Critical decisions in emergency medicine

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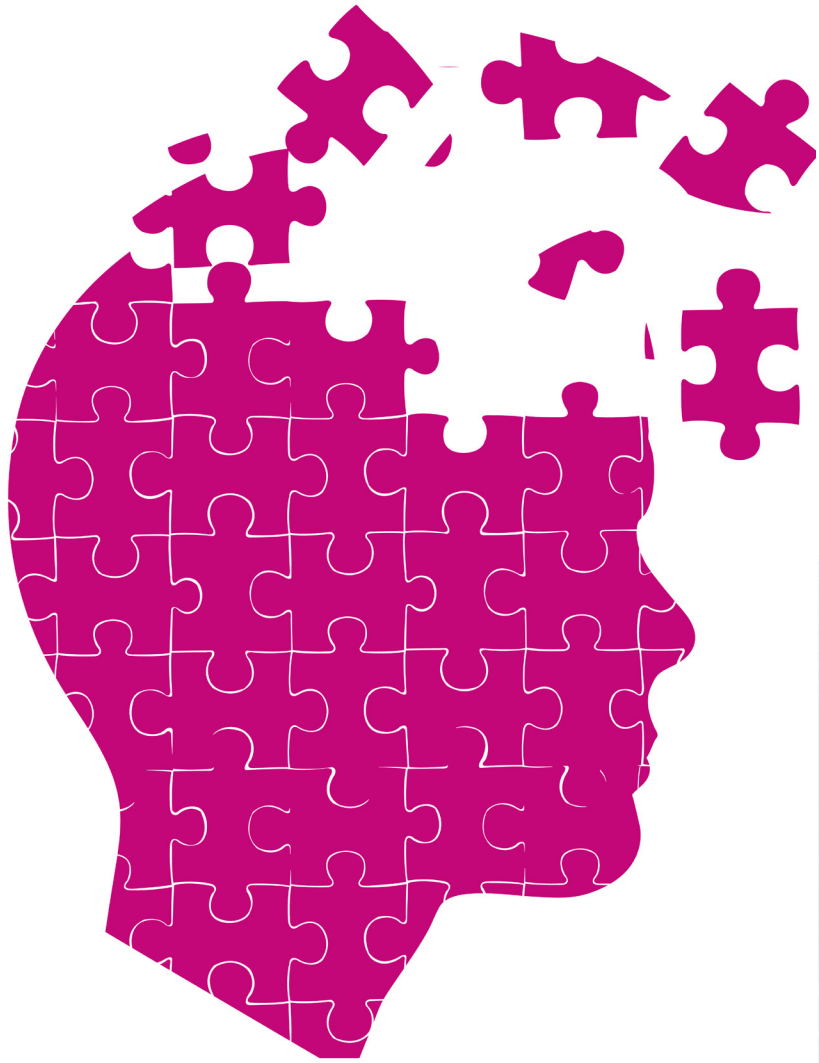

Race Against Time

Seizures are a common presentation in the emergency department. In the United States, an estimated 1% of the adult population is diagnosed with epilepsy. Additionally, multiple other disease processes can cause seizures. Emergency physicians must understand the range of seizure management, from a first-time seizure to status epilepticus, and specific treatment pathways for different seizure presentations to reduce neuronal damage as much as possible.

Sticks and Stones

Growth plate fractures account for a significant portion of pediatric fractures and, in some cases, can lead to bone growth arrest. Emergency physicians must understand how growth plate fractures are classified and their effects on patient morbidity to provide appropriate care and reduce the risk of complications associated with these common fractures.

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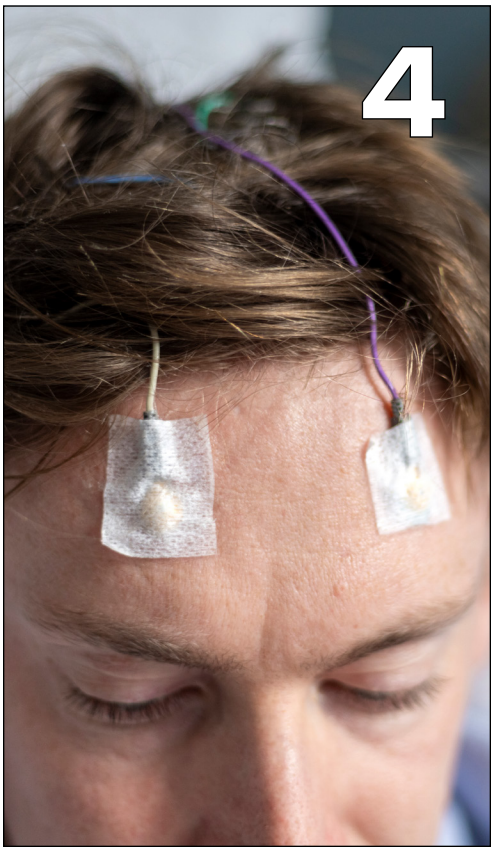

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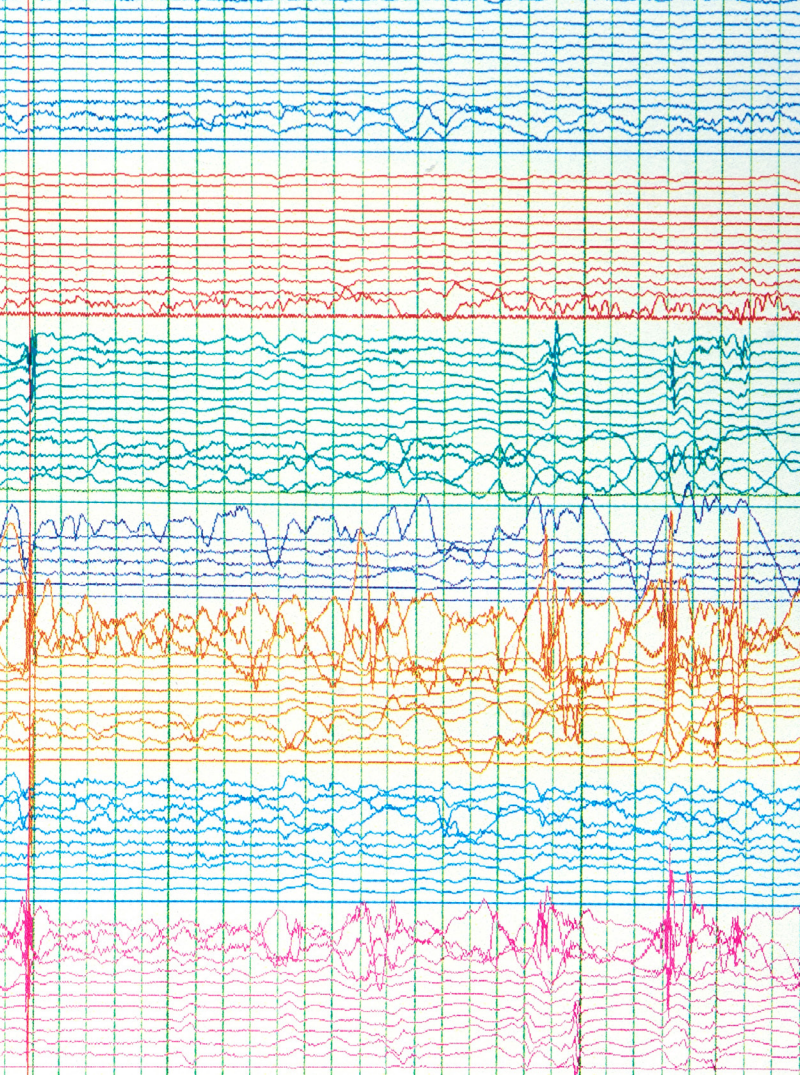

Race Against Time

Seizure Presentations

LESSON 3

By Andrew Piner, MD; and Wan-Tsu W. Chang, MD

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Reviewed by Danya Khoujah, MBBS, MEHP, FACEP

Objectives

On completion of this lesson, you should be able to:

1. Assess patients with first-time seizures.
2. Apply the critical timeline in the management of status epilepticus.
3. Describe the rationale for escalating antiseizure medications in the management of status epilepticus.
4. Recognize patients who need to be intubated for refractory status epilepticus.
5. Evaluate patients for reversible causes of seizure.

From the EM Model

12.0 Nervous System Disorders
12.9 Seizure Disorders
12.9.1 Epileptiform
12.9.1.3 Status Epilepticus

CRITICAL DECISIONS

- What is the workup for patients who present with a first-time seizure?
- What is the first-line treatment for status epilepticus?
- What is the second-line treatment for status epilepticus?
- How should refractory status epilepticus be managed?
- What reversible causes of seizures require disease-specific treatments?

Seizures are a common presentation in the emergency department. In the United States, an estimated 1% of the adult population is diagnosed with epilepsy.[1] Additionally, multiple other disease processes can cause seizures. Emergency physicians must understand the range of seizure management, from a first-time seizure to status epilepticus, and specific treatment pathways for different seizure presentations to reduce neuronal damage as much as possible.

CASE PRESENTATIONS

CASE ONE

A 33-year-old woman is brought in via EMS for an evaluation of seizures. Full body convulsions with loss of consciousness at home prompted her husband to call 911. He indicates that she has a history of hypertension but is otherwise healthy. The EMS crew states that when they arrived at the home, the patient was no longer seizing. They estimate the seizures lasted about 1 minute based on the dispatcher's information. During transport, the patient was groggy and slow to respond, but her vital signs remained normal. She is now beginning to return to her baseline.

CASE TWO

A 45-year-old man with a history of epilepsy presents via EMS for a seizure. The EMS crew was a basic life support unit and was unable to administer any medications. The crew provided supplemental oxygen and checked the patient's glucose level using a finger-stick test. The patient is now continuously seizing as he is moved over to the hospital stretcher.

CASE THREE

A 60-year-old man presents for evaluation of a seizure. He was found seizing by EMS, who administered midazolam 10 mg IM, which resolved the convulsions. He arrives after the seizure, confused and weak but moving all extremities and tolerating his secretions. He is tachycardic and afebrile, with a MAP of 72 mm Hg. The patient's family states that the patient has felt unwell for the past few days and has been experiencing nausea, vomiting, and diarrhea. Point-of-care laboratory tests show normal potassium and glucose levels but a sodium level of 110 mEq/L. He begins seizing in the emergency department.

Introduction

Seizure presentations occur frequently in the emergency department. Although many classifications exist, the American College of Emergency Physicians (ACEP) describes a simple seizure (ie, generalized convulsive seizure) as "generalized movements with unresponsiveness reflecting excessive synchronous cortical electrical activity."[2]

Seizures range from a self-terminating event to a life-threatening illness that requires multidisciplinary care. Critical management of patients — from first-time presentation to refractory status epilepticus — involves understanding the clinical trajectory and optimal time frame for intervention. Critical management also includes addressing specific reversible conditions associated with seizures.

Although many patients present with seizure-like activity, some are diagnosed with other conditions like syncope, transient convulsive episodes, or movement disorders. Therefore, one of the most important tasks for emergency physicians is to obtain a thorough history from witnesses of the episode and preceding events. Many bystanders describe any convulsive-like episode as a seizure. When available, video recordings of the event are helpful.

If the seizure-like activity is thought to have been an actual seizure, then a key aspect of the evaluation is to determine whether it was provoked. An acute provoked event describes a seizure that occurs within 7 days from an inciting factor, such as trauma, a stroke, a toxin, or a metabolic abnormality. Seizures due to underlying pathology like preexisting brain lesions or conditions that are not in an acute phase of illness (eg, cerebral palsy or remote traumatic brain injury) are called remote symptomatic, or unprovoked, seizures.[2]

CRITICAL DECISION

What is the workup for patients who present with a first-time seizure?

Evaluation of First-Time Seizures

It is worth emphasizing the importance of a good history and physical examination in patients who experience seizures. The history and physical examination include a detailed neurologic examination and an assessment of provoking risk factors or traumatic sequelae of the seizure activity. In addition to loss of consciousness and a convulsive phase, lateral tongue biting is associated with an increased likelihood of having had a seizure as opposed to pure syncope. Because most isolated seizure episodes are self-limited, patients often present in a postictal phase. Unilateral weakness can be seen in a single limb or hemibody, a phenomenon called Todd paresis.

Basic laboratory studies should be considered for patients who present for evaluation of a first-time seizure. A finger-stick blood glucose level should be obtained in all these patients because both hypo- and hyperglycemia can present as seizures. Women and girls of childbearing age should be tested for human chorionic gonadotropin levels. An ECG should be performed to evaluate for an arrhythmia or

a cardiogenic cause of syncope. Serum chemistry tests, especially sodium levels, can be helpful. Liver function tests and blood alcohol levels are reasonable to obtain if alcohol use disorder is a suspected precipitant of the seizure event. Finally, if the history suggests an infectious process as the seizure's trigger, then chest imaging, a urinalysis, and a lumbar puncture may be necessary. Drug screens are unhelpful in evaluating for acute intoxications but can provide some context if substance withdrawal is suspected.[3]

Brain imaging should be obtained in all evaluations of a first-time seizure. In most emergency departments, the most available neuroimaging modality is CT. Although not always diagnostic, a CT scan has the benefit of being performed rapidly, and it can provide insight into a potential seizure trigger. Many patients eventually undergo an MRI study as part of their seizure workup, but it is unlikely to be completed during an emergency department visit if the patient has a normal neurologic examination and has returned to their baseline.[3]

An EEG is infrequently performed during the acute evaluation of a patient with a seizure who has returned to their clinical baseline, in part because of the test's limited availability. However, in a study that evaluated first-time seizure patients, EEG results were abnormal in approximately 25% of cases and were predictive of short-term seizure recurrence. The decision to obtain an EEG should be made in consultation with a neurologist or epilepsy specialist. Local resources, clinical management based on EEG findings, and the availability of close outpatient follow-up care may dictate whether an EEG is feasible.[3]

Risk Stratification

Risk stratification with seizure falls into two categories: risk of a significant underlying trigger for the seizure and risk of seizure recurrence. Patients at risk of significant underlying disease as a seizure trigger include those with a known acute precipitant, focal seizures, an immunocompromised state, a malignancy, substance use disorder, anticoagulant use, persistent neurologic symptoms, or a fever.[4] These patients warrant a more in-depth investigation related to their risk factors and may require more testing, as dictated by their history and physical examination.

The risk of seizure recurrence tends to be highest in patients with a structural brain abnormality, a nocturnal seizure, or an EEG abnormality. The literature suggests that if a patient is to have a hyperacute recurrence of a seizure, it will happen within a few hours of the presenting event. Patients with alcohol use disorder and alcohol withdrawal syndrome have the highest risk of early seizure recurrence — these patients account for nearly 50% of all recurrent seizures within a 24-hour period, with more than 85% of these recurrences occurring within 6 hours.[5]

Antiseizure medication should be considered for patients with a high risk of seizure recurrence. The choice of antiseizure medication is based on the etiology of the seizures, comorbidities, potential side effect and drug interaction profiles, pregnancy and lactation status, and ability for treatment continuation in the outpatient setting.

Discharge Recommendations

According to the ACEP 2014 policy, patients who return to their baseline after an unprovoked seizure without high-risk features can be safely discharged.[2] When discharging patients with seizures, however, both the community's resources and the patient's ability to adhere to the discharge plan must be considered. Guidance on safety precautions is important, including restrictions on driving, directions to never swim alone, and warnings to avoid dangerous activities like climbing until cleared by a physician. Accessible and timely outpatient follow-up care may require a multidisciplinary approach with social work, case management, pharmacy, neurology, and emergency department staff.

