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The Diagnostic Challenge of an Isolated Leukemic Presentation of Blastic Plasmacytoid Dendritic Cell Neoplasm (BPDCN) with Atypical FLT-TKD Mutation

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ABSTRACT

Blastic plasmacytoid dendritic cell neoplasm (BPDCN) is a rare and aggressive hematologic malignancy characterized by frequent cutaneous manifestations and diagnosed via immunophenotyping. BPDCN typically expresses CD56, CD4, and/or CD123 expression by flow cytometry and immunohistochemistry and is confirmed by the expression of plasmacytoid dendritic cell markers, including TCL1, CD303, CD304, and/or TCF4, in the absence of other lineage-specific markers. We report a rare case of BPDCN that presented without cutaneous lesions and with a diagnostically challenging immunophenotype consisting of dim CD4, lack of CD303, and expression of CD117. The patient also had an FMS-like tyrosine kinase (FLT3) missense mutation at a variant allele frequency (VAF) of 20% and a TP53 frameshift mutation at a VAF of 64%. Given the unusual presentation of BPDCN, high clinical suspicion was necessary for the diagnosis and clinical awareness of this presentation is critical to avoid diagnostic delays.

KEYWORDS: *BPDCN, tagraxofusp, immunophenotype, leukemia, pancytopenia*

INTRODUCTION

Blastic plasmacytoid dendritic cell neoplasm (BPDCN) is a rare and aggressive hematologic malignancy characterized by frequent cutaneous manifestations and often presents with bone marrow involvement. Recent discovery has shown that BPDCN derives from precursors of plasmacytoid dendritic cells (pDCs) and has a cell origin closer to myeloid than lymphoid precursors (Sapienza et al., 2019). Based on this discovery, BPDCN is now distinguished as its own category of myeloid malignancies separate from acute myeloid leukemia (AML) in the 2016 revision to the World Health Organization (WHO) classification of hematological malignancies (Arber et al., 2016). BPDCN is diagnosed via immunophenotyping and typically expresses CD56, CD4, and/or CD123 expression by flow cytometry and immunohistochemistry and is confirmed by the expression of plasmacytoid dendritic cell markers, including TCL1, CD303, CD304, and/or TCF4, in the absence of other lineage-specific markers.

In this case report, we highlight a rare case of BPDCN that presented without cutaneous lesions, and with a diagnostically challenging immunohistochemical and molecular profile with dim CD4, lack of CD303, expression of CD117, and presence of a FLT3 missense mutation at a significant VAF. Given the unusual presentation of BPDCN, clinical awareness is necessary to expedite diagnosis and avoid treatment delays.

CASE REPORT

A 32-year-old male with no significant past medical history presented to his primary care physician with fatigue and shortness of breath with exertion for 6 weeks. Initial labs were concerning for a macrocytic anemia with a hemoglobin of 11.7 g/dL. Patient continued to develop worsening exertional fatigue with inability to perform his usual exercise. Repeat labs several weeks later showed a hemoglobin of 8.4 g/dL, with a mean corpuscular volume of 108.9 µm3. Platelet count was 79,000 per µL and white blood cell count was 5.95 per µL with an absolute neutrophil count of 60 and 4% blasts. He was referred immediately to the Emergency Department (ED).

On presentation to the ED, the patient reported right rib pain, fatigue, and shortness of breath. The patient denied

alcohol, drug, or smoking use, and he took no medications. He worked as a nurse at a nearby hospital. He had no family history of cancer. He weighed 113.2 kg. His temperature was 98.3°F, pulse 80, respiratory rate 20, blood pressure 132/77 mm Hg, and O2 saturation of 100% on room air. On physical examination, he was not in distress but demonstrated tenderness to palpation in his right lateral ribs.

CT chest, abdomen, and pelvis showed small right-sided pulmonary nodules but was otherwise unremarkable. Peripheral blood smear (figure 1) revealed a population of morphologically immature cells with fine to reticulated chromatin, high nuclear to cytoplasmic ratio, inconspicuous nucleoli, slightly indented nuclei, and occasional perinuclear cytoplasmic vacuoles ("pearl necklace" appearance). Some of the cells had an eccentrically placed nucleus imparting a plasmacytoid morphology. Bone marrow biopsy (figure 2) revealed a hypercellular specimen for age (approximately 80% cellular) and high power (figure 2, insert) demonstrated a monotonous proliferation of cells with irregular nuclear contours and scant cytoplasm. Flow cytometric analysis of the marrow aspirate (figure 3) demonstrated an increased blast population of 68% that expressed CD117, CD56, CD123, dim CD4 and was negative for CD13, CD33, CD34, TdT, and other lineage-associated markers such as MPO, CD14, cytoplasmic CD3, CD19, and cytoplasmic CD22.

