Ninety percent of physicians use smartphones.[3] An Apple iPhone5s iOS7 (www.apple.com/ios/) with the factory-enclosed Compass application (Chapter 25, manuals.info.apple.com/MANUALS/1000/MA1565/en_US/iphone_user_guide.pdf) can be used to estimate the magnitude of head tilts, face turns, and chin-up or chin-down postures. In the Compass application, measurements obtained from the plane of the AHP are subtracted from the appropriate frontal or sagittal anatomic plane to yield estimations.

Face turns, or rotation along the longitudinal axis (*y*-axis), can be estimated by standing above the patient's head and orienting the phone parallel to the floor. The difference between the compass reading obtained from aligning the compass along the naso-occipital versus the midsagittal plane estimates the degree of face turn.

Head tilts toward the shoulder, or rotation around the naso-occipital axis (*z*-axis), can be estimated orienting the phone in front of the patient's face and subtracting the compass measurement of the nasomental line from the perceived sagittal plane.

Chin-up and chin-down postures, or rotation around the interaural axis (*x*-axis), can be estimated by standing at the patient's ear side and comparing the degree of flexion or extension with the angular separation from the frontal anatomic plane.

The free iHandy Level app (itunes.apple.com/us/app/ihandy-level-free/id299852753?mt=8) may also be used as an alternative to estimate head tilts and chin-up and chin-down postures. The Compass application in iOS7 also has an enclosed level, when the screen is swiped to the left.

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Chronic myelogenous leukemia presenting with bilateral optic disc neovascularization

Clinically evident ocular involvement is common in patients with leukemia and has been described in up to 50% of patients at the time of diagnosis.[1] The 2 main types of ocular involvement include direct leukemic infiltration of ocular tissues and secondary leukemic retinopathy (e.g., retinal hemorrhages, cotton wool spots) from hematological complications of leukemia like anemia and hyperviscosity.[2] Peripheral neovascularization, secondary to hyperviscosity and capillary nonperfusion, is common in patients with chronic myelogenous leukemia (CML), occurring in approximately 78% of cases.[3] However, CML rarely displays posterior pole proliferative retinopathy or optic disc neovascularization. We report a case of CML that presented with bilateral optic disc neovascularization.

A healthy 46-year-old white male presented with decreased vision (20/30 OD, 20/25 OS) associated with headaches. Dilated fundus examination revealed bilateral optic disc neovascularization, white-centered retinal hemorrhages, venous dilation with areas of telangiectasis, and retinal neovascularization (Fig. 1). Fundus fluorescein angiography indicated leakage from optic nerve and retinal neovascularization with widespread areas of peripheral nonperfusion and some posterior capillary dropout in both eyes (Fig. 1). The patient had an elevated white blood cell count of 243 900 cells/mL (normal 4300–5700 cells/mL) with a hemoglobin of 7.9 g/dL (normal 13.8–17.2 g/dL). Magnetic resonance imaging of the brain revealed a frontotemporal lobe lesion that was excised by craniotomy and determined to be a granulocytic sarcoma of myeloid cells. A BCR-ABL translocation [t(9:22)(q34;q11.2)] was present on fluorescence in situ hybridization resulting in the formation of the Philadelphia chromosome. The diagnosis of CML with severe leukemic retinopathy was made and the patient was treated with the tyrosine kinase inhibitor dasatinib.

Over the course of 5 months, despite good control of systemic disease, his CML retinopathy progressed with recurrent preretinal and vitreous hemorrhages in both eyes (Fig. 2). Treatment was initially performed with bilateral full panretinal photocoagulation. Despite photocoagulation, 3 months later, our patient developed bilateral tractional retinal detachments (Fig. 2). Pars plana vitrectomy including membranectomy, endophotocoagulation, and air-fluid exchange followed by gas-air exchange with 16% perfluoropropane (C_3F_8) was performed in both eyes. The final vision was 20/25 OU with no signs of active retinopathy (Fig. 2). The patient continues to be in remission of his CML.

