NK cell activity, ferritin ≥ 500 μg/L and soluble CD25 ≥ 2400 U/mL, being the last three considered as new criteria.[3,8]

The initial management to secondary HLH includes the administration of etoposide and dexamethasone, both of which could be restarted and increased gradually in case of reactivation.[3]

Additionally, disseminated histoplasmosis is an uncommon opportunistic infection in HIV patients caused by Histoplasma capsulatum fungi, with the Capsulatum variant being more likely in America and the Duboisii variant more likely in Africa.[1,4,8] Some cases of HLH due to disseminated histoplasmosis have been described, mostly of them caused by *H. capsulatum* var. capsulatum, but they are infrequent.

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Anterior bilateral uveitis and acute parvovirus B19 infection☆

Uveítis anterior bilateral e infección aguda por parvovirus B19

Parvoviruses are single-stranded DNA viruses, the only one of which known to affect humans is B19. Parvovirus B19 is a ubiquitous micro-organism that causes common infections of respiratory transmission. The prevalence of the virus in the general adult population ranges from 50% to 80%.[1]

The course of the infection usually features two phases: a viraemia phase, in which the patient presents flu-like signs and symptoms with malaise, myalgia, fever, headache and chills; and a second phase, characterised by the onset of dermatosis, vasculitis, joint diseases and other general symptoms.[2] In healthy subjects, the infection may go unnoticed or be concomitant with infectious erythema or joint diseases. In adults, skin lesions are usually absent and joint impairment is most notable, with a particular propensity for the cervical spinal vertebrae, shoulders, elbows, wrists, ankles and feet.[3]

The infection can be diagnosed in the early phase with the demonstration of IgM antibodies against the virus, and in the disease resolution period by seroconversion to specific IgG antibodies. Treatment of this infection is symptomatic and based on analgesics and anti-inflammatory agents.

We report the case of a 46-year-old male who visited the emergency department due to headache radiating towards the cervical area with night sweats and measurable fever. The patient had also been suffering from back pain and joint pain in the knees and ankles for several weeks. He had already sought treatment for these symptoms and been diagnosed with influenza. Laboratory testing in the emergency department showed C-reactive protein (CRP) 1.6 mg/dl and leukocytosis 12,400/mm.[3] A lumbar puncture ruled out meningitis.

As the patient presented significant bilateral conjunctival hyperaemia, he was assessed by Ophthalmology. The ophthalmological examination showed bilateral Tyndall++ with visual acuity of the unit, and examination of the eye fundus revealed no signs of vitritis or retinitis. The patient was diagnosed with bilateral anterior uveitis.

A decision was made to admit him to the internal medicinal department to continue evaluating his systemic condition. He underwent an echocardiogram which, along with negative blood cultures, ruled out endocarditis. Urine sediment and urine culture were also normal. No influenza virus or respiratory syncytial virus (RSV) was detected in the patient's pharyngeal exudate, and a Mantoux test was negative. A chest X-ray showed no pleural effusion, and the patient's knees and shoulders showed no abnormalities. Serologies were all negative (*Brucella* spp., Toxoplasma, syphilis, hepatitis B and C, human immunodeficiency virus [HIV], *Coxiella* spp., *Borrelia* spp. and *Rickettsia* spp.) with IgG positivity for herpes simplex virus, Epstein–Barr virus and IgG and IgM antibodies for parvovirus. Autoimmunity was also negative (antinuclear antibodies [ANAs], antimitochondrial antibodies [AMAs] and extractable nuclear antibodies [ENAs]). Ultimately, the patient's diagnosis was probable acute infection due to parvovirus B19 and bilateral anterior uveitis.

It is estimated that only 20% of cases of anterior uveitis are of infectious origin; they are largely caused by viruses belonging to the family Herpesviridae.[4] However, there are few reported cases of infection with parvovirus B19 and uveitis. The four previously published cases (Table 1) were patients under 18 years of age; this case was the first one in an adult.[5–8] There are

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Table 1
Published cases of uveitis associated with parvovirus B19 infection.

