Chapter 94 – Delirium and Dementia

Episode Overview

Core questions:
1. List the four key diagnostic criteria for delirium.
2. List six strong predisposing or precipitating factors for delirium.
3. List 15 causes of delirium.
4. Describe how to use a screening tool for delirium (MMSE vs. CAM).
5. List 3 potential medications used for chemical restraint.
7. Compare delirium with dementia.
8. List important diagnostic studies for the workup of delirium.
9. List four diagnostic criteria for dementia.
10. List 10 specific causes of reversible dementia.
11. List 10 causes of non-reversible dementia.

Wisecracks:
1. Explain how to differentiate between psychosis, delirium and dementia.
2. How does Aricept work?
3. Describe the pathophysiology of Alzheimer's dz and list RFs for its development.
4. What is the triad of normal pressure hydrocephalus?

Rosens in Perspective:

“Delirium is characterized by a fluctuating neurobehavioral disturbance typically progressing over a short period. It is a direct consequence of an acute systemic or central nervous system (CNS) stressor. Dementia, on the other hand, tends to follow a more gradual course, with evolution occurring over months to years. Although patients with dementia exhibit confusion, unlike delirium, manifestations of autonomic nervous system abnormalities are minimal or absent and a disturbance in level of consciousness usually is not a feature.”

These big topics fall under the term neurobehavioural disturbances. Here is the big three step approach for us in the ER:
1. Take a full collateral hx and do a physical exam to determine if this is delirium or dementia
2. Rapidly treat any underlying cause of delirium
3. “Establish a supportive environment and employ pharmaceutical adjuncts as needed”

Core questions:
[1] List the four key diagnostic criteria for delirium

BOX 94.1
Diagnostic Criteria for Delirium
FOUR KEY CHARACTERISTICS
• The disturbance develops over a short time period, represents a change from baseline attention and awareness, and tends to fluctuate in severity during the day.
• Disturbance in attention and awareness.
• There are additional disturbances in cognition, such as memory, disorientation language, visual spatial ability, or perception.
• The disturbances are not better explained by another preexisting, established, or evolving neurocognitive disorder and do not occur in context of a coma.

1. Acute onset with fluctuating course
2. Disturbs attention and awareness (inattention)
3. Disturbed Perception
4. NOT better explained by another neurocognitive disorder

Important to note these things on history (collateral hx is crucial):
- Inattention
- Short term memory impairment
- Sleep-wake cycle changes
- Inquiry about disturbed perceptions - hallucinations or delusions

On physical exam:
- Autonomic system dysfunction:
  - Elevated or decreased:
    - Pulse, RR, temp, BP.
- Wernicke’s K.O.A.
  - Remember that magnesium is a cofactor in thiamine utilization!! Replace BOTH.

[2] List six strong predisposing or precipitating factors for delirium

See table 94.1 for the whole list.
Here are the ones with a STRONG level of evidence:
- Predisposing variables:
  - Age
  - Dementia
  - Hypertension
- Precipitating variables
  - Coma
  - Previous delirium
  - Emergency surgery
  - Mechanical ventilation
  - Trauma
[3] List 15 causes of delirium

Delirium is caused by a deregulation of neurotransmitter synthesis and metabolism.

DIMES:
- Drugs
  - ETOH - intox or withdrawal
  - Other street / recreational drugs
  - Sedative / hypnotics
  - Digoxin / lithium / quinidine / salicylate / antiepileptics
  - Over the counter (anticholinergics), TCA,
  - MANY others
- Infection
  - Meningitis, encephalitis, sinusitis, otitis media
  - Pneumonia
  - Peritonitis, pyelonephritis, cholecystitis, etc.
  - Cellulitis
- Metabolic
  - hypo/hypernatremia
  - Hyperosmolarity
  - Hypercapnia
  - Hypoxia
  - Hyperglycemia
  - Uremia
  - Liver failure
- Environmental
  - Hypothermia (usually < 35 deg. C)
  - Hyperthermia (usually > 42 deg. C)
- Structural
  - CVA, TBI, SAH, status epilepticus


Delirium can be present in a patient who is oriented to person, place and time; therefore we need a more comprehensive assessment tool!

