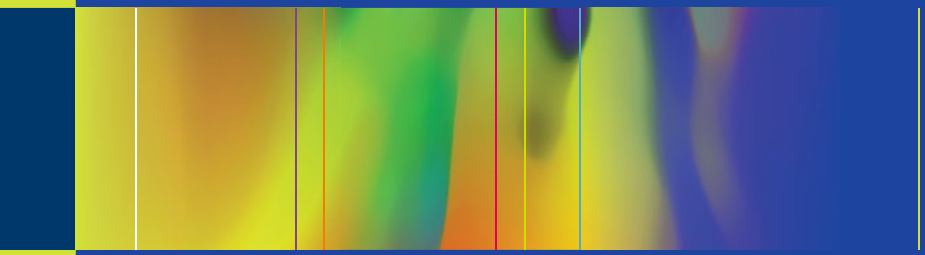
Kimberly D. Nordstrom
Michael P. Wilson
Editors



Quick Guide to Psychiatric Emergencies

Tools for Behavioral and Toxicological Situations

Springer

Quick Guide to Psychiatric Emergencies

Kimberly D. Nordstrom
Michael P. Wilson
Editors

Quick Guide to Psychiatric Emergencies

Tools for Behavioral and Toxicological Situations

Springer

Editors
Kimberly D. Nordstrom
Department of Human Services
Office of Behavioral Health
Denver, CO, USA

Michael P. Wilson
University of Arkansas for
Medical Sciences
Little Rock, AR, USA

American Association for
Emergency Psychiatry
Parker, CO, USA

ISBN 978-3-319-58258-0 ISBN 978-3-319-58260-3 (eBook)
https://doi.org/10.1007/978-3-319-58260-3

Library of Congress Control Number: 2018935599

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

To my very patient and very loving husband and son who enjoyed a lot of ski weekends in order to help me complete this book! To my parents—for always being there.

KN

To my supportive family and friends who always know the right time and place to tell a funny joke. There were many laughs during the preparation of this book, and for that, I will always be grateful.

MPW

Preface

I met Dr. Wilson years ago through the American Association for Emergency Psychiatry, during the creation of the "BETA Project." Since that time, we have been asked to speak and write together on many topics having to do with behavioral emergencies: Dr. Wilson giving an emergency medicine perspective while I give insights from the world of emergency psychiatry. We have had many lectures on "Medical Mimics," those disorders that mimic psychiatric disease. At every lecture, we have been asked to put our thoughts down in handbook form. This is the result. We brought a few of our friends along with us to help in the effort. We have had fun creating this handbook and hope you find it useful.

Denver, CO — Kimberly D. Nordstrom
Little Rock, AR — Michael P. Wilson

Acknowledgements

First we have to acknowledge the other authors of this handbook: Paul Borghesani, Julien J. Cavanagh, Bryan Corbett, Caitlin A. Kieltyka, Ernest C. Nwachukwu, Jagoda Pasic, Archana A. Shah, Christopher S. Sharp, Teresa Y. Smith, Paul Zarkowski, and Leslie S. Zun. When Dr. Wilson and I began writing this book (alone) we quickly realized that it was a huge undertaking (distilling large amounts of research to bullet-point facts). We got smart and invited some friends—those well known in the field of behavioral emergencies and a couple of talented junior colleagues.

We would also like to acknowledge colleagues who have been supportive in this and many of our endeavors: Gary Vilke, Michael Allen, Scott Zeller, Seth Powsner, Robert House, Kama Guluma, and Steve Hayden.

Contents

Contributors

Paul Borghesani, M.D., Ph.D. Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, University of Washington School of Medicine, Seattle, WA, USA

Julien J. Cavanagh, M.D. Department of Neurology, State University of New York—Downstate Medical Center, Brooklyn, NY, USA

Bryan Corbett, M.D. Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA

Caitlin A. Kieltyka, M.D. Department of Psychiatry, School of Medicine, University of Colorado-Denver, Denver, CO, USA

Kimberly D.Nordstrom, M.D., J.D. Department of Human Services, Office of Behavioral Health, Denver, CO, USA

American Association for Emergency Psychiatry, Parker, CO, USA

Ernest C. Nwachukwu, M.D. Department of Emergency Medicine, University of Chicago, Chicago, IL, USA

Jagoda Pasic, M.D., Ph.D. Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, University of Washington, Seattle, WA, USA

Archana A. Shah, M.D., M.B.A. Department of Emergency Medicine, University of Chicago, Chicago, IL, USA

Christopher S. Sharp, M.D. Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA
Department of Psychiatry, University of Colorado—Denver School of Medicine, Denver, CO, USA

Teresa Y. Smith, M.D., M.S.Ed., F.A.C.E.P. Department of Emergency Medicine, Kings County Hospital, State University of New York, Brooklyn, NY, USA

State University of New York Downstate Medical Center, Kings County Hospital, Brooklyn, NY, USA

Shameeke Taylor, M.D. Department of Emergency Medicine, University of Chicago, Chicago, IL, USA

Michael P. Wilson, M.D., Ph.D. University of Arkansas for Medical Sciences, Little Rock, AR, USA

Paul Zarkowski, M.D. Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, University of Washington, Seattle, WA, USA

Leslie S. Zun, M.D., M.B.A. Department of Emergency Medicine, Chicago Medical School, North Chicago, IL, USA

Department of Emergency Medicine, Mount Sinai Hospital, New York, NY, USA

American Association for Emergency Psychiatry, Parker, CO, USA

Section I
Evaluation of Patients

Section Editor: Les S. Zun, M.D.

Chapter 1
Medical and Psychiatric History

Ernest C. Nwachukwu and Leslie S. Zun

Introduction

Taking a good medical/psychiatric history is an essential first step in appropriately triaging and ultimately diagnosing a patient presenting with a behavioral emergency. Taking the information gathered during the initial interview along with physical exam findings can help to better differentiate primary psychiatric disorders from their medical mimics. The differential for behavioral emergencies is very broad and differs by each individual presentation. Using your interview to determine whether elements of the family history, medical history, social history, sexual

E.C. Nwachukwu, M.D.
Department of Emergency Medicine, University of Chicago, Chicago, IL 60637, USA

L.S. Zun, M.D., M.B.A. (✉)
Department of Emergency Medicine, Chicago Medical School, North Chicago, IL 60064, USA

Department of Emergency Medicine, Mount Sinai Hospital, New York, NY 10029, USA

Department of Emergency Medicine, Sinai Health System, Chicago, IL 60608, USA
e-mail: ZUNL@SiNAi.Org

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_1,

history, medications, or environmental exposures are contributory factors to a patient's presentation can be important in obtaining a good general picture. Often, collateral information obtained from friends, caregivers, and family can be key to elucidating the true cause of a behavioral complaint.

History of Present Illness (HPI)

When getting the HPI, obtain information regarding the onset, duration, character, exacerbating/alleviating factors, timing and severity of the complaint. Each of these parts of the HPI can help to understand the exact cause of the complaint.

- Character: What symptoms is the patient experiencing? Describe the change in behavior subjectively (patient perspective) and objectively (bystander perception).
- Onset/Duration: When did these symptoms start and how long have they been present? Was the onset associated with anything (recent start of or abrupt discontinuation from a medication)?
- Exacerbating/Alleviating Factors: Any precipitating or mitigating factors present?
- Timing: Are the symptoms constant or do they relapse/remit?
- Severity: How has the patient's work, school or home life been affected?

Past Medical History

The past medical history can clue you into comorbid conditions that can either predispose to medical causes of behavioral changes or exacerbate underlying psychiatric illness.

- CNS
  - Dementia, CVA, normal pressure hydrocephalus, space-occupying lesion

- Infectious
  - Pneumonia, UTI, Bacteremia, Intra-abdominal, Meningitis, Encephalitis
- Trauma
  - Subdural, Epidural, Subarachnoid, Concussion, Diffuse Axonal Injury
- Electrolyte
  - Sodium, Glucose, Calcium
- Endocrine
  - Thyroid, Adrenal, Pituitary
- Metabolic
  - Uremia, encephalopathy, hypoxemia, hypercarbia

Past Psychiatric History

The past psychiatric history can give clues about prior psychiatric exacerbations that may be similar to the current presentation. The psychiatric portion of the history can also give valuable information about a patient's risk of harm to his/herself.

- Does the patient have any prior psychiatric diagnoses?
  - When was the patient last hospitalized?
  - Where does the patient receive his/her mental health care?
  - Any history of similar symptoms during an exacerbation?
- Does the patient have any history of suicide attempts or aggressive behavior in the past?
  - Suicide Risk Assessment
  - Is there any current homicidal ideation or psychosis (auditory/visual hallucinations or delusions) present?

Medications/Allergies

Medication and allergy questions are aimed at determining whether a new medication, change in dose or medication could be the cause of the symptoms. It can also help give information on agents that one could use to help control behavior in the acute setting.

- What are the current medications?
  - Are medications being used appropriately (taken as prescribed and not to excess)
  - Any new medications?
  - Any recent dosage changes?
  - Any medical interactions between current medications and newly prescribed medications?
- What are the medication allergies and any history of a serious side effect to medications?
  - Any history of QT prolongation or concomitant QT prolonging medications being taken?
  - Any history of severe akathisia (extreme restlessness caused by medications)?

Family History

Family history questions can aid the clinician in determining whether or not the current presentation is similar to another family members. Does the patient have a family history of a psychiatric illness or a medical illness that could be the cause of or a factor in the current presentation?

- Any family history of psychiatric illness?
- Any family history of medical illness?
  - Is there a similar age of onset?

Social History

Social history should be aimed at obtaining information on patient's functional abilities such as employment and schooling as well as chronic or acute illicit drug use. Assessing functional abilities can give a baseline and help show any deterioration that may be occurring. Regarding the assessment of substance use, many times substance use can precipitate behavioral emergencies or acute intoxication can be

misinterpreted as an emergency by a patient's family/friends. It is important to note that questions regarding substance use are used strictly to provide medical care and are not asked to incriminate the patient or for punitive purposes. The kind of substance and frequency of use can also help to determine whether the presentation is primarily due to intoxication or withdrawal. When trying to undertand a person's substance use and patterns, it may be helpful to have the following questions in mind:

- Does the patient have any history of recent illicit drug use?
  - What agent?
  - Time of last use?
- Any history of chronic drug or alcohol use
  - Time of last use?
    - Pattern of past use (binge or regular user).
    - Amount of use (commonly given in amount of money used each week or in weights)
  - Has the patient ever experienced withdrawal in the past?

Sexual History

Sexual history can be important in determining the risk for latent diseases that predispose to immunosuppression or have direct effects on the CNS.

- Does the patient have any history of high-risk sexual behaviors?
  - i.e. multiple partners, MSM (men who have sex with men), inconsistent use of barrier contraception

Environmental Exposures

Environmental exposures encompasses travel and occupational history as well as exposure to the elements.

- Does the patient have any history of recent travel?
  - Which region did you travel to?
  - When did you travel?
- What is the patient's occupation?
- Has the patient been exposed to the elements?
- Any recent chemical exposures?

Tool

"SAMPLE"

Signs/Symptoms
Allergies
Medications
Past pertinent history
(SF-PMS) Social, Family, Psychiatric, Medical, Sexual
Last oral intake
Events prior to presentation

Chapter 2
Physical Exam

Shameeke Taylor, Archana A. Shah, and Leslie S. Zun

Introduction

The physical examination is an important tool for a clinician in the evaluation and subsequent care of a patient. The process of gathering information on the physical and mental state of a patient is key in determining the differential diagnosis and allows for the use of directed, rather than universal, laboratory studies. The physical examination is a necessary skill for psychiatrists and ED physicians in order to prevent

S. Taylor, M.D. • A.A. Shah, M.D., M.B.A. (✉)
Department of Emergency Medicine, University of Chicago, Chicago, IL 60637, USA
e-mail: archana.shah@uchospitals.edu

L.S. Zun, M.D., M.B.A.
Department of Emergency Medicine, Chicago Medical School, North Chicago, IL 60064, USA

Department of Emergency Medicine, Mount Sinai Hospital, New York, NY 10029, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_2,

physical illness from going undetected in patients that present with concern for psychiatric ailments. With the evolution of medicine, medical knowledge and discovery increased exponentially but the base of the physical examination remained the same: a discipline rooted in the astute use of the five senses. Evaluation of the psychiatric patient takes an eye for detail, patience and a curious mind.

The physical examination begins as soon as you walk into the patient's room. Key information can be ascertained even before the clinician begins talking to the patient.

What Do You Smell?

- Abnormal odors? (does the patient/room smell of urine, alcohol, blood etc.?)

What Do You See?

- Personal appearance: hygiene (covered in dirt, stool, blood etc), clothed vs unclothed, hair clean?, unkempt?, dressed appropriate for season (multiple jackets in the summer vs shorts in the winter)?, bruised?, signs of infestation/scratching?, signs of self-harm?
- Interaction with staff (cooperative, combative?)
- Alone?, With family, friends?, Police?, Security?, (Important, as you can gather information from people present about recent behavior or history of illness, medication compliance etc.)
- Staring into space?, Huddled in the corner?, In restraints?
- Involuntary movements/twitching?
- Is the patient clutching a body part? (chest, extremity, head, etc.?) (This is important as it can tell information about possible injuries.)
- Clothing: rips/tears? signs of infestation (bed bugs, etc.)? blood or other secretions?

What Do You Hear?

- Patient responding to voice or command of person present in room or not? Is patient talking to themselves? Yelling or screaming?

The more formalized exam:

Orientation

- Is the patient alert and able to respond to questions/commands?
- Oriented to person, place and time? Is the person oriented to the situation?

Head

- Visible trauma/deformity (may be tied to reason patient presents to the facility or as consequence of struggle to be brought to the facility), signs of infestation, facial flushing, twitches or stereotypical movements

Eyes

- Pupillary constriction/dilation, presence of nystagmus, equal and reactive to light, photosensitivity, open/closed during conversation, reacting to stimuli in room (signs for drug use/overdose, sign of possible intracranial process etc)

Nose

- Epistaxis, rhinorrhea, deformity (possible trauma vs cocaine or other illicit substance use)

Ears

- Hyperacusis/phonophobia, tenderness

Throat

- Dentition/oral hygiene, foreign bodies, erythema, ulceration, dry mouth vs hypersalivation/drooling

Cardiovascular

- Murmurs, rubs, gallops, irregular beats, tachycardia/bradycardia, bruising, reproducible tenderness

Back

- Tenderness to palpation, step-offs/deformity, bruising, rashes

Pulmonary

- Clear to auscultation, diminished breath sounds, rales/rhonchi

Abdominal

- Tenderness to palpation, palpable liver edge, rigidity, normoactive bowel sounds

GU

- Signs of trauma/bruising, bleeding, discharge (look for signs of possible sexual abuse)

Extremities

- Scars, fresh open wounds to wrists/arms (self injurious behavior etc), track marks (sign of IV drug use), asterixis, muscle rigidity

Neuro exam

- Normal cranial nerve exam? Normal movement of all four extremities? Gait stable? Is sensation intact?

Mental Status Exam

Rule of Thumb: if you do not know the "fancy" word, just describe what you are seeing and hearing (example: psychomotor agitation = extreme fidgeting)

- Appearance (as mentioned previously, paying attention to grooming habits, cleanliness); physical movements—slowed or is the patient fidgety (psychomotor retardation and agitation, respectively)
- Alertness (level of consciousness, attentive to commands and questions)
- Speech (rate: slow, normal, fast; pressured quality?; volume: soft, normal or loud; long pauses that do not appear purposeful?)
- Behavior/Attitude: (pleasant, cooperative, forthright, appropriate given the circumstances? Or in the other extreme agitated, combative, withholding)
- Mood (this is the patient's stated feeling: angry, happy, sad, depressed, etc.) The patient may experience a feeling of shifting inappropriately between these moods or a mood not compatible given the present situation
- Affect (this is the outward expression: does the patient appear happy, sad, angry, etc.?) Note fluctuation/range

*Normal range or fluctuation is when the affect changes appropriately based on the conversation—person becomes sad looking when discussing mom's death but happy when noting a child's birth. "Lability" is when the fluctuations are rapid and do not appear to be based on thought content.

- Thought Process (logical comments and responses given present situation/questions versus tangential or even further on the spectrum, disorganized thoughts)
- Thought Content (this truly means the *content of the thought*: are there delusions or are there any thoughts of suicide or homicide "ideations")
- Perceptions (any form of hallucination present)
- Memory (three object assessment, asking the patient to remember three objects and repeat the names of those objects in 5 min)
- Ability to perform calculations (tests ability to focus on simple tasks, basic calculations, spelling backwards)
- Insight (does the patient have insight regarding the current situation)
- Judgement (reasonable response to posed questions about their actions for specific situations or scenario. Also discernible given reason behind presentation and known recent actions)
- Reasoning (interpretation of a given phrase or quote, responses range from concrete to abstract to bizarre/odd responses)

Quick Guide for the ED Interview

- Make sure that you are safe with the patient. If highly agitated, do not sit down. Keep at least two arm lengths (yours & the patient's) away. Offer medication if indicated. See the related chapter on agitation.
- Obtain collateral information from family, friends, paramedics, or police. Check the chart or police papers.
- Begin with the question (posed to yourself): "Could there be a medical cause for the patient's symptoms?" Do not label the patient as "psych" and triage to a low designation. Consider the full differential.

Tool

S	Senses (smell, sight, sound)
C	Complete head to toe (unclothed)
A	Appearance (general and psych specific, including affect)
N	Neuro (orientation, nerves, cognition, mental status)

Chapter 3
Laboratory Testing and Studies

Leslie S. Zun

Introduction

The work up portion of the medical evaluation of a patient presenting with behavioral or psychiatric symptoms is one means to determine if the presentation is caused by or exacerbated by a medical problem. The prime determinate of the causation is the history and physical and rarely the testing.

Testing

Essential testing on presentation

- Glucose and oxygenation level
- Vital signs when able to obtain safely

L.S. Zun, M.D., M.B.A. (✉)
Department of Emergency Medicine, Chicago Medical School, North Chicago, IL 60064, USA

Department of Emergency Medicine, Mount Sinai Hospital, New York, NY, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA
e-mail: zunl@sinai.org

K.D. Nordstrom, M.P. Wilson (eds.), *Quick Guide to Psychiatric Emergencies*,
https://doi.org/10.1007/978-3-319-58260-3_3,

Reasons to do testing

- Should be clinically based
- New onset of psychiatric illness
- Red flags of medical illness
  - Age > 45 years old
  - Exposure to toxins or drugs
  - Substance intoxication or withdrawal
  - No prior psychiatric/medical history
  - Abnormal vital signs
  - Physical examination findings
  - Cognitive deficits
  - Focal neurologic findings

Convenience for psychiatric facility (example: if you are admitting a patient and the facility does not have an on-sight lab)

Valuable tests

- Complete Blood Count (with differential only if directed)
- Electrolytes
- Comprehensive Metabolic Profile
- Urinalysis
- Pregnancy test
- Patients on meds that need blood levels such as Lithium, Depakote, Tegretol

Drug screen and alcohol level

- Only indication: Altered mental status without etiology
  - Many problems with drug and alcohol testing
    - Most drugs or their metabolites are positive for 1–3 days or longer after use
  - "Routine urine toxicologic screens for drugs in alert, awake, cooperative patients do not affect ED management and need not be performed as part of the ED assessment" (ACEP Guideline)

- Blood alcohol concentrations do not correlate with the degree of intoxication
- "The patient's cognitive abilities, rather than a specific blood alcohol level, should be the basis on which the clinicians begin the psychiatric assessment." (ACEP Guideline)
- Urine drug screens are limited
- Does not test for all drugs
- Interference with meds
- False negative and false positives
- Intoxication is a clinical diagnosis; not a lab diagnosis
- Clinical Assessment of intoxication
  - Level of consciousness
  - Cognitive function
  - Neurologic function
  - Coordination
  - Gait
  - Nystagmus

Tests of uncertain or no value

- CPK
- Troponin
- EKG

Advanced testing rarely indicated

- CT Scan Head
- Chest radiograph
- EEG
- Lumbar puncture

Rule of Thumb Regarding Patient Types and Need for Testing

New-onset psychiatric complaint-full battery of testing
Chronic mentally ill with the same complaint or presentation-no testing indicated

Pediatric patient treated the same as adult for testing
Geriatric patient-assess other medical problems
Substance Use Disorder and Psychiatric presentation-focused testing may be indicated
Medical Illness and Psychiatric presentation-focused testing may be indicated

Tool

Testing protocol to determine if testing is necessary

Medical clearance checklist	
Yes	No
1. Does the patient have new psychiatric condition?	
2. Any history of active medical illness needing evaluation?	
3. Any abnormal vital signs prior to transfer?	
4. Any abnormal physical exam (unclothed)?	
5. Any abnormal mental status indicating medical illness?	
If no to all of the above questions, no further evaluation is necessary.	
If yes to any of the above questions, tests may be indicated.	

References

1. Emembolu FN, Zun LS. Medical clearance in the emergency department: is testing indicated? Prim Psychiatry. 2010;17(6):29–34.
2. Nordstrom K, Zun LS, Wilson MP, Ng AT, Bregman B, Anderson EL. Medical evaluation and triage of the agitated patient: consensus statement of the American Association for Emergency Psychiatry project Beta medical evaluation workgroup. West J Emerg Med. 2012;13(1):3–10.
3. Zun LS, Downey LA. Application of a medical clearance protocol. Prim Psychiatry. 2007:65–9.

Chapter 4
Suicide Risk Assessment

Kimberly D. Nordstrom

Introduction

There are many different assessments for suicide in the literature and hospitals tend to use a combination of different assessments in creating their own. In the current environment of electronic health records, hospitals will commonly have a risk assessment of some sort embedded or available to add to a note template. Risk assessments on the inpatient unit may have a different focus than a risk assessment in the emergency department, where the decision is focused on whether the person requires admission or can safely be discharged.

Elements: There are four basic elements of risk assessments: current thoughts, past self-harm behaviors, risk factors, and protective factors.

K.D. Nordstrom, M.D., J.D.
Department of Human Services, Office of Behavioral Health, Denver, CO 80236, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA
e-mail: Nordstrom_kimberly@yahoo.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_4,

Current Thoughts: What does "SI" mean?

First, it is important to frankly ask if a patient is having any thoughts of life not worth living, wanting to be dead, and thoughts of killing self. If any of these are positive, ask for more information.

How to further question—"Thoughts of killing yourself":

Are thoughts vague?
Is there a plan?
Are means readily available?
Is there imminence?
Have they already begun to act or prepare for a plan?

Past self-harm behavior: "Not all cutting is the same."

A history of self-harm behavior can mean many different things. Was the behavior a way to self-soothe, a rehearsal, or an aborted attempt? All of these could lead to significant harm but the intent of each is different. If a clinician only asks "have you ever hurt yourself before," you will catch all of these acts and may assume that they were all related to a failed suicide attempt.

How to question—"History of hurting yourself":

Have you ever purposely hurt yourself? Why/what was the purpose?
Have you ever attempted suicide? Tell me what happened—means, if the attempt was purposely aborted, if the person is currently feeling the same way as previously.

Risk Factors: These should be separated as dynamic and static. A static risk factor is something that you cannot really change but informs overall risk of the class—divorced 60 year old men. Dynamic factors are those that you may help, even in the acute care setting.

Static risk factors:

Past history of suicide attempts
History of mental health or substance issues
Family history of suicide attempts
History of abuse

Chronic illness/pain
Recent discharge from a psychiatric unit

Dynamic risk factors:

Current thoughts of suicide
Intoxication
Acute pain
Current issue in personal relationship (break up, family strife)
Feelings of hopelessness, worthlessness
Current depression, depression + anxiety
Recent loss: homelessness, employment, personal (separation or divorce)
Access to lethal methods (guns, cache of medications)

Protective Factors: Protective factors need to be asked and considered in overall risk but it is important not to imagine that 1 protective factor negates 1 risk factor; all factors needs to be considered together.

**Religious beliefs; this can go either way so you have to ask specifically about the patient's belief system and how it relates to suicide (some believe in a 'forgiving God who will understand', others believe that 'suicide is the ultimate sin.')
Close relationship with natural supports (friends, family, other)
Belief that others rely on the person
Good relationship with treatment team—therapists, clinicians
Skill in problem-solving and frustration tolerance
Access to care and interest in receiving care

The lists above are not exhaustive. Each individual may be able to describe his or her own risk and protective factors, if asked.

Treatment interventions: Once you have asked the questions about current thoughts, past behaviors, risk factors and protective factors, the next step is to take this information

and determine risk. It is usually fairly simple to determine high and low risk, with obvious interventions. The tough job, after assessing 'moderate' risk, is determining next steps. Begin with acute-care treatment interventions. After any intervention, reassess risk.

Targeting dynamic risk factors:

Intoxicated patient: giving the patient time to sober then reassess risk (though remember: sober does not necessarily equal safe)

Acute pain: treat the pain and underlying issue causing pain; refer for further treatment

Interpersonal issue: consider a family meeting (may want to have social work lead this)

Access to lethal means: set up a plan with patient AND family to have means removed (example would be to have a family member or friend take guns out of the home)

Current depressed mood: supportive and solution-focused therapy in acute setting; if able to be discharged, quick follow up set with primary care or mental health

Next Steps

Low risk:

- Connect with current treatment team and coordinate follow up
- Have the patient create a safety plan while in the ED
- Give the patient information as to what to do if suicidal thoughts return (depends on locality)
  - Crisis phone numbers
  - Addresses for crisis stabilization units/walk-in centers
  - Return to the ED

Moderate risk:

Try targeting risk factors.

If a psychiatric consult team is available, ask for a consultation for further treatment options
May consider time for observation (if there was an acute issue, such as a loss or intoxication)
Have the patient work on a safety plan, to include natural supports
If able to discharge (with a very firm safety plan), coordinate with outpatient and give crisis information, as noted above
If patient still feels suicidal, may need to admit into the hospital

High risk:

Begin treatment as noted above (moderate risk) while awaiting admission

Tool

Safety plans come in all shapes and sizes—one easy way to conceptualize a safety plan is to think of "Red Light, Yellow Light, Green Light." Even when someone is very overwhelmed, this concept is easy for them to digest. If your hospital or clinic does not have a ready-made safety plan available, simply take a piece of paper and write out those few words with space between the color of light.

Explanation to patient:

GREEN LIGHT: "Next to the green light, write those things that you can do when you're feeling depressed that help buoy your mood and keep you from having suicidal thoughts. Do you like to watch certain shows, call friends, take a bath…what helps?"

YELLOW LIGHT: "Next to the yellow light, write activities you can do when you are feeling really down and even having vague suicidal thoughts like 'sometimes I wish I didn't exist.' When you feel like this, can you call a close friend, a supportive family member, or a counselor? Are there

activities that can soothe the mind—like journaling? If the feelings worsen, could you call the National Suicide Prevention Hotline: 1-800-273-8255 or our local crisis hotline _____?"

RED LIGHT: [Suicidal thoughts are changing from vague to actionable] "Let's talk about what can keep you safe when you are in crisis."

The point is for the patient to create his or her own safety plan with as much detail as possible. If a particular family or friend is associated with the plan, suggest having the patient call the family or friend while in the ED to discuss the plan.

References

1. For FREE pocket cards of the "Suicide Assessment Five-step Evaluation and Triage" (SAFE-T) http://www.integration.samhsa.gov/images/res/SAFE_T.pdf.

Section II
Psychiatric Illness

Section Editor: Caitlin A. Kieltyka, M.D.

Chapter 5
Agitation

Caitlin A. Kieltyka and Kimberly D. Nordstrom

Introduction

Patients with agitation are frequently encountered in an emergency department (ED) or psychiatric emergency service (PES). Agitation is not a disease state but actually a cluster of symptoms. Core symptoms and signs of agitation generally include irritability, excessive or semi-purposeful motor activity (also known as "psychomotor agitation"), heightened responsiveness to internal and external stimuli, and an unstable course. Agitation can be associated with both psychiatric and non-psychiatric conditions. Until agitation is appropriately treated, evaluation and treatment of the underlying issue may be delayed.

C.A. Kieltyka, M.D. (✉)
Department of Psychiatry, School of Medicine, University of Colorado-Denver, Denver, CO 80045, USA
e-mail: Caitlin.Kieltyka.MD@gmail.com

K.D. Nordstrom, M.D., J.D.
Department of Human Services, Office of Behavioral Health, Denver, CO 80236, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_5,

Symptoms

Internal restlessness
Feeling out of control
Feeling irritable/argumentative
Heightened responsiveness to stimuli (internal and external)

Signs

Increased and excessive semi-purposeful motor activity (pacing, etc.)
Irritability
Verbal outbursts
Physical aggression

Life-Threatening Symptoms/Signs Related to Medical Causes

Symptoms

Loss of memory, disorientation
Severe headache
Extreme muscle stiffness or weakness
Heat intolerance
Unintentional weight loss
Psychosis (new onset)
Difficulty breathing

Signs

Abnormal vital signs: pulse, blood pressure, or temperature
Overt trauma
Anisocoria (unequal pupil size)
Slurred speech
Incoordination

Seizures
Hemiparesis

Differential: When a patient first arrives to the ED or PES and demonstrates agitation, it is helpful to think of agitation as falling into one of four main groups to guide immediate treatment. These include agitation associated with delirium; agitation due to intoxication; agitation associated with psychosis; and undifferentiated agitation (see "Tool" at end of chapter). After immediate treatment, further workup can be performed to determine the underlying cause of agitation. An extensive (though not exhaustive) differential of causes of agitation include the following:

Acute pain
Head trauma
Infection
Encephalitis
Encephalopathy
Toxins
Metabolic issue
Hypoxia
Hyperthyroidism
Neurologic disease
Supratherapeutic dosing of medications
Recreational drug intoxication
Recreational drug withdrawal
Exacerbation of primary psychiatric disorder

Treatment

Acute:

- In all cases, verbal de-escalation techniques should be tried.
- In order to determine best pharmacological treatment.
  - If the agitation is associated with delirium and alcohol or benzodiazepine withdrawal is not suspected, consider

medical causes first in the ED setting. Common causes include hypoglycemia, hypoxia, head injury, or thyroid disease. Treat the underlying cause first before giving medications (best evidence is for oral risperidone and oral olanzapine).

- If the agitation is associated with delirium and this is likely due to alcohol or benzodiazepine withdrawal, benzodiazepines are the first line treatment.
- If the agitation is associated with stimulant intoxication, benzodiazepines are also first line.
- If the agitation is because of a CNS depressant (like alcohol), protect the airway first. Non-pharmacologic interventions such as simply turning down the lights are first line. If medications are needed, first-generation antipsychotics like haloperidol are likely safer than benzodiazepines.
- If the agitation is associated with psychosis in a patient with a known psychiatric disorder, second-generation antipsychotics are preferred (best evidence for risperidone or olanzapine). If the home medication (anti-psychotic) is known, consider dosing with this medication.
- If the cause of the agitation is unknown and there is no psychosis, treat like alcohol or benzodiazepine withdrawal.
- If the cause of the agitation is unknown and there is psychosis, treat as if the patient had a known psychiatric disorder.
- Restraints are occasionally needed if the agitation is severe and the patient is actively trying to hurt self or others. Restraints should be used as a last resort only and avoided if at all possible. If used, frequent reassessment and patient debriefing is necessary.

Long-term treatment: The long-term treatment is really one of prevention. Agitation is an acute condition but the disease state that produces agitation may be chronic. If the agitation is related to a chronic illness, the treatment for the primary illness may need to be modified to help prevent breakthrough symptoms.

Tool

Protocol for Treatment of Agitation

References

1. Lindenmayer JP. The pathophysiology of agitation. J Clin Psychiatry. 2000;61(Suppl 14):5–10.
2. Nordstrom K, Zun LS, Wilson MP, et al. Medical evaluation and triage of the agitated patient: consensus statement of the American Association for Emergency Psychiatry Project BETA Medical Evaluation Workgroup. West J Emerg Med. 2012;13(1):3–10.
3. Wilson MP, Pepper D, Currier GW, et al. The psychopharmacology of agitation: consensus statement of the American Association for Emergency Psychiatry Project BETA Psychopharmacology Workgroup. West J Emerg Med. 2012;13(1):26–34.

Chapter 6
Anxiety

Caitlin A. Kieltyka and Kimberly D. Nordstrom

Introduction

Anxiety is defined as the psychological and physiological manifestations of excessive worry. While anxiety is a normal reaction that all humans experience, it can become problematic if the anxiety overcomes one's ability to function. Generalized Anxiety Disorder is characterized by excessive worry more days than not for a period of at least 6 months. A Panic Attack is characterized by an abrupt surge in intense fear or discomfort that usually peaks within minutes and includes a host of physiological and psychological symptoms such as palpitations, diaphoresis, chest pain, a fear of losing control and/or a fear of dying. A Panic Attack can be a solitary presenting symptom or can be part of another psychiatric illness such as Panic Disorder, Generalized Anxiety Disorder, or Major Depressive Disorder.

