University of CINCINNATI
Emergency Medicine
ANNALS OF B POD
Since 1970 - Leadership - Excellence - Opportunity
Volume XI Issue IV
Summer Issue 2018

B POD HIDDEN FROM SIGHT

mimics and imitators

ANNALS OF B POD
SUMMER 2018

B pod poses a unique environment where providers are faced with challenging and interesting cases on a daily basis. In the Summer 2018 issue of Annals of B Pod, we focus on the hidden diagnoses that can lurk within the pod. From stroke-like presentations to baclofen withdrawal, this issue details the intricacies of managing these challenging cases. Explore the evidence and master the management of these complex and interesting cases in this issue of Annals of B Pod.

bpodcase

Stroke Mimics

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History of Present Illness

The patient is a female in her sixties who presents to the emergency department (ED) with a chief complaint of fall. Her symptoms at initial evaluation include right upper extremity weakness and sensory loss, right lower extremity weakness, and neck pain.

The patient reports that earlier in the evening, she had consumed a significant amount of wine before retiring to bed. Her partner was with her during this time and states she was last seen normal approximately 120 minutes prior to arrival to the ED. She subsequently fell when attempting to get up from bed to use the restroom. The patient provides variable history as to whether there was right leg weakness precipitating the fall or whether the right leg weakness developed after the fall. She denies any prodromal symptoms preceding the fall. She does endorse striking her head but denies loss of consciousness. First responders report that the patient had initially complained of right hand numbness and weakness on scene. However, there are no complaints of facial weakness, headache, vision changes, nausea, vomiting, or abdominal pain. The patient does not use anticoagulants.

Past Medical History
Transient ischemic attack
Hypertension
Hyperlipidemia
Renal cell carcinoma

Past Surgical History
Roux-en-Y gastric bypass
Nephrectomy

Medications
Buspirone
Gabapentin
Lorazepam

Vitals

T 97.5 HR 74 BP 147/86 (101) RR 14 SpO2 100%

Physical Exam

The patient appears her stated age, in a cervical spine collar and in no apparent distress. She has some small abrasions and a small area of ecchymosis to the forehead; otherwise the ear, nose, and throat exams are unremarkable. There is no midline spine tenderness. Pulmonary and cardiac auscultations are normal. The abdomen is soft, non-distended, and non-tender. The skin is warm and well perfused, and there are strong peripheral pulses in bilateral upper and lower extremities. Cranial nerves are intact with no dysarthria, aphasia, or facial weakness. She cannot lift her right upper or lower extremities against gravity. She has diffusely reduced sensation to light touch throughout the right upper and lower extremities.

Labs

	12.4	
5.7		243
	37.9	

139	103	15	115
4.1	25	1.23	

Trop: <0.04

INR: 1.1

Imaging

CT Head: No evidence of intracranial hemorrhage or mass effect. Mild narrowing of the lateral ventricles and mild sulcal effacement out of proportion for patient's age.
CT-Angiogram Head/Neck: Normal
CT Cervical spine: Multilevel cervical spondylosis, worst at C4-5 without acute fracture or listhesis.

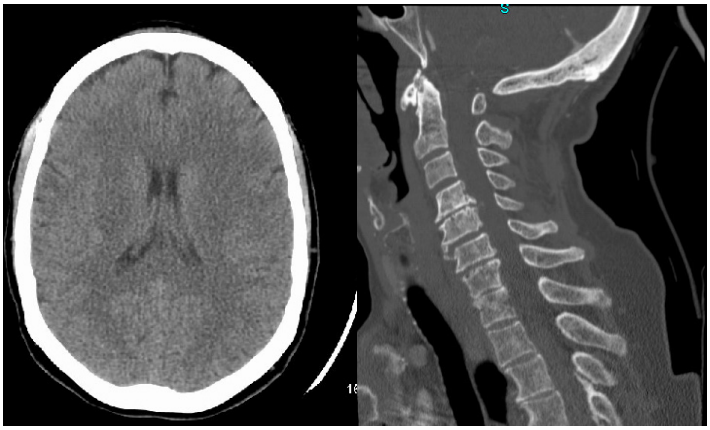

Image 1: Normal non-contrast head and cervical spine CT

Hospital Course

The patient's presentation was concerning for stroke. The patient had multiple risk factors for stroke including a prior history of a transient ischemic attack (TIA). The patient arrived to the ED within the window for thrombolytic intervention for acute stroke. After appropriate history taking, a thorough physical examination with an NIH stroke scale of 4, and imaging evaluation, it was felt that she did not have any contraindication to the use of systemic thrombolytics and would benefit from treatment given the degree of impairment. The stroke team was consulted, reviewed the case, and evaluated the patient at bedside. After discussion of the risks and benefits with the patient, tPA was initiated with a bolus approximately 60 minutes after arrival to the ED (door to needle) and approximately 180 minutes after the patient's "last seen normal" time.

The patient underwent an MRI head without contrast approximately twelve hours after tPA administration that showed no acute infarct, hemorrhage, or mass. An MRI cervical spine at that time revealed a large right central disc osteophyte complex at C4-C5, with severe central canal stenosis and cervical spinal cord compression. Interestingly, there was no midline spinal tenderness on exam, both the upper and lower extremities were affected, and CT of the cervical spine showed no acute fracture.

Given these findings, neurosurgery was consulted and evaluated the patient. They found an essentially unchanged physical exam with an NIH stroke scale of 4 with the same deficits. The patient underwent an anterior cervical discectomy and fusion (ACDF) at C4-C5 with polyether ether ketone (PEEK) implant placement on hospital day two following normalization of coagulation testing. She was discharged to inpatient rehab, and at post-op day 14 was found to have improved right upper and right lower extremity strength and was able to ambulate with a walker.

Discussion

This case represents a stroke mimic presenting to the ED that was treated with systemic thrombolytics. While spine injuries are an uncommon etiology of stroke mimic, this case reminds us that ED physicians must maintain a wide differential diagnosis even in the midst of directing a rapid imaging evaluation and minimizing door-to-needle times for patients with acute stroke-like symptoms.

In an aging population with increased incidence of chronic disease, the incidence of both stroke and stroke mimics can be expected to increase over time in developed countries.[1] Stroke mimics have been estimated to represent 5-31% of stroke presentations to the ED.[2] Seizures, syncope, and sepsis were the most common respective etiologies in one meta-analysis, together comprising about half of stroke mimic patients.[3] A substantial proportion of these patients can be classified as having no neurologic cause (*i.e.*, functional). One single-center study of admissions to a hyperacute stroke unit listed psychiatric conditions as final diagnoses in more than one-third of cases.[4] However, many etiologies of stroke mimics have therapies that should be instituted quickly by emergency providers.

High on the list of treatable stroke mimics is hypoglycemia. ED physicians must measure glucose (with intervention and re-evaluation as needed) prior to thrombolysis because isolated hypoglycemia can produce acute neurologic deficits. While expanded use of prehospital glucometers offers significant benefits, physicians should repeat the glucose testing on arrival to the ED.

Emergency physicians must always consider additional cardiovascular pathology in a patient who presents with neurologic defecits, especially aortic dissection. Prior to administering systemic thrombolytics, ED physicians should always ask about any history of chest pain and obtain a screening chest x-ray to evaluate for a widened mediastinum. Although aortic dissection cannot always be completely ruled out prior to thrombolytics, emergenyc physicians should keep this high on the differential.

