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Lesson 13 Subtle Poisonings ........................................ Page 2
Emergency physicians often are called on to be the local toxicologists in the emergency department. Many toxic exposures are obvious, but others can present in a more subtle fashion, in which case, significant overdoses may be overlooked. The emergency physician must be able to recognize these subtle presentations.

Lesson 14 STEMI Mimics ........................................ Page 10
An elevated ST segment on a patient’s ECG can indicate an acute myocardial infarction, but it can also indicate other life-threatening conditions. This lesson examines pericarditis, aortic dissection, and Brugada syndrome, three STEMI lookalikes that must be quickly differentiated from STEMI in order to institute appropriate treatment and, just as importantly, to prevent inappropriate treatment.

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Mary Anne Mitchell

Mary Anne Mitchell, ELS
Managing Editor, Critical Decisions in Emergency Medicine
Subtle Poisonings

Kevin M. Eanes, MD, and Brian E. Burgess, MD, FACEP

Objectives

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

1. Describe the subtle clinical manifestations of acetaminophen, salicylate, and sulfonylurea poisonings.
2. Explain which patients require N-acetylcysteine for acetaminophen toxicity.
3. Discuss which patients require transfer to a liver transplant center for acetaminophen toxicity.
4. Explain why urine alkanization increases salicylate excretion.
5. Discuss the indications for hemodialysis in patients with chronic salicylate toxicity.
6. Describe admission criteria for patients with sulfonylurea toxicity.
7. Discuss treatment options for sulfonylurea toxicity.

From the EM Model

17.0 Toxicologic Disorders
   17.1 Drugs and Chemical Classes

Drug toxicity is a growing public safety issue. In 2008 alone, the nation’s poison control centers were contacted for 2.5 million cases.1 Poisoning is the third leading cause of injury-related mortality, and from 1992 to 2002 mortality rates from poisoning increased 121%.2–3 As a result, emergency physicians are going to be expected to manage more toxic exposures each year, failure to recognize these poisonings in the emergency department can lead to increased morbidity and mortality.4

It is not sufficient to definitively diagnose a single overdose when patients report what, how much, and when they took the overdose. Emergency physicians must also be alert for an overdose when patients deny any ingestion. Emergency physicians must recognize the subtle presentations of significant poisonings (Table 1); it is the overdoses that patients do not report that have the greatest potential for harm.

Case Presentations

Case One

A 24-year-old obese woman presents complaining of abdominal pain, vomiting, and malaise. She describes the pain as a constant, nonradiating, dull ache in her right upper quadrant that has been getting progressively worse over the past 48 hours. She has never had this pain before, and there are no alleviating or exacerbating factors. She has no significant past medical or surgical history, takes no medications, and has no known drug allergies.

Her initial vital signs are blood pressure 100/60, pulse rate 110, respiratory rate 18, temperature 36.4°C (97.5°F), and oxygen saturation 99% on room air. The patient is diaphoretic with mild epigastric and right upper quadrant abdominal tenderness and no rebound or guarding; Murphy sign is negative. Laboratory findings include an AST 1,250, ALT 1,140, alkaline phosphatase 160, total bilirubin 1.5, lipase 62, and WBC 14.5; the rest of the CBC and basic metabolic panel results are within normal limits. Fairly certain of a diagnosis of gallbladder disease, the emergency physician performs bedside ultrasonography, which, to his surprise, reveals a thin-walled gallbladder without stones or pericholecystic fluid.

On further questioning, the patient tearfully confides that she has been depressed and under considerable stress since losing her job and has made suicide attempts by intentionally overdosing on acetaminophen. Her initial ingestion was 3 days prior when she took approximately 20 of the 500-mg tablets. When she did not experience any symptoms by the following day she ingested an additional 40 tablets. She denies any co-ingestants and now denies active suicidal ideation.

Case Two

A 78-year-old woman with a history of hypertension, type 2 diabetes, and rheumatoid arthritis is brought in by her husband because
Critical Decisions

- What is a toxic acetaminophen level?
- Which patients require N-acetylcysteine for acetaminophen toxicity?
- Which patients should be transferred to a liver transplant center following acetaminophen overdose?
- What is a toxic salicylate level?
- Which patients with salicylate poisoning should receive a bicarbonate infusion?
- Which patients require hemodialysis for salicylate toxicity?
- Which patients with sulfonylurea toxicity require hospital admission?
- How should refractory hypoglycemia caused by sulfonylureas be managed?

She has been having difficulty breathing. Her spouse notes that she has become gradually more short of breath over the past 2 days, and she has become increasingly confused this evening. Her medications are metoprolol, lisinopril, glipizide, low-dose aspirin, and simvastatin.

Her vital signs are blood pressure 110/78, pulse rate 104, respiratory rate 29, temperature 36.8°C (98.2°F), and oxygen saturation 88% on room air. Bedside glucose is 136. Physical examination reveals a patient in mild respiratory distress without retractions or prolonged exhalations. She is somnolent but opens her eyes to voice. Her heart is tachycardic, without murmurs, rubs, or gallops, and there is no jugular venous distention. Lung examination reveals bibasilar rales and no wheezes or rhonchi. Her abdomen is soft, nontender, and nondistended, with no pulsatile mass. There is no lower extremity edema or palpable cords. An ECG shows sinus tachycardia without significant ST changes. A portable chest radiograph reveals moderate pulmonary edema.

The patient is placed on noninvasive positive-pressure ventilation to improve her oxygenation. An arterial blood gas is drawn with the following results: pH 7.27, PCO₂ 18, HCO₃⁻ 13, PO₂ 110, and oxygen saturation 93%. A basic metabolic panel reveals sodium 132, potassium 3.9, chloride 96, bicarbonate 14, BUN 58, creatinine 2.3, and glucose 141. Lactate is 1.3, BNP is 149, and CBC and cardiac marker results are normal. Further discussion with the husband reveals that the patient had been suffering from a rheumatoid arthritis flare for the past week and had been taking extra doses of aspirin for the pain.

**Case Three**

A 5-year-old girl presents to the emergency department with the chief complaint of being “sleepy.” Her father notes that for the past 6 hours she has been very quiet and “acting tired.” She had previously been in her normal state of health, and this has never happened to her before. The father says that she has not had any fevers, cough, congestion, difficulty breathing, abdominal pain, vomiting, diarrhea, or rashes. She last ate 2 hours ago.

Initial vital signs are blood pressure 98/52, pulse rate 110, respiratory rate 18, temperature 36.1°C (97°F), and oxygen saturation 100% on room air. Bedside glucose is 68.

The patient is somnolent but awakens easily to voice and is able to follow commands. Examination reveals no evidence of trauma. Her pupils are 5 mm and briskly reactive, and her cranial nerves, strength, and reflexes are all normal. An HEENT examination reveals normal tympanic membranes, moist mucous membranes, and unremarkable tonsils. The cardiovascular, respiratory, and abdominal examinations are all unremarkable. The skin examination is normal and reveals no rashes.

A basic metabolic panel reveals sodium 136, potassium 4.2, chloride 106, bicarbonate 24, BUN 17, creatinine 0.6, and glucose 62. The CBC reveals WBC 7.9, hemoglobin 13.3, platelets 207, and a normal differential. Urinalysis and chest radiograph are both unremarkable.

While the physician is reviewing these results, the nurse interrupts to report that the patient is becoming increasingly less responsive.

**Acetaminophen**

A recent study revealed that each year acetaminophen-associated overdoses account for 56,000 emergency department visits and 26,000 hospitalizations. Despite readily available therapies, more than 450 Americans die annually from acetaminophen-associated toxicities, and approximately 100 of those are unintentional.

Most acetaminophen (85%) is conjugated by the liver and excreted by the kidneys. Approximately 5% to 10% is oxidized by the cytochrome P450 system to form a toxic metabolite, N-acetyl-p-benzoquinoneimine (NAPQI). Under normal conditions NAPQI is rapidly metabolized by glutathione to a nontoxic compound. When patients ingest toxic doses of acetaminophen, the normal conjugation pathways become saturated, and the P450 pathway plays a more prominent role. Eventually, glutathione stores are depleted, and excess NAPQI causes hepatotoxicity.

