https://doi.org/10.1182/blood.2025032360>.
- [68] J. Mascarenhas, M. Kremyanskaya, A. Patriarca, F. Palandri, T. Devos, F. Passamonti, R. K. Rampal, A. J. Mead, G. Hobbs, J. M. Scandura, et al. MANIFEST: pelabresib in combination with ruxolitinib for Janus kinase inhibitor treatment-naïve myelofibrosis. *J Clin Oncol.*, 41(32):4993–5004, 2023. URL <https://doi.org/10.1200/JCO.22.01972>.
- [69] J. Mascarenhas, R. S. Komrokji, F. Palandri, B. Martino, D. Niederwieser, A. Reiter, B. L. Scott, M. R. Baer, R. Hoffman, O. Odenike, et al. Randomized, single-blind, multicenter phase II study of two doses of imetelstat in relapsed or refractory myelofibrosis. *J Clin Oncol.*, 39(26):2881–2892, 2021. URL <https://doi.org/10.1200/JCO.20.02864>.
- [70] N. Pemmaraju, J. S. Garcia, J. Potluri, J. G. Harb, Y. Sun, P. Jung, Q. Q. Qin, S. K. Tantravahi, S. Verstovsek, and C. Harrison. Addition of navitoclax to ongoing ruxolitinib treatment in patients with myelofibrosis (REFINE): a post-hoc analysis of molecular biomarkers in a phase 2 study. *Lancet Haematol.*, 9(6):e434–e444, 2022. URL [https://doi.org/10.1016/S2352-3026\(22\)00116-8](https://doi.org/10.1016/S2352-3026(22)00116-8).
- [71] A. M. Vannucchi and P. Guglielmelli. JAK2 mutation-related disease and clinical phenotype in myeloproliferative neoplasms. *Hematology Am Soc Hematol Educ Program*, 1:480–488, 2017. URL <https://doi.org/10.1182/asheducation-2017.1.480>.
- [72] G. G. Loscocco, G. Rotunno, F. Mannarelli, G. Coltro, F. Gesullo, F. Pancani, L. Signori, C. Maccari, M. Esposito, C. Paoli, A. M. Vannucchi, and P. Guglielmelli. The prognostic contribution of CBL, NRAS, KRAS, RUNX1, and TP53 mutations to mutation-enhanced international prognostic score systems for primary myelofibrosis. *Am J Hematol.*, 99(1):68–78, 2024. URL <https://doi.org/10.1002/ajh.27136>.
- [73] I. Plo and W. Vainchenker. Myeloproliferative neoplasms. *N Engl J Med.*, 395(8):788–802, 2026. URL <https://doi.org/10.1056/NEJMra2507867>.
- [74] R. Shao, C. Ryder, L. Wang, H. Zhang, L. Moscinski, M. Martin, M. Shebes, J. Y. Li, and J. Song. A review of the pathological and molecular diagnosis of primary myelofibrosis. *Cancers (Basel)*, 18(1):50, 2026. URL <https://doi.org/10.3390/cancers18010050>.
- [75] A. Tefferi, N. Gangat, T. L. Lasho, Y. Elala, A. Pardanani, K. H. Begna, C. A. Hanson, R. P. Ketterling, and A. Pardanani. Revised cytogenetic risk stratification in primary myelofibrosis: analysis based on 1002 informative patients. *Leukemia*, 32(5):1189–1199, 2018. URL <https://doi.org/10.1038/s41375-018-0018-z>.