Diagnosis and treatment of hepatic encephalopathy

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Background. Hepatic encephalopathy (HE) is a neuropsychiatric complication of liver cirrhosis which symptoms may vary from imperceptible to severe ones. In recent years, there have been some changes of fundamental hepatic encephalopathy pathogenesis and treatment. The early HE on the stage of minimal hepatic encephalopathy (MHE) is rarely diagnosed and treatable condition worldwide and in Lithuania as well, however, this HE stage is responsible for the cognitive disorders which impair the quality of life of cirrhotic patients. According to resent data, MHE can be diagnosed in up to 70% of cirrhotic patients.

Aim. To evaluate new diagnostic and treatment strategies for HE and especially MHE for further use in clinical practice to cure the quality of life of cirrhotic patients and prevent clinical manifestation of HE.

Methods and materials. This article is based on relevant original publications and reviews in English (1991–2012) that were retrieved by a selective key word based search in the Medline and PubMed databases.

Results. It is recommended not to decrease an amount of proteins in food and consume products containing more branched-chain amino acids. Non-absorbable disaccharides (lactulose) are still the drugs of the first choice, though recent data show significant concerns about their effectiveness. Rifaximin is increasingly used all over the world for hepatic encephalopathy treatment. Other drugs for HE treatment are of secondary importance. Lactulose, probiotics are recommended for minimal hepatic encephalopathy treatment.

Diagnosis, especially of minimal hepatic encephalopathy, remains complicated. There are no reliable and validated blood indicators to establish minimal hepatic encephalopathy diagnosis, and to follow up treatment efficacy. Psychometric and neurophysiologic methods, visualisation methods are used more in scientific researches. Computerized methods, such as inhibitory control and critical flicker frequency tests, are also promising.

Conclusions. Further studies are necessary to design proper algorithms of hepatic encephalopathy diagnostic and treatment.

Key words: hepatic encephalopathy, minimal hepatic encephalopathy, diagnosis, treatment
INTRODUCTION

Hepatic encephalopathy (HE) is a neuropsychiatric complication of liver cirrhosis which symptoms may vary from imperceptible to severe, invaliding, and even lethal ones. Minimal hepatic encephalopathy (MHE) is also important because it impairs patients' cognitive functions and quality of life (1, 2).

Clinically manifested hepatic encephalopathy (HE) diagnosis is based on the clinical state of the patient. To evaluate its severity, West-Haven's criteria (Table) accepted in 1977 and the Glasgow Coma Scale are most widely used. Diagnostics of 1–2 degree HE according to these criteria is very subjective. West-Haven's criteria may be interpreted differently by different researchers and may have negative influence on the results of multicenter studies.

MHE has no symptoms and may be diagnosed only by using neuropsychometric or neurophysiological methods.

Despite HE in our country is diagnosed using West-Haven criteria, the initial stage of HE, MHE, is neither diagnosed nor treated worldwide and in Lithuania as well, although it is found in more than 70% of cirrhotic patients and appreciably impairs the quality of life of such subjects. That is why we conducted a review of resent achievements in diagnosis and treatment tools for both HE and MHE with further implication of them in clinical practice for the cure of the quality of life of cirrhotic patients and for prevention of clinical manifestation of HE.

There are a few types of neuropsychometric tests – “paper-pencil” tests, which do not need any special provisions and computerized tests. They help to evaluate patient's concentration, reaction speed, ability to suspend a response to stimulus and visuo-spatial functioning.

The “golden standard” for diagnosis of MHE is the PHES (Psychometric Hepatic Encephalopathy Score) battery (3). It is a set of subtests such as the number connection test A, number connection test B, digit symbol test, line tracing test, serial dotting test. MHE is proved if deviation from the normative standard is >2 SD in two or more tests. PHES tests battery is validated for populations in Germany, Italy and Spain. In the USA this battery is not validated, and another set of psychometric tests, namely, RBANS (Repeatable Battery for the Assessment of Neurological Status), is used. It takes as long as PHES – about 20–25 min. The assessment of results is complicated in both sets. Results depend on the education and age of the patient.

