

From the Retina to the Bone Marrow: Ophthalmic Revelation of Acute Myeloid Leukemia

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Abstract

Case Report

Acute myeloid leukemia [AML] is a hematological malignancy characterized by the accumulation of immature myeloid cells in the bone marrow and peripheral blood. Ocular manifestations are frequent and may result from direct leukemic infiltration, hematological abnormalities, or opportunistic infections. We report a case in which acute bilateral visual loss was the inaugural manifestation of AML type 5. A 26-year-old woman with no prior medical history presented with sudden bilateral visual loss associated with asthenia, dyspnea, and amenorrhea. Fundoscopic examination revealed bilateral papilledema, Roth spots, peripapillary hemorrhages, and macular star exudates consistent with leukemic retinopathy. Laboratory workup demonstrated severe pancytopenia, and bone marrow biopsy confirmed AML type 5. The patient received transfusion support and standard chemotherapy. At eight weeks, visual acuity improved from 20/50 to 20/25 bilaterally, with near-complete fundoscopic resolution and normalization of retinal nerve fiber layer thickness on OCT. This case underscores the diagnostic significance of ophthalmic examination in detecting underlying hematological malignancies. The triad of papilledema, retinal hemorrhages, and Roth spots in a young patient with systemic symptoms should prompt urgent hematological workup. Close ophthalmologist-hematologist collaboration is essential to optimize both visual and vital prognosis.

Keywords: Acute myeloid leukemia, Ocular manifestations, Papilledema, Roth spots, Visual loss, Leukemic retinopathy.

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INTRODUCTION

Acute leukemia is a group of hematological malignancies arising from abnormal proliferation of hematopoietic precursors [1]. Based on the cell lineage involved, they are classified into acute myeloid leukemia [AML] and acute lymphoblastic leukemia [ALL]. AML predominantly affects older adults, with a median age at diagnosis of 68 years, whereas ALL, though mainly a pediatric disease, carries a particularly poor prognosis when it occurs in adults [2,3].

Ocular involvement is a well-recognized complication of acute leukemia, reported in up to 50% of patients at the time of diagnosis, and may affect all intraocular structures [1]. It arises through two distinct mechanisms: direct infiltration of ocular tissues—including the retina, choroid, optic nerve, iris, ciliary body, and anterior chamber or indirect involvement secondary to hematological abnormalities such as

anemia, thrombocytopenia, and hyperviscosity [4]. Indirect manifestations include retinal, pre-retinal, and vitreous hemorrhages, Roth spots, cotton-wool spots, hard exudates, venous tortuosity, retinal vein occlusions, and, less commonly, neovascularization [5]. Papilledema, though less frequently reported, may also occur as a consequence of optic nerve infiltration, raised intracranial pressure secondary to leukemic meningeal involvement, or severe anemia-induced hypoxia [6]. Notably, ophthalmic manifestations may represent the initial presentation of systemic disease or serve as an early marker of hematological relapse, underscoring the diagnostic role of the ophthalmologist in the leukemia care pathway [4,7]. We report the case of a 26-year-old patient whose sudden visual loss was the first revealing sign of acute myeloid leukemia type 5.

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CASE PRESENTATION

A 26-year-old female patient with no prior medical history presented with sudden bilateral visual loss, without redness or pain. On history taking, she reported marked asthenia, dyspnea on minimal effort, and secondary amenorrhea for 5 months. Best-corrected visual acuity [BCVA] was 20/50 bilaterally. Intraocular pressure was at 15 mmHg. The conjunctivae appeared pale, while the anterior segment was within normal limits. Dilated fundus examination uncovered a striking bilateral picture: major papilledema with peripapillary

flame-shaped hemorrhages, Roth spots, macular star-shaped exudates, and diffuse vascular tortuosity [Fig. 1]. Multimodal imaging was performed. Optic nerve head OCT demonstrated significant bilateral peripapillary retinal nerve fiber layer [RNFL] thickening. Macular OCT revealed no macular edema but confirmed the presence of exudates in both eyes. Fluorescein angiography showed no evidence of vasculitis or posterior uveitis. Orbital and cerebral MRI demonstrated normal brain parenchyma with no optic nerve infiltration and normal cisternae and ventricular system, effectively ruling out meningeal intracranial hypertension.

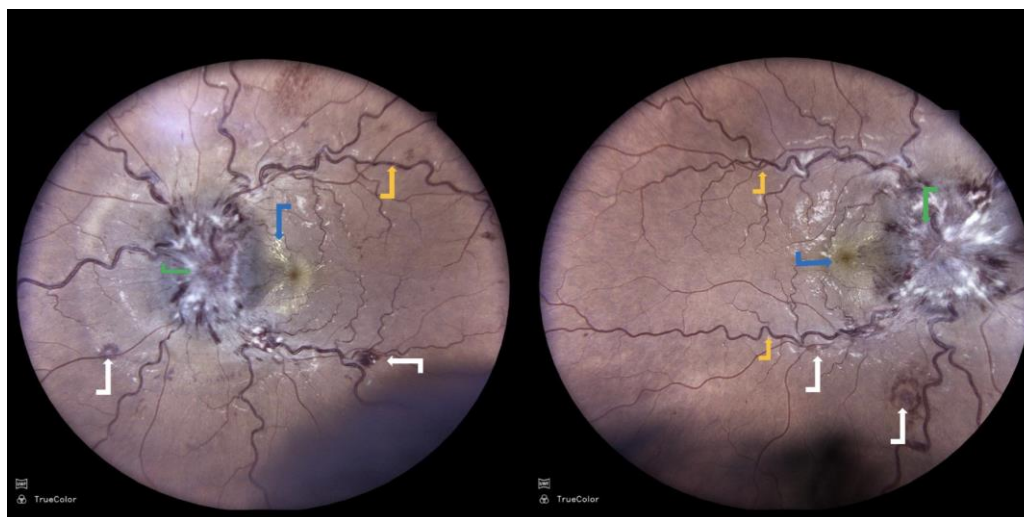


Fig. 1: Fundus photographs at initial presentation, showing bilateral leukemic retinopathy. Green arrows indicate bilateral optic disc edema, white arrows highlight Roth spots [white-centered retinal hemorrhages], blue arrows denote macular star exudates, and yellow arrows illustrate vascular tortuosity

Laboratory workup revealed severe pancytopenia, as summarized in Table 1. A peripheral blood smear showed 12% monocytes, 16% promonocytes, and 3% blasts. Infectious workup and

serologies were negative. In light of these findings, the patient was admitted to the hematology department for further diagnostic workup.