“A review of 11 bedside instruments used to identify the presence of delirium in adults concluded that the best evidence supported the use of the CAM as the best, and the Mini Mental State Exam as the least accurate test...In medical and surgical settings, the CAM has a sensitivity of 94 to 100 percent and a specificity of 90 to 95 percent ” Uptodate
Confusion Assessment Method (CAM)

4 Key Features:

1. Acute onset and fluctuating course
2. Inattention
3. Disorganized thinking
4. Altered Level of consciousness

The diagnosis of delirium requires the presence of features 1 AND 2 plus either 3 OR 4.

Check out these other FREE delirium instruments/tools:
https://www.hospitalelderlifeprogram.org/delirium-instruments

[5] List 3 potential medications used for chemical restraint

“The ideal sedating drug should have: low toxicity with minimal anticholinergic effects, ease of administration, short half-life, minimal effects on the cardiovascular and respiratory systems, and no effect on the seizure threshold” -- Rosen’s 9th Ed.

Top three choices:

- **Haldol** (haloperidol) - 1st Gen. antipsychotic
  - Tried, trusted, true. We have extensive clinical experience with it.
  - Wide dose range based on age, and comorbidities
    - 0.5 - 10 mg IM**
- **2nd generation antipsychotic** -
  - “(risperidone, olanzapine, ziprasidone, aripiprazole) may have equal or better efficacy and fewer side effects (especially akathisia and dystonia) for management of acute agitation in the psychiatric population.”
- **Benzodiazepines:**
  - Great choice, either solo or with an antipsychotic
  - Great for acute undifferentiated agitation, intoxication, or withdrawal.
  - **go to is LORAZEPAM**
    - Because diazepam has a very long half life.

“We recommend either a benzodiazepine or antipsychotic (typical or atypical) used as monotherapy. As an alternative, a combination of a low-dose antipsychotic plus benzodiazepine (eg, haloperidol 5 mg IM plus lorazepam 2 mg) can be used. The combination approach has been found to be superior to either class alone in the treatment of undifferentiated acute agitation and has the added benefit of minimizing adverse effects.” - Rosen’s 9th Ed.
List 2 potential side effects of Haldol administration

1. EPS:
   “Studies of the acute administration of haloperidol report an 8% to 30% incidence of extrapyramidal side effects with akathisias being most common and acute dystonia occurring in less than 10% of Patients.”
2. Prolonging the QTc:
   “more so when given intravenously, but this effect is clinically insignificant in most patients and does not require a pretreatment electrocardiogram. Caution is warranted with use of this agent in patients taking medications that prolong the QTc (eg, class IA and class III antiarrhythmics, certain antibiotics, inhibitors of the cytochrome P450 system) and in patients with acute coronary ischemia, uncompensated congestive heart failure, or hepatic dysfunction. The QTc effect is not usually concerning when the haloperidol is given intramuscularly.”

Compare delirium with dementia

Delirium is a medical emergency, while dementia isn’t. “Dementia is not a single disease entity but rather a highly variable clinical syndrome characterized by a gradually progressive deterioration of cognitive function.”

A word on delirium prognosis: “After an episode of acute delirium, younger patients may experience mild cognitive dysfunction that lasts weeks to months. Elders, on the other hand, often experience persistent decline in their baseline level of functioning, with loss of at least one activity of daily living after acute delirium.”

