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Idiopathic Multicentric Castleman Disease, Plasmacytic Variant, TAFRO Phenotype: A Case Report

Enfermedad de Castleman multicéntrica idiopática, variante plasmocelular, fenotipo TAFRO: reporte de caso

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ABSTRACT

Introduction: Castleman disease is a rare lymphoproliferative disorder whose diagnosis requires integrating clinical, imaging, and histopathological findings. It has a morphological classification (unicentric or multicentric) and a histopathological one (hyaline-vascular, plasmacytic, or mixed variants). The multicentric form may present TAFRO phenotype (thrombocytopenia, anasarca, fever, renal dysfunction, and organomegaly), which can be potentially fatal. This case highlights aggressiveness, initial diagnostic discordance, and early therapeutic decisions in a tertiary-care oncology hospital. **Case presentation:** A 44-year-old man with progressive abdominal pain, vomiting, and constipation, with peritoneal signs and petechiae. CT showed bowel obstruction, hepatosplenomegaly, and generalized lymphadenopathy. Laparotomy with appendectomy was performed; lymph nodes were consistent with lymphoid hyperplasia. He developed anasarca, pleural effusions, ascites, thrombocytopenia, acute kidney injury, and elevated inflammatory markers. **Diagnosis and interventions**: A confirmatory axillary lymph node biopsy established Multicentric Castleman Disease with TAFRO phenotype. Escalated antibiotics, hemodialysis, Dexamethasone, and a single dose of Rituximab were administered. Positron Emission Tomography demonstrated lymph nodes with moderate tracer uptake. He progressed to septic shock and multiorgan dysfunction and died despite mechanical ventilation and intensive support. **Conclusions:** Idiopathic multicentric Castleman disease with TAFRO can have fulminant clinical presentation. Early suspicion, repeating biopsies when clinicopathologic discordance exists, and timely access to advanced studies and targeted therapies are key to clinical outcomes. This case underscores the need for diagnostic protocols and early multidisciplinary management.

Keywords: Castleman's Disease, Lymphadenopathy, HHV-8, Interleukin-6, TAFRO, POEMS, Case report.

RESUMEN

Introducción: La enfermedad de Castleman es un trastorno linfoproliferativo infrecuente cuyo diagnóstico requiere integrar hallazgos clínicos, imagenológicos e histopatológicos. Tiene una clasificación morfológica (unicéntrica o multicéntrica) e histopatológica (variantes hipervascular, plasmocelular o mixta). La forma multicéntrica puede presentar el fenotipo TAFRO (trombocitopenia, anasarca, fiebre, disfunción renal y organomegalia), el cual puede ser potencialmente fatal. Este caso destaca la agresividad, la discordancia diagnóstica inicial y las decisiones terapéuticas tempranas en un hospital oncológico de tercer nivel. **Caso clínico:** Hombre de 44 años con dolor abdominal progresivo, vómitos y constipación; con signos peritoneales y petequias. El estudio tomográfico mostró obstrucción intestinal, hepatoesplenomegalia y adenopatías generalizadas. Se realizó laparotomía con apendicectomía; ganglios compatibles con hiperplasia linfoide. Desarrolló anasarca, derrames pleurales, ascitis, trombocitopenia, lesión renal aguda y marcadores inflamatorios altos. En cuanto al

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diagnóstico e intervenciones, se realizó biopsia axilar confirmatoria de enfermedad de Castleman multicéntrica idiopática variante plasmocelular, fenotipo TAFRO. Se administraron antibióticos escalonados, hemodiálisis, dexametasona y una dosis de rituximab; la tomografía por emisión de positrones mostró adenopatías de avidez moderada. Evolucionó a choque séptico y disfunción multiorgánica; falleció pese a ventilación mecánica y soporte intensivo. **Conclusiones:** La enfermedad de Castleman mutlicéntrica idiopática con fenotipo TAFRO puede tener presentación clínica fulminante. La sospecha temprana, la repetición de biopsias cuando hay discordancia clínico-patológica y el acceso oportuno a estudios avanzados y terapias dirigidas son claves para la evolución clínica. Este caso subraya la necesidad de protocolos diagnósticos y manejo multidisciplinario precoz.

