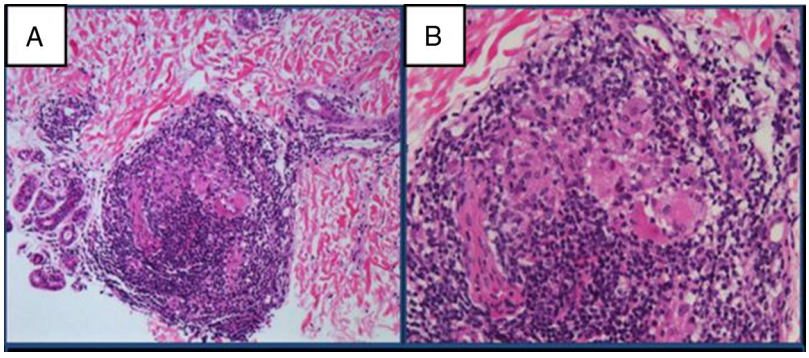


**Figura 2.** Anatomía patológica de la biopsia cutánea. Inflamación crónica y granulomatosa con presencia de células gigante y corona linfocitaria densa periférica de distribución perivascular, perianexial y perineural. Hematoxilina-eosina ×10 (A) y ×20 (B).

las técnicas de amplificación genómica (PCR) para la detección e identificación de *M. leprae* han supuesto un importante avance[6]. La sensibilidad de la PCR en formas paucibacilares está entre el 50 y el 80%. La técnica de Genotype-leprae-DR permite además analizar la resistencia a rifampicina, quinolonas y dapsona.

La OMS recomienda el tratamiento combinado[7]: doble terapia con rifampicina y dapsona en formas paucibacilares durante 6 meses y añade en las multibacilares un tercer fármaco (clofazimina), y prolonga la duración del tratamiento a 12 meses. La quimioprofilaxis en convivientes no está indicada.

En este caso queremos resaltar la importancia de estudiar a los contactos que conviven con pacientes con lepra, sobre todo formas multibacilares, ya que la lepra es una enfermedad curable, si se realiza el tratamiento adecuado y previene la transmisión a otras personas.

## Financiación

No se ha recibido ninguna financiación para la elaboración de este documento.

## Conflicto de intereses

Los autores no tienen ningún conflicto de intereses.

## Agradecimientos

Al Dr. Juan José Palacios Gutiérrez, laboratorio de Medicina, Área Microbiología. Hospital Universitario Central de Asturias, por el diagnóstico molecular de *M. leprae*.

Al Dr. José Ramón Gómez Echevarría, director médico de Lepra Fontilles, director de cursos de Leprología, por su ayuda con la orientación clínica, diagnóstico y tratamiento del caso.

A la Dra. Joanny Alejandra Duarte Luna, Servicio de Anatomía Patológica. Hospital Universitario de Getafe, por el diagnóstico anatomo-patológico de la biopsia de la piel.

## Anexo. Material adicional

Se puede consultar material adicional a este artículo en su versión electrónica disponible en doi:10.1016/j.eimc.2019.10.012.

## Bibliografía

1. Norman F, Fanciulli Ch, Pérez Molina JA, Monge-Maillo B, Lopez-Velez R. Imported and autochthonous leprosy presenting in Madrid (1989-2015): A case series and review of the literature. Travel Med Infect Dis. 2016;14:331e–49e.
2. Gonçalves Barreto J, Cipriani Frade MA, BernardesFilho F, da Silva MB, Spencer JS, Salgado GC. Leprosy in children. Curr Infect Dis Rep. 2017;19:23.
3. WHO. Global leprosy update, 2015: Time for action, accountability and inclusion. Weekly epidemiological record 2 September 2016, 91th year. No 35, 2016, 91, 405-420.
4. Ramos JM, Romero D, Belinchón I. Epidemiology of leprosy in Spain: The role of the international migration. PLoS Negl Trop Dis. 2016;10:e0004321, http://dx.doi.org/10.1371/journal.pntd.0004321.
5. Eichelmann K, González González SE, Salas-Alanis JC, Ocampo- Candiani J. Lepra: puesta al día. Definición, patogénesis, clasificación, diagnóstico y tratamiento. Actas Dermosifiliogr. 2013;104:554–63.
6. Nóbrega Martinez AN, Talhari C, Moraes MO, Talhari S. PCR-based techniques for leprosy diagnosis: From the laboratory to the clinic. PLoS Negl Trop Dis. 2014;8:e2655.
7. World Health Organization. Regional Office for South-East Asia. Guidelines for the diagnosis, treatment and prevention of leprosy, 2018.