Pearls

- Benzodiazepine administration should not be delayed for intravenous access in cases of status epilepticus. Intramuscular midazolam (0.2 mg/kg, maximum of 10 mg) is an effective option while intravenous access is obtained.
- The appropriate loading dose of levetiracetam for status epilepticus is 60 mg/kg, with a maximum dose of 4.5 g.
- Consider reversible causes in refractory status epilepticus cases including hypoglycemia, hyponatremia, eclampsia, and pyridoxine deficiency.
- Nonconvulsive seizures should be considered in patients who fail to return to their baseline neurologic status, especially after prolonged or recurrent convulsive seizures.

CRITICAL DECISION

What is the first-line treatment for status epilepticus?

Status Epilepticus

Most isolated seizure episodes resolve spontaneously. However, ongoing seizures that last longer than 5 minutes will require abortive therapy because neuronal damage occurs within 30 minutes of prolonged seizure activity.[6] Status epilepticus is defined as seizures that last longer than 5 minutes or recurrent seizures without a return to the baseline neurologic state. The diagnosis of status epilepticus can describe convulsive seizure activity, although patients can also have ongoing or recurrent nonconvulsive electrographic seizure activity that requires an EEG for detection.

Resuscitation

Timely treatment of status epilepticus is important and begins with basic resuscitation concepts. Figure 1 offers a suggested timeline for the management of status epilepticus. Airway management in the actively seizing patient includes placing the patient in the lateral decubitus position if possible and suctioning secretions. Although antiseizure medications, especially benzodiazepines, are feared to contribute to respiratory depression, generalized convulsive seizure activity results in ineffective ventilation; therefore, withholding seizure treatment is unwarranted. A meta-analysis found that benzodiazepine administration in patients with status epilepticus did not increase the incidence of hypoventilation or the need for intubation.[7] Supporting the ventilatory status and oxygenation may be achieved with as little as supplemental oxygen and manual displacement of the tongue with a nasopharyngeal airway or jaw thrust. Care should be taken to avoid the extremes of hyper- or hypoxia and hyper- or hypocarbia because these conditions can exacerbate seizures.

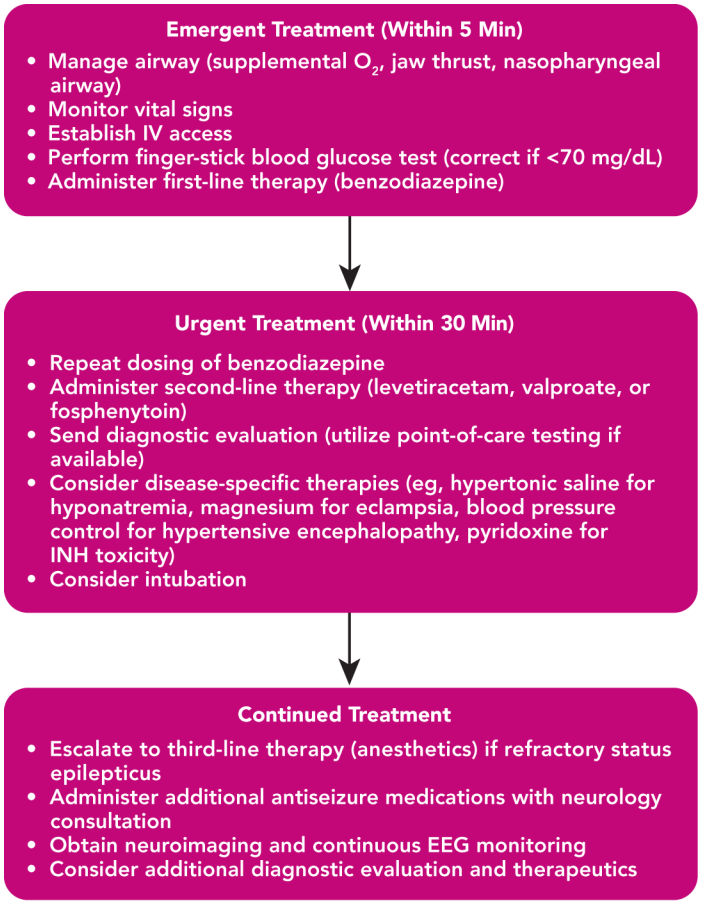

FIGURE 1. Treatment algorithm for status epilepticus. *Credit:* ACEP.

The seizing patient is often tachycardic and hypertensive due to a hyperadrenergic state. Persistent tachycardia and hypertension after apparent cessation of clinical seizure activity may reflect nonconvulsive seizure activity. Until the etiology of the seizure is known, rapidly lowering blood pressure should be avoided because lowering MAP can lower cerebral perfusion in acute brain injury with elevated intracranial pressure.

First-Line Therapy

Seizures that do not terminate within 5 minutes are unlikely to do so without pharmacologic intervention. First-line treatment of status epilepticus is benzodiazepines (*Table 1*). If intravenous access is unavailable, midazolam can be given intramuscularly with similar efficacy to intravenous lorazepam. Lorazepam has a slower onset of action when administered intramuscularly, making it less favorable for intramuscular administration. Diazepam should not be given intramuscularly, because of its erratic absorption; it should be administered only intravenously when lorazepam or midazolam cannot be given. Commercial forms of diazepam for rectal or intranasal administration also exist.[8] Benzodiazepines can be repeated for a second dose after 5 minutes if the seizure activity is ongoing. If intravenous access cannot be obtained, intraosseous access should be placed. Consider administering second-line agents in addition to the second dose of benzodiazepines.

The most common pitfall in the treatment of status epilepticus is underdosing benzodiazepines. Despite evidence-based guidelines, benzodiazepines are often given at smaller-than-recommended doses. Even when multiple doses are given, patients receive a cumulatively inadequate dose.[9]

	Medication	Loading Dose (by actual body weight)	Therapeutic Level	Considerations
First-Line Therapy (Can repeat in 5 min)	Lorazepam	0.1 mg/kg IV (max 4 mg)		
	Midazolam	0.2 mg/kg IM/IV (max 10 mg)		Preferred if no IV access
	Diazepam	0.2 mg/kg IV (max 10 mg)		Active metabolite with long half-life
Second-Line Therapy	Levetiracetam	60 mg/kg IV (max 4.5 g)	25-60 mg/mL	
	Valproate	40 mg/kg IV (max 3 g)	Total drug level: 80-140 mg/L Free drug level: 4-11 mg/L	Avoid in cases of hepatic impairment and in female patients of childbearing age
	Fosphenytoin	20 mg PE/kg IV (max 2,000 mg)	Total drug level: 15-25 µg/mL Free drug level: 1.5-2.5 µg/mL	Can be given IM if no IV access Preferred over phenytoin for fewer cardiac adverse effects
Third-Line Therapy (Patients should be intubated and placed on continuous EEG monitoring)	Propofol	1-2 mg/kg IV bolus, can repeat every 5 min to max 10 mg/kg 30-50 µg/kg/min IV infusion		Contraindications: · Soy and egg allergies · Hypertriglyceridemia · Propofol infusion syndrome
	Midazolam	0.2 mg/kg IV bolus, can repeat every 5 min to max 2 mg/kg 0.2-2.9 mg/kg/hr IV infusion		
	Ketamine	1.5 mg/kg IV bolus, can repeat every 5 min to max 4.5 mg/kg 0.3-7.5 mg/kg/hr IV infusion		Hypertension
Other Antiseizure Medications	Phenobarbital	15-20 mg/kg IV	20-50 mg/L	Avoid in advanced liver disease and profound shock
	Lacosamide	400 mg IV	5-10 mg/L	PR interval prolongation

TABLE 1. Antiseizure medications for status epilepticus

Underdosing can, in turn, lead to treatment resistance because of cellular and molecular changes associated with ongoing seizure activity.

CRITICAL DECISION

What is the second-line treatment for status epilepticus?

Many antiseizure medications are used in the management of epilepsy; however, many of them need to be initiated slowly to minimize adverse effects, making them imperfect agents in the treatment of status epilepticus. The Established Status Epilepticus Treatment Trial (ESETT) evaluated the use of levetiracetam, valproate, and fosphenytoin in patients with convulsive status epilepticus that is unresponsive to benzodiazepine treatment. The study found no significant difference between these second-line therapies in seizure cessation at 1 hour.[10] Adequate drug dosing was important in this trial; levetiracetam was administered at 60 mg/kg (max 4.5 g), valproate at 40 mg/kg (max 3 g), and fosphenytoin at 20 mg/kg (max 1.5 g). Based on this trial, use of levetiracetam in the acute setting has gained popularity because of its similar performance and safety to valproate and fosphenytoin, as well as its less severe side effects.

CRITICAL DECISION

How should refractory status epilepticus be managed?

Third-Line Therapy

Patients with continued or recurrent seizure activity despite appropriately dosed benzodiazepine and antiseizure medication are in refractory status epilepticus. Approximately half of the patients in ESETT still had evidence of clinically apparent seizures 1 hour after treatment with second-line therapy. Seizure recurrence was also seen in approximately 10% of ESETT patients in all treatment groups.[10] Given the risk of neuronal damage with continued seizure activity, treatment must be escalated in a timely manner.[11]

Management of refractory status epilepticus requires intubation to initiate third-line therapy with anesthetic agents. The choice of an induction agent should ideally be one with antiseizure properties to facilitate seizure control. Propofol is a reasonable option for induction because it is commonly used

for postintubation sedation. However, propofol does carry a risk of hypotension at the dose needed to suppress seizure activity; propofol-related infusion syndrome is also a concern when using the drug for a prolonged time. An alternative third-line therapy is benzodiazepines. Although not generally preferred as an induction or sedative agent, benzodiazepines do have a favorable hemodynamic profile relative to propofol when used as an infusion. If a benzodiazepine infusion is used for seizure control, midazolam is preferred because lorazepam and diazepam infusions are associated with the risk of propylene glycol toxicity. An emerging third-line therapy in the hemodynamically challenging patient is ketamine. As an *N*-methyl-d-aspartate (NMDA) and glutamate receptor antagonist, ketamine provides an alternative mechanism of action for seizure control.[12] Importantly, management of refractory status epilepticus with these anesthetic agents often requires much higher doses than commonly used for general sedation; thus, vasopressor support may also be needed (see *Table 1*).

Additional antiseizure medications are often needed to aid in seizure control and for ultimately weaning the patient off anesthetic agents. The choice of additional antiseizure medications is often based on their mechanisms of action and potential drug interactions and should be guided by consultation with a neurologist.

Other Considerations

Intubation in status epilepticus should be performed with a paralytic agent. Although the use of neuromuscular blockade can obscure the evaluation of convulsive seizure activity, reversal agents can be given if available. The risk of rhabdomyolysis-associated hyperkalemia from prolonged seizure activity may need to be taken into consideration when choosing a paralytic agent.

Diagnostic evaluation in status epilepticus should include serum drug levels of antiseizure medications because medication nonadherence is common. Many antiseizure medications are hepatically metabolized by cytochrome P450 (CYP450) isozymes and, thus, are susceptible to drug interactions with CYP450 inductors and inhibitors. However, serum drug level results are unlikely to arrive in time to direct treatment decisions in the emergency department.

Other common etiologies of status epilepticus are drug intoxication or withdrawal, meningoencephalitis, hypoxic-ischemic brain injury, structural brain lesions, and metabolic derangements. A subset of previously healthy patients can present with new-onset refractory status epilepticus without an apparent identifiable etiology. These patients often require extensive infectious, autoimmune, and paraneoplastic workup.

EEG

Patients who remain encephalopathic or have abnormal movements or behaviors that are concerning for subclinical seizures benefit from EEG evaluation for nonconvulsive seizures. Continuous video EEG is superior to routine 30-minute EEG in the detection of nonconvulsive seizures given the potential intermittent nature of these electrographic events. If continuous video EEG is unavailable, a routine EEG can still provide valuable information such as a potential seizure focus and can help with identifying patients who should undergo prolonged EEG monitoring.

New and emerging rapid- or limited-EEG technologies have become commercially available. These devices generally incorporate a reduced number of electrodes embedded in a headset or cap, which allows for application by clinicians other than trained EEG technicians. Some of the technology also includes an automated algorithm for seizure detection. These devices have increased the availability of EEG monitoring at hospitals without EEG technicians and epileptologists.

The DECIDE study showed that application of a limited EEG system took, on average, 5 minutes in an ICU setting.[13] This technology has also been shown to aid in the disposition and treatment decision of encephalopathic patients in the emergency department.[14] However, these rapid EEG systems are no substitute for traditional full-montage EEG or interpretation by an epileptologist.

✕ Pitfalls

- Failing to consider that the seizure is secondary to an underlying condition. Always search for precipitating factors or reversible causes.
- Delaying or withholding benzodiazepines for fear of respiratory depression. Untreated convulsive seizure activity has a higher risk of cardiopulmonary complications.
- Giving inadequate doses of benzodiazepines and antiseizure medications. Weight-based dosing per guideline recommendations is generally well tolerated.

CRITICAL DECISION

What reversible causes of seizures require disease-specific treatments?

Treatment for the underlying cause of a seizure is as important as treatment for the seizure. The classic reversible cause of seizures is hypoglycemia. All patients who present with a seizure should have a point-of-care glucose test, and dextrose-containing fluid should be given if the blood glucose level is less than 70 mg/dL. Other notable causes of seizures include hyponatremia, eclampsia, alcohol withdrawal, and isoniazid (INH) toxicity.

Hyponatremia

Hyponatremia is a common electrolyte disturbance seen in the emergency department. Its evaluation and management are critically linked to its etiology, severity, and rate of development. The risk of seizures increases with decreasing serum sodium levels, with most seizures occurring at serum sodium levels less than 120 mEq/L.[15] Seizing hyponatremic patients require prompt intervention. In addition to benzodiazepine administration, patients also need hypertonic saline for treatment of cerebral edema. The preferred treatment in this situation is 3% NaCl (2 mL/kg or ~150 mL). If 3% NaCl is unavailable, 75 mL of 8.4% sodium bicarbonate, which contains a similar sodium load, can also be used.[16] These treatments should elicit a 3 mEq/L rise in the serum sodium level. Repeat dosing can be given if the seizure continues or the serum sodium level fails to adequately increase. Point-of-care laboratory tests are helpful because the typical laboratory turnaround time can result in a significant delay in definitive therapy.

Hypertensive Encephalopathy, Eclampsia, and PRES

Hypertensive encephalopathy is a spectrum of conditions marked by severely elevated blood pressure and neurologic symptoms including headache, altered mental status, visual disturbances, and seizures. Syndromes like eclampsia and posterior reversible encephalopathy syndrome (PRES) share many clinical features. Common prevailing theories are that impaired cerebral autoregulation mechanisms and endothelial dysfunction result in blood brain barrier disruptions that lead to cerebral edema and intracranial hemorrhage.

Eclampsia is also a disease spectrum that spans from gestational hypertension to preeclampsia and eclampsia. Most cases of eclampsia are preceded by neurologic signs of headache, visual changes, photophobia, or mental status changes — although warning symptoms are sometimes absent. Half of eclampsia cases occur postpartum, and approximately 90% of these cases occur within 1 week of delivery.[17] Approximately 90% of patients with eclampsia have white matter changes on MRI that are similar to patients with PRES.[18] The Magpie study showed that magnesium therapy in preeclampsia reduced rates of eclampsia and maternal mortality.[19] A loading dose of magnesium 4 to 6 g over 20 minutes followed by an infusion of 1 to 2 g/hr leads to higher rates of seizure termination than benzodiazepines and phenytoin in cases of eclampsia. Obstetrics should be called early with these patients because an emergent delivery is needed in many of these cases.

