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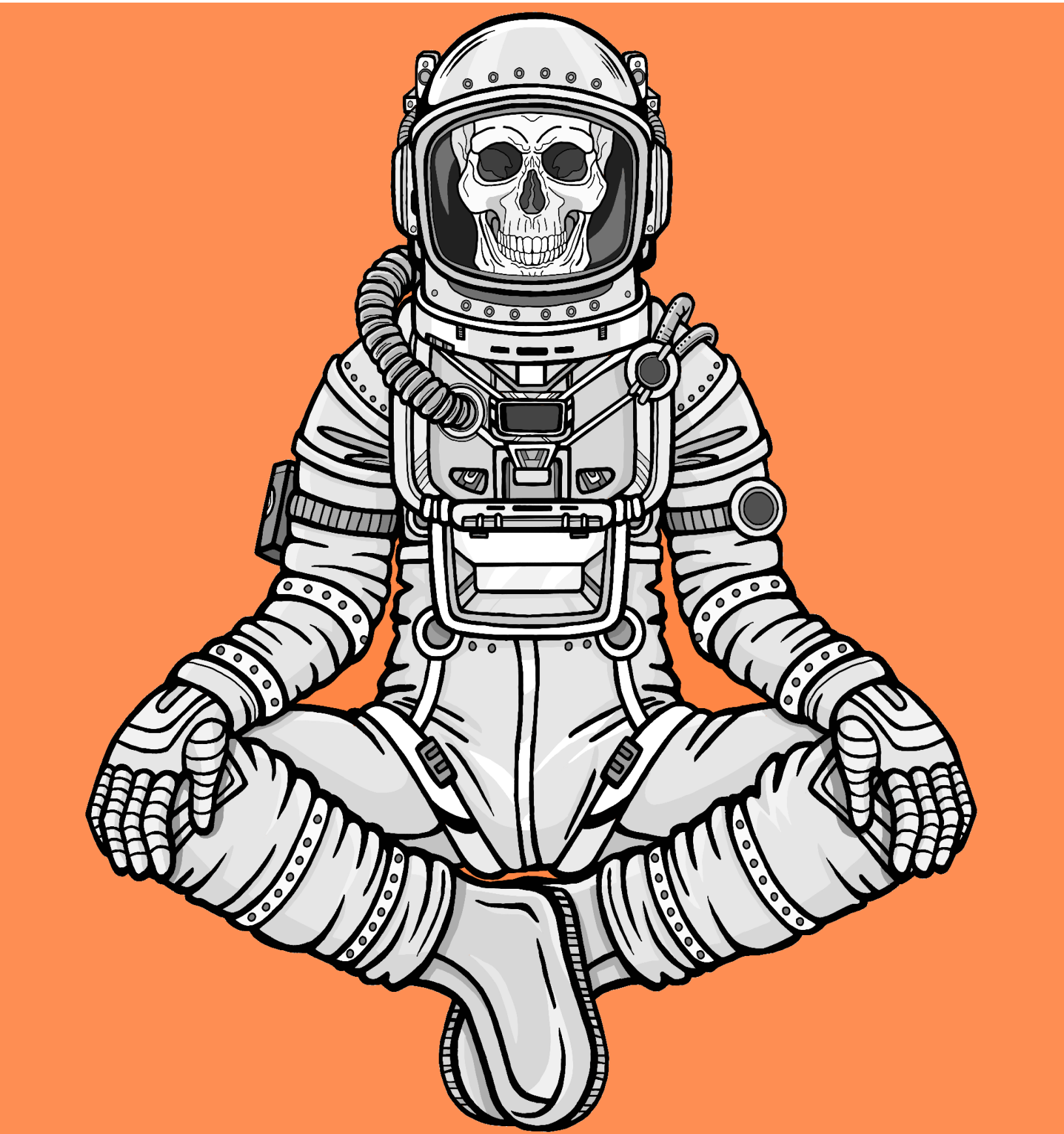

ANNALS OF B POD
WINTER 2020

NEUROCYSTICERCOSIS

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History of Present Illness

The patient is a Hispanic male in his late 20s who is brought in to the emergency department (ED) by family members with a chief complaint of headache, abnormal behavior, and confused speech. He reports gradual onset of a headache two days ago with pain located on the left side of his head and radiating to the front. He denies vision changes, neck pain, fevers, nausea, vomiting, or prior history of headaches. Later in the ED course, the patient provides a different story, complaining of generalized body aches for one hour and stating that he is seeking a primary care doctor for a checkup .

Collateral information obtained from family members reveals that the patient has been exhibiting strange behaviors at home. He has reportedly been more lethargic than usual, having memory issues as evidenced by mixing up past and present events, and talking "out of his head." They also report a history of fevers, night sweats, and chills for the past two days. Additionally, the patient provides incorrect information regarding how he got to the hospital and who brought him in, and when confronted about this misinformation, seems to be confabulating.

Past Medical History
None

Past Surgical History
None

Medications
None

Allergies
No known drug allergies

Social History
Originally from Mexico, has lived in the USA for 8-9 years. No international travel during this time-frame.
Works construction in Dayton, OH.
Smokes cigarettes, denies alcohol or drug use.

Physical Exam

T
36.3

HR
66

BP
138/73

RR
16

SPO2
97% on RA

The patient is non-toxic appearing and in no acute distress. He is drowsy but arouses to voice and is able to converse and follow commands. He is oriented to self and location, but not to date or time. Pupils are equal and reactive to light, extraocular movements are intact, and there is no evidence of nystagmus. His cranial nerves are intact. Strength, sensation and reflexes in bilateral upper and lower extremities are normal. He has no clonus, ataxia or tremors. No meningismus is noted. The remainder of his exam, including HEENT, cardiac, pulmonary, abdomen and skin, is unremarkable.

Diagnostic Tests

8.9 / 48.7	16.5 / 380	
3.7	142 / 27	106 / 0.91 / 16 / 100

LFTs: AST 17 / ALT 15
Urine: 40 ketones, trace protein
EtOH: <10
UDS: THC positive
Influenza: negative

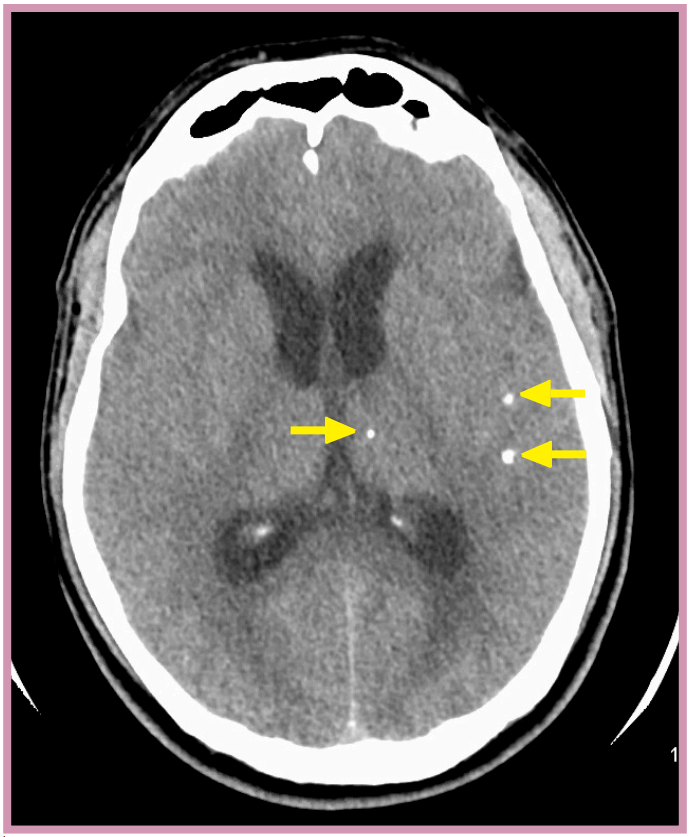


Image 1 : Representative image of CT head demonstrating multiple punctate foci of calcification (depicted by yellow arrows) with moderate ventriculomegaly. Findings concerning for neurocysticercosis with hydrocephalus.

Hospital Course

The patient had findings concerning for neurocysticercosis with hydrocephalus on non-contrast head CT. Neurology and neurosurgery were consulted, and an MRI brain with and without contrast was obtained. The MRI demonstrated a cystic mass in the third ventricle with an enhancing central nodule, consistent with intraventricular neurocysticercosis resulting in obstructive hydrocephalus. Lumbar puncture was deferred given the obstructive hydrocephalus, and the patient was admitted to the neurology service on a step-down status for further management. Ophthalmology performed a dilated eye exam that ruled out any signs of ocular involvement.

While inpatient, infectious disease was consulted. The patient was started on dexamethasone 0.1 mg/kg/day immediately, and began albendazole 15 mg/kg/day and praziquantal 50 mg/kg/day on hospital day two. Additional infectious work up, including HIV, strongyloides, and quantiferon TB tests were negative. Cysticercosis serum testing was positive. On hospital day three, neurosurgery performed endoscopic resection of the cyst in the third ventricle and placed an external ventricular drain. The patient tolerated the procedure well and his post-operative CT scan demonstrated immediate improvement in his hydrocephalus. The external ventricular drain was removed on hospital day four. The patient was to continue anti-helminths for a total of ten days and remain on steroids during this course. His neurologic exam improved throughout the hospital course, and he was discharged on hospital day seven. He followed up with neurosurgery at two and six weeks postoperatively and was noted to be recovering well with only intermittent headaches.

Discussion

Epidemiology and Pathophysiology

Cysticercosis is an infection caused by the larval stage of the tapeworm Taenia solium, commonly referred to as the "pork tapeworm." Among its infectious manifestations is neurocysticercosis, which is the most common parasitic infection of the central nervous system and one of the most common causes of epilepsy worldwide.[1] Neurocysticercosis is endemic to Latin America, Africa, South East Asia, India, China and Nepal.[1] It is not endemic to the United States, and cases are mainly due to immigration or travel from endemic countries rather than local transmission.

Humans become infected with neurocysticercosis after ingestion of T. solium eggs that have been shed in the stool of a human tapeworm carrier, typically via contaminated food or water. Following ingestion, embryos hatch in the small intestine, invade the bowel wall, enter the blood stream and then spread throughout the body, including the brain, muscle, and liver. The parasite then forms cysts in the tissue over a period of three to six weeks; those located in the brain and central nervous system cause neurocysticercosis. Of note, it is a common misconception that neurocysticercosis infection is caused by eating undercooked pork infected with T. solium. Individuals who ingest undercooked infected pork can acquire an intestinal tapeworm infection, but only individuals who ingest the eggs in the stool of human carriers develop neurocysticercosis.[2,3]

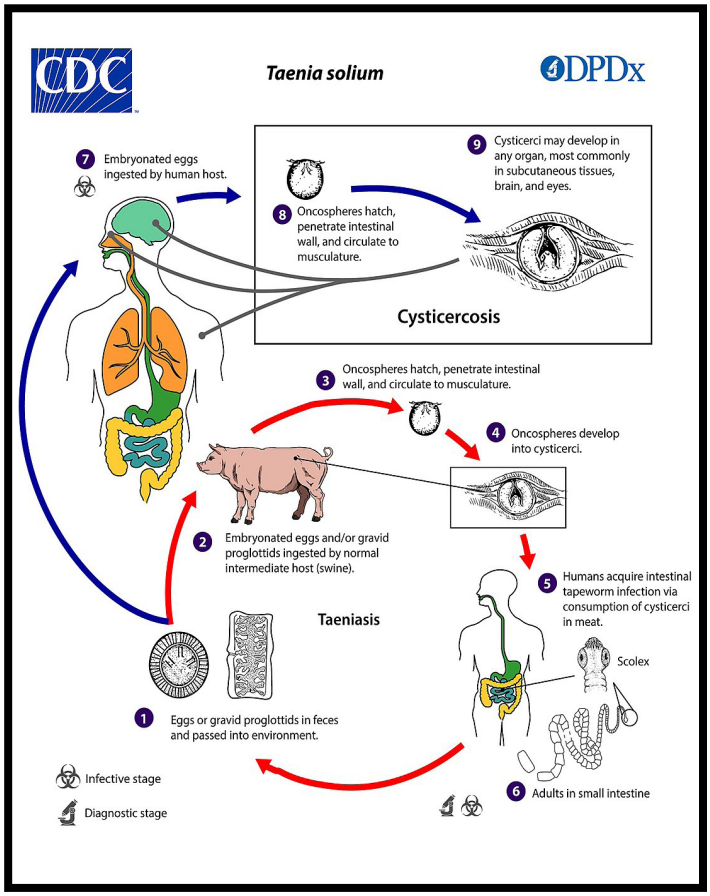
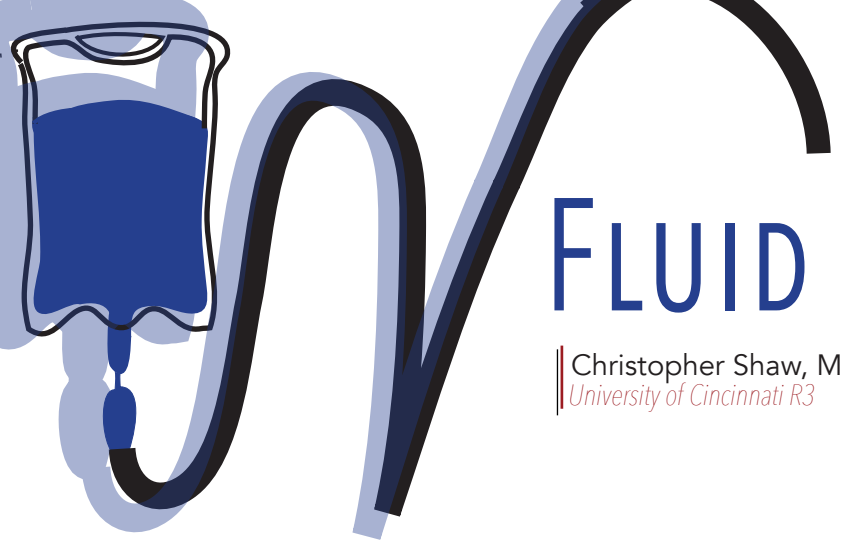


Figure 1: Life cycle of *Taenia Solium*, pork tapeworm. Courtesy of https://www.cdc.gov/dpdx/cysticercosis/index.html

Neurocysticercosis
continued on page 14



Fluid Resuscitation

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History of Present Illness

The patient is a male in his 70s who presents to the emergency department (ED) with dyspnea and hypoxemia. He has a past medical history of atrial fibrillation on rivaroxaban, diabetes mellitus type 2, and rheumatoid arthritis on prednisone.

