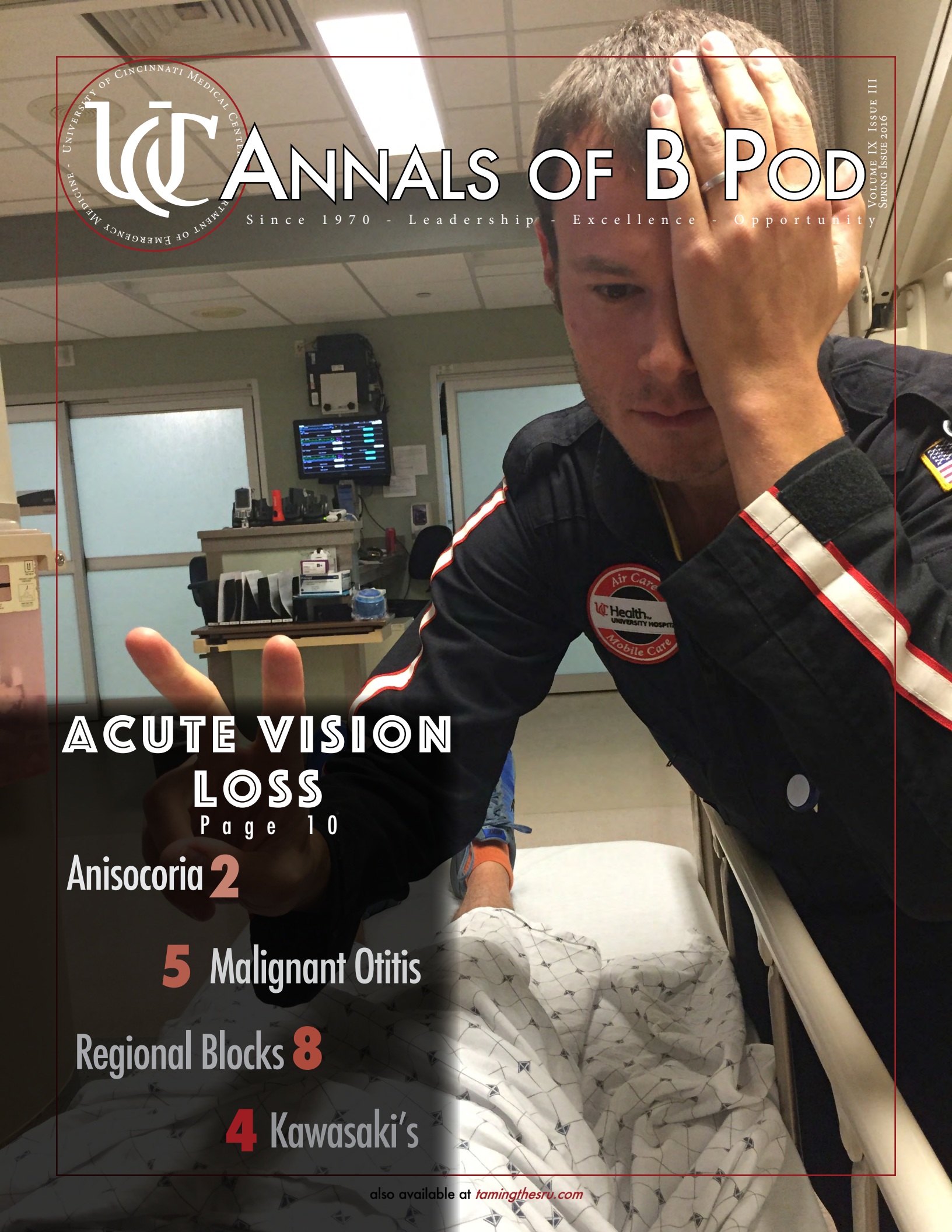

Annals of B Pod

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Volume IX Issue III
Spring Issue 2016

ACUTE VISION LOSS

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also available at *tamingthesru.com*

EMERGENCY MEDICINE
ANNALS OF B POD
SPRING 2016

#fillintheedges

Spring is here and new beginnings are right around the corner. Step-ups are looming and we will find out the names of our new interns any day. It is time for interns to polish their efficancy, for the R2s to finish their off-service rotations in anticipation of the great variety of the SRU, the R3s to finishing refining their ability to run an effective team. The R4s #fillintheedges of their careers as residents so they can go forth as prepared attendings. In anticipation of these new beginnings, this issue of Annals of B-Pod focuses on cases that #fillintheedges.

The progression through residency can mirror patient interactions. Our paitents presentations often have blurry edges, sometimes literally, as in our acute vision loss case. This issue of AoBP provides great examples of how everytime we #fillintheedges of a paitent's story, we also #fillintheedges of our ever growing skill set as emergency medicine physicians.

#bpodcase

Anisocoria:

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 symptoms were gradual 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 experienced no relief from her symptoms. On the day of presentation, the patient notes 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 tearing 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	Past Surgical History	Medications
Migraines	None	Cyclobenzaprine, Prednisone

Physical Exam

T98.6 HR 82 RR 15 BP 139/79 O2 Sat 95%

The patient is an alert, well-developed, well-appearing female in no acute distress. Her eye exam reveals normal conjunctiva with non-icteric sclera. Her extra-ocular movements are intact. Her right pupil is 5mm and reactive to 4mm, her left pupil is 3mm reactive to 2mm. She has no photophobia. Her head is atraumatic with mild tenderness to palpation over the right temporal region. She has clear tempanic membranes bilaterally and moist mucus membranes. Her neck exam reveals no cervical lymphadenopathy, no tenderness, and no carotid bruit. She has normal respiratory effort, normal breath sounds, and good air movement. Her cardiovascular 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 extremities and the remainder of her muskuloskeletal exam is normal. Her skin is warm and dry without rashes or nodules. She is alert, oriented to person, place, time, and situation. Her cranial nerves are intact with the exception of her anisocoria. She has good strength in her bilateral upper and lower extremities. She has normal finger-to-nose and heel-to-shin tests bilaterally. Her gait is normal.

Work-up & 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 ophthalmic arteries. Mild irregularity of the right vertebral artery as it courses intracranially may represent a small pseudoaneurysm.

Hospital Course

This patient was a non-toxic appearing female with progressive 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 segment of V3 with a small pseudoaneurysm 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 underwent an outpatient cerebral angiogram which revealed findings consistent with fibromuscular dysplasia. 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 for monitoring of the dissection. She continues to have mild anisocoria but no other neurologic deficits.

Discussion

Neck pain is a fairly common emergency room complaint for which there are countless etiologies, from the benign to the life-threatening. Vertebral artery dissection is a diagnosis on the more dangerous end of that spectrum. It involves creation of 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[1]. Frequently, these patients are otherwise healthy with no significant risk factors for cerebrovascular disease.

The most frequent initial complaint in vertebral dissection is posterolateral neck pain. This is often progressive over a period of days to several weeks and often precedes any neurologic symptoms. Non-specific headaches are common as well. The development of neurologic symptoms is often what leads to the diagnosis. Posterior circulation symptoms 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 symptoms. 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 lumen is a nidus for thrombus formation and subsequent emboli 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 it leaves the transverse foramen of the cervical bodies[2]. However, intracranial dissections carry a poorer prognosis as the intracranial arteries have a thinner adventitia and are more prone to bleeding[3].

