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 META VIR 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 both the FSI and P-III-P correlate with META VIR 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 reported 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 phosphorylation of c-Jun on serine 73 [10]. We began investigations of non alcoholic steatohepatitis (NASH) mediated fibrosis [11,12] and reported that ribavirin but not interferon inhibited fibrosis also associated with HCV and that this effect was mediated by the block of phosphorylation of c-Jun on serine 73 [13]. We reported that PTX decreased NASH sera-stimulated FSI (our patented diagnostic test for fibrosis using HSCs as the target cell) and c-Jun phosphorylation associated with 'H-thymidine incorporation and Western analysis respectively, such that NASH patient sera stimulated HSC proliferation and decreased NASH sera-stimulated phosphorylated c-Jun in HSCs [14]. Our recent published [15] and unpublished data indicate that PTX decreased the FSI; that the FSI correlates well with the META VIR fibrosis score in HCV patients and may be predictive of fibrosis in this cohort; this intellectual property is patented [16]. Pancreatic fibrosis shows remarkable though not surprising similarities to hepatic fibrosis. From the Binkley and colleagues early studies on molecular basis of pancreatic fibrosis [17] to the more recent studies of Krantz and colleagues on the molecular understanding of the role of TGFβ in this disorder [18], the diseases run in parallel. It would be exciting to work more closely with those who are making such great advances in the area of pancreatic fibrosis and facilitate the development of novel therapeutics that could potentially cure both.

References


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