

combination therapy. Comprehensive molecular characterization included RNA sequencing with subsequent differential gene expression analysis, pathway enrichment studies, and protein interaction network mapping. Mechanistic findings were confirmed through immunohistochemical and immunoblotting techniques.

Results: The SB-SF combination demonstrated enhanced anticancer efficacy compared to SB alone. Transcriptomic profiling identified 45 differentially expressed genes associated with HCC suppression, particularly those involved in proliferation control, oxidative stress response, and apoptotic regulation. The combination therapy significantly downregulated key oncogenic markers including NF- κ B-p65, COX-2, and β -catenin, suggesting its potential as a cost-effective therapeutic approach that warrants further clinical investigation.

Conclusions: The study reveals a multifaceted mechanism by which SF augments SB's anticancer activity in HCC. The combined treatment modulates critical oncogenic pathways including NF- κ B and Wnt/ β -catenin signaling while rebalancing apoptotic regulators through decreased Bcl-2 and increased Bax/caspase expression. Additionally, it suppresses proliferative markers such as Ki-67 and PCNA while attenuating inflammatory mediators including TNF- α and MMP-9. These coordinated effects demonstrate potent anti-tumorigenic, anti-angiogenic, and pro-apoptotic activity, highlighting the therapeutic promise of this combination approach for HCC treatment.

Conflict of interest: None

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#20

CONNECTIVE TISSUE GROWTH FACTOR AS A MARKER OF FIBROSIS IN PATIENTS WITH CHOLESTASIS

Jessica Mejía Ramírez¹, Fatima Higuera de la Tijera¹, Carolina Guzman², Ángel Daniel Santana Vargas³, Viridiana López Ladron de Guevara¹, Cristian Yamin Sánchez Sánchez¹, Diego Fernando Abendaño Rivera¹, Maria Argentina Díaz Castro¹, Kevin Vázquez Hernández¹, Karla Marysol Meixueiro Olivera¹, Jose Luis Pérez Hernández¹

¹ Servicio de Gastroenterología y Hepatología del Hospital General de México "Dr. Eduardo Liceaga".

² Laboratorio de Hígado, Páncreas y Motilidad. Unidad de Medicina Experimental. Hospital General de México "Dr. Eduardo Liceaga".

³ Dirección de Investigación del Hospital General de México "Dr. Eduardo Liceaga".

Introduction and Objectives: Connective Tissue Growth Factor (CTGF) is a multifunctional protein, plays a crucial role as a mediator in fibrogenic pathways involved in the development and progression of liver disease.

To establish the correlation between serum CTGF values by ELISA and the degree of liver fibrosis determined by transitional elastography in patients with cholestasis diagnosed with primary biliary cholangitis (PBC).

Materials and Methods: A descriptive and analytical prolective study that included patients with cholestasis diagnosed with PBC, patients with cirrhosis due to hepatitis C and a control group of healthy subjects. Serum CTGF levels and the degree of fibrosis were quantified. Statistical analysis: CTGF means were compared using a univariate model with etiology as an between-group factor and degree of fibrosis as an within-group factor, significance level 5%.

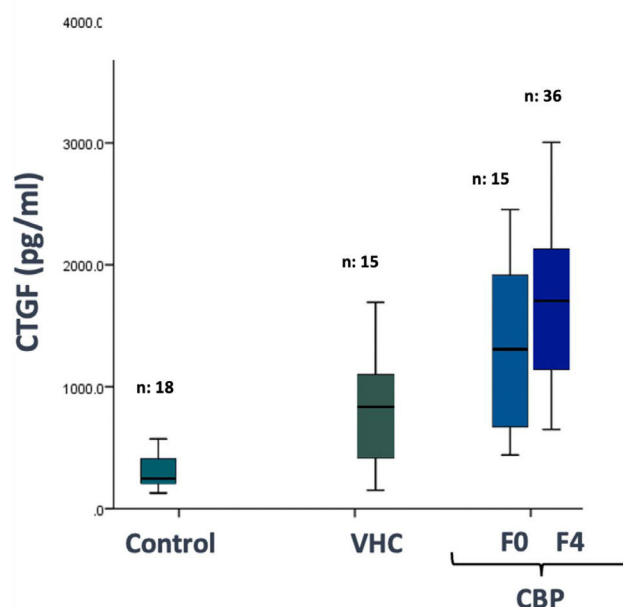
Results: 51 patients with PBC, 15 HCV and 18 controls were included. Age 48 ± 15 years, 75% women. The level of CTGF in patients with cholestasis increased with increasing degree of fibrosis. PBC (F0) 1342.4 ± 669.9 ; PBC (F4) 1679.9 ± 623.3 . HCV (F4) 816.3 ± 431.6 , CT 314.1 ± 163 . Statistically significant differences ($p < .001$) were found between groups CT vs PBC (F0); CT vs CBP (F4), HCV (F4) vs PBC (F4) and CT vs HCV (F4). Figure 1

Conclusions: There is a direct relationship between serum levels of CTGF of patients with cholestasis and degrees of fibrosis by transition elastography, perhaps it can be considered as a marker of fibrosis induction in patients with cholestasis.

Conflict of interest: None

Serum CTGF levels and the degree of fibrosis

Figure 1. Serum CTGF levels and the degree of fibrosis



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#21

CONNECTIVE TISSUE GROWTH FACTOR ASSESSMENT: DETERMINATION OF CUT-OFF POINTS FOR LIVER FIBROSIS IN CHOLESTASIS

Jessica Mejía Ramírez¹, Fatima Higuera de la Tijera¹, Carolina Guzman², Ángel Daniel Santana Vargas³, Viridiana López Ladron de Guevara¹, Cristian Yamin Sánchez Sánchez¹, Diego Fernando Abendaño Rivera¹, Maria Argentina Díaz Castro¹, Kevin Vázquez Hernández¹, Karla Marysol Meixueiro Olivera¹, Jose Luis Pérez Hernández¹

¹ Service of Gastroenterology and Hepatology. Hospital General de México "Dr. Eduardo Liceaga".

² Laboratory of Liver, Pancreas and Motility. Experimental Medicine Unit. Hospital General de México "Dr. Eduardo Liceaga".

³ Direction of Research. Hospital General de México "Dr. Eduardo Liceaga".