The presentation of acetaminophen poisonings can be very nonspecific; acetaminophen toxicity should be considered in patients who present...
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with mild gastrointestinal complaints. Rumack et al described four separate clinical stages.\(^6\) Stage I occurs before any hepatic injury has occurred. Nonspecific symptoms include nausea, vomiting, malaise, and pallor and can easily be mistaken for a viral illness or any number of disease processes. Some patients will even be asymptomatic in this stage. Stage II occurs within 24 to 72 hours and marks the beginning of hepatic injury. Symptoms, if present from stage I, may resolve, leading the patient or emergency physician to believe that the patient is improving. Symptoms can be similar to other forms of hepatitis accompanied by elevated liver enzymes. Stage III occurs 72 to 96 hours after ingestion and marks maximum hepatotoxicity. Synthetic functions of the liver are affected as evidenced by abnormal PT/INR, glucose, bilirubin, phosphate, ammonia, and lactate levels. Those who survive stage III will progress to recovery, stage IV. Most patients will have normalization of liver enzymes by day 7, although this may be delayed in severe poisonings.

**CRITICAL DECISION**

What is a toxic acetaminophen level?

In general, acetaminophen toxicity occurs after a dose of more than 150 mg/kg. For patients who present after a single overdose with a known time of ingestion, the Rumack nomogram is a sensitive predictor of toxicity.\(^7\) The original nomogram defines a 4-hour toxic acetaminophen level as greater than 200 mcg/mL. Patients with acetaminophen levels higher than this have a 60% risk of hepatotoxicity (arbitrarily defined as AST >1,000 IU/mL), 1% risk of renal failure, and 5% risk of mortality.

To enhance sensitivity and provide a layer of safety, the United States uses a modified Rumack nomogram with a 4-hour toxic level defined as levels above 150 mcg/mL. This modified nomogram was prospectively validated in 11,195 patients with acute acetaminophen overdose.\(^8\) Patients with an acetaminophen level below 150 mcg/mL have a 1% risk of hepatotoxicity; their symptoms will resolve spontaneously without

<table>
<thead>
<tr>
<th>Ingestion</th>
<th>Subtle Clinical Manifestations</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen*</td>
<td>Nausea, vomiting, malaise, abdominal pain</td>
<td>Charcoal; NAC</td>
</tr>
<tr>
<td>Salicylate*</td>
<td>Acute: nausea, vomiting, tinnitus, hyperventilation</td>
<td>Sodium bicarbonate; glucose; dialysis</td>
</tr>
<tr>
<td>Carbon monoxide*</td>
<td>Headache, dizziness, nausea, flu-like illness</td>
<td>100% oxygen, consider hyperbaric oxygen</td>
</tr>
<tr>
<td>Sulfonylurea*</td>
<td>Fatigue, dizziness, nausea, hypotension, focal neurologic deficits, seizures</td>
<td>Dextrose, octreotide</td>
</tr>
<tr>
<td>Iron*</td>
<td>Nausea, vomiting, diarrhea, abdominal pain</td>
<td>Deferoxamine</td>
</tr>
<tr>
<td>Digoxin*</td>
<td>Acute: nausea, vomiting, hyperkalemia, lethargy, bradyarrhythmias</td>
<td>Digoxin-specific antibody fragments (Fab)</td>
</tr>
<tr>
<td>Dilantin*</td>
<td>Nystagmus, ataxia, lethargy, slurred speech</td>
<td>Supportive care</td>
</tr>
<tr>
<td>Organic mercury*</td>
<td>Paresthesias, headaches, ataxia, dysarthria</td>
<td>Irreversible; may consider succimer</td>
</tr>
<tr>
<td>Lithium*</td>
<td>Chronic: tremor, nystagmus, ataxia, polyuria</td>
<td>Dialysis</td>
</tr>
<tr>
<td>Arsenic*</td>
<td>Peripheral neuropathy, cranial nerve palsy, progressive ascending paralysis</td>
<td>Dimercaprol; consider dialysis in renal failure</td>
</tr>
<tr>
<td>Lead*</td>
<td>Memory loss, insomnia, myalgias, peripheral neuropathy, hyperreflexia</td>
<td>Dimercaprol; edetate calcium disodium</td>
</tr>
<tr>
<td>Strychnine*</td>
<td>Involuntary muscle contractions, hyperthermia</td>
<td>Supportive care; benzodiazepines</td>
</tr>
<tr>
<td>Amanita phalloides* (death cap mushroom)</td>
<td>Delayed (up to 48 hours) nausea, vomiting, and diarrhea followed by progressive hepatic failure</td>
<td>NAC, penicillin, alpha lipoic acid, liver transplant</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>Bradycardia, heart blocks, hypotension, altered mental status, hyperglycemia, nausea, vomiting*</td>
<td>IV fluids, pressors, calcium gluconate, IV insulin, glucagon</td>
</tr>
<tr>
<td>(β)-Blockers</td>
<td>Bradycardia, heart blocks, hypotension, hypoglycemia(^6)</td>
<td>IV fluids, pressors, IV insulin, glucagon, pacing</td>
</tr>
<tr>
<td>Clonidine</td>
<td>Lethargy, coma, respiratory depression, pinpoint pupils, bradyarrhythmia(^6)</td>
<td>IV fluids, pressors, naloxone</td>
</tr>
</tbody>
</table>


\(^b\) Especially in pediatric patients
treatment, and there is a 0% risk of mortality. Any patient with a single acute overdose with a known time of ingestion should have an acetaminophen level checked and plotted on the modified nomogram to predict toxicity.

The Rumack nomogram has its limitations, however. The nomogram has no role in delayed presentations or chronic ingestions. Because it is the metabolites of acetaminophen that are toxic, a therapeutic or negative acetaminophen level does not guarantee patient safety. Patients with delayed presentations or chronic ingestions should have liver enzymes measured along with an acetaminophen level. In 2004, Daly et al demonstrated that patients with delayed presentations (more than 24 hours after ingestion) or chronic ingestions who had an acetaminophen level of less than 10 mcg/mL and an AST below 50 IU/mL had a 0% risk of developing hepatotoxicity. Patients suspected of chronic, repeated ingestions and with detectable serum acetaminophen levels (>10 mcg/mL) or elevated liver enzymes should be presumed to have acetaminophen toxicity.

**CRITICAL DECISION**

**Which patients require N-acetylcysteine for acetaminophen toxicity?**

The use of N-acetylcysteine (NAC) for the prevention and treatment of hepatotoxicity in acetaminophen overdose has been established as both safe and effective. Although there are a variety of oral and intravenous regimens, the most commonly used is the 20-hour intravenous NAC regimen. The initial loading dose is 150 mg/kg delivered over 15 minutes, followed by a 50-mg/kg dose delivered over 4 hours, followed by a 100-mg/kg dose delivered over 16 hours. The emergency physician must decide not only which patients might benefit from NAC but also when to begin administration.

For acute overdoses, in which an acetaminophen level is available within 8 hours of the time of ingestion, the emergency physician has the luxury of time. Smilkstein et al demonstrated that NAC administration within the first 8 hours is essentially 100% effective at preventing hepatotoxicity. This does not imply that physicians should unnecessarily delay NAC administration; rather, this knowledge should reassure the clinician that an acetaminophen level can be checked and plotted on the nomogram without negative consequences.

Because patients are at greater risk of morbidity and mortality if they are not treated with NAC within 8 hours of ingestion, all patients with delayed presentations should be empirically treated with NAC prior to any laboratory evaluation. If the acute ingestion occurred within the past 24 hours, the acetaminophen level should be plotted on the nomogram. If the patient’s level is above the toxic level, NAC should be continued, and the patient should be admitted for further treatment. Likewise, all patients with ingestions that occurred more than 24 hours before presentation and those with chronic or multiple ingestions should be empirically treated with NAC. These patients with acetaminophen levels of more than 10 mcg/mL or AST levels above 50 IU/mL should be presumed to have toxicity and should be admitted for continuation of treatment. Remember that patients who smoke and chronically consume alcohol, those who are fasting or debilitated, and those who take medications that delay gastric emptying (opiates, anticholinergics) or induce liver enzyme activity (phenytoin, phenobarbital, carbamazepine, rifampin, andisoniazid) are more susceptible to acetaminophen toxicity.

Even patients who present in fulminant hepatic failure benefit from NAC. Keays et al demonstrated that patients with fulminant hepatic failure who received NAC had improved survival rates (48% versus 20%), a lower incidence of cerebral edema (40% versus 68%), and fewer episodes of hypotension requiring pressor support (48% versus 80%). If there is any doubt, err on the side of caution and administer NAC until the case can be discussed with the local poison control center. Activated charcoal (1g/kg up to 50 grams) should be administered within 4 hours of an ingestion for decontamination.