Approximately 48 hours prior to this presentation, the patient was admitted to the hospital after being brought in by a concerned family member who found him disoriented at home with a diarrheal illness. On the patient's initial presentation to the ED, he was found to be febrile and in atrial fibrillation with rapid ventricular rate. While conversant, he was mildly confused with clear lungs and a soft, non-tender abdomen. The patient was resuscitated with intravenous fluids and empiric antibiotics, and was admitted to the medical floor where he remained on a continuous infusion of 0.9% normal saline (NS). He was briefly transferred to the intensive care unit (ICU) on hospital day 1 after becoming hypotensive, where the fluid rate was doubled and he received additional boluses of NS. Over the next 24 hours, his hemodynamics stabilized and his diarrhea resolved. An infectious work up revealed no bacterial source for his symptoms, antibiotics were discontinued, and he was discharged after a 36-hour hospital stay.

Less than 12 hours following discharge, the patient is brought back to the ED by emergency medical services who found him to be hypoxic to 81 % on room air. Upon arrival to the ED, the patient is unable to provide any history due to respiratory distress.

Past Medical History

Atrial fibrillation, chronic
Rheumatoid arthritis
Diabetes mellitus, type 2

Past Surgical History

Total knee arthroplasty

Medications

Rivaroxaban
Carvedilol
Metformin
Prednisone

Allergies

No known drug allergies

T
n/a

HR
170

BP
156/99

RR
34

SpO2
78% on
10L NC

Physical Exam

The patient is a diaphoretic, ill-appearing male who appears his stated age. His neck exam has jugular venous distension to the angle of the mandible with 2+ lower extremity edema in cool lower extremities with 1+ pulses bilaterally. The patient is in moderate respiratory distress, with suprasternal retractions and on auscultation there are diffuse inspiratory crackles and intermittent expiratory wheezes. The patient is tachycardic with an irregularly irregular rhythm without appreciable murmurs.

Diagnostic Tests

ALT 87 / AST 109 / T.bili <0.2 / Alk phos 67 / Albumin 4.0
Troponin <0.01 Pro-BNP 2,132
VBG: pH 7.13 / pCO2 29 / pO2 61 / HCO3 9 / base excess –19
Lactate – 4.8
Urinalysis – SG > 1.030, blood moderate, pH 6.0, protein 30, nitrite negative, leukocyte esterase negative
ECG – atrial fibrillation with rapid ventricular response, rate 151, left axis deviation, T wave inversions in 1 and aVL, minimal ST depression in V4-V6

Imaging:
Chest radiograph – diffuse bilateral air space opacities, more prominent in the right base. Consistent with pulmonary edema, although consolidation cannot be excluded in the right middle and lower lobe.

Hospital Course

The patient was immediately placed on non-invasive positive pressure ventilation with an FiO2 of 100%, given a bolus of furosemide, and placed on a nitroglycerin infusion at an escalating dose. Given the concern for possible sepsis, specifically legionella pneumonia with recent diarrheal illness, broad spectrum antibiotics were given. The patient's minute ventilation was calculated to be approximately 20 liters per minute. Upon admission to the ICU, his minute ventilation remained unchanged, although his FiO2 was weaned to 50%. Cardiology was consulted for acute decompensated heart failure, and the patient was started on infusions of both amiodarone and furosemide. A transthoracic echocardiogram showed a newly

reduced left ventricular ejection fraction (EF) of 15-20% with mild right ventricular dysfunction and no valvular pathology. Over the course of 24 hours, the patient was diuresed four liters and was weaned off positive pressure ventilation to nasal cannula. Intravenous amiodarone and furosemide were transitioned to an oral regimen. Urine legionella assay was negative. Blood cultures returned as no growth after 48 hours. He was discharged on hospital day five and referred for implantable cardioverter-defibrillator placement given his severely reduced EF.

Discussion

This elderly gentleman presented in respiratory distress with an anion gap metabolic acidosis, non-anion gap metabolic acidosis, and a respiratory acidosis. Although there are several pathophysiologic processes at play, this patient's clinical course may have been impacted by the fluid therapy he was given during his initial inpatient stay, during which he received approximately seven liters of NS over the first 24 hours of his hospitalization. Throughout his initial admission his bicarbonate decreased from 15 to 12, while his sodium and chloride went from 129 and 99 to 146 and 117, respectively. His large volume, chloride-liberal fluid administration may have played a role in the short span between admissions.

Intravenous fluid (IVF) is one of the most commonly provided treatments in the ED and inpatient settings. Nearly 30 million patients receive IVF annually in the United States alone.[1] It is so routine that physicians may forget that the potential benefits of fluid resuscitation can be outweighed based on fluid choice and the volume prescribed. The remainder of this discussion will focus on the options when choosing IVF, including the composition of each fluid and the available data regarding the use of each in particular patient populations.

Intravenous Fluid Options

Colloid v Crystalloid

At the most fundamental level, fluids can be divided into crystalloid and colloid agents. Crystalloids are an array of dissolved salts designed to mirror the osmotic composition of plasma. Colloids are comprised of larger molecules, such as albumin or synthetic carbohydrates, aiming to hold volume in the intravascular compartment through the exertion of oncotic force. Both options were designed to achieve the same goal: optimize intravascular volume and restore perfusion in patients suffering from shock. In practice, large scale trials have led many to avoid colloids as some studies have demonstrated evidence of harm with use of colloids, including increased need for renal replacement therapy and even mortality.[2,3] Albumin, while safe to use in most critically ill non-trauma patients, has not been found to improve mortality or patient-centered outcomes, such asventilator or ICU-free day.[4,5] In 2013, the Colloids vs. Crystalloids for the Resuscitation of Critically Ill (CRISTAL) trial, a large, multicenter, randomized investigation, found no difference in 28-day mortality between groups of hypovolemic shock patients who received crystalloid versus colloid resuscitation after being admitted to the ICU.[6] In the face of large trials showing no benefit to increased harm, and a hefty price tag compared to crystalloid options, colloids are rarely utilized as a primary mode of resuscitation.

Crystalloid v Crystalloid

The world of crystalloid is made up of NS and balanced solutions. Balanced solutions, which are designed to more closely reflect the ionic composition and pH of human plasma, are also referred to as chloride restrictive, or buffered, crystalloids. The most common varieties of balanced solutions are lactated Ringer's (LR), Plasma-Lyte A (PL), and Normosol-R. The composition of these fluids is strikingly different from NS (see Figure 1). Historically, due to a combination of availability, pricing, and precedent, NS has been the default resuscitative fluid in the majority of health care settings. However, observational data has linked NS to increased incidence of acute kidney injury,[7] renal replacement therapy, post-operative complications,[8] and even mortality[9,10] when compared to buffered solutions.

Mounting retrospective and observational data prompted large scale, randomized, prospective trials to evaluate the effects of fluid choice on outcomes. Most of these trials were focused in the realm of critical care and demonstrated no significant difference in incidence of AKI[11], or mortality[11-13]. Astudy published in 2018 by the SALT-ED investigators served as the first prospective randomized trial of fluid resuscitation in the ED.[14] Comprised of more

Intravenous Crystalloid Solutions

	Na+	K+	Ca2+	Mg2+	Cl-	Osmolarity
Human Plasma	**135-145**	**4.0-5.0**	**2.2-2.6**	**1.0-2.0**	**95-110**	**291**
0.9% Normal Saline	154	0	0	0	154	308
Lactated Ringer's	130	4.5	2.7	0	109	280
Plasma-lyte	140	5	0	1.5	98	294
Normosol-R	140	5	0	3	98	296

Table 1: Electrolyte composition and osmolarity of most commonly used intravenous crystalloid solutions. Table adapted from REBELEM

Intravenous Fluid
continued on page 13

SONO Series

Regional Wall Motion Abnormalities

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Background

Chest pain continues to be one of the most common chief concerns in patients presenting to emergency departments (ED) across the United States, accounting for more than 7 million ED visits annually.[1] Acute coronary syndrome, a critical and time-dependent spectrum of diagnoses, only contributes to an estimated 12-13% of these visits.[2] Of this total, approximately 70% will be secondary to non-ST-segment elevation acute coronary syndromes (NSTE-ACS).[3] Although the mismatch of oxygen supply to oxygen demand in NSTE-ACS can be due to a variety of etiologies, it is difficult to predict which of these patients have occult, but significant, coronary artery insults that can progress to myocardial infarction and necrosis if treatment is delayed. Thus, emergency physicians continue to face difficulties in determining which thresholds of conventional diagnostic modalities render a more morbid and mortal outcome, leading many of these patients to go underdiagnosed.

In order to understand the utility of echocardiography in the diagnosis of NSTE-ACS, we must first identify the limitations of current conventional diagnostic approach.

Electrocardiogram

While the electrocardiogram (ECG) is considered standard of care, it continues to be an imperfect diagnostic tool. Studies have demonstrated that only 40-65% of initial ECGs are indicative of ischemia in patients later diagnosed with acute myocardial infarction, with upwards of 20% of initial ECGs found to be completely normal.[3-7] ECGs are even less sensitive and specific in diagnosing NSTE-ACS, where findings can be subtle and difficult to interpret, especially in the setting of previous infarctions and conduction abnormalities. Several studies have demonstrated serial 12-lead ECG monitoring to have increased diagnostic utility, and national recommendations encourage providers to order serial ECGs every 15-30 minutes if clinical suspicion for ACS remains high, however sensitivity still falls shy of 70%.[6,7] Therefore, ECGs continue to be a suboptimal test in diagnosing this highly morbid disease spectrum.

Troponin

Troponin is a myocardium-specific serum marker that rises a few hours after myocardial injury, ischemia or infarct.[3] Sensitivity and specificity are high, however trends in troponin are generally valued over an absolute value in NSTE-ACS. Studies have demonstrated that upwards of 80% of patients who present with myocardial injury in the form of STEMI or NSTE-ACS will have troponin elevations within 2-3 hours.[3,8-10] However, this can be difficult to interpret if time of symptom onset is unclear. Therefore, the American Heart Association (AHA) currently recommends trending troponins out to at least six hours from initial symptom onset, and if clinical suspicion remains high and the patient is at intermediate- to high-risk for a coronary event, may require trending out to 24 hours in order to optimize ability to detect true ACS pathology.[3] A significant amount of time can elapse waiting for resulting of this test which may compromise outcome if intervention for true cardiac ischemia is being delayed. Furthermore, troponin is often elevated in clinical situations other than primary coronary pathology; any stress on the myocardium can lead to myocardial leak of troponin as seen in hypotension, sepsis, tachydysrhythmias, heart failure, myocarditis, renal failure, to name just a few. Thus, there may be diagnostic uncertainty, especially in the setting of co-morbid illnesses. The implementation of high-sensitivity troponins may alleviate some of this delay and diagnostic uncertainty, however further evaluation will be required after widespread use of these newer assays.