PRES is a clinical and radiologic syndrome characterized by neurologic symptoms including headache, encephalopathy, and seizures, as well as subcortical white matter vasogenic edema. Although frequently associated with hypertension, PRES is also associated with renal failure, immunosuppressive medications, and autoimmune disorders.[20] Treatment of PRES includes controlling blood pressure and removing any precipitating factors. Blood pressure should be gradually reduced by no more than 20% within 1 to 2 hours to avoid the risk of cerebral, coronary, and renal ischemia. Titratable agents are preferred for smooth reduction in blood pressure and decreased blood pressure variability. If an acute ischemic stroke or intracranial hemorrhage is present, then disease-specific guidelines should be followed regarding blood pressure targets.

Alcohol Withdrawal

Chronic alcohol use modulates γ-aminobutyric acid (GABA) and glutamate activity, leading to suppression of GABA inhibitory activity and upregulation of excitatory glutamate activity that manifests as characteristic neurologic and autonomic symptoms of alcohol withdrawal. Alcohol withdrawal is typically seen in patients within 12 to 48 hours of cessation of or reduction in alcohol intake. A blood alcohol level does not rule alcohol withdrawal in or out, because withdrawal symptoms can occur at any level.[21] Approximately 8% of patients with alcohol withdrawal experience withdrawal seizures within 12 to 24 hours, with 50% of these patients progressing to delirium tremens.[22] The best management strategy is prevention of symptom progression with administration of GABA agonists.

Debate exists about whether a symptom-triggered or schedule-based approach is superior and whether treatment with benzodiazepines or barbiturates is superior. Phenobarbital's pharmacokinetic profile and its modulation of both glutamate and GABA pathways make it an enticing option for treatment

of both seizures and withdrawal, but physicians should be familiar with both medication classes because not all patients are suited for phenobarbital.

Benzodiazepines are preferred in cases of advanced cirrhosis or if an alternative diagnosis is suspected. If benzodiazepines are used for withdrawal seizures, then standard dosing for status epilepticus should be used and escalated as appropriate. Diazepam has active metabolites and may be superior to lorazepam. Conventional antiseizure medications may be ineffective, especially phenytoin, but can be considered until the clinical scenario is more apparent.

If a barbiturate strategy is desired, a 15- to 20-mg/kg loading dose can be used to terminate the seizures. A serum phenobarbital level can be obtained for monitoring the therapeutic window and guiding repeated administrations. Some studies have shown a link between phenobarbital use and reductions in benzodiazepine use, intubation, and respiratory failure that requires intubation in patients with alcohol withdrawal.[23] Refrain from combining phenobarbital with other sedative agents because of the extremely long half-life of barbiturates.

Importantly, patients in alcohol withdrawal can also have other concomitant etiologies for their seizures. Neuroimaging is advised in nearly all of these patients because the rate of coexisting trauma is high. EEG monitoring is recommended in patients with continued encephalopathy or unreliable examinations. Additionally, given the risk of thiamine deficiency with chronic alcohol use, empiric thiamine supplementation should be considered: 100 mg IV daily if the patient displays normal mentation or 500 mg IV up to three times daily if Wernicke encephalopathy is a concern.[21]

Pyridoxine Deficiency

A rare but reversible cause of refractory seizures is pyridoxine (vitamin B6) deficiency. This deficiency as a cause occurs most classically in the setting of INH toxicity but has also been postulated in long-standing alcohol use disorder. Pyridoxine deficiency leads to decreased synthesis of GABA neurotransmitters, which increases susceptibility to seizures that can be refractory to benzodiazepines. INH, a tuberculosis treatment, is often the culprit: It directly inactivates pyridoxine. A patient who has taken INH and presents with new-onset refractory seizures should be given pyridoxine as a 1 g to 1 g—equivalent dosing. Higher doses may be needed for pediatric accidental ingestions or intentional overdoses. If the amount ingested is unknown, then 5 g can be given empirically.[24] A common laboratory finding is elevated liver enzymes; this finding can also occur in acetaminophen toxicity, a diagnosis which should be co-investigated to ensure timely treatment.

Summary

Emergency physicians play a pivotal role in the management of seizures, ranging from workup and anticipatory guidance for first-time seizures to timely interventions for status epilepticus. They also watch for reversible conditions associated with seizures that require specific treatments, especially in cases of refractory seizures. Benzodiazepines remain the mainstay of initial therapy, and appropriate dosing is of the utmost importance. Levetiracetam, valproate, or fosphenytoin can be used as second-line antiseizure medications.

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CASE RESOLUTIONS

CASE ONE

The patient returned to her normal mental status. Her blood pressure remained normal, and her subsequent neurologic examination revealed no focal findings. She stated that she had a strong family history of seizures but had never had a seizure before. Laboratory studies including serum electrolyte and human chorionic gonadotropin levels were unremarkable. She was discharged with neurology and primary care follow-up appointments in 1 week to expedite further evaluation with EEG and MRI. Anticipatory guidance, including driving precautions, was provided.

CASE TWO

The patient presented in status epilepticus, and the seizure failed to terminate with administration of intramuscular midazolam. Supplemental oxygen was applied, intravenous access was obtained, and a dose of lorazepam was given. A brief chart review revealed a history of seizures and nonadherence to levetiracetam. EMS also reported that the patient was found at the bottom of the staircase. He was loaded with levetiracetam 4.5 g and was intubated after being given ketamine and rocuronium. He underwent a head CT scan, which showed a traumatic subdural hematoma. The EEG showed no further seizure activity. The patient underwent craniotomy for subdural hematoma evacuation and recovered well in the ICU. He was extubated on hospital day 3 and was transferred to a subacute rehabilitation center.

CASE THREE

The patient presented with a known reversible cause of his seizure, acute hyponatremia. He was given 150 mL of 3% saline, and his seizure terminated. Repeated laboratory assessment showed an improvement in serum sodium level to 114 mEq/L, along with an elevated serum creatinine level. Careful evaluation of the patient's medical record showed that he was recently started on a thiazide diuretic. Bedside ultrasound revealed no hydronephrosis or urinary retention. He was admitted to the ICU for close monitoring of his neurologic status and electrolyte derangements.

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Weakness and Syncope

By Peter Vandersteenhoven, MD, MS; and William Brady, MD, FACEP
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Objectives

On completion of this article, you should be able to:

- Discuss the clinical significance of second-degree AV block type II and second-degree AV block type II with high-grade AV block in terms of diagnostic and management considerations.
- Identify the clinical risk associated with various ECG presentations of second-degree AV block type II, both with and without high-grade AV block.

CASE PRESENTATION

A 59-year-old man presents after a syncopal event. His history includes past myocardial infarction, diabetes mellitus, and hypertension. At the time of his syncopal event, he also experienced significant weakness and dizziness. He denies chest pain, dyspnea, and other complaints. He is ill appearing, with vital signs that include BP 85/63 and P 50. Lead II ECG rhythm strip is shown (*Figure 1*).

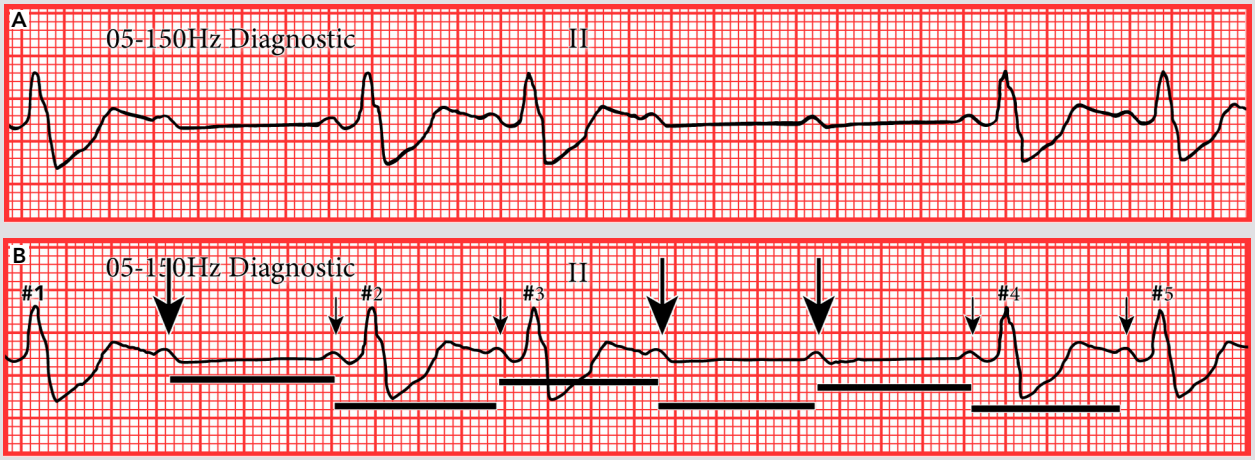

FIGURE 1. Lead II ECG rhythm strip. (*A*) Second-degree AV block type II with high-grade AV block. (*B*) Annotated rhythm strip showing second-degree AV block type II with high-grade AV block.

ECG Findings With Discussion

The patient's ECG demonstrated an irregular bradycardic rhythm with a wide QRS complex (0.12 sec); the average rate was approximately 50 bpm. P waves were noted and appeared to be associated with some of the QRS complexes (*large arrows*) with consistent, normal PR intervals (approximately 0.18 sec); other P waves (*small arrows*) were not associated with a QRS complex. The P-P intervals were consistent across the rhythm strip (*horizontal lines*), regardless of the presence of a QRS complex. The rhythm was considered a second-degree atrioventricular (AV) block type II with high-grade AV block. The first three beats (#1-#3) demonstrate a typical pattern of second-degree AV block type II. The next two beats — unaccompanied, consecutive P waves (*small arrows*) after these beats — do not demonstrate conduction to the ventricle; thus, no QRS complexes are noted, which illustrates the high-grade nature of this AV block.

AV block presents in four distinct patterns, including first-degree, second-degree, and third-degree AV block. Second-degree AV block is further subdivided into type I and type II. *Second-degree AV block type II* is diagnosed when the PR interval is fixed and unchanging without progressive lengthening, with eventual appearance of a P wave, and without a resultant QRS complex — the so-called *dropped* or *nonconducted* beat (*Figure 2*). This pattern can then repeat itself, either in a regular or irregular pattern. Second-degree AV block type II can also present with either a narrow or wide QRS complex and with more than one consecutive nonconducted P wave, a feature that is termed high-grade AV block.

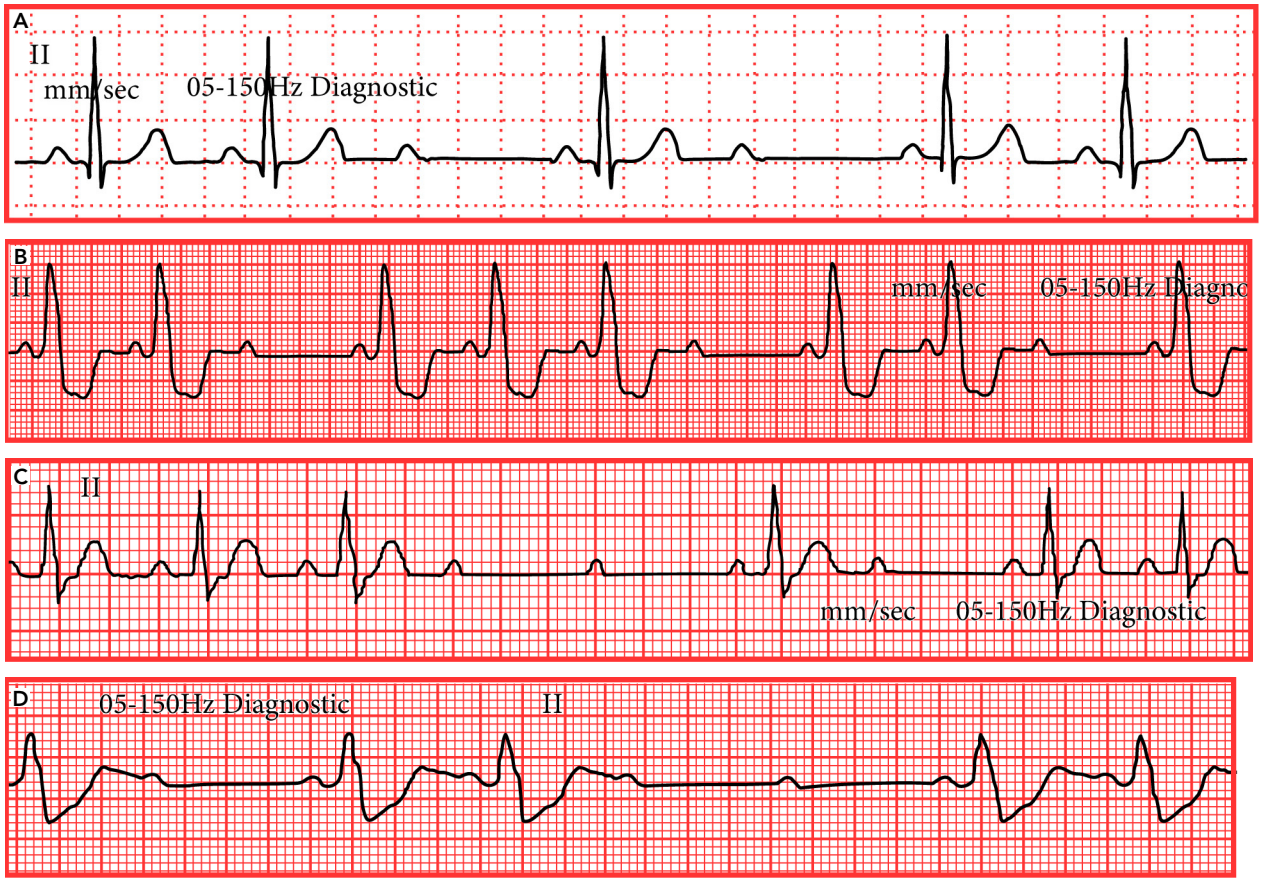

FIGURE 2. Second-degree AV block type II with (*A*) a narrow QRS complex, (*B*) a wide QRS complex, (*C*) a narrow QRS complex and consecutive nonconducted P waves, consistent with second-degree AV block type II with high-grade AV block, and (*D*) a wide QRS complex and consecutive nonconducted P waves, consistent with second-degree AV block type II with high-grade AV block.

In general, *second-degree AV block type II* is a high-risk form of AV block that is associated with significant cardiovascular risk. This block is the least common form of AV block but is the most frequent conduction abnormality that results in death. Patients who present with this form of conduction abnormality are acutely ill and have a considerable risk of further decompensation, often progressing to having a complete heart block and, frequently, cardiac arrest and death. Although all forms of second-degree AV block type II are associated with significant risk, the four presentations of this conduction abnormality in order of increasing degree of cardiovascular jeopardy are:

- Second-degree AV block type II with a narrow QRS complex (see *Figure 2A*);
- Second-degree AV block type II with a wide QRS complex (see *Figure 2B*);
- Second-degree AV block type II with a narrow QRS complex and high-grade AV block (see *Figure 2C*); and
- Second-degree AV block type II with a wide QRS complex and high-grade AV block (see *Figure 2D*).

The most frequent cause of second-degree AV block type II is extensive anterior wall myocardial infarction. Less common causes include infiltrative, fibrotic, or sclerotic cardiac disorders (eg, lymphoma, other cardiac tumors, amyloidosis, collagen vascular diseases).

