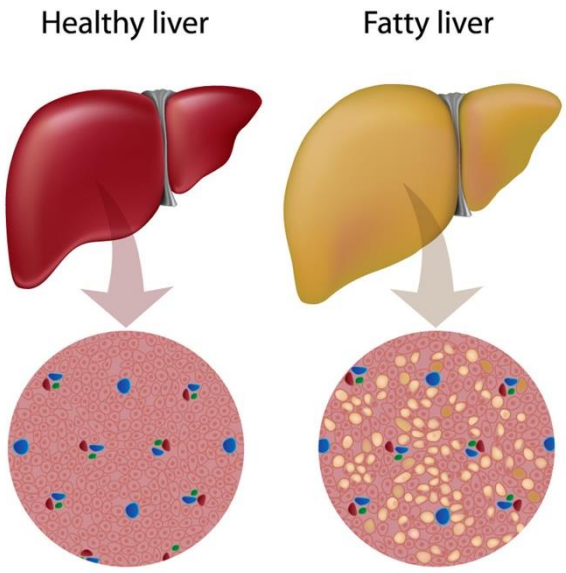
Fatty Liver Disease – "First Hit"



The liver is one of the major organs to synthesize triglycerides as the body's energy supply and cholesterol to support bile, Vitamin D, hormone production and cellular membranes. Fatty liver, or hepatic steatosis, is characterized by excess fat especially triglycerides built up in the liver cells.

Triglyceride synthesis is under feedback regulation through activation and deactivation of the sterol receptor element binding protein-1c (SREBP-1c). The abnormally increased activation of this protein causes over production of cellular lipids resulting in accumulation of excessive fat in the liver. Research has shown that malnutrition, especially a deficiency of choline and a high fructose diet with a high degree of sweetness such as high fructose corn syrup found in our processed food upregulate SREBP-1c activation. Studies suggest that high fructose intake is the major cause of fatty liver disease with increased fat accumulation in the liver. Nonalcoholic fatty liver disease (NAFLD) represents a continuum of hepatic injuries, which progress from simple fatty liver to steatohepatitis (NASH). According to the most widespread and prevailing model of the "two-hit hypothesis", the lipid accumulation in the hepatocytes is the "first hit".

Although the accumulated neutral fat seems harmless, the liver cell will undergo lipoapoptosis, a process of apoptosis or programed cell death caused by exposure to an excess of fatty acids. Lipoapoptosis is a prominent feature of NASH and is associated with the severity and progression of NASH / improve NASH and prevent its progression to fibrosis.

Nonalcoholic Steatohepatitis (NASH) – "Second Hit"

Fatty liver or hepatic steatosis is a silent condition. Patients usually have no symptoms and the disease does not progress quickly unless additional cellular events occur. This is known as the "second hit" when it will further develop to steatohepatitis, inflammation of the liver leading to liver damage and scarring. Patients with liver inflammation or NASH usually have elevated serum triglyceride levels. Liver damage can cause elevated liver enzymes such as ALP, AST, and ALT in their blood. Symptoms of liver inflammation include fatigue, weight loss, nausea, loss of appetite, pain in the upper right quadrant of the abdomen, burning or stabbing pain on the skin, spider veins and insomnia. Severe liver inflammation can also cause symptoms of genital inflammation, formication and skin crawling.

The second hit is characterized by increased pro-inflammatory cytokine production. The "second hit" includes stress induced blood flow reduction to the liver and/or bacteria endotoxins from the gut. The liver receives one-third of its blood supply from the hepatic artery which is oxygenated blood. Stress can cause reduced blood flow from the hepatic artery to the liver resulting in reduced oxygen supply to the liver. This can cause oxidative stress with increased reactive oxygen species (ROS) or free radicals which then catalyze lipid peroxidation. Oxidative stress and lipid peroxidation can injure liver cells, triggering an inflammatory response with pro-inflammatory cytokines production leading to steatohepatitis.

