Original Research

HISTOPATHOLOGICAL PATTERNS AND CLINICOPATHOLOGIC CORRELATES OF NON-ALCOHOLIC STEATOHEPATITIS IN INDIAN ADULTS

A Shamsath Nisha[1]
[1]Assistant Professor, Department of Pathology, Madurai Medical College, Tamil Nadu, India.
Corresponding Author: Dr. A Shamsath Nisha
Assistant Professor, Department of Pathology, Madurai Medical College, Tamil Nadu, India.
Email: samsath_nisha@yahoo.co.in

ABSTRACT

Background: Non-alcoholic fatty liver disease (NAFLD) is increasingly common worldwide, with a substantial proportion progressing to non-alcoholic steatohepatitis (NASH) and fibrosis. In India, metabolic risk factors are rising, and biopsy studies suggest that over 60% of NAFLD patients have NASH, with ~30–35% showing advanced fibrosis. Histologic diagnosis of NASH (macrovesicular steatosis, ballooning degeneration, and inflammation) is the gold standard. We examined liver biopsy samples from Indian adults with NAFLD to characterize histopathological patterns and correlate these with clinical features.

Materials and Methods: We conducted a retrospective analysis of 150 adult patients with biopsy-proven NAFLD (exclusion of other etiologies) at a tertiary center in India. Liver biopsies were scored by standard criteria (Brunt et al.) for steatosis grade (1–3), lobular inflammation (0–3), hepatocyte ballooning (0–2), portal inflammation (0–3), and fibrosis stage (0–4). NASH diagnosis required steatosis ≥5%, ballooning, and lobular inflammation. Clinical data (age, gender, BMI, diabetes, hypertension, metabolic syndrome, liver enzymes) were recorded. Continuous variables were compared by t-test or Mann–Whitney U test and categorical by chi-square. Logistic regression identified factors independently associated with NASH. Statistical significance was set at $p<0.05$.

Results: Among the 150 NAFLD patients (mean age 47±12 years, 60% male), 45 (30%) had definite NASH, 50 (33%) had borderline NASH, and 55 (37%) had simple steatosis (no NASH). NASH patients were older (52.1±11.3 vs 44.5±12.1 years, $p=0.004$), had higher BMI (28.5±4.3 vs 24.8±3.5 kg/m², $p<0.001$), and greater prevalence of type 2 diabetes (44% vs 17%, $p=0.001$), hypertension (33% vs 15%, $p=0.02$) and metabolic syndrome (67% vs 33%, $p<0.001$) than non-NASH patients (Table 1). ALT and AST levels were significantly higher in NASH (mean ALT 80 vs 45 U/L, AST 70 vs 40 U/L; $p<0.001$ for both). On histology (Table 2), moderate-to-severe steatosis (grade 2–3) was seen in 75% of NASH cases; ballooning (score ≥1) was present in 89% and lobular inflammation (score≥1) in 89%. Mallory–Denk bodies were noted in 12 (26.7%) of NASH biopsies. Fibrosis was present in 30 (66.7%) of NASH patients: stage 0 (none) in 15 (33.3%), stage 1 in 10 (22.2%), stage 2 in 7 (15.6%), stage 3 in 6 (13.3%), and stage 4 (cirrhosis) in 7 (15.6%). Thus advanced fibrosis (stage ≥3) occurred in 24.4% of NASH patients, similar to the ~30% reported in Indian series. Figure 1–3 show representative histology. In multivariate analysis (Table 3), BMI >25 kg/m² (OR 4.2; 95% CI 2.2–8.1; $p<0.001$), diabetes (OR 3.5; 95% CI 1.8–6.8; $p<0.001$), and age >50 years (OR 2.1; 95% CI 1.2–3.6; $p=0.01$) were independently associated with NASH. Female gender and mild ALT elevation were not significant predictors.

