

Delirium

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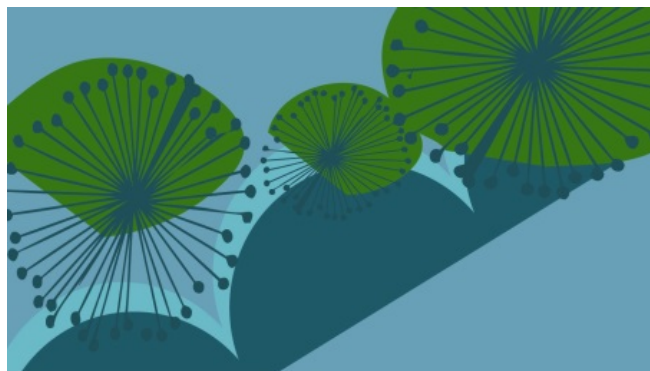
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PSYCH - LITE

PSYCHIATRY THAT'S EASY TO READ



ROB SELZER & STEVE ELLEN

Delirium

- Definitions – why is it missed so often?
- Clinical features
- Prevalence, Costs and Recognition etc.
- Causes – systemic and neurological
- Treatment

Terminology

- Terminology varies
- neurologists tend to use acute confusional syndrome (ACS) or acute brain syndrome
- definitions vary according to background of author
- seen as a manifestation of widespread brain dysfunction

Terminology

- “a change in mental state in which *the most salient deficits* occur in overall attentional tone” (neurologist)
- “acute change in cognitive state characterised by fluctuating consciousness and inattention” (physician)
- DSM-IV criteria list 3 categories: “disturbance of consciousness *and* change in cognition *of rapid onset*”

Delirium: Clinical Features

- A transient organic brain syndrome
 - acute onset and fluctuating course
 - global impairment of cognitive function
 - altered level of consciousness
 - reduced attention and concentration
 - increased or decreased psychomotor activity
 - disordered sleep/wake cycle
 - perceptual disturbances (hallucinations, illusions)

Delirium: Clinical Features

- ***Associated features:***
 - irritability
 - distractibility
 - mood disturbance - depression /anxiety
 - nocturnal exacerbation and day-time somnolence
- ***Prodromal symptoms:***
 - restlessness
 - anxiety
 - sleep disturbance

Clinical features

Some focal neurological findings can also be seen e.g.:

- Extensor plantar responses
- Coarse tremour, myoclonus or asterixis
- Asymmetric reflexes
- Asymmetric power in severe cases, especially elderly and often in hypoglycaemic states

Subtypes

- Hyperalert/hyperactive
 - agitation
 - hyper-reactivity
 - aggression
 - hallucinations
 - delusions

(Olofsson et al, 1995; Camus et al, 2000)

Subtypes

- Hypoalert/hypoactive
 - reduced reactivity
 - motor and speech retardation
 - facial inexpressiveness
- Mixed states (most common)

Camus et al 2000

Other Clinical Presentations



- “non-compliance” and denial
- anxiety and panic
- “crescendo pain”
- “adjustment problems”
- suicidal ideation and actions
- staff and family conflict

*Kissane & Smith, 1996; Saravay, 1987; Coyle et al, 1994;
Kinkel, 1997; Akechi et al, 1999; Farrell & Ganzini, 1995*

Incidence

Studies conflict:

- 10% of all hospital in-patients
- 30-50% of in-patients on geriatric wards
- 40% of post-operative patients (but very wide variation – greatest in bypass patients)
- (Dyer et al 1995 meta analysis of 26 published studies – selected from 374)

Recognition

- Recognition

- generally under-diagnosed and under-treated
- un-recognised in 20%
- common misattributions:
 - > depression
 - > anxiety
 - > non-organic psychosis

Faisinger et al, 1991, Margolis, 1994, Saravay, 1987

Risk factors

- increasing age
- pre-existing cognitive impairment
- drug abuse
- anticholinergic medications
- polypharmacy
- NOT gender, race, marital status, nor education

Pathophysiology

- poorly understood despite 2500 years of recognition (Hippocrates) and a hundred years of research
- adequate function of the CNS depends on the metabolic integrity of its constituent neurons and glia
- polysynaptic circuits appear to be especially vulnerable to toxic/metabolic insults
- believed to arise from any disruption in the “cholinergic axis”

Costs and morbidity

- Most studies suggest that mortality at least doubles (10-65%)
- Poor prognostic indicator for all surgery
- Markedly lengthens inpatient stay
- Excess annual health expenditure of \$US2 billion (*Inouye 1994*)

Causes

D-drugs

E-endocrine

L-Lungs

I-ischaemia

R-renal

I-infections

U-unknown

M-metabolic

Causes

Systemic vs. neurological

Systemic by far commonest:

- ***Toxic/metabolic***
- Hypovitaminosis
- Infection
- Endocrine
- Hypoxia
- other

Special toxic causes

- Alcohol intoxication and withdrawal
- Drug intoxication and withdrawal
- Chemical toxins and poisoning
- Carbon monoxide