One of the most popular computerized psychometric tests is the Inhibition Control Test, where

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria</th>
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<tbody>
<tr>
<td>Grade 0</td>
<td>Lack of detectable changes in a personality or behaviour</td>
</tr>
<tr>
<td></td>
<td>No asterix</td>
</tr>
<tr>
<td>Grade 1</td>
<td>Trivial lack of awareness</td>
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<tr>
<td></td>
<td>Euphoria or anxiety</td>
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<td></td>
<td>Shortened attention span</td>
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<tr>
<td></td>
<td>Impaired performance of addition</td>
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<tr>
<td></td>
<td>Asterixis may be present</td>
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<tr>
<td>Grade 2</td>
<td>Lethargy or apathy</td>
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<tr>
<td></td>
<td>Minimal disorientation for time or place</td>
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<td></td>
<td>Inappropriate behaviour</td>
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<td></td>
<td>Subtle personality changes</td>
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<td></td>
<td>Inappropriate behaviour, slurred speech</td>
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<td></td>
<td>Impaired performance of subtraction</td>
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<tr>
<td>Grade 3</td>
<td>Somnolence to semi-stupor, but responsive to verbal stimuli</td>
</tr>
<tr>
<td></td>
<td>Confusion</td>
</tr>
<tr>
<td></td>
<td>Gross disorientation</td>
</tr>
<tr>
<td></td>
<td>Asterixis is usually absent</td>
</tr>
<tr>
<td>Grade 4</td>
<td>Coma (unresponsive to verbal or noxious stimuli)</td>
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concentration and ability to suppress response to stimulus is assessed (4). The patients follow changing letters on the computer screen and have to react when a certain sequence of letters appears, and ignore the combination of letters which is similar to it. If there are 5 mistakes from 40 attempts, MHE might be diagnosed. The test is sensitive (5, 6) and is commonly used in the USA.

Another computerized test, popular in Great Britain, is the CDR (Cognitive Drug Research) test. It helps to evaluate the ability to keep concentration, quality of short- and long-term memory and speed. CDR test has a high correlation with psychometric tests, though it is appropriate for MHE diagnostic, unfortunately, takes a long time.

Neurophysiologic exploratory methods of HE are also available. One of them is the critical flickering frequency test characterized by high sensitivity, specificity, and high correlation with psychometric tests. It is widely used in clinical trials. This test is based on the fact that retinal glial cells in patients with HE undergo similar changes (swelling, termed hepatic retinopathy) to those seen in cerebral glial cells. Patients watch the decreasing frequency light pulses and fix when they start to see them as discrete ones. MHE is diagnosed if a critical flicker frequency is below 39 Hz. The benefit of this test is that results are not influenced by patient’s sex, education, and operating skills. Only age can have a partial influence. A critical flickering frequency test is quick and cost effective, fairly sensitive and specific for MHE diagnostics (7–9).

Electroencephalogram (EEG) is not very specific for HE diagnosis. The most sensitive of all EEG is EEG with computerized data analysis. P300 induced potentials test may be valuable for MHE diagnostics, though both EEG and the latter test require specially prepared personnel and equipment, therefore cannot be used widely in ambulatory environment (10).

Neurovisualization tests are magnetic resonance imaging (MRI) and magnetic resonance spectroscopy (MRS). They are not valuable for MHE diagnostics and useful, maybe, only for treatment evaluation (e. g., medical treatment or after liver transplantation) (11–13).

Using MRI, T1 weighted imaging hyperintensive signals because of manganese accumulation in basal ganglions are seen not only for patients with liver cirrhosis, but also for patients with non cirrhotic portal hypertension. This intensity has no correlation with a degree of HE severity.