Conclusion: In this Indian cohort, NASH was present in about one-third of biopsy-proven NAFLD patients. NASH was strongly associated with obesity, diabetes, and metabolic syndrome, underscoring metabolic dysfunction as a driver of disease. Histologically, most NASH biopsies showed moderate-to-severe steatosis, ballooning degeneration, and varying degrees of fibrosis, with nearly one-quarter having advanced fibrosis (stage 3–4). These findings align with reports that Indian NAFLD often presents with aggressive histology. Early recognition of at-risk patients (e.g. older, obese, diabetic individuals) is crucial, as NASH carries a high risk of progression to cirrhosis and hepatocellular carcinoma.

Keywords: NAFLD, NASH, Steatosis, Fibrosis, India, Histopathology, Metabolic Syndrome.

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most common chronic liver disease globally, affecting roughly 25–30% of adults [1].

www.ajmrhs.com
eISSN: 2583-7761

Date of Received: 04-06-2026
Date Acceptance: 12-06-2026
Date of Publication: 11-07-2026

NAFLD ranges from benign steatosis to non-alcoholic steatohepatitis (NASH), the latter defined by macrovesicular steatosis, hepatocyte ballooning, and lobular inflammation. NASH is clinically important because it can progress to fibrosis, cirrhosis, and hepatocellular carcinoma (HCC)[2]. Indians bear a high burden of NAFLD due to prevalent obesity, type 2 diabetes mellitus (T2DM), and metabolic syndrome [3]. Community surveys in India report NAFLD prevalence ranging widely

(~10–50%), with pooled estimates around 30–40%[4][5]. Notably, even "lean" individuals can have NAFLD in India due to high visceral adiposity[6]. Importantly, Indian cohorts appear to have more aggressive disease: over 60% of biopsied NAFLD patients had histologic NASH, and 30–35% showed advanced fibrosis. Explant studies confirm that >60% of presumed cryptogenic cirrhosis and increasing numbers of "non-B, non-C" HCC cases in India are attributable to NASH.

Liver biopsy remains the gold standard for diagnosing NASH and staging fibrosis, despite limitations of sampling error[7]. Histologic scoring systems (Brunt et al.[11], NASH-CRN NAS, SAF) grade steatosis (grade 1: ≤33%; grade 2: 33–66%; grade 3: ≥66% of hepatocytes), lobular/portal inflammation, and ballooning, and stage fibrosis (0–4). By consensus, NASH requires steatosis ≥5% plus ballooning and inflammation. In clinical trials, scores such as NAS (0–8) and fibrosis stage are used to monitor progression and response.

To better define NASH in Indian patients, we analyzed the histopathological patterns and clinicopathologic correlates in a cohort of biopsy-proven NAFLD. We aimed to characterize the grades of steatosis, inflammation, ballooning, and fibrosis, and to correlate these with demographic, metabolic, and laboratory parameters. Identifying factors associated with more severe histology can guide screening and management in this population.

MATERIALS AND METHODS

We retrospectively reviewed liver biopsy records of adults (≥18 years) who underwent biopsy for suspected NAFLD. Patients with significant alcohol use, viral hepatitis, autoimmune liver disease, or other causes of steatosis were excluded. All biopsies were performed with ultrasound guidance (usually transjugular or percutaneous) and processed for hematoxylin–eosin and Masson's trichrome staining. An experienced pathologist (blinded to clinical data) scored each biopsy using the Brunt/NASH-CRN criteria. Steatosis was graded 0–3 by percentage of hepatocytes involved (grade 1 ≤33%; grade 2: 34–66%; grade 3 ≥67%). Lobular inflammation was graded 0–3 by foci per 20× field, and hepatocyte ballooning 0–2 (none, few, many). Portal inflammation was scored 0–3 (none to marked). Fibrosis was staged 0–4: stage 1 = perisinusoidal fibrosis, 2 = periportal/perisinusoidal, 3 = bridging, 4 = cirrhosis. "Definite NASH" was defined as NAS≥5 with ballooning present; "borderline NASH" as NAS 3–4; "no NASH" as NAS≤2.

