CASE REPORT

# Chronic Myeloid Leukemia With Intense Thrombocytosis: A Case Report

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## ABSTRACT

A case report of Chronic Myeloid Leukaemia (CML) with extreme thrombocytosis. A premenopausal woman with an age of 45-year-old was admitted with chief complaints of fatigue and generalized body weakness, upper abdominal pain and two episodes of recent gum bleeds without fever or other bleeding manifestations. No other comorbidities. On Examination of US Abdomen gross splenomegaly and mild hepatomegaly are observed. On investigation, patient was also observed with extreme thrombocytosis (10,500,000/mm$^3$) and leucocytosis with moderate anemia. By the above investigation of leucocytosis, patient was investigated for CML and found to be positive for BCR-ABL by Reverse Transcription PCR (RT-PCR). And prescribed with imatinib 400 mg/day and achieved a complete haematological response by the end of 3 months. On evaluation, patient responded well to imatinib treatment. In above case of CML, atypical presentation of disease was identified and treated with kinase inhibitors.

**Key Words:** Leukocytosis, BCR-ABL, RT-PCR, Thrombocytosis

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## INTRODUCTION

Chronic myeloid leukemia is a hematological malignancies arising from reciprocal t (9,2), (q34; q11.2) *BCR/ABL1* gene in wild primitive hematopoietic stem cells. Myeloid cells predominate at all series of maturation, and there may be an Elevated in Thrombocyte count (ET) and erythroid and megakaryocytic cells at shows peripheral blood smear; leukocytosis is the main clinical finding, though [1,2]. An elevated in myeloid precursor stem cells, neutrophils, basophils count, and eosinophils count is present at a peripheral blood smear and presence of underlying Philadelphia chromosome as a cytogenetic aberration or BCR-ABL gene transcript as a molecular chromosomal aberration. A few CML patients can have mild to moderate elevated platelets count with an acquired JAK2V617F point mutation in about 50% of cases [3]. CML may be two phases a biphasic or triphasic phase. This phase further classified toward the initial- chronic phase and a terminal blastic phase, which is preceded by an accelerated phase in 60% to 80% of cases [4]. In these particular groups of cases chromosome and thrombocythemia mostly present shows women [5]. This case study presents clinical findings hematological parameters and molecular genetic analysis data from this case of CML [6].

## CASE PRESENTATION

A 45 years old, 43.7 kg premenopausal lady was presented to oncology hospital. She was experiencing fatigue and generalized body weakness, upper abdominal pain. The vitals symptoms showed blood pressure 130/90 mm/hg, temperature 98°F, pulse rate 82 beats/min on admission day. After generalized examination than advised to the complete blood count, USG Abdominal and pelvis, peripheral blood smear and they are suspecting? CML so advised to BCR/ABL gene quantitative analysis. After clinical findings laboratory reports were patient had gross splenomegaly, mild hepatomegaly small cystic lession in the left lobe of the liver bilateral grade-1 renal parenchyma changes, peripheral blood smear finding to chronic myeloid leukemia with thrombocytes. And also Philadelphia chromosome or *BCR /ABL*-1 fusion gene analysis by Fluorescent *In situ* Hybridization (FISH) technic Bone marrow aspiration and the positive result for the Philadelphia chromosome test led to the diagnosis of CML.

### Treatment

The basis on the medical investigation physician-prescribed Tab Imatinib 400 mg OD but patient, not affordable drug cost than physician change to (Hydra) Cap Hydroxyurea-500 mg (QID) and (Zyloric) Tab Allopurinol -100 mg (OD) Tab metoclopramide 10 mg (TID), Tab Ranitidine 150 mg (BD) review after one week with CBC and BCR/ ABL gene Analysis report. Patient came with reports with *BCR /ABL* gene analysis sample RT-PCR has to show the presence of *BCR/ABL* gene transcript, in the mean, while at the time of diagnosis, risk classification was performed which revealed high Sokal, high Hasford and low Eutos risk scores [7]. Imatinib therapy (400 mg/day) was initiated and then stop the Cap Hydroxyurea-500 mg start to Tab Imatinib-400 mg OD for a one month review with CBC, serum creatinine, urea. After 3rd-month review, a complete haematological response was achieved. on regular follow-up, the patient was irregular than haemoglobin 6.4 g/dl, Blas cells 14%, myelocytes 26% then advised 2 pints of PRBC transfusion after Blood transfusion review with one month and also continuing Tab Imatinib 400 mg but the patient was irregular follow-up. After two months patient came with chief complaining of breathleness wheezing, Generalized weakness, Hb-5.9 g/dl Blast cells 06%, myelocytes 17% than advice to 2 pints of PRBC transfusion and Nebulization with Duolin+Budecort every 6$^{th}$ hourly, DNS with MVI 75 ml/hr receiving supportive medication patient hemodynamically stable and discharge review with one month with CBC. On regular follow-up Patient two episodes of gum bleeding and generalized weakness advised to Tab (Pause) Tranexamicacid-500 mg and IV fluids NS/DNS 75 ml/hr then a patient hemodynamically stable review after one month and continuing regular treatment follow-up.

