Progressive Multifocal Leukoencephalopathy

VZV Meningitis

Traumatic Hyphema

Retrobulbar Hematoma

Epistaxis

Fitz-Hugh Curtis

EKG Corner: Hypothermia

Splenetic Infarct

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Now you know...

**Quality Matters**

Daniel Axelson, MD
University of Cincinnati R3

You’ve undoubtedly (…hopefully!) mastered your ABCs at this stage, but do you know your PQRS? You’ve heard of EBM, but are you brushed up on VBM?

Well, as of 2017 you better be.

Because in 2017 your value as a physician, and thus your Medicare physician reimbursement, can be penalized or rewarded based on quality and cost. Let’s explore.

PQRS stands for Physician Quality Reporting System. It is a quality data reporting system to CMS, the Centers for Medicare and Medicaid services, i.e. the government payer for healthcare services, i.e. the source your salary. It’s a way the government tracks how efficacious and cost-effective you are as a provider. Since 2007, this program has been voluntary, and physician-groups have been given financial incentives for reporting this cost and outcome data to the PQRS. But as of 2015 such reporting is mandatory, enforced by penalties in physician reimbursement from CMS for non-compliance. Such penalties can take effect starting in 2017. The penalties apply to every physician and every physician group, regardless of size or specialty. Additionally, CMS will begin publicly reporting PQRS data in 2016 on all physicians nationally.

Scoring grows ever finer. Yes, you’ve quite literally being watched.

VBM stands for Value Based Modifier. It is a score tied to quality and cost measures on the part of each individual physician. The score is composed of a Quality Composite Score, which is calculated from things such as patient safety, efficiency, and effective clinical care, and a Cost Composite Score, which is calculated from things such as total costs and disease specific costs. (Figure 1) Physicians will now be classified as at, below, or above average in quality and cost measures, and this will affect their reimbursements. (Figure 2) The Affordable Care Act requires that CMS apply this VBM to every physician starting in 2017.

There will be more government regulation over the healthcare you provide. Now you know.


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### Physical Exam

T 98.7  HR 68 RR 22 BP 220/65 SpO2 95% on RA

Exam reveals a Caucasian female with significant trauma to her right eye, face, lip, sitting up in bed in no acute distress but with bloody clothing. The HEENT exam reveals a right eye which is swollen shut with significant surrounding ecchymoses and swelling over maxilla. It is proptotic, with significant chemosis and a fixed mid dilated pupil not reactive to light. She is able to tell light vs dark but has no improvement in her vision. She is able to move her right eye only very slightly. The rest of her HEENT exam reveals a normal left eye exam, swelling over her right maxilla but no pain on palpation of the face and no Battle’s sign. Her neck, cardiovascular, pulmonary, abdominal, and remaining neurological exam are unremarkable.

### Hospital Course

Patient presented one hour after facial trauma with a proptotic right eye, only light/dark differentiation in that eye, as well as loss of extra-ocular movements and an afferent pupillary defect. Together, these findings were classic for retrobulbar hematoma. This was corroborated on her maxillofacial CT (Figure 1). Due to the patient’s significant vision deficits on presentation, a right lateral canthotomy with cantholysis was performed urgently at bedside, with successful release of the upper and lower canthal ligaments. After her procedure, the patient had no improvement in her vision, and worsened from light/dark perception to complete vision loss. Her IOP remained persistently elevated in above 90 mm Hg. Her IOP was acutely managed with timolol drops as well as mannitol, with some improvement in her IOPs. She was admitted to the trauma service for syncope workup as well as management of her ophthalmologic issues as well as multiple orbital and mid-face fractures. The patient ultimately suffered complete vision loss in her right eye.

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### Discussion

Retrobulbar hematoma, which can also be conceptualized as “orbital compartment syndrome,” is a vision threatening condition and ophthalmologic emergency. Retrobulbar hemorrhage has been described after trauma as well as after facial surgery, and this presentation may be delayed up to days after injury.1 This is an uncommon condition, even in the setting of orbital fracture – only 0.6-0.8% of patients with orbital fracture have a coexisting retrobulbar hematoma. However, it is quite morbid, as patients who present with vision loss in the setting of retrobulbar hemorrhage have a 44-52% chance of permanent blindness. The retina may tolerate approximately two hours of ischemia before vision loss is irreversible.2

