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AA Amyloidosis Revealing HHV-8-Associated Multicentric Castleman Disease: A Case Report and Literature Review

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Abstract

Background/Objectives: Multicentric Castleman disease (MCD) is a rare systemic lymphoproliferative disorder driven by interleukin-6 (IL-6) dysregulation and frequently associated with human herpesvirus 8 (HHV-8) infection. AA amyloidosis is a severe complication of chronic inflammatory states, resulting from extracellular deposition of serum amyloid A (SAA) protein. We report a case of HHV-8-associated MCD revealed by AA amyloidosis with hepatic and salivary gland involvement.

Methods: A 40-year-old Algerian woman with a six-year history of progressive gastrointestinal symptoms and chronic inflammation was evaluated for biopsy-proven AA amyloidosis involving the liver and minor salivary glands. Diagnostic workup included HHV-8 PCR, autoantibody screening, MEFV gene mutation analysis, bone marrow examination, and CT imaging.

Results: HHV-8 viremia was positive (25,000 copies/mL), while autoimmune and genetic testing, including MEFV mutation screening, were negative. Clinical findings included chronic hypotension, normocytic anemia with hemolytic features, and subcentimeter lymphadenopathy on CT. A diagnosis of HHV-8-associated MCD with secondary AA amyloidosis was established. The patient was treated with rituximab (375 mg/m² weekly for four weeks) combined with valganciclovir, resulting in viral clearance, normalization of inflammatory markers, and improvement in hemoglobin.

Conclusions: MCD should be considered in patients with unexplained chronic inflammation and AA amyloidosis. Early recognition and targeted therapy with rituximab are essential to prevent irreversible organ damage. Excisional lymph node biopsy with HHV-8 immunostaining is critical for definitive diagnosis.

Keywords: Castleman Disease, HHV-8, AA Amyloidosis, Rituximab, Proteinuria

1. Introduction

Castleman disease (CD) is a rare and heterogeneous lymphoproliferative disorder encompassing unicentric and multicentric forms. Multicentric Castleman disease (MCD) is characterized by systemic inflammation, generalized lymphadenopathy, and cytokine dysregulation, particularly involving interleukin-6 (IL-6) [1, 2]. MCD can be classified into HHV-8-associated, idiopathic, and POEMS-associated forms [2]. HHV-8 plays a central pathogenic role through viral IL-6 production and B-cell proliferation. AA amyloidosis is a severe complication of chronic inflammatory conditions resulting from persistent elevation of serum amyloid A protein and its tissue deposition. When associated with MCD, it significantly worsens prognosis, particularly in cases with renal involvement [3, 4].

Furthermore, the management of rare diseases such as MCD in resource-limited settings poses significant challenges. Algeria, as a middle-income country, faces constraints in access to targeted therapies and specialized diagnostic tools. Rituximab, a key therapeutic agent for HHV-8-associated MCD, was introduced in Algeria in 2006 but remains a high-cost medication with very restricted prescription, limited to specialized hospital settings and requiring specific authorization for off-label indications.

This case report documents what appears to be the first use of rituximab for the treatment of Castleman disease in the Algerian healthcare system, highlighting both therapeutic progress and persistent challenges in the management of rare diseases in resource-limited settings.

2. Case Report

2.1 Patient Information

A 40-year-old woman from the high plateaus of Algeria was admitted to the Nephrology Department of Bab El Oued University Hospital in December 2024 for evaluation of biopsy-proven AA amyloidosis. Her medical history included five pregnancies (one living child, one cesarean section, and three spontaneous abortions). Her family history was notable for maternal death and a relative's death from undocumented gastrointestinal neoplasia.

2.2 History of Present Illness

The patient's symptoms began six years prior to admission (around 2018) with progressive and recurrent episodes of nausea, vomiting, and diffuse abdominal pain. Over the following years, she underwent multiple medical consultations and investigations. In 2022, a computed tomography (CT) scan revealed subcentimeter lymph nodes in both thoracic and abdominal regions (Fig 1). Subsequent biopsies of the liver (Fig 2) and minor salivary glands (Fig 3) confirmed the diagnosis of AA amyloidosis, with positive immunostaining for anti-SAA antibodies. She had previously been hospitalized in other centers for etiological workup, but no definitive diagnosis had been established.

