AUCTORES
Globalize your Research

International Journal of Clinical Case Reports and Reviews
Madeeha Subhan Waleed

Open Access

Case Report

A Rare Case of CML with Abnormally High TLC Count

Madeeha Subhan Waleed[1]*, Waleed Sadiq[2]
[1]Graduate, Ayub Medical College, Pakistan.
[2]Resident Physician, Staten Island University Hospital, New York, NY, USA.
***Corresponding Author:** Madeeha Subhan Waleed, Graduate, Ayub Medical College, Pakistan.

Received date: October 06, 2020; Accepted date: October 19, 2020; Published date: October 28, 2020
Citation: M S Waleed, W Sadiq. (2020) A Rare Case of CML with Abnormally High TLC Count. International Journal of Clinical Case Reports and Reviews. 4(1); DOI: 10.31579/2690-4861/074

Abstract

CML is a neoplastic transformation of the primitive hemopoietic stem cells.CML presents with increased TLC count and low lap score.Diagnosis is made using peripheral smear,flow cytometry or bone marrrow biopsy.The incidence of this disease has remained stable for the last decade.We present to you a young male with CML and abnormally high WBC count of 175 000/uL ,low LAP score and anemia.Philadelphiahia chromosome (9:22 translocation) is positive.

Keywords: neoplastic transformation; hemopoietic stem cells; TLC; CML

Introduction

Chronic myelogenous (CML) or chronic granulocytic leukemia (CGL) is a malignancy of the white blood cells.it accounts for 15% of the adult leukemias.in severe cases.it has three phases i.e chronic, accelerated and the blast phase.Blast crisis is defined as the presence of ≥ 30% marrow blasts [1]. The translocation of 9,22 chromosome leads to a fusion protein bcr-abl tha is an active tyrosine kinase .chronic myeloid leukemia (CML) patients receive imatinib which is a tyrosine kinase inhibitors as first-line therapy achieve good cytogenetic and molecular responses [2,3].

Case Presentation

A 39 year old previously healthy male presented to the hospital with weightloss,left sided chest discomfort and tightness for two months.These symptoms started gradually.The pain was burning in character which didnot radiate anywhere.There were no aggravating factors.It was intially relieved by pain killers but then slowly it increased to the point where he decided to seek medical attention.There was no associated shortness of breath,sputum production,cough or hemoptysis.Moreover,he also complained of generalized fatigue,malaise low grade fever for 2 months.He lost about 7 kgs during this period.On review of system there was no headache,visual changes,vomiting,urinary or bowel problems. Regarding his social history he is an electrician ,non-alcoholic and nonsmoker but eats naswar(sun dried tobacco leaves).On examination, he had no edema,pallor,petechia,pallor,clubbing or lymphadenopathy.Cardiovascular and respiratory exam was unremarkable.On abdominal exam,there was no tenderness.The spleen was palpable two finger breadth below the costal margin and the bowel sounds were hypoactive.It was decided to admit him and appropriate laboratory investigations were ordered.The patient was started on ceftriaxone and vancomycin since he was febrile.

Laboratory Workup: **Complete blood picture:**

Hemoglobin	9.3 g/dl
Tlc count	170750/mm3
Rbc count	2.98 mil/mm3
Platlet	279,000/mm3
metamyelocytes	20%
myelocyts	18%
blasts	5%

***Peripheral smear*:**

There was marked leuckocytosis with bimodal peak of neutrophils and immature granulocytes.Anemia with rbc morphology showing anisocytosis ,hypochromia,pokilocytosis with pencil cells. Platelets were adequate.Peripheral film was suggestive of chronic myeloid Leukemia.
ESR: 76/1st hour.
Blood metabolic profile:

Blood metabolic profile:

Total bilirubin	0.7mg/dl
Cknac	50 U/l
calcium	10.8 mg/dl
ASAT	23 U/l
ALAT	19 U/l
Alkaline phosphatase	433 U/l
Creatnine	0.9 mg/dl
Urea	24 mg/dl
sodium	140 mmol/l
potassium	3.2 mmol/l

Chest Xray: Normal study

Philidalpheia chromosome was positive.

Urine R/E: Normal

LAP score: 02

Diagnosis was confirmed with flow cytometry.

Discussion

CML occurs in 7% to 15% of all leukemias in adults with approximately 1 to 1.5 cases per 100,000 population [4, 5]. Asymptomatic cases of CML have increased from 15% to about 40% of all cases.A European study published in 2007 estimates the CML incidence to be 1-2 cases per 100,000 people every year [6].The activity of leukocyte alkaline phosphatase is reduced in nearly all patients at diagnosis [7].According to Medscape total WBC count 20,000-60,000 cells/µL in CML with mildly increased basophils and eosinophils while in our patient it was 175,000/uL. The most common feature of CML is an increased WBC count, usually above 25,000/µL [8]. Some patients have wide cyclic variations in their WBC count, with peak counts every few days or separated by up to 70 days [9]. The WBC differential usually shows granulocytes in all stages of maturation, from blasts to mature granulocytes that look morphologically normal.Most patients have mild anemia on diagnosis.

Conclusions

Patient suffering from CML may have a subtle presentation.Blood work up will often lead to diagnosis of CML.They may present with TLC count greater than 100,000 which is usually a feature of Acute Leukemia.Extreme leukoctytosis can cause leukostasis which itself contribute to additional complications.Imitanib should be started in bcr-abl positive patients to bring wbc count in the normal range and the response should be monitored as some patients might require leukaphereis.As CML is one of the four main types of leukemia that affects and accounts for 15 % of all the leukemias worldwide approximately,so proper workup and therapy should be started immediately to decrese mortality and morbidty.

References

1. Silver RT. (1968) Characteristics of the terminal phase of chronic granulocytic leukemia. Blood. 445–459.
2. O'Brien SG, Guilhot F, Larson RA et al. (2003) Imatinib compared with interferon and low-dose cytarabine for newly diagnosed chronic-phase chronic myeloid leukemia. N Engl J Med. 348, 994–1004.
3. Hughes TP, Kaeda J, Branford S et al. (2003) Frequency of major molecular responses to imatinib or interferon alfa plus cytarabine in newly diagnosed chronic myeloid leukemia. N Engl J Med. 349, 1423–1432.
4. Brincker H. (1982) Population-based age- and sex-specific incidence rates in the 4 main types of leukaemia. Scand J Haematol. 29:241–249.
5. Call TG, Noel P, Habermann TM. (1994) Mayo Clin Proc. 69:315–322.
6. M, Rohrbacher and J, Hasford. (2009) Epidemiology of chronic myeloid leukaemia (CML). Advances in Biology and Therapy of Chronic Myeloid Leukaemia. 22:295–302.
7. Rosner F, Schreiber ZR, Parise F. (1972) Leukocyte alkaline phosphatase. Arch Intern Med. 130:892–894.
8. Canellos GP. (1976) Chronic granulocytic leukemia. Med Clin North Am. 60:1001–1018.
9. Inbal A, Akstein E, Barak I et al. (1983) Cyclic leukocytosis and long survival in chronic myeloid leukemia. Acta Haematol. 1983, 69:353–357.