Palabras clave: enfermedad de Castleman, linfadenopatía, HHV-8, interleucina-6, TAFRO, POEMS, reporte de caso.

1. Introduction

Castleman disease (CD) comprises a spectrum of very rare lymphoproliferative disorders whose evaluation requires integrating clinical manifestations, imaging, and histopathology [1]. It was first described in the 1950s by Benjamin Castleman as an enlarged lymph node characterized by an increased number of lymphoid follicles, involuted germinal centers with capillary proliferation [2]. The Castleman Disease Collaborative Network (CDCN) classifies CD as unicentric Castleman disease (UCD) and multicentric Castleman disease (MCD). The latter is subdivided into human herpesvirus 8 (HHV-8)-associated MCD, POEMS-associated MCD (polyneuropathy, organomegaly, endocrinopathy, monoclonal plasma cell disorder, and skin changes), and idiopathic MCD (iMCD) [2,3]. Histopathologic classification comprises the hyaline-vascular (HV) variant with regressive germinal centers and prominent vascularity; the plasmacytic (PC) variant, in which hyperplastic germinal centers and plasmacytosis predominate; and the mixed variant with overlapping features [2].

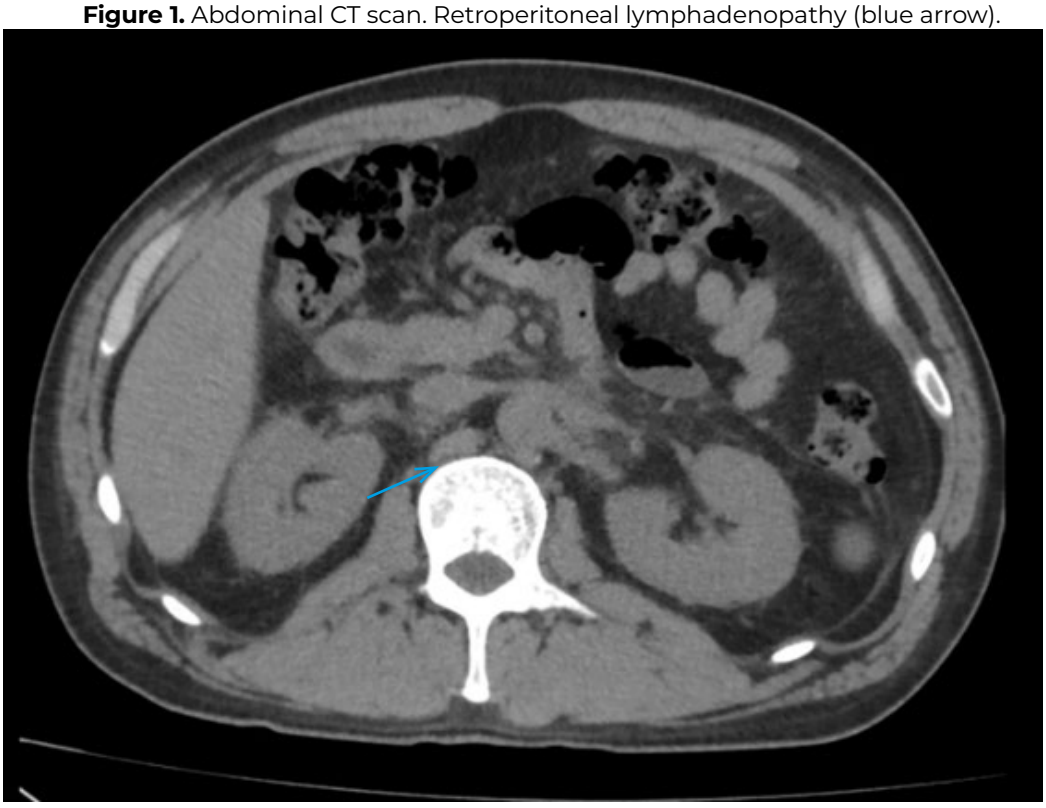
iMCD may present with TAFRO phenotype (thrombocytopenia, anasarca, fever, reticulin fibrosis, renal dysfunction, and organomegaly), a clinically very aggressive variant whose diagnosis requires histopathologic findings and clinical criteria. The other phenotype, with a milder clinical presentation that does not meet all the aforementioned criteria, is known as iMCD-not otherwise specified (iMCD-NOS). Despite advances in diagnosis and treatment, iMCD remains rare, underdiagnosed, and potentially fatal [4].