Patient	Age (years)	Sex	Erythema	Autoantibodies	Treatment for uveitis	Reference
1	18	Female	Yes	Unknown	Topical corticosteroid	5
2	6	Female	No	Rheumatoid factor	Topical corticosteroid	6
3	5	Female	Yes	ANAs	Topical corticosteroid	7
4	9	Male	Yes	Negative	Topical corticosteroid	8
5	46	Male	No	Negative	Topical corticosteroid	Case reported

two theories with regard to ocular involvement in parvovirus B19 infection: one is direct invasion by the virus; the other is induction of autoimmunity.[8] In our case, no autoantibodies were detected, hence we presumed direct invasion by the virus to be the cause of the uveitis. Furthermore, our patient did not present the typical infectious erythema either. He was treated symptomatically with paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs); his systemic signs and symptoms improved. His bilateral anterior uveitis responded to topical corticosteroid treatment with no associated complications and good subsequent management.

We report the only case of anterior bilateral uveitis associated with probable acute parvovirus B19 infection in an adult that has been published to date. Influenza-like signs and symptoms along with joint pain and subsequent development of uveitis with IgM positivity for parvovirus B19 led to the diagnosis. Uveitis should be considered an uncommon complication in the second phase of parvovirus B19 infection. Further similar cases need be reported if we are to understand the pathophysiological mechanisms of eye involvement in cases of infection with this virus.

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Absence of maternal HHV-6-specific T-cell response in the setting of human Herpesvirus-6 chromosomal integration: Potential impact on congenital infection

Ausencia de respuesta de células T específicas materna en el contexto de la integración cromosómica del herpesvirus-6 humano: impacto potencial en la infección congénita

Human herpesvirus 6 (HHV-6) is a ubiquitous pathogen, whose primary infection is often asymptomatic and manifests as exanthema subitum in only 20% of cases.[1]

HHV-6 has the capacity to integrate into the human genome (ciHHV-6). However, the mechanisms involved in HHV-6 integration are still largely unknown.[2–4] HHV-6 Integration into the gametes results in Mendelian transmission to offspring, with 1% of the worldwide human population carrying ci-HHV-6.[5]

It is well established that congenital HHV-6 infection results from the germline passage of ci-HHV-6 and the transplacental passage of maternal HHV-6 infection by either maternal primary infection or maternal virus reactivation.[6] The severity of HHV-6 congenital disease largely depends on the intensity and duration of viral replication, and is related to the maternal anti-HHV6 immune response and the implementation of antiviral treatment. HHV-6 congenital infections are usually asymptomatic[6,7] most likely due to the immune protection provided by maternal immunity. Nevertheless, the impact of the absence of maternal HHV-6-specific immune response in the setting of ci-HHV6 on the development of congenital HHV-6 infections is unknown.[8]

We describe the case of a 35-year-old pregnant woman without a relevant clinical history. Fetal screening sonography at week 12 of the pregnancy showed an isolated severe fetal pleural effusion. Amniocentesis was subsequently performed with a negative result for trisomy 13, 18 and 21. Nevertheless, real-time PCR in amniotic fluid was positive for HHV-6A and negative for other infections (varicella zoster virus, cytomegalovirus, Epstein–Barr virus, toxoplasmosis, herpesvirus 1 and 2 and Parvovirus B19). The patient presented a self-limited abdominal rash at weeks 8 of pregnancy as the only clinical symptom. Given the presence of massive fetal pleural effusion, the patient terminated the pregnancy at 22 weeks. The fetal autopsy showed a prominent pleural effusion, although no other macroscopic alterations were identified. Since HHV-6A had been previously detected in the amniotic fluid, the presence of HHV-6 real-time PCR was investigated in fetal tissues. Hence, all fetal tissues evaluated, including brain and lung tissues, showed the presence of HHV-6A. Four months after the abortion, the patient was referred to the infectious diseases unit of the hospital. HHV-6 viral loads were monitored in the patient by quantitative PCR in plasma (5.083 copies/ml) and whole blood (4.896.701 copies/ml) samples. A HHV-6 antiviral treatment with 900 mg of oral valganciclovir every 12 h was initiated. After four