

Case report

Combination naturopathic therapy in Primary Biliary Cirrhosis

A VARIETY OF NATURAL PRODUCTS are purported to be of special efficacy in liver disorders, especially hepatitis (1-3). "Liv-52," an *Ayurvedic* preparation, has also been the subject of considerable study with regard to hepatic ailments (4). In late 1991, I began to employ these agents in a form of herbal polypharmacy, with dietary modification, in the management of a 66 year-old caucasian woman with primary biliary cirrhosis (PBC) stage II, diagnosed by a liver biopsy in 1989. The patient also has a positive ANA and is serotyped as A_1, MN, Rh+, Le^A+ Le^B- (non-secretor).

The patient's history revealed an elevated GGTP (53 u/L; normal: 5.0- 45) in 1985, discovered during a workup for a fever of unknown origin. Protein electrophoresis at that time revealed a slightly elevated Alpha 1 fraction (0.33 gm/dL; normal: .10-.30) and a slightly lowered Beta fraction (0.39 gm/dL; normal: .50-1.10). Throughout the time of her treatment (September 1991 to February 1993), no allopathic medication other than antibiotic therapy for a case of bronchitis was taken. There have been a total of five office visits (9/91, 2/92, 5/92, 9/92 and 2/93). On each visit, a liver profile including bile acids, was performed at an independent medical laboratory. These values are displayed as coordinates on the graph as *Figure 1*. On 10/92, liver studies were ordered by a local gastroenterologist. They are also included.

PBC, a form of cholestatic liver disease, is increasingly being recognized. Middle-aged women constitute more than 90% of the cases. PBC is characterized by portal inflammation and necrosis of biliary cells in the small and medium bile ducts. A circulating antimitchondrial antibody is present in the serum of more than 95% of the patients and an elevated serum level of IgM is usually encountered (5).

The terminal phase of PBC is characterized by hyperbilirubinemia. The only cure for the disease is liver transplantation, although post-transplantation recurrences have been reported (6). Current drug therapy centers around the use of Bile Acid Therapy (BAT), usually in the form of ursodeoxycholic acid (UDCA) or chenodeoxycholic acid (CDCA) to "modify components of the endogenous bile acid pool"(5).

Although these agents are among the least toxic of the PBC therapeutic agents, BAT, to many researchers, fails to show definite benefit and, in a few individuals, actually accelerated the progression to liver failure (7). BAT's mode of action also remains unclear. Originally thought to replace toxic bile acids, it is now thought to possess its main mode of action through immunomodulation (8).

As shown in *Figure 1*, this patient had a positive response to the combination herbal therapy. The initial protocol (box "A") included Catechin ("SB313," THORNE RESEARCH, Sandpoint ,ID), 2 capsules BID and *Silybum marianum* ("Hepaguard," "Silymarin Phytosome," PHYTOPHARMICA, Green Bay, WI; "Thisylin Pro" MADAUS-MURDOCK, Springville, UT), 2 capsules BID. As the patient had moderate hypercholesterolemia (average: 264mg/dL), I prescribed *Crataegus oxy*. 5-1 SE (SCIENTIFIC BOTANICALS, Seattle, WA), 1/2 tsp BID ostensibly for its "solvent-like effects on the arteries "(9), and also because of the abundant antioxidant components found in the plant. The patient was also instructed to follow a diet derived from serotype analysis, in this case vegetable, fruit, soya and fish based. The patient adapted to these modifications well.

Alkaline phosphatase levels were most responsive to treatment, and declined from 781 u/L at initiation of the protocol in September 1991 to 480 u/L at the most recent visit (February 1993). In February 1992, Liv-52 (BOMBAY DRUG CO., Bombay, India) was introduced, 3 tablets BID (box "B"). Serum bile acids rose to 396 mcg/dL in the period from February 1992 to May 1992, then declined to 161 by September 1992. This decline coincided with the introduction (boxes "C","D") of *Glycyrrhiza glab.* 5-1 SE (SCIENTIFIC BOTANICALS Co., Seattle WA) 1/2 tsp BID; in conjunction perhaps with a response to the Liv-52. In September 1992, the *Glycyrrhiza* was replaced by oral Stronger Neo-Minophagen C "Glycyron" (MINOPHAGEN DRUG Co, Japan) 3 tablets BID (box E). This coincided with a decline in the GGTP from a high of 483 u/L in September 1992 to a low of 347 in February 1993. AST (SGOT) and ALT (SGPT) also showed improvement (AST 117 to 67 u/L, ALT 133 to 49u/L in the time period from September 1991 to February 1993). A sonogram taken in October 1992 showed mild splenomegaly, yet normal liver margins.