2-Dimensional Transthoracic Echocardiography

Two to three percent of highly morbid and mortal ACS pathologies continue to be discharged from the ED.[11] Echocardiography can be used to increase sensitivity and specificity for significant acute cardiac disease, primarily NSTE-ACS, in the setting of non-diagnostic ECGs and lab work. In fact, the AHA and several other national agencies have recommended the use of two-dimensional transthoracic echocardiography (2DTTE) as a class I recommendation when encountered with possible ACS.[12] Regional wall motion abnormalities (RWMA) diagnosed with 2DTTE have long been demonstrated to provide value in detecting myocardial ischemia

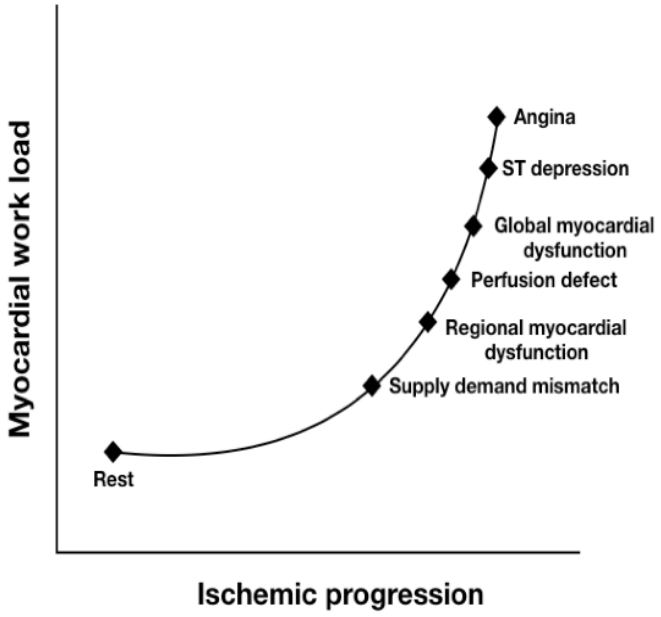
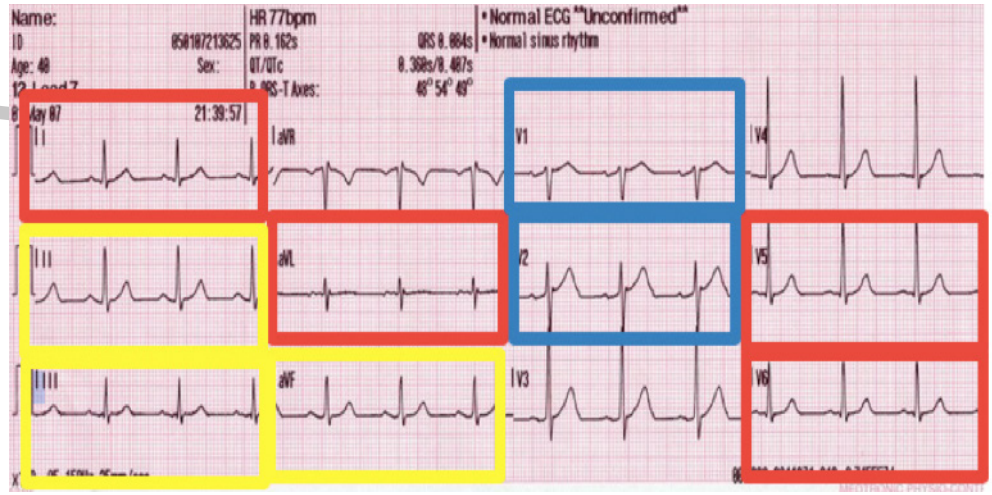


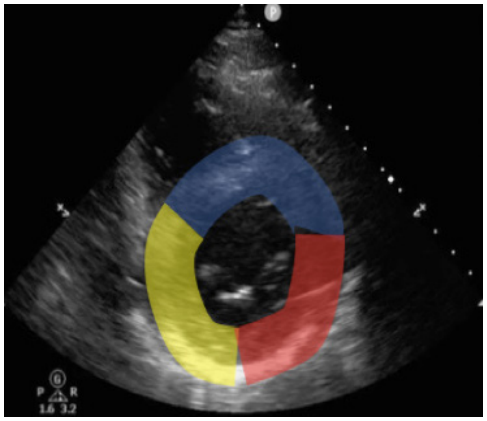
Figure 1: Pathophysiologic and clinical progression of coronary ischemia. Vascular dysfunction precedes regional wall motion abnormalities, which can be apparent prior to ECG changes. Figure adapted from Beller, 1988.

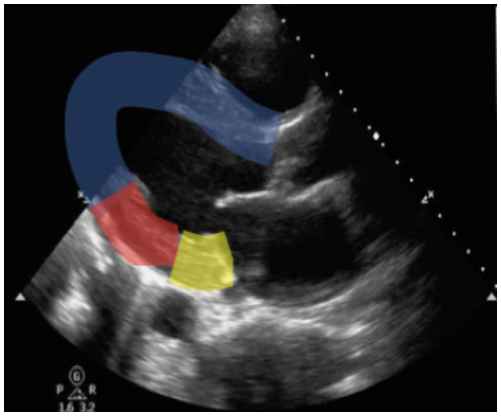
that will benefit from invasive intervention or inpatient monitoring. Robust literature published in the 1980s-1990s demonstrated sensitivities ranging 83-91% and specificities ranging 71-100% that outperform ECG, and rival myocardial perfusion imaging.[13-19]

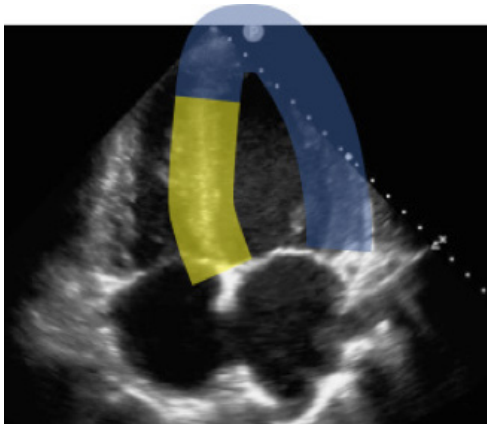
Discussion

Experimental and clinical studies have demonstrated that the earliest clinical manifestation of myocardial ischemia is RWMA, followed by ECG changes and onset of anginal symptoms (Figure 1).[20-22] RWMA develop within seconds of coronary artery occlusion during animal and human coronary angioplasties,[21,22] therefore proving that 2DTTE can be utilized immediately upon patient presentation if NSTE-ACS is suspected. Even if the patient's symptoms resolve spontaneously in the ED or shortly after administration of analgesics, RWMA have been shown to last anywhere from one to 24 hours after resolution of anginal symptoms.[22-27] Moreover, as little as five minutes of coronary disruption can lead to myocardial dysfunction for up to six hours after reperfusion, indicating some degree of myocardial stunning that occurs with even transient ischemic injury.[22-24] The delay in return of myocardial function may correlate with the length of anginal symptoms, degree of coronary occlusion, area of affected myocardium and presence of collateral blood flow. Thus, the highest yield of using 2DTTE to detect RWMA will be during acute symptoms, and while a negative study during active symptoms likely indicates another disease process, a negative study obtained after resolution of symptoms cannot definitively rule out NSTE-ACS.[28]









BLUE: Anterior = left anterior descending artery

YELLOW: Inferior = right coronary artery

RED: Lateral = left circumflex artery

Figure 3: Vascular territories on electrocardiogram matched to the corresponding myocardial regions as seen on transthoracic echocardiogram. Views include (from left to right) parasternal short, parasternal long, and apical four chamber.

Echocardiographically, the myocardium thickens during systole, followed by symmetric excursion. During active ischemia, there will be abnormal thinning of the myocardium during systole and asymmetry in myocardial excursion. RWMA can be identified as akinesia, hypokinesia or dyskinesia of the affected myocardium. The evaluation of wall thickening is preferred to wall motion abnormality in assessing for acute myocardial dysfunction, however, these changes can be more subtle in the setting of previously damaged myocardium, as scarred tissue will also appear thin during contraction.[29] Thus, findings on 2DTTE are more helpful when applied in the appropriate clinical setting - if ECG abnormalities are present, whether acute with ST segment and T wave changes, or chronic with Q waves, correlating RWMA with the expected lesion on ECG is helpful to discern acute from chronic pathology.

The American Heart Association and several other national committees sought to standardize a method to identify location and degree of wall motion abnormalities found on 2DTTE, and developed a 17-segment, quantitative evaluation schema.[30] While studies completed in the 1980s-1990s were primarily performed by radiologists or cardiologists, this is a cumbersome and impractical method to employ in the emergency department. Thus, in the setting of emergency physician performed echo, evaluation is condensed to a 3-segment assessment, with each analyzed segment anatomically correlating with a coronary artery perfusion territory: anterior left ventricular (LV) wall correlates with the left anterior descending artery; inferior LV with the right coronary artery; lateral with the left circumflex artery (See Figure 2).[27,31-34] Each wall is assessed in two orthogonal views at minimum, collected via a parasternal long, parasternal short and apical four

Regional Wall

continued on page 12

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CONNECT THE DOT

Case 1

History of Present Illness

The patient is a male in his early thirties with a past medical history of celiac disease and herpes simplex virus induced-erythema multiforme who presents to the emergency department (ED) with a chief complaint of a progressive, painful rash that has been worsening over the past three days. The patient's symptoms began with a cold sore on his lip, followed by lesions on his bilateral upper extremities. The lesions then spread to his palms, soles, trunk, back, neck, periorbital and perioral regions. He reports subjective fevers, odynophagia, and a tingling sensation of the left eye. He denies visual changes, eye pain, shortness of breath, abdominal pain, vomiting, urinary or bowel symptoms, or genital lesions. He denies recent tick exposure.

The patient was seen an outside ED on day two of symptoms, where he was treated with methylprednisolone, cetirizine and encouraged to follow up as an outpatient. His symptoms continued to worsen, so he presented to the same ED later that evening, where he was treated with dexamethasone and hydrocortisone cream and discharged. Despite these therapies, he has had no relief and now complains of severe pain and worsening lesions, particularly in his mouth and on his hands.

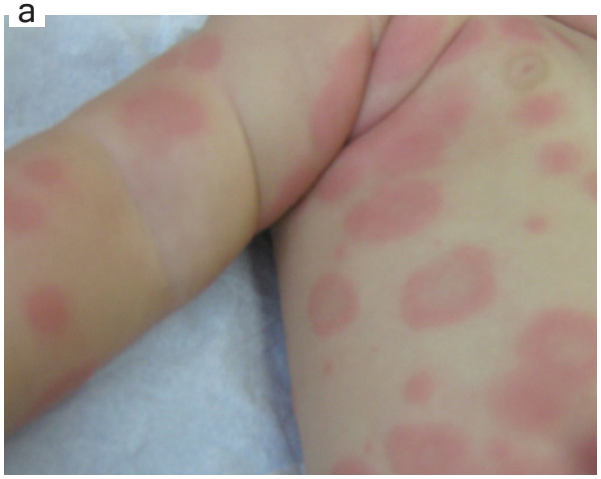
Physical Exam

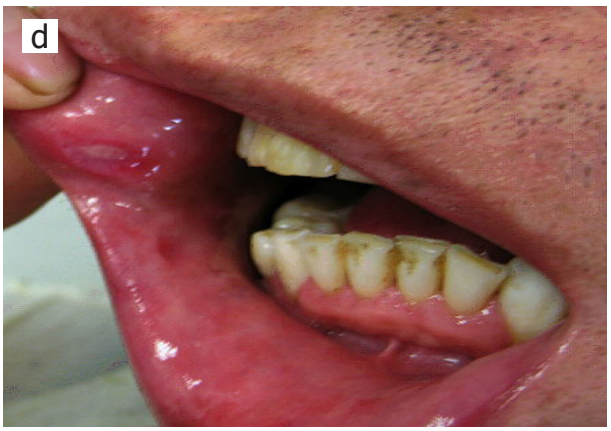
Vitals T36.3 / HR 54 / BP 129/75 / RR 12 / SpO2 94% on room air. The patient is a well-appearing male who appears uncomfortable but is in no distress. There are diffuse 0.5-3 cm variable eruptions, including tender papules and vesicles, over the trunk and extremities. The rash involves the palms and soles, and targetoid papules are noted on bilateral hands. There are also many scattered pigmented macules from older lesions. On the posterior scalp, there is a large 8x8 cm irregular pink plaque. No perianal lesion is noted, but there is an eruption on the right lateral penis with white scale. There is mucosal involvement of the anterior gingiva and palate. Ophthalmologic, cardiovascular, pulmonary, abdominal, and neurologic exams are within normal limits.

