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Hairy Cell Leukemia Treated with Rituximab and Cladribine in Abidjan

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Abstract

Hairy cell leukemia (HCL) is a rare hematologic malignancy, accounting for approximately 2% of all leukemias, and is characterized by the proliferation of mature "hairy" B lymphocytes Diagnosis is based on the morphologic identification of tumor cells in the peripheral blood and/or bone marrow, followed by demonstration of specific markers by immunophenotyping; failure to assess these markers may lead to confusion with other lymphoid neoplasms Diagnosis remains challenging in sub-Saharan Africa because of limited technical resources, and very few studies have been conducted. We report the case of a 48-year-old patient with hairy cell leukemia who was diagnosed and managed in our department at Cocody University Hospital in Abidjan with cladribine and rituximab, resulting in complete remission.

Keywords

Hairy Cell Leukemia, Immunophenotyping, Targeted Therapy, Abidjan

1. Introduction

Hairy cell leukemia is a rare mature B-cell lymphoid malignancy with distinctive cellular morphology (<3% of leukemias in France). Its diagnosis is primarily cytologic, may sometimes be difficult, and can be confused with other chronic lymphoproliferative disorders [1] [2]. Considerable progress has been made in understanding the biology of hairy cell leukemia (HCL) and other hairy-cell proliferative disorders. In Black Africa, only fragmentary studies have been conducted,

precluding an accurate estimate of prevalence. In one of the largest series from sub-Saharan Africa, Dokekias *et al*. identified ten cases over seven years at Brazzaville University Hospital in Congo in 2003 [3]. The series by Randriamahazo T.R. *et al*. in 2012 identified two cases at Antananarivo University Hospital in Madagascar [4]. The study by Sangaré A. *et al*. also reported two cases in 1990 at Cocody University Hospital in Abidjan, Côte d'Ivoire [5].

The BRAF V600E mutation is the molecular hallmark of the disease in classic forms, although it is not sufficient on its own to generate the disease. Detection of this mutation is useful for diagnosis and may support the use of BRAF inhibitors (BRAFi) [6]. The role of non-canonical BRAF mutations and mutations in other genes within coding or non-coding regions remains to be clarified and may be involved in disease progression [6].

The therapeutic approach to HCL has evolved considerably in recent years. Immunochemotherapy combining cladribine (CDA) and rituximab is now considered a standard first-line treatment for fit patients with symptomatic classic HCL [7]. In relapsed or refractory disease, several targeted therapeutic options are available, including BRAF inhibitors such as vemurafenib or dabrafenib, particularly in combination with anti-CD20 monoclonal antibodies, as well as MEK inhibitors, Bruton tyrosine kinase inhibitors, and BCL-2 inhibitors [2] [7]. Treatment selection should take into account previous therapies, duration and quality of response, patient age and comorbidities, as well as the biological profile of the disease, and should ideally be discussed in a multidisciplinary setting [2] [7].

The present report describes a case of the leukemic form of hairy cell leukemia.

2. Case Report

Mr. E.S., a 48-year-old employee of a private company residing in one of the municipalities of Abidjan, was referred to the Clinical Hematology Department of Cocody University Hospital in January 2023 for persistent pancytopenia associated with splenomegaly.

He reported no particular medical or surgical history. However, a history of long-term exposure to toxic substances and prolonged use of oral herbal medicines was identified.

The clinical history dated back approximately seven months before consultation, with the sudden onset of physical asthenia associated with the appearance of a mass in the left hypochondrium that progressively increased in size, with a sensation of abdominal heaviness, evolving in a context of low-grade fever.

On admission, the patient was afebrile at 37˚C, had a fair general condition with a WHO performance status of 1, and weighed 57 kg. Conjunctival pallor, type IV splenomegaly, and symmetrical lymphadenopathy measuring 1 to 3 cm were also noted.