LEARNING POINTS

- In general, second-degree AV block type II is a high-risk form of AV block that is associated with significant cardiovascular risk. It is the least common form of AV block but is the most frequent conduction abnormality that results in death.
- There are four distinct ECG presentations of second-degree AV block type II. They are (from least to most risky):
  - Second-degree AV block type II with a narrow QRS complex (see *Figure 2A*);
  - Second-degree AV block type II with a wide QRS complex (see *Figure 2B*);
  - Second-degree AV block type II with a narrow QRS complex and high-grade AV block (see *Figure 2C*); and
  - Second-degree AV block type II with a wide QRS complex and high-grade AV block (see *Figure 2D*).

Rapid evaluation and management are crucial in these presentations. Hemodynamic stabilization is achieved with intravenous fluid boluses, vasopressor infusion, and extrinsic pacing (either transcutaneous or transvenous pacing). Rapidly correctable causes of second-degree AV block type II should be tested for and, if appropriate, treated. Acute coronary syndrome, usually large myocardial infarction, is frequently involved and should be treated in standard fashion to restore conduction and an adequate heart rate. Many patients ultimately require placement of a permanent right ventricular pacemaker after stabilization in the ICU.

CASE RESOLUTION

The patient's blood pressure declined further as his lethargy progressed. Transcutaneous pacer pads were placed on him and activated with appropriate electrical and mechanical capture. The patient improved: His blood pressure increased to 100 mm Hg systolic, and his mental status normalized. Basic laboratory studies were normal, including serum potassium levels; high-sensitivity troponin levels were elevated above the 99th percentile, ultimately with up-trending values. The patient was found to have an extensive non–ST-elevation myocardial infarction with pronounced anterior wall hypokinesis. A permanent right ventricular pacemaker was inserted on hospital day 4. The patient was discharged on hospital day 6 and was well at his 6-month follow-up visit.

ADDITIONAL READING

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A Limping Child With Fever

By Stephen Sandelich, MD; and Grace Hwang, MPH
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Reviewed by Ann M. Dietrich, MD, FAAP, FACEP

Objective

On completion of this article, you should be able to:

- Discuss evaluation and treatment for multifocal osteomyelitis in pediatric patients.

CASE PRESENTATION

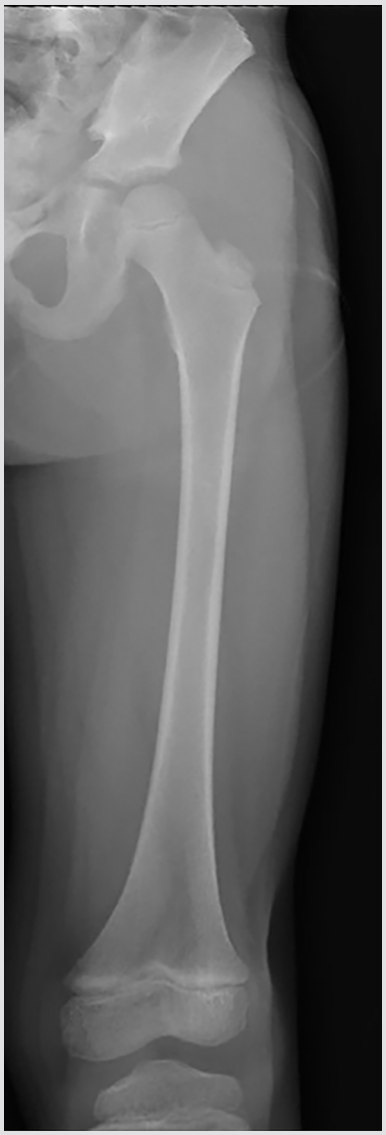

FIGURE 1. Anteroposterior x-ray of the left femur. No cortical erosion and no acute fractures or dislocations are present. Soft tissues are unremarkable. No knee effusion and no radiographic abnormalities of the left femur are noted.

A 4-year-old boy with a history of sickle cell trait presents with chief complaints of fever and limping. His mother reports that the patient has had intermittent hip pain for the past 6 days, which appears to be worsening. He has also had an intermittent fever, with a maximum temperature of 38.7°C (101.7°F). Prior to this presentation, the patient was seen at an outside hospital, where x-rays of his hip were reportedly normal. At that time, he had elevated inflammatory markers: a C-reactive protein level of 57 mg/L (the upper limit of normal is 10 mg/L) and an erythrocyte sedimentation rate (ESR) of 43 mm/hr. His WBC count was normal, and a respiratory viral panel was negative. He was discharged home with instructions to follow up with his pediatrician. Because the patient's pain and fever continued, his parents became more concerned and brought him to the emergency department.

On arrival, he is afebrile and has normal vital signs. On physical examination, he is generally well and nontoxic appearing. His musculoskeletal examination is significant for difficulty with ambulation and pain over the left hip, without overt swelling, erythema, or fluid collection. He has elevated inflammatory markers, including a C-reactive protein level of 57 mg/L, ESR of 43 mm/hr, and procalcitonin level of 0.13 ng/mL. His WBC count is 9.7×10^9/L. X-rays of his pelvis and femur are negative for avascular necrosis, fractures, and slipped capital femoral epiphysis (*Figure 1*). Lyme serology is also negative.

Because of his significant symptoms but negative x-rays, an MRI is performed. The MRI study shows left abdominal adductor muscle myositis and multifocal osteomyelitis without evidence of subperiosteal collections (*Figure 2*). No septic arthritis or joint involvement is noted. Orthopedic surgery is consulted; however, there are no collections for them to drain.

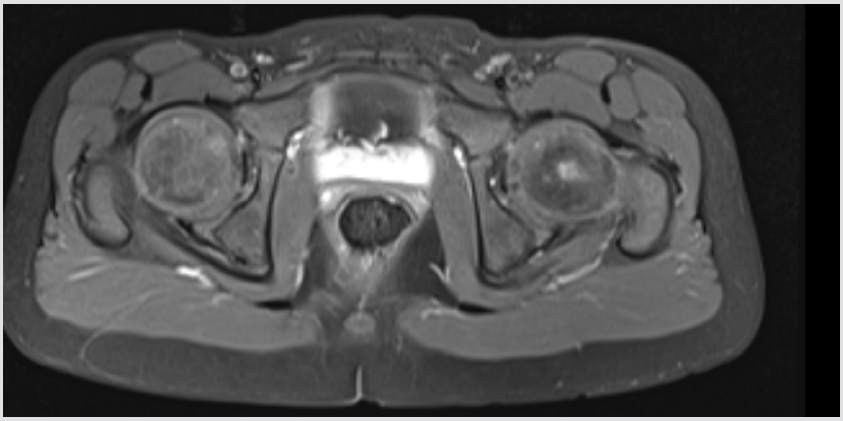

FIGURE 2. MRI study of the pelvis, axial T1 post contrast with fat saturation. Focal area of a decreased T1 signal with enhancement in the posterior aspect of the left lateral femoral condyle is shown.

Discussion

The differential diagnosis for a child with a limp depends significantly on the presence or absence of fever. In febrile patients with localized bone pain and systemic signs of infection, osteomyelitis is a key consideration because of its risk of sepsis, morbidity, and limb deformity.[1] Infectious etiologies such as septic arthritis, pyomyositis, and reactive arthritis must also be prioritized. Other conditions to consider are inflammatory conditions like transient synovitis — which can mimic septic arthritis but, instead, often follows a viral illness — and systemic illnesses like juvenile idiopathic arthritis.[2] In afebrile patients, noninfectious causes predominate, including trauma (eg, occult fractures or ligament injuries), orthopedic conditions such as slipped capital femoral epiphysis and Legg-Calvé-Perthes disease, and other pathologies like avascular necrosis and benign bone tumors (eg, osteoid osteoma).[1] A careful history and physical examination, inflammatory markers, and imaging are essential in distinguishing between these conditions, identifying cases of osteomyelitis, and providing timely management.

Pediatric osteomyelitis is commonly caused by bacterial pathogens that invade the bone through hematogenous spread, direct inoculation, or contiguous extension from adjacent tissues.[3] The most frequent causative organism is *Staphylococcus aureus* (including methicillin-resistant strains), particularly in community-acquired infections. Other pathogens include *Kingella kingae* (in children younger than 5 years, especially in cases associated with upper respiratory tract infections), *Streptococcus pyogenes*, and *Streptococcus pneumoniae*. Gram-negative bacteria such as *Pseudomonas aeruginosa* may be involved in osteomyelitis secondary to penetrating trauma. In neonates, *Escherichia coli* and group B streptococci are additional considerations.[4] The virulence factors of these pathogens, including biofilm formation and immune evasion mechanisms, complicate treatment and contribute to the chronicity and recurrence of the infection.[3,4]

No standard diagnostic criteria exist for osteomyelitis; however, because of the risk of sepsis, morbidity, and limb deformity associated with the condition, treatment is initiated for patients of high clinical concern. Although bone biopsy is the gold standard diagnostic test, it is rarely done. More commonly, diagnosis is made based on a constellation of clinical, laboratory, and imaging findings. Clinically, children with osteomyelitis present with localized bone pain, tenderness, swelling, and erythema; reduced limb use; and systemic symptoms like fever.[3] Laboratory tests typically reveal elevated inflammatory markers, including C-reactive protein and ESR. Leukocytosis may be variable. Blood cultures are positive in only 30% to 50% of cases, but they can be used to guide targeted antibiotic therapy.[5]

Many imaging modalities are used to characterize osteomyelitis. Initially, plain x-rays are used to rule out fractures or other bone pathologies. However, early osteomyelitis changes may not be apparent on an x-ray. By contrast, MRI is the most sensitive and specific imaging modality for detecting bone inflammation, abscess formation, and associated soft tissue involvement.[6] When multifocal osteomyelitis is suspected, positron emission tomography–CT (PET-CT) is an efficient but sparingly used method to scan the entire body.[7] If MRI or PET-CT is not readily available, ultrasound in conjunction with C-reactive protein levels and a WBC count on admission can be used to diagnose multifocal osteomyelitis. Ultrasound has a reported 74% to 76% sensitivity and a 63% specificity for detecting fluid collections and subperiosteal abscesses.[8,9] If noninvasive diagnostics are inconclusive, bone biopsy or aspiration can confirm the diagnosis and identify the causative pathogen, particularly for chronic or culture-negative cases.

Multifocal osteomyelitis and chronic recurrent multifocal osteomyelitis (CRMO) are distinct conditions in the pediatric population that share overlapping features but differ in their etiology, pathophysiology, and management.[10] Multifocal osteomyelitis typically refers to an infectious process caused by hematogenous spread of pathogens like *S. aureus*. It often presents acutely with systemic signs of infection (eg, fever, leukocytosis, elevated inflammatory markers) and radiographic evidence of multiple sites of bone involvement. By contrast, CRMO is an autoinflammatory disorder characterized by noninfectious, recurrent episodes of bone inflammation that often involve the metaphyses of long bones, vertebrae, and clavicles. Unlike multifocal osteomyelitis, CRMO patients often have mild or absent systemic symptoms and sterile cultures or biopsies. MRI can identify multifocal lesions in both conditions, but lesions in CRMO may persist on imaging despite clinical resolution.[3,10] As such, correctly identifying patients with CRMO is imperative to prevent unnecessary radiation exposure, bone biopsies, and prolonged antibiotic courses.[10]

The treatment of osteomyelitis in the pediatric population involves prompt initiation of antimicrobial therapy tailored to the suspected or confirmed pathogen. Empiric treatment typically targets *S. aureus*, including methicillin-resistant strains, by using agents such as vancomycin or clindamycin.[3] Targeted antimicrobial therapy can be guided by blood cultures, bone biopsy, or molecular diagnostics. Initial therapy is often administered intravenously for 1 to 2 weeks, followed by an oral antibiotic course for 4 to 6 weeks to ensure complete resolution.[4] Surgical intervention, such as debridement or drainage,

may be necessary in cases of abscess formation, subperiosteal collections, or failure of medical management.[3] Close monitoring of clinical response, inflammatory markers, and imaging findings is essential to guide therapy duration and ensure treatment success. Multidisciplinary care, including care from infectious disease and orthopedic specialists, can optimize outcomes.

CASE RESOLUTION

The patient was admitted to the inpatient pediatric service for management of multifocal osteomyelitis. He was treated with empiric intravenous cefazolin and ibuprofen (for pain control). His pain improved, and his fever resolved — his C-reactive protein level trended downward to 2 mg/L. Blood cultures showed no growth by day 5. He was discharged after 3 days with a 4-week course of enteral cephalexin and close outpatient follow-up care with a pediatric infectious disease specialist. At his follow-up visit, he had clinical improvement, including improvement with pain and no further episodes of fever.

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Balloon Tamponade for Unstable GI Bleeds

By Daniel Ruiz-Betancourt, MD; and Laura Welsh, MD
Harvard Affiliated Emergency Medicine Residency Program

Reviewed by Andrew J. Eyre, MD, MS-HPEd, FACEP

Objective

On completion of this article, you should be able to:

- Explain when and how to use balloon tamponade for upper GI bleeds.

Bridwell RE, Long B, Ramzy M, et al. Balloon tamponade for the management of gastrointestinal bleeding. *J Emerg Med.* 2022 Apr;62(4):545-548.

KEY POINTS

- Balloon tamponade devices have demonstrated a success rate of upward of 90% in securing hemostasis in patients with hemodynamically unstable GI bleeding.
- Balloon tamponade is indicated in hemodynamically unstable upper GI bleeding when there is a delay in endoscopy, an inability to perform endoscopy, a failed endoscopy, or a need for stabilization prior to facility transfer.
- Intubation and resuscitation should be performed *prior* to the use of balloon tamponade devices.

GI varices are among the most severe complications of liver cirrhosis. The prevalence of esophageal varices in patients with cirrhosis ranges from 40% to 95%, and approximately one-third of these patients experience a variceal bleed. Variceal bleeding is a life-threatening emergency that can rapidly result in hemodynamically unstable hemorrhage and airway compromise. Each episode of bleeding can have a mortality rate up to 30%. As such, it is critical that emergency physicians learn how to optimally manage patients who present with variceal bleeding. In general, the medical management of variceal bleeding includes airway protection, blood product transfusion, and administration of antibiotics and vasoactive medications. Balloon tamponade is reserved for unstable patients with refractory bleeding despite maximal medical therapy, and it functions as a bridge to definitive management, such as variceal band ligation, sclerotherapy, or a transjugular intrahepatic portosystemic shunt procedure.

Balloon Tamponade Devices

Balloon tamponade devices function by exerting direct pressure to stop varices from bleeding. These devices have demonstrated variable efficacy, ranging from 30% to 90% in controlling severe upper GI bleeding. The three major balloon tamponade devices are the Linton-Nachlas tube (LNT), Sengstaken-Blakemore tube (SBT), and Minnesota tube (MT). The LNT has a single 600-mL gastric balloon and three ports, including a balloon inflation port and two separate gastric and esophageal suction ports. The SBT and MT have two balloons, one gastric and one esophageal. The SBT differs from the MT in that it has a 250-mL gastric balloon and only three ports, including a single gastric suction port and two separate gastric and esophageal balloon inflation ports. The MT has a 500-mL gastric balloon and four ports: two separate gastric and esophageal balloon inflation ports and two separate gastric and esophageal suction ports. Data are insufficient to suggest head-to-head superiority between any of the balloon tamponade devices.

Indications, Contraindications, and Complications

Balloon tamponade devices should not be used in all patients with upper GI bleeding. Balloon tamponade is indicated for *hemodynamically unstable* upper GI bleeding when there is a delay in endoscopy, an inability to perform endoscopy, a failed endoscopy, or a need for stabilization prior to facility transfer. The two major contraindications are placement in hemodynamically stable patients and placement in patients who are not intubated. Relative contraindications include esophageal strictures and prior esophageal or gastric surgeries. Twenty percent of patients may experience complications

from balloon tamponade. Complications include aspiration, airway obstruction, esophageal perforation, and mucosal ulceration. Furthermore, variceal rebleeding is seen in approximately 50% of patients after balloon tamponade devices are removed.

