

Delirium

Delirium is a qualitative disorder of consciousness^[1] characterized by a sudden change in behavior, attention deficit and a fluctuating course. It is very common occurring in **10-30 %** of hospitalized patients, more often in those older than 65 years. It is often a neglected diagnosis, mainly because it is not considered or thought of. The diversity of the clinical picture has given delirium many different names, which has caused confusion in understanding the term, for example *acute brain failure, acute cerebral syndrome, acute cerebral insufficiency, acute state of confusion, acute organic syndrome, exogenous psychosis, metabolic encephalopathy, organic psychosis, toxic encephalopathy, toxic psychosis* ..^[2]

Etiology and differential diagnosis

Delirium is a syndrome that can have many causes, and therefore the differential diagnosis is very broad. The most common causes of delirium in elderly patients include **metabolic disorders** (e.g. mainly pneumonia and urinary tract infections, sepsis), **strokes** (delirium as a non-specific consequence) and **medication** (anticholinergic and narcotic agents). The most common cause of delirium **in young people** is the abuse of drugs and alcohol excess.^[2]

MAIN CAUSES OF DELIRIUM

Metabolic disorders	Hepatic encephalopathy, uremia, hypoglycemia, hypercalcemia, Hypomagnesemia/Hypermagnesemia, other disbalances in electrolytes, acidosis, hyperosmolar coma, endocrinopathies (thyroidal, parathyroidal, hypophysal), porphyrias, vitamin deficiencies (thiamin, nicotininc acid, folic acid), toxic and industrial exposure (carbon monoxide, organic solvents, copper, heavy metals) ^[2]
Medications and addictive substances	Overdose syndrome (benzodiazepines, barbiturates, alcohol), amphetamine, cocaine, nicotin, caffeine, phencyclidine, halucinogens, inhalants, narcotics, antiparkinsonics, sedatives, steroids, ^[2] lithium, antiarythmics, digitalis, opiates, antihypertensive agents ^[1]
Infections	Meningitis, encephalitis, cerebral abscess, neurosyphilis, Lyme neuroborreliosis, cerebritis, systemic infections with sepsis ^[2]
Neurologic	Ikterus, epilepsy, head trauma, hypertensive encephalopathy, brain tumors, migraine ^[2]
Perioperative	Specific surgeries (heart, orthopedic or opthtalmologic!), consequence of anesthesia or given medication, hypoxia and anemia. ^[2]
Other	Brain vasculitides, hyperviscose syndrome, trauma, dehydration, sensoric deprivation, ^[2] postoperative states, sleep deprivation, psychosocial stressors, cardiovascular disease (arrhythmia, heart failure, AMI) ^[1]

The most significant **risk factors** include **higher age**, preexisting cognitive dysfunction (mainly **dementia**), **sleep deprivation**, **immobilization**, **disorders of vision or hearing** and **dehydration**.^[2] Other risk factors include males, polymorbidities, history of delirium, CMP, neurologic disease, gait disturbances, falls, chronic liver or kidney failure.^[1]

Patophysiology

Patophysiology of delirium **is not fully understood**. Because several structures such as **ARAS** are responsible, **polymodal association cortex** and their **connection with the limbic cortex and thalamem**, delirium **can be the consequence of disorders of these structures**.

The second explanation explains the role of **neurotransmitters**, specifically **dysbalances between cholinergic and dopaminergic systems**. **Anticholinergics** and the consequent decrease in acetylcholine in the synaptic cleft can induce clinically and in the EEG delirium. In contrast inhibitors of acetylcholinesterase (donepezil, rivastigmin) increase concentration of acetylcholine in the synaptic cleft. They are used for example in the treatment of Alzheimers disease, in which the can improve the clinical course through their effect on attention. **Dopamine** appears to function reciprocally way to acetylcholinu, and therefore its administration in the form L-DOPA, or other jiných antiparkinsonics, can cause delirium. **Hypoglycemia, hypoxia, and other metabolic disturbances** can disrupt acetylcholinergic transmission and cause delirium.^[2]

Clinical presentation

Delirium is characterized by a reduced ability to concentrate and maintain attention, with a sudden onset and fluctuating course throughout the day .^[2]

3 basic **types of deliria according to the psychomotoric activity are distinguished**:

- *hyperactive* – agitation, restlessness, hallucinations, delusions, hypervigilance,
- *hypoactive (stuporous)* – low spontaneity, sedation, lethargy, latency of responses, reduction of movements,
- *combines* – alteration and combination^[1] .

