Top 5 neuro signs never to ignore

By Shauna Berry, MD; Ama Sadaka, MD; and Andrew G. Lee, MD

Let’s examine case-based examples to emphasize the top five neuro-ophthalmic disorders that should not be overlooked. These include:

- Acute painful loss of vision in elderly due to giant cell arteritis
- Acute painful ophthalmoplegia due to orbital apex disease
- Acute painful bitemporal hemianopsia due to pituitary apoplexy
- Acute painful anisocoria with a small pupil due to a Horner syndrome
- Acute painful anisocoria with a large pupil due to a pupil involved third nerve palsy from aneurysm

Clinicians should be aware of the distinctive clinical and radiographic findings for each of these potentially vision- or life-threatening conditions.

**Acute painful vision loss in the elderly** A 75-year-old male presents with acute vision loss OD to the 20/200 level associated with new headaches. This presentation is classic for giant cell arteritis (GCA). GCA should be considered in any elderly patient with any acute neuro-ophthalmic complaint with or without pain.

The most common presenting symptoms of GCA, however, are intermittent or constant visual loss, new onset headaches, jaw claudication, and scalp tenderness.1-4

Hayreh et al reported 106 of 363 patients with biopsy-proven GCA. The odds of a positive biopsy were 9.0 times greater with jaw claudication ($P = 0.0001$), 3.4 times greater with neck pain ($P = 0.0085$), 2.0 times greater with an erythrocyte sedimentation rate (ESR) of 47 to 107 mm/hour ($P = 0.0454$), 3.2 times greater with C-reactive protein above 2.45 mg/dl ($P = 0.0208$), and 2.0 times greater for age 75 years or more ($P = 0.0105$).5

The treatment of choice for GCA is immediate high-dose systemic steroids; however the route is still controversial.6-8

Some authors have suggested high-dose IV steroids (e.g., methylprednisolone 1000 mg/day) for patients with visual loss or neurologic symptoms.3,6,9 In a retrospective study, the visual acuity of patients treated with high-dose IV steroids (1000 mg for three days) followed by oral steroids was significantly improved compared to those on oral steroids alone.6,5 No head-to-head IV vs. by mouth trial of steroids for GCA has performed.

A temporal artery biopsy (TAB) remains the gold standard for the diagnosis of GCA.6-6 Although a floridly positive or completely negative TAB can be relatively straightforward, it is important to always read the entire body of the ocular pathology report.

The finding of “no active lymphocytic infiltrate or no giant cells” can still be due to GCA because “healed or treated” GCA can produce findings. Focal disruption of the internal elastic lamina, areas of fibrosis, irregular intimal thickening, and lymphocytes in the adventitia may suggest healed arteritis.10 Immunohistochemistry stains for CD 68 positive macrophages might helpful in such cases to help establish diagnosis of “healed or treated” giant cell arteritis.

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1. **FIGURE 1** Sagittal T1 weighted, magnetic resonance imaging (MRI) of the brain without contrast shows a sellar and suprasellar mass in the sella turcica with a hemorrhagic central core compressing the optic chiasm.
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Top neuro signs

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2. Acute painful bitemporal hemianopsia A 22-year-old female presents at 4:45 p.m. on a Friday with a severe headache, 20/20 visual acuity OU but blurred vision, and a normal fundus exam. Visual field shows a bitemporal hemianopsia.

This is another uncommon but classic neuro-ophthalmic presentation of an emergent condition, pituitary apoplexy. Although typically the acute onset of severe headaches and painful vision loss (bitemporal hemianopsia) is common in apoplexy, some patients have less severe or absent headache, and visual loss may be unilateral or bilateral and may or may not be the classic bitemporal hemianopic visual field loss.

The incidence of pituitary adenoma is approximately seven per 100,000 per year. Typically, the slow growth of benign pituitary adenomas produces painless and progressive neuro-ophthalmic symptoms. In contrast, pituitary apoplexy often produces acute and painful visual loss.

