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Regular review

Delirium: optimising management

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Delirium is a complex neuropsychiatric syndrome with an acute onset and fluctuating course; it is common in all medical settings. Delirium occurs in about 15-20% of all general admissions to hospital¹; it occurs with higher frequency in elderly people and in those with pre-existing cognitive impairment.² Delirium has many synonyms, reflecting its ubiquitous nature rather than distinct conditions. These synonyms include acute brain failure, acute confusional state, and post-operative psychosis. Delirium has not been well studied owing to methodological difficulties and a lack of consensus about its definition. Thus, delirium has been underappreciated as an independent entity that requires therapeutic intervention beyond identification of the syndrome and amelioration of the underlying cause. The development of a clearer definition, improved detection and assessment tools, and recognition of the significant independent morbidity associated with delirium have substantially changed this situation. These developments coupled with a greater awareness of the needs of an increasingly large population of elderly people³ make a review of the day to day management of patients with delirium timely.

Methods

This review is based on the results of a Medline search for articles published between 1980 and 1999 using the key words "delirium," "acute confusion," "management," and "treatment"; as well as hand searching for articles in major journals in general and old age medicine and psychiatry published during the past five years; inspection of recent treatment guidelines published by the American Psychiatric Association²; and a review of references cited within these sources. Because of variability in the methodological quality of research into delirium, articles were selected for inclusion on the basis of an appraisal of the usefulness and validity of the studies.

Clinical features

The symptoms of delirium are wide ranging, and although they are non-specific, their fluctuating nature is highly characteristic and is a valuable diagnostic indicator. The core disturbance involves an acute generalised impairment of cognitive function that affects orientation, attention, memory, and planning and organisational skills. Other disturbances, such as those

Summary points

Delirium is especially common in elderly patients and poses a substantial challenge for clinicians

Delirium comprises a wide range of symptoms, but the prevailing narrow definition impedes diagnosis and efforts to improve treatment

Diagnosis can be improved by clinicians becoming more aware of hypoactive presentations, incorporating cognitive assessment into routine practice, and using simple screening instruments

Environmental strategies for treatment are free of adverse effects but are underutilised

Neuroleptics (such as haloperidol) continue to be used as first line treatment, but benzodiazepines are indicated in specific situations

of the sleep-wake cycle, thought processes, affect, perception, and activity levels, are underemphasised in diagnostic systems but contribute substantially to problems in identifying and managing delirium. Depending on which symptoms are apparent, delirium may be mistaken for a variety of disorders including dementia, mood disorders, and functional psychoses.

Identification

Delirium is underidentified in clinical practice: non-detection rates of 33-66% are typically reported.⁴ The agitated, disturbed image of delirium tremens is an inaccurate and damaging stereotype because it represents the minority of cases, and the existence of this stereotype is linked to the underdetection of somnolent or hypoactive cases. Failure to diagnose the disorder does not merely reflect preferences in terminology but represents an actual failure to recognise and treat the disorder appropriately and is associated with a poorer outcome.⁵ Detection can be improved by implementing educational programmes⁵ and by putting greater emphasis on routine cognitive testing and the use of screening instruments. The Confusion

Differential diagnosis of delirium

	Diagnosis			
	Delirium	Dementia	Depression	Schizophrenia
Onset	Acute	Insidious	Variable	Variable
Course	Fluctuating	Steadily progressive	Diurnal variation	Variable
Consciousness and orientation	Clouded; disoriented	Clear until late stages	Generally unimpaired	Unimpaired but patient may be perplexed in acute stage
Attention and memory	Poor short term memory; inattention	Poor short term memory without marked inattention	Poor attention but memory intact	Poor attention but memory intact
Psychosis present?	Common (psychotic ideas are fleeting and simple in content)	Less common	Occurs in small number (psychotic symptoms are complex and in keeping with prevailing mood)	Frequent (psychotic symptoms are complex and often paranoid)
Electroencephalogram	Abnormal in 80-90%; generalised diffuse slowing in 80%	Abnormal in 80-90%; generalised diffuse slowing in 80%	Generally normal	Generally normal

Assessment Method is widely used because it is reliable, brief, and applicable to a variety of settings.⁴ Unfortunately, routine cognitive assessment is less common in the technological world of modern medicine and knowledge of a patient's prior cognitive status is often minimal. Given that delirium may be the sole indicator of serious illness, any patient experiencing a sudden deterioration in mental status is best presumed delirious until proven otherwise.