<table>
<thead>
<tr>
<th></th>
<th>DELIRIUM</th>
<th>DEMENTIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>onset</td>
<td>Acute</td>
<td>slow</td>
</tr>
<tr>
<td>awareness</td>
<td>Reduced</td>
<td>Clear</td>
</tr>
<tr>
<td>alertness</td>
<td>Fluctuates</td>
<td>Normal</td>
</tr>
<tr>
<td>Orientation</td>
<td>impaired</td>
<td>impaired</td>
</tr>
<tr>
<td>memory</td>
<td>impaired</td>
<td>impaired</td>
</tr>
<tr>
<td>Perception</td>
<td>Hallucinations</td>
<td>Intact</td>
</tr>
<tr>
<td>Thinking</td>
<td>Disorganized</td>
<td>Vague</td>
</tr>
<tr>
<td>Language</td>
<td>Slow</td>
<td>Word finding difficulty</td>
</tr>
</tbody>
</table>

What are those four diagnostic criteria for delirium?
- Acute onset with fluctuating course
- Inattention
- Altered perceptions / disorganized thinking - cognitive, memory,
- NOT explained by a preexisting neurocognitive disorder
[8] List important diagnostic studies for the workup of delirium

These are guided by your comprehensive hx and physical exam:

- Vital signs
- Point of care glucose
- CBC
- Electrolytes, BUN, Crt, extended lytes, osmolality
- Urinalysis
- CXR
- CT head
- Maybe
  - ABG (carbon monoxide, cyanide, hypercarbia, acidosis)
  - Drug levels
  - Troponin, Liver enzymes, TSH
  - B12, folate, heavy metals, toxin screens of blood/urine/sweat, toxic alcohols,
  - MRI brain
  - LP - CSF analysis
  - EEG (nonconvulsive status epilepticus)

Up to 16% of patients have no underlying cause for delirium found despite intensive investigations

[9] List four diagnostic criteria for dementia

BOX 94.3
Diagnostic Criteria for Dementia

A. Cognitive decline from a previous level of performance in one or more cognitive domains: Complex attention, executive function, learning and memory, language, perceptual motor function, or social cognition.
B. The disorder has an insidious onset and gradual progression.
C. The deficits do not occur exclusively during the course of a delirium.
D. The cognitive deficits are not better explained by another mental disorder, such as major depression or schizophrenia.

Said another way:
- **IS:**
  - Insidious onset, with gradual progression
  - Cognitive decline - function, learning, memory, social cognition
- **IS NOT:**
  - Occurring during delirium
  - No explained by depression or schizophrenia (or another neurocog. disorder)
BOX 94.2 Causes of Dementia

PRIMARY DEGENERATIVE DEMENTIAS
  ● Alzheimer’s disease
  ● Lewy bodies disease
  ● Frontal lobe disease (Pick’s disease)
SUBCORTICAL DEMENTIAS
  ● Parkinson’s disease
  ● Huntington’s disease
VASCULAR DEMENTIA
  ● Multi-infarct dementia
INTRACRANIAL PROCESSES
  ● Space occupying lesions (tumor, subdural hematoma)
  ● Hydrocephalus
  ● CNS infections (HIV-1, neurosyphilis, chronic meningitis)
  ● Repetitive head trauma
ENDOCRINOPATHIES
  ● Addison’s and Cushing’s diseases
  ● Thyroid and parathyroid disease
NUTRITIONAL DEFICIENCIES
  ● Thiamine
  ● Niacin
  ● Folate
  ● Vitamin B12
TOXIC EXPOSURES
  ● Heavy metals
  ● Carbon monoxide
  ● Carbon disulfide
DRUGS
  ● Psychotropics
  ● Antihypertensives
  ● Anticonvulsants
  ● Anticholinergics
DEPRESSION
  ● Pseudo-dementia


“Dementia is classified as either irreversible (primary degenerative) or potentially reversible (secondary); it is further classified according to the degree of cognitive impairment.
  ● Mild dementia implies some impairment of work and social activities; however, the capacity for independent living remains intact.
  ● With moderate dementia, independent living is hazardous, and some degree of supervision is necessary.
  ● With severe dementia, continual supervision and often custodial care are needed.”
Reversible causes:

- **Adverse drug reactions**
  - Psychotropic drugs
  - Antihypertensive medications
  - Anticonvulsants
  - Anticholinergics
  - L-dopa
  - Heavy metal poisoning
  - Carbon monoxide / carbon disulfide
  - Trichlorethylene
  - ETOH abuse

- **Endocrinopathies / Metabolic abnormalities**
  - Hypothyroidism
  - Parathyroid disease
  - Addison's disease
  - Cushing’s disease
  - Panhypopituitarism
  - Thiamine deficiency
  - Niacin deficiency
  - Vitamin B12 def.
  - Folate def.

