

### 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.<sup>1</sup> Visceral leishmaniasis is a frequent complication in patients with human immunodeficiency virus (HIV) infection.<sup>2</sup> Antimonial therapy achieves response rates greater than 50 % in HIV-infected individuals<sup>3</sup> 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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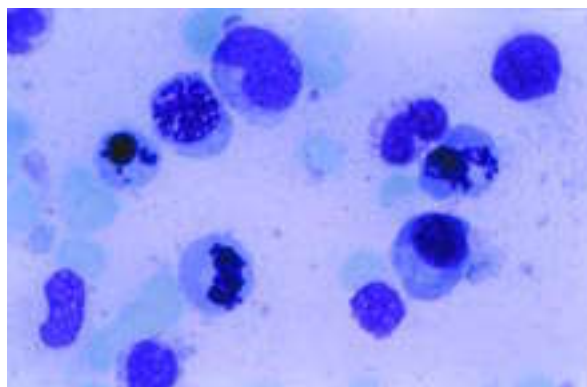


Figure 1. Bone marrow aspirate smear. Severe nuclear red cell dysplasia. May-Grünwald-Giemsa stain.

#### References

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