Leishmaniasis infection in allogeneic stem cell transplantation. An “accidental finding”?

Anna Komitopoulou

“Hygeia Hospital”, Kifissias Ave & 4 Er. Stavrou, Maroussi 151 23, Athens, Greece

Correspondence: Anna Komitopoulou
E-mail: annakom@yahoo.com
Received: January 27, 2015
Published online: February 11, 2015

Leishmaniasis is a parasitic disease caused by intracellular protozoa of genus Leishmania. Depending on patient’s immunity status, it presents as visceral, cutaneous and mucocutaneous form. Visceral form (VL) has the highest incidence among other clinical manifestations and is caused by L. donovani and L. infantum (synonym L.chagasi).

While leishmaniasis is mainly a disease of immunocompetent population which can be easily controlled if suspected, it can evolve in severe impairment in immunocompromised patients, especially transplant recipients, with 10—20% of hosts relapsing despite initial specific treatment [1-7].

Although VL is thoroughly investigated in recipients of solid organs, especially renal recipients, information about bone marrow transplanted patients is lacking and limited reports have been documented in the literature so far [8-11].

The diagnosis of leishmaniasis infection might delay or be missed initially by physicians who attend patients after haematopoietic stem cell transplantation (HSCT) because symptoms of the disease can be obscure. Furthermore, specific findings that characterize disease onset like pancytopenia, hepatomegaly and splenomegaly can be attributed to complications related to HSCT [10, 12-15]. Specifically, complications presenting after HSCT like Graft Versus Host Disease (GVHD), cytomegalovirus (CMV) reactivation or coexistent infections may have similar clinical and laboratory manifestations with leishmania infection. So, symptoms like fever, skin rash, sweats, weight loss or findings like pancytopenia can be initially correlated to post transplant complications. In addition, atypical sites of parasitic infection are common in these immunocompromised patients, unlike immunocompetent population [2,4,10,12-16].

Another important issue is that the diagnosis is usually certified by demonstration of parasites in smears or their culture in tissue and by polymerace chain reaction (PCR) positivity, since leishmania antibody detection has poor sensitivity in immunocompromised patients due to the lack of antibody production. Lack of antibody production is mainly related to inhibition of T-helper (Th-1) and T-helper (Th-2) responses in these patients which have been accepted to produce immunity against leishmania infection and correlated with antibody B-cell promotion respectively. (17-23) Sensitivity of PCR is higher in tissues (eg. bone
marrow or spleen), while it varies in peripheral blood because it depends on the circulating parasite load. Nevertheless in contrast to serology, PCR is reported to be useful in the follow up of treatment efficacy.\textsuperscript{[24-28]}

Liposomal amphotericin B is currently the first line of treatment in immunosuppressed patients while the issue of secondary prophylaxis, since VL often relapses in immunocompromised hosts, is still under investigation.\textsuperscript{[29-31]}

In conclusion, VL is a probable infection in subjects who have undergone HSCT. Its diagnosis is challenging because post transplant complications have similar clinical and laboratory manifestations with this specific infection. Physicians should be alert for investigation of leishmania, which is more often accidentally revealed, since early therapeutic intervention may inhibit deteriorating symptoms in hosts with already altered immunity.

References


