

Assessment of Dietary Management of Patients with Cirrhosis Liver

Shadia Mohamed Idris¹, Ebtesam AL Ali²

¹University of Bahri, Sudan

²University of Hail, KSA

Abstract: *It is very important to maintain proper nutrition and appropriate to the case of cirrhosis of the liver. The objective of this study was designed to assess the dietary intake liver cirrhosis patient and highlighting the dietary. Management during the one set of disease. Design this research study the assessment of dietary management of liver disease among patient in Hail to make dietary management practice more applicable of grate use for all family member of the liver disease patient. 28 Questionnaire was set to collect information from patients with liver disease / 2013. Patient with liver Cirrhosis are interview to collect information about the dietary intake. It was found that the majority of the respondents in these study their age between (21-40y). 64% most were Male 86% with education up to (Academic), 46% were smoker, 61% with economic status [Medium 2000 - less than 5000], and 50% were employed, 54% were Married. Conclusion The cases of this disease, showed poor eating habits, lack of interest in the nutritional side and dependence on junk food, and lack of exercise activities which may affecting the level of treatment on the nutritional side.*

Keyword: Liver Cirrhosis, Nutritional management, Sudan

1. Introduction

1.1 Structure

The liver is the largest gland in the body, weighing about 1500 g. The liver has two main lobes: the right and left. The right lobe is further divided into the interior and posterior segments; the right segmental fissure, which cannot be seen externally, separates the segments. The externally visible calceiform ligament divides the left lobe into the medial and lateral segments. The liver is supplied with blood from two sources: the hepatic artery which supplies about one third of the blood from the aorta; and the portal vein, which supplies the other two thirds and collects blood drained from the digestive tract [1].

About 1500 ml of blood per minute circulates through the liver and exits via the right and left hepatic veins into the inferior vena cava. Just as there is a system of blood vessels throughout the liver, there also exists a series of bile ducts. Bile, which is formed in the liver cells, exits the liver through a series of bile ducts that increase in size as they approach the common bile duct. It is a thick, viscous fluid secreted from the liver stored in the gallbladder and released into duodenum when fatty foods enter the duodenum. It emulsifies fats in the intestine and forms compounds with fatty acids to facilitate their absorption [1].

1.2 Functions

The liver has the ability to regenerate itself. Only 10% to 20% of functioning liver is required to sustain life, although removal of the liver will result in death within 24 hours. The liver is integral to most metabolic functions of the body and performs more than 500 tasks. The main functions of the liver include metabolism of carbohydrate, protein, and fat; storage and activation of vitamins and minerals; formation and excretion of bile; conversion of ammonia to urea; metabolism of steroids; and action as a filter and flood chamber. The liver plays a major role in carbohydrate metabolism. Galactose and fructose, products of

carbohydrate digestion, are converted into glucose in the hepatocyte or liver cell [1] [2].

The liver stores glucose as glycogen [glycogenesis]. and then returns it to the blood when glucose levels become low [glycogenolysis]. The liver also produces "new" glucose [gluconeogenesis]. from precursors such as lactic acid, glycolytic amino acids, and intermediates of the tricarboxylic acid cycle. Important protein metabolic pathways occur in the liver. Transamination and oxidative deamination are two such pathways that convert amino acids to substrates that are used in energy and glucose production as well as in the synthesis of nonessential amino acids [1].

Blood-clotting factors such as fibrinogen; prothrombin; and serum proteins, including albumin, α -globulin, β -globulin, transferrin, ceruloplasmin, and lipoproteins are formed by the liver. Fatty acids from the diet and adipose tissue are converted in the liver to acetyl-coenzyme A [CoA]. by the process of β -oxidation to produce energy. Ketone bodies are also produced. The liver synthesizes and hydrolyzes triglycerides, Phospholipids, cholesterol, and lipoproteins as well.

The liver is involved in the storage, activation, and transport of many vitamins and minerals. It stores all the fat soluble vitamins in addition to vitamin B12 and the minerals zinc, iron, copper and magnesium. Hepatically synthesized proteins transport vitamin A, iron, zinc, and copper in the bloodstream. Carotene is converted to vitamin A, folate to 5-methyl tetrahydrofolic acid, and vitamin D to an active form [25-hydroxy-cholecalciferol]. by the liver [1] [2],

In addition to functions of nutrient metabolism and storage, the liver forms and excretes bile. Bile salts are metabolized and used for the digestion and absorption of fats and fat-soluble vitamins. Bilirubin is a metabolic end product from red blood cell destruction; it is conjugated and excreted in the bile.

