WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 14, Issue 11, 44-59. Review Article ISSN 2277–7105

VITAMIN B6 DEFICIENCY AND ITS LONG TERMS EFFECTS ON PATIENTS WITH LIVER DISEASES

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Article Received on
04 April 2025,

Revised on 04 May 2025,
Accepted on 24 May 2025

DOI: 10.20959/wjpr202511-36652

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ABSTRACT

The liver is a important gland in the body and play role in the body functions such as blood clotting and various metabolism. Most people are taking illnesses substances which cause liver damage. Liver disease is defined as acute or chronic liver on the basis of whether the history of disease is less than or greater than 6 months respectively and disturbance of liver functions that causes liver damage. The various liver diseases have various sign symptoms and causes. The mainly liver cirrhosis is irreversible disease. In cirrhosis replacement of liver tissue by fibrosis, scar tissue and regenerate nodules causes loss of liver function. The etiological of which involves Alcohol consumption, Hepatitis A, B and C, Non alcoholic fatty liver disease and Primary Biliary Cholangitis. History and physical exam, laboratory test and imaging test are the diagnosis test of cirrhosis. Vitamin B6 is a water soluble Vitamin. There are three different natural form of vitamins b6 pyridoxine, pyridoxamine and pyridoxal which normally present in foods. Humans are depend on external sources to cover their vitamin B6 requirements and the pyridoxal 5 – phosphate (PLP) form is of major importance.

Objective

1. To study the sign, symptoms and effects of vitamin B6 deficiency on body.
2. To study on various liver diseases and specially in liver cirrhosis
3. Evaluate the effect of vitamin b6 deficiency on liver.
4. To study long term effects vitamin B6 deficiency on cirrhosis.

KEYWORDS: liver disease, vitamin B6 deficiency, cirrhosis, amino acid metabolism.

INTRODUCTION

The liver is a important gland in the body. It lies in the upper quadrant of the abdominal and weight about 1.5 kg. It play important role in body functions such as blood clotting, cholesterol glucose metabolism or iron metabolism, protein production reduces oxidative stress.[2]

Most people are taking illnesses substances which cause liver damage. **Liver disease is defined as acute or chronic liver on the basis of whether the history of disease is less than or greater than 6 months respectively and disturbance of liver functions that causes liver damage.** The liver lose the functions in the body by injury. There are many type of liver disease that can caused by virus, damage from drug and chemical, obesity, attacks for own immune system that can permanently damage the liver or bile duct. Liver disease is major cause of death every year approximately 29 million people suffer various liver problems. Number of liver disease are occur by deficiency of Vitamin B6 and augmented level of bilirubin in the body. It is the result of degradation of haemoglobin of dead red blood cells. The formation of red blood cell depend upon vitamin B6. Vitamin b6 is responsible for formation of red blood cell. Deficiency of Vitamin b6 causes several liver problems which is harmful. Hepatitis is inflammation of liver. Inflammation is swelling that happens when tissue of the body are injured or infected. It is causes by viral infection. Hemochromatosis is caused by more absorption of iron or excess iron stored in liver. Wilson's disease is a genetic disorder which excess copper store in the body. This disease occurs in between age of 5 to 35. Autoimmune liver disease is immune system starts attacking the liver then result loss of functions and inflammation of the liver. Liver cirrhosis is a late stage of scarring or fibrosis of liver. It is happens by many forms of liver disease.[1,2]

Sign and symptoms of liver disease: Liver disease doesn't always cause noticeable signs and symptoms.[3]

- Skin and eyes appear yellowish
- Abdominal pain and swelling
- Swelling in the legs and ankles
- Itchy skin
- Dark urine color
- Pale stool color
- Chronic fatigue
- Nausea or vomiting
- Loss of appetite
- Decreased energy or weakness and Jaundice

Risk factors

- Heavy alcohol consumption
- Obesity and Type 2 diabetes
- Tattoos and body piercing
- Injecting drugs by the using shared needles
- Family history of liver disease
- Exposure to certain chemicals or toxins
- Sexual activity
- Accidental exposure to blood or needlee tick
- Personal and daily habits.[3]

