Diagnostic and Therapeutic Challenges

Edited by H. Richard McDonald

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This case is submitted by Drs. Francisco J. Ascaso, Maria Rojo, and Enrique Minguez from the Department of Ophthalmology, ''Lozano Blesa'' University Clinic Hospital, Zaragoza, Spain, for the Diagnostic and Therapeutic Challenges Section of Retina; and commented by Dr. Steven M. Cohen, Clearwater, Florida, and Dr. Sam S. Yang, Walnut Creek, California.

Case Report

On October 5, 2008, an apparently healthy 51-year-old postmenopausal woman had a sudden onset of bilateral metamorphopsia and dyschromatopsia. She was under treatment with raloxifene hydrochloride (Evista, Eli Lilly and Co., Indianapolis, IN) and calcium supplements for osteoporosis prevention for 2 years. Her ocular, medical, and family histories were unremarkable. Best-corrected visual acuity was 20/30 in both eyes, and the anterior segment was normal. Intraocular pressure was 15 mmHg in both eyes. Fundus examination revealed a well-delineated oval area of serous retinal detachment involving both maculae, resembling a bilateral central serous chorioretinopathy (CSC) (Figure 1A, B). Fluorescein angiogram (FA) showed no active leakage point arising from the macular neurosensory retinal detachments. Her electroretinographic and electrooculographic findings were normal. Her chemistry profile was normal except for elevated serum values of liver enzymes (AST 47 U/L, GGT 134 U/L) and decreased serum calcium levels (7.8 mg/dL). Hemogram, coagulation studies, proteinogram, and inmunoglobulins (Ig A, Ig G, Ig M) all were normal. Mantoux test and serological tests for syphilis and toxoplasmosis were negative.

The patient was treated with 325 mg a day of oral acetazolamide. Ten days later, she reported a bilateral worsening of the central vision. Best-corrected visual acuity was 20/50 in both eyes. Fundus examination showed an enlargement of the neurosensory retinal detachment in both macular areas, associated with multiple variable-sized yellowish serous elevations of the sensory retina along the temporal vascular arcades (Figure 1C, D). Optical coherence tomography (OCT) demonstrated multiple blister-shaped hyporeflective lesions compatible with serous neurosensory retinal detachments, confirming the hydrodynamic separation of the sensory retina from the retinal pigment epithelium (RPE). The tallest and widest lesions were subfoveal. The foveal depression was lost, but the thickness of the overlying retina appeared normal. No fusiform or polypoidal thickening of the hyperreflective layer was shown by OCT to suggest the presence of occult neovascularization (Figure 2A, B). Late-phase FA identified minimal diffuse mottled hyperfluorescence in both eyes sparing the fovea and hypofluorescence of the denser, thicker, yellow lesions. The absence of chorioretinal vascular compromise seemed to contribute to the minimal expansion of the dye into serous cavities late into the study (Figure 3A, B). Indocyanine green (ICG) angiography showed early hyperfluorescence in the posterior pole of the choroid, which became prominent in the late phase. Indocyanine green angiography revealed neither occult polypoidal nor other forms of choroidal neovascularization (Figure 4A, B). After corticosteroid therapy (oral prednisone 1 mg/kg/day tapered to zero at about 3 months), the patient was asymptomatic, the serous retinal detachments resolved (Figure 1E, F), and the visual acuity improved to 20/20 in both eyes, remaining stable until now.

Neurosensory retinal detachments occur when there is an abnormal collection of extravascular fluid within the subretinal space. Serous retinal detachments with no RPE detachments have been reported in patients with paraproteinemia, toxemia of pregnancy, severe hypertension, disseminated intravascular coagulopathy, thrombotic thrombocytopenic purpura, and Goodpasture syndrome.[1]

The patient, who was apparently healthy, presented initially with bilateral macular sensory retinal detachment simulating that seen in idiopathic CSC. A few days later, an unusual presentation of multiple, slightly elevated, yellow lesions associated with a thin pink rim of what was interpreted as subretinal fluid was present in both eyes. Corticosteroids, which act in many ways to decrease vascular permeability, seem to worsen the RPE and neurosensory retinal detachments in patients with idiopathic CSC.[2] However, the present report points out the complete resolution of multiple serous neurosensory retinal detachments into an atypical CSC pattern after corticosteroid therapy.

