Delirium: moving beyond the clinical diagnosis

Delirium: além do diagnóstico clínico

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Abstract

Delirium is a common mental disorder that has been associated with increased length of hospital stay and health costs, as well as higher morbidity and mortality rates in later life. To date, psychiatric interventions have mostly been limited to the clinical diagnosis of *delirium* and treatment of the behavioural and psychological complications of the acute episode, although this seems to have a negligible impact on the course and long-term outcome of patients. This paper reviews the development of recent strategies designed to reduce the incidence and complications of *delirium*, and proposes that an effective management plan must always include the basic components of primary, secondary and tertiary prevention.

Keywords

Delirium. Confusional state. Therapeutics. Case management. Risk factors. Primary prevention.

Resumo

Delirium é um transtorno mental comum que tem sido associado a permanência hospitalar prolongada, aumento nos custos com cuidados médicos e maior morbidade e mortalidade entre idosos. De forma geral, o manejo de pacientes tem se limitado ao tratamento das complicações advindas do episódio agudo e dos distúrbios comportamentais e psicológicos associados ao delirium, embora isto pareça ter um impacto desprezível sobre o curso da doença e o prognóstico dos pacientes no longo prazo. Este artigo revisa o desenvolvimento de estratégias desenhadas com o objetivo de reduzir a incidência e as complicações clínicas do delirium e propõe que um tratamento efetivo de pacientes com delirium deve sempre incluir medidas básicas de prevenção primária, secundária e terciária.

Descritores

Delírio. Estado confusional. Terapêutica. Administração de caso. Fatores de risco. Prevenção primária.

Introduction

The concept of delirium has been known since the time of the ancient Greek and Roman physicians. The word itself is derived from the Latin *de* (from, away) and *lira* (track, furrow) suggesting an understanding, even from that time, that the state was a change from normal consciousness and behaviour. Today it is still the most common mental health issue for the elderly, affecting 14-56% of elderly hospitalised medical patients and 6-24% of nursing home patients. In the general community the point-prevalence of delirium is yet undefined, though it may be 1% of the population over 55 years-old. Even in the twenty-first century the syndrome is often unrecognised or not diagnosed in up to 67% of cases or it is misdiagnosed as dementia or other psychiatric illnesses such as depression or schizophrenia, or perhaps worse, accepted as a normal part of aging.

Delirium is mostly studied in hospital environments. The presence of a delirium leads to poorer hospital outcomes, longer hospital admissions and increase in the resources used.7-10 Studies looking at the longer-term prognosis show an increase in the rate of hospital re-admission, the need for nursing home placement and increase in mortality and morbidity, as well as clear decline in function.¹⁰⁻¹⁴ Delirium causes the individual patient and their families distress and hardship, but beyond that there is a marked economic cost to every nation's healthcare system. 15 The cost of delirium can be quantified in hospital systems and make up a large proportion of health budgets.³ However, there are still marked hidden costs, such as further morbidity, increased care needs (especially care needs provided by unpaid carers), nursing home placements and readmissions to acute hospital that go unaccounted for in considering the economic burden of delirium.

Diagnosis and clinical features

Delirium is a neuropsychiatric syndrome characterised by disturbances in attention and consciousness that are acute in onset and have a fluctuating course. 16,17 It usually involves a generalised cognitive impairment that historically has an acute onset and is also fluctuating, and generally involves disturbances in orientation, attention, memory, language, planning and organisational skills and other executive functions. Other disturbances include affect, psychomotor activity, thought processes and content, abnormal perceptions and speech, which are often under emphasised as diagnostic items in some classification systems such as the DSM-IV.17 However, these neuropsychiatric disturbances are often the causes of the behavioural presentation of the patient (Table 1).

Neuroimaging appears to have a limited role in assisting with the diagnosis of delirium, although its use may contribute to clarify the diagnosis of intra-cerebral pathology (space-occupying lesions, haematoma, haemorrhage, and infarct).

The use of electroencephalograms (EEG) has role in the evaluation of delirium but its usefulness may be limited in the assessment and diagnosis of the individual with delirium due to difficulty of the procedure on an agitated patient.

However, an EEG can be useful in those individuals where the diagnosis is in doubt or where non-convulsive status epilepticus is suspected.