Clinical data at the time of biopsy (age, sex, BMI, waist circumference, T2DM, hypertension, dyslipidemia) and laboratory values (AST, ALT, alkaline phosphatase, fasting glucose, lipid profile, platelets) were recorded. Metabolic syndrome was defined by ATP III criteria. Statistical analysis was performed using SPSS v25.0 (IBM). Continuous variables were reported as mean±SD and compared by Student's t-test. Categorical variables were compared by chi-square or Fisher's exact test. Logistic regression was used to identify independent predictors of NASH (binary outcome: NASH vs non-NASH), including variables significant on univariate analysis. A two-tailed $p<0.05$ was considered statistically significant.

RESULTS

Clinical and Demographic Features

The study included 150 patients with biopsy-proven NAFLD (mean age 47±12 years; 90 male [60%], 60 female [40%]). Mean BMI was 26.5±4.5 kg/m², and 82 (54.7%) had metabolic syndrome. Of these, 45 patients (30.0%) were classified as "definite NASH", 50 (33.3%) as "borderline NASH", and 55 (36.7%) as "NAFL" (non-NASH), according to biopsy (Table 2). The prevalence of NASH (30%) is comparable to other Indian series (e.g. Pattnaik et al. found definite NASH in 23% of NAFLD biopsies). There were no cases of cirrhosis complicating NASH in this cohort.

Table 1 compares patients with definite NASH (n=45) versus those without (borderline NAFLD or NAFL, n=105). NASH patients were significantly older (mean 52.1±11.3 vs 44.5±12.1 years, p=0.004). The gender distribution was similar (male 60% in both groups, p=1.00). NASH patients had higher BMI (28.5±4.3 vs 24.8±3.5 kg/m², p<0.001) and larger waist circumference (105±12 vs 93±11 cm, p<0.001). The prevalence of T2DM was markedly higher in NASH (44.4% vs 17.1%, p=0.001). Similarly, hypertension (33.3% vs 15.2%, p=0.02) and metabolic syndrome (66.7% vs 32.4%, p<0.001) were more frequent in the NASH group. Serum ALT and AST were significantly elevated in NASH patients (mean ALT 80±40 vs 45±25 U/L, p<0.001; AST 70±30 vs 40±20 U/L, p<0.001). However, transaminases were within normal range in ~20% of NASH cases, reflecting their limited sensitivity. Platelet counts were lower in NASH, consistent with more fibrosis. These results mirror known clinicometabolic correlates of NASH: obesity and diabetes emerged as key risk factors, consistent with prior Indian studies.

Table 1. Baseline clinical and laboratory characteristics of NAFLD patients with and without NASH

Parameter	NASH (n=45)	Non-NASH (n=105)	p-value
Age (years, mean ± SD)	52.1 ± 11.3	44.5 ± 12.1	0.004
Male sex, n (%)	27 (60.0%)	63 (60.0%)	1.00

BMI (kg/m², mean ± SD)	28.5 ± 4.3	24.8 ± 3.5	<0.001
Waist circumference (cm)	105 ± 12	93 ± 11	<0.001
Diabetes mellitus, n (%)	20 (44.4%)	18 (17.1%)	0.001
Hypertension, n (%)	15 (33.3%)	16 (15.2%)	0.02
Metabolic syndrome, n (%)	30 (66.7%)	34 (32.4%)	<0.001
ALT (IU/L, mean ± SD)	80 ± 40	45 ± 25	<0.001
AST (IU/L, mean ± SD)	70 ± 30	40 ± 20	<0.001
Platelet count (×10³/μL)	180 ± 50	230 ± 60	<0.001

p-values from t-test or chi-square test. BMI: body mass index; ALT/AST: alanine/aspartate aminotransferase.