**CRITICAL DECISION**

**Which patients should be transferred to a liver transplant center following acetaminophen overdose?**

Although patients with fulminant hepatic failure still benefit from NAC administration, it is important to identify which patients will require a liver transplant for survival. The King criteria predict death from fulminant hepatic failure if the patient does not receive a liver transplant. The King criteria are a serum pH below 7.3 after adequate fluid resuscitation; creatinine above 3.3 mg/dL; INR above 6.5, and grade III or IV encephalopathy. If any of the above criteria are met, serious consideration should be given to transferring the patient to a regional liver transplant center for definitive management (Table 2).

**Salicylates**

Analgesics are involved in 17% of all overdoses, and isolated salicylate poisoning occurs in 3% of all poisoning fatalities. It is critical that emergency physicians recognize salicylate toxicity, because mortality...
rates triple when the diagnosis is not made in the emergency department.4

Salicylates produce a constellation of acid-base disturbances. Salicylates stimulate the respiratory center in the brainstem, causing hyperventilation and a respiratory alkalosis.13 They also disrupt the tricarboxylic acid (Krebs) cycle and uncouple oxidative phosphorylation.16 The result is an accumulation of pyruvic and lactic acids as well as ketone bodies from fatty acid metabolism, producing a wide anion gap metabolic acidosis. Ultimately, patients with acute lung injury or central nervous system depression will develop a respiratory acidosis. Acute respiratory acidosis that occurs early during the course of an aspirin overdose should alert the emergency physician that a possible co-ingestion with a respiratory depressant may have occurred.

Acute salicylism is heralded by a triad of hyperventilation, tinnitus, and gastrointestinal upset.17 Chronic salicylism is a more subtle poisoning and can be mistaken for sepsis, unstable angina, stroke, congestive heart failure, hyperthyroidism, or failure to thrive.18,19 Compared to acute poisoning, chronic salicylism is more likely to occur in elderly patients, who can present with altered mental status or respiratory distress.

**CRITICAL DECISION**

**What is a toxic salicylate level?**

Salicylate toxicity occurs with doses greater than 150 mg/kg. Physicians have been trying to correlate salicylate levels to toxicity since Done introduced his nomogram in 1960.20 Done retrospectively examined 38 pediatric cases of acute salicylate ingestion and calculated peak serum concentrations based on an assumed constant half-life. However, salicylates do not have a constant half-life, and certain elimination mechanisms are saturable while others follow first-order kinetics.21 Calculation of peak serum concentration is inaccurate; Dugandzic et al attempted to validate Done’s work and found that the nomogram accurately predicted clinical toxicity less than 50% of the time.22

Salicylate toxicity is best determined by the patient’s clinical picture. Although an elevated salicylate concentration could indicate the etiology of the patient’s symptoms, the data should not guide therapy. Laboratory evaluation is particularly inaccurate in chronic salicylism. Salicylate levels correlate poorly with chronic toxicity, and one third of patients with salicylate toxicity have normal serum bicarbonate and anion gaps.19 As always, emergency physicians should treat the patient and not the numbers.

**CRITICAL DECISION**

**Which patients with salicylate poisoning should receive a bicarbonate infusion?**

Salicylates are mainly excreted by the kidney. Like all acids, they cross cell membranes and are reabsorbed in their non-ionized form and are eliminated in the urine when ionized. As weak acids, salicylates are predominantly ionized in an alkaline pH. Urine alkalinization promotes salicylate elimination by keeping salicylates in their ionized form. Since pKa is a logarithmic function, small changes in urine pH have a significant effect on elimination. This theoretical effect has been demonstrated clinically in the medical literature; urine alkalinization to a pH of 7.5 to 8 has been shown to significantly increase salicylate elimination and decrease salicylate half-life.23,24

The American Academy of Clinical Toxicology recommends that “urine alkalinization should be considered as first-line treatment for patients with moderately severe salicylate poisoning who do not meet the criteria for hemodialysis.”23 A case report demonstrated that urine alkalinization was superior to dialysis in the initial elimination of salicylates, and some experts recommend prompt urine alkalinization even in patients who meet criteria for dialysis.24,25

Maintaining a serum pH around 7.5 is recommended even if the patient is alkalemic because of an initial respiratory alkalosis. This promotes diffusion of tissue and brain salicylate into the extracellular fluid. Frequent monitoring of the blood pH is imperative. Supplemental glucose administration is also recommended, because aspirin depresses central nervous system glucose levels. Patients with altered mental status despite a normal blood glucose level should receive glucose infusions.

Hypokalemia in this population should be aggressively managed because of the potassium/hydrogen ion exchange within the renal tubules. Activated charcoal (1g/kg up to 50 grams) should be administered within 4 hours of an ingestion for decontamination. Intubation, if required, should be completed while maintaining appropriate minute ventilations so that the pH remains 7.5 to 7.59.

**CRITICAL DECISION**

**Which patients require hemodialysis for salicylate toxicity?**

Multiple studies have demonstrated the safety and effectiveness of hemodialysis in salicylate poisoning.24-28 Experts recommend hemodialysis for the following indications: renal failure, congestive heart failure, acute lung injury, persistent neurologic disturbances, hemodynamic instability, severe acid-base or electrolyte disturbance despite urine alkalinization, hepatic compromise with coagulopathy, or a salicylate concentration above 100 mg/dL regardless of symptoms.25,29 It is best to consult the nephrology service early, even if the patient does not require hemodialysis during the initial treatment.

**Sulfonylureas**

Sulfonylureas are a class of oral hypoglycemic agents that are commonly prescribed for type 2 diabetes. In a review of 1,418 cases of drug-induced hypoglycemia, sulfonylureas were involved in
nearly two thirds of the cases. Sulfonylureas stimulate release of endogenous stores of insulin from the pancreas. The clinical manifestations of sulfonylurea overdose are all related to hypoglycemia. Mild cases can present as lethargy or confusion, while more severe cases can present as focal neurologic deficits, coma, or seizure. Other nonspecific clinical manifestations such as dizziness, anxiety, nausea, palpitations, and diaphoresis can occur. These symptoms of hypoglycemia will worsen without prompt treatment. There is no specific test to diagnose sulfonylurea overdose. The diagnosis is made either when the patient or a witness reports the ingestion or when a patient with potential access to the drug is hypoglycemic.

**CRITICAL DECISION**

Which patients with sulfonylurea toxicity require hospital admission?

Sulfonylureas have an average duration of action of 24 hours, with duration of action ranging from 12 hours (tolbutamide) to 72 hours (chlorpropamide). Hypoglycemic episodes can be both delayed and precipitous. As a result, experts recommend that any adult patient who is hypoglycemic from a sulfonylurea poisoning should be admitted. Furthermore, there are cases in the medical literature of pediatric exposures in which hypoglycemia occurred more than 12 hours after presentation. For this reason, many experts also recommend that all pediatric patients be admitted for 24 hours of observation if they ingest even a single sulfonylurea tablet.

**CRITICAL DECISION**

How should refractory hypoglycemia caused by sulfonylureas be managed?

Sulfonylureas produce hypoglycemia by stimulating the release of endogenous insulin stores. Serum insulin concentration can reach supra-physiologic levels after an overdose, which can cause a profound hypoglycemia that is refractory to dextrose infusion.

Octreotide is a somatostatin analogue that inhibits sulfonylurea-mediated insulin release. It has been shown to be both safe and effective in cases of sulfonylurea toxicity. The recommended dosing is 1 or 2 mcg/kg in children or 50 to 100 mcg in adults. Octreotide can be administered either subcutaneously or intravenously and may be repeated in 12 hours as needed.

Boyle and colleagues demonstrated that insulin levels are five times higher when patients with sulfonylurea poisoning are treated with dextrose infusion than when octreotide is used. As a result, patients treated with octreotide require less dextrose during their hospitalization. McLaughlin et al discovered that patients treated with dextrose alone were 27 times more likely to become hypoglycemic compared to those treated with both dextrose and octreotide. Dextrose infusions are usually given as follows: 5 to 10 mL/kg of D5W for neonates; 2 to 4 mL/kg of D5W for children; and 1 to 2 mL/kg of D5W for adults.

Patients at risk of increased toxicity include the elderly, those with impaired renal function, patients with poor oral intake, and those on multiple medications (increased risk of drug interactions). Activated charcoal may be given within the first 2 hours of ingestion to inhibit absorption.

