

High Protein, Low Carbohydrate, High Non-Trans Fat, and Decitabine for Survival-ITP and LGC Leukemia

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Abstract—

Introduction: Therapy of TP53, mutation of MSD/AML with Niclosamide plus Azacitidine, is the end-stage phase of ITP/LGC Leukemia. The Constant therapy is unbroken with CHOP or R-CHOP as the second line therapy, plus nutrition HPLCHF, and decitabine ever since the early phase which should be given. Aims: No transfusion should be held since this early phase. Cytokine storm is the inducer of this advancement. Hypothesis: High Protein Low Carbohydrate High Fat (HPLCHF) and decitabine without transfusion should be the winner for survival ITP/LGC. Liquid therapy and aspirin are the first line symptomatic during the cytokine storm phase. Two dead cases of Large Granular Chronic Leukemia support this study.

Method: Hybrid My library and ChatGPT based, with academic search engine using Science Direct and EBSCOHost MEDLINE full text using keywords ITP/MDS/AML, and therapy, and case reports.

Result: Variables drugs is recorded such as pro-apoptotic agent Venetoclax a Bcl2- inhibitor, Fostamatinib a spleen tyrosine Kinase inhibitor, decitabine/azacitidine hypomethylation agent, CHOP (chemotherapy with prednisone) and splenectomy are given in the thrombocytopenia last moment, with MOD end-stage mortality less than 5 days due Multi/X/Total Antibiotic Resistance. Low carbohydrate, high-fat, high protein should be given in this early prediction low albumin level of severe thrombocytopenia in Dengue Lymphoma Malignant Fever (DFLM).

Discussion: Pathogenesis ITP, MDS, AML therapy in all phases, and the recorded mutation genetic/epigenetic, and immune landscape of TP53 and other mutant AML & higher risk MDS treated with azacitidine/decitabine.

Conclusion: HPLCHF nutrition and decitabine ever since the early phase should be given to support the body to cure themselves.

Keywords— ITP; Splenectomy; Prednisone; Low carbohydrate; often Protein nutrition; Decitabine.

I. INTRODUCTION

Anemia Immune Thrombocytopenia (ITP) a.k.a. Previously Idiopathic Thrombocytopenia Purpura progressed to MDS/AML or is a Life-threatening situation “bridge therapy” to surgery or invasive procedure,¹ splenectomy and or chemotherapy. ITP due to Defective Peripheral Immune Response often has high prevalence causes of this disease in our everyday practice.¹ ITP turns to MDS which increases bone marrow increasing of CD 34-blast, and CD16-granulocytes,² then progresses to AML. Germline genetic (and epigenetic) have been recorded in MDS and AML traditional outpatients.³ AML is an aggressive hematological malignancy with a higher incidence in older people.⁴ Clonal hematopoiesis (Ch) is a common premalignant state in the blood and confers an increased risk of blood cancers and all-cause mortality.⁵ Variables drugs are recorded such as pro-

apoptotic agent Venetoclax a Bcl2- inhibitor,^{4,6,7} Fostamatinib a spleen tyrosine Kinase inhibitor,⁸ decitabine/azacitidine hypomethylation agent,^{7,9,10} due to R-loop dysregulation and resultant genomic instability in the disease progression of MDS & AML,¹⁰ a therapy strategy for Myeloid cancers.¹⁰ Erythropoietin agents¹¹ and/or with RBC transfusion,¹² which reported as a cause of first hospitalization due the decreasing hemoglobin which drops common everyday Quality of Life (QL).¹¹ Daratumumab in refractory autoimmune cytopenia.¹³ Plasmapheresis (plasma exchange) for inflammation cytokine as the pathogenesis of MDS is yet to be fully established.^{12,14}

Novel Anti-CD38 monoclonal antibody for treating ITP,¹⁵ Notable treatment advances have been made for patients with MDS/ neoplasm to live longer and better for what will likely remain a largely incurable disease,¹⁶ describe the failure to complete recovery. This study brings to parsimony and calm reaction therapy like in fluidic only therapy on DHF in 16.000 platelet count is usually successful.^(Case Report)

II. METHOD

Review and case report of therapy in all phases of ITP and LGC using hybrid recommendation of My library Google Scholar and Academic Search engine i.e., ScienceDirect and EBSCOHost MEDLINE full text, and ChatGPT. This hybrid Search used keywords ITP/MDS/ATP and therapy. Bayesian network and analysis are being used in including and excluding the references.

