East African Medical Journal Vol. 95 No. 8 August 2018
PREVALENCE OF HEPATIC STEATOSIS AS DIAGNOSED ON UNENHANCED ABDOMINAL COMPUTED TOMOGRAPHY
Naushad Karim, Radiology Department, Aga Khan University, Nairobi, Kenya M.Med, Radiology(AKU - Nairobi), MBChB (UON), P.O. Box 39768, 00623 Nairobi. Rodney Adam, Radiology Department, Aga Khan University, Nairobi, Kenya, Doctor of Medicine (U. of Illinois). Sudhir Vinayak, Radiology Department, Aga Khan University, Nairobi, Kenya M. Med Radiology (UON), M.B.Ch. B (UON).

Corresponding Author: Naushad Karim, Radiology Department, Aga Khan University, Nairobi, Kenya, P.O. Box 39768, 00623 Nairobi. E-mail: naushadkarim@gmail.com.

PREVALENCE OF HEPATIC STEATOSIS AS DIAGNOSED ON UNENHANCED ABDOMINAL COMPUTED TOMOGRAPHY

N. H. Karim, R. Adam, and S. Vinayak

ABSTRACT

Background/Objectives: **The prevalence of non-alcoholic fatty liver disease (NAFLD) has been found to be lower in the African American population when compared to European American or Hispanics, even after controlling for obesity and insulin resistance. The prevalence of hepatic steatosis in the local population is unknown. No studies looking at the association between metabolic syndrome and non-alcoholic fatty liver disease have been done in the local population. The aim of this study is to determine the prevalence of hepatic steatosis in patients undergoing unenhanced abdominal Computed Tomography (CT) at Aga Khan University Hospital, Nairobi**

Subjects/Methods*:* A cross-sectional analytical study of resident indigenous African patients undergoing an unenhanced CT abdomen at Aga Khan University Hospital, Nairobi's (AKUHN) Radiology department. Data from 246 patients who meet the inclusion and exclusion criteria was collected. Metabolic syndrome was diagnosed using the WHO definition.

Results: **Of the 246 patients, 39.3% were female and 60.7% were male. Only 77 patients consented to undergo testing for fasting lipid profile. This limited the number of patients who could be diagnosed with metabolic syndrome. Out of the 246 patients, 33 had hepatic steatosis giving a prevalence of 13.4%, at a P value of 0.05 and a confidence interval of 9.0 to 17.8.Although a large number of people reported occasional/social alcohol intake, only 10 patients had alcohol uptake threshold meeting the criteria used. The causes of hepatic steatosis were mainly attributable to non-alcoholic fatty liver disease; only 2.5% had hepatic steatosis due to alcohol consumption. Obesity was found to be a strong risk factor for hepatic steatosis and patients with elevated BMI were up to 4 times more likely to have hepatic steatosis. Diabetes was also found to be a strong risk factor for**

hepatic steatosis, diabetics were 3 times more likely to have steatosis when compared to non-diabetics.

Conclusions: **The prevalence of hepatic steatosis was 13.4%. There was a strong association of hepatic steatosis and diabetes, with diabetics 3 times more likely to have hepatic steatosis. An association was found between the components of metabolic syndrome and hepatic steatosis.**

INTRODUCTION

Non communicable diseases are becoming an increasing health burden in sub-Saharan Africa with increasing urbanization and adaption of Western lifestyles. It has been estimated that from 1990 to 2020 the burden of non-communicable diseases in Africa will have doubled, a large portion of the victims will be middle aged, in contrast to the Western world where a large proportion are the elderly(1).

Metabolic syndrome represents a constellation of various factors that are related to an increased risk of a cardiovascular event. Data about the prevalence of metabolic syndrome in Kenya is scarce; however, a single study has shown a higher rate in Kenya's urban population compared to the rural population (2). Metabolic syndrome is thought to result from hypoxia of abdominal adipocytes, resulting in a low grade inflammation and resultant development of obesity related co morbidities(3).

Non-alcoholic fatty liver disease is considered to represent another feature of metabolic syndrome. Insulin resistance and hyperinsulinemia play a central role in the pathogenesis of both entities.Non-alcoholic fatty liver disease can range from hepatic steatosis to steatohepatitis, which can progress to cirrhosis. Liver biopsy remains the gold standard for diagnosis of hepatic steatosis. However, it is invasive and may potentially suffer from sampling errors. Hepatic steatosis may be diagnosed on unenhanced CT if the hepatic attenuation is less than 40 HU, or if the attenuation of the liver is at least 10 HU less than the spleen (4)(5).

Over the past three decades non-alcoholic fatty liver disease has emerged as one of the most common causes of liver cirrhosis in the West, and a large proportion of individuals who had previously been classified as having cryptogenic cirrhosis are now believed to have cirrhosis secondary to non-alcoholic steatohepatitis(6).The prevalence of hepatic steatosis in the local population is unknown. No studies looking at the association between metabolic syndrome and non-alcoholic fatty liver disease have been done in the local population.

The aim of this study is to determine the prevalence of hepatic steatosis in patients undergoing unenhanced abdominal Computed Tomography (CT) at Aga Khan University Hospital, Nairobi (AKUHN).

MATERIALS (SUBJECTS) AND METHODS

Area of study: This study was carried out atthe Aga Khan University Hospital, Nairobi which is a private, tertiary teaching and referral hospital in Kenya. AKUHN is home to one of the most sophisticated radiology facilities in East and Central Africa. The Radiology Department provides a comprehensive range of imaging services, including both routine and specialized procedures. It has a cutting-edge picture archiving and communication system (PACS). This system allows digital

images to be viewed immediately upon completion of an examination, which significantly reduces reporting turnaround times. The system also allows any facility in East Africa to easily and confidentially send AKUHN an image for a second opinion.

Study population: The study population consisted of patients undergoing an unenhanced abdominal CT at AKUHN Radiology department who fulfilled the eligibility criteria.

Study design: The study was across sectional analytical study.

Inclusion criteria: These included resident indigenous African patients who are to undergo an unenhanced abdominal CT (for suspected urolithiasis; unrelated to exposure and outcome of interest in this study) at AKUHN and who are above the age of 18 years.

Ethical considerations: Approval to perform the study was granted from the Dissertations Committee of Aga Khan University. An informed consent was sought from all patients who participate in the study.

Data collection: There was consecutive recruitment of patients (24 hours). Patient requiring unenhanced abdominal CT were presented to the CT scanning area. Radiologist on duty explained the details of the study and obtained consent. Questionnaires were filled and weight and height obtained. If the patient required any clarification, the primary investigator was contacted. The patient was given a form for fasting lipid profile and blood glucose to be done the following morning. If patient was an inpatient, then samples were collected in the morning before breakfast. Blood pressure was obtained from the referring department. These data enabled the diagnosis of metabolic syndrome using the WHO criteria to be made. Patients CT scan was examined by the principal investigator with 3 years' experience and a consultant with 11 years' experience.

Data analysis: Statistical analysis was carried out using STATA version 11.2. The accepted level of significance was 95% confidence intervals and exact P value for effects.

RESULTS

Of the 246 patients, 39.3% were female and 60.7% were male. Only 77 patients consented to undergo testing for fasting lipid profile. This limited the number of patients who could be diagnosed with metabolic syndrome. The general characteristics of the study population are given in Table 1. Out of the 246 patients, 33 had hepatic steatosis giving a prevalence of 13.4%, at a P value of 0.05 and a confidence interval of 9.0 to 17.8. The prevalence of hepatic steatosis was 15.5% in males and 10.6% in females (Table 2). Diabetes was found to be a strong risk factor hepatic steatosis, diabetics were 3 times more likely to have steatosis when compared to non-diabetics (Pearson chi test = 8.8 and P value = 0.003). There was no significant association between alcohol intake with hepatic steatosis (Pearson Chi test = 1.01 and P value = 0.315) (Table 3). Although a large number of people reported occasional/social alcohol intake, only 10 patients had alcohol uptake threshold meeting the criteria used in the study. Obesity was also found to be a strong risk factor for hepatic steatosis and patients with elevated BMI were up to 4 times more likely to have hepatic steatosis (Odds Ratio = 4.38 and P value = 0.0001).The causes of hepatic steatosis were mainly attributable to non-alcoholic fatty liver disease; only 2.5% had hepatic steatosis due to alcohol consumption (Table 4).

