Aplastic Anemia Associated with Amiodarone Therapy

I.S. Izidore S. Lossos
Y. Yaacov Matzner

Jerusalem
Prof. Yaacov Matzner, Hematology Unit, Hadassah University Hospital, P.O. Box 24035, 91240 Jerusalem (Israel)

Amiodarone, iodine-containing benzofuran derivative, is a potent antiarrhythmic drug effective against a variety of supraventricular and ventricular cardiac arrhythmias [1]. Its applicability is limited by numerous adverse effects including cardiac, pulmonary, hepatic, thyroid, gastrointestinal, dermatologic, ophthalmologic and neurologic abnormalities [1-3]. Hematologic side effects are rare and to the best of our knowledge, only 2 cases of amiodarone-induced thrombocytopenia have been reported [4]. We describe a patient who developed severe aplastic anemia during amiodarone therapy.

A 68-year-old man was admitted to the Department of Medicine for evaluation of spontaneous ecchymoses and oral mucosal bleeding of 2 months’ duration. His previous history included multifocal ventricular premature beats treated during the last 3 years with amiodarone 200 mg/day and uneventful left hemicolectomy for carcinoma of the colon, Duke A, 8 months prior to the present admission.

Physical examination revealed diffuse purpura and ecchymoses. Ophthalmoscopic inspection demonstrated brownish opaque corneal deposits attributed to amiodarone therapy. Laboratory studies revealed normocytic normochromic anemia (hemoglobin 97 g/l), thrombocytopenia (2 × 10^9/1), mild leukopenia (3 × 10^9/1 with 50% neutrophils) and reticulocyte count, corrected for hematocrit, 0.3%. Bone marrow aspiration and biopsy showed absence of megakaryocytes and the few cells present consisted mainly of lymphocytes and plasma cells. The overall cellularity was less than 25% establishing the diagnosis of severe aplastic anemia. All other laboratory studies, including serologic tests for possible viral etiology, were noncontributory.

The patient deteriorated rapidly despite discontinuation of the amiodarone therapy. Hemoglobin and white cell count decreased gradually and platelet count remained extremely low resulting in profound pancytopenia. Treatment with corticosteroids and cyclosporine A was given, but the patient succumbed to Klebsiella pneumoniae sepsis 2 months after the initial presentation.

Postmortem examination confirmed the diagnosis of severe aplastic anemia. There was no evidence for metastases of his colon carcinoma.

This case demonstrates a possible association between amiodarone and severe aplastic anemia. The fact that amiodarone discontinuation did not lead to improvement in the blood count may be explained by the long half-life of the drug in the blood or by an irreversible damage. Though a simple coincidence cannot be ruled out in a single case history, physicians should be aware of this possible side effect of amiodarone. Further reports will be required to clarify the cause-effect relationship between the drug and aplastic anemia.
References