Although uncommon, scleritis is a dangerous immune-mediated disease that can potentially threaten the structural integrity of the eye and may be indicative of potentially life-threatening systemic vasculitis.¹ Data on the genetic factors of scleritis is lacking, but it is thought that genes affecting systemic autoimmune diseases impact scleritis as well.² Differentiating between episcleritis and scleritis and posterior and anterior scleritis can be challenging for physicians. An accurate diagnosis is critical to properly treat the disease and prevent vision loss. In the following summary of a roundtable discussion, experts comment on the complexity in diagnosing scleritis, compare the efficacy of therapeutics based on patient presentation, and review treatment plans for patients who are refractory or nonresponders.

**DIAGNOSING SCLERITIS**

**Melissa Toyos, MD:** What percentage of patients in your practice have scleritis?

**Stephen D. Anesi, MD, FACS:** Scleritis accounts for 10% to 15% of the patients I see in my practice.

**David S. Chu, MD:** I agree; 10% to 15% sounds right to me as well.

**Thomas A. Albini, MD:** It is a small number. I see more posterior segment inflammatory disease, but scleritis makes up about 5% of my uveitis patients. The cases are often among the most painful cases and the most challenging to treat.
Robert C. Wang, MD: I’d say about 50% of my practice is treating uveitis and 5% is treating scleritis.

Dr. Toyos: The literature tells us that most scleritis patients are between ages 30 and 50, and that women are affected by scleritis twice as much as men. Its prevalence is estimated to be about six cases per 100,000 people, but it is higher in patients with rheumatoid arthritis or granulomatosis with polyangiitis (formerly known as Wegener granulomatosis). How do scleritis patients typically present in your office? What symptoms do you normally see?

Dr. Chu: Most of my scleritis patients are referred to me from optometrists and other ophthalmologists, and they typically present with chronic scleritis that isn’t improving, despite months of treatment or scleritis that’s recurred. They typically have a painful eye that’s tender to palpation and redness. It’s important to note that not every scleritis patient has pain. If the patient appears to have scleritis but doesn’t have pain, it could still be scleritis. This is especially true in children; their scleritis could be painless.

Dr. Anesi: Many of my patients are referred to me for pink eye or uveitis, and they actually have scleritis. I’ve actually had patients with uveitis who later develop scleritis independently. Scleritis can crop up later, especially in patients with autoimmune disease. Symptoms that help to differentiate scleritis from other types of eye redness are exquisite tenderness, pain with eye movement or pain that awakens patients from sleep. The association between scleritis and systemic disease has been reported in up to 50% of cases, the most common being rheumatoid arthritis, followed by granulomatosis with polyangiitis, inflammatory bowel disease, and systemic lupus erythematosus, among others.

Dr. Albini: My patients often complain of focal or pinpoint tenderness in the involved area. I typically ask patients to lightly close their eyes and gently touch their eyes through the eyelids. If they have scleritis, you will see signs of pain readily. Eye pain is the most consistent symptom. In the setting of pain, enlargement of the deep scleral vessels makes the diagnosis.

Dr. Wang: The most distinctive symptom of scleritis is orbital pain, which is found in about 60% of patients. Many of my scleritis patients present with deep, achy ocular pain that they have a difficult time verbalizing. I agree that not everyone will complain of classic scleritis pain symptoms, which makes the examination so important. Differentiating between the different types of scleritis and between scleritis and episcleritis can be challenging.

Posterior scleritis, which is a risk factor for decreased vision, is difficult to diagnose because there is no redness when the anterior sclera is not involved, pain is more variable, and examination may show elevation of the adjacent retina and choroid. Up to 12% of scleritis is posterior. Distinguishing between scleritis and episcleritis is not straightforward either, but there are some differences. Patients with scleritis, which is typically bilateral, can experience extreme pain, have symptoms for years, and have a violaceous vessel color. Patients usually describe a dull, achy pain with a slow onset that typically increases over several days. Patients with episcleritis, however, present with minimal pain, have symptoms for days or months, and have a light pink vessel color. Further, episcleritis is almost always unilateral and acute; onset may occur overnight. The signature clinical feature of episcleritis is sectorial injection in the nasal or temporal quadrant. Cells are not usually in the anterior chamber.

I was trained to examine the patient under normal lights, not under slit lamps, because you can miss that deep purple hue indicative of scleritis. If I’m not sure, I’ll use the phenylephrine blanching technique, which is blanching congested conjunctival and superficial episcleral blood vessels with either the 2.5% or the 10% concentration. If the deep episcleral plexus does not blanch, it’s scleritis; if the redness disappears, it’s episcleritis.