Acute hemorrhage or infarct of the pre-existing pituitary adenoma leads to compression of the surrounding anatomical structures (e.g., cavernous sinus or optic chiasm) and thus can produce the clinical findings of acute, severe headache, vision loss, ophthalmoplegia, altered consciousness, and hypopituitarism. In one study of 62 patients with pituitary apoplexy, the average patient age was 51 years. The majority of patients (81 percent) had no previous history of pituitary tumor. The most common presenting symptom was headache (87 percent), followed by decreased vision (56 percent), visual field defects (34 percent), and cranial nerve palsy (45 percent).

The compression or infarction of the normal pituitary gland may prevent the release of hormones, leading to hypopituitarism. Most patients (73 percent) exhibit a deficiency of at least one hormone produced by the anterior; Panhypopituitarism, a deficiency of all anterior pituitary hormones, is a life-threatening endocrine emergency that may require urgent hormonal supplementation.

Emergent imaging with a non-contrast CT head and neurosurgical consultation is indicated in patients with acute compressive symptoms or diminished mental status in pituitary apoplexy. The hyperdense signal from hemorrhage in acute apoplexy may be difficult to differentiate from other hyperdense lesions in the pituitary region such as a meningioma, Rathke cleft cysts, craniopharyngioma, and aneurysms. Magnetic resonance (MR) scan with and without contrast is useful in differentiating these lesions.

Neuro-surgical intervention may be necessary in patients with acute neurological symptoms, including neuro-ophthalmic symptoms. These patients require long-term follow-up for both visual and endocrinologic sequelae after pituitary apoplexy.

3. Acute painful ophthalmoplegia A 45-year-old male with uncontrolled diabetes mellitus presents to the emergency room with acute painful ophthalmoplegia and diabetic ketoacidosis (DKA). Mucormycosis, most commonly cerebro-rhino-orbital mucormycosis, is a rare but aggressive fungal infection that can affect immunocompromised or metabolically compromised patients but especially patients in DKA.

Diabetes mellitus is a common risk factor; however, 20 percent have no identifiable cause. Other risk factors include immunosuppression, metabolic ketoacidosis, underlying neoplasm, acute renal failure, severe burns or trauma, and steroids.

Rapid recognition of disease and treatment initiation improves the survival rate. Early symptoms include sinus tenderness, headaches, and blood-tinged or purulent rhinorrhea. Rapid angioinvasion and tissue infarction may produce a black necrotic eschar over the infected area, but this is a late and negative prognostic finding. Painful ophthalmoplegia, chemosis, diminished acuity, and proptosis can be present with orbital invasion.

Rhinocerebral mucormycosis has a high mortality rate, and thus it is imperative to rapidly recognize and treat the infection.

Imaging with CT may be preferred over MRI because it is faster and provides better sinus and bone detail.

Treatment involves correcting underlying systemic findings, such as DKA along with early aggressive surgical debridement and antifungal therapy with amphotericin.
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tericin B or posaconazole.16 In a study by Vehreschild et al, surgery and concomitant antifungal treatment with amphotericin B had the highest survival rate (70 percent) among 929 mucormycosis cases.16

4 Acute painful anisocoria greater in the light A 65-year-old diabetic male presents with acute onset ophthalmoplegia and anisocoria. The pattern of ophthalmoplegia in a third nerve palsy involves the extraocular muscles mediating adduction, supraduction, and infrafraction as well as the levator muscle, ciliary muscle, and iris sphincter.3,17

The third nerve palsy can be partial or complete based on the extent of the lesion and the pupil may or may not be involved.17 In a third nerve palsy, the anisocoria is greater in the light and is characterized by a dilated and poorly reactive or non-reactive pupil on the ipsilateral side.

