A 49-year-old man with a general deterioration since 6 months was admitted for acute febrile purpura with pulmonary symptoms. Pancytopenia (32 000 platelets/mm$^3$), elevated lactate dehydrogenase, liver enzymes, ferritin and triglyceride levels were found. HIV-1 infection (viral load, 1.8×10$^6$ copies/ml; CD4 cell count, 185 cells/μl) and Pneumocystis jirovecii pneumonia were diagnosed. As thrombotic thrombocytopenic purpura and haemophagocytic syndrome were suspected, intravenous immunoglobulins were administrated. Transient Epstein–Barr virus and cytomegalovirus reactivations were recorded, without evidence for organ damage. Human herpesvirus type 8 PCR was not performed. Fluorodeoxyglucose positron emission tomography (FDG-PET)/CT disclosed hypermetabolic lymph nodes (standardised uptake value (SUV) max, 2.2 to 5.2) with an increased metabolic activity of the bone marrow, the spleen, the liver (SUVmax, 2.6, 3.6 and 2.1, respectively) and the colonic omentum, strongly evocative of malignant lymphoma (figure 1A). Two bone
narrow and three node biopsies found non-specific lymphocytic activation, only. Six months after the introduction of highly active antiretroviral therapy, the CD4 cell count reached 571/mm³, the viral load became undetectable and all pathologic findings disappeared, including abnormal metabolic activities on FDG-PET/CT (figure 1B). During HIV infection, activated lymphocytes increase their glucose utilisation and consequently the ¹⁸F-FDG uptake of lymphoid tissues. ¹⁸F-FDG uptake highly correlated with viral load and is mainly located in mesenteric and ileocecal areas during late disease.¹ Splenic ¹⁸F-FDG uptake seems to be less frequent in chronic HIV infection in comparison with other chronic viral infections, and mainly suggests HIV-associated lymphoma.¹–³ This report highlights the potential for viral infections such as chronic HIV-1 infection, to mimic a malignant lymphoma on FDG-PET/CT.

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