Dr. Toyos: What is your approach to diagnosing these patients?

Dr. Chu: We have patients complete an intake questionnaire, which helps us save time when reviewing the symptoms and the systems. The intake form is comprehensive, but if a patient comes in with scleritis, we concentrate on the patient’s history of autoimmune disease, osteoarthritis, and rheumatoid arthritis. It’s also important to assess for other collagen-vascular diseases, and focus on that in the physical examination, blood test, and rheumatology consult. We want to know what else may be hiding in their immune system.

Dr. Albini: I ask for history of rheumatoid arthritis or systemic vasculitis and order some basic laboratory screening. Recognizing associated systemic conditions is critical to the patient’s overall care, but usually does not change the treatment of scleritis. The most difficult problem is identifying infectious cases, so I look for corneal changes or intraocular inflammation to tip me off. Continued observation as more aggressive therapies, like oral steroids, are initiated is critical because patients who do not respond to high-dose steroids are at risk for infectious causes.
Dr. Chu: Most scleritis cases are noninfectious. The patient’s history is key to diagnosing infectious scleritis, as the majority of cases have an identifiable factor that triggered the symptoms. The most common include prior ocular surgery such as pterygium excision, cataract extraction, and scleral buckle. If the patient has a prior history of ocular surgery, then I am automatically suspicious of infection and am quick to take a scleral biopsy to identify the organism. These patients can present with infectious scleritis years after surgery. If the scleritis is unilateral and not responding to therapy, you should suspect a herpetic etiology. It’s estimated that herpes viruses account for 7% of all scleritis cases.

Dr. Anesi: Scleritis and uveitis have similar symptoms at presentation: redness and pain. However, with scleritis, there’s almost always a distinct lack of light sensitivity, photophobia, or visual changes. Uveitis is extremely symptomatic, poses a threat to vision, and typically occurs in older people. Bright light tends to exacerbate symptoms. Uveitis is typically unilateral and patients present with increased vascular dilation over the ciliary body. If you have someone with just redness in their eye and the deep scleral vessels are involved, you can often tell that from the examination. However, if the patient has posterior scleritis, it becomes more challenging to diagnose because you don’t know if it’s the eyeball or the part of the orbit. It’s all very closely associated.

When it comes to working up the patient, unilateral versus bilateral symptoms make a big difference to me. In the unilateral cases, especially with nodular involvement, I oftentimes think herpetic. Herpes simplex virus type 1 is very common in the population. I test everyone for the presence of herpes IgG 1 and 2. Most cases of orofacial herpes infection are caused by type 1, but type 2 can cause scleritis as well, especially in younger patients.

The intake questionnaire is particularly helpful. For example, if the patient has keratitis, you’re going to think either herpes or rheumatoid arthritis. If the scleritis is bilateral, you should suspect a collagen vascular disorder, rheumatoid arthritis, or vasculitis.

I recommend doing a serologic panel. I test for IgM and will even look for inflammatory markers indicative of underlying inflammatory disease. Other diagnostic tests include angiotensin-converting enzyme (ACE) for sarcoidosis; antinuclear antibody (ANA) for lupus; cytoplasmic antineutrophil cytoplasmic antibody (c-ANCA) for Wegener granulomatosis; perinuclear antineutrophil cytoplasmic antibody (p-ANCA) for vasculitis and polyarteritis nodosa; fluorescent treponemal antibody absorption (FTA-ABS) and rapid plasma reagin/venereal disease reference laboratory (RPR/VDRL) for syphilis; enzyme-linked immunosorbent assay (ELISA) for Lyme disease; and rheumatoid factor (RF) for rheumatoid arthritis.

Dr. Wang: When diagnosing scleritis, I look for the five most common underlying causes: rheumatoid arthritis, lupus, polyarteritis nodosa, granulomatosis with polyangiitis, and relapsing polychondritis. The challenge is there is not a single blood test available to diagnose these conditions, although I do order blood work as part of the assessment. For relapsing polychondritis, for example, you make a clinical diagnosis and then get a biopsy. If someone doesn’t fit the typical clinical picture, I dive deeper into their history to determine why. Is the patient on any medication that could be causing their condition? Bisphosphonates have been known to cause scleritis. And, although rare, scleritis can develop in patients taking infliximab.

Dr. Toyos: How do you differentiate between posterior and anterior scleritis?