Hepatocytes detoxify ammonia by converting it to urea, 75% of which is excreted by the kidneys. The remaining urea finds its way back to the gastrointestinal tract. The liver also metabolizes steroids. It inactivates and excretes aldosterone, glucocorticoids, estrogen, progesterone, and testosterone. It is responsible for the detoxification of substances including drugs and alcohol.

Finally, the liver acts as a filter and flood chamber by removing bacteria and debris from blood through the phagocytic action of Kupffer cells located in the sinusoids and by storing blood backed up from the vena cava as in right heart failure.[3].

Diseases of the liver can be acute or chronic, inherited or acquired. Liver disease is classified in various ways: acute viral hepatitis, fulminant hepatitis, chronic hepatitis, nonalcoholic steatohepatitis [NASH], alcoholic hepatitis and cirrhosis, cholestatic liver diseases inherited disorders, and other liver disease.[3].

Cirrhosis is a slowly progressing disease in which healthy liver tissue is replaced with scar tissue, eventually preventing the liver from functioning properly. The scar tissue blocks the flow of blood through the liver and slows the processing of nutrients, hormones, drugs, and naturally produced toxins. It also slows the production of proteins and other substances made by the liver. Anything that damages the liver can cause cirrhosis including [3].

1.3 Cholestatic Liver Diseases

1.3.1 Alcoholic Cirrhosis

Clinical features of the third stage, alcoholic cirrhosis vary. Symptoms can mimic those of alcoholic hepatitis; or patients can develop gastrointestinal bleeding, hepatic encephalopathy, or portal hypertension [i.e. elevated blood pressure in the portal venous system caused by the obstruction of blood flow through the liver]. and other symptoms of liver disease. They can also develop ascites, the accumulation of fluid, serum protein, and electrolytes within the peritoneal cavity caused by increased pressure from portal hypertension and decreased production of albumin [which maintains serum colloidal osmotic pressure]. A liver biopsy usually reveals micro nodular cirrhosis, but it can be macro nodular or mixed. Prognosis depends on abstinence from alcohol and the degree of complication, already developed Ethanol ingestion creates specific and severe nutritional abnormalities [3].

Steatorrhea resulting from bile acid deficiency is also common in alcoholic liver disease affecting fat-soluble vitamin absorption. Vitamin A deficiency can lead to night blindness [22] [23]. Thiamin deficiency is the most common vitamin deficiency in alcoholics [22]. Folate deficiency can occur as a result of poor intake, impaired absorption, accelerated excretion, and altered storage and metabolism. Inadequate dietary intake and interactions between pyridoxal-5'-phosphate [active coenzyme of vitamin B6]. and alcohol reduce vitamin B6 nutritive [23].

Deficiency of all B vitamins and vitamins C, D, E, and K is also common [22]. Hypocalcemia, hypomagnesaemia, and hypophosphatemia are not uncommon among alcoholics;

furthermore, zinc deficiency and alterations in other micronutrients can accompany chronic alcohol intake [22].

1.3.2 Primary Biliary Cirrhosis

Primary biliary cirrhosis [PBC] is a chronic cholestatic disease caused by progressive destruction of small and intermediate-size intra-hepatic bile ducts. PBC typically presents with a mild elevation of liver enzymes with physical symptoms of pruritus and fatigue. Treatment with ursodeoxycholic acid can slow progression of the disease. Several nutritional complications from cholestasis occur with PBC, including osteopenia, hypercholesterolemia, and fat-soluble vitamin deficiencies [4].

1.3.3 Sclerosing Cholangitis

Sclerosing cholangitis is another chronic cholestatic liver disease. Fibrosing inflammation of segment of extra hepatic bile ducts, with or without involvement of intrahepatic ducts, characterizes the disease. Progression of the disease leads to complications of portal hypertension, hepatic failure [liver function diminished to 25% or less], and cholangiocarcinoma. Primary sclerosing cholangitis [PSC] is the most common type of sclerosing cholangitis [4].