This case of a man treated at SOLCA Guayaquil Hospital is notable for its unusual initial presentation as acute abdomen, its rapidly progressive course as TAFRO phenotype, and the discordance between initial clinical-radiological and histological findings, reflecting the diagnostic and therapeutic challenges posed by this rare entity.

2. Case presentation

A 44-year-old Ecuadorian mixed-race man, with no relevant personal, family, or surgical history and no toxic habits. He presented to the emergency department with 10 days of abdominal pain of progressively increasing intensity, associated with vomiting and constipation. He had dry oral mucosa, peritoneal guarding, multiple abdominal petechiae, and absent bowel sounds. An abdominopelvic CT scan showed bowel obstruction secondary to terminal ileitis with an appendicolith, hepatosplenomegaly, and mesenteric, retroperitoneal, and inguinal lymphadenopathy (Figure 1). Exploratory laparotomy was performed as an emergency surgical indication, with appendectomy, and intraoperative findings included grade III appendicitis and multiple mesenteric and bilateral iliac lymph nodes >1 cm. Lymph node biopsy showed reactive lymphoid hyperplasia. He underwent a stable postoperative course, and he was discharged 48 hours later. One month later, on an outpatient appointment, a new biopsy of a right axillary lymph node conglomerate was performed due to persistent generalized lymphadenopathy.

Figure 1. Abdominal CT scan. Retroperitoneal lymphadenopathy (blue arrow).



Source: Intranet SOLCA-Guayaquil

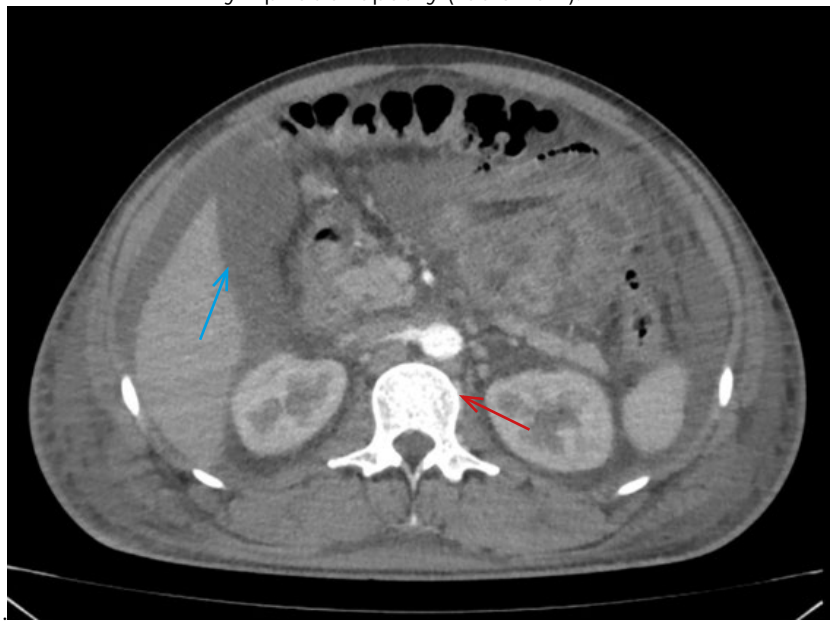
On day 36 from the initial clinical presentation in the emergency department, the patient was readmitted for abdominal pain and lower-extremity edema. Initial laboratory studies showed hyponatremia, hyperkalemia, acute kidney injury (KDIGO stage 2), and tumor lysis syndrome (Table 1). Whole-body CT revealed multiple supra- and infra-diaphragmatic lymphadenopathy, pleural effusion, ascites, and hepatomegaly, with a radiologic impression of a lymphoproliferative syndrome (Figure 2). On day 37, he developed leukocytosis and elevated acute-phase reactants and received Ampicillin/Sulbactam 1.5 g every 6 hours, with negative microbiological reports. On day 38, he had bleeding at the right axillary puncture site with a drop in hemoglobin level and transfusion requirement. The hematology department started Dexamethasone 8 mg every 12 hours due to suspicion of a lymphoproliferative disorder. On day 39, acute-phase reactants increased and antibiotic coverage was switched to Cefepime 1 g every 8 hours to treat a urinary infection, with a clinically stationary course. On day 42, ultrasound-guided therapeutic paracentesis was performed, removing 4,700 mL of ascitic fluid. Cytology was negative for malignancy.

