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Liver cirrhosis - disease awareness and dietary behavior

Marskość wątroby - świadomość schorzenia, a postępowanie dietetyczne

Summary

Cirrhosis is a condition defined histologically characterized by multiple clinical consequences. Following the generalized damage to the flesh, comes to fibrosis and normal liver architecture changes and disruption of the vascular system. With progression of liver failure, symptoms of metabolic impairment of the function and portal hypertension. Among the most common causes include the alcohol and hepatotropic viruses (hepatitis B, D and C). If left untreated, can lead to the transformation of liver cancer (hepatocellular carcinomacell, HCC). The goal of treatment is to eliminate the etiological agent, inhibiting the further progress of the disease and prevention of complications. Among the treatment options is a diet. This article contains the current state of knowledge about liver cirrhosis, as well as dietary tips which can slow down the progression of the disease.

Keywords: liver cirrhosis, HCV, HBV, alcohol, diet

Streszczenie

Marskość wątroby jest stanem definiowanym histologicznie, charakteryzującym się wieloma konsekwencjami klinicznymi. W następstwie uogólnionego uszkodzenia mięszu, dochodzi do włóknienia i przemiany prawidłowej architektury wątroby oraz dezorganizacji układu naczyniowego. W miarę pogłębiania się niewydolności wątroby pojawiają się objawy upośledzenia jej czynności metabolicznej i nadciśnienia wrotnego. Wśród najczęstszych przyczyn wymienia się alkohol oraz wirusy hepatotropowe (WZW B, D lub C). Nieleczona doprowadzić może do przemiany nowotworowej wątroby (rak wątrobowo komórkowy). Celem leczenia jest wyeliminowanie czynnika etiologicznego, zahamowanie dalszego postępu choroby oraz zapobieganie wystąpieniu powikłań. Wśród możliwości leczniczych duże miejsce zajmuje dietoterapia. Poniższy artykuł zawiera aktualny stan wiedzy dotyczący marskości wątroby, jak również wskazówki dietetyczne mogące spowolnić postęp choroby.

Słowa kluczowe: marskość wątroby, HCV, HBV, alkoholizm, dieta

Introduction

Cirrhosis is a condition defined histologically characterized by multiple clinical consequences. Following the generalized damage to the flesh, comes to fibrosis and normal liver architecture changes and disruption of the vascular system. With progression of liver failure, symptoms of impaired its metabolic activity and / or portal hypertension (decompensated cirrhosis). Cirrhosis without decompensation defined as compensated (Sass and Chopra, 2009; Wojcik and Piekarska, 2005; Szczeklik and Gajewski, 2011).

In the past, liver cirrhosis was considered irreversible disease of the course. Subsequent studies have confirmed the possibility of reversing the process of fibrosis. The condition for this is to remove the primary damaging agent, which is the cause of cirrhosis. Withdrawal fibrosis is observed in the case of successful treatment of chronic hepatitis C and hemochromatosis and patients with alcoholic liver disease who have stopped drinking (Strader et al., 2004).

Liver cirrhosis is a common condition among men, but the incidence depends on the amount of alcohol consumed and the prevalence of infections with hepatotropic viruses (geographical variation). Morbidity caused by cirrhosis of the liver is 200-300/100 000 population, and mortality-2% / year. The disease develops at any age with an increased incidence of male population (Blei and Cordoba, 2001; Szczeklik and Gajewski, 2011, Riley TR, Bhatti, 2001).

Every year in Poland, about 1500 cases of viral hepatitis B, and most of them are forms of chronic HBV infection. According to epidemiological studies conducted in Poland in 2009, to 26 thousand people (about 2% of the population) is infected with HCV. In most cases, HCV infection is not recognized prior to the disclosure of his irreversible consequences such as liver cirrhosis and primary liver cancer (Habior, 2001, Foster, 2009; Szczeklik and Gajewski, 2011).