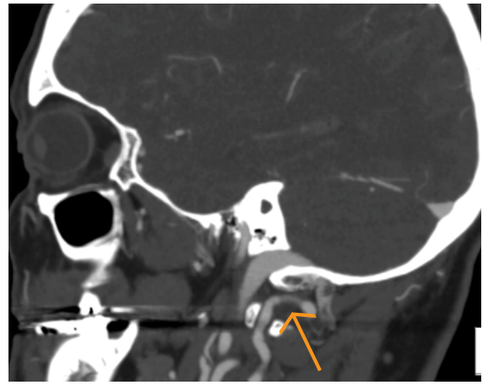

Image 1: CT angio of the Head: Arrow points to an area of vertebral artery dissection at the V3 segment

Dissections are diagnosed with vascular imaging studies such as CTA or MRA which may reveal a "string sign," where vessel filling appears to taper into a thin string[1]. 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 characteristics, clinical presentation, and local practice patterns. Antiplatelet 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 infarct or mass effect, NIH stroke scale >15, or those with intracranial extension 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 free thrombus near the dissection site, or patients with multiple ischemic events in the same distribution.

Similar to the management of acute ischemic stroke, patients who present within three to 4.5 hours of symptom onset, have no intracranial extension of their dissection, and have none of the classic exclusion criteria for tPA may be eligible for systemic antithrombotic therapy. Likewise, those who present within six to 12 hours and have significant neurologic compromise may be considered for intra-arterial thrombolysis[4]. This is often used in patients who present with basilar artery occlusion because of its high morbidity and 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 TIAs despite adequate therapy may also benefit from this procedure.

Ultimately, emergency department management 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 clinical presentation concerning for dissection is crucial. Involving consultants such as neurointerventional radiology and neurosurgery will help guide initial treatment and timing. In the acute period, treatment of hypertension in patients with vertebral dissection is generally not indicated, and allowing for cerebral autoregulation is preferred[5]. There is no specific blood pressure beyond which hypertension must be treated in the acute setting, yet gentle blood pressure lowering 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.

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3. Galtes I., Borondo J., Cos M., et al. Traumatic vertebral artery dissection. Foren Sci Inter. 2012; 214: e12-e15.
4. Redekop G. Extracranial carotid and vertebral artery dissection: A review. Can J Neurol Sci. 2008; 35:146-152.
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Persistent Pyrexia:

When the fever won't quit

Kari Gorder, MD
University of Cincinnati R2

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

Fully immunized

Social History

Attends daycare. No sick contacts. No recent travel.

Physical Exam

T101.8 HR 135 RR 24 BP 87/60 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 aspect of the bilateral hands and feet with some extension onto the arms and legs. No desquamation noted. There is some mild swelling of the hands and feet.

Lab Work-up

13.1 / 11.2 × 253 / 38.2

ESR: 57 CRP: 17 LFTs: normal
UA: >50 WBCs, negative nitrites

Hospital Course

This patient presented with one week of fevers, conjunctivitis, 1ymphadenopathy, rash, and irritability. Her urine did reveal >50 WBCs; however, her urine culture was ultimately negative. Her constellation of symptoms was concerning for Kawasaki Disease (KD). She was started on IVIG and high dose aspirin with significant improvement in her symptoms and persistent fever. An EKG and echocardiogram were both normal. She was discharged on low-dose aspirin. A follow-up echocardiogram revealed no coronary artery involvement, so her aspirin was stopped, and she continues to do very well.

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. Like most autoimmune disorders, its etiology is unknown, although multiple theories for its pathogenesis exist. The general consensus is that there is one or more infectious agents which trigger a pro-inflammatory host response in genetically susceptible individuals, which may explain the viral syndromes often seen in KD patients prior to their diagnosis. There is some geographic and seasonal variance, and in the United States it is seen primarily in the late winter and early spring. If KD goes untreated, it results in coronary artery aneurysms in up to 25% of children. As such, timely identification and treatment is of the utmost importance for these patients.

KD is primarily a clinical diagnosis that requires a high index of suspicion for the emergency medicine physician. The symptoms are a direct result of the widespread vascular involvement. Diagnostic criteria include a fever lasting five or more days that is poorly responsive to Tylenol or ibuprofen and the presence of at least four of the following: bilateral, nonpurulent conjunctivitis that is most often limbic-sparing; mucosal inflammation, most classically of the lips and tongue ("strawberry tongue"); a diffuse, polymorphous rash; painful swelling of the hands and feet; and non-painful cervical lymphadenopathy, with at least one lymph node equal to or greater than 1.5 cm in diameter. These symptoms can

Continued on page 15

Diagnostic Criteria for Kawasaki Disease

≥ 5 DAYS OF FEVER without other explanation
AND four of the five criteria below:

1. Bilateral conjunctival injection
2. Oral mucous membrane changes
   - Strawberry tongue
   - Injected pharynx
   - Fissured, injected, dry, or cracked lips
3. Peripheral extremity changes
   - Erythema or edema of the hands or feet (acute phase)
   - Periungual desquamation (convalescent phase)
4. Polymorphous truncal erythematous rash
5. Cervical lymphadenopathy

Not Just an Ear Infection:

Malignant Otitis Externa

Isaac Shaw, MD
University of Cincinnati R1

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 last three weeks, 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 Ciprodex, but it 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 non-bloody non-bilious emesis and tactile fevers. She denies any previous history of similar symptoms, and has not had ear infections since childhood. She does not wear hearing-aids nor does she regularly instrument or irrigate her ears.

Past Medical History

Hypertension, Diabetes Mellitus Type II, Hyperlipidemia

Medications

Hydrochlorothiazide, Insulin glargine, Insulin lispro
Atorvastatin

Physical Exam

T97.4 HR 60 RR 16 BP 108/59 O2 Sat 96%

Exam reveals a female patient who is alert and in no acute distress. The patient's head is normocephalic and without trauma. 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 secondary to extensive soft tissue edema of the canal. There is mild tenderness to the pre-auricular area and with movement of the tragus. Decreased hearing in the right ear compared to left. The patient is awake, alert, and oriented to person, place, time, and situation. The patient has subjectively decreased sensation along the right upper and lower face, but otherwise CNII-XII intact. Normal gait. Sensation intact. Strength is equal and symmetric in the upper and lower extremities. Normal finger-nose-finger, normal alternating hand movements. The patient's cardiopulmonary, abdominal, extremity, and skin exams are all normal.

Lab Work-up

139 | 107 | 13 / 3.9 | 26 | 0.84 × 206

ESR: 30
CRP: 2.4
HgbA1C: 11.6

13.7 / 4.8 × 234 / 41

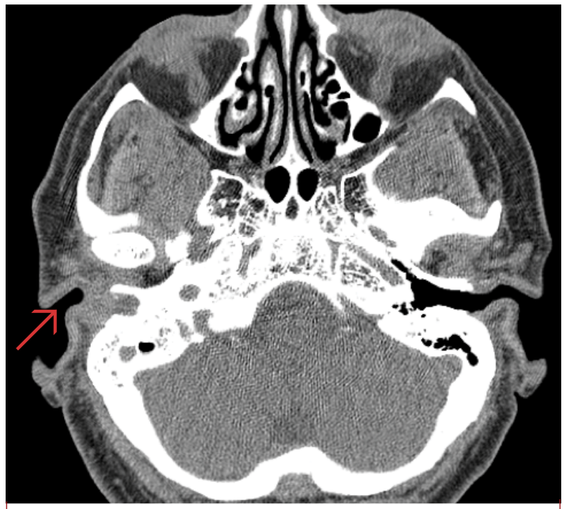


Figure 1: CT Orbit-Sella-IAC Impression: Occlusion of the external auditory canal of the right by soft tissue density. There is associated swelling of the cartilaginous portion of the external auditory canal. The process appears chronic with remodeling of the bony external auditory canal and minimal if any suspected erosive changes.