III. RESULTS

This study also recorded GvHDs therapy which does not respond to standard Systemic steroids which is given Ruxolitinib (JAK1 inhibitor for MS)¹⁷ and Fostamatinib for curing thrombocytopenia got successful tapering and discontinuation of corticosteroid while maintaining platelet count above 50,000/uL.¹⁸ Avatrombopag (a thrombopoietin receptor agonist) plus fostamatinib combination with multifactorial ITP is also recorded.¹⁹ Patients with thrombocytopenia usually do not experience serious bleeding until their platelet count is very low.

TABLE 1
RECORDED THERAPY IN ITP/MDS/AML, PHARMACOLOGY FUNCTION, PROGNOSIS

Studies, years	Phase	Agents/Surgery	Pharmacology function combat	Prognosis
¹ Gonzalez-Lopez, 2023	Refractory ITP	Chemotherapy/Splenectomy	Splenomegaly	MOD sepsis
¹⁸ Goel R, 2024	ITP	Standard Systemic Steroid (SSS) + Fostamatinib	Antiinflammation	Successful tapering
¹⁷ Teshima T, 2024	Acute GvHD	Ruxolitinib as MS inhibitor	JAK1 inhibitor	Respond to SSS again
²⁰ He Q, 2024	cGv-HD	SHR0302 + prednisone	JAK1 Inhibitor	1 st line Rx/
¹⁸ Goel R & ⁸ Gonzalez-Lopez 2024	ITP	Fostamatinib	Spleen TKI	High efficacy rate
³ Banaszaky LG, 2024	MDS/AML	Driven Germline genetic/epigenetic	Traditional out patients	Genetic counseling
¹⁵ Chen Y, 2024	ITP	Anti-CD38	Anti-cancer monoclonal antibody	Boosted platelet level
¹³ Hu U, 2024	Refractory autoimmune cytopenia	Daratumumab	Anti-CD38 monoclonal antibody	Effective Rx/
⁵ Waarts MR, 2024	Premalignant state	CRISPR/Cas9	Editing	Identify genetic dependencies in mutant HSPCs
⁴ Salamero O, 2020 ⁶ Wang D, 2024 ⁷ Kobayashi, 2024	AML	Venetoclax pro-apoptotic agent	A Bcl2-inhibitor	Phase I oke; Inhibit entering mitotic phase; Complete remission

⁹ Enjeti A, 2024	Genomic instability ITP/MDS/AML	Decitabine/azacitidine hypomethylation agent,	Gene silencing	The most MDS Rx/
¹¹ Battaglia MR, 2024	Hemoglobin drops until disturb everyday QL common living	EPO agents RBC transfusion	Fit for everyday living	LR-MDS std Rx/ in the future
¹² Topping J, 2024	Cytokine storm (CS) pre- MDS	Plasmapheresis	Decreasing inflammation cytokine	For Dx/ and Px/ CS to first RBC transfusion
²¹ Gangat N, 2024	Essensial thrombocytopenia in JAK2 and CALK- mutated	Aspirin	Anti-inflammation Anti-cytokine storm	with a lower pregnancy loss
¹⁰ Zhang F, 2024	Splicing factors are frequently mutated in pts. with MDS&AML	induce to anemia and ITP progress to MDS/AML	Rx/ strategy for Myeloid Ca with mutations in SRSF2	R-loop disruption by PRAP activation
²² Rathje, 2024	GvHD acute & chronic	Anti-T-Lymphocyte Globulin (ATLG)	Inflammation and immune reaction	Lower rates of severe cGvHD
²³ Salma RS, 2018	Non-Hodgkin Lymphoma high prevalence	CHOP and R-CHOP	Responses	Still a challenge (partial remission)
²⁴ Zeidan AM, 2024	TP53 mutant AML and higher risk MDS	Azacitidine	Hypomethylation agents	Integrated Rx/
²⁵ Maslah N, 2024	MSD/AML	Niclosamide plus Azacitidine	TP53 mutation + hypomethylation agents	Niclosamide is an anthelmintic
²⁶ Kammerer, 2021	Cancer	HPLCHF	Decrease body fat	Lifestyle diet