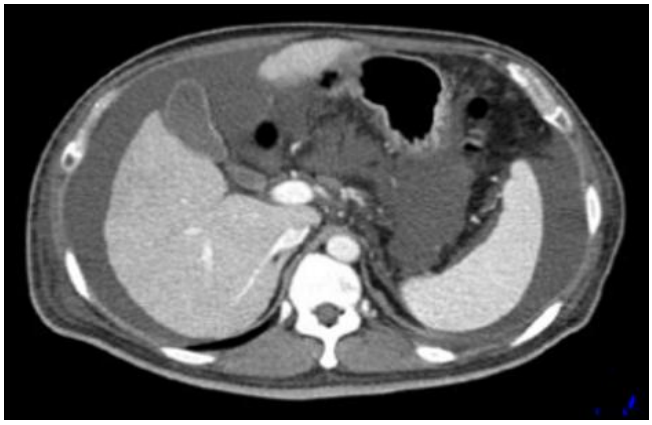

Fig 1: Chest, abdomen and pelvis CT scan showing hepatosplenomegaly with subcentimeter lymphadenopathy

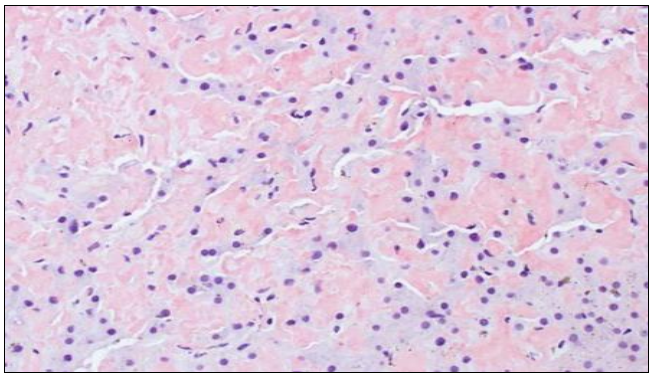

Fig 2: Hepatic amyloid deposition. Hematoxylin and eosin stain, low magnification: near-complete effacement of the acinar architecture with extensive deposition of pale eosinophilic, hyaline, amorphous, acellular material within the sinusoids and portal tracts

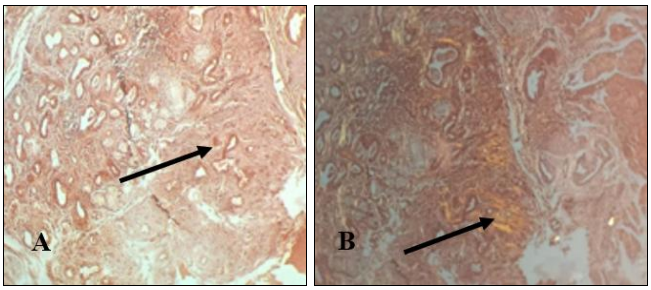

Fig 3: Minor salivary gland biopsy. (A) Medium-magnification view showing chronic lymphocytic sialadenitis (Chisholm and Mason grade 2) on hematoxylin and eosin staining. (B) Congo red staining under polarized light demonstrating yellow-green birefringence around salivary ducts and in perivascular areas, confirming AA amyloid deposition

2.3 Clinical Findings on Admission

On admission, the patient was in relatively good general condition, afebrile, and euvolemic, with no lower limb edema. Blood pressure was chronically low at 90/60 mmHg, not associated with orthostatic symptoms. Cardiorespiratory examination was normal. Mild skin pallor was noted, and she reported a history of intermittent ecchymotic spots. Abdominal examination was unremarkable. Urinalysis revealed proteinuria (1.2 g/24 h) and leukocyturia.