We asked several experts for their opinion.

Dr. Steven M. Cohen (Clearwater, Florida):

This 51-year-old healthy woman developed acute vision loss in each eye from a serous retinal detachment in the center of the macula and subsequently developed multifocal, yellow, well-delineated, polymorphous, serous retinal detachments in and at

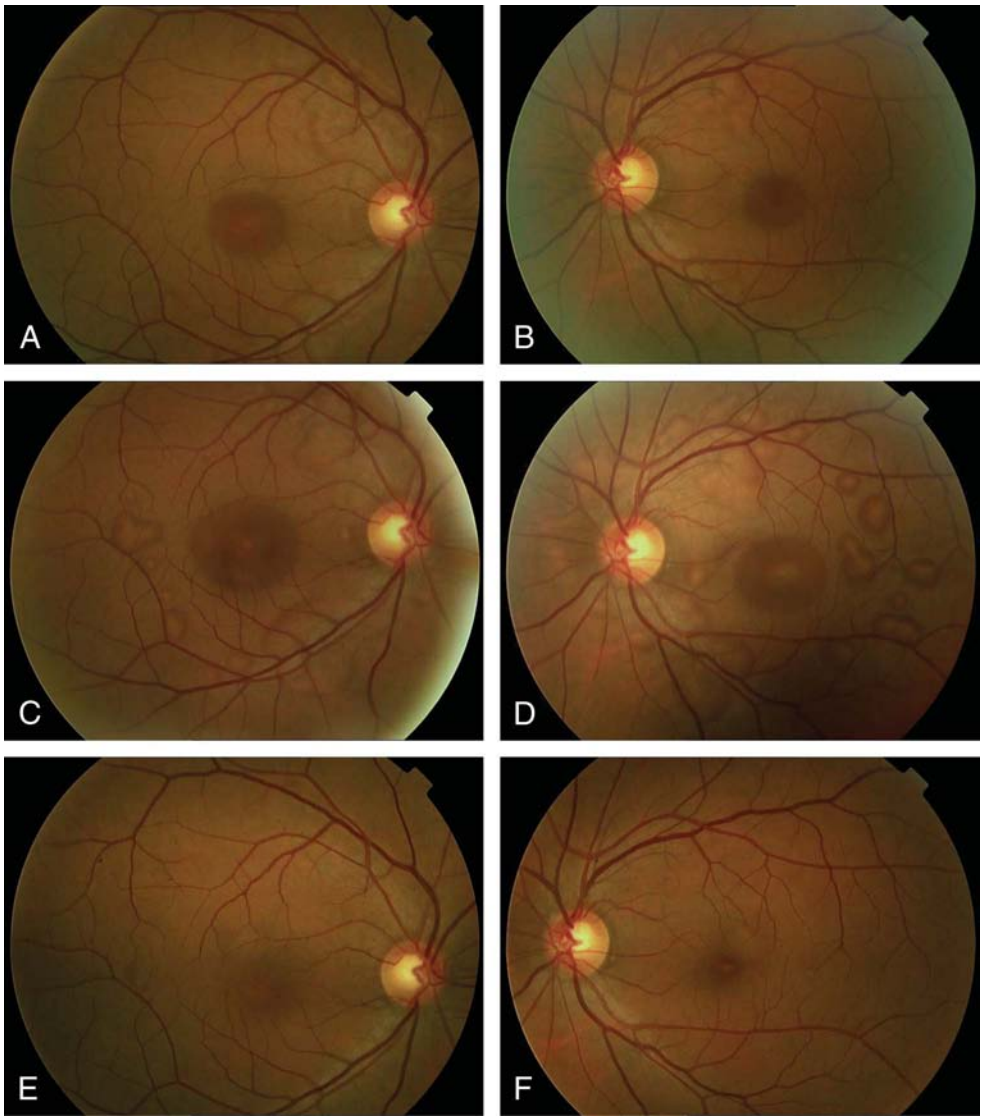

Fig. 1. **A**, **B**, On October 5, 2008, fundus examination of an apparently healthy 51-year-old woman revealed a well-delineated oval area of serous retinal detachment involving both maculae, resembling a bilateral CSC. **C**, **D**, Ten days later, fundoscopy showed an enlargement of the neurosensory retinal detachment in both macular areas, associated with multiple variable-sized yellowish serous elevations of the sensory retina along the temporal vascular arcades. **E**, **F**, After corticosteroid therapy (oral prednisone 1 mg/kg/day tapered to zero at about 3 months), the patient was asymptomatic and the serous retinal detachments resolved.

the edges of the macula in each eye. Her signs and symptoms resolved within 3 months during which time the authors treated the patient first with oral acetazolamide and then with oral corticosteroids.

This case presents several diagnostic challenges. One challenge is the accurate characterization of the multifocal, polymorphous, yellow subretinal lesions. The patient's clinical course, OCT, and FAs suggest that these multifocal pockets of fluid are serous retinal detachments. The fundus photographs, however, show lesions that look more like RPE detachments because they are dome shaped, sometimes have irregular edges, and fail to coalesce even when close to one