The skin lesion showed a localized area of fibrosis and vascular proliferation consistent with an angiobroma.

Comment. Tuberous sclerosis, otherwise known as Bourneville disease, is an autosomal dominant syndrome with an incidence of 1 case per 6000 to 29900 people per year. Despite its capability of following classic Mendelian patterns, approximately two thirds of cases are sporadic and seem to arise from new somatic mutations. Genetic research has implicated mutations at 2 gene loci, 9q34 (TSC1) and 16p13 (TSC2), in the pathogenesis of tuberous sclerosis. These genes are thought to be tumor suppressors. Historically, the clinical criteria for diagnosing tuberous sclerosis include facial adenoma sebaceum, mental deficiency, seizures, and retinal astrocytomas (hamartomas), although not all of the criteria need to be met to make the diagnosis. In addition to skin and retina, hamartomas have been identified throughout the body in locations such as the iris, brain, bones, kidneys, lungs, heart, and tongue. Bony abnormalities in patients with tuberous sclerosis have been previously described in the skull, long bones, pelvis, and metacarpal and metatarsal bones, with the hands and feet the most commonly involved. These lesions can be focal or diffuse; tend to be round, oval, or flame shaped; and range in size from a few millimeters to several centimeters. These bony lesions typically manifest as small cortical cysts or areas of dense periosteal sclerosis, the result of fibrous replacement of the normal trabecular bone pattern, and can produce an undulation in the bony contour. Bony abnormalities can be found in the cranium in up to 45% of patients with tuberous sclerosis. Radiographs of the skull may reveal sclerotic areas of dense bone formation, often due to hyperostosis of the diploic trabeculae of 1 or both tables of the cranial vault. The calvaria is thinned, marrow spaces are replaced by fat, and the periosteum is thickened.

After an extensive review of the peer-reviewed English-language literature using MEDLINE, we found only 1 report describing an osseous abnormality of the facial skeleton and orbit in a patient with tuberous sclerosis and none with histopathologic confirmation of a bony hamartoma in this location. Breningstall et al reported a case of a sclerotic lesion of the sphenoid bone in a 9-year-old girl with tuberous sclerosis who had left-sided proptosis. Skull radiographs and computed tomography revealed marked sclerosis and thickening of the left wing of the sphenoid bone, which was attributed to fibrous dysplasia. However, no histopathologic analysis was provided.

To our knowledge, this case of a large bony hamartoma arising from the inferior orbital rim in a patient with tuberous sclerosis provides the first clinicopathologic confirmation of a bony hamartomatous lesion involving the facial skeleton in the setting of tuberous sclerosis. Physicians treating patients with tuberous sclerosis should be aware of this presentation.

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Case 2. A 74-year-old woman had uneventful phacoemulsification of the right eye with posterior chamber IOL implantation in October 2000. She had noninsulin-dependent diabetes for 14 years and her preoperative BCVA was 6/24 OD. Postoperative BCVA on her follow-up visit in December 2000 was 6/24 OD. She had a quiet eye and early diabetic maculopathy. The patient was discharged to the opticians for further care.

The patient was seen again after 15 months in January 2002 complaining of blurred vision of 3 months’ duration. Posterior capsular opacification was diagnosed and she underwent Yag laser capsulotomy 3 months later. On her follow-up visit 3 months after this procedure, her visual acuity was still hand motions and additional Yag laser treatment was proposed. The possibility that this patient had had a vitreous hemorrhage was also suggested. A B-scan showed vitreous opacities and the patient was scheduled to undergo right vitrectomy for nonresolving vitreous hemorrhage. The patient was seen by the ophthalmic consultant preoperatively and opacification of the IOL was diagnosed. The patient had an IOL exchange with posterior chamber IOL in October 2002 (Figure 2).

At her last visit in January 2003, BCVA was hand motions. She was noted to have advanced untreatable diabetic maculopathy. However, she was happier with the quality of her vision and is being followed up in the diabetes clinic.

Case 3. A 75-year-old woman underwent uncomplicated phacoemulsification of the left eye with posterior chamber IOL implantation in November 2000. Her preoperative BCVA was 6/12 OS. On her last postoperative clinic visit in December 2000 she could read 6/9 OS unaided and was very pleased with that result. She was discharged from our care.

She was seen again, nearly 2 years later, in November 2002 complaining of blurred vision. Her BCVA was 6/60 OS. This time her ocular condition was immediately diagnosed as opacification of the IOL and she is awaiting an IOL exchange. The third patient could not be reached.

Comment. Silicone, hydrogel, and acrylic (hydrophilic and hydrophobic) are the principal materials used for manufacturing IOLs that can be folded to be inserted into the eye through a small self-sealing incision. There have been reports of varying degrees of opacification in all of these materials mentioned above.

The hydrophobic acrylic lenses (AcrySof; Alcon, Fort Worth, Tex) have been known to show glistening in the early postoperative period, resulting rarely in blurred vision. This has been attributed to the formation of small fluid-filled vacuoles within the optic of the IOL. In vitro studies have suggested that a change in the temperature of the surrounding environment may be the cause for this vacuolation.1,2

Some patients with hydrogel lenses (Hydroview model H60M; Bausch & Lomb Surgical, Rochester, NY) had significant reduction in visual acuity at 1 year postoperatively. This was attributed to late postoperative deposition of calcium on the surface of the lens. No definitive cause has been found for this.3

Silicone IOLs were the first foldable lenses and have been known to undergo brownish discoloration and central haze within the first 6 weeks postoperatively.4,5 This usually is not visually debilitating and has been attributed to light scatter from a layer of water vapor within
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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 systemic glucocorticoid administration.3 4 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% isoconazole 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-