Table 1

General Characteristics

	Male	Female	All
Average age (*n=246*)			47.8 years
Age group – 18 – 40 years	41.4 %	37.2 %	39.8 %
- 41 – 60 years	35.2 %	33 %	34.3 %
- > 60 years	23.4 %	29.8 %	25.9 %
BMI			
-Normal	110 (73.8 %)	69 (71.3 %)	179 (72.8 %)
-Overweight	35 (23.4 %)	26 (26.6 %)	61 (24.7 %)
-Obese	4 (2.8 %)	2 (2.1%)	6 (2.5 %)
Blood pressure >140/90 mm Hg	21 (14 %)	14 (14.4 %)	35 (14.3 %)
Medication causing steatosis	0	1 (1 %)	1 (0.4 %)
Alcohol	7 (4.7 %)	3 (3.1 %)	10 (4.2 %)
Diabetic/abnormal glucose	24 (16 %)	16 (16.5 %)	40 (16.3 %)
Abnormal lipid profile (total n=77; abnormal *n=21*)	16 (37.2%)	5 (14.7 %)	21 (27.3 %)
Metabolic syndrome (*n=77*)	4 (2.7 %)	3 (3.1 %)	7 (2.8 %)

Table 2

Prevalence of Hepatic Steatosis in the Two Sexes

Hepatic Steatosis	**Female**	**Male**	**Total**
No	87 (89.4%)	126 (84.5%)	213 (86.6%)
Yes	10 (10.6%)	23 (15.5%)	33 (13.4%)
Total	**97 (100%)**	**149 (100%)**	**246 (100%)**

Table 3

Relationship of Alcohol Intake and Diabetes with Hepatic Steatosis

	Hepatic Steatosis		**Total**
	No	**Yes**	
Alcohol			
No	204 (95.7%)	32 (97%)	236 (95.8%)
Yes	9 (4.3%)	1 (3%)	10 (4.2%)
Diabetic/abnormal sugar			
No	184 (89.3 %)	22 (10.7 %)	206 (100%)
Yes	29 (72.5 %)	11 (27.5 %)	40 (100%)

Table 4

Causes of Hepatic Steatosis

Causes of Hepatic Steatosis	**Frequency**	**Percentage**
Non-Alcoholic Fatty Liver Disease(NAFLD)	32	97.5 %
Alcoholic fatty liver	1	2.5 %
Medication	0	0%

DISCUSSION

This is the first study looking at prevalence of hepatic steatosis in sub-Saharan Africa. The prevalence of hepatic steatosis in the study population of 13.4% (CI 9.0 – 17.8) contrasts with that found in a study in urban African Americans, which showed a prevalence of 24% (confidence interval 21-26) (7). Although the current study was not designed to assess the lifestyles of the participants, the difference in prevalence when compared to urban African Americans may be due to different lifestyles, such as diet and physical activity. Another possible reason for the difference in prevalence may be the higher prevalence of elevated BMI and diabetes in the American population. The prevalence in the current study population is similar to the prevalence of hepatic steatosis in China (Prevalence 16.73%, CI 13.9 – 19.5) (8). This may be due to similarities in lifestyle and prevalence of elevated BMI.

The prevalence of hepatic steatosis in the current study was 15.5% in males and 10.6% in females. There was no difference in the prevalence of hepatic steatosis in the two sexes in urban African Americans; however there was a marked difference in the prevalence of hepatic steatosis in Caucasians with 42% prevalence in urban Caucasian males and 24% in urban Caucasian females (7). The higher prevalence in Caucasian males was attributed to higher alcohol intake. A higher prevalence was also found in Chinese males; however, no cause was attributed to this higher rate of prevalence(8). The current study shows a trend towards a higher prevalence in the male sex despite similar rates of diabetes and elevated BMI in the 2 sexes; however, it was not powered to assess the significance of the difference in prevalence in the 2 sexes or to explore the causes of differences in prevalence in the two sexes.

The causes of hepatic steatosis in this study population were mainly attributable to non-alcoholic fatty liver disease; only 2.5% had hepatic steatosis due to alcohol consumption. This is in contrast to the 16.4% prevalence of hepatic steatosis attributed to alcohol consumption in urban North America (7). The prevalence of hepatic steatosis attributable to alcohol consumption in China is 4.5%, this is similar to this study(9).

In the current study, obesity was found to be a strong risk factor for hepatic steatosis and patients with elevated BMI were up to 4 times more likely to have hepatic steatosis. 28.4% of patients of patients in the current study with elevated BMI had hepatic steatosis in contrast to the 80% prevalence in patients with elevated BMI in Japan (10). In the current study, 56% of patients with hepatic steatosis had elevated BMI. This is similar to the study done in urban North America, where two thirds of the patients with hepatic steatosis had elevated BMI(7). Obesity has also been shown to be a significant risk factor for the development of steatosis in the Chinese population, with the rise in prevalence in hepatic steatosis over the last 10 years attributed to the increased prevalence of elevated BMI (11).