C.A. Kieltyka, M.D. (✉)
Department of Psychiatry, School of Medicine, University of Colorado-Denver, Denver, CO 80045, USA
e-mail: Caitlin.Kieltyka.MD@gmail.com

K.D. Nordstrom, M.D., J.D.
Department of Human Services, Office of Behavioral Health, Denver, CO 80236, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_6,

Signs/Symptoms

The Diagnostic Statistical Manual-5 of the American Psychiatric Association lists the following sign and symptoms:

- **Generalized Anxiety Disorder**
  - Excessive anxiety and worry occurring more days than not for at least 6 months, about a number of events or activities
  - The individual finds it difficult to control the worry
  - The anxiety and worry are associated with three (or more) of the following symptoms
    - Restlessness or feeling keyed up or on edge
    - Being easily fatigued
    - Difficulty concentrating
    - Irritability
    - Muscle tension
    - Sleep disturbance
- **Panic Attack/Panic Disorder**
  - Panic Attack
    - Abrupt surge of anxiety with at least four of the following symptoms
      - Palpitations
      - Diaphoresis
      - Trembling or shaking
      - Feeling short of breath
      - Sensation of choking
      - Chest pain or discomfort
      - Nausea or abdominal distress
      - Feeling dizzy or light-headed
      - Chills or heat sensations
      - Paresthesia (numbness or tingling)
      - Derealization (feelings of unreality) or depersonalization (not feeling like oneself)
      - Fear of losing control
      - Fear of dying

  - Panic Disorder
    - Requires recurrent panic attacks along with at least one of the following lasting for at least 1 month following a panic attack
      - Persistent concern or fear of additional panic attacks or their consequences
      - A significant change in behavior related to the attacks, such as avoiding places or actions such as exercise

Differential

The differential is large. A broad risk of categories and representative samples are used here.

- Psychiatric
  - Social Anxiety Disorder (anticipatory anxiety focused on upcoming social situations)
  - Obsessive-Compulsive Disorder (intrusive and unwanted thoughts, urges or images as opposed to excessive worry about future events)
  - Posttraumatic Stress Disorder (where symptoms that occur following a trauma [E.g. nightmares, flashbacks, re-experiencing] are the primary symptom and anxiety only occurs in the context of this disorder)
  - Adjustment Disorder (anxiety occurs in response to a stressor within 3 months of the onset of that stressor and does not persist more than 6 months after the termination of the stressor or its consequences)
  - Depressive Disorders, Bipolar Disorders, Psychotic Disorders (where mood or psychotic symptoms are the primary symptom and anxiety only occurs in the context of those disorders)
- Non-psychiatric
  - Substance/medication induced (psychostimulants, levothyroxine, albuterol, steroids, caffeine, illicit stimulants, marijuana)
  - Endocrine: pheochromocytoma, hyperthyroidism, thyrotoxicosis

  - Cardiac: arrhythmias, angina, myocardial infarction
  - Pulmonary: pulmonary embolism, COPD, asthma
  - Neurologic: seizure

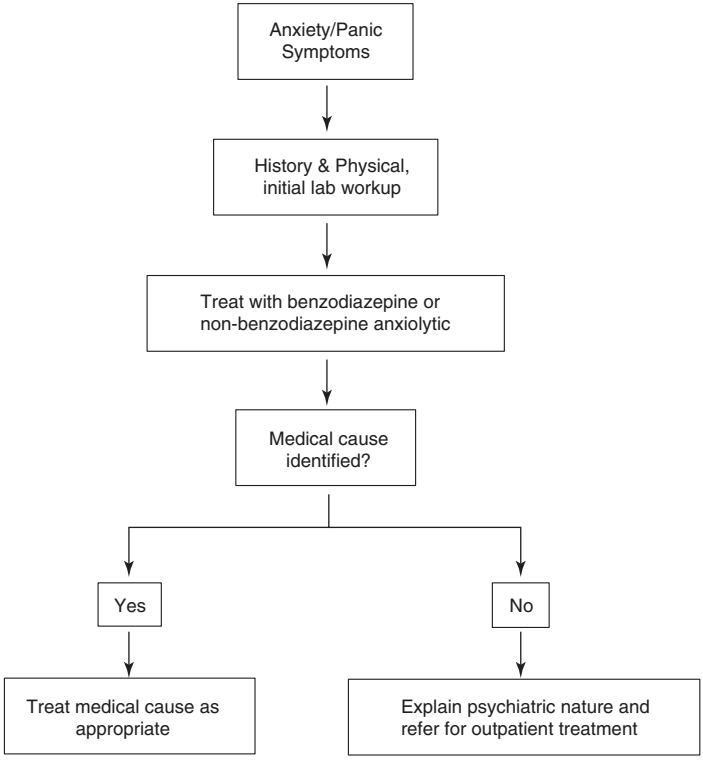
Initial Workup

- HPI:
  - Actual symptoms
  - Precipitating or exacerbating events
  - Duration
  - Timing
  - Severity
  - Be sure to utilize collateral sources of information (family members, friends) if available
  - Be sure to not dismiss physical complaints as "just anxiety," as the patient may have a serious medical condition that is making them anxious
- Suicide Risk (Suicide Risk Assessment, see Chap. 4)
- Substance use (including caffeine)
- Current medication list, as well as supplements
- Past psychiatric and medical history
- Family psychiatric and medical history
- Physical Exam:
  - Full system exam including Mental Status Exam
  - Review vital signs
- Laboratory evaluation should be directed based on history, physical exam, vital signs and differential diagnosis. Common studies to narrow the differential include:
  - TSH
  - Urine toxicology
  - EKG
  - EEG (rarely used in the ED setting)

Treatment

- Initial treatment should focus on reassuring and calming the patient, though this will likely not prevent recurrence and return to the ED
  - Can use a benzodiazepine such as lorazepam or diazepam
  - If concerned about substance use, could use a non-benzodiazepine anxiolytic such as diphenhydramine, hydroxyzine, or propranolol
- Explanation of the psychiatric etiology of the symptoms
  - Try something like "your symptoms are a physical manifestation of psychological anxiety and stress" and put into context of patient's current psychosocial stressors
  - Try to avoid using phrases that are akin to "it's all in your head" as this could be experienced as disparaging
- Secondary treatment could include teaching the patient relaxation techniques. A quick an easy technique such as diaphragmatic breathing could be taught. (See Tool 2 at the end of this chapter)
- Explore the patient's sleep hygiene. A pattern of disrupted sleep can be related to an increase in daytime anxiety. Elements of sleep hygiene include:
  - Setting regular sleep and wake times
  - No caffeine in the hours before bed
  - No vigorous exercise in the hours before bed (stimulates body)
  - Relaxation activities prior to bed: warm shower, reading, etc.
- Referral for psychiatric outpatient treatment and/or therapy to direct patient toward definitive treatment and thus decrease recurrence and return to the ED

Tool



Tool #2

DEEP BREATHING MADE SIMPLE:

Ask the patient to show how he or she breathes when trying to relax. In most cases, the breathing will be fast and you will see the shoulders rise. Point this out to the patient. Now place your hand on your abdomen (showing a patient how to correctly breathe goes a long way). Do 2 or 3 slow, deep, diaphragmatic breaths. Point out that your hand is moving out and in. Have the patient practice this a few times with you (make sure the patient places a hand on his or her abdomen). Point out that the hand is correctly moving out and in and the shoulders are not moving. That's it. You're done.

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
2. Buccelletti F, Ojetti V, Merra G, Carrocia A, Marsiliani D, Mangiola F, Calabò G, Iacomini P, Zuccalà G, Franceschi F. Recurrent use of the emergency department in patients with anxiety disorder. Eur Rev Med Pharmacol Sci. 2013;17:100–6.

Chapter 7
Catatonia

Caitlin A. Kieltyka and Kimberly D. Nordstrom

Introduction

Catatonia is a condition marked by changes in muscle tone or activity. Negative symptoms (like lack of movement) are often quickly recognized as catatonia but purposeless movements and other agitated behaviors are commonly missed. The incidence of agitation is unknown, as it is often unrecognized. There are three types of catatonia and patients may move between types. Catatonic types include (1) withdrawn or retarded, (2) excited, and (3) malignant. Malignant catatonia is the most severe type and is considered a medical emergency. Catatonia may be caused by a primary psychiatric illness or medical condition (such as neurological disorders, metabolic disorder, rheumatological disorders, and infections).

C.A. Kieltyka, M.D. (✉)
Department of Psychiatry, School of Medicine, University of Colorado-Denver, Denver, CO 80045, USA
e-mail: Caitlin.Kieltyka.MD@gmail.com

K.D. Nordstrom, M.D., J.D.
Department of Human Services, Office of Behavioral Health, Denver, CO 80236, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_7,

Though historically associated with Schizophrenia, catatonia is probably most often caused by mood disorders (depression and mania) and medical/neurological causes.

The Diagnostic Statistical Manual-5 of the American Psychiatric Association lists the following sign and symptoms, noting that three or more are required for the diagnosis:

Symptoms/Signs

Stupor
Cataplexy (full muscle weakness while conscious)
Waxy flexibility
Mutism
Negativism (resistance of external suggestions or internal stimuli, such as hunger)
Posturing
Mannerism (idiosyncrasy)
Stereotypy (persistent repetition of a movement or sound)
Agitation, not influenced by external stimuli
Grimacing
Echolalia (meaningless repetition of another person's vocalizations—sounds, phrases, parts of words or words)
Echopraxia (meaningless repetition of another's movements)

Life-Threatening Symptoms/Signs (Associated with Malignant Catatonia)

Fever (exclude infection first)
Autonomic instability
Labile blood pressure
Tachycardia
Tachypnea
Diaphoresis
Rigidity
Delirium

Differential Diagnosis

Neuroleptic malignant syndrome
Malignant hyperthermia
Thyroid storm
Infection
Metabolic derangement
Neurological abnormalities

Initial Workup

- HPI:
  - Actual symptoms
  - Precipitating or exacerbating events
  - Duration
  - Timing
  - Severity
  - Be sure to utilize collateral sources of information (family members, friends) if available
- Suicide Risk (Suicide Risk Assessment, see Chap. 4)
- Substance use (including caffeine)
- Current medication list, as well as supplements
- Past psychiatric and medical history
- Family psychiatric and medical history
- Physical Exam:
  - Full system exam including Mental Status Exam
  - Review vital signs
- Laboratory evaluation
  - No confirmatory labs; labs are used to identify underlying disorders
  - Simple test for withdrawn or agitated catatonia is a "test dose" of lorazepam: symptoms/signs should normalize quickly, but will return.

Treatment

Withdrawn and Agitated Catatonia

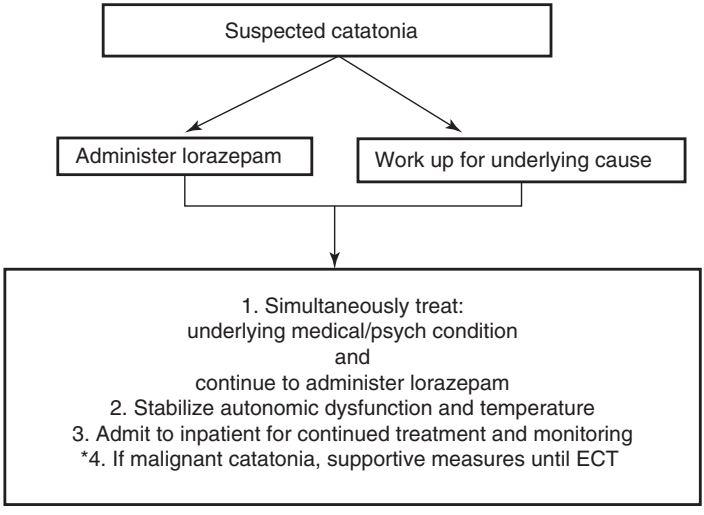
- Lorazepam (effective doses range from 6–21 mg over 24 h)
- 2nd line: Electroconvulsive Therapy (ECT)

Malignant Catatonia

- ED Management
  - Focus is on airway protection, autonomic stability, fever reduction
  - Supportive: compression stockings, passive range of motion
- Admission to hospital for electroconvulsive therapy (first line treatment); though may treat with benzodiazepines until ECT is available.

Avoid antipsychotics as they may worsen catatonia and may induce neuroleptic malignant syndrome.

Tool



References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
2. Seethalakshmi R, Dhavale S, Suggu K, et al. Catatonic syndrome: importance of detection and treatment with lorazepam. Ann Clin Psychiatry. 2008;20(1):5–8.
3. Kroll KE, Kroll DS, Pope JV, Tibbles CD. Catatonia in the emergency department. J Emerg Med. 2012;43(5):843–6.

Chapter 8
Depression

Caitlin A. Kieltyka and Kimberly D. Nordstrom

Introduction

The experience of depressed mood is not, in and of itself, a psychiatric or medical issue. It is when the experience is persistent and disrupts functioning of the individual that it should become a focus for further exploration and treatment. The term depression can mean many things, and only some patients will meet criteria for Major Depressive Disorder. Depressed mood may be caused by a reaction to stress or grief, a primary psychiatric disorder, a side effect of certain medications, and as a symptom of many illnesses and disease processes. It is important to go beyond asking about mood. Most of the psychiatric and medical causes can be easily

C.A. Kieltyka, M.D. (✉)
Department of Psychiatry, School of Medicine, University of Colorado-Denver, Denver, CO 80045, USA
e-mail: Caitlin.Kieltyka.MD@gmail.com

K.D. Nordstrom, M.D., J.D.
Department of Human Services, Office of Behavioral Health, Denver, CO 80236, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_8,

differentiated if a thorough history is obtained. Also, it is paramount to also ask about anxiety (as depression and anxiety are often co-occurring and lead to a higher likelihood of suicide) and about suicidal thoughts.

Signs/Symptoms of Major Depression

The Diagnostic Statistical Manual-5 of the American Psychiatric Association lists the following sign and symptoms:

- Depressed mood or irritability
- Anhedonia (decreased enjoyment or pleasure in otherwise pleasurable activities)
- Trouble with concentration
- Decreased motivation
- Feelings of worthlessness or helplessness
- Psychomotor changes (agitation or retardation): objective activation or slowing of movement

Vegetative Symptoms:

- Trouble with sleep (insomnia/hypersomnia)
- Change in appetite (increased or decreased); may lead to weight changes
- Decreased energy; feelings of fatigue

Thoughts of suicide or recurrent thoughts of death

Differential

Primary Psychiatric: For all diagnoses of depressive disorders, there must be impairment in function for the individual. Medical causes (including substances) for the depression must first be ruled out or treated.

- Bereavement: simple versus persistent and complex
- Persistent Depressive Disorder (Dysthymic Disorder): duration of at least 2 years

Adjustment disorder with depressed mood: occurs within 3 months of the onset of a stressor; once the stressor has ended, the symptoms should extinguish by 6 months

Major Depressive Disorder (MDD): criteria need to be present for at least 2 weeks

Bipolar Disorder, most recent episode depressed: at least 2 weeks of meeting full MDD criteria, while also having a history of mania or hypomania

Schizoaffective Disorder, Depressive Type: at least 2 weeks of meeting full MDD criteria and a period of psychosis with no mood symptoms

Schizoaffective Disorder, Bipolar Type, most recent episode depressed: at least 2 weeks of meeting full MDD criteria, history of manic/hypomanic episodes and a period of psychosis with no mood symptoms

Borderline Personality Disorder: marked instability of mood—dysphoria to euphoria, each mood episode usually lasting hours and rarely, days.

Premenstrual Dysphoric Disorder: changes in mood and irritability that occurs 1 week prior to menses and diminishes with the start of menses

General Medical Conditions/Medical Etiology

Substances:

Intoxication: depressed mood may occur during intoxication of depressants/sedatives

Withdrawal: depressed mood may occur with withdrawal, especially of stimulants (dopamine wash out)

Persistent: may have persistent depressed mood due to lasting biological effects of the drug and to the social/occupational/other effects of long-term use

Medications: many medications have been associated with mood changes, this is detailed elsewhere

Primary medical:

Medical causes of depressed mood is extensive and listed below by category only:

- Delirium
- Dementia
- Neurological conditions: (Inherited and Acquired)
  - Neurodegenerative Disorders
  - Infectious
  - Vascular
  - Inflammatory
  - Neoplastic and paraneoplastic
  - Traumatic brain injury
- Chronic Pain
- Sleep Disorders
- Endocrine Disease (such as hypothyroidism)
- Metabolic Disorders (such as Vitamin B12 deficiency, diabetes)
- Cardiovascular Disease

Initial Workup:

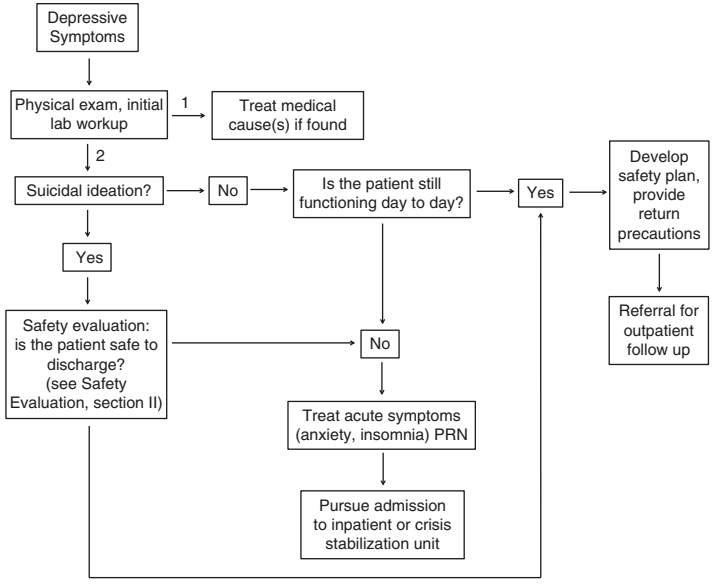
- HPI:
  - Actual symptoms
  - Precipitating or exacerbating events
  - Duration
  - Timing
  - Severity
  - Be sure to utilize collateral sources of information (family members, friends) if available
- Suicide Risk (Suicide Risk Assessment, see Chap. 4)
- Substance use (both acute use and chronic use are important)
- Current medication list, as well as supplements
- Past psychiatric and medical history
- Family psychiatric and medical history

- Physical Exam:
  - Full system exam including Mental Status Exam
  - Review vital signs
- Laboratory evaluation: all studies should be directed by differential diagnoses

Treatment

- Treatment of depression in the Emergency Department is dependent on severity of depression (is the patient able to continue to work and function day to day) and risk assessment (does the patient have an elevated imminent risk to harm self, is the patient able to safety plan; see Elements of a Thorough Risk Assessment chapter)
- If psychiatric hospitalization is warranted, would defer medication management to accepting psychiatric team, but can treat symptoms PRN (i.e. medications for acute anxiety or insomnia)
  - Inpatient, Crisis Stabilization Units, or Step-Down units can be considered for disposition, depending on level of severity and safety assessment
  - If the plan is inpatient but it appears the patient will be boarding for a significant period, can consider starting a low dose selective serotonin reuptake inhibitor. If anxiety is present, consider starting at half normal dose, as SSRIs can worsen anxiety on the short-term
- If psychiatric hospitalization is not warranted, plan for outpatient follow up coordination (social work would be able to help with this); give specific return precautions (i.e. if suicidal ideation returns or worsens)
- Would not recommend prescribing an antidepressant from the emergency department as these medications can have significant side effects (including precipitating mania or suicidal ideation in some cases) unless there is (confirmed) close follow-up. If an antidepressant is prescribed, also make sure to clearly state conditions that should warrant return to the ED.

Tool



Tool #2

Mnemonic Device for Depression

"SIG E CAPS"

Sleep disturbance (usually decreased—trouble with sleep initiation or maintenance but can also have marked increase of sleep)

Interest (loss of interest in things that were previously enjoyable)

Guilt sensation and worthlessness

Energy loss and fatigue

Concentration problems

Appetite problem (usually decreased with marked weight loss but could also be significantly increased)

Psychomotor agitation or retardation

Suicidality

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
2. Butler C, Zeman AZJ. Neurological syndromes which can be mistaken for psychiatric conditions. J Neurol Neurosurg Psychiatry. 2005;76(Suppl I):i31–i3.

Chapter 9
Mania

Caitlin A. Kieltyka and Kimberly D. Nordstrom

Introduction

Mania is the pathognomonic feature of Bipolar I Disorder and is characterized by changes in mood, behavior, sleep, energy and cognition. Manic symptoms can cause significant disruption in patients' lives, including impacting ability to maintain employment and interpersonal relationships. Mania can lead to potentially devastating personal and financial consequences and requires careful evaluation and management.

C.A. Kieltyka, M.D. (✉)
Department of Psychiatry, School of Medicine, University of Colorado-Denver, Denver, CO 80045, USA
e-mail: Caitlin.Kieltyka.MD@gmail.com

K.D. Nordstrom, M.D., J.D.
Department of Human Services, Office of Behavioral Health, Denver, CO 80236, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_9,

Signs/Symptoms

The Diagnostic Statistical Manual-5 of the American Psychiatric Association lists the following sign and symptoms, noting that three or more are required for the diagnosis (four are required if the mood is only irritable). The symptoms must be present for most of the day for at least 7 days, or any amount of time if the patient has been psychiatrically hospitalized.

- Abnormally elevated, expansive or irritable mood
- Inflated self-esteem or grandiosity
- Decreased need for sleep (tireless insomnia)
- More talkative than usual or pressure to keep talking
- Flight of ideas or racing thoughts
- Distractibility
- Increase in goal-directed activity or psychomotor agitation
- Excessive involvement in activities that have a high potential for painful consequences (spending significant amounts of money, sexual risk-taking)

Differential

- Psychiatric
  - Hypomanic Episode (symptoms must be present for most of the day for 4 days, generally less severe than Manic episode)
  - Generalized Anxiety Disorder
  - Panic Disorder
  - ADHD
  - Personality Disorder with rapid change in mood (E.g. affective instability of Borderline Personality Disorder)
- Non-psychiatric
  - Substance-induced
    - Prescription medications: psychostimulants, steroids
    - Recreational substances: amphetamine, cocaine, alcohol, phencyclidine

  - Cushing's Syndrome
  - Multiple Sclerosis
  - Traumatic brain injury
  - Cerebrovascular accident
  - Electrolyte abnormalities
  - Hyperthyroidism
  - Dementia
  - Delirium
  - Akathisia: medication side-effect

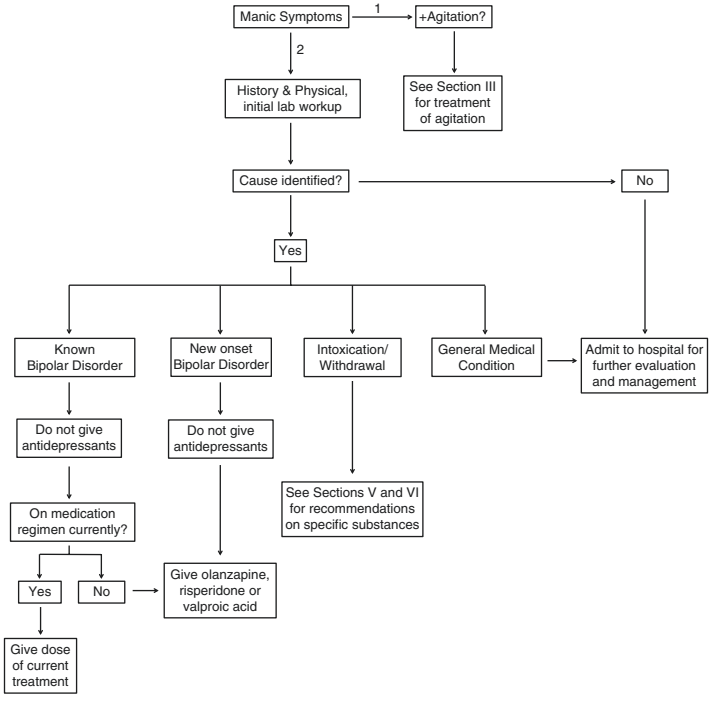
Initial Workup

- HPI:
  - Actual symptoms
  - Precipitating or exacerbating events
  - Duration
  - Timing
  - Severity
  - Be sure to utilize collateral sources of information (family members, friends) if available
- Suicide Risk (Suicide Risk Assessment, see Chap. 4)
- Substance use (including caffeine)
- Current medication list, as well as supplements
- Past psychiatric and medical history
- Family psychiatric and medical history
- Physical Exam:
  - Full system exam including Mental Status Exam
  - Review vital signs
- Laboratory evaluation should be directed based on history, physical exam, vital signs and differential diagnosis. Common studies to narrow the differential include:
  - TSH
  - CBC
  - BMP
  - Urine toxicology (if the patient is denying substance use but substance use is suspected)

Treatment

- If the patient is primarily agitated, see "Agitation" for treatment recommendations
- If the manic symptoms are found to be substance-induced, see corresponding chapters for specific substance treatment recommendations
- Do not give antidepressants to a patient if there is concern for mania
- If the patient has a known Bipolar Disorder and is on a medication regimen currently, could give dose of current regimen. There is controversy as to how to discontinue an antidepressant. If the patient is on an antidepressant, it is probably most wise to begin a taper rather than abruptly discontinue. The exception is the self-tapering fluoxetine.
- If the patient is not on a current medication regimen, could consider loading dose of valproic acid (30 mg/kg), especially if the patient is suspected to remain in the emergency environment for a significant period of time
- Could consider initiation of an antipsychotic that has been found to be helpful for manic phase of bipolar disorder such as olanzapine

Tool



Tool #2

Mnemonic Device for Mania

"DIG FAST"

Distractibility and easy frustration

Indiscretion and erratic disinhibited behavior

Grandiosity

Flight of ideas

Activity is significantly increased

Sleep need is decreased (total sleep decreased without daytime fatigue)

Talkativeness is greatly increased (hyperverbal, pressured)

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
2. Kemp DE, Johnson E, Wang WV, Tohen M, Calabrese JR. Clinical utility of early improvement to predict response or remission in acute mania: focus on olanzapine and risperidone. J Clin Psychiatry. 2011;72(9):1236–41.

Chapter 10
Psychosis

Caitlin A. Kieltyka and Kimberly D. Nordstrom

Introduction

Psychosis is a condition broadly defined as a loss of touch with reality. Its presentation includes hallucinations, delusions, and disorganization of thought and behavior. Most clinicians will encounter patients with psychosis, so it is important to be familiar with the workup and acute management of this disorder. Psychosis can be related to psychiatric conditions such as mood or psychotic disorders, or nonpsychiatric conditions such as general medical conditions and intoxication or withdrawal states.

C.A. Kieltyka, M.D. (✉)
Department of Psychiatry, School of Medicine,
University of Colorado-Denver, Denver, CO 80045, USA
e-mail: Caitlin.Kieltyka.MD@gmail.com

K.D. Nordstrom, M.D., J.D.
Department of Human Services, Office of Behavioral Health,
Denver, CO 80236, USA

American Association for Emergency Psychiatry,
Parker, CO 80134, USA

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_10,

Signs/Symptoms of Primary Psychosis

- **Hallucinations:** false auditory, visual, tactile, gustatory (taste), or olfactory perceptions (auditory and visual hallucinations are most common in primary psychosis)
- **Delusions:** fixed false beliefs
- **Disorganization:** evidenced through mental status exam
  - Thought blocking (long pauses in speech)
  - Tangentiality (answering questions in a partially related way)
  - Perseveration (intense focus on one topic or idea with difficulty moving on to a new topic)
  - Grossly disorganized behavior (e.g. purposeless actions such as picking up and setting down a piece of paper)
- **Responding to internal stimuli**: patient is observed to be talking or whispering to herself or himself (usually represents a patient's response to hearing voices or seeing things)
- **Negative Symptoms**: diminished emotional expression, avolition (decreased motivation to do purposeful actions)
- **Agitation/Aggression**: not a sign of psychosis per se but the psychotic patient may become agitated because of the psychotic experience or from the underlying disorder
- **Absence of physical findings**

Differential Diagnosis

Primary Psychiatric

- Schizophrenia
- Schizoaffective Disorder
- Major Depressive Disorder with Psychotic Features
- Bipolar Disorder with Psychotic Features
- Delusional Disorder
- Brief Psychotic Disorder
- Schizophreniform Disorder
- Schizotypal Personality Disorder
- Obsessive-compulsive and related disorders

General Medical Conditions

- Toxic state (intoxication, withdrawal)
- Delirium
- Seizure/post-ictal state
- Major neurocognitive disorder (Dementia)
- Electrolyte imbalances
- Hepatic encephalopathy
- Brain tumor
- Head injury
- Infection
- Sexually transmitted infections: HIV, Syphilis
- Hyperthyroidism or hypothyroidism
- Medications (e.g. steroids, interferon)

Initial Workup

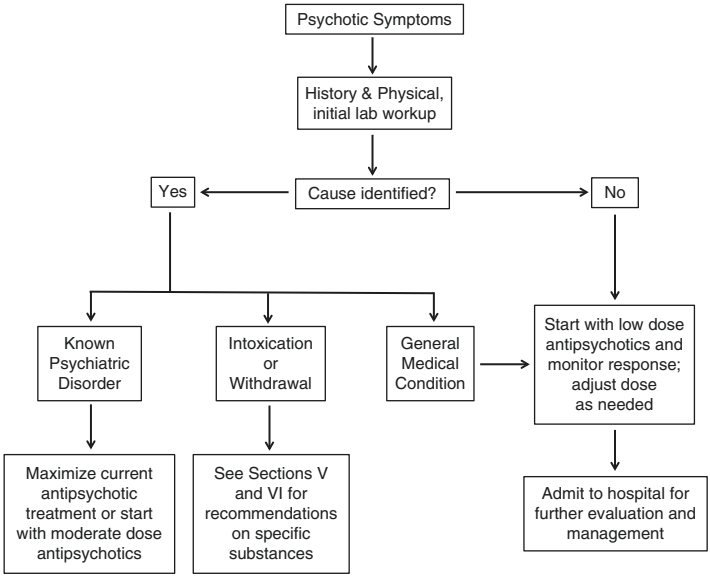
- HPI:
  - Actual symptoms
  - Precipitating or exacerbating events
  - Duration
  - Timing
  - Severity
  - Be sure to utilize collateral sources of information (family members, friends) if available
- Suicide Risk (Suicide Risk Assessment, see Chap. 4)
- Substance use (including caffeine)
- Current medication list, as well as supplements
- Past psychiatric and medical history
- Family psychiatric and medical history
- Physical Exam:
  - Full system exam including Mental Status Exam
  - Review vital signs
- Laboratory evaluation should be directed based on history, physical exam, vital signs and differential diagnosis. Common studies to narrow the differential include:
  - CBC
  - TSH

  - BMP (can expand to CMP if hepatic etiology is suspected)
  - Urine toxicology (if the patient is denying substance use but substance use is suspected)
  - Further *directed* workup to consider:
    - HIV/RPR
    - CT/MRI
    - CXR
    - EEG

Treatment

- Antipsychotics
  - If patient has a known psychiatric disorder, would try to maximize current antipsychotic treatment first, or start with moderate dose antipsychotics
    - E.g. olanzapine 10 mg, risperidone 1–2 mg, haloperidol 5–10 mg, perphenazine 8–12 mg
  - If new onset psychosis or unclear cause, would start with low dose and increase slowly based on response
    - E.g. olanzapine 5–10 mg, risperidone 0.5–1 mg, haloperidol 2.5–5 mg, perphenazine 4–8 mg
  - If psychosis is related to substance intoxication or withdrawal, see specific substance chapter for recommendations
  - Use atypical antipsychotic when trying to minimize risk of extrapyramidal side effects (akathisia, Parkinsonism)
  - Use typical antipsychotic when trying to minimize metabolic side effects (weight gain, increased Hgb A1c)
- Benzodiazepines, if agitated and not otherwise contraindicated
- Discontinue offending agent if known

Tool



References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
2. Schwartz P, Case MW, Berezin J. The psychotic patient. In: Riba MB, Ravindranath D, Winder SW, editors. Clinical manual of emergency psychiatry. 2nd ed. Arlington, VA: American Psychiatric Association; 2016. p. 105–31.

Section III
Medical Disorders

Section Editor: Christopher S. Sharp, M.D.

Chapter 11
Adrenal Crisis/Adrenal Insufficiency

Christopher S. Sharp and Michael P. Wilson

Introduction

Adrenal crisis is an acute deterioration in a patient with adrenal insufficiency, which is usually manifested as hypovolemic shock. Adrenal insufficiency may be secondary to infections especially tuberculosis, metastasis of cancer, bilateral adrenal hemorrhage, or bilateral adrenalectomy (primary or Addison's disease); pituitary tumors, pituitary surgery, pituitary radiation, or head trauma (secondary); or long-term steroid use (glucocorticoid-induced). Adrenal crisis may be the first presentation in up to 50% of patients with adrenal insufficiency.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_11,

If a patient has unexplained circulatory collapse, hypotension, vomiting, or diarrhea, the diagnosis of primary adrenal insufficiency should be considered. In patients with hyperpigmentation, hyponatremia, hyperkalemia, acidosis, and hypoglycemia, the diagnosis should be strongly suspected.