In general, PSC lacks any apparent etiology and usually occurs in association with inflammatory bowel disease. Like PBC, PSC may be an immune disorder because of its strong association with human leukocyte antigen haplotypes, auto antibodies, and multiple immunologic abnormalities. Seventy to 90% of patients with PSC also have inflammatory bowel disease. Patients with PSC are also at increased risk of fat-soluble vitamin deficiencies resulting from steatorrhea associated with this disease [5].

1.3.4 Hepatic Osteodystrophy

Hepatic osteodystrophy may occur from vitamin D and calcium malabsorption, resulting in secondary hyperparathyroidism and osteomalacia or rickets [7].

No treatment slows progression of the disease or improves survival. Ursodeoxycholic acid may improve laboratory values [serum bilirubin, alkaline phosphates and albumin]. but has no effect on survival [6].

1.3.5 Inherited Disorders

Inherited disorders of the liver include hemochromatosis, Wilson's disease, α -1-antitrypsin deficiency, protoporphyria, cystic fibrosis, glycogen storage disease, amyloidosis, and sarcoidosis. The first three disorders are the most commonly inherited disorders resulting in liver failure. Hemochromatosis is an inherited disease of iron overload patients with hereditary hemochromatosis absorb excessive iron from the gut and may store 20 to 40 g of iron compared with 0.3 to 0.8 g in normal persons.

A gene, HFE, is associated with hereditary hemochromatosis [8]. Hepatomegaly, esophageal varices, ascites impaired hepatic synthetic function, abnormal skin pigmentation, glucose intolerance, cardiac involvement, hypogonadism, arthropathy, and hepatocellular carcinoma may develop. Early diagnosis includes clinical, laboratory and pathologic testing, including elevated serum transferrin levels. Increased transferrin saturation are suggestive of

hemochromatosis [8]. Life expectancy is normal if phlebotomy is initiated before the development of cirrhosis or diabetes mellitus [9].

1.3.6 Wilson's Disease

Wilson's disease is an autosomal-recessive disorder associated with impaired biliary copper excretion. Copper accumulation various tissues including the liver, brain, cornea, and kidneys. Low serum ceruloplasmin levels and the presence of Kayser-Fleischer rings, [greenish yellow pigmented rings encircling the cornea just within the corneal margin, formed by copper deposits], confirm the diagnosis, although patients with this disease may consult a physician before these confirming symptoms develop. Patients can present with acute, fulminant, or chronic active hepatitis. Liver and neurologic signs may be the first signs of illness [10].

Copper-chelating agents and possibly zinc supplementation [to inhibit intestinal copper absorption and binding in the liver], are used to treat Wilson's disease once it is diagnosed. Copper chelation improves survival but does not prevent cirrhosis. Transplantation corrects the metabolic defect [8]. A low-copper diet is implemented if other therapies are unsuccessful. If this disease is not diagnosed until onset of fulminant failure, survival is not possible without transplantation [11].

1.3.7 Other Liver Diseases

Liver disease has several other causes. Liver tumors can be primary or metastatic, benign or malignant. Hepatocellular carcinoma usually develops in cirrhotic livers. The highest risk occurs in those with HBV, HCV and hereditary hemochromatosis [12]. The liver can be affected when there is systemic disease such as rheumatoid arthritis, systemic lupus erythematosus, polymyalgia, rheumatic orotemporal arthritis, polyarteritis nodosa, systemic sclerosis [12].

1.4 Diagnosis

Cirrhosis of the liver is diagnosed through several methods:

- Physical exam. During a physical exam, doctor can observe changes in how liver feels or how large it is [a cirrhotic liver is bumpy and irregular instead of smooth].
- Blood tests. If doctor suspects cirrhosis, you will be given blood tests to find out if liver disease is present.
- Other tests. In some cases, other tests that take pictures of the liver are performed, such as a computerized tomography [CT scan], ultrasound, or another specialized procedure called a radioisotope liver/spleen scan.
- Biopsy. doctor may decide to confirm the diagnosis by taking a sample of tissue [biopsy], from the liver.
- Surgery. In some cases, cirrhosis is diagnosed during surgery when the doctor is able to see the entire liver. The liver also can be inspected through a laparoscope, a viewing device that is inserted through a tiny incision in the abdomen.[13].