another. Optical coherence tomography is the best tool for characterizing structural changes in the retina and surrounding tissues. Because the OCT findings are consistent with serous retinal detachments, I agree with the authors that these lesions most likely represent atypical, multifocal, sometimes irregular, unusually yellow-colored, serous retinal detachments.

A second challenge is to determine the source of the multifocal polymorphous subretinal fluid collections. While in homeostasis, there is no accumulation of fluid in the subretinal space because the surrounding tissues efficiently remove excess subretinal fluid and prevent the formation of serous retinal detachments. Marmor and Yao list three requirements for the formation of a serous retinal detachment: a source of fluid, a defect in the blood–retinal barrier allowing the fluid access to the subretinal space, and a surrounding area of impaired subretinal fluid removal.[3]

Possible sources of fluid that can cause a serous retinal detachment include the liquid component of the vitreous blood in the retinal capillaries and blood in

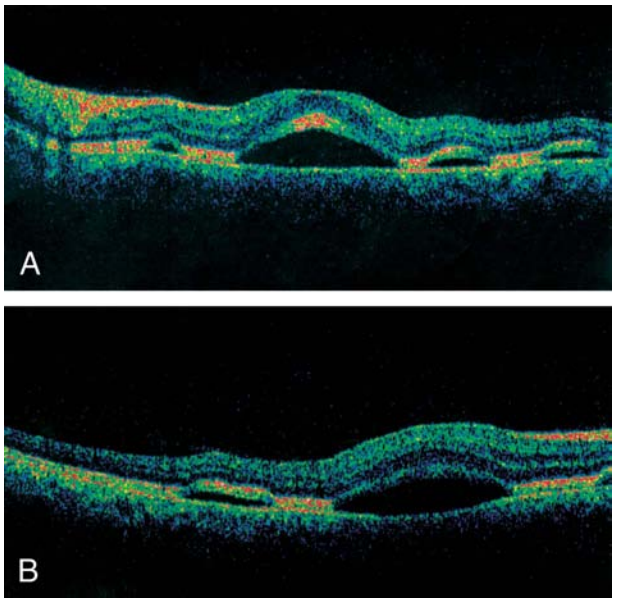

Fig. 2. A, **B**, Optical coherence tomography, at the same time as Figure 1B and C, demonstrated multiple blister-shaped hyporeflective lesions compatible with serous neurosensory retinal detachments, confirming the hydrodynamic separation of the sensory retina from the RPE. The tallest and widest lesions were subfoveal. The foveal depression was lost, but the thickness of the overlying retinal appeared normal. No fusiform or polypoidal thickening of the hyperreflective layer was shown by OCT to suggest the presence of occult neovascularization.