Course of the disease from diagnosis to the extreme histological liver failure lasts a long time in different ways. 10 years experienced only 10-20% of patients. It depends on the etiology and treatment undertaken (Blei and Cordoba, 2001).

Liver cirrhosis is considered to be pre-cancerous condition, which can develop hepatocellular carcinoma cell (HCC). The risk of malignant transformation is high, especially in patients with cirrhosis, a viral infection (HCV infection mostly) (Ryder, 2003).

Etiology

Cirrhosis is the final stage of many chronic liver diseases. Among the most common causes include the alcohol and hepatotropic viruses (hepatitis B, D and C). Other factors include a proven connection: autoimmune hepatitis, metabolic diseases, diseases of biliary tract, disease with impeded venous return, sarcoidosis, syphilis, drugs and toxins. The factors of the relationship unconfirmed include hepatitis G, mycotoxins, malnutrition, obesity, diabetes and schistosomiasis. In case it fails to establish the etiology, we are dealing with cryptogenic cirrhosis or disease Hindu children (Strader et al., 2004; Bhatti, 2001).

Risk factors for cirrhosis of the liver, in addition to the already mentioned alcohol abuse and chronic hepatitis B and C, male gender, genetic predisposition, and possibly smoking (Habior, 2001; Strader et al., 2004).

Pathogenesis

The pathogenesis of liver cirrhosis is still not fully elucidated. We know, however, that this process is the result of disturbed homeostasis between the formation and deposition of extracellular matrix and its degradation and destruction (Blei and Cordoba, 2001; Zahorska-Markiewicz and Małeck-Tender, 2009).

Under the influence of chronic damaging factor hepatocyte, comes to its launch necrosis and inflammation. Thus released cytokines and growth factors, stimulate the stellate cells (Ito cells), leading to their proliferation and transformation into myofibroblasts. The latter, in turn, begin to produce various cellular matrix components that accumulate in the spaces underendothelium of Disse (fibrosis about sinus). Consequently, this leads to kapilarizations bays probably responsible for clinical signs of fibrosis (portal hypertension, liver metabolic dysfunction) (Arroyo and Colmenero, 2003; Zahorska-Markiewicz and Małeck-Tender, 2009).

There are four levels of activity in cirrhosis: Io-phase inactive cirrhosis (fibrosis limit does not include parenchyma cells), IIo-bridging fibrosis with partial tuberosity (partial destruction of the plaque border); IIIo-bridging fibrosis tuberosity (complete destruction of the plaque border), IVo-cirrhosis of the liver (fibrosis ongoing, active inflammation, infiltration of eosinophils, plasma cells, macrophages, cholestasis, liver cell necrosis nodes and beyond nodules, malignant transformation) (Grant et al., 2004).

Type of cirrhosis

Due to the different distribution criteria, we distinguish several types of diseases. Having morphological changes (capacity regenerative nodules) cirrhosis divided into: micronodular (nodules up to 3 mm, all of equal size), macronodular (nodes size from 3 mm to several centimeters) and mixed; due to the etiology of: after necrotic cirrhosis (after hepatitis), portal vein cirrhosis (alcohol-induced), dysmetabolic cirrhosis, biliary cirrhosis (primary and secondary); due to functional changes in: compensated and decompensated cirrhosis (Blei and Cordoba, 2001).

The clinical picture

The clinical picture liver cirrhosis depends on several factors: the duration of illness, number of parenchyma, and portal circulation disorders of the treatment. In less than half of the patients with liver cirrhosis (30-40%), the disease was completely asymptomatic and detected incidentally during testing for other reasons or during surgery (Habior, 2001; Piekarska, 2008; Zahorska-Markiewicz and Małeck-Tender, 2009; Wojcik and Piekarska, 2005).