It is very important to maintain proper nutrition and appropriate to the case of cirrhosis of the liver. Cirrhosis is a case in which the patient is suffering from edema, malnutrition, lack of energy and vitamins. So it is important to consult a dietitian and keep on feeding grounds [1].

1.5 Complications

- Variceal bleeding. Variceal bleeding is caused by portal hypertension, which is an increase in the pressure within the portal vein [the large vessel that carries blood from the digestive organs to the liver]. This increase in pressure is caused by a blockage of blood flow through the liver as a result of cirrhosis. Increased pressure in the portal vein causes other veins in the body to enlarge [varices], such as those in the esophagus and stomach, to bypass the blockage. These varices become fragile and can bleed easily, causing severe hemorrhaging and fluid in the abdomen [13].
- Confused thinking and other mental changes [hepatic encephalopathy]. Hepatic encephalopathy most often occurs when cirrhosis has been present for a long time. Toxins produced in our intestines are normally detoxified by the liver, but once cirrhosis occurs, the liver cannot detoxify as well. Toxins get into the bloodstream and can cause confusion, changes in behavior, and even coma [13].

1.6 Other Serious Complications of Cirrhosis of the Liver Include

- Kidney failure
- Reduced oxygen in the blood
- Diabetes
- Changes in blood counts
- Increased risk of infections
- Excessive bleeding and bruising
- Breast enlargement in men
- Premature menopause
- Loss of muscle mass

Most of these complications can initially be treated with medicines or dietary changes. Once treatment for these complications becomes ineffective, a liver transplant is considered. Almost all of the complications can be cured by liver transplantation; however, in many circumstances, careful management can reduce the harmful effects of cirrhosis and delay or even prevent the need for a liver transplant [13].

1.7 Treatment

Although there is no cure for cirrhosis of the liver, there are treatments available that can stop or delay its progress, minimize the damage to liver cells, and reduce complications. The treatment used depends on the cause of cirrhosis of the liver. Cirrhosis has many clinical manifestations. Several major complications of cirrhosis and end-stage liver disease [ESLD], including malnutrition, ascites, hyponatremia, hepatic encephalopathy, glucose alterations, fat malabsorption, hepatorenal syndrome, and osteopenia have nutritional implications. When appropriate nutrition therapy is provided to patients with liver disease, malnutrition can be reversed, and clinical outcomes improved [14] [15].

1.9 Nutrition Assessment

Before appropriate nutrition therapy can be implemented, a nutrition assessment must offer to determine the extent and

cause of malnutrition. Objective parameters that may be helpful when monitored serially include anthropometric measurements and dietary intake evaluation [18] [19]. The best way to perform a nutrition assessment may be to combine these parameters with the subjective global assessment [SGA]. approach. The SGA has been used to evaluate patients with liver disease and transplantation and has demonstrated an acceptable level of reliability and validity [16] [17].

1.9.1 Problems in Feeding

Because anorexia, nausea, dysgeusia, and other gastrointestinal symptoms are common, adequate nutrition intake is difficult to achieve. With ascites, early satiety is also a frequent complaint. Smaller, more frequent meals are better tolerated than three traditional meals. In addition, evidence suggests that frequent feedings also improve nitrogen balance and prevent hypoglycemia. Oral liquid supplements should be encouraged, and, when necessary enteral tube feedings used. Adjunctive nutrition support should be given to malnourished patients with liver disease if their intake is less than DRI levels of 0.8 g of protein and 30 calories per kilogram of body weight daily and if they are at risk for fatal complications from the disease. Esophageal varices are usually not a contraindication for tube feeding [20].

