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Plasmacytoid dendritic cell leukemia - case study and diagnosis

Leucemia de células dendríticas plasmocitoides - estudio y diagnóstico de un caso

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Abstract

Plasmacytoid dendritic cell leukemia (pDCL) is a rare and aggressive neoplasm that affects the skin, lymph nodes, and may spread to the bone marrow. Recognized by the WHO as a distinct entity since 2016, it has a median survival of 12 to 16 months. Although sometimes associated with Epstein-Barr virus and HIV, its exact etiology remains uncertain. Genetic characteristics include deletions in chromosomes 4, 9, and 13, and overexpression of oncogenes.

The case study describes a 49-year-old patient with a history of type 2 diabetes mellitus who presented with symptoms of leukopenia, neutropenia, and thrombocytopenia. Agranulocytosis was initially suspected, but subsequent tests confirmed plasmacytoid dendritic cell leukemia and its progression.
Keywords: Leukemia, Cells, Dendritics, Case Reports

Resumen

La leucemia de células dendríticas plasmacitoides (pDCL) es una forma rara y agresiva de cáncer que afecta la piel y los ganglios linfáticos y puede extenderse a la médula ósea. Desde 2016 la OMS la reconoce como una entidad independiente, con una expectativa de vida media de 12 a 16 meses. Aunque a veces se relaciona con los virus Epstein-Barr y VIH, su causa exacta sigue siendo desconocida. Genéticamente se caracteriza por deleciones en los cromosomas 4, 9 y 13, y la sobreextensión de oncogenes

El caso clínico describe a un hombre de 49 años con diabetes mellitus tipo 2 que presentó síntomas de leucopenia, neutropenia y plaquetopenia. Inicialmente se sospechó de agranulocitosis, pero las pruebas confirmaron leucemia de células dendríticas plasmocitoides y su progresión.
Palabras clave: Leucemia, Células, Dendríticas, mieloide aguda, neoplasia maligna

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1 Introduction

Blastic plasmacytoid dendritic cell neoplasia (BDPCN) is a malignant disease whose precursor is the plasmacytoid dendritic cell (PDC). It is characterized by affecting the skin and lymph nodes, although it can evolve to a disseminated form and infiltrate the bone marrow. Initially classified as acute myeloid leukemia (AML), the World Health Organization (WHO) recognized it as an independent entity in 2016. This neoplasia is distinguished by its aggressive behavior, rapid systemic dissemination, and a median survival of 12 to 16 months. Its incidence is low, representing less than 1% of malignant neoplasms, and is more common in men than women, with a ratio of 3:1, predominating in the sixth decade of life; however, there are case studies in patients under 40 years of age (1,2).

The etiology of NBCDP is not well defined. Although it has been associated with Epstein-Barr virus (EBV) and human immunodeficiency virus (HIV), studies have not established a clear correlation. At the molecular level, this neoplasia presents deletions in chromosomes 4 (4q34), 9 (9p13-p11 and 9q12-q34) and 13 (13q12-q31), which contain several tumor suppressor genes with low expression of Rb1 and LATS2, in addition to high levels of oncogenes (HES, RUNX2, FTL3) without association with amplification (2).

The typical clinical picture of NBCDP includes skin lesions, lymphadenopathy, and organomegaly, consistent with the descriptions in this report. Skin lesions vary in size, shape, and color and may present as tumors, nodules, and plaques, generally affecting the face, trunk, and extremities (2,3).

Since NBCDP shares several common clinical and diagnostic features with acute leukemias and aggressive T-cell lymphomas, differential diagnosis is challenging and requires a thorough workup, in particular the detection of the defining immunophenotype is essential (3).

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Diagnosis requires extensive immunophenotypic analysis due to overlap with other neoplasms, in addition to clinical and histopathological correlation. There is no standardized treatment; however, improved outcomes have been observed in patients treated with a regimen similar to that for acute lymphocytic leukemia and consolidation with allogeneic transplantation (4).