During adjusted cirrhosis symptoms are most often unspecific. As the disease progresses accompanied by: weakness and increased fatigue, loss of appetite and weight loss, bloating, nausea and / or vomiting, right upper abdominal pain, the state of low-grade fever, itchy skin, decreased sex drive, menstrual disorder and infertility, painful gynecomastia, painful muscle cramps, bleeding gums and nose (Habior, 2001; Piekarska, 2008; Zahorska-Markiewicz and Małeck-Tender, 2009; Wojcik and Piekarska, 2005).

The adjusted form of liver cirrhosis there is no significant signs. At the time of onset of signs of decompensation (decompensated cirrhosis) occurs: the characteristic silhouette of the body (slim upper and lower limbs with enlarged abdominal circumference), ascites (accumulation of fluid in the peritoneal cavity), gynecomastia (enlargement of the

mammary glands in males resulting non-neoplastic proliferation of glandular tissue), and testicular atrophy, hirsutism in women (excess hair mature areas androgens sensitivity), swelling of the salivary glands, enlarged liver and spleen. Skin symptoms manifest themselves: jaundice (yellow coloring of whites, mucous membranes and skin, caused by accumulation of bilirubin in the tissues), stellate angioma, palmar and plantar erythema, telangiectasias (dilated presence of small blood vessels), tufts of yellow, leukonychia (color change nail), loss of hair on the chest and under the arms of men, Dupuytren's contracture (palmar fascia contracture), dilated veins of collateral circulation in the abdomen ("head of Medusa"), smoothing language (Habior, 2001; Piekarska, 2008; Zahorska-Markiewicz and Małeczka-Tender, 2009; Wojcik and Piekarska, 2005).

Ratingliver failure

The clinical significance and prognostic value in cirrhosis of the liver is split progress of the failure of the Child and Pugh criteria into three steps, from the smoothest- A by B, the most severe failure- C, based on the determined serum levels of albumin, bilirubin and prothrombin, and the presence of ascites or encephalopathy (Table 1).

Tab. 1. Classification of liver failure according to Child and Pugh criteria

parameter	points according to the degree of abnormality		
	1	2	3
encephalopathy	not	1-2 degree	3-4 degree
ascites	not	moderate	strained
bilirubin (mg/dl) (in primary biliary cirrhosis, PBC)	< 2	2 – 3	>3
albumin (g/dl)	> 3,5	2,8 -3,5	< 2,8
prothrombin time (in "s" more than normal)	1 –4	5 -10	> 10
total score	5 -6	7 -9	>10
result	A	B	C

Patients in the Class A there is no indication for liver transplantation (compensated cirrhosis), and patients in the class B and C are eligible for transplant (decompensated cirrhosis).

Diagnostics

Diagnosis of cirrhosis based on laboratory tests, imaging, endoscopic and morphological (histological). Among the irregularities laboratory findings are: thrombocytopenia, macrocytic anemia (hemolysis indicates Wilson's disease), leukopenia, increased alanine aminotransferase and aspartate aminotransferase (inflammatory etiology), increased alkaline phosphatase (cholestatic etiology), gamma-glutamyl-transferase (alcoholic etiology, autoimmune and viral), decreased cholinesterase activity, polyclonal hypergammaglobulinemia (due to the current collateral circulation), hyperglycemia (due to impaired insulin metabolism in hepatocytes), hypertriglyceridaemia (alcoholic etiology), hypercholesterolemia (cholestatic etiology), increased serum alpha-feto-protein (values > 200 µg/ml indicate hepatocellular carcinoma cell). In decompensated cirrhosis, serum appears: hyperbilirubinemia (with a predominance of conjugated bilirubin), hypoalbuminemia, plasma coagulation (prothrombin time, deficiency of factor V and antithrombin III), hypoglycemia, increased creatinine, and ammonia, hyponatremia, hypo-/hyper-kaliemia, hypokalaemic alkalosis (Habior, 2001; Piekarska, 2008; Szczeklik and Gajewski, 2011).

