median OS for non-AML M3 patients was 28±%17.2 months (95% CI: 0.00-61.8) (p=0.002). Survival duration was shorter in IR and HR groups than the SR group.Cumulative survival rates at 1 and 5 years were significantly longer in AML-M3 patients than non-AML M3 patients. For AML-M3, cumulative survival was 94%±%6 at both time points. In non-AML M3 patients, 1-year and 5-year cumulative survival rates were 73% and 43%, respectively (p = 0.002). In relapsed patients, median OS after relapse was 3±0.8 months (95% CI:1.23-4.76) (p=0.000). No significant difference in survival rates was observed between the AML-BFM 2004 and AML-BFM 2012 protocols. **Conclusion:** In the present study, key factors influencing survival in pediatric AML included risk group, age at diagnosis, induction response at diagnosis, and time-to-relapse. Among relapsed patients, the initial risk group also affected survival. Leukapheresis had no impact on survival. Mortality remains high in non-AML M3 cases. Further research is required to develop genetically defined treatment subgroups in pediatric AML. We recommend more stringent risk stratification for IR patients under the AML-BFM 2012 protocol, and advocate for larger studies aimed at creating standardized, personalized treatment protocols for all patients.

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OP 02

TP53-Deleted Mixed Phenotype Acute Leukemia with Widespread Nodal Disease: Complete Remission after HyperCVAD plus Azacitidine

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Introduction: Mixed phenotype acute leukemia (MPAL) is rare and clinically aggressive, particularly when accompanied by TP53 deletion and complex karyotype. Nodal presentations can mimic lymphoma, delaying definitive therapy. We report a young woman with MPAL (B/Myeloid) and extensive nodal involvement who achieved complete remission (CR) with HyperCVAD plus azacitidine. **Methods:** Single-patient case review of prospectively collected data. Diagnostic work-up included complete blood counts, bone marrow (BM) aspirate/biopsy with immunohistochemistry (IHC), multiparameter flow cytometry, cytogenetics/FISH, PCR panel for recurrent fusions, and FDG PET-CT. Treatment consisted of HyperCVAD combined with azacitidine. Response was assessed morphologically, by PET-CT, and by minimal residual disease (MRD) testing. **Results:** A 37-year-old woman presented with fatigue, bilateral cervical and axillary lymphadenopathy, and pancytopenia. BM was normo−to-hypersellular (cellularity ∼50 −65%) with blast proliferation; reticulin 0−1/4. IHC showed CD34+, CD117+, CD33+, heterogeneous CD3 and rare TdT; PAX5 was positive in marrow sections, while CD20, MPO, and CD13 were negative. Excisional axillary-node pathology revealed blast infiltration (CD34+, CD117+, CD33+, CD3+, CD5+, CD10+, BCL2+, Ki-67 ∼30%; PAX5 and MPO negative), supporting leukemic involvement. Flow cytometry identified a 53% blast population expressing CD33, HLA-DR, and aberrant CD7, negative for CD19, CD10, surface CD3, and MPO—consistent with **MPAL (B/Myeloid)** in the aggregate clinicopathologic context.Cytogenetics demonstrated **complex hyperdiploidy (85−92 chromosomes)** with **trisomy 8** and **tetrasomy 10**; FISH detected **TP53 (17p) deletion**. TEL/AML1, PML/RARA, BCR/ABL, AML/ETO were negative by FISH; PCR for BCR-ABL, PML-RARA, and FLT3 was negative. Baseline PET-CT showed **widespread FDG-avid nodal disease** (cervical, axillary, mediastinal, abdominal, retroperitoneal; SUVmax ∼4 −10) without visceral uptake. First-line **HyperCVAD plus azacitidine** was administered with standard supportive care. End-of-treatment evaluation demonstrated **morphologic CR**, **MRD negativity**, and **metabolic complete response** by PET-CT. The patient remained in remission on early surveillance. **Discussion:** This case highlights three practice points. (1) **Nodal MPAL can masquerade as lymphoma**; integrated BM, node histology, flow, and molecular profiling are essential to prevent misclassification and treatment delay. (2) **TP53 deletion with complex karyotype** portends high risk; nonetheless, **HyperCVAD plus azacitidine** achieved deep response, suggesting potential synergy of epigenetic priming with intensive chemotherapy in adverse-genetic MPAL. (3) Discordant lineage signals (e.g., PAX5 IHC positivity with B-lineage markers absent on flow, and MPO negativity despite myeloid antigen expression) illustrate real-world diagnostic ambiguity in MPAL and the need to rely on the totality of evidence rather than any single assay. **Conclusion:** In TP53-deleted, complex-karyotype **MPAL with extensive nodal disease**, **HyperCVAD plus azacitidine** induced **MRD-negative CR** with metabolic clearance. This experience supports considering epigenetic-augmented intensive regimens in high-risk MPAL and underscores the diagnostic value of coordinated marrow−node evaluation.