Hospital Course

The patient's initial presentation was consistent with erythema multiforme major as a leading diagnosis. Basic laboratory studies were unremarkable, and additional labs were sent to assess for drug rash with eosinophilia and systemic symptoms (DRESS) syndrome, human immunodeficiency virus (HIV), syphilis, and glucose-6-phosphate dehydrogenase (G6PD) deficiency, which were negative. The patient was treated with intravenous (IV) fluids and pain medication in the ED, and dermatology was consulted for recommendations on management. Based on his prior medical history of erythema multiforme secondary to herpes simplex virus, he was clinically diagnosed with erythema multiforme and biopsy was deferred. He was prescribed topical steroids and valacyclovir in the inpatient setting, and dressings were used to cover his lesions. He remained stable and was discharged home in stable condition with valacyclovir, topical clobetazole, ibuprofen, acetaminophen, and tramadol with follow up in dermatology clinic.

The patient was seen in dermatology clinic three weeks later,and his active cutaneous lesions had been replaced by scattered dyspigmented patches over this palms, soles, extremities, and trunk. Clobetasol ointment was discontinued given that he no longer had active lesions, and he was started on a suppressive dose of valacyclovir.





Images 3-8: Erythema multiforme is depicted by cutaneous findings , classically described as targetoid lesions on the trunk (a) and extremities (b). Mycoplasma pneumoiae ass

TS: A TALE OF TWO RASHES

Case 2

History of Present Illness

The patient is a previously healthy African female in her late twenties who presents with a chief complaint of productive cough and a rash. She was seen in the ED three days ago for her cough and was diagnosed with community acquired pneumonia, for which she was prescribed doxycycline and azithromycin. She has been compliant with her antibiotic regimen, however notes she has developed a desquamating rash, purulent drainage from her lips and mouth, and erythema and purulent drainage from bilateral eyes. The rash has progressed to form papules over bilateral upper and lower extremities. She denies fever, chest pain, shortness of breath, nausea, vomiting, and abdominal pain. The patient has recently moved to the United States from Western Africa three months prior to her presentation.

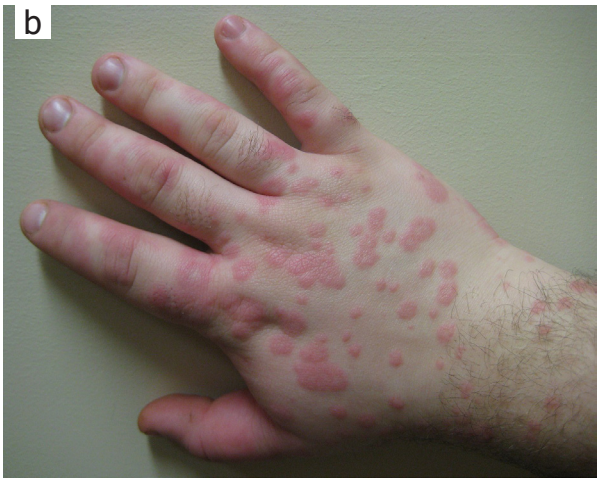
Physical Exam

Vitals T 39.1 / HR 118 / BP 106/60 / RR 27 / SpO2 96% on room air. The patient is an awake, alert, ill-appearing female. She exhibits desquamation of the upper and lower lips with purulent drainage from her oral mucosa, as well as bilateral conjunctival injection with purulent drainage. An erythematous, maculopapular rash is present on the upper and lower extremities. Many lesions have necrotic centers, and some lesions appear to be pustular. One erythematous lesion is present at the left lower quadrant of the abdomen, and scattered lesions are present on the patient's back. Genitalia exhibits erythema and purulent drainage, but no pustules or necrolysis. Lung sounds are diminished throughout, with wheezes auscultated at the right middle lobe. Cardiovascular, abdominal, and neurologic exams are within normal limits.

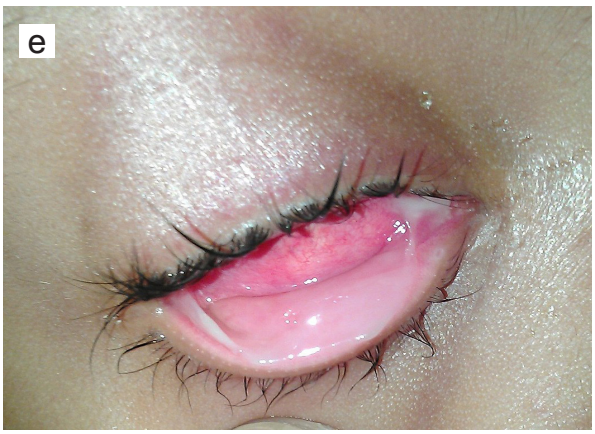
Hospital Course

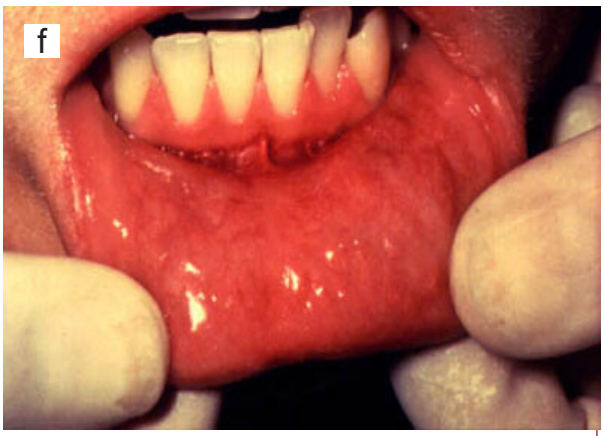
Basic laboratory studies were unremarkable. HIV screening was negative. Sputum culture and blood cultures showed no growth. Urine legionella antigen and mycoplasma pneumonia DNA studies were negative. Chest x-ray was within normal limits.

The patient's initial presentation was concerning for stevens johnson syndrome versus an infectious etiology of her rash, including mycoplasma pneumonia associated mucositis given her recent history of pneumonia. Dermatology, burn surgery and ophthalmology were consulted during the patient's hospital course to assist with management.



Upon admission, the patient was treated with IV fluids, levofloxacin, and standard wound care. A respiratory viral panel and quantiferon gold testing were negative. Mycoplasma IgM resulted positive, supporting a diagnosis of mycoplasma pneumonia-associated mucositis. Ophthalmology evaluated the patient on hospital day three and noted pseudomembranes and new staining of the bulbar conjunctiva in bilateral eyes. Ophthalmic moxifloxacin was started and the patient was taken to the operating room for bilateral amniotic membrane graft placements and symblepharon ring placement. Following the procedure, she was started on daily prednisone 60 mg. She required multiple days to heal from an ophthalmologic standpoint, however was ultimately discharged on hospital day 15 with a prednisone taper, and bacitracin and cliloxan ointment to bilateral eyes. At discharge, her rash was noted to have eroded and crusted over, and she was discharged with topical application of mild barrier cream for moisturization.





The patient followed up in ophthalmology clinic two days after discharge and was noted to have almost complete resolution of bilateral conjunctival staining. She also reported significant improvement in her skin lesions.

ociated mucositis can involve the conjunctiva (c,e), oral mucosa (d,f). Images courtesy of common.wikimedia.org.

Discussion

Erythema multiforme (EM - Case 1), mycoplasma pneumonia associated mucositis (MPAM - Case 2), and Stevens-Johnson Syndrome (SJS) present with similar clinical features and are often considered to be varying points along the same disease spectrum. Mycoplasma pneumonia is known to be a cause of all three disease processes, but some studies argue that there are subtle, yet important, differences in pathogenesis, presentation, and disease course between the three disease processes.[1]

Epidemiology and Clinical Presentation

Erythema Multiforme (EM)

Erythema multiforme is an immune-mediated condition characterized by cutaneous targetoid lesions, primarily on the face and extremities.[2] The disease occurs mostly in adolescents and young adults, with a male to female ratio of 3:2.[2] The etiology of EM is almost always infectious, with the herpes simplex virus and Mycoplasma pneumoniae being two major causes. Less commonly, EM is caused by other infectious pathogens or drugs, such as adenovirus, cytomegalovirus, Epstein-Barr virus, varicella zoster virus, viral hepatitis, non-steroidal anti-inflammatory drugs (NSAIDs), sulfonamides, and other antibiotics.[2]

The classic EM rash presents with asymptomatic targetoid lesions with or without mucosal involvement. There are two classifications of EM: EM major, which develops in approximately twenty percent of EM cases and signifies mucosal involvement, and EM minor, which involves only cutaneous symptoms.[3] Although the majority of patients will have asymptomatic cutaneous lesions, some patients, such as the one described above, do report pain or pruritus associated with dermatologic findings. The disease is usually self-limited and resolves within a few weeks, and very rarely does it recur or persist.[2] In severe cases, complications can occur, such as cutaneous scarring or ocular scarring with subsequent visual impairment.

Mycoplasma Pneumonia-Associated Mucositis (MPAM)

Mycoplasma pneumonia associated mucositis is an immune-mediated condition that is different from erythema multiforme in that cutaneous involvement is often sparse or nonexistent.[4] It is most common in children and adolescents,[5] with 66% of documented cases occurring in males.[6] It occurs in less than 10% of patients who acquire a Mycoplasma pneumoniae infection.[7]

MPAM primarily presents with involvement of oral and/or ocular mucosa. When cutaneous lesions are present, they are commonly maculopapular or vesicular and present on the trunk and extremities.[8] Prodromal symptoms, including respiratory symptoms, are almost always present,[1] and while upper respiratory tract disease is more common, pneumonia may occur as well.[9] Ocular complications, such as those present in the patient described here, are some of the most common sequelae of mycoplasma mucositis and can cause scarring and visual impairment. More rarely, central nervous system complications may ensue. These are relatively more common in children and include aseptic meningitis, meningoencephalitis, seizures, peripheral neuropathy, transverse myelitis, cognitive abnormalities, and cerebellar ataxia.[10,11] MPAM may also be associated with esophagitis and erosive bronchitis.[2] The disease course is most often mild but depends on severity of lesions and degree of ocular involvement.[1]

Mycoplasma pneumoniae is a documented cause of mucositis regardless of preceding antibiotic administration, but the large number of cases in which patients were receiving antibiotics at the presentation of mucocutaneous disease increases the suspicion that antibiotics "intensify the dermatosensitive potential of Mycoplasma pneumoniae,"[8] such as in the patient case presented above.

Diagnosis

EM and MPAM are primarily clinical diagnoses based on patient history and visual assessment of lesions. The differential diagnosis of these presentations will therefore include other skin conditions, including SJS, toxic epidermal necrolysis (TEN), urticaria, erythema nodosum, bullous pemphigoid, or dermatitis herpetiformis.[12] As discussed previously, EM will typically present with an acral distribution of papular targetoid cutaneous lesions, while MPAM will typically present with mucosal lesions with possible cutaneous involvement. Although atypical lesions may also occur, the typical target EM lesion is characterized by a "dusky central disk" with a more peripheral "infiltrated pale ring" and surrounding "erythematous halo."[2] Atypical EM lesions may include two rings only, or may be vesicles overlying a darker center and surrounded by erythema. However, EM lesions will always be papules, not macules.[2] Knowledge of HSV infection should increase suspicion for EM.

In cases of diagnostic uncertainty, biopsy of skin lesions, direct immunofluorescence, herpes serology, and serum antibodies may be used.[2] Cold agglutination studies may also assist with a diagnosis of MPAM. While these studies may not be necessary in a patient who will be discharged from the emergency department, they can assist admitting teams and consulting services in gaining a clearer picture of the patient's disease process.

Treatment

Management of patients with EM and MPAM will depend on the specific signs and symptoms exhibited by each patient, but generally entails supportive care. Both diseases are generally self-limited and do not have a specific cure.

Erythema Multiforme (EM)

While some physicians advocate for the use of systemic corticosteroids for severe cases of EM, there is insufficient evidence proving efficacy, and there is a significant risk of adverse side effects.[2] Three case reports did show significant improvement in duration of fever and vesicular eruptions with use of IV methylprednisolone, and another two cases demonstrated that IV methylprednisolone stopped disease progression and prevented recurrences. Other larger studies, however, linked corticosteroid treatment to longer hospital stays and increased complications.[13] Since EM can be managed with supportive care only, the possible benefits of steroid use have not been demonstrated to sufficiently outweigh the risks.

