the IOL that may have diffused into the silicone material.

Clinically significant postoperative opacification of hydrophilic acrylic IOL (model SC60B-OUV; Medical Developmental Research, Inc, Clearwater, Fla) has been reported. This has been reportedly due to the presence of granular deposits within the optic of the lens, which were naturally occurring calcium produced by the patient’s body.8

In our department we performed 8 cases of routine uneventful phacoemulsification with implantation of the Aquasense IOL (Ophthalmic Innovations, Inc) by 3 different surgeons between October and November 2000. This was a single-piece foldable hydrophilic acrylic IOL (water content, 25%).

The patients in 3 of these cases had significant visual deterioration due to total opacification of the IOL more than a year after surgery. The whole lens had diffuse opacification within its substance and was uniformly distributed. All of them had a similar pattern of opacification with minimal variation.

No apparent cause for the opacification of the IOL could be found. The company’s managing director in the United Kingdom was contacted (Ophthalmic Innovations, Inc, oral communication, November 2002). He explained that the opacification was due to an interaction between the silicone sleeve, used to hold the IOL in the vial, and the acrylic material of the IOL itself. This apparently imparted a negative charge to the IOL resulting in its opacification.

To our knowledge no other case has been reported in the literature where the whole IOL has opacified. The company refused to give out “proprietary” information on the exact number of IOLs that had been implanted, but an educated guess would be over 500. Apparently all these IOLs have been withdrawn and a modified version without the silicone sleeve has been introduced. Apparently no opacification has been reported so far with this new version. We have had no further experience with this IOL.

The authors have no relevant financial interest in this article.

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Central Serous Chorioretinopathy After Local Application of Glucocorticoids for Skin Disorders

Central serous chorioretinopathy (CSCR) is a common disease characterized by the accumulation of subretinal fluid at the posterior pole of the fundus; it typically affects young and middle-aged adults, with men affected more commonly than women. The exact pathogenic mechanism of CSCR remains unclear. There is accumulating evidence that both endogenous and exogenous glucocorticoids may be implicated in the pathogenesis of the disease.1,2 Regarding the role of exogenous glucocorticoids, CSCR has been reported as a complication of intravenous, intramuscular, oral, epidural, inhaled, and intranasal glucocorticoid administration. We describe 2 patients who developed CSCR after prolonged treatment with glucocorticoids applied locally to the skin for dermatological indications.

Report of Cases. Case 1. A 32-year-old man complained of decreased vision and metamorphopsia in the right eye. Best-corrected visual acuity was 20/25 OD and 20/20 OS. Fundus examination results were normal in the left eye but in the right eye revealed a well-circumscribed, shallow, serous detachment of the sensory retina. The clinical appearance was consistent with CSCR, and the diagnosis was confirmed by means of fluorescein angiography, which showed a leakage point at the superior macula, spreading slowly in an inkblot configuration into the subretinal space (Figure 1).

The medical history of the patient was remarkable for seborrheic dermatitis involving the central face, eyebrows, eyelids, and scalp. The disease had been diagnosed 2 years earlier, and 1% hydrocortisone acetate cream was prescribed for topical application. After the initial prescription, the patient used the cream without further medical consultation when symptoms were exacerbated. The 1% hydrocortisone acetate cream was used for 4 weeks, 3 to 4 times daily, before the development of CSCR.

Case 2. A 37-year-old man was referred to us for blurred vision in the left eye of 1 week’s duration. He had a history of CSCR in the contralateral eye, 5 years previously, for which he had been treated with laser photocoagulation at another institution. Best-corrected visual acuity was 20/20 OU. Funduscopy of the right eye revealed scars from previous laser photocoagulation at the superior macula. In the left eye, there was a well-delineated area of serous detachment temporal to the fovea. Small yellowish precipitates were visible at the posterior aspect of the detached retina. Fluorescein angiography revealed a leakage point at the upper pole of the detachment (Figure 2).

The medical history of the patient was remarkable for pityriasis versicolor, for which he was treated with local application of 0.1% diflucortolone valerate cream in combination with 1% isocochonolate nitrate. The patient used the cream occasionally and was being treated for 3 weeks before symptoms began. Notably, the patient was also being treated with 0.1% diflucorto-
valerate cream during the first episode of CSCR.

Comment. We describe 2 patients who developed CSCR during treatment with glucocorticoids applied locally to the skin for dermatological disorders. Notably, 1 had a history of CSCR in the contralateral eye, which had also developed while he was being treated with glucocorticoid cream applied locally.