1.9.2 Nutrient Requirements

- Energy
Energy requirements vary among patients with cirrhosis. In general, energy requirements for patients with ESLD and without, ascites are about 120% to 140% of the REE. Requirements increase to 150% to 175% of REE if ascites, infection, or malabsorption is present or if nutritional repletion is necessary. This equates to about 25 to 35 calories per kilogram body weight; estimated dry body weight should be used in calculations to prevent overfeeding. Oral nutritional supplements or tube feeding can be effective in increasing or ensuring optimal intake in malnourished patients and reducing complications and prolonging survival [21].
- Carbohydrates
Determining carbohydrate needs is often challenging in liver failure because of the primary role of the liver in carbohydrate metabolism. Liver failure reduces glucose production and peripheral glucose use. The rate of gluconeogenesis is decreased with preference for lipids and amino acids for energy. Alterations in the hormones insulin, glucagon, cortisol, and epinephrine are responsible in part for the preference for alternative fuels.[21].
- Lipid
In cirrhosis, plasma free fatty acids, glycerol, and ketone bodies are increased in the fasting state. The body prefers lipids as an energy substrate, and lipolysis is increased with active mobilization of lipid deposits, but the net capacity to store exogenous lipid is not impaired. A range of 25% to 40% of calories as fat is generally recommended [21].

- Protein
Protein is by far the most controversial nutrient in liver failure, and its management is also the most complex. Cirrhosis has long been thought of as a catabolic disease with increased protein breakdown and inadequate resynthesis, resulting in depletion of visceral protein stores and muscle wasting. Protein kinetic studies have been able to demonstrate increased nitrogen losses only in patients with fulminant hepatic failure or decompensate disease but not in patients with stable cirrhosis [25]. Patients with cirrhosis also have increased protein use. At least one study [26], suggests that 0.8 g of protein per kilogram per day is the mean protein requirement to achieve nitrogen balance in patients with stable cirrhosis. Therefore, in uncomplicated hepatitis or cirrhosis without encephalopathy, protein requirements range from 0.8 to 1 g/kg of dry weight per day to achieve nitrogen balance. To promote nitrogen accumulation or positive balance, at least 1.2 to 1.3 g/kg daily is needed [26]. In situations of stress such as alcoholic hepatitis or decompensate disease [sepsis, infection, gastrointestinal bleeding, severe ascites], at least 1.5 g of protein per kilogram per day should be provided.[25].

- Vitamins and Minerals
Vitamin and mineral deficiencies occur in alcoholic liver disease as a result of reduced intake and alterations in absorption, storage, and ability to convert the nutrients to their active forms [22]. Vitamin and mineral supplementation is needed in all patients with ESLD because of the intimate role of the liver in nutrient transport, storage, and metabolism, in addition to the side effects of drugs. Vitamin deficiencies can contribute to complications deficiency of pyridoxine, thiamin, or vitamin B12 can result in neuropathy. Confusion and ocular disturbances can result from a thiamin deficiency; impaired dark adaptation can occur from vitamin A deficiency; and hepatic osteodystrophy or osteopenia can develop from vitamin D deficiency [23].

Deficiencies of fat-soluble vitamins have been found in all types of liver failure, especially in cholestatic diseases in which malabsorption and steatorrhea occur. Therefore supplementation is necessary using water-soluble forms' Intravenous or intramuscular vitamin K is often given for 3 days to rule out vitamin K deficiency as the cause of a prolonged prothrombin times [22] [23]. Water-soluble vitamin deficiencies associated with alcoholic liver disease include thiamin [which can lead to Wernicke's encephalopathy], pyridoxine [B6], cyanocobalamin [B12], folate, and niacin [B3]. Large doses [100 mg]. of thiamin are given daily for a limited time if deficiency is suspected [23].

Mineral nutritive is also altered in liver disease. Iron stores may be depleted in patients experiencing gastrointestinal bleeding; however, iron supplementation should be avoided by persons with hemochromatosis or hemosiderosis [24]. Elevated serum copper levels are found in cholestatic liver diseases. Because copper and manganese are excreted primarily via bile, supplements should not contain these minerals. Wilson's disease is a disorder of abnormal copper metabolism in which urinary excretion is high, serum levels are low, and excess copper in various organs causes severe damage. Oral chelating agents such as zinc acetate or /d-

penicillamine are the primary treatment. A vegetarian diet may be useful as adjunctive therapy because copper is less available [27]. Dietary copper restriction is not routinely prescribed unless other therapies are unsuccessful.

