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Case Report
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The Rare Transition of Polycythaemia Vera to Chronic Myelogenous Leukaemia and Management in a Low-Resource Setting

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Abstract

Polycythaemia Vera (PV) is a cancerous condition that is characterized by excessive red blood cell production, carrying a risk of Vascular thrombosis and bleeding. PV often progresses to leukaemia or myelofibrosis; a transition to Chronic Myelogenous Leukaemia (CML) is, however, rare. A young boy, a daily wage farm worker and the sole breadwinner for the family, presented to the Primary Health Centre with a respiratory infection. His skin and eyes were unusually red. He had a massive spleen and a modest liver enlargement. Clinical findings and blood examination findings were consistent with both PV and CML. Following the patient's refusal of referral services, the Primary Health Centre (PHC) initiated affordable cytoreductive therapy with Hydroxyurea (HU) and aspirin. The cost of conventional PV treatment is exorbitant. HU has been shown to benefit both PV and CML. The initial evaluation cost was 25 USD, with daily medication costing 0.25 USD. A charity partly covered travel expenses for each visit to the center. This case of PV highlights a rare disease progression and demonstrates that an affordable HU option can benefit both PV and CML in low-resource settings.

Introduction

Polycythemia Vera (PV) is classified as a myeloproliferative neoplasm (MPN) by the World Health Organization [1]. There is a trilineage hematopoiesis in PV, i.e., increased production of red blood cells, white blood cells, and platelets. Characteristically, PV has mutations in exon 14 or exon 12 of JAK2 and has a high rate of vascular events such as thrombosis and bleeding.

Current treatment strategies in PV focus on preventing thrombotic complications rather than prolonging survival [2]. PV transforming to Chronic Myelogenous Leukaemia (CML) is a lesser-known progression compared to leukemic or myelofibrotic progression. We report a case of PV progressing to CML and a successful treatment with an affordable cytoreductive therapy using hydroxyurea.

Case Presentation

A 21-year-old boy from a remote subcentre, a daily-wage farm worker, presented to the Kamshet Primary Health Centre (PHC) with cough and fever. The ruddy skin and the redness of the eyes and nails were striking (picture). The blood report showed haemoglobin (14 g%) with the prominence of nucleated RBCs on the peripheral smear. White cell count was very high (2,64,200 cm^3) and fell within the range of hyperleukocytosis, with abundant immature white cells: band forms 11%, myelocytes 13%, promyelocytes 4%. There were no myeloblasts. Eosinophilia (9%) was also noted. The platelet count was 4,70,000 cm^3. The abdominal examination revealed an enlarged liver and a massively enlarged spleen, which was confirmed on abdominal USG. The chest X-ray showed a patch of pneumonia, for which antibiotics were prescribed. On a follow-up visit a week later, he felt better, and the chest signs of pneumonia had disappeared. The patient declined a referral to tertiary care and chose treatment at the PHC. With a proliferation of all three cell lines, cytoreduction was initiated with Hydroxyurea (HU), an affordable option, at an initial dose of 500 mg twice daily, later increased to 1 g twice daily. Aspirin, a blood thinner, was started at 75 mg once daily. The peripheral blood picture remained unchanged at the six-month follow-up, yet he reported feeling better. Aspirin was stopped when he had a bleeding nose. The dose was increased to 2 g/day when he complained of erectile dysfunction, although hesitatingly, since the PHC was ill-equipped to deal

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Figure 1: A photograph showing the ruddy complexion of the skin and injected conjunctivae.