Bleeding into the orbital space increases pressure in a closed cavity. With increased intra-orbital pressure, the globe is displaced anteriorly until it is tethered by the canthal ligaments, resulting in a compressive neuropathy. The optic nerve stretches and suffers ischemia as the globe is displaced anteriorly. Additionally, the pressure in the central retinal artery cannot overcome increased IOP leading to retinal ischemia. Together, these factors result in the classic proptotic eye with an afferent pupillary defect and progressive visual loss. Additionally, patients may have eye pain, ophthalmoplegia, and findings on fundoscopic exam such as a cherry red macula and nerve head pallor. Intracocular pressures with these secondary findings are typically >40 mm Hg.3

Lateral canthotomy with cantholysis is the treatment of choice for decompression of the orbit after retrobulbar hematoma. The only contraindication to lateral canthotomy include suspected globe rupture. See Page 31 for a description of the procedure.
Weakness in a HIV patient: Progressive Multifocal Leukoencephalopathy

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History of Present Illness
The patient is a 30-year-old male who presented to the emergency department with a chief complaint of left-sided weakness. The patient said that over the last month he has had progressively worsening weakness of his left arm and leg. He first noticed this about a month ago, when his thumb began slipping off of the TV remote. Over the course of the month, he developed the inability to raise his left arm off of the bed without the assistance of his right arm, and was having difficulty taking care of himself. When his family was in the room he said he had been taking his HIV medications for at least a year prior to the diagnosis. Before the implementation of HAART, 3.3% of HIV patients presented with PML, however now the incidence is 1.6% of HIV patients. Non-adherence to HAART medications also increased the risk of death from PML for our patient, approximately 5% of patients with PML on HAART die within 1 year while 50% not on HAART die within the same timeframe. PML contributed to 14% of AIDS related deaths in 2005 and even though HAART has increased survival from 0-30% to 38-62%, its effect on PML is the smallest amongst all AIDS-related illnesses.

The patient was admitted to the hospital for treatment of suspected progressive multifocal leukoencephalopathy (PML). He was restarted on his HIV medications and other infectious sources of neurologic pathology were ruled out, including toxoplasmosis, tuberculosis, and Herpes Simplex Virus. He did have a lumbar puncture that was positive for the John Cunningham (JC) virus, which was expected with his MRI findings consistent with PML. He was discharged to rehab with continued neurologic deficits in his left upper and left lower extremities. Rameron was started prior to discharge along with HAART medications and sulfamethoxazole/trimethoprim prophylaxis. Approximately 1 month after diagnosis, he was readmitted for failure to thrive and had a repeat brain MRI (Figure 2) significant for widely progressive PML. At this time he was unable to swallow or talk secondary to weakness and his family decided to pursue hospice care. The patient passed away approximately 6 weeks after the diagnosis was made.

Discussion
Progressive multifocal leukoencephalopathy (PML) is an infection of the central nervous system by the JC virus that affects patients with HIV. It is a ubiquitous virus that is transmitted via both inhalation and ingestion. The incidence in HIV patients with CD4 counts greater than 200 is much less than those with CD4 counts less than 200. 0.07 vs 0.7 infections/person years. Our patient was more susceptible to the infection because he had a CD4 count of 12 upon arrival. He also had not been on his Highly Active Anti-Retroviral Therapy (HAART) medication for at least a year prior to the diagnosis. Before the implementation of HAART, 3.3% of HIV patients presented with PML, however now the incidence is 1.6% of HIV patients. Non-adherence to HAART medications also increased the risk of death from PML for our patient, approximately 5% of patients with PML on HAART die within 1 year while 50% not on HAART die within the same timeframe. PML contributed to 14% of AIDS related deaths in 2005 and even though HAART has increased survival from 0-30% to 38-62%, its effect on PML is the smallest amongst all AIDS-related illnesses.

The JC virus is a polyomavirus that infects and destroys oligodendrocytes. Patients present with partial neurologic deficits that worsen with time as the lesion moves along white matter tracts. This infection pattern leads to the most common presenting complaints including speech difficulties, gait disturbances, and like our patient, limb weaknesses. Physical exam often reveals coordination deficits, cognitive deficits, and limb paresthesias that correlate with the most common sites of infection: subcortical white matter, white matter in the cerebellar pons, and in the brainstem. Patients typically present after the disease has progressed for weeks to months, at which time they finally have enough deficits to notice a change in functionality. Our patient waited to seek treatment until he couldn’t use his TV remote anymore but when questioned further, had been developing signs of deficits for approximately a month.