Tube Placement

Prior to placing the tube, ensure that the patient is intubated and resuscitated. Next, ensure that all equipment is available, present at the bedside, and checked for leaks. Place the patient in supine at 45°. The estimated length of the tube is measured from the bridge of the nose to the xiphoid process. The balloon is then lubricated and inserted through the oral cavity and, subsequently, into the esophageal cavity using Magill forceps, being careful not to damage the balloons. The manometer or syringe is attached to the gastric balloon port, and the gastric balloon is partially inflated with 50 mL of air; a chest x-ray is performed to ensure intragastric balloon positioning. Once its position is confirmed, the gastric balloon should be fully inflated. While inflating the gastric balloon, ensure that pressure does not exceed 15 mm Hg (pressure that exceeds 15 mm Hg suggests esophageal placement).

Once the gastric balloon is fully inflated, tie one end of rolled gauze to the external end of the device and tie the other end of the rolled gauze to a 1 L bag of intravenous fluids. Hang the bag of fluids over an IV pole to generate 1 to 2 lb of constant traction. Aspiration and irrigation of the gastric and esophageal aspiration ports should be performed. If hemostasis is not achieved and an SBT or MT is being used, the manometer should be placed on the esophageal balloon port, and the esophageal balloon should be inflated to reach 30 to 40 mm Hg. A repeat chest x-ray should be obtained to ensure final placement. After successful placement, continuous suction can be used through the aspiration ports for the first 12 hours. If a balloon tamponade device is needed for longer than 24 hours, the esophageal portion of the device, if present, should be deflated every 6 hours for a few minutes to limit mucosal ulceration.

Critical Decisions in Emergency Medicine's LLSA literature reviews feature articles from ABEM's 2025 Lifelong Learning and Self-Assessment Reading List. Available online at acep.org/moc/llsa and on the ABEM website.

Suprapubic Aspiration

By Steven J. Warrington, MD, PhD
MercyOne Siouxland, Sioux City, Iowa

Objective

On completion of this article, you should be able to:

- Use suprapubic aspiration to collect a urine sample when necessary.

Introduction

Suprapubic aspiration is not a commonly used procedure but may be an option for obtaining a urine sample in difficult situations, such as the presence of phimosis, adhesions, or strictures. Additionally, the use of point-of-care ultrasound can provide physicians unfamiliar with suprapubic aspiration a degree of comfort and added safety when performing the procedure.

Contraindications

- Infection at the planned puncture site

Benefits and Risks

Suprapubic aspiration's primary benefit is that it allows for collection of a urine sample so that the sample can be tested for infection. The procedure is of particular benefit when a catheter cannot be passed to collect a sample, such as in patients with phimosis or labial adhesions. The risks of suprapubic aspiration include failure of the procedure, introduction of infection, bleeding-related complications, allergic reactions, and injury of nearby structures.

TECHNIQUE

1. **Obtain** consent from the patient or caregiver. Provide an anxiolytic or analgesic at this time, if needed.
2. **Obtain** the necessary equipment and have another person restrain the patient, if necessary (eg, if the patient is a young child).
3. **Place** the patient supine and in a frog-legged position and then identify the bladder. Use point-of-care ultrasound for targeting the entry and aspiration site, if needed.
4. **Prepare** the site and equipment for a sterile procedure.
5. **Place** local anesthetic at the intended aspiration site.
6. **Insert** and advance the needle toward the bladder while continuously aspirating the syringe. Point-of-care ultrasound can also be used during this part of the procedure to allow for direct visualization.

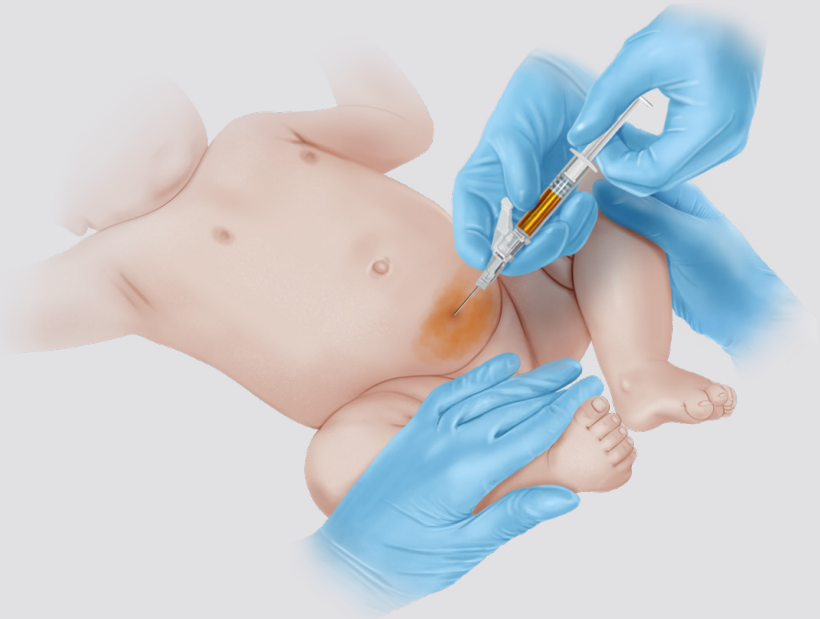

FIGURE 1. Suprapubic aspiration allows for the collection of a urine sample when collection via catheterization is not possible. *Credit:* ACEP.

7. **Attempt** to collect urine from an alternative entry point or angle or with a new set of equipment if no urine was obtained on the first attempt.
8. **Remove** the needle after urine is collected. Dress the site, if needed, and ensure appropriate handling of the specimen to send for testing.

Alternatives

Multiple alternatives exist for urine sample collection. Appropriate methods will depend on clinical needs and the patient's age, anatomy, and ability to tolerate the procedure. Many adults and adolescents can provide a urine sample on their own; alternative methods like suprapubic aspiration are mainly needed for young pediatric patients. Transurethral catheterization generally allows for collection of a urine sample in young patients and is less invasive than suprapubic aspiration. In patients with difficult anatomy that prevents the use of transurethral catheterization, placement of a suprapubic catheter may be an alternative to suprapubic aspiration.

Special Considerations

The use of point-of-care ultrasound during suprapubic aspiration has been suggested for potentially increasing safety and accuracy. Although research is limited, ultrasound appears to increase success with obtaining a urine sample. Furthermore, ultrasound can increase physicians' confidence when performing suprapubic aspiration.

Scaphoid Fractures

By Diego Riveros, MD, CAQSM; and Dana Huong Tran, BS

University of South Florida Morsani College of Medicine, Tampa and Loyola Stritch School of Medicine, Maywood, Illinois

Objectives

On completion of this article, you should be able to:

- Recognize clinical and imaging presentations of scaphoid fractures.
- Discuss how to manage scaphoid fractures to avoid nonunion.

CASE PRESENTATION

A 20-year-old man presents with left wrist pain. He is a college football lineman. The pain started as sharp wrist pain 3 weeks ago after he struck down a football. About 1 year ago, he had a fracture to his left wrist. He did not completely adhere to bracing, tried playing through the injury, and has had worsening pain, with difficulty fully extending his left wrist. He has no other new injuries.

On examination, the patient has tenderness to palpation at the anatomical snuffbox. He demonstrates active wrist extension but is unable to flex past midline. He also has full active range of motion in all digits; normal radial and ulnar deviation at the wrist; normal wrist flexion; and preserved motor function of the anterior interosseous nerve, posterior interosseous nerve, and ulnar nerve. His wrist is fully neurovascularly intact. X-rays are taken and reveal a nonunion of his scaphoid fracture (*Figure 1*).

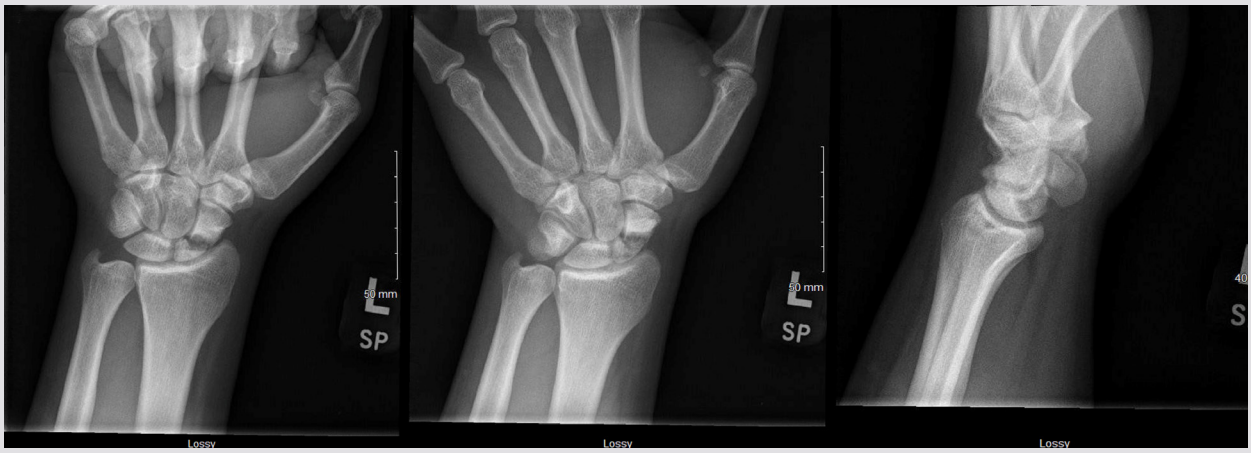

FIGURE 1. X-ray of the patient's wrist. *Credit:* Dana Huong Tran, BS.

Discussion

The scaphoid bone plays a crucial role in wrist stability and function. Scaphoid fractures account for 60% to 90% of all carpal fractures.[1] One of eight carpal bones in the wrist, the scaphoid bone is located on the radial side of the wrist and bridges the proximal and distal carpal rows. The bone has poor vascular supply — its main blood supply from the dorsal carpal branch of the radial artery is retrograde, meaning blood flows from distal to proximal. This vascular supply can lead to challenges with healing when the scaphoid bone is injured. Prompt diagnosis and management are needed to prevent long-term complications.

The most common mechanism for a scaphoid fracture is a fall on an outstretched hand. Other mechanisms include direct blows to the wrist from sport or motor vehicle collisions. Patients with scaphoid injuries often present with radial-sided wrist pain, tenderness in the anatomical snuffbox, pain on axial compression of the thumb, or tenderness over the scaphoid tubercle. Initial imaging includes standard x-ray views of the wrist. An additional posterior-anterior view with the wrist in ulnar deviation should be considered for suspected scaphoid fractures. Importantly, negative initial x-rays do not rule out the possibility of a scaphoid fracture, which can be difficult to visualize on initial x-rays. CT or MRI can be considered for further imaging, although empiric immobilization in a thumb spica splint or cast is the typical initial management for highly suspected scaphoid injuries with negative initial x-rays.[2,3] Empiric

immobilization mitigates the risk of complications, such as further displacement of the fracture, and avoids additional costs of advanced imaging in the emergency department. Repeat x-rays after 10 to 14 days of immobilization are advised. Most scaphoid fractures are more evident after this waiting period because bone resorption at the edges of the fracture lead to greater radiographic lucency. Nonadherence to immobilization can lead to fracture displacement and nonunion.[4] Confirmed stable fractures are often managed nonoperatively with immobilization for 6 to 12 weeks, while displaced or unstable fractures are managed through surgical fixation.[2]

Nonunion of scaphoid fractures is a significant concern and occurs in approximately 10% of scaphoid fracture cases when an occult fracture leads to a delayed diagnosis or inadequate immobilization.[5] Nonunion is a condition in which the fractured scaphoid fails to heal properly, and it can lead to scaphoid nonunion advanced collapse (SNAC), a progressive degenerative condition associated with wrist pain, decreased range of motion, and functional impairment. Open reduction and internal fixation (ORIF) is often required in cases of established nonunion to prevent SNAC. To prevent progression of scaphoid fractures to nonunion, physicians must maintain a high level of suspicion in patients with clinical signs of scaphoid fracture despite negative x-rays and must appropriately immobilize these injuries.

CASE RESOLUTION

Because of poor treatment adherence, the patient experienced a nonunion of his scaphoid fracture. A thumb spica was placed during his visit. The patient followed up with a hand surgery specialist and had an ORIF 1 month later. Postoperatively, he developed a pulmonary embolism and was placed on rivaroxaban. He continued to recover from the procedure.

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A Stubborn Urinary Tract Infection

By Joshua S. Broder, MD, FACEP

Dr. Broder is a professor and the residency program director in the Department of Emergency Medicine at Duke University Medical Center in Durham, North Carolina.

Objectives

On completion of this article, you should be able to:

- Explain a role of imaging for recurrent UTI.
- Name some risk factors for colovesical fistula formation.
- Select CT protocols optimized for detection of colovesical fistula.

CASE PRESENTATION

A 64-year-old man with Crohn disease and chronic obstructive pulmonary disease (COPD) presents with abdominal pain. He has had multiple recent emergency department visits during which he complained of dysuria and was prescribed antibiotics for UTI. He is currently homeless and has lost 36 kg (80 lb) in the past 5 months, during which time he has had intermittent hospitalizations for COPD exacerbations, falls, and surgical repair of a hip fracture. Currently, he denies fever or blood in his stool. In the past week when he urinates, he has noted air and what he describes as diarrhea from his penis.

His vital signs are BP 98/77, P 90, R 22, and T 36.5°C (97.7°F); SpO_2 is 97% on room air. He weighs 43 kg (94 lb) and has a body mass index of 14 kg/m^2. The patient is alert but cachectic. He is tachypneic and has wheezing on auscultation. His cardiac examination is normal. He has a scaphoid abdomen with diffuse tenderness (*Figure 1*). His genital examination is unremarkable.

Review of the patient's record shows numerous urinalyses over the past 2 months, each with more than 100 WBC/high-power field. His blood test reveals a WBC count of 13 × 10^9/L. His renal function is normal, and his albumin level is 2 g/dL. An abdominal-pelvic CT scan with intravenous contrast is obtained (*Figure 2*).

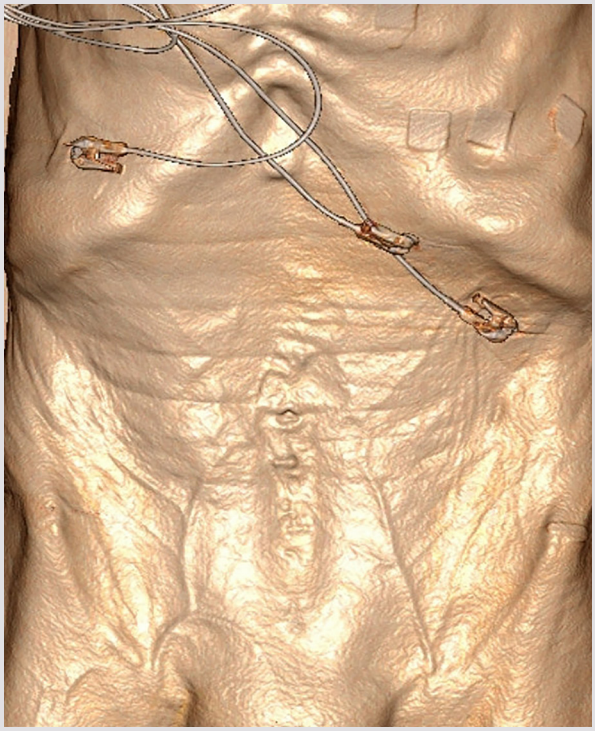

FIGURE 1. A skin surface reconstruction demonstrates the patient's severe cachexia.