Neurological causes

- Multifocal brain lesions – HT, vasculitis, DIC, air and fat emboli, SAH
- Infections – meningitis, encephalitis and brain abscess
- Non-convulsive status epilepticus
- Trauma
- Neoplastic conditions

Treatment- 4 goals

- **Recognition** (hardest part)
- **Investigation** (easiest part)
 - a) to see if this is delirium
 - b) to find the cause of the delirium
- **Symptomatic** treatment
- **Definitive** treatment

Investigations- for diagnosis of delirium

- History from nurses/notes- MSE on admission, fluctuation etc
- History from Family- usual level of cognitive function, rate of decline
- Serial MMSE
- EEG- sometimes useful

Investigations-for cause

- Careful review of drug history
- Corroboration with family members regarding medications at home, diet and pre-morbid function

Investigations- for cause

- Toxic/metabolic screen
- MSU +/- blood cultures
- CXR and abdominal films
- CT/MRI brain
- EEG – which should show diffuse slowing to be diagnostic
- LP in cases where no obvious cause apparent

Investigations

- A common mistake is to consider the symptoms to be psychiatric if no clear cause is found.
- No clear cause is found in about 20% of cases.
- Severe mental illness can lead to confusion, but this is rare, and when it is present, it is usually associated with dementia.

Symptomatic Treatment

- Aimed at specific indications (*Carter, 1995*)
 - > aggression
 - > safety
 - > hallucinations
 - > distress
 - > capacity to cooperate with care

Symptomatic Treatment

1. Medications

- very limited research base
- antipsychotic agents
 - > to treat agitation and psychotic symptoms
 - > superior to benzodiazepines
- haloperidol 1-2mg every 2-4 hours
 - > elderly: 0.25-0.50mg every four hours
 - > +/- short acting benzodiazepine (lorazepam in US studies), however I prefer diazepam (valium), as available, easy to prescribe, doses rarely mucked up!!
- Olanzapine 2-5 mg **Q/IM**

Symptomatic Treatment

1. Medications

- Precautions
 - Increased sensitivity to extrapyramidal effects of DA antagonists (eg HIV Infection)
 - Drug interactions (eg anticholinergic effects)
 - Pharmacokinetics
 - Polypharmacy
 - Sedation (eg hydration needs)

Symptomatic Treatment

2. Environment

- environmental and supportive interventions
 - optimal level of environmental stimulation
 - familiarize environment & optimize orientation
 - reduce sensory impairments
 - address fear and demoralization
 - liaison roles
 - Watch safety: attendant Vs Special nurse Vs in safer room

Definitive Treatment

- Obviously depends on the cause, however,
- Research findings support:
 - Discontinuation of unnecessary psycho-active medication
 - attention to hydration
 - change of opioid or dose modification
 - use of antipsychotic drug if needed

Prevention

- primary
 - > identify risk groups
 - > enhance environment
 - > modify causative factors (eg drug treatment practices)
- secondary
 - > early identification through cognitive monitoring
 - > early intervention - identify key early symptoms (eg sleep disturbance, agitation, irritability, somnolence)
 - > Identify and address precipitants

Prevention

- Tertiary Prevention
 - maintain and maximise function
 - > relief of distress
 - > reduce demands
 - > identify predisposing factors (eg dementia)
 - > environmental factors
 - > intervention with family and staff: goals of care
 - > address key tasks for patient
 - engaging with staff and family
 - participation in care at realistic level

Screening and Diagnosis



1. Screening Instruments

Confusion Rating Scale (*Williams et al, 1988*)

Clinical Assessment of Confusion (*Vermeersch, 1990*)

2. Diagnostic Instruments

Confusion Assessment Method (*Innouye et al, 1990*)

Delirium Symptom Interview (*Albert et al, 1992*)

3. Delirium Severity Rating Scales

Delirium Rating Scale (*Trzepacz, 1999*)

Memorial Delirium Assessment Scale

(*Breitbart et al, 1997*)

Delirium: Impact on Family

- roles and functions (esp decision-making)
- demoralization and fatigue
- functions needed and lost & demands on carers
- impact of behavioural change
- attribution for these changes
- conflict: family, staff
- children
- ?bereavement outcome

Issues for Staff

- > confrontation with regression and fragmented mental states
- > high care demands amidst poor cognitive function
- > therapeutic pessimism and hopelessness
- > blame from the family

Issues for Staff

- > overestimating decisional capacity
- > difficulty establishing realistic limits/safety for the dying patient
- > negotiating amidst complex ethical pressures and frameworks (eg autonomy and self determination)

Issues for Staff

- > re-establishing relationship with the patient post-delirium
- > impact on team/unit function
- > responding to family distress and patterns of response

Conclusion

- Delirium
 - common clinical syndrome
 - often missed
 - often inappropriately attributed to psychiatric cause
 - significant impact on patient, family and staff
 - significant benefits from comprehensive treatment