Features that differentiate delirium from other disorders are listed in the table. Typically delirium differs from dementia by virtue of its acute course and reversibility, but the boundaries are blurred in cases in which there is comorbidity, a prolonged delirious state, or Lewy body dementia (with its fluctuating course and symptoms that frequently include psychosis); they are further blurred by evidence that delirium symptoms frequently persist beyond the acute treatment phase.⁵ However, the presentation of delirium is the same regardless of whether dementia is present because symptoms of delirium will dominate when they co-occur.⁷ Psychological symptoms of depression are common in patients with delirium: up to 42% of patients referred to psychiatry services for consultations for suspected depressive illness have delirium.⁸ Distinguishing delirium from depression is particularly important since in addition to delaying appropriate treatment, many antidepressants have marked anticholinergic activity and can aggravate delirium. The investigation of suspected delirium is reviewed in detail elsewhere.²⁻⁴

Risk factors and causes

The causes of delirium are many. In a typical case, predisposing and precipitating factors interact with multiple aggravating or perpetuating factors, which influence the course. The multifactorial nature is often underemphasised, but studies that have accounted for the possibility of multiple causes have found that between two and six factors may be present in any single case.⁹ It is therefore vital to be aware of risk factors and, having identified an explanation for delirium, remain vigilant as to the possibility of additional factors. Attempting to identify and treat a single cause is overly simplistic: each case needs detailed, repeated assessment for multiple potential factors.

Delirium is caused by factors in the patient as well as by pharmacological and environmental factors (box). Age, pre-existing cognitive impairment, severe comorbidity, and exposure to medication are robust

predictors of the risk of delirium.^{1-2, 10} Models of causation that quantify the role of predisposing factors and precipitating insults have shown that cumulative interactions with the baseline risk are especially predictive. If vulnerability at baseline is low, patients are resistant to delirium despite exposure to significant precipitating factors, but if vulnerability at baseline is high, delirium is likely to occur with exposure to only minor precipitating factors.¹⁰

Although many risks for delirium reflect the enduring characteristics of the patient, some factors can be modified to prevent onset. At the very least, patients at high risk warrant close observation for emergent delirium and prompt intervention. Medications are implicated in 20-40% of cases: most

Risk factors for delirium

Patient's factors

Individual:

- Age
- Pre-existing cognitive deficit
- Severe comorbidity
- Previous episode of delirium
- Personality before illness

Perioperative:

- Course of postoperative period
- Type of operation (for example, hip replacement)
- Emergency operation
- Duration of operation

Specific conditions:

- Burns; AIDS; fracture; hypoxaemia; organ insufficiency; infection; metabolic disturbances (for example, dehydration, low serum albumin concentration)

Pharmacological factors

- Treatment with many drugs
- Dependence on drugs or alcohol
- Use of psychoactive drugs or alcohol

Specific drugs that may cause problems

- Benzodiazepines
- Anticholinergic agents
- Narcotics

Environmental factors

- Extremes in sensory experience (for example, hypothermia)
- Deficits in vision or hearing
- Immobility or decreased activity
- Social isolation
- Novel environment
- Stress

prescribed drugs can cause delirium but benzodiazepines, narcotics, and drugs with anticholinergic activity have a particular propensity.¹¹ Many drugs and their metabolites may unexpectedly contribute to causing delirium because their anticholinergic effects are unrecognised. This was illustrated by a study that identified sufficient anticholinergic activity to cause significant impairments in memory and attention in elderly patients; this activity occurred in 10 of the 25 drugs most commonly prescribed to elderly people including theophylline, digoxin, and warfarin.¹¹ It is therefore prudent to minimise exposure to drugs and to reduce doses or stop administration of high risk compounds especially during high risk periods, such as the perioperative period. Many risk factors may simply be markers of general morbidity, and studies showing the preventive impact of modification of these risk factors are lacking but important. None the less, preliminary evidence indicates that interventions that reduce sensory deficits, immobility, sleep disturbance, dehydration, and cognitive impairment can reduce the number of episodes of delirium and their duration.¹²