the choriocapillaris. Physiologic removal of subretinal fluid is driven by osmotic pressure, hydrostatic pressure, and active solute-linked water transport across the RPE (a pump). The most important of these transport mechanisms is the active RPE pump.[4]

The imaging studies all suggest that the source of the subretinal fluid is the leaky choriocapillaris. The OCT line scans show that the retina is of normal thickness, suggesting that the subretinal fluid did not traverse the retina but rather originated from beneath the retina in the choriocapillaris. The FA shows very slow accumulation of fluorescein in the subretinal space through the compromised RPE blood–retinal barrier. The ICG angiogram also shows the choriocapillaris to be excessively leaky.

Because a leaky choriocapillaris is not sufficient to cause a serous retinal detachment, a third challenge presented by this case is to offer a reasonable explanation for the loss of two distinct and important functions of the RPE: the loss of the barrier function of the RPE that prevents influx of fluid from the choriocapillaris into the subretinal space and the loss of the active pump that transports fluid from the subretinal space to the choriocapillaris.[5] Normal RPE structure and function can be disrupted by inflammation, infiltration, or choroidal ischemia. Each of these three potential causes of RPE disruption has a limited differential diagnosis.

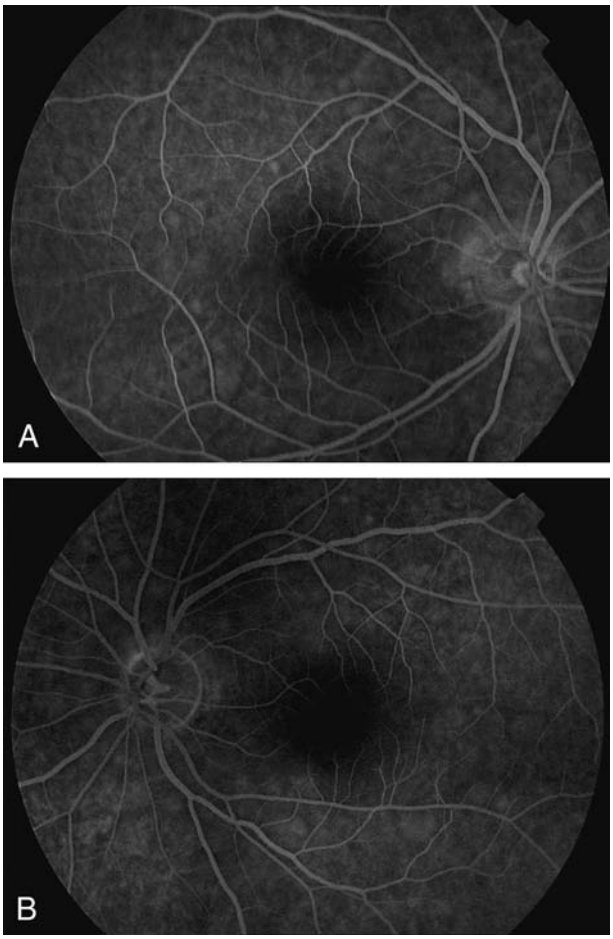

Fig. 3. A, **B**, Late-phase FA, at the same time as Figure 1B and C, identified minimal diffuse mottled hyperfluorescence in both eyes sparing the fovea and hypofluorescence of the denser, thicker, yellow lesions. The absence of chorioretinal vascular compromise seemed to contribute to the minimal expansion of the dye into serous cavities late into the study.