Since EM is an immune-mediated process, another possible treatment option for severe cases is intravenous immunoglobulin (IVIg). While evidence regarding its use in EM is limited, one case study demonstrated 80% improvement without significant adverse effects in a patient with persistent, severe EM that had been unresponsive to multiple rounds of steroids, acyclovir, and azathio-

prine.[14] Furthermore, IVIg has been shown to be effective in treating SJS and TEN,[15] therefore providers may consider empiric IVIg therapy if there is any diagnostic uncertainty

Although anti-HSV drugs have no effect on an active episode of EM, they can prevent recurrences in HSV-associated EM.[2] A double-blind, placeb-controlled studye demonstrated that a six-month course of acyclovir 400 mg twice daily achieved successful suppression of recurrent EM.[16] Valacyclovir and famciclovir have greater oral bioavailability than acyclovir and can be tried as alternatives to the above regimen. The goal of using antiviral agents is to achieve a recurrence-free period of several months at which point the dosage can be reduced and, hopefully, eventually discontinued.[17] When anti-HSV drugs have not worked to prevent recurrences, azathioprine and thalidomide have been found to be helpful.[2]

Mycoplasma Pneumonia-Associated Mucositis (MPAM)

For MPAM in particular, there are no evidence-based guidelines regarding management.[1] As such, current guidelines recommend discontinuation of the culprit drug, if that is the etiology of disease onset, and supportive management. . This includes standard wound care such as topical corticosteroids for cutaneous lesions, and oral antihistamines, topical corticosteroid gel, or mouthwash containing lidocainefor painful oral lesions. Ophthalmology consultation should be obtained for any ocular involvement in order to prevent long term sequelae.

Benefit of antibiotic use in mucocutaneous presentations may be limited, however use can often prevent neurologic and pulmonary complications later in the disease course, thus is commonly prescribed. Studies of steroid use in MPAM show similar conclusions as those studying steroids for EM: fever and hospital course are decreased, but complications may be increased. Similarly, there is limited evidence that IVIg is beneficial, but may be more helpful in cases with severe mucositis, such as cases so severe as to cause respiratory distress.[18]

Most patients with the above pathologies can be safely discharged. However, if symptoms are severe, or if there is any concern for SJS/TEN, a burns specialist or dermatologist should be consulted.[19] In severe cases, patients may have electrolyte abnormalities and should be corrected as needed. These patients will require hospital admission.

Summary

EM and MPAM are two clinically diagnosed conditions that are frequently thought to be along the same spectrum of dermatologic pathologies. However, there are some key differences that aid in diagnosis: EM is primarily a dermatologic condition, while MPAM is predominantly a mucositis. EM may be infectious or drug-induced, while MPAM is always associated with Mycoplamsa pneumoniae, and therefore respiratory symptoms are essentially universal. Both conditions focus on supportive care and discontinuing culprit drug use, if indicated, for management. They may also require specialty care involvement, such as ophthalmology, dermatology, pulmonology, or intensivists, depending on extent of systemic dissemination of the disease process. In severe cases, corticosteroids and IVIg may be beneficial, however more definitive research needs to be conducted to support routine use in these conditions.

1. Caravan, T. N., et al. Mycoplasma pneumoniae - induced rash and mucositis as a syndrome distinct from Stevens-Johnson syndrome and erythema multiforme: A systematic review. Journal of the American Academy of Dermatology. Volume 72, Issue 2, Pages 239-245. February 2015.
2. Roujeau JC. Erythema multiforme. In: Fitzpatrick's Dermatology in General Medicine, Wolff K, Goldsmith LA, Katz SI, et al (Eds), McGraw-Hill Companies, Inc, 2012. Chapter 39.
3. Huff, J. C., Weston, W. L., Tonnesen M.G. Erythema multiforme: a critical review of characteristics, diagnostic criteria, and causes. Journal of American Academy of Dermatology. 1983. 8:763.
4. Canavan TN, Mathes EF, Frieden I, Shinkai K. Mycoplasma pneumoniae-induced rash and mucositis as a syndrome distinct from Stevens-Johnson syndrome and erythema multiforme: a systematic review. J Am Acad Dermatol 2015; 72:239.
5. Meyer Sauteur PM, Goetschel P, Lautenschlager S. Mycoplasma pneumoniae and mucositis--part of the Stevens-Johnson syndrome spectrum. J Dtsch Dermatol Ges 2012; 10:740.
6. Bukhari, E., et al. Mycoplasma pneumoniae-associated mucositis syndrome: A rare and clinically challenging disease in a Saudi child. Journal of Taibah University Medical Sciences. August 2017. Volume 12, Issue 4, p. 356-359.
7. Figueroa, F. MPAM-Mycoplasma pneumonia associated mucositis: A diagnostic challenge. Cardiovascular Pharmacology Open Access. Volume 7. 2018.
8. Cherry JD. Anemia and mucocutaneous lesions due to Mycoplasma pneumoniae infections. Clin Infect Dis 1993; 17 Suppl 1:S47.
9. Mansel, JK, et al. Mycoplasma pneumoniae pneumonia. Chest. March 1989. 95(3):639-46.
10. Candler, PM, Dale, RC. Three cases of central nervous system complications associated with Mycoplasma pneumoniae. Pediatric Neurology. August 2004. 31(2): 133-8.
11. Smith, R., Eviatar, L. Neurologic manifestations of Mycoplasma pneumoniae infections: diverse spectrum of disease. A report of six cases and review of the literature. Clinical Pediatrics (Phila). April 2000. 39(4): 195-201.
12. Erythema Multiforme. NORD (National Organization for Rare Disorders). https://rarediseases.org/rare-diseases/erythema-multiforme/. Accessed January 25, 2020.
13. Yeung, A.K., Goldman, R.D. Use of steroids for erythema multiforme in children. Canadian Family Physician. November 2005. 51(11): 1481–1483.
14. Momin, SB, et al. Review of Intravenous Immunoglobulin in the Treatment of Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis. Journal of Clinical and Aesthetic Dermatology. February 2009. 2(2): 51–58.
15. Singh, MN, et al. Severe erythema multiforme responding to high dose intravenous immunoglobulin therapy. Journal of the American Academy of Dermatology. March 2004. Volume 50, Issue 3, Supplement, Page P95
16. Tatnall, FM, et al. A double-blind, placebo controlled trial of continuous acyclovir therapy in recurrent erythema multiforme. British Journal of Dermatology. February 1995. 132(2):267-70.
17. Lamoreux, MR, et al. Erythema Multiforme. American Family Physician. December 2006. 74(11):1883-1888.
18. Parris, RS, et al. More than Meets the Eye. A 23-Year-Old Woman with Rapidly Progressive Respiratory Failure, Mucositis, and Rash. Annals of the American Thoracic Society. December 2015. Vol 12, No 12.
19. Creamer D, Walsh SA, Dziewulski P, et al. U.K. guidelines for the management of Stevens-Johnson syndrome/toxic epidermal necrolysis in adults 2016. Br J Dermatol 2016; 174:1194.
20. Aurelian, L., et al. Herpes simplex virus (HSV)-associated erythema multiforme (HAEM): a viral disease with an autoimmune component. Dermatology Online Journal. February, 2003. 9(1):1.

	Epidemiology	Common Associations	Clinical Presentation	Treatment
Erythema Multiforme (EM)	Male Adolescents or young adults	HSV infection	Primarily cutaneous EM major (20%) involves mucosa	Symptom management +/- Corticosteroids, IVIg Antivirals for suppression
Mycoplasma Pneumonia Associated Mucositis (MPAM)	Male Children or adolescents	*Mycoplasma pneumoniae* Upper and lower respiratory symptoms Preceding antibiotic use	Primarily mucosal +/- Cutaneous	Symptom management Antibiotics +/- Corticosteroids, IVIg

Table 3: Epidemiology, common associations, clinical presentations and treatments of erythema multiforme and mycoplasma pneumonia associated mucositis.

continued from page 7

view. The degree of RWMA is assessed qualitatively and dichotomously, either demonstrating wall motion abnormality or not. This methodology has been shown to be efficient without sacrificing quality in identifying large RWMA for the purposes of ED evaluation of significant ischemic pathology.[14]

The degree of RWMA detected has been shown to correlate with the degree of ischemia or infarction, meaning multivessel disease will correlate with a larger and more severe myocardial dysfunction as detected on 2DTTE.[19,27,35] This indicates a larger swathe of at risk of myocardium that may benefit from early invasive therapy. Furthermore, when comparing global LV systolic dysfunction assessment with focal wall motion abnormalities, more focal abnormalities have been determined to be a greater predictor of morbidity and mortality in ACS, especially in the NSTE-ACS cohort.[26] The finding of any RWMA at rest is associated with an eightfold increase in adverse cardiac events in the first 48 hours, and fourfold in the next two years.[5,36] Thus, despite equivocal ECG and troponin levels, 2DTTE may more readily identify patients that are at high risk of progressing to myocardial infarction and require early invasive intervention.

Although identifying RWMA can be difficult, several recent studies demonstrate emergency physician are be able to utilize 2DTTE with short periods of training. A case series completed with emergency physician-performed point-of-care ultrasounds for three patients presenting with anginal equivalents with initially non-diagnostic conventional work up identified significant RWMA that correlated with severe single vessel coronary artery stenosis.[27] These studies were completed by ultrasound-fellowship trained ED physicians, with no formal echocardiography training, after a brief 10-minute didactic training video. Another study demonstrated statistically significant improvement in post-test performance in identifying RWMA after a 30-minute module depicting qualitative changes in normal and abnormal echocardiograms.[37] Standardized approach to echocardiogram, including assessment of RWMA, also improves the detection of other mimickers of NSTE-ACS, including aortic dissection, pericardial effusion, valvular pathology, acute heart failure, pulmonary embolus, and others, when utilized routinely by residents.[38,39] Thus, it is feasible for emergency physicians to perform this in real-time with brief didactic and clinical application, and can provide diagnostic clarity with relative ease and widespread availability.

Technical Difficulties

Pitfalls in assessing for RWMA are twofold: during image acquisition and during image interpretation. Images are best analyzed when sufficient quality is obtained to assess for abnormalities, and that those abnormalities can be compared in multiple views. Oftentimes image acquisition can be difficult due to body habitus, underlying pulmonary disease or inability to reposition the patient. Suboptimal images run the risk of leading to a false positive or false negative. Additionally, image interpretation can be difficult, specifically when delineating between acute versus chronic changes. Furthermore, significant valvular pathology, previous cardiomyopathy, acute myocarditis and bundle branch blocks will all make it difficult to assess which changes are new or old, ischemic or non-ischemic. Thus, it can be helpful to compare previous echocardiography imaging with current imaging to assess for acute change. Furthermore, experimental data has shown that a certain degree of myocardial thickness (>20%) and ventricular mass (>5%) needs to be affected in order to be detected as RWMA on 2DTTE.[40] On the other hand, a non-transmural injury only including subendocardial regions or a transmural infarct affecting only a small portion of the ventricular wall are unlikely to cause significant morbidity or mortality compared to the occult lesions causing obvious RWMA.

Take Home Points

- 2DTTE can be beneficial in identifying patients that may benefit from early invasive intervention if suspicion for NSTE-ACS is high and conventional work up is non-diagnostic
- Three cardiac views are required at minimum: parasternal long, parasternal short, apical four
- RMWA can be evident by myocardial thinning during contraction or asymmetric myocardial excursion as witnessed on two orthogonal views
- Correlations with ECG abnormalities and previous echocardiograms can be crucial in identifying acute versus chronic changes

