Conclusion

Le syndrome de Mazabraud est rare mais très probablement sous-estimé. L'imagerie et en particulier l'IRM, en montrant des images caractéristiques de la DF et du myxome, permet de poser le diagnostic de syndrome de Mazabraud et par-là de surseoir à la biopsie.

Références

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Visceral leishmaniasis in renal transplant patient

Visceral leishmaniasis (VL) is a parasitic disease, caused by protozoa of the genus Leishmania, transmitted by the bite of insect vectors, the phlebotomine sand flies. In Tunisia, it occurs under an endemic mode and its clinical profile is typical of the Mediterranean infantile form. Since 1985, the prevalence of VL has increased significantly in several countries, mainly in Southern Europe, due to immunosuppression associated with HIV infection [1]. Other immunosuppressive states can occasionally lead to the appearance of clinically overt VL in previously asymptomatic patients, such as neoplasic disease, use of corticosteroids and cytotoxics [2].

In the last 20 years, the increasing frequency of organ transplantations and improvement of the associated immunosuppressive treatments have led to the recognition of several cases of VL complicating organ transplantation [3]. In this context, VL is fatal without antileishmanial treatment which constitutes a difficult challenge.

We report a case of VL in renal transplant recipient treated by liposomal Amphotericin B (Ambisome®, Gilead).

Case report

A 29-year-old Caucasian man, with end- stage renal disease which etiology was not determined, received a kidney allograft in 2009 from a deceased donor. He originally came from the North of Tunisia (Tunis), and he has always lived there, before and after transplantation. He was hospitalized 17 months after transplantation, with complaints of high fever. Physical examination revealed hepatosplenomegaly and laboratory tests revealed pancytopeny. Diagnosis was done by direct finding of the parasite in smears of bone marrow and by positive serology. Retrospectively, the serological testing of stored serum sample collected before transplantation was negative. Serology of the donor was not possible. The patient was treated with Glucantime®, a pentavalent antimonial (Aventis, Paris, France), at 20 mg/kg per day endovenously. However, he presented at the 7th day a pancreatitis and the antimonial was stopped. Ambisome® was initiated at 3 mg/kg per day endovenously for 5 days plus one more dose a week apart for 4 months, with complete resolution of clinical and laboratory findings. Since, there is no evidence of relapse.

Conclusion

VL should be considered in the differential diagnosis of fever and/or pancytopeny occurring after organ transplantation in Tunisian patients. VL may be caused by ex-novo sand fly transmission, reactivation of latent infection, or transmission via an infected allograft or blood transfusion. The number of asymptomatic cases in endemic countries has led to the recommendation of a routine serological testing for leishmaniasis in the pre-operative check-up of transplant patients and donors. Moreover, transplant patients living or travelling in endemic areas should be tested regularly for leishmaniasis. The combination of these simple measures should lead in the future to a decrease in the frequency of VL occurring in transplant patients.

Liposomal Amphotericin B is presently the treatment of choice to VL in renal transplants.

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