2 Clinical case

A 49-year-old married man, a driver from Azogues and resident in Cuenca, with a secondary education, presented a history of type 2 diabetes mellitus treated with metformin and two episodes of COVID-19. He also had a family history of type 2 diabetes mellitus in his parents and siblings. He initially presented with lower back pain associated with colitis and intolerance to dairy and citrus fruits. These symptoms persisted, leading him to seek medical attention due to persistent lower back pain and food intolerance, which prompted additional testing.

During the consultation, her vital signs were: temperature of 36 degrees Celsius, heart rate of 80 beats per minute, respiratory rate of 20 breaths per minute, blood pressure of 120/76 mmHg, oxygen saturation of 88%, and capillary blood glucose of 444 mg/dL. Physical examination revealed a poor general appearance and edema of the right lower extremity with petechiae.

Initial blood tests revealed leukopenia, neutropenia, and low platelet count, while imaging studies appeared normal. Agranulocytosis was initially suspected, but the neutropenia persisted and became severe, prompting a bone marrow aspiration and biopsy (bone marrow analysis and immunophenotype). Preliminary results showed 48% myeloid blasts, suggesting possible acute leukemia. After repeat testing, the diagnosis of acute myeloblastic leukemia was confirmed, and chemotherapy was prescribed, managing pegfilgrastim (6 mg subcutaneous).

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The patient's progress was unfavorable, with petechiae on the lower extremities and intermittent fever, suggesting infection. Blood tests showed a leukocytosis of 81,050 and a hemoglobin of 8.1 mg/dL. An abdominal CT scan revealed an abdominal collection with upper intestinal obstruction, leading to a total colectomy with ileostomy. The patient's progress continued unfavorably, developing septic and hypovolemic shock with acute renal failure, and he died four months after diagnosis.

3 Analysis

Myelodysplastic syndromes (MDS) are clonal diseases of the bone marrow characterized by ineffective hematopoiesis, presenting as morphological dysplasia of the hematopoietic elements and peripheral cytopenias, with a high risk of progressing to acute leukemia in the future (5).

This condition is more common in patients who have received chemotherapy, radiation therapy, or both. In some cases, patients are asymptomatic, and diagnosis is made through a complete blood count or bone marrow examination, which may reveal anemia, thrombocytopenia, pancytopenia, and neutropenia (5).

Most patients present with signs and symptoms related to cytopenias, such as pallor, epistaxis, hematochezia, hematuria, bruising, and petechiae (6).

A group of clonal hematopoietic stem cell disorders, the myelodysplastic syndromes, are characterized by the presence of multiple hematopoietic stem cell mutations, most frequently in genes involved in RNA splicing (7,8).

The following chart describes the progression of hematopoietic stem cell disorders, focusing on myelodysplastic syndromes and their progression to leukemia. Its contents are detailed below:

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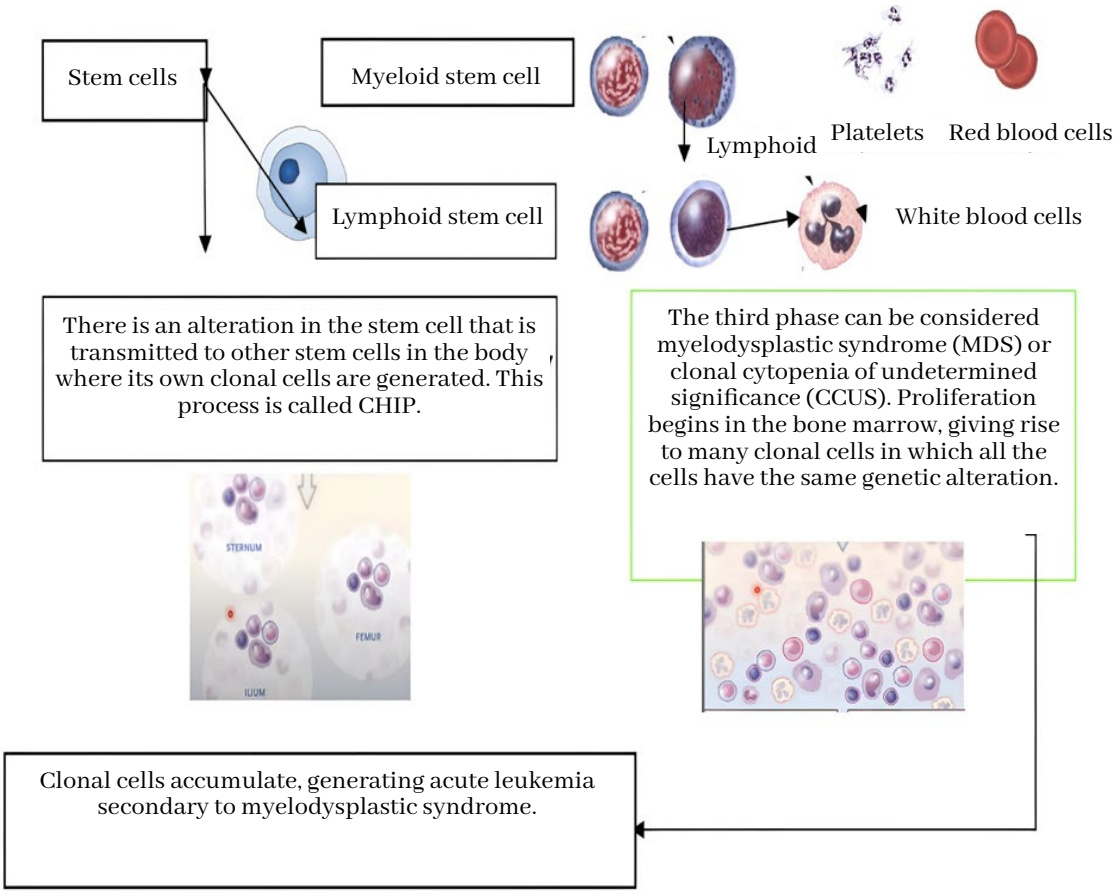
Figure 1



Pathophysiology of leukemia. Prepared by: Dután I, González C, Matute N, Rodríguez A.

Myelodysplastic syndromes (MDS) according to their morphological and genetic characteristics. It includes subtypes such as those with dysplasia of one or more cell lines, ring sideroblasts, isolated deletion of chromosome 5, and refractory anemia with excess blasts (types I and II). Unclassifiable variants and those with myeloproliferative characteristics, such as chronic myelomonocytic leukemia, are also described. This classification allows for diagnosis and prognosis in patients with MDS, as shown below:

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Table 1: Classification of myelodysplastic syndrome

TYPE OF MDs
MDs with single-line dysplasia
MDs with multilineage dysplasia
MDs syndrome with ring sideroblasts • with single-line dysplasia • with multiline dysplasia
MDs with isolated detection of chromosome 5
Refractory anemia with excess blasts • Type I • Type II
Unclassifiable MDs
Myelodysplastic/myeloproliferative syndrome
Chronic myelomonocytic leukemia • Type 0 • Type I • Type II

Discussion

Among the main differential diagnoses are acute acute myeloid leukemia (AML) with monocytic differentiation, chronic myelomonocytic leukemia, nasal type extranodal NK T-cell lymphoma, and panniculitic T-cell lymphoma (9).

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Differentiating between a**cute myeloid leukemia (AML) and** plasmacytoid dendritic cell leukemia (pDCL) requires an assessment of clinical, immunophenotypic, morphological, and genetic characteristics. Clinically, both AML and pDCL present similar symptoms, including fever, fatigue, cachexia, and cytopenias, which may be due to bone marrow infiltration by malignant cells. pDCL is usually associated with skin lesions, hepatomegaly, and lymphadenopathy (10).

Morphologically, acute myeloid leukemia cells have Auer bodies, whereas pDCL cells lack them. In AML, leukemic cells display myeloid markers such as CD13, CD33, CD117, and MPO (myeloperoxidase). In contrast, pDCL displays an immunophenotypic profile with expression of CD4, CD56, CD123, and certain specific markers BDCA-2 and TCL1 (10,11).

