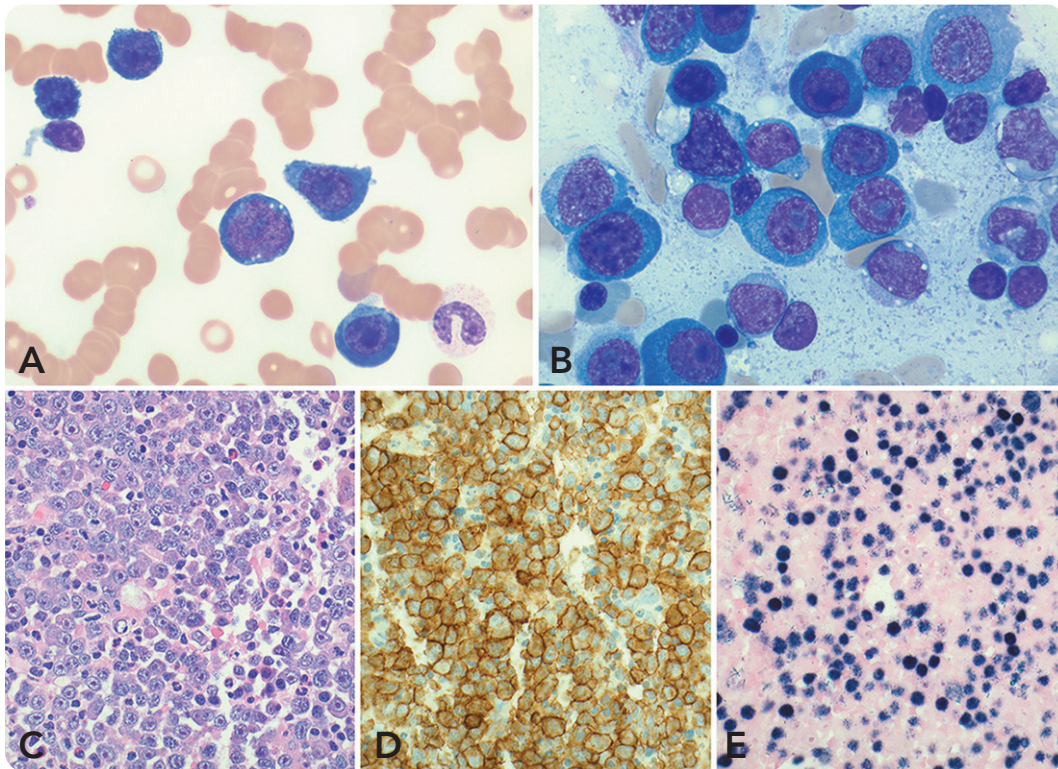


Plasmablastic myeloma/leukemia variant of monomorphic posttransplant lymphoproliferative disorder

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A 73-year-old man with a 2-year history of bilateral lung transplantation presented with progressive fatigue, nausea, and night sweats over the past 2 months. Laboratory tests showed abnormal serum components: creatinine, 460.0 $\mu\text{mol/L}$; lactate dehydrogenase, 3073 U/L; immunoglobulin A (IgA) lambda paraprotein, 43.7 g/L; free gamma light chain, 8.2 g/L; and Epstein-Barr virus (EBV) load, 1 555 723 IU/mL. Complete blood cell count demonstrated leukocytes $13.7 \times 10^9/\text{L}$, hemoglobin 74 g/L, and platelets $59 \times 10^9/\text{L}$. Blood smear examination showed 34% blastoid cells (panel A: Wright-Giemsa stain, original magnification $\times 1000$) that were identified as plasma cells with aberrant CD56 and absent CD19 and CD20 by flow cytometry. Bone marrow biopsy revealed markedly increased large cells with plasmablastic morphology (panel B: aspirate smear, Wright-Giemsa stain, original magnification $\times 1000$; panel C: core biopsy, hematoxylin and eosin stain, original magnification $\times 400$) that were positive for CD138 (panel D: original

magnification $\times 400$), lambda light chain restriction, and EBV latency 1 infection (panel E: EBV-encoded RNA in situ hybridization, original magnification $\times 400$). Cytogenetic studies detected a complex karyotype that included $t(8;14)(q24;q32)/MYC-IGH$. A radiologic scan demonstrated no lymphadenopathy or bone lytic lesions. Thereafter, diagnosis of a plasmablastic myeloma/leukemia variant of monomorphic posttransplant lymphoproliferative disorder (M-PTLD) was rendered. The patient's condition deteriorated without therapy specifically designated for treating myeloma/lymphoma, and he expired 7 days after the diagnosis.

The myeloma variant of M-PTLD is rare. The variant with leukemic manifestation has not yet been reported in any detail. Our patient shows plasmablastic morphology and an *MYC-IGH* genotype that might have been the pathologic factors driving the lethal leukemic presentation and catastrophic clinical course.