izing questions (e.g., neuro-ophthamology, pediatric ophthalmology), adding new questions, and adding detailed explanations to the answers.

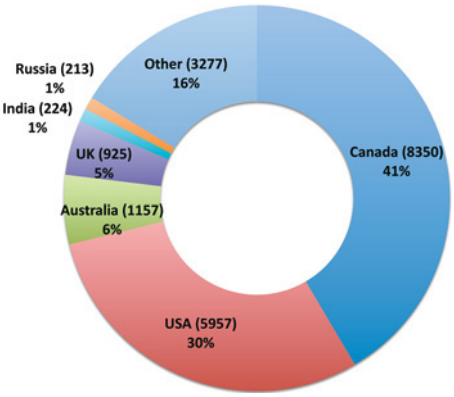

Fig. 2—Visitors to QuizMD by country of origin (total of 20 103 visitors over 12 months). Traffic to OphthoStudent.com is approximately 10% of all traffic to QuizMD.

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Silicone oil–induced bilateral granulomatous uveitis

A 65-year-old male with hypertension, aortic bypass surgery, femoral artery aneurysm, transient ischemic attacks, and Waldenstrom's macroglobulinemia presented with vitreous hemorrhage and nontraumatic retinal tear OS. The retinal tear was treated with laser photocoagulation. One month later, vitreous hemorrhage recurred and did not clear for 2 months. A pars plana vitrectomy with gas tamponade was carried out. One month postoperatively, total retinal detachment with proliferative vitreoretinopathy grade D-3 developed. Repeat vitrectomy, extensive membrane dissection, laser endophotocoagulation, and perfluorocarbon liquid injection followed by silicone oil/perfluorocarbon exchange were carried out. Postoperatively, the retina was initially reattached but, 2 months later, a cyclitic membrane formed, followed by phthisis.

One month later, the patient complained of floaters in the previously normal fellow eye. Visual acuity was 20/30 OD. There was granulomatous anterior uveitis. Ophthalmoscopy revealed vitreous cells, optic disc edema, and small multifocal deep retinal or choroidal yellowish nodules predominantly anterior to the equator. For diagnostic reasons, enucleation of the phthisical OS was carried out.

Histopathologic study of the enucleated OS showed chronic inflammatory cells and large vacuolated spaces in the cyclitic membrane and retina and in macrophages and foreign body giant cells (Fig. 1). No histopathologic features of sympathetic ophthalmia were identified. Electron microscopy of the vacuoles within the cytoplasm

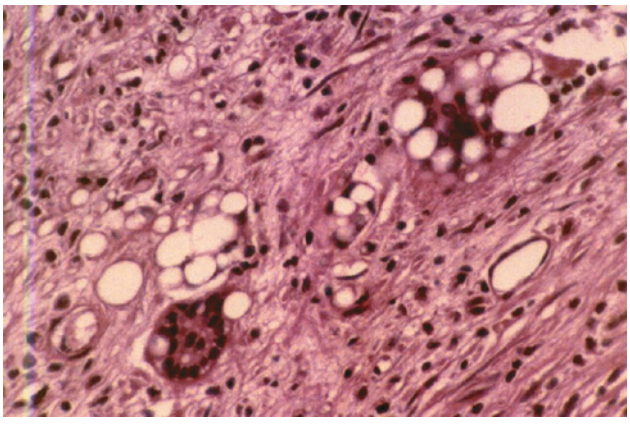

Fig. 1—Histopathologic staining of enucleated eye showing evidence of individual macrophages, as well as foreign body giant cells containing vacuoles (hematoxylin and eosin; original magnification × 100).

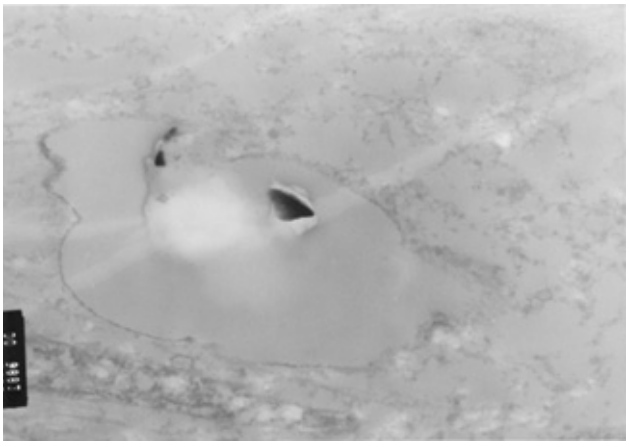

Fig. 2—Electron photomicrograph of same specimen showing a vacuole in a foreign body giant cell.

of these macrophages and giant cells showed greyish, amorphous debris that was positive for silicone on energy dispersive x-ray analysis (Figs. 2 and 3), indicating that the material in the vacuoles was silicone, not perfluorocarbon liquid.

