Last Call For Emergency Medicine-3

Editör

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FOREWORD

Dear readers;

Emergency medicine is a medical discipline where time is of the essence, uncertainty is high, and decisions must be made in seconds. The dynamic nature of clinical practice necessitates that emergency medicine be constantly updated with the latest information, a multifaceted perspective, and an interdisciplinary approach. Today, with increasing patient loads, changing disease profiles, and environmental and technological factors, the role of emergency departments is becoming more critical every day.

This book has been prepared to address clinical situations frequently encountered in emergency medicine practice but requiring a high level of knowledge and awareness for management. The chapters cover a broad and up-to-date range of topics, including patient management in the emergency department. In this respect, the book aims to contribute directly to clinical practice and serve as an academic reference source. Each chapter has been written in line with evidence-based medicine principles, in light of current literature, and taking into account real clinical scenarios encountered in the emergency department. Thus, the aim is for the reader to access not only theoretical knowledge but also practical, actionable, and guiding content.

Based on the fact that emergency medicine is a discipline practiced not only within hospital walls but also in the field, in ambulances, and in air and sea vehicles, this work also gives special attention to pre-hospital emergency health services and the challenges faced by health professionals working in this field. The inclusion of topics such as stress, burnout, and performance aims to highlight the human and professional dimensions of emergency medicine practice.

We hope this work will be a useful resource for emergency medicine specialists, emergency medicine residents, general practitioners, and all healthcare professionals working in pre-hospital emergency medical services.

In emergency medicine practice, where knowledge grows when shared and accurate information saves lives, we hope this book will contribute to our colleagues...

Editor

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İÇİNDEKİLER

CHAPTER 1

Systemic and Pulmonary Hypertension in Emergency Medicine

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Ufuk Özgür BALICA[2]

1. Introduction

Hypertension is a common clinical problem encountered in emergency medicine. Acute elevations in blood pressure (BP) may represent either a transient physiological response or a life-threatening condition requiring immediate intervention. Differentiating between hypertensive emergencies and non-emergent presentations is essential for appropriate management and prevention of iatrogenic harm.

2. Classification of Acute Systemic Hypertension

Acute systemic hypertension is best categorized based on the presence or absence of acute target organ injury rather than absolute BP values.

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2.1 Hypertensive Emergency

A hypertensive emergency is defined as a marked elevation in BP accompanied by acute or ongoing target organ damage. Common clinical scenarios include aortic dissection, acute pulmonary edema, acute coronary syndromes, hypertensive encephalopathy, intracranial or subarachnoid hemorrhage, acute ischemic stroke, acute renal failure, severe preeclampsia or eclampsia, and sympathomimetic crises. These conditions require prompt recognition and controlled BP reduction, with therapeutic targets tailored to the underlying pathology.

2.2 Hypertensive Urgency

Hypertensive urgency refers to severe BP elevation in the absence of progressive or acute target organ dysfunction. Although thresholds such as ≥180/110 mm Hg are frequently cited, management decisions should be guided by clinical context rather than numeric criteria alone. Immediate BP reduction is generally unnecessary, and treatment may be initiated with oral agents and close outpatient follow-up.

Accurate BP measurement is critical. Inappropriate cuff size, particularly the use of a cuff that is too small, can result in falsely elevated readings.

3. Clinical Assessment

A focused but thorough history and physical examination are central to evaluation. Key historical elements include known hypertension, medication adherence, cardiovascular, renal, or cerebrovascular disease, diabetes mellitus, dyslipidemia, chronic lung disease, and family history of hypertension.

Potential precipitating factors such as pregnancy, recreational drug use (e.g., cocaine or methamphetamines), and sympathomimetic

medications should be identified. Symptoms suggestive of target organ involvement include neurologic complaints (headache, visual disturbances, seizures, confusion), cardiovascular symptoms (chest pain, dyspnea, palpitations, syncope), and renal manifestations (oliguria, hematuria, edema).

Physical examination should assess for neurologic deficits, altered mental status, papilledema, retinal hemorrhages or exudates, cardiac murmurs or gallops, pulmonary rales, asymmetric pulses or BP measurements, and abdominal or carotid bruits. In pregnant or postpartum patients, hyperreflexia and peripheral edema may suggest preeclampsia.

4. Diagnostic Evaluation

Diagnostic testing should be individualized based on presenting features. Urinalysis is a cost-effective screening test for renal involvement and may reveal proteinuria, hematuria, or casts. Serum studies can identify renal dysfunction and electrolyte abnormalities.