Symptoms

Weakness
Acute abdominal pain out of proportion to physical exam
Nausea
Disorientation
Vomiting/diarrhea

Signs

Acidosis (low bicarbonate)
Hypotension
Fever
Altered level of consciousness
Hyponatremia
Hyperkalemia (unreliable)
Decreased serum bicarbonate
Hyperpigmentation of skin (hyperpigmentation of oral mucous membranes is considered pathognomonic)
Unexplained hypotension unresponsive to vasopressors

Life-Threatening Symptoms/Signs

Symptoms

Extreme disorientation

Signs

Hemodynamic instability or shock

Differential

Adrenal crisis may often be confused with sepsis, especially if the precipitating event was infection. Other diagnoses can usually be excluded with a careful medication history, including asking about any recent medication or dose changes. However, other causes to consider include infection, thyroid storm, non-convulsive status epilepticus, neuroleptic malignant syndrome, or excited delirium syndrome.

Testing

This is useful to identify the precipitating cause, and should include basic labs and cultures. Testing for suspected adrenal insufficiency may include serum cortisol level and plasma ACTH. Decreased cortisol and increased ACTH confirm the diagnosis.

Treatment

Treatment involves intravenous fluid resuscitation, with at least 1 L of normal saline over the first hour, and steroid replacement. Typically, intravenous hydrocortisone 100 mg is administered in the emergency department, with tapering doses of steroids administered in the hospital.

Potential Pitfall

Cortisol can suppress antidiuretic hormone. Particularly if normal saline is being administered (which contains 154 mEq/L of sodium), hyponatremia may be corrected too rapidly.

Tool

If any of the following, suspect adrenal crisis
Hypotension refractory to vasopressors
Vomiting/diarrhea
Hyponatremia/hyperkalemia/hypoglycemia/acidosis
Hyperpigmentation (especially oral)

If adrenal crisis is suspected, send plasma ACTH and serum cortisol. Administer IV normal saline. Administer IV hydrocortisone 100 mg.

References

1. Puar THK, Stikkelbroeck NMML, Smans LCCJ, Zelissen PMJ, Hermus RMM. Adrenal crisis: still a deadly event in the 21st century. Am J Med. 2015;129(3):339-e1.
2. Husebye ES, Allolio B, Arit W, et al. Consensus statement on the diagnosis, treatment, and follow-up of patients with primary adrenal insufficiency. J Intern Med. 2014;275:104–15.

Chapter 12
Chronic Pain

Christopher S. Sharp

Introduction

Chronic pain (CP) exacerbation is a common reason for emergency department visits, accounting for 10–16% of all visits, though it is not the ideal setting for management. CP is pain that endures past the normal time of healing though time frames vary by definition. Exacerbations of CP are typically referred to as "break through pain" or "flare ups." The etiologies of chronic pain are myriad but, in general, pain can be placed into one of four mechanisms: neuropathic, musculoskeletal, inflammatory, and visceral. In the United States opioid pain prescriptions have increased dramatically since the late 1990s, unfortunately so too has opioid misuse and overdose. The American College of Emergency Physicians (ACEP) policy recommends against the routine prescription of opioids for acute CP exacerbations.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA
e-mail: chris.s.sharp@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_12,

Symptoms

Site-specific pain
Anxiety
Depression

Signs

Hypertension
Tachycardia
Point tenderness

Life-Threatening Symptoms/Signs

Pain itself is not life-threatening however is associated with elevated risk of suicide and proper safety assessment should be performed (see related chapter).

Differential

- Acute injury
- Depression
- Anxiety
- Opioid use disorder

Initial Workup

Careful history
A thorough physical exam is the most important component of the evaluation
Functional assessment
Record review (including state prescription drug monitoring programs where available)
Urine drug screening

Imaging to rule out acute injury (when indicated)
Documented monitoring of pain and functional level at regular intervals

Treatment

In CP the goal of treatment is generally pain reduction rather than elimination of pain entirely.

- Ascertain whether non-opioid analgesics will be adequate for initial pain management
- Comorbid anxiety may be treated with low-dose benzodiazepines (such as lorazepam or diazepam) or hydroxyzine 25–50 mg
- If practical, consider past prescription patterns and existing pain contracts
- If opioids are prescribed at discharge, prescribe the lowest practical dose for a limited duration
- Educate the patient on non-medicinal forms of treatment
  - Consider review of acceptable exercises for musculoskeletal pain, if appropriate

Tool

Biological mechanisms of pain

	Example	**Description**	**Exam**
Neuropathic	Diabetic neuropathy	• Burning, stinging • "Pins and needles"	• Allodynia • Hyperalgesia • +/– Sensory or motor deficits
Musculoskeletal	Degenerative joint disease	• Aching and dull • +/– Sharp pain with movement or weight bearing	• Localized tenderness • Joint deformity • +/– Weakness

	Example	Description	Exam
Inflammatory	Rheumatoid arthritis	• Sharp, lancinating • +/− Pain increase with movement	• Tenderness • Erythema • Edema • Increased warmth • +/− Signs of infectious process
Visceral	Endometriosis	• Varied	• Varied

References

1. Calati R, Bakhiyi CL, Artero S, Ilgen M, Courtet P. The impact of physical pain on suicidal thoughts and behaviors: meta-analyses. J Psychiatr Res. 2015;71:16–32.
2. Cantrill SV, Brown MD, Carlisle RJ, Delaney KA, Hays DP, Nelson LS, O'Connor RE, Papa A, Sporer KA, Todd KH, Whitson RR. Clinical policy: critical issues in the prescribing of opioids for adult patients in the emergency department. Ann Emerg Med. 2012;60(4):499–525.
3. Ducharme J. Acute pain and pain control: state of the art. Ann Emerg Med. 2000;35(6):592–603.
4. Institute for Clinical Systems Improvement. Health care guideline: determine the pain generator. 7th ed. 2016. https://www.icsi.org/guideline_sub-pages/pain/determine_the_pain_generator/. Accessed 14 Dec 2016.
5. McLeod D, Nelson K. The role of the emergency department in the acute management of chronic or recurrent pain. Australas Emerg Nurs J. 2013;16(1):30–6.
6. Olsen JC, Ogarek JL, Goldenberg EJ, Sulo S. Impact of a chronic pain protocol on emergency department utilization. Acad Emerg Med. 2016;23(4):424–32.
7. Poulin PA, Nelli J, Tremblay S, Small R, Caluyong MB, Freeman J, Romanow H, Stokes Y, Carpino T, Carson A, Shergill Y. Chronic pain in the emergency department: a pilot mixed-methods cross-sectional study examining patient characteristics and reasons for presentations. Pain Res Manag. 2016;2016:3092391.

Chapter 13
Delirium

Christopher S. Sharp

Introduction

Delirium is a clinical diagnosis characterized by transient, usually reversible, cerebral dysfunction which develops over a short period of time. It can occur at any age but is seen more often in individuals over the age of 65 as well as those with compromised mental status, such as patients with dementia. The cardinal features of delirium include decreased attention, and "waxing and waning" orientation. Diagnosis is obtained from a careful history, from collateral sources such as family and caregivers, and cognitive exam. There is no laboratory test or study to diagnose delirium but studies can help identify the underlying cause. There are various screening tools for delirium which can be helpful in the ED.

There are two forms of delirium: hypoactive, presenting as withdrawn (very similar to retarded catatonia) and hyperactive, presenting as agitated, with heightened arousal and hypervigilance.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA
e-mail: chris.s.sharp@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_13,

Symptoms

Confusion
Agitation
Emotional changes: Irritability, depression, anxiety
Hallucinations, illusions (misinterpreting something in the environment)
Delusions

Signs

Disturbance in attention and awareness
Cognitive: disorientation, memory issues, fluctuating consciousness
Neurological: motor abnormalities, dysarthria, tremor, asterixis (depending on cause)

Life-Threatening Symptoms

These are based on the underlying disorder.

Differential

Dementia
Psychosis (several diagnoses)
Depression

Causes

Infection
Withdrawal
Acute Metabolic
Trauma
CNS pathology
Hypoxia

Deficiencies (vitamin, calories, fluids)
Endocrine
Acute vascular (MI)
Toxins
Heavy metals

Testing

Testing should be directed by key findings in history and physical exam and may include:

Complete blood cell count with differential
Complete metabolic panel—for electrolyte abnormalities, diabetic ketoacidosis, hyperosmolar nonketotic states, liver and renal failure.
Thyroid Stimulating Hormone
Vitamin B-12, folate, calcium
Urinalysis to rule out urinary tract infection (especially in elderly patients)
Blood cultures
Drug screen including alcohol level
Arterial blood gas
Neuroimaging
  CT scan
  MRI
Chest X-ray

Treatment

- Airway management, circulatory support, IV fluids, as indicated
- Aggressive cooling measures as needed
- Treat with first or second generation antipsychotic if delirium is not caused by alcohol or benzodiazepine withdrawal.
- Avoid benzodiazepine use unless alcohol or benzodiazepine withdrawal is suspected
- Treatment of underlying cause

Tool [2]

The Confusion Method Assessment Instrument
Acute Onset

1. Is there evidence of an acute change in mental status from the patient's baseline?

Inattention [*The questions listed under this topic were repeated where applicable]

2. A. Did the patient have difficulty focusing attention, for example, being easily distractible, or having difficulty keeping track of what was being said?
   Not present at any time during interview.
   Present at some time during interview, but in mild form.
   Present at some time during interview, in marked form.
   Uncertain.
   B. (If present or abnormal) Did this behavior fluctuate during the interview, that is, tend to come and go or increase and decrease in severity?
   Yes.
   No.
   Uncertain.
   Not applicable.
   C. (If present or abnormal) Please describe this behavior

Disorganized Thinking

3. Was the patient's thinking disorganized or incoherent, such as rambling or irrelevant conversation, unclear or illogical flow of ideas, or unpredictable switching from subject to subject?

Altered Level of Consciousness

4. Overall, how would you rate the patient's level of consciousness?

   Alert (normal)
   Vigilant (hyperalert, overly sensitive to environmental stimuli, startled very easily).

Lethargic (drowsy, easily aroused).
Stupor (difficult to arouse).
Coma (unarousable).
Uncertain

Disorientation

5. Was the patient disoriented at any time during the interview, such as thinking that he or she was somewhere other than the hospital, using the wrong bed, or misjudging the time of day?

Memory Impairment

6. Did the patient demonstrate any memory problems during the interview, such as inability to remember events in the hospital or difficulty remembering instructions?

Perceptual Disturbances

7. Did the patient have any evidence of perceptual disturbances, for example, hallucinations, illusions, or misinterpretations (such as thinking something was moving when it was not)?

Psychomotor Agitation

8. Part 1

   At any time during the interview, did the patient have an unusually increased level of motor activity, such as restlessness, picking at bedclothes, tapping fingers, or making frequent sudden changes of position?

Psychomotor Retardation

8. Part 2

   At any time during the interview, did the patient have an unusually decreased level of motor activity, such as sluggishness, staring into space, staying in one position for a long time, or moving very slowly?

Altered Sleep-Wake Cycle

9. Did the patient have evidence of the sleep-wake cycle, such as excessive daytime sleepiness with insomnia at night?

References

1. Inouye SK, VanDyck CH, Alessi CA, Balkin S, et al. Clarifying confusion: the confusion assessment method. A new method for detection of delirium. Ann Intern Med. 1990;113:941–8.
2. Inouye SK. The short Confusion Assessment Method (Short CAM): training manual and coding guide. Boston: Hospital Elder Life Program; 2014.
3. National Institute for Health and Clinical Excellence. Clinical guideline 103—Delirium. London: NICE; 2010.

Chapter 14
Dementia

Christopher S. Sharp

Introduction

Dementia is a syndrome of acquired cognitive deficits which interfere with executive functioning that typically occurs in older adults. There are many different potential etiologies including Alzheimer's disease, vascular dementia, and Lewy body dementia. Though significant neurocognitive decline is the hallmark of dementia neuropsychiatric symptoms often drive clinical presentation. These symptoms can occur with all forms of dementia and include depression, anxiety, agitation, psychosis, disinhibition, and sleep disturbance. Diagnosis is obtained through a careful history, collateral sources, cognitive exam, and neuroimaging. Dementia is often first recognized in emergency settings.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA
e-mail: chris.s.sharp@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_14,

Symptoms

Cognitive impairment (impaired recall, impaired judgment, etc.)
Agitation
Emotional changes: Irritability, depression, anxiety
Hallucinations, illusions (misinterpreting something in the environment)
Delusions

Signs

Cognitive: memory issues, impaired judgment
Psychiatric: apathy, agitation

Life-Threatening Symptoms

These are based on the underlying cause of dementia.

Differential

Delirium
Psychosis (several diagnoses)
Depression (common cause of "reversible dementia")
Anxiety
Sensory deficit
Metabolic conditions (hypothyroidism)
Nutritional conditions (B12 deficiency)
Substance use disorder
Medication side effects

Testing

Brief cognitive testing such as the Mini-Mental Status Exam (MMSE), the Montreal Cognitive Assessment (MOCA), and the Six-Item Screen (SIS) can help

both identify dementia and differentiate from delirium. Consciousness, attention, and orientation are typically preserved until late in the progression of dementia. Those who screen positive for cognitive impairment should be referred for neuropsychiatric testing.

Laboratory testing for potentially reversible causes: thyroid stimulating hormone, vitamin B12 level, HIV testing, and rapid plasma reagin (RPR) to rule out syphilis.

Urine and blood drug screening.

Neuroimaging.

CT scan.

MRI.

May also consider evaluation of hearing and vision.

Additional labs such as metabolic panels, urinalysis, and complete blood count can be useful in differentiating from delirium.

Treatment

- Supportive measures (airway management, circulatory support, IV fluids) as indicated
- Non-pharmacological behavioral interventions such as light and music therapy
- There are no FDA approved medications for agitation or psychosis in patients with dementia; however, psychotropic medications are often used off-label for this purpose. Atypical antipsychotics are the most frequently utilized. Use of antipsychotics should be low-dose and short-term if possible. Be aware that antipsychotics carry a Black Box Warning in geriatric patients due to the risk of increased mortality.
- Benzodiazepines are an alternative treatment option for agitation but should be avoided if possible due to the increased fall risk and worsening cognitive impairment.
- Treatment of any identified reversible causes.

Tool

Dementia versus delirium tool

	Dementia	Delirium
Onset	Insidious	Acute/abrupt
Course	Slow deterioration	Fluctuating "waxing & waning"
Attention	Intact early; worsens	Impaired
Awareness	Intact until late	Impaired
Orientation	Intact early; worsens	Impaired
Memory	Poor short-term memory	Poor working memory; impaired immediate recall
Delusions/ thought process	Fixed delusions/ impoverished thoughts	Changing and short-lived delusions/disorganized thoughts
Sleep pattern	Sleep-wake reversal common	Fragmented; disrupted

References

1. Callahan CM, Unverzagt FW, Hui SL, Perkins AJ, Hendrie HC. Six-item screener to identify cognitive impairment among potential subjects for clinical research. Med Care. 2002;40(9):771–81.
2. Downing LJ, Caprio TV, Lyness JM. Geriatric psychiatry review: differential diagnosis and treatment of the 3 D's-delirium, dementia, and depression. Curr Psychiatry Rep. 2013;15(6):1–10.
3. Hirschman K, Paik HH, Pines JM, McCusker CM, Naylor MD, Hollander JE. Cognitive impairment among older adults in the emergency department. West J Emerg Med. 2011;12(1):56–62.
4. Koenig AM, Arnold SE, Streim JE. Agitation and irritability in Alzheimer's disease: evidenced-based treatments and the black-box warning. Curr Psychiatry Rep. 2016;18(1):1–10.

Chapter 15
Encephalitis

Christopher S. Sharp and Michael P. Wilson

Introduction

Encephalitis is defined as an inflammatory disease of the brain with neurologic dysfunction as a result of direct infection of the brain parenchyma, a post-infectious process, or non-infectious process (such as NMDA-receptor antagonism). Neurologic dysfunction may manifest as seizures, decreased level of consciousness, ataxia, or other focal neurologic dysfunction, and importantly is not due to another infectious process of the brain. Encephalitis is sometimes confused with encephalopathy, which is defined as a state of altered mental status without inflammation.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_15,

As the exact etiologic agent often varies between geographic locations, travel history is important to obtain. Other key history points: time since onset of symptoms, rashes, sick contacts with similar symptoms, animal/insect contact (especially ticks), sexual contacts, vaccinations, and immunosuppressed status (medication or advanced HIV).

Symptoms

Headache
Personality changes
Neck stiffness
Nausea
Photophobia

Signs

Altered mental status
Fever (not universally true)
Disorientation/altered level of consciousness
Seizures
Focal neurological deficits
Vesicular rash (this is unreliable). In Ramsay-Hunt syndrome, the rash may be inside the ear, so don't forget to check there.
Vomiting

Life-Threatening Symptoms/Signs

Symptoms

Marked personality changes

Signs

Dysphasia/speech impairment
High fever or other unstable vital signs
Prominent neurological deficits

Differential

Encephalitis may be difficult to definitively diagnosis given the myriad potential etiologies. The main determination is between encephalitis and meningitis.

Testing

- CT or MRI, lumbar puncture, and appropriate cultures are mandatory.
- On the lumbar puncture opening pressure is helpful and the following CSF tests should be ordered:
  - Cell count and differential
  - Glucose
  - Protein
  - Lactate
  - HSV-1 and HSV-2
  - VZV DNA
  - CMV DNA
  - VDRL
  - Gram stain/culture
  - Consider an India Ink stain or cryptococcal antigen if the CSF pressure is elevated, as this may help exclude other causes.
  - Consider adding polio, rabies, and oligoclonal bands if the patient has paralysis.
- Keep an extra tube of CSF in case additional studies are needed.

- LP results usually indicate CSF pleocytosis with a predominance of lymphocytes with an increased number of CSF erythrocytes. That can sometimes be confused with a traumatic tap. Generally, the CSF glucose to serum glucose ratio is normal, although slight decrease may sometimes be found.
- MRI may show HSV lesions of the brain more accurately than CT but is often harder to obtain. CT scans may indicate low-attenuation changes.

Treatment

Supportive Care

Treat with IV acyclovir at the dose of 10–15 mg/kg three times daily.

Higher doses should be considered in patients who are younger without renal impairment. Lower doses of acyclovir may also be effective, but have less evidence.

Acyclovir should be administered early in the course of the disease, often before the exact causative agent can be identified.

Typically, patients are hospitalized at a level of care where neurological checks can be performed often.

Consider antibiotics if bacterial infection cannot be ruled out.

Tool

- If signs/symptoms of meningitis or encephalitis, administer IV antibiotics, obtain CT/MRI, and perform lumbar puncture.
- Tips for distinguishing meningitis from encephalitis
  - CSF gram stain typically indicates organisms (although imperfect reference standard with sensitivities reported 60–90%)

- CSF lactate typically >4.0 mmol/L
- If uncertain if encephalitis, administer IV acyclovir at 10 mg/kg with IV antibiotics

References

1. Derber CJ, Troy SB. Head and neck emergencies: bacterial meningitis, encephalitis, brain abscess, upper airway obstruction, and jugular septic thrombophlebitis. Med Clin N Am. 2012;96:1107–26.
2. Studahl M, Lindquist L, Eriksson B-M, et al. Acute viral infections of the central nervous system in immunocompetent adults: diagnosis and management. Drugs. 2013;73:131–58.
3. Venkatesan A, Tunkel AR, Block KC, et al. Case definitions, diagnostic algorithms, and priorities in encephalitis: consensus statement of the International Encephalitis Consortium. Clin Infect Dis. 2013;57:1114–28.

Chapter 16
Head Injury

Christopher S. Sharp

Introduction

Head injury is defined by a history of blunt or penetrating trauma to the head with physical evidence of trauma. It is sometimes accompanied by loss of consciousness. Head injury is commonly seen in the emergency department, averaging more than two million visits annually in the United States. It ranges in severity from mild head wounds to debilitating, sometimes fatal brain injuries. Head injury is the leading cause of death in trauma. There are numerous potential etiologies including falls, assault, sports injuries, and motor-vehicle collisions. The most widely used means of classification is the Glasgow coma scale (GCS) with a score of eight or lower indicating severe injury.

C.S. Sharp, M.D.
Psychiatric Emergency Service,
Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA
e-mail: chris.s.sharp@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_16,

Signs/Symptoms

- Altered mental status
- Drowsiness
- Headache
- Physical evidence of head trauma

Life-Threatening Symptoms/Signs

- Coma
- Focal neurologic deficits
- Evidence of skull fracture
- Emesis (particularly multiple episodes)
- Seizures
- Leakage of cerebrospinal fluid from the nose or ears
- Unequal pupils
- Cushing's triad (irregular respirations, bradycardia, hypertension)
- Posturing (either decorticate or decerebrate)

Differential (depends largely on the severity of head trauma)

Coma (other sources)
Cerebral vascular incident
Delirium
Psychosis
Personality Disorder (if outward manifestation largely behavioral)

Testing

CT scan is the investigation of choice for head trauma. Measurement and monitoring of intracranial pressure (ICP) is often necessary for severe injuries. Elevation of

ICP above 19 mmHg is associated with poor outcomes and mortality.

Treatment

- Airway management and supportive care
- Admission to Neuro-ICU for moderate to severe injuries
- Neurosurgery consult when appropriate
- Monitoring and management of ICP
  - Interventions to lower ICP such as mannitol, mild (34–35 °C) hypothermia, and hyperventilation have inconclusive evidence
- Antiepileptic drugs carbamazepine and phenytoin may reduce early seizures though have not been shown to reduce late seizures, neurologic disability, or mortality

Tool

The Glasgow Coma Scale:

Eye Opening

Spontaneous	4
To speech	3
To pain	2
None	1

Best Verbal Response

Oriented	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
None	1

Best Motor Response	
Obeys commands	6
Localizes pain	5
Withdrawal (normal flexion)	4
Abnormal flexion (decorticate)	3
Extension (decerebrate)	2
None	1

A score of 13 or higher correlates with mild brain injury, 9–12 is mild injury, eight or less is severe brain injury.

References

1. Burgess S, Abu-Laban RB, Slavik RS, Vu EN, Zed PJ. A systematic review of randomized controlled trials comparing hypertonic sodium solutions and mannitol for traumatic brain injury implications for Emergency Department Management. Ann Pharmacother. 2016;50(4):291–300.
2. Gaw CE, Zonfrillo MR. Emergency department visits for head trauma in the United States. BMC Emerg Med. 2016;16(1):1.
3. Maconochie I, Ross M. Head injury (moderate to severe). BMJ Clin Evid. 2010;2010.
4. Sternbach GL. The Glasgow coma scale. J Emerg Med. 2000;19(1):67–71.

Chapter 17
Hepatic Encephalopathy

Christopher S. Sharp and Michael P. Wilson

Introduction

Hepatic encephalopathy (HE) is a brain dysfunction caused by liver disease. HE results in altered mental status, ranging from neurological or psychiatric symptoms to coma. The pathophysiology is complex and not well elucidated. HE is usually triggered by some event, thought to be (in order of frequency): infections, GI bleeding, diuretic overdose, or electrolyte disorder.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_17,

Symptoms

In mild form, hepatic encephalopathy can be largely nonspecific affecting only cognitive tests of attention and memory. In more severe form:

- Personality changes
- Lethargy
- Confusion

Signs

- Disorientation to time/date
- Motor movement abnormalities such as hyperreflexia, muscular rigidity, asterixis

Signs of severe liver disease include caput medusae (enlargement of paraumbilical veins), abdominal distention, jaundice, and scleral icterus (presence of jaundice in the eye).

Life-Threatening Symptoms/Signs

Symptoms

Extreme lethargy or somnolence

Signs

Coma
Intracranial hypertension and cerebral edema
Seizures

Differential

- Alcohol use
- Toxins
- Infection, especially spontaneous bacterial peritonitis
- Electrolyte or glucose disorders, including diabetic ketoacidosis
- Intracranial bleed
- Primary psychiatric disorders
- Dementia

Testing

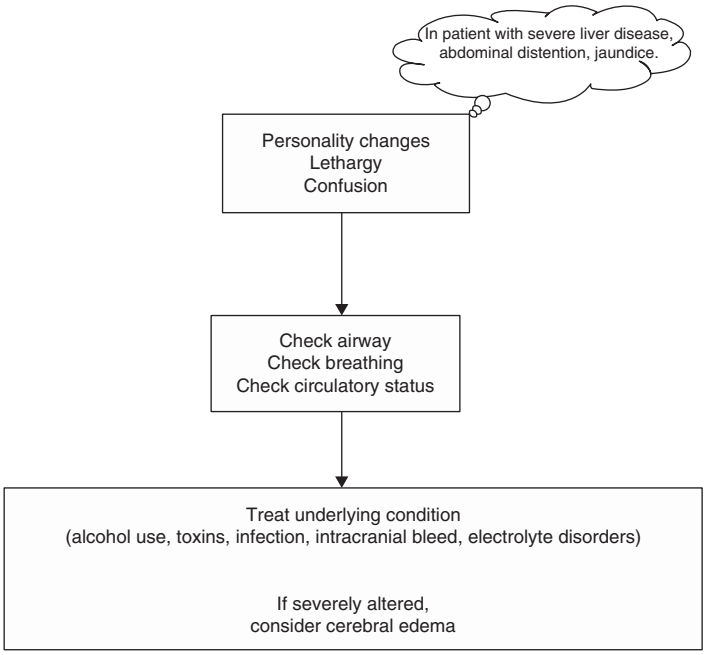
Ammonia is typically elevated, however, per 2014 guidelines, "High blood-ammonia levels alone do not add any diagnostic, staging, or prognostic value in HE patients." Still, a normal ammonia level should cast doubt on the diagnosis of HE.

Treatment

Treatment of hepatic encephalopathy is targeted to correcting the underlying problem. Lactulose 15–30 mL may be given either orally or rectally for hyperammonemia.

Tool

Encephalopathy



References

1. Vilstrup H, Amodio P, Bjaj J, Cordoba J, Ferenci P, Mullen KD, Weissenhorn K, Wong P. Hepatic encephalopathy in chronic liver disease: 2014 practice guidelines by the American Association for the Study of Liver Disease and the European Association for the Study of the Liver. Hepatology. 2014;60(2):715–35.

Chapter 18
Hypercalcemia

Christopher S. Sharp and Michael P. Wilson

Introduction

Hypercalcemia is the presence of elevated serum calcium, which typically ranges from 8.5 to 10.5 mg/dL. Hypercalcemia is most often caused by overactivity of the parathyroid glands or from cancer. Other causes of increased calcium include sarcoidosis, Paget disease, multiple endocrine neoplasia, tuberculosis, vitamin D toxicity, and certain medications. The effects of this increased calcium can be seen throughout the body and range from very mild to severe symptoms.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_18,

Symptoms

Mild forms are largely asymptomatic. In more severe hypercalcemia:

- Fatigue
- Depression
- Muscle cramps or weakness
- Paresthesias
- Constipation
- Abdominal pain

Signs

- Confusion
- Seizures
- Renal calculi
- Coma
- On EKG, shortened QTc interval and prolonged PR interval.
- Of note: Chvostek's sign (irritability of facial nerve when tapping over the region of the facial nerve) and Trousseau's sign (spasm induced by pressure over an extremity, typically seen when inflating a blood pressure cuff) are typically seen only in hypocalcemia, and should not be seen here.

Often remembered by the mnemonic "stones, bones, groans, psychiatric overtones."

Life-Threatening Symptoms/Signs

Symptoms

Severe muscle cramps

Signs

Altered mental status.
EKG Rhythm may indicate AV block in severe cases.

Differential

While hypercalcemia is a common laboratory error, it should never be ignored. If repeat testing confirms the elevated value, then further steps must be taken to determine the etiology. The vast majority of cases result from malignancy and hyperparathyroidism. Other etiologies include rhabdomyolysis, thiazide diuretics, and ingestion of calcium-containing products.

Testing

Blood chemistry is typically sufficient to show hypercalcemia. Testing should include potassium, as hypokalemia is often associated. As hyperparathyroidism is often responsible, PTH levels should be considered but may be unavailable in the acute setting.

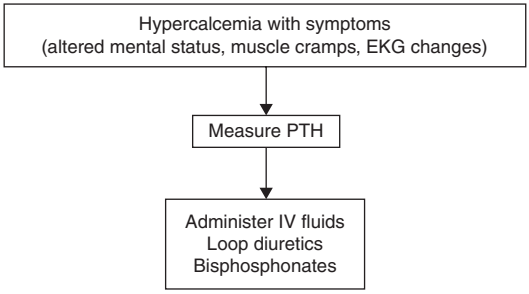
Treatment

Mild hypercalcemia (<12 mg/dL) often needs no treatment. Moderate to severe hypercalcemia should be treated first with aggressive intravenous fluids that are titrated to good urine output (typically 1–2 mL/kg per hour). When euvolemic, loop diuretics (such as furosemide) may be administered. Bisphosphonates and calcitonin which decrease bone resorption are first-line therapy, but may take several hours to work.

For patients that are seizuring or severely altered, treatment also involves supportive care for airway.

Tool

Hypercalcemia



References

1. Chang WT, Radin B, McCurdy MT. Calcium, magnesium, and phosphate abnormalities in the emergency department. Emerg Med Clin N Am. 2014;32(2):349–66.
2. Akeely Y, Vilke GM, Wilson MP. Psychiatric emergencies for clinicians: the ED management of hypercalcemia. In development.
3. Maier JD, Levine SN. Hypercalcemia in the intensive care unit a review of pathophysiology, diagnosis, and modern therapy. J Intensive Care Med. 2015;30(5):235–52. doi:10.1177/0885066613507530.

Chapter 19
Hypercortisolism (Cushing's Syndrome)

Christopher S. Sharp and Michael P. Wilson

Introduction

Hypercortisolism or Cushing's syndrome is the presence of elevated amounts of cortisol in the body. Corticotropin releasing hormone (CRH) is released in the hypothalamus, causing release of ACTH from the anterior pituitary. ACTH then acts on the adrenal glands (in the adrenal cortex) to produce cortisol. The most common cause of Cushing's syndrome is exogenous steroids. If elevated ACTH is produced by a pituitary tumor, the patient is instead said to have Cushing's disease.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_19,

The clinical syndrome caused by hypercortisolism is indistinguishable from metabolic syndrome, typically manifested as central obesity syndrome, insulin resistance (hyperglycemia), hypertension, and dyslipidemia.

Symptoms

Fatigue
Weakness
Irregular menstruation

Signs

Obesity
Hyperglycemia
Hypertension
Dyslipidemia
Striae (linear discolorations of skin most often seen in breasts, hips, thighs, buttocks, or abdomen)
Posterior cervical fat pad (buffalo hump)

Life-Threatening Symptoms/Signs

Symptoms

Extreme weakness

Signs

Marked hyperglycemia
Marked hypertension

Differential

Cushing's syndrome is indistinguishable from metabolic syndrome.

Testing

Plasma ACTH level can help identify Cushing's in the emergency setting.

For diagnosis, elevated midnight cortisol (obtained in a sleeping unstressed patient) is perhaps the earliest marker for Cushing's syndrome.

The overnight dexamethasone suppression test is most often used. In this test, a 1 mg dose of dexamethasone is given in the evening, with a measurement of serum cortisol in the morning. Although the diagnostic value of this test has been questioned, a level below 50 nmol/L is often used to exclude Cushing's.

Treatment

First-line treatment of Cushing's disease typically involves surgery, either of the pituitary or of the adrenal glands.

Medical therapies are available but typically not started in the emergency setting.

Management of Cushing's syndrome in the emergency setting is usually supportive. If the patient is taking exogenous steroids, these should be discontinued. The patient may need intravenous fluids and insulin if the hyperglycemia has caused dehydration.

Tool
Typical findings in Cushing's syndrome
Obesity
Hyperglycemia
Hypertension
Striae
Posterior cervical fat pad

If Cushing's syndrome is suspected, provide supportive care with insulin and intravenous fluids. A dexamethasone suppression test may be useful, but is typically not performed in the emergency setting.

References

1. Hamrahian AH, Yuen KCJ, Hoffman AR. AACE/ACE disease state clinical review: medical management of Cushing disease. Endocr Pract. 2014;20(7):746–57.
2. Lindholm J. Cushing's disease, pseudo-Cushing states and the dexamethasone test: a historical and critical review. Pituitary. 2014;17:374–80.
3. Raff H, Findling JW. A physiologic approach to diagnosis of the Cushing syndrome. Ann Intern Med. 2003;138:990–1.
4. Tritos NA, Biller BMK, Swearingen B. Management of Cushing disease. Nat Rev Endocrinol. 2011;7:279–89.

Chapter 20
Hypoglycemia

Christopher S. Sharp

Introduction

Hypoglycemia is a potentially life-threatening condition that results from low glucose.

