

Leukemic Retinopathy in Accelerated Chronic Myeloid Leukemia: A Rare Case from Indonesia



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ABSTRACT

Leukemia retinopathy is an uncommon and severe complication in chronic myeloid leukemia. Proper handling of this complication can save the patient from blindness. A 44-year-old man with accelerated-phase Chronic Myeloid Leukemia (CML) presented with visual blurring and floaters for the past one month associated with abdominal fullness and low-grade fever. There was splenomegaly, retinal neovascularization and Roth spots. Severe leukocytosis ($658.55 \times 10^9/L$), positivity for BCR-ABL p210, with additional laboratory findings were suggestive of CML. Hydroxyurea, allopurinol and tyrosine kinase inhibitors were initiated with improvement of hematological parameters but without halting the progression of vision loss. This case illustrates the need for early funduscopy and OCT to prevent serious irreversible complications, in conjunction with other systemic therapies.

Keywords: Leukaemia, Myelogenous, Chronic, Retinal Haemorrhage; Cotton Wool Spots, Ocular Manifestations, Vision Loss.

How to Cite this Article: Pamugkas J, Notopuro PB, Hajat A. Leukemic Retinopathy in Accelerated Chronic Myeloid Leukemia: A Rare Case from Indonesia. 2026;42(2):204-206. **Doi:10.36351/pjo.v42i2.2255**

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*Received: October 11, 2025
Revised: January 20, 2026
Accepted: February 06, 2026*

INTRODUCTION

CML is caused by a genetic abnormality that involves a reciprocal translocation of chromosomes 9 and 22 in hematopoietic stem cells and results in the formation of the BCR-ABL fusion oncogene. The recorded annual global incidence of CML is 1-2 per 100,000 adults, with the incidence of CML in Asia lower than in the Caucasian population.¹ CML is also associated with vision problems, more specifically, retinopathy caused by hyperleukocytosis, thrombocytopenia, and anaemia. Although retinal infiltrates, a hallmark of acute leukaemia, are absent in CML, the disease is still associated with certain retinal pathologies. Dot and flame-shaped retinal haemorrhages, cotton wool spots,

and retinopathy characterized by venous engorgement and multilayered haemorrhages are all part of the retinal findings in CML.² CML is associated with a unique vascular pathology, which explains the absence of retinal neovascularization, a pathological hallmark of ischemic conditions like diabetic retinopathy and retinal vein occlusion.³ Although Tyrosine Kinase Inhibitors (TKIs) are the most effective treatment for CML and are widely adopted as initial therapy, management of retinal complications associated with CML remains a challenge due to the potential for blindness. We highlight the need for early identification and a multidisciplinary management approach for patients with CML and ocular involvement. We also examined the correlation between hematologic parameters and retinopathy, which is still an understudied issue in Indonesia.

CASE REPORT

A 44-year-old man visited the ophthalmology clinic for blurred vision and black, thread-like floaters in both eyes that had persisted for a month. He also complained of systemic symptoms, such as

intermittent low-grade fever, cold sweats and abdominal fullness. The physical examination of the abdomen revealed abdominal fullness and splenomegaly.

The patient demonstrated a decrease in visual acuity (1/60 OD, 2/60 OS), with no abnormalities in extraocular movements, colour vision (assessed by Ishihara chart) and anterior segment examination. On fundoscopy, the optic disc margin neovascularization was noted, along with the presence of hard exudates, pigmentary changes in clumps, and Roth spots. Laboratory investigations revealed haemoglobin levels of 10.1 g/dL, haematocrit of 27.2%, leukocyte count of $658.55 \times 10^9/L$, and platelets at $445 \times 10^9/L$. The peripheral blood smear was normocytic normochromic, along with anisopoikilocytosis, pronounced leucocytosis with 17% of the leukocytes being myeloblasts showing advanced phase of CML (Figure 1).

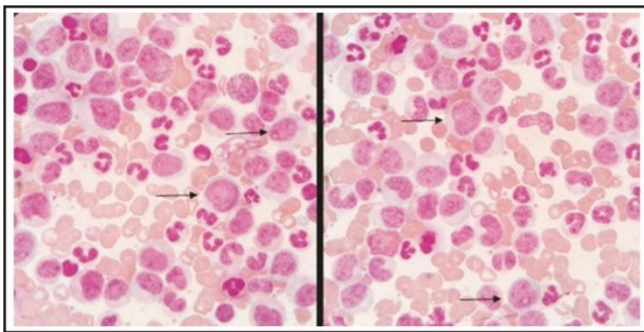


Figure 1: Peripheral blood smear showing myeloblasts (17%).