Vitamin are organic nutrient tests are essential for life. Body needs vitamin to function properly, we cannot produce most vitamin in body. They have to obtained through the food we eat. Vitamins are fatty liver and water soluble. The fat-soluble vitamins are A, D, E and K. The others are water soluble this are Vitamin C and B complex switch consisting of Vitamins B1,B2, B6,B12, niacin, folic acid , biotin, pantothenic acid, choline.[7]

Vitamin B6 is a water soluble Vitamin . There are three different natural form of vitamins b6 pyridoxine, pyridoxamine and pyridoxal which normally present in foods. Humans are depend on external sources to cover their vitamin B6 requirements and the pyridoxal 5 – phosphate (PLP) form is of major importance. Pyridoxal 5 – phosphate and pyridoxamine 5 phosphate are the active coenzyme forms of vitamin B6. Vitamin B6 coenzyme forms perform wide variety of functions in the body. It is extremely versatile and including in more

than 100 enzyme reactions, mostly in protein metabolism. Conversion of amino acid tryptophan to niacin or to the neurotransmitter serotonin also depend on PLP. PLP participate in the synthesis of the heme compound in haemoglobin, of nucleic acid in DNA. It is store in muscle tissue. The amount of vitamin B6 in adults age 19 to 64 need is about 1.4 mg /day in men and 1.2 mg /day women.[4,5,6]

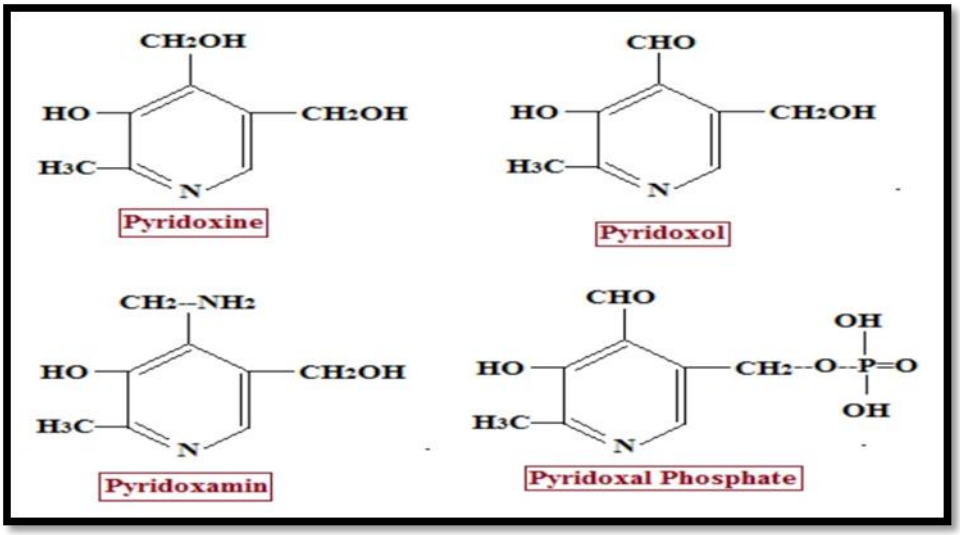

Fig:1 structure vitamin B6.

Source of vitamin B6:- there are many sources of Vitamin B6 including Chicken, Some fish, peanuts, soya beans, wheatgerm, oats, bananas, milk, whole grains, dried beans.

Functions of Vitamin B6

- Vitamin B6 helps the body to make antibodies. It is needed to fight many diseases.
- To maintain normal nerve function and developing of nervous system.
- It helps help to body make hemoglobin. It carries oxygen in the red blood cells to the tissue.
- To control blood sugar in normal ranges.
- Maintaining proper balance of sodium and phosphorus.
- It helps to reducing muscle spasm, cramps and numbness.