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OP 03

Dasatinib-Induced Progressive Enterocolitis Mimicking Inflammatory Bowel Disease in a Patient with Chronic Myeloid Leukemia: A Case Report

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Introduction: Dasatinib is a potent second-generation tyrosine kinase inhibitor widely used in chronic myeloid leukemia (CML) treatment, particularly in patients intolerant to imatinib. While generally well-tolerated, dasatinib can cause various adverse effects including pleural effusions, cytopenias, and gastrointestinal symptoms. However, progressive enterocolitis resembling inflammatory bowel disease (IBD) is rarely reported and poses diagnostic challenges due to clinical and endoscopic similarities to IBD. **Case Report:** A 71-year-old female with a 20-year history of achalasia was diagnosed with chronic myeloid leukemia in 2021 following evaluation

for leukocytosis and typical hematological findings. Initial treatment with imatinib 400 mg daily was discontinued due to severe facial edema. Subsequently, dasatinib 100 mg daily was initiated as second-line therapy. Concurrent with dasatinib initiation, the patient developed new gastrointestinal symptoms previously absent in her medical history, including abdominal pain, intermittent diarrhea, altered bowel habits, and occasional hematochezia. These symptoms progressively worsened over subsequent years despite achieving hematological remission. Physical examination in 2021 revealed stable vital signs with mild diffuse abdominal tenderness without hepatosplenomegaly. Laboratory investigations confirmed BCR-ABL positivity establishing CML diagnosis, with leukocytosis (WBC >50,000/μL) and normal renal and hepatic function. Hematological remission was maintained throughout 2022-2025 follow-up period. Colonoscopy performed in February 2022 revealed minimal terminal ileal hyperemia with edematous and granular colonic mucosa, raising suspicion for ulcerative colitis or Crohn's disease. Histopathological examination of biopsies showed chronic active colitis with cryptitis, terminal ileitis, and eosinophilic infiltration, but lacked granulomas or specific features diagnostic of IBD. Previous biopsies from November 2021 demonstrated similar chronic active colitis and cryptitis without diagnostic specificity. Despite endoscopic findings suggestive of IBD, the absence of characteristic histopathological features and progressive symptom worsening during dasatinib therapy raised suspicion for drug-induced enterocolitis. In 2025, when gastrointestinal symptoms significantly intensified, dasatinib was discontinued. Remarkably, within approximately two months of dasatinib discontinuation, all gastrointestinal symptoms completely resolved, providing strong evidence for drug-induced etiology rather than IBD. **Discussion:** This case demonstrates a rare but clinically significant adverse effect of dasatinib therapy. While gastrointestinal symptoms are recognized side effects of tyrosine kinase inhibitors, progressive enterocolitis mimicking IBD is uncommon and poses diagnostic challenges. The temporal relationship between dasatinib initiation and symptom onset, progressive worsening during treatment, and complete resolution following discontinuation strongly supports drug-induced etiology. The endoscopic findings, while concerning for IBD, lacked supporting histopathological evidence, which is crucial for IBD diagnosis. The mechanism underlying dasatinib-induced enterocolitis remains unclear but may involve disruption of intestinal epithelial barrier function or immune-mediated inflammatory responses. The eosinophilic infiltration observed in biopsies suggests possible allergic or hypersensitivity reaction. Clinicians should maintain high suspicion for drug-induced enterocolitis in CML patients receiving dasatinib who develop new gastrointestinal symptoms, particularly when symptoms are progressive. Careful correlation between clinical presentation, endoscopic findings, and histopathological examination is essential to avoid misdiagnosis and inappropriate immunosuppressive therapy. **Conclusion:** Dasatinib can cause progressive enterocolitis mimicking IBD in CML patients. Complete symptom resolution following drug discontinuation confirms the diagnosis and highlights the importance of considering drug-induced etiology before initiating immunosuppressive therapy for presumed IBD in patients receiving tyrosine kinase inhibitors.

