15 The Patient with Delirium and Dementia in the Emergency Department

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Introduction

You are working in the emergency department one night when a young male brings in his agitated brother with a history of schizophrenia, stating, "His schizophrenia is acting up again." You watch as the patient is brought back to a room, intermittently thrashing about the stretcher. You are able to calm him long enough to obtain vital signs and check his glucose, which is shockingly low. The nurse swiftly obtains IV access and gives him dextrose-containing fluids. To the brother's astonishment, the patient's status immediately reverts back to normal. You realize this change in mental status was secondary to delirium caused by hypoglycemia. The patient reports he forgot to eat after taking insulin, causing this predicament. As further diagnostic studies are underway, you learn of an impending case of Alzheimer's dementia found wandering outside the nursing home …

According to DSM-5, dementia and delirium are considered neurocognitive disorders, which represent cognitive decline from a previously attained level of functioning [1]. Since symptoms of delirium, dementia, and primary psychiatric disorders often overlap, this chapter will focus on how to differentiate those with medical illnesses so appropriate treatment is provided.

Delirium

The DSM-V criteria for the diagnosis of delirium consist of a disturbance in attention (ability to focus and sustain attention) and awareness (reduced orientation to the environment) that develops over a short period of time. Delirium tends to fluctuate in severity throughout the day and is associated with an additional cognitive deficit (memory, disorientation, language, or perception) attributable to a medical condition, substance intoxication, or withdrawal. Delirium may be considered hyper- or hypoactive, and the patient may switch between the two states [1].

Patients with hyperactive delirium may present as hypervigilant, restless or agitated, paranoid, and can report perceptual disturbances. The hypoactive form will present as increased lethargy, somnolence, psychomotor retardation, and might often be overlooked by physicians or be mistaken for depression [2–3]. Approximately 10% of older patients in the emergency department present with delirium, which was only

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acknowledged by healthcare providers in 25% of cases [4]. Of those patients admitted to the hospital with known dementia, 46% had superimposed delirium, wherein the recognition of such cause is unknown [5].

Causes of Delirium

The three most prevalent causes of delirium are toxic ingestions or withdrawal, infection, and fluid/electrolyte imbalance, but these are often overlooked in the acute setting [6]. A study in Mississippi found 64 cases of patients admitted to psychiatric facilities who were found to actually have unrecognized medical emergencies. In this case series, the diagnoses most often missed were severe intoxication, drug/alcohol withdrawal/delirium tremens, and prescription drug overdose [6]. An expanded but not all-inclusive list of delirium etiologies can be seen in Table 15.1.

Table 15.1 Causes of delirium

Category	Cause
Emergency	Shock
	Hypoxia
	Hypoglycemia
	Electrolyte/acid–base disturbance
	Thyroid storm
	Hyperthermia/hypothermia
	Delirium tremens
	Toxic alcohol ingestion
	Wernicke's encephalopathy
	Intracranial disturbance (trauma, abscess, etc.)
Medications/drugs	Illicit drugs/alcohol
	Drug and alcohol withdrawal
	Anticholinergic medications/drugs
	Medications in overdose
Neurologic	Seizure/post-ictal state
	Hypertensive encephalopathy
Endocrine	Hyperthyroidism/hypothyroidism
	Diabetes mellitus and DKA/HHS
Metabolic	Hepatic encephalopathy
	Renal failure
Infectious	Encephalitis/meningitis
	Neurosyphilis
	Urinary tract infection
	Pneumonia
	Sepsis

Intoxications

A significant number of the undiagnosed emergencies in the aforementioned study were secondary to drug and alcohol intoxication. In ethanol intoxication (such as beer, wine, or liquor), patients may present with euphoria, emotional lability, and disinhibition. They may appear flushed and diaphoretic, with slurred speech and incoordination. Patients who have consumed toxic alcohol (such as methanol, ethylene glycol, or isopropyl alcohol) may also present with the same symptoms as in ethanol ingestion, bolstering the importance of adequate history. Opiate intoxication presents with pinpoint pupils, nausea, vomiting, decreased respiratory drive, decreased blood pressure associated with lethargy, and, at times, agitation. Benzodiazepine intoxication often results in drowsiness with slurred speech. However, some cases of paradoxical excitation might exhibit hallucinations, hostility, psychosis, delirium, and seizures. Users of synthetic cannabinoids (such as "K2" or "Spice") may present with nausea, burning sensation in their eyes, dilated pupils, hot flushes, diaphoresis, and agitation. Stimulant use like cocaine and methamphetamines can lead to a sympathomimetic response, which is characterized by increased heart rate, blood pressure, and temperature; dilated pupils; psychosis; agitation; and extreme, possibly violent, muscular activity. Similarly, phencyclidine (PCP) intoxication may present with euphoria, auditory hallucinations, and visual distortions, nystagmus, disconnection from reality, and aggression, which may be severe, with "superhuman strength" [7].