Vitamin B6 deficiency :- Vitamin b6 is present in most foods and dietary deficiency through rare. It is caused biochemical change or low concentration of vitamin B-complex and low concentration of PLP. vitamin B6 deficiency is associated with microscopic anemia, electroencephalographic abnormality, dermatitis with scaling in the lips and cracks at the corners of mouth, swollen tongue. In infants vitamin B6 deficiency causes irritability

abnormally acute hearing and convulsive seizures. End-stage renal disease, chronic renal insufficiency and kidney disease can cause vitamin b6 deficiency. In current research deficiency can result from malabsorption syndrome such as celiac disease, crown's disease. Certain genetic disease such as homocystinuria can also cause vitamin B6 deficiency. Some medications such as antiepileptic drugs can leads to deficiency over time.[7]

Causes

- Low vitamin intake
- Pregnancy
- Alcoholics and smoker
- Older people
- Antibiotics
- Diabetes And malabsorption.

Sign and symptoms

- Skin rashes and mood change
- Cracked and sore lips
- Sore and Glossy Tongue
- Weakened Immune function
- Tiredness and Low Energy
- Tingling and Pain in hands and Feet
- Seizures

Content

Liver cirrhosis

Cirrhosis was firstly introduced by Laennec in 1826.The term obtained from Greek word scirrhus on the orange or tawny surface of the liver. The development of liver damage to cirrhosis may occur weeks to year. It is a chronic disease in which diffuse destruction and fibrotic regeneration of hepatic cells and necrotic tissue replaced by fibrotic tissue, normal liver, structure and vasculature is altered, impairing blood and lymph flow. Its result in hepatic insufficiency and Portal hypertension. Cirrhosis is defined as a consequence of chronic liver disease characterized by replacement of liver tissue by fibrosis, scar tissue and regenerate nodules causes loss of liver function. It may arise as a consequence of an exogenous toxic, infectious, autoimmune, vascular process. Patients are firstly diagnosed then

they are identified with changing of the anatomy and function of liver through clinical examination, biochemical tests, imaging and/or histological findings.[8,9]

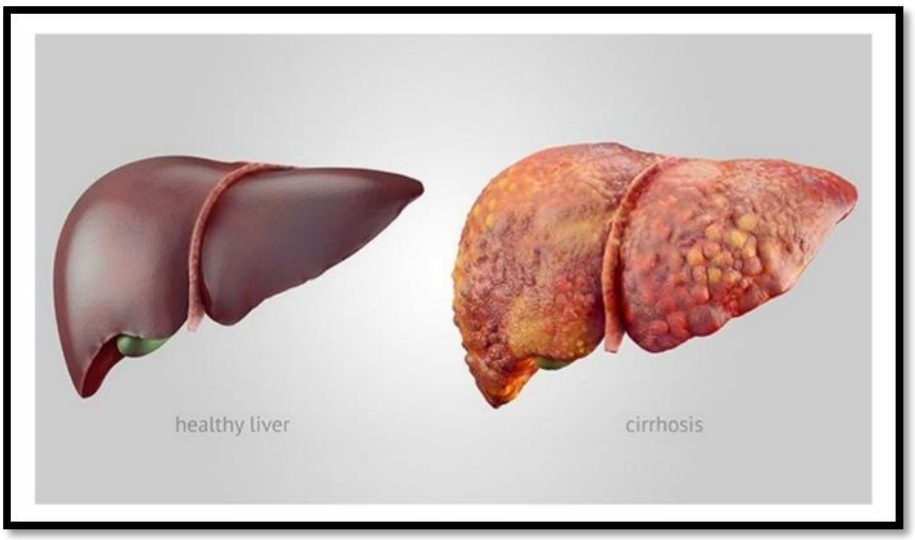

Fig 2: liver cirrhosis.