Between days 43 and 48, he had progressive increased lower-extremity edema and on day 49, left thoracentesis was performed with drainage of 940 mL. Cytology result was negative for malignancy. Colonization by a Carbapenemase-resistant bacterium was identified on rectal swab culture, and an extended-spectrum beta-lactamase-producing organism was identified on stool culture. On day 50, given a hyperdynamic circulation, fever, and elevated procalcitonin. The Infectious Diseases department changed antibiotic coverage to Meropenem 1 g every 12 hours and Tigecycline 50 mg every 12 hours. Between days 51 and 57, he developed worsening renal function, metabolic acidosis, and refractory oliguria, requiring three sessions of intermittent hemodialysis. On day 54, histologic and immunophenotypic results from the right axillary lymph node were compatible with Multicentric Castleman Disease (MCD), plasmacytic variant, HHV-8 negative, without criteria for POEMS. Therefore, it was classified as idiopathic (iMCD) (see Table 2). On day 55, bone marrow biopsy and aspirate were performed without evidence of cells with a pathologic phenotype.

Table 1. Summary of significant laboratory results

Parameter	Reference range	First admission Day 1	Second admission Day 36	Peak or nadir	Day of Peak or nadir
White blood cells (x10^3/µL)	5,00 - 9,50	14,51	11,47	58,26	Día 60
Hemoglobin (g/dL)	12,00 - 16,00	11,30	8,5	6,70	Día 38
Hematocrit (%)	36,00 - 48,00	34,8	26,4	21,00	Día 38
Neutrophils (%)	37 - 72	72	68	97	Día 60
Platelets (x10³/µL)	150,00 - 450,00	180,00	168,00	28,00	Día 60
C-reactive protein (mg/L)	0,00 - 0,50	9,6	13,20	20,30	Día 60
Procalcitonin (ng/mL)	<0,5	0,12	6,86	15,50	Día 60
Urea (mg/dL)	16,60 - 48,50	35,60	90,30	285,00	Día 51
Creatinine (mg/dL)	0,70 - 1,20	1,34	2,05	2,31	Día 60
Uric acid (mg/dL)	3,5–7,0	7,70	11,70	11,70	Día 36
Sodium (mEq/L)	135 - 145	130	121	121	Día 38
Potassium (mEq/L)	3,50 - 5,30	4,93	7,19	7,75	Día 51
Chloride (mEq/L)	98 - 109	101	95	95	Día 36
Total calcium (mg/dL)	8,60 - 10,00	-	8,92	7,43	Día 56
Ionized calcium (mmol/L)	4,80 - 5,60	-	4,19	3,30	Día 59
Phosphorus (mg/dL)	2,50 - 4,50	3,92	4,81	8,91	Día 59
Albumin (g/dL)	3,50 - 5,20	2,84	2,27	1,37	Día 60
Total protein (g/dL)	6,60 - 8,70	8,33	6,82	3,65	Día 60
Total protein (g/dL)	< 7	-	-	21,120	Día 60

Figure 2. Contrast-enhanced abdominal CT scan. Free fluid in the cavity (blue arrow). Lymphadenopathy (red arrow).



