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A case of histoplasmosis in a patient with MDS/MPN-U

TO THE EDITOR: We would like to share the following intriguing case with readers of **Blood Research**. A 72-year-old HIV-negative man presented with a 2-month history of gradual-onset generalized weakness, low-grade fever, and loss of weight and appetite. On examination, he had pallor and hepatosplenomegaly. His hemogram findings revealed a nearly normal hemoglobin level (11.8 g/dL), leukocytosis (15.8×10^9/L), and thrombocytopenia (78×10^9/L). Differential counts showed 3% circulating blasts apart from 6% immature myeloid cells, 4% basophils, and 2% monocytes. A bone marrow aspirate was hypercellular with 5% blasts along with dyserythropoiesis (36%, including 32% ring sideroblasts), dysgranulopoiesis (11%), and dysmegakaryopoiesis (40%). The trephine biopsy showed dyshemopoiesis along with World Health Organization grade 2 reticulin fibrosis. Tests for the detection of *BCR-ABL1* fusion gene and *JAK2* mutations were negative. A diagnosis of myelodysplastic syndrome/myeloproliferative neoplasm-unclassifiable (MDS/MPN-U) was made [1].

Subsequently, the patient was lost to follow-up, but 7 months later, he visited the emergency room with a high-grade fever with chills and rigors, altered sensorium, and irrelevant talking. On examination, he had neck rigidity, a positive Kernig sign, and right lower limb monoparesis along with hepatosplenomegaly. A computed tomographic scan of his head and cerebrospinal fluid examination were normal. Blood and urine bacterial cultures were sterile. At this time, the hemogram revealed anemia (hemoglobin level, 6.0 g/dL), thrombocytopenia (19×10^9/L), and leukocytosis (63.5×10^9/L) with a differential similar to that obtained 7 months previously. A bone marrow aspirate also revealed a picture similar to that of the previous marrow aspirate. The striking finding noticed at the time of peripheral blood and marrow evaluation was the presence of intracytoplasmic yeast forms (within neutrophils) conforming to *Histoplasma* species (Fig. 1). These were confirmed to be *Histoplasma capsulatum* based on fungal culture studies from peripheral blood (Fig. 1) as well as gene sequencing of the internal transcribed spacer region of the fungus. The patient died on the same day as the bone marrow procedure.

We present this case because this patient highlights an unusual morphological coexistence of a neoplastic and infective disorder [2]. A predisposing factor might have been the dysplastic neutrophils with defective phagocytic and microbicidal activity [3]. The case illustrates the importance of morphology in the era of genomics as well as the value of close interdisciplinary cooperation in diagnostic hematology. It also reinforces the dictum that hematopathologists must always stay on the alert for uncommon infections in unusual specimens, especially in tropical countries.

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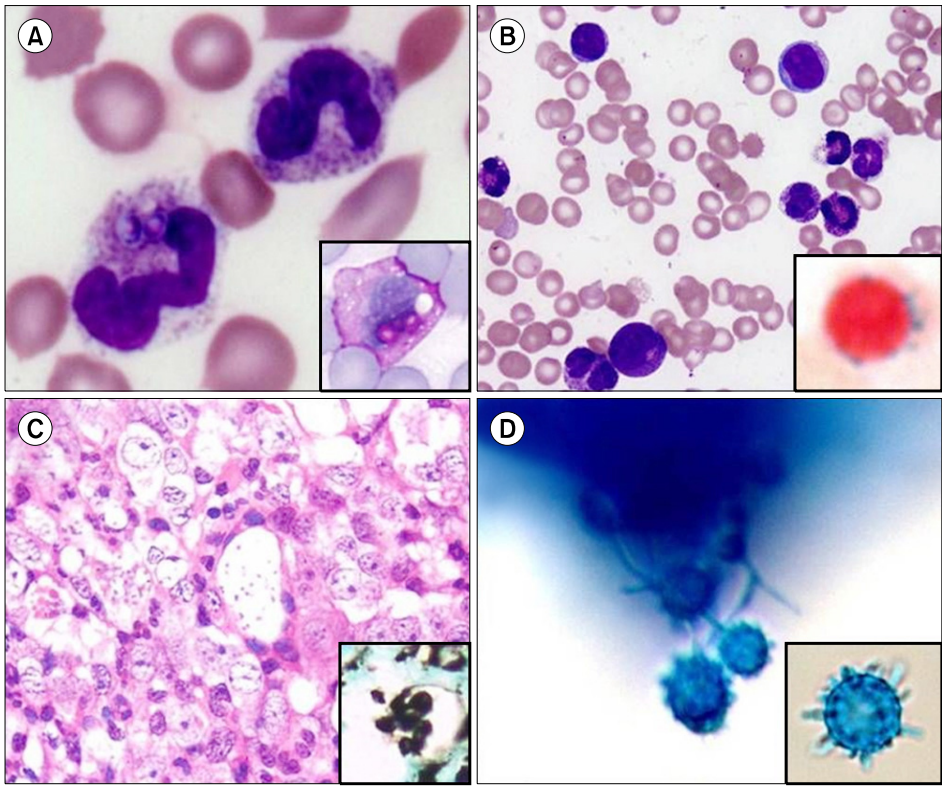

Fig. 1. **(A)** Peripheral blood shows neutrophils with toxic granules and two intra-cytoplasmic yeast forms of *Histoplasma capsulatum* in the cell on the left (May-Grunwald Giemsa, ×1,000). Inset: The fungus was brightly positive for periodic acid–Schiff stain (hematoxylin counterstain, ×1,000). **(B)** Hemodilute bone marrow smears showed dysgranulopoiesis with 5% blasts (May-Grunwald Giemsa, ×1,000). Inset: Ring sideroblasts comprised 32% of all cells (Perls' Prussian Blue reaction with neutral red counterstain, ×1,000). **(C)** Bone marrow biopsy showed intracellular yeast forms (hematoxylin and eosin, ×400). Inset: These were positive for Grocott's silver methenamine stain (methyl green counterstain, ×1,000). **(D)** Lactophenol cotton blue wet-mount preparation of the isolated mold shows thick-walled and tuberculate macroconidia, with a close-up in the inset (lactophenol cotton blue stain, ×1,000).

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A case of thrombotic thrombocytopenic purpura in late pregnancy

TO THE EDITOR: Thrombotic thrombocytopenic purpura (TTP) was first described by Eli Moschowitz about 90 years ago [1]. TTP is caused by a severe deficiency in the protein A disintegrin and metalloprotease with thrombospondin 1 motifs 13 (ADAMTS13), a metalloprotease that cleaves ultra-large von Willebrand factor (vWF) multimers [2]. Deficiency in this enzyme causes the accumulation of large vWF multimers, which increase platelet adhesiveness and impair fibrinolytic activity with subsequent thrombotic occlusion of the microvasculature. TTP is a rare and potentially life-threatening disorder. The incidence rate of suspected TTP-hemolytic uremic syndrome (TTP-HUS) in the general population is 11 cases per 1,000,000 people [3], compared to the estimated incidence of one in 25,000 deliveries [4]. The clinical features of TTP may resemble those of the more common pregnancy complications, such as preeclampsia or of hemolytic anemia, elevated liver enzymes, and low platelet count (HELLP) syndrome. However, the management of TTP is completely different from that of preeclampsia/HELLP. Here, we present the case of a young woman who developed TTP in late gestation.