Cancer cells, MDS, and ITP consume glucose at a higher rate and induce insulin resistance. Anemia and hypoglycemia are found in these thrombocytopenia patients. Dextrose infusions on the day or first hospitalization due to Quality of Life (QL) to get transfusion is also a common symptomatic treatment, where the next reaction is reported. Stress and its treatments can cause the body to release various hormones that increase blood sugar. This can weaken the immune system, and the fear of hypoglycemia makes the patients drink glucose by feeling the drop of QL. Cauda pancreas pancreatectomy with splenectomy has been the case reported. Glucose can be supplied from gluconeogenesis, protein synthesis disrupts from many viral infections, and could not do apoptosis. Protein should be supported by external supply the whole day for everyday QL. High-fat diet without saying how high the fat is, refers to the popular diet ketogenic which is 75% fat, 20 % protein, and 5% carbohydrate. HPLCHF is a lifestyle diet to drop bodyweight successfully. Also, the well-known trans fats which is the worst for health, e.g. shortening and margarine.

Therapy of TP53 mutation of MSD/AML with Niclosamide plus Azacitidine,²⁵ is the end-stage phase of ITP/LGC. The Constant therapy is unbroken with CHOP or R-CHOP²³ as the second line therapy, plus nutrition HPLCHF and decitabine ever since the early phase, which should be given. Supportive care in lower-risk MDS with anemia ITP is also rapidly evolving.¹¹

IV. DISCUSSION

Immune Thrombocytopenia (ITP) a.k.a. previous well-known Idiopathic Thrombocytopenia Purpura, is an Autoimmune bleeding/without bleeding or fibrous disorder characterized by immoderate reticuloendothelial platelet (Mononuclear Phagocytic System) destruction & inadequate compensatory platelet production.²⁷ In all phases, ITP needs specific metabolism so that becomes MDS (Myelodysplastic Syndrome)^{2,12} or myelofibrosis.²⁸ In the next advanced progress when the body cannot recover, MDS transforms to AML.^{1,2,27}

The aims of this study record the success and the failure of therapy in finding references in association with complete recovery, not refractory ITP/MDS/AML. This study discusses each therapy as follows:

4.1 Transfusion, like Graft versus Host Disease (GvHDs):

To suppress the immune system reaction, steroids are needed. The reaction needs immunosuppressive drugs, CHOPs, antifungal, antiviral, and antibiotics. Thrombocyte donors attack recipients' cells, also RBC, etc. The etiology of GvHDs is the cytokine storm reaction. The mutated TP53 and other mutations, block apoptosis, and decrease JAK1 & 2, a group of kinases expressed on many cells, surfaces that mediate cytokine signaling.^{20,29} One-third of patients with Acute GvHD are resistant to standard systemic steroids, and need second-line approach such as JAK1 and 2 inhibitors,¹⁷ anti-T Lymphocyte globulin.²² Aspirin therapy is associated with a lower pregnancy loss in both JAK2- and CALK-mutated essential thrombocytemia.²¹ Complete response ITP are recorded with prednisolone.³⁰

4.2 Luspatercept & ESA (erythropoietin stimulating agents)¹¹:

This EPO agent is better than Transfusion and is given since the patients need to be hospitalized for the drop of hemoglobin which decreases Quality of Life. Luspatercept in these with SF3B1 mutations has an EPO level < 500 U/L.

4.3 Lenalidomide:

Lenalidomide, is T Immunosuppressive therapy in del 5q syndrome, has both efficacy & durability of response.¹¹ Lenalidomide is an immunomodulator.

4.4 Imetelstat:

Telomerase inhibitor is though in the phase III trial support, may become a standard option¹¹. Imetelstat binds to the telomerase RNA component with high affinity, directly inhibiting telomerase activity.

4.5 Bone Marrow Transplantation:

Bone Marrow Transplantation using ATLG (anti-T Lymphocyte Globulin) depletion.

4.6 Supportive^{11,31}:

The gut microbiome in ITP affects health through human metabolism, immune modulation, and maintaining physiological,²⁷ while older people who cannot make protein anymore have a higher incidence of progressing to AML.⁴

4.7 Ruxolitinib³² and Fostamatinib⁸:

Ruxolitinib, inhibits JAK1 & 2, Belumosudil inhibits ROCK2, Ibrutinib inhibits BTK in Systemic Sclerosis (SSc) save for treatment of chronic GvHD (cGvHD)²⁹ Ruxolitinib in JAK1 inhibitor-naïve myelofibrosis.²⁸ Gilteritinib with/without Venetoclax improved outcomes when sequenced early for salvage.³³ JAK1 Inhibitor SHRO302 + prednisone for first-line treatment of cGvHD may have less effect on hematopoiesis.²⁰ Rx/ Fostamatinib in Thrombocytopenia give successful tapering & discontinuation of corticosteroids while maintaining platelet count above 50,000/uL.¹⁷