2.4 Diagnostic Workup

Laboratory investigations performed in April 2024 revealed normocytic anemia with a hemoglobin level of 10.8 g/dL, while white blood cell and platelet counts were within normal ranges. Inflammatory markers showed an elevated erythrocyte sedimentation rate (ESR) of 62 mm/h, with a C-reactive protein (CRP) level of 151 mg/L. Renal function was preserved, with a serum creatinine level of 0.53 mg/dL (approximately 46.8 µmol/L) and normal urea levels (0.3 g/L). Liver function tests demonstrated mild cytolysis, with an aspartate aminotransferase (AST) level of 43.50 U/L and elevated total bilirubin at 4.91 mg/dL, predominantly unconjugated. Hemolysis workup revealed haptoglobin levels < 0.0 g/L (normal range 0.3-2.0 g/L), elevated lactate dehydrogenase (LDH) at 600 U/L (normal < 250 U/L), and the presence of schistocytes on peripheral blood smear. The direct antiglobulin test (Coombs test) was negative. These findings were suggestive of mild hemolytic anemia with a negative autoimmune workup, consistent with a microangiopathic or inflammatory mechanism.

Immunological evaluation showed positive HHV-8 DNA in peripheral blood, with a quantitative PCR viral load of 25,000 copies/mL. Serological tests for HIV, hepatitis B virus (HBV), and hepatitis C virus (HCV) were negative. Autoimmune markers, including antinuclear antibodies (ANA), anti-double-stranded DNA (anti-dsDNA), and antineutrophil cytoplasmic antibodies (ANCA), were negative, while complement levels (C3 and C4) were within normal limits. Serum protein electrophoresis revealed hypoalbuminemia associated with decreased alpha-1, alpha-2, and beta-1 globulin fractions, along with polyclonal hypergammaglobulinemia.

Bone marrow examination demonstrated normal cellularity (grade 2-3) with no evidence of hematological malignancy within the limits of the specimen analyzed. Genetic testing for familial Mediterranean fever, including *MEFV* gene mutation screening, was negative. Imaging studies, including CT of the chest, abdomen, and pelvis, showed

hepatosplenomegaly associated with subcentimeter lymphadenopathy, without evidence of renal abnormalities.

2.5 Diagnosis and Management

Based on the constellation of findings, the diagnosis of HHV-8-associated multicentric Castleman disease with secondary AA amyloidosis was established. The patient was initiated on rituximab (375 mg/m² weekly for 4 weeks) along with valganciclovir for active HHV-8 infection.

2.6 Follow-Up and Clinical Improvement

After eight months of treatment with rituximab, the patient experienced a mild relapse with low viral load, necessitating an additional course of rituximab. Subsequent biological evolution indicated HHV-8 clearance, improved liver function, normalization of inflammatory markers, and an increase in hemoglobin to 12 g/dL, with no evidence of schistocytes on blood smear. The evolution of key biological parameters before and after treatment is summarized in Fig 4.

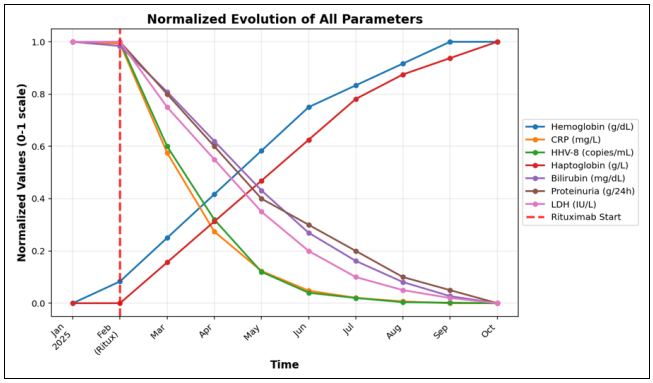

Fig 4: Evolution of key clinical and biological parameters before and after rituximab therapy. Hemoglobin (g/dL), CRP (mg/L), HHV-8 viral load (copies/mL), haptoglobin (g/L), total bilirubin (mg/dL), proteinuria (g/24h), and LDH (IU/L) are shown over time. Rituximab was initiated in February 2025. Rapid normalization of CRP, HHV-8 viral load, and LDH was observed, with improvement in hemoglobin and resolution of hemolysis markers

3. Discussion

We report a rare case of HHV-8-associated multicentric Castleman disease (MCD) revealed by AA amyloidosis with hepatic and salivary gland involvement. This observation highlights several key aspects, including diagnostic delay, pathophysiological mechanisms, renal implications, and therapeutic challenges.

