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Post transplant lymphoproliferative disease: detection of tumor by Tc99m sestaMIBI and treatment with rituximab

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Post transplant lymphoproliferative disease (PTLD) is often difficult to diagnose because of nonspecific clinical features, and unusual and/or extranodal presentations.

A 40-year-old-man with acute lymphoblastic leukemia (ALL) underwent allogeneic peripheral blood stem cell transplantation (PBSCT) in December 2001. Cyclosporine and prednisolone were given for 8 months as GVHD prophylaxis. In April 2002, liver function tests were found to be abnormal. CMV Ag was positive. CMV hepatitis was suspected but the patient refused a liver biopsy, and he was treated with ganciclovir for 2 weeks. He was well until July 2003, when skin GVHD developed, which was treated with systemic steroids. At the end of July 2003, routine biochemical analysis showed hypergammaglobulinemia and elevated β2-microglobulin. Physical examination was negative except minimal skin excoriation over the extremities. Laboratory parameters were as follows: hemoglobin 11.5 g/dl, white blood cells 11.8×10^9/l, platelets 393×10^9/l, LDH 613 IU/l, AST 59 IU/l, total protein 9.1 g/dl, albumin 2.5 g/dl, β2-microglobulin 8.2 ng/dl and IgG 4911 mg/dL; PTLD was suspected.

Whole-body CT scans and gallium (Ga) scanning showed no abnormality. PET scan was not performed. A Tc99m sestaMIBI scan showed multiple small focal uptake in the inguinal region bilaterally. There was no mediastinal accumulation except physiologic biodistribution of 99mTc sestaMIBI (Figure 1). Inguinal node biopsy showed a characteristic variable mixture of cells of different sizes and types: lymphoplasmacytoid forms, plasma cells, large transformed cells of centroblastic or immunoblastic morphology, as well as small lymphocytes resembling cleaved follicle center cells. The large cells were reactive with CD20 and small lymphocytes were mostly reactive with UCLH-1. Latent membrane protein for EBV was negative (Figure 2a–c).

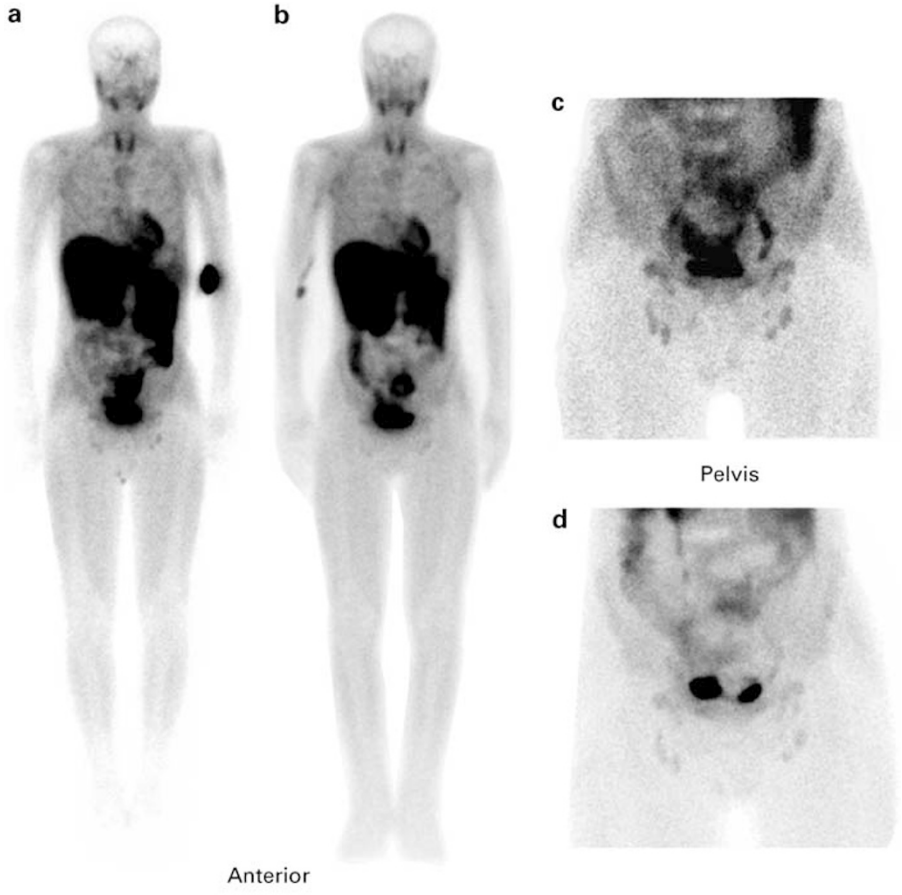


Figure 1 Tc99MIBI scanning: (**a**) anterior whole-body before Rituximab; (**b**) anterior whole-body after rituximab; (**c**) pelvic planar before rituximab and (**d**) pelvic planar after rituximab.

