

**AGE-RELATED MORPHOLOGICAL CHANGES IN ALCOHOLIC LIVER
CIRRHOSIS: A PATHOMORPHOLOGICAL AND HISTOLOGICAL
ANALYSIS IN UZBEKISTAN, 2020–2025**

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ABSTRACT

Alcoholic liver cirrhosis (ALC) causes 1.48 million deaths yearly, with rising prevalence in younger groups (25–34 years) and age-specific morphological patterns. This study examines age-related pathomorphological and histological changes in ALC among Uzbeks from 2020–2025, focusing on steatosis, fibrosis, and hepatocellular carcinoma (HCC). Analyzing 150 biopsy/autopsy specimens with H&E, Masson's trichrome, and METAVIR scoring, results show younger patients (25–34) with 80% microvesicular steatosis and rapid F3–F4 fibrosis (2–3 years), while older (50+) exhibit 65% nodular regeneration and 15% HCC. Statistical tests (Chi-square, ANOVA) confirm significant differences in fibrosis ($p < 0.01$) and necroinflammation ($p < 0.05$). Findings support age-tailored diagnostics and prevention, amid Uzbekistan's rising alcohol use (8.1 liters/capita by 2025), offering insights for resource-limited settings.

Keywords: alcoholic liver cirrhosis, age-related changes, pathological anatomy, histology, fibrosis, steatosis, hepatocellular carcinoma, Uzbekistan, epidemiology, morphology.

INTRODUCTION

Alcoholic liver disease (ALD) spans steatosis to cirrhosis, causing 8% of global liver deaths in 2019. ALC features irreversible fibrosis, nodules, and complications like portal hypertension and HCC. Global trends (2023–2025) show rising ALC in youth (15–34), with 19-fold prevalence and 2-fold mortality increases. In Uzbekistan, alcohol intake rose from 7.2 to 8.1 liters/capita (2020–2025), boosting ALC cases. Younger patients progress rapidly to fibrosis; older to decompensated cirrhosis and HCC. Pathological anatomy reveals these patterns via histology and macroscopy, but Uzbek data is limited. This study analyzes 2020–2025 specimens to detail age differences, integrating global and local insights for tailored strategies.

RELEVANCE OF WORK

ALC's global burden (1.48 million deaths/year) disproportionately affects youth (25–34) due to post-COVID stressors and rising alcohol use. In Uzbekistan, consumption hits 8.1 liters/capita by 2025, doubling young adult ALC incidence. Pathologically, youth show rapid fibrosis (F3–F4 in 2–3 years) and microvesicular steatosis; older, nodular regeneration and HCC. Limited Uzbek histological data impedes targeted interventions like youth screening or elder HCC monitoring. This study's novelty: age-specific patterns in Uzbekistan via specimen analysis, aiding diagnostics and policies like alcohol restrictions.

PURPOSE

To explore age-related morphological/histological ALC changes in Uzbeks (2020–2025), with objectives:

1. Characterize steatosis, fibrosis, necroinflammation in groups (25–34, 35–49, 50+) using METAVIR/Ishak.
2. Compare macro/micro changes, emphasizing fibrosis, nodules, HCC.
3. Correlate histology with clinical data (e.g., MELD, alcohol dose).
4. Suggest age-specific diagnostics/prevention to lower ALC burden.

RESULTS AND DISCUSSION

Methods: Retrospective analysis of 100 biopsies and 50 autopsies from Uzbek centers (2020–2025), stratified by age: 25–34 (n=50), 35–49 (n=60), 50+ (n=40). Used H&E/Masson's for steatosis/fibrosis (METAVIR), necroinflammation (Ishak); autopsies for macro features. Stats: Chi-square, ANOVA, Cox regression; clinical data from records.

Results: Age differences significant ($p < 0.01$). Youth (25–34): 80% microvesicular steatosis, 60% F3–F4 in 2–3 years, high necroinflammation (Ishak 4–6, $p < 0.05$), tied to 80 g/day alcohol. Middle (35–49): 50% mixed steatosis, 70% F2–F3 (4–5 years). Older (50+): 65% nodules, 15% HCC, 85% F4. Macro: Youth enlarged/pale livers; older shrunken/micronodular. Cox: Age (HR 1.03, $p < 0.01$), fibrosis (HR 1.15, $p < 0.001$) predict mortality.

Discussion: Youth rapid fibrosis matches global data, driven by high alcohol/inflammation; microvesicular steatosis indicates mitochondrial issues. Older HCC from cumulative exposure, per EASL guidelines. Uzbekistan's alcohol rise

worsens youth burden; limited FibroScan hinders detection. Suggest histological screening for youth. Limits: Retrospective bias; need prospective studies.

CONCLUSION

Study reveals ALC patterns in Uzbekistan: rapid fibrosis in youth (25–34), nodules/HCC in older (50+). Advocate age-specific tools like FibroScan for youth, HCC surveillance for elders. Push alcohol policies and awareness. Future: Probe markers (TGF- β , IL-6) for better models.

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