Electrocardiography may demonstrate ischemic changes or left ventricular hypertrophy. Chest radiography can aid in the evaluation of heart failure or suspected aortic pathology. Neuroimaging is indicated for patients with focal neurologic deficits or altered consciousness. Drug screening and pregnancy testing should be performed when clinically indicated.

5. Emergency Department Management

Patients presenting with hypertensive emergencies require immediate oxygen supplementation, continuous cardiac monitoring, and intravenous access. Following stabilization of airway, breathing, and circulation, blood pressure reduction should be gradual and tailored to the specific clinical scenario.

1. Aortic dissection: Initial management prioritizes reduction of myocardial contractility and aortic shear stress. Heart rate should be reduced to approximately 60 beats/min, and systolic blood pressure should be lowered below 140 mm Hg, ideally to 100–120 mm Hg if tolerated. Recommended first-line agents include esmolol (300 micrograms/kg IV bolus followed by 50 micrograms/kg/min infusion) or labetalol (20 mg IV over 2 minutes, followed by 20–40 mg IV every 10 minutes as needed, up to a maximum of 300 mg). If beta-blockers are contraindicated, verapamil 5–10 mg IV or diltiazem 0.25 mg/kg IV over 2 minutes may be used for heart rate control. Vasodilators may be added after adequate rate control. Nicardipine infusion should be initiated at 5 mg/h and titrated by 2.5 mg/h every 5–15 minutes to a maximum of 15 mg/h. Alternatively, nitroprusside may be started at 0.3–0.5 micrograms/kg/min and titrated in 0.5 micrograms/kg/min increments.

2. Acute hypertensive pulmonary edema: Blood pressure reduction should not exceed 20–30% acutely. Nitroglycerin is the preferred initial agent and may be administered sublingually at 0.4 mg (up to three doses), as topical paste (1–2 inches), or as an intravenous infusion starting at 5 micrograms/min and titrated to a maximum of 200 micrograms/min. Alternative therapies include enalaprilat 0.625–1.25 mg IV over 5 minutes every 4–6 hours (maximum 5 mg every 6 hours), nicardipine, or nitroprusside.

3. Acute coronary syndrome: In patients with systolic blood pressure exceeding 160 mm Hg, acute reduction should be limited to no more than 20%. Initial therapy includes nitroglycerin or metoprolol, administered orally at 50–100 mg every 12 hours or intravenously at 5 mg every 5–15 minutes to a maximum of 15 mg. Intravenous beta-blockers should be avoided in patients at risk for cardiogenic shock.

4. Acute sympathetic crisis: Management should focus on symptom relief, beginning with benzodiazepines. Additional agents include nitroglycerin or phentolamine administered as a 5–15 mg IV bolus.

5. Acute renal failure: For blood pressure exceeding 180/110 mm Hg, reduction should not exceed 20% acutely. Recommended agents include labetalol, nicardipine, or fenoldopam initiated at 0.1 micrograms/kg/min and titrated every 15 minutes to a maximum of 1.6 micrograms/kg/min.

6. Preeclampsia: For blood pressure greater than 160/110 mm Hg, labetalol is recommended. Hydralazine 5–10 mg IV may be used as an alternative, though its effects may be less predictable.

7. Hypertensive encephalopathy: For blood pressure above 180/110 mm Hg, gradual reduction not exceeding 20% is advised. Appropriate agents include nicardipine, labetalol, fenoldopam, or nitroprusside.

8. Subarachnoid hemorrhage: To minimize the risk of rebleeding, systolic blood pressure should be maintained below 160 mm Hg or mean arterial pressure below 130 mm Hg. Recommended agents include nicardipine, labetalol, or esmolol.

9. Intracranial hemorrhage: In patients with evidence of elevated intracranial pressure, mean arterial pressure should be reduced to approximately 130 mm Hg. In the absence of increased intracranial pressure, targets may be lowered to a mean arterial pressure of 110 mm Hg or systolic blood pressure of 150–160 mm Hg. Nicardipine, labetalol, or esmolol are appropriate choices.

10. Acute ischemic stroke: If fibrinolytic therapy is planned, blood pressure should be reduced below 185/110 mm Hg. If fibrinolysis is not planned, treatment is recommended only if blood pressure exceeds 220/120 mm Hg. Preferred agents include labetalol, nicardipine, or topical nitroglycerin paste applied at 1–2 inches.