Etiology of liver cirrhosis

The etiological spectrum of cirrhosis have different among different geographical population worldwide. In various Countries have high alcohol consumption and it has the leading causes of cirrhosis. Countries have alcohol consumption is low or provided have showed Viral hepatitis is has the most common cause of cirrhosis. In India various causes of cirrhosis are found in earlier study hepatitis B was found to be leading causes of cirrhosis in the young also in adult. Regional variation also be observed autoimmunity, Wilson disease and alcohol causes of cirrhosis in India.[11]

1. Alcohol use -: The liver is the first target organ of ethanol to injury because here most of the metabolism occurs. More alcohol usage causes three types of chronic liver disease such as steatosis (fatty liver), steatohepatitis, fibrosis and cirrhosis . Alcohol is most common causes of liver cirrhosis and hepatic transplantation in the world . In alcoholics, other factors interfere in liver damage such as genetic predisposition, female gender, infection with hepatitis B or C virus and malnutrition.
2. Hepatitis C:- Hepatitis C virus (HCV) is an infectious disease which affects the liver and causing acute or chronic hepatitis. The transmission of HCV is occur such as blood transfusions, use of syringes of injecting drug users, use of equipment or medical material without sufficient sterilization, sexual transmission Also cause of transmission during

childbirth. It is a slowly progressive disease which causes persistent inflammation. It will progress to cirrhosis in 20 to 30 years.

3. Hepatitis B :- Hepatitis B virus (HBV) is an infectious disease and causes by fulminant hepatitis, or a chronic condition. It is characterized by constant inflammation which can progress to cirrhosis or hepatocellular carcinoma. HBV is leads to transmitted by direct contact with blood and liquids of body parts such as saliva, semen and vaginal secretions of infected people. It can occur through the use of non-sterile material such as needles, syringes, instruments surgical, sexually or parentally or vertically.
4. Non-alcoholic fatty liver disease :-Non-alcoholic fatty liver disease (NAFLD) is a chronic disease that includes mild conditions of simple steatosis, which have a no impact on morbidity or short-term mortality while a more serious condition such as non-alcoholic steatohepatitis, which is fat accompanied by Inflammatory infiltrate with different degrees of fibrosis causing the liver cirrhosis. NAFLD is linked to obesity and diabetes.
5. Autoimmune diseases:- *T*hey represent etiology of cirrhosis, including autoimmune hepatitis, primary biliary cholangitis, and primary sclerosing cholangitis.

a. Autoimmune hepatitis :- It is the chronic inflammation of the liver that is characterized by the presence of elevated levels of gamma globulins, autoantibodies and hepatitis interface in the histopathological study. The treatment is based on the use of immunosuppressive therapy with corticosteroids.
b. Primary sclerosing cholangitis (PSC) :- It is a chronic disease of unknown etiology. It is specified by an inflammatory reaction and progressive fibrosis of intrahepatic or extrahepatic bile ducts. Related to inflammatory bowel disease since about 70 to 90% of patients with PSC suffer from it.
c. Primary Biliary Cholangitis :- It is a progressive autoimmune liver disease that causes cholestasis and then cirrhosis. The damage is observed in small and medium-sized intrahepatic bile ducts. It causes to biliary excretion and fibrosis. It occurs regularly in women between the fourth and the sixth decade of life. The progress of the disease is slow.
d. Cryptogenic cirrhosis :- Cryptogenic cirrhosis is a diagnosis of exclude of the chronic liver disease. Its etiology could not be determined after perform all the clinical, laboratory and histological . It is mostly occur in NAFLD.

6. Other causes:- Other causes are seen in patients as cardiac conditions, drugs, genetic diseases such as hemochromatosis, Wilson's disease, and alpha 1 antitrypsin deficit.[10]

Pathophysiology of cirrhosis

Liver play an important role in synthesis of protein like albumin, in clotting factors, complement factors and detoxification and storage of vitamin A. It participate in the metabolism of lipids and carbohydrate. cirrhosis is often followed by hepatitis and steatosis independent of the causes. If the coach is resolved at this stage the changes are completely reversible. In cirrhosis scar tissue developed replaced by normal parenchyma and block the portal flow of blood to organ and affect the normal function. Research shows the important role of stellate cell in the development of cirrhosis which generally store vitamin A. Hepatic parenchyma damage due to the inflammation and activate stellate cell and it increases fibrosis and obstructs the blood flow in the circulation. The formation of fibrous tissue bands separate hepatocytes nodules which replace the entire liver architecture. Chronic injury to the liver result in inflammation, necrosis and subsequently fibrosis.[12]