In delirium, EEG findings include slowing of posterior dominant rhythm and increased generalised slow-wave activity, except for alcohol or sedative withdrawal delirium.

Until 1980 and the publication of the DSM-III, there was no discrete criteria for delirium, which could not be distinguished from other acute brain syndromes.^{1,22} Even today one of the many synonyms still in use for delirium is

acute brain syndrome. ¹⁶ The term acute was then synonymous with the idea of reversibility rather than its temporal meaning of now. This of course led to difficulties in classification and research, but even with the tightening up of diagnostic criteria with the DSM-IV and the ICD-10, there is still confusion in this area. ^{16,17} This has led to the development of reliable and valid, sensitive and specific, structured and simple to use screening instruments to assist in the diagnosis and screening for the presence of delirium. These include *The Delirium Rating Scale, Delirium Rating Scale-Revised-98, The Confusion Assessment Method*, and *The Delirium Symptom Review*. ²³⁻²⁷ The *Confusion Assessment Method* has been shown to be valid and reliable in the Portuguese version. ²⁸

Delirium subtypes

There have been attempts to sub-classify delirium on the basis of phenomenology. Most work has been on the psychomotor activity. Classifications of hyperactive type (15-29%), hypoactive type (19-43%), mixed type (43-52%) and no psychomotor disturbance (0-14%) have been proposed.²⁹⁻³¹ It is not clear if there is a relationship between the subtype and risk factors for delirium or aetiologies, except for the suggestion that delirium from drug withdrawal states has a hyperactive presentation.32 However, there is a suggestion that hypoactive delirium states lead to longer hospital stay, inferring a possible worse outcome and this may have clinical significance.^{29,30} This is probably confounded by the increased likelihood of missed diagnosis of hypoactive delirium compared to the mixed and hyperactive types. Obviously more research is needed to fully glean the clinical significance of such a subclassification.

Table 1 - Clinical features of delirium.

Cognition

Consciousness
 Attention
 Clouding or reduced awareness of the environment.
 Reduced ability to focus, sustain or shift attention.

3. Disorientation Especially to time and place

With increased severity even to person.
4. Language Poor word finding, dysnomia and impove

Poor word finding, dysnomia and impoverished speech, impaired writing, perseveration, non-fluent aphasia, poor comprehension.

5. Memory Immediate recall and recent memory. Difficulties in learning

Executive function Impairment in planning and organisation.
 Impairment in performing goal directed tasks.

Psychotic symptoms

1. Delusions Often non-systematised.

2. Abnormal perceptions Hallucinations, illusions, misinterpretations

Sleep disturbances

1. Sleep-wake cycle disturbances Daytime sleepiness, nighttime arousal, difficulty falling asleep.

Psychomotor disturbances

Retardation, agitation and fluctuation between the two. Dysarthria, dysphagia, tremor, ataxia, dyspraxia, falls and seizures.

Affective disturbances

1. Aroused Excitability, irritability, agitation, facetious.

2. Lability Fluctuations in mod

Dysphoria Depression, perplexed, fear and suicidal ideations.
 Apathy

Autonomic Disturbances

Tachycardia, dilated pupils, fever, sweating, pallor or flushing, constipated or diarrhoea, excessive pilomotor response.

Other work to attempt to see a relationship between aetiology or risk factors and phenomenological expression of delirium is showing little in the way of relationship. However, the work is still in its infancy and there are many methodological difficulties that need to be considered in this type of research³². It is hoped that there is a possible relationship between phenomenological expression of delirium and aetiology, risk factors, patho-physiology and immediate and long-term prognosis.

An understanding of the different phenomenological subtypes of delirium in relation to the different pathophysiological changes that lead to the clinical expression of the syndrome may lead to the development of more refined management protocols.

Pathophysiology of delirium

Despite delirium being recognised for a very long time, the patho-physiology of delirium is poorly understood. O'Keeffe describes the main theory as that delirium represents the clinical manifestation of diffuse, reversible

Table 2 - Management strategies/interventions of delirium for the psychiatrist.