Although ED visits for hypoglycemia declined from 2006 to 2011, events still occur frequently. Mild hypoglycemic events may occur as often as twice weekly in type 1 diabetes mellitus. Hypoglycemic events may occur as often in type 2 diabetes mellitus due to pharmacologic interventions. Hypoglycemia cannot be defined by its symptoms as they are triggered by the lower plasma glucose values following an initial hypoglycemic event. However, a workgroup for the American Diabetes Association/Endocrine Society notes that 70 mg/dL (3.9 mmol/L) is a reasonable alert value.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, Denver, CO, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA
e-mail: chris.s.sharp@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_20,

Symptoms

Weakness
Shakiness
Anxiety
Irritability
Hunger

Signs

Sweating
Confusion/disorientation
Agitation

Life-Threatening Symptoms/Signs

Extreme disorientation

Differential

Although low glucose is usually reliably tested by portable glucose monitors and laboratory testing, the exact etiology of the hypoglycemia is sometimes more difficult to elucidate. Some causes to consider include:

infection
improper use of diabetic agents
non-convulsive status epilepticus.

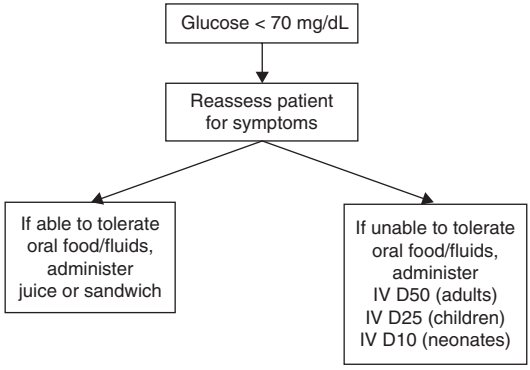
Testing

Measurement of serum glucose levels reliably confirms hypoglycemia.

Treatment

Treatment is supportive. If the patient is awake and able to take food orally, glucose-rich foods such as juice may be sufficient. If the patient is unable to tolerate oral food or fluids, intravenous D50 may be given in adults. In children, intravenous D25 is preferable, given usually 2 mL/kg. In neonates, administer D10 at 2 mL/kg.

Tool



References

1. Bonds DE, Miller ME, Dudi J, et al. Severe hypoglycemia symptoms, antecedent behaviors, immediate consequences and association with glycemia medication usage: secondary analysis of the ACCORD clinical trial data. BMC Endocr Disord. 2012;12(1):5. doi:10.1186/1472-6823-12-5.
2. Leese GP, Wang J, Broomhall J, et al. Frequency of severe hypoglycemia requiring emergency treatment in type 1 and type 2 diabetes. Diabetes Care. 2003;26:1176–80.

3. Seaquist ER, Anderson J, Childs B, et al. Hypoglycemia and diabetes: a report of a workgroup of the American Diabetes Association and the Endocrine Society. Diabetes Care. 2013;36:1384–95.
4. Wang J, Geiss LS, Williams DE, Gregg EW. Trends in emergency department visit rates for hypoglycemia and hyperglycemic crisis among adults with diabetes, United States, 2006–2011. PLoS One. 2015;10(8):e0134917. doi:10.1371/journal.pone.0134917.

Chapter 21
Hyponatremia

Christopher S. Sharp and Michael P. Wilson

Introduction

Hyponatremia is the presence of low amounts of serum sodium, usually defined as less than 135 mEQ/L.It can be further classified in relation to osmolality (hypotonic, isotonic, hypertonic) and volume status (hypovolemia, euvolemia, hypervolemia).

History should include medication use (especially diuretics), alcohol use (especially large quantities of beer), and substance use (especially MDMA or Ecstasy).

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, 777 Bannock St., Denver, CO 80204, USA

Department of Psychiatry, University of Colorado—Denver School of Medicine, Denver, CO, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_21,

Symptoms

In mild form, hyponatremia is largely asymptomatic.

- Polydipsia
- Muscle cramps
- Headaches

Signs

- Confusion
- Coma

*Pay close attention to indicators of volume status (mucous membranes, presence of JVD, lung crackles, pretibial edema) which may indicate hypervolemia.

Life-Threatening Symptoms/Signs

Symptoms

Severe muscle cramps

Signs

Altered mental status
Refractory seizures

Differential

- pseudohyponatremia

Pseudohyponatremia occurs when measured levels are low, but serum sodium is actually normal. Serum osmolality is

normal. This commonly occurs in hyperglycemia, and can be corrected using the formula: serum sodium = 1.6 (measured glucose in mg/dL—100).

Testing

Blood chemistry or a venous blood gas with electrolytes is sufficient to show hyponatremia. Serum osmolality should then be checked in order to classify the hyponatremia as hypotonic (dilutional). Urine sodium and urine creatinine are often useful for calculating fractional excretion of sodium (FENa), which is typically less than 1% unless the site of sodium loss is in the kidney. Measurement of TSH and pro-BNP may be considered in some patients to exclude other causes.

Treatment

Treatment of severe hyponatremia (typically 124 mEQ/L or below) is usually best done in the hospital in a monitored bed. In these patients, the rate of sodium correction should be less than 1 mEQ/h to prevent demyelination syndrome.

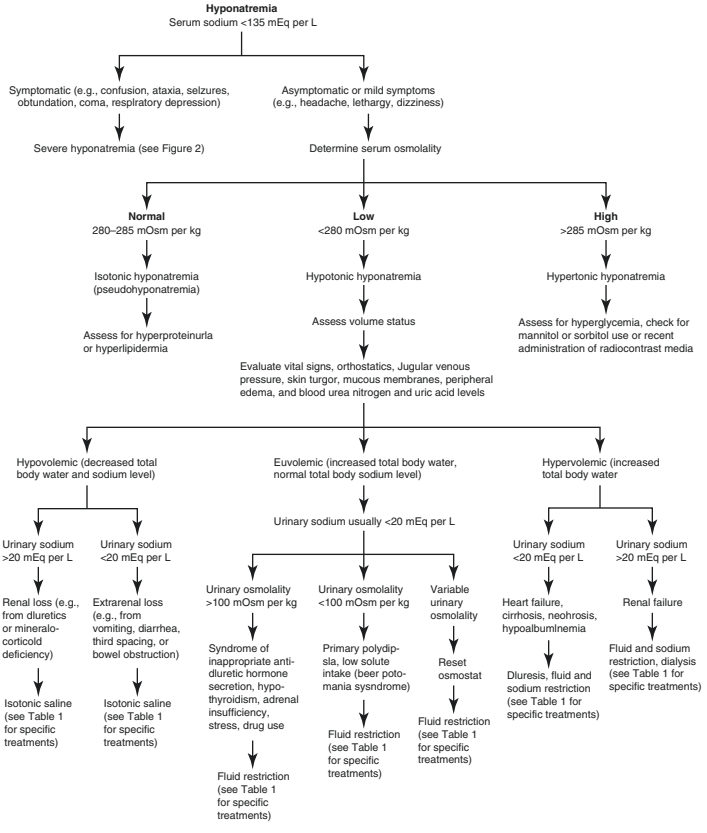
If the patient is seizuring or severely altered, administer 100 ml (up to 150 ml) of a bolus of 3% hypertonic saline to rapidly correct the sodium.

For patients that are not seizuring or severely altered, treatment is based on volume status.

- Hypovolemic hyponatremia (typically from volume depletion via vomiting, diarrhea, or increased diuretic use): treatment is with cautious intravenous fluids, being careful not to overcorrect the sodium level as above. Treat the underlying condition.
- Euvolemic hyponatremia is commonly caused by SIADH, but may also be caused by hypothyroidism or glucocorticoid deficiency.

Tool

Evaluation of Hyponatremia



From Braun MM et al. Diagnosis and management of sodium disorders: Hyponatremia and hypernatremia. AM Fam Physician. 2015; 91(5): 299-307

References

1. Vilstrup H, Amodio P, Bjaj J, Cordoba J, Ferenci P, Mullen KD, Weissenhorn K, Wong P. Hepatic encephalopathy in chronic liver disease: 2014 Practice Guidelines by the American Association for the Study of Liver Disease and the European Association for the Study of the Liver. Hepatol. 2014;60(2):715–35.
2. Braun, Barstow CH, Pyzocha NJ. Diagnosis and management of sodium disorders: Hyponatremia and hypernatremia. Am Fam Physician. 2015; 91(5):299–307.

Chapter 22
Hypothyroidism

Christopher S. Sharp and Kimberly D. Nordstrom

Introduction

Patients with hypothyroidism commonly present to the ED with a diverse range of physical and psychological complaints. This leads to a wide differential and can cause unnecessary testing if this diagnosis is not high on the list. In the most severe form, hypothyroidism can lead to myxedema coma, detailed in the chapter of that name.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, 777 Bannock St, Denver, CO 80204, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

K.D. Nordstrom, M.D., J.D. (✉)
Department of Human Services, Office of Behavioral Health, Denver, CO, USA

American Association for Emergency Psychiatry, PO Box 3948, Parker, CO 80134, USA
e-mail: Nordstrom_kimberly@yahoo.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_22,

Symptoms

General: fatigue, lethargy, cold sensitivity, weight gain
Neuro/muscular: memory issues, muscle weakness
Gastrointestinal: constipation
Hair/skin/nails: hair loss and thinning, dry skin, brittle nails
Genitourinary: changes in menstrual cycle
Psychological: depressed mood, anxiety, irritability

Signs

General: weight gain
Head/neck exam: loss of scalp hair, coarse facial expression, goiter
Neuro/muscular: psychomotor retardation (slowing of movements), hyporeflexia with delayed relaxation, ataxia, pain/numbness to limbs (peripheral neuropathy)
Gastrointestinal: abdominal distension
Skin/hair/nails: dry skin, loss of scalp hair, axillary and pubic hair
Extremities: pitting edema or non-pitting edema (myxedema pretibial)

Life-Threatening Symptoms/Signs

Related to Myxedema coma (please see Chap. 23)

Significant hypothermia
Impaired mental status (may be unconscious)
Mortality related to respiratory failure and sepsis

Differential

Thyroiditis
Thyroid lymphoma
Addison's Disease
Chronic fatigue syndrome
Euthyroid sick syndrome
Mononucleosis
Lithium-induce goiter
Menopause
Major depressive Disorder (or other depressive disorder)
Anxiety disorder
Severe symptoms: adrenal crisis, sepsis, multi-system failure

Causes

Autoimmune disease (Hashimoto's thyroiditis)
Iodine deficiency
Pituitary disorder
Post/peripartum hypothyroidism (produce antibodies to thyroid gland)
Secondary to treatment for hyperthyroidism: s/p surgery, radiation therapy
Response to medication--lithium

Laboratory Studies

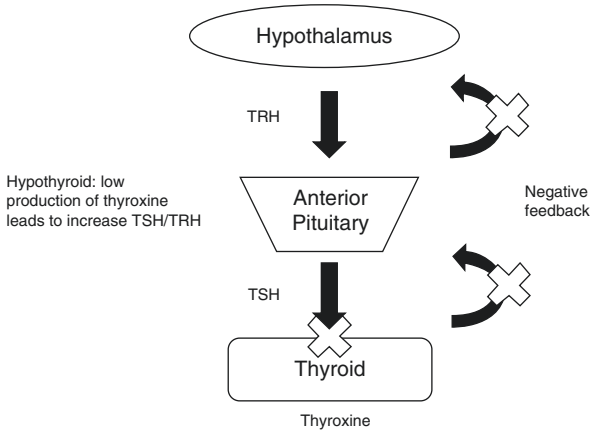
TSH
T3
Free T4

Treatment

- Replenish thyroid hormone (levothyroxine); alert patient to the fact that feelings of anxiety may occur with initiation of this hormone (known side effect)
- Educate the patient on the chronic nature of hypothyroidism and link the symptoms that have been experienced to this education
- Patient to follow up with primary care physician for titration of hormone

Tool

Hypothyroid



References

1. Garber JR, Cobin RH, Gharib H, Hennessey JV, Klein I, et al. Clinical practice guidelines for hypothyroidism in adults cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. Endocr Pract. 2012;18(6):988–1028.

Chapter 23
Myxedema Coma

Christopher S. Sharp and Kimberly D. Nordstrom

Introduction

Myxedema crisis or coma usually occurs after a major insult to the system, such as a myocardial infarct, cerebral vascular accident or sepsis, in the setting of hypothyroidism. Infections and sepsis are the leading precipitating factors. It is a life-threatening condition with high mortality rates. By definition, myxedema crisis/coma presents as impaired mental status (unconsciousness) and life-threatening signs related to the primary insult/crisis.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, 777 Bannock St, Denver, CO 80204, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

K.D. Nordstrom, M.D., J.D. (✉)
Department of Human Services, Office of Behavioral Health, Denver, CO, USA

American Association for Emergency Psychiatry, PO Box 3948, Parker, CO 80134, USA
e-mail: Nordstrom_kimberly@yahoo.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_23,

Life-Threatening Symptoms/Signs (Largely Related to Underlying Insult)

Neurocognitive: poor cognitive function, obtundation, lethargy, seizures, coma
Cardiovascular: bradycardia, hypotension, arrhythmias, cardiogenic shock, low cardiac output
Respiratory: hypoxia, hypercarbia, pleural effusion, pneumonia
Renal: hyponatremia, fluid retention
Gastrointestinal: constipation, paralytic ileus, toxic megacolon

Differential

Myocardial infarct
Cardiogenic shock
Thyroiditis
Adrenal crisis
Cardiovascular accident
Sepsis

Laboratory Studies

Thyroid Panels: TSH, T3, Free T4
Complete blood count
Complete metabolic panel
Arterial blood gas monitoring
Blood cultures
Creatine phosphokinase
Lactate dehydrogenase
Serum cortisol
Electrocardiogram
Directed radiographic studies
Other directed labs

Treatment

- airway management
- fluid management
- supportive care
  - rewarming (careful: vasodilation may precipitate hypotension)
  - correct associated hyponatremia and hypoglycemia
- Management of precipitating condition (CVA, sepsis, etc.)
- thyroid hormone replacement
  - American Thyroid Association recommends combination T3 (short term) and T4.
- If concomitant primary adrenal insufficiency, glucocorticoid therapy is indicated.
- admission to ICU

Tool

Common laboratory findings

		Notes
Sodium	Decreased	Tough situation: fluid supplementation for hypotension but fluid restriction for hyponatremia; do not right too quickly (myelinolysis)
Glucose	Decreased	
Creatine phosphokinase	Increased	
Lactate dehydrogenase	Increased	
Aspartate transaminase	Increased	
CO2 (ABG)	Increased	Check serial ABGs
Cortisol	Decreased	Related to primary or secondary adrenal insufficiency (would expect hyperkalemia and hypercalcemia); also thyroid hormone replacement may increase cortisol clearance

References

1. Jonklaas J, Bianco AC, Bauer AJ, Burman KD, Cappola AR, et al. Guidelines for the treatment of hypothyroidism: prepared by the american thyroid association task force on thyroid hormone replacement. Thyroid. 2014;24(12):1670–751.
2. Mathew V, Misgar RA, Ghosh S, Mukhopadhyay P, Roychowdhury P, et al. Myxedema coma: a new look into an old crisis. J Thyroid Res. 2011;2011:1–7.

Chapter 24
Non-Epileptic Seizures

Christopher S. Sharp

Introduction

Psychogenic nonepileptic seizures (PNES) are paroxysmal behaviors which resemble epileptic seizures however, unlike epilepsy, there is no electrophysiological correlate. While there is no clinical evidence for epilepsy, there is usually evidence for psychogenic factors which may have caused the seizure. The incidence of PNES is fairly low in the general population but may occur in up to 30% of patients referred to epilepsy monitoring units. PNES most commonly occur in women (80% of all cases) aged between 15–35 years. PNES lasting longer than 30 min are referred to as nonepileptic psychogenic status (NEPS) or "pseudo status" and represent the most common PNES emergency presentation. Failure to recognize PNES can result in

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, 777 Bannock St, Denver, CO 80204, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA
e-mail: chris.s.sharp@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_24,

unnecessary testing and treatment. In fact, an average of over $100,000 is spent on each patient with PNES before the diagnosis is made.

Signs/Symptoms

PNES can be indistinguishable from epileptic seizures and thus share many of the same signs and symptoms

- Mental status changes, dissociation in particular
- Convulsive motor activity (tonic contractures, clonic jerking, asynchronous body movements)
- Somatization or unexplained neurologic symptoms
- Progressive onset
- Waxing/waning course
- Abrupt termination

Life Threatening Signs/Symptoms

PNES are nonlethal; however patients should be screened for suicidality given likelihood of psychiatric comorbidities.

Differential

The primary differential is between PNES and epileptic seizures

Testing

A careful history can often yield many diagnostic clues for PNES:

- Trauma history (such as sexual or physical abuse)
- Dissociation
- Unexplained somatic symptoms
- Comorbid personality disorder (particularly borderline, avoidant, and dependent personality disorders)

If still differentiating from epileptic seizures the workup is quite similar

- Laboratory studies: basic metabolic panel and electrolytes, complete blood count, liver function tests, antiepileptic drug (AED) levels
- Electroencephalography (EEG)
- Imaging (must balance the value of imaging with the cost of delaying treatment)

Prolonged video EEG is the gold standard for diagnosing PNES though it is not readily available in most emergency departments. Unfortunately, EEG is of limited utility without direct observation.

Treatment

- The first step in treatment once PNES is diagnosed is to openly and nonconfrontationally explain the diagnosis.
- Anti-epileptic drugs are not indicated; however in the patient already receiving them the withdrawal of such medications should be done by the prescribing neurologist.
- Long term treatment recommendations typically involve therapies such as cognitive behavioral therapy, individual and group psychotherapy, and family therapy.

Tool

Differentiating PNES from epileptic seizures

	PNES	Epileptic seizures
Video EEG	Captured event with no EEG correlate	EEG correlates with event
Arise during sleep	No	Yes
Typical event duration	20–805 s	10–140 s
Waxing waning course	Possible	No
Termination	Abrupt	Gradual
Motor manifestations[a]	• Complex movements (writhing, flailing, whole body thrashing) common • Side to side head movements or body turning • Asynchronous body movements (alternate clonic movements) • Periods of prolonged body flaccidity	• Complex movements uncommon • Side to side head movements uncommon • No asynchronous movements
Tachycardia	Rare	Common
Urinary incontinence	Rare	Common
Crying during seizure	Common	Rare

	PNES	Epileptic seizures
Eyes at onset	Closed	Open
Eyes throughout episode	Closed	Intermittently open & closed
Forced eye closure (resistance to examiner opening eyes)	Present	Absent
Mouth during tonic phase	Closed	Open
Tongue bite	Tongue tip, lip, or buccal	Lateral
Postictal speech patterns	Emotional	Flat, monotone
Postictal headache	Rare	Common
Postictal recall of events occurring during seizure	Yes	No

[a]Frontal lobe epilepsy seizures can manifest similarly to PNES. For the purposes of this tool, epileptic siezures are non-frontal lobe convulsive epileptic seizures

References

1. Bodde NMG, Brooks JL, Baker GA, Boon PAJM, Hendriksen JGM, Mulder OG, Aldenkamp AP. Psychogenic non-epileptic seizures—definition, etiology, treatment and prognostic issues: a critical review. Seizure. 2009;18(8):543–53.
2. Devinsky O, Gazzola D, LaFrance WC. Differentiating between nonepileptic and epileptic seizures. Nat Rev Neurol. 2011;7(4):210–20.
3. Dworetzky BA, Weisholtz DS, Perez DL, Baslet G. A clinically oriented perspective on psychogenic nonepileptic seizure–related emergencies. Clin EEG Neurosci. 2015;46(1):26–33.

4. Hendrickson R, Popescu A, Ghearing G, Bagic A. Thoughts, emotions, and dissociative features differentiate patients with epilepsy from patients with psychogenic nonepileptic spells (PNESs). Epilepsy Behav. 2015;51:158–62.
5. Siket MS, Merchant RC. Psychogenic seizures: a review and description of pitfalls in their acute diagnosis and management in the emergency department. Emerg Med Clin North Am. 2011;29(1):73–81.

Chapter 25
Seizures

Christopher S. Sharp and Michael P. Wilson

Introduction

Seizures are defined as transient occurrences of signs or symptoms related to abnormal excessive or synchronous neuronal activity in the brain. Acute seizures comprise approximately 1% of all emergency department visits. Acutely, most seizures are identified by motor symptoms, such as clonic jerking. The most common seizure emergencies are acute repetitive seizures, an abrupt increase in seizure frequency compared to baseline, and status epilepticus (SE), at least

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, 777 Bannock St, Denver, CO 80204, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_25,

30 min of continuous seizure activity or multiple seizures without return to neurological baseline. Convulsive seizures are easily recognized; however nonconvulsive seizures are less clear yet are present in nearly 20% of patients with altered mental status (AMS) who receive electroencephalography (EEG). A period of AMS occurring in the period following a seizure is referred to as a postictal state.

Signs/Symptoms

Seizures have a wide variety of possible manifestations, however common signs and symptoms include:

- Mental status changes (confusion, amnesia, catatonia, psychosis, delirium, agitation, etc.)
- Altered sensation (visual, gustatory, olfactory, etc.)
- Convulsive motor activity (tonic contractures, clonic jerking)

Life-threatening Symptoms/Signs

- Hyperthermia
- Hypertension (though progresses to hypotension as status epilepticus progresses)
- Cardiac arrhythmias
- Rhabdomyolysis

Differential

Most seizures are unprovoked or occur from progression of symptomatic causes; however in hospitalized patients the vast majority of seizures or SE have an acute symptomatic cause. Potential causes include:

- traumatic brain injury
- stroke
- hemorrhage

CNS infections or tumors
metabolic abnormalities (for example, hyponatremia)
alcohol withdrawal
Illicit substances and medications can also lower the seizure threshold
*Epileptic seizures should also be differentiated from psychogenic nonepileptic seizures

Testing

Laboratory studies should be directed but could include:
- Basic metabolic panel, calcium, magnesium, phosphate to rule out metabolic causes
- CBC
- Liver function tests
- Troponin
- Antiepileptic drug (AED) levels, particularly if patient is known to be prescribed an AED such as phenytoin or valproic acid
- HCG level for women of reproductive age

Other studies:
- EEG
- Imaging (must balance the value of imaging with the cost of delaying treatment)
- Lumbar puncture & cerebrospinal fluid analysis if safe to do so and there is suspicion for encephalitis or subarachnoid hemorrhage

Treatment

- Airway management and respiratory support as indicated
- Place patient in left-lateral decubitus position
- Remove any foreign objects from mouth
- Cardiac monitoring
- Correct fluid and electrolyte imbalances
- If hypoglycemic (<80 mg/dL) administer 100 mg of thiamine followed by 20–50 g of dextrose 50% solution

- First line treatment for managing SE: benzodiazepines
  - Lorazepam—0.1 mg/kg at a rate of 2 mg/min
  - Midazolam—0.2 mg/kg, initial dose of 10 mg IM
  - Diazepam—0.2 mg/kg at a rate of 5 mg/min
- Second line treatment: AEDs
  - Phenytoin—20 mg/kg IV loading dose at a rate 50 mg/min; 100 mg every 6–8 h maintenance dose
  - Valproate—20–40 mg/kg loading dose; 4–6 mg/kg every 6 h maintenance
  - Levetiracetam—2000–4000 mg loading dose; 10–15 mg/kg every 12 h maintenance
  - Lacosamide—200–400 mg loading dose; 200–300 mg every 12 h maintenance
- Third line interventions for SE include propofol, pentobarbital, and ketamine

Tool

Potential Causes of Provoked Seizures

Drugs of Abuse	Alcohol
	Stimulants
	Ecstasy
	Phencyclidine (PCP)
	Lysergic acid diethylamide (LSD)
Infection/ Inflammation	Meningitis
	Encephalitis
	Cerebritis
Lesions	Tumors
	Stroke
	Hemorrhage
Systemic	Eclampsia
	Thyrotoxicosis
	Extreme fever

Metabolic Disorders	Hypoglycemia, Hyperglycemia
	Hyponatremia, Hypernatremia
	Hypocalcemia
	Hypomagnesemia
Antibiotics	Penicillins
	Isoniazid
	Rifampin
	Antimalarials
	Metronidazole
Antiarrhythmic agents	Digoxin
	Lidocaine
Antidepressants	Bupropion
	Cyclics
Antipsychotics	Clozapine
	Haloperidol
Pain Medications	Tramadol
	Demerol
	Fentanyl
Miscellaneous Medications	Baclofen
	Phenytoin (at supratherapeutic levels)
	Calcineurin inhibitors (cyclosporine, tacrolimus)
	Lithium
	Chemotherapeutic agents
	Multiple sclerosis medications
Withdrawal from	Opiates
	Alcohol
	AEDs (especially benzodiazepines and barbituates)
Trauma	

References

1. Fisher RS, Boas WVE, Blume W, Elger C, Genton P, Lee P, Engel J. Epileptic seizures and epilepsy: definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). Epilepsia. 2005;46(4):470–2.
2. Slattery DE, Pollack CV. Seizures as a cause of altered mental status. Emerg Med Clin North Am. 2010;28(3):517–34.
3. Foreman B, Hirsch LJ. Epilepsy emergencies: diagnosis and management. Neurol Clin. 2012;30(1):11–41.

Chapter 26
Thyrotoxicosis

Christopher S. Sharp and Kimberly D. Nordstrom

Introduction

Technically, thyrotoxicosis is any hyperthyroid state (from mild to severe). A severe, usually sudden burst of thyroid hormone is termed a "thyroid storm." A thyroid storm is a life-threatening, medical emergency. There are many causes for this condition but Grave's Disease is by far the most common. Other etiologies to consider include: toxic adenoma, multinodular goiter, and intake of toxic levels of exogenous thyroid. Diagnosis is usually suspected by overt symptoms and signs and then confirmed through laboratory testing. A further work up is indicated to determine etiology.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, 777 Bannock St, Denver, CO 80204, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

K.D. Nordstrom, M.D., J.D. (✉)
Department of Human Services, Office of Behavioral Health, Denver, CO, USA

American Association for Emergency Psychiatry, PO Box 3948, Parker, CO 80134, USA
e-mail: Nordstrom_kimberly@yahoo.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_26,

Symptoms

Weight loss (with increase or no change in diet)
Palpitations
Tremor
Sweating
Increased sensitivity to heat (intolerance)
Fatigue
Muscle weakness
Brittle hair
Insomnia
Psychiatric: anxiety, irritability

Signs

Enlarged thyroid gland
    Thyroid bruit
    Multinodular goiter
Exophthalmos
    Periorbital edema
    Conjunctival edema
    Lid lag
    Extraocular muscle dysfunction
    Proptosis
Pretibial myxedema
Tachycardia
Arrhythmia
Tremor

Life-Threatening Symptoms/Signs (Thyroid Storm)

Symptoms are sudden and severe
Tachycardia (usually greater than 140)
Atrial fibrillation
Hypertension
High fever (can be as high as 106°F)
Dehydration

- Nausea/vomiting →dehydration
- Diarrhea→dehydration
- Diaphoresis→dehydration

Psychiatric: extreme agitation, irritability, psychosis
Confusion, disorientation
Unconsciousness
Death

Differential

- Cardiac
  - Cardiac arrhythmia
  - Congestive Heart Failure
  - Angina
- Pulmonary
  - Pulmonary Edema
- Endocrine
  - Diabetes Mellitus
  - Addison disease
- Neurologic
  - Essential tremor
  - Neurocognitive disorder
- Psychiatric
  - Insomnia
  - Bipolar mania
  - Schizophrenia, both florid psychosis and catatonia
  - Anxiety/panic disorder
  - Attention Deficit Hyperactivity Disorder
- Other
  - Chronic fatigue syndrome
  - Myopathy
  - Irritable bowel syndrome
  - Malignancy
  - Infection
  - Medication reaction/interaction
  - Stimulant Use
  - Neuroleptic malignant syndrome
  - Malignant hyperthermia

Laboratory Studies

TSH: low
FT_4, T_3: elevated
Electrocardiogram

Treatment

Airway management and respiratory support as indicated
Aggressive supportive care such as cooling measures for hyperthermia and fluid resuscitation
Administer propranolol for control of cardiac arrhythmias
Initiate thyrostatics to reduce thyroid hormone levels: propylthiouracil (PTU) or methimazole
After at least 1 hour of PTU administration, give a saturated solution of potassium iodide to inhibit thyroid hormone production and release.
Begin dexamethasone to reduce peripheral thyroid hormone deiodination.
*Once the patient has been stabilized, the underlying cause of thyroid disease should be accurately investigated.

Tool

Testing in hyperthyroid versus thyroid storm

Study	Hyperthyroid	Thyroid storm
Free T_4	Elevated	Elevated (rapid change)
T_3	Elevated	Elevated (rapid change)
TSH	Reduced	Reduced
EKG	Tachycardia; possible arrhythmia	Supraventricular tachycardia Arrhythmia: atrial fibrillation
BMP	Possible dehydration	Dehydration
CBC	Possible mild leukocytosis	Marked leukocytosis
UA	Associated dehydration	Dehydration

References

1. Bahn RS, Burch HB, Cooper DS, Garber JR, Greenlee MC, Klein I, et al. Hyperthyroidism and other causes of thyrotoxicosis: Management guidelines of the American Thyroid Association and American Association of Clinical Endocrinologists. Endocr Pract. 2011;17:456–520.
2. Papi G, Corsello SM, Pontecorvi A. Clinical concepts on thyroid emergencies. Front Endocrinol (Lausanne). 2014;5:102.
3. Swee DS, Chung CL, Lim A. Clinical characteristics and outcome of thyroid storm: A case series and review of neuropsychiatric derangements in thyrotoxicosis. Endocr Pract. 2015;21(2):182–9.
4. Vaidya B, Pearce SH. Diagnosis and management of thyrotoxicosis. BMJ. 2014;349:g5128. doi:10.1136/bmj.g5128.
5. Klein I, Danzi S. Thyroid disease and the heart. Circulation. 2007;116(15):1725–35. doi:10.1161/circulationaha.106.678326.
6. Franklyn JA, Boelaert K. Thyrotoxicosis. Lancet. 2012;379(9821):1155–66. doi:10.1016/S0140-6736(11)60782-4.
7. Iagaru A, McDougall IR. Treatment of thyrotoxicosis. J Nucl Med. 2007;48:379–89.

Chapter 27
Wernicke-Korsakoff Syndrome

Christopher S. Sharp and Kimberly D. Nordstrom

Introduction

Wernicke-Korsakoff Syndrome (WKS) is related to a B-1 (thiamine) deficiency. Many understand the relationship with alcohol use disorders but B-1 deficiency can be associated with any disorder or illness that affects absorption or usage of thiamine (such as gastrectomy, hyperemesis gravidarum, sepsis). It is commonly taught that WKS presents with the clinical triad of confusion, ataxia and nystagmus but this cannot be relied upon.

C.S. Sharp, M.D.
Psychiatric Emergency Service, Denver Health Medical Center, 777 Bannock St, Denver, CO 80204, USA

Department of Psychiatry, University of Colorado-Denver School of Medicine, Denver, CO, USA

K. D. Nordstrom, M.D., J.D (✉)
Department of Human Services, Office of Behavioral Health, Denver, CO, USA

American Association for Emergency Psychiatry, PO Box 3948, Parker, CO 80134, USA
e-mail: Nordstrom_kimberly@yahoo.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_27,

Symptoms

- Confusion
- Loss of memory
- Lack of coordination
- Double or blurry vision
- Abnormal eye movements
- Anxiety
- Hallucinations

Signs

- Mental status changes
- Loss of memory (retrograde and anterograde)
  - Confabulation
- Oculomotor abnormalities
  - Horizontal nystagmus (most common)
  - Retinal hemorrhage
  - Conjugate gaze palsy
- Loss of muscle coordination
  - Wide-based gait
  - Unsteady gait
- Abnormal reflexes
- Muscle weakness
- Polyneuropathy
- Hypotension
- Tachycardia

Life-Threatening Symptoms/Signs

- Hypothermia
- Heart failure with lactic acidosis
- Related infection (increases overall mortality)
- Spastic paralysis
- Seizures
- Coma

Differential

Alcohol or other substance intoxication
Auto-immune illness (multiple sclerosis, lupus)
Head trauma or mass
Infection
Metabolic derangements
Medication toxicity
Migraines
Stroke

Laboratory Studies

No urgent study for confirmation of diagnosis; a thiamine level (usually a send out) can be measured
Electrolytes for related abnormalities
CBC for related infection
If severe symptoms: consider Lactate and CPK

Treatment

- Airway management and respiratory support as indicated
- Thiamine should be administered parenterally in any patient with suspected Wernicke's encephalopathy
- Correct fluid and electrolyte, particularly magnesium, imbalances
- Correct hypoglycemia with glucose loading, though timing is controversial
- If possible, MRI should be performed to support the diagnosis

Tool

Caine Criteria (sensitivity: 85%; specificity 100%)
Any 2 of the following 4 signs is sufficient to identify Wernicke's encephalopathy

1. Nutritional deficiencies (signs of malnutrition on physical exam or laboratory study)
2. Oculomotor abnormalities
3. Cerebellar dysfunction
4. Altered mental status or memory impairment

References

1. Caine D, Halliday GM, Kril JJ, Harper CG. Operational criteria for classification of chronic alcoholics: identification of Wernicke's encephalopathy. J Neurol Neurosurg Psychiatry. 1997;62(1):51–60.
2. Isenberg-Grzeda E, Kutner HE, Nicolson SE. Wernicke-Korsakoff-syndrome: under-recognized and under-treated. Psychosomatics. 2012;53:507–16. doi:10.1016/j.psym.2012.04.008.
3. Day E, Bentham PW, Callaghan R, Kuruvilla T, George S. Thiamine for prevention and treatment of Wernicke-Korsakoff Syndrome in people who abuse alcohol (Cochrane Review). Cochrane Libr. 2013;7:1–20.
4. Galvin R, Brathen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA, et al. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. Eur J Neurol. 2010; 17:1408–18.
5. Day GS, del Campo CM. Wernicke encephalopathy: a medical emergency. CMAJ. 2014;186(8):E295. doi:10.1503/cmaj.130091.
6. Lough ME. Wernicke's encephalopathy: expanding the diagnostic toolbox. Neuropsychol Rev. 2012;22(2):181–94. doi:10.1007/s11065-012-9200-7.
7. Thomson AD, Cook CH, Touquet R, Henry JA. The royal college of physicians report on alcohol: guidelines for managing Wernicke's encephalopathy in the accident and emergency department. Alcohol Alcohol. 2002;37:513–21. doi:10.1093/alcalc/37.6.513.
8. Schabelman E, Kuo D. Glucose before thiamine for Wernicke encephalopathy: a literature review. J Emerg Med. 2012;42(4): 488–94.

