

Metabolic and toxic encephalopathies and neuropathies

Metabolic and toxic encephalopathies and neuropathies are secondary, acquired disorders that arise as a result of changes in the internal environment of the organism, for example as a result of some internal diseases ("metabolic encephalopathies").

Anoxic ischemic encephalopathy

Etiology: hypotension, cardiopulmonary insufficiency

Causes: myocardial infarction, cardiac arrest, hemorrhagic shock, asphyxiation, CO intoxication

- hypoxia mostly affects the cortex and subcortical structures

Clinical picture:

- hypoxia → restlessness, anxiety, disorders of attention and motor coordination
- anoxia → loss of consciousness, generalized convulsions (tonic)
- prolonged anoxia → coma (mydriasis, extinguished photoreaction) to brain death

Treatment: nootropics

Posthypoxic (postanoxic) syndrome: persistent coma or other disorders of consciousness, apallic syndrome, dementia, parkinsonian syndrome, cerebellar syndrome, choreoathetosis, Korsak amnestic syndrome, intentional or action myoclonus^[1]

Hypercapnic encephalopathy

Etiology: increased partial pressure of CO₂ → chronic emphysema, respiratory insufficiency, chronic respiratory acidosis

Clinical picture: headaches, somnolence, psychomotor retardation, asterixis, possibly. confusion to coma

Hypoglycemic encephalopathy

Etiology: excessive dose or increased secretion of insulin

Clinical picture: nervousness, headaches, palpitations, anxiety, sweating, tremors, motor restlessness, muscle spasms, myoclonus, hyperreflexia, ev. confusion to coma, epileptiform convulsions, focal neurological symptoms (convulsions, paresis)

Treatment: glucose i.v.

Hyperglycemic coma

Etiology: ketoacidosis, hyperosmolality, hypoxemia

Hepatic encephalopathy

Etiology: chronic liver insufficiency with portacaval shunt

Clinical picture: confusion, convulsions, asterixis, metabolic tremor, spastic pyramidal phenomena, ataxia standing and walking, choreoathetosis

Treatment: lactulose

Uremic encephalopathy

Clinical picture: fatigue, apathy, increased irritability, confusion, muscle twitches to myoclonus, metabolic tremor, asterixis, convulsions, epileptiform seizures

Endocrine encephalopathy

Causes:

- administration of ACTH or corticosteroids
- psychotic clinical picture
- hypothyroidism
- confusion, mania or depression, muscle weakness

Diabetic polyneuropathy

Etiology: chronic hyperglycemia

Alcoholic polyneuropathy

Etiology: deficiency, nutritional and vitamin deficiency (especially thiamine), direct toxic effect of alcohol on peripheral nerves

Clinical picture: symmetrical mixed sensory and motor involvement acral on the lower limbs, dysautonomia, hyperhidrosis, axonal type neuropathy

Treatment: abstinence, adequate nutrition, vitamins B1 and B12

Toxic polyneuropathies

Etiology: organophosphates, carbon disulfide, lead, mercury, hexacarbons, isoniazid (therefore administer pyridoxine as a preventive measure), cytostatics (vincristine - therefore we administer with glutamic acid), amiodarone, statins

Links

Related Articles

- Toxoinfectious encephalopathy
- Encephalopathy
- Hyperglycemic Coma
- Heavy metal poisoning
- Alcohol intoxication
- Anoxia

References

1. {{#switch: book |book = *Incomplete publication citation*. AMBLER, Zdeněk. *Fundamentals of Neurology*. Prague : Galen, 2006. pp. 253. 978-80-7262-438-6. |collection = *Incomplete citation of contribution in proceedings*. AMBLER, Zdeněk. *Fundamentals of Neurology*. Prague : Galen, 2006. pp. 253. {{ #if: 80-7262-433-4 |978-80-7262-438-6} } |article = *Incomplete article citation*. AMBLER, Zdeněk. 2006, year 2006, pp. 253, |web = *Incomplete site citation*. AMBLER, Zdeněk. Galen, ©2006. |cd = *Incomplete carrier citation*. AMBLER, Zdeněk. Galen, ©2006. |db = *Incomplete database citation*. Galen, ©2006. |corporate_literature = AMBLER, Zdeněk. *Fundamentals of Neurology*. Prague : Galen, 2006. 978-80-7262-438-6}, s. 253.

Literature

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