Dr. Wang: I recommend an ultrasound examination or CT of the orbit, which can demonstrate a thickening of the sclera. B-scan ultrasonography and orbital magnetic resonance imaging (MRI) can also successfully detect posterior scleritis. On the ultrasound, you’re looking for choroidal thickening, scleral nodules, distended optic nerve sheath, fluid in Tenon capsule, or retinal detachment. Optical coherence tomography (OCT) can also be used to look for nodular posterior scleritis.

Dr. Albini: Both are painful. Anterior scleritis is readily apparent (Figure 1). Posterior scleritis may be more subtle,
but multimodal imaging like OCT and fluorescein angiography may demonstrate nonspecific changes, like choroidal thickening. Ultrasound is critical because tenons infiltration, especially the “T sign” around the nerve strongly suggests the diagnosis of posterior scleritis.

**Dr. Anesi:** I agree; OCTs are very helpful looking for areas of retinal thickening if posterior scleritis is suspected. Patients with posterior scleritis will often experience pain, but they don’t always have vision changes. If vision changes do occur, it’s usually around the nerve, resulting in visual field defects. You’re looking for the classic T sign of fluid around the optic nerve, but you don’t see that very often. More frequently we see thickening of the posterior retinal or choroidal scleral complex. Occasionally you’ll see some fluid or a subtle enhancement in one area versus the other comparable side of the other eye. That may tip you off or give you a hint that this is probably something in the posterior sclera that’s active (Figure 2).

**Dr. Toyos:** The literature tells us that fluorescein and indocyanine green angiography can help distinguish posterior scleritis from other issues, such as central serous retinopathy, and uncover optic nerve edema or serous retinal detachment, which are other characteristics of posterior scleritis. Does anyone use fluorescein on these patients?

**Dr. Wang:** Not routinely for me.

**Dr. Chu:** Not really for scleritis patients. For imaging posterior scleritis, I will sometimes use a widefield fundus camera to potentially pick up peripheral choroidal effusion, which may be evidence of posterior scleritis. Chorioretinal folds (Figure 3) in the macula are sometimes picked up on imaging as well.

**Dr. Anesi:** I do recommend an angiogram in a patient with vision changes associated with posterior orbital pain. You want to ensure there isn’t a hot nerve or some type of vasculitis that you didn’t catch on the exam if there’s an absence of uveitis that you can’t see.

**Dr. Toyos:** I agree. In select situations, especially if you have the technology, an angiogram can be helpful.

## Treating and Managing Scleritis

**Dr. Toyos:** What treatment do you recommend for patients with scleritis?

**Dr. Albini:** I typically offer oral NSAIDs first, especially for milder cases. In more severe cases, or cases that have failed a 2-month course of NSAIDs, I start oral steroids. If the scleritis recurs more than annually after tapering off steroids, I treat with immunomodulatory therapy (IMT), typically methotrexate. In really aggressive cases I have had good luck with subconjunctival steroid injections or biologics like anti-TNF or rituximab, if I can get them covered off-label. In necrotizing anterior scleritis, I always start with oral steroids and avoid subconjunctival injection.

**Dr. Anesi:** You want to get the patient worked up as quickly as possible, but there are some things you can do in the meantime while you’re waiting for the blood work. The first priority is improving their symptoms. For example, if the patient is in pain, one can often address this with topical steroids, regardless of whether scleritis is noninfectious or even viral, though one should be mindful of avoiding these if there is any viral keratitis, unless also using a systemic antiviral medication. Anything that’s anterior will usually respond to a topical steroid, but if it’s more severe or bilateral, one can consider prescribing systemic corticosteroids acutely as well, unless they have comorbidities that would preclude steroid use. I typically recommend prednisone 1 mg per kg body weight up to 60 mg daily, which seems to help.
If I suspect viral involvement, I’ll start the patient on valacyclovir 1 g three times daily or acyclovir 800 mg five times daily as a trial to see if this alone will help abate symptoms, usually even without starting steroids. Both agents are fast-acting, and patients will start improving within a week on these doses, which are reserved for more active viral disease. These are very effective and almost completely benign, although there is a concern with anyone with kidney disease because of rare renal toxicity.

Dr. Toyos: What are the primary comorbidities you need to be mindful of before prescribing oral steroids? For me, the first thing that comes to mind is unstable cardiovascular disease, gastrointestinal ulcers, and uncontrolled diabetes.

Dr. Anesi: I agree. You must also be careful when prescribing oral steroids in anyone who is being treated for malignancy. You want to ask their oncologist to confirm that you can prescribe prednisone.