Hospital Course

The patient's exam and CT findings (Figure 1) were most consistent with malignant otitis externa. She was started on IV vancomycin and cefepime and admitted to medicine after consultation with Otolaryngology (ENT). As an inpatient, an MRI was obtained (Figure 2) which re-demonstrated soft tissue thickening of the right external auditory canal and adjacent skull base osteomyelitis of the right mastoid. ENT performed debridement of the R external auditory canal. The tissue biopsies showed no evidence of malignancy. Following debridement ENT was able to evaluate the tympanic membrane and made the diagnosis of suppurative labyrinthitis. Tympanostomy tubes were then placed with subsequent resolution of her vertigo. Per infectious disease recommendations a PICC line was placed and she was dischaged on a 6-week course of antibiotics. During her hospitalization, she was placed on a new insulin regimen and given diabetes education as she was found to have uncontrolled diabetes secondary to medication noncompliance.

Discussion

Malignant otitis externa, also known as necrotizing external otitis, is an aggressive infection of the external auditory canal with adjacent skull base osteomyelitis. Populations most susceptible to this infection include the elderly, patients with diabetes, and those with immunosuppressive conditions such as HIV.[1]

The vast majority of these infections are monomicrobial infections of Pseudomonas aeruginosa, but they can also be due other microorganisms such as Staphylococcus aureus, Proteus mirabilis, Candida species, and Aspergillus species.[2] Risk factors for development of malignant otitis externa are similar those for classic otitis externa. These include ear instrumentation such as recent ear surgery and hearing aid use, and ear irrigation.[3]

Diagnosis of malignant otitis externa is most commonly made on physical exam,

Continued on page 15

#pediatrics

Annals of B Pod + PEMBlog: The Febrile Infant

Benjamin Ostro, MD
University of Cincinnati R4

Imagine it's your first moonlighting shift as a 4th 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 in time. 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… You muster your remaining energy and make your way toward the patient's room.

The patient is an otherwise healthy ex-full term 6-week old male. The mother denies any prenatal or neonatal complications. Patient has been in a good state of health until yesterday evening when he became irritable, febrile, and refused oral intake. These symptoms have persisted throughout the last 24 hours with a Tmax of 103. When asked about associated symptoms the mother endorses decreased urine output and periods of decreased activity. She denies cough, rash, cyanosis, decreased body tone, diarrhea, bloody stool, foul-smelling urine, and seizures. The patient has not yet received his 2 month shots. There has been no recent travel and no known sick contacts.

Physical Exam

T 102.5 HR 180 BP 70/45 RR 45 O2 Sat 97%

The infant is intermittently irritable but consolable. He does not interact or make eye contact with the care provider and only intermittently with the mother and is appropriately sized for his age. His fontaneles are flat, he has mild erythema of both tympanic membranes, mild clear rhinorrhea, normal conjunctiva, a normal oropharyx and pupils are equal, round, and reactive to light. He has non-labored breathing without retractions, faint wheezing at the right lung base but his lungs are otherwise clear to auscultation. His cardiac exam reveals a regular tachycardia without any murmurs, rubs, or gallops. His abdomen is soft and non-tender without any masses or hepatosplenomegaly. His extremities are well perfused, he has brisk cap refill. His skin, neck, and neurological exam are all normal.

As you finish your assessment the mother, who has tears welling in her eyes, asks you "is he going to be okay? You want to tell the mother that everything is fine, but at the same time you know that your assessment wasn't 100% reassuring. Although your suspicion is low, you cannot rule out a serious bacterial infection (SBI) at this point. As you sit next to the mother carefully contemplating what to say, your phone rings. "Thank God!", 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 regarding 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 Vukovic, a clinical fellow in Pediatric Emergency Medicine, to help us answer some of the critical questions surrounding management of the febrile infant.

AOBP: You have no identifiable source of infection. The differential ranges from 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 SBI to be 9.4%. Notably, they found that those less than two weeks had the highest prevalence of SBI (25%), while those less than one month (13%) and between one and three months (7.1%) had less prevalence of disease. In this study, they found that UTI was the most common source of SBI (prevalence 15-94%) whereas bacteremia was less common (prevalence 0-41%) and meningitis even more rare (prevalence 0-26%).

Infants at Low Risk for Meningitis

Presenting Features:	Work-up results:
29 to 56-60 days old	WBC ≥ 5,000 and ≤ 15,000
Full-term (≥37 weeks gestation)	Band:Neutrophil < 0.2 (Bands/bands + neutrophils)
No prolonged NICU stay	Urine WBC < 10 /HPF
No chronic medical problems	Negative urine Gram stain
No systemic antibiotics within 72 hours	Chest x-ray (if obtained) without infiltrate
Well-appearing and easily consolable	
No infections on exam	

Table 1: Features and work-up results of infants who are at low risk of meningitis

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: Does this child need a lumbar puncture?

CCHMC: This is a question that is dependent on your overall plan for the child. In Baker's study (Philadelphia, 1993), 1.2% of patients had bacterial meningitis. However, etiologies of meningitis in these cases included Haemophilus influenza b and Streptococcus pneumonia, both of which have become increasingly less notable players in invasive bacterial disease given the overall success of vaccine initiatives. More recently, bacterial meningitis is being cited at a prevalence of 0.3% in this age group. See Table 1 for features of infants at low risk for meningtis.

A study by Paquette et al. in 2011 looked retrospectively at 392 infants between 30 and 90 days with abnormal urinalyses, 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 685 cases of SBI evaluation in infants < 3 mos 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: 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:
0-21 days#	Ampicillin/Cefotaxime/ Acyclovir*
22-28 days	Ampicillin/Cefotaxime
29-56 days	Cefotaxime or Ceftriaxone

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

#gentamicin is potentially ototoxic and has become less commonly used – though you could substitute for cefotaxime in the infant <14 days.

	Rochester	Boston	Philadelphia
Age	<90 days	28-89 days	29-56 days
Temp	Not Specified	38 C	38.2 C
History/Physical Exam	Term No perinatal complications Not hospitalized longer than mom No history of underlying disease No previous antibiotic use No ear, soft tissue, joint or bone infection	No allergies to B-lactams No antibiotics in last 48h No ear, soft tissue, joint or bone infection	Presumed to be immunocompetent
CBC	5000-15000	<20,000	<15,000
Band	<1500		B:N <0.2:1
Urine Analysis	<10 WBC per HPF	<10 WBC per HPF or neg for LE	<10 WBC and few/no bacteria
CSF		< 10 x 10^6 cells/L	< 8 in non-bloody
CXR			no infiltrate

Table 2: Low risk criteria broken down by original study

Only start empiric antibiotics on the aforementioned patient if you get an LP. ceftriaxone or cefotaxime would suffice. If there is pleocytosis on the CSF or a positive gram stain consider adding vancomycin for Staph coverage and acyclovir for HSV. If you are worried about HSV send a CSF PCR along with hepatic profile (may show transaminitis which is a clue to invasive HSV disease) along with swabs of skin, mucous membrane and eyes for PCR. Get an enterovirus PCR if it is the right season (August-October).

AOBP: If everything comes back normal, what is the most appropriate disposition if there is still concern for SBI? Discharge with close PCP follow up? Admit and wait on culture results?

CCHMC: If our patient was still "low risk" following completion of our work up, then 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 xray if ordered then you can consider discharge home. The patient should be consolable and have an improved HR with defervescence. 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?
- Is their PCP comfortable with them going home?
- Would the parents be more comfortable in the hospital? On antibiotics (after LP, of course)?

