lymphohistiocytosis (HLH) due to *Bartonella henselae* in a human immunodeficiency virus (HIV) patient with the acquired immunodeficiency syndrome [1]. Although the idea that *Bartonella* itself induced HLH is intriguing and might be an overlooked cause in HLH cases, the report raises questions regarding the suggested relationship.

First, it remains unclear why the authors favor *Bartonella henselae* over other plausible causes of HLH. The patient has an advanced HIV infection with uncontrolled Epstein-Barr virus (EBV) and human herpesvirus type 8 (HHV8) replication. These are recognized triggers for HLH, especially in HIV-infected patients [2–5]. Moreover, other established triggers in HLH patients with HIV such as cytomegalovirus or histoplasmosis infection were not ruled out.

Second, alternative diagnoses than bartonellosis with secondary HLH can still be considered. Peliosis hepatitis is not *Bartonella* specific, and the presence of *Bartonella* in liver tissue was not confirmed by immunohistochemistry or by polymerase chain reaction. A HIV infection with Centers for Disease Control and Prevention (CDC) B and CDC C events can also explain the clinical context. The same accounts for EBV or HHV8 related multicentric Castleman disease (MCD). A normal positron emission tomography can still lower the MCD likelihood [6, 7]. Histopathological lymph node examination is the preferred method to rule out MCD in pro-inflammatory patients.

Third, longitudinal measurements of bacterial, viral, and HLH related clinical or immunological markers are not presented. However, these factors correlate with HLH activity and could have supported the suggested relationship between bartonellosis and HLH [8–10].

Last, HLH in adults is a reflection of a disbalanced immunological reaction, often induced by an infectious trigger. Treatment must be directed towards this trigger, sometimes facilitated by immunosuppressive and chemotherapeutic agents. The observed treatment response following a single infusion of etoposide, antiretroviral therapy and doxycycline only reflects that HLH is secondary to one of the treated triggers and does not favor *Bartonella*-induced HLH.

In conclusion, we are not convinced that we should consider *Bartonella henselae* in idiopathic HLH cases based on this single observation in which pivotal information needs to be clarified. The case is unique in a way that *Bartonella* has never been demonstrated in a patient with HLH. However, in our opinion there is not enough evidence presented to demonstrate the unique role for *Bartonella* causing HLH in this case to justify a general recommendation for immunocompromised patients with HLH.

**Note**

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