ORIGINAL ARTICLE

Evaluation of GIT Mucosa, Hepatic Parenchyma and Blood Biochemistry in Obesity

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Abstract

Background: ***Obesity has a negative effect on health. By 2035, it is anticipated that over 51% of the population will be affected by obesity. Obesity is a leading cause of various gastrointestinal and hepatic diseases.***

Objectives: ***To evaluate the association of various grades of obesity with endoscopic changes in the gastro-colonic mucosa, hepatic fibrocirrhotic changes, and blood chemistry abnormalities.***

Methods: ***A case-control study included 72 subjects with different grades of body mass index (BMI) recruited from an outpatient clinic. Subjects were categorized into obese (BMI>30) (n=36) and non-obese (BMI<30) (n=36). Non-obese subjects were categorized into normal BMI (18-24.9kg/m2) (group I) and overweight (BMI 25-29.9kg/m2) (group II). Obese subjects were categorized into obese (BMI 30-34.9kg/m2) (group III) and morbidly obese (BMI ≥ 35kg/m2) (group IV). Blood chemistry was assessed. Ultrasonography and Fibroscan were done for fibrocirrhotic liver changes. Gastro-colonic endoscopic screening was performed to detect GIT mucosal changes.***

Results: ***Obese subjects with BMI ≥30kg/m2 had fibrocirrhotic changes in liver parenchyma and higher values of routine blood tests (p-value < 0.05). Absence of hepatic fibro-cirrhotic changes (F0) was seen more often in group I (88.2%) and group II (63.2%) in comparison to other groups (p value <0.0001). Moderate hepatic fibro-cirrhosis (F7.1 to F10) was recognized in group III (40%) compared to the rest of the study populations. Gastro-colonic endoscopic screening did not reveal any characteristic macroscopic lesion that indicates a specific GIT disease.***

Conclusion: ***Obesity is associated with hepatic fibrocirrhotic changes, high baseline of routine blood test results and undetectable gastro-colonic macroscopic mucosal lesions.***

Keywords: BMI; Liver fibrosis; GIT endoscopy; Obesity

1. Introduction

Nonalcoholic hepatic steatosis (NAHS) is the prevalent liver disease in children and teenagers that has advanced courses and may be an end-stage organ failure. It includes simple liver steatosis, nonalcoholic steatohepatitis, and liver cirrhosis .[1] Elevated body mass index and abnormal lipid levels are recognized as the leading causes of NAHS.[2]

In recent years, NAHS occurrence has risen, becoming a significant health problem that affects more than a quarter of the global population 3. Obesity is linked to numerous diseases and conditions, especially diabetes mellitus, restricted airflow during sleep, cardiovascular diseases, a specific type of malignancy, and joint diseases.[4]

Accepted 06 February 2025.
Available online 28 February 2025

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https://doi.10.21608/aimj.2025.446429

There are increased risks of several gastrointestinal disorders in obesity, such as esophageal adenocarcinoma, erosive esophagitis, gastro esophageal reflux disease, squamous metaplasia of esophagus, gastritis, gastric malignancy, colonic diverticulae, colonic polyps and cancer, chlolelithiasis, acute pancreatitis, pancreatic malignancy, liver diseases including NAHS, cirrhosis and hepatocellular carcinoma.[5]

This research was conducted to assess the relationship between various grades of body mass index and endoscopic changes in gastric and colonic mucosa, hepatic tissues fatty-fibrotic changes, and blood chemistry abnormalities.

2. Patients and methods

A case-control study recruited 72 subjects with different grades of BMI from the outpatient clinic at Al-Azhar University Hospital. The body mass index (BMI) was calculated in all participants, then divided into non-obese (BMI<30) (n=36) and obese (BMI>30) (n=36). Non-obese subjects were further categorized into normal subjects with (BMI 18-24.9 kg/m2) (group I) (n=17) and overweight (BMI 25-29.9 kg/m2 (group II) (n=19). Obese subjects were further divided into obese (BMI 30-34.9 kg/m2) (group III) (n=20), and morbid obese (BMI ≥ 35 kg/m2) (group IV) (n=16).