The pathologic features of cirrhosis include when fibrous septa can regenerating nodules. It is loss of the normal lobular architecture within the nodules and cause to reducing blood flow throughout the liver. Spleen congestion causes hypersplenism and increased sequestration of platelets. There are two types of cirrhosis based on A) Micro nodules cirrhosis the regenerate nodules is size less than 3mm and presents in whole liver. It is caused by alcohol induced damage and biliary tract disease. B) Macro nodules cirrhosis are associate various Suze of nodules. The normal acini in the larger nodules and caused by chronic viral hepatitis.[8]

Macroscopically in this stage liver is enlarged and that time progress of disease becomes small. Surface of liver is irregular and color becomes yellow. Three microscopic types depend on size of nodules: a) Micro nodules or Laennec's cirrhosis or Portal cirrhosis is the nodules Size less than 3mm. In macro nodules cirrhosis or post necrotic cirrhosis nodules size is larger than 3mm and mixed cirrhosis have different size of nodules. Different entities can damage the liver by different ways which causes special dysfunction. In cardiac cirrhosis fibrosis in the tissue surrounding the hepatic veins. In primary biliary cirrhosis the fibrosis is present around the bile duct. In alcoholics cirrhosis neutrophils infiltrate occurs in the liver.[12]

Sign and symptoms

- Fatigue
- Easily bleeding

- Loss of appetite and Nausea
- Edema
- Weight loss and ltchy skin
- Yellow discoloration in the skin and eyes
- Spider like blood vessels on the skin
- Redness in the plams of the hands

Diagnosis of liver cirrhosis

History and physical exam

The most of patients with liver cirrhosis are mostly asymptomatic or have nonspecific symptoms like asthenia, weight loss, decreased lividness, among others, which delays the diagnosis.

It is important to research personal history in search of pathologies which predispose to cirrhosis like the presence of metabolic syndrome and autoimmune disorders. You must examine risk factors through an in depth history with specialize in the private and social history of alcohol consumption. Factors related to hepatitis B and C infection (use of injectable drugs, unprotected sex, tattoos, blood transfusions, vaccination), use of hepatotoxic and/or herbal medication, case history of diseases that have a genetic predisposition like hemochromatosis, genetic disorder and alpha-1 antitrypsin deficiency should even be considered within the history taking In the physical examination you will find a selection of manifestations whose presence should make us suspect the disease: asterixis, ascites, collateral abdominal circulation "jellyfish head", spider veins and telangiectasias, palmar erythema, changes within the nails, Dupuytren's contracture, gynecomastia, hepatomegaly, splenomegaly, bruising, testicular atrophy and jaundice.[10]

Imaging Test

- Abdominal computed tomography (CT scan) :- CT scan is combine procedure for specially x-ray equipment with sophisticated computer. It produce multiple, digital images or pictures of the liver. CT scan can help determine the grimness of cirrhosis and other liver diseases.[12]
- Abdominal Ultrasound :- It is a type of imaging exam. In this sound waves are used to create pictures of the inside of the abdomen and pelvis as well as images of the liver. Doppler ultrasound is used for evaluation of blood flow to and from the liver.

- Elastography:- This can examine the stiffness of liver. It can help diagnose how severe the scarring is in your liver known as liver fibrosis. It is untreated cause liver fibrosis can eventually lead to cirrhosis of the liver which is irreversible. Elastography can help detect stiffness of the liver caused by liver fibrosis earlier than other imaging tests. The test can be take place by ultrasound or MRI.[12]
- Body magnetic resonance imaging (MRI) :- This imaging exam uses a powerful magnetic field, radio frequency pulses and a computer. It can produce detailed image of the liver allow for measurement of damage caused by various liver diseases.[12]
- Magnetic resonance cholangiopancreatography (MRCP) :- It is special type of MRI protocol It is designed for to detect infected a part of the liver and gallbladder. The biliary system that is part of your liver. It may be used to help determine the reason of: jaundice; pancreatitis.