MRS shows intensification of glutamine and glutamate signals, and decreases intensity of myo-inositol and choline signals (because of homeostatic compensative mechanisms) (14). MRS does not help to diagnose MHE.

Other MRI methods, such as magnetization transfer ratio (MTR), fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI), improved diagnostics of cerebral oedema associated with HE. A regression of brain changes after liver transplantation was detected using these methods.

Laboratory tests are not very valuable for HE diagnostics yet. There is no correlation between concentration of ammonia in the blood and severity of C type HE, as against that in case of A type HE (15). Estimation of ammonia in the arterial blood is more precise for HE diagnosis than in venous one, though following of dynamic concentration changes is necessary. There are small studies showing usefulness of laboratory markers such as cGMP (16, 17), IL (IL-6, IL-18) (18) for diagnosis of MHE in cirrhotic patients, though more detailed clinical trials are necessary.

HE TREATMENT

The main aim of HE treatment is to eliminate all factors which had provoked it and to administer a proper empiric therapy. Following these principles, the condition of most patients improves within 24–48 hours of treatment initiation. If HE still persists, it is recommended to re-estimate the diagnosis of HE and to correct missed precipitating factors. If there is ascites, it is necessary to perform diagnostic paracentesis. A short course of antibiotics is recommended for severely affected patients with hepatic coma while waiting for bacterial culture growth results.

It is recommended to avoid sedatives, especially benzodiazepines. If the patient is very agitated, haloperidol might be prescribed.

Diet recommendations for patients with HE

In the twentieth century all patients with cirrhosis and HE were recommended to follow low protein diet. In recent years this point of view has cardinally changed.
For 20–60% of patients with liver diseases nutritional insufficiency was diagnosed (19). Particular attention was paid on protein insufficiency, which can occur in different stages of liver diseases, though more often in later ones. Insufficient nutrition has negative effect on both short- and long-term survival of patients with acute or chronic liver diseases (20), also it is related to higher incidence of bleeding during operation, and higher mortality rate. These patients are treated longer in ICU after operation and their treatment costs are higher (21).

In 2003, Cochrane community published conclusions, which were based on high quality clinical trials that there are no significant evidences about positive effects of branched-chain amino acids on symptoms of HE. However, an assessment of different quality trials showed efficacy of branched-chain amino acids for treatment of HE. It was recommended to continue research in this field with larger groups of patients and extend the duration of trials (22). In 2003 Marchesini and in 2005 Muto had conducted two large randomized controlled clinical trials. The results showed that branched-chain amino acids have a positive influence on patients’ condition and survival. Muto et al. ascertained that additional supplementation of diet with preparations of branched-chain amino acids had statistically significant effects on the increase of albumin concentration in the blood, improvement of the quality of life, decrease of the mortality rate, the decreased rate of bleeding from oesophageal varicose veins, and reduction in the development of hepatocellular carcinoma (23). Marchesini et al. also described a positive effect of preparations of branched-chain amino acids on patients’ survival rate, nutritional state and biochemical indices, Child-Pugh functional class and shortening of hospitalization rate, though a significant effect on HE was not observed (24). The use of branched-chain amino acids is also limited by an unpleasant taste and high costs of preparation.

According to 2006 guidelines of the European Society for Parenteral and Enteral Nutrition (ESPEN), patients with liver cirrhosis have to consume not less than 1.2 g/kg proteins per day. It is also recommended to supply diet of HE patients with preparations of branched-chain amino acids (level of evidence A). In advanced liver cirrhosis such diet improves clinical outcomes (level of evidence B) (25). It is very important to take enough proteins with food and to keep a lean body for effective neutralizing of the increased concentration of ammonia in the blood. A replacement of meat-based proteins with plant-based ones and following of “vegetable diet” (sometimes supplemented with milk and cheese) are recommended. Long-term restriction of protein intake is indefensible both for clinically significant persistent HE and minimal impairment of cognitive functions (26). Short-term protein reduction in food is permissible only in case of episodic severe clinical encephalopathy.

