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Original Research Article

The impact of NAFLD on diabetic complications in patients with T2DM

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ABSTRACT

Background: This study investigates the relationship between non-alcoholic fatty liver disease (NAFLD) and type 2 diabetes, focusing on the associated macrovascular and microvascular complications. The aim is to assess the impact of the temporal sequence of these conditions on patient outcomes.
Methods: An observational study was conducted over three months (October 2023 to December 2023) at Katihar Medical College, enrolling 91 patients diagnosed with type 2 diabetes and NAFLD. Participants were grouped based on the duration of NAFLD and type 2 diabetes, and a range of clinical, laboratory, and diagnostic assessments were conducted to evaluate macrovascular and microvascular complications. Statistical analyses were performed to compare the groups.
Results: The study found that Group C had significantly higher BMI, WHR, triglycerides, ALT, AST, γ-GT, and uric acid levels compared to Groups A and B. The prevalence of CAD and hypertension was significantly higher in Group C. Group B had a longer NAFLD duration and higher ALT and AST levels than Group A. These findings suggest a strong association between metabolic dysfunction, NAFLD, and diabetic complications.
Conclusion: The study concludes that the temporal sequence of NAFLD and type 2 diabetes significantly influences the prevalence of macrovascular complications, particularly coronary artery disease and hypertension, in affected patients.

Keywords: NAFLD, Type 2 diabetes, Coronary artery disease, Hypertension, Microvascular complications, Diabetic retinopathy

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is defined by the buildup of fat within the liver in the absence of significant alcohol intake.[1] Epidemiological research highlights that NAFLD has become a notable public health issue in China and various regions of Asia.[2–5]

This condition includes a range of liver abnormalities, from basic fat accumulation (steatosis) to inflammation (steatohepatitis), damage to hepatocytes, fibrosis, and, in more severe instances, advancement to cirrhosis or hepatocellular carcinoma. NAFLD is notably common in individuals diagnosed with type 2 diabetes mellitus (T2DM), impacting an estimated 50–75% of those living with this condition.[6,7] Furthermore, a multitude of research endeavours have demonstrated a prominent relationship between NAFLD and T2DM.[8–11] Studies indicate that NAFLD functions as a significant predictor of complications related to T2DM. Research suggests that NAFLD might be involved in increasing the risk of chronic kidney disease and cardiovascular disease.[12] Cross-sectional studies have illustrated a significant correlation between NAFLD and elevated carotid intima-media thickness, as well as an increased coronary artery calcium score, both of which serve as indicators of cardiovascular risk.[13–16] Furthermore, there is a connection between NAFLD and the onset of early left ventricular diastolic

dysfunction, as well as diminished myocardial perfusion, in individuals diagnosed with T2DM.[17] The condition is likewise linked to an increased prevalence and incidence of chronic kidney disease in individuals with T2DM.[18]

Numerous investigations into the association between NAFLD and diabetic complications have yet to clarify the temporal order of disease manifestation. The relationship between NAFLD in individuals with pre-existing T2DM and underlying metabolic syndrome risk factors is still ambiguous.

It is also uncertain whether NAFLD that occurs prior to the onset of T2DM indicates a longer duration of diabetes and its related complications. This study seeks to explore the variations in complications that arise depending on whether NAFLD manifests in individuals who already have T2DM or whether T2DM develops in individuals with a prior diagnosis of NAFLD.

METHODS

Study design

This observational study took place over three months (October 2023 to December 2023), at Katihar Medical College, Katihar, Bihar, India. A total of 91 patients were retrospectively recruited for the study. Ethical approval was obtained, and the study was carried out as per the standard protocols.

Study participants

The study included individuals who either abstained from alcohol or had an ethanol intake of <20 g/day. Patients with a history of chronic liver disease or those testing positive for hepatitis B or C were excluded.

Furthermore, individuals diagnosed with NAFLD had not received any prior medical treatment aimed at preventing liver injury before their inclusion in the study. The treatment plans for T2DM, hypertension, coronary artery disease (CAD), and other internal conditions were determined in accordance with the best clinical interests of the patients.

Patient grouping and diagnostic criteria

Participants were divided into three distinct groups as per their NAFLD status, as determined through abdominal ultrasonography, alongside the duration of their T2DM.

Group A

Patients diagnosed with T2DM.

Group B

Patients whose T2DM had been present longer than their NAFLD diagnosis.

Group C

Patients who were diagnosed with NAFLD before the onset of T2DM.

