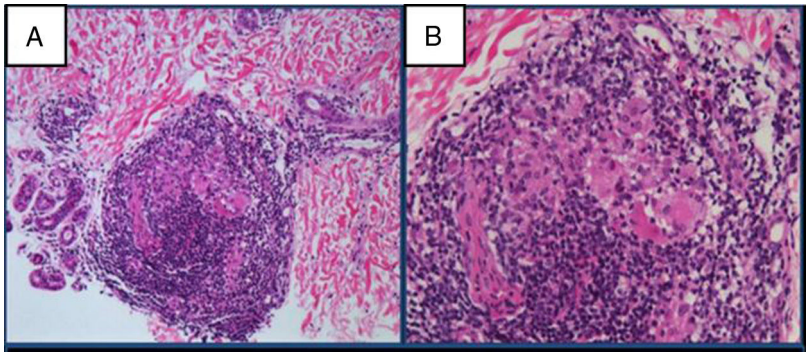

Figure 2. Pathology of the skin biopsy. Chronic, granulomatous inflammation with the presence of giant cells and a peripheral dense lymphocytic crown with a perivascular, periadnexal and perineural distribution. Haematoxylin-eosin ·10 (A) and ·20 (B).

a sensitivity of 50% in samples of nasal mucosa, earlobe and skin lesions. In tuberculoid (paucibacillary) leprosy, bacilli are very difficult to detect and genome amplification (PCR) techniques for the detection and identification of *M. leprae* have meant a significant advance in these cases.[6] The sensitivity of PCR in paucibacillary forms is from 50% to 80%. The GenoType Leprae-DR technique also makes it possible to analyse resistance to rifampicin, quinolones and dapsone.

The WHO recommends combined therapy[7]; double therapy with rifampicin and dapsone in paucibacillary forms for six months and, in multibacillary forms, adding a third drug (clofazimine) and prolonging the duration of treatment to 12 months. Chemoprophylaxis in cohabitants is not indicated.

In this case, we wish to highlight the importance of studying contacts who live with leprosy patients, especially with multibacillary forms, as leprosy is a curable disease if properly treated and this prevents transmission to other people.

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Conflicts of interest

The authors have no conflicts of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.eimce.2019.10.010.

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HIV-1 primary infection and acute hepatitis A: Beware of co-infection!

Primoinfección por VIH-1 y hepatitis A aguda: cuidado con la co-infección

Case report

We report a case of a 27-year-old man without relevant medical history. He was born in Costa Rica and was at that moment visiting Barcelona. He presented to the emergency department with a 3-day history of fever, headache and polyarthralgia. On clinical exam hepatosplenomegaly, jaundice and fever (39 °C) were documented. He referred a negative HIV serology performed 6 months before in his country, and reported sex with other men.

Initial laboratory test revealed altered liver parameters. Total bilirubin was 6.5 mg/dL (predominantly conjugated), aspartate aminotransferase 885 IU/L, alanine aminotransferase 2847 IU/L, alkaline phosphatase 238 IU/L, gamma-glutamyl transferase 864 IU/L and prothrombin time 67%.

HAV Ig G	positive	Chikungunya virus Ig M	negative
HAV Ig M	positive	Dengue virus Ig G	positive
HBs Ag	negative	Dengue virus Ig M	negative
Anti-HBs IgG Antibody	negative	EBV Ig G	positive
Anti-HBc Ig G Antibody	negative	EBV Ig M	negative
Anti- HBc Ig M Antibody	negative	CMV Ig G	positive
HCV Ig G	negative	CMV Ig M	negative
HCV RNA quantitative	undetectable	Anti-T. pallidum Ig G	positive
Antibodies HIV 1/2 + p24 Ag	negative	Anti-T. pallidum Ig M	negative
Chikungunya virus Ig G	negative	VDRL	negative

