ACUTE VISION LOSS

Page 10

Anisocoria 2

5 Malignant Otitis

Regional Blocks 8

4 Kawasaki’s

also available at tamingthesru.com
Vertebral Artery Dissection

Jessica Merriam, MD
University of Cincinnati R1

History of Present Illness

The patient is a 55 year old female with a past medical history of migraines who presented to the ED with a chief complaint of right-sided facial pressure. She reports that her right eye was gradually in onset and began with mild right-sided neck pain about two weeks prior. Shortly after the neck pain began, she developed right ear pressure with intermittent sharp pain along the right side of her neck. She was seen by her primary care provider for these complaints and was provided with antibiotics and steroids for a presumed ear infection. However, she experi-enced no relief from her symptoms. On the day of presentation, the patient noted that her pupils were unequal. She complains of a moderate “fullness” behind her right eye, but denies pain with range of motion of her eyes. She denies vision changes, double vision, changes in sensation, or weakness in her arms and legs. She reports no recent fevers and has had no increased tearting or redness in her eyes. She has had occasional migraines in the past but states that this is very different from those.

Past Medical History

Migraines

Past Surgical History

None

Medications

Cyclobenzaprine, Prednisone

Physical Exam

Ty6.8 HR 82 RR 15 BP 119/72 O2 Sat 95%

She has clear tympanic membranes bilater-ally and moist mucus membranes. Her neck reveals no cervical lymphadenopathy, no tenderness, and no carotid bruit. She has normal respiratory effort, normal breath sounds, and good air movement. Her cardi-ovascular exam reveals a regular rhythm without any murmurs, gallops, or rubs. Her abdomen is soft, non-distended, non-tend-er without guarding or rebound. She has no edema in her lower extremity and the remainder of her musculoskeletal exam is normal. Her skin is warm and dry without any rashes or lesions, with normal turgor and normal coloration. Her person, place, time, and situation. Her cran-ial nerves are intact with the exception of her anisocoria. She has good strength in all extremities, normal sensation, and normal movement of both limbs. Her cranial nerves are intact with the exception of her anisocoria. She has good strength in all extremities, normal sensation, and normal movement of both limbs.

Workup & Imaging

WBC 11.5 with 91% neutrophils. Normal electrolytes CRP 10.4 ESR 6

Normal CXR Normal non-contrast head CT

CT Angio Head and Neck: Short segment of prominent irregularity involving the distal right V3 segment concerning for dissection. No flow limiting stenosis or occlusion identified, specifically normal enhancement of the bilateral carotid arteries. Mild irregularity of the right vertebral artery as it courses intracranially may represent a small pseudoneuroanomaly.

Vertebral Artery Dissection

The patient was a non-toxic appearing female with prominent right neck pain and new-onset anisocoria. Because of this neurologic deficit, a CT angiogram was obtained in the ED and revealed a dissection of the distal vago-vascular exam reveals a regular rhythm without any murmurs, gallops, or rubs. Her abdomen is soft, non-distended, non-tender without guarding or rebound. She has no edema in her lower extremity and the remainder of her musculoskeletal exam is normal. Her skin is warm and dry without any rashes or lesions, with normal turgor and normal coloration. Her person, place, time, and situation. Her cranial nerves are intact with the exception of her anisocoria. She has good strength in all extremities, normal sensation, and normal movement of both limbs. Her cranial nerves are intact with the exception of her anisocoria. She has good strength in all extremities, normal sensation, and normal movement of both limbs.

Vertebral Artery Dissection

The patient was a non-toxic appearing female with prominent right neck pain and new-onset anisocoria. Because of this neurologic deficit, a CT angiogram was obtained in the ED and revealed a dissection of the distal side of the right vertebral artery. An MR angiogram confirmed these findings. Neurosurgery and neurointerventional radiology were consulted. She was started on aspirin and discharged home two days after admission. She under-went an outpatient cerebral angiogram which revealed findings consistent with fibromuscu-lar displaasia. Going forward, patient will be treated with aspirin indefinitely and will need re-evaluation with an MR angiogram every six months for the next five years. The patient was discharged with an improved anisocoria. She continues to have mild anisocoria but no other neurologic defi-cits.

Discussion

Neck pain is a fairly common emergency room complaint for which there are count-less etiologies, from the benign to the life-threatening. Vertebral artery dissection is a diagnosis on the more dangerous end of that spectrum. It involves a false lumen within a vessel and is often initiated by small intimal tear in the vessel wall. The most feared complications of vertebral artery dissection are posterior circulation stroke and vessel rupture leading to subarachnoid hemorrhage. The incidence of vertebral artery dissection is 1.5 per 100,000 and affects primarily people ages 35 to 50%. Frequently, these patients are otherwise healthy with no significant risk fac-tors for cerebrovascular disease.