Dr. Toyos: What percentage of your scleritis patients do you think are on or have been on oral steroids?

Dr. Wang: About 90% of my patients have already been treated and have used steroids. Some patients have been on various steroids chronically and don’t want to take them anymore. Most of the time I’m trying to find something else to reduce flaring or quiet it quickly, and then something more for long-term management.

Dr. Albini: Most of my patients are on oral steroids. I would estimate probably 75% of my patients. Then I try to taper to less than 10 mg daily by 2 months and switch to a steroid-sparing agent like methotrexate.

Dr. Chu: Most of my patients have tried topical steroids without resolution of the inflammation. I generally start scleritis patients, except for those with necrotizing variety, on oral NSAIDs (meloxicam or diclofenac) while I’m waiting on the blood work. I find that it’s effective in a large percentage of patients with noninfectious scleritis (Figure 4), and that therapy alone may be enough to resolve up to 50% of anterior scleritis cases. I move on to oral steroids if the patient doesn’t respond to oral NSAIDs or if they don’t respond quickly enough.

Figure 4. These images show a patient before (A) and after (B) treatment with oral NSAIDs.

Dr. Toyos: What’s your initial steroid dose?

Dr. Chu: Generally, 1 mg/kg/day up to 60 mg. For patients with necrotizing scleritis or peripheral ulcerative keratitis, I may go up to 120 mg/day, depending on the size of the patient. Those are special circumstances. I typically don’t exceed 60 mg at the initial treatment. You have to consider the side effects.

Dr. Toyos: I think we’re all too familiar with the robust side-effect profile of traditional oral steroids, and we all try to minimize the potential damage. Adverse events from oral steroids include but are not limited to osteoporosis and fractures; adrenal suppression; hyperglycemia and diabetes; cardiovascular disease and dyslipidemia, dermatological and gastrointestinal issues; psychiatric disturbances; and immunosuppression. That said, they can be effective. One international study (68 eyes) suggested subconjunctival triamcinolone acetonide injection (2 to 40 mg) for nonnecrotizing, noninfectious anterior scleritis can successfully treat the disease, with only a few patients experiencing elevated intraocular pressure and without necrosis or scleral melt over the long term.

What are your favorite steroid-sparing drugs when the course of steroids don’t work? How long do you give the steroids before you move into the next phase?

Dr. Chu: NSAIDs are the first-line treatment. I’ll add an oral steroid if the NSAID alone is not working. If that combination controls it, then I taper the steroid. If the scleritis flares, I’ve traditionally put the patient on methotrexate. We conducted a small study and found methotrexate to be corticosteroid sparing and well tolerated in the treatment of chronic, noninfectious, and nonnecrotizing scleritis. Of the 56 patients/84 eyes with scleritis in the study treated with methotrexate, 56.4% had complete control of inflammation sustained over at least 28 days and was achieved at or before 6 months of treatment.
“OSTEOPOROSIS AND ULCERS ARE TWO THINGS I WORRY ABOUT FOR EVERYONE TAKING STEROIDS. I USE A STEP-LADDER APPROACH TO TREATING VARIOUS TYPES OF OCULAR INFLAMMATION IN OUR CLINIC.”

A more recent retrospective review looked at the clinical records of noninfectious scleritis patients from the University of Ottawa Eye Institute between Sept. 1, 2010, and Dec. 31, 2014, (21 eyes) who were treated with methotrexate. A total of 69.2% and 92.3% of eyes had corticosteroid-sparing success at 3 and 12 months, respectively. Inflammation control was achieved in 61.9% and 90.5% of patients at 3 and 12 months, respectively.29

However, I said “traditionally” because that paradigm is changing as the treatment of rheumatoid arthritis evolves. The most common condition associated with noninfectious scleritis is rheumatoid arthritis, so the scleritis treatment follows that treatment pattern. If the rheumatologist is using biologics to treat the rheumatoid arthritis, then to that clinician, the first-line treatment is TNF-alpha inhibitors (infliximab, adalimumab, and etanercept).30 We find that our choice for patients are TNF-alpha inhibitors as an early steroid-sparing therapy if patients are either steroid-dependent or they failed NSAIDs.

Dr. Toyos: The TNF-alpha inhibitors are not without significant risk of side effects, such as numbness, vision problems, leg weakness, and chest pain.31 But I’ve had several patients, long-term steroid users, who developed a gastrointestinal ulcer and osteoporosis, and experienced mood swings and irritability, who elected to remain on steroids in light of the potential side effects of infliximab, adalimumab, and etanercept. The side effects for long-term steroid use are significant, but the side effects of TNF-alpha inhibitors are often worse.