The diagnosis of PML is typically made, as in our patient, in the following sequence: there is clinical suspicion followed by radiological identification with MRI and then detection of JC virus with CSF PCR. The only treatment for PML is to initiate HAART, which was done promptly upon admission to the hospital. Unfortunately, this only halts progression of the disease in half of patients and ultimately was unable to stop the progression in our patient. HAART cannot reverse the damage already done to the neurons and will leave residual neurologic deficits even those in whom therapy works to stop progression. Poor prognostic factors include lower CD4 counts, higher plasma HIV RNA, higher JC viral loads in the CSF, and any presence of brainstem lesions. Our patient had a CD4 count of 12 and a very high plasma HIV RNA load, both of which were predictive of his poor prognosis. Survival time after a diagnosis of PML pre-HAART was improved from approximately 1 year to around 1.8 years today. Sadly, our patient died within 2 months of diagnosis, from likely aspiration, the most common cause of death in this population.

In the era of HAART, PML has become more uncommon and therefore this particular patient does not present everyday in the ED. However, 60-70% of HIV patients develop CNS complications (Figure 4) and 20% of visits to the ED by HIV+ patients are for neurologic complaints. There is a body of literature attempting to guide the specific management of progressive multifocal leukoencephalopathy.

CNS Infections in HIV

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Prevalence</th>
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<tbody>
<tr>
<td>CMV</td>
<td>Primary CNS Lymphoma</td>
</tr>
<tr>
<td>Cryptococcus</td>
<td>Toxoplasmosis</td>
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<tr>
<td>Progressive Multifocal Leukoencephalopathy</td>
<td></td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>CMV</td>
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</tbody>
</table>

CD4 Count

0 50 100 200 250

Figure 1: CT head without contrast: Vasogenic edema in the right frontal lobe, concerning for an infectious or neoplastic lesion.

Figure 2: Initial MRI findings suggestive of progressive multifocal leukoencephalopathy in the right frontoparietal white matter and right thalamus with possible other foci of disease in the left corona radiata.

Figure 3: Repeat brain MRI one month later showing extension of the lesions associated with progressive multifocal leukoencephalopathy.

Figure 4: CNS infection risk by CD4 count.

Continued on page 7
History of Present Illness

The patient is a 22-year-old female with no past medical history presenting with rib pain. She reports 3 days of pain over the right costal margin. She described it as progressively worsening, moderate to severe, achy in quality, and intermitent. The pain is aggrivated by deep inspiration. She has no history of gallstones or abdominal surgeries. She denies nausea, vomiting, diarrhea, fever, vaginal discharge, dysuria, urinary frequency, chest pain, shortness of breath, and cough. Her last menstrual period ended a few days prior. She is sexually active with one partner and has no history of STIs. She has never been pregnant.

Past Medical History

Seasonal allergies

Social history

Non-smoker, sexually active with one partner, occasional condom use

Medications

Loratadine

Allergies

No known

Physical Exam

T 37 HR 120 BP 18 918/67 SpO2 100% on RA

Medications Allergies

Loratadine

No known

Discussion

Our patient presented with colicky right upper quadrant (RUQ) abdominal pain that was initially concerning for gallbladder, kidney, or lung pathology. The workup included a complete blood count, basic metabolic panel, liver function tests, urinalysis, urine pregnancy test, and chest x ray which were remarkable only for a leukocytosis of 15.2.

Over the course of her Emergency Department stay her pain was difficult to control and her tachycardia persisted despite IV fluids. She was admitted to the hospital and 6 hours after admission, her tachycardia with a regular rhythm. Her abdominal exam demonstrated tenderness to palpation, right side, a negative Murphy’s sign, and no rebound or guarding.

Acute PID

Causes acute onset of lower abdominal pain and tenderness of the pelvic organs on exam. It is complicated by tubo-ovarian abscess, acute salpingitis, peritubalitis (Fitz-Hugh-Curtis), endometritis, and pelvic peritonitis.

Subclinical PID

A subclinical infection of the upper reproductive tract with symptoms that usually do not prompt the patient to seek medical care, can cause long-term sequelae, specifically infertility.

Chronic PID

A rare indolent infection associated with tuberculo-sis or actinomycosis.