1. National hospital ambulatory medical care survey: 2016 emergency department summary tables. https://www.cdc.gov/nchs/data/nhamcs/web_tables/2016_ed_web_tables.pdf. Accessed Dec 9, 2019.
2. Benjamin EJ MP. Heart disease and stroke Statistics—2019 update: A report from the american heart association. Circulation. 2019.
3. Amsterdam E, Wenger N, Brindis R, et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevtion acute coronary syndromes: A report of the american college of cardiology/american heart association task force on practice guidelines. Circulation. 2014;130:344-426.
4. McGuinness J, Begg T, Semple T.
First electrocardiogram in recent myocardial infarction British Medical Journal. 1976:449-451.
5. Sabia P, Afrookteh A, Touchstone D, Keller M, Esquivel L, Kaul S.
Value of regional Wall Motion Abnormality in the emergency room diagnosis
of acute myocardial infarction
A Prospective study using two-dimensional echocardiography Circulation. 1991;84:85-92.
6. Fesmire F, Percy R, Bardoner J, Wharton D, Calhoun F. Usefulness of automated serial 12-lead ECG monitoring during the initial emergency department evaluation of patients with chest pain. Annals of Emergency Medicine. 1998;31:3-11.
7. Pope J, Ruthazer R, Beshansky J, Griffith J, Selker H. Clinical features of emergency department patients presenting with symptoms suggestive of acute cardiac ischemia: A multicenter study Journal of Thrombosis and Thrombolysis. 1998;6:63-74.
8. MacRae A, Kavsak P, Lustig V, et al. Assessing the requirement for the 6-hour interval between specimens in the american heart association classification of myocardial infarction in epidemiology and clinical research studies. Clinical Chemistry. 2006;52:812-818.
9. Thygesen K, Mair J, Katus H, et al. Recommendations for the use of cardiac troponin measurement in acute cardiac care. European Heart Journal. 2010;31:2197-2206.
10. Garg P, Morris P, Fazlanie A, et al. Cardiac biomarkers of acute coronary syndomre: From history to high-sensitivty cardiac troponin. Internal and Emergency Medicine. 2017;12:147-155.
11. Pope H, Aufderheide T, Ruthazer R, et al. Missed diagnosis of acute cardiac ischemia in the emergency department. The New England Journal of Medicine. 2000;342:1163-1170.
12. ACCF/ASE/AHA/ASNC/HFSA/HRS/SCAI/SCCM/SCCT/SCMR 2011 appropriate use criteria for echocardiography. Journal of the American College of Cardiology. 2011.
13. Horowitz R, Morganroth J, Parratto C, Chen C, Soffer J, Pauletto F. Immediate diagosis of acute myocardial infarction by two-dimensional echocardiography. Circulation. 1982;65:323-329.
14. Loh I, Charuzi Y, Beider A, Marshall L, Ginsburg J. Early diagnosis of nontransmural myocardial infarction by two-dimensional echocardiography. American Heart Journal. 1982;104:963-968.
15. Mahmoud M. Echocardiography in the evaluation fo chest pain in the emergency department. Polish Journal of Radiology. 2017;82:798-805.
16. Muscholl M, Oswald M, Mayer C, von Scheidt W. Prognostic value of 2D echocardiography in patients presenting with acute chest pain and non-diagnostic ECG for ST-elevation myocardial infarction. International Journal of Cardiolgoy. 2002;84:217-225.
17. Kontos M, Kurdziel K, McQueen R, et al. Comparison of 2-dimesional echocardiography and myocardial perfusion imaging for diagnosing myocardial infarction in emergency department patients. American Heart Journal. 2002;143:659-667.
18. Kontos M, Arrowood J, Jesse R, et al. Copmarison between 2-dimesional echocardiography and myocardial perfusion imaging in the emergency department in patients with possible myocardial ischemia. American Heart Journal. 1998;136:724-733.
19. Peels C, Visser C, Kupper A, Visser F, Roos J. Usefulness of two-dimesional echocardiography for immediate detection of myocardial ischemia in the emergency room. The American Journal of Cardiology. 1990;65:687-691.
20. Beller G. Myocardial perfusion imaging for detection of silent myocardial ischemia. The American Journal of Cardiology. 1988;61:22-26.
21. Hauser A, Vellappillil G, Ramos R, Gordon S, Timmis G, Dudlets P. Sequence of mechanical, electrocardiographic and clinical effects of repeated coronary artery occlusion in human beings: Echocardiographic observations during coronary angioplasty. Journal of the American College of Cardiology. 1985;5:193-197.
22. Wohlgelernter D, Cleman M, Highman A, et al. Regional myocardial dysfunction during coronary angioplasty: Evaluation by two-dimensional echocardiography and 12 lead electrocardiography. Journal of the American College of Cardiology. 1986;7:1245-1254.
23. Gerber B, Wijns W, Vanoverschelde J, et al. Myocardial perfusion and oxygen consuption in reperfused noninfarcted dysfunctional myocardium after unstable angina. Journal of American College of Cardiology. 1999;34:1939-1946.
24. Jeroudi M, Cheirif J, Habib G, Bolli R. Prolonged wall motion abnormalities after chest pain at rest in patients with unstable angina: A possible manifestation of myocardial stunning. American Hearth Journal. 1993;127:1241-1250.
25. Kalvaitis S, Kaul S, Tong K, Rinkevich D, Belcik T, Wei K. Effect of time delay on the diagnostic use of contrast echocardiography in patients presenting to the emergecy department with chest pain and no ST segment elevation. Journal of the American Society of Echocardiography. 2006;19:1488-1493.
26. Muller J, Hillis G, Oh J, Reeder G, Gersh B, Pellikka P. Wall motion score index and ejection fraction for stratification after acute myocardial infarction. American Heart Journal. 2006;151:419-425.
27. Frenkel O, Riguzzi C, Nagdev A. Identification of high-risk patients with acute coronary syndrome using point-of-care echocardiography in the ED. American Journal of Emergency Medicine. 2014;32:670-672.
28. Sasaki H, Charuzi Y, Beeder C, Sugiki Y, Lew A. Utility of echocardiography for the early assessment fo patients with nondiagonstic chest pain. American Heart Journal. 1986;112:494-497.
29. Esmaeilzadeh M, Parsaee M, Maleki M. The role of echocardiography in coronary artery disease and acute myocardial infarction. The Journal of Tehran University Heart Center. 2012:1-13.
30. Cerqueira M, Weissman N, Dilsizian V, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart A statement for healthcare professionals from the cardiac imaging committee of the council on clinical cardiology of the american heart association Circulation. 2002;539-542.
31. Ortiz-Perez J, Rodriguez J, Meyers S, Lee D, Davidson C, Wu E. Correspondence between the 17-segment model and coronary arterial anatomy using contrast-enhanced cardiac magnetic resonance imaging. Journal of American College of Cardiology Cardiovascular Imaging. 2008;1:282-293.
32. Teran F, Vanyo L. Interpretation of cardiac regional wall motion abnormalities by echocardiography – A simplified approach. https://sinaiem.org/education/regional-wall-motion-assessment/. Accessed 12/14, 2019.
33. Johnson B, Lovallo E, Frenkel O, Nagdev A. Detect cardiac regional wall motion abnormalities by point-of-care echocardiography. https://www.acepnow.com/article/detect-cardiac-regional-wall-motion-abnormalities-point-care-echocardiography/?singlepage=1&theme=print-friendly. Accessed 12/14, 2019.

than 13,000 subjects, this study demonstrated no difference in hospital-free days between patients admitted to non-ICU settings who were randomized to receive chloride restrictive fluids versus NS. However, patients receiving NS were more likely to die, receive new renal replacement therapy, or suffer from persistent renal dysfunction within 30 days. Theseoutcomes defined the major adverse kidney event (MAKE-30) composite endpoint. These findings were echoed in the concurrently published SMART trial, which demonstrated an increased incidence of MAKE-30 among patients admitted from the ED to the ICU resuscitated with NS compared to buffered crystalloid.[13] The authors of each study noted that these findings are hypothesis-generating, given that each was designed primarily to assess mortality rather than the composite outcome. Some critics argue that further large-scale investigation of IVF for all comers is not warranted, and that clinicians should individualize fluid choice based on pathophysiology.[15] Fortunately, there are a number of previously published investigations that shed light on fluid choice in a number of common illness states.

Considerations in Specific Patient Populations

Sepsis

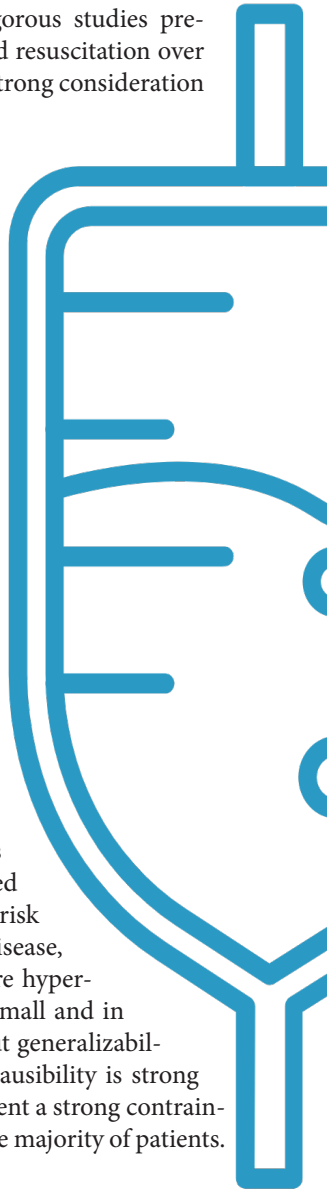
Fluid administration has been an integral piece of emergent sepsis management since the advent of early goal directed therapy.[16] While some components of Dr. Rivers' protocol have faded, aggressive fluid resuscitation remains a cornerstone of treatment. Subgroup analysis of patients with severe sepsis or septic shock in the Saline versus Albumin Fluid Evaluation (SAFE) study demonstrated a non-significant lower odds of mortality in patients receiving albumin (aOR 0.71, 95% CI 0.52-0.97).[17] However, albumin has not been shown to be superior to crystalloid in subsequent trials, and given the high price associated, it is not recommended for routine use.[18] High quality data guiding the choice between crystalloids in sepsis are lacking. A small, retrospective review of 115 septic patients receiving balanced fluids or NS in the ED demonstrated that significantly fewer patients in the balanced group died during their hospitalization.[19] In patients receiving both, each increase of 1% in the proportion of balanced fluid was associated with a 2.3% decrease in the odds of in-hospital mortality. Further study is certainly warranted to determine the true effect size of balanced resuscitation in patients with sepsis, a disease process that certainly continues to fill the beds in the ED worldwide.

Diabetic Ketoacidosis

Patients suffering from diabetic ketoacidosis (DKA) receive aggressive fluid replacement early in their clinical course, due in part to the osmotic diuresis of severe hyperglycemia. Professional society guidelines recommend NS as the initial fluid for restoring intravascular volume.[20] LR and PL have been compared to NS in several small studies over the past decade. In a retrospective trial in Australian ICUs, nine patients in the PL-only group had quicker time to resolution of acidemia and less hyperchloremia than the fourteen patients in the NS-only group.[21] Similarly, in a prospective, randomized, double blinded trial of NS versus LR in DKA, the LR arm returned to a normal pH more quickly than the NS arm, although this did not reach statistical significance.[22] Further, a third prospective, randomized study demonstrated that patients receiving balanced crystalloid were significantly less likely to develop hyperchloremic metabolic acidosis than patients resuscitated with NS.[23] The absence of methodologically rigorous studies precludes strong recommendations for balanced resuscitation over NS, but these data serve as a foundation for strong consideration on a case-to-case basis.

Hyperkalemia

Unlike NS, LR and PL both contain potassium. At first glance, supplying a patient afflicted with hyperkalemia more potassium in his or her IVF may seem like a problem. However, consideration of potassium ion exchange and affect of acid-base buffers suggests otherwise. By definition, serum potassium is greater than 5-5.5 millimoles per deciliter in hyperkalemia. Adding any fluid with a concentration less than the serum potassium is unlikely to lead to an increase in the serum value for several reasons. First, adding a hypotonic solution to a fixed volume does not increase tonicity. Second, the pH of the supplied fluid has an effect on serum potassium, with relatively acidic solutions pulling potassium out of the intracellular space, and alkalizing fluid pushing the ion into the cells. Based on these premises, one might expect balanced fluids to decrease serum potassium when compared to NS. Several studies in populations at high risk of hyperkalemia, primarily end-stage renal disease, have demonstrated NS tends to lead to more hyperkalemia.[24-26] These trials were all relatively small and in a selected population, raising concerns about generalizability. That said, the argument of biological plausibility is strong enough that hyperkalemia should not represent a strong contraindication to potassium-containing fluids in the majority of patients.



Traumatic Brain Injury

While patients suffering from critical illness, sepsis, DKA, or hyperkalemia should likely be treated with balanced resuscitation, traumatic brain injury (TBI) represents a different problem all together. The complex interplay of the neurovascular cellular system (consisting of the endothelium and surrounding support cells of the central nervous system) with plasma components is key to maintaining cerebral water volume.[27] Hypotonic fluids can have multiplied effects on a disrupted blood brain barrier, thereby increasing the likelihood of developing cerebral edema. In a post-hoc analysis of the SAFE study, patients with TBI were more likely to die when treated with albumin rather than NS.[28] While there are no large-scale trials pitting NS against balanced solutions, a retrospective comparison of prehospital fluid for patients with TBI demonstrated an increased adjusted risk for mortality at 30 days for patients receiving LR compared to NS.[29] Prospective trials may be forth coming, but for the time being, the physiologic argument against administering hypotonic fluid to patients with significant intracranial injury is enough to justify the use of normal saline in this patient population.

IV Fluids
continued on page 14

IV Fluids

continued from page 13

Summary

NS has taken hold as the de facto resuscitative fluid in the majority of health care institutions in the developed world. Recent data suggest that balanced fluids may provide benefit to patients suffering from a wide array of critical illnesses. Outside of TBI, chloride restrictive resuscitation should be the primary strategy in the modern ED.

