Ethanol and Opioid Intoxication and Withdrawal

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KEY POINTS

- Most organ systems in the body can be affected by ethanol consumption. Important associated disease states are electrolyte disturbances, traumatic injuries, infectious diseases, and primary central nervous system, gastrointestinal, and cardiovascular complications.
- Ethanol causes depressant effects, but abrupt cessation in long-term users causes hyperstimulation and dangerous withdrawal syndromes.
- Alcohol withdrawal is a spectrum of diseases ranging from minor signs and symptoms, such as anxiety and mild tremor, to severe withdrawal, including autonomic instability and delirium.
- Supportive care is the mainstay of treatment for acute ethanol intoxication and withdrawal. Benzodiazepines constitute the major form of pharmacotherapy for withdrawal syndromes.
- For admitted patients, underlying liver disease, need for intubation, hyperthermia, persistent tachycardia, and use of physical restraints are all associated with increased risk of death in alcohol withdrawal.
- Patients who have a history of major withdrawal and are currently in withdrawal or have significant associated disease states should be admitted for further treatment.
- Brief interventions in alcohol-dependent patients in the emergency department have been shown to have positive effects.
- Opioid intoxication is characterized by depressed central nervous system activity, respiratory depression, and miosis.
- Patients with opioid withdrawal syndrome can present with yawning, piloerection, and mydriasis.

ETHANOL

EPIDEMIOLOGY

Ethanol use is a common part of our society, as evidenced by the knowledge that approximately 80% of adults in the United States have consumed ethanol-containing beverages during their lifetimes. Mild to moderate consumption (up to one drink/day for women and two drinks/day for men) has been shown to have beneficial cardiovascular effects, including a decreased risk of myocardial infarction and stroke, as well as overall decreased mortality (Box 154.1).

Despite these possibly beneficial effects of alcohol, it has been found to be a top 10 cause of preventable deaths among all age groups in the United States. Additionally, approximately 9% of adults meet the diagnostic criteria for alcohol abuse and alcoholism. This maladaptive behavior can lead to numerous individual medical complications and societal problems, including motor vehicle collisions, assaults, homicide, suicide, and domestic violence. An estimated 7.6 million emergency department (ED) visits per year are for alcohol-related diseases and diagnoses.

Alcohol withdrawal is seen in the ED in various forms and stages, including early withdrawal, hallucinosis, seizures, and fully developed delirium tremens (DT). DT, a severe withdrawal syndrome defined by the presence of tremors, seizures, and delirium, develops in 5% of patients who develop symptoms of alcohol withdrawal and itself carries a 5% to 15% risk of mortality. Among patients admitted to the hospital with a diagnosis of alcohol withdrawal, the following clinical features have been found to be associated with an increased risk of death: underlying liver disease, the need for endotracheal intubation, hyperthermia, persistent tachycardia, and the use of physical restraints.

PATHOPHYSIOLOGY

ALCOHOL INTOXICATION

Ethanol is readily absorbed from the gastrointestinal (GI) tract and is primarily metabolized by the liver through the alcohol dehydrogenase pathway (Fig. 154.1). Metabolism of ethanol differs in men and women. Although alcohol dehydrogenase is found in gastric mucosa and other tissues, women seem to have less ability to metabolize it by the gastric route. Long-term ethanol users or those with high alcohol levels also use a second pathway, the microsomal ethanol-oxidizing system.

Ethanol is a central nervous system (CNS) depressant involving multiple receptors and pathways. Likely its greatest effect is in enhancing gamma-aminobutyric acid (GABA) inhibitory action. Ethanol is also known to block the excitatory N-methyl-D-aspartate (NMDA) glutamate receptor, thus leading to further CNS depression.

The level of CNS depression depends on many factors affecting absorption and elimination, including age, weight,
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gender, the presence of food, gastric motility, the speed of consumption, and long-term alcohol use. Ethanol intoxication in most states is legally defined as a blood alcohol concentration (BAC) of 80 to 100 mg/dL (0.08 to 0.1% BAC). Elimination rates vary greatly, but a rate of 20 mg/dL/hour can be assumed for most intoxicated patients in the ED, regardless of initial alcohol level or chronic alcohol use.\textsuperscript{16,17} Because alcohol follows zero-order kinetics, some sources advocate drawing two ethanol levels to determine the individual patient’s exact rate of ethanol clearance, although this is most often medically unnecessary.

