



Delirium in somatic practice on the example of a clinical case

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Delirium

It is an organic psychosyndrome characterized by an acute onset and variability, in which the main symptomatology consists of cognitive disorders, observed at any age.

Delirium often occurs with acute physical illnesses. The risk of developing delirium increases with the initial chronic pathology of the brain, chronic intoxication.

Symptomatology consists of the signs of confused consciousness and its decline, disorientation and disorganized thinking, inability to distinguish dreams and illusions from true hallucinations, confusing utterances, disturbances in the sleep-wake cycle, mnestic disorders, increased or decreased psychomotor activity.

Neurological causes

- Cerebrovascular disorders: ischemic stroke, hemorrhagic stroke, transient ischemic attack
- Trauma: traumatic brain injury and subdural hematoma
- Migraine: confusional migraine, where the consciousness is altered
- Seizure: including postictal state and nonconvulsive status epilepticus
- *Tumor*: primary or metastatic brain tumor, meningeal carcinomatosis
- *Inflammation or infection*: CNS vasculitis, meningitis, encephalitis, meningoencephalitis

Non-neurological causes

- *Endocrine disorders*: Cushing syndrome, adrenal insufficiency, pituitary insufficiency, thyroid disorders
- *Hematologic disorders*: thrombocytopenia, polycythemia, leukemic blast cell crisis, hyperviscosity syndrome
- **Drugs:** anticholinergics, antihistamines, antiemetics, antihypertensives, benzodiazepines, corticosteroids, dopamine agonists, muscle relaxants, recreational drugs, antispasmodics, digoxin, hypnotics, opioids, sedatives, tricyclic antidepressants
- Infection: sepsis, systemic infection, urinary tract infection, pneumonia, fever
- Injury: burns, fat embolism, hypothermia, heatstroke, electrical injury

Non-neurological causes

- *Metabolic disorders*: fluid and electrolyte disorders such as hypernatremia, hyponatremia, hypocalcemia, hypercalcemia, dehydration, hyperthermia, hypoglycemia, hyperosmolality, hypoxia.
- Vascular and circulatory abnormality: cardiac arrhythmia, heart failure, shock, hypoperfusion states, anemia
- Vitamin deficiency: Vitamin B12 deficiency, thiamine deficiency
- *Substance withdrawal*: Withdrawal from alcohol, benzodiazepines, opioids, or barbiturates

Other causes

- Liver failure
- Long ICU stays
- Sensory deprivation
- Sleep deprivation
- Change of environment
- Postoperative states
- Urinary retention
- Mental disorders

Diagnostic criteria of the Delirium

- Disturbance in attention (reduced ability to direct, focus, sustain, and shift attention) and awareness.
- Change in cognition (memory deficit, disorientation, language disturbance, perceptual disturbance) that is not better accounted for by a preexisting, established, or evolving dementia.
- The disturbance develops over a short period (usually hours to days) and tends to fluctuate during the course of the day.
- There is evidence from the history, physical examination, or laboratory findings that the disturbance is caused by a direct physiologic consequence of a general medical condition, an intoxicating substance, medication use, or more than one cause.

Assessment instruments of the Delirium

- Confusion Assessment Method (CAM)
- Delirium Symptom Interview (DSI)
- Confusion Assessment Method for the Intensive Care Unit (CAM-ICU)
- Intensive Care Delirium Screening Checklist (ICDSC)
- Delirium Detection Scale (DDS)
- Memorial Delirium Assessment Scale (MDAS)

DD of the Delirium

- Complete blood cell count: Infection; Anemia
- Electrolytes: Hypo- und Hypernatremia
- Glucose: Hypoglycemia; Diabetic ketoacidosis; Hyperosmolar nonketotic states
- Renal and liver function tests: Liver failure; Renal failure
- Thyroid function test: Hypothyroidism
- Urine analysis: Urinary tract infection
- Microbiological tests to identify the causative agent of the Infection
- Drug-screening
- Magnetic resonance imaging (MRI) of the head: Strokes; Hemorrhages; Structural lesions
- Lumbar punction: CNS Infection

Prognosis

- In patients who are admitted with delirium, mortality rates are 10-26%.
- Patients who develop delirium during hospitalization have a mortality rate of 22-76% and a high rate of death during the months following discharge.
- In patients who are elderly and patients in the postoperative period, delirium may result in a prolonged hospital stay, increased complications, increased cost, and long-term disability.

