presented with neurological symptoms resulting from intraorbital and leptomeningeal disease. Leptometrial disease as the initial manifestation of CLL is extremely rare (2). A large-scale CLL autopsy study reported brain and leptomeningeal involvement in 20% and 8% of cases, respectively. This study demonstrated that CNS involvement in CLL patients is underdiagnosed. Another study revealed orbital involvement in 14 of 97 autopsies (14%) of CLL patients (3). None of the studies demonstrated a correlation between leptomeningeal spread and CLL stage or duration. Standard risk factors for CNS involvement in CLL have not been systematically investigated (4).Clinical manifestations of CNS involvement in CLL are heterogeneous and include headache, cranial nerve palsies, cerebellar findings, visual problems, and motor or sensory deficits. Imaging studies do not provide sufficient evidence of CNS involvement in CLL. The diagnosis is usually confirmed by lumbar puncture. In the present case, the CSF sample showed widespread lymphocytes. In this case, a CSF sample contaminated with peripheral blood leukocytes as a result of a traumatic lumbar puncture is unlikely, as no erythrocytes and no myeloid cells were observed in the sample. The optimal treatment for CLL patients with CNS involvement is unclear. Most such patients receive treatment that includes intrathecal chemotherapy, either with or without radiotherapy or systemic chemotherapy. The most commonly used intrathecal chemotherapy agents are methotrexate, cytarabine, and corticosteroids, used alone or in combination. Our patient is currently a high-risk patient and responded well and rapidly to chemoimmunotherapy. In general, the prognosis for patients with CLL with neurological involvement is poor. Systemic chemoimmunotherapy is the most effective treatment for rapid symptom resolution in this patient group.

https://doi.org/10.1016/j.htct.2025.106139

PP 06

A CASE OF HAIRY CELL LEUKEMIA ASSOCIATED WITH CD10 EXPRESSION: THE SIGNIFICANCE OF AN ATYPICAL IMMUNOPHENOTYPIC PROFILE

Mehmet Soylu [1], Damla Çağla Patır [2,*]

[1] *Ege University, Faculty of Medicine, Department of Microbiology, Türkiye*
[2] *Ege University, Faculty of Medicine, Department of Hematology, Türkiye*

Case report: Hairy cell leukemia (HCL) represents a distinct subtype of mature B-cell lymphoproliferative disorders, predominantly affecting older individuals, with a median age of onset around 55 years. The disease exhibits a marked male predominance, with a male-to-female ratio of approximately 5:1. The spleen and bone marrow are the primary sites of involvement, and the majority of patients present with splenomegaly and pancytopenia at the time of diagnosis. Flow cytometric immunophenotyping (FCI) is an indispensable tool for the definitive diagnosis of HCL. The disease exhibits a characteristic immunophenotypic profile, defined by the absence of markers such as CD5, CD10, and CD23, and the presence of high-level or aberrantly bright expression of CD20, CD22, CD11c, and CD25. Furthermore, HCL cells are typically positive for CD103 and CD123. The current case report presents an atypical case of HCL with unexpected CD10 expression. A 36-year-old male with no comorbidities presented with a 1.5-month history of fatigue and exertional dyspnea, as well as B-symptoms. Physical examination revealed a palpable spleen in the left upper quadrant, with a dull percussion note over Traube's space, supporting the presence of splenomegaly. Initial complete blood count showed pancytopenia: a white blood cell count of 6210/μL (neutrophils: 190/μL; lymphocytes: 3120/μL; monocytes: 2880/μL), a hemoglobin level of 9.1 g/dL, and a platelet count of 56,000/μL. Further laboratory investigations for the etiology of anemia showed an iron level of 67 μg/dL, a transferrin saturation of 25%, a folate level of 9.4 ng/mL, and a vitamin B12 level of 270 pg/mL. A contrast-enhanced whole-body computed tomography (CT) scan revealed no pathological lymphadenopathy, but the spleen was measured at 140 $\times$ 60 mm. Peripheral blood smear analysis suggested the presence of atypical lymphoid cells with abundant cytoplasm. For a definitive diagnosis, a bone marrow biopsy was performed, and flow cytometry was conducted. Flow cytometry on the bone marrow samples demonstrated an increased percentage of B-lineage lymphocytes. These cells were positive for CD19, lambda light chain, CD20, CD22, FMC7, CD79a, CD27, CD11c, CD25, CD103, and CD10. HCL is a distinct lymphoproliferative disorder with an established immunophenotype essential for diagnosis. Our case, however, demonstrates atypical immunophenotypic features, posing a diagnostic challenge due to unexpected CD10 expression. While this occurrence is rare, a previous study identified aberrant CD10 expression in approximately 10% of HCL cases, suggesting such instances are not isolated. Although CD10 is a hallmark of other B-cell malignancies, such as follicular and Burkitt lymphomas, its presence should not automatically exclude HCL. In our case, the diagnosis was confirmed by the co-expression of the entire classic HCL marker panel. This highlights the crucial role of a comprehensive immunophenotyping panel, rather than reliance on a single marker, especially when faced with suboptimal morphological features or limited cell numbers. This case emphasizes the importance of expanding the differential diagnosis for CD10(+) B-cell lymphomas to include HCL. In conclusion, our case serves as a valuable reminder that the immunophenotypic profile of HCL can be more diverse than typically understood. Further research into the clinical and prognostic implications of this rare CD10 expression is warranted.

https://doi.org/10.1016/j.htct.2025.106140