**Outcome and follow-up:** This patient also responded very well to treatment with normalization of platelet count at 3 weeks and complete hematological response at the end of 3 months.

## DISCUSSION

Although, thrombocytosis is an predictable effect in CML. The cases of CML presenting with isolated thrombocytosis is rare and CML patients presenting with isolated thrombocytosis and different clinical and laboratory is typical. As the Splenomegaly and hepatomegaly, the common findings of CML (at 48-80% and 10-20%, respectively). In our case, patient is present with splenomegaly and hepatomegaly,



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thrombocytosis, leucocytosis, low haemoglobin, eosinophilia or basophilia, which are to be the common laboratory findings in classic CML cases but LDH and uric acid levels were within normal limits. Base on the high Sokal (score >1.2), high Hasford (score >1480) and low Eutos (score≤87) risk scores can be used for predicting the molecular responses in CML [7]. Initially first line tyrosine kinase inhibitors considered as a good progressive response treatment by Shailendra prasad Verma et.al case report as also be seen in our case report [8-10] (Table 1).

**Table 1 :** Relevant investigations performed during hospital stay.

| Hb (g/dl) | WBC cum/mm | Platelets / mm³ | N % | B % | E% | L% | Sr .Cr mg/dl | Urea mg/dl |
|---|---|---|---|---|---|---|---|---|
| 8.3 | 53,100 | 10,500000 | 50 | 10 | 5 | 10 | 0.85 | 21 |
| 8.5 | 8,400 | 10,900000 | 65 | 2 | 07 | 28 | 0.8 | 24 |
| 9.2 | 4,100 | 1,80000 | 63 | 5 | 0.7 | 30 | 0.9 | 18 |
| 11.4 | 7000 | 2,40000 | 60 | 3 | 0.9 | 31 | 0.9 | 18 |
| 11.9 | 35,300 | 5,8000 | 66 | 4 | 0.9 | 28 | 0.9 | 17 |
| 12.6 | 12,400 | 6,20000 | 70 | 3 | 10 | 19 | 0.9 | 24 |
| 9.4 | 7,6900 | 8,97000 | 87 | 4 | 5 | 22 | 0.9 | 22 |
| 7.2 | 2,18000 | 1,42000 | 50 | 0.5 | 2 | 0.4 | 0.9 | 22 |
| 6.4 | 40,6900 | 1,81000 | 60 | 1 | 0.1 | 0.9 | 1 | 25 |
| 5.9 | 45300 | 185000 | 47 | 0.8 | 0.5 | 1 | 0.9 | 22 |
| 7.3 | 396400 | 141000 | 60 | 5 | 0.9 | 0.4 | 0.9 | 25 |
| 6.4 | 293000 | 164000 | 52 | 3 | 2 | 0.5 | 0.1 | 21 |

## CONCLUSION

They also reported a case of CML mimicking with essential thrombocytosis in that cases patient advised for the FISH and RT-PCR for appropriate diagnosis. For Morphological examination, bone marrow aspiration, and biopsy can be advised in which we can observe megakaryocytes that were smaller and had a typical hyper lobed round nuclei are noted in CML patients. As in some of the studies, leucocytosis, basophilia, peripheral immature cells, and splenomegaly may be absent and the onset of disease or the initial presentations to the hospital but may develop later. By the above discussion we concluded that every case of Essential thrombocytosis must be tested for the Philadelphia chromosome. Some literatures also reveals case reports of Philadelphia positive (Ph+) ET without features of CML in peripheral blood. As by the above case report we advise the patients with extreme thrombocytosis should be investigated for BRC-ABL translocation for early diagnosis of CML. In our case, transcript analyses revealed a transcript that is confirmed by RT-PCR, and the patients responded extremely well to imatinib.

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