1.Glassford NJ, Bellomo R. The complexities of intravenous fluid research: questions of scale, volume, and accumulation. Korean J Crit Care Med 2016;31:276–299.
2.Myburgh, J. A., Finfer, S., Bellomo, R., Billot, L., Cass, A., Gattas, D., ... & McGuinness, S. (2012). Hydroxyethyl starch or saline for fluid resuscitation in intensive care. New England Journal of Medicine, 367(20), 1901-1911.
3.Perner, A., Haase, N., Guttormsen, A. B., Tenhunen, J., Klemenzson, G., Åneman, A., ... & Bendtsen, A. (2012). Hydroxyethyl starch 130/0.42 versus Ringer's acetate in severe sepsis. New England Journal of Medicine, 367(2), 124-134.
4.SAFE Study Investigators. (2004). A comparison of albumin and saline for fluid resuscitation in the intensive care unit. New England Journal of Medicine, 350(22), 2247-2256.
5.Caironi, P., Tognoni, G., Masson, S., Fumagalli, R., Pesenti, A., Romero, M., ... & Iapichino, G. (2014). Albumin replacement in patients with severe sepsis or septic shock. New England Journal of Medicine, 370(15), 1412-1421.
6.Annane, D., Siami, S., Jaber, S., Martin, C., Elatrous, S., Declère, A. D., ... & Trouillet, J. L. (2013). Effects of fluid resuscitation with colloids vs crystalloids on mortality in critically ill patients presenting with hypovolemic shock: the CRISTAL randomized trial. Jama, 310(17), 1809-1817.
7.Weinberg, L., Li, M. H. G., Churilov, L., Armellini, A., Gibney, M., Hewitt, T., ... & Bellomo, R. (2018). Associations of fluid amount, type, and balance and acute kidney injury in patients undergoing major surgery. Anaesthesia and intensive care, 46(1), 79-87.
8.Shaw, A. D., Bagshaw, S. M., Goldstein, S. L., Scherer, L. A., Duan, M., Schermer, C. R., & Kellum, J. A. (2012). Major complications, mortality, and resource utilization after open abdominal surgery: 0.9% saline compared to Plasma-Lyte. Annals of surgery, 255(5), 821-829.
9.Shaw, A. D., Raghunathan, K., Peyerl, F. W., Munson, S. H., Paluszkiewicz, S. M., & Schermer, C. R. (2014). Association between intravenous chloride load during resuscitation and in-hospital mortality among patients with SIRS. Intensive care medicine, 40(12), 1897-1905.
10.Raghunathan, K., Bonavia, A., Nathanson, B. H., Beadles, C. A., Shaw, A. D., Brookhart, M. A., ... & Lindenauer, P. K. (2015). Association between initial fluid choice and subsequent in-hospital mortality during the resuscitation of adults with septic shock. Anesthesiology: The Journal of the American Society of Anesthesiologists, 123(6), 1385-1393.
11.Young, P., Bailey, M., Beasley, R., Henderson, S., Mackle, D., McArthur, C., ... & Reddy, S. (2015). Effect of a buffered crystalloid solution vs saline on acute kidney injury among patients in the intensive care unit: the SPLIT randomized clinical trial. Jama, 314(16), 1701-1710.
12.Semler, M. W., Wanderer, J. P., Ehrenfeld, J. M., Stollings, J. L., Self, W. H., Siew, E. D., ... & Rice, T. W. (2017). Balanced crystalloids versus saline in the intensive care unit. The SALT randomized trial. American journal of respiratory and critical care medicine, 195(10), 1362-1372.
13.Semler, M. W., Self, W. H., Wanderer, J. P., Ehrenfeld, J. M., Wang, L., Byrne, D. W., ... & Guillamondegui, O. D. (2018). Balanced crystalloids versus saline in critically ill adults. New England Journal of Medicine, 378(9), 829-839.
14.Self, W. H., Semler, M. W., Wanderer, J. P., Wang, L., Byrne, D. W., Collins, S. P., ... & Shaw, A. D. (2018). Balanced crystalloids versus saline in noncritically ill adults. New England Journal of Medicine, 378(9), 819-828.
15.Vincent, J. L., & De Backer, D. (2016). Saline versus balanced solutions: are clinical trials comparing two crystalloid solutions really needed?. Critical Care, 20(1), 250.
16.Rivers, E., Nguyen, B., Havstad, S., Ressler, J., Muzzin, A., Knoblich, B., ... & Tomlanovich, M. (2001). Early goal-directed therapy in the treatment of severe sepsis and septic shock. New England Journal of Medicine, 345(19), 1368-1377.
17.SAFE Study Investigators. (2011). Impact of albumin compared to saline on organ function and mortality of patients with severe sepsis. Intensive care medicine, 37(1), 86-96.
18.Caironi, P., Tognoni, G., Masson, S., Fumagalli, R., Pesenti, A., Romero, M., ... & Iapichino, G. (2014). Albumin replacement in patients with severe sepsis or septic shock. New England Journal of Medicine, 370(15), 1412-1421.
19.Sethi, M., Owyang, C. G., Meyers, C., Parekh, R., Shah, K. H., & Manini, A. F. (2018). Choice of resuscitative fluids and mortality in emergency department patients with sepsis. The American journal of emergency medicine, 36(4), 625-629.
20.Kitabchi, A. E., Umpierrez, G. E., Miles, J. M., & Fisher, J. N. (2009). Hyperglycemic crises in adult patients with diabetes. Diabetes care, 32(7), 1335-1343.
21.Chua, H. R., Venkatesh, B., Stachowski, E., Schneider, A. G., Perkins, K., Ladanyi, S., ... & Bellomo, R. (2012). Plasma-Lyte 148 vs 0.9% saline for fluid resuscitation in diabetic ketoacidosis. Journal of critical care, 27(2), 138-145.
22.Van Zyl, D. G., Rheeder, P., & Delport, E. (2011). Fluid management in diabetic-acidosis—Ringer's lactate versus normal saline: a randomized controlled trial. QJM: An International Journal of Medicine, 105(4), 337-343.
23.Mahler, S. A., Conrad, S. A., Wang, H., & Arnold, T. C. (2011). Resuscitation with balanced electrolyte solution prevents hyperchloremic metabolic acidosis in patients with diabetic ketoacidosis. The American journal of emergency medicine, 29(6), 670-674.
24.Weinberg, L., Harris, L., Bellomo, R., Ierino, F. L., Story, D., Eastwood, G., ... & Mount, P. F. (2017). Effects of intraoperative and early postoperative normal saline or Plasma-Lyte 148® on hyperkalaemia in deceased donor renal transplantation: a double-blind randomized trial. BJA: British Journal of Anaesthesia, 119(4), 606-615.
25.Khajavi, M. R., Etezadi, F., Moharari, R. S., Imani, F., Meysamie, A. P., Khashayar, P., & Najafi, A. (2008). Effects of normal saline vs. lactated ringer's during renal transplantation. Renal failure, 30(5), 535-539.
26.O'Malley, C. M., Frumento, R. J., Hardy, M. A., Benvenisty, A. I., Brentjens, T. E., Mercer, J. S., & Bennett-Guerrero, E. (2005). A randomized, double-blind comparison of lactated Ringer's solution and 0.9% NaCl during renal transplantation. Anesthesia & Analgesia, 100(5), 1518-1524.
27.Ertmer, C., & Van Aken, H. (2014). Fluid therapy in patients with brain injury: what does physiology tell us?.
28.SAFE Study Investigators. (2007). Saline or albumin for fluid resuscitation in patients with traumatic brain injury. New England Journal of Medicine, 357(9), 874-884.
29.Rowell, S. E., Fair, K. A., Barbosa, R. R., Watters, J. M., Bulger, E. M., Holcomb, J. B., ... & PROMMTT Study Group. (2016). The impact of pre-hospital administration of lactated ringer's solution versus normal saline in patients with traumatic brain injury. Journal of neurotrauma, 33(11), 1054-1059.

Neurocysticercosis

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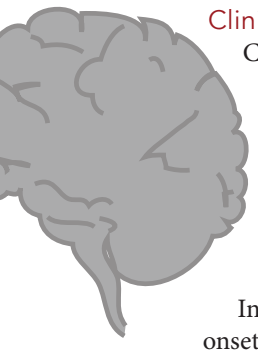

Clinical Presentation

Clinical manifestations and severity of neurocysticercosis vary widely based on characteristics of the infection and host (number, size, location of cysts and intensity of host's immune response). The symptomatology of neurocysticercosis can be separated into two main groups, intraparenchymal or extraparenchymal.

Intraparenchymal lesions are more common, with onset usually occurring a few years from initial infection.[4]Location and degenerating stage of cysts affect the timing and symptomology demonstrated, with newer developing cysts instigating an intense inflammatory response, and older calcified cysts that may be asymptomatic with only intermittent flares of inflammation.[4] In general, intraparenchymal cysts are most commonly associated with focal seizures with secondary generalization and headache, less commonly with altered vision and focal neurologic deficits, and rarely, psychiatric symptoms.[2,3] If a large number of parenchymal cysts are present, leading to a significant host inflammatory response, the patient may present with a clinical picture resembling encephalitis. Because the onset of symptoms can be subacute and subclinical, many cases are identified incidentally when neuroimaging is performed for other reasons in patients who are asymptomatic or who have less severe presentations of the disease.[5]

In contrast, extraparenchymal cysts, particularly cysts located in the intraventricular and subarachnoid spaces, are associated with symptoms of elevated intracranial pressure, such as headache, nausea, vomiting, and visual disturbances due to a massive inflammatory response or direct obstruction of cerebrospinal fluid flow, and may be accompanied by altered mental status. Mobile lesions in the third or fourth ventricle can cause intermittent obstruction depending upon patient positioning, leading to episodic symptoms. Spinal cord lesions are exceedingly rare, and can cause radicular pain and sensimotor deficits.[2,3] Lastly, ocular lesions can involve any portion of the globe and cause impaired vision, diplopia, and blindness. Ophthalmologic examination to rule out ocular involvement is paramount prior to initiation of antiparasitic therapy as treatment can otherwise cause ocular inflammation and blindness if parasites are not surgically removed from the eye prior to systemic therapy.[6] Direct visualization of the parasite can be done via fundoscopic exam.

Diagnosis

Diagnosis of neurocysticercosis starts with a travel history, since patients who have not been to an endemic country are unlikely to have the disease. However, suspicion must remain high in patients who have ever traveled to endemic areas, even for brief periods of time, as disease course can be indolent and develop several years after initial infection. CT or MRI of the head confirm the diagnosis. Radiographic findings can vary, including cystic lesions, enhancing lesions and calcifications, and findings can demonstrate cysts in different stages. Pathognomonic findings include identification of the scolex of the tapeworm within the cystic lesion. [will include representative image]. CT is more sensitive for identifying calcifications and ocular involvement, whereas MRI can help detect smaller lesions and evaluate for degenerative change and edema.[3,6] Serologic testing should be performed as confirmation, especially when CT/MRI are suggestive but not diagnostic. The serologic test of choice is enzyme-linked immunoelectrotransfer blot (EITB) using parasite glycoproteins performed on serum.[6,7] Sensitivity for serum testing is generally greater than cerebrospinal fluid testing, but is directly related to the number of lesions, and thus can be unreliable in single lesion cases. If clinical suspicion remains high despite equivocal findings using the diagnostic testing above, brain biopsy can be performed to confirm a diagnosis.