Section IV
Substance Intoxication

Section Editor: Julien J. Cavanagh, M.D.

Chapter 28
Acute Intoxication: General Considerations

Julien J. Cavanagh

Introduction

- Acute intoxication with recreational drugs is a very common reason for visits to the emergency room.
- These intoxications cause both medical and psychiatric symptoms which must be approached simultaneously.
- Intoxication with multiple drugs is common and clinicians must exert extreme caution when assessing patients.
- Intoxication with a single drug does not always follow typical clinical presentation:
  - Users often genuinely think they're taking one drug but take another

→Example: user purchases "liquid Ecstasy" (GBL) believing he is taking MDMA

  - Street drugs can contain mixing products and byproducts that are active.

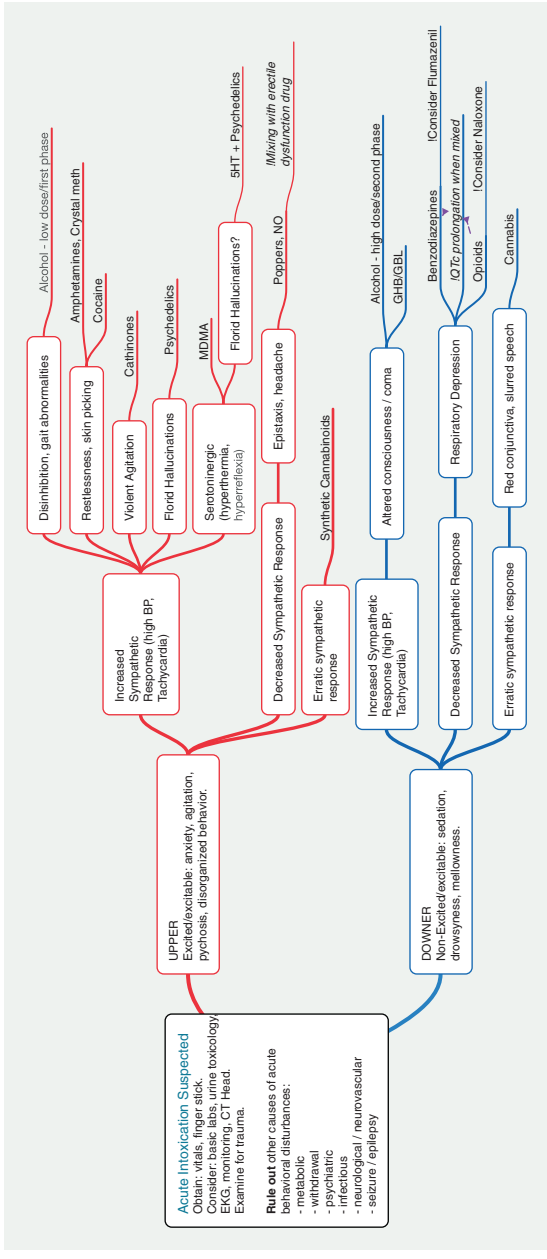
J.J. Cavanagh, M.D.
Department of Neurology, State University of New York—Downstate Medical Center, 450 Clarkson Ave, Brooklyn, NY 11203, USA
e-mail: Julien.cavanagh@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.), *Quick Guide to Psychiatric Emergencies*,
https://doi.org/10.1007/978-3-319-58260-3_28,

- Clinical assessment can orient toward a particular drug (see diagnosis orientation tree) but clinicians must be ready to reconsider their diagnosis as a patient's presentation evolves.
- Clinicians must be particularly cautious when managing psychiatric symptoms, especially agitation:
  - Use of antipsychotics is ill-advised in case of intoxication with many drugs because of:
    - Risk of QTc prolongation
    - Increased risk of NMS with certain drugs
    - Benzodiazepines are often the safest choice
      - Caution in risk of respiratory depression
- Some clinical presentations can appear as intoxication when they are in fact withdrawal syndromes.
- All patients must be reassessed psychiatrically once medically stable/clear "the next day" to evaluate for:
  - Suicidal risk
  - Persisting psychiatric symptoms (psychosis, mood disorder…) resulting from intoxication with the drug
  - Untreated psychiatric disease independent of drug use
- All patients must be counseled
  - Detox and rehab options vary by drug use and user
- All users must benefit from HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did patient have unprotected sex immediately prior to visit).
  - Educate about safe sex practices
  - Follow guidelines and consider Post-Exposure Prophylaxis.
- Encourage repeat users to consider Pre-Exposure Prophylaxis.

Tool

DECISION TREE



Chapter 29
Phenethylamines Intoxication (Amphetamines, Methamphetamine, Cathinones, and "Designer Amphetamines)

Julien J. Cavanagh and Teresa Y. Smith

Introduction

- History, chemical profile
  - Phenethylamines—commonly named amphetamines—have been used in virtually every human culture for centuries to provide increased alertness and euphoria.
  - In the modern western world, amphetamine was the first phenethylamine to be synthetized, in the late nineteenth century. Initially used a nasal deconges-

J.J. Cavanagh, M.D. (✉)
Department of Neurology, State University of New York—Downstate Medical Center, 450 Clarkson Ave, Brooklyn, NY 11203, USA
e-mail: julien.cavanagh@downstate.edu

T.Y. Smith, M.D., M.S.Ed., F.A.C.E.P.
Department of Emergency Medicine, Kings County Hospital, State University of New York, Brooklyn, NY, USA
e-mail: Teresa.Smith@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.), *Quick Guide to Psychiatric Emergencies*,
https://doi.org/10.1007/978-3-319-58260-3_29,

tant, it rapidly attracted attention for its other properties (cognitive enhancement, euphoriant, etc.) leading to the synthesis of numerous sister-molecules, many of them becoming drugs of abuse.
  - Today, phenethylamines constitute a large family of drugs. Some of them are of clinical interest and have gained approval for medical use. These authorized amphetamines are usually long-acting and used in conditions such as attention deficit, narcolepsy, or even depression.

Reason for Visit to the Emergency Department

- Typical clinical presentation:
  - "Violently agitated young person brought in by the police after he was reportedly harassing and attacking people and property on the street while wearing little to no clothes."
  - Person in her thirties brought to the emergency department in altered mental status by EMS after friends weren't able to wake her up the morning after a private party."
- Symptoms
  - Psychotic symptoms such as hallucinations, delusions, derealization/depersonalization, mental and motor automatism.
  - Episode of intense agitation in relation either with anxiety or psychotic phenomenology.
  - Agitation can easily lead to unpredictable violence. Correlation between quantity of substance used and intensity of agitation has been demonstrated.
  - Skin-picking is a common symptom and is most likely the result of delusions of formication ("meth-mites").
  - Stereotypies consisting of purposeless behaviors such as searching in one's purse or disassembling/reassembling objects for hours.

- Signs
  - Elevated blood pressure, tachycardia
  - Hyperthermia
  - Diaphoresis
  - Seizure
  - Altered Mental Status
  - Mechanical injuries, skin abscesses, anal abrasions, infectious complications from IV drug use

Differential Diagnosis

- Other commonly abused drugs that lead to increased sympathetic response, such as cocaine or MDMA, may present with a clinical picture identical to amphetamine intoxication
- Amphetamine intoxication may also present similar to anticholinergic overdose; differentiating factors may include the presence of diaphoresis and clear pressured speech in amphetamine use, as opposed to anhydrosis and muffled speech in anticholinergic poisoning.
- Differentials for hyperthermia with altered mental status include CNS infections—meningitis, encephalitis.

Labs/Testing/Imaging

- Metabolic panel, Liver Function Tests, CPK, Troponin
- Most common labs abnormalities are:
  - metabolic acidosis
  - elevated creatine kinase,
  - elevated troponin (demand ischemia)
  - transaminitis
  - hyponatremia
  - hyperkalemia/hypokalemia
- Urine toxicology should be obtained but should not delay appropriate management.

Medical and Psychiatric Management

- The main stay of treatment is benzodiazepines.
  - Benzodiazepines can help with sympathomimetic symptoms (hypertension and tachycardia)
  - Also serve as a sedative for medical restraint.
  - Offer neuroprotection against seizure event
- Consider*:*
  - IV, fluid bolus
  - Electrolytes correction

Caution

- Like other exciting drugs, phenethylamine use can be associated with trauma
- Physical restraining may potentate rhabdomyolysis
- We recommend extreme caution in the use of first generation antipsychotics such as butrophenones (haloperidol) as they may lower seizure threshold and also prolong QTc.

Disposition

- Among occasional users, acute medical and psychiatric complications of phenethylamines are usually self-limited and respond well to simple therapy (benzodiazepine, fluids, electrolytes correction)
  - Discharge is usually possible within 24–48 h
- Persistent psychosis
  - Psychiatric symptoms such as hallucination and persecutory delusions can persist up to a week.
  - Usually milder symptomatology; agitation is rare outside of the acute intoxication.
  - Psychiatry consult is warranted to assess danger to self and others in particular in context of persecutory delusions.
  - Short stay in psychiatry unit should be considered.

- All users should be counseled
  - Repeat/chronic users can be oriented towards peer support groups (e.g.: narcotics anonymous) or mental health clinics.
  - Inpatient detox programs rarely accept synthetic drugs users.
- HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did patient have unprotected sex immediately prior to visit).
  - Educate about safer sex practices.
  - Follow guidelines and consider Post-Exposure Prophylaxis.
  - Encourage repeat users to consider Pre-Exposure Prophylaxis.

Further Information

- Most phenethylamines today are Schedule I drugs and are considered drugs of abuse.
- Phenethylamines carry a positive alpha and beta adrenergic action. They also stimulate the dopaminergic, serotoninergic, and noradrenergic neurotransmission either by direct agonist action or recapture inhibition.
- The two most important phenethylamines remain amphetamine and methamphetamine.
- There are however multiple variations on the phenethylamine structure, generating an endless supply of new drugs commonly named "designer amphetamines." Each new molecule is *de facto* legal until they are identified by authorities and made unlawful to consume.
- Cathinones are a subgroup of phenethylamines derived from the khat plant. MDPV, Mephedrone, and Methcathinone are the most used cathinones. MDPV is widely known by its street name "bath salts." The slang term came to include all cathinones.
- It is believed that modifications in the ring structure of the phenethylamine molecule modulates the intensity of the

dopaminergic activity. This explains the hallucinatory properties of cathinones and designer amphetamines in general.

- How does the drug present, how is it consumed?
  - Phenethylamines can present as powder, pills, or crystals (hence the terms crystal-meth or bath salts).
  - Street names for amphetamines: speed, amped, coast-to-coast, upper, wake-up …
  - Street names for methamphetamines: crystal, go-fast, Ice, P2P, Tina, T …
  - Street names for cathinones: bath salts, Ivory wave, bloom, cloud nine, lunar wave, vanilla sky, white lightning, scarface …
  - Oral consumption: pills, powder or crushed crystals are ingested directly, dissolved in water, juice, or coffee ("trucker coffee"), or wrapped in paper ("parachuting"). The onset of action of the drug is around 30 min.
  - Snorting: the drug is crushed and snorted with a straw. The onset of action is a few minutes.
  - Smoked: most often in a glass pipe. The onset of action is a few minutes.
  - Vaporized: also known as "hot railing." The drug is heated with a red-hot glass stem. The vaporized drug is inhaled. The onset of action is a few seconds.
  - Anal absorption ("booty bumping"): the drug is either dissolved in water and introduced in the rectum with a syringe, or simply crushed and deposited in the anal canal with a finger. The onset of action is a few minutes.
  - IV: also known as slamming. The drug is dissolved in water and injected intravenously. The onset of action is a few seconds.

  - Intramuscular ("muscling") and subcutaneous ("skin popping") are also mentioned. However, they are rarely used for crystal forms as the absorption is poor.

- Context of using
  - Phenethylamines can be considered "party drugs" and are usually consumed in a festive context. However, users developing addiction will use in any context.
  - Methamphetamines and cathinones are commonly used in the context of sex encounters. The arousal and disinhibition generated by the drug, as well as erection modulation explains why the drug is used in gatherings where multiple sex partners are involved. The use of methamphetamine or cathinones within the gay communities has become a public health concern in recent years in particular because of the danger of spreading of HIV and other STIs, but also because of the significant increase in mortality among users.

- Desired effect
  - Subjects using phenethylamines experience an increase in arousal, alertness, and pleasure. This state of euphoria translates in a subjective feeling of performing better socially, sexually, or even artistically. Users experience disinhibition which explains all sorts of risk conducts.
  - Cathinones and designer amphetamines can generate intense perception disturbance such as hallucinations. While these might be sought after by users, they can also prove to be very disturbing and undesirable.

References

1. Rusyniak DE. Neurologic manifestations of chronic methamphetamine abuse. Neurol Clin. 2011;29(3):641–55. doi:10.1016/j.ncl.2011.05.004.
2. Spiller HA, Ryan ML, Weston RG, Jansen J. Clinical experience with and analytical confirmation of "bath salts" and "legal highs" (synthetic cathinones) in the United States. Clin Toxicol (Phila). 2011;49:499.
3. Singleton J, Degenhardt L, Hall W, Zabransky T. Mortality among amphetamine users: a systematic review of cohort studies. Drug Alcohol Depend. 2009;105:1–8. doi: 10.1016/j.drugalcdep.2009.05.028.
4. http://www.intheknowzone.com/substance-abuse-topics/methamphetamine.html

Chapter 30
Cocaine Intoxication

Julien J. Cavanagh and Teresa Y. Smith

Introduction

For thousands of years, coca plant leaves have been consumedby indigenous populations in South America. It is used for its stimulant and hunger suppressing properties. In the second half of the nineteenth century, Western medicine started being interested in cocaine, as an anesthetic and a stimulant. In the twentieth century, cocaine became a widely used recreational drug. Cocaine has several modes of action. It, notably, inhibits recapture of both serotonin and dopamine. It blocks the dopamine transporter, therefore increasing dopamine concentration in the synaptic cleft. It also blocks sodium channels which explains its an anesthetic properties.

J.J. Cavanagh, M.D. (✉)
Department of Neurology, State University of New York—Downstate Medical Center, 450 Clarkson Ave, Brooklyn, NY 11203, USA
e-mail: julien.cavanagh@downstate.edu

T.Y. Smith, M.D., M.S.Ed., F.A.C.E.P.
Department of Emergency Medicine, Kings County Hospital, State University of New York, Brooklyn, NY, USA
e-mail: Teresa.Smith@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_30,

Reason for Visit to the Emergency Department

- Typical clinical presentation:
  - "Young adult presenting to the emergency room agitated with chest pain, anxiety, agitation; exam remarkable for mydriasis"
- Symptoms
  - severe agitation
  - psychosis
  - anxiety
- Signs

– Hypertenson	– Tremor
– Tachycardia	– Severe agitation
– Hyperthermia	– Psychosis
– Nausea	– Seizure
– Diaphoretic	– Focal neurologic deficits
– Mydriasis	– Headache
– Picking of the skin (formication)	– Choreiform-like movements

Differential Diagnosis

- Other drugs of abuse that lead to increased sympathetic response, such as MDMA or cathinones, may present with a clinical picture identical to amphetamine intoxication.
- Cocaine intoxication may also present similarly to anticholinergic overdose; differentiating factors may include the presence of diaphoresis and clear pressured speech in cocaine use, as opposed to anhydrosis and muffled speech in anticholinergic poisoning.

Labs/Testing/Imaging

- ECG (for potential ischemic changes).
- Non-contrast head CT (hemorrhagic and ischemic findings).
- Check labs (elevated troponin, elevated creatine kinase, and hyperkalemia/hypokalemia).

Medical and Psychiatric Management

Given cocaine can produce a wide spectrum of symptoms, treatment is based on the area of the body that is affected (most commonly cardiopulmonary, neurologic, and psychiatric).

Consider:

- Benzodiazepines for sympathomimetic symptoms, agitation, or seizure.
- IV, fluid bolus.
- Aspirin and nitroglycerin for "cocaine chest pain."

Caution

- No beta blockers for hypertensive emergencies (unopposed alpha blockade).
- Crack cocaine –can cause a variety of pulmonary complications, including those described as "crack lung."
- Adulterants of cocaine—levamisole (causes agranulocytosis, cutaneous vasculitis, and leuokoencephalopathy).

Disposition

- Among occasional users, acute medical and psychiatric complications of cocaine are usually self-limited and respond well to simple therapy (benzodiazepine, fluids, electrolytes correction).
  - Discharge is usually possible within 24 h

- Persistent psychiatric symptoms
  - Usually milder symptomatology; agitation is rare outside of the acute intoxication.
  - Among regular users, withdrawal depression is common ("crash"). In that context, psychiatry consult can be warranted to assess affect and suicidal risk. Short stay in psychiatry unit can be considered.
- All users should be counseled:
  - Repeat/chronic users can be oriented towards peer support groups (e.g.: narcotics anonymous) or mental health clinics.
  - Inpatient detox programs rarely accept cocaine users.
- HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did patient have unprotected sex immediately prior to visit).
  - Educate about safer sex practices.
  - Follow guidelines and consider Post-Exposure Prophylaxis.
  - Encourage repeat users to consider Pre-Exposure Prophylaxis.

Further Information

- How does the drug present, how is it consumed?
  - Cocaine, in its purest form, appears as a white powder. It is often "cut" with other products such as talc or confection sugar but also pharmacologically active products such as PCP or synthetic amphetamines.
  - It is usually snorted but can also be injected or smoked.
  - Crack or crack-cocaine is a less pure product that looks like brown brittle crystal commonly called "rock". It is smoked with a glass pipe.
- Context of using
  - Cocaine is culturally associated with the rich and famous. It is in fact widely used in categories of populations.

  - Crack cocaine is generally consumed by users with lesser means because of its very cheap price.
- Desired effect
  - Sensation of euphoria, of being powerful, successful, limitless.
  - Diminished sensation of fatigue, impression of increased performances.
  - This increased sensation of self confidence leads to risk taking behaviors.

References

1. Almeida RR, Zanetti G, Souza ASJ, Souza LS, Silva JL, Escuissato DL, Irion KL, Mancano AD, Nobre LF, Hochbegger B, Marchiori E. Cocaine-induced pulmonary changes: HRCT findings. J Bras Pneumol. 2015;41(4):323–30.
2. Brody SL, Slovis CM, Wrenn KD. Cocaine-related medical problems: Consecutive series of 233 patients. Am J Med. 1990;88(4):325–31.
3. Rich JA, Singer DE. Cocaine-related symptoms in patients presenting to an urban emergency department. Ann Emerg Med. 1991;20(6):616–21.
4. Larocque A, Hoffman RS. Levamisole in cocaine: unexpected news from an old acquaintance. J Clin Toxicol. 50(4):231–41.

Chapter 31
Ethanol (Alcohol) Intoxication

Julien J. Cavanagh and Teresa Y. Smith

Introduction

Alcoholic beverages exist since the dawn of humanity and are part of the culture of many civilizations since antiquity. Ethanol is obtained through fermentation of countless products leading to an infinite variety of flavors. It is believed that ethanol binds several receptors in the human brain, including GABA receptors. This profile explains many of the empirical effects of alcohol.

J.J. Cavanagh, M.D. (✉)
Department of Neurology, State University of New York—Downstate Medical Center, Brooklyn, NY, USA
e-mail: julien.cavanagh@downstate.edu

T.Y. Smith, M.D., M.S.Ed., F.A.C.E.P.
State University of New York Downstate Medical Center, Kings County Hospital, Brooklyn, NY, USA
e-mail: Teresa.Smith@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_31,

Reason for Visit to the Emergency Department

- Typical clinical presentation:
  - "Violently agitated individual brought by the police for erratic behavior in a public place. Patient presents with slurred speech, enlarged gait, and disinhibition."
  - "Unconscious individual found on the street near a bar. Smells of alcohol and vomit."
- Symptoms
  - Consumed at low to moderate dose, alcohol causes euphoria, disinhibition and can lead to agitation and potentially violence if environmental triggers are encountered (interpersonal conflict, policing, etc.).
  - Alcohol exacerbates pre-existing psychiatric conditions such as schizophrenia, bipolar disorder, or personality disorders. It rarely causes psychosis (alcoholic hallucinosis).
  - Due to its disinhibiting effect, alcohol increases suicidal risk.
  - At higher dose, consciousness becomes altered and behavior becomes more and more disorganized.
- Signs

– Agitation	– Slurred speech
– Lethargy leading to unresponsiveness	– Unsteady gait
– Respiratory depression	– Urinary incontinence
– Hypotension	– Nausea/vomiting
– Tachycardic	
– Nystagmus	

Labs/Testing/Imaging

- Blood alcohol level is a very poor predictor of behavior (or medical state) as every individual's tolerance is different. The test remains of clinical interest as it offers confirmation (or not) of a clinical impression.
- Blood glucose (fingerstick) should be obtained immediately, as patients often presents with hypoglycemia.
- Are also found commonly:
  - hypoglycemia,
  - starvation ketosis (anion gap lactic acidosis),
  - hypokalemia, hypomagnesemia,
  - may also see hypocalcemia and hypophosphatemia.
- ECG (check for QTc prolongation)
- Consider head CT imaging to rule out head trauma

Medical and Psychiatric Management

- Mostly supportive treatment during acute alcohol intoxication.
- IV, fluid bolus
- Benzodiazepines are commonly used for the management of agitation in acute alcohol intoxication. However, they should be used with caution as they may worsen respiratory depression.
- Butrophenones (haloperidol) can be considered for agitation, but usage should be careful as this may lower seizure threshold and also further prolong QTc.
- Thiamine and Folic acid supplementation should be given in chronic alcohol abuse.

Caution

- Good physical examination (undress the patient to search for evidence of trauma and/or other pathology).

- Pay attention to alternate and/or concomitant diagnoses.
- Chronic alcoholism may lead to:
  - subsequent trauma (particularly occult head trauma),
  - electrolyte disturbances,
  - GI bleeds,
  - other illnesses not easily picked up due to inability to get a proper history of present illness.
- Alcohol mixed with energy drinks may cause increased alcohol consumption thus increasing the risk of alcohol-related consequences.
- Consider other alcohols (methanol or ethylene glycol) in cases of severe metabolic acidosis and increased osmolar gap.
- Consider other interventions/diagnostics: if patient does not return to normal mental status after an adequate period of observation/metabolism, or with impaired consciousness where alcohol levels are found to be less than 350 mg/100 mL.
- Alcohol withdrawal is lethal, and requires proper medical detoxification and vitamines supplementation. The mainstay of alcohol withdrawal treatment is benzodiazepines.

Disposition

- Ethanol elimination rate in non-chronic alcoholism is 15–20 mg/100 mL/h (non-fasting).
- Most acutely intoxicated patients can be discharged within 12–24 h.
- Alcohol-induced psychiatric symptoms rarely persists
- Psychiatric consult should be ordered for patient with persisting psychiatric symptoms, in particular suicidal ideation.
- All users should be counseled:
  - Inpatient detox programs do accept alcohol users. Different criteria of eligibility need to be met, depending on the institution.
  - Once completed, such program can orient patient to an acute rehab program, outpatient clinic, or twelve-step programs such as AA.

- HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did patient have unprotected sex immediately prior to visit).
  - Educate about safer sex practices.
  - Follow guidelines and consider Post-Exposure Prophylaxis.
  - Encourage repeat users to consider Pre-Exposure Prophylaxis.

Further Information

- How does the drug present, how is it consumed?
  - Ethanol is mainly consumed through alcoholic beverages.
  - Some users consume ethanol through disinfectants products or similar vehicles.
  - While the vast majority of users consume alcohol orally, some self-administer ethanol rectally or vaginally by soaking a tampon or similar devices with liquor or another alcoholic solution.
- Context of using
  - The French call it "*Art de vivre*", a mix of taste for delicacy and pleasure of sharing the moment with friends and acquaintances. This way of consuming alcohol is found in many cultures and traditions and the consensus is to not consider it abuse.
  - Abuse takes various forms and can break many stereotypes. From the homeless user consuming cheap liquor to the jet-setter drinking *grand cru*, there are many clinical presentations for which clinicians must be prepared.
- Desired effect
  - At low dose, users experience increased euphoria, self-confidence and sociability, as well as reduced anxiety.
  - At higher dose, ethanol alters consciousness which explains why many abusers use it as a sleep-inducer.

References

1. Jones AW. Evidence-based survey of the elimination rates of ethanol from blood with applications in forensic casework. Forensic Sci Int. 2010;200(1–3):1–20.
2. O'Brien MC, McCoy TP, Rhodes SD, Wagoner A, Wolfson M. Caffeinated cocktails: energy drink consumption, high-risk drinking, and alcohol-related consequences among college students. Acad Emerg Med. 2008;15(5):453–60.
3. Morgan MY. Acute alcohol toxicity and withdrawal in the emergency room and medical admissions unit. Clin Med. 2015;15(5):486–9.
4. Mullins PM, Mazer-Amirshahi M, Pines JM. Alcohol-related visits to US emergency departments, 2001–2011. Alcohol Alcohol. 2017;52(1):119–25. doi:10.1093/alcalc/agw074.
5. Shawn LK, Hoffman RS. Implementing evidence-based changes in emergency department treatment: alternative vitamin therapy for alcohol-related illnesses. Ann Emerg Med. 2013;61(1):119–20.
6. Shy BD, Hoffman RS. A descriptive comparison of alcohol-related presentations at a Large Urban Hospital Center from 1902 to 2009. J Med Toxicol. 2012;8(3):271–7.

Chapter 32
GHB, GBL Intoxication

Julien J. Cavanagh and Teresa Y. Smith

Introduction

Gamma-Hydroxybutyric acid is a general anesthetic. It was first synthetized in France by Henri Laborit. Its chemical structure is close to GABA therefore explaining both its euphoric and sedative profile by way of action on GABA receptors. Gamma-Butyrolatone results from dehydration of GHB. It has no action of its own. It behaves as a prodrug of GHB. Lactonase transforms GBL back into GHB. Because of its fat solubility, GBL has a higher potency and faster onset of action. 1,4-Butanediol or 1,4BD is another close parent to GHB and GBL but is less potent.

The main metabolism steps of GHB, GBL, and 1,4BD use alcohol deshydrogenase and aldehyde deshydrogenase. This

J.J. Cavanagh, M.D. (✉)
Department of Neurology, State University of New York—Downstate Medical Center, Brooklyn, NY, USA
e-mail: julien.cavanagh@downstate.edu

T.Y. Smith, M.D., M.S.Ed., F.A.C.E.P.
Department of Emergency Medicine, State University of New York, Kings County Hospital, Brooklyn, NY, USA
e-mail: Teresa.Smith@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_32,

pathway is shared with alcohol. This explains the cumulative effect of these drugs and alcohol.

GHB's medical use today is limited to narcolepsy.

Reason for Visit to the Emergency Department

- Typical clinical presentation:
  - "Young adult brought to the emergency department unresponsive and in respiratory distress after they were found unconscious on the floor at a private party"
- Symptoms
  - Users can present with anxiety and occasionally agitation.
  - Known users presenting with delirium are more likely to be withdrawing from the drug.
  - Users most often present with impaired consciousness.
- Signs
  - tachycardia
  - hypertension
  - insomnia
  - nausea/vomiting
  - diaphoresis
  - tremor
  - respiratory depression
  - unconsciousness/coma
  - altered mental status
  - delirium (particularly in the withdrawal state)

Differential Diagnosis

- Acute intoxication with low dose of GHB can mimic effects of MDMA. Increased temperature and hyponatremia are typically not found in GHB intoxication.

- Massive intoxication with other sedative drugs such as alcohol or benzodiazepines can give a similar presentation to GHB intoxication.

Labs/Testing/Imaging

- Comprehensive metabolic panel:
  - Hyperkalemia, Hypokalemia
- Elevated troponin.
- Elevated creatine kinase.
- Monitor vitals, oxygenation, arterial blood gases.
- Testing in urine can document the diagnosis.

Medical and Psychiatric Management

- Mainstay of the treatment during acute intoxication is supportive:
  - Maintain hemodynamic stability with IV fluid, bolus
- Acute respiratory failure can be fatal especially in cases of association with other sedative drugs such as alcohol or benzodiazepines:
  - Protection of airway might require intubation.
  - Respiratory distress might warrant ICU transfer
- Withdrawal state can also be lethal.

Caution

- Avoid neuroleptics (ineffective and may lead to dystonic reactions).
- Avoid antihypertensive agents (unless treating underlying hypertension). They are ineffective for symptomology control; avoid beta-blockers.

- Severe intoxication/overdose may cause severe altered mental status (even coma), which resolves within a few hours. With supportive care, patients usually fully recover.

Disposition

- Among occasional users, complications of GHB intoxications such as altered mental status, delirium, and sedation are usually self-limited and respond well to supportive treatment and allow discharge within 24 h.
- Massive intoxications or co-intoxication can justify transfer to medical ICU followed by transfer to medicine.
- Acute intoxication can be followed by withdrawal syndrome and its own lot of complications.
- All users should be counseled:
  - Repeat/chronic users can be oriented towards peer support groups (e.g.: narcotics anonymous) or mental health clinics.
  - Inpatient detox programs rarely accept GHB users, although they might consider it in context of co-morbid alcohol use disorder.
- HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did patient have unprotected sex immediately prior to visit).
  - Educate about safer sex practices.
  - Follow guidelines and consider Post-Exposure Prophylaxis.
  - Encourage repeat users to consider Pre-Exposure Prophylaxis.

Further Information

- How does the drug present, how is it consumed?
  - GHB and GBL both present as a salt. The powder is most often ingested after dissolution in water or another liquid. Some users inject dissolved GHB although this is rare.

  - GHB is often dissolved in alcoholic beverages in the party context. This also allows to mask its salty taste.
  - GBL is sometimes sold as a dietary supplement, although illegal in most developed countries.

- Context of using and desired effect
  - At low dose, GHB provokes euphoria, disinhibition, increased empathy, interest in sex. It is sometimes labelled as "liquid ecstasy".
  - "Chemsex" or "PNP/Party and Play" parties constitute typical contexts of using where GHB is used to increase/enhance sexual performances.
  - At higher does, GHB is sedative. Sleep can be the desired effect.
  - GHB is sometimes used by athletes as a doping agent. It has been shown to increase Growth Hormone secretion in men.
  - GHB has been called the "rape drug". Dissolved in the victim's drink, it can allow the perpetrator to take advantage of his/her victim who will have limited to no recollection of events.

References

1. Hockenhull J, Murphy KG, Paterson S. An observed rise in gamma-hydroxybutyrate-associated deaths in London: evidence to suggest a possible link with concomitant rise in chemsex. Forensic Sci Int. 2017;270:93–7.
2. Mason PE, Kerns WP. Gamma hydroxybutyric acid intoxication. Acad Emerg Med. 2002;9(7):730–9.

Chapter 33
Inhalant (Alkyl Nitrites, Nitrous Oxide, Hydrocarbons) Intoxication AKA: Poppers

Julien J. Cavanagh and Teresa Y. Smith

Introduction

There is a virtually infinite number of volatile substances of abuse that can be inhaled: glue, gasoline, spray paint, household products. These substances all have different effects. Nitrous oxide, known as whippets, act on NO receptors. They induce smooth muscle relaxation and hypotension. Alkyl Nitrites, known as poppers, are NO receptor agonists and have similar effect to whippets. They were first synthetized by Ballard in 1844 and used in angina.

J.J. Cavanagh, M.D. (✉)
Department of Neurology, State University of New York—Downstate Medical Center, Brooklyn, NY, USA
e-mail: julien.cavanagh@downstate.edu

T.Y. Smith, M.D., M.S.Ed., F.A.C.E.P.
Department of Emergency Medicine, State University of New York, Kings County Hospital, Brooklyn, NY, USA
e-mail: Teresa.Smith@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_33,

Reason for Visit to the Emergency Department

- Typical clinical presentation:
  - "Male individual presents with weakness, headache, epistaxis, low blood pressure after he starts feeling unwell during a sexual encounter"
- Symptoms
  - Psychiatric effects of poppers and whippets are typically temporary and last minutes.
  - Other inhalants can cause agitation and psychosis such as ideas of grandeur and hallucinations
- Signs

– Epistaxis	– Headache
– Facial rash	– Tremor
– Nausea/vomiting	– Seizure
– Anorexia	– Slurred speech
– Nystagmus and/or diplopia	– Tinnitus
– Coughing/wheezing	
– Muscular weakness	

Differential Diagnosis

- Diagnostic difficulties are most likely to stem from difficulty in identifying the product(s) used by the patient.