Choroidal ischemia can cause serous retinal detachments in patients with hypertensive retinopathy, giant cell arteritis, pregnancy, severe renal disease, systemic lupus erythematosus, polyarteritis nodosa, Goodpasture syndrome, Wegeners granulomatosis, relapsing polychondritis, and thrombotic thrombocytopenia purpura.[6] Choroidal and RPE inflammation and infection can cause serous retinal detachment in patients with Harada disease, posterior scleritis, sarcoidosis, tuberculosis, toxoplasmosis, and sympathetic ophthalmia. Finally, choroidal and RPE infiltrates can cause serous retinal detachment in patients with idiopathic uveal effusion, benign reactive lymphoid hyperplasia, choroidal tumors, and bilateral diffuse uveal melanocytic proliferation.

In this case, localized inflammation or ischemia is probably disturbing the choriocapillaris and RPE. The authors do not report whether the patient experienced

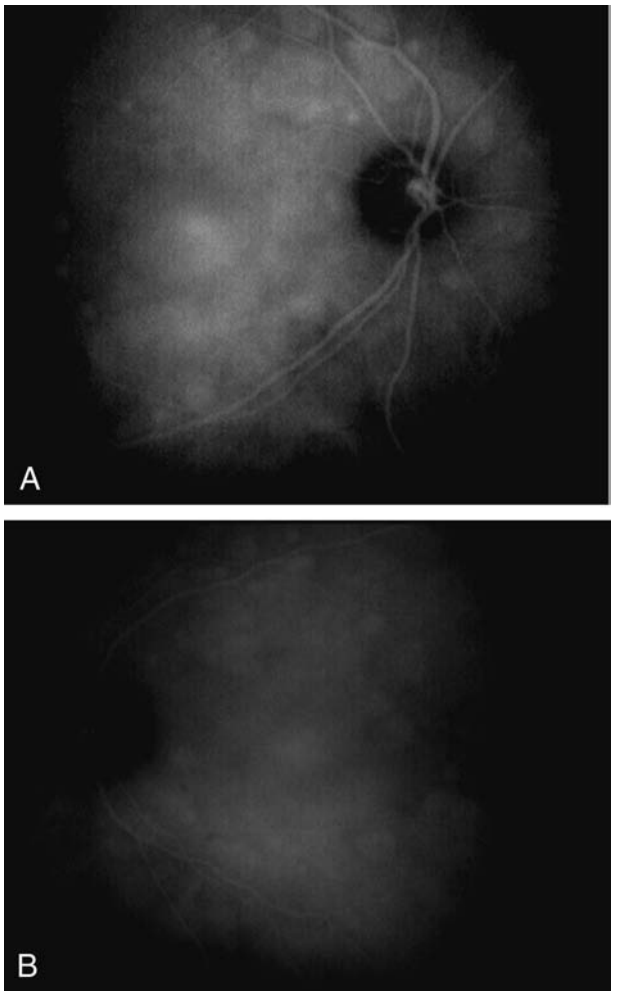

Fig. 4. A, **B**, Indocyanine green angiography, at the same time as Figure 1B and C, showed early hyperfluorescence in the posterior pole of the choroid, which became prominent in the late phase. Indocyanine green angiography revealed neither occult polypoidal nor other forms of choroidal neovascularization.

eye pain or pain on eye movement. The absence of eye pain or pain on eye movement would be evidence against the presence of diffuse inflammation of the posterior pole. Also, the normal EOG suggests that there is not widespread compromise of the RPE. Also, the absence of staining of the optic nerve head and retinal vessels in the FA is evidence against the presence of diffuse inflammation of the posterior pole.

