Pancreatic and Hepatic Fibrosis: Remarkable Similarities

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Quantification of liver fibrosis by non-invasive means is a major challenge that has stimulated the search for new approaches. The prognosis and clinical management of chronic liver diseases are highly dependent on the extent of liver fibrosis, as complications mainly occur in patients in the advanced stages. This is particularly true in patients with chronic hepatitis C (HCV). Liver biopsy with METAVIR analysis is the gold standard for assessing liver fibrosis associated with HCV [1-3]. It is an invasive and expensive procedure that is not well accepted by many patients especially when repeated examinations are needed. Moreover, its accuracy in assessing fibrosis is questionable, as reproducibility is poor due to sampling errors, and even in adequately sized specimens, intra-observer and inter-observer discrepancies are seen [4-7]. Our research shows that a special type of blood test which we have developed, the ‘Fibrogenic Stimulation Index’ (FSI), can identify patients with liver fibrosis. The FSI assesses the stimulation of proliferation of fibroblasts and hepatic stellate cells (HSCs), the key cell involved in hepatic fibrosis, in response to patients’ sera samples. The FSI is a cell based assay of fibrosis that assesses the ability of patients’ sera samples to stimulate proliferation of selected target cells. The type of fibrosis to be assessed is matched with a specific target cell. The FSI is a non-invasive test which uses patients’ sera samples in an in vitro assay which can detect and quantify the degree of fibrosis based on a patient’s blood sample. Our data suggests that FSI correlates with procollagen type III peptide (P-III-P) another measure of hepatic fibrosis and that the FSI and P-III-P correlate with METAVIR fibrosis score. Further study in the larger cohort of HCV patients indicates that FSI is a positive predictor of fibrosis in HCV patients. We were the first to report on the role for platelet derived growth factor (PDGF) in experimental models of hepatic fibrosis [8,9] and we showed that PTX blocked fibrosis via an effect on PDGF and that this occurred by blocking c-Jun phosphorylation of c-jun on serine 73 [10]. We began investigations on the role for platelet derived growth factor (PDGF) in experimental models of hepatic fibrosis [8,9] and we showed that PTX blocked fibrosis via an effect on PDGF and that this occurred by blocking c-Jun phosphorylation of c-jun on serine 73 [10].

References

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