Peripheral neovascularization is common in patients with CML and was first described in 1968.[3] Hyperviscosity causes peripheral nonperfusion and ischemia

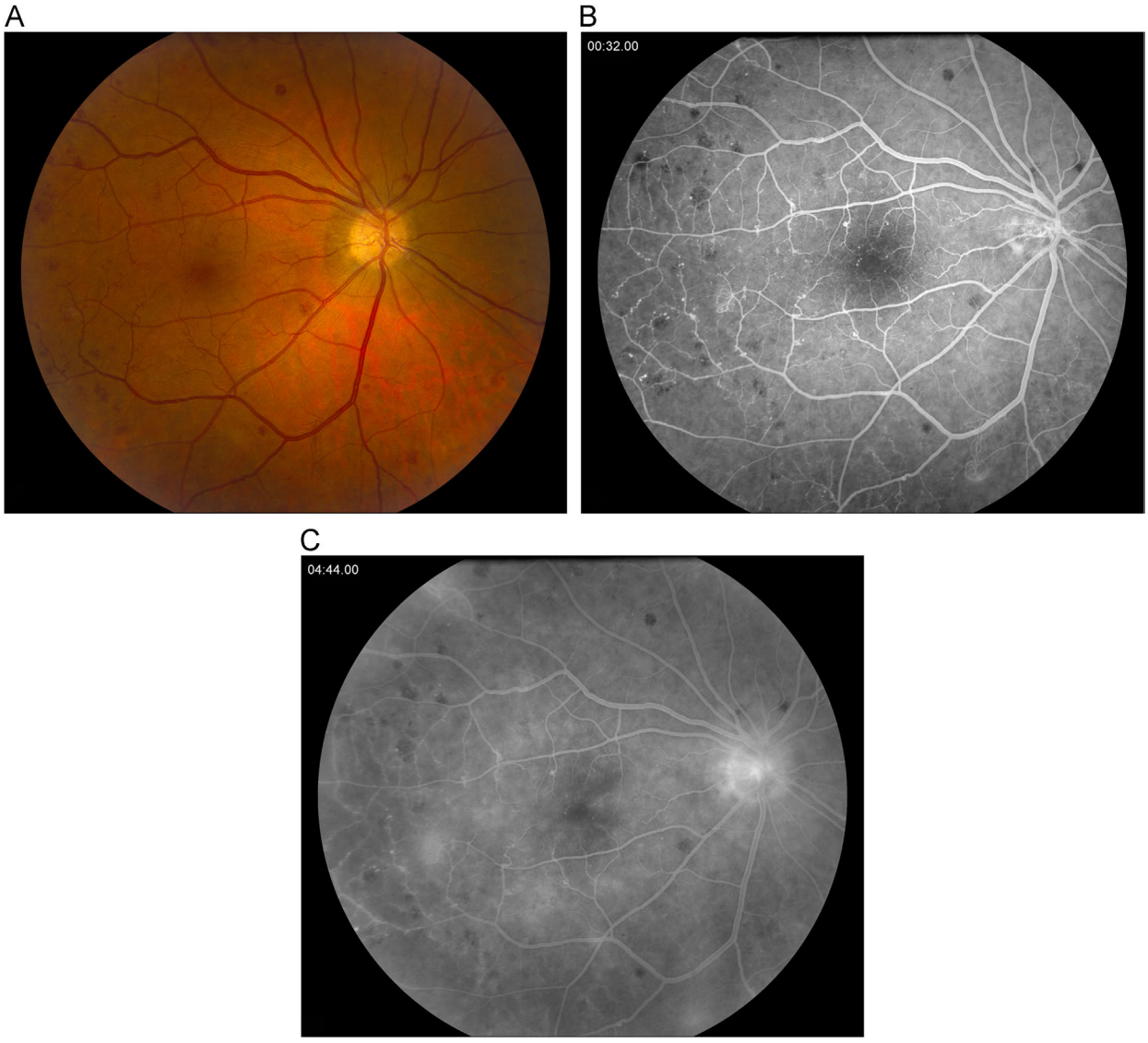

Fig. 1—Fundus photography and intravenous fluorescein angiogram of the right eye at initial presentation. A, There are extensive white-centered hemorrhages with areas of retinal telangiectasis and venous dilation. Optic disc neovascularization is evident. B, Arteriovenous phase of the angiogram reveals nonperfusion and capillary dropout with telangiectatic vessels throughout the macula. C, Late phase of the angiogram reveals leakage from optic disc neovascularization. The left eye was similar with presence of white-centered hemorrhages and optic disc neovascularization.

resulting in capillary dropout with possible formation of neovascularization. This predilection for the retinal periphery in CML can present with advanced proliferative retinopathy[4]; however, this tends to spare the posterior pole within the arcades.[5] Even more rare is the presence of optic disc neovascularization in CML because this is almost always associated with acute leukemia.[6] In contrast with proliferative diabetic retinopathy, it is extensively reported that the neovascularization observed in patients with CML typically remains in the midperiphery to far periphery and spares the optic nerve.[4–8]

Our case illustrates that, although rare, CML retinopathy may initially present with bilateral optic disc neovascularization. The case is instructive that once the capillary dropout occurred, the neovascularization and traction developed despite good control of the systemic disease with normalization of the high white cell count. The rapid progression of retinopathy also highlights the importance of prompt diagnosis and treatment, and we recommend peripheral blood analysis be ordered in patients who present with white-centered hemorrhages or retinal hemorrhages not in keeping with known ocular or systemic diseases. As a corollary, we suggest to our medicine colleagues that all patients with newly diagnosed leukemia of any type be referred for ocular examination regardless of whether they are symptomatic.