You can admit the child without antibiotics 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 & Vukovic and a list of references for this topic at tamingthesru.com/annals-of-bpod.

Regional Anesthesia

Face - Mouth - Wrist - Ankle

Aalap Shah MD
Nicholas Ludmer MD
University of Cincinnati R2

Supraorbital Nerve

for forehead lacerations

palpate supraorbital notch above pupil
insert laterally and inject when over notch

Tip: place pressure from below to prevent anesthetic from entering eyelid

Infraorbital Nerve

for unilateral anterior dental pain or facial lacerations

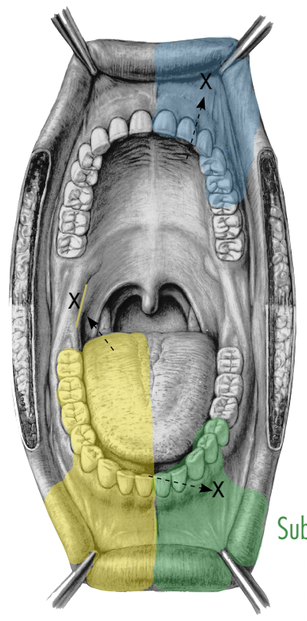
palpate infraorbital notch parallel with incisor aim towards finger until you reach the notch and inject 3cc

Tip: you should be able to palpate the needle under the surface near the notch

Inferior Alveolar Nerve

for unilateral mandibular dental pain

place thumb on mandibular ramus
inject 2cc 1/4 between raphe and thumb



Submental Nerve

inject 2cc around mental foramen beneath the 2nd bicuspid

Table 1: UCMC Local Anesthetics[1,7,8,9]

Anesthetic	Class	Onset (min)	Duration (min)	Maximum dose	Available at UCMC	Clinical pearls
Lidocaine	Amide	1 - 5	30-90	4 - 5 mg/kg (max total dose 300 mg)	1%, 2% PF 0.5%, 1%, 2%, or 4%	Most commonly used. May be combined with bupivacaine to create a short onset and extended duration (pay close attention to max dose when combining, additive effects).
Lidocaine + epinephrine		1 - 5	60-180	5 - 7 mg/kg (max total dose 500 mg)	1:100,000 as 1% or 2% 1:200,000 as 0.5%, 1%, 1.5%, or 2%	
Bupivacaine	Amide	5 -10	3-8 H	2 mg/kg (max total dose 175 mg)	0.125%, 0.25%, 0.75% PF 0.25%, 0.5% 0.75%	Epinephrine does not extend duration, however does decrease risk of toxicity by local vasoconstriction
Bupivacaine + epinephrine		5 - 10	3-8	3 mg/kg (max total dose 225 mg)	1:200,000 as 0.25%, 0.5%, and 0.75%	
Mepivicaine	Amide	1 - 5	30-120	4-5 mg/kg (max total dose 300 mg)	PF 1%	Very similar onset and duration as lidocaine
Tetracaine	Ester	10	8H	1 mg/kg	1%	

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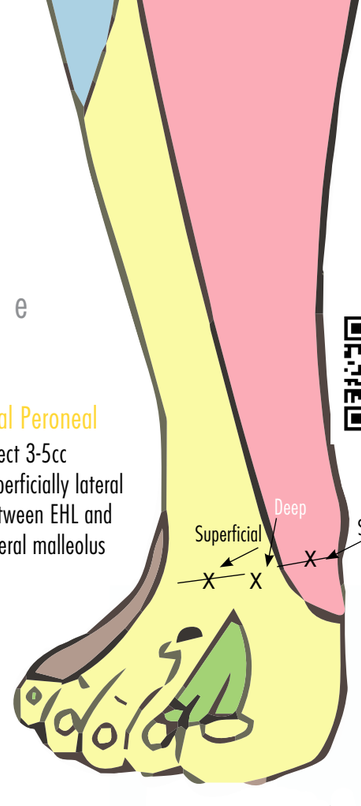
Ankle

5 blocks 3 deep, 2 superficial a lot of over-lapping areas make blocking adjacent areas or the whole ankle best



Saphenous

create a wheel between medial malleolus and AT tendon

Tip: dorsiflexion will help reveal the AT tendon

Sural

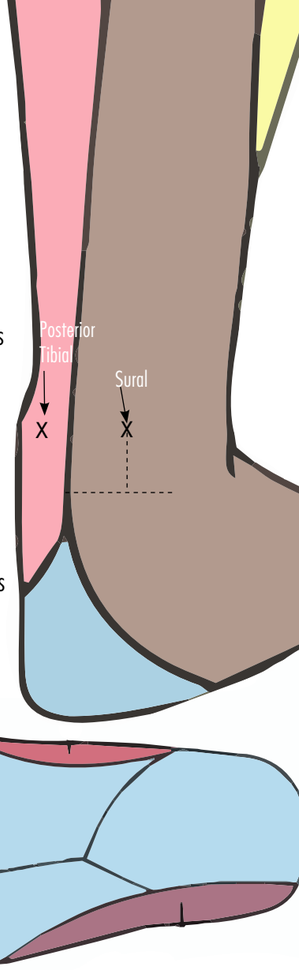
1cm superior of midpoint of achilles and lateral malleolus
inject 3-5cc superficially

Superficial Peroneal

inject 3-5cc superficially lateral between EHL and lateral malleolus

Posterior Tibial

1cm superior to midpoint of achilles and medial malleolus posterior to posterior tibial artery
inject 3-5cc at 1cm deep

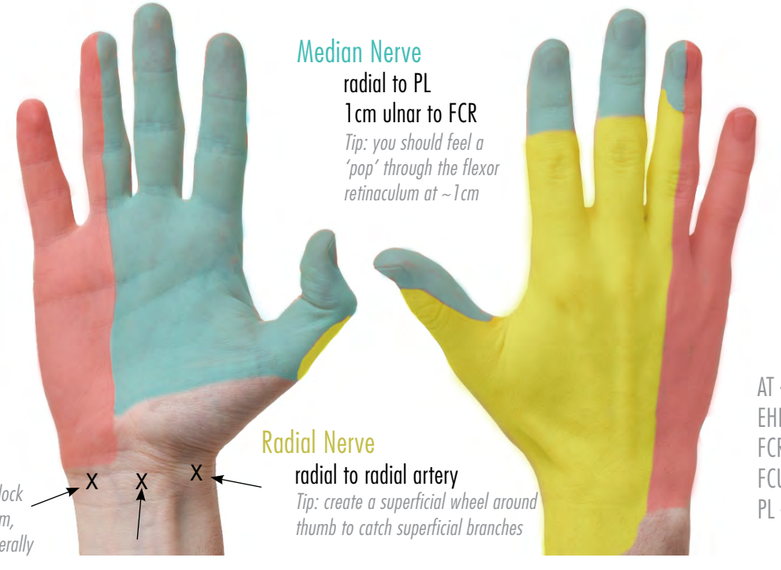


Deep Peroneal

inject 1-3cc 1cm deep between EHL and AT tendons

Wrist

3 blocks, each with 3-5 cc anesthetic proximal to the wrist crease



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

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

Radial Nerve

radial to radial artery

Tip: create a superficial wheel around thumb to catch superficial branches

Key

AT - Anterior Tibialis
EHL - Extensor Hallicus Longus
FCR - Flexor Carpi Radialis
FCU - Flexor Carpi Ulnaris
PL - Palmaris Longus

#bpodcase

Vision Loss: Thinking about the *other* left lower quadrant

Collins Harrison, MD
University of Cincinnati R1

History of Present Illness

The patient is a 74 year-old African-American female with 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.