Withdrawal

In a similar context, those in withdrawal may also suffer behavioral disturbances. Patients with opiate withdrawal may have dilated pupils, nausea, vomiting, diarrhea, abdominal pain, runny nose, excessive tearing, and insomnia. Opiate withdrawal is unpleasant but not life-threatening. Benzodiazepine withdrawal, however, may be life-threatening. Benzodiazepine withdrawal is characterized by agitation, hallucinations, confu-

sion, tremors, restlessness, and seizures. Likewise, alcohol withdrawal can also be life-threatening. Alcohol withdrawal is characterized by four stages, all of which do not necessarily occur in a single individual. The first stage consists of tremulousness, which begins 6–24 hours after cessation of alcohol and is characterized by high blood pressure, high heart rate, tremor, and a normal mental status. The second stage, or alcoholic hallucinosis, consists of visual hallucinations and formication (tactile hallucinations) with preserved sensorium. The third stage, which typically peaks at 24–48 hours, can present with seizures. The fourth and potentially lethal stage is delirium tremens [7], which occurs after between 24 and 96 hours of abstinence and consists of elevated heart rate, elevated blood pressure, hyperthermia, confusion, hallucinations, agitation, and disorientation. The mortality for delirium tremens may be up to 20%. Ethanol level can still be elevated (greater than 0) during withdrawal [8].

Overdose

Prescription medication overdose can also present as behavioral disturbances. MAOI (monoamine oxidase inhibitor)-related hypertensive crisis can occur as a result of a "cheese reaction" due to consumption of tyramine-rich food. This reaction is characterized by sympathomimetic symptoms, which begin within 30–90 minutes from ingestion. A typical presentation includes headache, elevated blood pressure, elevated heart rate, diaphoresis, and agitation, which may lead to seizures and coma. Carbamazepine in overdose can lead to dizziness, heart conductance disturbances, restlessness, confusion, aggression, drowsiness, and, eventually, coma. Lithium toxicity can lead to potentially lethal outcomes. Acute manifestation includes nausea, vomiting, tremor, agitation, and weakness. Progression of acute toxicity can manifest as confusion, slurred speech, decreased blood pressure, neuromuscular involvement leading to ataxia, renal failure, convulsions, and coma. Anticholinergic toxicity is associated with a wide variety of medications, ranging from tricyclic antidepressants to diphenhydramine use. A mnemonic used to remember the symptoms of anticholinergic toxicity is "Hot as a hare, dry as a bone, blind as a bat, and mad as a hatter," which note the cardinal symptoms of fever, dry skin, dry mucous membranes, dilated pupils, and agitation with altered mental status [7]. Medication overdose and toxicity occur in patients of all ages, and prudent care must be taken to appropriately care for these patients.

Other Important Considerations

Hypoxia or anoxia, temperature fluctuations, electrolyte imbalances such as glycemic shifts, abnormal acid–base status, and hypo or hypernatremia may also contribute to altered mental status and should be investigated further [7].

Infection is another common cause of delirium, particularly in the elderly. Young patients with delirium secondary to infection are more likely to be suffering from meningitis or encephalitis, while elderly patients are more likely to have pneumonia or a urinary tract infection.

Identification and Workup of Delirium

In patients with altered mental status or acute behavioral disturbances, a thorough history and physical examination is the first step in achieving an accurate diagnosis. This examination should place emphasis on identifying underlying medical conditions, like urinary tract infections or recent falls, toxidromes, or medication interactions. There are several key characteristics to further assist in differentiating between a primary psychiatric illness and delirium. Primary psychiatric illness does not cause decreased level of consciousness, abnormal vital signs, or focal neurologic deficits. In other words, unless there is an underlying medical condition or intoxication, the patient should be alert and neurologically intact. And while psychiatric disorders may also include perceptual disturbance like auditory and visual hallucinations, visual or tactile should

raise suspicion for a medical etiology [6]. Additionally, new onset of psychosis in older adults should also prompt further workup [7]. Tests to consider in a delirious patient include, but are not limited to, complete blood count, glucose, basic metabolic panel, liver function tests with ammonia level, urinalysis, chest X-ray, lumbar puncture, electrocardiogram, and/or a head CT [7].