Previous inflammatory disease (PUD)

FHC is a rare extra-pelvic complication of a genital infection that involves the peri-hepatic capsule. While most cases have been described in women in association with PID, in rare cases it has been reported in men.1 A prospective cohort study in 117 incarcerated adolescents documented a 4% incidence of Fitz-Hugh-Curtis syndrome in those with mild to moderate PID.6 Clinical features include severe RUQ abdominal pain, which may have a pleuritic component and radiation to the right shoulder. Aminotransferases are usually normal or only mildly elevated. If a CT is obtained it may show inflammatory changes in the pelvis and peritoneal regions. CT imaging may also reveal subtle perihepatic enhancement.

Continued on page 15

of the Academic Medical Colleges (AAMC) annual Learn, Serve, Lead meeting in Baltimore as a member of the Organization of Resident Representatives (ORR). Before I delve into the themes of this year’s meeting, let me give a little background on the AAMC. The AAMC is a not-for-profit association that represents 450 American medical schools, approximately 400 major teaching hospitals and health systems, including 51 VA medical centers and 90 academic and scientific societies. They are not a regulatory body and do not make specific recommendations. They do, however, make suggestions with the overall goal of improving undergraduate and graduate medical education. Thus, most of those in attendance at the meetings have an interest in academic medicine and range from medical students to undergraduate medical educators to resident medical educators.

Who did I meet? Within the ORR, I met residents of specialties, including preventative medicine, surgery, dermatology, ophthalmology, etcetera. This diversity allowed us to compare notes and focus on broad topics that span specialties with the goal of improving medical education globally. The conference also invites the Organization of Student Representatives, Council of Deans, Council of Teaching Hospitals and Customized Assessment Services, Group on Women in Medicine and Science, Council of Faculty and Academic Societies, Group on Student Affairs, and many other small committees. These committees allow for a wide range of idea sharing for innovative solutions.

The overarching topic of this conference focused on the underserved and resiliency within medical education. The main speaker was Eugene Robinson, who is a Pulitzer prize winning columnist and editor of the Washington Post. He reflected on the presidency of Barack Obama, the passage of healthcare reform, and more recently the civil unrest in Ferguson and Baltimore. Mr. Robinson set the tone for the conference by underscoring the AAMC’s commitment to decrease and hopefully end the racial and socioeconomic gap felt by the most vulnerable in our medical system.

Regarding resiliency, several smaller (up to 100 attendees) focused on the need of resiliency across all levels of training and the need to address the problem early. The AAMC brought Frank Warren, the creator of the Post-Project to help frame this topic for us. Post Secret involves people sending secrets on postcards from all across the world, and it has been published online and in books. Mr. Warren got the idea as a psychiatric social worker working for the suicide hotline and understood how freeing it can be for people to share their secrets. This is directly applicable to the medical field as medical professionals often do not want to admit inadequacies or failures for fear of being shamed or rejected. This underlying message is unconsciously relayed to medical students as they feel pressure to not disclose their inadequacies for fear of being found “not as smart” as their peers. This is thought to foster the development of impostor syndrome and lead to loneliness and depression.

Ultimately, I walked away from the conference with about 11 new ideas for projects, invigorated about the future possibilities for medical educators, and excited to have met a group of people so dedicated to this field.
The patient is a 45-year-old male who presents after sustaining an injury to his right eye with a fishing hook. He states that a three-barbed hook pierced his eye while fishing with his friend. On gross inspection, the hook was noted to have pierced the inferior eyelid causing an obvious right open globe and there was a large hyphema. While he was initially able to count fingers at four feet in his superior visual field, his visual acuity quickly deteriorated to light perception only. Extravascular movements were intact and caused movement of the hook. Ophthalmology was consulted and a CT was obtained. The patient was then taken to the OR for anterior chamber washout, open globe repair, and removal of the fish hook. He was discharged following the surgery with next day follow up with ophthalmology.