Past Medical History

Hypertension, NSTEMI status post drug eluding stent, Osteoarthritis

Medications

Aspirin, Atorvastatin, Chlorthalidone, Clopidigrel, Metoprolol, Benazepril, Pantoprazole

Social History

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

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. Neurologic exam revealed intact extraocular movements and her pupils were equal and reactive bilaterally. Confrontational visual field exam was remarkable for bilateral left lower quadrantanopia. Visual acuity was 20/40 bilaterally. Cranial nerves III - XII were intact. She ambulated with a cane and gait was at baseline. 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 pulses bilaterally.

Work-up

EKG: normal sinus, unchanged from prior, Glucose 104, INR 1.2
CT Head: Age-indeterminate infarction involving the parasagittal 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.

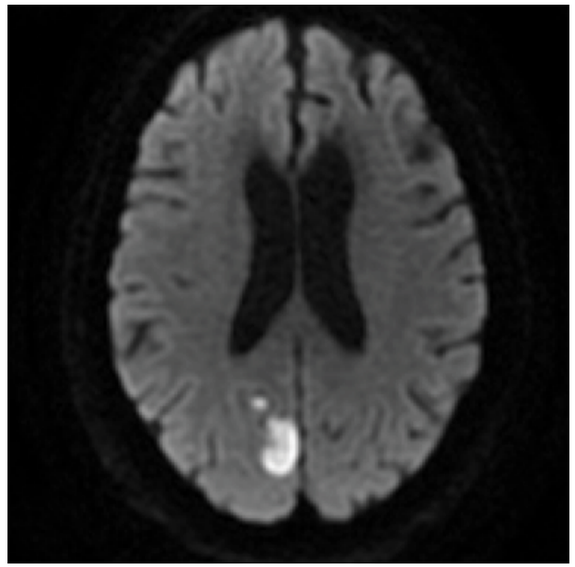


Image 1: MRI of the brain w/o contrast: Acute ischemic infarct in the right occipital region (DWI).

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 (tPA). The patient was admitted to the neurology service and underwent MRI of the brain (Image 1) that showed an acute ischemic infract in the right occipital regions, moderate to severe chronic microvascular disease and remote right cerebellar and left basal ganglia infarcts. 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 emboli. As such, the patient underwent a cardiac MRI that revealed no evidence of mural thrombus or vegetations. 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, this 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 exam 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 occipital cortex, and will cause a specific visual field defect based on location.

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

- Retinal detachment
- Vitreous detachment
- Retinal artery occlusion
- Central retinal vein occlusion
- Vitreous Hemorrhage
- Neuro-ophthalmologic vision loss (commonly infarction, tumor, AVM or migraine disorders)

While a slit lamp exam and ultrasonography may be helpful in the work up of retinal and vitreous pathologies, these modalities will not be of much value in diagnosing the majority of neuro-ophthalmologic lesions.

An accurate history and directed physical exam is key in the ED workup of acute-onset vision loss. Confrontational visual field testing, or Donder's Test, can be performed in seconds as part of the bedside exam.[2] 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 180º visual field, the examiner should move the hand slowly to a more central position until the patient confirms visualization of the target. Once visualization is confirmed, use this at the starting point for stationary testing. To perform stationary testing, the examiner holds up a certain number of fingers peripherally, equidistant between the examiner and the patient. The patient is asked to 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 recognized loss of vision in her left eye, confrontational visual field testing revealed vision loss in the left lower quadrant of both eyes, termed homonymous left lower quadrantanopia. Identification of that deficit helped to rule out primary opthalmalogic causes of vision loss such as retinal ischemia, retinal or vitreous detachment, and vitreous hemorrhage leading to an appropriate workup and the ultimate diagnosis.

The visual cortex is located in the occipital lobe, as depicted in Figure 2. The left hemispheric visual cortex receives signals from the right visual field, and the right hemispheric visual cortex receives signals from the left visual field via the optic radiations. The inferior division of the optic radiation, or Meyer's Loop, contains input from the superior visual field quadrants, and travels around the inferior horns of the lateral ventricles in the temporal lobe. The superior division, or Baum's Loop, contains input from the inferior visual field quadrants, and travels in the parietal lobe and has a much more direct path to the visual cortex. If a lesion is isolated to one division in one hemisphere only, the resulting visual defect is called quadrantanopia, which implies that only the respective superior or inferior quadrant of the visual field is affected. However, a lesion in the visual cortex itself can also cause a quadrantanopia if isolated to the area where the optic radiation terminates. The subtle differences in these presentations are only detectable by perimetry testing, which cannot be done at the bedside.

At the conclusion of the history and exam, the lesion in our patient was isolated to the right hemispheric superior optic radiation passing through the parietal lobe or in the visual cortex itself. A retrospective study in JAMA Neurology found that the location and frequency of lesions causing inferior quadrantanopia were most commonly the occipital lobe (76%), followed by the parietal lobe (22%) and temporal lobe (2%).[3] The study also found that if the lesion is in the parietal lobe, i.e. the optic radiations, then it will most often have associated parietal lobe deficits such as paresthesia, hemiparesis, neglect and aphasia. Lesions in the occipital lobe do not commonly have localizing symptoms on physical exam.

The ED workup of vision loss should be based on the history and exam and tailored appropriately. If cerebral infarction is suspected, as in our patient, and if the patient is presenting within a three-hour window, a non-contrasted CT head and CTA head and neck should be obtained emergently to determine eligibility for IV-TPA. The stroke team should also be contacted.

Additional studies should include CBC, renal panel for creatinine and glucose specifically, and coagulation studies. An EKG should be obtained, as the most common cause of a cardioembolic stroke is atrial fibrillation. Other sources of cardiogenic embolism include a mural thrombus on a hypokinetic wall segment, endocarditis, prosthetic heart valve thrombosis, rheumatic heart disease, and paradoxical embolism via a patent foramen ovale or atrial septal defect. Cardioembolic sources account for an estimated 20-25% of ischemic stroke. 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 proven to be superior to echo for detection of vegetations, and should only be utilized when there is still high suspicion for cardiac source and echocardiogram is nondiagnositic.[6] 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 head and neck or with carotid ultrasonography.

Continued on page 13

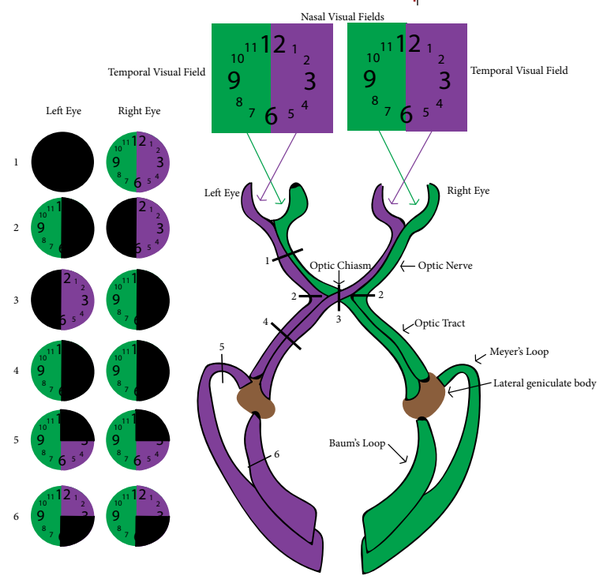


Figure 2: Visual Field Pathways, location of potential lesions causing neuro-ophathalomic vision loss and their associated deficits.[10]

#pharmconsult

Losing Your Breath

Treating Pulmonary Arterial Hypertension

Melissa Kincaid, PharmD
University of Cincinnati

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 arterial pressure of ≥ 25 mmHg. The prevalence of PAH is estimated to be 15-50 cases per million individuals, with approximately 1,000 new cases reported in the United States annually.[1,2] PAH is characterized by dyspnea on exertion, dizziness or syncopal episodes, chest pain, heart palpitations, and other symptoms 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.[3]

One of the major classes of medications used in the treatment of pulmonary hypertension is prostacyclins. Prostacyclin analogues exert their mechanism of action by eliciting a vasodilatory effect on the pulmonary vasculature, as well as inhibiting platelet aggregation and exhibiting antiproliferative effects on the pulmonary endothelial cells. Prostacyclins are formulated in various routes of administration, including parenteral, enteral, and inhalation. The 2014 CHEST guidelines for the treatment of adults with pulmonary arterial hypertension base treatment recommendations on patients' World Health Organization (WHO) functional class (FC) and treatment history (Table 1). The guidelines include recommendations regarding the use of prostacyclins (generally reserved for more severe cases) and indicate which route of administration should be utilized.