Assessment of Delirium

Several instruments for diagnosing and assessing the severity of delirium have been created. The Confusion Assessment Method (CAM) and the Delirium Rating Scale-Revised Edition (DRS-R-98) are both based on DSM criteria and are considered reliable and valid. The CAM is considered to be relatively easy to use and understand. DRS-R-98 is relatively comprehensive and sensitive to change, and so may be useful for monitoring patients over time [9].

Management of Delirium

The management of delirium includes identifying and managing the underlying cause. Measures must be taken to monitor and ensure safety of an agitated patient and staff. Environmental interventions like minimizing noise, fall prevention, proper illumination, limiting use of restraints, cueing, and redirection are an integral part of treatment [10]. Pharmacological intervention may be required in cases of severe agitation to ensure safety of patient and staff. Both typical (haloperidol) and atypical antipsychotics (olanzapine, risperidone, quetiapine, and aripiprazole) have been shown to be equally effective in the treatment of delirium. Atypical antipsychotic use is often preferred, due to lower risk of extra-pyramidal side effects and better tolerability. These are typically offered in oral formulations, with olanzapine being the only parenteral option available in intramuscular form. Finally, acutely delirious patients should be admitted for stabilization until the delirium resolves [11–12].

Dementia

The DSM-5 defines dementia, now known as major neurocognitive disorder, as cognitive decline with impairment in cognitive performance that may affect independence [1]. Several subtypes of dementia can lead to alterations in a patient's personality and emotional control causing irritability and disinhibition.

As compared to delirium, dementia is insidious in onset, with a clear sensorium and no alterations in attention. Each type varies in age of onset, progression, and features, and can manifest behavioral disturbances [13–14]. Table 15.2 below summarizes several subtypes of dementia with their age of onset, progression, and features.

Several studies have shown that patients with previously diagnosed psychiatric disorders are at increased risk of developing dementia. Delirium also accelerates cognitive decline in dementia [15–16]. In 2015, investigators in Denmark revealed that patients with schizophrenia had an almost two-fold increased risk of dementia, particularly in patients less than 65 years old [17]. Another Denmark study showed that the risk of dementia increases with the number of episodes of depression and bipolar disorder that lead to admission. The study calculated that the rate of dementia increased 13% with every depressive episode requiring admission, and 6% with every bipolar episode leading to admission [18]. Furthermore, in patients diagnosed with depressive "pseudo-dementia," when cognition and function could be entirely restored to normal if depression was adequately treated, approximately 40% ultimately developed dementia. With the above in mind, it is prudent to monitor the cognition of patients with psychiatric disorders, as dysfunction may not be minor or temporary [19].

Table 15.2 Types of dementia

Type of dementia	Age of onset	Onset and progression	Cognitive features	Behavioral features
Alzheimer's	Early: 40s–50s; late: 70s–80s	Insidious and gradual	Decline in memory	Depression, apathy, irritability, agitation, combativeness, wandering
Frontotemporal	20s–80s; 50s most often	Insidious and gradual	Decline in executive function; spares learning/memory and motor function	Disinhibition, apathy, loss of inertia, compulsive, hyperorality
Lewy body	50s–80s	Insidious and gradual	Fluctuating decline in executive function	Visual hallucinations, delusions, Parkinson's, and REM sleep behavior disorder features
Vascular	Variable	Variable	Decline in executive function	Variable
Traumatic brain injury	Variable	Presents immediately	Variable	Loss of emotional control, personality changes, suspicion, irritability, aggression
HIV	Variable	Variable with fluctuant course	Variable	Loss of emotional control, irritability
Prion	Variable	Insidious and rapid	Variable	Problems with appetite, anxiety and sleeping
Parkinson's	50s–80s	Insidious and gradual	Variable	Apathy, depression, anxiety, hallucinations, delusions, personality changes, REM disorder
Huntington's	30s–40s	Insidious and gradual	Variable	Depression, apathy, irritability, OCD symptoms, disinhibition, impulsivity, impaired insight

Workup of Dementia

An accurate and detailed history is essential to diagnosis of dementia. Onset of symptoms, progression, and level of functional impairment are important considerations during assessments. Evaluation to identify potentially reversible causes like vitamin deficiency can proceed simultaneously [20].