Treating patients with delirium

Although delirium indicates the existence of an underlying pathology, it has significant independent morbidity; patients with delirium require longer hospitalisation than control patients without delirium; and there

is a high frequency of complications (such as falls, infections, and pressure sores) in patients with delirium. Additionally, patients with delirium are more likely to subsequently need care in an institution. The negative impact of delirium may also include an increased risk of death.¹³ Treatment should be aimed at the specific symptoms of delirium, and efforts should be made to identify and treat underlying causes. Diagnosis and treatment occur concurrently, and regular evaluation of progress is important. Because of its serious nature, an episode of delirium is often best managed in hospital because aggressive investigation and treatment can be facilitated; however, this advantage must be balanced against the potentially deleterious effects on elderly people or those who have cognitive impairment of a sudden change in environment. In the United Kingdom patients with delirium may give informed consent during lucid periods, but in patients deemed incompetent urgent interventions are governed by common law doctrine—that is, treatment may be given without informed consent if medical colleagues would generally consider it appropriate and a reasonable person would want it. The competitive benzodiazepine antagonist flumazenil has been used to temporarily restore mental capacity in patients with delirium and hepatic failure to allow them to participate in decisions about treatment or personal affairs.¹⁴

During the postoperative period patients are at high risk for delirium, but delirium occurring at this time is particularly amenable to therapeutic efforts.¹⁵ A large, prospective, multicentre study directly implicated surgery and anaesthesia as factors contributing to the development of both short term and long term postoperative cognitive impairment,¹⁶ but there remains uncertainty about the specific factors that contribute to delirium (such as the type and duration of procedure, the circumstances of the operation, and the pharmacological agents used) (box). Nevertheless, systematic strategies to detect and manage the condition, which involve providing preoperative psychological support (education and reduction of anxiety), the use of patient controlled analgesia, and careful postoperative management, have significant benefits over traditional reactive care and can reduce the incidence of delirium.¹⁵

Supportive and environmental measures

Patients who have recovered from delirium have reported that simple but firm communication, reality orientation, a visible clock, and the presence of a relative all contribute to a heightened sense of control during delirium (box).¹⁷ Many supportive measures (for example, attention to noise, lighting, and mobility levels) (box) reflect basic features of a good therapeutic environment, protect against delirium, and should be applied routinely to all patient care settings. Other efforts that are made specifically in response to symptoms of delirium (for example, helping patients to reorient themselves), should be specifically detailed in treatment plans. Preliminary evidence suggests that nurses trained in managing patients with delirium improve outcomes by limiting risk factors, enhancing recognition of the condition, and encouraging standardised treatment.¹⁸

Environmental factors in treating delirium

Providing support and orientation

Communicate clearly and concisely; give repeated verbal reminders of the day, time, location, and identity of key individuals, such as members of the treatment team and relatives

Provide clear signposts to patient's location including a clock, calendar, chart with the day's schedule

Have familiar objects from the patient's home in the room

Ensure consistency in staff (for example, a key nurse)

Use television or radio for relaxation and to help the patient maintain contact with the outside world

Involve family and caregivers to encourage feelings of security and orientation

Providing an unambiguous environment

Simplify care area by removing unnecessary objects; allow adequate space between beds

Consider using single rooms to aid rest and avoid extremes of sensory experience

Avoid using medical jargon in patient's presence because it may encourage paranoia

Ensure that lighting is adequate; provide a 40-60 W night light to reduce misperceptions

Control sources of excess noise (such as staff, equipment, visitors); aim for <45 decibels in the day and <20 decibels at night

Keep room temperature between 21.1°C to 23.8°C

Maintaining competence

Identify and correct sensory impairments; ensure patients have their glasses, hearing aid, dentures. Consider whether interpreter is needed

Encourage self care and participation in treatment (for example, have patient give feedback on pain)