Arantxa Berzosa-Sánchez [a,*], Beatriz Soto-Sánchez [a], Juana Begoña Cacho-Calvo [b] y Sara Guillén-Martín [a]

[a] *Servicio de Pediatría, Hospital Universitario de Getafe, Getafe, Madrid, España*
[b] *Servicio de Microbiología, Hospital Universitario de Getafe, Getafe, Madrid, España*

* Autor para correspondencia.
*Correo electrónico:* aranire@msn.com (A. Berzosa-Sánchez).

https://doi.org/10.1016/j.eimc.2019.10.012


## 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 |

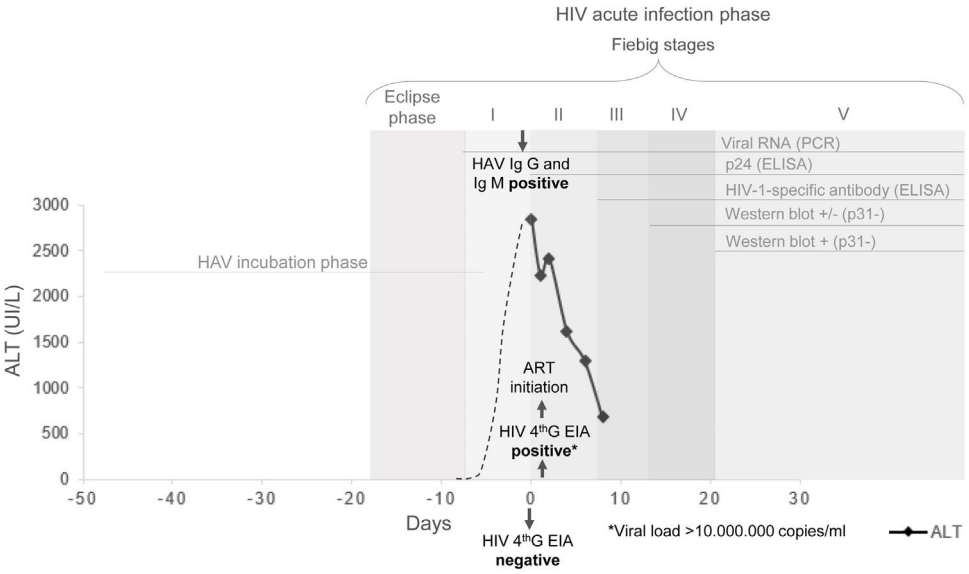


**Fig. 1.** (A) Serologies and molecular markers performed at admission. (B) Time line of HAV and HIV serological, biochemical and virological markers of the index case. On the background, laboratory staging of HIV infection. ART: antiretroviral therapy.

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

JA has received research funding from Gilead Sc and ViiV Healthcare, out of the current work.

NAD: none to declare.

## Bibliografía


1. Romero A, González V, Granell M, Matas L, Esteve A, Martró E, et al. Recently acquired HIV infection in Spain (2003–2005): introduction of the serological testing algorithm for recent HIV seroconversion. Sex Transm Infect. 2009;85:106–10.
2. Le Vu S, Le Strat Y, Barin F, Pillonel J, Cazein F, Bousquet V, et al. Population-based HIV-1 incidence in France, 2003–08: a modelling analysis. Lancet Infect Dis. 2010;10:682–7.
3. EACS guidelines. Version 10.0; 2019.
4. Fisher M, Pao D, Brown AE, Sudarshi D, Gill ON, Cane P, et al. Determinants of HIV-1 transmission in men who have sex with men: a combined clinical, epidemiological and phylogenetic approach. AIDS. 2010;24:1739–47.
5. Fiebig EW, Wright DJ, Rawal BD, Garrett PE, Schumacher RT, Peddada L, et al. Dynamics of HIV viremia and antibody seroconversion in plasma donors: implications for diagnosis and staging of primary HIV infection. AIDS. 2003;17:1871–9.
6. Freidl GS, Sonder GJ, Bovée LP, Friesema IH, van Rijckevorsel GG, Ruijs WL, et al. Hepatitis a outbreak among men who have sex with men (MSM) predominantly linked with the EuroPride, the Netherlands, July 2016 to February 2017. Eurosurveillance. 2017;22:1–5.
7. Comelli A, Izzo I, Casari S, Spinetti A, Bergamasco A, Castelli F. Hepatitis a outbreak in men who have sex with men (MSM) in Brescia (Northern Italy), July 2016–July 2017. Infez Med. 2018;26:46–51.
8. Charre C, Ramiere C, Roque-Afonso AM, Chidiac C, Zoulim F, Godinot M, et al. Hepatitis a outbreak in HIV-infected MSM and in PrEP-using MSM despite a high level of immunity, Lyon, France, January to June 2017. Eurosurveillance. 2017;22:1–4.
9. Beebeejaun K, Degala S, Balogun K, Simms I, Woodhall SC, Heinsbroek E, et al. Outbreak of hepatitis A associated with men who have sex with men (MSM), England, July 2016 to January 2017. Eurosurveillance. 2017;22:1–6.
10. Carrillo-Santisteve P, Tavoschi L, Severi E, Bonfigli S, Edelstein M, Byström E, et al. Seroprevalence and susceptibility to hepatitis A in the European Union and European Economic Area: a systematic review. Lancet Infect Dis. 2017;17:e306–19.



