Inflammation-related transcriptional differences associated with fibrosis severity in MASLD: Findings from a Turkish cohort

Deniz Cansen Kahraman[1], Esra Nalbat[1], Dilara Turan Gökçe[2], Saba Kiremitçi[3], Ramazan Idilman[2]
[1]Cancer Systems Biology Laboratory, Graduate School of Informatics, Middle East Technical University, Ankara, Turkiye
[2]Department of Gastroenterology, Faculty of Medicine, Ankara University, Ankara, Turkiye
[3]Department of Medical Pathology, Faculty of Medicine, Ankara University, Ankara, Turkiye

Background: In metabolic dysfunction–associated steatotic liver disease (MASLD), progression from mild (F0/F1) to significant fibrosis (>=F2) represents a key prognostic threshold guiding clinical management. Although inflammatory mechanisms contribute to fibrotic progression, inflammation-related molecular features distinguishing these stages across populations remain incompletely defined. This study aimed to identify inflammation-associated transcriptional differences linked to fibrosis severity in a Turkish MASLD cohort.

Method: FFPE liver biopsy samples from 36 MASLD patients were analyzed and classified as F0/F1 (n = 23) or >=F2 (n = 13). The cohort had a mean age of 47 years, with females comprising 66% of participants. Targeted transcriptomic profiling was performed using the NanoString nCounter Human Inflammation Panel (249 genes). Differentially expressed genes (DEGs) were identified and functional enrichment analysis was carried out using Enrichr. Protein–protein interaction networks were examined using STRING.

Results: Body mass index and sex distribution did not differ significantly between fibrosis groups. Metabolic and cardiovascular comorbidities, including diabetes mellitus, hypertension, hyperlipidemia, and coronary artery disease, were more common in patients with >=F2 fibrosis but did not reach statistical significance. Transcriptomic analysis identified 23 inflammation-related DEGs distinguishing early from significant fibrosis. Patients with >=F2 fibrosis showed increased expression of chemokines and chemokine receptors involved in inflammatory cell recruitment, including CCL2, CCL5, CCL19–21, CXCL1, CXCL6, CXCL8, CXCL9–10, and CXCR4. Profibrotic mediators (TGFB1–3) and innate inflammatory signaling components (TLR2, TLR8, TNF, STAT1) were also upregulated. Network analysis revealed a chemokine- and innate inflammation–centered interaction core with highly connected hub genes and peripheral nodes linked to oxidative stress and complement-related inflammatory pathways.

Conclusion: Significant fibrosis is characterized by distinct inflammation-related transcriptional signatures that are independent of major clinical covariates, underscoring the potential utility of inflammation-focused gene expression profiles for fibrosis risk stratification.

Keywords: MASLD (metabolic dysfunction-associated steatotic liver disease), liver fibrosis staging, inflammatory transcriptomics

Details

Status	:	Saved Draft. Not Submitted Yet
Presentation Preference	:	Oral Presentation
Abstract Category/Topic	:	MASLD
Language	:	English
Saved:	:	3.1.2026 22:54:18
Submit:	:	

Confidential to Author and Editor

Note to Editor	:	This study was supported by the Turkish Association for the Study of the Liver (TASL) and Cancer Systems Biology Laboratory (CanSyL)-METU.
Presenter	:	Deniz Cansen Kahraman (cansen@metu.edu.tr)

Close

Print