Arrange treatments to allow maximum periods of uninterrupted sleep

Maintain activity levels: ambulatory patients should walk three times each day; non-ambulatory patients should undergo a full range of movements for 15 minutes three times each day

Environmental strategies are free from adverse effects, but they are underutilised and are often applied only in response to behavioural disturbance rather than in response to the degree of cognitive impairment.¹⁹ The fact that these strategies are most commonly used in hyperactive patients may reflect the prevailing idea that “severe” delirium is associated with hyperactive, disturbed patients; the reality is that these patients have better outcomes than patients who are underactive and less disturbed.²⁰ It remains unclear whether better outcomes in hyperactive patients reflect underlying causes that are more treatable or differences in treatment.

Family members or caregivers can answer questions about what a patient’s mental status was before illness and facilitate efforts to reassure and reorient patients. Explaining delirium to family members is important because caregivers who are upset or ill informed can exacerbate a patient’s distress. Delirium may herald the terminal stages of illness, and it can shape enduring memories of loved ones as “crazy” or disturbed unless it is explained and managed sensitively. Because symptoms of delirium are often not fully resolved at the time the patient is discharged from hospital, relatives frequently play crucial roles in planning and monitoring care.

Drug treatment

Drug treatment of delirium requires careful consideration of the balance between the effective management of symptoms and potential adverse effects. Prescribing is often influenced by pressure from relatives, time constraints, or difficulties in communication between medical and nursing staff. The use of psychotropic drugs complicates the ongoing assessment of mental status, can impair the patient’s ability to understand or cooperate with treatment, and is associated with a greater incidence of falls. It is therefore important to clarify the reasons for using drugs to treat delirium: is the primary aim to alleviate delirium or to contain problem behaviour? Sedative compounds can improve agitation but may worsen cognitive impairment. A minority of patients require sedation to protect themselves. Less medication is required in cases in which delirium is identified early by screening,²¹ but there is a lack of studies of the effectiveness of pharmacological prophylaxis in high risk populations.

Antipsychotic drugs

Antipsychotics are the cornerstone of pharmacological treatment. Neuroleptics ameliorate a range of symptoms, are effective both in patients with a hyperactive or hypoactive clinical profile, and generally improve cognition.^{2 21 22} The onset of their action is rapid: improvement is usually evident within hours or days and thus occurs before underlying causes are treated.²³ Neuroleptics are superior to benzodiazepines in treating delirium that has been caused by factors other than alcohol withdrawal or sedative hypnotics.² Chlorpromazine, droperidol, and haloperidol have similar efficacy, but haloperidol is preferred because it has fewer active metabolites, limited anticholinergic effects, less sedative and hypotensive effects, and can be administered by different routes.^{2 24} Although the use of high potency antipsychotic drugs like haloperidol brings an increased risk of extrapyramidal side effects,

Pharmacological treatment of severe disturbance in delirium^{24–26 34 39}

- Administer 0.5–10 mg haloperidol (intramuscularly or intravenously) depending on level of disturbance and likely tolerance (having considered age, physical status, and risk of side effects)
- Observe patient for 20–30 minutes. If the patient remains unmanageable but has not had any adverse effects, double the dose and continue monitoring:
 - Repeat the cycle until an acceptable response occurs or side effects occur
 - Patient should be manageable not obtunded
- Up to 2 mg of lorazepam may be administered intravenously or intramuscularly every four hours and may be beneficial in allowing a lower dose of antipsychotics to be used in cases in which extrapyramidal side effects occur:
 - Monitor respiratory functions and level of sedation carefully
 - Consider administering flumazenil if there is evidence of significant toxicity
- Upper limits on doses have not been clearly established, but up to 100 mg of intravenous haloperidol every 24 hours is generally safe as is up to 60 mg intravenous haloperidol every 24 hours if benzodiazepines are used concomitantly

the actual reported incidence is low.^{21 25 26} Moreover, intravenous administration of haloperidol seems to be less likely to cause extrapyramidal side effects in patients with delirium.²⁷ Droperidol is more suitable when a faster onset of action or greater sedation is required. Pimozide is a potent calcium antagonist and may be more appropriate for treating delirium that is accompanied by hypercalcaemia.²⁸