In house next generation sequencing (NGS) panel for common myeloid associated molecular abnormalities revealed a TP53 frameshift variant (p.N131Cfs*27) at a variant allele frequency (VAF) of 63.5%, a FLT3 missense variant (p.D835H) at a VAF of 19.8%, a KRAS point mutation (p.A146T) at a VAF of 5.6% and 3 NRAS substitutions (p.G12S, p.G12D, p.G12V) at VAF of 4.3%, 4.1% , and 2.7%, respectively. Karyotype revealed an abnormal complex, composite clone with deletions of chromosomes 2q and 6q, loss of 3p and 19p due to translocation between chromosomes 12 and 17, loss of 12p (including ETV6) and 17p (including TP53), and loss of chromosomes 17 and 18. Although these findings are nonspecific, complex karyotype including deletions of 12p and 6q are frequently detected in cases of BPDCN (Tang et al., 2018). Though our NGS panel is not validated to detect copy number alterations, the data was suggestive of IKZF1 deletion.

Patient was started on induction therapy with cytarabine and daunorubicin for a presumptive diagnosis of acute myelomonocytic leukemia (AMML). A repeat bone marrow biopsy on day 14 revealed a reduced yet persistent population of blasts accounting for 32% of all cells analyzed by flow cytometry. The blast population expressed the same immunophenotype as that of the initial biopsy except CD4 expression showed brighter expression by flow cytometry (figure 4). Additional immunohistochemical stains (figure 5) were ordered which showed TCL-1 expression and negative CD303. This combination of CD4, CD56, CD123, and TCL-1 positivity in the absence of other lineage defining markers confirmed a diagnosis of BPDCN. MYC IHC, the expression of which has been found to correlate with treatment response (Sakamoto et al., 2018), was negative. On genomic analysis of residual disease, although the original TP53 frameshift variant was present at a similar VAF (70%), FLT3, KRAS, and NRAS variants were negative. The lack of FLT3 and other variants on follow up testing suggests elimination of these subclones with initial treatment.

Biopsy performed on a left shin lesion was negative for involvement by BPDCN. The patient was started on tagraxofusp and received intrathecal chemotherapy with methotrexate for central nervous system prophylaxis. His hospital course was complicated by platelet transfusion refractoriness for which he was found to have Glycoprotein IV platelet antibody requiring platelet-matched transfusions. He was discharged to home. On day 20 of tagraxofusp, a repeat bone marrow biopsy showed no residual BPDCN or myeloid blast population.

Despite an excellent initial remission to tagraxofusp, after six cycles the patient had an aggressive relapse. Bone marrow biopsy showed marked disease involvement of the marrow with 63% of cells identified as blasts. By flow cytometry, the cells expressed CD117, CD56, CD123, HLA-DR and a small subset Tdt. By immunohistochemistry, CD4 was weakly positive in rare cells, CD117 was weakly positive in the majority of neoplastic cells, and CD123 was positive in the majority of neoplastic cells as well. The phenotype was similar to the prior bone marrow biopsies. However, TCL-1 positive cells were fewer than prior and genomic profiling detected several aberrations that were different from prior, including an additional TP53 deletion and deletions in TET2 and CDKN2A. Furthermore, the previously detected FLT3 missense mutation and KRAS and NRAS variants were not detected. These findings suggest clonal evolution of disease. The patient was referred to a clinical trial involving anti-CD123 chimeric antigen receptor-modified T-cells (CART) but died 91 days after his relapse and before lymphodepletion due to complications from his disease progression.