Delirium usually develops rapidly within a few hours and usually resolves within a few days, followed by islet amnesia. Its course is **fluctuating**, tumultuous, **with a variable accompanying psychopathology**.^[1]

- altered consciousness (confused chaotic used to mental activity),
- disorientation,
- thinking is incoherent, fragmented, with paranoid assumptions to delusions, defective decision making, dream-like imagination,
- increased or decreased psychomotoric activity (restlessness, anxiety, excitement, agitation, aimless activity, bradypsychism),
- disorders of perception (most commonly the patient is unable to catch things that are happening around, in some cases hallucinations, mainly visual, tactile, less commonly acoustic),
- sleep-wake cycle disorders,
- hypoprosia,
- memory disorders especially short-term,
- increased suggestibility,
- other cognitive, behavioral or emotional abnormalities (e.g. disorders of writing) may be present

Diagnosis

Diagnosis of delirium occurs in 2 phases. In the first phase, *patient history, methods investigating attention* and a *diagnostic scale for delirium*

determine **whether it is delirium that is really present**. The **second phase, is focused on finding what is causing the delirium** with the help of *patient history, physical examination* or *laboratory examination*.^[2]

Patient history ^[2]

Is focused on the symptoms of delirium (see clinical presentation). Due to the nature of the syndrome, it is sometimes necessary to interview the patient's closest relatives. The aim is to find **changes in the patients behavior**, insomnia, patient's diseases, medication, recent trauma etc. In addition, numerous risk factors for delirium should be considered (see "Etiology and differential diagnosis")^[2]

Examination of mental status^[2]

Is a major part of the neurologic examination required for assessment of the patients **alertness and attention**. In the clinical practice a number of simple testing methods is used. Examples include:

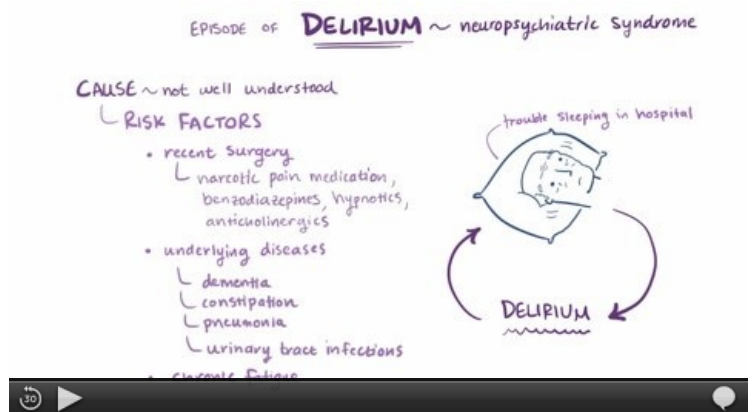
- *Digit span test*, in which the examiner says numbers in a monotone voice in one-second intervals and the patient is asked to repeat the same series of numbers after. If the patients say seven (plus minus two) numbers, it is a normal finding.
- *Serial reversal test*, in which the patient for example repeats, wholes set of numbers backwards, spells a word backwards or subtracts a number from a basic value (for example 7 from 100, this means. 93. 86, 79, etc.)
- *Vigilance A test*. In this test the patient lets the examiner know whenever they hear the letter A in the letters read out by the examiner.^[2]

Diagnostic scales and criteria for delirium^[2]

Routine examination of mental status **cannot distinguish delirium from dementia** and other cognitive deficits, thus specific criteria have been developed for the diagnosis of delirium.