The complete blood count showed normocytic normochromic anemia with a hemoglobin level of 9.9 g/dL, leukopenia at 2790/mm^3, grade IV neutropenia at 360/mm^3, lymphopenia at 1460/mm^3, and thrombocytopenia at 36,000/mm^3. Pe-

ripheral blood smear examination showed hairy cells. Bone marrow aspiration showed normal bone at puncture, easy aspiration, and grade V marrow cellularity with 58% bone marrow lymphocytosis composed of lymphocytes with cytoplasmic extensions. Peripheral blood immunophenotyping confirmed blood infiltration by a monotypic B-cell lymphoid population expressing $CD19^+$, $CD5^-$, $CD23^-$, $FMC7^+$, $CD79b^+$, $CD200^+$, **$CD103^+$, $CD11c^+$, $CD123^+$ and $CD25^+$,** with strong lambda light-chain expression, estimated at 63% of total lymphocytes. These findings were compatible with hairy cell leukemia. In addition, testing for the BRAFV600E mutation was positive. Taken together, these findings confirmed the diagnosis of **classic hairy cell leukemia (classic HCL).**

Staging and pretreatment assessment by thoracoabdominopelvic computed tomography showed homogeneous splenomegaly causing a mass effect on the left kidney; homogeneous hepatomegaly and uncomplicated gallstones; and the presence of celiac-mesenteric and lateral cervical lymphadenopathy. Serum protein electrophoresis was suggestive of an inflammatory syndrome. Malaria and amoebic serologies were positive; however, thick and thin blood smears for malaria and stool parasitological examination were negative, and the patient had no clinical features suggestive of active malaria or amoebiasis. Therefore, these serological findings were considered indicative of previous exposure rather than active infection. No specific antimalarial or anti-amoebic treatment was administered before immunochemotherapy. Viral serologies like hepatitis (HBsAg, anti-HBc antibodies, anti-HCV) and HIV serology were negative. Renal function and transaminase levels were normal. The coagulation profile was also normal, LDH levels were elevated, and the direct Coombs test and irregular antibody screening were negative.

Therapeutically, the patient received an R-CDA regimen consisting of cladribine 0.14 mg/kg/day intravenously over 2 hours for five consecutive days (days 1 - 5). Approximately 28 days after initiation of cladribine, rituximab at 375 mg/m^2 was administered intravenously once weekly for eight consecutive weeks. After the completion of therapy with rituximab a repeat BM examination for assessment of response was performed. The evaluation of minimal residual disease (MRD) was not performed.

Antimicrobial prophylaxis consisted of oral levofloxacin 500 mg once daily, oral valacyclovir 500 mg once daily, and oral fluconazole 400 mg once daily during the period of treatment-related immunosuppression. Tumor lysis syndrome prophylaxis consisted of oral allopurinol 300 mg once daily, initiated before immunochemotherapy, together with adequate hydration. Growth factors such G-CSF (Filgrastim) was administered subcutaneously at 5 μg/kg/day, starting 24 hours after completion of cladribine and continued until adequate neutrophil recovery. No clinically significant treatment-related toxicity, severe neutropenia, febrile neutropenia, or documented infection occurred during treatment.

At two months after treatment initiation, hematologic recovery was achieved, with hemoglobin 14 g/dL, platelet count 200×10^9/L, and neutrophil count 2.5 ×

10^9/L on two consecutive assessments performed two weeks apart. Clinically, splenomegaly had markedly decreased from Hackett grade IV at diagnosis to grade II. Because residual splenomegaly persisted, this assessment was not considered a complete remission.

At the six-month response assessment, the patient fulfilled the assessed criteria for complete remission. The complete blood count showed hemoglobin of 14 g/dL, a white blood cell count of 7.8×10^9/L, an absolute neutrophil count of 5.4×10^9/L, and a platelet count of 300×10^9/L. No hairy cells were detected on peripheral blood smear or bone marrow aspirate. Clinically, splenomegaly and lymphadenopathy had completely resolved, and computed tomography confirmed the disappearance of the previously documented tumor syndrome. Minimal residual disease assessment by multiparameter flow cytometry was not performed.

At the last recorded follow-up in May 2026, approximately three years after treatment, the patient remained in clinical and hematologic remission, with a normal complete blood count and no clinical evidence of disease recurrence. Routine surveillance consisted of clinical examination and serial complete blood counts; no repeat bone marrow evaluation or imaging was performed at this last follow-up.

3. Discussion

We report a case of hairy cell leukemia in a 48-year-old adult managed in the Hematology Department of Cocody University Hospital in Abidjan.

Hairy cell leukemia (from the Greek "tricho", meaning "hair") is a rare hematologic malignancy arising from late-activated post-germinal-center memory B cells [1]. Its distinctive feature is the presence of medium-sized mature lymphocytes with "hairy" cytoplasmic projections in the bone marrow, spleen, and occasionally extramedullary tissues [8].

It is a rare disease for which very few studies have been conducted [8]. In France, approximately 300 new cases are recorded each year [7]. On the African continent, published studies remain very limited [9]. Nevertheless, a case diagnosed in Côte d'Ivoire had previously been reported in 2020 by A. Condé *et al*., but the patient was not treated because appropriate therapeutic resources were unavailable [9].

