Plasma Lipoproteins and LCAT in Chronic Liver Disease

Once the presence of liver disease is established the clinician is mainly concerned with assessment of its severity. The choice of an appropriate liver function test (LFT) depends upon the clinical problem, the type and number of tests available and whether the problem is diagnostic, prognostic or requiring assessment of therapeutic effectiveness. So far no conventionally performed LFT correlates perfectly with histopathology, and therefore there is a continuous attempt to find the simplest test with maximum sensitivity and predictive value.

Biochemical tests presently available for evaluating chronic liver disease include serum albumin and globulin, BSP retention and prothrombin time. There are certain limitations in the interpretation of serum albumin levels. Serum albumin is static and does not accurately reflect the turnover of albumin. In alcoholic cirrhosis with ascites, the serum albumin levels are depressed but the size of the exchangeable albumin pool may be normal or increased. This is because the decreased level of albumin is more a reflection of increased volume of distribution rather than impaired synthesis. Rothschild et al found synthesis to be normal in 12 of 19 patients with cirrhosis and ascites and decreased in only seven. They also observed that albumin synthesis did not correlate with serum albumin levels and was a poor indicator of prognosis. Serum globulin and prothrombin time have their limitations and BSP estimation is too cumbersome to be used routinely.

The liver plays a major role in the synthesis and metabolism of plasma lipoproteins and therefore changes in lipoprotein fractions in the plasma occur in liver disease. The enzyme LCAT, which is synthesized and secreted only by the liver, catalyses the transfer of a fatty acid from the 2 position of phosphatidylcholine to the 3-OH group of cholesterol. Diseases affecting LCAT levels have profound effects on the metabolism of lipoproteins. Patients who have congenital deficiency of LCAT also have numerous abnormalities of lipoprotein composition, structure and function, manifested by low concentrations of cholesterol ester in the serum, persistence of HDL disks in the serum and failure of reverse cholesterol transport.

Low levels of LCAT have been observed in acute hepatitis, alcoholic liver disease and cholestatic liver disease. It is difficult to distinguish the effects of parenchymal disease from those of biliary obstruction.

Chadha et al, in this issue of the Journal, have compared the LCAT activity and lipid levels in 26 cirrhotics with those in 26 controls and have shown greater differences in the levels of lipid fractions between the two groups as compared to total proteins and albumin. They found no significant difference between ascitic and non-ascitic cases, as well as between alcoholic and non-alcoholic cirrhosis—the lipid profile and albumin being uniformly lowered in the patients. The authors conclude that plasma LCAT activity and cholesterol and its fractions serve as better guides than albumin in determining chronic hepatic dysfunction.

Despite the limitations on a practical level, it is generally held that reduction in serum albumin levels is an excellent indicator of severity of chronic liver disease. It is therefore justified to select serum albumin for comparison. However, to show the diagnostic superiority of one biochemical test over another, it is not just sufficient to show greater statistical difference between patients and controls in that parameter. More important points to be considered are its efficiency, sensitivity, specificity, and predictive value. The present study has limitations in assessing these due to the small sample size studied.

Measurement of lipoprotein fractions offers little in diagnostic terms due to a lack of specificity. The cost and time factor involved also put these tests at a disadvantage for routine testing. These studies are nevertheless helpful in understanding the mechanism underlying plasma lipid changes in liver disease. Lipoprotein abnormalities are responsible for some of the important metabolic changes in severe liver disease, viz malnutrition, hemolytic anemia, lowered humoral and cellular immunity, impaired water and electrolyte balance and renal failure, we need to consider ways to reverse them. Current attempts to influence lipoproteins and cell membranes include the infusion of LCAT.

Department of Gastroenterology, P N RAO
Punjab University, Hyderabad 500 012

V RADHA

The Journal is indexed and abstracted by EXCEPRTA MEDICA, Amsterdam, Netherlands, and indexed by INDEX MEDICUS and MEDLINE, Philadelphia, USA.
References

Protection of Health with ROC Products

Tonic Racplex:
A palatable ROBORANT containing therapeutic doses of B-Complex with Glycerophosphates and Lipotropic factors.
Bottles of 160 ml.

Rocfolin
CAPSULES:
Containing Riboflavin, Vit. B12, Nicotinamide and Folic Acid. A specific for Glossitis, Stomatitis, ulcers of the mouth and tongue, Sprue etc.
Containers of 20s and 100s.

Liporoc
LIQUID:
A very palatable liquid of 3 concentrated Lipotropic factors in Sorbitol-Glycerine base for protection of liver.
Bottles of 60 ml. and 200 ml.

ROC PHARMACEUTICALS
DADAR • BOMBAY 400 014
Tel.: 412 54 14

198 INDIAN J GASTROENTEROL Vol 6 No 4 OCTOBER 1987
LIPIDS IN LIVER DISEASE—RAO ET AL