Acute intervention

- Psychiatry intervention
 - Co-ordination of those healthcare workers and physicians providing care
 - Identify and correct aetiological/precipitating factors.
 - Assess Mental state and monitor to ensure continued safety
 - Assess psychiatric status of patient and family and establish therapeutic alliance.
 - Psychoeducation: patient, family and staff.
 - Assessment of competency and ability to consent.
- 2. Environmental interventions
 - Orientation
 - a. Encourage clear and concise communication.
 - Regular verbal reminders of time, place and purpose.
 - c. Encourage staff to always identify themselves and describe their purpose
 - d. Orientation paraphernalia in room, eg Signposts, calendar and
 - e. Use of the familiar eg family members or objects form home such as photographs.
 - Providing an environment that is not overstimulating nor understimulating
 - a. Removal of unnecessary objects or furniture with adequate space for moving around.
 - Use of single bed rooms
 - c. Use of adequate lighting. (use of night lights to avoid misperceptions)
 - d. Minimise amount of extraneous noise
 - e. Ambient room temperature.
 - Optimising competence
 - a. Identifying and correcting sensory impairments eg use of glasses, hearing aids.
 - b. Use of interpreters for foreign language speakers.
 - Treatment regime organised to allow minimal sleep disturbance.
 - Maintenance of activity ie ambulation or in bed full of movements for those that cannot ambulate.
- 3. Supportive interventions
 - Reduction of anxiety eg regular reassurance, regular reorientation. Psycho-education

 - Continuous supervision eg ono-to-one specialling.
- 4. Somatic interventions
 - Minimising unnecessary medications.
 - Judicious use of appropriate psycho-pharmacology with the goal to return mental state to baseline rather than sedation.
 - Minimising somatic distress eg adequate analgesia or other symptom
- 5. Post-delirium management planning

impairment of cerebral oxidative metabolism and neurotransmission.²⁰ Therefore, any process interfering with neurotransmitter function or with the supply or use of substrates can cause delirium. It is clear that the cholinergic pathways are involved but also disturbances of other neurotransmitter pathways such as dopamine, serotonin and gamma-amino-butyric acid have been reported.33-36 This suggests that the patho-physiological disturbance in the neurotransmitter systems that leads to delirium may be the expression of a disturbance or imbalance between different neurotransmitters, and that the disruption can be in any stage within the pathways.

Attempts to correlate the neuroanatomical disturbances of delirium with neurochemical imbalances indicate that the most likely brain areas involved are the pre-frontal cortex, thalamus, fusiform cortex, posterior parietal cortex and the basal ganglia.35 This may have major clinical implications as different neuroanatomical or neurochemical disturbances may be associated with specific clinical symptoms of delirium and, as such, may require different treatments and have diverse outcomes.³⁷ Like many disorders within psychiatric classifications, delirium is a heterogenic syndrome and research into its sub-classification on pathophysiological disturbances may unlock the key to further evidence based management practices.

Management

Management of delirium has been the subject of a number of recent reviews and practice guideline articles.³⁸⁻⁴¹ In addition, recent work has been published on the management of delirium on special cases, such as the terminally ill. 42,43 In all these publications there is a clear understanding that management starts with the education of our medical colleagues on how to decrease the risk of delirium amongst their patients (prevention), as well as how to detect its early signs and symptoms. The non-detection of delirium does lead to poorer outcomes, but can be improved with the implementation of educational programs.44

The keys to management of the acute syndrome of delirium are the introduction of adequate supportive and environmental measures (table 2). The aim of psychopharmacological treatment is to return the individuals mental state to as close as possible to baseline and not to increase sedation (which maybe at odds with the views or requests of other healthcare workers involved in the patient's care). A discussion of the pharmacological therapies is not within the scope of this article except to say that at present there have been no controlled trials of the atypical neuroleptics for treating behavioural disturbances of delirium. Education of the patient and his/her family, during and after the delirious episode, is seen as mandatory in providing holistic management and a good immediate outcome and perhaps decrease the likelihood of secondary psychiatric sequelae, such as post traumatic stress disorder and depression. But in moving beyond diagnosing and immediate management what is our role?