These results demonstrate that combination naturopathic treatment may represent a better means of managing PBC, a syndrome not currently curable by allopathic methods. The natural products used are derived from easily renewable resources and could represent considerable savings over current conventional management. Large-scale studies should now be undertaken to determine the usefulness of this protocol.

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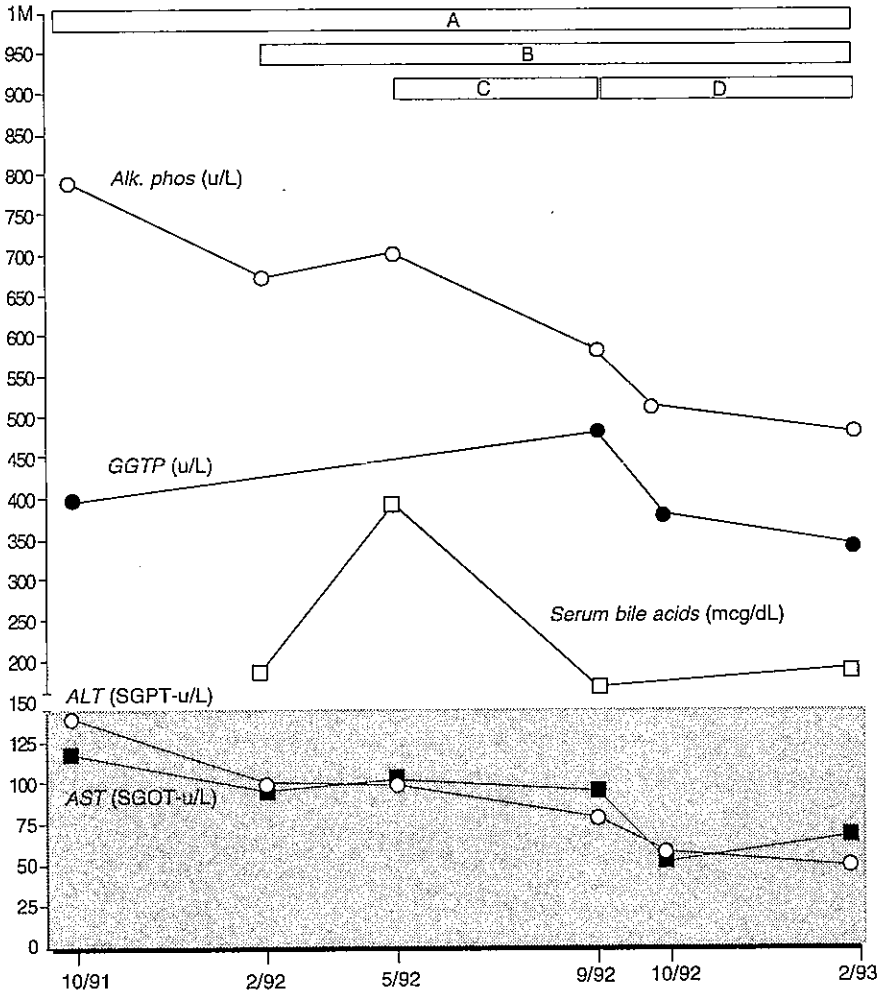

A: *Crataegus oxy.* 6-2 SE 1/2 tsp BID, milk thistle extract ["Hepaguard"]150mg (silymarin80%), catechin ["SB313," 400mg 2 BID.
B: Liv-52 3 tablets BID.
C: *Glycyrrhiza glabra*, 5-1 SE (11% glycyrrhizin) 1/2 tsp BID
D: Stronger Neo-Minophagen-C ["Glycyron"] 25mg monoammonium glycyrrhizinate, 25mg aminoacetic acid, 25mg DL-methionine; 3 BID

FIGURE 1: *Liver studies in a 66 year old woman with primary biliary cirrhosis.* Combination herbal treatment is box diagramed at the top. Shaded area denotes change in scale factor of Y axis (25 points per tic).

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