Prostacyclins have been shown in the literature to improve exercise capacity and hemodynamic parameters. Epoprostenol specifically has been found to have a benefit on survival in patients with pulmonary hypertension. In 1995, researchers conducted a prospective, randomized trial to compare the use of epoprostenol in addition to conventional therapy versus conventional therapy alone; conventional therapy consisted of anticoagulation, oral vasodilators, cardiac glycosides, and supplemental oxygen. Treatment with epoprostenol was associated with an increased survival compared to conventional therapy. All patients treated with epoprostenol in the study survived, whereas eight patients treated with conventional therapy alone died ($P < 0.001$).[5] More recently, a meta-analysis published by Zhang and colleagues reviewed the literature surrounding the use of prostacyclin therapy, including epoprostenol, treprostinil, iloprost, and beraprost. All four prostacyclin analogs demonstrated improvements in a 6-minute walk distance test (6MWD), functional capacity, and mortality rates, however specific outcomes differed based on the endpoint being evaluated and the agent utilized. Overall, the meta-analysis found epoprostenol to be the most recommended prostacyclin for use in the treatment of pulmonary hypertension based on increased improvements in exercise activity, functional capacity, and mortality.[6]

Parenternal Prostacyclins

The two parenteral prostacyclins available are treprostinil (Remodulin®) and epoprostenol (Flolan®, Veletri®). 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, confirmation of specialty pharmacy enrollment, and identification of plan for ongoing outpatient management. Parenteral prostacyclins should be dosed based on the patient's dosing weight, as a continuous infusion through a dedicated central line. The patient has a static dosing weight that is used throughout therapy, regardless of the patient's actual weight. Because the prostacyclins are dosed in ng/kg/min, any small fluctuation in weight being used for calculations can result in a big change in therapy, so verification of the patient's dosing weight is very important. Patient's dosing weight and current rate of infusion are on file with their Specialty Pharmacy and can generally also be found in the patient's chart in a PH clinic note if they are a UCMC PH patient. Treprostinil may be administered as a continuous intravenous or subcutaneous infusion, whereas epoprostenol may only be administered as a continuous intravenous infusion.

	Treatment-Naive	Disease Progression	Agents Used
WHO FC I	- Monitor for development of symptoms that warrant initations of pharmacotherapy; may initiate oral calcium channel blockers (CCBs) in patients with acute vasoreactivity		**CCBs**: amlodipine, diltiazem, nifedipine **ETRA**: ambrisentan, bosentan, macitentan **PDE-5 inhibitors**: sildenafil, tadalafil **Guanylate cyclase stimulator**: riociguat **Parenteral prostacyclin**: IV epoprostenol, IV treprostinil, SQ treprostinil **Inhaled prostacyclin**: treprostinil
WHO FC II	- Oral CCBs	If CCBs failed or contraindicated, endothelin receptor antagonist (ETRA), phosphodiesterase-5 inhibitor (PDE-5), or guanylate cyclase stimulator	
WHO FC III	- If CCBs failed or contraindicated, ETRA, PDE-5, or guanylate cyclase stimulator - With evidence of disease progression or poor clinical prognosis, initial treatment should include a parenteral prostacycline	With evidence of disease progression or poor clinical prognosis despite treatment with one or two classes or oral agents, consider addition or parenteral or inhaled prostacyclin	
WHO FC IV	- Parenteral prostacyclins are recommended as first-line monotherapy. - If patient is unable to manage parenteral prostacyclin, recommend inhaled prostacyclin in combination with an ETRA	Recommend addition of a second class of therapy	

Table 1: 2014 CHEST Treatment Guidelines for Adults with Pulmonary Arterial Hypertension Based on WHO Functional Class (FC) and Treatment History

Oral Prostacyclins

There is one oral prostacyclin product commercially available: treprostinil (Orenitram®). Oral treprostinil may be ordered in the emergency department as a continuation of patients' home therapy or patients may be admitted for initiation of therapy. Patients who are being initiated on oral treprostinil may only be admitted to MICU, MPCU, or CSD for further management, while patients continuing their home therapy may be admitted to any hospital unit. If more than two oral doses of treprostinil are missed, please consult the PH team.

Inhaled Prostacyclins

There are two inhaled prostacyclin products available: treprostinil (Tyvaso®) and epoprostenol (Flolan®). Inhaled treprostinil is non-formulary. 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 and pump to ensure the pump is functioning properly and 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 intravenous line placed in order to reinitiate therapy 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.[7] 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 to ensure the patient receives optimal care, including, but not limited to, calculating or verifying doses, ordering the medication, and compounding the medication.[3]

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Painless 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 as 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 intrarterial (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 basilar artery occlusion.[7] The American College of Chest Physicians recommandations, however, include patients with acute occlusions of any proximal cerebral blood vessel (e.g. ICA, MCA, basilar artery, vertebral artery) under the assumption that the pathophysiology and accessibility were believed to be similar for all major intracranial arterial locations.[8] In regards to mechanical clot retrieval, per the AHA/ASA guidelines, patients with an ischemic stroke caused by a proximal large artery occlusion in the anterior circulation are candidates for mechanical thrombectomy if presenting in less than 6 hours and without other contraindications. 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 vertebral arteries, basilar artery, or posterior cerebral arteries.[9] 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 neuroimaging.

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 hypertension, diabetes, hyperlipidema, obesity and tobacco use should also be managed meticulously to reduce the risk of future CVA.

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10 Adapted from Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing, and Allied Health, Seventh Edition. 2003 by Saunders, an imprint of Elsevier, Inc.

#minorcare

Mastering Minor Care

Benjamin Ostro, MD
University of Cincinnati R4

Closing the gap: Deep Sutures with Dr. Trott

For this installment of Mastering Minor Care we delve into some of the nuances of wound management. While many wounds are adequately repaired with simple interrupted sutures, not infrequently we are confronted with wounds that require more specialized suturing methods. One such method is deep sutures. Here to answer some questions regarding deep sutures is our wound management guru, and author of the book "Wounds and Lacerations: Emergency Care and Closures," Dr. Alexander Trott.

AOBP: When should I place deep sutures?

Traditionally, deep subcuticular closure is reserved for deep lacerations where a superficial cuticular closure will not close the deep portion of the wound. It might leave behind an open space that can fill with blood or other wound exudate that is thought to increased the risk of infection or increased scar formation.

Deep closures are also used to reduce tension of the superficial, cuticular closure.

However, as is common in wound care, there is no definitive research, either bench or clinical, that can shed light on these techniques and their outcomes.

