Conference 10/7/2020

CCU follow up

-John Cook, PGY1

45 y/o male with syncope vs seizure. Patient had a syncopal episode en route and was pulseless during this episode. Did resolve upon arrival to the ED. With concerning rhythm strip. EKG: NSR, rate 65,1st degree AV block, LBBB, no ischemia

Continued to have syncope with pulselessness of 5-10 seconds. Patient had 2nd degree type II and 3rd degree AV block.

-placed on dopamine gtt and push does epi, and placed on epi gtt

-Cardiology placed transvenous pacemaker

Acquired Complete Heart block:

-ischemia/infarction

-Neuromuscular disorders

-Infectious: Chaga, Lyme's

-Metabolic

-Cardiomyopathy

-latrogenic causes: medications

Patient also traveled to places endemic to Lyme's disease Lyme disease: treatment Doxycycline Disseminated Lyme disease: IV Ceftriaxone 2g daily

MICU follow up

Blaine Jordan, PGY1

75 y/o transferred from OSH (Jewish) presented with Digoxin toxicity with hyperkalemia and bradycardia, and Warfarin toxicity. Along with lactic acidosis and sepsis positive

Labs: WBC 18.3, Trop 0.0, Potassium 4.9 (was higher), INR: 7.2 Digoxin level >9 (normal high is >2)

Digoxin Toxicity

-Digoxin causes increased inotropy, increases vagal tone and decreased conduction through SA and AV node -Toxicity:

Cardiac: arrythmias basically any arrhythmia.

GI: vague

Neurological: AMS

Metabolic: hypo/hyperkalemia

Hyperkalemia: acute toxicity

EKG: Digoxin effect: Salvador Dali – ST Depressions in lateral leads

U waves prominent, flattened inverted or biphasic T waves, shorten QT

Treatment: Digoxin Antibody Fragments (FAB)

-indications: life threatening/ hemodynamically unstable arrythmia, Hyperkalemia >5.5, evidence of end organ damage

-10 vials adult and 5 vials for children

-Calcium not recommended for HyperK, and treating hyperK does not decrease mortality

CALL POISON CONTROL!

Capstone

Cal Staben, PGY3

Hypothermia: Core Temp <35oC

-Potassium: >7 mortality outcomes increase

-Cardiovascular:

EKG Changes: Osborn Wave-J Wave (32oC and below), prolonged $PR \rightarrow QRS \rightarrow QTc$ prolongation, bradycardia (50% <28oC), Hypotension and decreased CO (50% at <25oC) Arrythmia: VF and asystole

Vasoactive and electricity is not going to help \rightarrow need to warm >30oC

-CNS

Losing Shivering <31, loss of corneal reflexes 23-25oC and flat EEG <19oC

-Respiratory

Bradypnea, decreased compliance, respiratory acidosis, change in airway anatomy (Trismus), drug physiology (decrease doses)

-Coagulopathy: Thrombocytopenia, PTT Prolongation and DIC

Rewarming

-passive rewarming, Warm IVF, Invasive rewarming (bladder/gastric/peritoneal/thoracic), Dialysis/ECMO Frost bite

1st degree: mild edema

2nd degree: superficial vesiculation, erythema, edema (bulky warm dressing, lance blisters)

3rd degree, hemorrhagic blisters (lance blisters and pharmacologic treatment)

4th: complete extension through dermis into muscle and bone (most likely amputation)

Treatment:

-Field Management -prevention-socks, pain control (ibuprofen 12 mg/kg/day), rewarming

-Hospital: consider thrombolytics if deep <24 hours after thaw, *<u>Iloprost (vasodilate and platelet aggregation)</u> <u>within 48 hrs</u> after thaw, source control (amputation)*

Clinical Pathway-Management of Pneumonia

Zach Heppner, Michael Carter PGY2

Pneumonia -new lung infiltrate plus clinical evidence that the infiltrate is of an infectious origin, with new onset fever, purulent sputum leukocytosis and increase O2 demand

Viral Pneumonia, Fungal Pneumonia, Mycobacterium (Tb), Bacterial

САР

-Strep pneumo, H flu, Moraxella,
-Atypical: mycoplasma, Legionella, Chlamydia pneumoniae
-Aspiration: Klebsiella, anaerobes
-high macrolide resistance don't use as monotherapy (especially in Kentucky)
HAP/VAP (multi-drug resistant)
-Staph aureus, Pseudomonas, ESBL-GNB, VRE
-HCAP is no longer a thing since 2016

<u>Risk Stratification scores</u> PSI/PORT Score-See MD calc (score >90 patient should come in to the hospital) CURB-65 -BUN >19, RR >30, SBP<90 or DBP <60, Age >65. Score >3 admission

MDRO Risk factures: MRSA, pseudomonas, ESBL, VRE -risk factors: **BIGGEST RISK FACTOR prior IV abx in 90 days**

Outpatient Abx therapy for CAP

-no comorbidities: <u>Amoxicillin</u> or Doxycycline or Macrolide (remember in Kentucky macrolide as monotherapy is a no no)

-Comorbidities: Augmentin or Cephalosporin AND macrolide or doxycycline. OR monotherapy with respiratory Fluoroquinolone (Moxifloxacin, gemifloxacin and Levofloxicin)

Inpatient treatment CAP

-non severe: Beta-lactam _Macrolide OR respiratory fluoroquinolone (if prior MRSA add coverage) -Severe: Beta lactam + Macrolide or Beta-lactam +Respiratory fluroquinolone (add coverage if MRSA previously).