The most frequent initial complaint in vertebral dissection is posteriortel neck pain. This is often progressive over a period of days or weeks to months and may cause any neurologic symptoms. Non-specific head-aches are common as well. The development of neurologic symptoms is often what leads to the diagnosis. Posterior circulation symp-toms such as vertigo, nausea and vomiting, diplopia, ataxia, and dysarthria are common. Occasionally, vertebral artery dissection can lead to specific neurologic syndromes such as lateral medullary syndrome with findings of Horner’s, facial numbness, and contralateral limb anesthesia. Many patients will report a recent history of blunt or even minor neck trauma prior to the development of symp-toms. However, spontaneous dissections are also possible, particularly among patients with connective tissue disorders such as fibromuscular dysplasia, Ehlers-Danlos, and Marfan syndrome.

Regardless of the inciting event, once there has been a disruption in the intima, blood under arterial pressure can enter between the layers of the vessel wall and result in an intramural hematoma. This irregularity in the vessel wall can lead to a thrombosis event and subsequent embolus to the brain. In addition, the hematoma can expand enough that it can stenose or occlude the vessel resulting in cerebral ischemia. Finally, if the intramural collection of blood expands into the adventitia, the weakest part of the vessel wall, aneurysmal dilation or vessel rupture can occur. Dissections are most commonly found in the extracranial segment of the vessel as these areas are prone to the transverse foramen of the cervical bodies. However, intracranial dissec-tions carry a poorer prognosis as the intracranial arteries have a thinner adventitia and are more prone to bleeding.

Dissections are diagnosed with vascular imaging studies such as CTA or MRA, any of which may reveal a “string sign” where vessel filling appears to taper into a thin string. Conventional angiography is occasionally used to further characterize the extent and appearance of the lesion if no obvious cause of the dissection is identifiable.

Once diagnosed, treatment of vertebral dissections varies depending on patient char-acteristics, clinical presentation, and local practice patterns. Antiplaquet agents such as aspirin or Plavix are preferred over oral anticoagulants in patients who are at increased risk of bleeding. These patients include those with large intestinal or mass effect, NIH stroke scale ≥5, or those with intracranial exten-sion of the dissection. Oral anticoagulants are preferred in patients with higher risk for thromboembolic disease such as those with significant vessel stenosis or occlusion of the dissected vessel, those with a known true thrombus near the dissection site, or patients with multiple ischemic events in the same distribution.

Similar to the management of acute isch-emic stroke, patients who present within three to 4.5 hours of symptom onset, have no intracranial extension of their dissec-tion, and have none of the classic exclusion criteria for tPA may be eligible for systemic anithrombotic therapy. Likewise, those who present within six to 12 hours and have significant neurologic compromise may be considered for intra-arterial therapy. This is often used in patients who present with basilar artery occlusion because of its high mortality if untreated. Endovascular treatment such as stenting and angioplasty is another treatment modality.

Patients with significant stenosis or occlusion at the dissection site or those with associated aneurysm formation are often considered for endovascular repair. Additionally, patients with a history of recurrent TIA’s despite adequate therapy may also benefit from this procedure.

Ultimately, emergency department man-agement of vertebral dissections centers on making the diagnosis and determining if the patient is a candidate for tPA. Having a high index of suspicion and obtaining the appropriate imaging in patients with a clini-cal presentation concerning for dissection is crucial. Involving consultants such as neuro-interventional radiology and neurosurgery will help guide initial treatment and tming. In the acute period, treatment of verteb-real dissection generally not indicated, and allowing for cerebral autoregulation is preferred. If there is no specific blood pressure with which hypertension must be treated in the acute setting, yet gentle blood pressure lower-ing may be warranted when the systolic is greater than 200. These patients will require admission and telemetry monitoring in the acute period and repeat imaging over the next several years to re-evaluate the affected vessel. Overall, if managed appropriately, this disease process carries a good prognosis with a mortality rate of less than 5% and good functional recovery in 75% of patients.