The dose of an antipsychotic drug is determined by the route of administration, the patient’s age, the amount of agitation, the patient’s risk of developing side effects, and the therapeutic setting. Low dose oral haloperidol (1 mg to 10 mg/day) improves symptoms in most patients.^{21–23} Information on drug treatment in highly disturbed patients comes from studies of patients with general agitation rather than patients with delirium. A clear association between the successful control of agitation and an improved outcome in delirium has not been shown but it can be inferred from evidence linking poorer outcomes to the complications of untreated illness, such as non-compliance with treatment (for example, refusing medication) and immobility.²⁰ A treatment regimen for severe cases requiring prompt, aggressive control of symptoms is outlined in the box.

Olanzapine (5–10 mg) and risperidone (1.5–4 mg) have been used successfully in uncontrolled case series.^{29 30} These atypical compounds cause less sedation and fewer extrapyramidal effects, and studies of neuropsychological effects in normal elderly volunteers suggest that they have other advantages.³¹ However, they are only available in oral forms, and the advantage of using them for short term treatment, which is typical in delirium, is unclear.

Benzodiazepines

Benzodiazepines are first line treatment for delirium that is associated with seizures or withdrawal from alcohol or sedatives.³² They are also a useful adjunctive treatment for patients who cannot tolerate antipsychotic drugs because lower doses can be used³³ and their effects can be rapidly reversed with flumazenil. The therapeutic aims of drug treatment should be explicit since anxiolytic, sedative, and hypnotic effects

occur as doses are increased. Benzodiazepines can both protect against delirium and be a risk factor for it; this highlights the need for judicious use in patients dependent on alcohol or benzodiazepines. Lorazepam has several advantages owing to its sedative properties, rapid onset, and short duration of action; it also has a low risk of accumulation; there are no major active metabolites; and its bioavailability is more predictable when it is given intramuscularly. Lower doses are necessary in elderly patients, those with hepatic disease, or those receiving compounds that undergo extensive hepatic oxidative metabolism (for example, cimetidine and isoniazid). The recommended upper limits for intravenous lorazepam are 2 mg every four hours.³⁴ Giving adequate initial doses reduces the risk of paradoxical excitement (that is, disinhibition with worsening of behavioural disturbance).

Emerging therapies

Disturbances of cholinergic metabolism are implicated in cases in which delirium is caused by hypoxia, traumatic brain injury, or hypoglycaemia, or is drug related. Anticholinergic delirium is generally treated conservatively by withdrawing the offending agent and occasionally by administering physostigmine.² Other procholinergic agents used to counter cholinergic deficits in dementia have theoretical potential but are not recommended owing to the risk of causing adverse effects. Current smoking has been identified as a possible protective factor against delirium,³⁵ but the usefulness of nicotine replacement treatment in protecting against delirium has not been tested.

Trazodone and mianserin are antidepressant compounds that share antagonistic actions at 5-HT₂ (serotonin) receptors. Open studies of low dose treatment of delirium with these compounds have found a rapid reduction of non-cognitive symptoms in particular. This effect was independent of the mood altering actions of the drugs.²³⁻²⁶ Other reports have advocated the use of light therapy,³⁷ but the usefulness of this treatment needs to be more fully evaluated before it is used routinely.

Managing patients after discharge

Many patients with delirium are discharged before their symptoms are fully resolved; this factor must be accounted for in planning their care after discharge. The continuing need for rehabilitation must be explicitly documented. Problems with attention and orientation are especially persistent.⁶ Further episodes may be prevented by addressing risk factors such as medication and sensory impairment. The psychological sequelae of delirium have not been studied enough, but depression and post traumatic stress disorder have been described. Most patients dismiss the episode of delirium once it has passed, but a significant minority have lingering concerns that an episode of delirium may represent the first step towards loss of mental faculties and independence.¹⁷ Other patients experience "silent delirium" and are ashamed or afraid to admit to symptoms. A post-hospital visit to the treatment environment can facilitate adjustment and clarify the transient nature of delirium symptoms.³⁸