3.1 Diagnostic Consensus and the Critical Role of Lymph Node Biopsy

The diagnosis of HHV-8 -associated MCD rests on well-established international consensus criteria (Fajgenbaum *et al*., 2017). According to these criteria, the diagnosis requires:

Major criteria (both mandatory):

- Histopathological confirmation on an excisional lymph node biopsy showing characteristic features (atrophic germinal centers, expanded mantle zones with "onion-skin" appearance, interfollicular plasmacytosis) and positive HHV-8 immunohistochemistry (LANA-1 staining in plasmablasts).
- Multicentric lymphadenopathy involving at least two nodal stations, with lymph nodes ≥ 1 cm in short axis on imaging.

Minor criteria (at least two, including at least one laboratory abnormality):

- Constitutional symptoms (fever, night sweats, weight loss, fatigue)
- Hepatosplenomegaly
- Effusions (pleural, ascites, edema)
- Laboratory abnormalities: elevated CRP (> 10 mg/L), anemia (Hb < 12.5 g/dL in men, < 11.5 g/dL in women), thrombocytopenia (< 150 × 10⁹/L) or thrombocytosis (> 400 × 10⁹/L), hypoalbuminemia (< 3.5 g/dL), polyclonal hypergammaglobulinemia, or renal impairment/proteinuria.

In our patient, despite six years of symptoms and the presence of several minor criteria (chronic inflammatory syndrome, anemia, hypergammaglobulinemia, splenomegaly, and proteinuria), the diagnosis was delayed because a lymph node biopsy with HHV-8 immunostaining was not performed earlier. Subcentimeter lymph nodes were noted on imaging but were not biopsied, partly because they were not frankly enlarged and the clinical focus was on gastrointestinal and hepatic manifestations. This case underscores a critical educational point: excisional lymph node biopsy must be performed in any patient with unexplained chronic inflammation and multicentric lymphadenopathy, however subtle, when Castleman disease is suspected. The presence of AA amyloidosis should further raise suspicion, as it is a well-recognized complication of untreated MCD [3]. Relying solely on imaging or on less invasive biopsies (liver, salivary gland) may confirm the complication (amyloidosis) but not the underlying etiology. Definitive diagnosis of HHV-8-associated MCD requires direct demonstration of the virus in lymphoid tissue.

3.2 Diagnostic Challenges and Clinical Presentation

MCD is often difficult to diagnose due to its rarity and nonspecific clinical features. Symptoms may evolve insidiously over several years before the disease is recognized. In our patient, gastrointestinal complaints, intermittent ecchymotic lesions, chronic hypotension, and persistent inflammation persisted for six years prior to definitive diagnosis, illustrating the indolent yet progressive nature of HHV-8-associated MCD [1, 2, 5]. Such prolonged diagnostic latency has been described in previous studies, where patients frequently undergo multiple inconclusive evaluations before the correct diagnosis is reached [2].

The presence of subcentimeter lymphadenopathy, as observed in our case, may further complicate diagnosis, as it can be easily overlooked or considered nonspecific. However, even minimal lymph node enlargement in the setting of chronic inflammation should raise suspicion for a lymphoproliferative disorder. Understanding the pathophysiology of this disorder helps explain why such subtle clinical signs are associated with severe complications such as AA amyloidosis.

The etiological investigation of AA amyloidosis remains a cornerstone of our clinical practice. In a 20-year retrospective analysis from our center, we highlighted the diverse underlying causes of AA amyloidosis in the Algerian population, emphasizing that a systematic

diagnostic approach is essential to identify treatable inflammatory conditions [6]. The present case further underscores this principle, as the diagnosis of HHV-8-associated MCD was established only after a six-year diagnostic odyssey, and targeted therapy with rituximab resulted in favorable outcomes.