Laboratory tests

The laboratory tests is help to detect liver injury and determine devolvement of scarring tissue. This test is help to evaluate severity liver disease, particularly if the individual has some risk factor for developing cirrhosis. This tests may be to help diagnose the various cause and to monitor the affected person's health over time. This can include to diagnose the progress hepatocellular carcinoma.

Liver damage is first found by comprehensive metabolic panel (CMP) or a liver panel. It involving the following tests

- **Alanine aminotransferase (ALT)**:-ALT is an enzyme present the liver. The values of ALT are increased which causes the liver injury mainly including cirrhosis.[13]
- **Aspartate aminotransferase (AST)**:- AST is enzyme present in the liver as well as other body organs. AST is measured in people with liver injury, including cirrhosis.[13]
- **Alkaline phosphatase (ALP):**-ALP is enzyme present along bile ducts. ALP is usually normal or mildly measured in cirrhosis.[13]
- **Total bilirubin:-** Bilirubin is a substance formed only in the liver. It is increased with responsible many liver diseases. Bilirubin is usually normal or slightly measured until cirrhosis becomes far advanced.[13]
- **Albumin**:- Albumin is protein made in the liver . It is on decreased in cirrhosis.

- **Complete Blood Count (CBC):-** Complete blood count test is help to measure the red blood cell, white blood cells and platelets. Resulting the anemia may be present and if bleeding has occurred, and platelets are often decreased with cirrhosis.
- **Prothrombin Time (PT/INR) :-**Prothrombin is most clotting factors are produced by the liver. This test evaluates clotting function and results could also be prolonged with cirrhosis.[13]
- **Liver biopsy** It is involves taking a sample of liver tissue to judge the structure and cells of the liver. It can clearly indicate the presence of cirrhosis, but since the sample is small, a negative result might not rule cirrhosis out.[8]

Treatment

Pharmacologic treatment

Specific medical therapies could also be applied to many liver diseases so as to alleviate symptoms and primarily to avoid or delay the event of cirrhosis. Examples include prednisone and azathioprine for autoimmune hepatitis, interferon and other antiviral agents for viral hepatitis and C phlebotomy for hemochromatosis, ursodeoxycholic acid for primary biliary cirrhosis, and trientine and zinc for Wilson disease. These therapies gradually become less effective as the chronic disease progresses to cirrhosis. AS cirrhosis develops, treatment is aimed at controlling complications as they arise. Certainly, bleeding from varicose veins, ascites, and hepatic encephalopathy are among the major serious complications experienced by cirrhotic patients. However, one should also pay attention to the patient's chronic constitutional complaints.[14]

Analgesics

The use of painkillers in patients with liver cirrhosis may be problematic. Most hepatologists allow acetaminophen doses up to 2000 mg\/day for patients with liver cirrhosis. The use of non-steroidal anti-inflammatory drugs in patients with liver cirrhosis can cause gastrointestinal bleeding. Due to inhibition of prostaglandins and impaired renal blood flow, patients with liver cirrhosis are at risk of NSAID-induced renal failure. Patients with hepatic encephalopathy should use opioid analgesics with caution, otherwise they may worsen potential mental function. Many prescription drugs and over-the-counter drugs. The drug can be used as a combined preparation. Patients with liver cirrhosis should pay attention to reading. Before starting any new medicine, carefully label the medicine to avoid accidental overdose.[12]

Zinc deficiency

Zinc deficiency is common in cirrhosis patients. Zinc sulfate treatment at 220 mg orally twice a day can improve digestive disorders and may stimulate appetite. If not, Zinc is effective in treating muscle cramps and is an adjuvant for the liver encephalopathy.[14]

Drug Hepatotoxicity in the Patient with Cirrhosis

Drugs linked to drug-induced liver disease including NSAIDs, isoniazid, valproic acid, erythromycin, Amoxicillin clavulanate, Ketoconazole, Chlorpromazine and Ezetimibe. Statins are commonly associated with increased levels of alanine aminotransferase and should safely used in patients with chronic liver disease.[12]