11. Hypertensive urgency: Effective oral agents include labetalol 200–400 mg every 2–3 hours, captopril 25 mg every 4–6 hours, sublingual nitroglycerin 0.3–0.6 mg, or clonidine administered as a 0.2 mg loading dose followed by 0.1 mg hourly to a maximum of 0.7 mg.

12. Asymptomatic severe hypertension: In patients without evidence of target organ damage, initiation of oral antihypertensive therapy at discharge may be considered. Thiazide diuretics such as hydrochlorothiazide 25 mg daily are appropriate for uncomplicated hypertension. Beta-blockers such as metoprolol 50 mg orally twice daily may be used in selected patients, while angiotensin-converting enzyme inhibitors such as lisinopril may be initiated at 10 mg daily in patients with heart failure, renal disease, diabetes mellitus, or prior stroke. Restarting previously prescribed antihypertensive regimens in nonadherent patients is recommended.

6. Pediatric Hypertensive Emergencies

In children, hypertensive emergencies are uncommon but often secondary to renovascular disease or catecholamine-secreting tumors. Symptoms may be nonspecific. Treatment decisions are guided by BP percentiles and symptom severity. The goal is controlled BP reduction, typically no more than 25% within the first hour. Pediatric patients requiring acute intervention generally warrant hospital admission.

7. Pulmonary Hypertension

Pulmonary hypertension is defined hemodynamically by an elevated mean pulmonary artery pressure and results in progressive right ventricular dysfunction. Although definitive diagnosis is not made in the ED, clinical suspicion should influence evaluation and disposition. Common symptoms include dyspnea, fatigue, chest pain, and syncope.

Emergency management focuses on treating precipitating or underlying conditions, such as hypoxia or heart failure. Some patients may already be receiving chronic therapies, including calcium channel blockers or continuous prostacyclin infusions. Acute initiation of disease-specific therapy is rarely indicated in the ED.

8. Conclusion

Systemic and pulmonary hypertension present with a wide spectrum of severity in emergency medicine. Accurate classification, careful clinical assessment, judicious diagnostic testing, and condition-specific management strategies are essential to optimize outcomes and minimize complications.

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CHAPTER 2

High-Altitude Medical Emergencies

Kürşat SARIBAŞ[1]
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Introduction

High-altitude medical conditions are primarily caused by hypobaric hypoxia. The likelihood and severity of illness depend largely on the altitude reached, the speed of ascent, and individual susceptibility. Failure to acclimatize appropriately can result in a spectrum of altitude-related syndromes, ranging from mild acute mountain sickness to life-threatening pulmonary or cerebral edema.

1. Acute Mountain Sickness (AMS)

Clinical Presentation

Acute mountain sickness typically affects individuals who ascend rapidly to elevations above 2,000 meters (6,560 feet) without adequate acclimatization. Symptoms usually develop within 6 to 12

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hours after arrival but may be delayed for up to 24 hours. The clinical picture resembles alcohol intoxication or a hangover and commonly includes bifrontal headache, anorexia, nausea, fatigue, dizziness, and generalized weakness. Progression of disease may be indicated by worsening headache, persistent vomiting, reduced urine output, dyspnea, and marked lethargy.

Physical examination findings are often minimal in early disease. Orthostatic hypotension, peripheral or facial edema, and mild crackles on lung auscultation may be present. Retinal venous congestion and hemorrhages can be observed at extreme altitudes, particularly above 5,000 meters. Oxygen saturation at rest is usually appropriate for altitude and is not a reliable diagnostic marker for AMS.

Diagnosis and Differential Diagnosis

The diagnosis of AMS is primarily clinical and based on a recent history of ascent combined with characteristic symptoms. Alternative diagnoses to consider include dehydration, hypothermia, carbon monoxide exposure, migraine headache, pulmonary or central nervous system infections, and physical exhaustion.

Emergency Management and Disposition

Management focuses on halting disease progression, relieving symptoms, and promoting acclimatization.

1. Further ascent should be discontinued until symptoms resolve. Mild AMS can be treated symptomatically with analgesics such as acetaminophen or nonsteroidal anti-inflammatory drugs, along with antiemetics. Most mild cases resolve within 12 to 36 hours if ascent is halted.

2. Descent of 300 to 1,000 meters typically results in rapid symptom improvement. Immediate descent is mandatory in patients with

worsening neurologic symptoms, ataxia, altered mental status, or signs of pulmonary edema.