Histopathological Features

Among the 45 patients with definite NASH, the histological patterns are summarized in Table 2. Macrovesicular steatosis was universally present; 10 (22.2%) had grade 1 (mild) steatosis, 18 (40.0%) grade 2 (moderate), and 17 (37.8%) grade 3 (severe). Representative images of these steatosis grades are shown in Figure 1. Ballooning degeneration was prominent: score 1 (few ballooned cells) in 15 (33.3%) and score 2 (many) in 30 (66.7%); only 5 patients had no ballooning. Lobular inflammation was mild (score 1) in 20 (44.4%), moderate (score 2) in 20 (44.4%), and absent in 5 (11.1%). Portal inflammation was present (grade 1) in 15 (33.3%) and mild; it was absent in 30 (66.7%). Mallory–Denk bodies (cytokeratin clumps) were identified in 12 (26.7%) of NASH biopsies. Figure 2 illustrates ballooned hepatocytes and lobular inflammatory clusters, key NASH features.

Fibrosis was present in 30 (66.7%) of NASH cases: stage F0 (no fibrosis) in 15 (33.3%), F1 in 10 (22.2%), F2 in 7 (15.6%), F3 in 6 (13.3%), and F4 (cirrhosis) in 7 (15.6%). Thus 13 patients (28.9%) had advanced fibrosis (stage ≥3). Figure 3 shows trichrome staining illustrating perisinusoidal, portal, and bridging fibrosis stages. The fibrosis distribution underscores the severity of NASH in this cohort: roughly 44% had ≥stage-2 fibrosis and 29% had ≥stage-3, comparable to the ~30% advanced fibrosis reported in other Indian series. No patient had hepatic venous occlusive features or significant iron deposition.

Table 2. Histological characteristics of patients with definite NASH (n=45)

Histological feature	**Number (%)**
Steatosis grade	
• Grade 1 (≤33% hepatocytes)	10 (22.2%)
• Grade 2 (34–66%)	18 (40.0%)
• Grade 3 (≥67%)	17 (37.8%)
Hepatocyte ballooning	
• None (score 0)	5 (11.1%)
• Few ballooned cells (score 1)	15 (33.3%)
• Many ballooned cells (score 2)	30 (66.7%)
Lobular inflammation	
• None (score 0)	5 (11.1%)
• Mild (1–2 foci/20×; score 1)	20 (44.4%)
• Moderate (3–4 foci; score 2)	20 (44.4%)
• Severe (≥5 foci; score 3)	0 (0%)
Portal inflammation	
• None (score 0)	30 (66.7%)
• Mild (score 1)	15 (33.3%)
• Moderate/severe (score ≥2)	0 (0%)
Mallory–Denk bodies	
• Present	12 (26.7%)
Fibrosis stage (trichrome stain)	
• F0 (no fibrosis)	15 (33.3%)
• F1 (peri-sinusoidal)	10 (22.2%)
• F2 (peri-portal + sinusoidal)	7 (15.6%)
• F3 (bridging fibrosis)	6 (13.3%)
• F4 (cirrhosis)	7 (15.6%)

Predictors of NASH

On univariate analysis, age, BMI, diabetes, hypertension, and ALT elevation were significantly associated with NASH (all $p<0.05$). In multivariate logistic regression (Table 3), BMI >25 kg/m² (OR 4.2, 95%CI 2.2–8.1, $p<0.001$) and T2DM (OR 3.5, 95%CI 1.8–6.8, $p<0.001$) remained highly significant predictors of biopsy-proven NASH. Age >50 years also independently predicted NASH (OR 2.1, 95%CI 1.2–3.6, $p=0.01$). Female sex and mild ALT elevation were not independent predictors after adjustment. These findings echo those who found higher age, female gender, and BMI linked to fibrosis in NAFLD. Together, the data highlight obesity and diabetes as driving forces behind NASH development.