DISCUSSION

BPDCN is a rare and aggressive tumor with an incidence of 0.000045% (Sapienza et al., 2019). Cutaneous lesions are the hallmark of the disease and found in 85% of patients (Garnache-Ottou et al., 2019). Skin manifestations can include erythematous or violaceous nodules, plaques or tumors. Bone marrow involvement occurs in 96% of patients (Garnache-Ottou et al., 2019), and leukemic dissemination without cutaneous findings occurs in 23% of patients (Pagano et al., 2013). Immunophenotyping is the key to the diagnosis. Once the absence of specific lineage markers is noted, the expression of CD123, CD4, and/or CD56 should raise the possibility of BPDCN. BPDCN should then be confirmed by the expression of plasmacytoid dendritic cell markers,

including TCL1 (T-cell leukemia/lymphoma), CD303, CD304, and/or TCF4. Expression of isolated myeloid or lymphoid markers must not exclude the diagnosis.

This patient had an unusual presentation of BPDCN which delayed his diagnosis. First, he presented without cutaneous lesions and with an atypical immunophenotype with dim CD4 expression and expression of CD117, which is typically negative in BPDCN. Although CD4 is almost always expressed in BPDCN, the intensity of CD4 expression can be dim and/or partial; therefore, expertise in flow cytometric immunophenotyping (FCI), including experience in interpretating subtle immunophenotypic abnormalities, as well as technical aspects of FCI, are paramount to interpretation. Other possible reasons for the appearance of dim CD4 on the initial bone marrow evaluation could be due to physiologic differences between the leukemic cells present before and after therapy initiation. In addition, he expressed other uncommon immunophenotypic features for BPDCN, including negativity for the PDC-specific antigen CD303 and positivity for CD117. Lack of CD303 expression does not exclude this diagnosis if other PDC-specific antigens are expressed, and CD303 can be negative in up to 75% of cases that are positive for CD117 (Garnache-Ottou et al., 2019).

Lastly, this patient had an FMS-like tyrosine kinase 3 (FLT3) missense mutation at a significant VAF (20%). FLT3 mutations are usually associated with AML, but cases of FLT3 internal tandem duplication (ITD) have been reported in BPDCN (Pagano et al., 2013). Rare cases of BPDCN have been reported in the literature with FLT3 substitutions identified at a lower VAF more suggestive of a subclone or clonal hematopoiesis of indeterminate potential (CHIP) level of VAF unlike the more significant VAF seen in our case (Lei et al., 2017). Given our patient presented without skin findings, had an unusual immunophenotype for BPDCN, and had a FLT3 missense mutation at a significant VAF, a high index of suspicion was necessary for his diagnosis.

The origin of BPDCN has been strongly debated in recent years with new evidence suggesting the myeloid origin of BPDCN. In 2008, BPDCN was recognized as deriving from precursors of pDCs based on immunohistochemical evidence (Sapienza et al., 2014). In 2014, gene expression profiling showed molecular evidence that BPDCN most closely resembles resting pDCs and originates from a myeloid origin (Sapienza et al., 2014). Currently, neoplasms derived from pDCs are categorized as either BPDCN or mature pDC proliferations associated with myeloid neoplasms (MPDMN) (Facchetti et al., 2016). Whereas BPDCN is characterized by undifferentiated blast cells, MPDMN is associated with mature pDC proliferation in the setting of myeloid neoplasms, of which chronic myelomonocytic leukemia (CMML) is most common and rarely AML and myelodysplastic syndrome (MDS) (Zalmaï et al., 2021). Recently, a spectrum of pDC maturation distinct from BPDCN and MPDMN has been recognized in the disease group pDC-AML, which has been associated with a high frequency of somatic RUNX1 mutations (Pemmaraju, 2021; Xiao et al., 2021; Zalmaï et al., 2021). pDC-AML provides further evidence of a common origin between pDC and immature myeloid blasts and raises the prospect of a broader spectrum of pDC-associated neoplasms.

A clonal relationship between CMML and BPDCN has also been identified to suggest a common stem cell progenitor. BPDCN co-exists frequently with other myeloid malignancies, such as CMML and MDS and a number of case reports and case series have shown the clonal evolution of BPDCN from CMML (Espasa et al., 2021; Patnaik et al., 2018). BPDCN and CMML have also been found to share similar molecular alterations in early genetic events, such as TET2, SRSF2, and ZRSR2, to suggest a shared ancestral event (Batta et al., 2021; Brunetti et al., 2017; Espasa et al., 2021). Lastly, a high prevalence of bone marrow clonal hematopoiesis beyond an associated diagnosis of MDS or CMML has been found in BPDCN, which provides additional evidence of clonal evolution (Khanlari et al., 2022).