Persistent Pyrexia:
When the fever won’t quit

#communitycase

Kari Gorder, MD
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History of Present Illness
The patient is an otherwise healthy, fully immunized Indian-American 4-year-old female who presents with fever and rash. Per her father, the patient developed a fever approximately seven days ago. She has had daily, spiking fevers ranging from 101-103°F despite alternating use of acetaminophen and ibuprofen. Approximately three days ago, she developed bilateral conjunctivitis. She was tested by her pediatrician that day for strep throat, which was negative. At that time, her pediatrician did note a middle ear effusion and started the patient on amoxicillin. Yesterday, the patient began to develop a rash on her hands, arms, and feet. Her father also reports poor PO intake over the past few days and decreased urine output in the last 24 hours. The patient has been increasingly fussy and tearful over this time period as well. No nausea or vomiting.
Her father also endorses some intermittent rhinorrhea, nasal congestion and eye discharge.

Past Medical History
Social History

Full immunized
Attends daycare. No sick contacts.
No recent travel.

Physical Exam

T101.8 HR 135 RR 24 BP 97/60 O2 Sat 97%

Exam reveals an uncomfortable appearing, tearful and tired child. HEENT exam is notable for dry mucous membranes and some oropharyngeal erythema. Tongue and lips appear slightly red and cracked. There is pronounced bilateral limbic sparing conjunctivitis with a scant amount of yellow discharge. No photophobia. There is diffuse anterior cervical lymphadenopathy with the largest node measuring >1.5 cm. Cardiopulmonary exam is unremarkable. Skin exam reveals a fine, papular, erythematous rash involving the dorsal hands and feet. No desquamation noted. There is some mild fever is not relieving the patient.

Diagnostic Criteria for Kawasaki Disease

≥ 5 DAYS OF FEVER WITHOUT OTHER EXPLANATION AND FOUR OF THE FIVE CRITERIA BELOW:

1. BILATERAL CONJUNCTIVAL INFECTION
2. ORAL MUCOSA MUCUS CHANGES
   - Strawberry tongue
   - Injectionary papillae
   - Fissured, injected, dry, or cracked lips
3. PERIPHERAL EXTREMITY CHANGES
   - Erythema or edema of the hands or feet
4. POLYMORPHOUS TRUNCAL ERYTHEMATOUS RASH
5. CERVICAL LYMPHADENOPATHY

Discussion

Kawasaki Disease is an acute, systemic vasculitis of the medium-sized arteries that is primarily found in children between 6 months and 4 years of age. It is particularly prevalent in male children and those of Asian descent. First described in Japan in the 1960s, it is the leading cause of acquired heart disease in developed nations, and is second only to rheumatic fever in developing countries. Individuals most at increased risk for Kawasaki Disease are those with Down syndrome, premature infants, and children from Asian countries.

The patient in this case report presents with fever, conjunctivitis, and a rash on the hands and feet, consistent with Kawasaki Disease. The patient also has anterior cervical lymphadenopathy, which is a common finding in Kawasaki Disease. The patient’s fever and rash have been present for approximately seven days, which is consistent with the diagnosis of Kawasaki Disease.

The patient’s fever and rash have not responded to Tylenol or ibuprofen, and the patient continues to feel unwell. A decision is made to start the patient on intravenous immunoglobulin, which is a standard treatment for Kawasaki Disease.

Not Just an Ear Infection:
Malignant Otitis Externa

Isaac Shaw, MD
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History of Present Illness
The patient is a 50-year-old female with a past medical history of hypertension, hyperlipidemia, and diabetes who presents with a chief complaint of right ear discharge. The discharge has been present for three days, and she describes it as purulent drainage out of her right ear canal. She visited her primary care doctor as an outpatient, who prescribed her Ciprofloxacin but has not alleviated her symptoms. Over the last three days her discharge has become more copious and is now associated with mild right otalgia and vertigo. In addition, she endorses one day of nausea with vomiting.

Physical Exam

T97.4 HR 60 RR 24 BP 108/59 O2 Sat 96%

Exam reveals a female patient who is alert and in no acute distress. The patient’s head is nontender and without edema. The right ear exam reveals a purulent discharge in the external auditory canal. The canal is diffusely erythematous with a mild amount of granulation tissue. The tympanic membrane cannot be visualized due to extensive edema and granulation tissue.

Discussions

Malignant otitis externa, also known as necrotizing otitis externa, is an aggressive infection of the external auditory canal with adjacent skull base osteomyelitis. Populations most susceptible to this infection include the elderly, with diabetes, and those with immunosuppressive conditions such as HIV.

The patient’s fever is not resolving with antibiotics. She is referred to Otolaryngology (ENT) for evaluation and treatment. Malignant otitis externa is a more serious infection than an ordinary ear infection.