**Case Resolutions**

**Case One**

The young woman who attempted suicide with acetaminophen was treated with intravenous NAC and admitted to a monitored bed. The following morning, repeat liver enzymes showed improvement: AST of 540 and ALT of 616. The NAC was continued for an additional 20 hours, and the following day the patient’s liver enzymes had normalized. She was discharged from the hospital, and outpatient followup with a psychiatrist was scheduled.

**Pears**

- Always consider a toxic ingestion in the differential diagnosis.
- Pay close attention to the details of the patient’s presenting history.
- When initial clinical suspicions are wrong, ask more questions.
- Know the antidotes for common poisonings.
- Know your limitations—a clinical toxicologist is only a phone call away.
- Patients are more susceptible to acetaminophen toxicity if they smoke and chronically consume alcohol, if they are fasting or debilitated, or if they take medications that delay gastric emptying (opiates, anticholinergics) or induce liver enzyme activity (phenytoin, phenobarbital, carbamazepine, rifampin, and isoniazid).
- Urine alkalinization should be considered as first-line treatment for patients with moderately severe salicylate poisoning who do not meet the criteria for hemodialysis.

**Pitfalls**

- Not considering poisoning in your differential diagnosis.
- Assuming patients will report their toxic exposures.
- Failing to recognize subtle toxidromes.
- Failing to use the regional poison control center.
- Relying on serum salicylate levels and not on the patient’s clinical picture to determine aspirin toxicity.
- Relying on the acetaminophen toxicity nomogram when the patient has a chronic ingestion or multiple ingestions over time.
Case Two

In the case of the elderly woman who was brought in for difficulty breathing, her salicylate level was found to be 30 mg/dL. She was started on a bicarbonate infusion to increase salicylate excretion. Due to the patient’s altered mental status and acute renal failure, she was considered to be a good candidate for emergent hemodialysis. The patient was admitted to the ICU, and after dialysis and continued bicarbonate infusion she was alert and breathing comfortably on room air. She was observed in the hospital for an additional 48 hours and then discharged home without complications.

Case Three

The emergency physician immediately returned to check on the 5-year-old girl who was becoming less responsive. Her ABCs were found to be intact, but she barely roused to deep sternal rub. A repeat bedside glucose test revealed a blood glucose of 24. Half an ampule of D50 was administered, and the patient suddenly awakened and began to cry. Further discussion with the father revealed that he has type 2 diabetes and takes glyburide for glucose control; this information combined with the clinical picture pointed to a diagnosis of glyburide overdose.

The father was grateful that the emergency physician had identified what was wrong and asked if they could go home soon. The physician explained that the glyburide would remain in his daughter’s circulation for the next 24 hours and that she should be admitted. A dextrose infusion was started, and a 2-mcg/kg intravenous dose of octreotide was given to prevent further release of endogenous insulin. The patient remained euglycemic throughout her hospital stay and was discharged home 48 hours later.

Summary

Toxic exposures are a growing threat; it is essential that emergency physicians be able to diagnose and treat these patients effectively. Failure to recognize these subtle poisonings in the emergency department could lead to further morbidity and mortality.

References

The LLSA Literature Review

“The LLSA Literature Review” summarizes articles from ABEM’s “2011 Lifelong Learning and Self-Assessment Reading List.” These articles are available online in the ACEP LLSA Resource Center (www.acep.org/llsa) and on the ABEM Web site.

A Systematic Review of Medical Therapy to Facilitate Passage of Ureteral Calculi

Reviewed by Maame Yaa A. B. Yiadom, MD, MPH, and J. Stephen Bohan, MS, MD, FACEP; Harvard Affiliated Emergency Medicine Residency; Brigham and Women’s Hospital


Pain control is the usual initial treatment for patients presenting with a ureteral stone. Fluids are often given in an attempt to hasten stone expulsion. Recent studies have looked at the benefit of calcium channel blockers (nifedipine, verapamil, diltiazem) and α-antagonists (tamsulosin, terazosin, doxazosin, alfuzosin, prazosin) to assist stone passage. This study is a pooled analysis of randomized or controlled trials done on adult patients to review the effectiveness of these adjuncts.

Calcium channel blockers were shown to be a beneficial adjunct, with stone expulsion 1.5 times more likely to occur sooner than with standard treatment alone. The number needed to treat was 3.9. For two-thirds of the included studies, average stone size was less than 5 mm. The side effects noted were transient hypotension, dyspepsia, headache, palpitations, and reduction in mean blood pressure.

α-Antagonists also demonstrated a risk ratio of 1.59 and a number needed to treat of 3.3. Stone size in these studies ranged from 1.8 to 3 mm. There was a 2- to 6-day improvement in stone expulsion compared to controls. Side effects, although not consistently reported, included dizziness, headache, asthenia, nausea, and vomiting.

Highlights

- The results of this analysis suggest a significant benefit in time to stone passage rates when either a calcium channel blocker or an α-antagonist is added to standard therapy.
- Side effects were substantially more common with calcium channel blockers.
- A 2-week course of therapy is recommended.
- The preferred choice of pharmacologic agent has yet to be determined. At this stage, cost and side effects should guide the practitioner’s choice.
Critical Decisions in Emergency Medicine

Lesson 14

STEMI Mimics

Laurie A. James, DO, and Gary T. Giorgio, MD

Objectives

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

1. List the differential diagnosis for patients with ECGs showing ST-segment elevation.
2. Describe the physical signs and symptoms that can present with ST-segment elevation but that do not represent ST-segment elevation myocardial infarction (STEMI).
3. Identify the common ECG findings during each stage of pericarditis.
4. Identify the ECG abnormalities in thoracic aortic dissections.
5. Describe appropriate antihypertensive treatment in patients with an acute aortic dissection.
6. Describe the three ECG patterns seen in Brugada syndrome.

From the EM Model

1.0 Signs, Symptoms, and Presentations
1.3 Chest/Chest pain

Coronary heart disease continues to be the leading cause of death in the United States. Each year, there are approximately 200,000 fatal cases of acute myocardial infarction (AMI). Emergency physicians are responsible for immediately obtaining an ECG and accurately identifying patients that meet criteria for a ST-segment elevation myocardial infarction (STEMI). It is imperative that this diagnosis is made quickly, because the 1-year mortality risk is increased by 7.5% for each 30-minute delay in treatment. To complicate this process, there are many other diseases that mimic myocardial infarction, and they too must be rapidly identified. Many protocols are focused on quickly identifying STEMI and activating the appropriate management team. The preferred management of a STEMI is early reperfusion with percutaneous coronary intervention (PCI), with a door-to-balloon time of less than 90 minutes, or thrombolysis when PCI is not readily available. Indications for PCI include chest pain or equivalent symptoms at rest, with either ST-segment elevation in two or more contiguous leads (>2 mm in the precordial leads or >1 mm in the limb lead), ST-segment depression of more than 1 mm in the precordial leads, or a new left bundle-branch block.

However, emergency physicians are also responsible for considering other diseases that can mimic AMI. A “STEMI mimic” is a condition that has a clinical presentation and ECG findings similar to that of an acute ischemic coronary artery syndrome. In fact, the most common cause of ST-segment elevation in emergency department patients is left ventricular hypertrophy and not an AMI. It is important that these mimics be properly identified, because inadvertent administration of thrombolytics or PCI can be harmful to patients not experiencing STEMI and further delay the treatment of their actual disease.

The differential diagnosis is broad for patients with chest pain and ST-segment elevation. The differential can be organized into conditions related to a cardiac process but not affecting the coronary arteries, conditions of vascular origin but not coronary artery occlusion, and noncardiac conditions. This lesson addresses three important STEMI mimics that should not be missed—pericarditis, aortic dissection, and Brugada syndrome. In pericarditis there is inflammation of the pericardial sac that creates a diffuse ST-segment elevation on the ECG. In a thoracic aortic dissection, the dissection can extend into the coronary arteries and show ischemic-like changes on the ECG consistent with a STEMI. Brugada syndrome is a channelopathy associated with sudden cardiac death and can be mistaken for a STEMI because the ECG demonstrates a right bundle-branch pattern with J-wave elevation.
Case Presentations

**Case One**
A 34-year-old woman presents to the emergency department complaining of chest pain. She has had discomfort for 2 days, but tonight when she went to bed the pain increased. She describes the pain as a sharp, burning, left-sided chest pain. The pain has been constant and increases in intensity when she is lying down. She took acetaminophen without any relief. She also reports feeling more short of breath today. She denies any chronic medical problems but notes that she had an upper respiratory infection last week. Vital signs in triage are blood pressure 108/60, pulse rate 114, respiratory rate 18, temperature 37.3°C (99.1°F) orally, and oxygen saturation 97% on room air. On examination, the patient is ill appearing. She has dry mucous membranes. Her cardiovascular examination reveals a regular tachycardia with a rub noted when the patient is in a sitting position. No murmurs or gallops are heard. Her lungs are clear bilaterally to auscultation. Her abdomen is soft, nontender, and nondistended. Extremities are warm and dry with strong symmetrical peripheral pulses and no signs of peripheral edema. An ECG done at the bedside shows diffuse concave-upward, ST-segment elevation with PR-segment depression except in lead aVR, which has ST-segment depression and PR-segment elevation (Figure 1).

