What Is the Cause of Macrocytosis and Dyspnea in an Older Man?

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A 78-year-old man presented to the emergency department with a 3-week history of progressive shortness of breath and cough with blood-streaked, yellowish sputum. The patient had dyspnea on exertion limited to 2 blocks, 2-pillow orthopnea, paroxysmal nocturnal dyspnea, and nocturia. Neither fever nor chills were present. He had lost 7.2 kg (16 lb) during the last year.

Figure 1

Figure 2

A 78-year-old man presented to the emergency department with a 3-week history of progressive shortness of breath and cough with blood-streaked, yellowish sputum. The patient had dyspnea on exertion limited to 2 blocks, 2-pillow orthopnea, paroxysmal nocturnal dyspnea, and nocturia. Neither fever nor chills were present. He had lost 7.2 kg (16 lb) during the last year.

The medical history included chronic obstructive pulmonary disease, hypertension, and type 2 diabetes mellitus; medications were metformin, aspirin, metoprolol, and lisinopril. The patient smoked 2 cigars a day and denied excessive alcohol consumption. His brother died at age 39 of a myocardial infarction; 2 cousins had thalassemia.

Respiratory distress, pale skin, and increased jugular venous pressure (jugular venous column was 11 cm above the sternal angle) were noted. Lung examination revealed bilateral crackles halfway up, with decreased breath sounds and dullness to percussion. An S3 with a nonradiating, soft systolic murmur at the left sternal border was heard. The spleen was enlarged. The knees had bilateral 2+ pitting edema.

White blood cell count was 2500/?L; hemoglobin, 5 g/dL; hematocrit, 15.8%; platelet count, 128,000/?L; and mean corpuscular volume, 127 ?m3. Liver function tests and coagulation profile results were normal. Vitamin B12, folic acid, methylmalonic acid, and homocysteine levels were normal. The peripheral smear (Figures 1 and 2) demonstrated increased monocytes with bizarre, stippled, megaloblastic red blood cells and large platelets.

A chest film revealed cardiomegaly with pulmonary edema. The echocardiogram showed normal left ventricular function with mild tricuspid regurgitation. High-output congestive heart failure was diagnosed. The patient was admitted to the hospital; nitrates, morphine, angiotensin-converting enzyme inhibitors, and furosemide were given for the heart failure, and packed red blood cells were administered to increase the hemoglobin.
Because of the patient's anemia and splenomegaly, a hematologist was consulted. A bone marrow aspirate (Figures 3 - 5) revealed dyserythropoietic cells, increased marrow monocytes, a leftward shift, and ringed sideroblasts. The total blasts and promyelocytes were 11.5%; refractory anemia with excess blasts was identified. Six red blood cell transfusions stabilized the patient's condition; he was discharged from the hospital.

Early referral to a hematologist is warranted for patients with macrocytosis for which there is no obvious cause. This patient was referred to a hematologist. He refused all but symptomatic treatment.

The myelodysplastic syndromes (Table)—clonal hematologic disorders characterized by ineffective hematopoiesis—need to be considered in the differential diagnosis of patients with a macrocytic anemia, particularly those who are older than 50 years. These preleukemic conditions manifest as slowly developing anemia that is refractory to standard therapy and may develop into acute myelogenous leukemia (AML). Median age at onset is the seventh decade of life; approximately 60% of patients are men.¹
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Refractory anemia (20% - 30%)

Elderly; anemic < 1 % blasts; cytopenia at least 1 cell line, most of the time RBs < 5 %; no myelodysplasia; pancytopenia; low neutrophils; low platelets; low red blood cells; low white blood cells; low hemoglobin; low hematocrit; low mean corpuscular volume; low mean corpuscular hemoglobin; low mean corpuscular hemoglobin concentration; low serum iron; low transferrin saturation; low ferritin; low total iron-binding capacity; low reticulocyte count; low bone marrow biopsy; low bone marrow aspirate; low bone marrow smear; low bone marrow cytogenetics; low bone marrow histology; low bone marrow flow cytometry; low bone marrow immunohistochemistry; low bone marrow aspirate cytology; low bone marrow aspirate morphology; low bone marrow aspirate nucleation; low bone marrow aspirate differentiation; low bone marrow aspirate maturation; low bone marrow aspirate infiltration; low bone marrow aspirate necrosis; low bone marrow aspirate fibrosis; low bone marrow aspirate ossification; low bone 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Refractory anemia with excess blasts (30%)

Any age; symptoms of brief duration > 5% blasts in transformation; 5% - 20% blasts; normal morphology; peripheral leukoerythroblastosis.
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Chronic myelomonocytic leukemia (15% - 20%)
PATHOGENESIS
Most primitive progenitors give rise to red cells, platelets, neutrophils, eosinophils, and basophils. Neoplastic transformation occurs at various levels of development. In myelodysplasia, malignant transformation at the level of a myeloid stem cell can result in chromosomal abnormalities. Patients with balanced chromosomal translocation are more likely to have overt leukemia; those with unbalanced translocation (eg, trisomies or partial chromosomal deletions) are prone to ineffective hematopoiesis, which leads to increased marrow cellularity. Inadequate cell maturation results in peripheral cytopenias. Apoptosis, or programmed cell death, increases in patients who have myelodysplasia, with futile cycling of blood cell precursors and impaired production of mature blood cells. The sensitivity of red cell precursors to erythropoietin is decreased, as is the myeloperoxidase and microbicidal activity of neutrophils. Platelets are functionally defective.