3.3 Pathophysiology and Link with AA Amyloidosis

The pathogenesis of HHV-8-associated MCD is driven by viral infection of B cells, leading to overproduction of viral IL-6 and human IL-6. This cytokine storm induces systemic inflammation, hypergammaglobulinemia, and hepatic synthesis of serum amyloid A (SAA), which is the precursor protein of AA amyloidosis [2, 7]. Persistent elevation of SAA results in its misfolding and extracellular deposition as amyloid fibrils in various organs, particularly the kidneys, liver, and gastrointestinal tract (Bernabei *et al*., 2020). This mechanism explains the occurrence of AA amyloidosis in chronic inflammatory states, including MCD.

Although AA amyloidosis is a well-known complication of chronic inflammatory diseases, its association with Castleman disease remains rare. Previous reports suggest that this complication is more frequently observed in the plasmacytic subtype, which is characterized by high IL-6 levels [3]. Our case supports this mechanism, given the chronic inflammatory profile and hypergammaglobulinemia observed.

3.4 Renal Involvement and Prognostic Implications

Renal involvement is one of the most significant determinants of prognosis in AA amyloidosis. It typically manifests as proteinuria, which may progress to nephrotic syndrome and eventually end-stage renal disease if the underlying inflammatory process is not adequately controlled [8, 9]. In our patient, renal function was preserved at presentation, but the presence of proteinuria (1.2 g/24 h) suggests early renal amyloid deposition. This finding underscores the importance of early detection and close nephrological monitoring.

The critical therapeutic objective in AA amyloidosis is suppression of the circulating precursor protein, SAA, to concentrations below 10 mg/L. This target, initially established [10], has been consistently associated with improved overall survival and a reduced risk of progressive renal failure. More recently, Basset *et al* [11] developed and validated novel staging systems for AA amyloidosis that integrate SAA concentration, estimated glomerular filtration rate, and proteinuria to accurately predict both overall survival and the risk of progression to end-stage renal disease. These prognostic tools reinforce the concept that timely diagnosis and sustained control of the underlying inflammatory disorder are essential to prevent irreversible organ damage.

Given the central role of chronic inflammation in the pathogenesis of AA amyloidosis, controlling the underlying disease is paramount. In HHV-8-associated MCD, this relies on targeted therapies directed against the virus and the cytokine dysregulation it induces.

3.5 Therapeutic Considerations

Management of HHV-8-associated MCD in HIV-negative patients remains challenging due to the scarcity of large controlled studies. However, case reports and small case series support the use of rituximab-based therapy as an effective immunotherapeutic option. Rituximab, a monoclonal anti-CD20 antibody, targets B lymphocytes that harbor HHV-8 and contribute to cytokine dysregulation and systemic inflammation [12]. Clinical evidence from case reports suggests that rituximab alone can induce clinical remission in HHV-8-positive, HIV-negative MCD patients with good performance status and without life-threatening organ failure [13]. In addition, combination approaches using rituximab with cytotoxic chemotherapy and antiviral therapy such as valganciclovir have achieved complete remission in some HIV-negative MCD cases [14].

Recent therapeutic advances have expanded the armamentarium beyond rituximab. In HHV-8-associated MCD, rituximab remains the cornerstone, but combination with liposomal doxorubicin or etoposide may be required in severe cases [15, 16]. For the HHV-8-negative (idiopathic) form, siltuximab has demonstrated superior progression-free survival compared to rituximab-based regimens in a recent meta-analysis [7]. Emerging therapies currently under investigation include JAK inhibitors (ruxolitinib) for patients refractory to anti- IL-6 therapy [4], as well as mTOR inhibitors (sirolimus) [2], BTK inhibitors, and TNF-alpha inhibitors [4]. The 2024 NCCN guidelines have also introduced combination regimens such as BCD (bortezomib, cyclophosphamide, dexamethasone) and RVD (rituximab, bortezomib, dexamethasone) for severe forms of iMCD. These developments underscore the importance of precise etiological classification to guide treatment selection in MCD.

In the Algerian context, the availability of targeted therapies for rare diseases is limited. Rituximab was introduced in Algeria in 2006 but remains a high-cost medication with very restricted prescription, limited to specialized hospital settings and often requiring specific authorization for off-label indications. To our knowledge, this case represents the first documented use of rituximab for Castleman disease in Algeria. The successful response in our patient, despite limited access to advanced molecular monitoring and the absence of routine IL-6 or SAA measurements, underscores the potential for achieving clinical remission with targeted immunotherapy even in resource-limited healthcare systems. This experience may serve as a reference for clinicians in other low- and middle-income countries facing similar diagnostic and therapeutic constraints.