Discussion

The most common cause of colovesical fistula in one series was diverticular disease, accounting for over 70% of cases.[1] Trauma, radiation therapy, and iatrogenic (operative) injuries are other causes. Crohn disease is a risk factor for fistula formation to abdominal and pelvic organs and the skin; poor nutritional status is another risk factor.[2] Additionally, even in the presence of risk factors like Crohn disease, malignancy should be suspected in patients with fistulae because 11% to 20% of colovesical and colovaginal fistulae are malignant in etiology.[1,3] Recurrent or refractory urinary tract infections (UTIs) occur in almost 50% of patients with colovesical fistulae. Pneumaturia and fecaluria are considered pathognomonic.[1]

The American College of Radiology (ACR) Appropriateness Criteria for anorectal disease designates CT of the pelvis with intravenous contrast as "usually appropriate" for a suspected rectovesical fistula.[3] For suspected Crohn disease exacerbation, the ACR finds CT of the abdomen and pelvis with intravenous contrast "usually appropriate."[4] The ACR specifies that intravenous contrast is particularly helpful in characterizing fluid collections, abscess, and fistulae, and comments that CT without intravenous contrast is not useful.[3] Also, a noncontrast CT scan before administration of intravenous contrast is generally unnecessary for a suspected fistula.[3] The ACR recommends use of water-soluble contrast in either the bowel or bladder to opacify a fistula tract. The GI system and bladder should *not* both be intentionally filled with contrast: The point of intraluminal contrast is to identify when contrast inappropriately

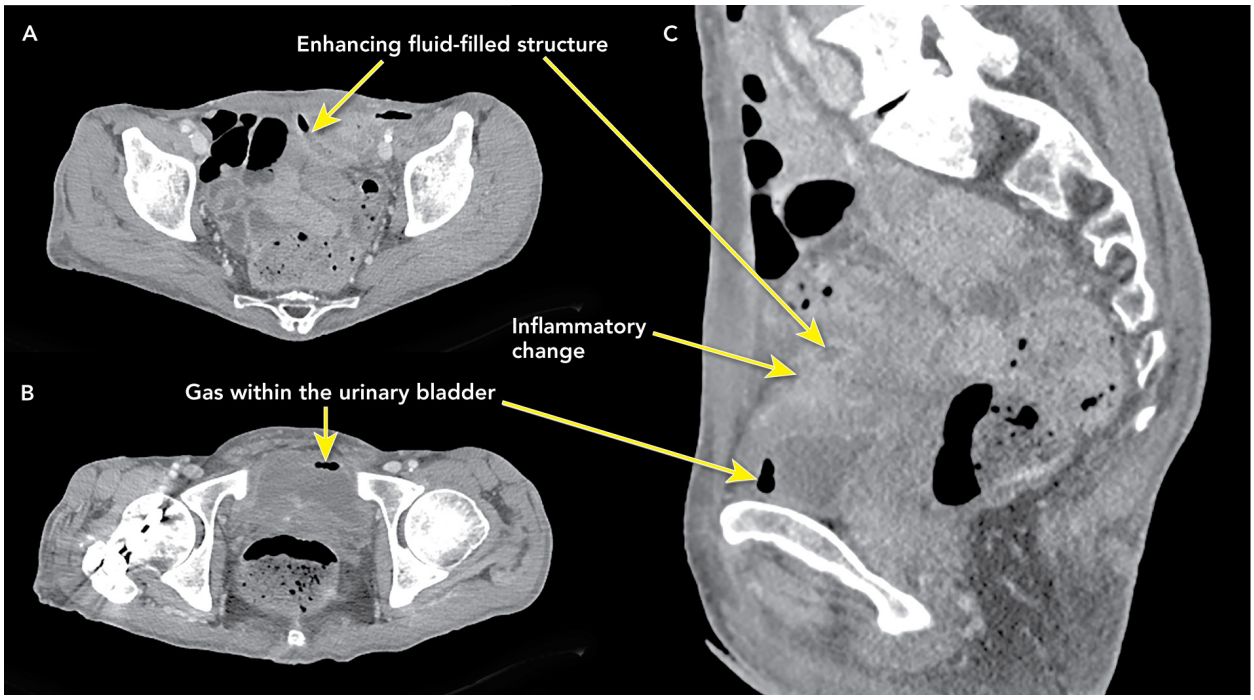

FIGURE 2. CT scan with intravenous contrast. (*A*) An enhancing fluid-filled structure is seen in this axial CT slice just cephalad to the bladder. Although a definite connection to the bladder is not identified, this structure and the intravesicular air seen in image B imply the presence of a colovesical fistula. (*B*) Gas is seen within the urinary bladder, consistent with a fistula to the gas-filled colon. The patient has no history of urinary catheterization, which could also have been a source of abnormal air within the bladder. (*C*) In this single sagittal image, gas is seen within the urinary bladder, consistent with a fistula to the gas-filled colon. An enhancing, fluid-filled structure lies cephalad to the bladder, with adjacent inflammatory change. These findings strongly suggest a fistula that connects the bladder and colon. This patient is so cachectic that he has a paucity of intraperitoneal fat; intraperitoneal fat is usually a useful CT "contrast agent" because it separates organs and can demonstrate inflammatory fat stranding.

transits from one system to the other to detect a pathological fistula. The bladder can be opacified either retrograde (ie, instilling contrast into the bladder through a urinary catheter) or anterograde (ie, administering intravenous contrast and then performing delayed CT imaging, after contrast has been excreted by the kidneys).[3] CT's sensitivity for colovaginal and colovesical fistula has been reported to be 76.5%, but the authors of the study did not report details on the route of contrast administration or the CT protocol.[1] If neither enteric nor bladder contrast is provided (as in the case presented), other clues such as air within the bladder or inflammatory changes of the bladder wall or adjacent bowel may suggest fistula.

For a persistent fistula between the nonsterile GI tract and bladder, UTI and colonization cannot be overcome with antibiotics. The condition requires surgical therapy for cure, and the specific procedure required depends on the etiology (eg, malignancy versus benign diverticular disease) and other patient-specific factors.

CASE RESOLUTION

The patient was admitted and evaluated for surgical repair. His nutritional status prevented immediate operative therapy. Urine cultures grew *Klebsiella pneumonia* and *Enterococcus faecium*. He was treated with intravenous antibiotics as a temporizing measure.

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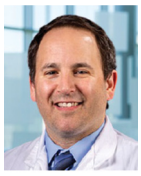
Feature Editor: Joshua S. Broder, MD, FACEP. See also *Diagnostic Imaging for the Emergency Physician* (Winner of the 2011 Prose Award in Clinical Medicine, the American Publishers Award for Professional and Scholarly Excellence) and *Critical Images in Emergency Medicine* by Dr. Broder.

Sticks and Stones

Pediatric Growth Plate Fractures

LESSON 4


By Bhakti Sanghani, DO; Joshua Kern, MD; and Prayag Mehta, MD

Dr. Sanghani is a resident and Dr. Kern and Dr. Mehta are assistant professors of emergency medicine at the University of Texas Southwestern Medical Center in Dallas.

Reviewed by Erika B. Crawford, MD, FAAP, FACEP

Objectives

On completion of this lesson, you should be able to:

1. Describe the anatomy and physiology of the growth plate.
2. Classify growth plate fractures using Salter-Harris classification.
3. Identify the imaging modalities needed to properly diagnose growth plate fractures.
4. Discuss treatment options in the emergency department for different types of growth plate fractures.
5. Recognize which growth plate fractures require orthopedic consultation in the emergency department.

From the EM Model

18.0 Traumatic Disorders
- 18.1 Trauma
  - 18.1.12 Pediatric Fractures
    - 18.1.12.1 Epiphyseal
      - 18.1.12.1.1 Salter-Harris Classification

CRITICAL DECISIONS

- What are growth plates and how do they normally develop?
- What are the different types of growth plate fractures?
- What is the best imaging modality to assess for growth plate fractures?
- How are growth plate fractures managed?
- What are the discharge instructions for patients with growth plate fractures?

Growth plate fractures account for a significant portion of pediatric fractures and, in some cases, can lead to bone growth arrest. Emergency physicians must understand how growth plate fractures are classified and their effects on patient morbidity to provide appropriate care and reduce the risk of complications associated with these common fractures.

CASE PRESENTATIONS

CASE ONE

A 10-year-old boy arrives with his anxious parents after he fell off his bicycle onto his right side 30 minutes ago. He is right-hand dominant. On arrival, he is crying but consolable and is holding onto his right wrist while not letting anyone touch his arm. On visual inspection, the patient has a slight deformity of his right distal forearm. Once access is established in his left arm, intravenous pain medications are given to allow for a more detailed physical examination.

The patient has mild edema and point tenderness over his right lateral wrist, with a dorsal deformity of his right distal radius. He has soft forearm compartments, a 2+ radial pulse, a normal capillary refill, and intact sensation over his right arm, wrist, and hand. He can flex and extend his elbow and wiggle the fingers of his right hand. He can supinate and pronate his hand but has significant pain with range of motion. X-rays are ordered.

CASE TWO

A 7-year-old girl presents with right ankle pain after she jumped in the air and landed incorrectly on her right ankle during ballet class 1 hour ago. She is brought in by her dad, who is carrying her because she refuses to walk or be put down due to pain. After intravenous access for pain medication is established, the patient is more agreeable to an examination from her dad's lap. She resists plantar flexion due to pain but can invert, evert, and dorsiflex her right foot. Some ecchymosis and edema are present along the posterior aspect of her ankle. Her calf is soft, and she has 2+ posterior tibial and dorsalis pedis pulses with intact sensation over her foot and ankle. Plain x-rays of her tibia, fibula, ankle, and foot are taken, including anterior-posterior, oblique, and lateral views of the right ankle.

CASE THREE

A 14-year-old boy presents with left ankle pain after a skiing accident 30 minutes prior to arrival. He fell roughly 5 feet down a snow ledge and landed on his left side. He did not pass out or hit his head but was unable to stand or ambulate afterward. When he landed, he heard a snap and felt immediate pain in his left ankle. Park rangers had to evacuate him. En route, EMS gave him 30 µg of intranasal fentanyl, which mildly relieved his pain.

On examination, his airway, breathing, and circulation are intact. On secondary survey, he has intact skin and no obvious deformity. Erythema and mild nonpitting edema are noted around his left ankle, with point tenderness to palpation over the medial malleolus. His posterior tibial and dorsalis pedis pulses are 2+ and symmetric bilaterally. Sensation is intact over the left foot, ankle, and leg. X-rays are taken.

Introduction

Growth plate fractures, also known as physeal fractures, are high-risk pediatric fractures because they occur in open growth plates and can lead to physeal arrest. Growth plate fractures account for 15% to 18% of all pediatric fractures, and most emergency physicians encounter these fractures at some point in their career.[1,2] A methodical approach helps in evaluating and treating these patients. Salter-Harris classification is used to describe the type of growth plate fracture, and fractures that are more likely to lead to growth abnormalities can be identified based on specific features. Some growth plate fractures can be managed in the emergency department, while others require consultation with an orthopedic surgeon.

CRITICAL DECISION

What are growth plates and how do they normally develop?

Long bones in humans include the femur, radius, ulna, humerus, tibia, fibula, and metacarpal bones. In children, immature long bones have two growth plates (one at each end) that contribute to

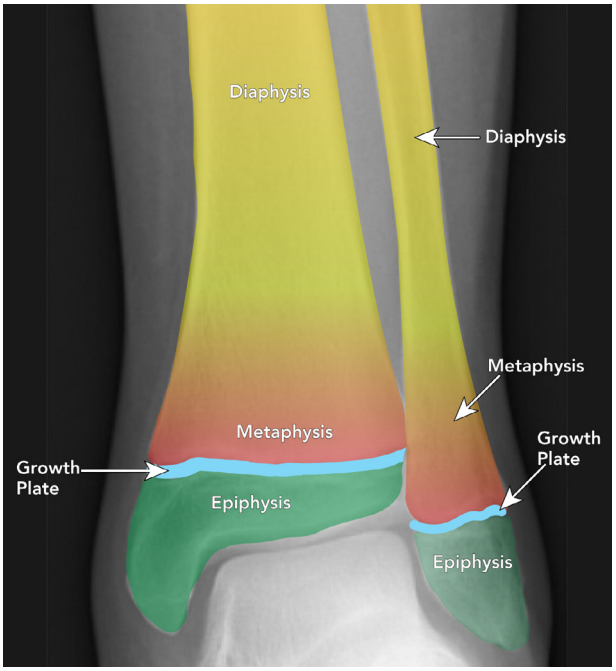


FIGURE 1. The areas of the tibia and fibula. The physeal plates in these long bones are depicted as blue lines that divide the epiphysis and metaphysis. *Credit:* ACEP.

longitudinal growth. The areas of a long bone include the epiphysis, physis, metaphysis, and diaphysis. The epiphysis is the end of the long bone that lies near the joint. The physis is the growth plate and lies next to the epiphysis. The metaphysis is between the physis and the shaft known as the diaphysis (*Figure 1*). Growth plates border both sides of the diaphysis.

During childhood, growth plates serve as the original template for long bone growth and its final shape. As children get older, growth plates gradually become less wide. Growth plates are made up of cartilage produced by cells called chondrocytes; these cells go through five phases of maturation that allow long bones to grow and lengthen. When these cells die, growth plates close and cartilage is replaced by mature bone in the process of endochondral ossification. An epiphyseal scar is left behind by the process. Growth plate closure signifies the end of long bone growth and occurs during adolescence.[3]

The process of endochondral ossification is coordinated by chondrocytes and many highly complex regulatory signaling pathways. Chondrocytes progress through zones of the growth plate during their maturation: the reserve, proliferative, hypertrophic, and ossification zones (*Figure 2*). The reserve zone includes a colloid matrix that is supplied by blood vessels and houses a germinal layer of cells that differentiate into chondrocytes in a resting or mitotically inactive state. When chondrocytes enter the proliferative zone, they divide by mitosis and organize into columns of cells within a proteoglycan matrix.

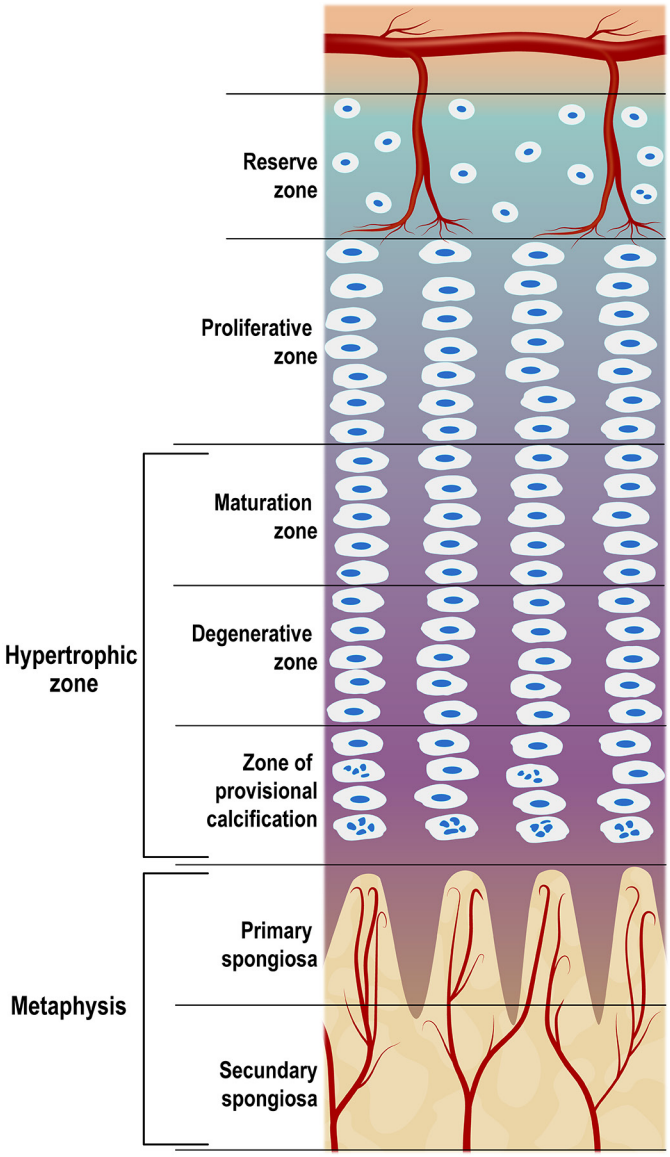


FIGURE 2. Zones of the growth plate. *Credit:* Tareq Al-Salamah, MBBS, MPH, FACEP; Yasser AlKhalife, MD; and Malak N. AlShebel.

