Med. J. Cairo Univ., Vol. 62, No.1 March (Suppl.): 227-233, 1994

Gall Bladder Volume and Function in Obese Individuals

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Abstract

This study was carried out on 50 obese and 50 average weight females with normal laboratory investigations. Gall bladder volumes were measured in fasting state and 1 1/2 hour after a liquid fatty meal by ultrasonography. The incidence of gall stones in the obese group was 28% compared to 2% in non-obese group. The fasting gall bladder volume in the obese group was (32.7 ± 8.9) which was significantly higher than non-obese group (16.1 ± 2.7) and the postprandial volume was also higher in the obese (14. 6 ± 5.02) than non-obese group (2.9 ± 1.07). The cases with morbid obesity had significantly larger fasting volume than the rest of obese females (39.3 ± 12.3 versus 27.7 ± 4.9) and also larger postprandial volume (25.7 ± 6.7 versus 11.7 ± 4.03). The indicence of gall stones in the morbidly obese was 40% compared to 14.3% in the rest of obese females. The volumes of gall bladder were also significantly higher in android obesity group comparted to gynoid obesity, with a higher incidence of gall stones in the android group. It is concluded that gall bladder contractility is impaired in obese females especially in morbid obesity and android obesity.

Introduction

THE association between obesity and gallbladder disease has been documented in several studies. Increased cholesterol production and secretion provides an explanation for the increased risk of gallbladder disease [1]. 50% of markedly obese patients have gallstones detected at surgery due to excessive biliary secretion of cholesterol, however, they have normal bile salt secretion [2].

High choiesterol diet and diet rich in polyunsaturated fats leads to excessive biliary cholesterol secretion [3].

Hyperlipidaemia and low plasma high density lipoprotein cholesterol had been reported to be associated with increased risk of gallstones [4]. Beside disturbed lipid pattern, defective gallbladder function may lead to gallstone formation [5].

The risk of gallstone formation is known to decrease if the obese loses weight, although at the period of active weight reduction the cholesterol saturation may actually increase [6].

Since the gallbladder itself is the site of stone formation, it is suggested that impaired gallbladder motor function may be a primary phenomenon antedating and even predisposing to gallstone formation [7].

Beside its storing and concentrating functions the gallbladder acts as a motor driving the enterohepatic circulation of bile acids. Any disturbance of the driving capacity of this motor may result in a decrease of the number of enterohepatic circulations, another factor incriminated in gallstone formation [8].

This study is undertaken to determine fasting and postprandial gallbladder volume and gallbladder motility in obese normal people and non-obese control individuals, to detect the role of impaired gallbladder emptying in gallstone formation in obese patients.

Material and Methods

Fifty obese and fifty average weight normal females were the subject of this study. Both groups were of comparable age.

The mean body mass index (BMI) of the obese females was 38.2 +/- 4.3 Kg/ m^2. The normal BMI is 20 - 25 Kg/m^2.

$$\text{The BMI} = \frac{\text{weight in Kg}}{\text{square height in meter}}$$

19 females had morbid obesity. Morbidly obese subjects had BMI above 40 Kg/m^2 [9]. As the prevalence of gallbladder disease in women is twice than men, we restricted our study to women.

None of the obese and non-obese groups were diabetic, taking medications known to alter smooth muscle contractility or taking oral contraceptive pills, pregnancy was also excluded.

The Waist/Hip (W/H) circumferences for obese females were measured and they are classified into 2 groups according to W/H ratio. Android obesity had a ratio above 1 and gynoid obesity had a ratio below 1.

After an overnight fast, the gallbladder volume was calculated from the real time ultrasonographic dimensions of the gallbladder. The linear transducer was placed on the right upper quadrant and rotated until the largest dimensions of the gallbladder were obtained on the screen, then the image was frozen and the volume of gallbladder was measured by computerized electronic calipers using the ellipsoid model volume = (L x W x H).

Where L is the maximum longitudinal dimension, W is the width and H is the height of the gallbladder [17].

Postprandial gallbladder volume after a liquid fatty meal was calculated as follows: Postprandial volume = (% remaining in gallbladder at 90 min.) x (fasting volume).

Results

The prevalence of gallstones in the obese group was 28% compared to 2% in the non-obese group. The fasting and postprandial gallbladder volume in the obese group were significantly higher than non-obese group (Table 1).

Females with morbid obesity (B. M. I. above 40) had significantly larger fasting and postprandial gallbladder volume than the rest of obese females (able 2).

Of the 14 obese women with gallstones 10 were asymptomatic (71.4%) and 25% had a family history of gallstones.

The fasting and postprandial gallbladder volumes in android obesity group were significantly higher than the gynoid group, and the incidence of gallstones was also higher in the android obesity group (table 3).

Table (1):Statistical Comparison between Normal & Obese Groups

	Normal (Mean ± S.D)	Obese (Mean ± S.D)	"*p*" Value	Significane
Number	50	50		
Age	35.2 ± 6.79	43.32 ± 11.23	non - sign.	
Height (m)	1.65 ± 0.08	1.60 ± 0.098	non - sign.	
Weight (kg)	64.0 ± 7.03	97.84 ± 12.91	non - sign.	
BMI	23.44 ± 0.99	38.22 ± 4.30	< 0.01	Sign.
F.V (ml)	16.14 ± 2.70	32.69 ± 8.95	< 0.01	Sign.
$1\frac{1}{2}$ hpp.v.	2.39 ± 1.07	14.63 ± 5.02	< 0.01	Sign.
Gallstone	2%	28%		

* F.V. : Fasting GB volume (ml)

*$1\frac{1}{2}$ hpp.v. :- $1\frac{1}{2}$ hour postprandial volume (ml)

Table (2): Statistical Comparison between Morbidly Obese Females and the Rest of Obese Group.