Diagnostic Criteria for Kawasaki Disease

≥ 5 DAYS OF FEVER WITHOUT OTHER EXPLANATION AND FOUR OF THE FIVE CRITERIA BELOW:

1. BILATERAL CONJUNCTIVAL INFECTION
2. ORAL MUCOSA MUCUS CHANGES
   - Strawberry tongue
   - Injectionary papillae
   - Fissured, injected, dry, or cracked lips
3. PERIPHERAL EXTREMITY CHANGES
   - Erythema or edema of the hands or feet
4. POLYMORPHOUS TRUNCAL ERYTHEMATOUS RASH
5. CERVICAL LYMPHADENOPATHY

Discussion

Malignant otitis externa, also known as necrotizing otitis externa, is an aggressive infection of the external auditory canal with adjacent skull base osteomyelitis. Populations most susceptible to this infection include the elderly, with diabetes, and those with immunosuppressive conditions such as HIV.

The vast majority of these infections are monomicrobial infections of Pseudomonas aeruginosa, but they can also be due to other microorganisms such as Staphylococcus aureus, Proteus mirabilis, Candida species, and Aspergillus species. Risk factors for development of malignant otitis externa are similar to those for classic otitis externa.

These include ear instrumentation such as recent ear surgery and hearing aid use, and ear irrigation.

Diagnosis of malignant otitis externa is most commonly made on physical exam.
Imagine it's your first moonlighting shift as a 4 year resident at a small rural community hospital. The nearest referral center for both adults and children is 90-minutes away by ground. The annual census of the emergency department is 15,000 patients per year, of which only 5% is pediatric. There are 2 hours left in your 12-hour shift and your energy is all but spent. You are looking forward to winding down at home after an extremely busy and high-acuity shift when your 35th patient of the day checks in. The patient's chief complaint is fever. You give yourself an internal fist pump thinking that you're about to see your 12th viral URI of the day and that you'll be in-and-out of that room no time in. In the midst of your premature celebration you scan the nursing note and see the age of the patient: 6 weeks... You're hopes of a quick and easy disposition suddenly melt away leaving you with many more questions regarding this patient's care than answers…

“Yikes!” you tell yourself and you rush out of the room. And as you make your way back to your work station a flood of questions surrounding management of the febrile infant hit you all at once…

Luckily you have made several close contacts at Cincinnati Children’s Hospital Medical Center ED over the course of your residency training and you decide to phone-a-friend. Joining us on this edition of Annals of B Pod is Dr. Brad Sobolewski, a pediatric emergency medicine attending and creator of PEMBlog, and Dr. Adam Yukovic, a clinical fellow in Pediatric Emergency Medicine, to help us answer some of the critical questions surrounding management of the febrile infant.

AOBP: If everything comes back normal, what is the most appropriate disposition for this patient population?

A pediatrician reviewing the patient's work-up notes that UTI was the most common source of SBI (prevalence 15-94%) whereas bacteremia was less common (prevalence 0-4%) and meningitis even more rare (prevalence 0-26%). This infant is ill, but non-toxic and represents a clinical and diagnostic dilemma. A slightly red tympanic membrane in the setting of a crying, febrile infant is not representative of acute otitis media. The current evidence supports multiple approaches – but only if the infant is well appearing. Doing “nothing” isn’t an option – but in general, WELL appearing babies can get blood and urine studies alone.

AOBP: Should you decide to do an LP, it will undoubtedly take a while to perform as your ED is full and your nurses and techs are already stretched thin. When, if ever, should empiric antibiotics be started? What antibiotics should be given?

CCHMC: Empiric antibiotics can be delayed until studies are obtained in the mildly ill or well appearing febrile neonate or infant. The antibiotic choices are:

- Age
- Antibiotics:

<table>
<thead>
<tr>
<th>Age</th>
<th>Antibiotics:</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-21 days</td>
<td>Ampicillin/Cefotaxime/Acyclovir*</td>
</tr>
<tr>
<td>22-28 days</td>
<td>Ampicillin/Cefotaxime</td>
</tr>
<tr>
<td>29-56 days</td>
<td>Cefotaxime or Ceftriaxone</td>
</tr>
</tbody>
</table>

*Note that HSV is highly unlikely outside of the first 3 weeks of age – unless that is the baby has seized or has an abnormal neurologic exam.

AOBP: Does this child need a lumbar puncture?