Conclusions

There has been a shift towards recognising delirium as a distinct entity requiring study in its own right. This has resulted in greater appreciation of the variety of the syndrome's symptoms and the development of accurate screening tools that can be readily applied in routine clinical practice. Optimal management of delirium primarily depends on reducing modifiable risk factors and detecting high risk cases early. Treatment requires multifaceted, interdisciplinary efforts that address both the underlying causes and the symptoms of delirium. The value of supportive and environmental strategies is increasingly being recognised, particularly in research designed and run by nurses. Typical neuroleptic drugs remain the cornerstone of treatment; however, their effectiveness in both acute and long term treatment in different populations in which delirium has different causes and for varying symptom profiles remains poorly studied. Benzodiazepines are the treatment of choice in delirium associated with specific causes, such as alcohol withdrawal, and are a useful adjunct treatment in other cases. Specific treatments for delirium, such as physostigmine and flumazenil, can be useful where rapid reduction in symptoms is desirable. Atypical neuroleptics and procholinergic agents have substantial treatment potential but have not been studied in depth. The symptoms of delirium frequently persist beyond the acute phase of treatment, therefore post-discharge treatment plans must focus on reducing ongoing risk factors and managing residual functional impairments.

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Lesson of the week

A painful hip as a presentation of Guillain-Barré syndrome in children

Tjun Tang, Charles Noble-Jamieson

Pain of the lower limb in children is a symptom of many conditions and can lead to diagnostic difficulties; the clinician may search for a musculoskeletal rather than a neurological cause. Guillain-Barré syndrome must be considered when a child presents with pain of the lower limb. Misdiagnosis is more common in the paediatric population because the child's history and cooperation in the neurological examination are often limited. Failure to diagnose Guillain-Barré syndrome and to initiate prompt treatment is potentially life threatening as the disease can quickly lead to respiratory failure and death from muscle paralysis. We describe a child with Guillain-Barré syndrome who initially presented with a painful hip.

Case report

A two year old boy presented with pain in his right leg. Three weeks earlier he had had tonsillitis, fever, and diarrhoea. His doctor had treated him with amoxicillin, and his symptoms subsequently settled. The pain in his leg started two days before admission. He limped and was reluctant to walk. The pain was precipitated by walking and was vaguely localised to the hip. He had no other joint symptoms or relevant history. On examination he was apyrexial but irritable. He had difficulty sitting and refused to stand or weight bear. Neurological examination revealed intact cranial nerves, although the gag reflex was depressed, which was partly attributed to his uncooperative behaviour. However, there was no indication that he had been choking.

Examination of the upper limbs revealed normal tone and power, but reflexes of the biceps, triceps, and supinator were diminished. The patient was distressed on flexion, extension, and internal rotation of the right hip. Otherwise the joints had full range of movement with no swelling or tenderness, although the patient preferred to lie with his hips flexed and mildly rotated externally. The patella and ankle reflexes were diminished bilaterally. The plantar responses were extensor.

Ultrasonography of the hips showed a symmetrical appearance with no synovial thickening or effusions. A radiograph of the hips showed a slightly irregular epiphysis on the right side but otherwise gave normal results. A bone scan was later performed, but no definitive abnormality was found.

Tests for blood count, erythrocyte sedimentation rate, and C-reactive protein concentration gave normal results. An orthopaedic opinion was sought, and an irritable right hip was considered likely. The patient was initially given paracetamol for analgesia, but because of increasing hip pain and irritability he was given Oramorph (Boehringer Ingelheim) and his right leg was placed on skin traction.

On day 4 after admission he continued to deteriorate; he mentioned pain in his neck, elbows, and wrist. He became weak, lethargic, and disinterested in feeds. He was unable to sit up independently. A musculoskeletal cause was considered, and he was referred to a rheumatologist at a tertiary centre. On examination only generalised limb weakness and hypotonia were found, and a neurological opinion was subsequently sought. By this time he had started to cough and was

Guillain-Barré syndrome should be part of the differential diagnosis of children with painful lower limbs

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