**Discussion**

A hyphema, defined as a collection of blood in the anterior chamber, is most commonly caused by trauma to the eye.2 Blunt trauma causes a hyphema by stretching or shearing anterior uveal structures. Penetrating trauma causes hyphema due to the direct vascular injury. Hyphema can also form spontaneously in conditions such as leukemia/lymphoma, ocular neoplasm, coagulopathies, and sickle cell disease.3 When evaluating a patient with a hyphema, a slit lamp exam should be performed to rule out a corneal abrasion, evaluate for traumatic iritis, and visualize the extent of bleeding and clot formation. Seidel’s test is a helpful test to evaluate for open globe injury. To perform this test, the clinician anesthetizes the globe with tetracaine and applies fluorescein just as one would to evaluate for a corneal abrasion. The eye is then examined with a Wood’s lamp or the blue cobalt slit lamp filter. If perfusion or leakage is present, the concentrated dye will be diffused by the aqueous humor from the anterior chamber.

Only once an open globe injury is excluded should a clinician obtain intra-ocular pressures. Intra-ocular pressures are necessary because patient’s with hyphema are at high risk for secondary glaucoma due to blockage of aqueous humor drainage by clotted blood.1 Patient’s with sickle cell disease are particularly susceptible to this pathology.5 Management of hyphema focuses on preventing further trauma, preventing rebleeding, and treating complications.3 An eye shield is typically applied to prevent further trauma.2 The head of the bed should be raised to at least 30 degrees whenever the patient is supine to prevent secondary glaucoma caused by blood settling posteriorly.2 A cycloplegic such as atropine can be used to immobilize the pupil to prevent further injury to vessels of the anterior chamber.3 Topical steroids can be considered to prevent secondary events associated with traumatic hyphema.6 Given the limited evidence to support these treatments, deci- sions to use these medications should be made on a case-by-case basis and in consultation with ophthalmology.5 Antibiotic ointments such as oral aminocaproic acid have been shown to reduce the rate of rebleeding, but do not improve visual acuity.5 Patients should be instructed not perform any strenuous activity that could lead to rebleeding. Rebleed- ing typically happens 2-5 days following the injury and is tracked with daily measurements of intraocular pressure.6

Indications for hospital admission in traumatic hyphema include involvement of greater than 50% of the anterior chamber, rebreeding, elevated intraocular pressure, suspected child abuse, or poor patient follow-up.7 Patient compliance is important, as they need next day follow-up with an ophthalmologist.

References:

Diffuses through the interstitial tissue and charged conformation. Upon injection into in which the molecule is in its water soluble terminal amine allows the molecule to transi-
matic ring as well as a terminal amine. The of the nerve2. Local anesthetics are either am-
in nerve fibers which disrupts depolarization pain by reversibly blocking sodium channels. The low pH of the drug formulation makes injection painful to the patient. Lidocaine may be buffered to decrease pain upon injection by co-admin-
istering sodium bicarbonate as a 1:9 mixture made of one part sodium bicarbonate 1 mEq/ mL to 9 parts 1% lidocaine1. This should be used promptly as the shelf life of the lidocaine will decrease upon buffering. Some local an-
esthetic are formulated with epinephrine to create local vasoconstriction for the purpose of decreasing systemic absorption of the an-
esthetic.3 Epinephrine may extend the du-
rations of action in certain anesthetics such as lidocaine (Table 1). Epinephrine formul-
ations should be avoided when the patient has comorbidities including severe hyper-
sension, hyperthyroidism, phaeochromocytoma, coronary artery disease, or suspected recent cocaine use.2 There is no maximum dose recommendation, however 40 mcg per 30 minutes is reported in dental literature.1 For reference, 300 mcg is the intramuscular dose to treat anaphylaxis4. A list of UCMC formu-
lar local anesthetics class, dose, and pearls are listed in Table 1.

True allergic reactions to local anesthetics are rare. It is carefully to important question the patient on their allergy as patients often mistake syncope or tachycardia for allergies. If the allergy is not a major reaction such as diffuse hives, throat swelling, or anaphylaxis a different anesthetic without preservatives or epinephrine may be used as adverse events are hypothesized to be from the additives then local anesthetics themselves. If the aller-
gy is major, neither amides nor esters should be used as there is a possibility of cross reac-
tivity among the classes. The patient may be referred to an allergist for further investigation and possible future use.

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Diphenhydramine is an alternative agent that can be considered for local-
coronary artery disease, or suspected recent cocaine use1,2. There is no maximum dose recommendation, however concern for necrosis limits its use to small skin procedures. Efficacy was measured by pinprick in a trial involving 24 blinded patients randomly given 0.5 mL of placebo, 1% lidocaine, 1% diphenhydramine, and 2% diphenhydramine. 1% lidocaine and 1% diphenhydramine displayed no difference (p=0.889). A desired 1% sol-
cation can be made by drawing up 50 mg/mL diphenhydramine with 4 mL of normal saline. Onset of action has been reported to begin within 5 minutes, and duration lasting 30-40 minutes.