Diabetes was also found to be a strong risk factor hepatic steatosis in this study. Diabetics were 3 times more likely to have steatosis when compared to non-diabetics. The prevalence of hepatic steatosis in diabetics in this study was 27.5%. This is much lower than the 62% prevalence of hepatic steatosis in diabetic in urban North America(7). An association between hepatic steatosis and diabetes was also found in the Chinese population, with a prevalence of 51.7% in

patients with elevated HbA1c compared to 25% in patients with a normal HbA1c (12).

In this study 23.8% of patients with dyslipidaemia had hepatic steatosis and although there was a trend towards an association between hepatic steatosis and dyslipidaemia with an odds ratio of 2.8 (P=0.09), this study was not powered to assess this. In the study done in urban North America, 61.5% of patients with dyslipidaemia had hepatic steatosis(7).

The number of patients who could be diagnosed with metabolic syndrome was limited by the low number of patients who consented to undergo testing for fasting lipid profile. Hence, the association between metabolic syndrome and hepatic steatosis could not be determined. However, the results from this study show an association between hepatic steatosis and components of the metabolic syndrome as given in table 11 above. In the study in urban North America, 78.9% of patients with metabolic syndrome had hepatic steatosis. In a study done in Taiwan, 20% of patients with mild steatosis had metabolic syndrome and 51.4% of patients with moderate to severe steatosis had metabolic syndrome(13).

This study gives an indication of the prevalence of hepatic steatosis in a private urban tertiary health care facility in Sub Saharan Africa although with the caution that it may not be representative of the rest of the population due to the selection bias of the more affluent members of society who frequent this hospital and whose lifestyle mirrors Western lifestyle and dietary habits.

REFERENCES

1. Jamison D. Disease and mortality in sub-Saharan Africa. World Bank Publications; 2006.

2. Christensen D, Friis H, Mwaniki D, Kilonzo B, Tetens I, Boit M. Prevalence of glucose intolerance and associated risk factors in rural and urban populations of different ethnic groups in Kenya. 2009;84(3): Diabetes Res Clin Pract. 2009;84(3):303–10.

3. Fezeu L, Balkau B, Kengne AP, Sobngwi E, Mbanya JC. Metabolic syndrome in a sub-Saharan African setting: central obesity may be the key determinant. Atherosclerosis. 2007;193(1):70–6.

4. Limanond P, Raman SS, Lassman C, Sayre J, Ghobrial RM, Busuttil RW, et al. Macrovesicular Hepatic Steatosis in Living Related Liver Donors: Correlation between CT and Histologic Findings. Radiology. 2004;230:276–280.

5. Joy V, Thava R, Scott BB. Diagnosis of fatty liver disease: is biopsy necessary? Eur J Gastroenterol Hepatol. 2003;15(5):539-543.

6. Dowman J, Tomlinson J, Newsome P. Pathogenesis of non-alcoholic fatty liver disease. Qjm. 2010;103(2):71–83.

7. Browning JD, Szczepaniak LS, Dobbins R, Nuremberg P, Horton JD, Cohen JC, et al. Prevalence of hepatic steatosis in an urban population in the United States: Impact of ethnicity. Hepatology. 2004;40(6):1387–95.

8. Zhu JZ, Zhou QY, Wang YM, Dai YN, Zhu J, Yu CH, et al. Prevalence of fatty liver disease and the economy in China: A systematic review. World J Gastroenterol. 2015;21(18):5695–706.

9. Fan JG. Epidemiology of alcoholic and nonalcoholic fatty liver disease in China. J Gastroenterol Hepatol. 2013;28(SUPPL 1):11–7.

10. Amarapurkar DN, Hashimoto E, Lesmana LA, Sollano JD, Chen PJ, Goh KL. 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.

11. Jiang Y, Xu Y, Bi Y, Wang L, Zhang M, Zhou M, et al. Prevalence and trends in overweight and obesity among Chinese adults in 2004–10: data from three nationwide surveys in China. Lancet [Internet]. 2015;386:S77. Available from: http://dx.doi.org/10.1016/S0140-6736(15)00658-3

12. Ma H, Xu C, Xu L, Yu C, Miao M, Li Y. Independent association of HbA1c and nonalcoholic fatty liver disease in an elderly Chinese population. BMC Gastroenterol. 2013;13(1):1–6.

13. Yang KC, Hung HF, Lu CW, Chang HH, Lee LT, Huang KC. Association of Non-alcoholic Fatty Liver Disease with Metabolic Syndrome Independently of Central Obesity and Insulin Resistance. Sci Rep [Internet]. 2016;6(March):1–10. Available from: http://dx.doi.org/10.1038/srep27034