Which came first: the iris neovascularization or the uveitis?

Abstract

A 67-year-old male presents to the clinic with acute onset blurred vision and classic symptoms of iridocyclitis. Examination revealed severe iridocyclitis and new iris neovascularization, possibly due to a history of pneumatic retinopexy.

Case History

A 67-year-old Caucasian male presents to the clinic with complaints of acute onset blurred vision in the right eye. As the day progresses the eye becomes more irritated, red and photophobic. The patient denies flashes or curtain of vision loss. He patient has a history of a horse shoe retinal tear with associated retinal detachment in the right eye, status post pneumatic retinopexy about a year ago. He has a history of amaurosis fugax, status post TIA and carotid endarterectomy. Systemic history is positive for diabetes mellitus type 2, hypertension, hyperlipidemia and congestive heart failure.

Pertinent Findings

Examination reveals visual acuity of 20/200 OD, no improvement with pinhole and 20/25 OS. Pupils were miotic and sluggish, no APD present. Slit lamp exam showed grade 2+ circumlimbal injection, grade 1+ diffuse injection and grade 1+ papillae OD, OS was clear. The right cornea had grade 3 diffuse corneal edema and haze, 4-5 keratic precipitates inferior on the corneal endothelium, the left cornea was clear. Anterior chamber of the right eye had grade 4 flare and debris and grade 2 cells, the left anterior chamber was deep and quiet. Examination of the right iris revealed small fine vessels running perpendicular to iris strands with an irregular course on top of the iris, most concentrated temporally but also present inferior, nasal and superiorly. Vessels did not extend into the angle or begin at the pupillary margin. A posterior synechiae from 3 to 10 o’clock was noted. The iris of the left eye was flat with no rubeiosis or iris vessels. IOP checked with goldman tonometry was 19 mmHg in the right eye and 12 mmHg in the left eye. Pupils were dilated, breaking the synechiae of the right iris, leaving behind a ring of pigment of the right anterior lens capsule. Posterior examination was negative for retinal vasculitis OU. In the right eye evidence of pneumatic retinopexy was apparent, laser scars superior temporal in the periphery extending into the mid periphery. No new holes, tears or detachments noted in either eye. The macula was flat and intact OU and the optic nerves were well perfused with normal margins. Gonioscopy was open to ciliary body 360 degrees OU with no neovascularization of the angle. Anterior segment photos of the iris vessels were also taken at that time. No lab/blood work was ordered during the first examination as this was the first occurrence of uveitis.

Differential diagnosis:

1. Acute iridocyclitis OD: this is the primary diagnoses as this patient exhibits almost all the signs and symptoms of anterior uveitis.
2. Acute angle closure glaucoma: can present as an acute red eye with associated pain and blurred vision. This does not fit as it is often transient and can be associated with nausea and headache.