**ALCOHOL WITHDRAWAL**

Alcohol withdrawal is best described as a pathologic excitation of the CNS and autonomic systems. GABA receptors are desensitized and downregulated in chronic ethanol use, with a resulting decrease in activity of the inhibitory effects of GABA when a patient reduces ethanol consumption. The excitatory glutamate neurotransmitter system is blocked by the NMDA receptor in the presence of ethanol, and this blockade leads to receptor upregulation in chronic alcoholism and excitation during withdrawal.\textsuperscript{17} With repeated episodes of alcohol withdrawal, the patient will have more severe withdrawal, a phenomenon known as “kindling.”\textsuperscript{18} Cessation of alcohol consumption may be inadvertent, as in the patient who is unable to tolerate oral intake because of vomiting or in the hospitalized patient whose access to ethanol is restricted.

**PRESENTING SIGNS AND SYMPTOMS**

Ethanol use is associated with many disease states affecting many organ systems in the body. The Wernicke-Korsakoff syndrome bears special mention. This syndrome complex is composed of two disease processes, Wernicke encephalopathy and the Korsakoff amnestic state, which can manifest individually or concomitantly.

Classically, Wernicke encephalopathy consists of the following: ocular abnormalities such as nystagmus and motor palsies, seen in 29% of cases; ataxia, seen in 23% of cases; and mental status change, seen in 82% of cases. Presentations with this classic triad are rare, with only 10% of confirmed cases having all three symptom types.\textsuperscript{19}

The Korsakoff amnestic state refers to the syndrome of memory deficits found in long-term alcohol abusers. Anterograde and retrograde amnesia is present, and confabulation is common. Thiamine deficiency is the cause, and ataxia and memory loss may persist despite treatment.

The CNS-depressive effects of ethanol range from diminished fine-motor control to coma and respiratory depression. Because of greater tolerance, patients with chronic alcoholism may exhibit a high level of functioning despite a high BAC. The intoxicated patient often presents with the smell of ethanol on the breath, slurred speech, emotional lability, and difficulty with coordination. Death can occur from respiratory depression or aspiration.

Alcohol is related to an estimated 35% of injury-associated ED visits.\textsuperscript{20} In the setting of trauma, ethanol intoxication generally should not lower the Glasgow Coma Scale score dramatically; whenever a low score is found, further CNS evaluation is warranted.\textsuperscript{21}

**ALCOHOL WITHDRAWAL**

The patient in ethanol withdrawal usually presents to the ED approximately 24 hours after a significant decrease or cessation of ethanol consumption. The patient is anxious, tremulous, tachycardic, hypertensive, and hyperreflexic and may complain of sleep and GI disturbances. Alcohol withdrawal syndrome represents a spectrum of disease, ranging from minor to major. An accompanying time frame within this spectrum of disease shows significant

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**BOX 154.1 Definition of One Standard Alcoholic Drink**

A standard alcoholic drink can be defined as one of the following:
- 12 fluid oz. regular beer
- 5 fluid oz. wine
- 1.5 fluid oz. 80-proof distilled spirits


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![Fig. 154.1 Alcohol dehydrogenase pathway, including the microsomal ethanol-oxidizing system (MEOS), the alternative metabolism seen in chronic alcoholics. NAD\(^+\), nicotinamide adenine dinucleotide; NADH, reduced nicotinamide adenine dinucleotide; NADP, nicotinamide adenine dinucleotide phosphate; NADPH, reduced nicotinamide adenine dinucleotide phosphate.](image-url)
overlap between timing and the signs and symptoms. Minor withdrawal begins within 6 to 24 hours and is characterized by hyperstimulation. Major withdrawal syndrome begins after 24 hours and peaks at approximately 48 to 72 hours. It may have any or all of the following features: progression of excitatory signs and symptoms, hyperpyrexia, seizures, altered mental status, hallucinations (visual, auditory, or tactile), and delirium.

True DT is rare and constitutes the most severe form of withdrawal, although patients may mistakenly equate it with generalized withdrawal syndrome. DT occupies the far end of the spectrum, and it consists of substantial tremor, autonomic hyperactivity, profound confusion, fever, and hallucinations (Fig. 154.2).22

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The diagnosis of ethanol intoxication is mainly one of exclusion, and a history consistent with ethanol consumption is important. The initial approach to the patient should be the same as for any patient with altered mental status. Traumatic injuries and coingestions (acetaminophen, illicit drugs, toxic alcohols) should be high on the differential diagnosis list (Box 154.2).