Zinc and magnesium levels are low in liver disease related to alcoholism, in part because of diuretic therapy. Calcium, as well as magnesium and zinc, may be malabsorbed with steatorrhea. Therefore the patient should take supplements of these minerals at least at the level of the DRI [27]. Medications may be given to control the symptoms of cirrhosis. Edema [fluid retention] and ascites [fluid in the abdomen] are treated, in part, by reducing salt in the diet. Drugs called diuretics are used to remove excess fluid and to prevent edema from recurring. Diet and drug therapies can help improve the altered mental function that cirrhosis can cause. Laxatives such as lactose may be given to help absorb toxins and speed their removal from the intestines [13].

Liver transplantation may be needed for some people with severe cirrhosis [13].

1.10 Dietary management

1.10.1 Prevention

There are several ways to reduce risk of developing cirrhosis as following

- a. Don't abuse alcohol. If you do drink alcohol, limit how much you drink and how often. Remember it's not only the heavy drinker who gets cirrhosis. If you drink more than 2 drinks a day, you are increasing your risk. A drink is a 5 oz glass of wine, 12-oz can of beer, or a 1 1/2 oz portion of hard liquor.
- b. Avoid high-risk sexual behavior such as unprotected sexual contact with multiple partners.
 - Be careful around synthetic chemicals, such as cleaning products and pesticides. If you come into contact with chemicals often, wear protective clothing and a facemask.
 - Get vaccinated against hepatitis B.
 - Eat a well-balanced, low-fat diet and take vitamins.[13].

2. Results & Discussion

Table 1: Socio-demographic characteristics n [%]

Characteristics		Total
Sample Size		28 [100]
Age Groups [year]	20-Oct	3 [10.7]
	21-40	14[50]
	≥ 41 years	11[39.3]
Sex	Male	18[64.3]
	Female	10[35.7]
Education	School	4[14.3]
	University	24 [85.7]
Employment	Employed	15 [53.6]
	Unemployed	13 [46.4]
Smoke	Yes	16[57.1]
	No	12[42.9]
economic status	Medium	17[60.7]
	High	11[39.3]
Marital Status	Married	15[53.6]
	Unmarried	13[46.4]

Table 1 Shows that the majority of the respondents in these study their age between [21-40yrs] 50%, most were Male 64.3% , with education up to university 85.7, 57.1% were smoker, 60.7% with economic status [Medium 2000 - less than 5000],and 53.6 % were employed, 53.6% were Married.

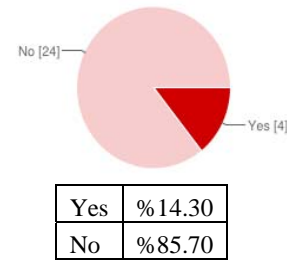


Figure 1: Genetics

Figure No. 1 Shows that the majority of the respondents in this study 85.7% they are not inherited the disease from their family.

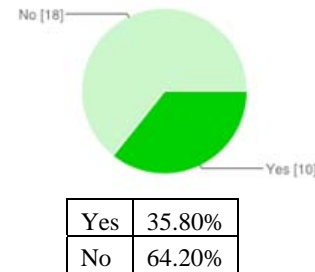


Figure 2: Knowledge about disease

Figure No.2 represents the knowledge of the respondents in these study, 64.2% have no knowledge about the dietary management of liver cirrhosis disease. The lack of knowledge about the disease may lead to more complication for the patient condition. So this patient needs more education and awareness about liver cirrhosis dietary management.

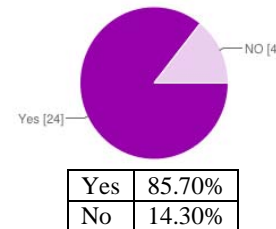


Figure 3: Spicy foods

Figure No. 3 Shows that the majority of the respondents in these study 85.7% used spicy in their meals. May be using spicy complicated the treatment of liver cirrhosis patients.

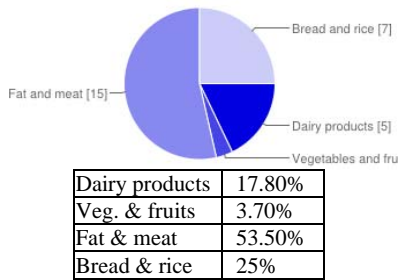


Figure 4: Food groups

Figure No. 4 Shows that the majority of the respondents in these study 53.5% consumed more fat and meat than the other type of food group on their daily life. More nutritional food should be offer to those with liver cirrhosis. Most patients eat a little fruit and vegetables, and a lot of carbohydrates and meat. Adequate food intake is recommended.