This patient's multiple serous retinal detachments in association with increased leakage of dye from the choriocapillaris on the ICG angiogram suggest that the patient may have CSC. The cause of hyperpermeability of the choriocapillaris in CSC is not yet known but has been associated with elevated catecholamines and corticosteroids. Catecholamines and corticosteroids may affect capillary fragility, capillary permeability, and choroidal vessel diameter.[6]

Most patients with CSC recover good vision without treatment. Although some treatments may speed visual recovery, no treatment has been shown to improve the final visual acuity of patients with acute CSC. Therefore, treatments with known risks should be avoided. Patients with chronic CSC with symptoms of at least 6 month-duration may benefit from intravitreal injection of a vascular endothelial growth factor inhibitor, focal laser, grid laser, and photodynamic laser.

There was a time when corticosteroids were used to treat patients with CSC. Interestingly, the first report of corticosteroids for use in macular disease describes a case of CSC that improved with escalating dose of systemic corticosteroids.[7] Today, many physicians avoid the use of corticosteroids in patients with CSC because sometimes corticosteroids worsen the course of the disease.[8]

Corticosteroids could affect the choriocapillaris and RPE because they suppress inflammation, reduce levels of vascular endothelial growth factor, and affect nitric oxide. Recent studies showing that corticosteroids alter the expression of nearly 2000 genes in the retina and RPE suggest that the effects of corticosteroids on these tissues are legion.[9] Although it is dangerous to administer corticosteroids to some patients with CSC, there may be other patients who benefit from corticosteroid therapy. This patient was treated with oral acetazolamide and oral corticosteroids. The effect of treatments is difficult to evaluate in patients with CSC because the disease is usually self limited. It may be that this patient recovered visual acuity despite the treatment with corticosteroids and not because of the treatment with corticosteroids.

Finally, although this case most closely resembles CSC, it is atypical. This patient is a woman, yet CSC is uncommon in women.[10] This patient had multifocal serous retinal detachments and bilateral symptomatic disease both of which are uncommon in CSC. In addition, the distribution of the serous retinal detachments in this patient's eyes–toward the edge of the macula with a different looking central lesion–was unusual and remarkably symmetric between the two eyes as you would see in the eyes of a patient with a retinal dystrophy or with ocular manifestations of a systemic disease. Also, the patient did not worsen on corticosteroids as patients with CSC sometimes will.

Many features of this patient's case are similar to those published with acute exudative polymorphous vitelliform maculopathy syndrome.[11–14] Acute exudative polymorphous vitelliform maculopathy is a rare inflammatory or immune-mediated disease of the retina and underlying RPE characterized by multifocal yellow subretinal deposits.[12] Like the patient in this report, patients with acute exudative polymorphous

vitelliform maculopathy present with acute bilateral vision loss; serous macular detachment; multifocal yellow subretinal lesions; ICG angiography evidence of increased permeability of the choriocapillaris; and subsequent resolution of signs and symptoms.[15] Unlike the patient in this report, patients with acute exudative polymorphous vitelliform maculopathy usually present with a marked headache a few days before vision loss; a preceding flu-like illness; abnormal electrooculogram; and persistent subretinal yellow deposits for months or years. Fewer than 20 cases of this rare disorder have been published, and there are variable clinical presentations.[11–15]

Because uncommon manifestations of common diseases are more common than uncommon manifestations of uncommon diseases, this case is more probably an atypical presentation of CSC than an atypical presentation of acute exudative polymorphous vitelliform maculopathy.

Dr. Sam S. Yang (Walnut Creek, California):

Dr. Ascaso et al presents a case of a previously healthy 51-year old woman with bilateral macular serous retinal detachment. Her ocular, medical, and family histories are reported as unremarkable. After a course of oral acetazolamide treatment, the bilateral neurosensory detachment enlarged with multiple new lesions of various sizes. The FA and ICG do not reveal any underlying choroidal neovascular lesions, but the ICG shows choroidal vascular hyperpermeability. After a course of oral corticosteroids, the serous retinal detachments completely resolved with vision recovery. This case raises the question of the etiology of bilateral multifocal serous neurosensory retinal detachment.

