The use of SSRIs for children and adolescents has increased in Canada by 44% between 2005 and 2009,[14] raising the possibility of greater incidence of cataracts in adolescent patients. The increased rate of antidepressant use in adolescents raises the concern that adverse effects should be more closely studied, particularly in children. Fluvoxamine has been shown to increase the risk for cataract formation in a large cohort of Canadian adults[3] but was never investigated in children. This epidemiologic study included 18 784 patients with cataract and 187 840 age-matched control subjects, and has shown an increased risk for cataract formation in patients taking fluvoxamine, venlafaxine, and paroxetine. We compared the incidence of cataract surgeries within a cohort of patients using fluvoxamine with control subjects and demonstrated a relative risk for cataract formation in this group of 1.51, suggesting a strong association between fluvoxamine use and cataract formation. The possibility that more frequent eye examinations are warranted for children and adolescents using SSRIs and specifically fluvoxamine should be investigated.

The effect of decreased visual acuity on quality of life is profound in children, especially when they are suffering from depression, anxiety, and other psychiatric disorders. Patients suffering from depression and anxiety might not have proper coping mechanisms and motivation to pursue intervention and report the changes, allowing the changes to go unnoticed and further exacerbating the child's mental state. Such a case of rapidly progressing bilateral cataract development and associated loss in vision in an adolescent emphasizes the importance of frequent eye examinations of any individual taking fluvoxamine. Further studies should be conducted to determine the specific risk for cataract development in adolescents and children taking fluvoxamine.

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Temporal changes in a giant macular hole formed secondary to toxoplasmic retinochoroiditis

It is well-known that ocular toxoplasmosis can cause posterior uveitis, including focal necrotizing retinochoroiditis, which is often accompanied by adjacent retinochoroidal scars, vitritis, macular edema, and retinal detachment.[1,2] Recently, some reports of a macular hole (MH) secondary to toxoplasmic retinochoroiditis as a rare complication of ocular toxoplasmosis have been presented, in which the pathogenesis of the MH associated with toxoplasmic retinochoroiditis was suggested to involve vitreomacular traction,[3,4] retinochoroidal ischemia,[5] or both. Unfortunately, the MH had already developed before initial examination in these reports; thus, symptoms preceding development were not well described. In this article, we present a case of newly acquired ocular toxoplasmosis with the development of a giant MH during treatment for posterior uveitis. Using optical coherence tomography (OCT), we examined the symptoms before and after MH formation.

A 59-year-old male visited our department with a 2-week history of visual disturbance and central scotoma in the left eye. He had a medical history of central serous chorioretinopathy in the right eye at 34 years of age. He also suffered from lymphocytic leukemia and underwent bone marrow transplantation 6 months before our initial examination, during which oral prednisolone (15 mg/day) and tacrolimus (1.75 mg/week) were prescribed.

At the initial visit, best corrected visual acuity (BCVA) was 20/200 OU. Intraocular pressure was normal, and slit-lamp examination of the anterior segment was unremarkable. Fundus examination revealed a newly acquired yellowish exudative lesion in the macula of the left eye (Fig. 1A) and old macular atrophy in the right eye. Spectral-domain OCT examination (Spectralis; Heidelberg Engineering, Heidelberg, Germany) revealed increased intraretinal reflectivity and thickening in this area, in addition to a partially detached posterior hyaloid membrane adjacent to the macula (Fig. 2A). Fluorescence angiography revealed early hypofluorescence in the central part of the lesion, with progressive hyperfluorescence at the margin. A serologic test for *Toxoplasma gondii* infection was positive (IgM: 1.9 IU/mL, IgG: 47 IU/mL), whereas antibodies against *Toxocara canis, Treponema pallidum* latex agglutination, hepatitis B surface antigen, hepatitis C antibody, human immunodeficiency virus, β-D-glucan, cytomegalovirus IgM, and an interferon-γ release assay (T-SPOT.TB) were negative or within normal ranges. Based on serologic and ophthalmologic examination findings, a diagnosis of toxoplasmic retinochoroiditis was made and oral acetylspiramycin (1200 mg/day) was initiated. After 8 weeks, the exudative lesion became cicatrized (Fig. 1B), and oral acetylspiramycin was stopped. OCT revealed a decrease in the retinal thickness of the macula and deteriorating vitreomacular traction (Fig. 2B). After 4 weeks, he exhibited sudden visual impairment in the affected eye. BCVA was decreased to 20/300, and fundus examination revealed a giant MH (2852 μm horizontally, 2571 μm vertically in spectral-domain OCT) and vitreous hemorrhage (Figs. 1C and 2C). To remove vitreous opacity and prevent progression to retinal detachment, we performed 25-gauge pars plana vitrectomy with internal limiting membrane peeling and sulphur hexafluoride gas tamponade. Although the MH was not fully closed, surgery resulted in a decrease in size (1814 μm horizontally, 1681 μm vertically; Figs. 1D and 2D). Three months