If patients are so constrained that they cannot even safely swallow drugs, both medicines and food may be administered via a nasogastric tube. Varicose nods in oesophagus are not contraindication for nasogastric tube insertion.

**PHARMACOLOGICAL THERAPY OF HE**

**Non-absorbable disaccharides (lactulose)**

The main target of HE treatment is the intestine where majority of ammonia is synthesized and absorbed. Non-absorbable disaccharides – lactulose and lactitol – have been widely used for HE treatment since 1970s. Lactic acid (the metabolism product of lactulose) acidifies the content of the intestine, changes a complex of intestinal bacteria, therefore Lactobacillus strain becomes predominant and ammonia producing bacteria are inhibited. Non-absorbable disaccharides also accelerate bowel passage.

Lactulose is used depending on the consciousness of patients. If a patient is comatose or unresponsive, lactulose is administered via a nasogastric tube 30 ml/2–4 hours, or rectally through enemas (300 ml lactulose with 700 ml water) every 4 hours depending on response. Doses per os are individual. Patients should pass stool 2–3 times per day, otherwise disbalance of electrolytes, dehydration, hypovolemia and worsening of HE symptoms may occur. Some patients do not tolerate this preparation because of meteorism, spastic abdominal pain, and unpleasant taste.

In 2004 Cochrane community conducted a systematic overview of clinical trials where non-absorbable disaccharides were administered for HE treatment. While some small clinical trials approved effectiveness of non-absorbable disaccharides, metaanalysis showed that in high quality clinical...
trials lactulose was as effective as placebo. In trials where lactulose was compared with antibiotics (neomycin, rifaximin), clinical effect of antibiotics was preeminent (27). However, there is no better choice, so far, and according to a long lasting clinical experience of lactulose administration worldwide non-absorbable disaccharides remain the first-choice medicines for HE treatment (28). In INASL consensus (2010) on MHE, lactulose is recommended as a medicine which improves psychometric indices and quality of life (evidence level 1b). The recommended dose of lactulose is 30–60 ml 2–3 times per day for 3–6 months (A, 1b) (10).

**Antibiotics**

For a long time antibiotics (neomycin, metronidazole, quinolones, and oral vancomycin) were considered to be the second-choice medicines for HE treatment when lactulose is not effective or a patient does not tolerate it. However, in recent years this point of view is changing.

Neomycin is one of the first antibiotics administered for treatment of HE. Although FDA approved neomycin only for treatment of acute HE (dose per os 1–3 g/6 hours for 6 days), but it is also administered for treatment of chronic HE (0.5–1.0 g/12 hours). Neomycin suppresses glutaminase in the wall of the intestine and synthesis of ammonia is reduced. It also inhibits urea producing bacteria. However, its wider clinical use is limited due to nephrotoxicity and ototoxicity. Long-term use of other antibiotics also has side effects. Metronidazole, which has a similar HE treatment efficacy as neomycin, causes peripheral neuropathies, intestinal disorders and dizziness (29, 30). Vancomycin has better tolerance profile, but resistance of bacteria is of concern. Rifaximin received FDA approval for treatment of HE in March 2010 (at an oral dose of 550 mg twice daily). This antibiotic has a minimal absorption in the intestine, very few side effects, and has no interaction with other medicines.

In 2010 Bass et al. published the results of an international randomized double-blind placebo controlled clinical trial. It was conducted to evaluate the efficacy of rifaximin for prevention of HE recurrences. For one group of patients rifaximin was administered twice daily, and for other group placebo was administered. The patients were followed up for 6 months. Lactulose was given to 91% of patients in both groups. Recurrences were observed more often in the placebo group compared with the rifaximin group (46% and 22%, respectively, \( P < 0.001 \)). The patients treated with the antibiotic were also less often hospitalized because of HE compared with the placebo group (14% and 23%, respectively, \( P = 0.01 \)). However, in the rifaximin group, solitary cases of colitis caused by *Clostridium difficile* were diagnosed (31, 32).