Our patient

- Name: S. M.
- Sex: male
- Age: 84 years
- Location: city citizen
- Occupation: former school director

Complaints

- General weakness
- Inverted perception of the world (upside down) but maintained right-left perception (all objects where they are should be)
- Disorientation
- Dyscoordination
- Panic und Anxiety



Anamnesis morbi

- patient was delivered to the ED by ambulance, as his neighbors called police because they haven't seen him around 2-3 days
- the disease started suddenly at night when the patient was going to the bed after watching TV
- these changes are followed by disorientation in space and coordination failure.
- patient was conscious and understood all, but could not move, call someone or reach the telephone for 3 days, so he had lied on his floor
- patient was oriented in time, his personality and place and he remembered everything

Anamnesis vitae

- widower since 2005
- he lives alone and he has son
- no known history of chronic illness;
- hereditary diseases are not identified
- allergic history is not burdened
- childhood infections no
- sexually transmitted diseases were denied
- smoker no, do not abuse alcohol

Objective examination

- Conciseness clear, state moderate severity, body position active, 38°C, SpO₂ -96%.
- Dental prosthesis of both jaws, swallow reflex is normal.
- Pale skin and mucosae. Thyroid: not enlarged, soft. Peripheral lymphatic nodes not enlarged.
- Musculoskeletal system no changes
- BR 16-17 / min
- Lung percussion: no clinically significant changes
- Lung auscultation: hard breathing
- Borders of the heart: without clinically significant changes.
- Heart auscultation: rrhythmic, heart tones muffled, no murmurs over all points of auscultation. HR 70 bts/min
- Pulse rrhythmic, normal, 70 bts/min. BP- 120/70 mm Hg.
- Abdomen: normal size, symmetric, unpainful.
- Liver: not enlarged, moderate density, no pain during palpation in right hypochondrium
- Spleen: not palpated
- Pasternatsky symptom positive from both sides
- Edemas: absent
- Neurological status without clinically significant changes.

Plan of survey

- Complete blood count, urinalysis
- Blood analysis for urogenital infections, tests for HIV, syphilis
- Biochemical panel
- Chest X-ray
- ECG
- CT-Brain + MRI Brain
- Angiography of cerebral vessels
- Consultations of urologist and neurologist

Complete blood count

	21.09.17	22.09.17	23.09.17	31.09.17	Normal range
Hemoglobin, g/L	15,9	12,7	13,8	13,8	13,5 – 17,5 mg/dl
Erythrocytes × 10 ¹² /l	5,4	4,4	4,8	4,7	4.5-5.9
Leukocytes × 10 ⁹ /L	12,5	12,7	13,1	4,7	3.5 - 8.5
Platelets \times 10 9 /L	231	159	294	285	160-370
Ht	0,45	0,37	0,40	0,43	0,40-0,53 1/1
MCV	29,2	28,9	28,9	29,2	80-96 fl
MCH (HbE)	35,3	33,2	34,1	33,5	28,0-33,0 pg
MCHC	12,8	12,8	12,6	12,8	31,5-36,0 g/dl
Normoblasts× 10 ⁹ /L	0,00	0,00	0,00	0,00	-

Conclusion: leukocytosis, exicosis

Urine analysis

Options	Results	Normal range
рН	6,0	5,0-7,0
Protein, g / l	Not detected	to 0.033
Glucose	Not detected	absent
Leucocytes, cells/hpf	0	6-8
Epithelium, cells/hpf	3	≤15-20
Bacteria	positiv	absent

Conclusion: UTI

Microbiological analysis Urin Culture

	Penicillin	Oxacillin	Ampicillin	Ampicillin+ Sulbactam	Meropenem	Cefazolin	Cefalexin	Tetracyclin	Sulfamethoxazol	Erythromycin	Doxycyclin	Ciprofloxacin	Vancomycin
E. coli	R	S	R	S	S	S	S	S	S	S	S	S	S

Biochemical test (1)

	Results	Normal range
INR	1,14	
PTT sec	30	26-36 sec
Quick, %	80	70-120%
LDH	280	< 250mg/dl
HDL-Cholesterol	30	> 35 mg/dl
LDL-Cholesterol	163	< 130 mg/dl
LDL-HDL Ratio	5,45	< 3
TAG	134	< 150 mg/dl
Cholesterol	204	< 200mg/dl
Uric acid	5,6	3,6-7,0 mg/dl
Folic acid	11,9	$3,1-20,5 \mu g/l$
Active Vit B12	> 256,0	> 50,0 pmol/l

Conclusion: dyslipidemia

Biochemical test (2)