- **Intracranial processes**
  - Tumours
  - Hydrocephalus
  - Chronic head trauma
  - HIV
  - Tuberculous or fungal meningitis
  - Toxoplasmosis, cryptococcus, CMV, herpes virus, VZV

- **Depression**

[11] **List 10 causes of non-reversible dementia.**

These are the primary degenerative diseases:

- **PRIMARY DEGENERATIVE DEMENTIAS**
  - Alzheimer’s disease
  - Lewy bodies disease
  - Frontal lobe disease (Pick’s disease)

- **SUBCORTICAL DEMENTIAS**
  - Parkinson’s disease
  - Huntington’s disease

- **VASCULAR DEMENTIA**
  - Multi-infarct dementia

- **Attributable causes:**
  - Anoxic encephalopathy
  - Hepatolenticular degeneration
  - Tumours
  - Slow virus infections
The top three are:
1. Alzheimer’s disease > 60%
2. Subcortical dementia and vascular dementia > 20%
3. Lewy body dementia (the type with well formed hallucinations - visual - and EPS)

**Wisecracks:**
[1] Explain how you differentiate between psychosis, delirium and dementia.

“The goals of ED evaluation for suspected dementia are:
(1) to recognize the signs and symptoms of undiagnosed and potentially reversible forms of dementia,
(2) to identify the manifestations of acute illness in the demented patient promptly, and
(3) to assess the clinical findings in lieu of the patient’s cognitive impairment and facilitate a safe disposition and expedited follow-up.” - Rosen’s 9th Ed.

<table>
<thead>
<tr>
<th>Psychosis</th>
<th>Delirium</th>
<th>Dementia</th>
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<tbody>
<tr>
<td>● Hx of psychiatric illness</td>
<td>● Acute onset, fluctuating course / level of consciousness</td>
<td>● Often denial of impaired cognitive function or social avoidance</td>
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<tr>
<td>● With depression: pt emphasizes cognitive dysfunction, failures and difficulties (dysphoria)</td>
<td>● Inattention</td>
<td>● insidious, progressive, without much fluctuation, and occurs over a much longer time (months to years). Attention is relatively intact, as are remote memories in the earlier stages</td>
</tr>
<tr>
<td>● Less fluctuation</td>
<td>● Abnormal vital signs (autonomic instability), labs, and examination</td>
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<tr>
<td>● agitation, delusions, and psychotic behavior</td>
<td>● Abnormal sensorium</td>
<td></td>
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<tr>
<td>● Highly systematized delusions</td>
<td></td>
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<tr>
<td>● NORMAL sensorium</td>
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</tbody>
</table>

It is common for dementia to first present with a superimposed delirium or depression!

[2] How does Aricept (Donepezil) work?

Cholinesterase inhibitors (donepezil, rivastigmine, and galantamine) increase cholinergic transmission by inhibiting cholinesterase at the synaptic cleft and provide modest symptomatic benefit in some patients with dementia.

They are not disease modifying, and often cause a functional improvement for 2-3 yrs in cases of mild dementia. They are known to cause cholinergic side effects.

[3] Describe the pathophysiology of Alzheimer’s dz and list RFs for its development

Anatomic changes:
- Cortical atrophy - in temporal and hippocampal regions - loss of grey matter → loss of white matter
Pathologic changes
- Extracellular deposition of B-amyloid protein and neurofibrillary tangles (abnormal protein TAU which is involved in the process of neural regeneration).
- Granulovascular degeneration - in the cortical blood vessels

Neurochemical changes:
- Decrease in acetylcholine

Risk Factors
- Age. 30-50% by age 85 yrs
- Family history
- Low education level
- Hypercholesterolemia
- Head trauma

[4] What is the triad of normal pressure hydrocephalus?

Think about this in the young patient (60 yrs or less) with:
- Progressive dementia
- Ataxia
- Urinary incontinence