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

maintained. **Discussion:** This case is unique in that: • **Sequential triple malignancy**: PV, CLL, and EGFR-mutant NSCLC rarely occur together. • **Therapeutic synergy**: Dual-targeted therapy produced durable responses in both solid and hematological malignancies. • **JAK2 loss**: Post-treatment JAK2 negativity suggests clonal competition or epigenetic remission; parallels have been observed with interferon-alpha in MPN[5]. • **Clinical implications**: Supports feasibility of combinatorial targeted therapy in elderly with multiple malignancies. Clonal hematopoiesis of indeterminate potential (CHIP) and aging likely predisposed this patient to multiple neoplasms [^6]. The "clonal competition hypothesis" posits that dominant clones (e.g., NSCLC with EGFR mutation) may suppress other clones (JAK2+) via shared niche or resource limitation. Limitations include single-patient observation; further genomic investigation (e.g., NGS) could clarify clonal evolution mechanisms. We recommend longitudinal monitoring of allele burden and expanded studies on multi-targeted therapy interactions. **Conclusion:** Conclusion Elderly patients with multiple sequential malignancies can benefit from tailored, low-toxicity targeted therapies. The unexpected disappearance of JAK2 mutation invites further investigation into clonal dynamics and epigenetic remission phenomena. This case enriches our understanding of cancer ecology in aging patients.

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OP 5

Autoimmune Hemolytic Anemia as the Presenting Feature of Chronic Lymphocytic Leukemia: Two Contrasting Cases Across Different Age Groups

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Introduction: Chronic lymphocytic leukemia represents the most common adult leukemia in Western countries, with autoimmune hemolytic anemia occurring as a complication in 5-10% of cases. AIHA as the presenting feature of CLL is uncommon, particularly in young adults where CLL incidence is extremely rare. The immunophenotypic heterogeneity of CLL, including atypical variants, may influence both clinical presentation and treatment response. **Case Reports:** Case 1: An 84-year-old female presented with progressive fatigue, weakness, and dyspnea. Laboratory evaluation revealed severe anemia (Hb: 9.3 g/dL), marked leukocytosis ($42.36 \times 10^3/\mu$L), and thrombocytopenia. Direct antiglobulin test was strongly positive (3+), confirming warm-type AIHA. Flow cytometry demonstrated classic CLL immunophenotype: CD19+ (93%), CD5+ (95%), CD23+ (84%), CD20+ (52%), with absent CD38 expression suggesting favorable-risk disease. Bone marrow biopsy confirmed CLL/SLL with 50% infiltration. Treatment with prednisolone rapidly resolved hemolysis, followed by ibrutinib therapy for CLL. The patient achieved sustained remission over 12 months with corticosteroid discontinuation after 3 months. Case 2: A 25-year-old male presented with dyspnea, palpitations, and fatigue. Initial workup revealed severe anemia (Hb: 7.8 g/dL), reticulocytosis (6.8%), and elevated LDH with spherocytes on peripheral smear. Direct antiglobulin test was strongly positive (4+). Investigation revealed lymphocytosis (14,200/mm^3, 68% lymphocytes) with atypical CLL immunophenotype: CD5+/CD19+/FMC7+/CD23-, distinguishing it from typical CLL while excluding mantle cell lymphoma through negative cyclin D1. TP53 abnormalities were absent. Initial prednisolone therapy provided insufficient response, prompting rituximab monotherapy (375 mg/m$^2 \times 4$ cycles). The patient achieved complete hematologic response with hemoglobin normalization (11.6 g/dL), reticulocyte count resolution, and lymphocytosis improvement. **Discussion:** These cases illustrate important clinical principles in CLL-associated AIHA management. The elderly patient presented with classic CLL immunophenotype and favorable prognostic markers (CD38-negative), supporting the choice of BTK inhibitor therapy appropriate for her age and comorbidities. The young adult case demonstrated atypical CLL immunophenotype (FMC7+/CD23-), representing a variant phenotype that required careful differentiation from mantle cell lymphoma. The treatment approaches differed significantly based on age and disease characteristics. The elderly patient benefited from targeted therapy (ibrutinib) combined with corticosteroids, while the young patient achieved excellent response with rituximab monotherapy after steroid failure. This highlights the importance of individualized treatment selection based on patient factors and disease biology. Both cases emphasize the critical role of comprehensive flow cytometric analysis in patients presenting with unexplained AIHA, regardless of age. Early recognition of underlying CLL enables appropriate targeted therapy and optimal outcomes. The contrasting immunophenotypes demonstrate the heterogeneity of CLL, with both classic (CD5+/CD23+) and atypical (CD5+/CD23-/FMC7+) variants capable of presenting with AIHA as the initial manifestation. **Conclusion:** AIHA may serve as the presenting feature of CLL across diverse age groups with varying immunophenotypic profiles. These cases underscore the importance of systematic flow cytometric evaluation in all AIHA patients and demonstrate that age-appropriate targeted therapies can achieve excellent clinical outcomes in both classic and atypical CLL variants.

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OP 6

HHV-8 Positive Kaposi Sarcoma in a Myelofibrosis Patient Treated with Ruxolitinib: A Rare but Clinically Relevant Association

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Introduction: Kaposi sarcoma (KS) is a rare vascular tumor strongly associated with human herpesvirus 8 (HHV-8) and typically seen in immunocompromised states such as HIV/AIDS or post-transplant settings. However, with the increasing use of immunomodulatory therapies in hematologic