**BOX 154.2 Differential Diagnosis of Ethanol Intoxication and Withdrawal**

**Intoxication**
- Traumatic head injury
- Cerebrovascular accident
- Metabolic derangements (hypoglycemia)
- Hypoxia
- Drug ingestion
- Central nervous system infections

**Withdrawal**
- Infections (meningitis, encephalitis, sepsis)
- Toxidromes (psychomimetic, anticholinergic)
- Thyrotoxicosis
- Neuroleptic malignant syndrome
- Heat stroke
- Acute psychosis
- Withdrawal from other sedative-hypnotic drugs (benzodiazepines, barbiturates) and from drugs used to treat spasticity (baclofen)

A history of previous ethanol withdrawal or of alcohol abuse with decreased intake or cessation of alcohol is the key to the diagnosis of ethanol withdrawal.

Diagnostic testing for the acutely intoxicated patient should be guided by suspicion of concomitant disease states and potential traumatic injury. A BAC measurement is necessary only to confirm a diagnosis or to guide treatment.

Patients presenting in withdrawal may mandate a comprehensive evaluation, but the same guidelines apply as for the intoxicated patient. The laboratory tests vary but may include a complete blood count, serum glucose measurement, blood chemistry panel with a full set of electrolyte measurements, urinalysis, toxicology screen, electrocardiogram, chest radiography, and head computed tomography. Lumbar puncture for cerebrospinal fluid analysis may be indicated if subarachnoid hemorrhage or CNS infection is in the differential diagnosis.

**TREATMENT**

**ALCOHOL INTOXICATION**

Supportive care is the mainstay of treatment for acute ethanol intoxication (Fig. 154.3). Airway and breathing must be assessed in the comatose patient, and endotracheal intubation, although rarely needed, should be used for airway protection if necessary. Circulation should be assessed, and isotonic intravenous fluids should be given initially for patients with hypotension or volume depletion.

In the comatose patient, naloxone (0.8 mg) should be considered, and glucose (25 to 50 g intravenously) should be given to a hypoglycemic patient. Thiamine (100 mg intravenously) can be given before glucose administration to prevent or treat Wernicke encephalopathy, but glucose administration need not be delayed. Electrolyte and thiamine replacement can be achieved orally if the patient is tolerating oral intake, is not at risk of aspiration, and is not being treated for active Wernicke encephalopathy. Routine multivitamin replacement with vitamin B12 and folate in patients presenting with alcohol intoxication is unnecessary.

**ALCOHOL WITHDRAWAL**

Patients who present to the ED with signs and symptoms of alcohol withdrawal should be evaluated using the Revised Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar) scale to aid in determining the severity of withdrawal (Box 154.3). Initial treatment should focus on resuscitation with fluids, replacement of electrolyte deficiencies, and evaluation and treatment of concomitant diseases.

Benzodiazepines are the mainstays of treatment for withdrawal and are usually initiated when CIWA-Ar scores are
The patient with uncomplicated ethanol intoxication may be discharged home after an evaluation for associated disease states if the following criteria are met: (1) the patient is not at risk of airway or breathing complications; and (2) a responsible, sober adult is able to monitor the patient for the next 24 hours. Otherwise, the patient should be monitored in the ED until he or she is clinically and legally sober.

Patients with major ethanol withdrawal require admission and may need an intensive care unit setting. Patients with minor withdrawal may be discharged after observation for 4 to 6 hours if mental status, vital signs, and laboratory study results remain within normal limits. A short course of benzodiazepines can be considered for the patient undergoing outpatient detoxification.

Referral to an outpatient treatment program is appropriate for all patients being discharged who are recognized as having a substance abuse disorder. Options include the use of inpatient versus outpatient treatment programs and referral to Alcoholics Anonymous (AA). Because of the high incidence of underlying social and psychiatric problems, referral to the ED’s social or psychiatric worker (if available) may also be helpful.

Patients presenting to the ED with alcohol-related issues should be screened for alcohol abuse and dependence. For patients thought to have or to be at risk for alcohol dependence, brief interventions have been shown to decrease at-risk drinking in the short term and provide an opportunity to provide information on long-term follow-up. The brief intervention is described as a four-step conversation with the patient and consists of the following: (1) broach the subject,
### SECTION XV  TOXICOLOGIC EMERGENCIES

#### EPIDEMIOLOGY

Opioids are a class of drugs that comprise natural, semisynthetic, and synthetic substances that provide analgesic and anesthetic properties by acting at opioid receptors. Box 154.4 gives the definition of terms associated with opioids. Box 154.4 gives the definition of terms associated with opioids. Between 2004 and 2008, the number of ED visits for nonmedical uses of prescription opioids increased by 111% and now is equivalent to the number of visits associated with illicit drugs. This change parallels the marked increase in opioid prescription rates seen since 2000; approximately 4 million patients in the United States are receiving long-acting, long-term opioid therapy.

