Metabolic Parameter and Insulin Resistance in Non Alcoholic Fatty Liver Disease Patient Attending Tertiary Care Hospital Coastal Andhra Pradesh

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INTRODUCTION

It has been reported that there are various metabolic abnormalities, obesity and type-2 DM associated with NAFLD in western world but there are very few study on metabolic parameters and insulin resistance associated with NAFLD in India. So this study was designed with an aim to determine the presence of component of metabolic syndrome and insulin resistance in non alcoholic fatty liver disease.

MATERIAL AND METHODS

This was a prospective observational and quantitative study, conducted in the department of general medicine Konaseema Institute of Medical Sciences from Jan 2018 to August 2019.

Selection of the patients: Based on exclusion and inclusion criteria, patients who underwent ultrasonography of abdomen for liver function test abnormality, or any other indication were enrolled for this study.

INTRODUCTION

It has been reported that non alcoholic fatty liver disease is most common liver disease and its prevalence ranges from 20% to 30% in western world.¹ The frequency of this disease varies with ethnicity.² The prevalence of non alcoholic fatty liver disease is also high in Indian sub continent here it ranges from 4% to 29%, which similar to western world.³,⁴ Non alcoholic fatty liver is defined as abnormality of the liver in the absence of alcohol consumption and presence of ≥5% hepatic steatosis in the absence of competing liver disease.⁵ Lonardo A et la in his review has concluded that non alcoholic fatty liver disease is considered to be both consequences and cause of metabolic syndrome. It’s link with metabolic syndrome is more complex.⁶ Pathogenesis of NAFLD is complex it starts with hepatic triglyceride accumulation or steatosis, insulin resistance, proceed to steatohepatitis / inflammation, increased level of hepatic expression of inflammatory cytokines, decreased level of adiponectin, oxidative stress, mitochondrial and endoplasmic reticulum dysfunction, followed by fibrosis, genetic predisposition also play a role. J.K. Dowman et al.⁷ It has been reported that there are various metabolic abnormalities, obesity and type–2 DM associated with NAFLD in western world but there are very few study on metabolic parameters and insulin resistance associated with NAFLD in India.⁸ So this prospective was designed with an aim to determine the presence of component of metabolic syndrome and insulin resistance in non alcoholic fatty liver disease.

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Table 1: Demography of the patients (group A) and control (group B)

| Variables          | Group A (Mean±SD) | Group B (Mean±SD) | P value |
|--------------------|-------------------|-------------------|--------|
| Age                | 45.525±9.041      | 42.675±7.86       | 0.707  |
| Sex                |                   |                   |        |
| M                  | 110               | 108               | .89629 |
| F                  | 40                | 42                |        |
| BMI (kg/m2)        |                   |                   |        |
| Normal (18.5 to 22.9) | 16            | 32                | 0.001032 |
| Over weight (23.0 to 24.9) | 44           | 64                |        |
| Obese (25 and above) | 80             | 54                |        |
| Waist circumference| 94.62±6.421       | 86.37±3.74        | 0.0262 |

Table 2: Metabolic Parameters in both groups

| Variables          | Group A(Mean±SD) | Group B(Mean±SD) | P value |
|--------------------|------------------|------------------|--------|
| FPG (mg/dl)        | 91.42±6.424      | 76.32±4.376      | 0.002  |
| TRPG (mg/dl)       | 126.33±8.426     | 112.73±7.7       | 0.0001 |
| LDL (mg/dl)        | 148.90±30.46     | 116.98±22.32     | 0.001  |
| HDL (mg/dl)        | 36.42±6.376      | 39.47±9.32       | 0.124  |
| Tg (mg/dl)         | 162.89±29.48     | 116.42±20.96     | 0.001  |
| Fasting insulin conc(uIU/ml) | 9.15±1.6 | 5.94±1.364 | 0.0026 |
| HOMA-IR            | 2.18±26          | 1.38±0.44        | 0.0001 |
| AST                | 68.26±18.42      | 28.84±12.42      | 0.001  |
| ALT                | 59.22±14.22      | 24.12±14.62      | 0.001  |
total cholesterol in group A was 218.64±26.34 mg/dl and in group B it was 162.67±24.336 mg/dl. These two value were statistically different as P value of 0.0001. In group A the mean of LDL concentration was 148.90±50.46 mg/dl and in group B it was 116.98±22.32 mg/dl, the P value was 0.001. The mean of HDL concentration was 36.42±6.376 mg/dl in group A and 39.429.32 mg/dl in group B which was not significant as P value was 0.124. Triglyceride concentration was 162.89±29.48 mg/dl in group A and 116.42±2096 mg/dl in group B, the P value was 0.001. The mean of fasting insulin concentration was 9.156±1.6 uIU/ml in group A and 5.94±1.364 uIU/ml in group B. The P value was 0.0026. The mean value of HOMA-IR was 2.18±0.26 in group A and 1.383±0.44 in group B. This difference is statically significant as P value is 0.0001. The mean of AST was 68.26±18.42 unit/litre in group A and 28.84±12.42 unit/litre in group B. The P value was 0.001. In group A the mean of ALT was 59.22±14.22 U/Lt and it group B it was 24.12±14.62 U/Lt, the P value was 0.001.