This anisocoria is key in differentiating between ischemic and compressive causes of complete third nerve palsy. The parasympathetic fibers travel along the superficial surface of the oculomotor nerve to innervate the ciliary muscle, making them more susceptible to a compressive injury vs. internally located motor fibers.17,18

The most worrisome compressive lesion causing a third nerve palsy is an aneurysm of the posterior communicating artery (PCOM).3,18 A neurologically isolated, painless, complete third nerve palsy without pupil involvement in a vasculopathic patient is generally at low risk for a compressive lesion and is more likely to be ischemic.3,18 However, a small percentage of PCOM aneurysms present with normal pupils initially, especially in partial third nerve palsies, and therefore neuroimaging and further work up may still be necessary.18

In an acute setting, a CT with CTA of the brain is the recommended first line imaging.3,19 A contrast CTA should be able to detect an aneurysm as small as 3 mm.20,21 If the CTA is negative, then the next line of testing is an MRI of brain and orbits with and without contrast and MRA.3,12

Despite the high combined sensitivities of CTA with MRI and MRA, standard catheter angiogram remains the gold standard depending on the index of suspicion for aneurysm.3

Prognosis of the third nerve palsy depends on the etiology. In cases of aneurysmal compression, improvement is generally seen following surgical or endovascular treatment. Third nerve palsies secondary to ischemia typically improve over four to 12 weeks.

5 Acute painful anisocoria greater in the dark A 35-year-old male presents with new onset headache and anisocoria after playing basketball. A right sided 2 mm ptosis, upside-down ptosis, and ipsilateral miosis was also present. The anisocoria was greater in the dark with a dilation lag of the pupil OD. The rest of the eye exam was normal.

Acute painful ptosis and miosis should be considered as an ipsilateral internal carotid dissection from damage to the oculosympathetic pathway producing a Horner syndrome (HS). Other causes for the HS include malignancy, stroke, and aneurysm.

Damage along any point of the oculo-sympathetic pathway can produce a HS. The first order neuron begins in the hypothalamus and descends posterolaterally in the brainstem to synapse in the cilio-spinal center of budge at the level of C8-T2. This second order neuron exits the spinal cord and travels over lung apex (Pancoast lung tumor) to synapse within the superior cervical ganglion at the bifurcation of the common carotid artery. The third order neuron ascends through the cavernous sinus via the adventitia of the internal carotid artery before traveling a short course on the abducens nerve and joining the first division of the trigeminal nerve where it travels through the superior orbital fissure to innervate Mullers muscle and iris dilator.3

Pharmacologic testing is often helpful in confirming the diagnosis, but neuroimaging is recommended for all patients with a suspected HS clinically.

Pain (e.g., headache, eye pain, face pain, or neck pain) is the most common presenting symptom of a Horner syndrome from a carotid artery dissection but may be absent or variable in severity. The incidence is approximately 2.6 per 100,000 in the United States, and it can occur spontaneously or secondary to trauma.3,22 Most patients experience a positive clinical outcome with resolution or recanalization in 80 percent.23

Apraclonidine is a direct acting, non-selective alpha-agonist (predominantly alpha-2 activity) commonly used in diagnosing the HS. As a result of denervation hypersensitivity after a HS, a positive apraclonidine test will reverse the anisocoria due to dilation from up-regulation of the post-synaptic alpha-1 effect in the eye with the HS and the normal alpha-2 effect in the fellow eye (which produces slight pupillary constriction). This test has a high sensitivity and specificity.3,22,24

In the acute ER setting, a CT-CTA of the head and neck to thoracic level (T2) is the preferred initial study, and we do not generally recommend waiting for confirmation.

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Eye banks create the cycle of giving
Donated ocular tissue journeys from recovery to transplant

By Diane M. Hollingsworth

The Human Tissue “box” (Figure 1) that shows up at your hospital or surgery center and its contents go through quite a journey, which we in the eye banking world call the Cycle of Giving, to reach your destination.