Nasal-type extranodal NK/T-cell lymphoma occurs predominantly in and around the nose, but can also affect other parts of the body. It is associated with Epstein-Barr virus (EBV) infection. Histologically, it has an angiocentric and angioinvasive pattern with a wide area of necrosis. Plasmacytoid dendritic cell leukemia can present with systemic symptoms and cutaneous involvement and is not usually limited to a specific region such as the nose. The patient's clinical presentation is also useful, since NK/T-cell lymphoma is characterized by nasal symptoms and obstructive masses, whereas dendritic cell leukemia does not show this pattern (12).

To differentiate between a panniculitis T-cell lymphoma (PPCTL) and a (pDCL) a complex evaluation must be made regarding its clinical presentation and some morphological characteristics (13).

LPCTL presents with subcutaneous skin lesions resembling panniculitis, primarily affecting the extremities. Histologically, there is an infiltrate of atypical lymphocytes in the subcutaneous tissue, with adipocyte necrosis and cytoskeletal formation. Lymphocytes in LPCTL display T cell markers such as CD3, CD4, or CD8, and a loss of markers found in normal T cells. Furthermore, they may express cytotoxicity, as evidenced by the expression of granzyme B, perforin, and TIA-1 (14).

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Blastic plasmacytoid dendritic cell neoplasia (BPDCN) is a rare entity characterized by the malignant proliferation of a blastic plasmacytoid dendritic cell. The true incidence of this disease is unknown due to its rarity and the difficulty of diagnosis (2).

In most patients, the condition presents with cutaneous plaques or nodules of variable size, shape, and color, primarily affecting the extremities, trunk, and face; progressing to bone marrow involvement (90%). Extracutaneous involvement is common and includes regional lymph nodes (40%–50%), splenomegaly (44%), and hepatomegaly (42%) (15).

In this particular case study, the patient presented for colitis. A physical examination revealed multiple purple spots on the trunk and lower extremities. A complete blood count revealed severe cytopenias with markedly decreased cellular and immunological immunity. He was referred to the hematology department, where a PAMO (Molecular Biology Report) was performed, confirming the condition.

Pemmaraju et al. (14), in their research emphasizes the heterogeneous nature of the disease, where patients may initially present with skin lesions, followed by progression to a systemic disease. In addition, Sánchez E (18), in his case analysis, agrees with the other authors about the presence of skin manifestations as an initial sign of this neoplasia, as it was in the patient in our study.

However, Pemmaraju et al. (14) contrast the above by indicating that in a significant group of patients, skin lesions may be absent or minimal, which complicates the initial diagnosis and delays therapeutic intervention. This aspect underlines the importance of a high clinical suspicion and the need for exhaustive immunophenotypic studies in any case of unexplained cytopenia or systemic symptoms that could be associated with BPDCN (14).

Flores-Angulo et al (1) highlight that the diagnosis of BPDCN is based on the identification of specific immunophenotypic markers, such as CD123, CD4, and CD56, through flow cytometry. These markers are

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critical for differentiating BPDCN from other hematologic malignancies, given that clinical manifestations may be nonspecific or shared with other diseases. In their reported case, the combination of histologic and immunophenotyping studies was key to establishing the definitive diagnosis. On the other hand, in the article by Avilés et al (2), the relevance of an early diagnosis to improve the patient's prognosis is emphasized. They underline that the diagnosis is often difficult due to the rarity of the disease and the lack of familiarity of many clinicians with BPDCN. Avilés et al (2) also mention that confirmation of the diagnosis often requires collaboration between hematologists, dermatologists, and pathologists, given that BPDCN can present characteristics that affect multiple systems, something that was also necessary in the case we are analyzing.

For their part, the article by Martini M, Russo V, and Fabbri A (13), in Annali dell'Istituto Superiore di Sanità (Italy) offers an in-depth insight into plasmacytoid dendritic cell leukemia, focusing on the molecular and biological aspects of the disease, unlike other studies that tend to focus on clinical aspects or specific case reports. While the Italian article provides a detailed perspective on the pathogenetic mechanisms of BPDCN and its implications for diagnosis and treatment, Spanish studies such as those by Flores-Angulo et al. and Avilés et al. present more practical approaches focused on clinical cases and management in local contexts due to limited access to resources and technology.