The liver receives two-third of its blood supply from the hepatic portal vein which comes from the digestive tract and is deoxygenated. If patients have digestive tract microbial infections, small intestinal bacterial overgrowth (SIBO), or leaky gut, the blood from the portal vein may contain high amounts of bacterial endotoxins. When the fatty liver is exposed to bacterial toxins, the "second hit", especially the endotoxin from gram-negative bacteria, the development of non-alcoholic fatty liver disease (NAFLD) can be triggered. Lipopolysaccharide (LPS), is bacterial endotoxin found in the outer membrane of gram-negative bacteria in bowel bacteria flora. Bacterial endotoxin, lipopolysaccharide (LPS), plays an important role in the pathogenesis of NAFLD. LPS triggers and accelerates the progress of NAFLD as it is a potent inducer of hepatic inflammation and also cause further increased triglyceride synthesis. Non-alcoholic fatty liver disease is an

irreversible process that starts with liver inflammation with the production of a wide array of pro-inflammatory cytokines and chemokines which enhance liver cell proliferation and collagen secretion in the extracellular matrix. High amounts of collagen deposits in the extracellular spaces can result is hardening of the liver, loss of blood infusion to liver cells, and subsequent development of liver cell death and fibrosis.

Wellness Recommendation

In TCM, fatty liver is condition caused by a Liver Yin deficiency with Liver Deficiency Heat. Treatment at this stage requires Brown and LC Balancer to nurture Liver Yin to eliminate the Deficiency Heat and help the liver resume its normal metabolic function including the feedback control of SREBP-1c in fat synthesis to reverse the fatty liver condition. The use of Brown and LC Balancer also helps clean out the accumulated excessive lipids in the liver, repair liver damage and therefore help reduce the elevated liver enzyme levels in the blood. Water Plantain Rhizome, an herbal ingredient in Brown, has been shown to have antisteatotic activity by alleviate simple fatty hepatocytes via ER stress inhibition, hepatic lipogenesis suppression, and transfer of lipids out of liver.[1] It also contains antioxidant activity and anti-lipoapoptic activity.[1] It also contains many hepatoprotective effects through lowering serum AST and ALT levels increased by high-fat diet.[1] American Ginseng, an herbal ingredient in LB Balancer, has shown a wide array of beneficial role in the regulation of liver functions and the treatment of liver disorders of acute/chronic hepatotoxicity, hepatitis, hepatic fibrosis/cirrhosis, hepatocellular carcinoma, and so on in various pathways and mechanisms.[3] Ginseng extracts have also been reported to show protective effects on hepatocytes.[3]

The protocol includes 3-6 weeks of Brown and LC Balancer. Patients can experience symptom improvement in 1 week. A diet that is rich in choline and low in sweetness is highly recommended to assist the reversal of fatty liver and prevent future development of the condition. If the patient is craving a sweet diet, the Weisslim is recommended which help normalize their eating habit and eliminate their cravings for sweets.

NASH

If the patient's fatty liver condition has progressed to steatohepatitis characterized by liver inflammation, Brown, LC Balancer, and Levera are recommended. Brown and LC Balancer nurture Liver Yin to help repair liver damage and reduce the elevated liver enzyme levels in the blood by increasing blood flow to the liver and controlling ROS generation through increasing the production of glutathione (a powerful antioxidant). Goji berry, an herbal ingredient in Brown, has been shown to significantly enhances the expression of the drug-metabolizing enzyme genes and intracellular glutathione level in hepatoma cells.[2] Levera removes Liver Heat to clear liver inflammation by removing the proinflammatory cytokines from the liver. Levera also helps reduce the triglyceride production from the liver and reduce the elevated serum triglyceride levels.

The protocol includes 3-6 weeks of Brown, LC Balancer, and Levera. Patients can experience symptom improvement in 1 week. Depending on the severity of inflammation, 3 months of the protocol may be required for significant improvement. If patients have intestinal bacterial infections or SIBO, Probiosis, PA and/or Silver are also required. If liver scarring, fibrosis or cirrhosis has been developed, 6 weeks to 3 months of Cirrhonin is also required to break down the scar tissue.

References:

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