The question of long-term use of antibiotics and related bacteria resistance remains unresolved (33). Some authors recommend taking the medicine continuously with one week break per month. Rifaximin causes peripheral oedemas and nausea. Also it is an expensive preparation (lactulose 30 ml × 4 is 2.2 $ vs rifaximin 400 mg × 3 – 18.42 $). Its cost effectiveness requires more detailed clinical research to evaluate and antibiotics are recommended. According to the results of a retrospective review of Mantry et al., hospitalization rate and duration decrease when rifaximin and lactulose are administered (34). Rifaximin improves psychometric test performance scores, health-related quality of life (35), driving performance in patients with MHE (36).

Results of meta-analysis revealed that rifaximin is as effective as other conventional oral HE treatment with better safety profile (37). It should be administrated for patients, who do not tolerate disaccharides or treatment is unsuccessful.

**Probiotics**

Using of probiotics changes the composition of intestinal bacteria (the proportion between urease producing and not producing bacteria changes) (38). Results of meta-analysis and systemic review show that administration of probiotics or symbiotics is effective for hepatic encephalopathy treatment (39, 40). MHE regresses, concentration of ammonia in the blood decreases, patients perform psychometric tests better, clinically expressed HE occurs less often as compared with patients who did not get this treatment (41–44). According to the consensus of INASL on MHE, probiotics not only decrease endotoxiaemia, but also improve the functional indices of the liver, psychometric tests results, and quality of life (1b) (10). Probiotics are well tolerated. The question of probiotics safety remains unresolved. Cirrhotic patients are considered being immunosuppressed therefore the administration of live bacteria preparation for these
patients is negotiable. A dose of probiotics also remains unclear. Prospective randomized controlled dose related clinical trials to evaluate effective doses are required (5, D) (10).

**L-ornithine-L-aspartate**

Preparations of L-ornithine-L-aspartate (LOLA) promote metabolism of ammonia, decrease its plasma concentration and symptoms of HE.

Ornithine and aspartate are the main substrates for synthesis of urea and glutamine in the liver and muscles (45, 46).

In one small clinical trial, one group of patients was treated with LOLA 18 g/day 2 weeks, and other group was given placebo. In the first group, concentration of ammonia in the blood significantly decreased and the mental activity, cognitive functions of the patients improved. A good effect of LOLA for both clinically expressed HE and MHE patients was observed (47).

Mittal et al. found that in patients with MHE LOLA, probiotics, and lactulose were equally effective for improvement of psychometric indices and life quality (48). In another study, one group of patients was administered 20 g LOLA intravenously for one week, other group got placebo. The results showed that LOLA decreased concentration of ammonia in the blood, improved the mental activity and cognitive functions in the patients with clinically expressed mild HE. Reliable results about its efficacy for patients with MHE and severe HE were not obtained (49). In recently published meta analysis, LOLA efficacy for patients with MHE was not ascertained, though for I and II grade clinically expressed HE LOLA was effective (50). The preparation is safe. Use of LOLA rarely causes intestinal disorders. For patients with renal insufficiency it should be administered with caution. According to INASL, further prospective randomized controlled clinical trials of LOLA efficacy for treatment of MHE are required (1b, A) (10).

**Zinc preparations**

Zinc deficiency is frequently found in cirrhotic patients (especially of alcoholic origin). Food supplements with zinc decrease concentration of ammonia in the blood by stimulating urea and glutamine synthesis in the muscles and liver. Zinc sulphate or acetate 600 mg/day were used in research. The results were controversial (51–54).

**L-carnitine**

In experimental trials on animals L-carnitine decreases concentration of ammonia in serum and cerebrospinal fluid due to stimulating synthesis of urea (55). A randomized placebo controlled clinical trial with 150 patients was conducted. During 90 days one group of patients was treated with L-carnitine 2 g twice per day, other group took placebo. In the first group a significant reduction of ammonia in the blood was observed, also the patients performed psychometric tests better (56). Further studies are necessary to approve the efficacy of L-carnitine (57).