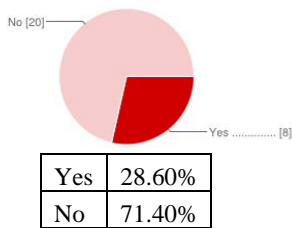


Figure 5: Exercises

Figure No. 5 Shows that the majority of the respondents in these study 71.4% were not practices any sort of exercise. The lack of exercise may be contributed to the management of the disease. The patients with liver cirrhosis need to practice any sort of exercise to help in the treatment. Knowledge about the benefit of exercise is recommended.

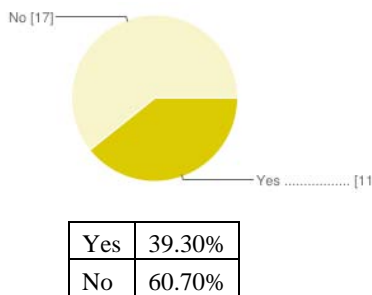


Figure 6: Other Diseases

Figure 6 Shows that the majority of the respondents in these study 60.7% are not complain from any disease other the liver cirrhosis. This means there is no relationship between the presence of disease and cirrhosis of the liver.

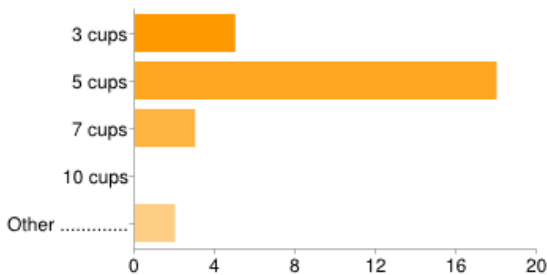


Figure 7: Drinking of water

Figure No. 7 shows that the majority of the respondents in these study 75% drinking 5 glasses of water / day. The need of water should be taken according to the condition of the patient as described in dietary management of liver cirrhosis.

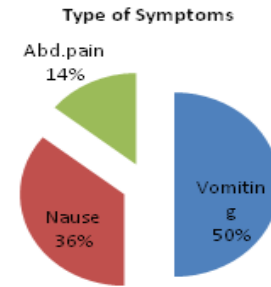


Figure 8: Type of symptoms

Figure No8. Represent the common symptoms most of the respondents complain from it. The highest symptom is vomiting 50%, followed by nausea then abdominal pain. Oral liquid supplements should be encouraged, and, when necessary enteral tube feedings used [20]

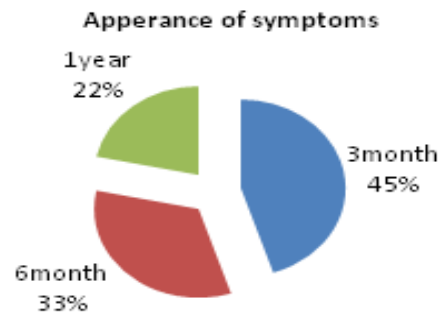


Figure 9: Time since the symptoms appeared

Figure 9 showed the time since the symptoms first appeared. The majority 45% of the respondent, their symptoms appear just before three month.

3. Conclusion

The cases of this disease showed poor eating habits, lack of interest in the nutritional side and dependence on junk food, and lack of exercise activities significantly affecting the level of treatment on the nutritional side.

4. Recommendation

- Diet low in fat and carbohydrates
- Reduce salt
- Eating fish
- When use the fat use olive oil
- Exercise or walking for 30 minutes daily
- Cooking, grilling is better than frying
- Diagnosis of the disease every 6 months

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Author Profile

Shadia Mohamed Idriss Bakheit, received the B.Sc, M.Sc, PhD, degree in Home Science/Nutrition from Ahfad university for women, university of Khartoum-Sudan, 1989, 1997, and 2000, respectively. She worked at Khartoum Teaching Hospital Sudan 1990-1997, University of Juba, College of Community Studies & Rural Development 1997-2011, University of Bahri-Sudan 2011. She worked now, as Assistant professor at university of Hail- Saudi Arabia-department of clinical nutrition

Ebtesam AL Ali University of Hail/KSA is in Department of clinical nutrition.