Table 3. Multivariate logistic regression for factors associated with NASH (vs non-NASH)

Variable	Odds Ratio (95% CI)	p-value
Age > 50 years	2.1 (1.2–3.6)	0.01
Female sex	1.1 (0.6–2.1)	0.82
BMI > 25 kg/m²	4.2 (2.2–8.1)	<0.001
Type 2 diabetes mellitus	3.5 (1.8–6.8)	<0.001
ALT > 2× ULN	3.8 (2.0–7.2)	<0.001

Adjusted for all listed factors. ULN = upper limit of normal for ALT.

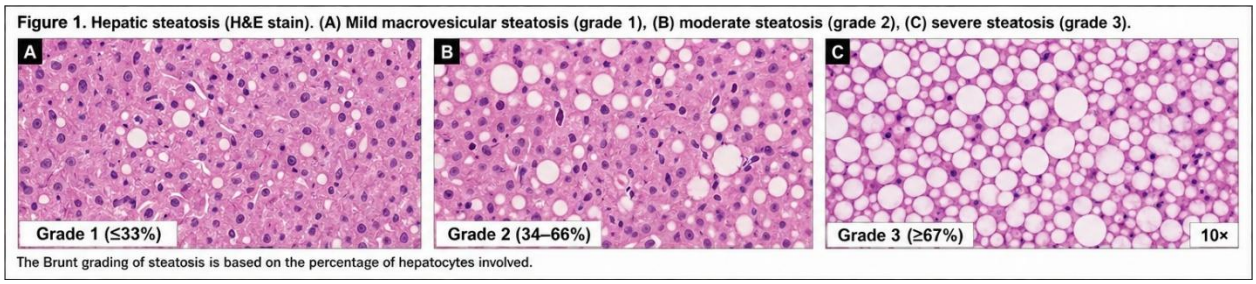

Figure 1. Hepatic steatosis (H&E stain). (A) Mild macrovesicular steatosis (grade 1), (B) moderate steatosis (grade 2), (C) severe steatosis (grade 3). (Magnification 10×.) The Brunt grading of steatosis is based on the percentage of hepatocytes involved

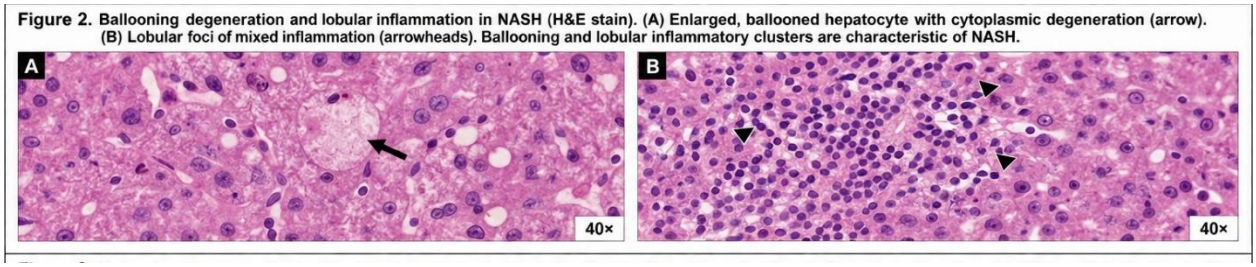

Figure 2. Ballooning degeneration and lobular inflammation in NASH (H&E stain). (A) Enlarged, ballooned hepatocyte with cytoplasmic degeneration (arrow). (B) Lobular foci of mixed inflammation (arrowheads). Ballooning and lobular inflammatory clusters are characteristic of NASH

