

INTEGRATED INFLAMMATION–FIBROGENESIS PANEL FOR STRATIFYING PATIENTS WITH VIRAL LIVER CIRRHOSIS

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Background. The progression from advanced fibrosis to cirrhosis is driven not only by hepatocyte injury but also by persistent systemic inflammation, activation of liver macrophages, and excessive extracellular matrix deposition. Conventional biochemical tests do not fully reflect these pathogenic mechanisms. Biomarkers such as interleukin-6 (IL-6), C-reactive protein (CRP), soluble CD163 (sCD163), procollagen type III N-terminal propeptide (PIIINP), and tissue inhibitor of metalloproteinases-1 (TIMP-1) may provide additional information on inflammatory activity, macrophage activation, and fibrogenesis.

Objective. To evaluate an integrated panel of inflammatory, macrophage activation, and fibrogenesis biomarkers for differentiating viral liver cirrhosis from advanced liver fibrosis.

Materials and Methods. The study included 79 patients with viral liver cirrhosis, 36 patients with F3–F4 liver fibrosis, and 30 healthy controls. Serum IL-6, CRP, sCD163, PIIINP, and TIMP-1 concentrations were determined using enzyme-linked immunosorbent assay. The levels of the biomarkers were compared among the study groups, and $p < 0.05$ was considered statistically significant.

Results. A marked increase in inflammatory markers was observed in patients with cirrhosis. IL-6 concentration reached 18.6 ± 7.4 pg/mL in the cirrhosis group, compared with 9.8 ± 3.7 pg/mL in the F3–F4 fibrosis group and 3.1 ± 1.2 pg/mL in controls ($p < 0.001$). Similarly, CRP levels were significantly higher in cirrhosis than in advanced fibrosis (11.9 ± 4.3 versus 6.2 ± 2.1 mg/L; $p < 0.001$), indicating a greater systemic inflammatory burden.

The marker of macrophage activation, sCD163, increased from 420 ± 110 ng/mL in F3–F4 fibrosis to 698 ± 185 ng/mL in cirrhosis ($p < 0.001$). This finding reflects enhanced Kupffer-cell activation and immune dysregulation in advanced liver disease. Markers of extracellular matrix turnover also showed significant differences. PIIINP concentration was 14.3 ± 4.1 ng/mL in cirrhosis compared with 8.7 ± 2.8 ng/mL in the F3–F4 group ($p < 0.001$), while TIMP-1 levels were 312 ± 74 and 198 ± 56 ng/mL, respectively ($p < 0.001$). The simultaneous increase in PIIINP and TIMP-1 suggests active collagen synthesis accompanied by reduced matrix degradation.

Conclusion. The combined assessment of IL-6, CRP, sCD163, PIIINP, and TIMP-1 provides a broader laboratory characterization of viral liver cirrhosis than routine biochemical testing alone. This integrated panel may be useful for identifying patients with active inflammation and progressive fibrogenesis, supporting laboratory-based risk stratification and individualized clinical follow-up.