In the hypertrophic zone, chondrocytes begin to terminally differentiate and increase in size; here, they produce collagen and incorporate lipids, glycogen, and alkaline phosphatase. The terminal phase of chondrocyte maturation occurs within the hypertrophic zone in the zone of provisional calcification, which is the last layer of the growth plate before the metaphysis. In the provisional calcification zone, the colloid matrix becomes calcified and chondrocytes die to get ready for osteogenesis. The calcified matrix is then invaded by blood vessels and replaced by osteocytes and a bony matrix in the ossification zone.[4]

Because the growth plate is composed of cartilage, it is a weak point in pediatric bone. The weakest part of the growth plate, and where most fractures occur, is the zone of provisional calcification; it is weaker than its surrounding osseous ligamentous structures because it serves as a transition point between calcified and noncalcified extracellular matrix proteins. After the growth plate closes, fracture risk decreases and injuries typically manifest as sprained ligaments instead.[5]

Blood supply to the physis is primarily from the epiphysis and, to a lesser extent, the metaphysis and perichondrial ring. Epiphyseal circulation supplies the physeal germinal and proliferative layers and is essential to healing physeal injuries. When the blood supply becomes compromised in fractures that separate the epiphysis from the physis, the risk of delayed wound healing and complications increases. Metaphyseal circulation supplies the zone of endochondral ossification through branches of the nutrient artery and supplies the periphery of the physis through branches of the perichondrial ring of LaCroix. The hypertrophic zone remains relatively avascular.

Skeletal growth typically occurs until age 14 years in girls and 16 years in boys. Distal tibia and fibula physeal plates fuse by 12 to 16 years in girls and 14 to 19 years in boys (or men).[6] Distal radius physeal plates fuse by 18 years in women and 19 years in men.[7] Growth plate closure is driven by estrogen produced during

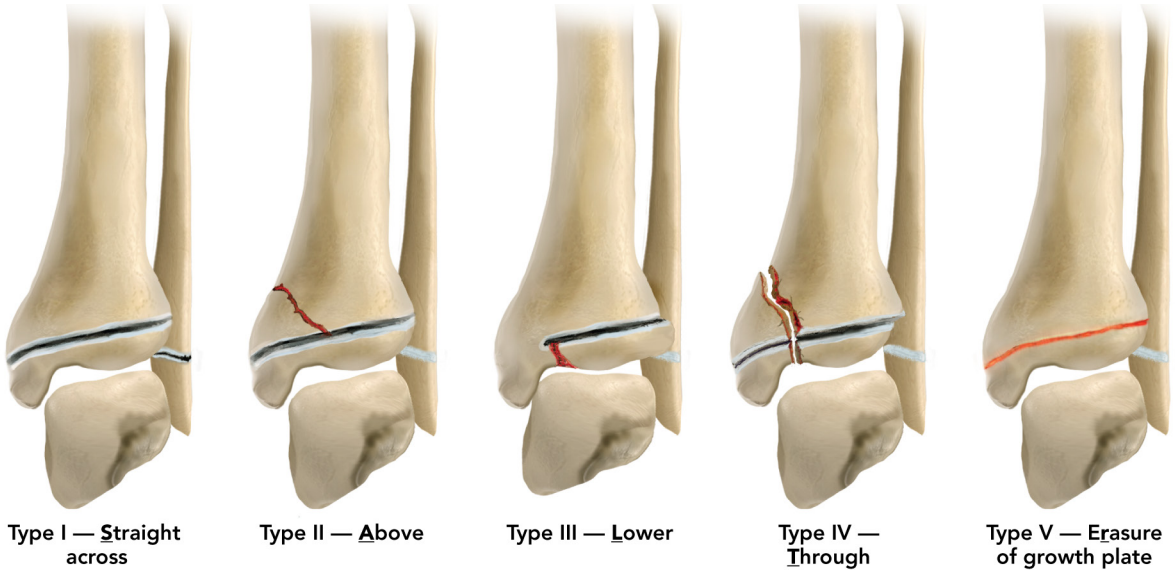


FIGURE 3. Salter-Harris classification of growth plate fractures. The acronym SALTR can be used to memorize the location of the five types of growth plate fractures. *Credit:* ACEP.

puberty. Estrogen increases the rate of chondrocyte transformation from the resting to the proliferative state, and higher levels of the hormone in girls contribute to their earlier physeal plate closure.

The physiology behind the final stage of growth plate closure is a point of scientific controversy. Theories include apoptosis, autophagy, hypoxia, or chondrocyte differentiation into osteoblasts. Growth plate closure occurs incrementally, usually with a transitional period of incomplete closure when the physis can be injured. Closure follows a distinct pattern, starting with the central portion of the growth plate followed by the anteromedial portions and, lastly, the anterolateral portion. A visible scar is left once the growth plate fuses.[4] Knowledge of bone development and vascular supply can help emergency physicians understand specific growth plate fracture healing patterns and morbidity.

CRITICAL DECISION

What are the different types of growth plate fractures?

In 1963, two Canadian orthopedic surgeons, Salter and Harris, developed a classification system to differentiate pediatric growth plate fractures. More than 50 years later, the Salter-Harris classification system is still the predominant method surgeons and physicians use to classify these fractures. The system describes five types of growth plate fractures, which are based on the injury's location (*Figure 3*). The acronym *SALTR* can be used to memorize the location of growth plate fractures.[8]

Type I or Straight Across (S)

Type I fractures occur through the hypertrophic zone due to a longitudinal force that causes separation of the epiphysis and physis from the metaphysis and causes a fracture line through the growth plate. Diagnosis is largely clinical, with patients exhibiting point tenderness over the physis and soft tissue swelling around the bone involved. If clinical examination is consistent with a type I fracture, even if x-rays are normal, physicians should treat these patients with appropriate immobilization. These fractures generally have a good prognosis and do not typically require surgery.[9]

An example of a Salter-Harris type I fracture is the slipped capital femoral epiphysis (SCFE), which occurs when the epiphysis of the proximal femur slips posteriorly and medially relative to the physis (*Figure 4*). The Klein line can be used to compare the positioning of both structures. Drawn parallel to the lateral aspect of the femoral neck,

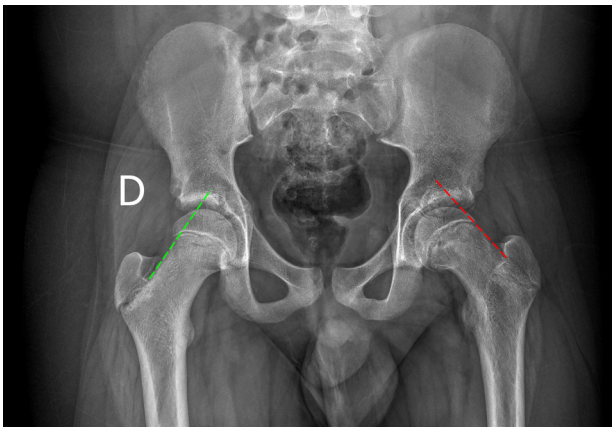


FIGURE 4. SCFE, a Salter-Harris type I fracture. The Klein line is abnormal on the left (*red*) femoral neck because it barely intersects the capital epiphysis. *Credit:* Copyright 2025 Dr. Leonardo Lustosa. Image courtesy of Dr. Leonardo Lustosa and Radiopaedia.org, rID: 160105. Used under license.

the Klein line normally intersects the lateral capital epiphysis on x-ray. In SCFE, however, the Klein line will barely, if at all, intersect the femoral head. SCFE is commonly seen in preadolescents or adolescents who present with a limp and atraumatic leg or hip pain. The most significant risk factor for SCFE is obesity, but periods of rapid growth and prior radiation can also increase the risk.[10]

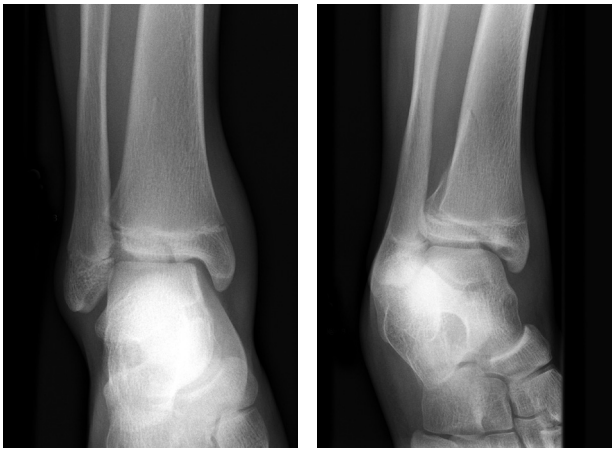


FIGURE 5. Triplane fracture. The fracture's configuration is an oblique fracture through the metaphysis, a horizontal fracture through the physis, and a vertical fracture through the epiphysis. *Credit:* Copyright 2025 Dr. Frank Gaillard. Image courtesy of Dr. Frank Gaillard and Radiopaedia.org, rID: 10438. Used under license.

Type II or Above (A)

Type II physeal fractures occur through the physis and metaphysis, with a fragment of the metaphysis still attached to the epiphysis. Commonly called the Thurstan Holland sign, the metaphyseal fragment is triangular. Treatment of type II fractures is usually closed reduction or casting. When these fractures are placed in a cast or splint, the prognosis is typically excellent.[9] Distal radius fractures are the most common type II physeal fractures followed by ankle fractures. Type II fractures of the distal tibia growth plate are high-risk fractures: They are at risk of premature physeal closure and angular deformity. Usually, these distal tibia growth plate fractures are treated with closed reduction, but sometimes, they are managed in the operating room with open reduction and internal fixation (ORIF). Recent literature found no significant difference in complications between these two treatment modalities.[11]

Type III or Lower (L)

Type III fractures begin intra-articularly and travel through the epiphysis into the physis. Proper anatomic reduction is of the utmost importance in minimizing post-traumatic arthritis and growth abnormalities of intra-articular fractures. An example of a type III Salter-Harris fracture is the Tillaux fracture, an intra-articular fracture of the anterolateral distal tibia epiphysis that occurs in adolescents during asymmetric physeal closure of the distal tibia growth plate. This fracture is typically caused by supination with an external rotation force or medial rotation of the leg on a fixed foot.[12] Anterior-posterior, lateral, and mortise views during radiographic assessment can identify the fracture; however, CT is more sensitive in evaluating degrees of displacement to determine proper treatment. Orthopedics must be consulted when managing these fractures. Fractures displaced less than 2 mm are managed with closed reduction and immobilization, while fractures displaced more than 2 mm are managed with closed reduction and percutaneous pinning (CRPP) or ORIF.[11] With proper reduction, the prognosis for these fractures is excellent.

Type IV or Through (T)

Type IV fractures begin intra-articularly and travel through the epiphysis, physis, and metaphysis. Because they are also intra-articular, proper reduction is paramount. These fractures are typically unstable and require surgical fixation to maintain reduction. They are also associated with growth disturbance that leads to morbidity.[4]

Triplane fractures — a complex, traumatic distal tibia fracture that occurs in children with partially fused growth plates — are a Salter-Harris type IV fracture (*Figure 5*). They usually occur from a force of supination and external rotation. The epiphysis is fractured through the sagittal plane, the metaphysis through the coronal plane, and the physis through the axial plane. Anterior-posterior and lateral plain x-rays must be taken to visualize triplane fractures. Closed reduction or surgery may be used to treat them depending on the degree of displacement and articular step-off.[13]

Type V or Erasure of Growth Plate (R)

Type V fractures are the rarest type of physeal plate fracture. They occur due to axial loading injuries, in which the physis is crushed by a force transmitted through the epiphysis and physis. Effects include disruption of the germinal matrix of the reserve zone of the physis and its vascular supply, which leads to bone growth arrest, limb length discrepancy, and functional impairment. Type V fractures often occur in joints that move in one plane, like the knee or ankle joint.[3] Their diagnosis is often made retrospectively: They can be difficult to diagnose because displacement of the epiphysis is unusual and initial x-rays are unremarkable. These fractures have a poor prognosis and lead to premature growth cessation.[9]

CRITICAL DECISION

What is the best imaging modality to assess for growth plate fractures?

Plain x-rays are usually sufficient for diagnosing growth plate fractures. X-rays should include at least two orthogonal views of the fracture site to discern displacement and injury pattern. Some fractures may not be easily detected on anterior-posterior view, so oblique and lateral views are recommended in some cases. Imaging the joints above and below the suspected area of injury is recommended to avoid missing any fractures, especially because pediatric patients may not be able to verbalize or localize their pain. For example, imaging the forearm can miss subtle distal radius physeal fractures that are more likely to be seen on wrist x-rays. Many orthopedic surgeons image the contralateral unaffected limb to compare baseline alignment, but this practice is not considered the standard of care and does not need to be routinely done in the emergency department.

Stress views are performed by applying pressure on a joint, such as weight-bearing or valgus or varus stress, to help determine the extent of injury. Although they are beneficial for specific patients and injuries, stress views for pediatric patients in the emergency department are not recommended because they can cause iatrogenic injury of the physis by displacing the fracture. Importantly, for Salter-Harris types I and V fractures, plain x-rays are often unremarkable if there is no significant displacement. In these cases, repeat imaging is critical, and management should be conservative with immobilization and close follow-up care. Type V fractures that are unremarkable on plain x-rays are often visualized on MRI.[3]

Obtaining advanced imaging for evaluation of pediatric growth plate fractures in the emergency department is usually unnecessary. Advanced imaging is sometimes ordered by orthopedic specialists and occasionally by emergency physicians if a fracture to a weight-bearing bone is highly suspected after negative plain x-rays. In the outpatient setting, CT scans are used to delineate fracture lines for preoperative planning of intra-articular physeal fractures. MRI studies are used to diagnose occult physeal fractures, chondral and osteochondral injuries, and ligamental injuries. The 2D assessment of complex injury patterns and intra-articular fracture geometry is useful for operative and nonoperative treatment planning. Arthrograms are used intraoperatively to outline the articular surface, evaluate for gaps or step-offs, and plan for percutaneous fixation.[14,15]

CRITICAL DECISION

How are growth plate fractures managed?