4.8 Selinexor²⁸:

Selinexor causes cell cycle arrest and apoptosis in cancer cells. Selinexor is a selective inhibitor of nuclear export, an oral drug that prevents access to the nucleo-cytoplasmic transport of nuclear protein in JAK/STAT and non-JAK/STAT pathway. Selinexor induces nuclear localization of tumor suppressor proteins (incl. p53), be a route to the selective induction of apoptosis, and inhibits the performance of DNA damage repair protein.³⁴

V. LIMITATION

The integrated genetic, epigenetic, and immune landscape of TP53 mutant AML and higher-risk MDS give medical care with azacitidine, has been talked of in Therapeutic Advance in Hematology, based on TP53 mutation MDS and AML,²⁴ which is Rx/ Niclosamide + Azacitidine has successfully totally recovered the sensitivity of TP53-mutated cells to hypomethylating agents. It's targeting TP53-mutated MDS/AML cells.²⁵ Inflammation reaction and immune reaction induce anemia, and ITP progresses to MDS/AML.¹² Inflammation storm and immune reaction in GvHD-free and relapse-free survival,²² has not been elaborated specifically. Splicing factors are frequently mutated in ITP patients which induces anemia and MDS/AML progression.¹⁰ Anti-CD38 monoclonal antibody has been used as a prognostic marker in leukemia: Daratumumab is effective in refractory cases.¹³ This study has been successful in the use of hypomethylation agents in all phases, but still has limitations in building deductive and inductive protein production for RBC, platelet, and antibody/anti-cytokine storm.

VI. CONCLUSION

HPLCHF nutrition and decitabine ever since the early phase should be given to support the body to cure themselves.

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CONFLICT OF INTEREST

The author declares no conflict of interest in any insights.