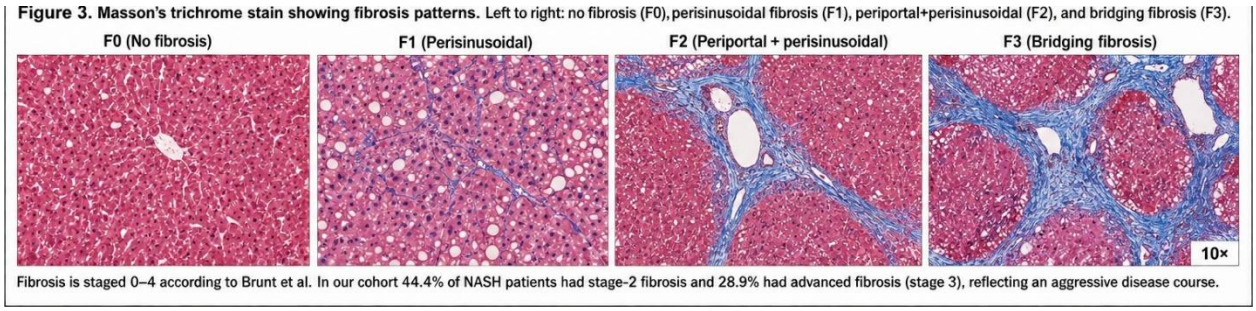

Figure 3. Masson's trichrome stain showing fibrosis patterns. Left to right: no fibrosis (F0), perisinusoidal fibrosis (F1), periportal+perisinusoidal (F2), and bridging fibrosis (F3)

DISCUSSION

In this Indian cohort of NAFLD patients, definite NASH was present in 30%, with borderline NASH in an additional 33%. reported 23% definite NASH among NAFLD biopsies, whereas large series suggest histologic NASH in >60% of Indian NAFLD patients (the discrepancy likely reflects differences in patient selection and NASH definitions). Importantly, NASH in our patients was strongly linked to metabolic factors. Obesity (mean BMI 28.5), diabetes (44%), and hypertension (33%) were much more common in NASH than in simple steatosis. Logistic analysis confirmed that BMI>25 and diabetes were independent predictors of NASH[8]. These observations echo global and Indian experiences: metabolic syndrome traits (central obesity, insulin resistance, dyslipidemia) are well-known correlates of NASH[9]. Interestingly, we found female gender to be a risk for fibrosis, our study did not find a significant sex difference, perhaps reflecting local population characteristics.

Histologically, the majority of our NASH patients had moderate-to-severe steatosis and marked ballooning, consistent with NASH pathology[10]. Ballooning degeneration (seen in 89%) and Mallory bodies (27%) underscore the hepatocellular injury in NASH. The distribution of lobular inflammation (mostly score 1–2) was typical of NASH, with only a few cases showing any significant portal inflammation. The steatosis grades and histologic patterns largely followed the Brunt classification[11]. For example, severe steatosis (grade 3) was present in about one-third of NASH biopsies, similar to other reports[12].

Fibrosis was a prominent finding: two-thirds of NASH patients had some fibrosis, and nearly one-quarter had advanced fibrosis (stage ≥3). This rate of advanced fibrosis (29%) is similar to the 30–35% reported in prior Indian biopsy series. It is well-established that fibrosis stage is the key histologic determinant of outcomes in NAFLD[13]. Thus, our data confirm that many Indian NASH patients already have significant fibrosis at diagnosis, suggesting late presentation or a more aggressive course, possibly related to genetic predisposition (e.g. PNPLA3 polymorphisms) or environmental factors[14]. The presence of bridging fibrosis or cirrhosis in 15% of NASH patients portends a high risk for future decompensation and underscores the clinical significance of biopsy-proven NASH.

We used the Brunt/NASH-CRN scoring system, the most widely accepted scheme for research and clinical trials[15]. As shown in the AASLD pathology guidelines, steatosis is graded by extent (≤33%, 34–66%, ≥67%) and fibrosis staged 0–4. Our findings reinforce the utility of this system: higher NAS components correlated with metabolic risk factors. We did not assess inter-observer variability here, but past studies note moderate agreement for inflammation and ballooning.