**Sodium benzoate, sodium phenyl butyrate and sodium phenyl acetate**

Sodium benzoate, sodium phenyl butyrate and sodium phenyl acetate are one more group of medicines which stimulate plasma ammonia reduction. Sodium benzoate connects to glycine and forms hippurates which are eliminated through kidneys. One mole of hippuric acid has one mole of nitrogen.

Symptoms of HE might be effectively controlled with 5 g twice per day of sodium benzoate (58). However, this medicine has an unpleasant taste, patient receives an excessive amount of sodium, which is not desirable for patients with heart, kidneys or liver insufficiency and ascites, and sometimes it causes nausea. The FDA has not approved sodium benzoate for treatment of HE.

Sodium phenyl butyrate in the human body turns into phenyl acetate, it reacts with glutamine forming phenyl acetyl glutamine which is eliminated with urine. In one mole of phenyl acetyl glutamine there are two moles of nitrogen. Sodium phenyl butyrate and intravenous sodium phenyl acetate combined with sodium benzoate are approved by the FDA only for treatment of hyperammonemia when there is a disorder of urea synthesis. Recently, clinical trials were conducted to evaluate the efficacy of these medicines for cirrhotic patients with severe HE.

**Alpha-glucosidase inhibitors**

Acarbose is an alpha-glucosidase inhibitor which is administered for treatment of diabetes mellitus type 2. This preparation taken before every meal slows down digestion of consumed carbohydrates. Proteins-fragmenting bacteria in the
large intestine are suppressed so less ammonia is produced.

Acarbose is not routinely used for HE treatment. A single study was conducted to evaluate efficacy of acarbose for patients with liver cirrhosis, diabetes mellitus type 2, and with mild HE. In the patient group who was treated with acarbose, concentration of ammonia in the blood significantly decreased, and they performed psychometric tests better than in the placebo group (59). However, this medicine causes diarrhoea, meteorism and tenesmus, increases activity of liver enzymes, and it is not tolerated very well. Furthermore, the manufacturer information leaflet states that one of contraindications for its use is liver cirrhosis.

Benzodiazepine receptor antagonists
For many years, pathogenesis of HE was explained by both ammonia and GABA theories. It was thought that in case of liver cirrhosis, higher than normal amount of endogenous benzodiazepines is circulated in the blood. Also sensitivity of GABA receptors for these substances increases in brain. This theory was the basis for treatment with benzodiazepine receptor antagonist flumazenil. Researches had denied this theory later (60). Recent data show that flumazenil efficacy was seen for a small group of patients and for a short period of time (61).

Dopamine receptor antagonists
Bromocriptine and L-dopa were administered for persistent HE with marked extra pyramidal symptoms. It was assumed that such symptoms develop because of increased dopaminergic activity. Though in recent years, due to advances in neurovisual (MRI) and molecular research, an excessive accumulation of manganese in basal nuclei was proved as a cause of extra pyramidal symptoms (62, 63). Clinical trials with manganese binding preparations are expected to confirm this assumption.

N-methyl-D-aspartate (NMDA) receptor antagonists
In case of liver cirrhosis high levels of glutamate stimulate NMDA receptors and, at the same time, activate many enzymes including nitric oxide synthase. On the other hand, NO activates soluble guanylate cyclase and cGMP concentration increases. These processes are important for circadian rhythms and long-term potentiation, which is important for memory and learning (64).

In animal experiments, memantine, a NMDA receptor antagonist, has a positive effect on all clinical symptoms, EEG data, ammonia concentration in cerebrospinal fluid, but not on blood ammonia (65).