The history of corneal transplants began more than 100 years ago in 1905 when the first surgery was performed.\(^1\) The first eye bank was established in New York City in 1944.\(^2\) As more eye banks were founded throughout the country, they formed a strong network which lead to the creation of the Eye Bank Association of America (EBAA) in 1961, the Cornea Society in 1975, and the development of medical standards for all accredited eye banks in 1980.\(^2\)

In conjunction with the expanding presence and impact of eye banks, the Uniform Anatomical Gift Act (UAGA) was passed in 1968, establishing a regulatory framework for the donation of organs, tissues, and eyes in the United States.\(^2\) This act ensured compliance with a donor’s wishes upon his death to donate to science, medicine, and education. This legislation has gone through two revisions over the years and has been adopted in some form in the majority of states.\(^2\)

In addition, most states have implemented a donation symbol on their driver’s licenses which confirms an individual’s designation to be a part of her state’s registry as an eye, organ, and tissue donor. Today, most donor registrations have evolved to first-person authorizations, a legally binding agreement that ensures the donor’s wishes are carried out at the time of death.

The symbiotic advancements in the fields of eye banking and state donation policies have had a profound impact. As recently as 10 years ago, corneal transplant surgeons had to put patients on a wait list until tissue became available. Now, due to the increased number of registered donors and ocular tissue available, surgeries can be scheduled in advance. Corneal transplants have become the most successful and most common form of human transplant performed.\(^3\)

Someone in need
The Cycle of Giving begins with the need for ocular tissue.

It could be an individual with a progressive eye disease requiring a corneal transplant to preserve or restore his sight. It could be someone suffering from glaucoma who needs a piece of sclera for her glaucoma shunt. Or, it could be a scientist in need of human ocular tissue to take

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his research to the next level.

The reasons may vary but the necessity is always there (Figure 2).

**A charitable act**

Next in the Cycle of Giving is the selfless act of individuals who committed to being a donor upon their passing or a family’s willingness to authorize the donation on behalf of their loved one.

At the time of their passing, hospitals are required by Centers for Medicare and Medicaid Services (CMS) conditions of participation to report the death to their designated Organ Procurement Organization (OPO).

The OPO will triage the donor’s information to the eye bank contracted with the hospital. The state’s first-person authorization registry will then be checked to see if the decedent chose to be a donor. If so, the eye bank will contact the next of kin (NOK) to inform them of their loved one’s wishes and to ask a series of medical and social history questions. If the decedent is not on the registry, the NOK will need to provide authorization in order to move forward with donation.

The medical and social history screening obtained by the eye bank, as well as a review of the donor’s medical information and records, is used to uncover any potential contraindications for donation as deemed by the U.S. Food and Drug Administration (FDA). This process is critical to ensuring the safety of ocular tissue for transplant.

**Recovery of donated tissue**

As soon as the eye bank completes the screening and determines that the individual is a suitable ocular donor, a trained eye bank technician is dispatched to the hospital, coroner’s office, or funeral home to procure the ocular tissue (Figure 3). The recovery must be completed within 24 hours of the individual’s death, or sooner, depending on certain research protocols.

The technician is trained to recover corneas, whole eyes, or specific ocular tissues following medical standards designed to cause the least amount of damage to the tissue as well as to treat the donor with the utmost level of dignity. In addition to the ocular recovery, the technician will draw a blood sample for serological testing of infectious diseases.

**Lab evaluation**

Ocular tissue is brought back to the eye bank, allowing the tissue evaluation process to begin (Figure 4). Each tissue is rated using specific criteria established by the eye bank’s medical director. The endothelial cell density of each cornea is determined using a specular microscope. The health of the cells and cell morphology are also thoroughly evaluated by the eye bank technician during this process.

Next, the ocular tissue is examined under a slit lamp looking for clarity, evidence of prior surgeries, trauma, foreign bodies, infections, dystrophies, and recovery damage to ensure the cornea is in the appropriate condition for transplant.

Most eye banks also utilize optical coherence tomography (OCT) to measure corneal thickness to ensure the tissue is prepared to a surgeon’s specifications.

The physical evaluation of the ocular tissue, donor medical and ocular history, and serologic testing results are reviewed as a whole in order to determine the suitability of the tissue for transplant. This final step is the eligibility determination, performed by an eye bank technician specifically knowledgeable in FDA, EBAA, and medical standards for one last review before a tissue is offered to a surgeon.

