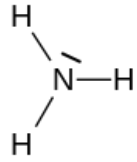
Hepatic Encephalopathy

Hepatic encephalopathy is a complication of chronic hepatic diseases of various etiology. Cirrhosis and portal hypertension are very frequent findings. However, there should be some **trigger**, which causes acute hepatic encephalopathy. Usually it is a process leading to increased levels of **ammonia** – main agent of brain damage.

Hepatic encephalopathy is usually acute or subacute state. However, sometimes is the brain damage reversible and the seizures recur (so called chronic hepatic encephalopathy). Unfortunatelly, they still progress and after months or years they lead to the irreversible damage.

Etiology



Ammonia structure

Reduced liver function (due to chronic hepatic disease) is a common finding, but are always other factors, which tilt the balance:

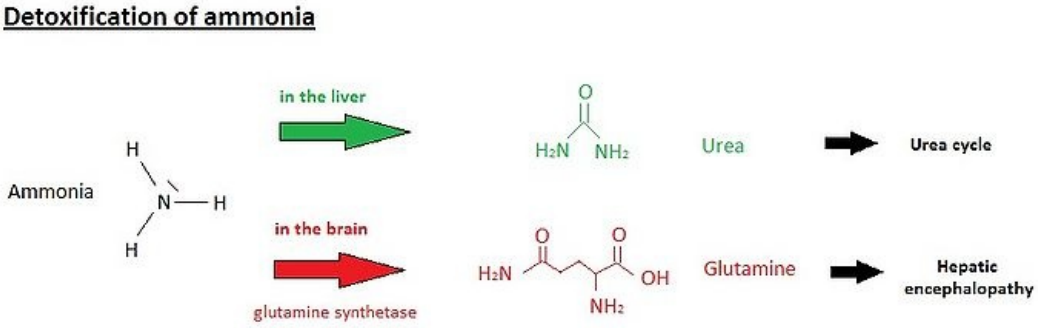
- bleeding in GIT
- infection
- high protein intake
- hypokalaemia
- drug involvement
- hypoxia
- constipation

Pathogenesis

There are few steps, which leads to hepatic encephalopathy. The first one is a **reduced detoxification ability of liver**, which is connected with high level of ammonia. Ammonia is normally transformed into urea and its elimination continues in urea cycle. If there is a cirrhosis, this metabolic way is defective.

The second problem are **changes in blood flow**. Blood from GIT then pass through the blood faults and don´t get to the liver at all.

Finally, **haematoencephalic barrier** of people who suffer from chronic hepatic disease is more permeable, so ammonia gets into the brain much easier. In the brain is ammonia transformed by enzyme called *glutamine-synthetase* into **glutamine**. Glutamine influences neurotransmitters – especially GABA and dopamine. The metabolism of brain is significantly reduced. If these changes occur fast then there is a risk of of **cerebral edema**. The **main affected areas** are basal ganglia (especially globus pallidum) and cerebellum.



Signs and Symptoms

Signs of chronic hepatic disease:

- icterus
- anorexia and fatigue
- ascites
- splenomegaly

Neurological changes:

- mind disorder
- changes in behavior
- flapping tremor
- drowsiness

- character changes
- disorders of consciousness – delirium or coma
- in late progression – dementia, rigidity, ataxia, chorea, hypokinesia

Diagnosis

We monitor levels of ammonia **in the blood**. From the auxiliary examination, just **EEG** with diffuse slow-motion delta waves.

Therapy

The most common therapy of hepatic encephalopathy is a combination of **lactulose**, which accelerates the passage of intestinal contents, and **antibiotics** to modify the intestinal microflora (reduce ammonia producing bacteria). Patient should also be on **low-protein diet** and drink no alcohol.

Links

Related Articles

- Cirrhosis
- Portal Hypertension
- Encephalopathy
- Urea Cycle
- Urea
- Neurotransmitters
- GABA
- Dopamine
- Brain Edema
- Glutamine
- Icterus
- Ascites
- Splenomegaly
- EEG

Bibliography

- KLENER, Pavel, et al. *Vnitřní lékařství.* 4th edition. 2011. ISBN 978-80-7262-705-9.
- BEDNAŘÍK, AMBLER, RŮŽIČKA,, et al. *Klinická neurologie : část speciální II.* 1st edition. 2010. ISBN 978-80-7387-389-9.