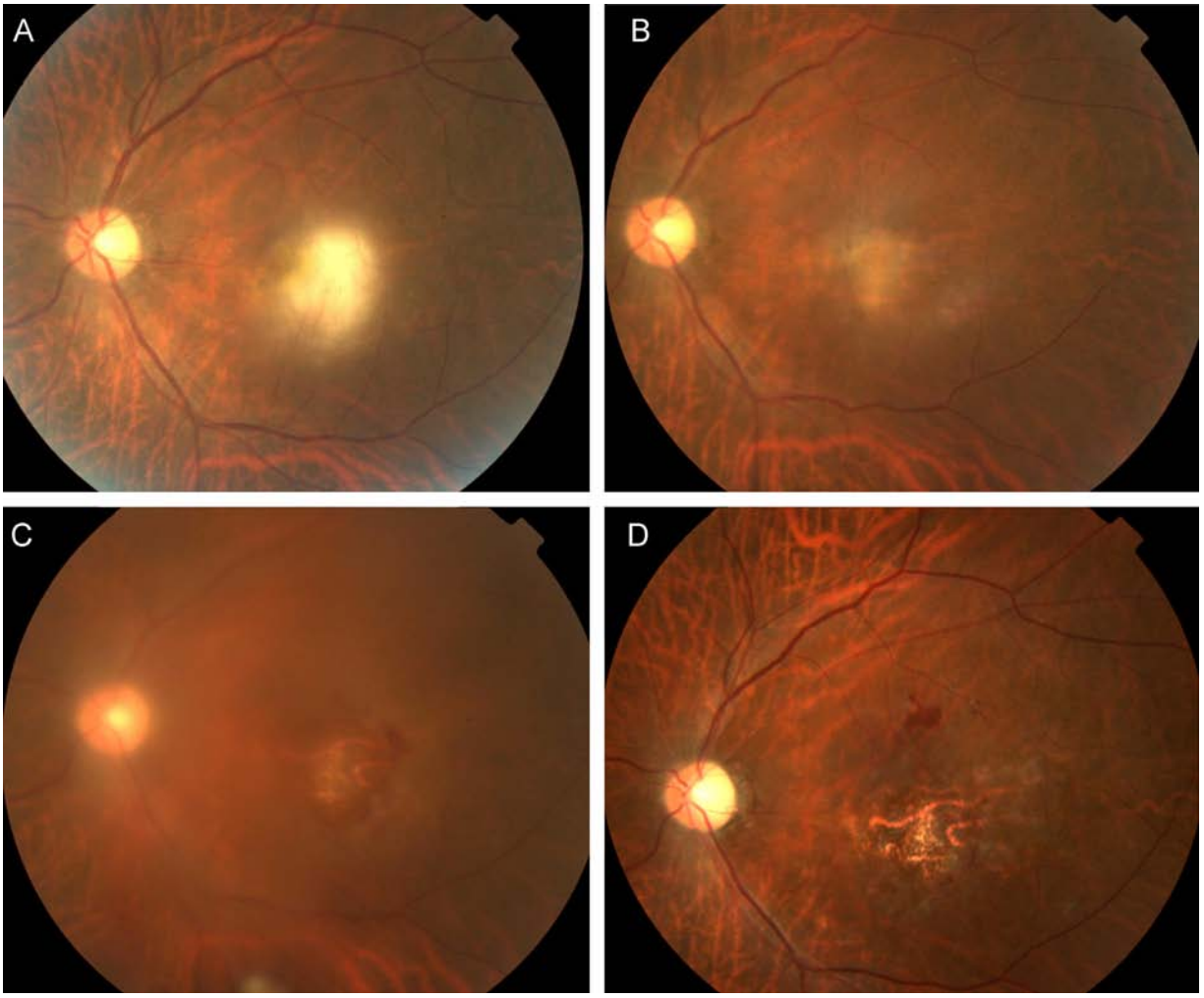

Fig. 1—A, Fundus image of the left eye obtained at initial examination showing a cream-coloured active retinal toxoplasmosis lesion in the fovea. B, After the initiation of oral treatment, the exudative lesion became cicatrized. C, At 12 weeks after the initial visit, a giant macular hole and vitreous hemorrhage developed. D, Following the performance of vitrectomy, vitreous hemorrhage resolved.