Since 2002, opioids have become the leading cause of death from unintentional drug overdose in the United States, and they account for more deaths than heroin and cocaine combined. Opioid overdose can occur in several situations, including intentional self-injury, unintentional prescription, recreational or pediatric ingestion, and drug packing and stuffing.

#### PATHOPHYSIOLOGY

Opioids as a medical class are defined by their agonist activity at opioid receptors, of which µ, κ and δ are the best described. Opioid receptor activity in the CNS, spinal cord, and GI system has widespread effects, including analgesia, anesthesia, euphoria, decreased GI motility, respiratory depression,
and miosis. Although the many opioids undergo varying types of initial conversion, all opioids undergo hepatic metabolism and renal elimination. These factors should be considered when caring for patients with hepatic and renal dysfunction.

Long-term opioid use can result in physiologic tolerance and dependency through changes in opioid receptor structure, receptor trafficking, and mechanism of action. Withdrawal can be precipitated in opioid-dependent individuals who decrease their intake or are given an opioid receptor antagonist such as naloxone. The timing of withdrawal symptoms depends on the variable half-life of the particular opioid.

Considered the opioid overdose antidote, naloxone is a competitive receptor antagonist that can reverse opioid activity and toxicity. Naloxone is used to reverse the dangerous consequence of respiratory depression in opioid overdose and can be given by various routes, including intravenous, subcutaneous, intramuscular, endotracheal, intranasal, and nebulized. Naloxone is not effective when given orally because of first-pass metabolism.

**PRESENTING SIGNS AND SYMPTOMS**

Respiratory depression leading to apnea is the primary life-threatening presentation of opioid overdose. Because respiratory depression is reliably accompanied by altered mental status or coma, a history of opioid use is often not readily available and should be considered in patients who are found unconscious and who have a decreased respiratory rate or miosis. Patients with milder opioid intoxication may present with nausea, vomiting, constipation, miosis, depressed CNS level, and depressed respiratory status. **Table 154.1** contains a more extensive list of these symptoms.

The patient presenting with acute opioid overdose must be evaluated for additional emergency diagnoses, including trauma, infection (particularly in patients who inject opioids), coingestion, electrolyte abnormalities, and complications of prolonged immobility such as rhabdomyolysis, compartment syndrome, and mononeuropathies.

Certain opioids have unique toxicities that must be considered during ED evaluation (**Table 154.2**). Methadone, an agent used primarily for addiction therapy, is very long acting, with a half-life of more than 24 hours, and can cause prolongation of the QTc interval and torsades de pointes. Propoxyphene, tramadol, and meperidine may cause seizures, even in therapeutic doses. Propoxyphene was taken off the market because of its tricyclic antidepressant–like sodium channel activity and association with wide complex tachyarrhythmias and negative inotropy, even at therapeutic doses. Fentanyl, particularly when given as a rapid injection, can cause chest wall rigidity that can be difficult to manage, even with naloxone and endotracheal intubation.

Noncardiogenic pulmonary edema is an uncommon complication of opioid overdose characterized by hypoxia despite resolution of altered mental status and bradypnea, production of frothy pink sputum, and chest radiograph evidence of diffuse pulmonary infiltrates. Opioid-related pulmonary edema is short-lived and infrequently requires intubation, but it mandates admission to the hospital until resolution of symptoms and hypoxia. In the alert patient, noninvasive ventilation can be considered for improved oxygenation.

Patients with opioid withdrawal syndrome present with similarly varied symptoms, but as a rule they appear uncomfortable. Their symptoms may include yawning, rhinorrhea, mydriasis, piloerection, nausea, vomiting, diarrhea, myalgias, and abdominal pain. An acute withdrawal syndrome is seen after the administration of naloxone, particularly in patients with long-term opioid use with dependence.

Vital signs may include tachycardia, normal blood pressure to hypertension, and tachypnea. Another common picture of...
opioid withdrawal is seen in the patient who has cancer or uses an opioid for chronic pain and who misses a dose of medication. Such a patient presents to the ED with nausea, vomiting, and abdominal cramping. The history usually uncovers missed opioid doses.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The differential diagnosis of mild opioid intoxication should include diagnoses that cause altered mental status and hypoventilation: hypoglycemia, head injury, and overdose of other medications (alcohols, benzodiazepines, barbiturates, tricyclic antidepressants). The differential diagnosis for the patient who is ill secondary to opioid overdose is similar to that for the patient in coma; infection, cerebrovascular accident, head trauma, and other overdoses should be considered (Box 154.5).