Imaging studies are performed in order to: determine the size and shape of the organ, accompanying the diagnosis of fatty liver cirrhosis, detection of lesions and the evaluation of symptoms of portal hypertension and liver blood flow. Among the imaging methods take into account the ultrasound, computed tomography, scintigraphy and possibly arteriography (Habior, 2001; Piekarska, 2008).

Endoscopy (ezofago-gastro-duodenoscopy) is performed to look for characteristics of portal gastropathy (esophageal varices), or changes in ulcer (Habior, 2001; Piekarska, 2008).

Histological examination of the liver section is made to substitute a clear diagnosis of liver cirrhosis and liver disease staging (Habior, 2001; Piekarska, 2008).

Complications

Complications of cirrhosis include: ascites, spontaneous bacterial peritonitis, gastrointestinal bleeding, jaundice, bleeding disorders, hormonal disorders, hepatic encephalopathy, hepatorenal syndrome, hepato-pulmonary syndrome, hypersplenism, portal vein thrombosis and hepatocellular carcinoma cell (Choudhury and Sanyal, 2003; Zahorska-Markiewicz and Małecka-Tender, 2009; Szczeklik and Gajewski, 2011).

Treatment

The goal of treatment is to inhibit the further progression of the disease and the prevention of complications. First of all, the aim must be to eliminate the causative factors. Absolute hospital bed and the regime recommended by exacerbations of cirrhosis and the occurrence of complications. Treatment of rest, dietary and pharmacological tests must be applied simultaneously. Only this will lead to a successful therapeutic effect (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewicz, 2003; Książczyńska, 2010; Szczeklik and Gajewski, 2011).

Accidental adjusted cirrhosis is alcohol abstinence and proper diet (Halsted, 2004; Gajewska, 2009).

Products recommended for the preparation of diet in liver cirrhosis include bread (wheat and rye milling low), small cereal, rice, small pasta, noodles lane, cottage cheese,

cottage cheese, skim milk, eggs (in limited quantities), lean meat (veal, poultry, lean fish such as cod, crucian carp bream), lean meats (ham, sirloin), meatballs, fresh butter, cream, olive oils (soya and sunflower) in the form of mashed potatoes, carrots, beets, spinach, pumpkin, celery, apples, strawberries, blueberries, apricots, honey, jams, weak tea, coffee, cereal, yogurt, kefir, fruit juices (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książdźyna, 2010).

Nutritional therapy in liver cirrhosis should be in each case individually. It depends on many factors and includes: disease duration, severity, complications, the operation of compensatory mechanisms. The most important factor that determines the choice of diet is the nutritional status of the patient and their clinical condition (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książdźyna, 2010).

Dietary treatment should include: a balanced diet in terms of supply of nutrients and energy, taking into account the deficiencies in minerals, vitamins and rich diet with adequate preparations which protects the liver (eg S-methylmethionine, phosphatidylcholine, polyunsaturated fatty acids, glutamine). Many difficulties make the right nutrition disorders of the gastrointestinal tract, such as anorexia, vomiting or nausea (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książdźyna, 2010).

Adequate energy intake is recommended for all patients with chronic liver disease. It is also a major factor in preventing protein-energy malnutrition. The adjusted cirrhosis and normal body mass caloric intake of foods should be compatible with standard saline: 10 000 to 11 700 kJ (2400-2800 kcal.). For patients malnourished amount of energy should be increased to 12 600 kJ (3000 kcal), and even more (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książdźyna, 2010).

In most cases, the patients is recommended the rich of proteins diet with an increased amount of energy. Note, however, that the protein is not used by the liver leads to increased conversion of nitrogen metabolites, contributing indirectly to an increase in the concentration of ammonia (encephalopathy factor). Therefore, patients should observe the reaction of your body to prescribed diet and if necessary make changes in it, and only after consultation with the doctor (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książdźyna, 2010).