- *The Confusion Assessment Method (CAM)* consists of 9 questions, that assess, whether there is a fluctuating course, inattention and whether either disorganized thinking or an altered degree of consciousness is present. This method has been modified (**ICUCAM**) for the diagnosis of patients in the ICU.
- *The delirium rating scale-revised-98 (DRS-R-98)* is a method, which enables the differentiation of delirium from dementia, depression and schizophrenia.
- *The memorial delirium assessment scale (MDAS)* is a scale which enables the determination of the severity of delirium in chronically treated patients.
- *The delirium symptom interview* may be helpful in the diagnosis of delirium.

Physical examination^[2]



Definition, pathogenesis, symptoms, complications, treatment.

Delirium may have some nonspecific symptoms such as high frequency postural tremor (8–10 Hz), asterixis, choreaform movements, gait instability etc. Behavioral changes such as agitation or psychomotor retardation, apathy, cataplexy appear.

Laboratory examination^[2]

Delirium is practically always accompanied with changes in the EEG. Disorganization of common brain rhythms and there decrease in rate occurs. Slower activity on EEG occurs in both clinical forms of delirium, hypoactive and hyperactive.

Another necessary part of the laboratory examination is the **examination** of *blood counts, glucose, electrolytes, urea a creatinin, transaminases, levels of ammonia, examination of thyroid gland function, blood gases, chest x-ray, ECG, examination of urine including tests for the presence of addictive substances.*

Examination of **evoked potentials** often shows extended latency times.

In case of doubts in diagnosis, a **lumbar puncture** can be performed. A puncture should be preceded by **CT** or **MRI** imaging, especially in case of a neurologic finding, or a suspicion of intracranial hypertension, intracranial expansion or head trauma.

Therapy^[2]

Therapy of delirium includes the following steps:

1. **Find the cause and eliminate it.**
2. **Symptomatic therapy focused on attention and correction of elektrolyte dysbalances.** In general, the use of medication should be restricted in confused patients, in whom the medication may worsen the condition. Adjusting the sleep-wake cycle can help many patients, especially in having higher quality night sleep.

Hypnotics can help. If the patient's behavior is dangerous, antipsychotics, especially atypical ones (**olanzapin, quetiapin, risperidon**) may be used. Benzodiazepines and anticholinergics should be avoided.

1. **Influencing the patient's environment.** The patient should not be deprived by lack of stimuli, but also not overstimulated by their excess. Noise in the room should be reduced, possibly also the number of unrelated visitors. On the contrary, the presence of a radio or television, glasses to improve eyesight, aids to improve hearing, allowing the patient to move, etc. can be beneficial.
2. **Correct communication and support** is very important for these patients; everything should be appropriately explained and patients should be supported emotionally, for example by frequent visits by their family.

Attention should be paid to risk factors, ie. mainly dehydration, cognitive dysfunction, sleep deprivations, immobilization, vision and hearing disorders.

Prognosis

In most cases **prognosis is good**. Delirium usually lasts from 2 days to 2 weeks, in elderly even longer. In one third of elderly patients partial delirium remains, even after being discharged from the hospital. This partial delirium meets only some of the criteria for delirium, and only one-fifth of post-delirium patients return to pre-delirium status. In addition these patients frequently limit their daily activities at home, have increased risk of falling, pressure ulcers and consequently mortality. ^[2]

Links

Related articles

- Consciousness and its disorders
- Delirium tremens

References

1. HANUŠ, Herbert. *Obecná psychiatrie*. 1. edition. Praha : Karolinum, 1997. ISBN 80-7184-382-2.
2. AUTHORS, Edited by Walter G. Bradley... [et al.]; with 120 contributing – BRADLEY, W. G. (Walter George). *Neurology in clinical practice. Vol. 2, The neurological disorders*. 4. edition. Philadelphia : Butterworth-Heinemann, c2004. ISBN 0-7506-7469-5.

Literature used

- AUTHORS, Edited by Walter G. Bradley... [et al.]; with 120 contributing – BRADLEY, W. G. (Walter George). *Neurology in clinical practice. Vol. 2, The neurological disorders*. 4. edition. Philadelphia : Butterworth-Heinemann, c2004. ISBN 0-7506-7469-5.

- HANUŠ, Herbert, et al. *Obecná psychiatrie*. 1. edition. Praha : Karolinum, 1997. ISBN 80-7184-382-2.