

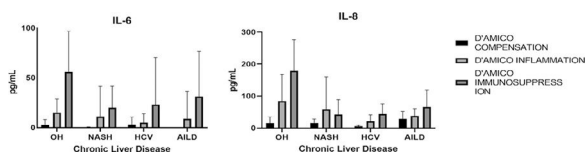
with HCV and AILD, increased serum levels of IL-8 and IL-6 were shown in decompensation stages (Figure 1).

**Discussion:** In this study, we demonstrated a significant increase in the pro-inflammatory cytokine profile in patients in the inflammation and immunosuppression phases. Fischer J et al. reported that a greater understanding of the mechanisms associated with immune dysfunction has led to the identification of possible therapeutic targets, with the intention of reducing the risk of infection and preventing decompensation events and disease progression.

**Conclusion:** This study demonstrated a significant increase in the pro-inflammatory cytokine profile (IL-8 and IL-6) as cirrhosis progresses. This is consistent with in the inflammation and immunosuppression phases, assessed by the Child-Pugh severity scales in stages B and C, D'amico from stages 3 to 5 and MELD> from 16 to 24 points and from 25 to 34 points. in the four etiologies included, being statistically significant.

The authors declare no conflict of interest.

This work was sponsored by PAICYT-UANL-SA830-19.



<https://doi.org/10.1016/j.aohep.2021.100645>

### CONNECTIVE TISSUE GROWTH FACTOR (CTGF) AS A PROMOTER IN THE DEVELOPMENT OF FIBROSIS IN PATIENTS WITH PRIMARY BILIARY CHOLANGITIS

E. Morales, C. Guzmán-Arriaga, M.A. Díaz-Castro, F. Higuera-de-la-Tijera, D. Santana-Vargas, E. Bautista Ubaldo, J.E. Lira-Vera, O. Morales-Gutiérrez, J.L. Pérez-Hernández

General Hospital of México Dr. Eduardo Liceaga.  
México City, México

**Introduction and Objectives:** Primary biliary cholangitis (PBC), an immune-mediated disease, is characterized by destroying the intrahepatic bile ducts, leading to progressive damage to the biliary tree, cholestasis, and development of progressive fibrosis leading to cirrhosis with all its complications. The development of fibrosis is multifactorial and includes connective tissue growth factor (CTGF). in a mouse model of cholestasis by bile duct ligation, the hepatic and serum increase in CTGF associated with the progression of fibrosis was demonstrated. Our goal was to determine the relationship between CTGF levels and their association with the development of fibrosis in patients with PBC.

**Material and methods:** Prospective, cross-sectional, and analytical study, including patients with PBC. The degree of fibrosis was determined by transient elastography (Fibroscan). Serum concentrations of FCTC-8pg/ml were quantified, for statistical analysis, the SPSS version 25.0 software was used; the medians (Q3, Q1) of CTGF, alkaline phosphatase, gamma-glutamyl-transpeptidase, and degree of fibrosis were compared with the Mann-Whitney U test with significance less than 0.05.

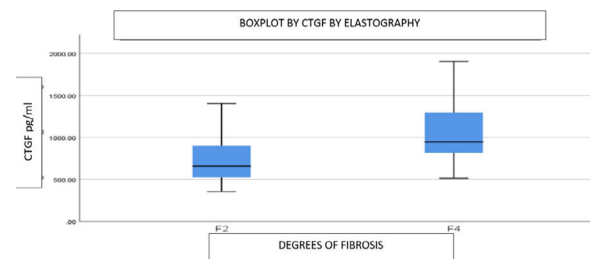
**Results:** We included 30 patients, 29 women (96.6%) and 1 man (3.4%), with a mean age of 55.5±12.4 years. Overexpression of CTGF protein was shown in 28 subjects (93.3%). Regarding the degree of fibrosis, all patients were categorized into one of two stages: Significant fibrosis (F2) and cirrhosis (F4). The F2 group had 11 patients with a median and standard deviation for CTGF of 915.9 and 522.9,

respectively; The F4 group had 19 patients who showed a median: 945.7 (1313.85-738.32); p:0.025. In relation to the differences between fibrosis levels and markers of alkaline phosphatase cholestasis, the median and interquartile ranges F2: 79 (180.60) F4: 169 (266-5.84) p: 0.066; GGT: F2: 1.51(7.7-1,04); F4: 1.2(2-1.92) p:0.746 In (Figure 1) The difference in medians of patients with different degrees of fibrosis and different concentration of CTGF is shown, confirming the association between peptide and the development and progression of fibrosis.

**Discussion:** According to the results obtained in patients with CBP and chronic cholestasis, the increase in CTGF showed significant differences between the degree of fibrosis and its levels; this could perhaps be interpreted as if it were an important factor for the development and progression of liver fibrosis, taking into account the antecedent of the initial study in mice with bile duct ligation and secondary cholestasis, where this factor was overexpressed at the hepatic and serum level in subjects with advanced fibrosis. It will be important to add more samples to this work and compare it with healthy controls to have better evidence.

**Conclusions:** Connective tissue growth factor (CTGF) probably participates directly in the processes of synthesis of extracellular matrix and therefore in the progression of fibrosis in subjects with primary biliary cholangitis, which makes it a possibility of a therapeutic target to develop in future studies.

The authors declare that there is no conflict of interest.



(Figure 1) The difference in medians of patients with different degrees of fibrosis and different concentration of CTGF is shown, confirming the association between peptide and the development and progression of fibrosis.

<https://doi.org/10.1016/j.aohep.2021.100646>

### Global real-world evidence of sofosbuvir/velpatasvir (SOF/VEL) as a highly effective treatment in underserved patient populations because of mental health disorders, incarceration or homelessness

L. Barrett<sup>1</sup>, S. Rosati<sup>2</sup>, M. Garcia-Retortillo<sup>3,4</sup>, H. Wedemeyer<sup>5</sup>, E. Teti<sup>6</sup>, FA Pérez Hernández<sup>7</sup>, M. Selfridge<sup>8</sup>, A. Wong<sup>9</sup>, S. Rodriguez-Tajes<sup>10</sup>, L.E. Morano-Amado<sup>11</sup>, C. Brixko<sup>12</sup>, E. Jimenez-Mutiloa<sup>13</sup>, J. O'Loan<sup>14,15</sup>, M. Milella<sup>16</sup>, F. Campanale<sup>17,18</sup>, G. Macedo<sup>19</sup>, M. Guerra-Veloz<sup>20</sup>, I. Maida<sup>21</sup>, R. Ranieri<sup>22,23</sup>, A. Martins<sup>24</sup>, A. Bascia<sup>25,26</sup>, M. Buti<sup>27</sup>, C.M. Fernandez-Rodriguez<sup>28</sup>, B. Conway<sup>29</sup>, J. Foucher<sup>30</sup>, S. Fagioli<sup>31</sup>, A. Ramji<sup>32</sup>, M. Fenech<sup>33</sup>, P. Ryan<sup>34</sup>, S. Borgia<sup>35</sup>, A. Mangia<sup>36</sup>, J. Mendez-Navarro<sup>36a</sup>, I. Ntalla<sup>37</sup>, C. Hernández<sup>38</sup>, M. Mertens<sup>38</sup>, K. Vanstraelen<sup>38</sup>, V. Calvaruso<sup>39</sup>

<sup>1</sup> NSHA/Dalhousie University, Infectious Diseases Department, Halifax, NS, Canada

<sup>2</sup> I.N.M.I. Lazzaro Spallanzani IRCCS, Rome, Italy