DIFFERENTIAL DIAGNOSIS
Hereditary dysplasias, such as hereditary sideroblastic anemia, Fanconi anemia, Diamond-Blackfan syndrome, Kostmann syndrome, and Schwachman syndrome, need to be included in the differential. Also consider vitamin B₁₂/folate deficiency; toxicity from drugs, alcohol, and chemotherapeutic agents; radiation or benzene exposure; renal failure; tuberculosis; autoimmune disease; viral infections, including those caused by the Epstein-Barr virus, parvovirus, and HIV; and paroxysmal nocturnal hemoglobinuria.

DIAGNOSIS
Patients with myelodysplasia may present with 1 or more of the following:

- Bleeding.
- Bruising.
- Dyspnea on exertion.
- Infection.
- Leukopenia.
- Progressive fatigue.
- Splenomegaly.
- Thrombocytopenia.

Confirmation of the diagnosis primarily relies on morphologic findings. Examine the bone marrow to detect cytogenetic abnormalities. Fluorescence in situ hybridization is an important tool for identifying clonal genetic abnormalities. Anemia, neutropenia, and/or thrombocytopenia are common findings. Bone marrow has normal or increased cellularity; megaloblastic red cell precursors with multiple nuclei or asynchronous maturation of the nucleus and cytoplasm may be noted. Ringed sideroblasts—erythroid precursors...
with iron-laden mitochondria—are seen occasionally. Mature neutrophils may be hypogranular and hypolobulated (pseudo-Pelger-Huët anomaly).

Megakaryocytes have few nuclear lobes and are small (micromegakaryocytes). Dysplastic abnormalities in all lines can include nuclear and cytoplasmic blebs, karyorrhexis, and misshapen nuclei. The number of myeloblasts is increased. Peripheral blood may have oval macrocytic red cells, hypogranular granulocytes with pseudo-Pelger-Huët anomaly, and giant platelets.

**ASSOCIATED SYNDROMES**

**The 5q syndrome.** This disorder is caused by a deletion of a long arm of chromosome 5. Approximately 70% of patients are women. The syndrome has a prolonged course with less than a 25% chance of progression to leukemia. The patient's platelet count usually is high. Red blood cell transfusions and management of iron overload are the mainstays of treatment.

**Hypoplastic myelodysplasia.** It is difficult to distinguish this disorder from aplastic anemia. The bone marrow has less than 25% cellularity. The natural history of myelodysplasia with hypocellular marrow is similar to normocellular or hypercellular myelodysplasia; however, it appears to be a distinct clinicopathologic entity that is characterized by marrow hypoplasia, macrocytosis, severe leukopenia and thrombocytopenia, and a low incidence of progression to acute leukemia. The disorder is unresponsive to conventional therapy; pyridoxine, folic acid, prednisone, anabolic steroids, retinoids, and lowdose cytosine arabinoside, which are minimally effective in normocellular disease, are not beneficial in patients with the hypoplastic disorder.14

**Childhood myelodysplasia.** The symptoms of the childhood disease are similar to those in adults. About one third of affected children have Down syndrome; more than 40% of patients develop AML.15

Usually, the disease is found in children younger than 2 years. Typically, their hemoglobin F level is greater than 10%. The presence of refractory anemia with excess blasts and refractory anemia with excess blasts in transformation is associated with a poor outcome.

**Bone marrow fibrosis.** Some patients with myelodysplasia have increased reticulin fibrosis in the marrow, a condition that can be confused with primary (agnogenic) myelofibrosis. However, myelodysplasia may be distinguished by trilineage dysplasia, prominent splenomegaly, and the absence of hepatomegaly.

**TREATMENT**

Myelodysplastic syndromes are highly resistant to treatment. Supportive therapies include transfusions, antibiotics, deferoxamine, and erythropoietin. A small number of patients may be helped by pyridoxine. Treatments similar to those used for patients with AML have been tried. The remission rate after induction of anthracycline and cytarabine is 50% to 60%; relapses occur in about 90% of those patients with myelodysplastic syndromes.16

Patients with preexisting myelodysplastic syndrome have a lower remission rate, shorter periods of remission, and a higher incidence of relapse. Other treatments that have been tried include topotecan, hexamethylene bisacetamide, 5-aza-2'-deoxycytidine, and amifostine (organic thiophosphate). Antithymocyte globulin and antibody against CD33 are also used. Granulocyte colony-stimulating factor, granulocyte-macrophage colony-stimulating factor, interleukins (IL-6 and IL-11), and interferon-α have been beneficial in a few patients. Stem-cell transplantation is now the primary curative therapy, especially for younger patients.

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