Introduction

Chronic hepatitis C is a viral disease with a prevalence of at least 3% worldwide ***(Alter 1995)***.

At least 70- 80% of infected patients progress to chronic hepatitis, which is highly co-related with the development of cirrhosis and hepatocellular carcinoma, In chronic liver disease, development of fibrosis is the first step towards progression to cirrhosis and its complications (such as organ failure, esophageal variceal bleeding and hepatocellular carcinoma), independent of the underlying etiology ***(Bataller et al., 2005)***. The wound healing or a scaring response to liver damage leads to hepatic fibrosis ***(Olaso et al., 1998)***.

Liver fibrosis results from chronic damage to the liver in conjunction with the accumulation of extra-cellular matrix proteins, which is a characteristic of most types of chronic liver diseases. ***(Friedman et al., 2003)***.

Liver biopsy provides information in three major areas. Firstly, it can. elucidate, cause, Secondly, it provides information on underlying pathologic processes , Thirdly, it can also act as a prognostic indicator. ***(Ryder et al., 2004)***.

Liver biopsy does have its limitations, and some have questioned whether it truly represents a gold standard reference test. In large studies of patients undergoing biopsy, pain was reported in 20% and severe complications in 0.57% ***(Cadranel et al., 2000)***.

Noninvasive tests for assessment of liver fibrosis can be classified in several ways, including based on the modality of the test (eg, serum

blood tests versus imaging), the constituents of the test (eg, direct markers versus indirect markers of fibrosis).

Examples of fibrosis biomarker components :

Direct

- Collagen and extracellular matrix components(HA, MMP 1, MMP8, PIII NP, Laminin)
- Hepatic stellate cell and fibrogenic cell mediators (TIMP 1,TGF b, Angiotensin II,YKL 40)

Indirect

- Portal hypertension (Platelet count and Spleen size)
- Synthetic parameters(Albumin)
- Liver enzymes and bilirubin (AST, ALT, AST/ALT ratio, GGT and Bilirubin)
- Miscellaneous (Cholesterol and Insulin resistance) ***(Kaul et al., 2002).***

Preclinical studies have reported a scientific rationale and experimental evidence supporting the use of many potential therapies for fibrosis. These therapies have been targeted to any of several different biologic targets (eg, inhibition of collagen synthesis, interruption of matrix deposition, stimulation of matrix degradation, modulation of stellate cell activation, induction of stellate cell death). In general, these therapies have been highly effective in animal models. Specific Antifibrotic Therapies (Studied in Human Subjects) Angiotensin II antagonists ***(Bataller et al., 2005),*** Pirfenidone, Colchicine and Silymarin ***(Lieber et al., 2003),*** Polyenylphosphatidylcholine, Ursodeoxycholic acid and Interleukin-10 ***(Nelson et al., 2003)***.