Treatment and Prognosis

Treatment of neurocysticercosis is focused both on treating the underlying infection as well as the complications of the disease pro-

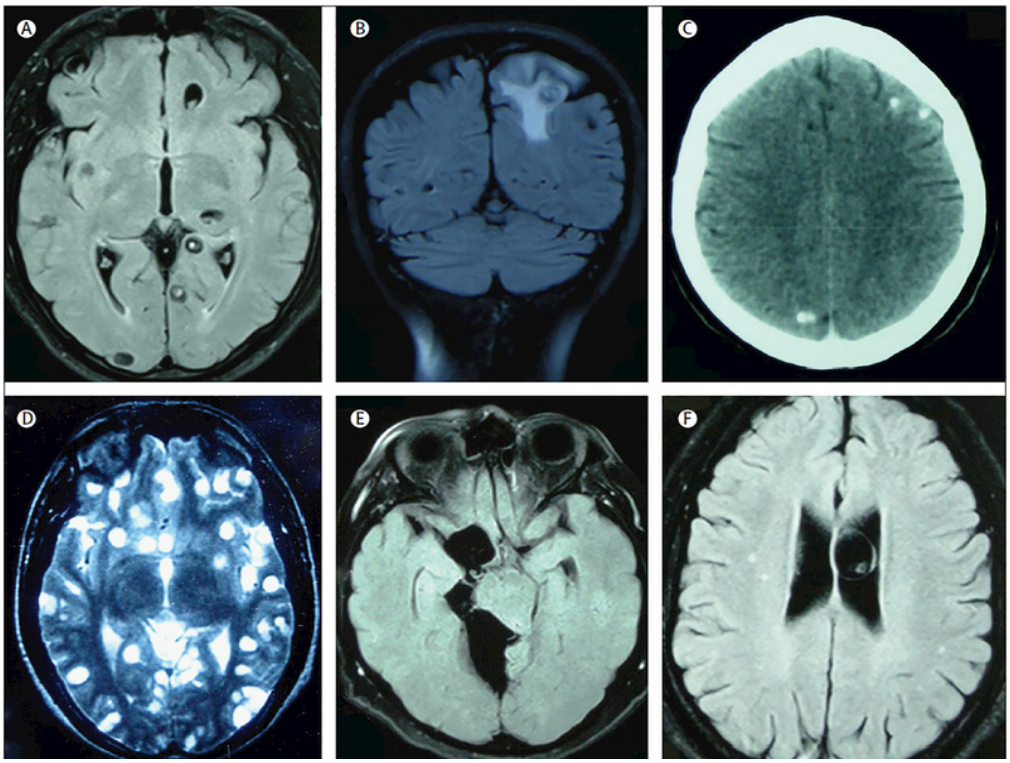

Image 12: MRI images depicting different findings indicating viable cyst, including brain calcifications (c), parenchymal cyst (d), basal subarachnoid cyst (e) and intraventricular cyst (f). Image courtesy of: Garcia HH, Nash TE, Del Brutto OH. Clinical symptoms, diagnosis, and treatment of neurocysticercosis. The Lancet Neurology. 2014 Dec 1;13(12):1202-15.

cess.[6] The order of the treatment employed largely depends on the severity of the clinical presentation. If a patient presents in status epilepticus or with severely increased intracranial pressure, measures for treating these life-threatening conditions outweigh initiation of antiparasitics. Thus, evaluation of airway protection along with quick initiation of anti-epileptics or emergent external ventricular drain placement take precedence, if needed. Furthermore, initiation of antiparasitic therapy can risk exacerbating severe neurologic symptoms due to increasing inflammatory response around the degenerating cysts, especially in patients with a large disease burden.

In treating less emergent complications, such as seizures and cerebral edema, first-line antiepileptic drugs (AED) are as effective in neurocysticercosis as in treating idiopathic epilepsy, and patients should remain on AED therapy for at least two years afterward to prevent recurrent seizures.[2] As discussed before, a significant inflammatory response can be seen in neurocysticercosis, and subsequently corticosteroids are universally used. A trial of dexamethasone 0.2-0.4 mg/kg per day should be initiated. Therapy can hasten resolution of active cysts, diminish seizure risk, and reduce likelihood of recurrent hydrocephalus. Duration of steroids may differ depending upon disease burden and location, but will usually accompany at least the duration of anti-parasitic treatment. Prior to initiation of long-term steroids, serum testing for latent tuberculosis and strongyloides should be completed.

In terms of antiparasitic treatment, both albendazole and praziquantel are cysticidal, resulting in eventual cyst resolution and calcification. However, use of these agents should be in conjunction with an infectious disease specialist, as location and number of cysts can affect the efficacy of each drug. Antiparasitic treatment is usually delayed until steroids have been administered in order to prevent worsening inflammatory response. Furthermore, a patient can have cysts in several different stages at the time of treatment, requiring a prolonged course until all cysts have resolved. Monotherapy can be used in patients with one to two cysts, with a typical regimen being albendazole 15 mg/kg per day divided in two doses. For patients with greater than two cysts, treatment is most effective with albendazole in addition to praziquantel 50 mg/kg per day divided in three doses.[2] Treatment duration is, at minimum, ten days. Follow up neuroimaging is usually conducted biannually until resolution of cysts are evident.[4] If cystic lesions persist, additional therapy is warranted.

Neurosurgical intervention, including neuroendoscopy, cyst removal and shunt placement, may offer the benefit of removing source of infection and reducing the length of antiparasitic and corticosteroid courses required. Obstructive hydrocephalus requires surgical intervention, as seen in our case, while communicating hydrocephalus may ultimately require a ventriculoperitoneal shunt.[6]

In terms of prognostication, intraparenchymal cysts tend to respond better to the above therapies when compared to extraparenchymal cysts, with single cysts portending a better outcome than numerous cysts. Extraparenchymal neurocysticercosis has a worse prognosis, thought to be secondary to larger parasite loads and less restricted growth of individual cysts.[3]

Summary

In summary, although neurocysticercosis is not endemic to the United States, clinical suspicion for this disease process must remain high in patients with any travel history to endemic regions, especially immigrants, as the disease process can be chronic and subclinical, and with high morbidity and mortality. Emergent diagnosis and immediate treatment for complications is paramount, and these patients should be co-managed with infectious disease, neurology, and neurosurgery, as long-term treatment and follow up is needed.

1. Fabiani S, Bruschi F. Neurocysticercosis in Europe: still a public health concern not only for imported cases. Acta tropica. 2013 Oct 1;128(1):18-26.
2. Garcia HH, Nash TE, Del Brutto OH. Clinical symptoms, diagnosis, and treatment of neurocysticercosis. The Lancet Neurology. 2014 Dec 1;13(12):1202-15.
3. Gripper LB, Welburn SC. Neurocysticercosis infection and disease–A review. Acta tropica. 2017 Feb 1;166:218-24.
4. Nash TE, Garcia HH. Diagnosis and treatment of neurocysticercosis. Nat Rev Neurol 2011; 7:584.
5. Carabin H, Ndimubanzi PC, Budke CM, Nguyen H, Qian Y, Cowan LD, Stoner JA, Rainwater E, Dickey M. Clinical manifestations associated with neurocysticercosis: a systematic review. PLoS neglected tropical diseases. 2011 May 24;5(5):e1152.
6. White Jr AC, Coyle CM, Rajshekhar V, Singh G, Hauser WA, Mohanty A, Garcia HH, Nash TE. Diagnosis and treatment of neurocysticercosis: 2017 clinical practice guidelines by the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH). Clinical Infectious Diseases. 2018 Feb 22;66(8):e49-75.
7. Tsang VC, Brand JA, Boyer AE. An enzyme-linked immunoelectrotransfer blot assay and glycoprotein antigens for diagnosing human cysticercosis (Taenia solium). Journal of Infectious Diseases. 1989 Jan 1;159(1):50-9.

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EKG focus

Shark Fin Sign: A STEMI Equivalent

A 55-year-old female presents to the emergency department with complaints of left-sided jaw pain and nausea for the past twenty minutes. She appears diaphoretic and in acute distress. While vital signs are being obtained, she becomes unresponsive and pulseless. Cardiac monitoring reveals a monomorphic, wide-complex tachycardia. The patient is defibrillated once with return of spontaneous circulation. A subsequent 12-lead electrocardiogram (ECG) demonstrates the following findings.

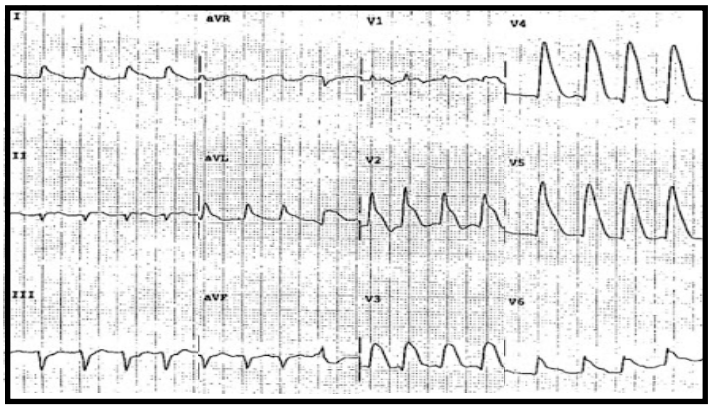

Figure 4: ECG depicting "Shark Fin" sign in leads V2-V6. Courtesy of: "Shark Fin": A Deadly ECG Sign that you Must Know! Dr. Smith's ECG Blog. http://hqmeded-ecg.blogspot.com/2018/06/shark-fin-deadly-ecg-sign-that-you-must.html.

The "Shark Fin" sign seen here, also known as the "Giant R-wave," is a unique phenomenon that occurs in the setting of acute myocardial ischemia and infarction. As cited in a few experimental studies, the first manifestation of transmural myocardial ischemia is an increase in R-wave amplitude and disappearance of the S-wave thought to be caused by cellular shifts in ions and change in membrane action potentials due to injured myocytes.[1,2] This then morphs into widening of the R-wave that causes eventual blunting between the elevated ST segment and growing R wave, and produces a monophasic concave structure that resembles a shark fin.[1] These findings are transient, found within the first several minutes after an acute myocardial insult, and have been documented to quickly evolve to the more conventional ECG presentations of ST elevation myocardial infarctions.[1,3] Thus, these findings are often not seen due to delayed patient presentation.

Clinically, when seen, the interpretation of this uncommon ECG finding in the critically ill can be mistaken for a myriad of other pathologies, likely making the incidence of this phenomenon greater than that acknowledged in the literature. Due to the obscuring of the QRS and ST segments, the "Shark Fin" can be misdiagnosed as an arrhythmia, such as ventricular tachycardia; a conduction abnormality, such as left and right bundle branch block; an electrolyte derangement, such as hyperkalemia; or a toxicologic process, such as tricyclic antidepressant overdose.[2] However, it is important to keep in mind that the "Shark Fin" sign is most prominent in the lead distribution correlating with the culprit artery, and oftentimes the remaining leads continue to maintain the clarity of their QRS-ST segments.[4,5] Thus, searching for the J-point in the unaffected segments can help differentiate between a widened QRS complex, as is common in the other pathologies listed above, from a blunted ST segment elevation, as characteristic of the "Shark Fin."[4,5]

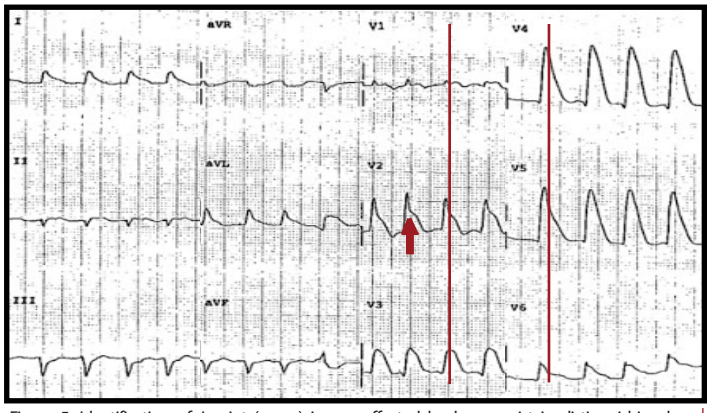

Figure 5: Identification of J-point (arrow) in non-affected lead can assist in distinguishing between QRS wave and ST segments (lines). Adapted from Figure 4.

Although there is a paucity of literature on this topic, there are a few case reports demonstrating an association between ventricular tachydysrhythmias early in clinical course and patients found to have the "Shark Fin" sign.[3,4] This may indicate that the increase in R-wave amplitude is related to the degree of myocardial ischemia occurring, with studies demonstrating this finding only in cases with severe transmural ischemia or a large area of myocardial tissue at risk. Thus, the "Shark Fin" sign has been found to hold a poor prognosis, making it paramount for emergency physicians to recognize it quickly and to arrange for emergent reperfusion interventions for best patient outcome.

1. Ekmekci A, Toyoshima H, Kwoczynski JK, Nagaya T, Prinzmetal M. Angina pectoris: V. Giant R and receding S wave in myocardial ischemia and certain nonischemic conditions. Am J Cardiol. 1961;7(4):521-532. doi:10.1016/0002-9149(61)90510-0.
2. Madias JE, Krikelis EN. Transient giant R waves in the early phase of acute myocardial infarction: Association with ventricular fibrillation. Clin Cardiol. 1981;4(6):339-349. doi:10.1002/clc.4960040606.
3. Madias JE. The "giant R waves" ECG pattern of hyperacute phase of myocardial infarction: A case report. J Electrocardiol. 1993;26(1):77-82. doi:10.1016/0022-0736(93)90068-O.
4. "Shark Fin": A Deadly ECG Sign that you Must Know! Dr. Smith's ECG Blog. http://hqmeded-ecg.blogspot.com/2018/06/shark-fin-deadly-ecg-sign-that-you-must.html.
5. Giant R-waves. What are they? Dr. Smith's ECG Blog. http://hqmeded-ecg.blogspot.com/2015/07/giant-r-waves-what-are-they.html.

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.

Case	Providers
Acetaminophen Overdose	Leech/Baxter
Hypercalcemia	Walsh/Ryan
Testicular Torsion	Meigh/Fernandez
Dural Venous Sinus Thrombosis	Jensen/Bryant
EBV Hepatitis	Klaszky/Fermann
Spinal Cord Contusion	Iparraguierre/Bryant
Capsular Warning Syndrome	Iparraguierre/Bryant