more episodes of bright red bleeding and required two additional erythrocyte transfusions. An upper endoscopy was performed, revealing no esophageal varices. A 2 × 3 cm angiodysplastic lesion was observed in the gastric corpus and was cauterized with an argon laser. A scintigraphy scan confirmed increased activity in the same area. Following the procedure, a fresh frozen plasma transfusion was administered and propranolol treatment was started. With the bleeding controlled, and given the concurrent angiodysplasia, a detailed work-up for coagulopathy was performed. The patient's von Willebrand factor activity was below 5%, the von Willebrand factor antigen was below 3%, and Factor VIII was less than 3.5%. Platelet aggregation tests were normal. Genetic analysis of the VWF gene identified a novel homozygous mutation, c.7176T>G p.(Tyr2392Ter), and a heterozygous mutation, c.817C>G p.(Ar273Gly). Considering the clinical presentation, laboratory findings, and genetic analysis in a patient with no parental bleeding history, a diagnosis of Type 3 von Willebrand disease was established. Gastrointestinal bleeding due to angiodysplasia in VWD is a well-known complication that typically arises in adults. The only previously reported pediatric case was by Aggoune et al., who described a 14-year-old with Type 3 VWD and duodenal angiodysplasia who required surgical resection for recurrent bleeding. In contrast, our patient's initial bleeding episode was successfully managed with a combination of argon laser cauterization and medical therapy. This case highlights the importance of considering VWD in pediatric patients who present with severe gastrointestinal bleeding, especially when routine coagulation tests show a prolonged aPTT and bleeding time in the absence of risk factors for esophageal varices. It also serves as a crucial reminder that angiodysplasia is a known complication of VWD that can present even in the youngest of patients

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Marrow-Dominant Marginal Zone Lymphoma with Plasmacytic Differentiation in a Frail and old patient: Immunophenotypic Pitfalls and Rituximab-Only Strategy

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Introduction: Marginal zone lymphoma (MZL) with plasmacytic differentiation can mimic other B-cell entities—particularly CLL/SLL and lymphoplasmacytic lymphoma (LPL)—and often presents in the elderly with cytopenias rather than bulky nodal disease. Correct classification is critical, as comorbidity and frailty frequently constrain treatment intensity. We report a bone marrow–dominant, plasmacytoid MZL in an 84-year-old woman successfully managed with rituximab monotherapy. **Methods:** We conducted a single-patient, retrospective case review of prospectively collected clinical, laboratory, pathology, and imaging data. Diagnostic workflow integrated complete blood count, immunoglobulin quantification, bone marrow histology with immunohistochemistry (IHC), multiparameter flow cytometry, and FDG-PET/CT. Treatment selection followed a frailty-adapted decision process. **Results:** An 84-year-old woman presented with progressive fatigue and dyspnea on exertion. Baseline labs showed leukocytosis with marked lymphocytosis and macrocytic pancytopenia (WBC 22.2 × 10^9/L; absolute lymphocytes 18.4 × 10^9/L; Hb 8.1 g/dL; MCV 105 fL; platelets 42 × 10^9/L). Immunoglobulins were not suggestive of LPL/WM (IgM 0.73 g/L; IgG 13.6 g/L; IgA 1.7 g/L). FDG-PET/CT revealed mediastinal/abdominal lymphadenopathy, splenomegaly, and a sternal cortical irregularity suspicious for osseous involvement. Bone marrow biopsy was hypercellular (∼95%) with ∼90% interstitial/patchy small-to-intermediate B-cell infiltration; reticulin fibrosis 0/4; Congo red negative. IHC supported a non-CLL, non-mantle phenotype: CD20 strong positive; CD5, CD23, CD10, cyclin D1, annexin A1, TRAP all negative. Flow cytometry demonstrated a clonal mature B-cell population (CD19+, CD20+, CD38+, cCD79a+) without CLL-type markers (CD5/CD23 negative). Plasmacytic differentiation was present, yet serum IgM remained normal, arguing against LPL/WM. Overall, findings established marginal zone lymphoma with plasmacytic differentiation, stage IV-A (marrow ± splenic/possible bone involvement). Given advanced age, cytopenias, and frailty, cytotoxic chemo-immunotherapy was deferred. The patient received rituximab monotherapy with antiviral prophylaxis and supportive care (transfusion as needed). Treatment was well tolerated; early follow-up showed clinical improvement with rising hemoglobin and platelet counts and reduction in lymphocytosis. **Discussion:** This case highlights three practice points. First, plasmacytic differentiation in MZL can masquerade as CLL or LPL/WM; a disciplined panel—CD5/CD23/cyclin D1 negativity with strong CD20 and compatible flow cytometry—prevents misclassification. Second, serological context matters: normal IgM helped exclude LPL/WM despite plasmacytoid histology. Third, in the very elderly/frail, rituximab monotherapy is a rational, lower-toxicity strategy that can reverse cytopenias and improve function when marrow disease predominates. The possible osseous signal on imaging further underlines the heterogeneity of MZL dissemination. Educationally, the case underscores integrating morphology, IHC, flow, and serology to secure diagnosis and tailor therapy beyond one-size-fits-all chemo-immunotherapy. **Conclusion:** Bone marrow-dominant MZL with plasmacytic differentiation presents diagnostic challenges but can be accurately classified through integrated morphology, IHC, flow cytometry, and serology. In very elderly or frail patients, rituximab monotherapy represents a rational and effective treatment strategy, offering hematologic recovery and functional benefit while minimizing toxicity.

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