Labs/Testing/Imaging

- CBC
  - Bone marrow suppression (benzene abuse)
- Basic chemistry
  - Hypokalemia and/or metabolic acidosis (tulane abuse)
  - Renal impairment (halogenated hydrocarbons)

- Liver panel
  - Liver impairment (halogenated hydrocarbons)
- Blood gas
  - Methemoglobinemia (nitrate exposure),
- ECG (for arrhythmias)

Medical and Psychiatric Management

- Mostly supportive care during acute intoxication:
  - Maintain hemodynamic stability
  - Supplemental Oxygen
  - IV, fluid bolus
- Monitor oxygenation and ventilation and/or airway
- Benzodiazepines for agitation/psychosis

Caution

- Mixing phosphodiesterase-5 inhibitors (e.g. Sildenafil/Viagra®) with nitrite oxide can result in acute hypotension, stroke, myocardial infarction.
- Some inhaled substances can lead to lead poisoning (gasoline inhalants; methemoglobinemia) due to nitrate exposure. This is typical to intoxication by ingestion and less common with inhalation. Treat accordingly.
- Nitrous Oxide-causes vitamin B12 deficiency.
- Maculopathy has been described among chronic users.

Disposition

- Among occasional users, acute medical and psychiatric complications of inhalants are usually self-limited and respond well to supportive therapy; discharge is usually possible within 24 h.
- All users should be counseled:
  - Repeat/chronic users can be oriented towards peer support groups (e.g.: narcotics anonymous) or mental health clinics.

  - Inpatient detox programs rarely accept inhalants users.

- HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did the patient have unprotected sex immediately prior to visit).
  - Educate about safer sex practices.
  - Follow guidelines and consider Post-Exposure Prophylaxis.
  - Encourage repeat users to consider Pre-Exposure Prophylaxis.

Further Information

- How does the drug present, how is it consumed?
  - There are all kind of methods to use inhalants: sniffing, bagging, or huffing
  - Poppers and whippets are liquid substances usually packaged in small bottles. They are inhaled by approaching the opened bottle to a nostril.

- Context of using and desired effect
  - Poppers and whippets became popular in the gay community in the 1970s and more generally during the sexual liberation movement.
  - They induce a brief moment of euphoria, intense excitement, and decupled interest for the sexual partner.
  - Poppers' smooth muscle relaxation effect

References

1. Wu LT, Schlenger WE, Ringwalt CL. Use of nitrite inhalants ("poppers") among American youth. J Adolesc Health. 2005;37(1):52–60.
2. Muller AA, Muller GF. Inhalant abuse. J Emerg Nurs. 2006;32(5):447–8.
3. Kurtzman TL, Otsuka KN, Wahl RA. Inhalant abuse by adolescents. J Adolesc Health. 2001;28(3):170–80.

Chapter 34
MDMA (Ecstasy, Molly) Intoxication

Julien J. Cavanagh and Teresa Y. Smith

Introduction

Ecstasy existed since 1912. It belongs to the amphetamine family and therefore presents with a sympathomimetic profile. MDMA/Ecstasy is distinguished from other amphetamines by its intense serotoninergic activity. It makes Ecstasy the drug of empathy and openness. This lead to attempts to use MDMA as a psychotropic adjunct in the 1970s during psychotherapy. But Ecstasy rapidly became a drug of abuse which lead the DEA to label MDMA as a schedule 1 drug in 1985. It has remained since then in the United States and in most countries.

J.J. Cavanagh, M.D. (✉)
Department of Neurology, State University of New York—Downstate Medical Center, Brooklyn, NY, USA
e-mail: julien.cavanagh@downstate.edu

T.Y. Smith, M.D., M.S.Ed., F.A.C.E.P.
Department of Emergency Medicine, State University of New York, Kings County Hospital, Brooklyn, NY, USA
e-mail: Teresa.Smith@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_34,

Reason for Visit to the Emergency Department

- Typical clinical presentation:
  - "Young adult brought to the Emergency Room for altered mental status in context of nightlife (clubbing/rave party). Presents with hypertension, tachycardia, hyperthermia."
- Symptoms:
  - MDMA is not a psychedelic drug and psychotic symptoms such as hallucinations and delusions are rarely described. However, illusions such as color or objects distortion are common.
  - Acute anxiety is common and can lead to agitation.
- Signs:
  - Elevated blood pressure, tachycardia
  - Hyperthermia
  - Diaphoresis
  - Seizure
  - Altered Mental Status
  - Hyponatremia is the most common electrolyte disturbance. It is thought to be the result of polydipsia (most likely an attempt of the user to prevent hyperthermia).

Differential Diagnosis

- Other drugs of abuse that lead to increased sympathetic response, such as cocaine, amphetamine, and methamphetamine, may present with a clinical picture identical to MDMA intoxication.
- MDMA intoxication may also present similarly to anticholinergic overdose; differentiating factors may include the presence of diaphoresis and clear pressured speech in MDMA use, as opposed to anhydrosis and muffled speech in anticholinergic poisoning.

- If patients present with serotonin syndrome, differentials may also include neuroleptic malignant syndrome, malignant hyperthermia, and sedative-hypnotic withdrawal.
- Differentials for hyperthermia with altered mental status include CNS infections—meningitis, encephalitis.
- Differentials for hypotonic hyponatremia: exercise-induced with over-hydration and persistent ADH secretion; hypothyroidism, adrenal insufficiency, pregnancy; diuretic use, CHF, cirrhosis, and SIADH.

Labs/Testing/Imaging

- Metabolic panel, Liver Function Tests, CPK, Troponin.
- Most common lab abnormalities are:
  - metabolic acidosis,
  - elevated creatine kinase,
  - elevated troponin (demand ischemia),
  - transaminitis,
  - hyponatremia,
  - hyperkalemia/hypokalemia.
- Urine toxicology should be obtained but should not delay appropriate management.

Medical and Psychiatric Management

- The mainstay of treatment is benzodiazepines.
  - Benzodiazepines can help with sympathomimetic symptoms (hypertension and tachycardia);
  - Also serve as a sedative for medical restraint.
- Consider*:*
  - IV, fluid bolus
  - Electrolytes correction
- Acute hyponatremia can be corrected rapidly with normal or hypertonic saline. The risk of osmotic demyelination syndrome (central pontine myelinolysis) is limited.

Caution

- Like other exciting drugs, MDMA use can be associated with trauma.
- Physical restraining may potentiate rhabdomyolysis.
- We recommend extreme caution in the use of first generation antipsychotics such as butrophenones (haloperidol) as they may lower seizure threshold and also prolong QTc.
- Severe acute hyponatremia can lead to hyponatremic encephalopathy and neurogenic pulmonary edema.
- Hyperthermia and hyponatremia are unlikely to be associated. Think of other causes if they are (e.g.: sepsis such as meningitis).
- Serotonin syndrome:
  - MDMA can cause serotonin syndrome
  - Usually reversible with resting
  - Mild symptoms include agitation, confusion, hyperthermia, and jaw clenching.
  - More severe cases involve significant worsening of these symptoms and may also include tachycardia, myoclonus, shivering, and hyperreflexia.

Disposition

- Acute medical and psychiatric complications of MDMA are usually self-limited and respond well to simple therapy (benzodiazepine, fluids, electrolytes correction). Discharge is usually possible within 12–24 h
- If psychiatric symptoms such as anxiety and depression persist after 24 h:
  - suicidal risk must be thoroughly evaluated,
  - psychiatric consult can be indicated.
- All users should be counseled:
  - Repeat/chronic users can be oriented towards peer support groups (e.g.: narcotics anonymous) or mental health clinics.
  - Inpatient detox programs rarely accept synthetic drugs users.

- HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did the patient have unprotected sex immediately prior to visit).
  - Educate about safer sex practices.
  - Follow guidelines and consider Post-Exposure Prophylaxis.
  - Encourage repeat users to consider Pre-Exposure Prophylaxis.

Further Information

- How does the drug present, how is it consumed?
  - MDMA can present as pills or powder. Pills are usually known as "Ecstasy" and are typically decorated with symbols such as a smiley face, a currency sign, or a brand logo.
  - Pills can be ingested, crushed and snorted, dissolved in water, or even injected.
  - Molly is a purer form of MDMA presenting as a crystal powder that is usually snorted.
  - MDMA has many street names among which: X, E, XTC, Adam, Beans, Candy, Dancing Shoes, Disco Biscuits, Doves, E-bomb, Egg Rolls, Happy Pill, Hug Drug, Love Drug, Malcolm (or Malcolm X), Scooby Snacks, Smartees, Sweets, Skittles, Thizz, Vitamin E, Vitamin X, Vowels, Drop, Double Drop, Thizzing, Flip or Flipping, Roll, Rolling, Cuddle Puddle, E-Puddle, E-tard, Raver, Raving, and many more.
- Context of using
  - MDMA is the drug of clubbing by excellence. This "experience enhancer" fits well with night life, partying, and making new acquaintances. It is also widely consumed in giant techno music gathering known as "rave parties".
  - It is usually consumed on site as the onset of action is around 30 min. It's hence often purchased illegally at the club/venue itself.

  - It is estimated that half of ecstasy pills do not contain active substance and have no effect at all. This fact is largely known and leads users to sometimes double down on their consumption, in hope to maximize their chances of a high.
- Desired effect
  - MDMA users describe their experience with the drug as an intense feeling of affective enhancement. Partners appear more attractive, music sounds louder and crispier, and sex becomes more pleasurable. Overall, this intense feeling of empathy could be described as the feeling one gets when a favorite team is winning. Ecstasy recreates this irrepressible need to hug one another, this feeling of a deep connection with others sharing the same experience.

References

1. Richards JR, Bretz SW, Johnson EB, Turnipseed SD, Brofeldt BT, Derlet RW. Methamphetamine abuse and emergency department utilization. West J Med. 1999;170:198–202.
2. Alldredoe BK, Lowenstein DH, Simon RP. Seizures associated with recreational drug abuse. Neurology. 1989;39(8):1037–9.
3. Leach JP, Mohanraj R, Borland W. Alcohol and rugs in epilepsy: pathophysiology, presentation, possibilities, and prevention. Epilepsia. 2012;53:48–57.
4. Mayer KH, Colfax G, Guzman R. Club drugs and HIV infection: a review. Clin Infect Dis. 2006;42(10):1463–9.
5. Green AR, King MV, Shortall SE, Fone KCF. Ecstasy cannot be assumed to be 3,4-methylenediozyamphetamine (MDMA). Br J Pharmacol. 2012;166(5):1521–2.

Chapter 35
Opiates, Opioids Intoxication

Julien J. Cavanagh and Teresa Y. Smith

Introduction

Opiates are the psychoactive products found in the opium/poppy plant, mainly morphine and codeine. Opioids designate a larger family of chemical compounds that are similar to morphine in their action (e.g.: heroin hydrocodone, oxycodone, fentanyl). The use of opiates is as old as written human history and is well described in the antiquity by Hippocrates. Opioids receptors were discovered in the early 1970s. There are three known opioid receptors: μ, δ and κ. They regulate response to pain and other processes such as hunger, thirst, and breathing. Opioids also downregulate GABA which results in upregulation.

J.J. Cavanagh, M.D. (✉)
Department of Neurology, State University of New York—Downstate Medical Center, Brooklyn, NY, USA
e-mail: julien.cavanagh@downstate.edu

T.Y. Smith, M.D., M.S.Ed., F.A.C.E.P.
Department of Emergency Medicine, State University of New York, Kings County Hospital, Brooklyn, NY, USA
e-mail: Teresa.Smith@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_35,

Reason for Visit to the Emergency Department

Typical Clinical Presentation

- "An adult is brought by EMS because he looked drowsy after a minor car accident. On exam, the patient presents slow/slurred speech and pinpoint pupils"

Symptoms

- Acutely intoxicated opioids/opiates users usually appear slow, sedated, or even unconscious
- Most acute psychiatric presentations stem from withdrawal symptoms. This can include anxiety, nervousness, and even psychopathic behaviors.
- Naloxone push can result in agitation. This is transient but can be intense and require sedation and restraint.

Signs

- Decreased respiratory rate
- Myosis
- Lethargy with depressed mental status
- Decreased GI motility

Differential Diagnosis

- Other sedative drugs such as benzodiazepines or barbiturates can give a similar presentation. Naloxone response confirms the diagnosis of opiate/opioid intoxication.

Labs/Testing/Imaging

- ECG (looking for QTc prolongation)
- Basic labs: CBC, metabolic panel
- Chest X-Ray

Medical and Psychiatric Management

Most of the treatment is based on the monitoring or correcting of the respiratory depression.

Consider:

- Supplemental oxygen,
- Monitoring oxygenation (SaO_2 saturation) and/or capnography,
- Naloxone (nebulized, IM, IV) for reversal of opiate intoxication,
- Nasal trumpet (reduces tongue collapse and airway obstruction).

Caution

- Naloxone can lower seizure threshold: Start with very low doses (0.04 mg).
- Naloxone is very short acting. Once it metabolizes, patient may return to depressed respiratory state.
- Recent studies have shown that by-stander naloxone administration (even with refusal to be transported to a hospital) may be safe if patient maintains normal vital signs and mentation. If in the ED, 1-h observation sufficient.
- Naloxone-related ARDS (may be attributed to opiates themselves vs. the administration of naloxone; little is known about the exact etiology)

- Cardiac arrest caused by opiate intoxication, likely due to respiratory failure, has very poor outcomes.
- Methadone: is very long acting, beware of prolonged QTc
- Fentanyl: very short acting, often prescribed as a patch especially if mixed with benzodiazepines.
- Hydrocodone or oxycodone: often combined with acetaminophen (check levels).
- Opiates combined with benzodiazepines cause QT prolongation.
- Loperamide: anti-diarrheal agent that is an opioid agonist often used for opiate withdrawal symptoms, high doses produces both QRS and QT prolongation.
- Krokodil (desomorphine + adulterant like gasoline): cause skin necrosis.
- Dextromethorphan: found in cough suppressants. Not a pure opioid agonist but in high doses, has similar opiate intoxication symptoms. Risk of serotonin syndrome.
- Recreational drugs like "purple drank" that use cough syrup contain codeine and promethazine (not usually dextromethorphan).

Disposition

- Depending on severity of intoxication and complications, patients can be discharged within several hours to several days.
- Most psychiatric symptoms will stem from withdrawal syndrome.
- All users should be counseled
  - Inpatient detox programs typically accept opiates users. Once completed, such programs can orient patients to an outpatient clinic.
  - Opiate dependence clinic are widely available and many offer substitution treatment such as methadone or buprenorphine. These programs obey very strict rules and provide tight monitoring of their patients.

  - Methadone program participants must carry a card with their clinic's address and emergency phone number. Clinicians ordering methadone coverage in the emergency context should contact the patient's clinic in order to document enrollment and latest dosage.
  - Many jurisdictions now allow Naloxone kits. They can be used IM or intranasally. All users and their family/entourage should be counseled about their existence and usage.
- HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did the patient have unprotected sex immediately prior to visit).
  - Educate about safer sex practices.
  - Follow guidelines and consider Post-Exposure Prophylaxis.
  - Encourage repeat users to consider Pre-Exposure Prophylaxis.

Further Information

- How does the drug present, how is it consumed?
  - Opioids can present as powder or tablets. They are ingested, chewed, snorted, or injected intravenously. Other modes of delivery such as patches also exist.
- Context of using
  - Like most recreational drugs, opioids can be used alone or as part as a group experience.
  - It is not uncommon to see users mix opioids with other substances such as cocaine or amphetamines (speedballing).
- Desired effect
  - Heroin and other opioids are the opposite of a "upper" drugs like cocaine or amphetamines. Users describe a feeling of slight euphoria such as "mellowness" and "contentment" with initial intakes causing little to no after (crashing) or withdrawal effects.

References

1. Horburger D, Kurkcivan I, Sterz F, Schober A, Stockl M, Stratil P, Uray T, Testori C, Weizer C, Haugk M. Cardiac arrest caused by acute intoxication-insight from a registry. Ann Emerg Med. 2013;10:1443–7.
2. Reed CR, Gloaser FL. Drug-induced noncardiogenic pulmonary edema. Chest. 1991;100:1120–4.
3. Willman MW, Liss DB, Schartz ES, Mullins ME. Do heroin overdose patients require observation after receiving naloxone? Clin Toxicol. 2016:1–7.
4. Wightman RS, Hoffman RS, Howland MA, Rice B, Biary R, Lugassy D. Not your regular high: cardiac dysrhythmias caused by loperamide. Clin Toxicol. 2016;54(5):454–8.

Chapter 36
Psychedelic Drug (LSD, PCP, Hallucinogenic Mushrooms) Intoxication

Julien J. Cavanagh and Teresa Y. Smith

Introduction

Many substances with psychedelic properties have been used throughout the ages. They come from plants and mushrooms and are used spiritually during ceremonies or recreationally. LSD is the first of these substances to have been synthetized in 1938 by Swiss scientist Albert Hofmann.

Many other substances are classified as psychedelic drugs: mescaline, psilocybin (mushrooms), peyote (cactus), DMT, 2C, other. Psychedelic drugs can be separated in dopaminergic (psylocibin, DMT) and serotoninergic agonists (mescaline, 2C). Depending on their receptor affinity profile, they generate different user experience. LSD is considered a "dirty" drug because of its affinity for a multitude of receptors.

J.J. Cavanagh, M.D. (✉)
Department of Neurology, State University of New York—Downstate Medical Center, Brooklyn, NY, USA
e-mail: julien.cavanagh@downstate.edu

T.Y. Smith, M.D., M.S.Ed., F.A.C.E.P.
Department of Emergency Medicine, State University of New York, Kings County Hospital, Brooklyn, NY, USA
e-mail: Teresa.Smith@downstate.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_36,

Reason for Visit to the Emergency Department

Typical Clinical Presentation

- "Middle-age individual brought to the emergency department for intractable vomiting, altered mental status, internal preoccupation. Strongly smells of incense."

Symptoms

- Psychedelic drugs can generate anxiety-provoking hallucinations. Panic attacks are common.
- Agitation can either stem from hallucinations, psychotic disorganization, or anxiety.

Signs

- hypertension
- tachycardia
- hyperthermia
- nausea/vomiting/diarrhea

Differential Diagnosis

- The large panel of psychedelic drugs make clinical diagnosis challenging.
- The phenomenologic signature of psychedelic drugs is hallucination and other florid psychotic symptoms.
- Other drugs of abuse with similar affinity for dopaminergic (amphetamines) and serotoninergic receptors (MDMA) can give similar symptoms.

Labs/Testing/Imaging

- Monitor vitals including temperature, oxygenation.
- Metabolic panel:
  - Electrolyte disturbances
  - Increased creatinine (acute kidney injury)
- Creatine kinase:
  - Rhabdomyolysis
- Urine toxicology:
  - Diagnostic aid, multiple intoxication.
- EKG:
  - QTc and other modifications relating to electrolytes disturbances.

Medical and Psychiatric Management

- Hyperthermia is the most common of the severe symptomatology.

Consider:

- Benzodiazepines for agitation or psychotic symptoms.
- May need to add neuroleptics (beware of QTc prolongation).
- IV, fluid bolus.

Caution

- Persistent psychosis may be the result of underlying psychiatric illness.
- PCP- often added to cigarettes or marijuana (usually in the form of dipping the cigarette). PCP causes bizarre and violent behavior and rotatory nystagmus.
- Some hallucinogens (tryptamines) are structurally related to serotonin (causes visual hallucinations and risk of serotonin syndrome in severe overdose).

Disposition

- Among occasional users, acute medical and psychiatric complications of psychedelic drugs are usually self-limited and respond well to simple therapy (benzodiazepine, fluids, electrolyte correction). Discharge is usually possible within 24 h.
- Persistent psychiatric symptoms:
  - Persistent psychotic symptoms following use of psychedelic drugs are described. They can be flashbacks of hallucinations or persisting hallucinations (hallucinogen persistent perception disorder).
  - Persistent psychotic symptoms can be accompanied by mood and anxiety disorders.
- All users should be counseled
  - Repeat/chronic users can be oriented towards peer support groups (e.g.: narcotics anonymous) or mental health clinics.
  - Inpatient detox programs rarely accept psychedelic drugs users.
- HIV/STIs risk reduction strategy:
  - Evaluate risk taking behavior in general and for current visit (i.e.: did the patient have unprotected sex immediately prior to visit).
  - Educate about safer sex practices.
  - Follow guidelines and consider Post-Exposure Prophylaxis.
  - Encourage repeat users to consider Pre-Exposure Prophylaxis.

Further Information

- How does the drug present, how is it consumed?
  - Psychedelic drugs can present as tablets/pills, liquid, decoction, or blotter form (stamp-size piece of paper soaked with the substance).

  - Mushrooms are usually consumed dry. They can be chopped and fill gel caps.

- Context of using
  - Ritual/cultural/spiritual ceremonies.
  - Recreational use in single or collective use.

- Desired effect
  - Hallucinatory experience, synesthetic experience (music sounding like color, odors smelling like sounds, etc.), derealization, depersonalization (out-of-body-experience).

References

1. Callaway CW, Clark RF. Hyperthermia in psychostimulant overdose. AnnEmergMed. 1994;24(1):68.
2. Kometer M, Vollenweider FX. Serotonergic hallcinogen-induced visual perceptual alterations. Curr Top Behav Neurosci. 2016:1–26.
3. Araujo AM, Carvalho F, Bastos ML, Guedes de Pinho P, Carvalho M. The hallucinogenic world of tryptamines: an updated review. Arch Toxicol. 2015;89(8):1–73.

Section V
Withdrawal Syndromes

Section Editor: Jagoda Pasic, M.D., Ph.D.

Chapter 37
Acute Withdrawal: General Principles

Jagoda Pasic

Introduction

Patients with substance use disorders are frequent utilizers of medical and psychiatric services in the emergency department. Recognition of common withdrawal syndromes is essential in formulating a differential diagnosis and providing appropriate medical care. In the DSM-5, withdrawal syndromes are uniquely defined for each substance class but share core features in that, (1) they occur after cessation of heavy and prolonged drug use, (2) the cause significant distress or impairment in social, occupational or interpersonal function and, (3) the symptoms cannot be better attributed to another mental or medical disease. In this section the major substance withdrawals include alcohol, benzodiazepine, barbiturate, cannabis, opioid and stimulant (amphetamine-type substances and cocaine). While abrupt cessation of other substances listed in the DSM-5, such as tobacco, can lead to withdrawal, focus has been placed on the most common withdrawal syndromes that can lead to medical concerns.

J. Pasic, M.D., Ph.D.
Department of Psychiatry and Behavioral Sciences,
Harborview Medical Center, University of Washington
School of Medicine, Seattle, WA, USA
e-mail: jpasic@uw.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_37,

Chapter 38
Alcohol Withdrawal

Paul Borghesani

Introduction

Over 70 million individuals worldwide suffer from alcohol use disorders (AUDs) and alcohol is thought to account for up to 1.8 million deaths each year. Over one third of patients admitted to intensive care units have AUDs while upwards of 8% of all hospitalized patients are treated for alcohol withdrawal symptoms (AWS). Low to moderate alcohol consumption produces euphoria and excitation via activation of glutamatergic neurotransmission while higher concentrations produce severe intoxication via GABAergic mechanisms. Acute withdrawal unmasks the hyper-excitatory state of the brain causing anxiety, agitation and autonomic activation characteristic of AWS. AWS typically begin 1–3 days after the last drink.

P. Borghesani, M.D., Ph.D.
Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, University of Washington School of Medicine, Seattle, WA, USA
e-mail: paulrb@uw.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_38,

Symptoms

Starting 6 h after cessation of drinking and lasting 6–48 h
Anxiety
Agitation
Irritability
Diaphoresis
Nausea/vomiting
Headache
Visual, tactile or auditory hallucinations (for up to 6 days)

Signs

Autonomic activation (tachycardia, tachypnea, hypertension)
Fever
Hand tremor
Ataxia
Hyper-reflexia

Serious and Potentially Dangerous Symptoms/Signs

Occurs in roughly 5–15% patients 6–48 h after last drink
- Seizures (generalized tonic-clonic with short post-ictal period)
- Alcoholic hallucinosis without severe autonomic hyperactivity

Delirium Tremens (DTs) occurring 48–96 h after last drink
- Severe autonomic hyperactivity (fever, hypertension, tachycardia)
- Delirium

Wernicke-Korsakoff Syndrome (WKS)

Differential

Alcohol withdrawal is a clinical diagnosis and as such a history of recent cessation of excessive drinking is essential. Although rarely mimicking historically defined WKS, other medical conditions are frequently co-morbid with AWS and need to be evaluated, including: head trauma, CNS infections, metabolic derangements, hepatic failure, GI bleeding and intoxication with other substances.

Wenicke-Korsakoff Syndrome (WKS):

*for full information, please see Chapter 27

WKS is characterized by mixed behavioral, cognitive and neurological symptoms caused by chronic thiamine (vitamin B1) deficiency of any etiology, including chronic alcoholism. It is rare to observe the classic triad of ophthalmalgia, ataxia and mental status changes and thus it has been suggested that if any of these symptoms are seen in conjunction with suspected malnutrition (e.g., in chronic alcoholism), WKS should be suspected. As the disease progresses, cognitive dysfunction deteriorates, most dramatically leading to overt confabulation. Treatment of WKS focusses on immediate parenteral thiamine replacement (200–500 mg TID IM/IV for 3 days) followed by long-term oral replacement (100–200 mg PO QD-BID). Initial parenteral replacement is necessary since alcohol reduces the absorption, metabolism, storage and physiologic effects of thiamine. Although behavioral and neurologic symptoms can resolve, restoration of cognitive function is rarely possible even with continued thiamine replacement and abstinence from alcohol.

Laboratory Studies

Blood alcohol level
Basic metabolic panel to assess for hypoglycemia and electrolyte disorders
Liver function tests to assess for hepatic injury
Complete blood count to assess for anemia and thrombocytopenia
Coagulation studies if acute bleeding is feared (e.g., with esophageal varices)

Treatment

- Benzodiazepines are first line of treatment of AWS and no individual agent has been shown to be clearly superior to others. Given the prevalence of hepatic dysfunction in chronic alcoholics and the elderly, lorazepam is an appropriate first choice. Diazepam and chlordiazepoxide, though hepatically metabolized, are good alternatives given their longer duration of action. Excessive treatment should be avoided and can result in symptoms similar to intoxication including sedation, ataxia and confusion.
- It is feasible to dose benzodiazepines through one of three strategies:
  - loading with long acting agents until the patient is sedated and without symptoms,
  - using a fixed dose/taper protocol based on clinical judgement, or
  - symptom triggered strategies (see table). Symptom triggered strategies are preferred unless severe AWS are of concern.
- Antiepileptic medications including carbamazepine (200–400 mg PO BID), valproate (250–500 PO TID) and gaba-

pentin (600 mg PO TID) have also been shown to reduce AWS and seizures. Advantages include their lack of abuse potential and utility in treating mood disorders but clinical unfamiliarity limits their widespread adoption.
- Antiadrenergic medications such as clonidine (0.1–0.2 mg PO BID) and propranolol (10–20 mg PO TID) can reduce heartrate, blood pressure and anxiety but are only recommended as adjuvants to benzodiazepines and antiepileptic medications because they don't prevent seizures or DTs.
- Treatment of WKS involves thiamine replacement in conjunction with benzodiazepines, and, prior to glucose replacement (if clinically indicated).

Long-Term Treatment

- Motivational interviewing and Screening, Brief Intervention and Referral to Treatment (SBIRT) to explore and facilitate long term treatment options.
- Abstinence can be promoted in motivated individuals willing to take disulfirm which inhibits aldehyde dehydrogenase and induces unpleasant physiologic effects if alcohol is consumed.
- Harm reduction is facilitated by oral treatment with naltrexone or acamprosate which can be considered after appropriate medical assessment.
- Some studies suggest oral treatment with gabapentin or topiramate reduces alcohol consumption in motivated individuals.

Tool

Alcohol withdrawal protocol

	Example Rxs	Benefits	Drawbacks
Loading dose	Diazepam 10–20 mg q1 h *or* Chordiazepoxide 25–100 mg q1–2 h	Compatible with short ED visits Less monitoring after initial treatment Limited staff training needed Mild sedation is a goal	Potentially reinforcing of short/frequent ED visits May mask symptoms/signs of other illness
Fixed dose	Lorazepam 2 mg PO/IV q2–6 h for 48–72 h with taper over next 7–10 days	Good choice for severe AWS or comorbid benzodiazepine dependence Limited staff training needed Less potential of symptom embellishment	Increased total duration and dose of benzodiazepines Potential for abuse if prescriptions provided

	Example Rxs	Benefits	Drawbacks
Symptom triggered	Lorazepam 2 mg PO q2–6 h for CIWA-Ar scores of >10 points [4]	Good choice for mild/moderate AWS Less total benzodiazepines Shorter duration of treatment Less risk of over/under medication	Trained personnel needed Potential for symptom embellishment

References

1. Jesse S, et al. Alcohol withdrawal syndrome: mechanisms, manifestations, and management. Acta Neurol Scand. 2017;135:4–16.
2. Isenberg-Grzeda E, Kutner HE, Nicolson SE. Wernicke-Korsakoff-syndrome: under-recognized and under-treated. Psychosomatics. 2012;53:507–16.
3. Sachdeva A, Choudhary M, Chandra M. Alcohol withdrawal syndrome: benzodiazepines and beyond. J Clin Diagn Res. 2015;9(9):VE01–9.
4. Sullivan JT, Sykora K, Schneiderman J, Naranjo CA, Sellers EM. Assessment of alcohol withdrawal: the revised clinical institute withdrawal assessment for alcohol scale (CIWA-Ar). Br J Addict. 1989;84:1353–7.
5. Strobbe S. Prevention and screening, brief intervention, and referral to treatment for substance use in primary care. Prim Care. 2014;41(2):185–213.

Chapter 39
Amphetamine Withdrawal

Jagoda Pasic

Introduction

DSM-V diagnostic criteria for stimulant withdrawal are combined for amphetamine and cocaine though clinically there are distinct differences. Acute withdrawal symptoms typically occur within 24 h after abrupt discontinuation of using high amounts of amphetamine-type substances over a prolonged period of time. There are two phases of withdrawal syndrome: (1) the initial "crash" that resolves within about a week, and (2) a subacute or protracted withdrawal symptoms generally resolve in 3 weeks. The withdrawal symptoms are experienced by amphetamine dependent individuals as severe and intolerant. This often compromises long-term success to achieve abstinence because a single use of amphetamine immediately removes the discomfort.

J. Pasic, M.D., Ph.D.
Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, University of Washington School of Medicine, Seattle, WA, USA
e-mail: jpasic@uw.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_39,

Initial Symptoms

Intense craving
Intense dysphoria
Agitation
Paranoia
Insomnia
Vivid, unpleasant dreams
Fatigue

Subsequent Symptoms

Depressive symptoms
Decreased energy
Anhedonia
Suicidal ideation
Increased appetite
Hypersomnia

Signs

Agitation/anxiety
Irritability
Lethargy

Serious and Potentially Dangerous Symptoms/Signs

Symptoms
Suicidal ideation

Signs
Agitation
Violent behavior

Differential

Cocaine Withdrawal
Major Depression
Bipolar Disorder
Alcohol Withdrawal
Hypothyroidism

Treatment

- Methamphetamine is the most common type of amphetamine-like substance being used, however, there is poor evidence for treating withdrawal syndrome.
- Treatment is supportive and symptom driven, e.g. agitation to be treated with benzodiazepines.
- Allowing patients to sleep may avoid the need for hospitalization.
- No evidence that pharmacological agents relieve withdrawal symptoms of amphetamine-like substances (one pilot study using modafinil).

Long-Term Treatment

- Chemical dependency treatment for Relapse prevention
- Contingency management
- No current evidence to support the clinical use of pharmacological agents

Tool

Amphetamine Withdrawal Mnemonic: "De-PANTS"
Depression (suicidal ideation)
Psychomotor Changes (agitation, later retardation)
Appetite Increase
Nightmares (unpleasant dreams)

Tiredness (fatigue)
Sleep (insomnia, later changes to hypersomnia)

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. Washington, DC: American Psychiatric Association; 2013.
2. Lee N, Pennay A, Hester R, McKetin R, Nielsen S, Ferris J. A pilot randomized controlled trial of modafinil during acute methamphetaminewithdrawal: feasibility, tolerability and clinical outcomes. Drug AlcoholRev.2013;32(1):88–95.doi:10.1111/j.1465-3362.2012.00473.x.
3. Pennay AE, Lee NK. Putting the call out for more research: the poor evidence base for treating methamphetaminewithdrawal. Drug Alcohol Rev. 2011;30(2):216–22. doi:10.1111/j.1465-3362.2010.00240.x.
4. Shoptaw SJ, Kao U, Heinzerling K, Ling W. Treatment for amphetamine withdrawal. CochraneDatabase Syst Rev. 2009;2:CD003021. doi:10.1002/14651858.CD003021.pub2.