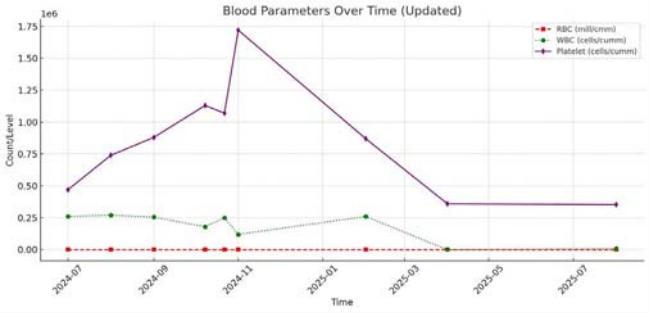

Figure 2: The graph shows changes in blood counts with treatment.

with any side effects that may crop up with the increased dose of HU. After the seventh month of treatment, a dramatic change was noted on the blood examination: Hb-10.3 g/dl, Total white cell counts 5400/ cu mm, P-20, L-60, E-8, and M-12. The platelet count was 3,63,000/ cu mm. No immature cells were spotted on the peripheral smear. The spleen was reduced to less than half its original size. The HU dose was decreased to 1.5 g/day.

Discussion

Our patient belonged to PV with a low-risk category for vascular events since he was less than 60 years old and had no history of thrombosis, as do more than half of PV cases at first presentation [3]. A high WBC count is also a risk factor for thrombotic events, even with a haematocrit level of ≤ 45, 4, and hence the aspirin therapy. Phlebotomy was not considered for our patient, nor was it possible at the PHC.

Cytoreductive drug therapy is recommended for the low-risk patients with PV in the presence of at least one of the following criteria: intolerance to phlebotomy, symptomatic, progressive splenomegaly, persistent/progressive leucocytosis, inadequate haematocrit control requiring frequent phlebotomies, persistently high cardiovascular risk, and persistently high symptom burden [5]. Our patient received HU since he had asymptomatic splenomegaly, hyperleukocytosis, and no access to phlebotomy.

PV progression/transformation to CML is a rarity, and PV and CML are mutually exclusive [6]. However, our patient has clinical and haematological findings of both PV and CML. PV features include ruddy skin colour, injected conjunctivae, and abundant normoblasts on the peripheral smear. CML features include massive splenomegaly, hyperleukocytosis, and mature and maturing granulocytes on the peripheral blood smear. Disease transformation to acute myelogenous leukaemia or myelofibrosis is well-known; our patient appears to have a rare transition from PV to CML [6].

Our resources did not permit us to perform genetic profiling and erythropoietin levels, or to demonstrate the Ph chromosome to aid the diagnosis. We also did not have interferon treatment for PV or tyrosine kinase inhibitors for treating CML. The economic costs of managing PV are substantial. Hence, their assessment is advisable in the treatment strategies [7]. Studies have shown that healthcare costs for patients with PV are higher and remain higher in patients with thrombotic events [8]. Indirect social costs are also high for patients with PV [9]. Without comparing the two settings, we can provide a bird's-eye view of the expenses incurred for our patients' medical care. Initial investigations included a complete blood count, a chest X-ray, and an abdominal ultrasound. These cost INR 2000 (approx. USD 25), and the recurring cost of HU and aspirin is INR 25 per day (approx. USD 0.25). The travel expenses incurred are INR 50 per PHC visit (USD 0.5). There was no cost to the patient on these counts, as a charity sponsored them. Loss of wages and travel expenses were partly compensated by paying him INR 100 (USD 1.25), though the actual cost could have been much higher. Even if the patient had agreed to seek specialist services in the public sector at no cost, the Out-of-Pocket (OOP) expenses could still have been considerable. In India, OOP expenditure accounts for more than three-quarters of the financial burden of health care, placing a strain on family resources [10], even when health insurance mechanisms are in place [11].

HU treatment would benefit both PV and CML. A study recommends HU for primary disease management following its successful use in 20 cases [12]. There are five reports of the successful use of HU in pregnancy. It may be the treatment of choice when leukapheresis is unavailable [13]. Affordability is a prime asset of HU therapy, especially when resources are scarce.

Conclusion

Here is a case demonstrating a rare transition from PV to CML, which was previously considered mutually exclusive. The patient shows clinical/haematological features of both. The response to HU has been good. HU may be a first-choice cytoreductive drug, especially when resources are scarce.

Ethical Statement

The appropriate ethics committee has reviewed the study. The patient provided informed consent prior to his inclusion in the study.

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