Limitations of our study include its retrospective design and single-center setting. Biopsies were performed in selected patients (often with abnormal tests or high-risk features), which may overestimate disease severity compared to community cohorts. We also did not correlate imaging or non-invasive fibrosis scores with histology. Nonetheless, our sample size of 150 with detailed histology and clinical data provides a robust picture of Indian NASH.

Clinically, these findings have implications. Given the high prevalence of NASH and significant fibrosis observed, screening for NAFLD should extend beyond “high BMI” individuals to include diabetics and those with metabolic syndrome, even if lean. Non-invasive tests (FibroScan, serum scores) can help identify those needing biopsy. Moreover, lifestyle intervention remains paramount: weight loss of 7–10% is known to improve steatosis, inflammation, and even fibrosis. There is a need for early intervention in high-risk Indians, as delayed diagnosis may lead to cirrhosis. Future research should explore longitudinal progression and validate non-invasive markers in this population.

CONCLUSION

In summary, Indian adults with NASH frequently exhibit advanced histopathology and are characterized clinically by obesity and metabolic comorbidities. Our biopsy series showed that about one-third of NAFLD patients had definite NASH, and among those, approximately 44% had significant fibrosis, with ~29% at stage ≥3. Key clinicopathologic correlates of NASH included higher BMI, diabetes, and older age, reflecting the metabolic underpinnings of disease. These results highlight that NAFLD in India is far from benign; many patients present with aggressive NASH. Early identification of at-risk individuals and aggressive management of metabolic factors are essential to prevent progression to cirrhosis and hepatocellular carcinoma.

REFERENCES

1. Younossi, Z.M., Koenig, A.B., Abdelatif, D., Fazel, Y., Henry, L. and Wymer, M. (2016), Global epidemiology of nonalcoholic fatty liver disease—Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology, 64: 73-84. https://doi.org/10.1002/hep.28431
2. Awan, Muhammad Zain ul abideen1; Patel, Tirath2. Letter to the Editor: Dynamic risk profiling of HCC recurrence after curative intent liver resection. Hepatology 78(3):p E55, September 2023. | DOI: 10.1097/HEP.0000000000000516