Chapter 40
Barbiturate Withdrawal

Paul Zarkowski

Introduction

Barbiturates share a common mechanism of action with benzodiazepines as an allosteric modulator of GABA receptors, but at higher serum levels, barbiturates also act as agonists at the GABA active site. For this reason, barbiturates have a lower therapeutic index than benzodiazepines. Barbiturates are classified according to duration of action as shown in the Tool. The barbiturates that produce withdrawal symptoms generally have short to intermediate action, with half-lives between 10 and 50 h. Secobarbital, at least 0.6 g/day for 30 days or 0.4 g/day for 90 days, is associated with physical dependence. Acute withdrawal symptoms may occur between 8 and 16 h after cessation of regular use. Seizures may occur between 24 and 115 h.

P. Zarkowski, M.D.
Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, University of Washington School of Medicine, Seattle, WA, USA
e-mail: pzark@uw.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_40,

Symptoms

- Restlessness
- Insomnia
- Difficulty concentrating
- Neurological
  - Dizziness
  - presyncope
  - headache
  - numbness/tingling
  - tinnitus
- Muscular
  - weakness
  - twitches
- Gastrointestinal
  - nausea/vomiting
  - diarrhea
- Psychiatric
  - agitation
  - anxiety/panic attacks
  - irritability
  - depression
  - psychosis
  - suicidal thoughts

Signs

Photophobia
Diaphoresis
Hyperreflexia
Tremor

Life-Threatening Symptoms/Signs

Seizures
Catatonia

Autonomic instability
Hyperthermia
Coma

Differential

Seizure Disorder
Alcohol withdrawal
Encephalopathy
Encephalitis
Sepsis
Metabolic derangement

Laboratory Studies

- No studies confirm diagnosis
- Consider urine toxicology for co-occurring substances
- All studies would be rule/out

Treatment

- Pentobarbitol 0.2–0.4 mg q4 to 6 h for acute stabilization.
- Oral phenobarbital 120 mg q1 to 2 h until resolution of withdraw symptoms or the presence of at least three of the following: nystagmus, drowsiness, ataxia, dysarthria, or emotional liability.
- Low dose antipsychotic if patient continues to be psychotic

Long-Term Treatment

As many cases of barbiturate dependence are initiated during treatment of migraine headaches, starting a non-habit forming alternative decreases the risk of relapse. In other cases, referral to a substance dependence treatment program with cognitive behavioral elements should be considered.

Tool

Barbiturate half life classification

Barbiturate generic name	**Barbiturate trade name**	**Classification of half life**	**Equivalent does to 10 mg of diazepam**
Thiopental	Pentothal	UltraShort	
Secobarbital	Seconal	Short	100 mg
Pentobarbital	Nembutal	Short	100 mg
Amobarbital	Amytal	Short	
Butalbital	Fiorinal, Fioricet	Medium	
Phenobarbital	Luminal	Long	30 mg

References

1. Raja M, Altavista MC, Azzoni A, Albanese A. Severe barbiturate withdrawal syndrome in migrainous patients. Headache. 1996; 36(2):119–21.
2. Robinson GM, Sellers EM, Janecek E. Barbiturate and hypnosedative withdrawal by a multiple oral phenobarbital loading dose technique. Clin Pharmacol Ther. 1981;30(1):71–6.
3. Sarrecchia C, Sordillo P, Conte G, Rocchi G. Barbiturate withdrawal syndrome: a case associated with the abuse of a headache medication. Ann Ital Med Int. 1998;13(4):237–9.
4. Sellers EM. Alcohol, barbiturate and benzodiazepine withdrawal syndromes: clinical management. CMAJ. 1988;139(2):113–20.
5. Sullivan JT, Sellers EM. Treatment of the barbiturate abstinence syndrome. Med J Aust. 1986;145(9):456–8.

Chapter 41
Benzodiazepine Withdrawal

Paul Zarkowski

Introduction

Benzodiazepine withdrawal is usually detected by a careful history. The patient may recount stopping a medication and, within a few days, having a myriad of symptoms. Physical dependence can occur as early as 3–6 weeks of therapeutic dosing.Withdrawal appears to be more severe after abrupt cessation of high dose benzodiazepines or rapid reduction of those with short half-lives. Withdrawal symptoms may present within 6–8 hours of decreasing levels of short acting benzos with half live less than 10 h, peaking in intensity on the second day and resolving on days three and four. Withdrawal symptoms from longer acting benzodiazepines may not present for a week, peaking on the second week and resolving on the third or fourth week.

P. Zarkowski, M.D.
Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, University of Washington School of Medicine, Seattle, WA, USA
e-mail: pzark@uw.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_41,

Symptoms

- Poor concentration
- Insomnia
- Sensory hypersensitivity
- Neurological
  - Dizziness/light-headedness
  - Headache
  - Numbness/tingling
  - Tremor
  - Tinnitus
- Muscular
  - Stiffness
  - Weakness
  - Twitches
- Gastrointestinal
  - Abdominal distension
  - Nausea/vomiting
  - Diarrhea
  - Loss of appetite and weight loss
- Psychiatric
  - Agitation
  - Anxiety/panic attacks
  - Irritability
  - Depression
  - Psychosis
  - Suicidal thoughts

Signs

- Akathisia & related physical restlessness
- Photophobia
- Hyper-reflexia
  - Tremor
  - Diaphoresis
  - Fasciculations
  - Paraesthesia: perioral extremities

Life-Threatening Symptoms/Signs

- Seizures
- Catatonia
- Autonomic instability
- Hyperthermia
- Coma

Differential

- Seizure Disorder
- Alcohol withdrawal
- Encephalopathy
- Encephalitis
- Sepsis
- Metabolic derangement

Laboratory Studies

- No studies confirm diagnosis
- Consider urine toxicology for co-occurring substances
- All studies would be rule/out

Treatment

- Long acting benzodiazepines can be given acutely (e.g., diazepam 20 mg per hour until symptoms are suppressed) and gradually tapered over 6–8 weeks.
- Consider antiepileptic drugs (carbamazepine and valproate) as they may provide the ability to accelerate taper the benzodiazepine while providing seizure prophylaxis
- Low dose antipsychotic if patient continues to be psychotic
- Hospitalization for severe withdrawal
- Consider symptom triggered withdrawal protocol for severe withdrawal

Long Term Treatment

Referral to chemical dependence treatment with cognitive behavioral elements.

Sustained abstinence from benzodiazepines can often be facilitated by addressing the symptoms of primary anxiety disorders with non-habit forming treatment alternatives before starting a benzodiazepine taper.

Tool

Benzodiazepine half-life comparison

Benzodiazepine, generic name	**Benzodiazepine, trade name**	**Half-life in hours [active metabolite]**	**Equivalent dose to 10 mg of diazepam**
Alprazolam	Xanax	6–12	1 mg
Lorazepam	Ativan	10–20	2 mg
Temazepam	Restoril	8–22	20 mg
Diazepam	Valium	20–100 [36–200]	–
Clorazepate	Tranxene	[36–200]	20 mg
Chlordiazepoxide	Librium	5–30 [36–200]	50 mg

References

1. Sellers EM. Alcohol, barbiturate and benzodiazepine withdrawal syndromes: clinical management. CMAJ. 1988;139(2):113–20.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders:DSM-5. Washington, DC: American Psychiatric Association; 2013.
3. Maxwell JS, Ward NG. Psychotropic drugs fast facts. 2nd ed.

Chapter 42
Cannabis Withdrawal

Jagoda Pasic

Introduction

Cannabis withdrawal occurs after cessation of prolonged and heavy cannabis use. Prior to DSM-V, cannabis withdrawal had not been recognized as a separate syndrome. Intense daily use of cannabis increases the risk of cannabis dependence and the risk of cannabis withdrawal after cessation. Earlier initiation of cannabis use, increased use of more potent forms of cannabis (e.g. the flowering heads of the female cannabis plant) and frequent use of water-pipes may lead to increased serum levels of tetrahydrocannabinol and a higher risk of dependence. Cannabis withdrawal causes functional impairment which is dependent on symptom severity, and is predictive of relapse to cannabis. Onset of symptoms is usually within 24–48 h of abstinence, typically reaching a peak within the first week.Symptoms may persist for up to 3–4 weeks. Withdrawal symptoms may cause discomfort, distress and functional impairment and are a significant barrier to achieving abstinence.

J. Pasic, M.D., Ph.D.
Department of Psychiatry and Behavioral Sciences,
Harborview Medical Center, University of Washington
School of Medicine, Seattle, WA, USA
e-mail: jpasic@uw.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_42,

Symptoms

- Anxiety
- Sleep difficulty (e.g. insomnia, nightmares)
- Decreased appetite or weight loss
- Depression
- Abdominal pain
- Sweating
- Fever
- Chills
- Headache

Signs

- Irritability, anger or aggression
- Depressed affect
- Nervousness or anxiety
- Restlessness
- Shakiness/tremor
- Constricted or blunted affect
- Diaphoresis

Life-Threatening Symptoms/Signs

The cannabis withdrawal syndrome is not life threatening and it is not associated with significant medical or psychiatric consequences.

Differential

- Anxiety syndrome, panic attack
- Depression
- Other substance intoxication or withdrawal
- Primary insomnia
- Side effect to medication
- Metabolic derangement

Treatment

Treatment is supportive that may address intolerable symptoms such as insomnia.

- There are no accepted pharmacotherapies for the treatment of cannabis withdrawal or cessation.
- The most promising are the newer tetrahydrocannabinol agonists, such as nabiximols and nabilone.
- Non-cannabinoid medications used to treat other substance-use disorders (clonidine, naltrexone, nefazodone, mirtazapine, bupropion, venlafaxine, divalproex, lithium, oxytocin, quetiapine, baclofen) have been largely negative for cannabis withdrawal.
- The evidence base for the anticonvulsant gabapentin and the glutamatergic modulator N-acetylcysteine is weak, and need further investigation.

Long-Term Treatment

- Most consistent evidence supports combination of Motivation Enhanced Therapy and Cognitive Behavioral Therapy with abstinence-based incentives (Cochrane Database).
- Harm reduction model may include switching to cannabidiol (CBD), a cannabinoid analogue.

Tool

Additive/ impurity	**Comment**
Cocaine, PCP	Adulterant to increase potency
Daminocide	Plant growth regulator banned by California as a carcinogen
Isopentane	Residual solvent used in the extraction for cannabis concentrates
Paclobutrazol	Pesticide not registered with EPA for use on food crops
Paraquat	Herbicide, causes Parkinson's disease

References

1. Copeland J, Pokorski I. Progress toward pharmacotherapies for cannabis-use disorder: an evidence-based review. Subst Abuse Rehabil. 2016;7:41–53.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. Washington, DC: American Psychiatric Association; 2013.
3. Gates PJ, Sabioni P, Copeland J, Le Foll B, Gowing L. Psychosocial interventions for cannabis use disorder. Cochrane Database Syst Rev. 2016;5:CD005336. doi:10.1002/14651858.CD005336.pub4.
4. Marshall KS, Gowing L, Ali R. Pharmacotherapies for cannabis withdrawal. Cochrane Database Syst Rev. 2014;12:CD008940. doi:10.1002/14651858.CD008940.pub2.
5. Milin R, Manion I, Dare G, Walker S. Prospective assessment of cannabis withdrawal in adolescents with cannabis dependence: a pilot study. J Am Acad Child Adolesc Psychiatry. 2008; 47(2):174–9.
6. Crippa JA, Hallak JE, Machado-de-Sousa JP, Queiroz RH, Bergamaschi M, Chagas MH, Zuardi AW. Cannabidiol for the treatment of cannabiswithdrawal syndrome: a case report. J Clin Pharm Ther. 2013;38(2):162–4. doi:10.1111/jcpt.12018.
7. Raber JC, Elzinga S, Kaplan C. Understanding dabs: contamination concerns ofcannabisconcentrates and cannabinoid transfer during the act of dabbing. J Toxicol Sci. 2015;40(6):797–803. doi:10.2131/jts.40.797.
8. Stone D. Cannabis,pesticidesand conflicting laws: the dilemma for legalized states and implications for public health. Regul Toxicol Pharmacol. 2014;69(3):284–8. doi:10.1016/j.yrtph.2014.05.015. Epub 2014 May 20.
9. Lanaro R, Costa JL, Cazenave SO, Zanolli-Filho LA, Tavares MF, Chasin AA. Determination of herbicides paraquat, glyphosate, and aminomethylphosphonic acid in marijuana samples by capillary electrophoresis. J Forensic Sci. 2015;60(Suppl 1):S241–7. doi:10.1111/1556-4029.12628. Epub 2014 Nov 20.

Chapter 43
Cocaine Withdrawal

Jagoda Pasic

Introduction

Of all illegal substances, cocaine leads to most emergency department visits in the US: per the Drug Abuse Warning Network, in 2011 there were 162 cocaine-related visits per 100,000 population in contrast to 83 heroin-related ED visits. However, there are no data that specifically pertain to cocaine withdrawal. Acute withdrawal symptoms or the "crash" typically occurs after repetitive use or binging. Initial withdrawal symptoms may occur 6–12 h after heavy use of cocaine with protracted withdrawal symptoms occurring up to 96 h after use. Abrupt cocaine cessation can cause significant distress and social and occupational impairment.

J. Pasic, M.D., Ph.D.
Department of Psychiatry and Behavioral Sciences, Harborview Medical Center, University of Washington School of Medicine, Seattle, WA, USA
e-mail: jpasic@uw.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_43,

Symptoms

- Intense dysphoria
- Distress
- Depressive symptoms
- Feelings of lassitude
- Increased appetite
- Sleep disturbance
- Vivid, unpleasant dreams
- Cocaine craving
- Paranoia
- Suicidal ideation

Signs

- Injected conjunctiva
- Excessive tearfulness
- Irritability
- Lethargy

Serious and Potentially Dangerous Symptoms/ Signs

Symptoms

- Suicidal ideation

Signs

- Agitation
- Violent behavior

Differential

- Major depression or other depressive disorders
- Bipolar Depression
- Amphetamine Withdrawal

Treatment

- Symptom driven, e.g., agitation to be treated with benzodiazepines.
- Allowing patients to sleep may avoid the need for hospitalization.
- Psychiatric admission may be indicated in patients who remain suicidal as cocaine use has been reported to be independently associated with attempted suicide, (OR: 1.96), though, such patients tend to have a short hospital stay.
- Antidepressants have no utility in treating acute cocaine withdrawal.
- Antipsychotics have no efficacy advantages over placebo in regard to cocaine abstinence.

Long-Term Treatment

- Chemical dependency treatment for relapse prevention
- Contingency management
- No current evidence to support the clinical use of pharmacological agents (negative trials with anticonvulsants, antipsychotics, antidepressants, disulfiram, dopamine agonists)

Tool

Common additives and effects

Additive/impurity	**Comment**
Benzocaine, lidocaine, procaine	Substitute to mimic analgesic effect, causes methemoglobinemia
Boric acid	Pesticide added to increase anesthetic effect
Caffeine	Substitute to mimic stimulant effect
Ether	Used as a solvent in free base cocaine, causes burns of throat and nasal sinuses
Mannitol	Added due to anticaking properties
Phenacetin	Metabolizes into acetaminophen, but banned by FDA as a carcinogen in 1983
Levamisole	Antiparasitic used as adulterant, causes vasculitis and agranulocytosis

References

1. Artenie AA, Bruneau J, Zang G, Lespérance F, Renaud J, Tremblay J, Jutras-Aswad D. Associations of substance use patterns with attempted suicide among persons who inject drugs: can distinct use patterns play a role? Drug Alcohol Depend. 2015;147:208–14.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. Washington, DC: American Psychiatric Association; 2013.
3. Ithman MH, Gopalakrishna G, Beck NC, Das J, Petroski G. Predictors of length of stay in an acute psychiatric hospital. J Biosafety Health Educ. 2014;2:119.
4. Kishi T, Matsuda Y, Iwato N, Carrell C. Antipsychotics for cocaine or psychostimulant dependence: systematic review and meta-analysis of randomized, placebo-controlled trials. J Clin Psychiatry. 2013;74:1169–80.
5. Minozzi S, Saulle R, De Crescenzo F, Amato L. Psychosocial interventions for psychostimulant misuse. Cochrane Database Syst Rev. 2016;9:CD011866. doi:10.1002/14651858.CD011866.pub2.
6. de Souza LM, Rodrigues RR, Santos H, Costa HB, Merlo BB, Filgueiras PR, Poppi RJ, Vaz BG, Romão W. A survey of adulterants used to cut cocaine in samples seized in the Espírito Santo state by GC-MS allied to chemometric tools. Sci Justice. 2016;56(2):73–9. doi:10.1016/j.scijus.2015.11.006.
7. Berman M, Paran D, Elkayam O. Cocaine-induced vasculitis. Rambam Maimonides Med J. 2016;7(4):e0036. doi:10.5041/RMMJ.10263.
8. Taylor RF, Bernard GR. Airway complications from free-basingcocaine. Chest. 1989;95(2):476–7.
9. Saraghi M, Hersh EV. Potential diversion of local anesthetics from dental offices for use ascocaineadulterants. J Am Dent Assoc. 2014;145(3):256–9. doi:10.14219/jada.2013.33.
10. Cunningham EE, Venuto RC, Zielezny MA. Adulterants in heroin/cocaine: implications concerning heroin-associated nephropathy. Drug Alcohol Depend. 1984;14(1):19–22.

Chapter 44
Opiate/Opioid Withdrawal

Paul Borghesani

Introduction

Opiates are naturally occurring substances (morphine, opium and codeine) while opioids are synthesized (heroin, oxycodone, hydromorphone, fentanyl, buprenorphine, methadone). They are prescribed worldwide to treat acute and chronic pain, diarrhea and cough. Their euphoric, sedating and anxiolytic effects make them common substances of abuse and approximately three million people in the United States and 16 million worldwide have a current or past opiate use disorder. Withdrawal typically occurs between 6–48 h after cessation of use with longer acting substances (e.g., methadone) causing a more delayed withdrawal. While intoxication and iatrogenic reversal can be life-threatening, natural withdrawal is not, requiring supportive care only.

P. Borghesani, M.D., Ph.D.
Department of Psychiatry and Behavioral Sciences,
Harborview Medical Center, University of Washington
School of Medicine, Seattle, WA, USA
e-mail: paulrb@uw.edu

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_44,

Symptoms

- Dysphoria
- Malaise
- Chills
- Yawning
- Nausea
- Vomiting
- Muscle cramps
- Insomnia

Signs

- Rhinorrhea (runny nose)
- Lacrimation (tearing)
- Increased bowel sounds and diarrhea
- Piloerection
- Mydriasis
- Myalgia and arthralgia
- Can be rated using the Clinical Opioid Withdrawal Scale (COWS)

Differential

Withdrawal from alcohol and sedative/hypnotics can appear very similar to opioid withdrawal but the former are routinely associated with tachycardia and hypertension that, while occurring during agitation of any etiology, are not the norm for opioid withdrawal. Intoxication with stimulants can appear similar to opioid withdrawal but the former is usually associated with far greater agitation and can be confirmed by history.

Laboratory Studies

- Urine toxicology including opioid differentiation, if possible
- Consider blood glucose to evaluate hypoglycemic coma
- Consider creatine kinase especially if patient was found down/immobile
- Consider chest radiograph to evaluate for aspiration
- Consider serum acetaminophen given opioid preparations and possibility of suicide attempt
- Consider electrocardiogram if methadone overdose is suspected

Treatment of Withdrawal

No specific pharmacotherapy is required and medications are for symptom management only

- Use clonidine 0.1–0.2 mg PO for anxiety, restlessness and hypertension
- Use antihistamines PO or IM for anxiety, restlessness, nausea and vomiting
- Use loperamide 4 mg PO for diarrhea and stomach cramps
- Use NSAIDs or acetaminophen for myalgias and arthragias
- Use baclofen 5–10 mg PO for muscle cramping
- Methadone 10–20 mg PO can be considered and usually relieves symptoms but does not reduce use. Not recommended for iatrogenic withdrawal.

Long-Term Treatment

- Referral to licensed providers delivering long-term treatment with methadone or buprenorphine.
- Consideration of antagonist therapy with naltrexone in motivated individuals seeking abstinence.
- Referral to chemical dependency treatment or Narcotics Anonymous.

Tool

Opiate/opioid Comparison table

Opiate/opioid	**Equivalent dose**	**Half-life in hours [including active metabolite]**	**Duration of acute effects**
Oxycodone	20–30 mg PO	2–3 h	3–6 h
Heroin	5 mg IV/SC/ Inhaled	< 10 min but has active metabolites	3–6 h
Buprenorphine	04 mg IM/IV 0.4 mg SL	2–3 h	8–12 h
Fentanyl	0.1 mg IM/ IV/SC	3–5 h	1–3 h
Hydrocodone	30 mg PO	3–5 h	4–8 h
Methadone	10 mg PO	12–150 h	3–12 h
Morphine	10 mg IM/IV/ SQ 30 mg PO	2–3 h	3–4 h 3–6 h
Codeine	75 mg IM/IV/ SQ 150 mg PO	2–4 h	4–6 h

Note: both the serum half-life and equivalent doses are given for drug naïve patients. Actual duration and extent of effects vary substantially between individuals

References

1. Schuckit MA. Treatment of opioid-use disorders. N Engl J Med. 2016;375(4):357–68.
2. Wesson DR, Ling W. The Clinical Opiate Withdrawal Scale (COWS). J Psychoactive Drugs 2003, 35:253–299, see http://www.mdcalc.com/cows-score-opiate-withdrawal/
3. Amato L, Davoli M, Minozzi S, Ferroni E, Ferri M. Methadone at tapered doses for the management of opioid withdrawal. Cochrane Database Syst Rev. 2013;2:CD3409.

Section VI
Toxicologic Syndromes

Section Editor: Bryan Corbett, M.D.

Chapter 45
Antidepressant Discontinuation Syndrome

Bryan Corbett and Kimberly D. Nordstrom

Introduction

The etiology of antidepressant discontinuation syndrome is not fully understood but it tends to occur after the abrupt discontinuation of an antidepressant, though may also occur even with a taper. It is known to occur with monoamine oxidize inhibitors (MAOI), tricyclic antidepressants (TCA), selective serotonin reuptake inhibitors (SSRI), serotonin-norepinephrine reuptake inhibitors (SNRI) and atypical antidepressants. The syndrome can occur with missing only one dose of medications with short half-lives, such as paroxetine and venlafaxine. Symptoms can last for weeks, sometimes longer. It is important to recognize this syndrome in order to avoid very costly work ups.

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA

K.D. Nordstrom, M.D., J.D (✉)
Colorado Department of Human Services, Office of Behavioral Health, Denver, CO, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA
e-mail: Nordstrom_kimberly@yahoo.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_45,

Symptoms (Associated with Serotonergic Agents)

General: flu-like symptoms, weakness, fatigue, lethargy, chills, headache, lightheadedness, dizziness
Sleep: Insomnia, hypersomnia
Sensory disturbances: 'shock–like' sensations and parathesias
Neuromuscular: myalgia, dystonia, imbalance
Gastrointestinal: nausea, vomiting, diarrhea, abdominal pain
Psychological: severe anxiety, irritability, agitation, depression, hallucinations

Signs

None specific to this syndrome

Life-Threatening Symptoms/Signs

Delirium has been associated with tricyclic and monoamine oxidase inhibitor withdrawal.

Differential (Related to Prominent Symptoms)

Influenza infection
Neurological Disorder
Gastrointestinal disturbance
Psychiatric: panic disorder, generalized anxiety disorder, depression

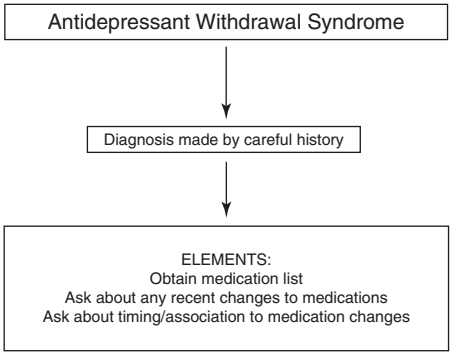
Laboratory Studies

None specific for syndrome
Consider electrolytes if patient with significant GI disturbance

Treatment

- Restart antidepressant with suggested plan for outpatient psychiatrist to taper slowly
- For severe TCA and MAOI withdrawal, consider use of benzodiazepines for severe agitation
- Supportive care
- Patient education

Tool



References

1. Fava GA, Gatti A, Belaise C, Guidi J, Offidani E. Withdrawal symtpoms after selective serotonin reuptake inhibitor discontinuation: a systematic review. Psychother Psychosom. 2015;84:72–81.
2. Schatzberg AF, Blier P, Delgado PL, Fava M, Haddad PM, Shelton RC. Antidepressant discontinuation syndrome: consensus panel recommendations for clinical management and additional research. J Clin Psychiatry. 2006;67(Suppl4):27–30.
3. Lejoyeux M, Ades J, Mourad S, Solomon J, Dilsaver S. Antidepressant withdrawal syndrome: recognition, prevention and management. CNS Drugs. 1996;5(4):278–92.
4. Shelton RC. Steps following attainment of remission: discontinuation of antidepressant therapy. Prim Care Companion J Clin Psychiatry. 2001;3(4):168–74.

Chapter 46
Anti-Muscarinic Toxicity

Bryan Corbett

Introduction

Anti-muscarinic toxicity, also commonly referred to as anticholinergic toxicity, is the result of muscarinic acetylcholine receptor blockade. Muscarinic acetylcholine receptors are found in the CNS as well as in the PNS at the parasympathetic postganglionic synapse. This receptor distribution explains the classic symptoms and signs of the toxidrome. Many medications have anti-muscarinic toxicity and the toxidrome is usually the result of supratherapeutic dosing of these medications. In addition to medications, various plants possess anti-muscarinic activity and can also cause the toxidrome. Recreational use of both medications and plants, presumably for the mind altering effects, is not uncommon particularly in the adolescent population.

Symptoms

Confusion
Disorientation
Somnolence

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA
e-mail: bcorbett1982@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_46,

Agitation
Difficulty urinating
Constipation

Signs

Hyperthermia, >100.4 °F or >38.0 °C
Mydriasis
Dry Mucous Membranes
Tachycardia
Urinary Retention
Flushing
Decreased Bowel Sounds
Lack of Sweating

Life-threatening Symptoms/Signs

Anti-muscarinic toxicity is generally not life threatening, however, fatalities have occurred. Deaths seem to be associated with hot environments where an inability or impairment in sweating predisposes one to hyperthermia. It should also be noted that many common medications with anti-muscarinic effects, such as diphenhydramine, also block sodium channels which can cause life-threatening arrhythmias and seizures. There is some question as to whether seizures occur in isolated anti-muscarinic toxicity or absent associated hyperthermia. Regardless, seizures indicate a significant poisoning.

Differential

The differential diagnosis for anti-muscarinic toxicity is broad and encompasses a number of diagnoses that can cause confusion and agitation. The tachycardia that usually accompanies anti-muscarinic toxicity can help to narrow the differential diagnosis down to a more select list. A key finding that helps to differentiate anti-muscarinic toxicity from many on this more select list is the lack of sweating, however, this is not pathognomic by any means.

Encephalitis/Meningitis
Excited catatonia
Heat-stroke
Malignant hyperthermia
Nonconvulsive status epilepticus
Pheochromocytoma
Serotonin syndrome
Neuroleptic malignant syndrome
Sympathomimetic intoxication (cocaine, methamphetamine, PCP)
Thyroid storm
Alcohol/benzodiazepine/barbiturate withdrawal
Withdrawal from intrathecal baclofen

Testing

- Diagnosis is clinical and based on typical signs and symptoms of anti-muscarinic toxicity along with a good history
- Laboratory workup should be guided by clinical context
- A good history of ingestion may prompt only a CBC, CMP, APAP level, and EKG
- An APAP level should always be checked as many OTC anti-muscarinic medications exist as combination products with acetaminophen
- Ammonia, TSH, Vitamin B12 level, HIV, CTH, LP, UA, CXR, ASA level should be considered in less clear cut cases of ingestion

Treatment

- Treatment is generally supportive
- Gastrointestinal decontamination with activated charcoal (AC) should be considered in patients presenting within 1 hour of ingestion. AC should not be used when the patient does not have a secure airway or there is risk of aspiration such as with depressed mental status, non-compliance, and seizures.

- Physostigmine, an acetylcholinesterase inhibitor, can be used for diagnostic and treatment purposes to reverse the CNS and PNS manifestations of anti-muscarinic toxicity
  - Dosing: In our practice, we administer 1 mg over 1–2 min, and repeat ×1 if suboptimal effect. The package insert recommends 2 mg slowly which may be repeated ×1 for life threatening signs.
  - QRS complex >100 ms is a generally accepted contraindication to physostigmine use.

Tool

Physical Exam Findings and End Organ Effects in Antimuscarinic Toxicity

Organ System/Examination Site	Effects/Findings
Heart Rate	Increased
Pupil Size	Increased
Skin	Dry (decreased diaphoresis)
Mucous Menbranes	Dry (decreased secretions)
GI	Constipation, decreased bowel sounds (decreased peristalsis)
Bladder	Urinary retention
CNS	Agitation, confusion, seizures

References

1. American Academy of Clinical Toxicology and European Association of Poisons Centres and Clinical Toxicologists. Position Paper: single-dose activated charcoal. Clin Toxicol. 2005;43:61–87.
2. Forrer GR, Miller JJ. Atropine coma-somatic therapy in psychiatry. Am J Psychiatry. 1958;115:455–8.
3. Centers for Disease Control and Prevention (CDC). Jimson weed poisoning—Texas, New York, and California, 1994. MMWR Morb Mortal Wkly Rep. 1995;44(3):41–4.
4. Patel RJ, Saylor T, Williams SR, Clark RF. Prevalence of autonomic signs and symptoms in antimuscarinic drug poisonings. J Emerg Med. 2004;26(1):89–94.
5. Physostigmine Salicylate Injection [package insert]. Lake Forest: Akorn. 2008.
6. Suchard JR. Assessing physostigmine's contraindication in cyclic antidepressant ingestions. J Emerg Med. 2003;25:185–91.

Chapter 47
Common Medications Which May Mimic Psychiatric Symptoms

Bryan Corbett

Introduction

Virtually any medication which crosses the blood-brain barrier may cause neuropsychiatric effects. Nonetheless, some common drug classes are more predisposed to this. Below are common classes which may present with alterations in mental status or behavior:

Name of drug class	Examples	Effects
Sympathomimmetics	Cocaine, methamphetamine	Agitation, paranoia, hallucinations
Antipsychotics	Risperidone, olanzapine, haloperidol	Neuroleptic malignant syndrome, antimuscarinic toxidrome, CNS depression, confusion

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA
e-mail: bcorbett1982@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_47,

Name of drug class	Examples	Effects
Antiepileptics	Topiramate, lamotrigine, levetiracetam, carbamazepine	Confusion, delirium, CNS depression, carbamazepine may cause antimuscarinic toxidrome
Calcium-channel blockers, Beta blockers	Amlodipine, verapamil, carvedilol, propranolol	Delirium, confusion (usually in setting of hypotension)
Antihistamines	Diphenhydramine, doxylamine	Antimuscarinic toxidrome, confusion, agitation
Benzodiazepines	Alprazolam, diazepam	CNS depression
Tricyclic Antidepressants	Amitriptyline, nortriptyline	Antimuscarinic toxidrome, CNS depression, confusion
Steroids	Prednisone, dexamethasone	Psychosis, insomnia, mania

Reference

1. The Medical Letter. 2008 December 15/29;50:1301/1302.Accessed at http://secure.medicalletter.org/sites/default/files/freedocs/w1301c.pdf on December 21 2015.

Chapter 48
Lithium Toxicity

Bryan Corbett

Introduction

Lithium, a monovalent cation, is used as a mood stabilizer in bipolar disorder as well as in the adjunctive treatment of other psychiatric illnesses. The CNS is the major target in therapeutic as well as toxic doses. A narrow therapeutic index, 0.6–1.2 mmol/L, and an inherently at risk population increase the risk of toxicity. Lithium is relatively rapidly absorbed from the GI tract with therapeutic dosing although sustained release preparations do exist. Tissue distribution, most importantly to the CNS, however, is more prolonged. This is clinically relevant in that three subtypes of toxicity are recognized; acute, chronic, and acute on chronic.

Acute Toxicity

In acute toxicity, the patient has no body burden of lithium prior to the ingestion in question. Initial levels can be quite high prior to redistribution but these values don't represent what

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA
e-mail: bcorbett1982@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_48,

tissue levels and thus clinical findings will be at equilibrium. Early on GI symptoms predominate with nausea, vomiting, and diarrhea. As the lithium distributes into tissues, specifically the CNS, neurologic manifestations may occur (see below).