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OP 4

CASE REPORT: A RARE TRIPLE MALIGNANCY – JAK2-POSITIVE POLYCYTHEMIA VERA, CHRONIC LYMPHOCYTIC LEUKEMIA AND EGFR-MUTANT STAGE IIIB NON–SMALL CELL LUNG ADENOCARCINOMA WITH UNUSUAL CLINICAL COURSE

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Case Description: A 73-year-old male was first diagnosed with PV (hemoglobin >18 g/dL, hematocrit >55%, JAK2 V617F positive) in 2016. He was managed with low-dose aspirin and phlebotomy; hydroxyurea was added later. In 2019, routine CBC showed persistent lymphocytosis (lymphocytes ~12 × 10^9/L). Flow cytometry demonstrated CD5+, CD19+, CD23+, FMC7− B-cells comprising 68% of lymphocytes, confirming Rai stage I CLL. No active treatment was initiated. In 2020, during evaluation for COVID-like respiratory symptoms, thoracic CT revealed a during evaluation for a COVID-19-suspected cough and dyspnea, thoracic CT revealed a 20 × 14 mm left upper lobe mass with mediastinal lymphadenopathy with mediastinal lymphadenopathy. Bronchoscopic biopsy confirmed adenocarcinoma. EGFR exon 21 L858R mutation was present; ALK and ROS1 were negative. PET–CT staged disease at IIIB. Standard chemoradiotherapy was declined by the patient. Erlotinib treatment was initiated in March 2020. Concurrent progression of CLL with B symptoms prompted introduction of chlorambucil 10 mg daily for 7 days in a 28-day cycle. At 3-month follow-up, CT scan showed near-complete regression of primary lung lesion and mediastinal nodes. CBC normalized. JAK2 V617F mutation, positive in 2016, was undetectable via allele-specific PCR (<1% allele burden). The patient exhibited ECOG 1 and continued erlotinib and chlorambucil with no grade ≥2 toxicity. **Timeline:** • 2016: PV diagnosis (JAK2 V617F+) → aspirin/phlebotomy • 2019: Rai stage IV CLL diagnosis+ chlorambucil • 2020: NSCLC diagnosis (EGFR L858R+), start erlotinib • 2021: Near-complete response, hematologic normalization, JAK2 negativity **Diagnostic Assessment:** Routine labs and molecular assays performed at a reference laboratory confirmed JAK2 mutation status. Flow cytometry was consistent with CLL immunophenotype. NSCLC diagnosis followed standard bronchoscopic sampling; molecular analysis used validated PCR panels and sequencing. **Therapeutic Intervention:** • **Erlotinib**: 150 mg PO daily as standard first-line for EGFR-mutant NSCLC[^3]. • **Chlorambucil**: 10 mg PO daily for 7/28 cycle for symptomatic Rai stage IV CLL, selected for low toxicity in elderly[^4]. **Follow-Up and Outcomes:** • **At 3 Months:** Dramatic radiologic regression; normalization of hematologic parameters; JAK2 mutation undetectable. • Continued stable on erlotinib + chlorambucil with no significant toxicity; quality of life