Seizures and their sequelae which include post-ictal state, focal seizures, or Todd's paresis can produce objective neurologic deficits even on serial exams. Syncope may be initially diagnosed as a stroke or insufficiency of the vertebrobasilar system, particularly if additional

Continued on page 11

Neurosyphilis

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History of Present Illness

The patient is a male in his thirties with a past medical history of Human Immunodeficiency Virus (HIV) who presents to the emergency department (ED) with a rash and eye pain. The rash has been present for the past six weeks and started as red spots on the trunk that spread to the extremities, including his palms and soles. The rash is neither pruritic nor painful, and recently his hands have begun to peel. He denies new sexual or environmental exposures. For the past week, he has been taking oral prednisone that was prescribed by his primary care provider, but his rash has not improved.

The patient also reports bilateral eye pain. The pain started in his right eye with purulent drainage and blurry vision one week ago. The symptoms then spread to his left eye several days later. He was diagnosed with bilateral uveitis at an outside facility and was transferred to a tertiary care center for further work up.

Past Medical History

HIV (Diagnosed 2012)
Previous STI Exposure

Past Surgical History

None

Medications

Prednisone

Allergies

None

Vitals

T 98.0 HR 103 BP 121/65 RR 22 SpO2 97%

Physical Exam

The patient appears thin and older than his stated age but is in no acute distress. He has bilateral conjunctival injection. Visual acuity is 20/150 in the right and left eyes individually. Intraocular pressures are within normal limits. Cell and flare are present bilaterally on slit lamp examination. Examination of the oropharynx shows multiple white patches along the buccal mucosa, uvula, and posterior oropharynx. Bilateral cervical and inguinal adenopathy are present. Lung, cardiac, and abdominal examinations are unremarkable. There is a diffuse erythematous maculopapular rash involving bilateral palms where desquamation is also noted (Image 1). On neurologic exam the patient has a normal mental status, is moving all four extremities, and has normal cranial nerve function.

Hospital Course

Ophthalmology was consulted for the patient's bilateral uveitis. Their examination revealed pan-uveitis concerning for ocular syphilis. This was additionally supported by his rash consistent with secondary syphilis and untreated HIV. A broad work-up was initiated to evaluate for additional causes of his pan-uveitis. The patient was given prednisolone, cyclopentolate, and phenylephrine eye drops and admitted to the intensive care unit for frequent drop administration. Dermatology and infectious disease were consulted on admission. Shortly into the patient's hospital course, the patient's CD4 count resulted at 71 with a high viral load. Trepia (Treponema pallidum agglutination assay) and rapid plasma regain (RPR) studies were also positive. Biopsies of the rash were consistent with secondary syphilis. Lumbar puncture was performed and CSF VDRL titer was 1:4 indicating neurosyphilis. His hospital course was complicated by gram-positive and gram-negative bacteremia. He was discharged with an improvement in his vision and rash after completing two weeks of intravenous penicillin. He continues to be seen by ophthalmology, dermatology, and infectious disease in the outpatient setting for ongoing treatment and monitoring of neurosyphilis and acquired immune deficiency syndrome (AIDS).

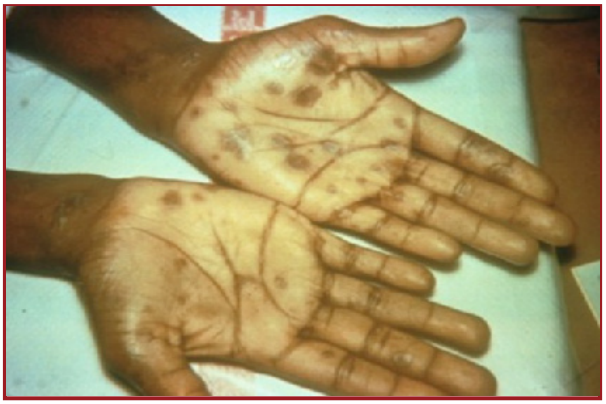

Image 1: Representative image of the palmar rash seen in secondary syphilis.[19]

Discussion

Syphilis is a sexually transmitted infection caused by the spirochete *Treponema pallidum*. If left untreated, the disease can progress through four clinical stages: primary, secondary, latent, and tertiary. After the primary stage of the disease, patients can present with multiple symptoms that mimic other infectious processes and diseases. Syphilis is

therefore known as "the great impostor."

The incidence of syphilis in the United States has varied since rates of infection have been recorded starting in the early 1940s. Penicillin was introduced in the late 1940s and infection rates significantly decreased.[1] In the 1980s, there was a surge secondary to increasing intravenous drug use and prostitution.[1] In 2000, the incidence of syphilis was at an all-time low after programs for aggressive screening and primary prevention were implemented.[1] However, syphilis has since been on the rise in recent years. The annual rate of syphilis has increased every year from 2005 to 2016.[1] In 2016, there were 27,814 cases of primary and secondary syphilis reported in the United States. This was a 17.6% increase from 2015 and 74.0% increased from 2012.[1] Sexually transmitted infections are on the rise in general and research is on-going to determine the cause. Some postulate that the rise of dating apps may play a role.[2]

Stage	Timing	Features
Primary	3-4 weeks after exposure	Painless chancre at inoculation site
Secondary	4-8 weeks after appearance of primary chancre	Rash, condyloma lata, systemic symptoms common
Latent	Early: <1 year after infection Late: >1 year after infection	Asymptomatic
Tertiary	1-10 years after infection	Benign: gummatous lesions Cardiovascular: aortitis, coronary arteritis CNS: tabes dorsalis, paresis

Table 1: Stages of Syphilis based on timing of initial infection and characteristic signs and symptoms

Primary syphilis classically presents with a chancre at the inoculation site. The chancre initially forms as erythematous papules that progresses to a painless ulcer. Spirochetes are present in these lesions and spread systemically via the lymphatic and hematopoietic systems. Secondary syphilis occurs four to ten weeks after the initial infection and can produce a wide variety of signs and symptoms. It is characterized by a generalized maculopapular rash that frequently involves the palms and soles. Secondary syphilis is classically characterized by condylomata lata which are papules at mucocutaneous junctions. Systemic symptoms such as fever, malaise, headaches, joint pains, and anorexia are common. Hepatic, renal, ocular, and central nervous system involvement also occur at this stage. The latent phase follows the secondary phase, and is a prolonged asymptomatic period following the secondary phase.

The latent stage is dichotomized into early and late stages. Early latent stage occurs less than one year after primary infection and late latent stage occurs greater than one year after primary infection. Tertiary syphilis is due to an obliterative endarteritis that can affect any organ system but is divided into three subtypes: benign, cardiovascular, and central nervous system. Benign tertiary syphilis presents with granulomas anywhere in the body that are called gummatous lesions. The cardiovascular subtype involves the aorta and coronary arteries and patients can present with classic symptoms of acute coronary syndrome. Patients with the central nervous system subtype present with tabes dorsalis and general paresis. Some infectious disease groups also describe a quaternary stage that is an aggressive form of neurosyphilis in patients with AIDS and causes necrotizing encephalitis.[3]

Neurosyphilis can occur any time after primary infection and is much more common in patients with HIV infection.[4] Infection of the central nervous system begins early, and patients with primary syphilis may have some degree of aseptic meningitis. Over time, the infection may progress and involve more structures in the central nervous system. Early infection generally affects the meninges and vasculature. Late infections progress to involve the parenchyma of the brain and spinal cord. There are six subtypes of neurosyphilis based on the clinical symptoms and structures involved. These include asymptomatic, acute syphilitic meningitis, meningovascular syphilis, tabes dorsalis, general paresis, and optic atrophy (Table 2). Although described below as separate entities, these syndromes can overlap.[5]

Asymptomatic Neurosyphilis

Asymptomatic neurosyphilis is characterized by positive VDRL serology in the CSF with no signs or symptoms of neurologic disease. CSF studies also show elevated protein and a very mild lymphocytic pleocytosis. This stage may resolve spontaneously without treatment.