Any disorder that can destabilize the complex balance of forces at the level of the RPE and choroidal circulation can affect the retina and retinal-pigmented epithelial adhesion, resulting in abnormal fluid accumulation and neurosensory detachment. In this case, several categories of etiologies should be considered. The patient clinically resembles idiopathic CSC. However, the patient's improvement with systemic steroids, which are well known to exacerbate the course of idiopathic CSC, would argue against this. The resolution of the neurosensory detachment with corticosteroids is more consistent with an underlying inflammatory etiology.

Several inflammatory diseases may associate with bilateral serous retinal detachment by compromising choroidal vascular permeability and choroidal interstitial fluid composition. In this case, Vogt–Koyanagi–Harada (VKH) syndrome, sarcoidosis, and posterior scleritis should be considered. Similar to VKH, this patient responded promptly with corticosteroids. Patients with VKH typically present with bilateral serous retinal detachment in the acute uveitic phase and may be accompanied with neurologic signs (meningismus and tinnitus) and integumentary manifestations (alopecia, poliosis, and vitiligo). The absence of uveitis and extraocular findings in this patient is clinically inconsistent with typical VKH. However, the clinical features of VKH can vary in atypical or incomplete variants of the disease. The early frames of her FA are not available. The late frames lack the multiple punctate hyperfluorescent pattern typically seen in VKH. In this case, lumbar puncture and B-scan ultrasound may be useful adjunctive tests to support the VKH diagnosis with the presence of cerebrospinal fluid pleocytosis and thickening of choroid.

Sarcoidosis also has variable ocular manifestations, including serous retinal detachment with or without anterior uveitis and focal choroidal granulomas.[16] Serum angiotensin-converting enzyme, lysozyme, and chest x-rays would be helpful to screen for this condition. Sarcoidosis may also manifest in the hepatic system. This patient's liver enzymes are elevated, which may prompt an imaging study such as ultrasound or CT scan to evaluate the hepatobiliary system for granulomatous nodules.

Posterior scleritis can also improve with corticosteroids. Although it may involve both eyes, posterior scleritis typically presents unilaterally. The patient's lack of eye pain, redness, and physical signs (choroidal folds, retinal striae, and disk edema) argues against posterior scleritis being the etiology. However, up to 15% of patients may not present with physical signs of posterior scleritis.[17] Obtaining both A- and B-scan ultrasound would be useful to look for both choroidal and scleral thickening and the presence of edema in the Tenon's space.

Other inflammatory systemic disorders can induce acute occlusion of the choriocapillaris or choroidal arterioles by vasculitis with fibrin–platelet thrombi and neutrophil invasion of the vessel wall. This may result in acute necrosis of the overlying RPE and serous retinal detachment. Systemic lupus erythematosus was reported to develop serous detachment in its primary disease.[18] A screening antinuclear antibody assay would be useful in the evaluation. Other collagen vascular diseases such as polyarteritis nodosa, Wegener granulomatosis, relapsing polychondritis, scleroderma, dermatomyositis, and Goodpasture syndrome were also reported to associate with focal detachment of neurosensory retina.[19] The medical history is not supportive of the constellation of systemic findings that would accompany these disorders. A complete physical

examination with sedimentation rate and C-reactive protein assays may be helpful to screen for these systemic inflammatory diseases.

Other categories in the differential list include disorders with abnormal clotting state that can also lead to occlusion of choroidal microvasculature. This patient's normal hemogram and coagulation studies ruled out disseminated intravascular coagulopathy and thrombotic thrombocytopenic purpura. Other conditions in this category include malignant hypertension, malignancies, preeclampsia, sepsis, and organ transplant.[19,20] Often, these patients have associated cotton wool spots, retinal hemorrhages, and intraretinal lipid. Furthermore, the ICG would show areas of patchy filling delay indicative of segmental choroidal vascular ischemia. The lack of these ocular findings in the patient and noncontributory medical history suggest that these etiologies are not likely to be the underlying cause.