Natalia Anahí Díaz [a,b], Juan Ambrosioni [b,*]

[a] *Infectious Diseases Service, Hospital Cesar Milstein, Buenos Aires, Argentina*

[b] *Infectious Diseases Service, Hospital Clinic-IDIPABS, Barcelona, Spain*

* Corresponding author.

*E-mail address:* AMBROSIONI@clinic.cat (J. Ambrosioni).



https://doi.org/10.1016/j.eimc.2019.11.003

0213-005X/



## *Mycobacterium lentiflavum*. Infección pulmonar en una paciente inmunocompetente

## Mycobacterium lentiflavum. *Pulmonary infection in an immunocompetent patient*

*Mycobacterium lentiflavum* se considera una micobacteria no tuberculosa (MNT) de crecimiento lento. Fue descrita como una nueva especie por primera vez en 1996[1].

Presentamos el caso de una mujer de 56 años, exfumadora desde hace 15 años, con un índice paquetes-años de 5. Sin antecedentes médicos de interés. Acudió al servicio de urgencias por presentar hemoptisis, con un volumen expectorado de hasta 150 ml en 10 h. El cuadro clínico motivó la realización de una broncoscopia urgente, en la que se observaron 2 coágulos de sangre (en el lóbulo superior y el lóbulo inferior derecho). A los 3 días se repitió una segunda broncoscopia para completar la exploración. Posteriormente, se realizó una tomografía computarizada (TC) torácica en la que destacó la existencia de bronquiectasias, imágenes de aspecto nodular, discretas áreas en vidrio deslustrado peribronquiales (en probable relación con sangrado reciente) y adenopatías hiliares en rango patológico.

Se tomaron 3 muestras respiratorias (2 aspirados bronquiales obtenidos en 2 broncoscopias distintas y un esputo) y se enviaron al laboratorio de Microbiología, donde se realizó examen microscópico y se cultivaron tanto en medio sólido (Coletsos) a 35 °C, como en medio líquido, utilizando el sistema de incubación y detección automática: sistema Bactec MGIT 960 (Becton Dickinson, Maryland, EE. UU.). En la tinción de auramina se observaron aislados bacilos ácido-alcohol resistentes en todas las muestras; sin embargo, no se detectó ADN especifico de *Mycobacterium tuberculosis* complex mediante la técnica comercial de PCR en tiempo real (BD MAX MDR-TB, BD New Jersey, EE. UU.) en ninguna de las muestras. Durante el ingreso, se inició tratamiento empírico con ceftriaxona y, dada la adecuada evolución clínica, fue dada de alta para continuar con el estudio de forma ambulatoria. Quedó pendiente la identificación de micobacterias.

A los 2 meses y medio se repitió la TC torácica, que mostró persistencia de las bronquiectasias, imágenes nodulares y adenopatías con desaparición de las discretas áreas en vidrio deslustrado (fig. 1). Las pruebas de función respiratoria, análisis de autoinmunidad y coagulación fueron normales.

Tras 2 meses de incubación, creció una micobacteria en las 3 muestras respiratorias, que se identificó como *M. lentiflavum* por el método de amplificación e hibridación reversa en tira (GenoType® Mycobacterium AS [Hain, Lifescience, Nehrn, Alemania]). Dado que se habían descartado otras etiologías y la paciente cumplía criterios clínicos, radiológicos y microbiológicos compatibles con infección por *M. lentiflavum*[2], se decidió iniciar tratamiento con azitromicina, etambutol y rifampicina, con adecuada tolerancia