Others have reported histopathologic evidence of granulomatous inflammation due to the presence of silicone oil in 1 eye. Histopathological examination of an eye enucleated for chronic retinal detachment treated 20 years earlier with silicone oil showed vacuoles that were termed "silicone granulomas" in preretinal membranes, the subretinal space, and the choroid.[1] Other clinical/histopathologic studies have confirmed the association of granulomatous inflammation following retinal detachment surgery with intraocular silicone oil injection.[2]

The potential antigenicity of silicone has been implicated in a wide variety of circumstances in which silicone implants, including silicone bands and sponges, have been inserted. Silicone oil has been shown to be a mediator in a number of destructive immunologic reactions in human beings and in animal models. In animal studies, silicone is a well known and powerful adjuvant in the production of antibodies to a wide variety of antigens, including rat thyroglobulin, bovine collagen II, and bovine serum albumin in mice.[3] In mice, intraperitoneal injection of silicone oil causes persistent elevation of serum immunoglobulin M and activates macrophages and interleukin-1 beta.[3]

Our case is unique in that granulomatous uveitis developed in the previously normal fellow eye in which silicone oil had not been inserted, which suggests that silicone oil may incite a granulomatous inflammatory response that may affect both eyes, including the eye that does not contain any silicone oil. The demonstration by energy dispersive x-ray analysis of silicone oil in the phagocytosed material in macrophages and foreign body giant cells in the diseased eye is strong evidence that silicone may be the inciting agent.

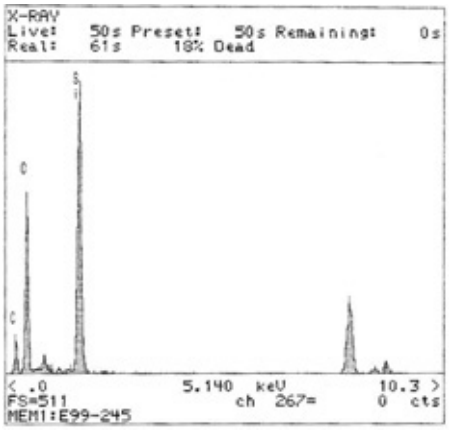

Fig. 3—Energy dispersive x-ray analysis of contents of vacuoles showing peak absorption, indicating silicone oil presence. (Si, silicone oil; O, oxygen; C, carbon.)

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Parry-Romberg syndrome associated with anterior uveitis and retinal vasculitis

Parry-Romberg syndrome is a progressive, hemifacial atrophy involving the skin, soft tissues, cartilage, and underlying bone.[1] Ocular involvement is well recognized and occurs in up to 40% of cases; enophthalmos has been reported as the commonest ocular manifestation.[2]

We describe a case of Parry-Romberg syndrome associated with anterior uveitis and retinal vasculitis.

A 33-year-old female presented with a history of progressive atrophy of the left side of her face starting before 18 years of age. Between 1999 and 2005, the patient underwent several aesthetic surgery procedures. The face examination revealed extensive atrophy of subcutaneous tissue, fat, and muscles on the left side. At the time of presentation, the patient complained of floaters in her left eye. Her visual acuity was 20/20 OU. Hertel measurements were 18 mm for both eyes. Slit-lamp and fundus examinations were within normal limits in the right eye. In the left eye, slit-lamp examination showed multiple medium-sized, fresh and small pigmented keratic precipitates, 2+ flare and cells in the anterior chamber, and cells in the anterior vitreous. No posterior synechiae or heterochromia were seen (Fig. 1). The intraocular pressures were within normal limits in both eyes. Fundoscopy showed areas of retinal pigment epithelial atrophy in the nasal and temporal quadrants, and focal perivenular sheathing. Fluorescein angiography showed focal staining of vessel walls and capillary leakage, especially in the inferior peri-