Cognitive disorders

Delirium

Delirium is characterized by global impairment of consciousness (clouding of consciousness), resulting in reduced level of alertness, attention, and perception of the environment, and thence cognitive performance. A number of other terms, such as acute confusional state, acute brain failure, and acute organic syndrome, have also been used, but delirium is the preferred term in both .ICD-10 and DSM-5

Epidemiology

The prevalence of delirium in the elderly is 1–2% in community samples, 8–17% in emergency departments, 18–35% on admission to hospital, with an overall occurrence in inpatients of 29–64%. Rates of about 8% have been reported in residential care homes. Delirium is much more common in the elderly than in younger people, and in individuals with diminished 'cerebral reserve', notably those with pre-existing dementia and other medical factors. Delirium is thus a common disorder in all medical settings, especially for older people. Moreover, it is associated with several adverse outcomes, discussed .below, and so its prompt recognition and treatment are important

Risk factors

:Predisposing factors

Dementia

Previous episode of delirium

Sensory impairment

History of cerebrovascular disease

Alcohol misuse

Older age

Precipitating factors

Substance-related: Prescribed medical drugs (e.g. steroids, digoxin, diuretics). Psychotropic medication (e.g. sedatives, opiates, hypnotics). Alcohol—intoxication, withdrawal, delirium tremens. Use of more than one drug

Physiological causes: Septicaemia. Infection (e.g. urinary tract, respiratory). Hypoxia (e.g. postoperative). Organ failure (e.g. renal, hepatic, cardiac). Abnormal albumin. Metabolic acidosis. Hypoglycaemia or hyperglycaemia. Dehydration

.Neurological causes: Post-ictal. Head injury. Space-occupying lesion Encephalitis

.Use of physical restraints

.Bladder catheter

.Pain

.Sleep deprivation

Constipation

Clinical features

The cardinal feature of delirium is disturbed consciousness. It is manifested as drowsiness, decreased awareness of one's surroundings, disorientation in time and place, and distractibility. At its most severe the patient may be unresponsive (stuporose), but more commonly the impaired consciousness is quite subtle. Indeed, the first clue to the presence of delirium is often one of its other features, which include mental slowness, distractibility, perceptual anomalies, and disorganization of .the sleep–wake cycle

Symptoms and signs vary widely between patients, and in the same patient at different times of day, typically being worse at night. For example, some patients are hyperactive, restless, irritable, and have psychotic symptoms, while others are .hypoactive, with retardation and perseveration

Repetitive, purposeless movements are common in both forms. Thinking is slow and muddled, but often rich in content ('dream-like'). Ideas of reference and delusions .(often persecutory) are common, but are usually transient and poorly elaborated

Visual perception is often distorted, with illusions, misinterpretations, and visual hallucinations, sometimes with fantastic content. Tactile and auditory hallucinations .also occur

Anxiety, depression, and emotional lability are common. The patient may be frightened, or perplexed. Experiences of depersonalization and derealization are sometimes described. Attention and registration are particularly impaired, and on .recovery there is usually amnesia for the period of the delirium

Aetiology

In practice, most cases are multifactorial, and sometimes idiopathic. In elderly people in medical settings, drugs (especially polypharmacy) and infection are probably the commonest causes; in other settings and populations, delirium has a wide range of causes and hence requires urgent and wide ranging .investigations

The pathophysiological basis of delirium is unclear. The severity of clinical disturbance correlates with the degree of slowing of cerebral rhythms on EEG, and the neurotransmitters dopamine and acetylcholine are implicated in a final common pathway. Inflammatory, metabolic, and genetic factors also .contribute

Management of delirium

Treatment

Delirium is a medical emergency. Although it is a clinical diagnosis, it is essential to identify and treat the underlying cause, and a range of investigations may be required. Many are routine (e.g. electrolytes, full blood count, urinalysis), whereas others are carried out .depending on the context (e.g. lumbar puncture). Vital signs need regular measurement

As delirium is often caused by drugs (due to side effects or withdrawal effects), these should always be suspected until there is evidence of another cause. The mainstay of treatment should be non-pharmacological, since there is little evidence that any medication improves .outcome, and antipsychotics (which are commonly used) have known adverse effects

General measures should always be used to relieve distress, control agitation, and prevent exhaustion. These include frequent explanation, reorientation, and reassurance. Unnecessary changes in the staff caring for the patient should be avoided. The patient should ideally be nursed in a quiet single room. Relatives should be encouraged to visit regularly. At night, lighting should be sufficient to promote orientation, while not preventing sleep. In many cases, use of these behavioural measures can be sufficient to manage the patient whilst the .cause of the delirium is being investigated and treated

Nevertheless, despite such interventions, in practice many patients with delirium are treated with medication to control agitation and distress, and to allow adequate sleep. Antipsychotics are usually first-line medication. They should be limited to cases with severe agitation (endangering the patient) and for psychotic symptoms causing distress (hallucinations or .delusions)

Haloperidol has conventionally been used, starting at a very low dose, and then carefully titrated to achieve the desired calming effect without excess sedation or side effects. If necessary, the first dose can be given intramuscularly, followed by doses every 6 hours. Atypical antipsychotics are now often prescribed instead of haloperidol; the same principles of careful administration and monitoring apply. Some causes of delirium require avoidance of antipsychotics, or particular caution when using them. This includes all patients with coexisting dementia, especially dementia with Lewy bodies