Many factors affect how growth plate fractures are managed, including patient age, suspected fracture type, fracture location, neurovascular status, joint stability, and whether the skin overlying the fracture is open or closed. Unstable fractures may need surgery to properly immobilize and reduce the segments. Fortunately, pediatric bones have a thicker periosteum than adult bones, which allows for better bone formation and faster healing.[16]

When patients with growth plate injuries present to the emergency department, consultation with orthopedic surgery, if available, is beneficial. These specialists can help with accurate diagnosis by recommending the appropriate imaging tests, which can also reduce imaging overutilization. Additionally, these specialists can help with identifying appropriate interventions and, potentially, providing intra-operative management, if needed, or outpatient surgical planning. Patients can also get established

✔ Pearls

- Pediatric growth plate fractures are painful. They should be treated with adequate analgesia in the emergency department, and a multimodal pain plan should be discussed with patients and caregivers at discharge.
- Salter-Harris types I and V fractures can be difficult to visualize on imaging. Maintain a high index of suspicion when x-rays are normal but signs and symptoms are consistent with one of these fractures. In cases of uncertainty, err on the side of caution and immobilize these patients. Also, ensure that they have adequate follow-up plans with orthopedic surgery for repeat imaging.
- When imaging an injured region, visualize the area in at least two different orthogonal planes, usually anterior-posterior and lateral views. In some cases, oblique views will need to be obtained. Additionally, visualizing the joints above and below the level of injury is necessary so that other injuries are not missed.
- When immobilizing a fracture, document its neurovascular status before and after immobilization. At discharge, discuss return precautions that could indicate neurovascular compromise, including any paresthesia, pain, or skin color changes.

with these orthopedic specialists for follow-up care and long-term management in the outpatient setting. With the widespread accessibility of electronic imaging and medical records within hospital systems, the practice of providing extensive details of fractures to consultants is largely outdated. However, emergency physicians should discuss the pertinent physical examination findings and any concerns for patient safety with consultants. Fracture descriptions to include during consultation include:

- Name of the injured bone;
- Injury location (eg, metaphysis, diaphysis, epiphysis, dorsal, volar);
- Fracture orientation (eg, transverse, oblique, spiral);
- Broken or intact skin overlying the fracture;
- Presence of any injury-related neurovascular compromise; and
- Fracture angulation, comminution, and displacement, if present.

Most orthopedic surgery colleagues evaluate their own x-ray images; however, emergency physicians should read the x-rays they ordered in the emergency department and discuss findings with these surgical colleagues as well.[17]

Growth plate fractures are incredibly painful. Studies have shown that pain is significantly undertreated for pediatric musculoskeletal presentations in the emergency department, including at times of extremity manipulation during the physical examination and before reduction and immobilization of fractures.[18] Emergency physicians should address these patients' pain with generous and appropriate analgesia and sedation. No one medication is more optimal than others. Ibuprofen, acetaminophen, and codeine are oral medications that can be used; however, they have a slower time of onset and may need to be combined with faster-acting medications for multimodal analgesia. Opioid medications for pediatric patients (if age appropriate) include intranasal fentanyl and intravenous morphine; intranasal ketamine is also an option. For reduction and immobilization of fractures, procedural sedation with ketamine or propofol (with continuous monitoring) can be used.

Displaced fractures should be reduced before being immobilized. The neurovascular status of the affected area should be documented both before and after immobilization. If neurovascular compromise has occurred, surgical intervention should be expedited to prevent acute complications like limb loss. Open reduction is preferred over forced reduction, instrumentation of the physis for manipulating fracture fragments, and multiple attempts at closed reduction; these less preferred methods can lead to iatrogenic physeal injury. Salter-Harris types I and II fractures are extra-articular physeal fractures. Attempts at closed reduction for these fractures should not be delayed more than 5 days from onset — delay could lead to iatrogenic physeal injury and growth arrest. Growth arrest is significantly more difficult to manage than malunion. Displaced Salter-Harris types III and IV fractures need to be reduced and stabilized by internal fixation regardless of their time of presentation. A closed, arthroscopic, or open approach may be done by orthopedic surgery.[15,16]

Closed reduction and casting of the affected bone provides the basis for fracture healing. Stable fractures that are at low risk of further displacement are immobilized sufficiently with splinting. Unstable fractures are at higher risk of displacement and usually require surgical intervention. Salter-Harris types I and II fractures are more likely to be managed nonoperatively with splinting, while types III and IV are more likely to require surgical fixation. The anatomic location, degree of displacement, and risk factors must all be considered when deciding between operative and nonoperative treatment. Lower-extremity casting compared to upper-extremity casting requires longer periods of immobilization to maximize stability and strength.[8] Repeated or forceful closed reduction can increase iatrogenic physeal damage. A physeal gap of up to 3 mm is tolerated, and alignment of the fracture fragments is more acceptable than anatomic reduction. If fracture fragments cannot be aligned, open reduction should be performed by an orthopedic surgeon to achieve alignment without further iatrogenic physeal injury.[15,16]

Many studies have been performed to examine the need for operative intervention over closed reduction and casting. The two procedures that orthopedic surgeons are most likely to perform are ORIF or CRPP. One of the indications for operative management includes post-reduction displacement. However, the degree of displacement needed for operative intervention depends on the anatomic location, and evidence varies on the amount of displacement needed at common anatomic locations, including the distal tibia physis, before operative management is recommended. Generally, more than 2 to 3 mm of displacement requires operative intervention, but emergency physicians should defer to recommendations from an orthopedist.[11,16] Other indications for operative management include an inability to perform closed reduction, open fractures, unstable fractures, and fractures with higher degrees of angulation (especially those with failed closed reduction).

The possibility of physeal arrest, which occurs in 5% to 10% of all growth plate fractures, should be discussed with patients and their families.[19] Premature growth arrest is the unexpected discontinuation of appositional or longitudinal bone growth that occurs after an insult to the physeal plate before skeletal maturity. Depending on the location of the injury, premature growth arrest can lead to limb length

discrepancy or angular deformity. Anatomic reduction does not guarantee that growth arrest will not occur. As such, outpatient follow-up care must be arranged with orthopedic surgery for observation and any further outpatient interventions. Generally, the higher the classification of the Salter-Harris fracture, the higher the likelihood of a poor prognosis, growth arrest, and need for surgery. However, the prognosis of growth plate fractures predominantly depends on the location of the injury, angulation or displacement of the fracture, fracture stability, and comorbid medical conditions.[19,20]

CRITICAL DECISION

What are the discharge instructions for patients with growth plate fractures?

Emergency physicians should relay important information at discharge to increase the chances of a good prognosis. Discharge instructions should include information on timely follow-up care when indicated, strict return precautions, the patient's weight-bearing status, and pain management prescriptions.

Adequate and timely follow-up recommendations depend on several factors including the nature of the fracture, concerns for patient compliance, the availability of appointments, the expected prognosis, and injury location. Initial follow-up visits for stable fractures should be scheduled within 3 to 7 days of presentation, with instructions for return precautions provided. Unstable fractures need more frequent reassessments post reduction to ensure proper maintenance of alignment, sometimes up to twice a week; be sure to communicate with orthopedic surgery colleagues to ensure these patients have proper follow-up appointments and discharge instructions. Displaced physeal fractures require patients to be monitored for growth disturbances for at least 1 year or until these patients are skeletally mature. If the physeal fracture is nondisplaced, the potential for growth arrest is low; follow-up care should be initiated by family if the patient develops new symptoms or a new deformity.[14] Patients with type I fractures can generally follow up with pediatricians and do not always require orthopedic referral.

For extremity fractures, emergency physicians should make sure that patients and their caregivers are aware of activity restrictions and the patient's weight-bearing status. They must also be made aware of return precautions, which include new or worsening symptoms such as pain, numbness, tingling, or skin discoloration. These symptoms can be associated with compartment syndrome, a feared complication of extremity fractures that is more likely to occur in patients who wear casts that are difficult to remove. Because of the pain associated with growth plate fractures, these patients should also be discharged with appropriate analgesia and instructions for a multimodal pain plan.

Summary

Growth plate fractures are among the most common pediatric fractures seen in the emergency department. They occur through the hypertrophic calcification zone of the physeal plate, which is the weakest point of developing bones. Growth plate fractures are described according to the Salter-Harris classification system. In the emergency department, plain x-rays with orthogonal views are generally sufficient to characterize physeal plate fractures, although CT imaging may be required in some cases. Management of physeal plate fractures depends on the location of the injury, fracture type, neurovascular status, degree of displacement, articular congruence, condition of the overlying skin, and patient age. If available, orthopedic surgery should be consulted for displaced fractures to determine the need for closed reduction versus surgical management. At discharge, physicians should review with patients and their caregivers the patient's weight-bearing status, activity restrictions, and return precautions to reduce the morbidity associated with these fractures. Orthopedic follow-up care is recommended for all Salter-Harris fractures except for type I fractures, which can start with pediatrician follow-up care instead.

✕ Pitfalls

- Excluding a Salter-Harris type I fracture based on normal x-rays. Patients with clinical findings consistent with a type I fracture should still be immobilized even if x-rays are normal.
- Ordering only a single-view x-ray. Multiple views of the injury area should be ordered because single views can miss growth plate fractures.
- Neglecting to provide thorough discharge instructions. Topics to cover at discharge are weight-bearing status, activity restrictions, return precautions, and follow-up instructions.
- Failing to consult orthopedic surgery for high-risk fractures (eg, Salter-Harris types III through V fractures, displaced fractures).

CASE RESOLUTIONS

CASE ONE

The patient's x-ray demonstrated a fracture through the distal right radius physeal plate with anterior displacement of the radial metaphysis, consistent with a Salter-Harris type I fracture. After consultation with an orthopedic surgeon, who evaluated the patient at bedside, closed reduction under procedural sedation with ketamine was performed. The emergency physician and orthopedic surgeon reduced the fracture; post-reduction plain x-rays confirmed the reduction. The fracture was then immobilized with a sugar tong splint, and the patient was discharged with instructions to follow up with a pediatric orthopedic surgery clinic in 1 week. At the follow-up appointment, repeat x-rays showed no displacement. The patient's right wrist fully recovered without growth plate arrest, worsening pain, or anatomic deformity.

CASE TWO

The lateral right ankle x-ray showed a nondisplaced fracture of the posterior tibia metaphysis that extended to disrupt the growth plate. A triangular metaphyseal fragment, or a Thurstan Holland fragment, was present along the posterior aspect, indicating a Salter-Harris type II fracture. The patient was placed in a posterior short leg splint, given crutches, and instructed on strict non–weight-bearing precautions. She had a scheduled follow-up appointment with a pediatric orthopedic specialist within 1 week of her presentation. One year later, the patient was able to participate in ballet and had no growth plate abnormalities, pain, or anatomic deformities.

CASE THREE

The patient was found to have a closed Salter-Harris type IV fracture of the distal fibula. He underwent ORIF and was sent home with instructions to remain non–weight-bearing on the right leg for 6 weeks. His distal fibula fracture carried a lower risk of physeal arrest and post-traumatic arthritis (compared with distal tibia fractures). He was able to follow up with the orthopedic clinic and was eventually able to bear weight without pain. He did not experience any growth arrest.

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Drug Box

Ceftazidime-Avibactam

By Frank LoVecchio, DO, MPH, FACEP
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Objective

On completion of this column, you should be able to:

- Discuss the indications for ceftazidime-avibactam.

Introduction

Ceftazidime-avibactam is a fixed-dose combination agent used for the treatment of gram-negative, multidrug-resistant complicated intra-abdominal and urinary tract infections, often when no other option is available. It is indicated for the treatment of *Escherichia coli*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae* and *oxytoca*, *Enterobacter cloacae* and *aerogenes*, *Citrobacter freundii* and *koseri*, and *Proteus mirabilis*.

Mechanism of Action

Ceftazidime is a third-generation β-lactam cephalosporin; avibactam is a non–β-lactam β-lactamase inhibitor. B-lactams are suicide inhibitors for transpeptidase, the enzyme responsible for the final cross-linking step in the synthesis of the peptidoglycan mesh on the bacterial cell wall. These agents ultimately lead to lysis and death of the bacterial cell. Over time, bacteria have developed resistance to such mechanisms by producing β-lactamases, enzymes that directly inactivate β-lactams. However, the coadministration of a β-lactamase inhibitor and a β-lactam can restore the effectiveness of β-lactams.

Indications

- Intra-abdominal infections in combination with metronidazole for complicated intra-abdominal infections
- Complicated urinary tract infections and pyelonephritis
- Hospital-acquired bacterial pneumonia and ventilator-associated bacterial pneumonia

Dosing

- Common dose is 2.5 g IV three times daily
- Adjust doses for kidney function
- No hepatic dose adjustment

Pharmacological Properties

- ***Half-life:*** ceftazidime (2.76 hr); avibactam (2.71 hr)
- ***Excretion in urine:*** ceftazidime (80%-90% unchanged); avibactam (100% unchanged)

Side Effects

Common complications include constipation, anxiety, nausea, vomiting, abdominal pain, and dizziness. Patients also may develop *Clostridioides difficile*–associated diarrhea, although this side effect is likelier with prolonged use.

Precautions

- ***Contraindications:*** previous hypersensitivity to ceftazidime, avibactam, or other cephalosporins or β-lactams
- ***Considerations:*** Dose adjustments are required in renal failure.
- ***Pregnancy:*** Animal studies report adverse effects with the use of avibactam. Ceftazidime is excreted in breast milk; excretion of avibactam is unknown.

Tox Box

Bupropion Toxicity

By David Carroll, MD; and Brandtly Yakey, DO
Michigan Poison and Drug Information Center
Reviewed by Christian A. Tomaszewski, MD, MS, MBA, FACEP

Objective

On completion of this column, you should be able to:

- Recognize and manage bupropion toxicity.

Introduction

Bupropion is a substituted monocyclic aminoketone (cathinone) used therapeutically for depression and smoking cessation. Related cathinones occur naturally in the shrub *Catha edulis* and are chewed recreationally to achieve a mild stimulant effect. Bupropion is an important cause of drug-related seizures, which can be idiosyncratic or dose dependent. In overdose, bupropion can impair intercellular cardiac conduction and lead to dysrhythmia and cardiogenic shock.

Sources of Exposure

- Most commonly oral ingestion of the extended-release ("XL") formulation
- Sustained and instant release formulations also available
- Intranasal and intravenous use less common

Mechanism of Action (Physiology/Toxicity)

- Inhibition of norepinephrine and dopamine reuptake transporters
- Noncompetitive antagonist at nicotinic receptors (α4β2 subunit)
- May inhibit ion flux through cardiac gap junctions in overdose

Pharmacokinetics/Toxicokinetics

- Absorption saturable in overdose; serum concentrations may rise over 8+ hr
- Highly protein bound, hepatic metabolism to active metabolites (eg, hydroxybupropion)

Risk Stratification

- Tachycardia, agitation, and altered mental status associated with delayed (≥8 hr) seizures
- Seizures, intentionality, age, and QRS widening with or without long QT interval associated with cardiotoxicity

Clinical Manifestations (Acute Overdose)

- ***Mild to moderate:*** tachycardia, diaphoresis, tremor, psychomotor agitation, and seizures
- ***Severe:*** status epilepticus, cardiogenic shock, and CNS depression (brain death mimic)

Diagnostics

- Point-of-care glucose test, ECG, acetaminophen concentration
- Serum bupropion (and metabolites) test not readily available; may be selectively useful

Treatment

- Activated charcoal if recent (~1-2 hr) ingestion; whole bowel irrigation if massive ingestion and intubated
- Benzodiazepines and/or phenobarbital for seizure cessation (with or without prophylaxis if high risk)
- Sodium bicarbonate for shock with QRS widening (*may* not be effective)
- Consultation for venoarterial extracorporeal membrane oxygenation if refractory cardiogenic shock
- No role for extracorporeal elimination (eg, hemodialysis)

Disposition

- Admit all extended-release ingestions if ≥900 mg or symptomatic