Acute Syphilitic Meningitis

Patients with acute syphilitic meningitis present with classic symptoms of meningeal irritation including headache, meningismus, nausea, and vomiting. Fever is not always present, especially if the patient is immunocompromised. Cranial neuropathies can also be present, with cranial nerve seven involved most commonly.

Meningovascular Syphilis

Meningovascular syphilis is a result of neurovascular endarteritis. There is fibroblastic proliferation of the intima of the cerebral blood vessels and luminal narrowing. Patients are more vulnerable to cerebrovascular thrombosis and present with stroke. This occurs about seven years after the initial infection if left untreated.

Continued on page 12

bpodcase

Baclofen Pump Failure

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History of Present Illness

A female in her 60s with a history of spastic quadraparesis secondary to paraneoplastic syndrome following breast cancer, currently managed by an intrathecal baclofen (ITB) pump, presents to the emergency department (ED) for concerns of baclofen pump malfunction. The patient has had changes in her speech, increased rigidity, tremors, and diaphoresis. Tthe patient's baclofen pump began to alarm at her skilled nursing facility early that day. The patient had a previous baclofen pump malfunction in 2013 that resulted in intubation and admission to an intensive care unit. The patient's intrathecal baclofen pump was placed in September 2005 by a neurosurgery group at an outside hospital and is currently managed by Physical Medicine and Rehabilitation (PMR).

Past Medical History

Ductal Carcinoma in Situ, Paraneoplastic Syndrome, Pulmonary Embolism

Past Surgical History

Anterior cervical decompression with fusion, Right breast needle core biopsy, Baclofen intrathecal pump insertion

Medications

Intrathecal Baclofen
Fluoxetine

Allergies

No known

Lab Work-up

WBC 14.7 H/H 14.5/42.6 Plt 312
BMP: 139/4.6/108/22/14/0.54/88
CK: 127
UA: -Nit/-LE/9 RBC/8 WBC

Vitals

T 98.1 HR 131 BP 194/114 RR 26 SpO2 98%

Physical Exam

The patient appears her stated age and is slow to respond, but oriented to person, place, and time. There are bilateral lower and right upper extremity contractures. The patient appears diaphoretic. She is tachycardic and tachypneic, but protecting her airway. Abdominal, cardiovascular, and pulmonary exams are otherwise benign. Neurologic exam is difficult to assess given the patients baseline contractures, but she is able to follow simple and two step commands, and no cranial nerve deficits are noted. There is no skin erythema overlying her baclofen pump and no alarms heard.

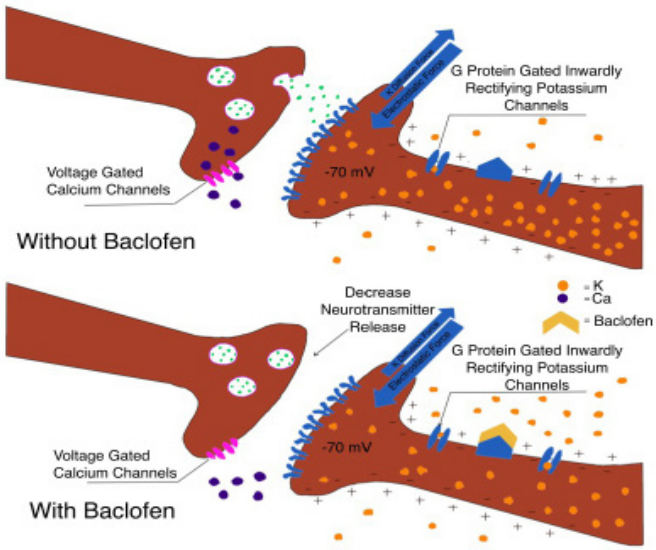

Figure 1: Diagram of synaptic cleft in the presence and absence of Baclofen.

Hospital Course

PMR was consulted and confirmed pump malfunction and the patient was quickly diagnosed with early baclofen withdrawal. She was given a total of 8 mg of lorazepam over 2 hours with little response. Pump replacement was recommended in addition to high dose oral baclofen in the interim for withdrawal symptoms. Neurosurgery was also consulted for operative repair or replacement of her baclofen pump. While in the ED, the patient became febrile with continued altered mental status, hypertension, tachycardia, and tachypnea. A dexmedetomidine drip was started in the ED and she was admitted to the Neuroscience Intensive Care Unit (NSICU) for further management of baclofen pump failure and baclofen withdrawal.

Throughout her hospitalization, the patient required several different medications for the management of baclofen

withdrawal including dexmedetomidine, lorazepam, cyproheptadine, oral baclofen, fentanyl, and diazepam. Neurosurgery successfully replaced the patient's intrathecal baclofen pump on hospital day two. PMR slowly up-titrated her intrathecal baclofen while weaning the multiple additional medications used to control her baclofen withdrawal. Her hospital course was complicated by sepsis secondary to a urinary tract infection which improved with antibiotics.

The patient was transferred from the NSICU to the neurology floor team on hospital day six. On hospital day nine, the patient's vital signs had normalized, she was at her baseline mental status, and her spasticity was improving. The patient was discharged from the hospital to a skilled nursing facility with PMR follow-up. At discharge, the patient's oral baclofen and cyproheptadine were still being down titrated.

Ten days following discharge the patient was seen by PMR at an outpatient visit and no adjustments to the baclofen pump were made at that time. The patient again presented to the ED 12 days after hospital discharge for lethargy concerning for baclofen toxicity. PMR was again consulted and her baclofen pump infusion rate was decreased. The patient was admitted to the medicine service and an extensive altered mental status work-up was completed and unremarkable. Her mental status returned to baseline following baclofen pump adjustment and she was discharged back to her nursing facility.

Discussion

Baclofen is a gamma-amino butyric acid (GABA) agonist that acts at both pre-synaptic and post-synaptic GABA receptors. Pre-synaptically, baclofen prevents calcium influx. Calcium is required for neurotransmitter release on the presynaptic terminal, therefore baclofen decreases the amount of presynaptic neurotransmitter release.[1] Post-synaptically, baclofen increases potassium efflux. A potassium ion gradient is present with high potassium levels within the neuronal stroma and low potassium levels in the extracellular fluid. This potassium gradient creates an overall negative resting membrane potential.

Baclofen opens potassium channels, resulting in increased efflux of potassium and neuronal hyperpolarization. This decreases the ability of the neuron to depolarize. Clinically, this results in inhibition of neurons. Therefore, baclofen is used in spastic conditions where imbalance of neuronal activity leads to increased excitation at the neuromuscular junction.

Baclofen has been found to be very effective in reducing spasticity when compared to other drugs. It exerts its effect by decreasing the hyperactivity of stretch reflexes, cutaneous reflexes, muscle spasms and clonus.[2] Baclofen does not readily cross the blood brain barrier, so high doses of oral baclofen are often required to achieve effect. These high oral doses result in many undesired side effects including muscle weakness, nausea, somnolence, and paresthesia. These side effects are experienced in approximately 25% - 75% of patients treated with oral baclofen.[2] The dose of baclofen can be decreased by greater than 100-fold by delivering ITB via a pump. The pump has an attached reservoir and is implanted in the abdominal wall, which is connected to a catheter extending into the anterior spinal canal and enters the intrathecal compartment. The rate and schedule of baclofen delivery is then titrated to effect.