CCHMC: A study by Paquette et al. in 2011 looked retrospectively at 392 infants between 30 and 90 days with abnormal TUTI, finding that only 4 ultimately had meningitis (1%). That being said, none of them were “well-appearing” on presentation nor met “low-risk” criteria. In 2010, Mintegi et al. retrospectively reviewed 665 cases of SBI evaluation in infants < 3 months of age. In their study, the incidence of bacterial meningitis was 0.3%, all of which were identified by a combination of laboratory data and clinical appearance. Four infants (0.6%) who did not initially have an LP were ultimately diagnosed with aseptic meningitis, and all four did well.

AOBP: You have no identifiable source of infection. The different sources of otitis media, to UTI, to early pneumonia, to bacteremia, to meningitis. What is the incidence of SBI in this patient population? What labs, if any, are indicated in this patient population?

CCHMC: In a large meta-analysis of infants less than three months of age with fever without a source (>$54,000 patients) published in 2012 by Hui et al., the authors found the prevalence of SIB was 0.3% in this age group.

AOBP: If Everything comes back normal, what is the most appropriate disposition for the patient population?

CCHMC: Our patient was still "low risk" following completion of our work up, but there is still a decision to make. For this specific child, if you obtained blood and urine and low-risk criteria were met, plus a negative chest x-ray if ordered then you can consider discharge home. The patient should be consolable and have an improved HR with defervescent. They should also be able to feed. It is your job as the Emergency Department physician to ask the following questions:

- What are the family's concerns?
- Will they be able to follow up tomorrow?
- Is it a holiday or weekend? Is the Primary Care Provider's office even open?
- Are they PCP comfortable with them going home?
- Would the parents be more comfortable in the hospital? On antibiotics (L.P. of course)?

You can admit the child without antibiotic regardless of whether or not the LP was performed. Again, any infant that gets antibiotics in this age range should be admitted 100% of the time.

Check out the rest of the interview with Drs. Sobolewski & Yukovic and a list of references for this topic at tamingthebursa.com/annals-of-bpod.
**Regional Anesthesia**

**Face - Mouth - Wrist - Ankle**

Aalap Shah MD
Nicholas Ludmer MD
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**Superficial Peroneal**

Inject 3-5cc superficially lateral between EHL and lateral malleolus.

**Median Nerve**

Radial to PL

1cm ulnar to FCR.

Tip: you should feel a ‘pop’ through the flexor retinaculum at ~1cm.

**Ulnar Nerve**

Radial to FCU

Tips: initially aim and block distally towards pisiform, then aim and block laterally towards tendon.

**Radial Nerve**

Radial to radial artery.

Tips: create a superficial wheel around thumbs to catch superficial branches.

**Saphenous**

Create a wheel between medial malleolus and AT tendon.

Tip: classification will help reveal the AT tendon.

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**Table 1: UCOC Local Anesthesia**

<table>
<thead>
<tr>
<th>Location</th>
<th>Dose</th>
<th>% in Local anesthetic</th>
<th>Maximum dose</th>
<th>Available at UCOC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Submental Nerve</td>
<td>1-3</td>
<td>60-90</td>
<td>0.03-0.3%</td>
<td>0.5-1.5%</td>
</tr>
<tr>
<td>Supraorbital Nerve</td>
<td>5-10</td>
<td>50-90</td>
<td>0.03-0.5%</td>
<td>0.5-1.5%</td>
</tr>
<tr>
<td>Infraorbital Nerve</td>
<td>1-3</td>
<td>60-90</td>
<td>0.03-0.6%</td>
<td>0.5-1.5%</td>
</tr>
<tr>
<td>Palmar cutaneous nerve of forearm</td>
<td>10-20</td>
<td>50-90</td>
<td>0.03-0.6%</td>
<td>0.5-1.5%</td>
</tr>
</tbody>
</table>

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**References**


Davis B. What dose of epinephrine contained in local anesthesia can be safely administered to a patient with underlying cardiac disease during a dental procedure? Journal of Canadian Dental Association. 2010;76:a36.


**History of Present Illness**

The patient is a 74-year-old African-American female with a history of hypertension, coronary artery disease status post drug-eluting stent, and iron deficiency anemia presenting with left-sided vision loss. Patient states that approximately two days ago she woke up with painless peripheral vision loss of her left eye only. She describes it as darkness in the lateral portion of her left eye. She reports that her vision returned to baseline throughout the day, however, she had visual deficits again when she awoke the next morning. Since that time she endorses persistent vision loss in the left periphery. She denies blurry vision, eye pain, headaches, recent trauma, flashes, and floaters. Furthermore, she also denies dizziness, numbness, weakness, dysarthria, dysphagia, fever, chills, nausea, vomiting, chest pain, shortness of breath, and palpitations. She reports adherence to her antihypertensive and anti-platelet medications.