As mentioned by the authors, paraproteinemias such as cryoglobulinemia, IgA and IgM gammopathies, and hyperviscosity syndrome were described with serous detachment of the macula.[19] Hypoproteinemia secondary to insufficient intake, synthesis, or absorption could also induce serous retinal detachment. However, the patient's normal protein electrophoresis and immunoglobulins ruled out these dysproteinemia conditions.

Last, a number of systemic infectious etiologies are associated with serous retinal detachment. Bacterial etiologies include tuberculosis and syphilis, which the authors ruled out with the negative Mantoux test and syphilis serologies. Other infectious etiologies such as Lyme disease and cat scratch disease can also present with serous retinal detachment.[21] Exposure history and serum titers for Lyme and cat scratch disease may be helpful. However, the rapid improvement with corticosteroids in this case suggests that an infectious etiology is low on the differential list.

In summary, the responsiveness to corticosteroids in this case of bilateral multifocal serous retinal detachment suggests an inflammatory etiology. Additional systemic workup would be helpful to narrow down the underlying cause. The patient will require future monitoring for recurrences as well as any new ocular and systemic manifestations.

Editor's Note:

Drs. Ascaso, Rojo, and Minquez present a 51-year-old woman with sudden onset metamorphopsia and decreased vision. Fundus examination revealed bilateral multiple serous detachment.

We have asked Dr. Steven Cohen and Dr. Sam Yang to help us with this case, and they have provided a differential diagnosis.

I. Choroidal ischemia
  A. Hypertensive retinopathy
  B. Giant cell arteritis
  C. Pregnancy
    1. Preeclampsia
  D. Severe renal disease
  E. Systemic lupus erythematosis
  F. Collagen vascular diseases
    1. Polyarteritis nodosa
    2. Goodpasture syndrome
    3. Wegener granulomatosis
    4. Relapsing polychondritis
    5. Scleraderma
    6. Dermatomyositis
  G. Coagulopathies
    1. Disseminated intravascular coagulopathy
    2. Thrombotic thrombocytopenic purpura
II. Choroidal Inflammation/Infection
  A. Inflammatory disease
    1. Acute exudative polymorphous vitelliform maculopathy syndrome
    2. VKH syndrome
    3. Posterior scleritis
    4. Sarcoidosis
    5. Sympathetic ophthalmia
  B. Infectious disease
    1. Bartonella
    2. Tuberculosis
    3. Lyme
    4. Toxoplasmosis
III. Choroidal Infiltrate
  A. Benign reactive lymphoid hyperplasia
  B. Choroidal tumors
  C. Bilateral diffuse uveal melanocytic proliferation
IV. Idiopathic
  A. Uveal effusion syndrome
  B. Central serous chorioretinopathy
    1. Steroid induced
V. Other
  A. Paraproteinemias
    1. Cryoglobulinemia
    2. Gammopathies IgA, IgM
    3. Hyperviscosity syndromes
  B. Hypoproteinemia
    1. Insufficient intake
    2. Insufficient synthesis
    3. Insufficient absorption

The differential diagnosis of serous retinal detachment is vast. The workup of such a patient has been reviewed by our consultants and is directed at ruling out those entities listed in the differential diagnosis. Dr. Yang feels that the responsiveness to corticosteroids suggests an inflammatory etiology. Dr. Cohen feels that this case most closely resembles central serous choroidopathy but notes irregularities in its presentation in this patient. The sticking point being that the patient improved with corticosteroids, a

therapy we would expect to worsen the condition, not improve it. He rightly notes that a claim that a treatment benefits a patient afflicted with a self-limited disease, like CSC, must be evaluated cautiously. Well said. Dr. Cohen also notes that patients with the rare entity, acute exudative polymorphous vitelliform maculopathy, may present with similar findings, although our patient did not have preceding headache or flu-like symptoms. Nevertheless, this is an intriguing possibility.

We thank our consultants for their excellent review of this interesting case.

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