Baclofen pumps are also associated with side effects. These are often related to a failure of delivery of baclofen to the intrathecal space, resulting in baclofen withdrawal. The rate of complications associated with ITB delivery is reported to be between 24-40%.[5,6] Complications more commonly involve catheter malfunction (catheter kink, migration, and disconnection) as opposed to an empty reservoir or failure of the pump itself. Symptoms of baclofen withdrawal will occur 24-48 hours after decreased delivery of baclofen to the intrathecal space. The presentation is often consistent with withdrawal from other central nervous system depressants such as benzodiazepines and alcohol. The patient will often present with neuropsychiatric symptoms (altered mental status, hallucinations, and confusion), increased spasticity, hypertension, tachycardia, hyperthermia, and seizures. It is important to differentiate the patient's current spasticity from the patient's baseline, as this patient population will have spasticity at baseline.

While baclofen withdrawal must be kept at the top of the differential for any patient with an ITB pump presenting with altered mental status and the previously described vital sign abnormalities, there are many other conditions which should be considered by emergency physicians. The differential diagnosis of baclofen withdrawal includes autonomic dysreflexia, sepsis, serotonin syndrome, neuroleptic malignant syndrome, malignant hyperthermia, benzodiazepine withdrawal, and alcohol withdrawal (Table 1).[1] These should be considered in every patient that presents with symptoms resembling baclofen withdrawal as this patient population is often at high risk for infection and polypharmacy. It is not unreasonable to undergo an infectious workup including blood and urine cultures while empirically treating for a presumed infection.

While the diagnosis of baclofen withdrawal is made on purely clinical grounds, the determination of a cause will further solidify the diagnosis. Imaging modalities such as an upright abdominal X-ray or CT scan are able to characterize the catheter and evaluate for discontinuity, kink, dislodgement, or other mechanical causes inhibiting the delivery of ba-

Continued on page 14

bpodcase

Spontaneous Pneumomediastinum

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History of Present Illness

The patient is a male in his mid-20s with a past medical history of spontaneous pneumomediastinum who presents to the emergency department (ED) with sudden onset shortness of breath and a "chipmunk"-like change in his voice. He denies any associated chest pain, fevers, cough, or leg swelling. He was diagnosed with spontaneous pneumomediastinum when he presented with similar symptoms.

Past Medical History
Spontaneous Pneumomediastinum

Medications
None

Past Surgical History
None

Allergies
None

Vitals

T 37 HR 113 BP 137/96 RR 12 SpO2 97% room air

Physical Exam

Physical exam reveals a well-appearing male in no distress. He is speaking in a nasal sounding, "hot potato" voice. Respiratory effort is normal with good air movement bilaterally. Heart rate is regular without murmurs, gallops, or rubs. Abdomen is soft, non-distended, and non-tender. Neurologic exam is normal.

Labs & Imaging

14.1			
14.5	173		
41.6			
139	103	10	81
3.3	29	1.05	

VBG: pH 7.41/pCO2 46/PO2 31/BE 3.6

Portable CXR: Questionable pneumomediastinum (Image 1).

CT Neck Soft Tissue with IV Contrast: Extensive pneumomediastinum extending into the neck, right greater than left.

CT Chest: Extensive pneumomediastinum, primarily along the aortic arch, trachea, and esophagus with extension to the diaphragmatic hiatus and cervical regions. No pneumothorax (Image 2).

Hospital Course

Thoracic surgery was consulted and recommended a fluoroscopic esophagram that did not reveal any evidence of a leak. He was admitted to the ED observation unit for close monitoring. The following day, a repeat chest x-ray showed stable pneumomediastinum, and repeat labs showed resolution of his leukocytosis. He remained afebrile and hemodynamically stable during his stay. He was able to tolerate a full diet without difficulty and was discharged 25 hours after presentation with close follow-up with his primary care physician.

Discussion

Pneumomediastinum and subcutaneous emphysema were first noted to occur during childbirth in the early 17th century.[1] It was not until the mid-20th century when Dr. Louis Hamman thoroughly described the multiple clinical features associated with pneumomediastinum.[2] In fact, the classic crunching sound appreciated with cardiac motion was described by Dr. Hamman and subsequently named Hamman's crunch.[3]

The epidemiology of pneumomediastinum largely depends on the underlying etiology and associated patho-

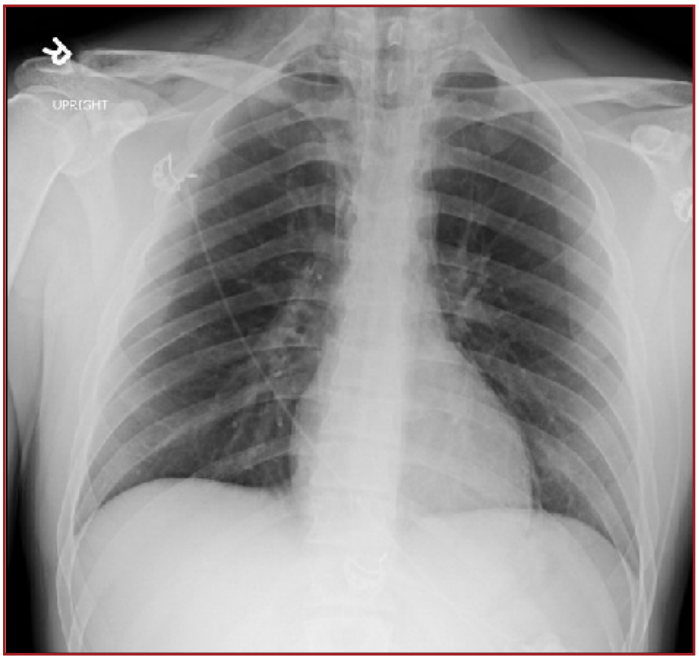

Image 1: Portable CXR showing questionable pneumomediastinum

physiology. Pneumomediastinum can be spontaneous or from a secondary cause. Predisposing conditions for spontaneous pneumomediastinum include smoking, asthma, idiopathic pulmonary fibrosis, and chronic obstructive pulmonary disease.[4] There have been case reports of spontaneous pneumomediastinum in patients with Ehlers-Danlos Syndrome, anorexia, and congenital adrenal hyperplasia.[5,6] Secondary causes include blunt or penetrating thoracic trauma, barotrauma during mechanical ventilation, rupture of a hollow viscus, infection from a gas-forming organism, recent interventions to the esophagus, or instrumentation of the tracheobronchial tree.[4]

One study found that one in every 368 people hospitalized for unexplained chest pain and no specific risk factors for a secondary pneumomediastinum were found to have spontaneous pneumomediastinum.[7] An underlying lung disorder was identified in up to 44 percent of patients with spontaneous pneumomediastinum.[3] Men are more likely to have both secondary and spontaneous pneumomediastinum.[3] Women are at increased risk of pneumomediastinum during labor.[8] The mean age for spontaneous pneumomediastinum is 27 compared to 39 for secondary pneumomediastinum.[4]

Macklin and Macklin first described the pathophysiology of spontaneous pneumomediastinum in 1944. They described rupture of alveoli under high pressure causing air to travel through the bronchovascular sheath into the mediastinum. Air continues to dissect through facial planes to other areas of the body such as the subcutaneous tissues, pleural space, peritoneum, retroperitoneum, pericardium, and intravascularly.[9] Precipitating factors of spontaneous pneumomediastinum cause elevated alveolar pressures and include inhalational drug abuse, emesis, upper respiratory infections, asthma exacerbations, diabetic ketoacidosis, and physical activities or straining.[3,10] Providers should attempt to identify any of these associated precipitating factors while obtaining the history of present illness.