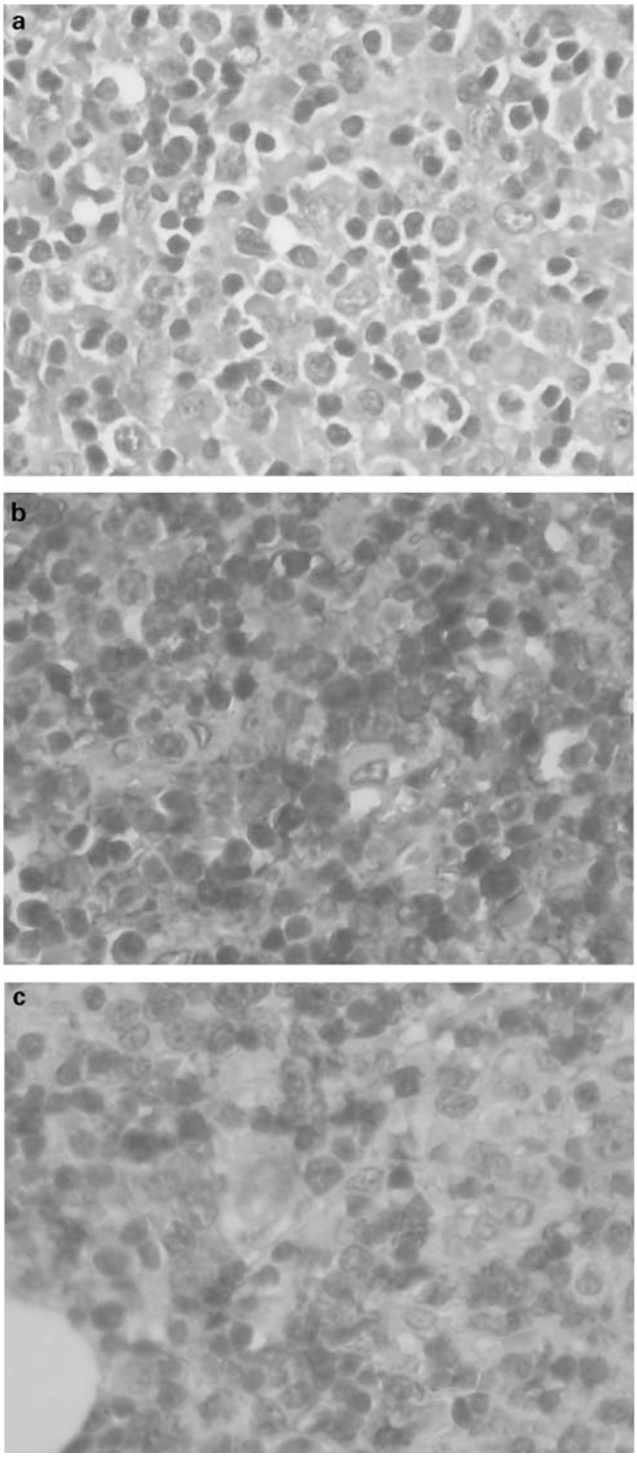


Figure 2 Characteristic mixture of cells of various sizes, some plasma cells, large transformed cells and small lymphocytes.

Rituximab 375 mg/m^2 was given weekly for four doses with response as seen by decreased sestaMIBI accumulation. Figure 1 shows the 99mTc sestaMIBI scan on anterior whole-body (a: before rituximab; b: after rituximab) and pelvic planar (c: before rituximab; d: after rituximab) images. He eventually died of a pulmonary infection.

The diagnosis of PTLD in PBSCT recipients is not always obvious,[1] and standard imaging techniques may be negative. In our case, CT and Ga scans were negative, although, unfortunately, a PET scan was not carried out. PET scanning may be particularly useful in PTLD.[2] Although 99mTc sestaMIBI is normally used for cardiac imaging, it has also been used for tumor imaging and has been found to be useful to detect the lymphomatous infiltrations in cases with lymphoma.[3,4] However intense sestaMIBI activity in the lower chest and abdomen as well as tumor size may limit the diagnostic sensitivity of this radionuclide in lymphoma cases. For this reason CT, PET and Ga scanning have not been replaced by 99mTc sestaMIBI.

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