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Ensefalopati hepatik pdf

BackHepatic Encephalopathy in Chronic Liver Disease The approach to a patient with hepatic encephalopathy depends upon the severity of the mental status changes and upon the certainty of the diagnosis. As an example, a patient with known cirrhosis and mild complaints of decreased concentration might be served best by an empiric trial of rifaximin or lactulose and a follow-up office visit to check its effect. However, a patient presenting to the emergency department with severe hepatic encephalopathy requires a different approach. General management recommendations include the following: Exclude nonhepatic causes of altered mental function. Consider checking an arterial ammonia level in the initial assessment of a hospitalized patient with cirrhosis and with impaired mental function. Ammonia levels have less use in a stable outpatient. Precipitants of hepatic encephalopathy, such as hypovolemia, metabolic disturbances, gastrointestinal bleeding, infection, and constipation, should be corrected. Avoid medications that depress central nervous system function, especially benzodiazepines. Patients with severe agitation and hepatic encephalopathy may receive haloperidol as a sedative. Treating patients who present with coexisting alcohol withdrawal and hepatic encephalopathy is particularly challenging. These patients may require therapy with benzodiazepines in conjunction with lactulose and other medical therapies for hepatic encephalopathy. Patients with severe encephalopathy (ie, grade 3 or 4) who are at risk for aspiration should undergo prophylactic endotracheal intubation. They are optimally managed in the intensive care unit. Most current therapies are designed to treat hyperammonemia that is a hallmark of most cases of hepatic encephalopathy. Diet In the late 19th century, it was recognized that the feeding of a high-protein diet to dogs that had undergone portosystemic shunt surgery could produce symptoms of abnormal coordination and stupor in the treated animals. In the 20th century, low-protein diets were routinely recommended for patients with cirrhosis, in the hope of decreasing intestinal ammonia production and in preventing exacerbations of hepatic encephalopathy. An obvious consequence was the worsening of preexisting protein-energy malnutrition. Protein restriction may be appropriate in some patients immediately following a severe flare of symptoms (ie, episodic hepatic encephalopathy). However, protein restriction is rarely justified in patients with cirrhosis and persistent hepatic encephalopathy. Indeed, malnutrition is a more serious clinical problem than hepatic encephalopathy for many of these patients. In the author's experience, it is an infrequent patient who is intolerant of a diet high in protein. Most patients with mild chronic hepatic encephalopathy tolerate more than 60-80 g of protein per day. Furthermore, one study administered a protein-rich diet (>1.2 g/kg/d) to patients with advanced disease awaiting liver transplantation, without inducing a flare of encephalopathy symptoms. [40] Another study randomized patients with severe episodic encephalopathy to either a low-protein diet or a high-protein diet, administered via nasogastric tube. [41] All patients received the same regimen of neomycin per nasogastric tube. Mental function improved at the same rate in both treatment groups. Importantly, patients receiving the low-protein diet had evidence of increased protein breakdown during the duration of the study. Diets containing vegetable proteins appear to be better tolerated than diets rich in animal proteins, especially proteins derived from red meats. This may be because of increased content of dietary fiber, a natural cathartic, and decreased levels of aromatic amino acids. Aromatic amino acids, as precursors of the false neurotransmitters tyramine and octopamine, are thought to inhibit dopaminergic neurotransmission and worsen hepatic encephalopathy. The author recommends that patients consume well-cooked chicken and fish in addition to vegetable proteins. Malnourished patients are encouraged to add commercially available liquid nutritional supplements to their diet. Patients rarely require specialized treatment with oral or enteral supplements rich in branched-chain amino acids. To evaluate the beneficial and harmful effects of branched-chain amino acids (BCAA) versus any control intervention for people with hepatic encephalopathy, Gluud and colleagues conducted a systematic review involving 16 randomized clinical trials that included 827 participants with hepatic encephalopathy. Primary