**Distributing tissue**

For each tissue requested by a surgeon, an eye bank distribution staff offers tissue specifically to meet the needs of an individual patient.

The surgeon specification includes the type of tissue best suited for each patient. The majority of corneal transplants are full thickness (for penetrating keratoplasty [PK], or eye bank-prepared partial thickness (for Descemet’s stripping with endothelial keratoplasty [DSEK] and Descemet’s membrane endothelial keratoplasty [DMEK]).

In order to accomplish all that is needed to meet each surgeon’s preferences, eye banks work 24 hours a day, seven days a week. Tissue not only needs to be recovered within 24 hours, it must also be evaluated, prepared, and in a surgeon’s hands within just a few days. Regulations state that tissue expires in 14 days, but surgeons in the United States typically require a much quicker turnaround (Figure 4).
Eye banks

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Regs and quality

The FDA regulates all eye banks and completes unscheduled inspections to ensure recipient safety and to prevent the transmission of communicable disease. It is also important for all medical professionals to ensure that their local eye bank is accredited by the EBAA. The EBAA inspects each eye bank at least once every three years, though inspections may become more frequent if a location receives infractions.

Each eye bank is required to have a quality assurance program to ensure it is inspection ready at all times and is adhering to the procedures established by the eye bank’s medical director, FDA, and EBAA.

Transplant

The Cycle of Giving completes with a corneal transplant recipient of human ocular donor tissue. Current EBAA statistics show that eye banks provided nearly 80,000 corneas for transplant in 2015, a far cry from the few used back in 1944.24 In addition, a 2013 study revealed that the lifetime benefit of corneal transplants is an extraordinary $5.5 billion.5

Many donor families long to know the outcome of their loved one’s tissue. Eye banks encourage discussion with transplant patients about the gift they receive. The eye bank can then facilitate anonymous correspondence between donor families and recipients, share donor and recipient stories, and advocate for donation.

The following examples are excerpts from correspondence between a donor mom and a recipient.6

The donor mom wrote, “To know that my daughter’s corneas are helping you see the world more clearly has brought me much joy over the years.”

The cornea transplant recipient wrote back, “Not one day goes by that I don’t think about my donor and give thanks.”

Outcomes

An additional service most eye banks provide to surgical facilities is a Handling of Human Eye Tissue Program. A representative can come out to a hospital or surgery center to verify compliance with joint commission standards and provide education on the best practices for inspection of the tissue, tissue information sheets, tissue labels, understanding post-operative outcome forms, and adverse reaction forms.

The Cycle of Giving is complex but extremely successful in the United States. This success, however, is not met everywhere around the world and much needs to be done.

Today, only one in 70 corneas required to restore sight are available worldwide. About half of the world is without access to corneal transplants, and the total need worldwide is estimated at more than 12 million. In order to fulfill this need and serve the global community, eye banks are working diligently to help develop eye banking in other countries.7

In addition to helping to develop global eye banking practices, eye banks are involved in developing alternative therapies to eliminate corneal blindness. Approaches such as the use of a selective portion of the cornea to help several patients, gene therapy, autologous endothelial cells, and synthetic corneas are being researched.

Eye banks will be working alongside corneal surgeons and researchers for optimal patient outcomes both today and in the future.

References

How I found my mentors

Mentors build confidence, offer advice, and expand career horizons

By Tami L. Hagemeyer, ABOC, FNAO

I have been fortunate in my professional life to have two mentors who have given me direction and helped expand my optical career into an amazing instrument of communication. I am now able to connect with my peers, and appreciate the opportunity to speak at optical conferences.

My youngest child was preparing for college. I felt ready to embark on my life with just my husband and myself; with friends telling me it would become my “new normal,” I felt consoled.

My quiet professional life had become a little too quiet. I had worked in a comfortable private practice for many years, and was beginning to become a little complacent. I began to feel restless.