Remember add coverage if IV abx in the past 90 days

COVID pneumonia

-RECOVER TRIAL

Effect of dexamethasone on 28-day mortality in COVID- mortality benefit based on severity Intubated group: more benefit

-Dexamethasone use

IDSA does not recommend

In severe pneumonia may cause more benefit

-Airway Isolation Precautions

Pathway- Please see Room 9er

Radiology-Chest CT

Dr. Van Bogaert

Protocols:

-CT chest with contrast: looking for confirmatory for pneumonia. You get okay visualizations of aorta, soft tissues and lungs.

-PE protocol: inject contrast looking at the pulmonary artery, and once it reaches a threshold \rightarrow then scan the patient and the contrast will be in the pulmonary arteries

-MAN, scan CT Chest abdomen and pelvis (a CTA chest essentially) – similar to PE protocol however shows aorta and its branches between

-CT non-Contrast: confirming pneumothorax, pleural effusion, pneumonia. You don't need to look at solid organs

Start on the inside and work your way out

-looking at vessels: window level abdominal angio

-looking on sagittal view can help you see pseudoaneurysm and confirm dissections

-always look at pulmonary arteries: incidental PE's can be found. Also looking at the sagittal view can help you view smaller PE's

Pulmonary vein is not as opacified as the pulmonary artery, so make sure you are looking at the artery for PE. Follow the filling defect back and the pulmonary vein will go into the atrium

Lung window

- Good for pneumothorax, pulmonary nodules show up well
- Look for bronchial injuries
- Pneumonias
- Pulmonary contusions

Bone Window

- Especially look in trauma patients

- Sagittal images are good for chest trauma- sternal fractures and first rib fractures, scapular fractures Best way to approach- step by step look at individual structures

Vessels \rightarrow heart, \rightarrow mediastinum, \rightarrow airway \rightarrow lungs \rightarrow bones

Toxic Gases

Dr. Bosse

Nerve Agents: potent organophosphates

- Usually a liquid
- Depends on temperature and pressure: can be a fine line between gas and a liquid/solid
- Toxic Gas Classification
 - o Irritants
 - High water and lower water solubility
 - High: rapid onset and upper airway
 - Poor: delayed onsent and lower lung injury
 - Simple asphyxiants
 - o Systemic (formerly chemical) asphyxiants

Anhydrous ammonia:

- Fertilizer, pesticide, pharmaceuticals, refridgerater gas, disinfectant
- Highly water soluble \rightarrow ammonia hydroxide and you get chemical burns
- Sx: nasal and throat eye irritation
- Tx: remove from source, symptomatic and supportive care

Chlorine

- Manufacturing non agricultural chemicals, paper industry, household bleach, water purification
- DON'T MIX HOUSEHOLD BLEACH and ACIDIC DRAIN CLEANER
- Intermediate water solubility, heavier than air and reactive and explosive
- Tx: remove from source control and supportive care
 - o Neutralization with nebulized sodium bicarb

Phosgene

- Low water solubility, colorless gas and 3.4x heavier than air
- Can smell like "musty hay"
- Can cause latent development of lung injury
- Tx: removal from source and symptomatic and supportive care
- Admission for observation of delayed signs and symptoms

Asphyxiants

Simple asphyxiants displace oxygen from environment (Helium, CO2, Nitrogen, methane. Ethane, CO)

- Signs and s related to hypoxemia
- Treatment: remove form source, supplemental oxygen, supportive care

Systemic

- Inhibit binding of O2 to the cells.
- Carbon monoxide, cyanide and hydrogen sulfide
- **CO**
 - Fires, engine exhaust, heating equipment, methylene chloride, cigarettes, endogenous production
 - Bind to Hbg and forms Carboxyhemoglobin

- Clinical effects: cardiac (tachycardia, arrhythmias) and neurologic (AMS, seizures, HA Very common)
 - Delayed neurological effects: Parkinsonism, peripheral neuropathy, behavioral disturbances, incontinence, dementia
- Lab Dx: Carboxyhemoglobin, can be determined on a VBG
- Tx: oxygen, and hyperbaric oxygen (does it work)
 - HBO indications: syncope, coma, seizure, AMS, COHbg >25% and pregnancy >15%

- Cyanide

- Plants, labs, industry, *nitroprusside*, laetrile (Cancer tx), acetonitrile, combustion/smoke inhalation
- Clinical manifestations: involvement of oxygen sensitive organs: CV and neurologic manifestations. May have a brady arrhythmia prior to demise
 - Bitter almond odor
- Labs: CN levels, metabolic acidosis, lactic acid
- Tx remove from source and decon with protective gear for liquid or solid exposures. Then symptomatic and supportive care
- Antidotes: nitrites, Thiosulfate and hydroxocobalamin
 - Indications for hydroxocobalamin- *Per Bosse* hemodynamic instability, metabolic acidosis with elevated LA, AMS
- Hydrogen Sulfide
 - o Bacterial decomposition of protein, sewer gas, industry, inhibition of Cytochrome oxidase
 - Antidote: amyl nitrite and sodium nitrite