REFERENCES

- [1] Gonzalez-Lopez TJ, Provan D, Barez A, Bernardo-Gutierrez A, Bernat S, Martinez-Carballeira DM, et al. Primary and secondary immune thrombocytopenia (ITP): Time for a rethink. *Blood Reviews*, 2023; 61:101112. <https://doi.org/10.1016/j.bire.2023.101112>
- [2] Azoulay D, Paz A, Stemer G. Bone marrow S-phase in Associated with Risk assessment and Shows Differential Correlation with levels of CD34+ and CD16 in Patients with Myelodysplastic Syndrome. *MedRxIV* 2024;D6. Preprint and has not been peer-reviewed. <https://doi.org/10.1101/2024.06.06.24308422>
- [3] Banaszak LG, Cabral PL, Smith-Simmer K, Hassan A, Brunner M, Fallon M, et al. Implementation of and Systems-Level Barriers to Guideline-Driven Germline Genetic Evaluation in the Care of Patients With Myelodysplastic Syndrome and Acute Myeloid Leukemia. *JCO Precision Oncol* 2024; June:8:e2300518. <https://doi.org/10.1200/PO.23.00518>
- [4] Salamero O, Somerville TCP, Molero A, Acuna E, Perez A, Cano I, et al. 1916 Robust Efficacy Signals in Elderly AML Patients Treated with Iadademstat in Combination with Azacitidine (ALICE Phase IIa Trial). (The Lancet Haematology 2024) 62nd ASH Annual Meeting and Exposition 2020: Dec 5-8.
- [5] Waarts MR, Mowla S, Boileau M, Benitez ARM, Sango J, Bagish M. CRISPR Dependency Screens in Primary Hematopoietic Stem Cells Identify KDM3B as a Genotype Specific Vulnerability in IDH2-and TET2-Mutant Cells. *Cancer Discovery* 2024; <https://doi.org/10.1158/2159-8290.CD-23-1092>
- [6] Wang D, He J, Liu S, Zhang H, Tang D, Chen P, Yang M. Anlotinib synergizes with venetoclax to induce mitotic catastrophe in acute myeloid leukemia. *Cancer Letters* 2024; Jul 1;593:216970. <https://doi.org/10.1016/j.canlet.2024.216970>
- [7] Kobayashi T, Sato H, Fukushi Y, Kuroki W, Ito F, Teshima K, Watanabe A, Fu...N. Overexposure to venetoclax is associated with prolonged-duration of neutropenia during venetoclax and azacitidine therapy in Japanese patients with acute myeloid leukemia. *Cancer Chemotherapy and Pharmacology* 2024 May 23. <https://doi.org/10.1007/s00280-024-04673-5>
- [8] Gonzalez-Lopez TJ, Bermejo N, Cardesa-Cabrera R, Martinez V, Aguilar-Monserrate G, Segura GP, et al. Fostamatinib effectiveness and safety for immune thrombocytopenia in clinical practice. *Blood* 2024;2024024250 <https://doi.org/10.1182/blood.2024024250>
- [9] Enjeti A, Ashraf A, Caillet V, Alam S, Silar J, Keer H, et al. Real-world study of the use of azacitidine in myelodysplasia in Australia. *eJHaem* 2024;1-8. <https://doi.org/10.1002/jha.2.911>
- [10] Zhang F, Sun J, Zhang L, Li R, Wang Y, Geng H, Shen C, et al. PARP Inhibition leads to synthetic lethality with key -factor mutations in myelodysplastic syndromes. *Br J of Cancer* 2024. <https://doi.org/10.1038/s41416-024-02729-0>
- [11] Battaglia MR, Cannova J, Madero-Marroquin R, Patel AA. Treatment of Anemia in Lower-Risk Myelodysplastic Syndrome. *Curr Treat Options, Oncol* 2024 May 30. <https://doi.org/10.1007/s11864-024-01217-0>
- [12] Topping J, Taylor A, Nadat F, Crouch S, Ibbostson A, Cermak J, Symeonidis A, Tatic A. Inflammatory profile of lower risk myelodysplastic syndrome. *Br J Haematol* 2024 May 21. <https://doi.org/10.1111/bjh.19530>
- [13] Hu Y, Wang Z, Ma J, Wang N, Meng J, Dong S, et al. The early and rapid response to daratumumab in children with chronic refractory immune thrombocytopenia from a referral single centre of China. *Br J Haematology* 2024, 03 Jun; <https://doi.org/10.1111/bjh.19553>
- [14] Picod A, Provot F, Coppo P. Therapeutic plasma exchange in thrombotic thrombocytopenic purpura. *La Presse Medicale* 2019;Nov;48(11)part 2: 319-327. <https://doi.org/10.1016/j.lpm.2019.08.024>
- [15] Chen Y, Xu Y, Li H, Sun T, Cao X, Wang Y, et al. A Novel Anti-CD38 Monoclonal Antibody for Treating Immune Thrombocytopenia. *New England J Med* 2024;390(23):2178-90 <https://doi.org/10.1056/NEJMoa2400409>
- [16] Efficace F, Buckstein R, Abel GA, Giesinger JM, Fenaux P, Bewersdorf JP, et al (23). Toward a more patient-centered drug development process in clinical trials for patients with myelodysplastic syndrome/neoplasms (MDS): Practical considerations from the International Consortium for MDS (icMDS). *HemaSphere* 2024 May;8(5): e69. <https://doi.org/10.1002/hem3.69>
- [17] Teshima T, Onishi Y, Kato K, Taniguchi S, Miyamura L, Fukushima K, et al. Ruxolitinib in steroid-refractory acute graft-vs-host disease. Japanese subgroup analysis of the randomized REACH2 trial. *Int J Hematol* 2024 May 25. <https://doi.org/10.1007/s12185-024-13772-6>
- [18] Goel R, Azhar W, Numerof RP, Chow D, and Shah B. Real-world experience with fostamatinib in patients with immune thrombocytopenia: Results of an observational study (FORTE). *J Clin Oncol* 42(16 suppl). https://doi.org/10.1200/JCO.2024.42.16_suppl.e23289
- [19] Mingot-Castellano ME, Bastida JM, Ghanima W, Sainz ER, Vazquez RN, et al. Avatrombopag plus fostamatinib combination as treatment in patients with multifactorial immune thrombocytopenia. *Br JHaem* 2024, Jun 19. <https://doi.org/10.1111/bjh.19602>