I thought it would pass; after all, I felt well respected and had earned the trust of my peers and patients. But it didn’t pass. Although I went through the motions of my day, I continued to suspect that there was more for me to do.

Finding an opportunity

I found myself looking for opportunities and felt limited by my abilities. At the time I thought because I had invested years to my optical career, I was too far gone to learn anything new. I felt my years of experience would hold me back; I may be incapable of becoming the professional I wanted to be.

It was at my lowest emotional point when I found an ad in the newspaper. A practice was looking for an optician; it was close to home and sounded perfect. At first I did not respond, but I began to work on my resume. With much courage, I gave my resume one last look and dropped it off.

After handing it to the team member behind the desk, I remember thinking about turning around and saying, "I’m sure I made a mistake, and I would like my resume back!" But I didn’t say it, and with both dread and excitement I held my head up, returned to my car, and drove to the local coffee shop.

New beginning

I ordered my coffee and sat there for what seemed like hours. I just sat there! My thoughts were muddled. I knew it was time for a change, but was it time for a change? After all, this was the next phase in my life; it was time to relax... right? What was I worried about, they probably won’t even call...right? I finished my coffee, reclaimed my jumbled thoughts, and drove home.

I was almost home when I received the call. I almost didn’t answer. The person on the other end said he had reviewed my resume and would love the opportunity to speak to me. Now this was serious! I would have to meet with him—my most dreaded thought was an interview. It had been more than 17 years since my last one.

As these thoughts began to whirl around my scattered brain, I calmly scheduled a time to meet. I did, however, give myself a few days in between to prepare for my (yikes!) interview.

On the day of the interview I felt strangely calm and a little apprehensive, but the feeling that I was about to embark on a new journey was beginning to pique my interest. When I arrived, I took some time to look at my surroundings. The office itself looked normal enough. It was a late winter afternoon and beginning to get dark. The office had a warm glow, and it felt homely and comfortable.

Meeting the doctor for the interview would prove to be just as comfortable. We hit it off and talked for more than an hour. I remember thinking that it was

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a good interview. I smiled as I drove home. My feeling of dread was gone, replaced by a sense of accomplishment.

After a few weeks, I was offered the job. This was an exciting time until I had the ridiculous thought that after more than 20 years of experience, I was about to become the “new girl.”

Adding something new
My transition into new employee status was painless. It wasn’t long before I felt like part of this amazing new team. My positivity when interacting with patients impressed our office manager, and soon I was asked to speak at our weekly office meeting.

Following the meeting, the doctor asked if I would be interested in speaking at eye conferences.

“What, speak in front of more than a few of my peers? No, thank you!” I replied.

He laughed and asked me to think about it. The thought sent shivers down my spine. I was sure I would make a complete fool of myself, so the subject was closed. Or so I thought.

That night, I asked my family their thoughts on the subject. To my surprise, they responded that I would be great at speaking, and I should reconsider.

With the help of the doctor, who had now become my new mentor, I developed a two-hour workshop on frame adjustment and repair.

The morning of my first conference, I arrived at the assigned room about 45 minutes early and I felt ready.

As time passed, I wondered why no one came into the room. Maybe no one signed up for my class. Then I got a text—the room had been changed, and I was in the wrong room!

I called my mentor. He ran to help me move. We ran into the correct room with two minutes to spare. In the end, I had an incredible class and received positive evaluations. It was an amazing experience.

During the same conference, I became acquainted with my second mentor. Attending his class, I found his speaking to be informative and engaging. I respected his thoughts, and I knew his support would be invaluable.

Following the class, I approached him. I asked for advice on building my new career. He responded with ideas that have helped him become a well-loved speaker. He encouraged me to remain in contact with him, and he has given me invaluable advice with numerous recommendations and referrals.

To say these two individuals have become mentors to me feels like an understatement. They have become my friends, counselors, and coaches.

Their guidance has helped to build my confidence. They have encouraged a continued growth for my pursuit in whatever I choose to do or wherever I may go with my optical career, which has become truly limitless.

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