Table 1: Abnormal Complete Blood Count

Parameters	Values	Reference Values	High H, low L, or normal N
Hemoglobin	4.7 g/dL	12.0 – 16.0 g/dL	L
Hematocrit	14.1%	36 – 46%	L
Mean Corpuscular Volume [MCV]	85.2 fL	80 – 100 fL	N
White Blood Cells	$3.3 \times 10^3/\mu\text{L}$	$4.0 - 10.0 \times 10^3/\mu\text{L}$	L
Platelets	$75 \times 10^3/\mu\text{L}$	$150 - 400 \times 10^3/\mu\text{L}$	L
Reticulocytes	12,000/ μL	25,000 – 100,000/ μL	L

Bone marrow biopsy showed a hypercellular marrow infiltrated to 82% by a monocytic component [monoblasts 19%, promonocytes 63%, monocytes 2%]. Myeloperoxidase was positive. Immunophenotyping identified 45% myeloid blasts expressing HLA-DR, MPO, CD33, CD64, and CD34, confirming the diagnosis of acute myeloid leukemia type 5. Management included transfusion support with red blood cell and platelet concentrates to correct the severe pancytopenia, followed by standard '7+3' induction chemotherapy [cytarabine for seven days combined with daunorubicin for 3 days]. Antibiotic prophylaxis with

piperacillin/tazobactam was administered throughout the post-chemotherapy aplasia period. Following completion of induction chemotherapy, the patient was subsequently managed by the hematology team for further disease-specific treatment.

At three weeks post-chemotherapy, visual acuity remained stable at 20/50; however, fundoscopic examination showed marked improvement with resolution of Roth spots and regression of retinal hemorrhages and papilledema, while macular star exudates persisted [Fig. 2].



Fig. 2: Regression of leukemic retinopathy three weeks after initiation of chemotherapy

At eight weeks post-chemotherapy, improvement was remarkable. BCVA reached 20/25 in the right eye and 20/20 in the left eye, with near-complete resolution of papilledema and hemorrhages [Fig. 3].

OCT confirmed normalization of RNFL thickness, an objective sign of recovery without major axonal sequelae.



Fig. 3: Near-complete resolution of fundus findings eight weeks after initiation of chemotherapy

DISCUSSION

Ocular manifestations occur in 28-50% of AML cases, with retinal hemorrhages and Roth spots as the most frequent findings, and may precede the hematological diagnosis, making the ophthalmologist a key first-line diagnostician [7,8]. In our case, the leukemic retinopathy was primarily driven by indirect mechanisms: severe anemia [Hb 4.7 g/dL] caused retinal hypoxia with capillary fragility, thrombocytopenia [$75 \times 10^3/\mu\text{L}$] led to hemorrhagic complications; and hyperviscosity induced venous stasis and bilateral papilledema [8,9].

In AML, papilledema may arise from indirect venous stasis due to hyperviscosity or from direct leukemic infiltration of the optic nerve [6,7]. Direct infiltration of the optic nerve was ruled out by normal MRI. Roth spots result from rupture of retinal capillaries followed by platelet-fibrin thrombus formation,

explaining their rapid resolution by week 3 under effective treatment [9-11]. While posterior segment involvement predominates, anterior segment manifestations, such as subconjunctival hemorrhages, lid ecchymosis, conjunctival chemosis, proptosis, uveitis, and hypopyon, may also occur [12,13]. Hypopyon, in particular, when present, is considered an early warning sign of CNS involvement or systemic relapse [4,7]. AML type 5, characterized by monocytic differentiation, exhibits a well-documented tropism for extramedullary sites such as the skin, gingiva, meninges, and eye. It was attributed to the migratory properties of monocytes and their capacity for endothelial adhesion and tissue infiltration [8,14]. Mastrogriuseppe *et al.* Recently highlighted the eye's potential role as a "sanctuary site" for leukemic cells in monocytic AML subtypes, where the blood-ocular barrier may limit chemotherapy penetration and favor local persistence of disease [14]. This biological particularity may partly explain the

severity and multifocality of ocular involvement observed at first in our patient and reinforces the ophthalmological monitoring throughout the course of treatment. The visual recovery observed in our patient, along with near-complete RNFL normalization on OCT, confirms the reversibility of indirect vascular lesions under systemic chemotherapy, consistent with the literature reporting resolution of fundus lesions within the first two months of induction treatment [12,15].

CONCLUSION

This case reminds us that the eye can be the first window through which a life-threatening disease reveals itself. In our patient, a routine examination not only led to the diagnosis of AML but also provided a tangible reflection of disease activity and treatment response over time. The rapid resolution of retinal lesions and the normalization of RNFL thickness mirrored the hematological recovery, reinforcing the idea that ocular findings are not merely a complication to manage but a clinical marker to follow. More broadly, this case advocates for a closer and more systematic collaboration between ophthalmologists and hemato- oncologists, where multidisciplinary expertise ultimately serves the patients' best interest.

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