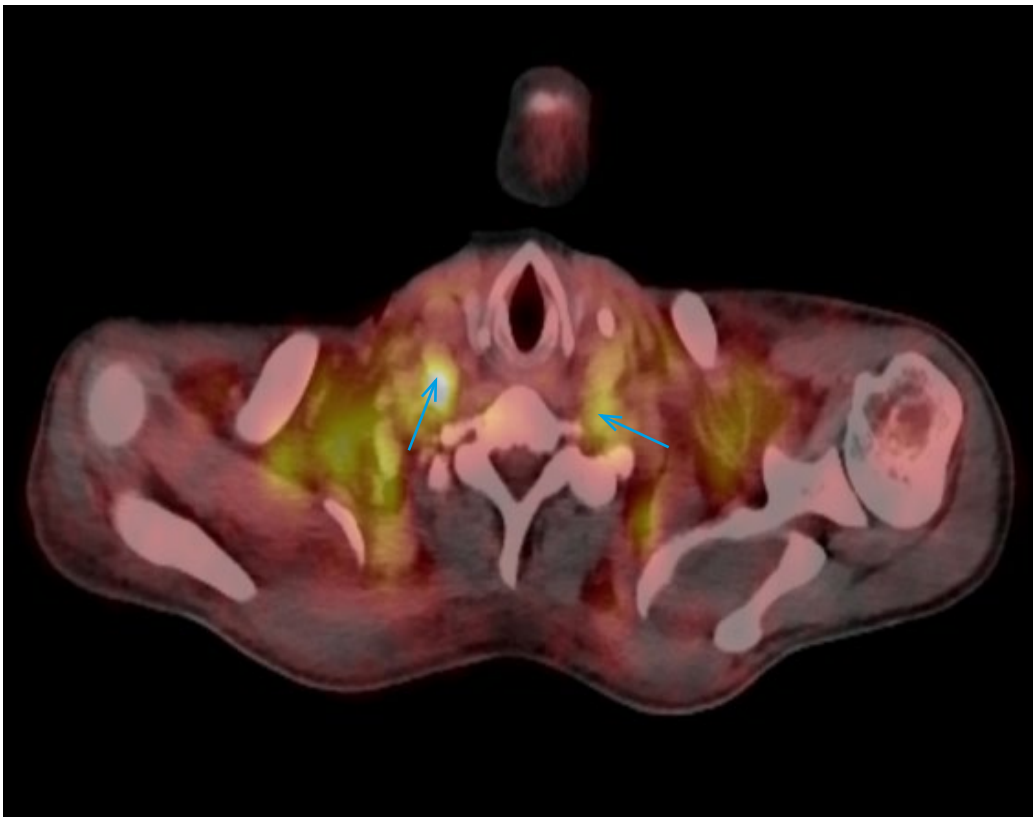
Source: Intranet SOLCA-Guayaquil

Table 2. Anatomopathologic and immunohistochemical correlation of right axillary lymphadenopathy with diagnosis idiopathic Multicentric Castleman disease, plasmacytic variant (TAFRO phenotype)

Study / marker	Result	Diagnostic relevance
Nodal morphology	Lymphadenopathy morphologically compatible with plasmacytic Castleman disease.	Compatible with a plasmacytic pattern of Castleman disease; absence of atypia or monoclonality suggests a polyclonal reactive process.
CD3	Normal expression in the interfollicular T-cell area.	Confirms preserved T-cell population; rules out neoplastic T-cell infiltrate.
CD10	Normal expression in germinal centers.	Preservation of follicular architecture, without clonal disruption.
CD20 (L26)	Normal expression in the cortical B-cell zone.	Maintains a polyclonal B-cell population; rules out B-cell lymphoma.
Kappa / Lambda	Slight predominance of kappa over lambda.	Polyclonal expression, compatible with reactive hyperplasia, not neoplastic.
CD138	Abundant mature plasma cells in the interfollicular region.	Cardinal finding of the plasmacytic variant of Castleman disease.
BCL-2	Normal expression in the interfollicular T-cell zone.	Absence of neoplastic overexpression; rules out follicular lymphoma.
KI-67	Increased in germinal centers; expression in plasma cells.	Maintains reactive follicular architecture.
CD23	Normal expression in follicular dendritic cell meshwork.	Mantiene arquitectura folicular reactiva.
EBER (ISH)	Negative.	Excludes Epstein-Barr virus infection.
IgG4	Negative (<20% of plasma cells).	Rules out IgG4-related disease.
IgG total	Polyclonal expression correlated with CD138+.	Reinforces the polyclonal hyperplastic component.
HHV-8	Negative.	Rules out HHV-8-associated CD; supports a diagnosis of idiopathic MCD (iMCD).
Nota al diagnóstico	Morphologic findings compatible with plasmacytic Castleman disease. No signs of malignancy are observed.	Defined as iMCD, plasmacytic variant, TAFRO phenotype due to association with thrombocytopenia, anasarca, fever, renal dysfunction, and organomegaly.