**Medications**

- Aspirin
- Atorvastatin
- Chlorthalidone
- Clopidogrel
- Metoprolol
- Torsapril
- Pantoprazole

**Social History**

Former 21 pack-year smoking history (quit 20+ years ago)

**Past Medical History**

Hypertension, NSTEMI

**Medications**

- Description of past drug eluding stent, Osteoarthritis

**Physical Exam**

T36.4 HR 74 RR 16 BP 167/109 O2 Sat 96%

The patient's exam revealed a well appearing, elderly African-American female who appeared alert, oriented and in no acute distress. Neurological exam revealed intact extraocular movements and her pupils were equal and reactive bilaterally. Strength and sensation was intact in all extremities. There was no cerebellar dysmetria or truncal ataxia. National Institute of Health Stroke Scale (NIHSS) was 1. Her pulmonary exam was unremarkable. Her cardiovascular exam revealed a regular rhythm, normal S1/S2 without murmur or gallops and she had 2+ peripheral pulse bilaterally.

**Workup**

EKG: normal sinus, unchanged from prior. Glucose 104. INR 1.2

**CT Head**

Age-independently infarction involving the paraseptal right occipital lobe. CTA Head & Neck: Diminished enhancement of distal cortical branches of the right posterior cerebral artery in the right parieto-occipital region corresponding to the region of suspected infarct on noncontrast CT.

**Hospital Course**

**Neurology** was consulted in the Emergency Department. Given the patient's symptom duration of >48 hours, the decision was made not to give tissue plasminogen activator (t-PA). The patient was admitted to the neurology service and underwent MRI of the brain (Image 1) that showed an acute ischemic infarct in the right occipital region, with adjacent edema without mass effect. The patient had no associated deficits. MRI is the gold standard for characterization of stroke and the diffusion weighted imaging (DWI) image confirmed the presence of acute ischemia. The etiology of her stroke was thought to be from thromboembolic phenomena based on her diffuse ischemic disease on imaging. Given her history of NSTEMI, neurology considered a cardiac source of the embolus. As such, the patient underwent a cardiac MRI that revealed no evidence of mural thrombus or vegetation. Throughout her hospital stay she had no improvement in her vision. She was, however, able to ambulate without problems. Occupational therapy evaluated the patient and recommended assistance at home and outpatient therapy for visual training. She was ultimately discharged home on hospital day two with continuation of all her home medications and instructions to follow up with neurology as an outpatient.

**Discussion**

The differential diagnosis for acute-onset vision loss is broad. However, there are several key features from the history that can be useful for the Emergency Physician in determining initial workup and management. Specifically, the patient experienced acute, painless, "unilateral" vision loss. The most common pathologies causing this presentation are listed in Table 1. Neuro-ophthalmologic vision loss is defined as vision loss that is not readily explained by an abnormality on physical examination of the eye for which a cause distal to the retina is suspected. The lesion can be located anywhere from the optic nerve to the cortical vision loss. Most common pathologies causing this presentation

**Table 1: Causes of Acute, Painless, Unilateral Vision Loss**

<table>
<thead>
<tr>
<th>Type of Vision Loss</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retinal detachment</td>
<td>Retinitis pigmentosa, diabetic retinopathy</td>
</tr>
<tr>
<td>Retinal vein occlusion</td>
<td>Central retinal artery occlusion</td>
</tr>
<tr>
<td>Retinal Hereditary</td>
<td>Neuro-ophthalmologic vision loss</td>
</tr>
</tbody>
</table>

An accurate history and directed physical exam is key to the ED workup of acute-onset vision loss. Confrontational visual field testing, or Donders Test, can be performed in seconds as part of the bedside exam. The examiner should assume a position directly across from the patient at arm's length, so that their eyes align on the same horizontal and vertical plane. The patient should cover their right eye with their right hand and vice versa when testing the opposite eye. With the examiner seated directly across from the patient, the patient should direct their gaze to the corresponding eye of the examiner. Starting outside the usual 180° visual field, the examiner should move the hand slowly to a more central position until the patient correctly identifies the target. Once visualization is confirmed, use this as the starting point for stationary testing. To perform stationary testing, the examiner should hold up a stationary target. The patient should correctly identify the number of fingers. The visual fields of both eyes overlap, therefore, each eye should be tested independently and all four quadrants (superior, inferior, left, right) should be tested.