Tonic complications may occur if dose exceeds the maximum recom-
ended dose or if drug is injected into a major vessel. Patients with renal or hepatic disease should receive a 50% dose reduction to avoid toxicity. Metallic taste, tinnitus, and tingling lips are the first signs of lidocaine toxicity, seizures may then occur, which may be treated with benzodiazepines. Supracuro has the highest risk of toxicity. If ventricular arrhythmias or cardia-
asthmatic agent of choice, in some cases right atrial electromechanical dissociation may be observed. 10 mg of lidocaine is administered as a bolus followed by 1 mg/minute until hemodynamic recovery is ob-
tained. Reversal of the sodium blockade may be overcome by administering sodium via so-
dium bicarbonate or hypertonic saline infusions or boluses. Table 2 summarizes the anesthetic ad-
verse effects.

In summary, retrobulbar hemorraghe is an uncommon but time-
sensitive, highly morbid condition that carries an approximately 50% chance of vision loss for the patient. Timely recognition and intervention at bedside with a lateral canthotomy with cantholysis can prevent further ischemic damage to the nervous structures of the eye and help to mitigate vision loss for the patient.

Table 2: Anesthetic Adverse Effects 6

Midazolam (Versed) 0.25mg/kg or 2mg/m 2

Diphenhydramine 50mg/m 2

Bupivacaine, IV over 5 minutes

Mepivacaine

Tetracaine,

Bupivacaine

Lidocaine

Table 1: UCMC Local Anesthetics 1,2

Anesthetic Class Onset Duration Max dose Available at UCMC Clinical Pearls

Lidocaine Ester 1.5 30-90 4-5mg/[max total dose 300mg] 1%, 2% PF 0.5%, 1%, 2%, or 4% Most commonly used. May be combined with bupivacaine to create a short sett and extended duration (pay close attention to max dose when combining, additive effects).

Lidocaine + epinephrine Ester 1.5 60-160 3-7mg/[max total dose 300mg] 1:100,000 as 1% and 2% 1:200,000 as 0.5%, 1%, 1.5%, or 2% Use with caution in patients with diminished cardiac reserve, severe hypertension, hepatitis, or cirrhosis. Not recommended for intrathecal use in pediatric or geriatric patients.

Bupivacaine Ester 5-10 3-8H 2mg/[max total dose 15mg] 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 Ester 5-10 3-8H 2mg/[max total dose 22mg] 1:200,000 as 0.25%, 0.5%, or 0.75% Similar onset and duration as lidocaine

Mepivacaine Ester 30-120 30-120 5-10mg/[max total dose 300mg] PF 1% Similar onset and duration as lidocaine

Tetracaine Amide BH BH 1mg/[max dose 800mg] 2% or 3% Locally vasoconstricting and used as a local vasoconstric-
tion

Chloroprocaine Amide Amide 30 30 10mg/[max total dose 800mg] 1% Less irritation than lidocaine. Use in small skin procedures.

Common Anesthetic Concentrations

2% 10mg/mL

1% 10mg/mL

0.75% 7.5mg/mL

0.5% 5mg/mL

0.25% 2.5mg/mL

0.125% 1.25mg/mL

1:10,000 10mcg/mL of epinephrine

1:200,000 20mcg/mL of epinephrine

Figure 1: Reactions undergone by local anesthetic medi-
cations from injection until the medication binds to and works sodium channels in neuronal membranes produc-
ing the sought after anesthetic effect.

Table 2: Anesthetic Adverse Effects 6

CNS Effects CV Effects

Metallic taste Bradycardia

Tinnitus Decreased myocardial contractility

Tingling lips AV block

Agitation Vasodilation

Seizures (dose dependent) Ventricular arrhythmias

Retrobulbar Hematoma

Continued from page 3

Ideally, decompression is performed as soon as possible after the injury, to mitigate the effects of ischemia on the nervous structures of the orbit. If decompression is unsuccessful in lowering IOP, adjunctive therapies include pharmacologic interven-
tions to decrease intra-ocular pressure by reducing the production of aqueous humor (similar to the acute treatment of glaucoma), including beta-blocker drops such as timolol and carbonic anhydrase inhibitors such as acetazolamide. Additionally, hypomoric agents such as mannitol may also be used to try to decrease edema contri-
buting to increased IOP.7 These patients require prompt ophthalmol-
ogy evaluation and follow-up.