Antipsychotics should be avoided in delirium associated with alcohol withdrawal (delirium tremens) or with epilepsy, because of the risk of seizures. In delirium tremens, a benzodiazepine is the standard .treatment (either lorazepam or chlordiazepoxide)

All drugs should be used with caution in liver failure because of the danger of precipitating hepatic .coma

Outcome

Many cases of delirium recover rapidly. The prognosis is related to the underlying cause, and is worse in the elderly and in those with pre-existing dementia or physical illness. Patients with a 'hypoactive' behavioural profile have a worse outcome than those who are 'hyperactive'. There is an elevated mortality rate following delirium, with an estimated 25% mortality at 3 months, although published estimates vary markedly; a recent meta-analysis of elderly patients found that an episode of delirium was associated with a twofold increased risk of death in the next 2 years

A meta-analysis reported a fivefold increase in incidence of dementia 2 years after an episode of delirium

Amnesia and amnesic disorders

Amnesia is loss of memory, and amnesic (also called amnestic) disorders or syndromes are those in which episodic memory is specifically and persistently affected, and with a decline from previous level of functioning. In ICD-10 and in routine clinical practice, these criteria together distinguish amnesic disorders from dementia, delirium, or neurodevelopmental .disorders

Amnesic disorders manifest as an inability to learn new information (anterograde amnesia) and to recall past events (retrograde amnesia). To make the diagnosis, there should be a significant impairment in social or occupational functioning, and evidence of a general medical condition which can be aetiologically related to the memory impairment. Korsakov syndrome (also called Korsakoff syndrome) is sometimes erroneously referred to .synonymously with amnesic disorder, but is in fact a specific form of it, as described below

Causes of amnesia

Transient

Transient global amnesia. Transient epileptic amnesia. Head injury Alcoholic blackouts. Post-electroconvulsive therapy. Post-traumatic stress disorder. Psychogenic fugue. Amnesia for criminal offence

Persistent (amnestic syndrome)

Korsakov syndrome. Herpes encephalitis. Posterior cerebral artery and .thalamic strokes. Head injury

Clinical features

The cardinal feature is a profound deficit in episodic memory. The full clinical picture is striking. There is disorientation for time, loss of autobiographical information (often extending back for many years), severe anterograde amnesia for verbal and visual material, and lack of insight into the amnesia. Events are recalled immediately after they occur, but forgotten a few minutes later. Thus the digit span, which tests the short-term memory store, is typically normal. New learning is grossly defective, but retrograde memory is variably preserved .and shows a temporal gradient, with older memories being better preserved

Other cognitive functions are relatively intact, although some emotional blunting and inertia are often observed. The other classic feature, seen particularly in Korsakov syndrome, is confabulation, in which gaps in memory are filled by a vivid and detailed but wholly fictitious account of recent activities, which the patient believes to be true. The confabulating patient is .often highly suggestible

Aetiology and pathology

- Amnesia results from lesions in the medial thalamus, other midline diencephalic structures, or medial temporal lobes (hippocampus and adjacent temporal cortex). Cases due to damage in the medial temporal lobe typically produce the ‘purest’ amnesia, with little in the way of disorientation or confabulation; these features are characteristic of thalamic and diencephalic .lesions

Korsakov syndrome

- The commonest cause of amnesic disorder is Korsakov syndrome, named after the Russian neuropsychiatrist who described it in 1889. The alternative term, **Wernicke–Korsakov syndrome**, was proposed by Victor et al. (1971), because the syndrome often follows an acute neurological syndrome called Wernicke's encephalopathy, consisting of delirium, ataxia, pupillary abnormalities, ophthalmoplegia, nystagmus, and a peripheral neuropathy. Korsakov syndrome is usually caused by thiamine deficiency, secondary to alcohol abuse, although it occasionally results from other causes, such as .hyperemesis gravidarum and severe malnutrition

- The classic neuropathological findings are neuronal loss, gliosis, and microhaemorrhages in the periaqueductal and paraventricular grey matter, the mammillary bodies, and the anterior and mediodorsal thalamus. Other causes of amnesic syndrome include tumours and infarcts in the medial .thalamus (diencephalic amnesia) and encephalitis

Investigations and Management

Alertness to the possibility of amnesic syndrome is essential; the patient may not fit the stereotype of chronic alcohol misuse associated with Korsakov .syndrome, and this is a potentially reversible condition

Useful findings from investigations include a reduced red cell transketolase level, which is a marker of thiamine deficiency, and an increased MRI signal in midline structures. In practice, Korsakov syndrome should be assumed to be the cause of amnesic syndrome until another aetiology can be demonstrated, and should be treated urgently with thiamine without awaiting the results of .investigations

Thiamine is given parenterally in an acute presentation, together with rehydration, general nutritional support, and treatment of supervening alcohol withdrawal. Thiamine replacement should always precede administration of intravenous glucose-containing solutions. Close liaison with physicians and .neurologists is important

Apart from thiamine, there are no interventions of proven efficacy for amnesic disorders. In the longer term, amnesic syndrome may require substantial rehabilitation and support, as the condition markedly impairs normal activities .and ability to provide self-care

Course and prognosis

- In the series of Victor et al. (1971), consisting of 245 patients with Wernicke–Korsakov syndrome, 96% of the patients presented with Wernicke's encephalopathy. Mortality was 17% in the acute stage, and 84% of the survivors developed a typical amnesic syndrome. There was no improvement in 50% of cases, complete recovery in 25%, and partial .recovery in the remainder