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Coexistence of Chronic Myeloid Leukemia and Hodgkin's Lymphoma: A Case Report at the National Hospital of Niamey (Niger)

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Abstract

Introduction: The coexistence of chronic myeloid leukemia and Hodgkin lymphoma (HL) is rare. In the few reported cases, HL occurs after a varying duration of treatment with tyrosine kinase inhibitors (TKIs). However, in our patient, the diagnosis of these two diseases was simultaneous. **Observation:** This is a 38-year-old female patient with no known medical history, hospitalized for vomiting and low back pain in a febrile context. There was a deterioration in WHO 3 general condition and generalized pruritus. Stage III splenomegaly and diffuse axillary, inguinal, laterocervical, and bilateral submaxillary adenopathies, non-symmetrical, were observed. Hepatomegaly, pulmonary consolidation syndrome, and generalized skin lesions were also noted. The blood count showed hyperleukocytosis at 22,339/mm^3 with neutrophil predominance at 19,500/mm^3. The blood smear revealed immature myelemia. The BCR-ABL transcript search was positive. The karyotype was not carried out due to the lack of resources. The lymph node biopsy and histology performed concluded that there was classic sclero-nodular HL and the immunohistochemistry performed confirmed the diagnosis. The patient received treatment with first-generation TKIs. Treatment was initiated 24 hours after diag-

nosis, at a dose of 400 mg per day and six (6) cycles of chemotherapy using the ABVD protocol, including two weeks between two cycles. The evolution was marked by the death of the patient despite a good clinical course, in a picture of septic shock following a superinfection of her skin lesions. **Conclusion:** The coexistence of CML with Hodgkin lymphoma is a poor prognostic condition. Despite its rarity, it should be considered in the presence of adenopathic polymorphism in chronic myeloid leukemia.

Keywords

Coexistence, CML, HL, Oncohematology, Niger

1. Introduction

Chronic myeloid leukemia (CML) is a hematological malignancy characterized by proliferation of granulosa cell lines, without maturation blockage [1]. In contrast, Hodgkin lymphoma (HL), also a hematological malignancy, belongs to the lymphoid lineage, characterized by pleomorphic lymphocytic infiltration of multinucleated giant cells (Reed-Sternberg cells) (see **Figure 1**) [2]. The coexistence of CML and HL is rare, although it is described in the literature. In the few reported cases, HL occurs after a more or less long duration of treatment with tyrosine kinase inhibitors (TKIs). However, in our patient, the diagnosis of these two diseases was simultaneous.

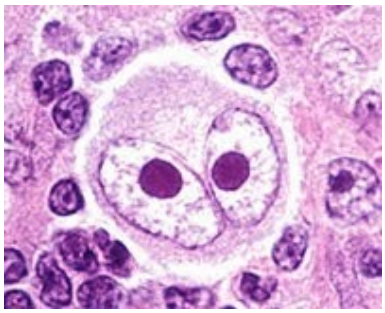

Figure 1. Reed-Sternberg cells.

2. Observation

A 38-year-old patient, first pregnancy, two parities, two living children, and none deceased (G1 P2 V2 Do). She is the first of three siblings and is not from a consanguineous marriage, with no known history of pathology. She was hospitalized for vomiting and low back pain in a feverish context.

A general examination revealed a conscious patient (Glasgow 15/15) with pale conjunctiva and mucous membranes, no jaundice; significant numerical weight loss of more than 10% of her body weight, physical asthenia, and an acute dry cough, generalized pruritus, and no lower limb edema. Her general condition was impaired (WHO 3).

Splenodal examination noted the presence of Hackett stage III splenomegaly, firm, painless, and non-compressive; diffuse polyadenopathies, axillary, inguinal, laterocervical, and bilateral submaxillary, non-symmetrical, the largest of which measured 5 cm in diameter.

At the digestive level, hepatomegaly with a hepatic arrow at 21 cm, with a regular lower border, homogeneous with a regular contour, without hepatojugular reflux and without ascites, was noted. Pleuropulmonary examination noted pulmonary consolidation in the right lung field.

At the cutaneous level, a lesion polymorphism was noted, consisting of papulonodular, inflammatory, well-defined, confluent lesions in places, disseminated on the trunk, thighs, and forearm. There were also ulcerative nodular lesions with inflammatory and crusty edges, tender to palpation, located on the trunk and neck. Erosive, oozing, rounded, well-defined lesions, measuring one (01) to two (02) centimeters in diameter, were located on the neck. Hypochromic macules, well-defined, confluent in places, were disseminated on the trunk.