1. Liberally inject surrounding soft tissue with 1% lidocaine with epinephrine.

2. Advance a hemostat from the lateral canthus to the outer orbital rim and clamp to devascularize the tissue. Hold for 30-90 seconds.

3. Use small, sharp scissors (like scissors) to cut from the lateral canthus to the outer canthal rim.

4. Use forceps to reflect the lower eyelid to visualize the inferior canthal tendon.

5. Cut the tendon (yellow dotted line) to decompress the globe.

6. If this does not result in reduced IOP, repeat for the upper canthal tendon (green dotted line).

In summary, retrobulbar hemorrhage is an uncommon but time-
sensitive, highly morbid condition that carries an approximately 50% chance of vision loss for the patient. Timely recognition and intervention at bedside with a lateral canthotomy with cantholysis can help prevent further ischemic damage to the nervous structures of the eye and help to mitigate vision loss for the patient.


Dr. Bryant: I agree with clearing the clot and am an Afrin fan. I then put the plastic nose clip on and let the coagulation cascade do its thing for 15-20 minutes. This, honestly, is all that is required in most bleeds in people with normal anatomy, not post op, not traumatic, not on anticoagulants, normal platelet count, etc. Silver nitrate is a next line for me. I actually tend to avoid silver nitrate because it’s painful and damages tissue. I don’t fail. You can always (and I said always) hold the nasal speculum while you hold the suction in one hand and the nitrate sticks in the other hand. I never use silver nitrate alone. You will need a bunch of them. I also make a PCA or a nurse be standing there to hand me more as I need. I will often use 10 or more sticks to get a septal bleeder stopped.

AoBP: What do you do if your first approach fails to stop the bleeding?

Dr. Bryant: I have followed all steps correctly, it should work. If not, I pack the antrum with Vaseline gauze strip for 1 hour then come back and try again. If it still does not work, I repack and call or send to ENT the next day. Failure usually means there is a complication (ASA, anticoagulant use) or technique is not adequate.

Dr. Hooker: I don’t fail. You can always (and I said always) stop an anterior bleed. I guess you could use Gel-foam, but I have never had to. The key is adequate suction while using the nitrate sticks. You may need someone to hold the nasal speculum while you hold the suction in one hand and the nitrate sticks in the other hand. I never use a rapid rhino. They are miserable and unnecessary.

Dr. Bryant: Gel-foam is a reasonably next step. I try another round of the above, and then use a small rapid rhino after some topical lidocaine (the best plan is to aerosolize it and spray like you’re going to NP scope with the 4% lidocaine).

AoBP: Do you ever place bilateral packing?

Dr. Bryant: Only if both sides can be demonstrated to be bleeding.

Dr. Hooker: I use electrocautery. It is too dangerous. You can burn right through the septum. I only use silver nitrate. You will need a bunch of them. I also make a PCA or nurse be standing there to hand me more as I need. I will often use 10 or more sticks to get a septal bleeder stopped.

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Terri Joseph, MD

History of Present Illness

The patient is an African-American female in her mid-30s with a history of diabetes and previous abdominal pain. The patient presented to our ED with diffuse hypokinesis and an increased ejection fraction. Transthoracic Echocardiography revealed a severely reduced ejection fraction (18%). CT Abdomen/Pelvic showed a hypoattenuated wedge consistent with splenic infarct (Image 1). The patient had stigmata of systemic embolus (Image 1). Transthoracic Echocardiography showed a severely reduced ejection fraction of 20-25% with diffuse hypokinesis.

Hospital Course

The patient was started on enoxaparin with a coumadin for presumed left ventricular thrombus that had embolized to the spleen. The patient had stigmata of systemic embolus (Image 1). Transthoracic Echocardiography showed a severely reduced ejection fraction of 20-25% with diffuse hypokinesis.

The Canary is in the Spleen

Ryan LaFollette, MD

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Refractory heart failure in a patient with hypertension, diabetes, and prior acute myocardial infarction. The patient presented with worsening shortness of breath and orthopnea.

Dr. Trott: Our patient is a 50-year-old man with a history of hypertension, diabetes, and prior acute myocardial infarction. The patient presented with worsening shortness of breath and orthopnea.