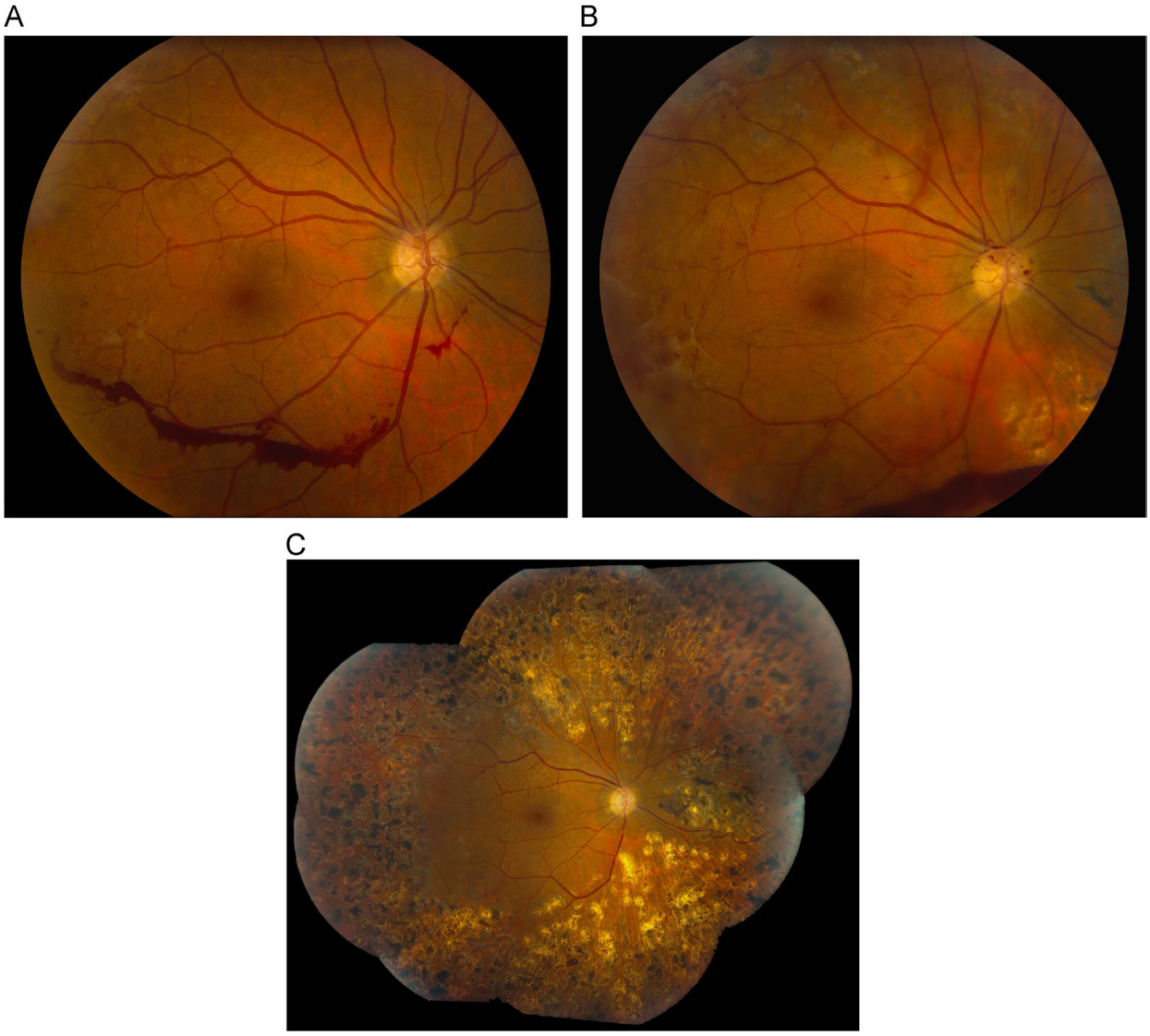

Fig. 2—Fundus photography for the right eye. A, Preretinal hemorrhage is noted along the inferotemporal arcade with venous dilation and optic disc neovascularization. B, Leukemic retinopathy progressed to tractional retinal detachment of the temporal macula with further preretinal and vitreous hemorrhage. C, Ultimate treatment with pars plana vitrectomy, membranectomy, and endophotocoagulation was needed to stabilize the eye. The left eye showed similar findings.

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