All patients underwent comprehensive clinical evaluations, including electrocardiograms, blood pressure measurements, and physical examinations such as Doppler ultrasound, electrophysiology assessments, fundoscopy, and cardiac computed tomography. Additionally, a detailed medical history was recorded for all participants.

Laboratory assessments

A series of laboratory investigations were conducted, including fasting blood glucose, glycated haemoglobin (HbA1c), and an oral glucose tolerance test. Liver and renal function tests were performed, along with lipid profile assessments and urinary albumin excretion tests. These biochemical evaluations provided essential insights into the metabolic and organ function status of the participants.

Assessment of diabetic complications

Macrovascular complications

The study examined macrovascular complications in individuals diagnosed with diabetes, with particular emphasis on CAD and hypertension. CAD was diagnosed upon the identification of stenosis exceeding 50% in significant coronary vessels, which include the left main, left anterior descending, left circumflex, and right coronary arteries, as confirmed through cardiac computed tomography.

Hypertension is characterised by a systolic blood pressure of 140 mmHg or higher and/or a diastolic pressure of 90 mmHg or higher. Measurements should be obtained while the individual is seated, utilising a mercury sphygmomanometer, and must be recorded on no fewer than three distinct occasions.

Microvascular complications

Microvascular complications evaluated included diabetic nephropathy, retinopathy, and peripheral neuropathy. Nephropathy was characterized by sustained protein presence in the urine across three successive assessments within a 12-month period.

Retinopathy was identified through signs such as retinal bleeding, lipid deposits, or swelling in the macula. Peripheral neuropathy was determined based on clinical features like ongoing numbness, tingling sensations, decreased perception of vibration, or the absence of reflexes in the knees or ankles.

Statistical analysis

Continuous variables were presented as mean±standard deviation, while categorical variables were expressed as percentages. Comparisons between groups were performed using the student's t-test for continuous variables.

RESULTS

The clinical and laboratory characteristics of the patients are shown in Table 1. The mean age of participants across the three groups was comparable, with group A having an average age of 55.7±11.5 years, Group B at 50.8±9.5 years, and group C at 51.5±10.6 years. The proportion of male participants was highest in group C (63.2%), followed by group A (54.1%) and group B (50.4%). Smoking prevalence was also greatest in group C (49.6%), with lower percentages in group A (42.5%) and Group B (40.7%).

Family history of T2DM was most frequently reported in group B (70.7%) and group C (63.2%), whereas group A had a slightly lower prevalence (51.6%). Similarly, a family history of hypertension was highest in group C (37.3%), compared to 28.0% in group A and 27.8% in group B. Coronary artery disease history showed minimal variation, with rates of 22.9% in group B, 15.0% in group C, and 14.9% in group A. Regarding metabolic parameters, BMI and waist-to-hip ratio (WHR) were significantly higher in groups B and C compared to group A.

The mean duration of diabetes was longest in group A (9.7±7.7 years), followed by group B (8.3±5.8 years) and Group C (5.0±5.2 years). Conversely, NAFLD duration was longest in Group B (10.1±7.0 years) compared to group C (3.1±4.0 years). Biochemical markers revealed notable differences in lipid and liver enzyme levels.

Triglyceride levels were significantly elevated in groups B and C compared to group A. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels were higher in Group B, whereas gamma-glutamyl transferase (γ-GT) was markedly increased in both groups B and C relative to group A (Table 1).

Table 1: Clinical and laboratory characteristics of study participants.