Liver transplantation

Liver transplantation has become an important strategy in the management of cirrhotic patients. Patient should be referred for examination for the liver transplant after the first sign of liver shift. Advances in surgical techniques, organs improved preservation and immunosuppression survival after surgery. In the early 1980s, the percentage of patients who survived 1 year and 5 years after hepatic resection 70% and 15% transplanted, respectively. Now the patient with 1-year survival rates of around 85-90% and 5 years survival rate greater than 70%. Quality of life after liver the transplant is good or excellent in most cases.[12,3]

Osteoporosis

Patients with liver cirrhosis can develop osteoporosis. Calcium and vitamin D are important for high-risk patients with osteoporosis, especially corticosteroids in chronic patients with primary cholestasis or cirrhosis and autoimmune hepatitis. Bone density studies that explore reduced bone mineralization may lead to tissue treatment with amino bisphosphonates.[14]

Vitamin B6 deficiency long term effect on cirrhosis

About 90% of patients with severe liver cirrhosis are deficient in vitamin B6. Some people think that supplementing these patients with vitamin B6 may cause a false decrease in serum AST concentration.

Vitamin B6 is a water-soluble B-complex vitamin that exists in many foods, such as pyridoxine, pyridoxal, pyridoxamine, and vitamin B6, mainly in the form of its biologically active coenzyme 5'-pyridoxal phosphate Exist and participate in a wide range of biochemical reactions, including amino acid and glycogen metabolism, nucleic acid synthesis,

hemoglobin, sphingomyelin and other sphingolipids. This study evaluated the impact of daily oral pyridoxine supplementation on patients with liver cirrhosis. Eight subjects received 25 mg of pyridoxine for 28 consecutive days. Before and after the supplementation period, the status was assessed by measuring fasting plasma vitamin levels and response to a 25 mg oral pyridoxine load.

Vitamin B, is present within the body in multiple vitamers. the most coenzymatically active being pyridoxal5'-phosphate (PLP). PLP is important to amino acid metabolism by its involvement in transaminase and decarboxylase reactions. It is also important in neurotransmitter, sphingolipid and heme biosynthesis and is an important cofactor for glycogen phosphorylase. Symptomatic B6 deficiency is rare due to the ubiquity of the vitamers in food.

After ingestion, the nonphosphorylated forms of vitamin B, cross the intestinal mucosa by passive diffusion, although some phosphorylation and dephosphorylation may occur within the mucosal cells. The absorbed species are rapidly phosphorylated once they reach the liver by the enzyme pyridoxal kinase. The pyridoxine (PNP) and pyridoxamine 5'-phosphates (PMP) are converted to PLP by a flavin-dependent oxidase. The liver releases PLP and pyridoxal (PL), the major circulating forms, which are tightly bound to albumin and hemoglobin, respectively. Current data indicate that PLP is not directly absorbed by peripheral cells; uptake occurs after hydrolysis to PL by alkaline phosphatase. Regulation of intracellular concentrations could also be associated with the binding of the rephosphorylated PLP to intracellular proteins during a complex which isn't readily hydrolyzed. Hepatic and renal aldehyde oxidases convert PL to 4-pyridoxic acid (4-PA), which is that the major form excreted within the urine. Patients with cirrhosis display deranged B6 metabolism. Fasting plasma PLP levels are significantly decreased and hepatic PLP is low. This discrepancy is thought to be due to heightened degradation of PLP rather than decreased hepatic production. The cirrhotic liver has normal levels of biosynthetic enzymes and a traditional rate of conversion of pyridoxine (PN) to 4-PA, but a rise in hepatic and serum alkaline phosphatase is found in cirrhosis. In addition, the plasma AUCpL of patients with liver cirrhosis is significantly higher. Therefore, the response to pyridoxine load supports the hypothesis that PLP degradation is increased in cirrhosis.