Chest pain, cough, and dyspnea are the most common presenting symptoms.[3,4] Other presenting symptoms include dysphagia, dysphonia, neck pain, and lightheadedness.[3] Up to 92 percent of patients will have evidence of subcutaneous crepitus and up to 52 percent of patients will have Hamman's crunch.[8]

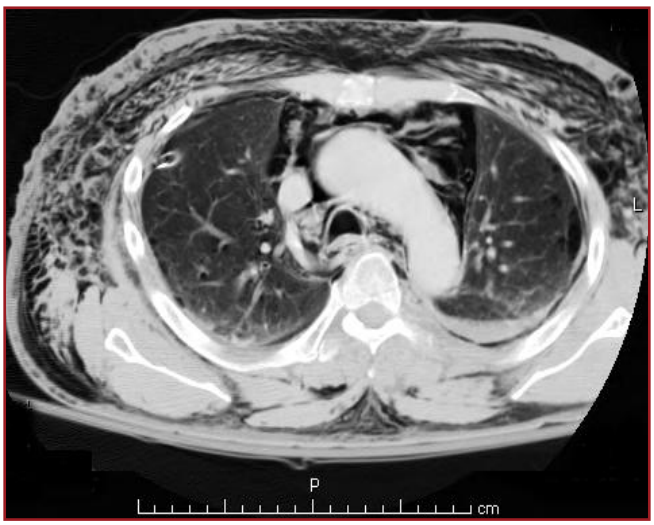

Image 2: Representative CT of pneumomediastinum and subcutaneous air [17]

Low-grade fever may be seen secondary to reactive inflammation.

Patients will often have leukocytosis in the acute setting, but this is a non-specific finding. There is no EKG finding that is pathognomonic for pneumomediastinum, although low voltage or an axis deviation might be seen.[8] Chest x-ray is the primary imaging modality to identify spontaneous pneumomediastinum and identifies 90 percent of cases. Ultrasound may reveal diffuse A-lines and poor visualization of the heart in parasternal and apical windows.[11] If a chest x-ray is normal but clinical suspicion is still high, a CT scan of the chest should be performed.[10] If pneumomediastinum is found on imaging, it is important to evaluate for an underlying insult before ruling it a spontaneous pneumomediastinum.[4] The esophagus specifically needs to be examined with contrast imaging studies to rule out esophageal catastrophe.[10,12] Esophageal rupture (Boerhaave syndrome) should be considered in patients with a history of severe vomiting, abdominal tenderness, pleural effusions, pneumopericardium, or pneumoperitoneum.[13] Once deadly causes of pneumomediastinum are ruled out, the diagnosis of spontaneous pneumomediastinum can be established.

Treatment should be targeted toward any identified underlying etiology. If esophageal rupture is identified, the patient will require emergent surgical consultation and broad-spectrum antibiotics.[14] If tracheobronchial tree rupture is discovered, the patient will require cardiothoracic surgery consultation and likely operative management. If pneumomediastinum occurs in a ventilated patient, PEEP and tidal volume should be minimized while maintaining appropriate oxygenation and ventilation. Any reversible causes of air trapping like bronchospasm should treated accordingly. Underlying pre-existing factors like obstructive pulmonary disease should be treated adequately and any cough should be suppressed using antitussives.[13]

Most patients who are ultimately diagnosed with spontaneous pneumomediastinum are admitted to the hospital for observation, analgesics, rest, oxygen therapy, and occasionally antibiotics. It is reasonable to administer a broad spectrum antibiotic intially, such as a 3rd generation cephalosporin, but do not need to be continued if the patient is afebrile and the cause of the pneumomediastinum is not due to esophageal perforation. Treatment with 100 percent supplemental oxygen will enhance reabsorption of mediastinal air six-fold.[8,13] The duration of supplemental oxygen therapy varies based on consultant recommendations but should be continued until clinical or radiographic improvement.

Complications include tension physiology of the pneumomediastinum compressing the heart and great vessels, pneumopericardium, pneumorrhachis (free air in the spinal canal), and pneumothorax.[10] If a pneumothorax is present, tube thoracostomy is recommended.[15] Tension pneumomediastinum may

Continued on page 15

Esmolol in Refractory Ventricular Fibrillation

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Approximately 209,000 cardiac arrests occur within the hospital each year.[1] Ventricular fibrillation is a common presenting rhythm for those that arrest from primary cardiac etiologies. The American Heart Association Guidelines currently recommend high quality CPR, defibrillation, and the administration of epinephrine and amiodarone for patients that present in pulseless ventricular tachycardia or ventricular fibrillation.[2] If the rhythm persists after three defibrillation attempts and administration of amiodarone, it is considered to be refractory ventricular fibrillation (RVF) and is associated with higher mortality rates.[3-4]

During cardiac arrest, patients may have high levels of exogenous catecholamines due to the administration of epinephrine. Epinephrine works on the α-1 receptors causing vasoconstriction in addition to the β-1 and β-2 receptors causing increased myocardial contractility and bronchodilation.[5] The activation of beta-receptors could lead to harmful effects that include an increase in myocardial oxygen demand, worsening ischemic injury, and a lowering of the arrhythmia threshold. Therefore, there is a theoretical benefit to administering beta-blockers for patients with RVF as beta-blockers would inhibit the deleterious effects caused by the beta agonistic properties of epinephrine.[6-7]

Pharmacokinetics of Esmolol	
Onset of Action	2-10 minutes (Quickest with loading dose)
Duration of Effect	10-30 minutes (Prolonged with high doses)
Half Life	9 minutes
Metabolism	Red blood cell esterases

Table 2: Summary of package insert for esmolol hydrochloride

Beta-blockers have been evaluated in human and animal studies in RVF.[8] It was found that esmolol has the most favorable pharmacokinetic profile due to its very rapid onset and short duration of action (Table 1).[9] Esmolol is a selective beta-blocker that competitively blocks the β-1 receptors with no intrinsic sympathomimetic or membrane stabilizing activity.

No prospective studies have evaluated the use of esmolol of in RVF, but a few retrospective studies assessing esmolol in RVF in humans have been completed.[6-7] A retrospective observational analysis from January 2011 to January 2014 was conducted to evaluate outcomes of patients who received esmolol versus patients who received standard of care in the emergency department during RVF.[6] Twenty-five patients were evaluated, six of whom received a loading dose of 500 mcg/kg of esmolol followed by an infusion of 0-100 mcg/kg/min. When comparing patients who received esmolol to those that did not, the following outcomes were found: higher rates of temporary return of spontaneous circulation (ROSC) (67% vs. 42%), sustained ROSC (67% vs. 32%), increased ICU admission rates (66% vs. 32%), and increased survival to hospital discharge (50% vs. 16%) with favorable neurologic outcomes (50% vs. 11%).[6]

A second retrospective study, designed as a pre-post trial, evaluated patients with out-of-hospital cardiac arrest from January 2012 to December 2015.[7] This study aimed to determine the effects of esmolol on sustained ROSC and survival with good neurological outcomes in patients with RVF.[7] Based on inclusion criteria, twenty-five patients were included in the pre-phase (received standard of care) and sixteen patients were included in the post-phase (received esmolol). The esmolol was given as a 500 mcg/kg loading dose followed by a continuous infusion of 0-100 mcg/kg/min. Sustained ROSC was higher in the esmolol group (56% vs. 16%; p=0.007). Survival with good neurological outcome was not found to be statistically significant despite two-fold better outcomes seen in the esmolol group when compared with standard of care (18.8% vs. 8%; p=0.36). These findings support the previous results that esmolol is associated with an increase in sustained ROSC; however, improved long-term outcomes were not found to be statistically significant.[7]

Although the evidence for the use of esmolol in RVF shows improved odds of sustained ROSC, the data examining long-term outcomes is controversial. The available literature is retrospective with small sample sizes. In order to strengthen the recommendation for use of esmolol in RVF, future prospective, randomized trials are needed to determine the overall benefit and outcomes.