Regarding treatment, Pemmaraju and Kantarjian (14) focus more on clinical innovations such as the use of tagraxofusp and other targeted therapeutic approaches that have demonstrated efficacy in clinical practice. These investigations provide insight into how therapies based on the latest advances are applied in the real-world management of BPDCN. While Martini et al (13) lays the groundwork for future research and potential treatments by unraveling the molecular mechanisms, providing a detailed insight into them; which is crucial for the development of new therapeutic strategies.

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Currently in Latin America in Chile, although NBCDP may initially appear as a localized cutaneous tumor, an aggressive treatment could be considered initially, including allogeneic hematopoietic progenitor cell transplantation (TAloCPH); considered the best option during the first complete remission, increasing overall survival and disease-free survival, without relapses within the first 27 months (2). In the studies conducted by Avilés et al (2), the use of HiperCVAD followed by TAloCPH with a mean follow-up of 14.6 months. At the time of the study, the first patient had died with disease, the second was in documented relapse in skin and bone marrow, and the third was alive with no evidence of neoplastic disease, demonstrating that the treatment received was not optimal for the patients (15).

5. Treatment

Treatment of dendritic cell leukemia, especially in cases of blastic plasmacytoid dendritic cell leukemia (BPDCN), usually includes: High-intensity chemotherapy with Hyper-CVAD regimens (cyclophosphamide, vincristine, doxorubicin, and dexamethasone), is essential to rapidly reduce the disease burden (16).

Tagraxofusp-erzs (SL-401) immunotherapy, a CD123-targeted agent, has been approved for the treatment of BPDCN. This agent binds to the CD123 receptor and delivers a toxin that kills cancer cells (17).

Other treatment alternatives include Hematopoietic Stem Cell Transplantation (HSCT), which seeks to replace diseased bone marrow with healthy hematopoietic stem cells from the donor, providing an opportunity for a potential cure (18).

To date, in our country there is not enough evidence on what is the best treatment, due to the lack of schemes for the management of this atypical neoplasia, however, it has been assured that the combination of

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conventional chemotherapy based mainly on the Hyper-CVAD scheme, together with adequate monitoring, prolongs survival (19).

As for the patient in our study, he was treated with two cycles of Pegfilgrastim with the aim of achieving an antineoplastic and immunomodulatory effect among other effects. He was also scheduled for HyperCVAD, which he received until his death (20).

6 Conclusions

Plasmacytoid dendritic cell leukemia (pDCL) is a rare and aggressive hematological malignancy that presents a significant challenge in both diagnosis and treatment. The described case study reveals the complexity of the disease, highlighting the need for a multidisciplinary approach to its management. Despite receiving appropriate medical care and aggressive treatment with chemotherapy, a Hyper-CVAD regimen, and pegfilgrastim , the patient showed an unfavorable clinical course, culminating in severe complications and a negative outcome. This case underscores the difficulty in early diagnosis and the urgent need for more effective and personalized treatments to improve the prognosis of patients with pDCL.

The diagnostic approach to plasmacytoid dendritic cell leukemia should be comprehensive and include a broad spectrum of immunophenotypic and genetic testing to differentiate it from other myeloid neoplasms and lymphomas. The lack of standardized treatment and the limited efficacy of current regimens highlight the need for continued research. The incorporation of new targeted therapies and improved understanding of disease biology could offer hope for improved management and potential improvements in survival for patients affected by this extremely rare and challenging neoplasm.

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7. Contribution of the authors

PF: Data collection, analysis of results, discussion, final review of the article.

ID: Data collection, analysis of results, discussion, final review of the article.

CG: Data collection, analysis of results, discussion, final review of the article.

AR: Data collection, analysis of results, discussion, final review of the article.

NM: Data collection, analysis of results, discussion, final review of the article.

8.- Approval of the ethics committee and consent to participate in the study

Interventional studies involving animals and humans, and other research requiring ethics committee approval, must state the authority/ institution that approved the study and provided the code of ethics.

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