Dr. Anesi: Osteoporosis and ulcers are two things I worry about for everyone taking steroids. I use a step-ladder approach to treating various types of ocular inflammation in our clinic. NSAIDs are helpful in nonnecrotizing mild-to-moderate anterior scleritis, if it’s not infectious. In patients with severe or nonresponsive disease, especially if there’s evidence of necrotizing disease or keratitis, I think about using immunosuppressive agents such as antimetabolites and/or biologics.

For severe cases of rheumatoid arthritis-associated scleritis, I turn to intravenous medications such as infliximab and rituximab. Rituximab is an effective treatment for recalcitrant noninfectious or idiopathic scleritis, with some patients experiencing long-term, durable, drug-free remission.32,33 I give it more aggressively than the standard two doses at 1,000 mg 2 weeks apart every 6 months. Instead I prescribe a dose of 375 mg per square meter body surface area weekly for 8 weeks, and then monthly to see how they respond. Patients often do well on this regimen.

The strategy is to elevate the aggressiveness of the medication profile as the patient needs it depending on the severity of the disease or the nonresponsiveness to less-aggressive medications. If I think it’s appropriate, I’ll always give them a chance to take an antimalab tight because it’s less expensive, doesn’t require an intravenous infusion, and it’s easier for insurance to approve. That said, I don’t hesitate to treat with biologics if necessary.

Dr. Wang: If a patient has a systemic condition, that drives the initial treatment. But if it’s purely scleritis, I’ve found that a subconjunctival injection of a steroid like triamcinolone does a great job. A paper by Sohn and colleagues34 showed a nearly 50/50 chance that area stays in remission for the next 5 years after that treatment.

For systemic disease, I do like biologics and will prescribe quickly adalimumab or infliximab for patients who are refractory for adalimumab. In patients who are especially recalcitrant, rituximab works well. Acthar Gel (Repository Corticotropin Injection) has also worked well for me. It comes with some controversy in its cost, but I’ve found it to be a great treatment. Side effects of Acthar are similar to steroids, including gastrointestinal bleeding, weight gain, increased risk of infections, cataract, glaucoma, and mood swings, but seem to occur much more infrequently.

USING ACTHAR IN THE CLINIC

Dr. Toyos: A recent retrospective medical review looked at the American Medical Association physician prescriber and Acthar prescriber databases, assessing physicians who prescribed Acthar for uveitis in the past year. To avoid any perception of cherry picking, the doctors were asked to report on the specific characteristics from their last six cases.35

Because the study was a retrospective review, it doesn’t have the same validity that a prospective trial would, but it did provide some interesting information. The researchers looked at 91 separate cases, and 81% of patients had blurred vision and the normal things you would expect with uveitis: light sensitivity, floaters, loss of visual acuity,
eye pain, and redness. In what can only be described as a retinal visual acuity scale, the researchers described mild disease as a visual acuity of better than 20/70. If the patient could drive legally, they had mild disease. If the patient couldn’t drive but was not technically legally blind, that was classified as moderate disease. If the patient was 20/200 but better than 20/400, they were classified as having severe disease.

Acthar was used for the first time in 91% of patients, with an average treatment duration of just over 16 weeks. The statistical analysis was good; 84% of patients had an improvement in symptoms and 16% stayed the same, but not one patient was worse after the administration of Acthar for uveitis. The majority of patients reported an improvement in vision and eye pain. Although any type of dosing regimen was allowable, most patients were prescribed the standard dose of Acthar (40 to 80 units once or twice weekly). The doses were then tapered.

We’ve discussed how many of our patients have already been treated with oral steroids when we see them. In this trial, 71 of 91 patients had been given oral prednisone (at least 10 mg) for 6 months or longer. Coming into the Acthar trial, patients were on an average of 2.5 concomitant medications. During treatment, that number was cut in half, and 3 months after Acthar initiation, many of these patients had terminated treatment, and only 20 patients remained on concomitant medication.

What was remarkable to me was that with the initiation of Acthar treatment, patients were tapered off their other medications, and most patients were able to come off their concomitant medication, even 90 days after Acthar administration. With that in mind, how do you use Acthar and what experiences have you had with it in the clinic?