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Stroke Mimics Continued from page 3

neurologic symptoms are present beyond loss of consciousness.[5] Sepsis can present as a stroke mimic by provoking recrudescence of prior neurologic deficits. Conversely, stroke can also provoke sepsis (*e.g.*, by increasing aspiration pneumonia risk), although this generally occurs over a longer time course.

Severe presentations of primary headache disorders can include focal neurologic deficits with up to 25% of migraine patients presenting with focal neurologic deficits.[6] The presence of a headache, however, can also be a symptom of stroke in up to 25% of cases. A thorough history, including family history for familial hemiplegic migraine, is essential to identify possible stroke mimics of primary headache disorders.

As in this case, spinal cord lesions can produce symptoms concerning for stroke. Subacute onset or history of diverse neurologic findings may suggest a demyelinating disorder, such as acute demyelination encephalomyelitis (ADEM) or multiple sclerosis (MS).[6]
Several studies have attempted to identify features that favor a stroke mimic as opposed to an acute ischemic stroke. A retrospective study of 960 prehospital patients found that patients with stroke mimics had lower systolic blood pressure measured by EMS providers (146.1 mmHg; 95% CI 142.5-148.6mmHg) compared to acute stroke patients (155.6mmHg; 95% CI 153.4-157.9mmHg).[7] While interesting, the utility of this finding for differentiating etiology in an individual acute patient is limited. A retrospective study of 10 years of data from the NIH Stroke Program of consults received from both ED patients and hospitalized inpatients (8187 patients; 30% with stroke mimic) found that lack of a history of hypertension, atrial fibrillation, or hyperlipidemia were factors associated with the greatest odds of a stroke mimic. Stroke mimics were more likely in younger patients, proportionally more common among women, and more common in patients arriving by private vehicle.[8]

While the breadth of the differential and complexity of individual patients can clearly create diagnostic uncertainty in the ED, data suggests that the majority of patients treated with thrombolysis have an acute ischemic stroke. Of patients treated with systemic thrombolytics for stroke-like symptoms, studies have reported 1.4% (95% CI 0.8% - 2.4%) as stroke mimics in a single Finnish center,[2] 1.8% (95% CI 1.5%-2.2%) in a multicenter European report, and 14.5% (95% CI 11.7% - 17.9%) in a single Tennessee center.[12]

A meta-analysis of nine studies reported 4.4% of patients treated with thrombolytics were diagnosed as stroke mimics, although there was significant risk of selection and reporting bias in these data since several studies were not prospectively collected.[12] Among the 392 patients ultimately diagnosed as stroke mimics who received thrombolytics, symptomatic intracerebral hemorrhage (sICH) occurred in 0.5% (95% CI 0%-2%) and angioedema occurred in 0.3% (95% CI 0%-2%).[12] A single article drawing on a 12-center European dataset contributed more than a quarter of the treated stroke mimics,[13] but there was no evidence of heterogeneity seen in the data.[12] While this data appears robust, there is likely some publication bias against disclosure of treatment with thrombolysis in stroke mimic. However, the overall sICH rate was similar to those reported in the cardiovascular literature for patients with myocardial infarction treated with thrombolytics and is likely an accurate reflection of the risk of sICH in patients with stroke mimics treated with tPA.[2]

Interestingly, the rate of sICH among stroke mimics treated with thrombolytics (<1%) is lower than among acute ischemic stroke (AIS) patients, which in the NINDS trial was approximately 6.4% in tPA treated patients compared to 0.6% of placebo-treated patients.[15] For physicians counseling patients where there is diagnostic uncertainty, the expectation that sICH is less likely if there is not a stroke is somewhat reassuring, although ideally thrombolysis would not proceed in patients with stroke mimics.

In summary, emergency physicians must maintain a broad differential for stroke-like symptoms. It is also paramount for physicians to be well versed in the risks of thrombolysis in order to have a meaningful and patient-centered discussion when treating patients who present to the ED with stroke-like symptoms.

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Continued on page 12

Stroke Mimics
Continued from page 11

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Neurosyphilis
Continued from page 5

Tabes Dorsalis

Tabes dorsalis is a slowly progressive degenerative disease of the posterior columns of the spinal cord. It typically develops 20 years after the initial infection. Symptoms include sensory ataxia and neuropathic pain. Pupillary irregularities like Argyll-Robertson pupil are also common in tabes dorsalis. The damage is often irreversible at this stage of infection.

of a strain of a *T. pallidum* with more affinity for the eye. Other theories include greater awareness of ocular complications or the overall rise in syphilis infections.[7] If ocular syphilis is recognized and treated early in the clinical course, vision is usually fully recovered. If left untreated, chronic progressive intraocular inflammation may result in glaucoma, chronic vitritis, retinal necrosis, and optic atrophy.[8]

Subtype	Timing After Initial Infection	Features
Asymptomatic	Anytime	Clinically silent; CSF (+) VDRL
Meningitis	0-2 years	Headache, meningismus, vomiting CSF: elevated protein, lymphocytosis
Meningovascular	7 years	Acute neurologic deficits
Tabes Dorsalis	20 years	Sensory ataxia, neuropathic pain Argyll-Robertson pupil
General Paresis	10-25 years	Dementia, psychosis, flaccid paralysis
Optic Atrophy	1-2 years	Posterior uveitis, conjunctivitis, scleritis

Table 2: Subtypes of neurosyphilis and highlights of clinical presentations

General Paresis

General paresis often develops 10 to 25 years after infection and is also known as general paralysis of the insane, dementia paralytic, or paretic neurosyphilis. It is a progressive dementia with psychotic features. If caught early, symptoms can be reversible with appropriate treatment.[6]

Optic Atrophy

When syphilis involves the eye, almost any ocular structure can be involved and results in ocular atrophy. Manifestations of ocular syphilis include conjunctivitis, episcleritis, scleritis, interstitial keratitis, iritis, uveitis, chorioretinitis, retinitis, and retinal vasculitis. Posterior uveitis and pan-uveitis are the most common findings. Ocular syphilis can occur at any time during the disease course and most commonly presents with syphilitic meningitis. A report from 2015 showed that the incidence of ocular syphilis is increasing and occurs in about 0.6% of all cases of syphilis.[7] The etiology of the increasing incidence of ocular syphilis remains unclear. Some experts postulate that it could be secondary to the outbreak

Diagnosis

The first step in the diagnosis of syphilis and neurosyphilis is to determine if the patient has ever been infected with *T. pallidium*. This is accomplished with serologic blood testing with both non-treponemal and treponemal tests. Non-treponemal testing detects antibodies against antigens released by the issue damaged by spirochetes (*e.g.*, cardiolipin). These are the Venereal Disease Research Laboratory (VDRL) and RPR tests. False-positive results can occur in other acute infections, chronic autoimmune disease, and intravenous drug use. These titers decrease after successful treatment and are used to monitor response to treatment as well as reinfection. Treponemal tests detect antibodies against spirochete antigens and are more specific than the non-treponemal tests. These include micro-hemagglutination T pallidum (MHA-TP), fluorescent treponemal antibody absorption test (FTA-ABS), and T. pallidum particle agglutination (TPA). Treponemal antibodies remain positive for life, do not correlate with disease activity, and are not useful in monitoring response to treatment. Generally, neither test should be used

in isolation when evaluating a patient with syphilis.[9]

Diagnosis of ocular syphilis is challenging as it can infect any structure of the eye. There is no pathognomonic presentation, and it mimics many other inflammatory conditions of the eye. If an inflammatory eye condition fails to improve with standard therapy, clinicians should consider syphilis in the differential. Emergency physicians need to have a high-index of suspicion and low threshold to obtain serologic testing as ocular syphilis cannot be ruled out by clinical presentation alone.