**Case Two**
An emergency dispatcher notifies the emergency physician that paramedics are en route with a 62-year-old man who is complaining of midsternal chest pain. The paramedics describe the patient’s vital signs as stable, but he is hypertensive. When the patient arrives, he is visibly uncomfortable and complains of tearing chest pain that does not radiate anywhere. The patient states that the pain started suddenly and was most severe at the time of onset. He is also complaining of nausea and lightheadedness and says he feels like he is about to die. The patient has a long history of hypertension and hyperlipidemia; he takes medications as directed. He has a 30-pack-year smoking history and currently smokes ½ pack per day. He denies any alcohol or drug abuse. Vital signs are blood pressure 210/108, pulse rate 106, respiratory rate 18, temperature 35.7°C (98.2°F), and oxygen saturation 97% on 2 liters of oxygen via nasal cannula. On examination, the patient is diaphoretic and visibly uncomfortable. The cardiovascular examination reveals a regular tachycardic rhythm, with a diastolic murmur heard over the second right intercostal space, and no rubs or gallops. His lung examination is clear to auscultation and his abdomen is soft and nontender, with no palpable masses. His back is nontender. The patient is alert and oriented. Cranial nerves II through XII are grossly intact. He is moving all of his extremities but has diminished femoral pulses. A bedside ECG shows ST-segment elevation in leads II, III, and aVF.

**Case Three**
A 42-year-old man is brought to the emergency department by his wife after he apparently fainted. The wife states that they were at home hanging a picture on the wall when the patient suddenly collapsed to the ground. She was able to “catch him” and ease him down. The patient was unresponsive for about 10 seconds. When he woke up he was acting normally but did not recall the episode. He denies feeling lightheaded or dizzy prior to the episode. He currently feels
fine and would like to go home. He denies any chest pain, shortness of breath, or recent illnesses. He denies any medical problems, and he does not take any medication or over-the-counter herbs or supplements. He denies tobacco, alcohol, and drug use. A family history reveals that the patient’s father died at age 38 of uncertain cause.

Vital signs are blood pressure 128/78, pulse rate 88, respiratory rate 16, temperature 36.6°C (97.9°F), and oxygen saturation 100% on room air. The patient is a well-appearing man, in no acute distress. Cardiovascular examination reveals regular rate and rhythm with no appreciable murmurs, and his lungs are clear to auscultation. His abdomen is soft, nontender, nondistended, and with no palpable masses. His extremities are warm and dry with symmetrical pulses and no signs of clubbing or edema. The patient is awake and oriented, with a nonfocal neurologic examination.

A bedside ECG shows ST-segment elevation in V1-V3 with T-wave inversion (Figure 2).

**Pericarditis**

Acute pericarditis is inflammation of the pericardium and is responsible for approximately 1% of cases of ST-segment elevation in the emergency department. The actual cause of pericarditis is difficult to determine, and it is often labeled idiopathic. Some identifiable etiologies include infection, autoimmune disorders, neoplasms, trauma, hypothyroidism, uremia, and ovarian hyperstimulation syndrome.

Patients with pericarditis typically complain of parasternal chest pain that is exacerbated by inspiration or the supine position. On cardiovascular examination, a friction rub may be heard. If present, a rub is best heard at the lower left sternal border when the patient is in a seated position and leaning forward.

In order to diagnose pericarditis, two out of the four following features must be present: pericarditic-typical chest pain, pericardial friction rub, ECG changes consistent with pericarditis pattern, and a new or worsening pericardial effusion.

The initial workup in the emergency department includes an ECG, which will show abnormalities consistent with epicardial inflammation in about 90% of cases. The ECG findings will vary depending on the stage of the disease, but during the acute phase, the ECG has diffuse ST-segment elevations, which can mimic a STEMI.

Several differences can be noted in the ECG when comparing pericarditis to an AMI. Specifically, in pericarditis, the ST-segment elevation begins at the J point and maintains normal concavity and is seen in multiple leads representing more than one coronary artery territory. Reciprocal changes will be absent. Pathologic

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**Figure 1.**

ECG of patient in case one
Q waves should not be seen unless the patient has had a preexisting or associated MI.

Another important ECG finding that is indicative of pericarditis is ST-segment depression and PR-segment elevation in lead aVR.11

A chest radiograph should also be performed in the emergency department. In the presence of a pericardial effusion, the cardiac silhouette will be enlarged.11 Blood work can show several nonspecific abnormalities, including an elevated WBC count, elevated erythrocyte sedimentation rate, and elevated C-reactive protein. Cardiac enzymes should be obtained. Elevated markers suggest involvement of the myocardium, and the differential diagnosis should include myocarditis as well as myocardial infarction.10,11

The management of pericarditis in the emergency department depends on the patient’s clinical presentation. When treating a stable patient suspected of having idiopathic or viral pericarditis, the primary therapy is directed at relieving pain and reducing inflammation. Ibuprofen should be dosed at 300 to 800 mg every 6 to 8 hours for several days to several weeks, until symptoms improve. If the pericarditis is associated with an AMI, aspirin is the preferred medication.12

CRITICAL DECISION
What is the appropriate disposition for patients with pericarditis?

New cases of acute pericarditis often result in hospitalization for further testing, including an echocardiogram. Patients at high risk for complications, including those with persistent fever, large pericardial effusion, concerns for cardiac tamponade, immunosuppressed state, acute trauma, anticoagulant therapy, elevated troponin, or failure to respond to nonsteroidal anti-inflammatory drug therapy within 7 days, should be admitted for further evaluation. Patients without these predictors may be treated in an outpatient setting. The American College of Cardiology, the American Heart Association, and the American Society of Echocardiography recommend an echocardiogram for all patients with suspected pericarditis and, in selective cases, further evaluation into the cause with tuberculin skin testing, ANA titers, HIV serology, and blood cultures.13

CRITICAL DECISION
What physical examination findings are concerning for cardiac tamponade?

Cardiac tamponade occurs when an accumulation of fluid within the pericardial sac is large enough to obstruct the flow of blood into the heart. The amount of fluid needed to produce a tamponade depends on how quickly the fluid accumulates;

![Figure 2.](image)

ECG of patient in case three
Critical Decisions in Emergency Medicine

it can range from 200 mL (when fluids develop quickly) up to 2,000 mL (when a patient has a slowly developing effusion). A high degree of suspicion is required to make this diagnosis, but key findings include hypotension, tachycardia, increased jugular venous pressure, diminished heart sounds, and pulsus paradoxus (>10 mm Hg drop in systolic blood pressure during inspiration). On ECG, there can be low voltage with electrical alternans. A bedside echocardiogram can easily identify a large pericardial effusion and cardiac tamponade. The treatment is a pericardiocentesis or placement of a pericardial window to remove the fluid.9

Thoracic Aortic Dissection

Thoracic aortic dissection affects nearly 4.5 million patients per year. Incidence peaks in patients between 45 and 65 years of age, and it is more common in men and blacks. There are many causes of aortic dissection, but the most important risk factor is systemic hypertension. When younger patients develop a dissection it is often associated with other preexisting conditions such as a bicuspid aortic valve, coarctation, Marfan syndrome, Ehlers-Danlos syndrome, adult polycystic disease, or pregnancy.14,15

Aortic dissections occur when there is a disruption in the aorta’s intimal layer. There are two systems used to classify aortic dissections: the Stanford and the DeBakey. The Stanford classification system is more widely used; it classifies dissections based on the region of the aorta involved. Stanford type A refers to dissections that involve the ascending aorta, and type B describes only those affecting the descending aorta. The risk of rupture depends on the size of the aneurysm and increases significantly when the diameter of the ascending aorta is larger than 6 cm or the diameter of the descending aorta is larger than 7 cm. In general, a thoracic aneurysm grows 0.1 to 0.4 cm per year.16

The classic presentation of an aortic dissection is the sudden onset of ripping or tearing chest pain that radiates to the back. However, in clinical practice the presentation is variable, and up to 60% of patients do not experience any back pain.6 Pain is usually described as maximal at the time of onset, and patients can present with other associated symptoms such as syncope, hemiparesis from involvement of the carotid arteries, extremity paralysis from limb or spinal cord ischemia, myocardial infarction, and abdominal pain. Neurologic symptoms are present in up to 20% of cases, with the most common findings being syncope and altered mental status. It is also important to note that an aortic dissection is painless in up to 10% of patients. Specific examination findings that should increase suspicion for a thoracic aortic dissection are a new diastolic murmur, asymmetric pulses, asymmetric blood pressures, and extremes of blood pressure.17

Acute thoracic aortic dissection is a dangerous STEMI mimic, because improperly treating these patients with thrombolysis can have serious consequences. The patient’s clinical presentation of chest pain and ECG findings can strongly suggest an acute coronary syndrome. In type A dissections, the ECG can show ST-segment elevation involving the right coronary artery territory. This occurs when the dissection propagates back towards the coronary artery ostium and then involves the right coronary artery. When this extension occurs, the right coronary artery is most commonly affected, but the dissection can also extend toward other territories, including the atrioventricular conduction system, which can cause a heart block.