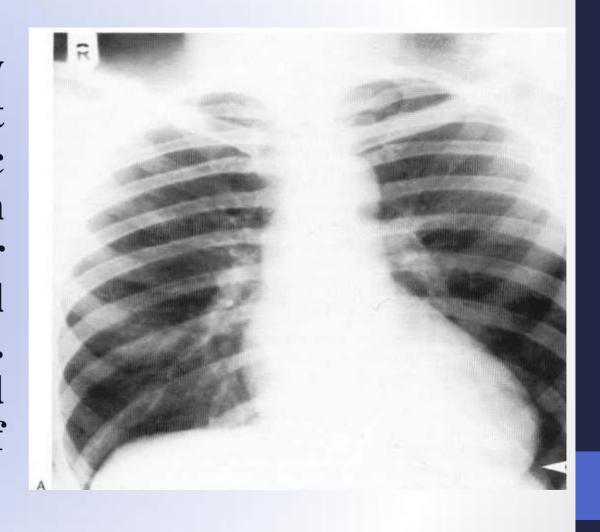
	21.09.17	22.09.17	23.09.17	31.09.17	Normal range
C-reactive protein	112,6	61,5	18,0	1,7	<5, mg/l
Total protein (pl)		58,4	66,1		66-83,0 g/l
Albumin			38,1		40,2-47,6 g/l
a1-globuline			3,7		2,1-3,5 g/l
γ-globuline			9,6		8,0-13,5 g/l
ALT	80	54	346	60	<50 U/l
AST	-	-	176	36	<50 U/l
AP (alc.phosp)		46	173	92	40-130 U/l
LDH		255	280		<250 U/I
γ-GT	28	22	293	72	<60 U/l
Total bilirubin			0,6	0,7	<1,2, mg/l

Biochemical test (3)

	21.09.17	22.09.17	23.09.17	31.09.17	Normal range
CK	6201	4100	190		<190 U/l
CK-MB	179	123			<25 U/I
Troponin I	34	33			<34 ng/l
Sodium	135	141	140	142	135-145 mmol/l
Potassium	3,8	3,45	4,23	4,27	3,40-4,50 mmol/l
Calcium		2,10	2,32		2,15-2,58 mmol/l
Lipase	31				8-78 U/I
Urea				27	18-55 mg/dl
Creatinine, mg/dl	0,8	0,8	1,0	1,0	0,7-1,2 mg/dl
GFR (CKD-EPI)	81	82	73	63	> 60 ml/min
Glucose	105				74-109 mg/dl
TSH	1,43		1,73		0,27-4,20 mU/l

Chest X-ray 21.09.17

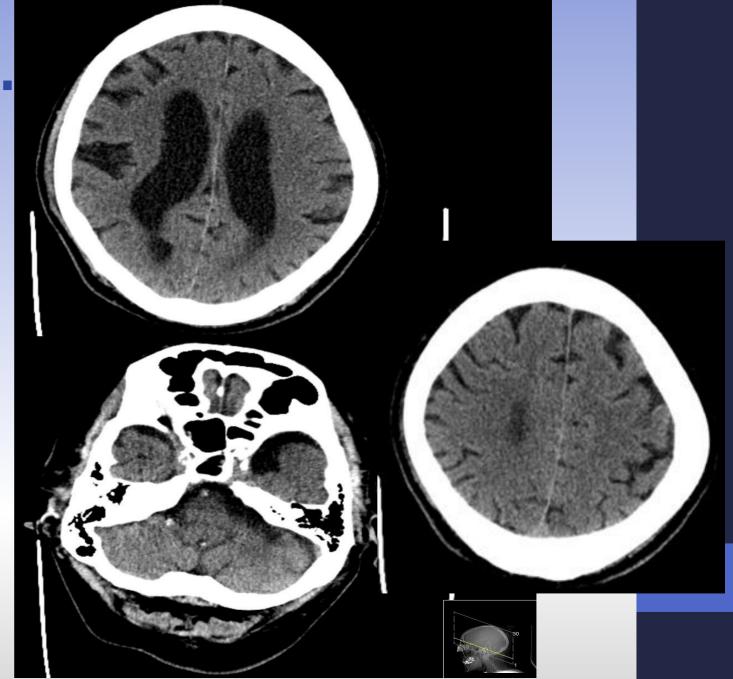
Heart shadow is changed by left ventricle enlargement without signs of cardiac insufficiency. Lungs are clean without infiltration atelectasis present. No pleural effusion seen, normal sinuses. Investigations are limited because of supine position of the patient



Brain CT Scan 22.09.