CSF analysis should be performed in all cases of ocular syphilis or in patients with syphilis and neurologic complaints. Lumbar puncture should be considered in any patient with HIV and syphilis at any stage, regardless of ocular or neurologic disease. Greater than five leukocytes, elevated protein, and presence of treponemal or non-treponemal antibodies in the CSF are diagnostic for neurosyphilis.[9] The preferred treatment of syphilis is parenterally administered penicillin G. The dose and duration of therapy vary by stage. Treatment for neurosyphilis is four million units of penicillin G intravenously every four hours for two weeks followed by 2.4 million units of benzathine penicillin intramuscularly weekly for three weeks. Patients with ocular syphilis should be treated similarly to patients with neurosyphilis even without CSF evidence
of neurosyphilis. Intravenous penicillin is required to achieve adequate intraocular levels for bactericidal effects. In penicillin allergic patients, penicillin desensitization should be performed as non-penicillin antibiotic regimens are less effective in the treatment of neurosyphilis.[10]

Successful treatment of neurosyphilis is confirmed by normalization of CSF studies and nonreactive VDRL serology. Neurologic examination and lumbar puncture should be performed every three to six months following treatment. Patients who do not have normalization of CSF studies by six months or decrease in VDRL titers by fourfold at one year should be retreated.[11]

Topical and systemic steroids can be used to treat ocular syphilis but only in the setting of appropriate antibiotic therapy. Topical steroids decrease inflammation from interstitial keratitis and anterior uveitis. Oral and intravenous steroids can be used to treat optic neuritis, scleritis, and posterior uveitis.[12] In cases of severe inflammation, topical therapies may need to be administered so frequently that these patients require admission to the intensive care unit.

Emergency physicians must be cognizant that rates of syphilis infections are on the rise and patients can have a wide spectrum of clinical presentations. It is important to ask historical questions to identify patients at risk including sexual behaviors, previous exposures to sexually transmitted infections, and HIV status. Have a low threshold to obtain serology and CSF studies. Ophthalmology should be consulted for further work up and management of inflammation in the eye concerning for ocular syphilis. Neurology should be consulted for patients with neurologic deficits in the setting of neurosyphilis. Patients with findings of ocular syphilis or neurosyphilis should be admitted to receive intravenous penicillin. They will also need to establish care with multiple specialists for long term management.

Syphilis is a sexually transmitted, chronic infection that can affect almost any organ system if untreated. Infection of the central nervous system can occur at almost any time during the infection and patients present with a wide range of symptoms. Ocular syphilis can also occur at any time and can infect any structure in the eye. Diagnosis is confirmed with treponemal and non-treponemal tests and analysis of the CSF. Providers should have a low threshold to perform a lumbar puncture in patients with syphilis and especially those with HIV. If caught early and treated adequately, the majority of neurosyphilis and ocular syphilis can be cured with minimal long term sequelae.

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Baclofen Pump Failure
Continued from page 7

clofen. The baclofen pump should also be interrogated to evaluate for pump malfunction and reservoir status as the patient may need a new pump or simply a baclofen refill. Baclofen pump refill varies based on institution, but can typically be completed by a PMR physician or the provider who manages the patient's baclofen pump. Baclofen pump refill should not be attempted in the ED prior to consultation with the physician who manages the patient's baclofen pump.

The treatment of baclofen withdrawal begins with early and aggressive resuscitative measures. Intravenous access should be obtained, the patient should be on cardiac monitoring, and airway, breathing, and circulation must be assessed and intervened on as necessary. The direct medical management of baclofen withdrawal centers on increasing GABA activity. Because these patients receive baclofen chronically, GABA receptors are down regulated resulting in hyperactive afferent nerve impulses. The goals of treatment in baclofen withdrawal are focused on prevention of central nervous system complications (seizures, altered mental status, and delirium), hyperthermia, extreme derangements in blood pressure, and spasticity. Oral baclofen alone is not adequate for baclofen withdrawal. The slow onset of oral baclofen (3-4 days) makes it ineffective in the acute care environment. However, these patients often have prolonged hospital stays and may benefit from oral baclofen later in their hospitalization, so its administration in the ED should be considered.[4]

Baclofen Withdrawal Differential Diagnosis	
Sepsis	Serotonin Syndrome
Meningitis/Encephalitis	Neuroleptic Malignant Syndrome
Alcohol Withdrawal	Benzodiazapine Withdrawal
Sympathomimetic Overdose	Malignant Hyperthermia

Table 1: Differential diagnosis in baclofen withdrawal.

ITB is ultimately what these patient's need, but is often not feasible in the ED. Previous case reports discuss ITB delivered via lumbar puncture for the management of patients experiencing baclofen withdrawal.[3,8,9] While there is minimal data, this is reportedly an effective treatment of ITB withdrawal. This procedure is technically challenging and can result in worsening complications if damage is caused to the catheter system. Therefore, only experienced physicians should deliver ITB via lumbar puncture, and this procedure should not be performed in the ED without neurosurgical consultation.

There are many alternative agents that can be utilized in the acute management of baclofen withdrawal. Benzodiazepines are the most common adjuvant therapy for baclofen withdrawal and should be administered based on withdrawal symptoms.[4] Lorazepam, diazepam, and midazolam can be used and should be titrated until vital signs are normal, spasms have decreased, and seizures have been controlled. Propofol is another medication that has been found to be useful in baclofen withdrawal. Propofol has presynaptic activity at GABA receptors, which will improve many of the symptoms of baclofen withdrawal caused by GABA down regulation. Similar to benzodiazepines, propofol should be titrated based on the patient's clinical symptoms. Propofol's short half-life is advantageous for titration, but has significant respiratory and hemodynamic side effects. If using propofol of ITB withdrawal, the patient will almost always require endotracheal intubation and admission to an intensive care unit.

When considering the plan for airway management in the ED, it is important to avoid succinylcholine given the potential for rhabdomyolysis and hyperkalemia from baclofen withdrawal. The neurocritical care unit is likely the most appropriate disposition for these patients given the extensive neurological monitoring required and the possiblity of neurosurgical intervention. Continuous EEG should be strongly considered, as the neurologic exam can be unreliable in the setting of deep sedation. This can be initiated in the ED, and if there is any evidence of seizure activity aggressive treatment should be initiated. Furthermore, vital signs and reassessments should take place frequently both in the ED and the intensive care unit until the patient's symptoms have resolved.

Additional novel treatments have been described for baclofen withdrawal that does not respond to high dose benzodiazepine therapy. There is significant symptom overlap between ITB withdrawal and serotonin syndrome and cyproheptadine can be an effective adjuvant treatment of baclofen withdrawal.[4] Baclofen withdrawal has spasticity reminiscent of neuroleptic malignant syndrome, and the use of dantrolene has additionally been reported by a few authors as further adjuvant treatment.[4] Although cyproheptadine and dantrolene should not be the initial treatment, they should be considered in baclfoen withdrawal refractory to benzodiazepines and propofol.