The ECG abnormalities observed in aortic dissection are nonspecific and can be mistaken for an acute coronary syndrome. Up to 15% of patients with aortic dissection show ischemic changes on the ECG. However, the most common ECG finding in aortic dissection patients is left ventricular hypertrophy and

<table>
<thead>
<tr>
<th>Stage</th>
<th>Duration</th>
<th>ECG Finding</th>
</tr>
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<tbody>
<tr>
<td>I</td>
<td>Hours to days</td>
<td>Diffuse ST-segment elevation (I, II, III, aVL, V2-V6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reciprocal ST-segment depression (aVR, V1)</td>
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<tr>
<td></td>
<td></td>
<td>Diffuse PR-segment depression (I, II, III, aVL, V2-V6)</td>
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<tr>
<td></td>
<td></td>
<td>Reciprocal PR-segment elevation (aVR, V1)</td>
</tr>
<tr>
<td>II</td>
<td>1 to 3 weeks</td>
<td>ST-segment normalization</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PR-segment normalization</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T-wave flattening</td>
</tr>
<tr>
<td>III</td>
<td>3 weeks to 2 months</td>
<td>Diffuse T-wave inversions (not seen in all patients)</td>
</tr>
<tr>
<td>IV</td>
<td>3 weeks to 2 months</td>
<td>ECG normalization or persistent T-wave inversion</td>
</tr>
</tbody>
</table>
nonspecific ST and T-wave changes. Studies have shown that up to 31% of patients have a normal ECG.15,18

The survival of patients with an acute aortic dissection depends on early diagnosis and aggressive treatment. During an acute dissection, it is estimated that the death rate increases 1% per hour, and if the dissection goes untreated 75% of patients will die within 2 weeks.6 A misdiagnosis of STEMI can result in the administration of anticoagulants, which would be detrimental to these patients.8 or they might be sent to the catheterization laboratory where the aortic dissection is discovered but the delay in diagnosis could be critical.

Emergency department evaluation should include an ECG, a chest radiograph, but if clinical suspicion is high, other noninvasive tests should be ordered, such as transesophageal echocardiogram, but if these may not be practical or available in the emergency department.17

**CRITICAL DECISIONS**

Can a chest radiograph reliably rule out the diagnosis of aortic dissection?

According to the International Registry for Aortic Dissection, 12% of patients with aortic dissection have chest radiographs that were read as normal. Although there are several radiographic findings associated with an aortic dissection, the most commonly cited is a widened mediastinum. Studies have shown that a widened mediastinum could be present in as few as 25% of cases and has an even lower sensitivity when the dissection is confined to the ascending aorta. Although a mediastinal width greater than 8 cm on an anteroposterior chest film is suggestive, the lack of this finding does not rule out dissection. Other findings indicative of dissection on chest radiograph include a blunted aortic knob, displacement of the aorta more than 5 mm past the calcified aortic knob, pleural effusion, tracheal or esophageal deviation, and depression of the left main stem bronchus.18,19

**CRITICAL DECISIONS**

What antihypertensive medications should be used for blood pressure control in a patient with an aortic dissection?

The blood pressure and heart rate goals for acute aortic dissection are a systolic blood pressure between 100 and 120 and a heart rate between 60 and 80 beats/min. Often the combination of a ß-blocker and vasodilator (such as nitroprusside) are effective in reaching these goals. Esmolol is an ultra–short-acting ß-blocker that can be quickly discontinued if hypotension develops. The loading dose for an adult is 250 to 500 mcg/kg over 1 minute followed by a maintenance infusion of 50 to 150 mcg/kg/min. Nitroprusside is often used in combination with a ß-blocker to reduce peripheral resistance. Nitroprusside is easily titratable at 0.5 to 3 mcg/kg/min. It is important that a ß-blocker be initiated first in order to avoid a reflex tachycardia that can result when nitroprusside is used alone.16,17

**Brugada Syndrome**

Brugada syndrome is an inherited cardiac ion channel dysfunction that is associated with syncope, cardiac arrest, and sudden cardiac death. The prevalence is highest among the Asian population and it is seen more commonly in men than women (8 to 10 times). The average age at presentation is between 30 and 50 years of age, with case reports of patients as young as 2 days and as old as 84 years.20

Although the cause of Brugada syndrome is unclear, the disease appears to be a type of channelopathy that affects transmembrane ion currents and disrupts the cardiac action potential. Many cases are linked to a mutation in the SCN5A sodium channel gene. Patients are prone to developing polymorphic tachycardias that can evolve into ventricular fibrillation and sudden cardiac death. If patients have a self-limiting arrhythmia they will present with near syncope, syncope, or aborted sudden death.20

Patients can present after a syncopal episode or cardiac arrest; however, some patients do not describe any symptoms. Their physical examination, including the cardiovascular examination, is usually

<table>
<thead>
<tr>
<th>Type</th>
<th>J Wave</th>
<th>ST Configuration</th>
<th>T Wave</th>
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<tbody>
<tr>
<td>1</td>
<td>&gt;2 mm</td>
<td>“Coved” type in leads V₁-V₃; ST segment elevated &gt;2 mm</td>
<td>Negative</td>
</tr>
<tr>
<td>2</td>
<td>&gt;2 mm</td>
<td>“Saddleback” ST-T wave; ST segment elevated &gt;1 mm</td>
<td>Positive or biphasic</td>
</tr>
<tr>
<td>3</td>
<td>&gt;2 mm</td>
<td>“Saddleback” ST-T wave; ST segment elevated &lt;1 mm</td>
<td>Positive</td>
</tr>
</tbody>
</table>
Pearls
- ST-segment elevation is found in a variety of conditions and is not always a sign of AMI.
- A chest radiograph will not show evidence of pericardial effusion until the fluid accumulation is more than 200 mL.
- All cases of acute pericarditis should be evaluated with an echocardiogram.
- Consider Brugada syndrome in any patient with right bundle-branch block and J-point elevation of more than 2 mm on ECG.

Pitfalls
- Failure to consider all differential diagnoses in patients with ST-segment elevation.
- Ruling out a thoracic aortic dissection based on a normal mediastinum on chest radiograph.
- Failing to consider aortic dissection in patients with syncope, hemiparesis, extremity paralysis, MI, or abdominal pain (only 60% have any back pain).
- Failing to obtain a 12-lead ECG on syncope patients.
- Failing to admit patients with acute pericarditis who have persistent fever, a large pericardial effusion or possible cardiac tamponade, trauma, or positive troponin, those who are taking anticoagulants, and those who have not responded after 7 days of treatment with nonsteroidal anti-inflammatory drugs.

normal. Brugada syndrome should be considered in all patients with syncope.

There are three different ECG patterns that have been identified as Brugada signs, labeled type 1, type 2, and type 3. All patients will have a right bundle-branch pattern with J-wave elevation of 2 mm or more. The ST-T configurations will vary between the three types (Table 3).20,21

Brugada syndrome is the most common cause of sudden cardiac death in structurally normal hearts; therefore it is essential that an emergency physician recognize these patterns. However, the pattern may be concealed, with only transient ECG abnormalities. The Brugada pattern may be revealed by provoking factors such as febrile illness, hyperkalemia or hypokalemia, hypercalcemia, and alcohol or cocaine use. Also, certain medications increase the risk of arrhythmic events. These include antiarrhythmics (flecainide, procainamide), β-blockers, nitrates, lithium, and psychotropic drugs including tricyclic antidepressants and selective serotonin reuptake inhibitors.22

CRITICAL DECISIONS
What is the appropriate disposition of a patient with Brugada syndrome?