In the above case, while the patient only reported painless, unilateral vision loss such as retinal ischemia, retinal or vitreous detachments, these pathologies may be ruled out by an echocardiogram as an inpatient. Cardiac MRI is the gold standard for characterization of cardiac sources of embolism, although it has never been proven to be superior to echo for detection of vegetation, and should only be utilized when there is still high suspicion for cardiac source and echocardiography is nondiagnostic. Thromboembolism from large or small vessel atherosclerotic disease is thought to be responsible for approximately 45% of ischemic stroke and is best visualized by CTA of the neck and/or carotid ultrasound.
Pulmonary arterial hypertension (PAH) is a rare disease of the pulmonary vasculature. PAH is diagnosed utilizing a right heart catheterization and is defined as a mean pulmonary artery pressure (mPAP) of more than 25 mmHg at rest. The prevalence of PAH is estimated to be 15-20 cases per million individuals, with approximately 1,000 new cases reported in the United States annually. PAH is characterized by dyspnea on exertion, chest pain, dizziness, or syncope, and is often associated with right ventricular failure. There are multiple proposed mechanisms behind the pathophysiology of pulmonary hypertension. Two major proposed mechanisms include decrease in prostacyclin production and increase in thromboxane A2 production, these changes result in decreased vasodilation and increased vasoconstriction, respectively, which inevitably culminates in right-sided heart failure.

One of the many classes of medications used in the treatment of pulmonary hypertension is prostacyclins. Prostacyclins analogues exert their mechanism of action by eliciting a vasodilatory effect on the pulmonary vasculature, their mechanism of action by elicting a vasoactive prostanoid, prostacyclin. Prostacyclin analogues exert a dosing regimen for treatment of PAH patients. Overall, the meta-analysis found epoprostenol to be the most recommended prostacyclin for use in the treatment of pulmonary hypertension based on improved exercise capacity, functional capacity, and mortality.

Parenteral Prostacyclins

The two parenteral prostacyclins available are treprostinil (Remodulin®) and epoprostenol (Flolan®, Veinlet®). These may be ordered as a continuation of patients’ home therapy or initiated in patients who meet clinical criteria and have completed the following prior to initiation: confirmation of third party payer source approval, completion of specialty pharmacy enrollment, and identification of pharmacy technicians. Inhaled epoprostenol is restricted to use in cardiac surgery patients only. Both inhaled prostacyclins require specialized devices for use. Patients presenting to the emergency department with symptomatic pulmonary hypertension should be assessed and treated immediately. Patients who present to the ED with established prostacyclin therapy should be continued on these therapies so as to avoid complications. It is especially important to evaluate the medication regimen when the patient presents to the ED due to the short half-life of prostacyclin therapy. If the patient has enough drug supply in the pump to continue until evaluated by the pulmonary hypertension team, patients who present with a malfunctioning pump or an occlusion in their catheter or line should have an inhaled dose of prostacyclin administered as soon as possible. Abrupt discontinuation of therapy may result in rebound pulmonary hypertension due to increased pulmonary artery pressure and vascular resistance, acute right ventricular failure, or death. Moreover, patients may become refractory to prostacyclin therapy and treatment options become limited. Pharmacists in the emergency department and on-call clinical pharmacists are available to assist with a variety of tasks in order to ensure optimal patient care, including, but not limited to, calculating or verifying doses, ordering the medication, and compounding the medication.

Inhaled Prostacyclins

There are two inhaled prostacyclin products available: treprostinil (Tyvaso®) and epoprostenol (Flolan®). Inhaled treprostinil is not formulated. Inhaled epoprostenol is restricted to use in cardiac surgery patients only. Both inhaled prostacyclins require specialized devices for use. Patients presenting to the emergency department with symptomatic pulmonary hypertension should be assessed and treated immediately. Patients who present to the ED with established prostacyclin therapy should be continued on these therapies so as to avoid complications. It is especially important to evaluate the medication regimen when the patient presents to the ED due to the short half-life of prostacyclin therapy. If the patient has enough drug supply in the pump to continue until evaluated by the pulmonary hypertension team, patients who present with a malfunctioning pump or an occlusion in their catheter or line should have an inhaled dose of prostacyclin administered as soon as possible. Abrupt discontinuation of therapy may result in rebound pulmonary hypertension due to increased pulmonary artery pressure and vascular resistance, acute right ventricular failure, or death. Moreover, patients may become refractory to prostacyclin therapy and treatment options become limited. Pharmacists in the emergency department and on-call clinical pharmacists are available to assist with a variety of tasks in order to ensure optimal patient care, including, but not limited to, calculating or verifying doses, ordering the medication, and compounding the medication.