Evaluating cognitive dysfunction requires involvement of family or other independent observers (not just the patient). Cognitive abilities should be screened and documented using the Folstein Mini-Mental State Examination (MMSE). A maximum of 30 points can be awarded based on responses that evaluate orientation, registration, attention, calculation, and visuospatial domains [21]. Typically, a score of 24 or less is considered suggestive of dementia, with a sensitivity and specificity of 87% and 82%, respectively. Limitations of the MMSE include few pure recall items and a relatively low sensitivity to early or mild cognitive impairment (MCI), particularly in highly educated individuals. It is also not sensitive to frontal lobe dysfunction. The Mini-Cog is a time-efficient and clinically efficient bedside measure with similar sensitivity and specificity for dementia. The test includes three-word recall and clock drawing test [22].

The Montreal Cognitive Assessment Battery (MoCA) was developed for the assessment of MCI and includes expanded assessments of visuospatial and executive function. It has excellent sensitivity for MCI (90%) compared to a clinical evaluation in a memory clinic [23]. It is now gaining popularity, as it is freely available and can be administered easily at the bedside. It is available in multiple languages, including both

a version for the visually impaired and a telephone version.

A complete physical and neurological examination is necessary to identify comorbid medical illnesses that maybe affecting cognition. Apraxia, focal deficits, gait abnormality, and pyramidal or extrapyramidal motor deficits can suggest neurological etiology. Laboratory testing should be considered to identify potentially reversible conditions that may mimic dementia. Common tests include complete blood counts (CBC), chemistry panels, erythrocyte sedimentation rate, thyroid function tests (thyroid-stimulating hormone, or TSH, and free thyroxine, or FT4), a vitamin B12 level, a thiamine level, and a syphilis screening [20].

Brain imaging (magnetic resonance imaging (MRI) or computed tomography (CT)) is recommended for all patients to identify structural, demyelinating, inflammatory, or vascular etiologies. There is no consensus guideline recommending one imaging modality over the other [24].

Management of Dementia

The best way to manage behavioral disturbances in dementia depends on the severity of the symptoms. Reversible causes like delirium, pain, comorbid medical illness, medications, and environmental factors must be addressed. In patients with mild–moderate disturbances, non-pharmacological approaches like cognitive training, exercise, family training, or environmental training should be the primary intervention [25].

Cholinergic deficiency contributes to the neuropsychiatric symptoms of Alzheimer's disease, and cholinomimetic therapies such as cholinesterase inhibitors (donepezil, rivastigmine, galantamine) are being utilized for treatment as well [26]. Low-dose atypical antipsychotics can be considered for management of agitation and behavioral disturbances, though literature supporting its use is limited. Antidepressants and anxiolytics can help with depression and anxiety. Lastly, in those with mania-like symptoms and aggression, mood stabilizers and/or atypical antipsychotics should be used. Prior to initiating medications, environmental factors should be adjusted whenever possible. Loneliness may be treated by interaction with those with positive relationships with the patient, videotapes of family members, and contact with animals. Boredom is best alleviated with both structured and unstructured stimulation, including but not limited to music and items with which to play, such as aprons with buttons. Patients with a need to wander should be encouraged to walk in sheltered gardens. Those who need to hoard should be provided bags and safe areas where they may "shop" for their treasures, after which they may be restored to their rightful owners [27].

As the population continues to age, there are increasing numbers of elderly patients with psychiatric disorders and dementia, leading to the formation of a geriatric psychiatry unit. These have become a key part of treatment, as patients with dementia are particularly vulnerable in acute hospitals to environmental change and communication difficulties. Some of the key characteristics of joint geriatric/psychiatric wards are joint medical care by geriatricians and psychiatrists, securing the home environment to facilitate rehabilitation and maintain independence, patient-centered care, dedicated multidisciplinary team and continuity of care, good community links to facilitate safe discharge, and specialized training of staff to manage behavioral problems without recourse to physical or pharmacological restraint. These wards have many potential advantages in hospital care of the frail and elderly, and are being evaluated for clinical and cost-effectiveness worldwide [28].

Patients presenting with behavioral disturbances may have these changes secondary to delirium and dementia. As discussed above, delirium is acute in onset and is characterized by a disturbance in consciousness with a change in cognition, while dementia is more gradual in onset and is characterized by intellectual impairment that may interfere with functioning. Care must be taken to ensure patients with neurocognitive disorders are accurately diagnosed and cared for to ensure the best outcomes.

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