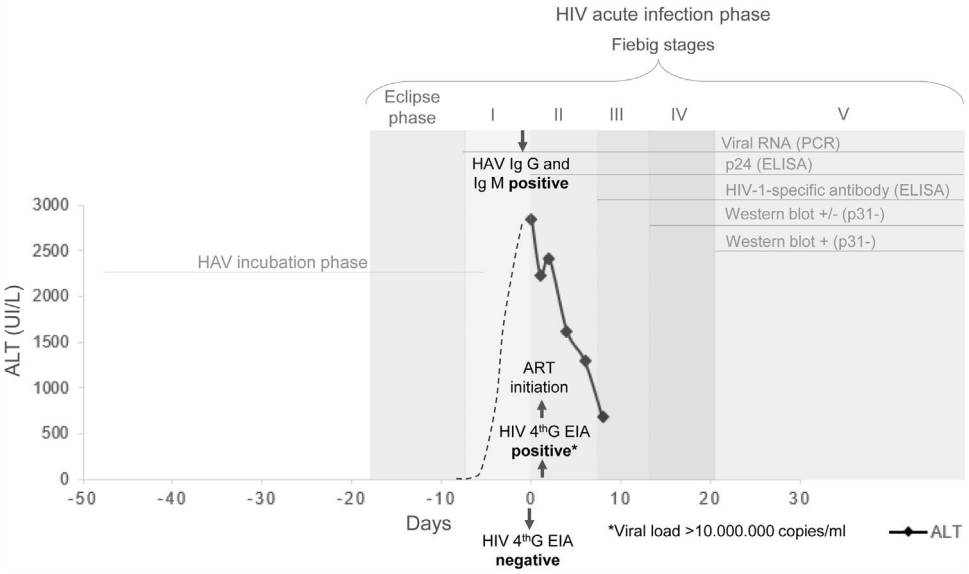

Fig. 1. (A) Serologies and molecular markers performed at admission. (B) Time line of HAV and HIV serological, biochemical and virological markers of the index case. On the background, laboratory staging of HIV infection. ART: antiretroviral therapy.

According to geographical origin of the patient, laboratory results and clinical presentation, primary hepatotropic viruses serologies (HAV, HBV, HCV, HDV, HEV) and viral load, EBV, CMV, dengue, chikungunya, syphilis serologies and HIV 1/2 4th generation EIA were requested (Fig. 1A). Acute hepatitis A infection was diagnosed, also with serological evidence to previous exposure to dengue, syphilis, CMV and EBV.

Re-exploring the patient 24 h later, polyadenopathies and rash were identified, unnoticed the day before. HIV-1 viral load was then requested, being >10,000,000 copies/ml and CD4+ T cells count was 224/mm^3 (17%). Retrospectively repeated EIA from that day resulted now positive, only 24 h later than the previously negative. Concomitant acute hepatitis A and severe acute primary HIV infection (PHI) were then the final diagnoses. TDF/FTC/dolutegravir at usual doses were immediately started with rapid remission of symptoms and liver tests progressive improvement over the following weeks. Virological, immunological and biochemical markers are shown in Fig. 1B.

A week later the patient's partner, a 65-year-old Spanish man, presented at hospital, reporting their last sexual intercourse being 18 days before. He had HIV negative test 2 years ago and was asymptomatic at that moment. HIV RNA, IgM and IgG HAV were requested. HAV vaccine/immune globulin was indicated depending on the results, but he decided to perform laboratory tests in another hospital. A week later he returned to the emergency department with fever and altered liver parameters. IgG and IgM HAV were positive confirming acute hepatitis A, HIV-1 viral load was undetectable, excluding HIV-1 infection.

Discussion

PHI can co-exist with other sexually transmitted infections (such as HAV) thus health professionals should be aware and always suspect them. PHI clinical symptoms may be non-specific and results of diagnostic tests rapidly modify, as in the current case, in 24 h. Sexual intercourse during PHI is associated to high transmission risk due to uncontrolled viral replication and extremely high HIV viral load in this phase, with frequent unawareness of infection.