Variables	Group A (n=30)	Group B (n=31)	Group C (n=30)
Age (in years)	55.7±11.5	50.8±9.5	51.5±10.6
Percentage of men	54.1	50.4	63.2
Percentage of patient with smoking history	42.5	40.7	49.6
Percentage of patients with family history of			
T2DM	51.6	70.7	63.2
Hypertension	28.0	27.8	37.3
CAD	14.9	22.9	15.0
Duration (in years)			
T2DM	9.7±7.7	8.3±5.8	5.0±5.2†
NAFLD	3.1±4.0	10.1±7.0	-
Clinical parameters			
Systolic blood pressure (mmHg)	125.3±15.7	128.2±12.4	130.0±14.2
Diastolic blood pressure (mmHg)	74.0±12.6	79.2±7.4	79.8±8.5
Body mass index (kg/m²)	23.8±3.8	27.9±3.6	28.8±3.5
Waist-to-hip ratio	0.85±0.05	0.94 ±0.03	0.94±0.04
Blood glucose (mmol/l)	8.1±2.4	8.8±2.3	8.2±2.3
Glycated haemoglobin (HbA1c) (%)	8.2±2.5	8.0 ±1.7	8.0±2.1
Triglycerides (millimoles per liter)	1.5±0.6	2.6±2.1	2.4±1.6
Total cholesterol (millimoles per liter)	5.6±0.9	6.0±1.3	5.9±1.0
High-density lipoprotein cholesterol (millimoles per liter)	2.2±0.3	2.2±0.2	2.2±0.2
Low-density lipoprotein cholesterol (millimoles per liter)	3.9±0.7	4.2±0.9	4.0±0.8
Alanine aminotransferase (units per liter)	16.2±8.0	26.2±12.5	16.2±8.0
Aspartate aminotransferase (units per liter)	15.7±5.1	24.6±15.7	23.6±19.2
Γ-glutamyl transferase (units per liter)	21.4±18.8	38.3±28.9	47.2±38.0
Blood urea nitrogen (millimoles per liter)	5.9±2.1	5.2±1.3	5.5±1.5
Creatinine (micromoles per liter)	64.4±28.3	61.8±14.7	65.3±14.8
Creatinine clearance rate	115.3±36.4	116.9±22.6	111.4±28.7
Uric acid (micromoles per liter)	267.3±81.8	311.9±78.4	333.4±104.0
Urinary microalbumin (milligrams per 24 hours)	42.1±70.8	55.9±79.5	34.5±59.1
Urinary total protein (grams per 24 hours)	0.19±0.36	0.24±0.45	0.16±0.18

Table 2: Incidence of diabetic complications among study participants.

Group	CAD (%)	Hypertension (%)	DR (%)	DN (%)	DPN (%)
A (n=30)	25	28	26	19	45
B (n=31)	23	28	31	20	35
C (n=30)	40	48	23	17	32

The incidence of diabetic complications among the study participants. CAD was more prevalent in group C (40%) compared to groups A (25%) and B (23%). Similarly, hypertension was reported in 48% of patients in Group C, significantly higher than the rates in groups A and B (28% each). Diabetic retinopathy (DR) was most frequently observed in group B (31%), followed by group A (26%) and group C (23%). Diabetic nephropathy (DN) occurred in 20% of patients in group B, 19% in Group A, and 17% in Group C, showing minimal variation. However, diabetic peripheral neuropathy (DPN) was significantly more common in group A (45%), followed by group B (35%) and group C (32%) (Table 2).

DISCUSSION

This research indicates that individuals diagnosed with both NAFLD and T2DM demonstrate a greater occurrence of significant CAD and hypertension when contrasted with those who have type 2 diabetes followed by NAFLD, as well as those who have type 2 diabetes in isolation. This indicates that the existence of diabetes among patients with NAFLD serves as a significant predictor of macrovascular complications, including CAD and hypertension. NAFLD is a prevalent condition intricately connected to metabolic syndrome and its various components, such as insulin resistance, hypertension, and diabetes mellitus among others. Prior research has demonstrated a reciprocal association between NAFLD and T2DM, revealing that NAFLD may facilitate the onset of diabetes while also serving as an independent risk factor for CAD.[19] The current results are consistent with earlier studies, indicating that both groups with NAFLD and diabetes displayed higher levels of BMI, waist-to-hip ratio, triglycerides, ALT, AST, and γ-GT when compared to the group with diabetes alone.[20–22] Interestingly, the levels of uric acid were found to be the highest in the group with NAFLD and T2DM, supporting the results of a Chinese cross-sectional study that established a significant association between increased uric acid and NAFLD.[23]