Confirmatory qualitative PCR for BCR-ABL p210, along with retinal ischemia was noted on OCT with superior macular thinning in the right eye and inferior-superior macular thickening in the left eye (Figure 2) along with Roth spots (Figure 3).

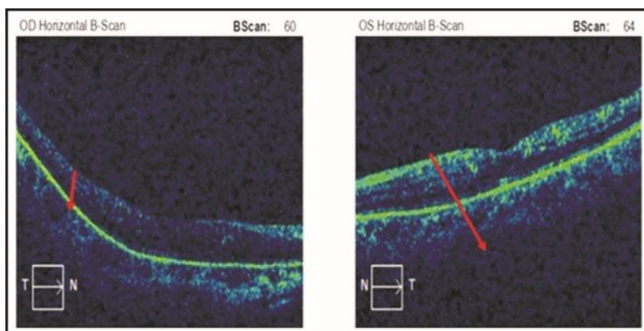


Figure 2: OCT of the right eye (macular thinning) and left eye (macular thickening).

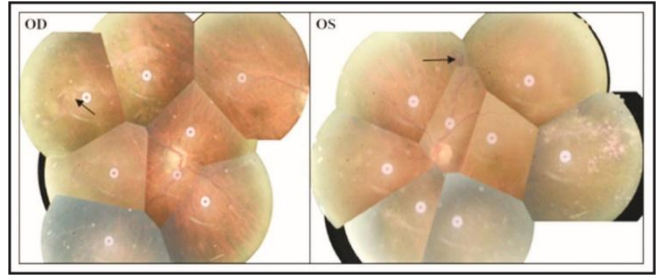


Figure 3: Fundus photo showing Roth spots and neovascularization.

To prevent tumour lysis syndrome, the patient was started on imatinib, hydroxyurea, and allopurinol. After two weeks, there was decrease in the leukocyte count and an improvement in the systemic symptoms. Visual impairment persisted, indicative of irreversible damage to the retina.

DISCUSSION

One of the CML complications is leukemic retinopathy. The condition is poorly documented in Indonesia.⁴ The patient in question demonstrated severe hyperleukocytosis, anaemia, and moderate thrombocytosis, coupled with blurred vision, floaters, Roth spots, and neovascularization, strongly indicating hematologic dysregulation and the presence of bilateral retinopathy due primarily to retinal microvascular disease.⁵ In CML, hyperleukocytosis may progress to leukostasis and blood hyper viscosity syndrome with the potential to compromise the perfusion of retinal capillary beds and precipitate capillary zone ischemia.⁶ These ischemic conditions, while differing in cause, may closely mimic the clinical picture of diabetic retinopathy, retinal vein occlusion and result in impairment of vision. Psychosocial stress associated with vision defect may further predispose to and aggravate the ischemic process due to the increase in blood hypoxemia. The hypoxemic effect of systemic anaemia, particularly of the normocytic type, encourages the progression of hypoxia-induced injury, while dysfunctional platelets, due to the hyperleukocytosis, aid in increased vascular collapse of petechiae.⁷

While rare, neovascularization can occur due to prolonged retinal ischemia caused by hyperleukocytosis and anaemia, as seen in this case.⁸ It illustrates the importance of implementing systemic therapy to prevent vision loss due to neovascularization. Targeted therapy using TKIs

remains the standard.⁹The patient experienced significant biological responses while on a combination of imatinib, hydroxyurea, and allopurinol.

This case highlights the importance of multidisciplinary approach to prevent irreversible vision loss. Direct haematology and ophthalmology consultation can ensure timely diagnosis and management of vision-threatening conditions. Although ocular ischemia produced by TKIs can lead to serious sequelae, ischemia should be monitored.

CONCLUSION

CML can cause vision problems as a complication of leukemic retinopathy. However, this is related to hematologic issues, not the direct leukemic infiltration. Loss of vision can be minimized with early diagnosis, a complete hematologic workup, and regular follow up by an eye specialist. Fundoscopy and OCT scans of the retina are critical, although the condition is managed primarily with systemic treatment. Clinicians should remain alert to the diagnosis and systemic symptoms of retinopathy.

Funding: This study was not funded by any organization.

Patient's Consent: Researchers followed the guidelines set forth in the Declaration of Helsinki.

Conflict of Interest: Authors declared no conflict of interest.

Acknowledgements

The authors appreciate the collaboration with the Hospital's Departments of Clinical Pathology and Ophthalmology for the support in patient diagnosis and management. The authors also acknowledge the Faculty of Medicine, Airlangga University, for assistance in the development of the case report.

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