Dr. Hooker: I think that the key is getting cocaine. With-
VZV Meningitis

Continued from page 8

VZV meningitis is a rare complication of VZV encephalitis. It is usually diagnosed by lumbar puncture, which reveals an increase in cerebrospinal fluid (CSF) pressure, pleocytosis, and elevated protein levels. The CSF glucose level is typically normal or only slightly decreased. The mortality rate of VZV meningitis is high, and early diagnosis and treatment are essential.


Preventing Post Lumbar Puncture Headaches

Step 1: Soak in sterile water for a full 30 seconds. (Not in saline)

Step 2: Have the patient gently blow their nose to rid the nasal cavity of any excess blood and clots.

Step 3: Insert the Rapid Rhino into the nasal cavity along the septal floor and parallel to the hard palate until the plastic proximal fabric ring is well within the nares.

Step 4: Using a 20ml syringe, inflate the Rapid Rhino with air. Stop inflation when the pilot becomes rounded and feels firm when squeezed.

Inserting a Rapid Rhino

Dr. Hooker: I perform bilateral nasal packing only when I have a posterior or unidentifiable source of bleeding. I use bilateral Foley catheters and 6 feet of gauze ribbons in each side. I then admit these patients to the ICU for observation.

Dr. Bryant: For the higher risk people (post op, traumatic, on anticoagulants, low platelet count) I have done the miserable thing of putting a rhino in both sides to get it stopped.

Dr. Trott: If the patient is sitting straight up, then a posterior bleed will be seen as active bleeding in the peritoneum leading to chronic right upper quadrant pain and which may require adhesiolysis surgery.

If history does not provide the clear answer; proper inspection with insertion of a nasal speculum can often help to define the origin of the bleeding.

If brisk bleeding continues, especially into the oropharynx, despite placement of bilateral anterior nasal packing, a posterior source of bleeding is likely.

If both history and physical examination still leave you in the dust, anterior nosebleed treatment failure may be the only method of differentiating the two. Although there is a paucity of evidence on this topic, it is reasonable to conclude that if brisk bleeding continues, especially into the oropharynx, despite placement of bilateral anterior nasal packing, a posterior source of bleeding is likely.

If the patient is sitting straight up, then a posterior bleed will be seen as active bleeding in the peritoneum leading to chronic right upper quadrant pain and which may require adhesiolysis surgery.

Given the presence of the patient's pain being isolated to above her umbilicus, and her initial denial of genito-urinary symptoms and low abdominal pain, a pelvic exam was not performed. In retrospect, this was an error which delayed diagnosis and increased the patient's ED length of stay. The "bilateral tubular space" of the CT pelvis were likely inflamed fallopian tubes from PID.

In summary, this was a common presentation of an uncommon disease. We should suspect Fitz-Hugh-Curtis in any sexually active female presenting with RUQ pain and have a low threshold to perform a pelvic exam early. Furthermore, in any female with abdominal pain for whom advanced imaging is being considered, a pelvic exam should be strongly considered, especially if there is a history of prior surgery or any doubt as to the reliability of the patient. Also remember that patients may downplay their symptoms, especially those involving sensitive issues or with potentially upsetting psychosocial ramifications.


The patient required intubation in the ED for airway protection and initial GCS of 3. He was rewarmed with warm IV fluids and a Bair Hugger warming unit. After 24 hours the patient’s temperature improved to 36.9°C (98.5°F). After rewarming the patient’s mental status improved and he was discharged on hospital day 5.

**EKG Changes Associated with Hypothermia**

1. Sinus bradycardia or atrial fibrillation with slow ventricular response. This is thought to precede more ominous arrhythmias such as ventricular fibrillation and asystole.
2. Interval prolongation; typically PR, QRS, and QT. This is due to slowing of myocardial conduction and prolongation of the cardiac cycle.
3. Presence of Osborn or “J” waves. The amplitude of the “J” wave is inversely related to the degree of hypothermia. They are commonly seen in the anterior and lateral precordial leads and lead II.

Irregular ventricular rate without P-waves consistent with atrial fibrillation. The heart rate in this patient is 61 bpm, showing a slow ventricular response.

The QT interval in this patient was 612ms. As heart rate slows the QT interval lengthens.

Osborn waves are a positive deflection in the terminal QRS.