The PET-CT (positron emission tomography/computed tomography) study on day 56 demonstrated hypermetabolic cervical lymph nodes at bilateral levels IIa, IIb, and IV (size up to 10 mm, SULmax 2.3-2.9) (Figure 3). In the axillary region, there were bilateral hypermetabolic lymph nodes at levels I-II (size up to 11 mm, SULmax 2.2-2.3), and a right axillary lymph node conglomerate (57 x 28 x 36 mm) without radiotracer affinity. Additionally, multiple lymph nodes up to 12 mm at intercavoaortic, lateroaortic, common iliac, and external iliac levels (SULmax ≤1.5) and heterogeneous radiotracer uptake in the axial and appendicular skeleton were reported as probable reactive marrow hyperplasia.

Figure 3. PET-CT. Bilateral hypermetabolic cervical lymph nodes (Blue arrow).



Source: Intranet SOLCA-Guayaquil.

On day 57, he received a single 500 mg dose of Rituximab. Between days 58 and 60, the patient developed acute deterioration of respiratory function, recurrent bilateral pleural effusion, increased inflammatory markers (predominantly interleukin-6 (IL-6)), and septic shock. He required invasive mechanical ventilation and inotropic support and was admitted to the Intensive Care Unit, where multiorgan failure worsened. He suffered a cardiorespiratory arrest and, after unsuccessful cardiopulmonary resuscitation, he was declared dead.

3. Discussion

In Ecuador, there is a limited number of publications on CD and a lack of epidemiologic data on its prevalence. Duchicela et al. [5] reported a 59-year-old patient with UCD, HV variant, in the cervical region treated surgically; and Cedeño A. et al. [6] reported another case in a 9-year-old with UCD, HV variant, in a left cervical nodule, with excision of the lesion. In contrast, the present report describes MCD with multiple lymphadenopathies, which is considerably rarer and clinically more aggressive. Its management is usually clinical rather than surgical.

This case corresponds to the plasmacytic (PC) histologic variant, which—according to Murakami et al. [7]—accounts for 86% of MCD cases overall; according to Liu et al. [8], it represents 33% and 44% of patients with iMCD in case series and clinical trials, respectively, included in their systematic review. Dispenzieri et al. [2] reported that in patients with iMCD-TAFRO, usual pathologic features are hyaline-vascular and mixed, which contrasts with the case presented here.

Regarding diagnosis, a strength of this case is that it met the clinical-analytic criteria proposed by the CDCN compatible with iMCD [2,3]. Conversely, the dissociation between the clinical presentation and the initial histopathologic result that prompted a second biopsy is considered a limitation. The

diagnostic challenge of iMCD, which often requires multiple biopsies and exclusion of other diseases, is similar to the case report by Semenchuk et al. [9], in which seven biopsies were obtained before the specific treatment was administered.

On PET/CT, which provides diagnostic and treatment response information, hypermetabolic lymphadenopathy was observed. It was consistent with CD descriptions in which radiotracer avidity is usually moderate and non-discriminative between inflammatory reactive and pathologic etiologies. This circumstance broadened the radiologic differential diagnosis and made histologic confirmation indispensable [10].

