Natural and Holistic Medicines Role of Treatment of Nonalcoholic Fatty Liver Disease

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With increasing rates of obesity and metabolic syndrome, the allied conditions of nonalcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH) have become increasingly common, such that they are now the number one cause of liver disease in Western countries. Nonalcoholic fatty liver disease (NAFLD) affects 10 to 46% of the United States population, while the worldwide prevalence is 6 to 35%, with a median of 20%. Actual numbers, of course, may be higher due to the silent nature of this metabolic syndrome companion.

NAFLD is subdivided into nonalcoholic fatty liver (NAFLD) and nonalcoholic steatohepatitis (NASH). The former presents without significant liver inflammation, while the latter is associated with hepatic inflammation that may not be distinguishable histologically from alcoholic steatohepatitis. As NAFL and NASH are both considered "silent" liver diseases, these pathologies are often asymptomatic and easy to miss clinically in the absence of proper screening. Although liver function tests (LFTs) are a part of the comprehensive metabolic panel (CMP), which many physicians routinely assess after age 50, this profile is often not performed unless there is a problem related to the liver that presented when the patient was younger, and many times, the CMP does not include gamma-glutamyltransferase (GGT). Thus, the tissue changes and low-level inflammatory state of NAFLD can go undiagnosed for many years and may even progress into hepatic cirrhosis, no different than that of end-stage liver disease associated with alcoholism or chronic viral hepatitis. If LFTs are assessed, many physicians pass off mild elevations in GGT, alanine aminotransferase (ALT), and asparate aminotransferase (AST) as "normal" without further screening, particularly in individuals who are overweight or obese. Even when physicians take the extra steps to rule out infectious hepatitis and biliary disease, the only recommendations typically suggested that may impact elevated liver enzymes (attributed to NAFLD) are alcohol abstinence, weight loss, and the proper medical management of blood sugar and cholesterol.

As we evaluate possible natural treatment strategies and interventions, it is important to consider the conditions that contribute to fatty liver changes and

hepatic inflammation. It is well understood that a prolonged state of immune=led defenses contributes to oxidative stress, inflammation, and eventual hepatocellular damage and injury, but what are the factors that exacerbate this?

Gut-Liver Connection?

An overlap between digestive system disease and hepatic inflammation (Fig8re1) has been well documented. **In embryological terms, the gut and the liver are intrinsically linked, with the liver budding directly from the foregut during development.** Increasing evidence shows that the gut and liver have multiple levels of associated interdependence, and disturbance of the gut-liver axis has been implicated in several conditions linked to obesity, including NAFLD.

Liver enzyme elevation and fatty liver changes are commonly seen in gastrointestinal conditions such as small intestinal bacterial overgrowth (SIBO), celiac disease, and inflammatory bowel disease (IBD), even in the absence of autoimmune liver or biliary disease. A recent meta=analysis also found that patients with gastroesophageal reflux disease were at a significantly increased risk of developing NAFLD (pooled odds ratio of 2.07). It doesn't stop there; an association has also been shown with Helicobacter pylori infection.

One common denominator among these conditions is the integrity, or lack thereof of the gut mucosal barrier. **"Leaky gut"**, the common term for increased intestinal permeability, has been demonstrated in each of these conditions, and it has not been a stretch for hepatologists and gastroenterologists to connect this common underpinning with NAFLD . With the compromised intestinal barrier that is hallmark to leaky gut, bacterial-derived endotoxin, also known as **lipopolysaccharide (LPS**) is able to pass into circulation and trigger a defensive inflammatory response.

In addition to alternations in the gut microbiome such as SIBO or H.pylori infection, a high-fat diet (HFD) has been shown to contribute to increased intestinal permeability and related endotoxemia. Endotoxemia contributes to intrahepatic cholestasis and related hepatocyte inflammation and damage. However, much like the gut-brain axis where there is communication in both directions, the cholestasis related to endotoxemia can further contribute to an altered balance of gastrointestinal flora and diminished motility.

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