AOBP: Are there any tricks of the trade that can improve wound healing when placing deep sutures?

Effective, high pressure irrigation using a 20 cc syringe capped with a standard splash shield remains the most important step in cleaning deep wounds. Also, debriding contaminated or non-viable wound tissue is essential to minimize risk of infection.

To further decrease the incidence of wound infection when placing deep sutures, it is preferable to use monofilament, absorbable suture material such as Monocryl. Braided suture material potentially has a higher risk of infection because of increased surface area of braiding. Place as few deep sutures as needed to close the deep space or reduce wound tension. In practice, I rarely use more than 3 deep closures per wound that does not need extensive repair. If the wound is deep, but not too deep, consider a horizontal mattress which has both a deep and superficial configuration.

When in doubt, leave them out. The body has remarkable healing powers without unnecessary interventions.

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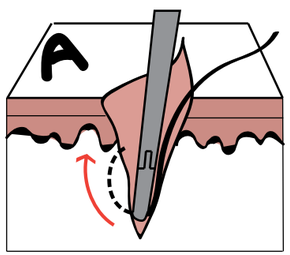
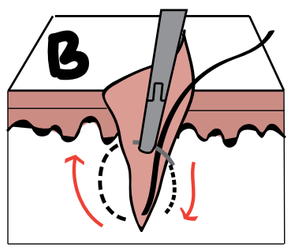
How do I place deep sutures?

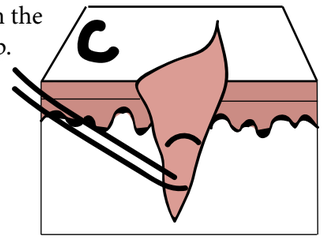
A. To start, the needle is inserted at the level of the superficial fascia and exits at the dermal-epidermal junction.



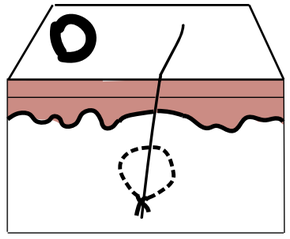
B. The needle is then re-armed with the driver and inserted at the dermal-epidermal junction on the contralateral side and exited at the level of the superficial fascia.



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 "trails."



Kawaski Disease Continued from page 4

occur together or separately, and appear in no particular order. Other clinical findings in these patients can include cough, rhinorrhea, diarrhea, vomiting, abdominal pain and arthritis. Although not a diagnostic criteria, one of the hallmarks of KD patients is their irritability. Patients are uncomfortable appearing and minimally consolable.

Although rare, the cardiac complications of KD are serious and life-threatening. Twenty to twenty-five percent of untreated children with KD will develop coronary artery aneurysms. These occur on average four to five weeks into the illness, but can be detected as early as day ten. These aneurysms can be very large (up to 8mm) and can go on to cause a myocardial infarction, which is the most common cause of death in patients with KD. MIs occur, on average, a year after the disease. Valvular pathologies, including acute stenosis or insufficiency of the mitral, tricuspid or aortic valves, can also occur.
The diagnosis is primarily based on presenting symptoms, but there are some laboratory findings that can help to guide the clinician. For those patients who do not meet the criteria above for KD but for whom the provider has a high clinical suspicion, supplementary lab criteria exist from the AHA for the diagnosis of incomplete KD. These include an albumin level less than 3.0; anemia for the patient's age; elevated ALT for age; a platelet count > 450,000 after 7 days of illness; a WBC of greater than 15,000; and urine with greater than 10 WBCs/HPF, which is almost always sterile pyuria secondary to urethritis. Many patients will also have an elevated ESR or CRP, but this is a nonspecific finding.

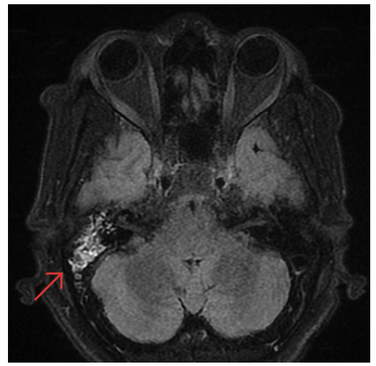
The treatment for KD involves high-dose aspirin and IVIG, both of which are chosen for their anti-inflammatory properties. Patients will often defervesce 24 to 48 hours after the initiation of this treatment. They will then need to be transitioned to low-dose aspirin once they are afebrile. They will remain on low dose aspirin for several weeks to months later. A baseline echocardiogram is usually done at the onset of the disease to evaluate for coronary artery involvement, and may be repeated as frequently as needed as the patient recovers. As the presenting symptoms may mimic many other benign viral illnesses of childhood, patients will often present to multiple providers before a diagnosis can be made. As such, it is of the utmost importance that emergency physicians maintain a high level of suspicion for Kawasaki Disease in the persistently febrile child. Although KD has the potential for significant morbidity, if recognized and treated appropriately most patients do very well with no significant life-long complications.

1. Yim et al. Update on Kawasaki disease: epidemiology, aetiology and pathogenesis. J. Paediatr. Child Health 2013. (49) 704-708
2. AHA Scientific Statement: Diagnostic Guidelines for Kawasaki Disease. Circulation. 2001 03: 335-336
3. Yim et al. An update on Kawasaki disease II: Clinical features, diagnosis, treatment and outcomes. J. Paediatr. Child Health. 2013. (49): 614-623
4. Kuo et al. Kawasaki Disease: An Update on Diagnosis and Treatment. Pediatrics & Neonatology. 2012. 53(1): 4-11
5. Newburger et al. Diagnosis, treatment and long-term management of Kawasaki disease. Circulation. 2004. (110) 2747-2771

Malignant Otitis Externa Continued from page 5

as laboratory inflammatory markers such as ESR, CRP and WBC count are frequently normal. Additionally, as inflammation of the skull base can be involved, these patients should be screened for any cranial nerve palsies. Cranial nerve CVII is most commonly affected. However, cranial nerves VIII-XII palsies have also been reported.[5] Aside from basic laboratory workup, including ESR and CRP, if ear discharge is present, a culture and gram stain should be sent. Both MRI and CT are useful imaging modalities to evaluate the anatomic extent of soft tissue inflammation, abscess formation, and intracranial complications.[5] More often, however, CT is the initial imaging modality of choice as it is more readily obtained in the emergency department and should include thin slides through the skull base, at UCMC the orbit-sella-IAC scan is ideal. An alternative imaging modality is a gallium-citrate bone scan which has very high sensitivity for detecting osteomyelitis and is useful for monitoring disease resolution after treatment.[6]

As the majority of cases of this disease are secondary to P. aeruginosa, anti-pseudomonal coverage with intravenous antibiotics and admission to the hospital are the standard of care. Ciprofloxacin has been the first-line antibiotic. Given growing resistance patterns of P. aeruginosa to fluroquinolones, however, cefepime or piperaillintazobactam should be considered.[7,8] As with other forms of osteomyelitis, a full six-week course of antibiotics is typically recommended. Although hyperbaric oxygen has been proposed as an adjuvant treatment in refractory cases, there is no clear evidence thus far to support its efficacy.[9] With the exception of debridement and biopsy to rule out maligiancy surgical intervention is rarely indicated in typical malignant otitis externa. Following treatment, recurrence rates can be has high as 20%.[10] Left untreated, it can cause serious complications including permanent sensorineural hearing loss, cerebral abscess, meningitis, dural sinus thrombosis, and death,[11] making prompt diagnosis in the Emergency Department crucial.

