Acute toxicity in erythroid bone marrow progenitors after antimonial therapy

Erythroid bone marrow damage has been described after the administration of several drugs, such as cloramphenicol or alcohol. Visceral leishmaniasis is a frequent complication in patients with human immunodeficiency virus (HIV) infection. Antimonial therapy achieves response rates greater than 50% in HIV-infected individuals with leishmaniasis. We report the case of an HIV-positive patient with visceral leishmaniasis and severe bone marrow dyserythropoiesis after antimonial therapy with sodium stibogluconate. A 37-year-old man with a diagnosis of acquired immunodeficiency syndrome was admitted to our hospital because of prolonged fever. He was receiving triple highly active antiretroviral therapy and secondary prophylaxis for P. carinii infection with cotrimoxazole. Blood tests showed hemoglobin (Hb) 10.4 g/dL and polyclonal hypergammaglobulinemia. Bone marrow aspirate smears revealed a very high number of Leishmania sp. Parenteral sodium stibogluconate therapy (850 mg/day) was started in an outpatient schedule. Three weeks later, the patient developed severe anemia (Hb 6.9 g/dL) and red blood cell transfusion was required. A new bone marrow aspirate showed karyorrhexis in most of nuclear red cell progenitors but no Leishmania invasion was observed (Figure 1). Antimonial therapy was stopped and clinical and biological recovery was achieved. A third myelogram, performed 4 weeks later, was normal. The patient remains alive after one year, with a Hb level of 12 g/dL and without relapse of visceral leishmaniasis despite secondary prophylaxis with antimonial compounds or amphotericin B not being administered.

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