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.

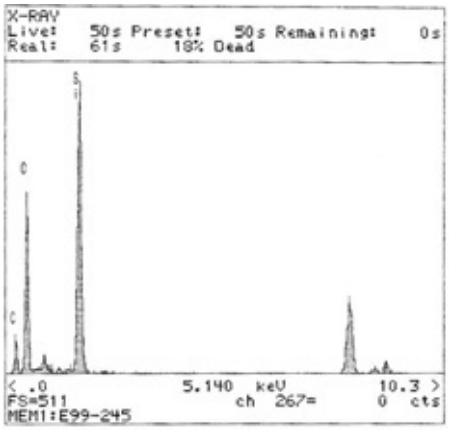

Fig. 3—Energy dispersive x-ray analysis of contents of vacuoles showing peak absorption, indicating silicone oil presence. (Si, silicone oil; O, oxygen; C, carbon.)

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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-

pheral retina (Fig. 2). Complete blood count, biochemical tests, erythrocyte sedimentation rate, C reactive protein, VDRL (Venereal Disease Research Laboratory) testing, HLA-B27, antinuclear antibody, rheumatoid factor, angiotensin-converting enzyme, and thorax computerized tomography were found to be normal or negative. No additional pathology was determined as a result of dermatology, rheumatology, or pulmonary medicine consultations.

Topically 1% prednisolone acetate and 1% tropicamide hydrochloride treatment was started. Fifteen days after the beginning of treatment, a decrease in the number of fresh keratic precipitates, cells, and flare was observed. One month after fresh keratic precipitates, the cells and flare had disappeared. Topical treatment was tapered gradually and discontinued. The patient is being followed.

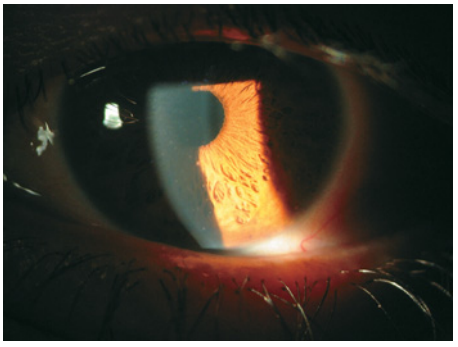

Fig. 1—Slit-lamp examination of the left eye showed multiple medium-sized, fresh and small pigmented keratic precipitates.

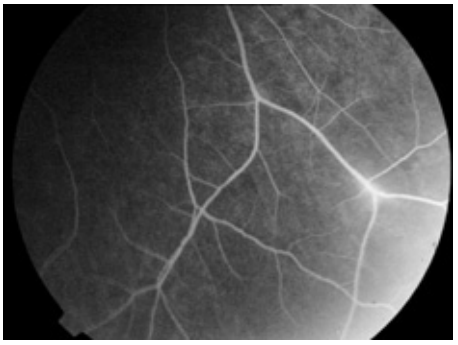

Fig. 2—Fluorescein angiography of the left eye revealed focal staining of vessel walls and capillary leakage, especially in the inferior peripheral retina.

Ocular complications such as enophthalmos, vitiligo, poliosis, ptosis, ectropion, neuroparalytic keratitis, strabismus, uveitis, heterochromia of the iris, retinal telangiectases, neuroretinitis, and macular edema have been described.[2–5] Our patient with Parry-Romberg syndrome showed an unusual association of anterior uveitis and retinal vasculitis.

Patients with Parry-Romberg syndrome who have received systemic immunosuppressive therapy for retinal vasculitis and neuroretinitis have been described in previous reports.[4,5] Karim et al.[4] used corticotherapy (1mg/kg daily) for bilateral vitreitis and unilateral neuroretinitis. Bellusci et al.[5] reported a patient with bilateral Parry-Romberg syndrome associated with retinal vasculitis and macular edema who underwent immunosuppressive therapy with cyclosporine-A (4mg/kg daily). We treated our case with only topical corticosteroids for anterior uveitis since the visual acuity was 20/20 and there was no macular edema. The anterior uveitis resolved completely within 1 month of topical corticosteroid treatment. The patient is still followed without systemic treatment.

It should be kept in mind that ophthalmic complications that can result in visual loss may occur in patients having Parry-Romberg syndrome with typical facial appearance and progress. Regular ophthalmic consultations should be advised.

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Tsukamurella*: an emerging opportunistic ocular pathogen*

Tsukamurella species are Gram-positive, weakly acid-fast bacilli belonging to the aerobic Actinomycetes family.[1–4] *Tsukamurella* taxonomically comprises at least 10 described species and behaves as a nonmotile and non-spore-forming pathogen rarely cultured from humans.[5–7] Limited information exists on the detection, identification, and treatment of this pathogen in human infections. Postulated risk factors for infection with *Tsukamurella* include the presence of a foreign body