Dr. Albini: I have used Acthar as a last resort in a handful of patients, with good results in some patients. In one pediatric intermediate uveitis case, a young girl who was on two antimetabolites and a biologic concurrently still had cystoid macular edema. When I added Acthar, it resolved. The problems is we really don’t know the efficacy or complications of the drug in a series of uveitis patients.

Dr. Chu: I use Acthar when patients don’t respond to the first few steps of the step-ladder approach, oral NSAIDs, antimetabolites such as methotrexate and mycophenolate, then biologics, or if they have certain contraindications to the biologics, like a history of multiple sclerosis. It’s another option to help control inflammation. I would give Acthar before an alkylating agent (chlorambucil and cyclophosphamide) in patients with scleritis.

Dr. Toyos: I agree. Alkylating agents have many nasty side effects like hair loss, low blood counts, oral ulcers, and numbness, among others.

Dr. Anesi: I don’t consider Acthar as part of the step-ladder treatment approach because it’s too new; it’s not the first thing we’d think of putting in that group of medications that we use in that order. It does have its place, however. I agree that we should use Acthar before an alkylating agent because the potential side-effect profile is much more benign for patients compared with those drugs. The efficacy of Acthar seems to be very good as well. I currently use it for different types of ocular inflammation, especially in patients who have had difficulties with other medications, allergies, adverse reactions, or contraindications. I also use Acthar in patients who have no good alternative, such as in a patient with Stevens-Johnson syndrome with severe recalcitrant inflammation and who has been reliant on steroids otherwise.

I think Acthar has potential benefit in any type of noninfectious inflammation that we treat.

Dr. Toyos: I agree. As a general ophthalmologist who doesn’t routinely send people to infusion centers, a small subcutaneous injection is a lot more accessible for doctors in my position. We’re all infinitely familiar with the side-effect profile of a traditional steroid. With Acthar, it has the additional benefits of the melanocortin pathway. We’re just starting to understand how that controls inflammation at the site, upregulates the T regulatory cells, and promotes health and may be protective to tissues where the inflammation is occurring.

Dr. Wang: For me, Acthar usually comes before the biologics due to insurance or the rheumatologist. If I put a patient on standard antimetabolites and that doesn’t help
control the inflammation, I’ll add Acthar for combination therapy. That tends to work well.

The side-effect profile is minimal. In the retrospective study mentioned earlier, there were about eight patients with diabetes in the study. Those had to be watched closely. We typically wouldn’t put a diabetic patient on steroids because it can fluctuate their blood sugar. But Acthar seemed to be safe with less steroid side effects than we’re used to seeing. It’s one of those nice places where I get a steroid-like antiinflammatory effect with minimal steroid profile.

**Dr. Tozos:** In our case report, our scleritis patient had not only uncontrolled scleritis, but also uncontrolled steroid response to glaucoma. He was on every topical glaucoma medication and oral acetazolamide, and his pressure was still in the mid-40s. But once we started Acthar, we were able to taper him off those topical antiinflammatories and topical glaucoma medications within a few weeks. That really stood out to me; it was remarkable. Acthar is technically a steroid, but it doesn’t have the traditional side effects associated with steroids.

**Dr. Chu:** We’re currently working on an open-label therapy trial of patients with scleritis using Acthar. We plan to enroll 30 patients for 6 months of treatment to see the response of a noninfectious, anterior scleritis to Acthar (NCT02931175). There are two study groups: group A received Acthar 80 mg injections twice a week and group B received Acthar 80 mg 3 times a week, after receiving daily loading dose of 80 mg for 14 days. The study is ongoing.

We are gathering the information actively and will hopefully have some data to report soon. In terms of the response, we started the trial because I have experience in using Acthar in scleritis. Patients may not respond 100% of the time, but I’ve found that most patients do respond (Figure 5).

**Dr. Tozos:** There are additional ongoing studies looking at Acthar. My clinic is currently recruiting for a study on the clinical efficacy of Acthar in patients with dry eye (NCT03287635). It’s also being explored in retinal vasculitis (NCT0366869), uveitis and panuveitis (NCT0366692, NCT02725177, and NCT03473964), and scleritis specifically (NCT03465111).

**Dr. Anesi:** The number of clinical trials is very exciting. We need these studies to show that it works for various types of ocular inflammation.

**Dr. Albini:** Clinical trial data involving Acthar will be critical for better understanding where this agent fits into our armamentarium. For example, it is really not clear to me yet that Acthar has fewer systemic side effects than steroids when used over long periods of time. I certainly hope it does.

**Dr. Tozos:** Thank you all for your comments on managing and treating scleritis in today’s real-world clinical environment.

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