Chronic Toxicity

Lithium has a narrow therapeutic index and even a small increase in dose or decrease in elimination can lead to toxicity when tissue stores already exist. Lithium is almost entirely renally cleared and chronic toxicity often occurs in situations that decrease GFR such as with NSAID or ACEI use. Dehydration such as with gastroenteritis or other sodium avid states (chronic diuretic use) also decrease clearance via an increase in lithium reabsorption given its similarity to sodium. Manifestations of chronic toxicity are mostly neurologic and are delineated below. Blood levels may not predict the severity of clinical findings.

Acute on Chronic Toxicity

Acute ingestion of lithium in an individual with pre-existing body stores will produce this subtype of toxicity. Presentation is a mix of acute and chronic toxicity with significant GI as well as neurologic findings discussed previously.

Gastrointestinal Manifestations

Nausea
Vomiting
Diarrhea

Neurological Manifestations

Tremor (can be present in absence of true toxicity)
Hyperreflexia
Choreoathetoid movements

Dysarthria
Nystagmus
Ataxia
Confusion
Agitation
Delirium
Seizures
Coma
Hyperthermia

Other Manifestations Associated with Lithium Use

Nephrogenic Diabetes Insipidus
Hypo and Hyperthyroidism
Hyperparathyroidism

Life-Threatening Symptoms/Signs

More severe neurological manifestations include CNS depression, agitation, seizures, delirium, and coma. Rarely cardiovascular manifestations such as heart block and other rhythm disturbances may occur.

Differential

The differential diagnosis of altered mental status or behavioral changes is large. The lack of specific vital sign abnormalities and varying presentation of lithium toxicity limits the ability to develop a succinct list of likely alternative diagnoses. Essentially any process that can result in alterations of mental status or affect behavior should be entertained as on the differential and assessed in the clinical context. The various neurological findings can help guide differential diagnosis development, however. A list of broad categories with individual representative examples is included to aid in

developing a reasonable differential given each patient's unique clinical context.

Vascular: vascular dementia
Infectious: encephalitits
Trauma: subdural hematoma
Autoimmune: Anti-NMDA encephalitis
Metabolic: hypo/hypernatremia
Neoplastic: cerebral metastases
Neurologic: normal pressure hydrocephalus
Medication (including alcohol and illicit drugs): intoxication or withdrawal

Testing

- Lithium level: marker of exposure, may not correlate with clinical severity
- A BMP is essential to assess sodium levels as well as BUN and Creatinine to assess renal function (remember lithium is almost entirely renally excreted)
- TSH in chronic users
- Urine osmolality and electrolytes if diabetes insipidus is suspected
- An EKG and APAP level in cases of known or suspected intentional ingestion
- Ammonia, Liver panel, Vitamin B12 level, HIV, CTH, LP, UA, CXR, ASA level should be considered in the clinical context

Treatment

- Decontamination
  - Activated charcoal does not bind lithium and has no role in decontamination
  - Whole bowel irrigation with polyethylene glycol (PEG) can be considered when sustained release preparations are ingested

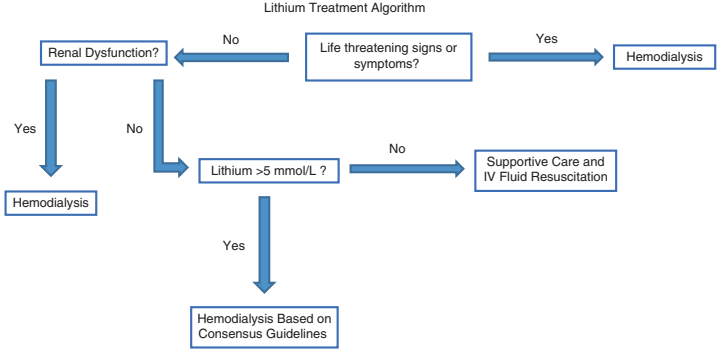
Give 1–2 L of PEG an hour until clear rectal effluent (this can be hard for the patient to tolerate)

- Other treatment is primarily geared towards maintaining lithium elimination by the kidneys
- IV normal saline to maintain intravascular volume and GFR (also addresses hyponatremia)
- Hemodialysis (HD)
  - Effectively removes lithium from blood compartment
  - May need multiple runs as blood levels will spike afterwards as intracellular lithium stores re-equilibrate
  - Consensus recommendations have been published and are as follows (it should be noted these are essentially expert consensus opinion with a level of evidence of 1D and 2D):

Impaired kidney function and lithium >4 mmol/L
Life threatening signs or symptoms
Lithium >5 mmol/L

  - Patients with severe neurological findings are also good candidates for early HD

Tool



References

1. Decker BS, Goldfarb DS, Dargan PI, Friesen M, Gosselin S, Hoffman RS, Lavergne V, Nolin TD, Ghannoum M. Extracorporeal treatment for lithium poisoning: systematic review and recommendations from the EXTRIP workgroup. Clin J Am Soc Nephrol. 2015;10:875–87.
2. Timmer RT, Sands JM. Lithium intoxication. J Am Soc Nephrol. 1999;10:666–74.
3. Waring WS. Management of lithium toxicity. Toxicol Rev. 2006;25(4):221–30.

Chapter 49
Neuroleptic Malignant Syndrome

Bryan Corbett and Michael P. Wilson

Introduction

Neuroleptic Malignant Syndrome (NMS) is the result of dopamine receptor blockade in the CNS. Classically it is characterized by the tetrad of altered mental status, rigidity, hyperthermia, and autonomic dysfunction. In practice, however, NMS exists on a spectrum and not all four features need be present for the diagnosis. It is also important to understand that less severe dopamine receptor blockade may present only with dystonia or akathisia. While not classified as NMS, these manifestations are the result of the same pathophysiologic mechanism.

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_49,

Any dopaminergic blocking medication may cause NMS. The typical antipsychotics, some of the most potent dopamine receptor blockers, are frequently implicated but atypical antipsychotics are also a known cause. Other medications used for nausea such as prochlorperazine (brand name Compazine) and promethazine (brand name Phenergan) also possess antidopaminergic activity and, in fact, belong to the family of typical antipsychotics. Withdrawal of dopamine agonists, such as occurs with drug holidays in Parkinson's patients, has also been implicated in the development of NMS. NMS is idiosyncratic and may occur after chronic use of antidopaminergic medications even without dosing changes. Onset is generally not abrupt and occurs over a number of days.

Symptoms

Altered Mental Status

- Confusion
- Disorientation
- Delirium
- Altered level of consciousness

Patients may complain of various issues related to rigidity such as difficulty walking.

Signs

Autonomic Dysfunction

- Tachycardia
- Hypertension
- Tachypnea
- Rigidity
- Hyperthermia>100.4 °F or >38.0 °C
- Hyperreflexia
- Tremor
- Diaphoresis

Life-Threatening Symptoms/Signs

NMS has a reported fatality rate ranging from 5–20%. As such any patient with the diagnosis of NMS is in a life-threatening situation. Specific concerning findings include severe rigidity which can result in muscle breakdown and resultant rhabdomyolysis and renal failure. Significant hyperthermia is also concerning, can result in multiorgansystem failure, and should be treated aggressively.

Differential

Antimuscarinic poisoning
Dystonic reaction
Encephalitis
Excited catatonia
Heat-stroke
Malignant hyperthermia
Meningitis
Nonconvulsive status epilepticus
Pheochromocytoma
Porphyria
Rabies
Serotonin syndrome
Strychnine poisoning
Sympathomimetic intoxication, cocaine, methamphetamine, PCP
Tetanus
Thyroid storm
Baclofen Withdrawal

Testing

- Diagnosis is based on history, clinical findings, and exclusion of other diagnoses.
- Criteria exist to aid in diagnosis as discussed below.

- Basic labs including a BMP, CBC, and UA
- A CPK should be checked to assess for muscle breakdown
- A liver panel, ammonia, TSH, CT head, LP, CXR, Vitamin B12 and Thiamine levels, HIV, RPR, and VDRL should be considered in the clinical context

Treatment

- Largely supportive, stabilize ABCs.
- IV fluid resuscitation
- Liberal use of benzodiazepines is a mainstay of treatment
- Aggressive cooling measures
- Patients with resistant hyperthermia can be intubated and paralyzed to prevent heat production from muscles
- Discontinuation of dopamine antagonists or re-institution of dopamine agonists
- Bromocriptine (a dopamine receptor agonist) can be used but its benefit over supportive care alone is debatable. In addition, it is only available as an oral preparation complicating administration in critically ill patients. Dosing is 2.5–10 mg 3–4× daily.

Tool

Tool 1:Diagnostic Criteria for NMS (While Helpful It Should Be Noted These Have Not Been Externally Validated and Are Based on Expert Opinion)

Recent dopamine antagonist exposure OR dopamine agonist withdrawal
Hyperthermia: >100.4 °F or >38.0 °C on at least two occasions
Rigidity
Mental status alteration

Creatinine kinase elevation at least 4 times the upper limit of normal

Sympathetic nervous system lability:

blood pressure elevation, ≥25% above baseline;
blood pressure fluctuation, ≥20 mm Hg (diastolic) OR ≥25 mm Hg (systolic) change within 24 h

Tachycardia ≥25% above baseline AND tachypnea ≥50% above baseline

Negative workup for other causes (CSF is characteristically normal)

Tool 2

Neuroleptic Malignant Syndrome Vs. Serotonin Syndrome

Differentiating Factors	Neuroleptic Malignant Syndrome	Serotonin Syndrome
Medication Type	Dopamine antagonists, withdrawal of dopamine agonists	Serotonin Agonist
Predictability	Idiosyncratic	Drug-drug interaction or overdose
Onset	Insidious	Rapid
Recovery	Prolonged	Generally rapid (excluding medications with long half life)
Neuromuscular findings	Lead-pipe rigidity	Clonus

References

1. Chang C-K, Harrison S, Lee W, et al. Ascertaining instances of neuroleptic malignant syndrome in a secondary mental health-care electronic medical records database: the SLAM BRC case register. Ther Adv Psychopharmacol. 2012;2(2):75–83.
2. Levenson JL. Neuroleptic malignant syndrome. Am J Psychiatry. 1985;142:1137–45.

3. Trollor JN, Chen X, Chitty K, Sachdev PS. Comparison of neuroleptic malignant syndrome induced by first- and second-generation antipsychotics. Br J Psychiatry. 2012;201:52–6.
4. Gurrera RJ, Caroff SN, Cohen A, et al. An international consensus study of neuroleptic malignant syndrome diagnostic criteria using the Delphi method. J Clin Psychiatry. 2011;72:1222–8.
5. Strawn JR, Keck PE, Caroff SN. Neuroleptic malignant syndrome. Am J Psychiatry. 2007;164(6):870–6.
6. Reulbach U, Dutsch C, Biermann T, et al. Managing an effective treatment for neuroleptic malignant syndrome. Crit Care. 2007;11:R4.
7. Sakkas P, Davis JM, Janicak PG, et al. Drug treatment of the neuroleptic malignant syndrome. Psychopharmacol Bull. 1991;27:381–4.
8. Rosebush PI, Stewart T, Mazurek MF. The treatment of neuroleptic malignant syndrome. Are dantrolene and bromocriptine useful adjuncts to supportive care? Br J Psychiatry. 1991;159:709–12.
9. Rosenberg MR, Green M. Neuroleptic malignant syndrome: review of response to therapy. Arch Intern Med. 1989;149(9):1927–31.

Chapter 50
Salicylate Toxicity

Bryan Corbett

Introduction

Multiple forms of salicylates exist; the most commonly encountered is acetylsalicylic acid, brand name Aspirin. All salicylates are non-steroidal anti-inflammatory drugs meaning they inhibit the enzyme cyclooxygenase (COX). COX inhibition ultimately inhibits prostaglandin formation which mediate inflammation and fever. They do, however, possess unique properties that are of consequence in toxicity which will be reviewed here. Salicylates destroy the proton gradient in mitochondria used to generate ATP. Clinically this manifests as metabolic acidosis and hyperthermia. Salicylates also directly stimulate the respiratory center of the brain leading to a primary respiratory alkalosis. Clinically this presents as tachypnea, and given the concurrent metabolic acidosis, produces the classic ABG finding of a mixed respiratory alkalosis and metabolic acidosis. Adult Respiratory Distress Syndrome (ARDS) and cerebral edema also occur and por-

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA
e-mail: bcorbett1982@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_50,

tend a poor prognosis. Both acute and chronic toxicity occur; the main difference being the more insidious onset in chronic and the associated lower serum salicylate concentrations. Chronic toxicity often occurs with repeated supratherapeutic doses of aspirin when being used to treat chronic conditions such as arthritis

Symptoms

Nausea
Abdominal pain
Tinnitus
Confusion
Agitation
Lethargy
Hallucinations

Signs

Vomiting
Tachypnea
Diaphoresis
Tachycardia
Hyperthermia
Seizures
Coma
ARDS

Life-Threatening Symptoms/Signs

Alterations in mental status indicate CNS involvement and along with ARDS indicate severe toxicity. Development of a respiratory acidosis rather than an alkalosis on arterial or venous blood gas (ABG/VBG) also indicates severe poisoning.

Differential

The differential diagnosis for salicylate toxicity is broad and the list below is not comprehensive. Tachycardia, tachypnea, and hyperthermia can be manifestations of many processes and salicylate toxicity is often confused for sepsis in older individuals. A key finding in identifying salicylate toxicity (other than history and levels) is a mixed primary respiratory alkalosis and metabolic acidosis on ABG or VBG.

Encephalitis/Meningitis
Heat-stroke
Malignant hyperthermia
Nonconvulsive status epilepticus
Pheochromocytoma
Serotonin syndrome
Neuroleptic malignant syndrome
Sympathomimetic intoxication (cocaine, methamphetamine, PCP)
Thyroid storm
Alcohol/benzodiazepine/barbiturate withdrawal
Withdrawal from intrathecal baclofen
Sepsis

Testing

- Serum salicylate levels (actionable levels discussed below)
- BMP to check renal function and assess for a gap acidosis
- A liver panel can be considered if there is concern for concurrent APAP ingestion
- ABG or VBG: early can only be respiratory alkalosis, later respiratory alkalosis and metabolic acidosis
- An EKG and APAP level in cases of known or suspected intentional ingestion
- CXR

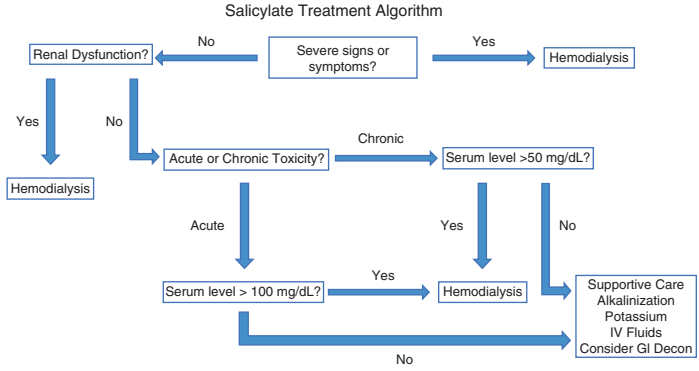
- CTH if altered mental status to assess for cerebral edema
- Ammonia, Vitamin B12 level, HIV, LP, UA, should be considered in the clinical context

Treatment

- Decontamination
  - Activated Charcoal (AC)
    - Aspirin has delayed and erratic absorption in overdose so AC can be administered greater than 1 h since ingestion
- Multiple doses of AC up to ~4 times can be given due to this delayed absorption
- Ideal dosing is a 10:1 ratio of AC/Salicylate, in practice doses greater than 50 g are hard to tolerate
- Do not give AC when there is concern for aspiration (i.e. depressed mental status or seizures)
  - Whole bowel irrigation with polyethylene glycol (PEG) can be used when enteric coated preparations are ingested
    - Give 1–2 L of PEG an hour until clear rectal effluent (this can be hard for the patient to tolerate)
- Elimination
  - Urinary and Serum alkalinization
    - Increases ionic form of aspirin which increased serum concentration and urine concentrations leading to increased renal elimination
    - Treat hypokalemia as this inhibits ability to raise urine pH
    - 1–2 ampule bolus of sodium bicarbonate followed by D5W spiked with 3 ampules of sodium bicarbonate at 1.5–2 times maintenance rate
  - Hemodialysis (HD)
    - Effectively eliminates salicylate
    - May need multiple runs if continued GI absorption
    - Indications:
      - ARDS, altered mental status, cerebral edema, severe electrolyte abnormalities or acidosis (severe signs or symptoms)

- Renal failure or AKI
- Specific levels (> 100 mg/dL or >50 mg/dL for acute and chronic poisoning respectively)
- Severe clinical findings should prompt initiation of HD despite serum concentrations

Tool



References

1. Barone JA, Raia JJ, Huang YC. Ann Emerg Med. 1988;17(1):34–7.
2. Hill JB. Experimental salicylate poisoning: observations on the effects of altering blood pH on tissue and plasma salicylate concentrations. Pediatrics. 1971;47:658–65.
3. Juurlink DN, Gosselin S, Kielstein JT, Ghannoum M, Lavergne V, Nolin TD, Hoffman RS. Extracorporeal treatment for salicylate poisoning: systematic review and recommendations from the EXTRIP workgroup. Ann Emerg Med. 2015;66:165–81.
4. LugassyDM. Salicylates (Chapter 39). In: HoffmanRS, HowlandMA, LewinNA, NelsonLS, GoldfrankLR. Goldfrank's toxicologic emergencies10th. New York: McGraw-Hill; 2015. pgs. 516–527.
5. Temple AR. Acute and chronic effects of aspirin toxicity and their treatment. Arch Intern Med. 1981;141:364–9.

Chapter 51
Serotonin Syndrome

Bryan Corbett and Kimberly D. Nordstrom

Introduction

Serotonin syndrome (SS) is the result of excess serotonergic activity in the CNS. It can occur with therapeutic use of multiple serotonergic medications or from supratherapeutic dosing of a single serotonergic medication. Classic manifestations include altered mental status, autonomic hyperactivity, and clonus. Like Neuroleptic Malignant Syndrome (NMS), SS exists on a continuum and not all above mentioned manifestations need be present for a diagnosis. Onset is typically rapid occurring over the course of minutes up to about 24 h.

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA

K.D. Nordstrom, M.D., J.D (✉)
Colorado Department of Human Services, Office of Behavioral Health, Denver, CO, USA

American Association for Emergency Psychiatry, Parker, CO 80134, USA
e-mail: Nordstrom_kimberly@yahoo.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_51,

Symptoms

- Agitation
- Disorientation
- Restlessness
- Delirium

Signs

- Clonus (typically more pronounced in the lower extremities)
- Hyperreflexia
- Muscular hyperactivity
- Diarrhea
- Akathisia
- Rigidity
- Hypertension
- Tachycardia
- Diaphoresis
- Hyperthermia
- Mydriasis
- Seizures

Life-Threatening Symptoms/Signs

Serotonin Syndrome is a medical emergency and should be treated as such. Life-threatening manifestations include rhabdomyolysis with resultant renal failure. Hyperthermia can lead to multi-organ system failure, cardiopulmonary collapse, and death. Episodes of disseminated intravascular coagulation have also been reported.

Differential

- Antimuscarinic poisoning
- Dystonic reaction

Encephalitis
Excited catatonia
Heat-stroke
Malignant hyperthermia
Meningitis
Nonconvulsive status epilepticus
Pheochromocytoma
Porphyria
Rabies
Serotonin syndrome
Strychnine poisoning
Sympathomimetic intoxication, cocaine, methamphetamine, PCP
Tetanus
Thyroid storm
Baclofen Withdrawal

Testing

- Diagnosis is based on history, clinical findings, and exclusion of other diagnoses.
- The Hunter Criteria (see below) are a set of decision rules used to diagnose SS. They are internally validated and found to have good agreement with the diagnosis by a clinical toxicologist.
- Basic labs including a BMP, CBC, and UA
- A CPK should be checked to assess for muscle breakdown
- A liver panel, ammonia, TSH, CT head, LP, CXR, Vitamin B12 and Thiamine levels, HIV, RPR, and VDRL should be considered in the clinical context

Treatment

- Largely supportive, stabilize ABCs.
- IV fluid resuscitation
- Liberal use of benzodiazepines is a mainstay of treatment
- Aggressive cooling measures

- Patients with resistant hyperthermia can be intubated and paralyzed to prevent heat production from muscles
- Discontinuation of all serotonergic medications
- Cyproheptadine, an early anti-histamine with anti-serotonergic activity, can also be used. It is only available as an oral preparation complicating administration in critically ill patients. No evidenced based dosing recommendations exist. A starting dose of 8 mg repeated as necessary is reasonable based on case reports.

Tool

Tool 1: Hunter Criteria

If any of the following, may diagnose SS:
Spontaneous clonus
Inducible clonus + agitation OR diaphoresis
Ocular clonus + agitation OR diaphoresis
Tremor + hyperreflexia
Hypertonia + temp above 38 + ocular clonus OR inducible clonus

Sensitivity 84%, specificity 97%

- How to induce clonus: Clonus refers to a persistent reflex contraction of a muscle after an initial stimulus. Clonus can often be best appreciated with regard to the Achilles reflex. To check for clonus, forcefully dorsiflex the foot at the ankle and maintain slight dorsal pressure on the foot.

Tool 2

Neuroleptic Malignant Syndrome Vs. Serotonin Syndrome

Differentiating Factors	Neuroleptic Malignant Syndrome	Serotonin Syndrome
Medication Type	Dopamine antagonists, withdrawal of dopamine agonists	Serotonin Agonist
Predictability	Idiosyncratic	Drug-drug interaction or overdose
Onset	Insidious	Rapid
Recovery	Prolonged	Generally rapid (excluding medications with long half life)
Neuromuscular findings	Lead-pipe rigidity	Clonus

References

1. Boyer EW, Shannon M. The serotonin syndrome. N Engl J Med. 2005;352:1112–20.
2. Dunkley EJC, Isbister GK, Sibbritt D, Dawson AH, Whyte IM. The hunter serotonin toxicity criteria: simple and accurate diagnostic decision rules for serotonin toxicity. QJM. 2003;96:635–42.
3. Graudins A, Stearman A, Chan B. Treatment of the serotonin syndrome with cyproheptadine. J Emerg Med. 1998;16:615–9.
4. Nordstrom K, Vilke GM, Wilson MP. Psychiatric emergencies for clinicians: The ED management of serotonin syndrome. J Emerg Med. 2016;50(1):89–91.

Chapter 52
Neuropsychiatric Complications of Steroids

Bryan Corbett and Michael P. Wilson

Introduction

Steroids are ubiquitous medications used to treat myriad disease processes. They are not without complications or side effects, however. In addition to weight gain, hyperglycemia, and immunosuppression, neuropsychiatric complications may also be present. Often referred to as "steroid psychosis" the neuropsychiatric complications of steroid use are much more diverse and not necessarily so obvious as the moniker implies. Manifestations range from an increased sense of well-being to hypomania, mania, depression, and frank psychosis. In addition, cognitive symptoms may be

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA

M.P. Wilson, M.D., Ph.D. (✉)
University of Arkansas for Medical Sciences, Little Rock, AR, USA
e-mail: mpwilso1@outlook.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_52,

present such as difficulty with attention, concentration, and memory impairment. Symptoms generally start within days to weeks of starting steroids. Cases have occurred hours after starting therapy as well as after discontinuation of steroids, however. The true incidence is unclear and various studies report a broad range of 2–60%. Higher doses of steroids do convey an increased risk for developing neuropsychiatric symptoms, however, doses as low as 2.5 mg of prednisone daily have resulted in symptoms. In addition, essentially all routes of administration have been associated with the development of symptoms including inhaled, intra-articular, epidural, topical, and of course oral. Outcomes are generally good with resolution of neuropsychiatric symptoms within 6 weeks for 90% of individuals. Cognitive insults may take months to fully resolve. Treatment details are discussed below.

Symptoms

Hypomania
Mania
Depression
Psychosis
Confusion
Anxiety
Memory impairment
Difficulty with attention

Life-Threatening Symptoms/Signs

Neuropsychiatric complications of steroid use are not themselves life threatening. The alterations in mood, behavior, and cognition do likely put the patient at increased risk for self-harm, harming others, and putting themselves and others in unsafe situations. As such, emergent diagnosis and treatment is imperative.

Differential

The differential diagnosis of altered mental status or behavioral changes is large. The lack of specific vital sign abnormalities, characteristic lab abnormalities, or physical exam findings with neuropsychiatric steroid complications limits the ability to develop a succinct list of likely alternative diagnoses. Essentially any process that can result in alterations of mental status or affect behavior should be entertained as on the differential and assessed in the clinical context. As such, a discrete list of differential diagnoses is beyond the scope of this chapter. A list of broad categories with individual representative examples is included, however, to aid in developing a reasonable differential given each patient's unique clinical context.

Vascular: vascular dementia
Infectious: encephalitits
Trauma: subdural hematoma
Autoimmune: Anti-NMDA encephalitis
Metabolic: hypo/hypernatremia
Neoplastic: cerebral metastases
Neurologic: normal pressure hydrocephalus
Medication (including alcohol and illicit drugs): intoxication or withdrawal

Testing

- Diagnosis is clinical and based on history of steroid use and exclusion of other causes of psychosis or neuropsychiatric symptoms
- Basic laboratory testing should include a BMP, CBC, and UA
- Further testing such as transaminases, ammonia, TSH, CT head, LP, CXR, Vitamin B12 and Thiamine levels, HIV, RPR, VDRL, and autoimmune markers should be guided by the clinical context

Treatment

- Discontinue steroids if possible. Taper if unable to stop secondary to active disease process
- Antipsychotics including olanzapine, risperidone, and haloperidol have been and can be used as adjunctive treatment
- Consider admission to rule out other causes of your patient's presentation as well as for an unsafe social situation

Tool

Incidence of Steroid Psychosis Versus Dose

Dose (in mg of prednisone daily)	Incidence
< 40 mg	1.3%
41-80 mg	4.6%
> 80 mg	18.4%

Adapted From: Boston Collaborative Drug Surveillance Program. Acute adverse reactions to prednisone in relation to dosage. Clin Pharmacol Ther 1972;13:694–8.

References

1. Dubovsky AN, Arvikar S, Stern TA, Axelrod L. The neuropsychiatric complications of glucocorticoid use: steroid psychosis revisited. Psychosomatics. 2012;53:103–15.
2. Fardet L, Petersen I, Nazareth I. Suicidal behavior and severe neuropsychiatric disorders following glucocorticoid therapy in primary care. Am J Psychiatry. 2012;169:491–7.
3. Judd LL, Schettler PJ, Brown ES, Wolkowitz OM, Sternberg EM, Bender BG, Bulloch K, Cidlowski JA, de Kloet ER, Fardet L, Joels M, Leung DYM, McEwen BS, Roozendaal B, Van Rossum EFC, Ahn J, Brown DW, Plitt A, Singh G. Adverse consequences of glucocorticoid medication: psychological, cognitive, and behavioral effects. Am J Psychiatry. 2014;171:1045–51.
4. Ross DA, Cetas JS. Steroid psychosis: a review for neurosurgeons. J Neuro-Oncol. 2012;109:439–47.
5. Sirois F. Steroid psychosis: a review. Gen Hosp Psychiatry. 2003;25:27–33.

Chapter 53
Valproic Acid Toxicity

Bryan Corbett

Introduction

Valproic acid (VPA) is used to treat seizure and bipolar disorders as well as for migraine prophylaxis. Acute overdoses can present with CNS depression and in severe cases cerebral edema and herniation can occur. Recovery can be prolonged as overdose peak plasma levels can be delayed up to 7 h. In addition, some VPA preparations are specifically designed for delayed absorption. In addition, in supratherapeutic dosing, enzymatic metabolism becomes saturated and the rate of clearance reaches a plateau. Even with therapeutic dosing, VPA may cause problems. Via multiple mechanisms, VPA inhibits the urea cycle and can lead to hyperammonemia which can also cause CNS depression and confusion. This may or may not occur in conjunction with a transaminitis. As a corollary, transaminitis may occur without elevated ammonia levels but this should prompt treatment nonetheless (discussed below) as further hepatic dysfunction and hyperammonemia could ensue.

B. Corbett, M.D.
Division of Medical Toxicology, Department of Emergency Medicine, University of California Health Sciences, San Diego, CA, USA
e-mail: bcorbett1982@gmail.com

K.D. Nordstrom, M.P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3_53,

Symptoms

Confusion
Disorientation
Somnolence
Lethargy

Signs

Depressed Mental Status
Ataxia
Seizures
Focal Neurologic Findings
Asterixis (in cases of hyperammonemia)
Hyperreflexia (in cases of hyperammonemia)
Metabolic acidosis
Leukopenia (severe toxicity)
Thrombocytopenia (severe toxicity)

Life-Threatening Symptoms/Signs

Cerebral edema can lead to herniation and death. While CNS depression can occur in the absence of cerebral edema, any patient with significant CNS depression warrants a CT head to assess for cerebral edema.

Differential

CNS findings in VPA toxicity are non-specific. VPA levels are available in real time to aid in diagnosis. Patients presenting with findings consistent with VPA toxicity who have access to VPA should have levels checked. Again, it should be noted that CNS findings can occur with normal VPA levels in the setting of hyperammonemia. Like in hepatic encephalopathy, however, clinical findings and ammonia levels do not always correlate. As such significant elevations in ammonia can

occur in the absence of clinical findings. Likewise, significant clinical findings can occur with modest ammonia elevations. The other myriad causes of alterations in mental status should be worked up within the clinical context. A list of broad categories with individual representative examples is included to aid in developing a reasonable differential diagnosis.

Vascular: vascular dementia
Infectious: encephalitits
Trauma: subdural hematoma
Autoimmune: Anti-NMDA encephalitis
Metabolic: hypo/hypernatremia
Neoplastic: cerebral metastases
Neurologic: normal pressure hydrocephalus
Medication (including alcohol and illicit drugs): intoxication or withdrawal

Testing

- VPA serum levels with repeat levels every 4–6 h when elevated
- CMP and ammonia levels are also helpful
- An APAP level should be checked in all intentional ingestions
- CTH in cases of CNS depression in known VPA ingestion
- CBC, TSH, Vitamin B12 level, HIV, LP, UA, CXR, ASA level should be considered within the clinical context

Treatment

- Treatment is generally supportive
- Decontamination
  - Activated Charcoal (AC)

    VPA may have delayed and prolonged absorption in overdose so AC can be administered greater than 1 h since ingestion

Ideal dosing is a 10:1 ratio of AC/VPA, in practice doses greater than 50 g are hard to tolerate

Do not give AC when there is concern for aspiration (i.e. depressed mental status or seizures)

  - Whole bowel irrigation with polyethylene glycol (PEG) can be considered when large doses or sustained release preparations are ingested

    Give 1–2 L of PEG an hour until clear rectal effluent (this can be hard for the patient to tolerate)
- Carnitine
  - Elevated ammonia
  - Elevated transaminases
  - In symptomatic individuals administer a loading dose of 100 mg/kg IV followed by maintenance dosing of 15 mg/kg IV every 4 h
- Hemodialysis
  - Serum levels >900 mg/L
  - Refractory shock
  - Cerebral edema
  - Coma
  - Persistent metabolic acidosis

Tool

Valproic Acid Serum Concentrations and Toxicity

Valproic Acid Concentrations in mg/L	Toxicity
50-100	Therapeutic Range
> 100-< 450	Limited Toxicity
> 450-< 850	Increased Risk of Moderate to Major Toxicity
> 850	Severe Toxicity, Coma, Cerebral Edema, Respiratory Depression, Hemodynamic Instability, Metabolic Acidosis

Adapted from: Spiller HA, Krenzelok EP, Klein-Schwartz W, Winter ML, Weber JA, Sollee DR, et al. Multicenter case series of valproic acid ingestion: serum concentrations and toxicity. J Toxicol Clin Toxicol. 2000;16:330–2.

References

1. Doyon S. Antiepileptics (Chapter 48). In: Hoffman RS, Howland MA, Lewin NA, Nelson LS, Goldfrank LR, editors. Goldfrank's toxicologic emergencies. 10th ed. New York: McGraw-Hill; 2015. p. 651–3.
2. Ghannoum M, Laliberte M, Nolin TD, Mactier R, Lavergne V, Hoffman RS, Gosselin S. Extracorporeal treatment for valproic acid poisoning: systematic review and recommendations from the EXTRIP workgroup. Clin Toxicol. 2015;53:454–65.
3. Lewis C, Deshpande A, Tesar GE, Dale R. Valproate-induced hyperammonemic encephalopathy: a brief review. Curr Med Res Opin. 2012;28:1039–42.
4. Perrott J, Murphy NG, Zed PJ. L-carnitine for acute valproic acid overdose: a systematic review of published cases. Ann Pharmacother. 2010;44:1287–93.
5. Spiller HA, Krenzelok EP, Klein-Schwartz W, Winter ML, Weber JA, Sollee DR, et al. Multicenter case series of valproic acid ingestion: serum concentrations and toxicity. J Toxicol Clin Toxicol. 2000;16:330–2.

Index

K. D. Nordstrom, M. P. Wilson (eds.),
Quick Guide to Psychiatric Emergencies,
https://doi.org/10.1007/978-3-319-58260-3