The patient described above was successfully managed with dexmedetomidine for severe baclofen withdrawal. While there is a wide consensus that dexmedetomidine is an effective treatment for alcohol withdrawal, there is only a single case report of successful management for baclofen withdraw-

al.[10] Dexmedetomidine is a highly selective alpha-2 adrenergic agonist with a superior safety profile in comparison to benzodiazepines or propofol. Dexmedetomidine was found to be non-inferior in terms of time spent at the target sedation range with shorter times to extubation and decreased respiratory depression.[11] This likely played a significant role in the avoidance of intubation in the patient described previously, making dexmedetomidine a promising option for the future treatment of ITB withdrawal.

While medical management of baclofen withdrawal is primarily focused on replacing intrasynaptic GABA, the hyperthermia and rhabdomyolysis often associated with ITB withdrawal must also be managed. Hyperthermia should be treated with aggressive cooling measures. The relief of spasticity and hyperthermia will stop ongoing muscle destruction and aid in the treatment of rhabdomyolysis. Intravenous fluids should be administered to maintain kidney perfusion and urine output to decrease kidney injury from rhabdomyolysis. These patients must additionally be monitored for electrolyte abnormalities associated with rhabdomyolysis, specifically hyperkalemia.

While ITB is central in the treatment of many spastic conditions, baclofen withdrawal is a dangerous complication of ITB cessation. While the differential must be broad, baclofen withdrawal must be considered in all patients with an ITB pump presenting with seizures, altered mental status, or abnormal vital signs. ITB is the ultimate treatment for baclofen withdrawal, however benzodiazepines or propofol are often effective temporizing measures. Although little evidence is currently present, dexmedetomidine was successfully used in the case presented and should be considered as a potential temporizing treatment in refractory ITB withdrawal. These medications should be aggressively titrated to effect based on improvement of the patient's symptoms. If necessary, the patient should undergo endotracheal intubation. Lastly, hyperthermia and rhabdomyolysis are often associated with ITB withdrawal and must be simultaneously managed. Recognition and initiation of the treatment of baclofen withdrawal in the ED is critical, and these patients should be admitted to the NSICU for further management.

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Pneumomediastinum
Continued from page 9

lead to cardiac tamponade or airway obstruction and requires emergent video-assisted thoracoscopic surgery or thoracotomy. Pneumopericardium can cause cardiac tamponade, which also requires emergent surgical evacuation by a cardiothoracic surgeon.

In general, spontaneous pneumomediastinum responds well to conservative therapy and symptoms usually start improving within one day.[16] Once discharged from the hospital, patients should have serial imaging until complete resolution. Follow-up with either a primary care provider or cardiothoracic surgeon is appropriate. Prophylactic antibiotics are not necessary in clear-cut cases of spontaneous pneumomediastinum but are most often considered in cases of an esophageal rupture.[15]

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17. Courtesy of Wikimedia Commons vis www.gruntdoc.con. https://upload.wikimedia.org/wikipedia/commons/f/f7/Subcutaneous_emphysema_chest_cropped.jpg.

EKG focus

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History of Present Illness

A 30 year old gentleman presents to the emergency department with severe chest pain that began while wrestling with his son. He describes the pain as crushing with radiation to his left arm. Patient denies a personal history of cardiac disease, though he notes his mother suffered her first myocardial infarction at the age of 40. On examination, the patient is in obvious distress and is diaphoretic. An electrocardiogram is performed and is notable for the presence of a second-degree artioventricular block, Mobitz type I (*i.e.*, Wenckebach; see Figure 1). The patient was admitted to the hospital under the cardiology service. He was taken to the catheterization suite, where percutaneous angiography revealed total occlusion of the right coronary artery. Patient underwent balloon angioplasty and drug eluting stent placement with TIMI grade 3 revascularization, after which he was discharged home on dual antiplatelet therapy.

Etiology of Wenckebach

The etiology of the faulty conduction varies between patients and may stem from underlying ischemic disease, myocardial scarring, or iatrogenesis (*i.e.*, use of beta- or calcium channel-blockers, cardiac glycosides, etc). While typically viewed as a benign conduction aberration, recent literature suggests that Wenckebach physiology is actually predictive of impending sinoatrial dysfunction and dysrhythmias, especially in elderly patients.[3] Furthermore, emergency providers tasked with the care of patients with presumed new or previously undiagnosed second-degree type I heart block must stabilize symptomatic or hemodynamically tenuous patients and rule out potentially reversible causes of AV nodal failure, including underlying coronary artery disease or adverse pharmacologic effects. In patients with persistently symptomatic Wenckebach physiology and no obvious reversible etiology, the American College of Cardiology recommends placement of a permanent pacemaker.

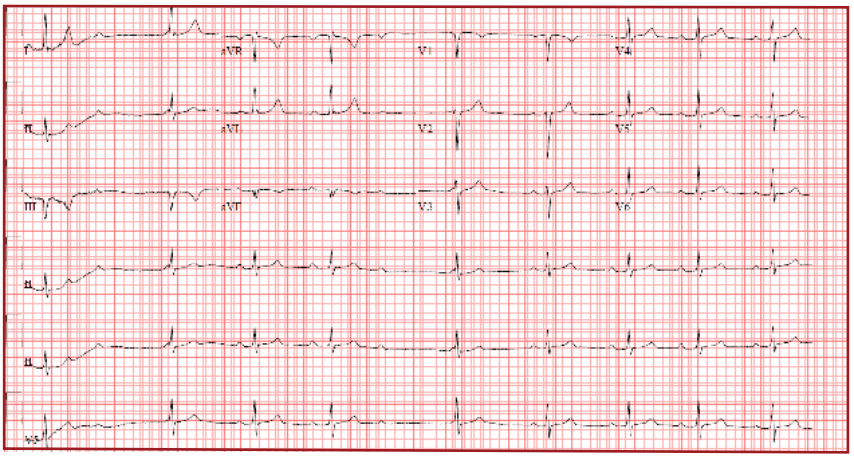
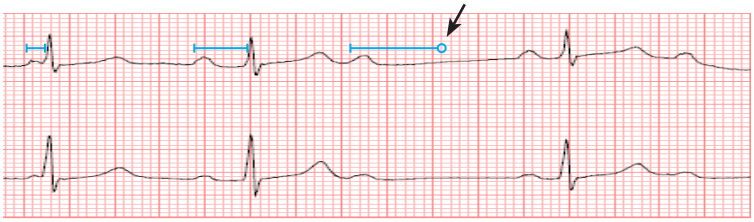


Figure 1. The patient's EKG demonstrating progressive P-R interval lengthening typical of second-degree atrioventricular block, Mobitz Type I.

EKG Features of Wenckebach



Progressive P-R interval prolongation (bars) precedes a dropped QRS complex (arrow), typical of Wenckebach physiology. Note also the subsequent P-R interval narrowing following the dropped beat as the node resets.

Second-Degree Atrioventricular Block, Mobitz Type I

Wenckebach physiology is one of two second degree electrophysiologic blocks characterized by progressive fatiguing of the atrioventricular node. This fatigue leads to progressive lengthening of the P-R interval preceding a nonconducted atrial impulse ("dropped beat"), after which the interval narrows once again. The ratio of conducted to non-conducted atrial impulses is often regular and reproducible, typically in a 2:1 or 3:2 fashion.

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EKG and Case referred by

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Annals of B Pod is always looking for interesting cases to publish!

Please submit cases via EPIC In Basket message to Dr. David Habib. Make sure to include the R1/R4 involved in the case.

Submitted B Pod Cases

Case	Providers
AC Separation	Banning
Baclofen Pump Failure	Gorder
WPW and 2nd Degree AV block	Burkhart
Stroke Mimic	Murphy
Intestinal Volvulus	Li
Pubic Symphysis Osteomyelitis	Skrobut
Nephrotic Syndrome	Benoit
Uveitis	Lagasse
Phenytoin Toxicity	Gottula