The only treatment proved effective for Brugada syndrome is an implantable cardiac defibrillator (ICD). At this time, there is no proven pharmacologic treatment. Patients seen in the emergency department with suspected Brugada syndrome after an episode of syncope should be hospitalized for cardiac monitoring and evaluation for an ICD placement.

Case Resolutions

Case One
In the case of the woman who presented with chest pain, her ECG showed diffuse ST-segment elevation (Figure 1), and a pericardial friction rub was heard on examination, indicating pericarditis. The physician checked basic laboratory work and cardiac enzymes, which were normal. A cardiologist was consulted, the patient was admitted for 24-hour observation, and an echocardiogram was performed the following morning. The echocardiogram showed a small pericardial effusion, with no structural abnormalities. The cause was presumed to be related to the patient’s recent upper respiratory tract infection. She was started on ibuprofen and had improvement of her symptoms prior to discharge.

Case Two
The emergency physician was appropriately concerned about a dissection in the 62-year-old man with midsternal chest pain. His ECG showed ST-segment elevation in a pattern consistent with a right coronary artery lesion; however, aortic dissection was included in the differential diagnosis because of the patient’s history of sudden ripping chest pain, new diastolic murmur, and asymmetric peripheral pulses. The emergency physician immediately started 2 large-bore intravenous lines and placed the patient on oxygen and cardiac monitoring. The patient was given morphine for pain and started on esmolol and nitroprusside. His pain improved as soon as his systolic blood pressure reached 110. A CT of the thoracic aorta showed an intimal flap involving the ascending aorta with a true and false lumen consistent with a type A dissection. A thoracic surgeon was emergently consulted, and the patient was taken to the operating room where a bypass graft of the coronary artery and aortic repair were performed.

Case Three
In the case of the man brought to the emergency department for syncope, the ECG showed an ST-segment elevation in leads V1 and V2, which the emergency physician recognized as a Brugada pattern (Figure 2). The patient had a type 1 pattern and was considered symptomatic because of his syncopal episode and thus at high risk for sudden cardiac death. The physician
checked electrolytes to rule out any precipitating factors and called the cardiologist. The patient was admitted to the hospital and had no other acute events. After consultation with the cardiologist, the patient received an ICD.

Summary

Chest pain accounts for approximately 5% of emergency department visits, and emergency physicians must consider etiologies ranging from life-threatening to benign conditions. An ECG is a relatively quick and cost-effective tool used by emergency physicians to help differentiate among AMIs, arrhythmias, conduction abnormalities, and electrolyte imbalances. ST-segment elevation requires the physician to quickly determine if the abnormalities are a result of coronary artery occlusion or if they represent a STEMI mimic. Emergency physicians must know the conditions in the differential diagnosis for ST-segment elevation in order to promptly recognize and appropriately treat these various conditions.

References


Correction

Please note that there was an error in the dosage of rocuronium in the February 2011 issue of Critical Decisions in Emergency Medicine. On page 24, the dosing for rocuronium should be as follows:

Dosing
For RSI: 0.6 to 1.2 mg/kg IV X 1
For endotracheal intubation and neuromuscular blockade induction: 0.6 mg/kg VI X 1
For neuromuscular blockade maintenance: 0.01-0.02 mg/kg IV when at 25% of recovery

This will be corrected in our online version.
A 50-year-old man with chest pressure and dyspnea.

Sinus tachycardia, rate 134, inferior myocardial infarction (MI) of uncertain age, T-wave abnormality consistent with inferior and anterior ischemia. Q wave in the inferior leads in association with T-wave inversions in the same leads is suggestive of a recent MI or a previous MI with new ischemia. Review of a previous ECG demonstrated that the Q waves were old but the T-wave inversions were new. Simultaneous T-wave inversions in the inferior and anteroseptal leads should prompt consideration of pulmonary embolism. In this case, the patient was treated for acute cardiac ischemia without resolution of the T-wave abnormality. He had an emergency coronary angiogram that showed no evidence of acute coronary obstruction. Finally, pulmonary embolism was considered; he then underwent computed tomography of the lungs that demonstrated several large pulmonary emboli.

Feature Editor: Amal Mattu, MD, FACEP
The Critical Image

A 30-year-old woman presenting with sudden-onset left flank pain and breathing difficulty. She denied anterior chest or abdominal pain and had no fever, vomiting, cough, or urinary complaints. She had a history of asthma but was otherwise healthy. The patient’s vital signs were blood pressure 108/62, heart rate 79, respiratory rate 18, room air SaO2 98%, and temperature 35°C (95°F). Urine hCG was negative. A chest radiograph was performed, followed by additional imaging.

The patient’s chest radiograph (left) demonstrates an abnormally high gastric air bubble, suggesting herniation of the stomach through a diaphragmatic defect. Normally, the left diaphragm is lower than the right, because of the larger size of the liver relative to the spleen. The stomach is usually visible below the left diaphragm. In this case, the diaphragm is obscured, and the costophrenic angle is not seen. The heart is shifted right by mass effect of herniated organs.

The CT with IV contrast (right) shows a full stomach and spleen in the left hemithorax, with near complete loss of left lung volume. The spleen and stomach enhance normally with IV contrast.

Diaphragmatic hernias can be congenital or acquired through surgery or trauma. Herniation of abdominal contents into the chest results in loss of lung volume and can result in strangulation of abdominal organs and intestinal obstruction. Compression of the pulmonary artery by herniated abdominal organs can result in shunt physiology and respiratory failure resembling pulmonary embolism, and thrombosis of the pulmonary artery can occur. Very rarely, cardiac compression resembling pericardial tamponade occurs.

In a symptomatic patient with diaphragmatic hernia, time-sensitive complications such as gastric or splenic volvulus or bowel obstruction should be considered. Common imaging modalities may be confounded. For example, ventilation-perfusion scan for pulmonary embolism may be nondiagnostic with a matched ventilation-perfusion defect because of lung compression. CT pulmonary angiography may be difficult to interpret because compression of pulmonary vessels can simulate non-filling due to thrombosis. CT with intravenous contrast can assess perfusion of herniated organs, which will not enhance if strangulated. Following a single injected bolus of contrast, images can be acquired first during pulmonary artery filling to assess for pulmonary embolism, and then after a delay of approximately 1 minute to assess for visceral perfusion. Fluoroscopic upper gastrointestinal (GI) series can demonstrate intestinal obstruction. MRI, ultrasonography, and direct visualization with laparoscopy also may be used in diagnosis.

The patient underwent an upper GI series showing normal gastric emptying. As neither visceral strangulation nor pulmonary embolism was detected, she was scheduled for elective repair of her hernia.

CME Questions

Qualified, paid subscribers to Critical Decisions in Emergency Medicine may receive CME certificates for up to 5 ACEP Category I credits, 5 AMA PRA Category 1 Credits™, and 5 AOA Category 2-B credits for answering the following questions. To receive your certificate, go to www.acep.org/criticaldecisionstesting and submit your answers online. You will immediately receive your score and printable CME certificate. You may submit the answers to these questions at any time within 3 years of the publication date. You will be given appropriate credit for all tests you complete and submit within this time. Answers to this month’s questions will be published in next month’s issue.