The genetic analysis of our patient highlights the clonal heterogeneity and evolution of BPDCN. The bone marrow biopsy after induction therapy with cytarabine and daunorubicin and the bone marrow biopsy at the time of disease recurrence both shared similar genetic mutations to the initial bone marrow biopsy as well as developed new genetic abnormalities. For example, the bone marrow biopsy at the time of disease recurrence showed the same TP53 variant as the original diagnosis as well as the previously identified IKZF1 mutation. However, a new TET2 variant and biallelic CDKN2A deletion were identified, and the previously identified FLT3 TKD, KRAS, and NRAS variants were not detected. These findings suggest clonal evolution of the disease following the eradication of a subset of clones by the initial therapy regimen. The TP53 rich subclone, which is generally associated with more aggressive neoplasms, persisted. Additionally, other previously minor clones appeared to have increased in prevalence either due to direct selective pressures from treatment or by chance mutations arising in the few surviving subclones that would eventually proliferate to become the dominant clones in the recurrent neoplasm. The fact that our patient had complete response to tagraxofusp and an insufficient response to the 7+3 regimen also provides further evidence of the BPDCN diagnosis.

The treatment of BPDCN is rapidly evolving but the overall prognosis of BPDCN is extremely poor. In 2018, tagraxofusp was approved by the US Food and Drug Administration (FDA) for treatment of BPDCN based on high response rates (Jen et al., 2020). Tagraxofusp is a CD123-directed cytotoxin consisting of recombinant human IL-3 truncated to diptheria toxin. In an open-label multicohort study of 47 patients with untreated or relapsed BDPCN, tagraxofusp led to a 90% overall response rate in 29 previously untreated patients,

and of these patients, 45% were successfully bridged to transplant (Pemmaraju et al., 2019). Survival rates in this previously untreated cohort at 18 and 24 months were 59% and 52%, respectively. In the 15 previously treated patients, the overall response rate was 67%, and the median duration of overall survival was 8.5 months (Pemmaraju et al., 2019). Prior to the introduction of tagraxofusp, therapies used for AML, ALL, and other types of lymphomas and leukemias were used.

After remission, allo-SCT is recommended. Retrospective reviews show that allo-SCT leads to a 3-year overall survival of 41% and that a myeloablative regimen is preferred (Roos-Weil et al., 2013). For those who cannot undergo allo-SCT, relapse is almost definite. However, given the novel treatment of tagraxofusp, long-term outcomes with tagraxofusp after remission and allo-SCT are unknown. Other targeted agents are also in development for BPDCN including humanized antibody-drug conjugates consisting of an anti-CD123 receptor monoclonal antibody conjugated to a DNA-alkylating agent, an anti-CD123/anti-CD3 bispecific monoclonal antibody, and anti-CD123 CART therapy (Economides, Konopleva, & Pemmaraju, 2019).

CONCLUSION

Given our patient presented without skin findings and had an unusual immunophenotype for BPDCN, the final diagnosis of BPDCN was challenging. In addition, to our knowledge, this is the first case report of BPDCN with a FLT3 missense mutation at a VAF of 20%. The patient had a complete response to tagraxofusp, but unfortunately relapsed after six cycles while preparing for allo-SCT. Given the unusual presentation of this rare disease, increasing awareness and recognition of such presentations of BPDCN is critical to expedite patients' treatment and increase chances of survival. Further research regarding the evolution of BPDCN is also integral to better understand, diagnose, and treat this disease.