outcomes included mortality (all cause), hepatic encephalopathy (number of people without improved manifestations of hepatic encephalopathy), and adverse events. The control groups received placebo/no intervention, diets, lactulose, or neomycin. In 15 trials, all participants had cirrhosis. Analyses showed that BCAA had a beneficial effect on hepatic encephalopathy. The authors found no effect of BCAA on mortality, quality of life, or nutritional parameters, but they recommended additional trials to evaluate these outcomes. [42] Cathartics Lactulose (beta-galactosidofructose) and lactitol (beta-galactosidosorbitol) are nonabsorbable disaccharides that have been in common clinical use since the early 1970s (the latter is not available in the United States). They are degraded by intestinal bacteria to lactic acid and other organic acids. Lactulose appears to inhibit intestinal ammonia production by a number of mechanisms. The conversion of lactulose to lactic acid and acetic acid results in the acidification of the gut lumen. [43, 44] This favors conversion of ammonia (NH3) to ammonium (NH4+); owing to the resultant relative impermeability of the membrane, the NH4+ ions are not easily absorbed, thereby remaining trapped in the colonic lumen, and there is a reduction in plasma NH3. [43, 44, 45] Gut acidification inhibits ammoniagenic coliform bacteria, leading to increased levels of nonammoniaogenic lactobacilli. [43] Lactulose also works as a cathartic, reducing colonic bacterial load. Initial lactulose dosing is 30 mL orally, daily or twice daily. The dose may be increased as tolerated. Patients should be instructed to reduce lactulose dosing in the event of diarrhea, abdominal cramping, or bloating. Patients should take sufficient lactulose as to have 2-4 loose stools per day. Great care must be taken when prescribing lactulose. Overdosage can result in ileus, severe diarrhea, electrolyte disturbances, and hypovolemia. Hypovolemia may be sufficiently severe as to actually induce a flare of encephalopathy symptoms. Higher doses of lactulose (eg, 30 mL q2-4h) may be administered orally or by nasogastric tube to patients hospitalized with severe hepatic encephalopathy. Lactulose may be administered as an enema to patients who are comatose and unable to take the medication by mouth. The recommended dosing is 300 mL lactulose plus 700 mL water, administered as a retention enema every 4 hours as needed. Lactulose has been the subject of dozens of clinical trials over almost four decades. Many small trials demonstrated the medication's efficacy in the treatment of hepatic encephalopathy. A controversial meta-analysis published in 2004 contradicted these trials and most physicians' clinical experiences. [46] When assessing high-quality randomized trials, lactulose was no more effective than placebo at improving encephalopathy symptoms. In trials comparing lactulose to an antibiotic (eg, neomycin, rifaximin), lactulose was actually inferior to antibiotic therapy. In subsequent years, multiple randomized trials have reinvestigated the efficacy of lactulose. In patients with minimal hepatic encephalopathy, lactulose was more effective than placebo in terms of improving patient performance on psychometric testing. [47, 48] Lactulose was studied in large randomized trials as secondary prevention against recurrent overt encephalopathy. [49, 50] In the study by Sharma et al, patients who were recovering from hepatic encephalopathy were randomized to receive lactulose (n = 61) or placebo (n = 64). Over a median follow-up of 14 months, 12 patients (19.6%) in the lactulose group developed overt hepatic encephalopathy as compared with 30 patients (46.8%) in the placebo group (P = 0.001). The authors concluded that lactulose effectively prevented the recurrence of overt hepatic encephalopathy in patients with cirrhosis. [49] Lactulose also appeared to be effective as primary prophylaxis against the development of overt hepatic encephalopathy, [51] although few physicians in the United States would advocate the use of lactulose for this indication. An updated meta-analysis published in 2013 included these studies and affirmed the utility of lactulose in the management of hepatic encephalopathy. [52] Antibiotics Neomycin and other antibiotics, such as metronidazole, oral vancomycin, paromomycin, and oral quinolones, are administered in an effort to decrease the colonic concentration of ammoniagenic bacteria. Initial neomycin dosing is 250 mg orally 2-4 times a day. Doses as high as 4000 mg/d may be administered. Neomycin is usually reserved as a second-line agent, after initiation of treatment with lactulose. Long-term