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TOPIC HIGHLIGHT

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Nutritional recommendations for patients with non-alcoholic fatty liver diseases

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Abstract

Fatty liver is the most common liver disease worldwide. Patients with fatty liver disease die primarily from cardiovascular disease and not from chronic liver diseases. Hyperglycemia and hyperinsulinemia induce lipogenesis, thereby increasing the hepatic pool of fatty acids. This pool is also increased by increased delivery of fatty acids through the diet or lipolysis in adipose tissue. Nutritional consultations and lifestyle modification are important in the treatment of non-alcoholic fatty liver disease (NAFLD). Among the dietary constituents, combination of vitamin D, vitamin E, and omega-3 fatty acids shows promise for the treatment of NAFLD.

Key words: Weight reduction; Non-alcoholic fatty liver disease; Physical activity; Nutrition; Fat

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Fatty liver is the most common cause of liver diseases in adults and children[1]. Fatty liver disease in humans is an insulin-resistant condition and the liver over-produces glucose and triglycerides due to impaired insulin action[2]. Fatty liver is an independent predictor of diabetes and cardiovascular disease[3]. There are three major sources for increased liver fat accumulation: excessive delivery of free fatty acids from lipolysis of superficial and visceral fat depots (60%), increased *de novo* hepatic lipogenesis (30%), and increased nutritional intake (10%)[4]. Recently, an increase in dietary cholesterol has been suggested to induce *de novo* fatty acid synthesis in hepatocytes *via* the LXRa-SREBP-1c pathway[5]. The most common cause of death in patients with non-alcoholic fatty liver disease (NAFLD) is coronary artery disease (CAD), and not chronic liver disease[6]. Fatty liver increases cardiovascular risk by classical (dyslipidemia, hypertension or diabetes) and by less conventional mechanisms. New emerging risk factors include leptin, adiponectin, pro-inflammatory cytokines such as interleukin-6, C-reactive protein and plasminogen activator inhibitor-1, which together lead to increased oxidative stress, lipotoxicity and endothelial dysfunction, which finally promote CAD[7]. When classical risk factors are superimposed on fatty liver accumulation, they may further increase the new metabolic risk factors, thus exacerbating CAD.

Several changes in dietary intake have occurred in the past few years, including increased energy intake (24%), and increases in added sugars, flour and cereal products, fruit, added fats and total fat intake[8]. Use of high fructose corn syrup (HFCS), which is used as sweetener in

soft drinks, has increased to comprise 41% of total added sweeteners. Sucrose accounts for 45% of the remainder. These changes have certainly contributed to the increase in prevalence of NAFLD, by increasing obesity and by direct fructose ingestion from soft drinks[9].

The review by Zelberg-Sagi describes elegantly the data regarding the association between dietary intake and NAFLD, and has focused on the dietary treatment of NAFLD beyond weight loss and physical activity. She has shown clearly that "good food may be a good medicine". The dietary interventions that seem to be beneficial in NAFLD are: (1) nutritional counseling with a multidisciplinary team including a dietitian, psychologist, and physical activity supervisor (behavior, educational, and motivational therapy); (2) aerobic exercise (walking 30 min daily, or > 5 km/d three times weekly); (3) restriction of calorie intake to < 30 kcal/kg per day, with a balanced diet that includes low levels of saturated and *trans* fats and simple sugars; (4) gradual weight loss (10% within 6 mo); (5) avoid rapid weight loss (> 1.6 kg/wk) as this can increase the progression of NAFLD; (6) management of accompanying conditions such as diabetes, obesity, and metabolic syndrome; (7) avoid foods with HFCS (soft drink), fast food (trans fats, and reduce red and processed meats), and genetically modified crops; (8) morbid obese patients [body mass index (BMI) > 40 or BMI > 35 with comorbidity] may be considered for referral for bariatric surgery; (9) use of vitamin E (400-800 IU/d), vitamin D (1000 IU/d), omega-3 fatty acids (1 g/d fish oil), and omega-9 fatty acids (olive oil) is recommended; and (10) trial of orlistat in patients who fail diet therapy. Use of metformin/pioglitazone if insulin resistance index (HOMA) > 2, with or without ursodeoxycholic acid (15 mg/kg per day).

However, whether any type of diet including weight loss diets can prevent steatohepatitis or fibrosis is uncertain because data on histology before and after dietary intervention are lacking. It is also uncertain whether bariatric surgery can prevent fibrosis and decrease the metabolic risk factors for CAD. It is important to establish the effects of diet composition on the natural course of NAFLD. Such data are not available at present. Of the dietary constituents, combination of vitamin D, vitamin E, and omega-3 fatty acids shows promise for the treatment of NAFLD.

REFERENCES

1 **Tiniakos DG**, Vos MB, Brunt EM. Nonalcoholic fatty liver disease: pathology and pathogenesis. *Annu Rev Pathol* 2010; **5**: 145-171

2 **Yki-Järvinen H**. Nutritional modulation of nonalcoholic fatty liver disease and insulin resistance: human data. *Curr Opin Clin Nutr Metab Care* 2010; **13**: 709-714

3 **Kotronen A**, Yki-Järvinen H. Fatty liver: a novel component of the metabolic syndrome. *Arterioscler Thromb Vasc Biol* 2008; **28**: 27-38

4 **Neuschwander-Tetri BA**, Caldwell SH. Nonalcoholic steatohepatitis: summary of an AASLD Single Topic Conference. *Hepatology* 2003; **37**: 1202-1219

5 **Yasutake K**, Nakamuta M, Shima Y, Ohyama A, Masuda K, Haruta N, Fujino T, Aoyagi Y, Fukuizumi K, Yoshimoto T, Takemoto R, Miyahara T, Harada N, Hayata F, Nakashima M, Enjoji M. Nutritional investigation of non-obese patients with non-alcoholic fatty liver disease: the significance of dietary cholesterol. *Scand J Gastroenterol* 2009; **44**: 471-477

6 **Söderberg C**, Stål P, Askling J, Glaumann H, Lindberg G, Marmur J, Hultcrantz R. Decreased survival of subjects with elevated liver function tests during a 28-year follow-up. *Hepatology* 2010; **51**: 595-602

7 **Targher G**, Day CP, Bonora E. Risk of cardiovascular disease in patients with nonalcoholic fatty liver disease. *N Engl J Med* 2010; **363**: 1341-1350

8 **Tappy L**, Lê KA, Tran C, Paquot N. Fructose and metabolic diseases: new findings, new questions. *Nutrition* 2010; **26**: 1044-1049

9 **Abid A**, Taha O, Nseir W, Farah R, Grosovski M, Assy N. Soft drink consumption is associated with fatty liver disease independent of metabolic syndrome. *J Hepatol* 2009; **51**: 918-924

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