In the appendages, punctiform leukonychia was noted on the fingers.

The rest of the clinical examination (cardiovascular, neurological, etc.) was unremarkable.

The blood count revealed: white blood cells at 22,339 cells/mm^3, predominantly neutrophils, with neutrophil polymorphonuclear leukocytes at 19,500 cells/mm^3. Hemoglobin level at 5 g/dL and platelets at 106,000 cells/mm^3. The blood smear revealed significant and polymorphic myelemia (blasts: 2%, promyelocytes: 1%, myelocytes: 35%, metamyelocytes: 25%, and neutrophils: 32%).

The myelogram was suggestive of chronic-phase CML with 3% blasts. Molecular biology testing for the BCR-ABL transcript was positive. The karyotype was not carried out due to the lack of resources.

Given this significant polyadenopathy at this stage of CML, we immediately performed a lymph node biopsy with anatomopathological examination, the histological appearance of which suggested a classical Hodgkin lymphoma with a sclero-nodular lymph node variant (**Figure 1**). The immunophenotyping performed revealed tumor cells with CD20−, CD30−, CD15+ phenotypes. CD3s mark hyperplastic T lymphocytes that are arranged in a rosette around the tumor cells. Abdominal ultrasound demonstrated homogeneous hepatosplenomegaly. CT and CT scans could not be performed due to lack of financial resources. The biological tests performed showed normal LDH at 325 ng/ml, positive CRP at 79.2 mg/l, negative HIV serology, and liver, kidney, and hemostasis tests were unremarkable. The patient was classified as Ann Arbor IIIBb.

The management consisted of treatment for CML with first-generation TKIs. Treatment was initiated 24 hours after diagnosis, at a dose of 400 mg per day, and 6 courses of chemotherapy using the ABVD protocol (Adriamycin, Bleomycin, Vinblastine, and Dacarbazine) for HL, including two weeks between two cycles. The evolution was marked by the death of the patient, despite good clinical progress, in a context of septic shock following a superinfection of her skin lesions.

3. Discussion

The coexistence of myeloid and lymphoid hematological disorders, whether simultaneous or sequential, in the same patient is extremely rare, with an incidence of less than 1% [3]. In the majority of cases, 66%, this association is sequential, compared to 34% simultaneous [4]. In our patient, the diagnosis of CML and HL was simultaneous.

Only cases describing the occurrence of HL after treatment of CML with tyrosine kinase inhibitors (TKIs) have been reported in the literature [5]. However, what is unique about our patient is that she had not received TKI treatment.

Lymphadenopathy usually appears in the accelerated or acute phase of CML and is generally moderate and symmetrical [6]. In our patient, the presence of asymmetric polyadenopathy in the chronic phase of CML prompted us to perform this lymph node biopsy, which detected this HL.

Several cases of CML coexisting with non-Hodgkin lymphomas and other myeloproliferative disorders have been reported in the literature, but very rarely with HL. Various types of hematological diseases are present, such as lymphocytic lymphoma [7], CLL [8], nodular marginal zone lymphoma [9], and multiple myeloma [10]. In our patient, it was a typical sclero-nodular HL.

In addition to hematological diseases, the likelihood of developing a malignant tumor increases in patients with CML due to the effective treatment of CML and improved long-term patient survival. Thus, prostate, colon, lung, breast, and melanoma cancers are the most frequently found [11].

The incidence of coexistence of CML and lymphoma is very rare and frequently occurs sequentially. Treatment of lymphoma by chemotherapy and/or radiotherapy facilitates the occurrence of CML by inducing immunosuppression. Conversely, in the case of CML, the mutagenic role of TKIs is not to be demonstrated. However, this theory would not explain cases where the diagnosis is simultaneous, as in our patient. This is why the hypothesis of a disease of the hematopoietic stem cell, externalized at two levels and on two daughter lines, seems the most attractive. This hypothesis is more consistent with current concepts that make CML a clonal disease of a totipotent cell, a common precursor of the lymphoid and myeloid lines, as supported by the presence of Ph1 in mature B lymphocytes and in acute transformations in a lymphoblastic mode [8].

4. Conclusion

Practitioners in general, and hematologists in particular, should be aware of the possibility of the concomitant presence of several hematological malignancies in the same patient. The presence of lymphadenopathy in a patient with CML may represent an acceleration or an acuity, but it may also be another distinct lymphoid hematological disease. When this is suspected, it is essential to carry out all the necessary analyses in order to make a precise diagnosis. Given that the treatment of CML differs from that of hematological lymphomas, it is important to make this distinction for appropriate patient management.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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