**DISCUSSION**

In present study we have evaluated the metabolic parameters and insulin resistance in 150 diagnosed cases of non alcoholic fatty liver diseases. In our study we have found that mean age of the patient was 45.525±9.041 yrs, Sangeetha Suresh et al has reported that majority of subject were between 41 to 50 yrs of age which support our study.¹¹ My observation also corroborates with the finding of Kalra S et al.¹² There is male predominance in our study, which is supported by the finding of Perumpail BJ et al.¹³ We have observed in our study that 53.4% patient with NAFLD were obese, 26.6% were overweight, but in control group only 36% were obese and 42% were overweight, so obesity is associated with NAFLD. We have also observed that increased waist circumference is also strongly associated with NAFLD. This finding corroborates with the observation of Perumpail BJ et al, Majumdar A et al and Farrel GL et al.¹⁴,¹⁵

Regarding various metabolic parameters, we have observed that fasting plasma glucose was significantly high in NAFLD patients than control (P=0.00214,0.001). This finding is supported by the work of NovaKovic T et al and Pardhe B.D et al.¹⁶ Total serum cholesterol was significantly higher (218.46±26.426 vs 162.67±24.336) in NAFLD group than the control group (P=0.0001). This finding is supported by the steedy of Agarwal AK et al and D Mahling DV et al.¹⁷ In our study HDL level was low in NAFLD group in comparison to control (36.42±6.376 mg/dl vs 39.97±9.32) which is supported by the work of Agrawal R et al.¹⁸ We have observed that there is hypertriglyceridemia and LDL level was also high. Accumulation of triglyceride in hepatocytes is considered the main pathogenic trigger in the process of pathogenesis of NAFLD, various author has concluded that patients with NAFLD has disrupted lipid profile.¹⁹,²⁰ Result of our study goes along with that.

In our study fasting insulin concentration and HOMA-IR are significantly higher in NAFLD group. The study group has significantly higher fasting insulin concentration than control (9.15±1.6 vs 5.94±1.364) and HOMA IR value was (2.18±0.26 vs 1.38±0.44) also high. So patients with NAFLD have higher FPG, fasting insulin conc and HOMA-IR even though they are not diabetic. This finding corroborates with the finding of Salgado et al.²¹

We have observed that both AST and ALT were significantly higher in NAFLD group than control group. This finding corroborates with the finding of NovaKovic et al.²²

**CONCLUSION**

To conclude most of the patient with NAFLD were obese but some having normal body weight also. Patients with NAFLD have higher FPG, dyslipidemia, fasting insulin concentration and HOMA-IR even though they are not diabetic. So insulin resistance, obesity, lipotoxicity and environmental factor are of major concern in development of non alcoholic fatty liver disease.

