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REPORT

A Protocol for Hepatitis C

New scientific studies appear regularly on treating hepatitis C. Here, the foundation publishes its newly revised protocol for the treatment of this disease.

Infection with the hepatitis C virus occurs from blood transfusions, needle sharing, working in a medical environment and sexual contact. Often, the infected individual does not know how he or she acquired this potentially lethal virus that has a high affinity for liver cells.

Hepatitis C used to be called non-A/non-B hepatitis and was not considered a significant health risk. There is now more research being conducted on hepatitis C than on any other cause of liver disease.

The hepatitis C virus does most of its damage by latching onto molecules of iron, and delivering massive free-radical damage to liver cells. These free radicals can mutate cellular DNA to cause hepatocellular carcinoma, and can kill large numbers of liver cells. Liver dysfunction causes havoc throughout the body. Successful eradication of the hepatitis C virus from the body requires that iron levels in the liver and blood be at very low levels, and thus it can be said that high stores of iron in the liver preclude successful therapy against the hepatitis C virus. It is mandatory to reduce iron levels in the body before initiating treatment with interferon and ribavirin combination therapy.

People are diagnosed with hepatitis C when a blood test reveals a positive reading for the hepatitis C antibody. While the hepatitis C antibody test can diagnose whether one may have the disease, the blood test that measures overall viral load is the polymerase chain reaction test (PCR). Standard tests to measure hepatitis C activity include the liver function tests SGOT, SGPT, GGTP and alkaline phosphatase. Hepatitis C antibody tests can accurately diagnose hepatitis C infection, but are not always precise in evaluating the success of treatments.

Via its free-radical attacks on liver cells, hepatitis viruses have been shown to induce liver inflammation, cirrhosis and primary liver cancer. Antioxidant supplements, in addition to anti-viral therapies, are used by scientists to protect against the lethal consequences of both hepatitis B and C.

In areas of China with high rates of hepatitis B and primary liver cancer, epidemiological surveys demonstrated that high levels of dietary selenium reduce liver-cancer incidence and hepatitis B infection. Animal studies showed that selenium supplementation reduced hepatitis B infection by 77.2 percent and precancerous liver lesions by 75.8 percent. In a four-year trial on 130,471 Chinese, those who were given selenium-spiked table salt showed a 35.1-percent reduction in primary liver cancer, compared with the group given salt without selenium added. A clinical study of 226 hepatitis B-positive people showed that one 200-microgram tablet a day of selenium reduced primary liver-cancer incidences down to zero. Upon cessation of selenium supplementation, primary liver cancer incidences began to rise, indicating that viral hepatitis patients should take selenium on a continuous basis.

Selenium also appears to be effective in suppressing the hepatitis C virus.

In patients with hepatitis C, particularly those who are HIV-positive, a systemic depletion of glutathione is present, especially in the liver. This depletion may be a factor underlying the resistance to interferon therapy. This finding represents a biological basis for N-acetylcysteine (NAC) and glutathione supplements as adjuvant (assisting) therapies.

The therapy approved by the Food and Drug Administration to treat hepatitis is a six-month regimen of interferon-alpha. While hepatitis C patients see only a 20-percent response to interferon mono-therapy, when the anti-viral drug ribavirin is combined with interferon the response rate improves by two- to tenfold. Even in patients who do not respond to interferon therapy by itself, inasmuch as there still is active viral activity in the liver, there is a significant reduction in primary liver cancer. Many hepatitis C patients have refused interferon therapy because of its toxic side effects and low rate of response (20 percent). However, a recent study showed that interferon therapy confers a 75-percent reduction in the risk of lethal primary liver cancer in hepatitis C patients, which warrants consideration of a six-month therapy with interferon (combined with ribavirin).

Studies show a systemic depletion of glutathione in hepatitis C patients, forming the basis for NAC and glutathione supplementation.

The most recent published study showed that, in hepatitis C patients who initially failed interferon therapy, the addition of ribavirin to a new round of interferon therapy produced a tenfold increase in the number of patients showing eradication of detectable hepatitis C virus. The Life Extension Foundation's protocol for hepatitis C includes:

1. The standard dose of interferon- alpha (3 million IU injected subcutaneously three times a week for six months) prescribed by an infectious-disease physician. Interferon is the FDA-approved therapy for treatment of hepatitis C. However, it works only in a minority of patients when used without ribavirin.
2. 1,000 to 1,200 mg a day of ribavirin (taken in three doses) for six months. Ribavirin increases the effectiveness of interferon therapy by up to tenfold.
3. The standard doses of Life Extension Mix and Life Extension Herbal Mix. Please note that some hepatitis C patients encounter liver enzyme elevations in response to the moderate doses of vitamin A, niacin and beta carotene in Life Extension Mix. If your liver-enzyme levels elevate after starting Life Extension Mix, discontinue it and take separately the other nutrients contained in Life Extension Mix. Beta carotene possesses unique immune-enhancing benefits that could help suppress the hepatitis C virus, but some hepatitis C patients cannot tolerate it.
4. High doses of green tea and garlic, as well as chelation therapy, to reduce serum and liver iron levels to a minimum. Iron promotes hepatitis virus-induced liver injury, and thus does not allow successful treatment with interferon. Verify that liver iron levels have been reduced before starting interferon therapy. Some people have to donate blood before going on interferon-ribavirin therapy in order to sufficiently reduce iron levels.
5. Liver-protecting nutrients and immune-boosting therapies such as 200 mg of milk thistle extract twice a day; 500 mg of licorice extract three times a day; 2,000 mg a day of garlic; 800 micrograms a day of selenium; 1,200 mg a day of N-acetylcysteine; 500 mg a day of glutathione; and vitamin C ranging from 5,000 to 20,000 mg a day.
6. S-adenosylmethionine (SAME) for the purpose of protecting and restoring liver cell function destroyed by the hepatitis C virus. SAME is in clinical trials in the United States for treating liver cirrhosis, and published research shows a significant benefit. The high cost of SAME may preclude some hepatitis C patients from being able to afford it. The suggested dose of SAME is 400 mg three times a day, to be taken with the methylation enhancing agents trimethylglycine (TMG), in a dose of 1,000 mg twice a day; folic acid in the dose of 800 micrograms three times a day; and methylcobalamin (a form of vitamin B12) in the dose of 5 mg twice a day taken sublingually.

Folic acid and vitamin B12 may also protect against ribavirin-induced anemia, which occurs in 10 percent of hepatitis C patients being treated with ribavirin. If anemia does develop using ribavirin, discontinue ribavirin until blood cell counts return to normal. Then resume ribavirin therapy.

For hepatitis C patients who fail the above regimen, the intake of 30 grams a day of whey protein concentrate could boost liver glutathione levels to help protect liver cells against hepatitis C-induced free radical liver damage.

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