The etiology of hairy cell leukemia remains incompletely understood. Several occupational and environmental exposures, including farming, pesticides, and petroleum products, have been investigated as potential risk factors, although the available epidemiological evidence does not establish a specific causal exposure [10]. Our patient had worked as a mooring worker for nearly 25 years, an occupation potentially involving exposure to various environmental and industrial agents. However, the specific substances, intensity, and duration of individual exposures were not documented or quantitatively assessed. Therefore, this occupational history should be regarded only as a possible unmeasured exposure, and no etiologic association with HCL can be inferred from this case.

Hairy cell leukemia predominantly affects men, with a male-to-female ratio of approximately 4:1, and the median age at diagnosis is approximately 55 years [2]. These epidemiological characteristics are consistent with our patient, who was a 48-year-old man.

Dokekias E. A. *et al*. reported a mean age of 43.3 years [3]. These findings are consistent with the age of our patient, who was 48 years old.

Clinically, our patient presented with massive Hackett grade IV splenomegaly associated with anemia. Splenomegaly was also a prominent clinical feature in previously reported sub-Saharan African series of hairy cell leukemia [3] [4].

The complete blood count at admission showed pancytopenia, a common hematologic manifestation of hairy cell leukemia resulting from bone marrow infiltration and splenic involvement [1]. However, leukocytosis was documented in the case reported by Condé *et al*. [9].

The immunophenotypic profile of peripheral blood lymphocytes observed in our patient was consistent with classic HCL, which typically expresses CD11c, CD25, CD103, and CD123 [1]. An immunological scoring system, comparable to the Matutes score used in chronic lymphocytic leukemia, has been developed for the diagnosis of hairy cell leukemia. It takes into account the expression of four markers: CD103, CD11c, CD25, and CD123. One point is assigned for positive expression and 0 points for negative expression. Hairy cell leukemia has a score of 3 or 4 in almost all cases, whereas variant forms or splenic lymphoma with villous lymphocytes have scores of 0 or 1 [1].

The BRAF V600E mutation is a genetic alteration present in more than 80% to 95% of cases of classic hairy cell leukemia (HCL). This specific activating mutation leads to uncontrolled proliferation of B lymphocytes and constitutes a major molecular diagnostic marker for distinguishing classic HCL from the hairy cell leukemia variant [1]. This marker was investigated in our patient and was positive.

According to the literature, the prognosis of classic HCL has markedly improved with purine analog-based therapy. In a large multicenter study of patients treated with cladribine, estimated overall survival was 95.3% at 5 years, 92.4% at 10 years, and 81.8% at 15 years [11]. Our patient received cladribine combined with rituximab and achieved complete remission at six months. At the last recorded follow-up in May 2026, approximately three years after treatment, he remained in clinical and hematologic remission without evidence of relapse.

4. Conclusion

Although rare, hairy cell leukemia should be considered, particularly in our practice setting in sub-Saharan Africa. It preferentially affects older male adults. The clinical presentation is dominated by pancytopenia associated with splenomegaly. Diagnosis combines cytology (peripheral blood smear and bone marrow aspiration) and immunophenotyping (flow cytometry). Detection of the BRAF V600E mutation strongly supports the diagnosis of classic HCL and helps distinguish it from other HCL-like disorders. The prognosis is favorable owing to treatment

with purine analogues, sometimes combined with rituximab.

Ethics Approval and Consent for Publication

According to the institutional policy of Cocody University Hospital, formal ethics committee approval was not required for this anonymized case report. Written informed consent for the publication of the patient's clinical information was obtained from the patient. Patient confidentiality and anonymity were maintained throughout the preparation of the manuscript.

Author Contributions

Kouakou Boidy, Lontsi Edgard and Nanho Danho Clotaire were involved in the clinical management of the patient and contributed to drafting the manuscript.

Kamara Ismaël, Atiméré Yao Nicaise, Silué Dohoma Alexis, Kouakou Affoué N'guessan Jeanne Ines, Djeket, Ruth, Assohou Koffi Emmanuela, Kouamé Norman Isaac Klébair and Kouadio Atta Emmanuel contributed to the critical revision of the manuscript.

Koffi Kouassi Gustave, Professor of Clinical Hematology and Head of the Department of Clinical Hematology at Cocody University Hospital, provided scientific supervision and critically reviewed the manuscript.

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

Conflicts of Interest

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

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