consequences to the patient through toxic impurities and microbial contamination.

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The authors report no conflicts of interest.

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Idiopathic or Secondary?

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I read with interest the clinical observation by Mohammad et al (1) of "idiopathic intracranial hypertension" associated with the topical application of vitamin A. Whether or not one has abandoned the term "idiopathic intracranial hypertension" for the term "primary pseudotumor cerebri," as recommended by De Simone et al (2), it would seem to me that this case does not fall under the rubric of "idiopathic" as it has at least a presumed etiology, that is, vitamin A, and would be better termed "secondary pseudotumor cerebri."

The author reports no conflicts of interest.

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Diagnosing Chronic Lymphocytic Leukemia With Temporal Artery Biopsy

We read with interest the report of Ghinai et al (1) documenting how the diagnosis of light-chain amyloidosis was established with temporal artery biopsy in a patient suspected of having giant cell arteritis (GCA). We would like to share our experience with a patient in whom temporal artery biopsy ultimately led the diagnosis of chronic lymphocytic leukemia.

A 74-year-old man developed acute painless vision loss in his left eye. On examination, visual acuity was 20/20, right eye and 20/40, left eye with diminished color vision in the left eye and a left relative afferent pupillary defect. The remainder of the ophthalmic examination was normal.

The patient's blood pressure was 123/86 mm Hg. Normal laboratory testing including erythrocyte sedimentation rate (4 mm/h), platelet count, glucose, cholesterol, and triglycerides. Leukocyte count was 12.6×10^9 cells/L (normal range: $3.5 - 10.0 \times 10^9$ cells/L). Echocardiography and ultrasonography of the carotid arteries were unremarkable.

Two months later, the patient complained of visual field loss in the right eye although visual field testing was unremarkable in that eye. There was an inferior altitudinal defect in the left eye.

Six months after his initial visual complaints, the patient reported acute, painless loss of vision in his right eye. Acuity was counting fingers, right eye and 20/30, left eye with a right relative afferent pupillary defect. The right fundus was normal and there was mild left optic disc pallor. The right visual field demonstrated central and inferior loss, whereas the left field was normal. Repeat testing including erythrocyte sedimentation rate, C-reactive protein, and platelet count was normal. A temporal artery biopsy was performed. There were no findings suggestive of GCA but,

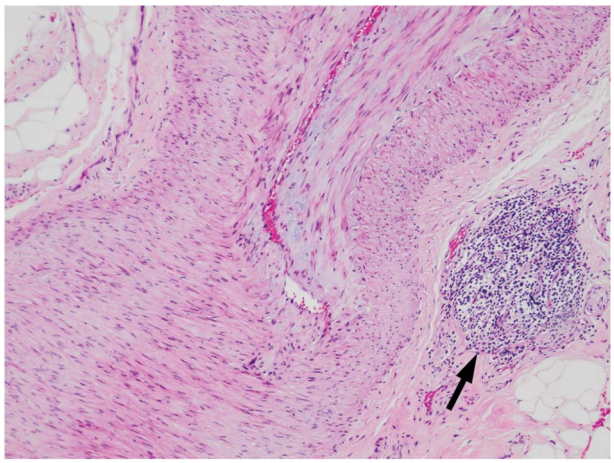

FIG. 1. Temporal artery biopsy shows no signs of giant cell arteritis. An aggregate of lymphocytes (*arrow*) is seen in the adventitia (hematoxylin and eosin, ×100).

within the surrounding tissue, lymphocyte aggregates were seen (Fig. 1).

Additional testing revealed a leukocytosis of 27×10^9 cells/L. Flow cytometry showed a monoclonal B-cell population of 44% which was kappa light chain positive and CD5, CD19, CD20, and CD23 positive. The immunoglobulin heavy chain gene mutation status was not mutated; 11q and 17p deletions were not found. Diagnosis of chronic lymphocytic leukemia (Rai stage II) was made.

Treatment was not begun because the patient did not meet the criteria for active disease (2). However, because of recurrent episodes of decreased vision in each eye, he was prescribed prednisone 60 mg a day for 7 days, which lead to subjective improvement in vision. Because the possible association of eye symptoms with chronic lymphocytic leukemia was present, and no other underlying causes were found, the patient was placed on a regimen of rituximab, chlorambucil, and prednisone each 28 days. After 6 monthly cycles, the patient was in full remission, and after 8 cycles, his hematologist switched him to rituximab maintenance monotherapy. The patient's leukocyte count normalized to 4.4×10^9 cells/L, and his visual acuity stabilized at 20/40, right eye and 20/20, left eye. Visual function has remained stable during follow-up.

Although the precise cause of our patient's bilateral optic neuropathy is uncertain, possibilities include vaso-occlusion or paraneoplastic syndrome due to chronic lymphocytic leukemia. Neither our clinical examination nor neuroimaging suggested leukemic infiltration of the optic nerves. During our patient's clinical course, GCA was in the differential diagnosis and a temporal artery biopsy was performed. It was the finding of perivascular lymphocytes on the biopsy specimen that prompted us to pursue a hematologic evaluation, and arrive at the correct diagnosis of chronic lymphocytic leukemia.

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The 14th Hoyt Lecture Ischemic Optic Neuropathy: The Evolving Profile, 1966–2015: Comment

I read with interest the Hoyt Lecture by Arnold (1) dealing with nonarteritic anterior ischemic optic neuropathy (NAION). The observation of a small cup-to-disc ratio in these patients was made by William F. Hoyt, but Arnold erroneously attributed this to Sohan Singh Hayreh from an article discussing the development of disc cupping after an attack of arteritic AION instead (reference 17 in Arnold's article). Yet, Bill Hoyt was the first to recognize this disc morphology in patients with NAION—what was