Painful Vision Loss

Continued from page 11

The administration of intravenous (IV) tissue plasminogen activator (tPA) is dependent on the time of presentation, the contraindications present, and the degree of neurologic deficits. Most practitioners would not administer tPA for isolated quadrantanopia in our patient even if within the 3-hour window; however, a thorough discussion of the risks/benefits with the patient and family should be had. If the patient is outside of the IV-tPA window, but less than 6 hours from symptom onset, there should be consideration of intracarotid (IA) tPA administration if a larger vessel occlusion is found. The AHA/ASA recommends IA-tPA in select patients with MCA occlusion within 6-hour of symptom onset but no recommendation is made for IA-tPA in posterior circulation stroke, including basal artery occlusion. The American College of Physicians recommendations, however, indicated no role for IA-tPA in patients with an ischemic stroke caused by a proximal large artery occlusion in the anterior circulation are candidates for mechanical thrombectomy if present after strokes in the anterior circulation territories. Additionally, although the benefits are uncertain and evidence is lacking, the use of stent retrievers may be reasonable for carefully selected patients with acute ischemic stroke who have occlusion of vertebrobasilar arterial territory, or other brainstem or cerebellar territories.” Our patient was unfortunately outside of the window for any of these acute interventions and the anatomical location of her lesion would not have been amenable to thrombectomy regardless. She will most likely have persistent visual field deficits as evidenced by her neuromonitoring.

In regards to long-term treatment, patients with a history of non-cardioembolic ischemic stroke should continue aspirin, clopidogrel, or aspirin/extended release dipyridamole. Additional contributing comorbidities such as diabetes mellitus and tobacco use should also be managed medically to reduce the risk of future CVA.

References


A. To start, the needle is inserted at the level of the superficial fascia and exits at the dermal-epidermal junction.

B. The needle is then re-armed with the driver and inserted at the dermal-epidermal junction on the contralateral side and exited at the level of the superficial fascia.

C. Crucial to this process is that the leading and trailing segments of the suture remain on the same side of the loop.

D. Using 3 or 4 throws, the knot is tied and buried at the level of the superficial fascia. The knot is cut leaving only 2mm “tails.”
History of Present Illness

The patient is a woman in her 60’s with a past medical history of diabetes, hypertension, and stroke, who presents with lightheadedness and confusion. The patient’s initial troponin was >30 ng/mL in the Emergency Department.

Patient Outcome

The patient was taken emergently from the ED to the cath lab where the patient was found to have an occluded left circumflex artery. The interventional cardiologist was able to place a drug eluting stent in the area of stenosis and restore distal blood flow.

Posterior STEMI (PMI)

PMI is the most commonly missed type of STEMI and when correctly identified only 30% meet door-to-balloon time. Isolated PMIs are difficult to identify due to EKG similarities with anteroseptal ischemia. The occurrence of isolated PMIs is unknown, but is thought to be <10% of all STEMIs.

EKGs and Case referred by

Drs. Dang and Otten
University of Cincinnati


Where can you find a PMI in an EKG?

In the anterior precordial leads, V1-V3!

ST segment depression

Prominent R waves

Upright T waves

Leads V1-V3 are an electrical mirror of the posterior leads V7-V9. ST depression in V1-V3 can represent ST elevation in leads V7-V9.

Prominent R waves in leads V1-V3 reflect Q waves in the posterior leads V7-V9. This occurs because V1-V3 are an electrical mirror of V7-V9.

Upright T waves in leads V1-V3 and ST depression in the same leads can help to differentiate a PMI from a septal MI or anteroseptal ischemia.

Posterior EKG Lead Placement

Lead placement for posterior leads V7-V9 along the posterior chest wall. V7 is placed at the left posterior axillary line, V8 is placed in the left mid scapular line, V9 is placed at the left paraspinal line.

ST segment elevation (≥1 mm) in the posterior leads, V7-V9, concerning for a PMI.

List of Submitted B Pod Cases

Case

- Dysfibrinemia with SMA thrombosis
- HIV & syphilis
- Giant cell arteritis w/ visual symptoms
- Spontaneous vertebral artery dissection
- Hermericrania Continua
- Brugada Type 3
- Familial Hypertriglyceridemia
- Malignant Otits Externa
- Occipital Infarct

Case Physicians

- Doerning
- Hamilton/Betz
- Beyde/Niziolek
- Merriam/Betz
- Denney/Adeoye
- March/Bonomo
- Whitford/Ostro
- Shaw/LaFollette
- Harrison/LaFollette

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