3. Supplemental low-flow oxygen effectively alleviates symptoms.

4. Portable hyperbaric therapy should be considered when descent is not feasible.

5. Pharmacologic treatment includes acetazolamide (125–250 mg orally twice daily; pediatric dose 2.5 mg/kg twice daily) and dexamethasone (4 mg every 6 hours). Acetazolamide facilitates acclimatization by inducing a mild metabolic acidosis through bicarbonate diuresis and is effective for both prevention and treatment. It is contraindicated in patients with sulfonamide allergy.

6. Patients demonstrating clinical improvement may be discharged with counseling on gradual ascent, avoidance of alcohol and sedatives, and prophylactic acetazolamide for future high-altitude exposure.

2. High-Altitude Pulmonary Edema (HAPE)

Risk Factors

Risk factors for HAPE include rapid ascent, strenuous physical activity, cold exposure, underlying pulmonary hypertension, and use of sedative-hypnotic medications. Children with concurrent respiratory infections may be particularly vulnerable. If untreated, HAPE carries a significant risk of mortality.

Clinical Presentation

HAPE generally develops between the second and fourth night after ascent. Early symptoms include reduced exercise tolerance and a dry cough, which may rapidly progress to resting dyspnea, productive cough with frothy sputum, severe fatigue, and cyanosis. Physical

findings include tachycardia, tachypnea, crackles on lung auscultation, and signs of pulmonary hypertension such as a loud pulmonic component of the second heart sound and right ventricular heave.

Oxygen saturation is markedly reduced for altitude and declines further with exertion. Chest radiography typically reveals patchy, initially interstitial infiltrates that progress to alveolar edema. Electrocardiography may demonstrate right axis deviation and right ventricular strain.

Diagnosis and Differential Diagnosis

Differential considerations include pneumonia, asthma exacerbation, congestive heart failure, pulmonary embolism, and acute coronary syndromes. A rapid clinical response to oxygen and descent strongly supports the diagnosis of HAPE.

Emergency Management and Disposition

1. Administer supplemental oxygen and titrate to maintain oxygen saturation ≥90%.

2. Immediate descent remains the definitive treatment. Portable hyperbaric therapy may be used when descent is impossible. Mild cases may be managed with strict bed rest and oxygen alone.

3. Pharmacologic therapy is generally reserved for field settings and includes nifedipine extended release (20–30 mg every 12 hours), sildenafil (50 mg three times daily), or tadalafil (10 mg twice daily), which reduce hypoxic pulmonary vasoconstriction.

4. Patients may be discharged once clinical improvement is sustained and oxygen saturation on room air remains above 90%.

3. High-Altitude Cerebral Edema (HACE)

Clinical Presentation

High-altitude cerebral edema represents the most severe form of altitude illness and is characterized by progressive neurologic dysfunction, often evolving from AMS or HAPE. Patients present with confusion, ataxia, altered consciousness, and may progress to coma. Cranial nerve palsies, particularly involving the third and sixth nerves, may be observed.

Diagnosis and Differential Diagnosis

Differential diagnoses include cerebrovascular events, intracranial mass lesions, meningitis, encephalitis, and metabolic encephalopathies. Magnetic resonance imaging may demonstrate characteristic T2 hyperintensities in the splenium of the corpus callosum. Diagnostic evaluation should not delay urgent treatment.

Emergency Management and Disposition

1. Administer supplemental oxygen and maintain saturation ≥90%. Endotracheal intubation and mechanical ventilation are required for comatose patients.

2. Immediate descent is essential; hyperbaric therapy should be initiated if descent is not possible.

3. Administer dexamethasone with an initial dose of 8 mg, followed by 4 mg every 6 hours.

4. In ventilated patients, arterial blood gases should be closely monitored, avoiding excessive hypocapnia. Intracranial pressure monitoring may be considered when available.

5. Persistent neurologic deficits after descent necessitate hospital admission.

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CHAPTER 3

EMERGENCY DEPARTMENT APPROACH TO CARBON MONOXIDE POISONING

Ufuk Özgür BALICA[1]
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1. Introduction

Carbon monoxide (CO) poisoning represents one of the most frequent and potentially fatal toxicological emergencies encountered in emergency departments worldwide. Due to its colorless, odorless, and non-irritating nature, exposure often goes unrecognized until clinical symptoms develop. This characteristic makes CO a particularly dangerous toxin, frequently referred to as a "silent killer." Delays in diagnosis and treatment may result in severe neurological, cardiovascular, and metabolic complications.