- symmetrical ventricle disposition, all structures are are good visualized.
- no signs of hemorrhage
- cortex area is good visualized without any pathological changes
- thalamus, gangli and cerebellum without pathological changes
- mild microangiopathy but no hemorrhage or ischemic focuses
- decreased brain volume

Conclusion: no clinically important changes were found



Diagnosis

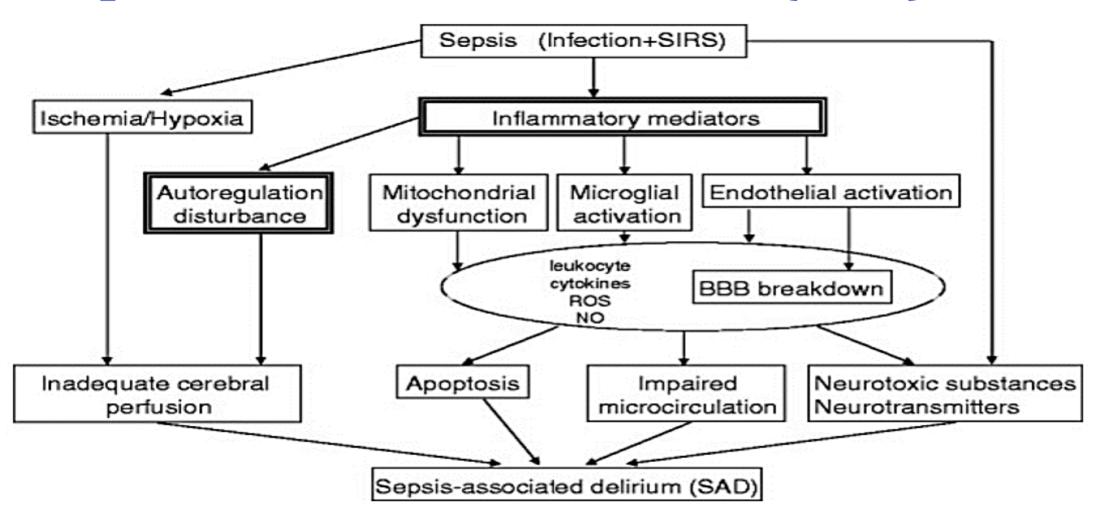
Urosepsis with Delirium and Exicosis

Treatment plan in the hospital

- Ampicillin+Sulbactam 3 g IV every 8 hours during 5 days
- Consumption of at least 2 liters of fluid per day
- Certroparin 3000 IE s.c. 1 time per day (Thrombosis prophylaxis)
- Melperon 5 ml per os 2-3 times per day if necessary

Against the background of the treatment, the patient's condition stabilized after 3 days, 7 days after the admission the patient was discharged under the supervision of his family doctor.

Sepsis-associated Delirium (SAD)



Delirium is defined as a "transient, usually reversible cause of cerebral dysfunction and manifests clinically with a wide range of neuropsychiatric abnormalities"

NCBLgov/BioMed Central

Recommendations

Treatment of urosepsis comprises four major aspects:

- early diagnosis;
- early goal-directed therapy including optimal pharmacodynamic exposure to antimicrobials both in the plasma and in the urinary tract;
- identification and control of the complicating factor in the urinary tract; and
- specific sepsis therapy.

Early adequate tissue oxygenation, adequate initial antibiotic therapy, and rapid identification and control of the septic focus in the urinary tract are critical steps in the successful management of a patient with urosepsis, which includes early imaging, and an optimal interdisciplinary approach encompassing emergency unit, urological and intensive-care medicine specialists.

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Sedatives are often used in the intensive care unit, and the use of benzodiazepines is related to the increased incidence of brain dysfunction, as assessed by symptoms of delirium. Dexmedetomidine, an alpha-2 agonist, has been associated with less delirium than continuous lorazepam and midazolam in critically ill patients (the MENDS trial).

Clinics (Sao Paulo). 2011 Oct; 66(10): 1825–1831.

Conclusion

The exact mechanism causing delirium is complex and involves the neurological impact arising out of the immune response causing "prolonged inflammation, brain cells activation, over expression of NO, dysfunction of intracellular metabolism and cell death" [Intensive Care Med. 2007;33:941–950]. However, to the best of our knowledge, only few case reports have ever noted delirium presenting as the only initial manifestation of urosepsis.

The brain may be affected in sepsis, similar to any other organ, but with many unique effects. Therefore, efforts should be made to understand the multiple clinical presentations and pathophysiology of SAE to target therapies toward the mechanistic pathways of the disorder rather than to merely control the symptoms of SAE.