The research revealed that 67.5% of patients with diabetes were diagnosed with NAFLD, aligning with earlier findings that suggest a prevalence of NAFLD ranging from 50% to 75% in individuals with diabetes.[6,7] An essential component of this study involved elucidating the temporal relationship of NAFLD with T2DM. These revealed that in 56.6% of patients presenting with both conditions, NAFLD emerged prior to the onset of diabetes, whereas in 43.4% of cases, diabetes was established before the development of NAFLD. This distinction holds significant importance, as it has not been thoroughly examined in prior studies. The research further indicated that the presence of diabetes alongside NAFLD correlated with a diabetes duration surpassing eight years, while the combination of NAFLD with diabetes was associated with an extended NAFLD duration exceeding ten years. Given that NAFLD and atherosclerosis share common risk factors such as obesity, diabetes, and hypertension, the discrepancies in disease duration could play a significant role in influencing diverse health outcomes.[24] The research provides additional evidence aligning with earlier findings that NAFLD serves as a predictor for cardiovascular events.[24] The build-up of fatty liver results in a rise in the free fatty acids, which in turn initiates low-grade inflammation and the progression of CAD.[25] Furthermore, NAFLD intensifies cardiovascular risk through its role in promoting insulin resistance and atherogenic dyslipidaemia.[18] The increased occurrence of dyslipidaemia and insulin resistance noted in the NAFLD with diabetes cohort highlights their heightened risk for coronary artery disease.[26] The research additionally revealed a greater occurrence of hypertension within the NAFLD group that also had diabetes, in contrast to the other two groups examined. This study is in agreement with earlier investigations that indicate even modest elevations in systolic blood pressure can heighten the risk of NAFLD.[27] Furthermore, managing hypertension in non-obese populations may play a vital role in preventing or mitigating the progression of NAFLD.[28]

Furthermore, research has indicated a correlation of the onset of fatty liver with the progression of hypertension.[29] A variety of mechanisms could elucidate this relationship, one of which is insulin resistance, which aids in disrupting blood pressure regulation among patients with NAFLD.[30] The dysregulation of the renin–angiotensin system in NAFLD may exacerbate hypertension through the promotion of hepatic inflammation and fibrosis.[31] Moreover, the fatty liver index has been proposed as an effective instrument for identifying patients with NAFLD who are at an elevated risk of developing hypertension.[32] The results underscore the importance of conducting diabetes screening among patients with NAFLD to pinpoint individuals who may be at heightened risk for cardiovascular disease and hypertension. Notably, the group with NAFLD and diabetes exhibited a reduced prevalence of diabetic peripheral neuropathy and retinopathy in comparison to the other groups, which may be attributed to their shorter duration of diabetes. A decline

in diabetic nephropathy was noted within this group; however, the variations did not reach statistical significance. Earlier research has indicated a negative correlation between non-alcoholic fatty liver disease (NAFLD) and complications associated with diabetes, including retinopathy, neuropathy, and nephropathy.[33] Similarly, research from Korea found that NAFLD was negatively associated with diabetic retinopathy and nephropathy but not with neuropathy.[34]

Nonetheless, additional research has associated NAFLD with an increased occurrence of chronic kidney disease and severe diabetic retinopathy.[35] The discrepancies observed in the results could be attributed to variations in the study populations, particularly the differences in glycated haemoglobin levels among the groups involved. Since earlier studies did not account for the sequence of the relationship between these two ailments, the discrepancies in the results could partly be explained by this factor. Increasing the sample size in subsequent studies may yield additional confirmation of these findings. This study highlights significant differences in clinical, laboratory, and metabolic characteristics among the three patient groups, with group B and C exhibiting higher BMI, WHR, and lipid levels compared to group A. Notably, group C demonstrated a higher prevalence of cardiovascular diseases and hypertension, while Group B had a longer duration of NAFLD. The findings underscore the need for targeted interventions for metabolic management, especially in patients with prolonged comorbidities such as diabetes and NAFLD. Further investigations are required to examine the long-term impact of these metabolic alterations on the manifestation of diabetic complications.

This study is limited by its cross-sectional design, which restricts the ability to establish causal relationships between NAFLD and T2DM. The relatively small sample size may limit the generalizability of the findings, and the reliance on ultrasound for diagnosing NAFLD instead of histopathological confirmation may introduce diagnostic inaccuracies. Furthermore, potential confounding factors such as physical activity, diet, and medication use were not fully accounted for. Future research with larger, longitudinal cohorts and more precise diagnostic tools is recommended to validate these findings and clarify the temporal and causal associations.

CONCLUSION

This study highlights a complex and variable relationship between non-alcoholic fatty liver disease (NAFLD) and type 2 diabetes mellitus (T2DM), with the sequence of disease onset influencing the prevalence of associated macrovascular and microvascular complications. Patients with NAFLD preceding T2DM exhibited a higher prevalence of coronary artery disease and hypertension, whereas those with longstanding T2DM showed a greater burden of diabetic peripheral neuropathy. The findings suggest that the temporal relationship between NAFLD and T2DM may significantly impact disease progression and complication profiles. To mitigate these risks, early screening and integrated management strategies targeting both hepatic and glycemic health are recommended.