NON-PHARMACOLOGICAL THERAPY OF HE

MARS (Molecular Adsorbents Recirculation System)
MARS helps to eliminate small and medium size water soluble molecules, and albumin-binded molecules. Worldwide, it is mostly used in cases of drug poisoning. MARS is not approved by the FDA for HE treatment. During MARS procedure bilirubin binds to albumin, biliary acids and ammonia are eliminated. Results of a multicentre randomized controlled trial with patients who had HE of grade 3–4 showed that after use of MARS for 6 hours per day for 5 days, in addition to standard pharmacological therapy, disappearance of HE symptoms was achieved much faster than with medicinal therapy only (66). The procedure is well tolerated.

TIPS (Transjugular Intrahepatic Portosystemic Shunt)
In 35% of patients after performing TIPS procedure HE develops. Condition of most patients improves when standard therapy is administered, though 3–7% invasive procedures are necessary. For some patients the diameter of a stent has to be minimized, for others a stent has to be even obturated (67).

Liver transplantation
Hepatic encephalopathy after liver transplantation is a reversible condition. Various MRI imaging findings after liver transplantation regress and disappear (68). Incomplete improvement might be in patients with hepatic myelopahty, acquired hepato-cerebral degeneration. Also there is opinion that after an overt HE bout subtle neurological abnormalities remain (69, 70). HE residual changes must be differentiated from intraoperative ischemic brain lesions and persistent portosystemic shunts after liver transplantation, previous stroke, alcohol abuse, trauma, drugs side effects. It is unknown if HE has a significant impact upon the clinical
outcome after liver transplantation. Until now, there is not priority for earlier liver transplantation for HE affected patients (71).

CONCLUSIONS

Hepatic encephalopathy diagnosis is based on patient’s clinical condition. HE grading by West-Haven’s criteria, especially stages 1 and 2, might be subjective and influence results of clinical trials. MHE is undiagnosed and untreated in most cases due to incompliance of psychometric tests and lack of validated blood tests.

A new approach to the HE pathogenesis allowed to start rifaximin for HE treatment and prophylaxis. Rifaximin has the same clinical effectiveness as disaccharides and oral antibiotics and causes fewer side effects than other oral agents. Rifaximin therapy is more expensive than lactulose. Lactulose is still recommended as the first line therapy.

There are still insufficient research data to design algorithms of hepatic encephalopathy diagnostic and treatment suitable for everyday clinical practice. Further studies are necessary.

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HEPATINĖS ENCEFALOPATIJOS DIAGNOSTIKA IR GYDYMAS

Santrauka


Tikslas. Apžvelgti hepatitis encefalopatijos diagnostikos ir gydymo naujoves bei jų pritaikymo klinikinėje praktikoje galimybę siekti pagerinti ciroze sergančių pacientų gyvenimo kokybę bei atitolinti klinikinių HE simptomų atsiradimą.

Metodai ir medžiaga. Šis straipsnis parašytas remiantis originaliais straipsniais ir apžvalgomis anglų kalba (1991–2012), kurie rasti Medline ir Pubmed duomenų bazėse naudojant raktažodžius.


Minimaliai hepatitis encefalopatijai (MHE) gydyma rekomenduojama laktuliozė ir probiotikai. MHE diagnostika išlieka sudėtinga – nėra patvirtintų kraujo tyrimų, kurie padėtų ją diagnozuoti bei stebėti gydymo efektyvumą. Psychometriniai ir neurofiziologiniai, taip pat vizualizacijos metodai daugiau taikyti mokslo tyrimui, kasdienėje praktikoje gali būti naudingi kompiuterizuoti metodai (psychometrinis inhibicinis kontrolės testas, kritinio mirgėjimo dažnio testas).

Išvados. Reikalingi tolesni klinikiniai tyrimai, kurie leistų sukurti klinikiniam pitaikymui tinkamą HE diagnostikos ir gydymo algoritmą.

Raktas: hepatitis encefalopatija, minimali hepatitis encefalopatija, diagnostika, gydymas