Discussion

This patient was diagnosed with acute iridocyclitis in the right eye and apparent iris vessels also in the right eye. Uveitis describes swelling of the uvea. This case exemplifies an example of anterior uveitis which involves inflammation of the iris and ciliary body. Acute anterior uveitis presents with sudden onset, unilateral pain, photophobia, and redness. Signs include miosis due to iris sphincter spasm and possible posterior synechiae. The corneal endothelium is often dusted with pigmented cells. Keratic precipitates can appear after a few days into the disease progression. A reaction in the anterior chamber, including cells and flare, indicate disease activity and severity. Grading of anterior chamber cells and flare is used to track the effect of treatment and the change in inflammation. The presence of flare reflects the presence of protein that results from a breakdown of the blood aqueous barrier. In severe cases of inflammation a hypopyon can result in which cells settle in the inferior part of the anterior chamber. A posterior synechiae can occur quickly and occurs because the iris becomes “sticky” from inflammation. A low IOP results from a reduction in aqueous humor production by the ciliary body. A dilated fundus exam must be completed to check for signs of retinal vasculitis and vitreous cells. Acute uveitis typically lasts for 4-6 weeks with appropriate treatment and management. Chronic uveitis differs with a few key presentations, such as the presence of large mutton fat keratic precipitates, which have a greasy appearance. Dilated iris vessels that run radially may be apparent. Iris nodules typically occur in granulomatous disease and can be either keoppe or busacca nodules. Keoppe nodules are small and sit on the pupillary border. Busacca nodules are in the mid peripheral pupil. Iris atrophy is characteristic for uveitis resulting from herpes simplex and herpes zoster infections. Chronic uveitis can last for months to years. Cases of chronic uveitis often go through multiple cases of remissions and exacerbations. Systemic work up and further investigation is not indicated in cases that are mild, unilateral or are the first incidence of inflammation. If a systemic diagnosis known to be associated with uveitis such as sarcoidosis is present, no further work up is needed. In cases of uveitis that have granulomatous inflammation, are recurrent or bilateral a systemic work up is needed in order to uncover the underlying cause of inflammation. In this particular case no signs of chronic uveitis were apparent and no systemic work up was ordered right away. The most common etiologies of anterior uveitis include injury or trauma to the eye, an autoimmune condition including sarcoidosis or ankylosing spondylitis, an inflammatory disorder including crohns or ulcerative colitis, an infection such as herpes zoster or simplex, syphilis, lyme disease or tuberculosis or in rare cases lymphoma. It is important to identify the underlying etiology of anterior uveitis as the underlying causes can be life threatening or debilitating and should be managed appropriately. The interesting finding in this case is the presence of neovascular-like iris vessels. The iris vessels were dilated tortuous vessels that ran perpendicular to the radial iris fibers. Along with the treatment of the eye inflammation the main goal of management of this patient is to determine the etiology of the irregular iris vessels. A small scale study, Neovascularization of the iris in rhegmatogenous retinal detachment, aimed to identify conditions associated with
neovascularization of the iris in rhegmatogenous retinal detachments. The disease courses were classified into three groups including: neovascularization of the iris without a history of vitreoretinal operation, neovascularization of the iris after unsuccessful vitreoretinal operation and neovascularization of the iris after surgical complications. The last classification is specifically pertinent in this case. As etiology of the iris vessels is attempted to be uncovered in this patient it is possible that the iris vessels arose from complications of the pneumatic retinopexy that the patient received almost a year ago from the date of examination. While the retina appears to be in tact on examination, it is possible that the stress of surgery led to the formation of iris vessels. Other etiologies that were explored were carotid artery stenosis resulting in lack of oxygen to the eye and subsequent iris neovascularization, given the patients systemic history of a stenosed carotid artery this is a plausible etiology. Both a retinal consult and carotid Doppler were ordered to further evaluate the etiology of the iris vessels. Both of these consults have been placed but have not yet been completed. Another question that this case brings to light is that are the iris vessels a result of chronic inflammation, that was subclinical until recently or are the leaking iris vessels the cause of the anterior uveitis.

A small study looked at vascular changes in the iris in patients with chronic uveitis all of varying etiologies. Along with clinical examination, a fluorescein angiography of the iris vessels was conducted. This study found that iris vessels seen in cases of chronic uveitis differ from vessels found in various vascular eye disease such as BRVO. Further studies need to be conducted looking at the pathophysiology of the iris vessels in chronic uveitis vs vessels arising from vascular eye disease.

**Treatment**

Treatment for anterior uveitis includes frequent dosing of a topical ophthalmic steroid and a strong cycloplegic drop to both paralyze the iris sphincter muscle which decreases patient pain but also the break any present posterior synechiae. The dosing of the steroid is largely dependent the severity of the initial presenting inflammation. This patient was placed on Prednisolone Acetate 1% Q1H OD only until follow up the next day along with cyclopentolate TID OD only. Frequent follow up is necessary to monitor the effect of treatment but also monitor the intraocular pressure. Once inflammation is shown to be improving the steroid can begin to be tapered. Tapering should be done with caution as a reversal of inflammation can recur quickly. This patient has responded well to treatment with a drastic decrease in inflammation. He still has an anterior chamber reaction of grade 1+ cells and was tapered to Pred Acetate 1% Q2H OD only. The iris vessels have since resolved even though the inflammation is still present.

**Conclusion**

This case is still awaiting the results of several tests in order to properly determine the etiology of both the uveitis and the apparent iris vessels. With both disorders being acute and new it is hard to say if the vessels arose from chronic inflammation or if the iris vessels arising from an unknown etiology are the cause of the increased inflammation. Chronic, recurrent,
granulomatous or severe cases of uveitis require further systemic testing in order to properly treat and manage the patient.

References: