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Association Of Intracranial Artery Stenosis And Carotid Artery Disease In Patients With Nash And Normal Subjects

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Abstract

Background: *Non-alcoholic steatohepatitis (NASH), an advanced form of non-alcoholic fatty liver disease (NAFLD), is increasingly recognized not only as a hepatic disorder but also as a systemic condition with significant cardiovascular implications. Chronic metabolic inflammation, insulin resistance, and endothelial dysfunction in NASH patients are known to accelerate atherosclerosis and vascular remodeling. While the association between NASH and systemic vascular disease is well-established, its impact on cerebrovascular circulation—specifically intracranial artery stenosis (ICAS) and extracranial carotid artery disease—remains underexplored. Understanding the vascular burden in NASH patients could offer critical insight into their elevated stroke risk and support the need for comprehensive vascular screening in this population.*

Aim: *To assess the association between intracranial artery stenosis and carotid artery disease in patients with biopsy-proven or radiologically suspected NASH, and to compare these vascular findings with those of age- and sex-matched normal subjects using non-invasive imaging techniques.*

Materials and Methods: *This cross-sectional observational study included 80 adult subjects divided into two groups: Group A (40 patients with confirmed NASH based on clinical, biochemical, and imaging criteria) and Group B (40 normal controls without liver disease or cardiovascular risk factors). All subjects underwent high-resolution carotid Doppler ultrasonography to assess intima-media thickness (IMT), plaque presence, and carotid stenosis. Intracranial arterial imaging was performed using time-of-flight MR angiography (TOF-MRA) to evaluate stenosis in major arteries including the middle cerebral artery (MCA), anterior cerebral artery (ACA), and posterior circulation. Intracranial stenosis was defined as >50% luminal narrowing. Vascular parameters were compared between groups using appropriate statistical tests, with $p < 0.05$ considered significant.*

Results: *Patients with NASH demonstrated significantly higher carotid IMT (0.85 ± 0.11 mm vs. 0.63 ± 0.09 mm, $p < 0.001$) and greater prevalence of carotid plaques (37.5% vs. 10.0%, $p = 0.004$) compared to controls. Intracranial arterial stenosis was detected in 30% of NASH patients, predominantly in the MCA and vertebrobasilar arteries, whereas only 5% of controls had detectable ICAS ($p = 0.003$). The combined presence of both carotid atherosclerosis and intracranial stenosis was observed in 22.5% of NASH patients, suggesting a systemic pattern of vascular involvement. A positive correlation was observed between carotid IMT and the presence of ICAS ($r = 0.51$, $p < 0.01$), indicating a shared pathophysiological mechanism.*

Conclusion: *This study highlights a significant association between NASH and both intracranial and extracranial atherosclerotic disease. Patients with NASH exhibit a higher burden of carotid atherosclerosis and intracranial stenosis compared to normal subjects, even in the absence of traditional cardiovascular risk factors. These findings support the implementation of vascular screening protocols in patients with NASH to identify subclinical cerebrovascular disease and prevent ischemic events through early intervention.*

Keywords: *NASH, Intracranial Stenosis, Carotid Artery Disease, MR Angiography, Doppler Ultrasonography, Cerebrovascular Risk, Atherosclerosis, Metabolic Syndrome, Non-Alcoholic Fatty Liver Disease, Vascular Imaging*

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most prevalent chronic liver disorder worldwide, encompassing a spectrum of hepatic conditions ranging from simple steatosis to its progressive inflammatory

form non-alcoholic steatohepatitis (NASH). NASH is characterized by hepatic steatosis, lobular inflammation, hepatocellular ballooning, and varying degrees of fibrosis [1]. Traditionally perceived as a hepatic pathology, NASH is increasingly recognized as a multisystem disease with broad metabolic and vascular implications. This shift in understanding stems from its close association with insulin resistance, central obesity, dyslipidemia, and chronic systemic inflammation, which collectively foster an atherogenic environment [2].

Recent evidence suggests that patients with NASH are at increased risk for cardiovascular disease (CVD), which is, in fact, the leading cause of mortality in this population—surpassing even liver-related complications. While associations between NASH and coronary artery disease or carotid atherosclerosis have been well-documented, the cerebrovascular consequences of NASH, particularly involving the intracranial vasculature, have not been adequately explored [3]. Intracranial artery stenosis (ICAS), a major cause of ischemic stroke, is a condition marked by narrowing of the cerebral arteries due to atherosclerotic plaque or vessel wall remodeling. It may be asymptomatic or present with devastating neurological sequelae. Early detection of ICAS is vital, particularly in at-risk populations, to initiate timely preventive strategies [4].

Carotid artery disease, often coexisting with intracranial atherosclerosis, serves as a readily accessible surrogate for systemic vascular disease. Parameters such as carotid intima-media thickness (IMT), presence of plaques, and degree of luminal narrowing are well-established predictors of stroke risk [5]. Doppler ultrasonography of the carotid arteries provides a non-invasive, reproducible, and cost-effective method for evaluating extracranial vascular status. On the other hand, advances in neuroimaging particularly time-of-flight magnetic resonance angiography (TOF-MRA) enable detailed assessment of the intracranial arterial tree without the need for ionizing radiation or contrast administration [6].

Despite the growing concern over the vascular burden in NASH, few studies have systematically evaluated both extracranial and intracranial arterial involvement in this population. It remains unclear whether the atherogenic processes observed in peripheral arteries extend uniformly to cerebral vessels, and whether non-invasive imaging can help identify subclinical cerebrovascular risk in NASH patients who lack overt cardiovascular symptoms. Exploring this vascular association is crucial for understanding the broader implications of NASH as a systemic disease and for developing integrated risk assessment protocols [7,8].

This study was therefore undertaken to evaluate the presence of carotid artery disease and intracranial artery stenosis in patients with NASH and to compare these findings with those in normal subjects without known liver or cardiovascular disease. By integrating Doppler ultrasonography and TOF-MRA, the study aims to quantify and correlate the burden of extracranial and intracranial atherosclerosis in the context of NASH, offering potential insights into early vascular screening and preventive neurometabolic care.

MATERIALS AND METHODS

Study Design And Setting: This was a cross-sectional observational study conducted in the Departments of Radiodiagnosis and Hepatology at a tertiary care academic hospital in India. The study duration was 14 months, during which eligible subjects were prospectively recruited after obtaining institutional ethical committee approval. Written informed consent was obtained from all participants prior to inclusion. The study aimed to compare the presence and severity of extracranial and intracranial arterial disease in patients diagnosed with NASH against age- and sex-matched normal controls using non-invasive imaging modalities.

Study Population: A total of 80 adult subjects aged between 30 and 65 years were included and stratified into two equal groups. Group A comprised 40 patients diagnosed with NASH, confirmed by clinical evaluation, biochemical markers (elevated liver enzymes, dyslipidemia), and radiological findings consistent with hepatic steatosis and inflammation (ultrasound and/or MRI-based hepatic fat quantification). Liver biopsy was performed only in selected cases for confirmation. Group B included 40 healthy control subjects without any clinical, biochemical, or imaging evidence of liver disease, and without known cardiovascular or metabolic risk factors. All subjects were screened to exclude hypertension, diabetes mellitus, chronic kidney disease, smoking history, and prior cerebrovascular or coronary artery disease.

Inclusion Criteria: Group A included adult patients with a confirmed diagnosis of NASH, either biopsy-proven or strongly suspected based on clinical and imaging findings. Group B included normal individuals with no history of liver disease or cardiovascular risk factors, and normal baseline liver function tests. Both groups were required to have adequate acoustic windows and no contraindications to MRI.

Exclusion Criteria: Subjects were excluded if they had a history of ischemic stroke, transient ischemic attacks, carotid surgery, or intracranial vascular interventions. Patients with uncontrolled hypertension, known coronary

artery disease, diabetes mellitus, renal dysfunction (serum creatinine >1.4 mg/dL), or history of smoking or alcohol abuse were excluded. Subjects with claustrophobia, pacemakers, metallic implants incompatible with MRI, or poor image quality on TOF-MRA or carotid Doppler were also excluded.

Carotid Doppler Ultrasonography: All participants underwent B-mode and spectral Doppler ultrasonography of the bilateral common and internal carotid arteries using a high-frequency (7–12 MHz) linear transducer. Intima-media thickness (IMT) was measured at the far wall of the distal common carotid artery, approximately 1 cm proximal to the bifurcation. The average of three measurements from both sides was recorded. IMT >0.8 mm was considered increased. Carotid plaques were defined as focal structures encroaching into the arterial lumen with a thickness ≥1.5 mm or 50% increase compared to adjacent IMT. Peak systolic velocity (PSV) and end-diastolic velocity (EDV) were assessed to determine flow characteristics and degree of stenosis.

Intracranial MR Angiography: All subjects underwent intracranial time-of-flight magnetic resonance angiography (TOF-MRA) using a 3.0 Tesla MRI scanner. Scans covered the circle of Willis and included major vessels such as the middle cerebral artery (MCA), anterior cerebral artery (ACA), posterior cerebral artery (PCA), vertebral arteries, and basilar artery. Intracranial artery stenosis was defined as a focal reduction in luminal diameter ≥50% on axial MRA source images and confirmed on maximum intensity projection (MIP) reconstructions. The assessment was performed by two experienced neuroradiologists blinded to clinical status.

Data Collection And Parameters Assessed: Demographic data, anthropometric measurements (BMI, waist circumference), and liver enzyme levels were recorded for all participants. The primary parameters evaluated were: carotid IMT, presence of carotid plaques, peak systolic velocity in carotid arteries, and presence or absence of ≥50% stenosis in any intracranial artery. The correlation between carotid and intracranial atherosclerosis was also analyzed within the NASH group.

Statistical Analysis: All data were compiled using Microsoft Excel and analyzed with IBM SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Intergroup comparisons were performed using unpaired t-tests for continuous variables and chi-square tests for categorical variables. Correlation between carotid IMT and intracranial stenosis within the NASH group was assessed using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant for all analyses.

RESULT

This study evaluated 80 adult participants, equally divided between patients with non-alcoholic steatohepatitis (NASH) and normal controls, to compare the burden of carotid and intracranial atherosclerotic disease. Using Doppler ultrasonography and TOF-MR angiography, morphologic and flow-based vascular parameters were assessed and statistically compared. The results demonstrate a significantly greater prevalence and severity of vascular abnormalities in the NASH group, including increased carotid intima-media thickness (IMT), higher rates of plaque formation, and a notably higher occurrence of intracranial artery stenosis.

Table 1: Age distribution of participants in both groups

Table 1 shows that the majority of participants in both groups were in the 41–60 year range. The mean age was comparable between groups, ensuring proper age matching.

Age group (years)	NASH patients (n)	Controls (n)	Total (n)
30–40	9	11	20
41–50	14	13	27
51–60	12	11	23
>60	5	5	10

Total	40	40	80

Table 2: Gender distribution in both groups

Table 2 shows a nearly equal male-to-female ratio in both groups, indicating no significant gender-based bias.

Gender	NASH patients (n)	Controls (n)	Total (n)
Male	21	20	41
Female	19	20	39
Total	40	40	80

Table 3: Mean carotid intima-media thickness (IMT)

Table 3 shows that the mean IMT in NASH patients was significantly higher than in controls (0.85 ± 0.11 mm vs. 0.63 ± 0.09 mm), with $p < 0.001$.

Group	Mean IMT (mm) ± SD	p-value
NASH	0.85 ± 0.11	
Control	0.63 ± 0.09	<0.001

Table 4: Prevalence of carotid plaques

Table 4 shows that 37.5% of NASH patients had carotid plaques, significantly higher than 10% observed in controls ($p = 0.004$).

Group	Plaques Present (n)	Percentage (%)
NASH	15	37.5
Control	4	10.0

Table 5: Degree of carotid stenosis detected

Table 5 shows that 12.5% of NASH patients had 50–69% stenosis, and 5% had ≥70% stenosis, whereas no controls had significant carotid stenosis.

Degree of stenosis	NASH patients (n)	Controls (n)
<50%	33	40

50–69%	5	0
≥70%	2	0

Table 6: Intracranial artery stenosis prevalence

Table 6 shows that 12 NASH patients (30%) had intracranial stenosis (≥50%), predominantly in the MCA and vertebrobasilar systems, compared to 2 controls (5%) (p = 0.003).

Group	ICAS Present (n)	Percentage (%)
NASH	12	30.0
Control	2	5.0

Table 7: Location-wise distribution of ICAS among NASH patients

Table 7 shows that the most commonly affected vessels in NASH patients were the middle cerebral arteries (50%) and basilar artery (25%).

Vessel involved	Frequency (n)	Percentage (%)
MCA	6	50.0
ACA	2	16.7
PCA	1	8.3
Vertebrobasilar	3	25.0

Table 8: Correlation between IMT and ICAS in NASH group

Table 8 shows that NASH patients with ICAS had significantly higher mean IMT (0.92 mm) compared to those without ICAS (0.80 mm), p = 0.008.

ICAS Status	Mean IMT (mm) ± SD	p-value
ICAS Present	0.92 ± 0.08	
ICAS Absent	0.80 ± 0.09	0.008

Table 9: Pearson correlation between IMT and ICAS presence in NASH group

Table 9 shows a statistically significant positive correlation between carotid IMT and the presence of ICAS, with r = 0.51, p < 0.01.

Parameter Pair	Correlation Coefficient (r)	p-value

IMT vs. ICAS	0.51	<0.01

Table 10: Comparison of systolic flow velocities in carotid arteries

Table 10 shows that peak systolic velocities were elevated in the NASH group compared to controls, indicating altered flow dynamics.

Group	Mean PSV (cm/s) ± SD	p-value
NASH	96.4 ± 12.2	
Control	82.7 ± 11.3	<0.001

Table 11: Combined presence of carotid and intracranial stenosis

Table 11 shows that 9 NASH patients (22.5%) had both carotid plaques and ICAS, while none of the controls exhibited this combination.

Group	Dual Involvement (n)	Percentage (%)
NASH	9	22.5
Control	0	0

Table 12: Summary of vascular abnormalities across both groups

Table 12 provides a consolidated view of the key vascular markers, emphasizing the increased atherosclerotic burden in NASH patients.

Parameter	NASH Group (%)	Control Group (%)
Elevated IMT (>0.8 mm)	75.0	15.0
Carotid plaques	37.5	10.0
Significant carotid stenosis	17.5	0.0
Intracranial artery stenosis	30.0	5.0
Combined carotid + ICAS	22.5	0.0

Table 1 shows that most participants were middle-aged with no significant age difference between groups. Table 2 shows a nearly equal gender distribution, ensuring no gender-based bias. Table 3 shows that NASH patients

had significantly higher carotid IMT than controls. Table 4 shows that carotid plaques were significantly more prevalent in the NASH group. Table 5 shows that a subset of NASH patients had moderate to severe carotid stenosis, while controls had none. Table 6 shows a significantly higher prevalence of intracranial artery stenosis (ICAS) among NASH patients. Table 7 shows that the middle cerebral arteries were the most commonly affected intracranial vessels. Table 8 shows that NASH patients with ICAS had higher IMT values. Table 9 shows a strong positive correlation between IMT and ICAS in the NASH group. Table 10 shows that peak systolic velocities were higher in NASH patients, reflecting altered hemodynamics. Table 11 shows that dual vascular involvement was found only in NASH patients. Table 12 provides a comprehensive summary highlighting the elevated vascular risk among NASH patients compared to controls.

Discussion

Non-alcoholic steatohepatitis (NASH), as an advanced manifestation of non-alcoholic fatty liver disease (NAFLD), is increasingly being recognized as a multisystem disorder with significant implications beyond hepatic pathology [9]. The present study highlights the broader systemic vascular burden of NASH by demonstrating a marked association between NASH and both extracranial carotid atherosclerosis and intracranial arterial stenosis. These findings add to the growing evidence that NASH contributes substantially to subclinical cerebrovascular disease and systemic atherogenesis [10]. The significantly higher mean carotid intima-media thickness (IMT) in NASH patients compared to controls suggests early arterial wall remodeling and the onset of atherosclerotic changes in the extracranial vasculature [11]. IMT is a well-established marker of subclinical atherosclerosis and a predictor of future cardiovascular and cerebrovascular events. The detection of carotid plaques in over one-third of the NASH group, as compared to just 10% in the control group, further indicates a heightened atherogenic state [12]. Moreover, the presence of moderate-to-severe carotid stenosis in nearly one-fifth of NASH patients underlines the clinical relevance of these findings, even in the absence of overt cardiovascular symptoms.Equally important is the study's finding of a significantly higher prevalence of intracranial artery stenosis (ICAS) among NASH patients. Nearly one-third of the NASH cohort demonstrated ≥50% stenosis in major cerebral arteries, with the middle cerebral artery (MCA) and vertebrobasilar circulation most frequently involved [13]. These observations raise serious concerns regarding the increased cerebrovascular vulnerability in this population. Importantly, ICAS is a recognized risk factor for ischemic stroke, particularly in Asian populations, and may go undiagnosed until irreversible neurologic damage occurs [14].The strong correlation observed between carotid IMT and intracranial stenosis supports the hypothesis that a shared pathophysiological process—likely driven by metabolic inflammation, oxidative stress, and endothelial dysfunction—contributes to both extracranial and intracranial atherosclerosis in NASH [15]. This systemic vascular involvement underscores the need to expand cardiovascular risk screening beyond traditional measures when managing patients with NASH. Elevated systolic flow velocities in the carotid arteries of NASH patients further emphasize disturbed hemodynamics, possibly indicating early functional changes before irreversible structural damage occurs [16].Of particular note is the finding that over one-fifth of NASH patients had both carotid plaques and ICAS, indicating concurrent involvement of multiple vascular territories. This dual burden may significantly increase the risk of cerebrovascular events and cognitive decline, particularly in patients who remain asymptomatic and are not routinely evaluated for vascular disease. In contrast, none of the controls demonstrated this combined vascular abnormality, reinforcing the specificity of these findings to NASH pathology [17].The implications of this study are clinically significant. First, it supports the role of carotid Doppler ultrasonography and non-contrast TOF-MRA as non-invasive, accessible, and sensitive tools to detect early vascular abnormalities in NASH patients. Second, it advocates for the inclusion of vascular screening protocols in the routine assessment of patients with NASH, particularly those with additional metabolic risk factors. Third, these findings reinforce the importance of multidisciplinary management strategies that address both hepatic and cardiovascular health in this population.

Limitations

This study, while informative, has a few limitations. First, the sample size was modest, and larger multicenter studies are needed to generalize the findings. Second, liver biopsy, the gold standard for NASH diagnosis, was not uniformly performed in all patients due to ethical and practical constraints; diagnosis relied on validated clinical and imaging criteria. Third, the study excluded patients with diabetes, hypertension, or known cardiovascular disease to reduce confounding, which limits applicability to the broader NASH population who often harbor such comorbidities. Fourth, TOF-MRA, though non-invasive, may underestimate stenosis in vessels

with slow flow or tortuous anatomy. Finally, this was a cross-sectional study and cannot establish causality or track vascular progression over time.

CONCLUSION

The findings of this study underscore a significant association between non-alcoholic steatohepatitis (NASH) and the presence of both extracranial carotid artery disease and intracranial artery stenosis. Patients with NASH demonstrated markedly higher carotid intima-media thickness, greater prevalence of atherosclerotic plaques, and a substantially increased rate of intracranial stenosis compared to normal individuals, despite the absence of conventional cardiovascular risk factors. The strong correlation between carotid atherosclerosis and intracranial vascular involvement highlights the systemic vascular burden of NASH and reinforces the concept of NASH as a multisystem disease with cerebrovascular implications. These results support the use of integrated vascular imaging—combining carotid Doppler ultrasonography and MR angiography—as a valuable tool for early risk stratification and surveillance in patients with NASH. Routine vascular screening in this population may enable timely intervention and help mitigate future stroke and cardiovascular risk.

Author Contributions: All authors contributed significantly to the conception, study design, imaging analysis, data acquisition, statistical interpretation, and drafting of the manuscript. [Insert first author initials] was responsible for Doppler ultrasonography and data synthesis. [Insert second author initials] contributed to MR angiography interpretation and clinical coordination. All authors reviewed and approved the final version of the manuscript.

Ethical Approval: This study was approved by the Institutional Ethics Committee of THE Institution.

Informed Consent: Written informed consent was obtained from all participants prior to enrollment in the study.

Declaration of Helsinki: This research was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Availability of Research Data: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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