In cirrhosis, normal amino acid metabolism is disrupted. Alterations in plasma levels of aromatic and branched-chain amino acids, hyperammonemia and accumulation of plasma

mercaptans have all been implicated in the pathogenesis of hepatic encephalopathy. Horowitz et al. demonstrated that the plasma clearance of methionine was diminished in cirrhotic patients after an oral methionine load. Because no plasma or urinary intermediates accumulated in association with the hypermethioninemia, they suggested that the dominant block in the degradation pathway was prior to homocysteine synthesis. However, multiple steps in this pathway are PLP dependent and might be expected to respond to B6 supplementation. This study investigates the effects of PN supplementation in a sample of patients with cirrhosis. We examined changes in the response to an oral PN load as a measure of repletion in addition to the determination of' fasting plasma PLP levels. The effects of PN supplementation on amino acid metabolism were determined using oral methionine and protein loads.[16]

Non-alcoholic fatty liver disease

Vitamin B6 (VitB6) can also be a common name with 6 vitamins: pyridoxine (PN), pyridoxal (PL), pyridoxamine (PM) and their respective pyridoxine 5'-phosphate (PNP) Phosphate, pyridoxal 5'-phosphate (PLP) and pyridoxamine 5'-phosphate (PMP). VitB6 is a cofactor for more than 150 enzymatic reactions involving the metabolism of amino acids, glucose and fat. PLP is that the biologically active kind of VitB6 and low levels of plasma PLP are related to disorder, stroke, phlebothrombosis, atrophic arthritis, inflammatory bowel disease, diabetes, and a number of other cancers. VitB6 intake and hepatic steatosis are negatively correlated and NAFLD have been found to have diets low in VitB6. In addition, NAFLD have low levels of plasma VitB6, and VitB6 administration has been shown to ameliorate hepatic lipid accumulation.

Although VitB6 deficiency may be associated with the progression of NAFLD, there is no efficacy of VitB6 with NAFLD. Therefore, study of therapeutic effect of VitB6 administration in patients with NAFLD. There have been no reports of the efficacy of VitB6 use in NAFLD patients, and this study reviews and provides primary known evidence that VitB6 administration improves hepatic lipid deposition in NAFLD patients.

Although the mechanism by which VitB6 enhances lipid accumulation in the liver has not been fully elucidated, a potential mechanism may involve the catabolism of homocysteine (Hcy). PLP, a physiologically active VitB6, functions as a coenzyme of cysteine-b-synthase (CBS) and cystathionine-g-lyase (CGL). Since CBS and CGL contribute to Hcy catabolism, insufficient VitB6 intake leads to Hcy accumulation. Hcy induces protein misalignment in the

endoplasmic reticulum (ER), leading to a stress response from the ER. ER stress induces activation of the transcription factor sterol-reactive protein 1c and induces the formation of de novo lipogenes. Thus, in support of this mechanism, it is believed that VitB6 deficiency induces an accumulation of lipids in the liver.

In NAFLD patients, plasma concentrations of PLP were lower than in healthy patients, while plasma levels of Hcy were higher. Supplementation with B vitamins, including B6, has also been reported to reduce blood levels of Hcy in subjects with one or more components of metabolic syndrome. Therefore, VitB6 supplementation can reduce liver fat through Hcy catabolism. The use of VitB6 significantly improved the accumulation of fat in the liver. As VitB6 is a reasonable agent with few side effects, it may be a possible new therapeutic agent for the treatment of NAFLD. Large-scale, long-term randomized controlled trials are needed to verify the therapeutic efficacy of VitB6 in NAFLD patients.[17]

CONCLUSION

Cirrhosis represents a common histological approach Various chronic liver diseases. Damage to Liver cells cause changes in liver function. Liver cirrhosis It is mainly diagnosed by liver biopsy and other serological test and Laboratory testing. At first, the liver the disease usually responds to treatment, but in advanced liver disease damage due to cirrhosis, cirrhosis and irreversible liver failure. This advanced stage leads to the end dead. When diagnosing liver disease, the condition causing the disease must be treated. Yes With early detection and proper treatment, liver damage can heal. In the middle stage of the disease, treatment can help heal the damage, but as the disease progresses, Therapeutic approaches focus on disease management and prolongation of diagnosis. vitamin B6 deficiency can be corrected in patients with cirrhosis using daily oral supplements of 25 mg PN. Although we have observed no direct benefit in amino acid metabolism, further studies are necessary to examine changes in other areas of B6 activity.

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