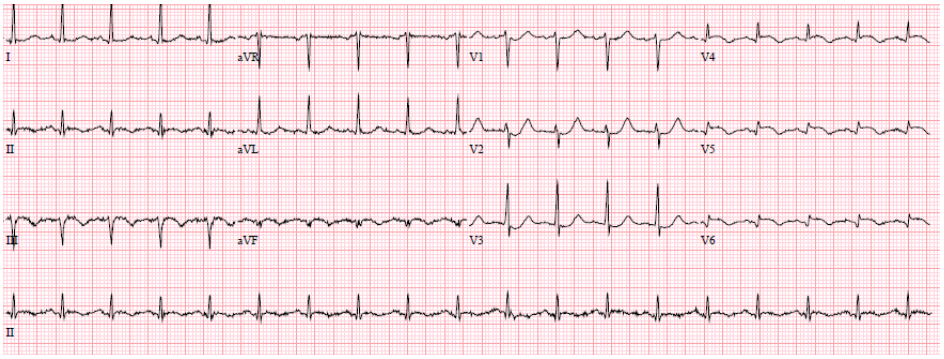


MRI Head w/o contrast: Extensive soft tissue thickening in the right external auditory canal with central non-enhancement and diffusion restriction.

1. Singh A, Al Khabori M, Hyder MJ. Skull base osteomyelitis: diagnostic and therapeutic challenges in atypical presentation. Otolaryngol Head Neck Surg 2005;133:121
2. Rutka J. Acute otitis externa: treatment perspectives. Ear Nose Throat J 2004; 83:20–21.
3. Jacobsen LM, Antonelli P. Errors in the diagnosis and management of necrotizing otitis externa. Otolaryngol Head Neck Surg. 2010;143(4):506.
4. Mani N, Sudhoff H, Rajagopal S, et al. Cranial nerve involvement in malignant external otitis: implications for clinical outcome. Laryngoscope 2007;117:907–910.
5. Grandis JR, Curtin HD, Yu VL. Necrotizing (malignant) external otitis: prospective comparison of CT and MR imaging in diagnosis and follow-up. Radiology 1995;196:499–504.
6. Stokkel MP, Boot CN, van Eck-Smit BL. SPECT gallium scintigraphy in malignant external otitis: initial staging and follow-up. Case reports. Laryngoscope 1996;106:338–40.5.
7. Joachims H, Danino J, Raz R. Malignant external otitis: treatment with fluoroquinolones . Am J Otolaryngol. 1988;9:102-105.
8. Berenholz L, Katzenell U, Harell M. Evolving resistant pseudomonas to ciprofloxacin in malignant otitis externa. Laryngoscope 2002;112:1619–1622.
9. Phillips JS, Jones SE. Hyperbaric oxygen as an adjuvant treatment for malignant otitis externa. Cochrane Database Syst Rev. 2013.
10. Singh A, Al Khabori M, Hyder MJ. Skull base osteomyelitis: diagnostic and therapeutic challenges in atypical presentation. Otolaryngol Head Neck Surg 2005;133:121–5.
11. Schwarz GA, Blumenkrantz MJ, Sundmäker WL. Neurologic complications of malignant external otitis. Neurology 1971; 21:1077.

EKG focus

Grace Lagasse, MD
University of Cincinnati R2



The EKG above highlights some of the EKG changes consistent with a PMI. There is ST segment depression and upright T waves in the anterior precordial leads, V1-V3. In this EKG, leads V4-V6 are actually posterior EKG leads and represent V7-V9, respectively. ST elevation can be seen in these posterior leads.

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 occurance of isolated PMIs is unknown, but is thought to be <10% of all STEMIs.

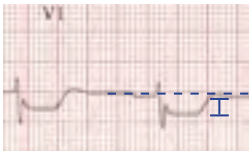
EKGs and Case referred by

Drs. Dang and Otten
University of Cincinnati

Brown DF, Nadel ES. Posterior wall myocardial infarction. J Emerg Med. 2004 Jul;27(1):75-8.
Khan JN, Chauhan A, Mozdiak E, Khan JM, Varma C. Posterior myocardial infarction: are we failing to diagnose this? Emerg Med J. 2012 Jan;29(1):15-8.
Madias JE, Bravidis D, Attari M. Posterior myocardial infarction and complete right bundle- branch block. Chest. 2002 Nov;122(5):1860-4.
Vasaiwala SC, Schreiber R. Posterior myocardial infarction: unique diagnosis to an elusive problem. Am J Emerg Med. 2008 May;26(4):520.e5-6.
Waldo SW, Brenner DA, Li S, Alexander K, Ganz P. Reperfusion times and inhospital outcomes among patients with an isolated posterior myocardial infartion: insights from the National Cardiovascular Data Registry (NCDR). Am Heart J. 2014 Mar;167(3):350-4.
Waldo SW, Armstrong EJ, Kulkarni A, Hoffmayer KS, Hsue P, Ganz P, McCabe JM. Clinical characteristics and reperfusion times among patients with an isolated posterior myocardial infarction. J Invasive Cardiol. 2013 Aug;25(8):371-5.

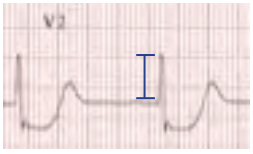
Where can you find a PMI in an EKG? In the anterior precordial leads, V1-V3!

ST segment depression



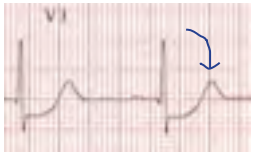
Leads V1-V3 are an electrical mirror of the posterior leads V7-V9. ST depression in V1-V3 can represent ST elevation in leads V7-V9.

Prominant R waves



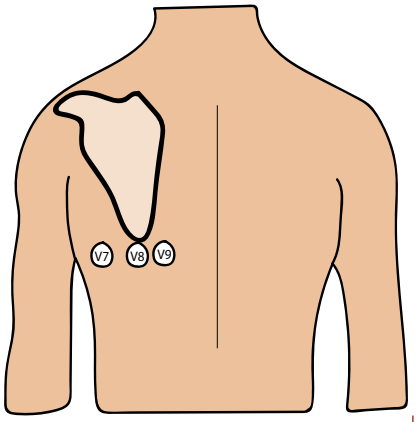
Prominant R waves in leads V1-V3 reflect Q waves in the posterior leads V7-V9. This occurs because V1-V3 are an electrical mirror of V7-V9.

Upright T waves

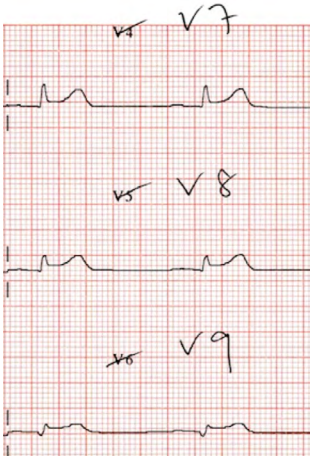


Upright T waves in leads V1-V3 and ST depression in the same leads can help to differentiate a PMI from a septal MI or anteroseptal ischemia.

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.

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List of Submitted B Pod Cases

Case	Case Physicians
Dysfibrinemia with SMA thrombosis	Doerning
HIV & syphillis	Hamilton/Betz
Giant cell arteritis w/ visual symptoms	Beyde/Niziolek
Spontaneous vertebral artery dissection	Merriam/Betz
Hermicrania Continua	Denney/Adeoye
Brugada Type 3	March/Bonomo
Familial Hypertriglyceridemia	Whitford/Ostro
Malignant Otits Externa	Shaw/LaFollette
Occipital Infarct	Harrison/LaFollette