**BIBLIOGRAPHY**

1. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M, Global epidemiology of nonalcoholic fatty liver disease—Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology. 2016; 64(2): 73–84.
2. Browning, J. D. et al. Prevalence of hepatic steatosis in an urban population in the United States: impact of ethnicity. Hepatology 2004;40(4):1387–1395.
3. Amarapurkar D, Kamani P, Patel N, Gupte P, Kumar P, Agal S, et al. Prevalence of non-alcoholic fatty liver disease: Population based study. Ann Hepatol. 2007;6(4):161-3.
4. Duseja A. Nonalcoholic fatty liver disease in India - A lot done, yet more required! Indian J Gastroenterol 2010; 29(4):217-25.
5. Zobair M. Younossi, Aaron B. Koenig, Dinan Abdelatif, Yousef Fazel, Linda Henry, and Mark Wymer. Global Epidemiology of Nonalcoholic Fatty Liver Disease—Meta-Analytic Assessment of Prevalence, Incidence, and Outcomes Hepatology 2016;64(1).
6. Lonardo A, Nascimbeni F, Mantovani A, Targher G. Hypertension, diabetes, atherosclerosis and NASH: cause or consequence? J Hepatol. 2018;68(2):335–52.
7. J. K. Dowman, J.W. Tomlinson and P.N. Newsome. Pathogenesis of non-alcoholic fatty liver disease. QJ Med 2010; 103(3):71–83
8. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin L,Flegal KM. Prevalence of overweight and obesity among US,children, adolescents, and adults, 1999-2002. JAMA. 2004;291(2):2847–50.
9. Sample size to estimate a proportion or apparent prevalence with specified precision http://epitools.ausvet.com.au/content.php?page=1Proportion
10. Bannor E, Targher G, Alberiche M, Bonadonna RC, Saggiani F,Zenere MB, et al. Homeostasis model assessment closely mirrors the glucose clamp technique in the assessment of insulin sensitivity: studies in subjects with various degrees of glucose tolerance and insulin sensitivity. Diabetes Care. 2000;23(4):57–63.
11. Suresh S, Rajanbabu B, Veetil VM, Hussain A, Veetil JN. A study on the altered glycemic and lipid parameters and prevalence of insulin resistance in nonalcoholic fatty liver disease. J Family Med Prim Care. 2018;7(1):93–97.
12. Kalra S, Vithalani M, Gulati G, Kulkarni CM, Kadam
Y, Pallivathukkal J, Das B, Sahay R, Modi KD. Study of prevalence of nonalcoholic fatty liver disease (NAFLD) in type 2 diabetes patients in India (SPRINT). J Assoc Physicians India. 2013;61(7):448-53

13. Perumpail BJ, Khan MA, Yoo ER, Cholankeril G, Kim D, Ahmed A. Clinical epidemiology and disease burden of nonalcoholic fatty liver disease. World J Gastroenterol. 2017;23(47):8263–8276.

14. Majumdar A, Misra P, Sharma S, Kant S, Krishnan A, Pandav CS. Prevalence of nonalcoholic fatty liver disease in an adult population in a rural community of Haryana, India. Indian J Public Health 2016;60(4):26–33.

15. Farrell GC. The liver and the waistline: Fifty years of growth. J Gastroenterol Hepatol. 2009;24(3):S105–S118.

16. Novakovic T, Mekic M, Smilic L, et al. Anthropometric and biochemical characteristics of patients with nonalcoholic fatty liver diagnosed by non-invasive diagnostic methods. Med Arch. 2014;68(1):22–26.

17. Pardhe, B.D., Shakya, S., Bhetwal, A. et al. Metabolic syndrome and biochemical changes among non-alcoholic fatty liver disease patients attending a tertiary care hospital of Nepal. BMC Gastroenterol 2018;18(3):109–11.

18. Agarwal A, Jain V, Singla S, Baruah B, Arya V, Yadav R, et al. Prevalence of non-alcoholic fatty liver disease and its correlation with coronary risk factors in patients with type 2 diabetes. J Assoc Physicians India. 2011;59:351–55.

19. Mahaling DU, Basavaraj MM, Bika AJ. Comparison of lipid profile in different grades of non-alcoholic fatty liver disease diagnosed on ultrasound. Asian Pac J Trop Biomed. 2013;3(11):907–12.

20. Agrawal R, Mishra S, Dixit VK, Rai S. Association of non-alcoholic fatty liver disorder with obesity. Indian J Prev Soc Med. 2009;40(6):126–129

21. von Tacer K, Rozman D. Nonalcoholic Fatty Liver Disease: Focus on Lipoprotein and Lipid Deregulation. Journal of Lipids. 2011:14.

22. Ginsberg HN, Zhang YL, Hernandez-Ono A. Regulation of plasma triglycerides in insulin resistance and diabetes. Archives of Medical Research. 2005;36(3):232–240.

23. Lúcia Farias de Azevedo Salgado A, de Carvalho L, Claudia Oliveira A, Nascimento dos Santos V, Gilberto Vieira J, Roberto Parise E. Insulin resistance index (HOMA-IR) in the differentiation of patients with non-alcoholic fatty liver disease and healthy individuals. Arq Gastroenterol. 2010;47(2):165–169.

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