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T-Cell Blast Crisis in Chronic Myelogenous Leukemia with Response to Tyrosine Kinase Inhibitors & Chemotherapy: A Case Report

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Abstract

Chronic Myeloid Leukemia (CML) is a myeloproliferative neoplasm characterized by the dysregulated production and uncontrolled proliferation of mature and maturing granulocytes with relatively normal differentiation. Clinically, CML presents in three phases: chronic, accelerated, and blast crisis. Approximately 5% of cases are diagnosed in the blast phase, with 70% being myeloid and the remaining 30% being lymphoid blast crisis.

We present a 54-year-old male patient with chronic-phase CML, who had been taking Imatinib, presenting with cervical lymphadenopathy and diagnosed with a T-cell CML blast crisis.

Keywords: CML; Blast crisis; T-cell

Introduction

Chronic Myeloid Leukemia (CML) is a myeloproliferative disorder associated with the Philadelphia chromosome t(9;22)(q34;q11) and the BCR-ABL fusion gene.

Clinically, CML manifests in three phases: the chronic phase, which is the most indolent form of the disease and accounts for about 85%-90% of cases; the accelerated phase, which accounts for about 10%; and the blast crisis phase, which accounts for approximately 5% of CML cases.[1]

CML usually progresses from the stable chronic phase to the more advanced blast crisis phase within 4-5 years without treatment. The advent of Tyrosine Kinase Inhibitors (TKIs) has significantly delayed or even prevented the transformation to advanced forms in the majority of chronic-phase CML cases.[2]

However, certain patients experience resistance to these medications, which occurs through mechanisms such as the accumulation of additional cytogenetic abnormalities, conferring a survival advantage to treated myeloid cells and leading to disease transformation into accelerated or blast crisis. The most common cytogenetic abnormalities include the presence of an additional Philadelphia chromosome, trisomy 8, and abnormalities of chromosome 17q.[3]

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The WHO defines CML blast crisis by the presence of one or more of the following: ≥20% peripheral blood or bone marrow blasts, large foci or clusters of blasts in the bone marrow biopsy, and the presence of extramedullary blastic infiltrates.

In the majority of cases, blasts are of myeloid origin, accounting for about 70%, with lymphoid blasts observed in approximately 30% of CML blast crises.[4]

Lymphoid blast crisis resembles Acute Lymphoblastic Leukemia (ALL) with Philadelphia chromosome positivity. Most reports of CML blast crisis with lymphoid transformation describe B-cell transformations, though reports on T-cell lymphoid blast crisis are sparse.[5]

Here, we report a case of a 54-year-old male patient with chronic-phase CML who was transformed into a T-cell blast crisis, evidenced by both morphologic and immunologic studies.

Case Presentation

A 54-year-old male patient, known to have chronic-phase CML, had been on Imatinib (400 mg once daily) for the past 3 years. He had regular follow-ups, adhered to his medication regimen, and had been in complete hematologic remission for the past 3 months.

The patient presented with a 3-month history of progressively enlarging bilateral cervical lymphadenopathy, without associated axillary, inguinal, or abdominal swelling. He also reported symptoms of easy fatigability, tinnitus, and vertigo associated with the cervical swelling but denied any history of fever or bleeding from any site.

On examination, he had multiple firm, nontender cervical lymphadenopathies, with the largest measuring 3 cm × 3 cm. There were no other abnormal physical examination findings.

Laboratory parameters included:

- WBC: 4.2 × 10^3/μL
- ANC: 2.6 × 10^3/μL
- Hgb: 13.7 g/dL
- Platelet: 184 × 10^3/μL
- LDH: 219 IU/L
- Cr: 1.0 mg/dL
- Total bilirubin: 0.7 mg/dL
- Direct bilirubin: 0.1 mg/dL
- AST: 42 IU/L
- ALT: 41 IU/L
- AlP: 125 IU/L

Lymph node biopsy revealed monotonous, predominantly medium to occasionally large lymphoid cells with a coarse hyperchromatic pattern, inconspicuous nuclei, and frequent mitosis with a "starry sky" pattern.

Immunohistochemistry (IHC) of the lymph node biopsy revealed T-cell lymphoblastic lymphoma, with tumor cells strongly positive for CD3, TdT, CD7, and CD45, focally positive for CD5, and negative for CD34, CD117, with Ki67 at 80%.

Bone marrow biopsy, along with IHC, showed T-lymphoblastic lymphoma with sheets of atypical medium-sized lymphoid cells that were diffusely and strongly positive for CD3, TdT, and CD45, focally positive for CD5, and negative for CD20, CD34, and CD117.

BCR-ABL fusion was detected at 95% by FISH, with no other cytogenetic abnormalities noted.

Discussion

It is well established that approximately 30% of CML blast crises are of the lymphoid phenotype, with B-lymphocytes being the most commonly reported type of lymphoid blast crisis. However, the exact proportion of T-cell lymphoid blast crises is not well documented.[5]

Accurate identification of the blast phenotype has therapeutic implications, as the treatment protocols for lymphoid blast crisis differ from those for myeloid blast crisis.

In myeloid blast crisis, the preferred initial treatment is the use of a TKI followed by allogeneic Hematopoietic Cell Transplantation (HCT) for eligible patients, as the disease is typically refractory to chemotherapy. The goal in treating myeloid blast crisis is to return patients to an earlier phase of the disease, followed by allogeneic stem cell transplantation.[6]

CML lymphoid blast crisis is usually treated similarly to Philadelphia-positive Acute Lymphoblastic Leukemia (ALL), with the use of tyrosine kinase inhibitors in combination with chemotherapy or steroids.[6]

In this case, the patient transformed into blast crisis while on Imatinib treatment. He was switched to Dasatinib 140 mg daily in combination with dexamethasone and vincristine, as his performance status was poor, and he was considered unfit for an intensive ALL chemotherapy regimen. After 3 months of treatment, the patient showed improvement in clinical symptoms, with disappearance of the cervical lymphadenopathy, but did not achieve clearance of peripheral and bone marrow lymphoid blasts.

Conclusion

Although lymphoid blast transformation of CML is rare, a high index of clinical suspicion is required when chronic-phase CML patients develop lymphadenopathy.

T-cell blast crisis is extremely rare, and its exact incidence is not clearly known. This case highlights the importance of immunohistochemistry and genetic studies in accurately diagnosing the type of CML blast crisis.

Disclosure

Verbal informed consent was obtained from the patient.

The patient voluntarily consented to the reporting of this case and the continuation of care.

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