This patient's iMCD was associated with TAFRO phenotype, meeting all histopathologic criteria (typical lymph node pathology, HHV-8 negative), four out of five major criteria (thrombocytopenia, anasarca, fever, organomegaly), and one minor criterion (elevated alkaline phosphatase without significant transaminase elevation). This phenotype is usually accompanied by a more aggressive clinical course, multiorgan failure, longer length of stay, and worse survival than (HHV-8)-associated MCD and iMCD-NOS [2,4,11].

iMCD can cause significant systemic complications and, although it is not considered a malignant disorder, according to Hoffmann C. et al. [12], it has an annual mortality rate of 23% to 49% with multiorgan dysfunction and sometimes progression to non-Hodgkin lymphoma. In line with scientific literature, the patient in this report developed multiorgan failure and septic shock, requiring intensive clinical support with hemodialysis and mechanical ventilation, with a fatal outcome.

Although histopathology enables the diagnosis of iMCD, histopathologic variants alone do not guide the therapeutic approach [1,2]. Conversely, extremely elevated IL-6 is consistent with iMCD pathogenesis and is considered the main mediator of the hyperinflammatory syndrome, as detailed in several reviews [1,4]. This agrees with studies such as that by Jitaru C. et al. [13], in which the anti-IL-6 agent Siltuximab is effective. In a case report of iMCD published by Sikora M. et al. [14] treatment with Tocilizumab (anti-IL-6) resulted in clinical improvement, normalization of inflammatory status, and remission.

Reviews recommend adding corticosteroids according to severity, with Rituximab, proteasome inhibitors, and immunomodulators as options when anti-IL-6 therapy is unavailable or the patient is refractory to the latter [4,15]. The rapid progression in this patient limited therapy to a single dose of Rituximab plus Dexamethasone prior to ICU transfer, and the severe clinical condition did not allow additional specific regimens to be used or assessing the response to different lines of therapy.

In contrast to patients with MCD, those with UCD present with localized masses are generally asymptomatic and have surgical resolution. It is usually curative, as in the case reported by Jaishanker S. et al. [16] describing excision of a retroperitoneal hyaline-vascular mass as well as the previously mentioned cases published in Ecuador [5,6].

Because it is a single-case report, this publication does not allow causal associations to be established nor findings to be generalized to the overall population with CD. This patient is the first published case of idiopathic Multicentric Castleman Disease with TAFRO phenotype in our institution.

4. Conclusion

This case illustrates idiopathic Multicentric Castleman disease, iMCD, plasmacytic variant, with TAFRO phenotype and a fulminant course, presenting with polyserositis, hyperinflammation, thrombocytopenia, and renal injury, confirmed by histopathology and immunohistochemistry. Moderate uptake on PET-CT and initial clinicopathologic discordance emphasize that iMCD requires a high index of clinical suspicion and availability of advanced laboratory and radiologic studies to apply an appropriate diagnostic approach. Although rare, this disease should be considered in the differential diagnosis of patients with lymphoproliferative syndromes. Development of protocols for diagnosis, early management, and close multidisciplinary follow-up is recommended.

5. Abbreviations

CD: Castleman disease

CDCN: Castleman Disease Collaborative Network

MCD: Multicentric Castleman disease

UCD: Unicentric Castleman disease

(HHV-8)-associated MCD: Human herpesvirus 8-associated multicentric Castleman disease

POEMS: Polyneuropathy, organomegaly, endocrinopathy, monoclonal plasma cell disorder, and skin changes

iMCD: Idiopathic Multicentric Castleman disease

HV: Hyaline-vascular

PC: Plasmacytic

TAFRO: Thrombocytopenia, anasarca, fever, reticulin fibrosis, renal dysfunction, and organomegaly

6. Administrative information

6.1 Additional files

None declared by the authors.

6.2 Acknowledgements

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6.3 Authors' contributions

Carlos Calle Caamaño; Andrea Plaza Rodríguez: Conceptualization, methodology, validation, formal analysis, investigation, project administration.

Diego Cruz Santos; Andrés Macías Gordillo: Conceptualization, investigation, visualization, writing-original draft, writing-review and editing.

All authors read and approved the final version of the manuscript.

6.4 Funding

None.

6.5 Availability of data and materials

Data are available upon request from the corresponding author. No other materials are reported.

6.6 Declarations

Ethics committee approval is not required for case reports.

6.7 Consent for publication

The patient provided written consent for publication of this case report.

7. Conflicts of interest

The authors declare that they have no competing interests or conflicts of interest.

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