treatment with this oral aminoglycoside runs the risks of inducing ototoxicity and nephrotoxicity because of some systemic absorption. Rifaximin (Xifaxan), a nonabsorbable derivative of rifampin, has been used in Europe for more than 20 years for a wide variety of gastrointestinal indications. Multiple clinical trials have demonstrated that rifaximin at a dose of 400 mg taken orally 3 times a day was as effective as lactulose or lactitol at improving hepatic encephalopathy symptoms. [46, 53, 54] Similarly, rifaximin was as effective as neomycin and paromomycin. Rifaximin had a tolerability profile comparable to placebo. It was better tolerated than both the cathartics and the other nonabsorbable antibiotics. A potential mechanism for rifaximin's clinical activity is its effects on the metabolic function of the gut microbiota, rather than a change in the relative bacterial abundance. [55] In 2004, rifaximin received approval by the FDA in the United States for the treatment of travelers' diarrhea. In 2005, it received orphan drug status as a treatment for hepatic encephalopathy. In March 2010, rifaximin was approved by the FDA to reduce recurrence of hepatic encephalopathy. The approval was based on a phase 3 clinical trial conducted by Bass et al. [56] Bass et al evaluated rifaximin's ability to reduce the risk of recurrent hepatic encephalopathy (HE). [56] In this double-blind, placebo-controlled, multinational, phase 3 clinical trial, 299 patients received either rifaximin 550 mg or placebo BID. Each group also received lactulose. Breakthrough episodes of HE occurred in 22% of patients treated with rifaximin and 46% of patients with placebo (P< 0.001). HE-related hospitalization occurred in 14% of patients treated with rifaximin and 23% of patients treated with placebo (P = 0.01). Peripheral edema and nausea are described in some rifaximin-treated patients. There are also questions whether long-term treatment with rifaximin can induce microbial resistance. Thus far, microbial resistance has not been reported in patients using the medication. It remains unclear whether diarrhea caused by Clostridium difficile occurs at a higher rate in rifaximin-treated patients than untreated patients. In the study by Bass et al, two rifaximin-treated patients and no placebo-treated patients developed C difficile infection. [56] Rifaximin was also examined in patients with minimal hepatic encephalopathy. In a large study by Sidhu et al, rifaximin was more effective than placebo in terms of improving patient performance on psychometric testing and in terms of improving health-related quality of life. [57] L-ornithine L-aspartate (LOLA) LOLA (Hepa-Merz) is available in Europe in both intravenous formulations and oral formulations. It is not available in the United States. LOLA is a stable salt of the two constituent amino acids. L-ornithine stimulates the urea cycle, with resulting loss of ammonia. Both L-ornithine and L-aspartate are substrates for glutamate transaminase. Their administration results in increased glutamate levels. Ammonia is subsequently used in the conversion of glutamate to glutamine by glutamine synthetase. LOLA was found to be effective in treating hepatic encephalopathy in a number of European trials. [58, 59] Zinc Zinc deficiency is common in cirrhosis. Even in patients who are not zinc deficient, zinc administration has the potential to improve hyperammonemia by increasing the activity of ornithine transcarbamylase, an enzyme in the urea cycle. The subsequent increase in ureagenesis results in the loss of ammonia ions. Zinc sulfate and zinc acetate have been used at a dose of 600 mg orally every day in clinical trials. Hepatic encephalopathy improved in two studies [60]; there was no improvement in mental function in two other studies. [61] Meena et al evaluated the correlation between low serum zinc levels in 75 patients with decompensated chronic liver disease (DCLD) and various stages of hepatic encephalopathy. There was a statistically significant association between low serum zinc level and the grade of HE (P = 0.001) or class of liver cirrhosis (P = 0.001). Additional studies are needed to establish the role of correcting hypozincemia to prevent worsening of cirrhosis and development of encephalopathy. [62] Sodium benzoate, sodium phenylbutyrate, sodium phenylacetate, glycerol phenylbutyrate Sodium benzoate interacts with glycine to form hippurate. The subsequent renal excretion of hippurate results in the loss of ammonia ions. Dosing of sodium benzoate at 5 g orally twice a day can effectively control hepatic encephalopathy. [63] Use of the medication is limited by the risk