	Obese (Mean ± S.D)	Morbidly Obese (Mean ± S.D)	"p" Value	Significance
Numbers	31	19		
BMI	35.5 ± 2.3	42.6 ± 3.1	< 0.01	Sign.
F.V. (ml)	27.7 ± 4.9	39.3 ± 12.3	< 0.01	Sign.
P.P. volume (ml)	11.7 ± 4.03	25.7 ± 7.6	< 0.01	Sign.
Gallstone	19.3 %	40%		

Table (3) : Statistical Comparison between Android & Gyoid Groups.

	Android Obeseity (Mean ± S.D)	Gynoid Obesity (Mean ± S.D)	"p" Value	Significane
Numbers	25	25		
Age	42.96 ± 12.59	43.68 ± 9.94		non -Sign.
Height (m)	1.61 ± 0.097	1.59 ± 0.099		non -Sign.
Weight (kg)	101:72 ± 14.33	93.96 ± 10.18		non -Sign.
BMI	38.94 ± 4.2	37.44 ± 4.32	> 0.05	
Waist / Hip Ratio	1.13 ± 0.09	0.89 ± 0.05		
F.V (ml)	35.36 ± 9.46	30.20 ± 7.70	< 0.05	Non Sign.
	16.28 ± 4.9	12.99 ± 4.68	< 0.05	Sign.
Gallstone	36 %	20%		

Discussion

Obesity is a major risk factor for gall bladder disease. The reported incidence of biliary tract disease in morbidly obese varies from 28 to 45.2% [2]. This incidence is 3 to 4 times greater than that reported in general population. The incidence of biliary disease in non-obese women who are less than 50 years old is 6 to 16.7%, but in obese women who are less than 50 years old the incidence is 31 to 39%. In contrast, obese men and women greater

than 50 years of age do not exhibit an increased risk of biliary disease when compared with nonobese control subjects, [10] which reflects that, in addition to obesity, hormonal and reproductive factors are important in the development of gall bladder disease.

The high incidence of cholesterol gall stones in morbid obesity has been attributed to abnormalities in bile composition [11]. Increased intake of energy or fat was associated with increased biliary cholesterol concentration over time and increased risk of gallstones among subjects below 50 years [10].

Pixley et al. [3], found a 1.9 fold increase in risk of developing gallstones in non-vegetarians compared with vegetarians, suggesting a protective effect of high intake of developing gallstones in non-vegetarians compared with vegetarians, suggesting a protective effect of high intake of fibre may be important in the aetiology of gallstones. Bran lowers the bile saturation index [12]. A diet high in sugar (sucrose) was associated with increased cholesterol saturation of bile and increased risk of cholesterol gallstones [13]. Increased intake of sucrose may increase the of gallstones formation by stimulating insulin secretion. Insulin increases gallstone formation by increasing the cholesterol and triglyceride synthesis by the liver [14].

In addition to abnormalities of bile composition a contributing factor for gallstone formation, abnormal gallbladder motility increases the risk of gallstone. Impaired gallbladder emptying and/or increased gall bladder volume occurs in diabetes, pregnancy, truncal vagotomy, and prolonged total parenteral nutrition [15], all these conditions have increased incidence of gallstones.

Gallbladder emptying studies have been suggested as a means of diagnosing functional gallbladder disease (biliary dyskinesia) and chronic cholecystitis [16]. Radionuclide determination of fractional gallbladder emptying and ultrasonographic gallbladder volume changes in response to a meal have been preformed by many investigators in average sized persons [17, 18].

We have reported increased fasting gallbladder volume and incomplete emptying in obese females, especially in morbid and upper body obesity. The incidence of gallstones was also higher in morbidly obese and android obesity group compared to the rest of obese group.

The sensitivity of detecting gallstones by ultrasonography is less than the sensitivity reported in general population (963.6% versus 92%) because of poor quality of ultrasonograms obtained through large adipose mass [2].

The etiology of the incomplete gallbladder emptying in morbidly obese remains unclear. Small cholesterol gallstones missed on ultrasonography may have secondarily altered gallbladder emptying [9].

The incomplete emptying in obesity may be secondary to cholecystokinin resistance, which is the hormone related to gallbladder contraction [9].

Rapid weight loss results in an increased incidence of gallstones [19]. Some of our obese females were under regimen with low calorie diet and this may lead to bile stasis.

Android obesity is associated with increased peripheral insulin resistance, hyperinsulinemia, glucose intolerance, hypertriglyceridemia and hypertension [20]. In addition, in this study, the gallbladder emptying was more reduced in android obesity than gynoid type, with higher incidence of gallstones. Hyperinsulinemia may be the contributing factor, as insulin is relevant to gallstone disease [14].

The finding that more than 70% of women with gallstones in this study were asymptomatic favours conservative management of gallstones in such subjects an aspect of controversy in the management of gall stones.

In conclusion, altered gall bladder motility occurs in obese females, which may play a role in the pothogenesis of gall stones among them.

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