Modern day concept of delirium

Much of the recent work with delirium has been devoted to the investigation of the multiple factors that contribute to its development. A distinct cause of delirium is often not found, but under close scrutiny there is rarely only one single factor.³ Inouye has proposed a model that takes account of the many factors that play a role in the development of delirium.⁴⁵ She suggests that there is an interaction between predisposing factors (the patient's vulnerability) and precipitating factors (insults or aetiological factors). Patients with high vulnerability (ie, multiple predisposing factors) require only minimal insults to develop delirium, whilst those with low vulnerability require multiple noxious insults to lead to a delirium. This model explains the relatively high number of cases where aetiological causes cannot be discerned. With this model we can then attempt to predict those individuals who are at risk to develop delirium and introduce preventative strategies in an attempt to decrease the incidence and consequences of delirium.

Prevention is a healthcare concept that psychiatry took a long time to embrace, though it has been discussed since Caplan in 1964.⁴⁷ The concept of primary, secondary and tertiary prevention have been clearly defined in psychiatry.⁴⁶ In relation to delirium, primary prevention would represent the introduction of measures designed to decrease the incidence of delirium. Secondary prevention would aim to decrease the severity and immediate morbidity of delirium, where as tertiary prevention would aim to address the long-term consequences of delirium, that is the high morbidity and mortality.

Inouye et al have looked at predictive models for delirium based on four risk factors: vision impairment, severe illness, cognitive impairment and high blood urea / creatinine ratio.47 Using this has enabled work to be set up to look at preventing the development of delirium in those who are at risk. Inouye et al then designed a multicomponent risk factor intervention study with the aim of decreasing the incidence and duration of delirium.48 The controlled trial put in place set protocols to deal with six risk factors: cognitive impairment, sleep deprivation, immobility, visual impairment, hearing impairment and dehydration on admission for those subjects not admitted with delirium. The study showed a significant decrease in the rate of delirium (9.9% in the intervention group versus 15.5% amongst controls) and duration in the number of days of delirium.⁴⁹ This study suggests that prediction of those at risk and early intervention can have an effect on the rate and severity of delirium and be cost effective.⁴⁸ More recent work has been successful in extending this work to specific groups of patients using secondary preventative strategy models.^{49,50}

But as stated previously, delirium is an important independent prognostic factor for longer-term outcomes, including high mortality rates, nursing home placement and functional decline. ¹⁰ It is still unclear the exact long term cognitive consequences of delirium, even though as far back as 1958, Engel and Romano had already suggested that delirium may be associated with permanent, irreversible brain damage. ⁵¹

As clinicians what is our role in decreasing this high morbidity and mortality especially considering that up to 52% of individuals with delirium are discharged from hospital with their symptoms unresolved?⁵² Are we doing enough in terms of tertiary prevention? Are we educating our medical colleagues to the importance and need of the initial diagnosis, appropriate management of risk factors and the need for longer term interventions? Rahkonen et al⁵³ have looked at post-discharge interventions in non-demented elderly individuals with delirium in an attempt to look at the effects of tertiary prevention interventions. The interventions included intensive nursing support and counselling, yearly rehabilitation by a multiskilled team and individual planned-community care services. After a three-year follow-up, the investigators observed a significant reduction in the time spend in long-term care facilities and decreased mortality(35% vs 18%).53 Obviously, more research is needed to clarify what type of long-term care provides the best outcomes, but this study shows that the mortality and morbidity associated with delirium in the longterm can, and should, be reduced.

Conclusion

The evidence shows that delirium is associated with marked mortality, morbidity, decline in function, increase need for health resources and increase admission into institutional care. But there is now evidence that primary prevention of delirium is possible and can significantly decrease its incidence. Education of others in the healthcare profession can increase awareness and detection rates, but this is not enough. Holistic management with environmental manipulations and patient support can decrease the duration and, perhaps, the severity of a delirious episode. In addition, post-delirious work that includes education, counselling and long-term community care and rehabilitation may markedly decrease mortality and morbidity.

The role of the psychiatrist has now moved well beyond that of a professional who is only responsible for the diagnosis and immediate acute management of patients with delirium. We, as a group, need to take responsibility for the overall management plan of patients, including the use of evidence-based programs of primary, secondary and tertiary prevention of delirium. It is our responsibility to advocate for the best possible treatment for patients, as these have been shown to decrease morbidity and mortality, as well as reduce the overall costs in the delivery of health services.

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