Diagnostic testing in opioid overdose usually does not guide treatment, given that the antidote is administered before test results are available. Tests are used to evaluate for complications of opioid toxicity including arrhythmias, acute lung injury, pulmonary edema, and comorbid diseases. Electrocardiograms and chest radiographs can be useful as adjuncts in these cases. Because intravenous drug abusers are prone to numerous and severe infections, the presence of fever or persistent altered mental status in this population should prompt a rapid and broad work-up.

Urine drug testing does not guide treatment in the acute setting of opioid overdose. Opioids can be detected for up to 36 hours in the urine, although false-positive results have been

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**Table 154.1** Opioid Intoxication and Withdrawal Signs and Symptoms by Organ System

<table>
<thead>
<tr>
<th>ORGAN SYSTEM</th>
<th>INTOXICATION</th>
<th>WITHDRAWAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central nervous system</td>
<td>Depression of activity</td>
<td>Excitation, restlessness, anxiety, seizures</td>
</tr>
<tr>
<td></td>
<td>Respiratory depression</td>
<td>(rare)</td>
</tr>
<tr>
<td></td>
<td>Increased parasympathetic activity</td>
<td>Tachypnea</td>
</tr>
<tr>
<td>Head and neck</td>
<td>Miosis (pinpoint pupils)</td>
<td>Mydriasis</td>
</tr>
<tr>
<td></td>
<td>Antitussive effect</td>
<td>Rhinorrhea</td>
</tr>
<tr>
<td>Cardiovascular system</td>
<td>Hypotension to normal blood pressure</td>
<td>Normal blood pressure to hypertension</td>
</tr>
<tr>
<td></td>
<td>Bradycardia to normal heart rate</td>
<td>Normal heart rate to tachycardia</td>
</tr>
<tr>
<td>Gastrointestinal tract</td>
<td>Constipation</td>
<td>Diarrhea</td>
</tr>
<tr>
<td></td>
<td>Nausea and vomiting</td>
<td>Nausea and vomiting</td>
</tr>
<tr>
<td>Genitourinary tract</td>
<td>Sphincter constriction/spasm</td>
<td>Sphincter relaxation</td>
</tr>
<tr>
<td>Musculoskeletal system</td>
<td>Relaxation and flaccidity</td>
<td>Myalgias</td>
</tr>
<tr>
<td>Psychiatric manifestations</td>
<td>Euphoria or dysphoria</td>
<td>Drug craving</td>
</tr>
</tbody>
</table>

**Table 154.2** Specific Opioid Toxicities

<table>
<thead>
<tr>
<th>COMPOUND</th>
<th>TOXICITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>Acute lung injury</td>
</tr>
<tr>
<td>Meperidine</td>
<td>Seizures</td>
</tr>
<tr>
<td>Methadone</td>
<td>QTc prolongation, torsades de pointes</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Chest wall rigidity</td>
</tr>
<tr>
<td>Propoxyphene</td>
<td>QRS prolongation, seizures</td>
</tr>
<tr>
<td>Tramadol</td>
<td>Seizures</td>
</tr>
</tbody>
</table>


**BOX 154.5** Differential Diagnosis for Opioid Overdose

- All components of differential diagnosis for a patient who is unconscious or in a coma
- Alcohol (ethanol, isopropyl, ethylene glycol, methanol) intoxication or overdose
- Barbiturate overdose
- Benzodiazepine overdose
- Clonidine overdose
- Tricyclic antidepressant overdose
- Infection
- Head trauma
- Cerebrovascular accident
found after ingestion of poppy seeds. The urine drug screen is also poorly sensitive for detecting use of synthetic opioids, including methadone, fentanyl, hydromorphone, hydrocodone, and oxycodone. Acetaminophen, salicylate, and ethanol measurements should be included in the evaluation for unknown ingestions. A serum chemistry panel and complete blood count can be helpful in the broader evaluation of the sick opioid-toxic patient. The serum creatinine kinase level may be elevated in patients with prolonged immobility and rhabdomyolysis. Many patients awaken after treatment with naloxone, admit to opioid overdose, and may not require any further testing.