- [20] He Q, Sun X, Niu J, Yang J, Wang Y, Huang C, et al. A Novel JAK1 Inhibitor SHR0302 Combined With Prednisone for First-Line Treatment of Chronic Graft-Versus-Host Disease: A Phase I Clinical Trial. *Cell Transplantation* 2024; May 26; <https://doi.org/10.1177/09636897241254678>
- [21] Gangat N, Singh A, Ilyas R, Loscocco GG, Elliot M, Begna K, et al. Aspirin therapy is associated with a lower risk of pregnancy loss in both JAK2 and CALR-mutated essential thrombocythemia-A Mayo Clinic study of 200 pregnancies. *Am J Hematol* 2024, Jun 12. <https://doi.org/10.1002/ajh.27416>
- [22] Rathje K, Gagelmann N, Salit RB, Schroeder T, Gurnari C, Pagliuca S, et al (19). Anti-T-lymphocyte globulin improves GvHD-free and relapse-free survival in myelofibrosis after matched related or unrelated donor transplantation. *Bone Marrow Transplant* 2024 May 21. <https://doi.org/10.1038/s41409-024-02291-6>
- [23] Salma RS, Sedana MP, Yudho SU. CHOP and R-CHOP Therapeutic Responses in Non-Hodgkin Lymphoma Patients in Dr. Soetomo General Hospital Surabaya. *Biomol and Health Sc J* 2018;1(2). <https://doi.org/10.20473/bhsj.v1i2.9244>
- [24] Zeidan AM, Bewersdorf JP, Hasle V, Shallis RM, Thompson E, et al. Integrated genetic, epigenetic, and immune landscape of TP53 mutant AML and higher risk MDS treated with azacitidine. <https://doi.org/10.1177/20406207241237904>
- [25] Maslah N, Rety S, Bonnamy M, Aguinaga L, Huynh T, et al. Niclosamide combined to Azacitidine to target TP53-mutated MDS/AML cells. *Leukemia*, 2024. <https://doi.org/10.1038/s41375-024-02281-z>
- [26] Kammerer U, Klement RJ, Joos FT, Sutterlin M, and Reuss-Borst M. Low Carb and Ketonic Diets Increase Quality of Life, Physical Performance, Body Composition, and Metabolic Health of Women with Breast Cancer. *Nutrients* 2021 Mar;13(3): 1029. <https://doi.org/10.3390/nu13031029>
- [27] Saki N, Hadi H, Keikhaei B, Mirzaei A, Purrahman D. Gut microbiome composition and dysbiosis in immune thrombocytopenia: A review of literature. *Blood Review* 2024, Jun 6:: 101219. <https://doi.org/10.1016/j.blre.2024.101219>
- [28] Mascarenhas J, Harrison C, Schuler TA, Liassou D, Garretson M, Miller TA, et al. Real-World Use of Fedratinib for Myelofibrosis Following Prior Ruxolitinib Failure: Patient Characteristics, Treatment Patterns, and Clinical Outcomes. *Clin Lymphoma Myeloma Leuk* 2024;24(2):122-32. <https://doi.org/10.1016/j.clml.2023.09.008>
- [29] Hong C, Jin R, Dai X, Gao X. Functional Contributions of Antigen Presenting Cells in Chronic Graft-Versus-Host Disease. *Front Immunol* 2021, Feb 24;12. <https://doi.org/10.3389/fimmu.2021.6114183>
- [30] Beyene DB, Sisay EA, Fentie AM, Gebremedhin A. Treatment outcomes and adherence to treatment in patients with immune thrombocytopenia in two Ethiopian teaching hospitals: a retrospective cohort study. *Sci Rep* 2024;14(1): 11917. <https://doi.org/10.1038/s41598-024-62372-w>
- [31] Barak G, Demmler-Harrison G, Rossetti L, Tubman VN, Walimbe AS, Asaithambi R, et al. Progressive Thrombocytopenia, Splenomegaly, and Abnormal Tone in an Infant with Growth Faltering. *Pediatrics* 2024, Jul 1;154(1):e2023064048. <https://doi.org/10.1542/peds.2023-064048>
- [32] Guglielmelli P, Mora B, Gesullo F, Mannelli F, Loscocco GG, Signori L. Clinical impact of mutated JAK2 allele burden reduction in polycythemia vera and thrombocythemia. *Am J of Hematology* 2024; 06 June. <https://doi.org/10.1002/ajh.27400>
- [33] Kugler E, Cohen I, Amitai I, Ram R, Frisch A, Nachmias B et al. Gilteritinib with or without venetoclax for relapsed/refractory FLT3-mutated acute myeloid leukemia. *B J Haem* 2024, May 23. <https://doi.org/10.1111/bjh.19548>
- [34] Bogani G, Monk BJ, Coleman RL, Vergote I, Oakin A, Rau-Coquard I, et al. Selenixor in patients with advanced and recurrent endometrial cancer. *Curr Probl Cancer* 2023, Dec;47(6):100963. <https://doi.org/10.1016/j.currproblcancer.2023.100963>.