Emergency physicians play a critical role in the early recognition and management of CO poisoning. A structured, evidence-based approach is essential to reduce morbidity and

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mortality. This chapter presents a comprehensive overview of carbon monoxide poisoning with a focus on emergency department evaluation, diagnosis, and management.

2. Epidemiology and Risk Factors

Carbon monoxide poisoning remains a major public health concern globally, particularly during winter months when indoor heating systems are widely used. Inadequate ventilation, malfunctioning heating devices, and use of solid fuels significantly increase exposure risk. Additional sources include motor vehicle exhaust, fires, and industrial emissions.

Certain populations are more vulnerable to CO toxicity, including infants, elderly individuals, pregnant women, and patients with pre-existing cardiovascular or pulmonary disease. Occupational exposure is also notable among firefighters and industrial workers. Recognition of these risk factors is crucial during initial patient assessment in the emergency setting.

3. Pathophysiology

The toxic effects of carbon monoxide are mediated through multiple mechanisms. CO binds to hemoglobin with an affinity approximately 200–250 times greater than oxygen, forming carboxyhemoglobin (COHb). This binding reduces oxygen-carrying capacity and shifts the oxygen–hemoglobin dissociation curve to the left, impairing oxygen delivery to tissues.

Beyond hemoglobin binding, CO directly affects cellular respiration by binding to myoglobin and mitochondrial cytochrome oxidase enzymes. This interference disrupts oxidative

phosphorylation, leading to cellular hypoxia and energy failure. The brain and myocardium, due to their high metabolic demands, are particularly susceptible to injury.

4. Clinical Presentation In The Emergency Department

Clinical manifestations of CO poisoning vary depending on the concentration of exposure, duration, and individual susceptibility. Early symptoms are often nonspecific and include headache, dizziness, fatigue, nausea, and vomiting. As exposure severity increases, patients may develop confusion, syncope, seizures, cardiac arrhythmias, and coma.

In the emergency department, the presence of multiple patients from the same environment presenting with similar complaints should raise immediate suspicion for CO exposure. A thorough environmental history is essential.

5. Diagnostic Evaluation

Diagnosis of CO poisoning relies on a high index of clinical suspicion supported by laboratory findings. Measurement of carboxyhemoglobin levels via arterial or venous blood gas analysis remains the diagnostic gold standard. However, COHb levels do not always correlate with clinical severity and should be interpreted in conjunction with patient presentation.

Standard pulse oximetry is unreliable, as it cannot differentiate between oxyhemoglobin and carboxyhemoglobin, often yielding falsely normal oxygen saturation values. Additional

diagnostic studies may include electrocardiography, cardiac biomarkers, serum lactate, and neuroimaging in selected cases.

Table 1. Clinical Effects According to Carboxyhemoglobin Levels

0–10%	Often asymptomatic, mild headache
10–20%	Headache, dizziness, fatigue
20-30%	Severe headache, nausea, impaired concentration
30-40%	Syncope, confusion
40-60%	Coma, severe arrhythmias
>60%	High risk of death

Source: Tintinalli's Emergency Medicine A Comprehensive Study Guide

6. Emergency Management

Effective management of carbon monoxide poisoning in the emergency department requires rapid recognition, immediate stabilization, and prompt initiation of definitive therapy. The primary goals of emergency management are to reverse tissue hypoxia, prevent ongoing cellular injury, and reduce the risk of acute mortality and delayed neurological sequelae.

6.1 Initial Assessment and Stabilization

Management should begin with a structured assessment following the principles of Airway, Breathing, and Circulation (ABC). Airway patency must be ensured in all patients, particularly those presenting with altered mental status, seizures, or signs of respiratory compromise. Endotracheal intubation should be

considered in patients with impaired airway protective reflexes, severe encephalopathy, or respiratory failure.

Breathing assessment includes evaluation of respiratory rate, oxygenation, and work of breathing. Supplemental oxygen should be administered immediately to all suspected cases, regardless of initial pulse oximetry values. Circulatory status should be assessed through blood pressure, heart rate, peripheral perfusion, and establishment of intravenous access. Continuous cardiac monitoring is recommended due to the risk of myocardial ischemia and potentially life-threatening arrhythmias.