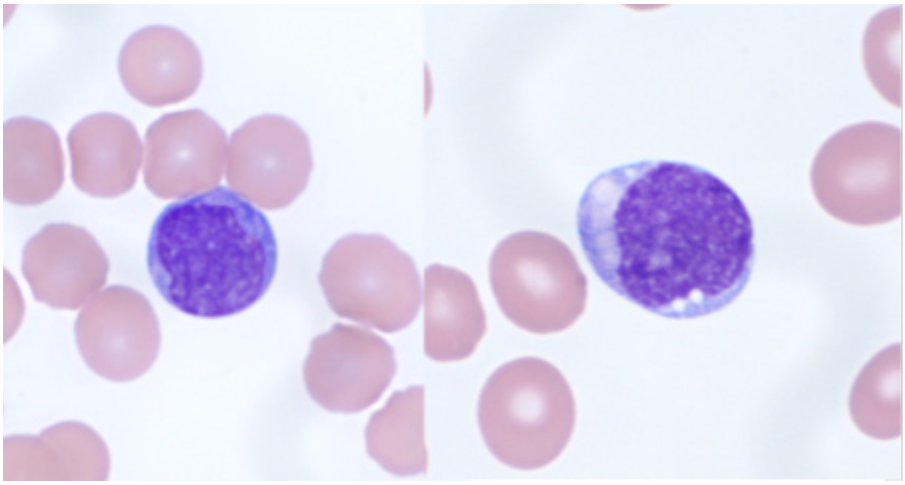

Figure 1. Peripheral blood smear demonstrating a blast population with fine to reticulated chromatin, high nuclear to cytoplasmic ratio, inconspicuous nucleoli, lobated nuclei, perinuclear cytoplasmic vacuoles ("pearl necklace"), and plasmacytoid morphology (right image).

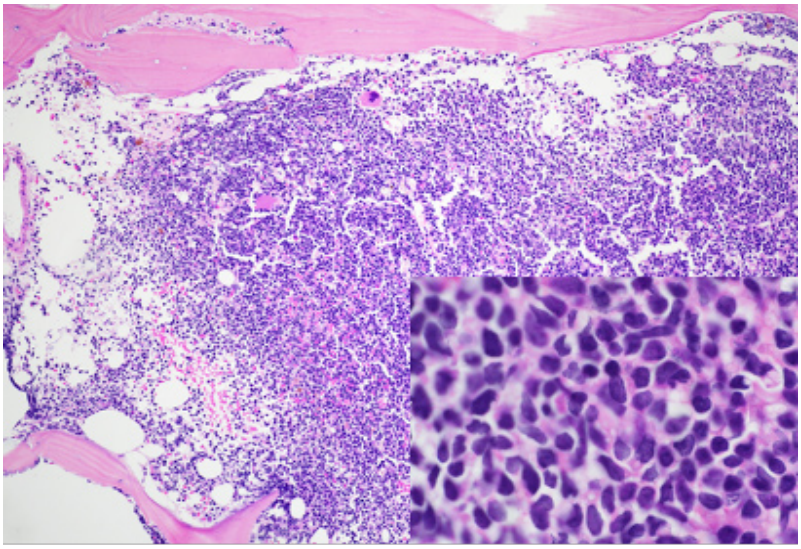

Figure 2. Core biopsy with hypercellular marrow for age (~80%). High power (insert) demonstrates monotonous proliferation of cells with irregular nuclear contours and scant cytoplasm.