The exact pathogenic mechanism of CSCR is unclear and is the subject of considerable controversy. Accumulating evidence suggests that glucocorticoids may contribute to the pathogenesis of CSCR. The development of CSCR has been described in association with conditions characterized by endogenous hypercortisolism such as pregnancy, stress, and endogenous Cushing syndrome. It has also been described as a complication of exogenous glucocorticoids administered via various routes—oral, intravenous, intramuscular, inhaled, intranasal, and epidural.

To our knowledge, this is the first report of patients developing CSCR after local application of glucocorticoids to the skin for the treatment of dermatological disorders. Percutaneous absorption of glucocorticoids applied locally to the skin is well described in the dermatological literature. Application of glucocorticoids may result in systemic absorption sufficient to cause hypercortisolism, adrenal suppression, and reduced glucose tolerance. Therefore, although a coincidental association cannot be excluded, the development of CSCR in our patients may be related to the local skin application of glucocorticoids. Even if this report does not prove that topical steroids cause CSCR, it provides evidence that raises this suspicion and suggests that further study of this potential association is warranted. The exact pathogenic mechanism by which glucocorticoids may be implicated in the development of CSCR is unknown and remains speculative. Proposed theories incriminate the effect of glucocorticoids either to the choroidal vasculature or to the function of the retinal pigment epithelium.

In conclusion, local skin application of glucocorticoids may be complicated by the development of CSCR. In such cases, discontinuation of glucocorticoid treatment should be considered whenever possible.

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Figure 1. In case 1, the fluorescein angiogram of the right eye shows pinpoint leakage at the superior macula (A) that spreads to the subretinal space (B) at later phases.

Figure 2. In case 2, the red-free photograph of the left eye shows serous retinal detachment (A), and the fluorescein angiogram of the left eye demonstrates the leakage point (B).
The authors have no relevant financial interest in this article.

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Ocular Involvement in Systemic Vasculitis Associated With Perinuclear Antineutrophil Cytoplasmic Antibodies

Antineutrophil cytoplasmic antibody (ANCA) formation to myeloperoxidase or perinuclear (p-ANCA) is infrequent in ocular disease and is reported in approximately 10% of patients with Wegener granulomatosis 1,2 as opposed to cytoplasmic (c-ANCA) positivity (antibodies to proteinase 3), which is well recognized. Higher levels of p-ANCA are also identified in microscopic polyangiitis, Churg-Strauss syndrome, inflammatory bowel disease, and crescentic glomerulonephritis. The sensitivity and specificity of both tests are high for a vasculitic process frequently associated with a systemic disease that requires prompt immunosuppressive therapy. 1,2

We report 4 cases of p-ANCA positivity showing a spectrum of severe ocular involvement. Two patients had been systemically well previously, but it was the ocular disease that prompted ANCA measurement.

Report of Cases. Case 1. A 76-year-old man had a 12-month history of red smarting eyes. Examination revealed bilateral peripheral ulcerative keratitis with normal visual acuity and no proptosis. Investigations revealed chronic renal failure and elevated lesions at the level of the retinal pigment epithelium showing a swollen optic disc and creamy white, elevated lesions at the level of the retinal pigment epithelium at the posterior pole in case 4. Magnetic resonance imaging of the orbits showed enlarged extraocular muscles, and renal biopsy results showed florid necrotizing glomerulonephritis. After treatment with high-dose steroids and azathioprine sodium, the patient’s signs resolved dramatically, leaving some residual diplopia controlled by prisms.

Case 3. A 67-year-old man with ulcerative colitis and a previous right serous retinal detachment had deteriorating vision in the left eye. Visual acuities were hand movements OD and 20/200 OS. Examination showed marked retinal pigment epithelial mottling and macular edema in both eyes and serous retinal detachment in the left eye. Results of fluorescein angiography and B-scan ultrasonography were used to confirm posterior scleritis. After therapy with immunosuppressants, the patient’s visual acuity improved to 20/20 OS. Two years later, a relapse occurred, and p-ANCA findings were positive. His titre subsequently normalized with treatment, although his vision continued to deteriorate.

Case 4. A 58-year-old man had a 15-month history of progressive left-sided headache, poor appetite, weight loss, and deafness. Cranial nerve examination revealed left trigeminal nerve involvement and sensorineural deafness. Visual acuity was normal. Funduscopy demonstrated a swollen right optic disc and bilateral white, elevated retinal pigment epithelial lesions (Figure 2). These lesions showed early hypo-