The diagnosis of opioid withdrawal can usually be obtained based on history and examination findings. However, because patients with opioid withdrawal can appear systemically ill, other emergency diagnoses such as infection, serotonin syndrome, and sympathomimetic or cholinergic toxicity should be considered.

Diagnostic testing in patients undergoing opioid withdrawal is guided by ruling out other causes of the presenting signs and symptoms. A comprehensive chemistry panel is useful when the patient has massive vomiting and diarrhea.

**TREATMENT**

Treatment of opioid intoxication and overdose should begin with assessment of the airway, breathing, and circulatory status of the patient. Airway adjuncts such as an oral or nasal airway can be used to improve the viable airway. In patients with a decreased respiratory rate or apnea, bag-valve-mask ventilation may be necessary before intubation or naloxone administration.

Rapid administration of naloxone may preclude the need for intubation. If signs and symptoms are consistent with opioid intoxication, the antidote should be given immediately while preparations are being made for intubation. Naloxone should be administered intravenously in apneic patients, with starting doses of 0.4 to 1 mg and 2 mg in those with cardio-pulmonary arrest. Naloxone can be repeated to reach the desired effect of increased respiratory rate. A higher dose of naloxone may be required for certain naloxone-resistant opioids (e.g., fentanyl, methadone, propoxyphene). Naloxone should take effect in minutes and has a duration of action between 20 and 90 minutes. Repeat dosing or continuous infusion may be necessary for patients who have ingested long-acting opioids such as methadone and extended-release formulations.

Hypotension should be treated with intravenous fluids according to resuscitation protocols. Blood glucose levels should be checked at the bedside, and patients with hypoglycemia should receive dextrose. Care should be taken to reevaluate the patient frequently and observe him or her for a return of respiratory depression.

Activated charcoal administration may be considered only in awake or intubated patients if they had a known recent oral ingestion and especially with coingestions. Opioid-induced seizures may respond to oxygen and naloxone administration. Seizures that do not respond to naloxone may be treated with benzodiazepines. Refractory seizures should prompt investigation of a complicating or additional process such as body packing, head trauma, or other ingestions. Hemodialysis is not indicated in opioid overdose because of the large and variable volume of distribution of these agents in the body.

Treatment of opioid withdrawal in the ED is aimed at stabilization of cardiopulmonary status and symptomatic therapy. Opioid replacement should be guided by the cause of the withdrawal: cessation of prescription medications, methadone
therapy for addiction, and decreased recreational intake. Administering missed doses of opioids and methadone replacement (20 mg orally or 10 mg intramuscularly) can be used to reverse withdrawal without overdose. Clonidine (0.1 to 0.3 mg orally every hour or in a sustained-release patch) can also help with high blood pressure and decrease withdrawal symptoms. Benzodiazepines can be used to aid in sedation and to temper withdrawal symptoms. Antiemetics can be given to the patient with persistent nausea and vomiting.

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uncomplicated opioid overdose can be monitored in the ED for 2 to 4 hours after reversal because the half-life of most opioids is in this range. However, patients who present with overdose from opioids with long half-lives, such as methadone, should be admitted to the hospital for continued airway monitoring. Depending on the type of opioid, the route of administration, and the amount taken, additional doses of naloxone may be required to keep the patient from experiencing opioid reintoxication.

Any patient who requires a second dose of naloxone should be observed for an extended time, and intensive care unit admission should be considered. Patients with complicated opioid overdoses requiring respiratory assistance and those with severe toxicity must be admitted to the hospital’s critical care unit.

Unlike alcohol withdrawal, opioid withdrawal is not life-threatening. Most patients may be discharged for outpatient treatment.

SUGGESTED READINGS


REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.

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RED FLAGS

• Incorrectly assuming the patient is intoxicated
• Not suspecting ethanol abuse in an older patient
• Not recognizing concomitant head injury, intoxication, or associated diseases
• Not aggressively treating signs and symptoms of withdrawal
• Inappropriately discharging an acutely intoxicated patient
• Not managing the airway in a timely manner
• Not evaluating for other causes, including head trauma, infection, and cerebrovascular accident, after repeated doses of naloxone or continued altered consciousness in the patient with suspected intoxication or opioid overdose
• Not considering opioid withdrawal in patients with cancer and in other patients with long-term opioid use and treating appropriately

FOLLOW-UP, NEXT STEPS OF CARE, AND PATIENT EDUCATION

Patients with opioid intoxication can probably be discharged from the ED after observation and evaluation of any active comorbid diseases at presentation. Patients with