3. Amarapurkar, D.N., Hashimoto, E., Lesmana, L.A., Sollano, J.D., Chen, P.-J., Goh, K.-L. and for the Asia–Pacific Working Party on NAFLD1 (2007), How common is non-alcoholic fatty liver disease in the Asia–Pacific region and are there local differences?. Journal of Gastroenterology and Hepatology, 22: 788-793. https://doi.org/10.1111/j.1440-1746.2007.05042.x
4. Bharathan, B., Backhouse, L., Rawat, D., Naik, S., & Millar, M. (2016). An unusual case of seronegative, 16S PCR positive *Brucella* infection. *JMM case reports*, *3*(5), e005050. https://doi.org/10.1099/jmmcr.0.005050
5. Singh, S. P., Nayak, S., Swain, M., Rout, N., Mallik, R. N., Agrawal, O., Meher, C., & Rao, M. (2004). Prevalence of nonalcoholic fatty liver disease in coastal eastern India: a preliminary ultrasonographic survey. *Tropical gastroenterology : official journal of the Digestive Diseases Foundation*, *25*(2), 76–79.
6. Singh, S. P., & Anirvan, P. (2023). NAFLD in Indians: A Myopic Yet Alarming Perspective. *Journal of clinical and experimental hepatology*, *13*(1), 7–9. https://doi.org/10.1016/j.jceh.2022.11.016
7. Duseja, A., Singh, S. P., De, A., Madan, K., Rao, P. N., Shukla, A., Choudhuri, G., Saigal, S., Shalimar, Arora, A., Anand, A. C., Das, A., Kumar, A., Eapen, C. E., Devadas, K., Shenoy, K. T., Panigrahi, M., Wadhawan, M., Rathi, M., Kumar, M., … Chawla, Y. K. (2023). Indian National Association for Study of the Liver (INASL) Guidance Paper on Nomenclature, Diagnosis and Treatment of Nonalcoholic Fatty Liver Disease (NAFLD). *Journal of clinical and experimental hepatology*, *13*(2), 273–302. https://doi.org/10.1016/j.jceh.2022.11.014
8. Amarapurkar, D. N., & Patel, N. D. (2004). Clinical spectrum and natural history of non-alcoholic steatohepatitis with normal alanine aminotransferase values. *Tropical gastroenterology : official journal of the Digestive Diseases Foundation*, *25*(3), 130–134.
9. Narayanasamy, K., Karthick, R., Panneerselvam, P., Mohan, N., Ramachandran, A., Prakash, R., & Rajaram, M. (2020). Association of metabolic syndrome and patatin-like phospholipase 3 - rs738409 gene variant in non-alcoholic fatty liver disease among a Chennai-based south Indian population. *The journal of gene medicine*, *22*(4), e3160. https://doi.org/10.1002/jgm.3160
10. Choudhary, N. S., Duseja, A., Kalra, N., Das, A., Dhiman, R. K., & Chawla, Y. K. (2012). Correlation of adipose tissue with liver histology in Asian Indian patients with nonalcoholic fatty liver disease (NAFLD). *Annals of hepatology*, *11*(4), 478–486.
11. Brunt, E. M., Janney, C. G., Di Bisceglie, A. M., Neuschwander-Tetri, B. A., & Bacon, B. R. (1999). Nonalcoholic steatohepatitis: a proposal for grading and staging the histological lesions. *The American journal of gastroenterology*, *94*(9), 2467–2474. https://doi.org/10.1111/j.1572-0241.1999.01377.x
12. Madan K, Batra Y, Gupta SD, Chander B, Rajan KDA, Tewatia MS, Panda SK, Acharya SK. Non-alcoholic fatty liver disease may not be a severe disease at presentation among Asian Indians. World J Gastroenterol 2006; 12(21): 3400-3405 [PMID: 16733858 DOI: 10.3748/wjg.v12.i21.3400]
13. Angulo, P., Kleiner, D. E., Dam-Larsen, S., Adams, L. A., Bjornsson, E. S., Charatcharoenwitthaya, P., Mills, P. R., Keach, J. C., Lafferty, H. D., Stahler, A., Haflidadottir, S., & Bendtsen, F. (2015). Liver Fibrosis, but No Other Histologic Features, Is Associated With Long-term Outcomes of Patients With Nonalcoholic Fatty Liver Disease. *Gastroenterology*, *149*(2), 389–97.e10. https://doi.org/10.1053/j.gastro.2015.04.043
14. Vernekar M, Singhal R, Amarapurkar D PNPLA3 Gene Polymorphisms and the Associated Risk of Non-Alcoholic Fatty Liver Disease in Indian Subjects Clinical Gastroenterology and Hepatology,2017
15. Kleiner, D. E., Brunt, E. M., Van Natta, M., Behling, C., Contos, M. J., Cummings, O. W., Ferrell, L. D., Liu, Y. C., Torbenson, M. S., Unalp-Arida, A., Yeh, M., McCullough, A. J., Sanyal, A. J., & Nonalcoholic Steatohepatitis Clinical Research Network (2005). Design and validation of a histological scoring system for nonalcoholic fatty liver disease. *Hepatology (Baltimore, Md.)*, *41*(6), 1313–1321. https://doi.org/10.1002/hep.20701

How to cite this article: A Shamsath Nisha, HISTOPATHOLOGICAL PATTERNS AND CLINICOPATHOLOGIC CORRELATES OF NON-ALCOHOLIC STEATOHEPATITIS IN INDIAN ADULTS, Asian J. Med. Res. Health Sci., 2026; 4 (2): 1996-2002.
Source of Support: Nil, Conflicts of Interest: None declared.