1. Which of the following indicates a toxic acetaminophen level?
   A. an acute ingestion of 4 grams in an adult
   B. an acute ingestion with a 4-hour acetaminophen level of 95 mcg/mL; the patient is nauseated
   C. an acute ingestion with a 4-hour acetaminophen level of 130 mcg/mL
   D. a chronic ingestion with an acetaminophen level of less than 10 mcg/mL; AST 275 IU/mL
   E. a delayed presentation of an acute ingestion, acetaminophen level of less than 10 mcg/mL; AST 35 IU/mL

2. Which of the following patients should receive NAC for acetaminophen toxicity?
   A. a patient with an acute ingestion who presents longer than 10 hours after ingestion; acetaminophen level 100 mcg/mL
   B. a patient with an acute ingestion with a 4-hour acetaminophen level of 100 mcg/mL
   C. a patient with an acute ingestion with a 6-hour acetaminophen level of 60 mcg/mL; AST 50 IU/mL
   D. a patient with a chronic ingestion with an acetaminophen level of less than 10 mcg/mL; AST 35 IU/mL
   E. a patient with a polypharmacy overdose, unknown ingestants, time of ingestion 2 hours ago

3. Which of the following criteria indicates that a patient should be transferred to a liver transplant center following an isolated acetaminophen overdose?
   A. creatinine is 2 mg/dL
   B. INR is 7
   C. patient is mildly confused
   D. patient is vomiting; creatinine is 2.4 mg/dL
   E. pH is 7.1 prior to fluid resuscitation

4. Which of the following symptoms requires dialysis in the setting of salicylate toxicity?
   A. altered mental status, resolved with urine alkalinization
   B. congestive heart failure
   C. salicylate level 55 mg/dL
   D. serum pH 7.38, creatinine 2 mg/dL
   E. tinnitus

5. Which of the following patients can be safely discharged following a sulfonylurea overdose?
   A. an alert 1-year-old boy with glyburide pill fragments in the mouth
   B. a 2-year-old girl, blood glucose 55 mg/dL, but asymptomatic
   C. a 7-year-old boy, normoglycemic, tolerating oral intake
   D. a 43-year-old man, initial blood glucose 84 mg/dL, tolerating oral intake and who remains normoglycemic after 24 hours of observation
   E. a 56-year-old woman, initially hypoglycemic but now normoglycemic and tolerating oral intake

6. Management of a toxic glyburide ingestion may require which of the following?
   A. bicarbonate infusion
   B. calcium gluconate
   C. dimercaprol
   D. naloxone
   E. octreotide administration

7. Overdose of which of the following may require hemodialysis?
   A. acetaminophen
   B. clonidine
   C. glyburide
   D. propranolol
   E. salicylates

8. Appropriate salicylate poisoning management includes:
   A. avoiding the use of activated charcoal
   B. calcium gluconate for hypocalcemia, which develops due to calcium/hydrogen ion exchange
   C. maintaining appropriate minute ventilations such that the serum pH remains 7.5 to 7.59
   D. maintaining a serum pH of 7.2 to 7.4
   E. octreotide infusion

9. Which of the following is correct regarding sulfonylurea poisoning?
   A. it can present with dizziness, anxiety, palpitations, and diaphoresis
   B. it can present with lethargy and confusion, but never focal neurologic deficits
   C. it is benign in children 5 to 10 years old
   D. it is diagnosed by using urinary antigen testing
   E. patients require more intravenous glucose when octreotide is used initially

10. Which of the following statements is true regarding salicylate poisoning?
    A. confused patients should never receive glucose infusions
    B. the Donde nomogram accurately predicts toxicity with greater than 95% accuracy
    C. salicylates are predominately ionized and excreted in an alkaline urine pH
    D. salicylates are predominantly metabolized by the liver
    E. salicylates cross membranes in their ionized form

11. Which of the following is most common cause of ST-segment elevation in the emergency department?
    A. acute myocardial infarction
    B. early repolarization
    C. left ventricular hypertrophy
    D. left ventricular aneurysm
    E. pericarditis
12. Which of the following is an example of a STEMI mimic that does not typically have a cardiac or vascular involvement?
A. Brugada syndrome
B. hypothermia
C. left bundle-branch block
D. left ventricular hypertrophy
E. pulmonary embolism

13. In acute pericarditis, where is a pericardial friction rub typically best heard?
A. patient seated and leaning forward, over left sternal border
B. patient seated and leaning forward, over right second intercostal space
C. patient seated and leaning forward, over point of maximal impulse
D. patient in supine position, over left sternal border
E. patient in supine position, over right second intercostal space

14. What physical examination findings are consistent with a pericardial tamponade?
A. hypertension, bradycardia, diminished heart sounds
B. hypertension, tachycardia, diminished heart sounds
C. hypotension, bradycardia, diminished heart sounds
D. hypotension, bradycardia, pulsus paradoxus
E. hypotension, tachycardia, diminished heart sounds

15. What is the most important risk factor for aortic dissection?
A. age
B. alcohol abuse
C. diabetes
D. hyperlipidemia
E. hypertension

16. When an aortic dissection propagates retrograde into the coronary arteries what is the most likely ECG finding and what is it a result of?
A. ST-segment elevation in anterior lead, caused by dissection into the left anterior descending coronary artery
B. ST-segment elevation in anterior lead, caused by dissection into the right coronary artery
C. ST-segment elevation in anterior lead, caused by a ventricular aneurysm
D. ST-segment elevation in the inferior leads, caused by dissection into the left anterior descending coronary artery
E. ST-segment elevation in the inferior leads, caused by dissection into the right coronary artery

17. According to the International Registry for Aortic Dissection, up to what percentage of patients with an aortic dissection have a chest radiograph that is initially read as normal?
A. 2%
B. 12%
C. 24%
D. 50%
E. 72%

18. Which of the following is correct regarding aortic dissection?
A. more than half of patients with acute dissection do not have radiating back pain
B. the most common ECG finding during a dissection is ST-segment elevation in leads II, III, aVF
C. a normal mediastinum on chest radiograph rules out an aortic dissection
D. a therapeutic goal is a systolic blood pressure between 130 and 150
E. when treating blood pressure, nitroprusside should be started before a β-blocker to avoid reflex tachycardia

19. Which of the following is correct regarding Brugada syndrome?
A. ECG findings show coved-type ST-segment elevations in leads V1-V6
B. ICD implantation is the only proven treatment to prevent sudden cardiac death
C. it is a channelopathy with mutation in the potassium channel gene
D. on autopsy, patients have a structurally abnormal heart
E. patients may be treated with flecainide

20. Which of the following most accurately describes ECG findings in type I Brugada syndrome?
A. left bundle-branch pattern with 2-mm elevation in lead V1-V3 and coved ST-T configuration
B. left bundle-branch pattern with 2-mm elevation in lead V4-V6 and coved ST-T configuration
C. right bundle-branch pattern with diffuse ST-segment elevation in limb and precordial leads
D. right bundle-branch pattern with 2-mm elevation in lead V1-V3 and coved ST-T configuration
E. right bundle-branch pattern with 2-mm elevation in lead V4-V6 with coved ST-T configuration

Change to CME Processes
Beginning with the July 2011 issue of Critical Decisions in Emergency Medicine, qualified subscribers who take the monthly posttest and submit their answers must make a score of 70% or better in order to receive a printable CME certificate.

Answer key for February 2011, Volume 25, Number 6

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
| A | B | D | E | B | A | C | C | B | C | E | A | D | E | A | B | E | C | E | E |

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Activated Charcoal

Ryan Squier, MD, Akron General Medical Center

Activated charcoal is often used in the emergency department as an adjunct treatment for overdoses and to prevent the absorption of orally ingested poisons or toxins. Clinical benefit is more likely if it is given within 1 hour of ingestion. Note that activated charcoal should not be used in cases of cyanide, mineral acids, caustic alkalis, organic solvents, iron, ethanol, methanol, or lithium ingestion. Although it may be used as an adjunct with a cathartic, it is not routinely recommended. In certain instances, multiple doses of activated charcoal may be considered; however, this has not been proven to improve clinical outcomes.

Activated Charcoal

Mechanism of Action
Porous material that absorbs toxins and gases in the gastrointestinal (GI) tract, helping with clearance and preventing absorption by the body. Potential for interruption of enterohemorrhagic and enteroenteric circulation

Indications
Acute poisonings and oral ingestion of toxic substances
Over-the-counter indications include irritable bowel syndrome, diarrhea, indigestion, and flatulence

Dosing
Adults: 1 g/kg orally (25-100 grams); 12.5 g/hr for multiple doses
Pediatrics: 1-12 years: 0.5-1 g/kg orally (25-50 grams)
<1 year old: 0.5-1 g/kg orally (10-25 grams)
0.25 g/kg/hr for multiple doses
EMS: Typically 12.5- to 25-gram doses premixed with water
Approximately 10 grams of activated charcoal is required for every 1 gram of toxin ingested

Side Effects
Vomiting with risk of aspiration, constipation/impaction
If given with cathartics—diarrhea, hyperternatemia, hypermagnesemia

Contraindications/Precautions
Contraindications: cyanide, acid, alkali, organic solvent, iron, ethanol, methanol, or lithium ingestion; GI tract not intact; risk of hemorrhage or GI perforation; airway not protected
Caution: some agents used for flavoring may reduce efficacy. If used with ipecac, ensure that vomiting has subsided prior to administration of activated charcoal
Pregnancy category C

Feature Editors: Michael S. Beeson, MD, MBA, FACEP; Steven Warrington, MD