of salt overload and by its unpleasant taste. The medication, also used as a food preservative, is available throughout many specialty chemical manufacturers throughout the United States. The author has limited its use to patients with severe encephalopathy symptoms. However, in the author's opinion, doses of sodium benzoate as low as 2.5 g orally three times per week significantly improved mental function in outpatients who had persistent encephalopathy symptoms despite cotherapy with lactulose and rifaximin. Sodium phenylbutyrate is converted to phenylacetate. Phenylacetate, in turn, reacts with glutamine to form phenylacetylglutamine. This chemical is subsequently excreted in the urine, with the loss of ammonia ions. Sodium phenylbutyrate (Buphenyl), intravenous sodium phenylacetate in combination with sodium benzoate (Ammonul), and glycerol phenylbutyrate (Ravicti) are approved by the FDA for the treatment of hyperammonemia associated with urea cycle disorders. [64] The latter is currently in clinical trials in cirrhotic patients with hepatic encephalopathy. [65] In a phase II trial involving 178 patients with cirrhosis (including 59 already taking rifaximin) who had experienced two or more hepatic encephalopathy (HE) events in the previous 6 months, glycerol phenylbutyrate (GPB), at a dose of 6 mL orally twice-daily, significantly reduced the proportion of patients who experienced an HE event, time to first event, and total events. [66] In addition, GPB was associated with fewer HE hospitalizations. For patients not on rifaximin at enrollment, GPB reduced the proportion of patients with an HE event, time to first event, and total events. Plasma ammonia was significantly lower in patients on GPB than in patients on placebo. Adverse events occurred in a similar proportion of patients in the GPB and placebo groups. The authors concluded that the results implicated ammonia in the pathogenesis of HE and suggested that GPB had therapeutic potential in this patient population. [66] L-carnitine L-carnitine improved hepatic encephalopathy symptoms in several small studies of patients with cirrhosis. [67] Whether the medication works by improving blood ammonia levels or whether it works centrally perhaps by decreasing brain ammonia uptake remains unclear. [68] Sleep disturbances are more common in patients with cirrhosis than in control subjects. Whether or not this relates to hepatic encephalopathy is unclear. A trial compared the histamine H1 blocker hydroxyzine with placebo in patients with cirrhosis and minimal hepatic encephalopathy. [69] Sleep efficiency and the patients' subjective quality of sleep improved in patients receiving hydroxyzine (25 mg) at bedtime. However, there was no accompanying improvement in cognition, as measured by neurophysiologic tests. The authors urged caution when prescribing hydroxyzine, on account of the risk of worsening encephalopathy in some patients. Hepatic encephalopathy is seen in about 1 in 3 patients who undergo the creation of a transjugular intrahepatic portosystemic shunt (TIPS). Typically, post-TIPS encephalopathy symptoms are well controlled with the use of rifaximin or lactulose. However, post-TIPS encephalopathy symptoms can be profound in some instances. In a study by Fanelli et al, 12 of 189 patients undergoing TIPS developed encephalopathy that was refractory to conventional therapy with lactulose. These patients subsequently underwent placement of an hourglass-shaped balloon-expandable polytetrafluoroethylene (ePTFE) stent-graft inside the original shunt. Encephalopathy symptoms resolved in all of the patients over the next 18-26 hours. [70] Of course, such a procedure is not expected to improve a patient's overall condition. At the end of a mean of 74 weeks of follow-up, only 5 of the 12 patients remained alive and in good clinical condition. Trebicka et al studied the outcomes of diameter of covered, self-expandable nitinol stents in patients with a TIPS. Of 185 patients, 53 received 8 mm stents and the remaining received 10 mm stents. Patients who received 8 mm stents survived significantly longer than patients who received 10 mm stents, regardless whether they were fully dilated or underdilated. The authors concluded that the 8 mm stent is associated with a prolonged survival compared to 10 mm stents, independent of liver specific prognostic criteria. [71] ensefalopati hepatikum pdf. ensefalopati hepatikum grade. ensefalopati hepatik grade. ensefalopati hepatikum scribd. ensefalopati hepatikum icd 10. ensefalopati hepatikum ppt. ensefalopati hepatikum medscape. ensefalopati hepatik jurnal

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