In patients with compensated cirrhosis optimum protein intake should be 1-1.5 g / kg / day. The dose by stimulating liver cell renewal processes, reduce fatty infiltration of liver, favoring tissue complement deficiency and synthesis of serum albumin, has a beneficial effect on the disease. Protein in about 60% should come from animal products, and should cover up to 15-20% of energy (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książdźyna, 2010).

Consumption of sugar but does not exceed 300-400 grams / day (approximately 70% of daily energy intake), including 50-70 g digestible sugars summer (in honey, jams and confectionery). Please note that this is not artificially colored products with the addition of preservatives. An excess of these sugars in the diet causes an increased synthesis of triglycerides in the liver and leads to severe steatosis of the liver. The demand for carbohydrates covers mainly starch contained in cereals and potatoes (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książdźyna, 2010).

The daily amount of fat should be limited and should not exceed 1.0 g / kg / day, which is about 25% of total energy intake. The diet should be eliminated animal fats with high melting such as lard or bacon at the expense of unsaturated fats, high in nutrition (soybean oil, sunflower oil, olive oil) (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003, Książczyńska, 2010).

It is important that the diet was varied and included in its composition to the mineral salts and vitamins. These components, as a result of malabsorption, there are respectively the quantity stored and used. This causes latent deficiencies in the body. If your diet does not cover the increased need for vitamins, the doctor will advise you to take pre-vitamin supplements. Antioxidant vitamins (E, C, A and carotenoids) contained in green leafy plants, play an important role in slowing the progression of liver disease. They should be used in an amount of 150-200g per day. Deficiency of fat-soluble vitamins can be seen in patients with biliaryinsufficiency (cholestasis), alcoholism, fat intolerance, patients taking certain medications (cholestyramine). Supplementation of these vitamins may need patients with fatty diarrhea (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książczyńska, 2010).

In the feeding of patients with cirrhotic liver plays an important role fiber. It is given a similar dose to healthy people. By eliminating constipation, which is a common problem, fiber improves intestinal peristalsis. In addition, fractions soluble fiber reduces insulin resistance and favorably alter plasma lipid profile (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książczyńska, 2010).

In the feeding patients should be small and frequent meals (7-8/dobę), with an evening snack. Additional last meal should provide about 200-300 calories (15-20% of the total daily requirement) and consist largely of carbohydrates. Doing so will shorten the life of hunger and improves nitrogen balance (small reserve of glycogen stimulates faster glikogenezy at night). In addition, all patients should be bound by the absolute prohibition of drinking alcohol (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książczyńska, 2010).

The demand for energy in patients with decompensated cirrhosis of the liver for energy is 35-40 kcal / kg / day and protein intake is about 1.2-1.5 g / kg / day. These values result from impaired protein consumption and aminoaciduria and severity of decline. Fats are administered in a dose of 1 g / kg m.c. / Day. In the case of liver failure associated with cholestasis, the supply should be reduced to 0.8 g / kg / day (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książczyńska, 2010; Gajewska, 2009).

It is recommended to split meals for four to six a day, eaten in the same time interval (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książczyńska, 2010).

If you have varicose veins of the esophagus, the consistency of the diet should be modified. In this case, avoid harsh and hard vegetables or fruit, meat, with significant amounts of connective tissue and hard bread. Patients with ascites preparations should use high energy, but with a low content of sodium in order to reduce the retention of body fluid. Malnourished patients should be supplemented electrolytes and vitamin deficiency (Kaniowska and Grzymisławski, 2008; Ciborowska and Rudnick, 2009; Hryniewiecki, 2003; Książczyńska, 2010).

However, the primary method of treatment of patients with decompensated cirrhosis of the liver, liver transplantation is still (Murray and Carithers, 2005)

Conclusions

Cirrhosis is a chronic condition in which the liver parenchyma is incorrect reconstruction. The consequence of this situation is a dysfunction of the organ, which is not only a threat to our health, but often also for life. Of great importance in the treatment of this pathology plays a proper diet. Scrupulous observance of the correct principles of healthy eating in a short time can slow or even halt further progression of the disease.

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