About 20% of diagnosis in Spain and other European countries are made during acute infection (detection of p24 Ag and/or HIV-RNA in absence of HIV antibody) or during recent infection (HIV antibody detection up to 6 months after infection).[1–3] Immediate ART initiation controls symptoms, minimize viral reservoirs and decrease transmissibility.[4]

Hepatitis A is an acute, self-limiting disease transmitted by faecal–oral route through contaminated food or water or through

person-to-person contact, including sexual contact. If sexual transmission of HAV is suspected, particularly among men-who-have-sex-with-men (MSM), PHI should be excluded. PHI presents clinically as a mononucleosis-like or flu-like syndrome, easily missed in the context of a concomitant acute hepatitis. HAV incubation period could potentially be shorter than serological evidence of HIV infection. HIV RNA become positive 7–10 days after HIV exposure, p24-antigen approximately 14 days and HIV antibodies at around 21 days.[5] Thus, if concomitant or near exposure to HAV and HIV are suspected, HIV RNA should be requested to exclude PHI. HIV-VL is frequently extremely high in this period, with increased transmission risk. Complete seroconversion to HIV takes approximately 3 months until last WB band become positive (usually p31 band).

Sexually transmitted outbreaks of acute hepatitis A have been reported in the last years in Europe among MSM, particularly HIV-positive.[6–9] Hepatitis A infection is not more severe among HIV-infected individuals, but HAV viraemia is higher and longer, increasing transmission risk. In high-income countries anti-HAV IgG in general population is usually low (<50% by the age of 30 years).[10] Therefore, when HAV is introduced in groups at particular high-risk, as MSM population, outbreaks may occur, stressing the importance of preventive measures, particularly vaccination. As for HBV and HCV, HAV must also be considered as a potential co-infection in the context of PHI.

Conflict of interest

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Mycobacterium lentiflavum. Pulmonary infection in an immunocompetent patient☆

Mycobacterium lentiflavum. *Infección pulmonar en una paciente inmunocompetente*

Mycobacterium lentiflavum is considered as a slow-growing non-tuberculous mycobacterium (NTM). It was first described as a new species in 1996.[1]

We present the case of a 56-year-old woman, a former smoker with a five-pack-year history who had stopped 15 years previously, with no relevant medical history. She went to the Accident and Emergency department with haemoptysis, with an expectorated volume of up to 150 mL in 10 h. In view of her symptoms, she was given an emergency bronchoscopy, in which two blood clots were detected (in the right upper and lower lobes). Three days later, a second bronchoscopy was performed to complete the examination. The patient subsequently had a chest computed tomography (CT) scan, which showed bronchiectasis, nodular-looking images, discrete peribronchial ground-glass areas (likely related to recent bleeding), and hilar lymphadenopathy in the pathological range.

Three respiratory samples were taken (two bronchial aspirate obtained from the two different bronchoscopies and one sputum) and sent to the Microbiology laboratory, where microscopic examination was performed and they were cultured in solid medium (Coletsos) at 35 °C, and in liquid medium, using an automatic incubation and detection system: BACTEC MGIT 960 system (Becton Dickinson, Maryland, USA). In the auramine staining, acid-alcohol-fast bacilli were observed in all the samples. However, no *Mycobacterium tuberculosis* complex-specific DNA was detected by the commercial real-time PCR technique (BD MAX MDR-TB, BD New Jersey, USA) in any of the samples. While in hospital, the patient was started on empirical treatment with ceftriaxone and, in view of her adequate clinical progress, she was discharged to continue investigations as an outpatient. Identification of mycobacteria was still pending.

At two and a half months, the chest CT scan was repeated, showing persistence of bronchiectasis, nodular images and lymphadenopathy with disappearance of the discrete ground-glass areas (Fig. 1). The patient's respiratory function tests, autoimmunity tests and coagulation were all normal.

After two months of incubation, mycobacteria grew in the three respiratory samples, identified as *M. lentiflavum* by the strip-based reverse hybridisation and amplification method (GenoType® Mycobacterium AS [Hain Lifescience, Nehren, Germany]). As other aetiologies had been ruled out and the patient fulfilled clinical, radiological and microbiological criteria compatible with infec-

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