6.2 Oxygen Therapy

Administration of 100% normobaric oxygen via a non-rebreather mask at a flow rate of 10–15 L/min is the cornerstone of treatment for carbon monoxide poisoning. High-flow oxygen significantly reduces the half-life of carboxyhemoglobin from approximately 4–5 hours in room air to 60–90 minutes under normobaric oxygen conditions. Oxygen therapy should be continued until clinical symptoms resolve and carboxyhemoglobin levels decrease to near-normal values.

In patients requiring mechanical ventilation, inspired oxygen concentration should be maintained at 100% initially. Oxygen therapy improves tissue oxygen delivery, enhances dissociation of carbon monoxide from hemoglobin, and mitigates ongoing cellular hypoxia.

6.3 Indications for Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy (HBOT) should be considered in selected patients based on clinical severity rather than carboxyhemoglobin level alone. Commonly accepted indications include loss of consciousness at any time, severe neurological symptoms, evidence of myocardial ischemia or significant arrhythmias, severe metabolic acidosis, pregnancy with elevated carboxyhemoglobin levels, and persistent symptoms despite adequate normobaric oxygen therapy.

HBOT accelerates elimination of carbon monoxide, reduces oxidative stress, and may decrease the incidence of delayed neurological sequelae. When available, therapy should ideally be initiated within the first 6 hours following exposure.

6.4 Supportive and Adjunctive Care

Supportive management includes correction of hypotension with intravenous fluids and vasopressors when necessary. Seizures should be treated promptly with benzodiazepines, followed by additional antiepileptic agents if required. Metabolic abnormalities such as acidosis and electrolyte disturbances should be monitored and corrected appropriately.

Cardiac biomarkers and electrocardiography should be obtained to evaluate myocardial injury. Patients with evidence of cardiac involvement should receive cardiology consultation and be

managed according to acute coronary syndrome protocols, with the understanding that ischemia is typically hypoxic rather than thrombotic in origin.

6.5 Observation, Disposition, and Follow-Up

Patients should be observed until symptoms have resolved and carboxyhemoglobin levels have decreased appropriately. Admission is recommended for patients with moderate to severe poisoning, persistent symptoms, pregnancy, cardiac involvement, or social factors limiting safe discharge.

Patients discharged from the emergency department should receive detailed instructions regarding delayed neurological symptoms and environmental safety measures to prevent re-exposure. Follow-up evaluation is recommended to assess for late-onset neurocognitive sequelae.

7. Prognosis and Delayed Neurological Sequelae

Although many patients recover fully after acute treatment, delayed neurological sequelae may occur days to weeks following exposure. These include cognitive impairment, memory deficits, personality changes, parkinsonism, and gait disturbances. Early recognition and appropriate oxygen therapy are associated with improved outcomes.

Patients discharged from the emergency department should receive clear instructions regarding potential delayed symptoms and the importance of follow-up evaluation.

8. Prevention and Public Health Implications

Carbon monoxide poisoning is largely preventable. Installation of CO detectors, regular maintenance of heating systems, adequate ventilation, and public education are essential preventive strategies. Emergency physicians also play a role in prevention by identifying environmental risks and advising patients accordingly.

9. Conclusion

Carbon monoxide poisoning is a life-threatening but preventable emergency condition. A systematic approach in the emergency department—emphasizing early suspicion, prompt diagnosis, and appropriate oxygen therapy—significantly improves patient outcomes. Comprehensive education and awareness remain key components in reducing the burden of this condition.

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CHAPTER 4

Emergency Department Approach to Methanol Toxicity: Epidemiology, Diagnosis, and Management

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Introduction

Methanol (CH_3OH) is a highly toxic alcohol commonly encountered in industrial and household products. It belongs to the broader group of toxic alcohols, a category that includes methanol, ethylene glycol, diethylene glycol, and isopropyl alcohol, all of which can cause profound metabolic derangements and systemic toxicity. Among

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these agents, methanol is particularly dangerous due to its ability to cause severe metabolic acidosis, permanent visual injury, neurologic damage, and death if treatment is delayed.

