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The effect of type 2 diabetes mellitus on fasting gallbladder volume

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ABSTRACT: One hundred type 2 diabetic patients and 100 age and sex matched controls underwent real time ultrasonography to determine the effect of type 2 diabetes mellitus on fasting gallbladder volume. The mean gallbladder volume of diabetic patients was 27.6 ± 15.4 ml compared with 24.3 ± 12.8 ml in non-diabetic controls ($P > 0.05$). It is suggested that type 2 diabetes mellitus could be a risk factor for increased fasting gallbladder volume in Nigerians.

Key Words: Type 2 diabetes mellitus; Gallbladder volume; Ultrasonography.

Introduction

The pathogenic mechanism(s) by which GS forms is generally agreed to be due to (i) alteration in the composition of bile, (ii) stasis and (iii) infection (1,2). The risk factors for cholesterol GS include increasing age, female gender, multiparity, obesity, rapid weight loss, high animal fat diet, drugs such as contraceptive pills and ileal disease or resection. Others are liver cirrhosis, haemoglobinopathy and diabetes mellitus (3).

Gallstone disease (GSD) in patients with diabetes mellitus is largely due to dyslipidaemia, leading to the alteration in the composition of bile. Impairment of gallbladder motility and contraction, as a result of hyperglycaemia and diabetic autonomic neuropathy, also cause bile stasis and promote cholesterol gallstone crystal formation (4). Studies have also shown that type 2 diabetes mellitus is an independent predictor for increased gallbladder volume.

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Diabetes mellitus (DM) is one of the risk factors for cholesterol gallstones. Individuals with diabetes mellitus are reported to have 2- to 3-fold increase in the incidence of cholesterol gallstones (4). Studies have shown a higher prevalence of GSD in patients with DM (5 – 8) and that DM is an independent predictor for increased gallbladder volume. They have also shown that fasting gallbladder volumes are significantly increased in patients with multiple gallstones, and that diabetic patients with autonomic neuropathy show a decreased gallbladder emptying rate (4,5).

The present study is, therefore, to determine the effect of type 2 diabetes mellitus on fasting gall bladder volume.

Materials and Methods

The study was a prospective one. The setting of the study was the Medical Out-Patient Department (MOPD) of the University of Ilorin Teaching Hospital (UITH), Ilorin, Nigeria. Approval for the study was obtained from the Research and Ethical Committee of the UITH. Verbal and informed consent was obtained from the participants.

One hundred type 2 diabetic patients and 100 age- and sex-matched controls undwerwent real time ultrasonography (USS) using real time ultrasound scanner from Sony Incorporated, Japan to determine the presence of GSD and to measure the fasting gallbladder volume.

All consenting patients with confirmed diagnosis of DM [(by the WHO criteria of 1999: fasting plasma glucose concentration equal to or greater than 7.0 mmol/L (126 mg/dL), 2hr postprandial glucose equal to or greater than 11.1 mmol/L (200 mg/dL)] attending the DM Clinic of the MOPD were recruited into the study. Patients labelled as having type 2 DM were those whose age at onset of the disease was equal to or greater than 40 years, those who did not require insulin for survival or those who were not ketosis prone. Controls were recruited from normal hospital health workers, patients with minor ailments such as malaria or upper respiratory tract infection without DM.

Only patients with haemoglobin genotype Hb AA were recruited into the study. Both the study group and the controls were matched for age and sex. The examinations were done in the morning following an overnight fast (to prevent gallbladder contraction) without sedation. Fasting gallbladder volumes were determined.

Longitudinal and transverse scans of the right upper quadrant (RUQ) was done in both the supine and left lateral positions. Patients with known risk factors for GSD, such as haemolytic disorders like sickle cell disease and pregnancy, were excluded from the study. Patients who have had cholecystectomy were also excluded. Blood glucose was determined using 5 ml of blood collected in fluoride oxalate bottles. The blood samples were centrifuged and plasma separated. Trinder's analytical method was used for glucose estimation (9).

Statistical Analysis

The data obtained were analysed with Epi-Info statistical software, version 6.1.

Results

At the conclusion of the study, one hundred patients each for the diabetic group and controls completed the study. They were all native Nigerians and all had HbAA genotype. Their ages ranged between 25 and 78 years with a mean of 52.9 ± 10.7 years for the diabetic group and 25 – 75 years with a mean of 52.9 ± 10.7 years for the controls. Both groups were similar in age ($P > 0.05$) (Table 1).. The Body Mass Index (BMI) ranged from 15.6 kg/m^2 to 43.1 kg/m^2 with a mean of 26.1 ± 5.7 kg/m^2 for the experimental group and 14.7 kg/m^2 to 34.5 kg/m^2 with a mean of 23.5 ± 5.4 kg/m^2 for the controls. The mean BMI for the experimental subjects was slightly above the normal range (i.e. pre-obese) while the mean BMI for the control group was within the normal range (18.5 – 24.9 kg/m^2). The diabetic patients had a significantly higher mean BMI than the controls ($P < 0.01$)

The Waist Hip Ratio (WHR) ranged from 0.85 to 1.24 with a mean of 0.97 ± 0.08 for the experimental group and 0.81 to 1.19 with a mean of 0.95 ± 0.07 for the controls. The difference between the two groups was not statistically significant (Table 1).