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REFERENCES

1. Adams LA, Angulo P, Lindor KD. Non-alcoholic fatty liver disease. CMAJ. 2005;172:899–905.
2. Hou XH, Zhu YX, Lu HJ, Chen HF, Li Q, Jiang S, et al. Non-alcoholic fatty liver disease's prevalence and impact on alanine aminotransferase associated with metabolic syndrome in the Chinese. J Gastroenterol Hepatol. 2011;26(4):722-30.
3. Fan JG, Saibara T, Chitturi S, Kim BI, Sung JJ, Chutaputti A. Asia-Pacific Working Party for NAFLD. What are the risk factors and settings for non-alcoholic fatty liver disease in Asia-Pacific. J Gastroenterol Hepatol. 2007;22(6):794-800.
4. Amarapurkar DN, Hashimoto E, Lesmana LA, Sollano JD, Chen PJ, Goh KL; Asia-Pacific Working Party on NAFLD. How common is non-alcoholic fatty liver disease in the Asia-Pacific region and are there local differences. J Gastroenterol Hepatol. 2007;22(6):788-93.
5. Fan JG, Farrell GC. Epidemiology of non–alcoholic fatty liver disease in China. J Hepatol. 2009;50:204–10.
6. Akbar DH, Kawther AH. Nonalcoholic fatty liver disease in Saudi type 2 diabetic subjects attending a medical outpatient clinic: Prevalence and general characteristics. Diabetes Care. 2003;26:3351–2.
7. Gupte P, Amarapurkar D, Agal S, Baijal R, Kulshrestha P, Pramanik S, et al. Non-alcoholic steatohepatitis in type 2 diabetes mellitus. J Gastroenterol Hepatol. 2004;19(8):854-8.
8. Leite NC, Salles GF, Araujo AL, Villela-Nogueira CA, Cardoso CR. Prevalence and associated factors of non-alcoholic fatty liver disease in patients with type-2 diabetes mellitus. Liver Int. 2009;29(1):113-9.
9. Younossi ZM, Gramlich T, Matteoni CA, Boparai N, McCullough AJ. Nonalcoholic fatty liver disease in patients with type 2 diabetes. Clin Gastroenterol Hepatol. 2004;2(3):262-5.
10. Targher G, Bertolini L, Padovani R, Rodella S, Tessari R, Zenari L, et al. Prevalence of nonalcoholic fatty liver disease and its association with cardiovascular disease among type 2 diabetic patients. Diabetes Care. 2007;30(5):1212-8.
11. Kim CH, Park JY, Lee KU, Kim JH, Kim HK. Fatty liver is an independent risk factor for the development of Type 2 diabetes in Korean adults. Diabet Med. 2008;25(4):476-81.

12. El Azeem H, Khalek E-S, El-Akabawy H. Association between nonalcoholic fatty liver disease and the incidence of cardiovascular and renal events. J Saudi Heart Assoc. 2013;25:239–46.
13. Sung KC, Wild SH, Kwag HJ, Byrne CD. Fatty liver, insulin resistance, and features of metabolic syndrome: relationships with coronary artery calcium in 10,153 people. Diabetes Care. 2012;35(11):2359-64.
14. Targher G, Bertolini L, Padovani R, Poli F, Scala L, Zenari L, et al. Non-alcoholic fatty liver disease is associated with carotid artery wall thickness in diet-controlled type 2 diabetic patients. J Endocrinol Invest. 2006;29(1):55-60.
15. Targher G, Bertolini L, Padovani R, Rodella S, Zoppini G, Zenari L, et al. Relations between carotid artery wall thickness and liver histology in subjects with nonalcoholic fatty liver disease. Diabetes Care. 2006;29(6):1325-30.
16. Kim HC, Kim DJ, Huh KB. Association between nonalcoholic fatty liver disease and carotid intima-media thickness according to the presence of metabolic syndrome. Atherosclerosis. 2009;204:521-5.
17. Bonapace S, Perseghin G, Molon G, Canali G, Bertolini L, Zoppini G, et al. Nonalcoholic fatty liver disease is associated with left ventricular diastolic dysfunction in patients with type 2 diabetes. Diabetes Care. 2012;35(2):389-95.
18. Targher G, Byrne CD. Clinical Review: Nonalcoholic fatty liver disease: A novel cardiometabolic risk factor for type 2 diabetes and its complications. J Clin Endocrinol Metab. 2013;98:483–95.
19. Targher G, Kendrick J, Smits G, Chonchol M. Relationship between serum gamma-glutamyltransferase and chronic kidney disease in the United States adult population. Findings from the National Health and Nutrition Examination Survey 2001-2006. Nutr Metab Cardiovasc Dis. 2010;20(8):583-90.
20. Speliotes EK, Massaro JM, Hoffmann U, Vasan RS, Meigs JB, Sahani DV, et al. Fatty liver is associated with dyslipidemia and dysglycemia independent of visceral fat: the Framingham Heart Study. Hepatology. 2010;51(6):1979-87.
21. Fraser A, Harris R, Sattar N, Ebrahim S, Davey Smith G, Lawlor DA. Alanine aminotransferase, gamma-glutamyltransferase, and incident diabetes: the British Women's heart and health study and meta-analysis. Diabetes Care. 2009;32(4):741-50.
22. Musso G, Gambino R, Cassader M, Pagano G. Meta-analysis: natural history of non-alcoholic fatty liver disease (NAFLD) and diagnostic accuracy of non-invasive tests for liver disease severity. Ann Med. 2011;43(8):617-49.
23. Xie Y, Wang M, Zhang Y, Zhang S, Tan A, Gao Y, et al. Serum uric acid and non-alcoholic fatty liver disease in non-diabetic Chinese men. PLoS One. 2013;8(7):67152.
24. Hamaguchi M, Kojima T, Takeda N, Nagata C, Takeda J, Sarui H, et al. Nonalcoholic fatty liver disease is a novel predictor of cardiovascular disease. World J Gastroenterol. 2007;13(10):1579-84.
25. Targher G, Bertolini L, Scala L, Zoppini G, Zenari L, Falezza G. Non-alcoholic hepatic steatosis and its relation to increased plasma biomarkers of inflammation and endothelial dysfunction in non-diabetic men. Role of visceral adipose tissue. Diabet Med. 2005;22(10):1354-8.
26. Yan L, Wang S, Fu Y, Wang X, Zheng X, Zheng Y, et al. The impact of non-alcoholic fatty liver disease on islet function in type 2 diabetes. Zhonghua Nei Ke Za Zhi. 2015;54(3):197-200.
27. Wu SJ, Zou H, Zhu GQ, Wang LR, Zhang Q, Shi KQ, et al. Increased levels of systolic blood pressure within the normal range are associated with significantly elevated risks of nonalcoholic fatty liver disease. Medicine (Baltimore). 2015;94(19):842.
28. Aneni EC, Oni ET, Martin SS, Blaha MJ, Agatston AS, Feldman T, et al. Blood pressure is associated with the presence and severity of non-alcoholic fatty liver disease across the spectrum of cardiometabolic risk. J Hypertens. 2015;33(6):1207-14.
29. Sung KC, Wild SH, Byrne CD. Development of new fatty liver, or resolution of existing fatty liver, over five years of follow-up, and risk of incident hypertension. J Hepatol. 2014;60:1040–5.
30. Latea L, Negrea S, Bolboaca S. Primary non-alcoholic fatty liver disease in hypertensive patients. Australas Med J. 2013;6:325–30.
31. Paschos P, Tziomalos K. Nonalcoholic fatty liver disease and the renin-angiotensin system: Implications for treatment. World J Hepatol. 2012;4:327–31.
32. Huh JH, Ahn SV, Koh SB, Choi E, Kim JY, Sung KC, et al. A Prospective Study of Fatty Liver Index and Incident Hypertension: The KoGES-ARIRANG Study. PLoS One. 2015;10(11):143560.
33. Lv WS, Sun RX, Gao YY, Wen JP, Pan RF, Li L, et al. Nonalcoholic fatty liver disease and microvascular complications in type 2 diabetes. World J Gastroenterol. 2013;19(20):3134-42.
34. Kim BY, Jung CH, Mok JO, Kang SK, Kim CH. Prevalences of diabetic retinopathy and nephropathy are lower in Korean type 2 diabetic patients with non-alcoholic fatty liver disease. J Diabetes Investig. 2014;5(2):170-5.
35. Targher G, Bertolini L, Rodella S, Zoppini G, Lippi G, Day C, et al. Non-alcoholic fatty liver disease is independently associated with an increased prevalence of chronic kidney disease and proliferative/laser-treated retinopathy in type 2 diabetic patients. Diabetologia. 2008;51(3):444-50.

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