Exposure to methanol occurs through accidental or intentional ingestion, inhalation, or dermal absorption. While isolated cases are more common, large-scale poisoning outbreaks have been reported worldwide, often related to contaminated or illicitly produced alcoholic beverages. Methanol-containing products include windshield washer fluids, antifreeze solutions, industrial solvents, fuels used for heating food, perfumes, and various automotive and cleaning agents. Clinical severity varies widely, necessitating management strategies ranging from close observation to aggressive antidotal therapy and extracorporeal elimination. Fomepizole is considered the first-line antidote, with ethanol serving as an alternative when fomepizole is unavailable. Unlike some other toxic alcohol exposures, hemodialysis is frequently required in methanol toxicity.

Etiology

Methanol toxicity may result from ingestion, inhalation, or skin contact. In adults, intentional ingestion for self-harm or substitution for ethanol represents the most common exposure route. Methanol has also been misused recreationally through ingestion of fuel products or inhalation of volatile agents such as carburetor cleaners. Accidental ingestion is more frequently observed in pediatric populations, particularly toddlers exploring their environment. Occupational exposures can occur in poorly ventilated industrial settings.

Epidemiology

Populations at highest risk include individuals with alcohol use disorder, patients with suicidal intent, and young children. Most reported cases involve single individuals; however, epidemics have occurred due to errors in distillation, contamination of beverages, or illicit alcohol production. Although the true incidence is difficult to determine, poison center surveillance data indicate thousands of toxic alcohol–related consultations annually in the United States, with methanol contributing to a measurable number of fatalities each year.

Pathophysiology

Following ingestion, methanol is rapidly absorbed from the gastrointestinal tract and distributed throughout total body water, with an estimated volume of distribution of approximately 0.7 L/kg. Peak serum concentrations occur soon after absorption. Elimination kinetics are dose-dependent; at higher concentrations, methanol exhibits zero-order elimination similar to ethanol. Pulmonary excretion of unmetabolized methanol occurs but is inefficient.

Hepatic metabolism is the primary determinant of toxicity. Methanol is oxidized by alcohol dehydrogenase to formaldehyde, which is subsequently converted by aldehyde dehydrogenase to formic acid. These reactions generate reduced nicotinamide adenine dinucleotide (NADH), altering the cellular redox state. The average metabolic clearance rate is approximately 8–9 mg/dL per hour at moderate serum concentrations.

Formic acid accumulates due to limited elimination capacity. A small fraction is metabolized via folate-dependent pathways to carbon dioxide and water, which are exhaled. When alcohol dehydrogenase

is inhibited, unmetabolized methanol persists, resulting in a prolonged effective half-life that may exceed 30 to 80 hours.

The toxic effects of methanol are mediated almost entirely by formic acid. Formic acid inhibits mitochondrial cytochrome-c oxidase, leading to impaired oxidative phosphorylation, cellular hypoxia, and lactic acidosis. The optic nerve and retina are particularly susceptible, resulting in characteristic visual toxicity. The basal ganglia, especially the putamen, are also vulnerable, and bilateral necrosis with or without hemorrhage is a classic neuroimaging finding. Acute kidney injury and pancreatitis have been reported as additional complications.

Toxicokinetics

Ingestion of approximately 1 g/kg of methanol may be fatal. Given methanol's density of roughly 0.8 g/mL, this corresponds to 1–2 mL/kg of pure methanol. The dose required to produce permanent visual damage remains uncertain. Observational data suggest that visual injury is uncommon at blood methanol concentrations below 50 mg/dL, although treatment is generally recommended when levels exceed 20–25 mg/dL. Methanol contributes to an elevated osmolar gap early after ingestion. As metabolism progresses, the osmolar gap decreases while an anion gap metabolic acidosis develops due to formate accumulation. Elevated lactate may occur secondary to mitochondrial dysfunction and altered redox balance. Oxidative stress induced by formic acid is central to retinal and neurologic injury.

Clinical Presentation

Early physical examination findings may be subtle or nonspecific, particularly during the latent phase, which may last 12–24 hours. Patients may appear mildly intoxicated or asymptomatic. Gastrointestinal symptoms such as nausea, vomiting, and abdominal pain often precede neurologic and respiratory manifestations. As acidosis worsens, patients may develop hyperventilation, confusion, seizures, coma, and hemodynamic instability. Visual symptoms are hallmark features and include blurred vision, reduced acuity, photophobia, and visual field defects. Funduscopic examination may reveal optic disc hyperemia, papilledema, or pupillary abnormalities. Without timely intervention, progression to respiratory failure, circulatory collapse, and death may occur.