Table 1: Demographic and anthropometric data of the study subjects (n = 100).

Variables	Range		Mean ± SD		P-value
	DM	Controls	DM	Controls	
Age (Years)	25 – 78	20 – 75	52.9 ± 10.7	49.0 ± 12.5	0.062 (NS)
BMI (kg/m^2)	15.6 – 43.1	14.7 – 34.5	26.1 ± 5.7	23.5 ± 5.4	0.0065 (S)
WHR	0.85 – 1.24	0.81 – 1.19	0.97 ± 0.08	0.95 ± 0.07	0.208 (NS)

S = Significant; NS = Not significant.

Table 2: Age distribution of patients with gallstones.

Age Group	DM Subjects			Controls		
(Years)	N	GS	% GS	N	GS	% GS
20 – 29	1	0	0	1	0	0
30 – 39	12	1	6.7	12	1	14.3
40 – 49	26	2	13.3	26	2	28.6
50 – 59	28	3	20.0	28	2	28.6
60 – 69	25	6	40.0	25	1	14.3
70 -79	8	3	20.0	8	1	14.3
Total	100	15	100	100	7	100

N = Number of patients; GS = Number of patients with gallstones; % GS = Percentage of patients with gallstones.

Table 3: Relationship between gallstones and gallbladder volume.

	DM Patient	Control	P-value
Gallbladder volume	27.6 ± 15.4	24.3 ± 12.8	0.189 (NS)

Seventy-nine (79%) of the patients fell within the age group 40 – 69 years. Eleven of the diabetic patients with GS (73.3%) were in the age range 40 – 69 years with six (40%) of them in the age range 60 – 69 years, i.e. seventh decade of life. There was a steady increase in the incidence of GS in diabetic patients with age, with a peak incidence in the seventh decade, i.e. 60 – 69 years, and a decline in the eighth decade, i.e. 70 – 79 years.

Four patients (57.1%) in the control group with GS were in the age group 40 – 59 years. The peak incidence (57.1%) was also in the age group 40 – 59 years, i.e. fifth and sixth decades, with a steady decline towards the eighth decade, i.e. 70 – 79 years (Table 2).

Sex distribution of patients with gallstones

Fifty (50%) were males for the diabetic group and controls and fifty (50%) were females. In the DM group, seven of the patients with GS (46.7%) were males while eight (53.3%) were females, a male to female ratio of 1:1.14. The difference is, however, not statistically significant (P = 0.198).

Prevalence of cholelithiasis in the study population

Fifteen diabetic patients had GS while 7 control subjects had GS. This gives a prevalence rate of 15% in the diabetic patients and 7% in the controls.

Discussion

From the study, it was found that the mean gallbladder volume in diabetic patients was 27.6 ± 15.4 ml compared with that in non-diabetic controls 24.3 ± 12.8 ml. However, the difference was not statistically significant (P > 0.05). These findings are in agreement with those of Chapma *et al*. (5) and Bucceri *et al*. (10) who compared the gallbladder volume in diabetic patients and non-diabetic controls. They found that the variations in gallbladder volumes between diabetic patients and non-diabetic controls were influenced by the presence of GS. Chapma *et al*. (5) also demonstrated that non-insulin dependent diabetes mellitus (NIDDM) is an independent predictor for increased gallbladder volume.

Hahms *et al*. (4) also found that gallbladder volume in diabetic patients was significantly greater compared with that of controls. Pazzi *et al*. (11) in a review of gallbladder motor function in DM proposed that the mechanism of gallbladder emptying abnormalities in diabetes mellitus may represent a manifestation of denervation caused by visceral neuropathy, a decreased sensitivity of the smooth muscle of the gallbladder to plasma cholecystokinin and/or decreased cholecystokinin receptors on the gallbladder wall. Hahms *et al*. (4) suggested that impairment of gallbladder motility complicated by autonomic neuropathy causes stasis and results in cholesterol GS crystal formation and GS growth.

An extensive review of the literature has shown that similar work has not been done on an African population. The data presented here, therefore, serve to provide baseline information for subsequent studies. Our conclusion is that type 2 diabetes is a likely risk factor for increased fasting gallbladder volume in Nigerans.

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