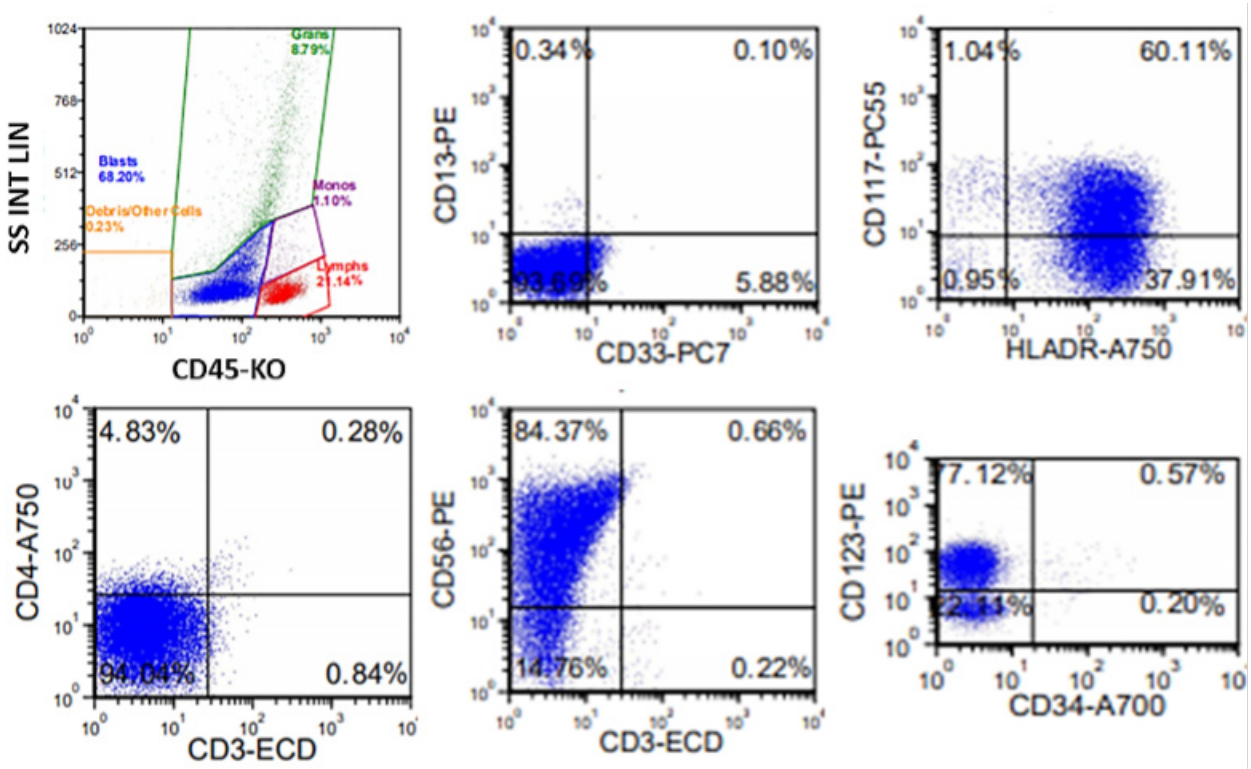

Figure 3. Flow cytometry from initial marrow biopsy demonstrating 68% blast population with expression of CD117, HLA-DR, CD56, CD123, dim CD4 with negative CD13, CD33, CD34, and CD3.

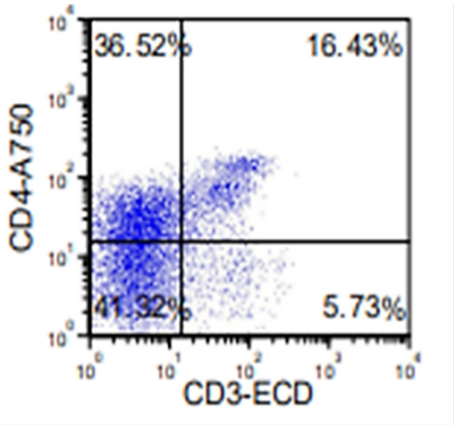

Figure 4. Flow cytometry scatterplot from post treatment marrow specimen demonstrating brighter CD4 expression within the blast population.

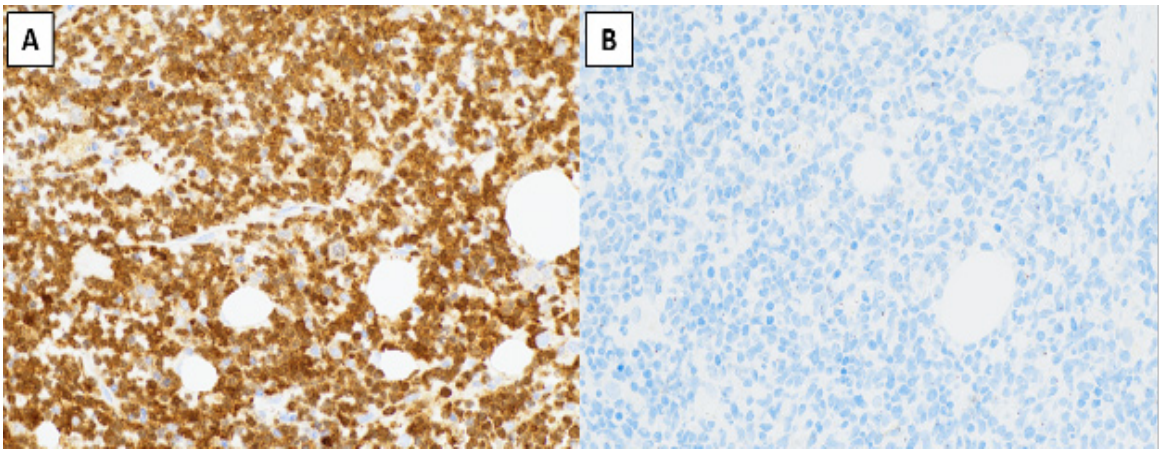

Figure 5. Immunohistochemistry demonstrating expression of TCL-1 (A) and lack of CD303 (B).

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