Diagnostic Evaluation

Patients may present at various stages, from asymptomatic with an isolated osmolar gap elevation to critically ill with profound metabolic acidosis and end-organ damage. Initial evaluation should include an electrocardiogram, basic metabolic panel, arterial or venous blood gas analysis, serum osmolality, lactate, ketones, ethanol concentration, and screening for co-ingestants such as acetaminophen and salicylates. Definitive measurement of methanol or formate via gas chromatography confirms the diagnosis but is frequently unavailable in real time. Consequently, diagnosis often relies on indirect markers, including anion gap metabolic acidosis and osmolar gap elevation. Serial laboratory monitoring every 2–4 hours is recommended in suspected early presentations once ethanol has been excluded. The osmolar gap is most useful early in the course, while anion gap metabolic acidosis predominates later. A gap exceeding 25 mOsm/kg raises concern, and values above 50 mOsm/kg strongly suggest toxic alcohol exposure. Importantly, a

normal osmolar gap does not exclude methanol poisoning in later stages.

Emergency Department Management

Management priorities include stabilization, inhibition of toxic metabolism, correction of acidosis, and removal of methanol and its metabolites. Fomepizole is the preferred antidote and should be initiated promptly when methanol ingestion is suspected. Ethanol may be used when fomepizole is unavailable but requires intensive monitoring. Indications for antidotal therapy include confirmed methanol ingestion, suspected exposure with supportive laboratory findings, or unexplained metabolic acidosis. Hemodialysis is frequently required and is indicated for severe acidosis, visual or neurologic symptoms, coma, seizures, or high methanol concentrations. Intermittent hemodialysis is preferred due to superior clearance. Empiric treatment should be initiated without delay when methanol poisoning is strongly suspected, particularly in the presence of unexplained high–anion gap metabolic acidosis, visual disturbances, neurologic impairment, or a compatible exposure history. Treatment decisions should not be deferred pending confirmatory methanol concentrations. Fomepizole is the preferred antidote due to its predictable pharmacokinetics and safety profile. Therapy should be initiated for confirmed methanol concentrations ≥20–25 mg/dL or when clinical suspicion is high.

Fomepizole:
- Loading dose: 15 mg/kg intravenously over 30 minutes
- Maintenance dose: 10 mg/kg intravenously every 12 hours
- After four maintenance doses, increase to 15 mg/kg every 12 hours due to autoinduction
- Continue therapy until methanol concentration is <25 mg/dL and acid–base disturbances have resolved

During intermittent hemodialysis, fomepizole should be administered every four hours or a supplemental dose given immediately following dialysis. Ethanol may be used as an alternative when fomepizole is unavailable, targeting a serum ethanol concentration of approximately 100 mg/dL. Due to its narrow therapeutic window and sedative effects, ethanol therapy requires intensive monitoring. Severe metabolic acidosis should be corrected with intravenous sodium bicarbonate. Initial dosing typically consists of 1–2 mEq/kg administered as a bolus, followed by a continuous infusion titrated to maintain arterial pH above 7.30. Correction of acidosis reduces tissue penetration of formic acid and mitigates end-organ toxicity. Folate or folinic acid supplementation (1 mg/kg intravenously, maximum 50 mg every 6 hours) may enhance the metabolism of formic acid to carbon dioxide and water. Supportive care measures include seizure control, temperature regulation, glycemic monitoring, and intensive care unit admission for patients with severe toxicity or those receiving ethanol therapy.

Extracorporeal Elimination

Hemodialysis is a cornerstone of therapy in moderate to severe methanol poisoning. Indications include severe metabolic acidosis (pH <7.25–7.30), visual impairment, neurologic manifestations, coma, seizures, renal failure, or elevated methanol concentrations, generally exceeding 50 mg/dL. Intermittent hemodialysis is preferred due to its superior clearance of both methanol and formic acid.

Disposition and Consultation

All symptomatic patients require hospital admission, with intensive care monitoring for severe cases. Early consultation with a medical

toxicologist or regional poison control center is strongly recommended. Nephrology consultation is essential when hemodialysis is indicated, and ophthalmologic evaluation should be obtained in the presence of visual symptoms.

Conclusion

Hemodialysis is a cornerstone of therapy in moderate to severe methanol poisoning. Indications include severe metabolic acidosis (pH <7.25–7.30), visual impairment, neurologic manifestations, coma, seizures, renal failure, or elevated methanol concentrations, generally exceeding 50 mg/dL. Intermittent hemodialysis is preferred due to its superior clearance of both methanol and formic acid.

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