Overall, early initiation of rituximab therapy in HHV-8-associated MCD, even in the absence of HIV infection, is crucial to achieve disease control, prevent organ damage, and reduce the risk of secondary complications such as AA amyloidosis.

3.6 Prognosis and Long-Term Outcomes

The prognosis of patients with MCD-associated AA amyloidosis largely depends on the control of the underlying inflammatory process. Effective suppression of IL-6 activity can lead to a reduction in SAA levels and stabilization of amyloid deposits (Bernabei *et al*., 2020). In some cases, regression of amyloid deposits has been reported following successful treatment of the underlying disease, particularly when SAA levels are maintained below 10 mg/L [10, 17]. However, once significant organ damage occurs, particularly renal involvement, the prognosis remains guarded [8].

Our patient showed favorable clinical and biological evolution following rituximab therapy, with normalization

of inflammatory markers and viral clearance, highlighting the importance of early and targeted treatment (Fig 4). Nevertheless, long-term follow-up is essential to monitor for potential relapse of MCD and progression of amyloid deposition.

4. Study Limitations

As a single case report, this study has inherent limitations. The rarity of HHV-8-associated MCD precludes generalizable conclusions, and the diagnostic workup relied on a single institution's experience. The long diagnostic delay (six years) reflects the nonspecific presentation of MCD, but also introduces recall bias in the clinical history. Histopathological confirmation of AA amyloidosis was obtained from liver and salivary gland biopsies; a renal biopsy was not performed, and the degree of renal amyloid deposition remains unknown. The patient's response to rituximab and valganciclovir was favorable, but long-term follow-up data are still limited. Despite these limitations, this report highlights the importance of considering MCD in patients with unexplained chronic inflammation and AA amyloidosis, and underscores the value of early, targeted therapy.

5. Conclusion

This case emphasizes the need for heightened clinical awareness of MCD in patients with unexplained chronic inflammation and AA amyloidosis. Early diagnosis, accurate etiological classification, and prompt initiation of targeted therapy are essential to improve outcomes and prevent irreversible organ damage. Excisional lymph node biopsy with HHV-8 immunostaining remains the cornerstone of definitive diagnosis and should be performed early in the diagnostic workup.

In resource-limited settings, access to targeted therapies such as rituximab may be delayed or restricted, yet this case demonstrates that when available, early treatment can yield favorable outcomes. This underscores the need for continued efforts to improve access to essential medicines for rare diseases in low- and middle-income countries.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki. Ethical review and approval were waived for this case report by the Ethics Committee of the University of Health Sciences Dr. Youcef El Khatib, Faculty of Medicine, Algiers, Algeria, as it involved retrospective analysis of anonymized clinical data.

Informed Consent Statement: Informed consent was obtained from the patient for the publication of this case report and any accompanying images. A copy of the written consent form is available for review by the Editor-in-Chief of this journal.

Data Availability Statement: The data presented in this case report are available on request from the corresponding author due to privacy and ethical restrictions.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

Abbreviation	Definition
ANA	Antinuclear antibodies
ANCA	Antineutrophil cytoplasmic antibodies
anti-dsDNA	Anti-double-stranded DNA
AST	Aspartate aminotransferase
CD	Castleman disease
CRP	C-reactive protein
CT	Computed tomography
ESR	Erythrocyte sedimentation rate
HBV	Hepatitis B virus
HCV	Hepatitis C virus
HHV-8	Human herpesvirus 8
HIV	Human immunodeficiency virus
IL-6	Interleukin-6
iMCD	Idiopathic multicentric Castleman disease
LDH	Lactate dehydrogenase
MCD	Multicentric Castleman disease
MEFV	Mediterranean fever gene
MMGR	Laboratory of Metabolic and Rare Genetic Diseases
PCR	Polymerase chain reaction
SAA	Serum amyloid A

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