Routine blood tests including CBC, lipid profile, fasting and post prandial blood sugar, HbA1C, liver and kidney function tests, serum uric acid were assessed. Ultrasonography was done for detection of liver changes that were categorized into three categories: minimal, moderate and marked. Fibroscan was done for assessing fibrotic changes of the liver by measuring fibrosis score (F0-F4). Upper endoscopy and colonoscopy were done to address the macroscopic GIT mucosal changes.

Exclusion criteria included age less than 18 years old, Diabetes Mellitus, end-stage organ failure, known organic upper GIT or colonic diseases, chronic liver, viral or autoimmune disorders, and malignancy.

The findings were processed and analyzed using Microsoft Excel 365 and SPSS's IBM Statistics, version 29.0.1. Data were represented as mean ± SD. A one-way analysis of variance to compare groups was used. When P-values proved significant, the student's test for paired and unpaired data was used to identify differences between groups. The correlation coefficient (r) was used to compare two variables. The chi-square test (X2) was utilized to evaluate the difference between two proportions. A level of probability P≤0.05 was significant.

Ethics approval

This research was endorsed by the ethical committee at the department of Hepatology, Gastroenterology, and Infectious diseases and by the ethical committee at Al-Azhar faculty of medicine. Written consent was endorsed by subjects.

Sample size calculation

We were planning a study of subjects in which we would regress the values of patients against those of controls. Prior data indicated that the standard deviation of the control is 0.9, and the standard deviation of the regression errors will be 1.94. If the true slope of the line obtained by regressing patients against control is 1.4, we will need to study 35 subjects at least for each group of BMI, to be able to reject the null hypothesis that this slope equals zero with probability (power) 90%. The Type I error probability associated with this test of the null hypothesis is 0.05. The formula for calculating a z-score is $z = (x-\mu)/\sigma$, where x is the raw score, μ is the population mean, and σ is the population standard deviation.

3. Results

Table 1. Age distribution in each study group.

	AGE SUBGROUPS/YEAR OLD				
STUDY GROUPS	18 – 30	>30 – 40	>40 – 50	>50 – 60	>60
GROUP I (N=17)	7(41.2%)	6(35.3%)	1(5.9%)	3(17.6%)	0(0%)
GROUP II (N=19)	11(57.9%)	3(15.8%)	3(15.8%)	2(10.5%)	0(0%)
GROUP III (N=20)	7(35%)	7(35%)	4(20%)	1(5%)	1(5%)
GROUP IV (N=16)	1(6.2%)	8(50%)	2(12.5%)	3(18.8%)	2(12.5%)
P VALUE:	0.001	<0.001	0.007	0.021	NA
I VS. II	0.194	0.950	<0.001	<0.001	NA
I VS. III	<0.001	0.003	0.046	0.759	NA
I VS. IV	<0.001	<0.001	0.294	0.012	NA
II VS. III	<0.001	<0.001	0.318	0.034	NA
II VS. IV	<0.001	0.003	0.023	<0.001	0.023
III VS. IV					

Age subgroup 18-30 years old were prevalent more often in group II (overweight) with high significance when compared to other groups, while age subgroups (30-40 years old) was prevalent more often in group III (obese) & IV (morbid obese) with high significance compared to group I & II, and significance in group IV relative to group III (p-value < 0.05). Beyond 40 years of age, changes were statistically insignificant (Table 1).

Table 2. Gender composition in each group.

STUDY GROUPS		GENDER	
		Female	Male
GROUP I (N=17)		10(58.8%)	7(41.2%)
GROUP II (N=19)		13(68.4%)	6(31.6%)
GROUP III (N=20)		11(55%)	9(45%)
GROUP IV (N=16)		9(56.3%)	7(43.7%)
P VALUE:	I VS. II	0.039	0.039
	I VS. III	0.445	0.445

I VS. IV	0.614	0.614
II VS. III	0.007	0.007
II VS. IV	0.015	0.015
III VS. IV	0.793	0.793

Female gender was seen more often in group II (overweight) relative to group I (normal) & IV (morbid obese) with significant difference when compared with group III (obese) (p-value < 0.05). Male gender was seen more often in group III (obese) with high significant difference compared to group II (overweight) and significant difference in group I (normal) & IV (morbid obese) relative to group II (overweight) (p-value < 0.05) (Table 2).

Table 3. Clinical history in individual study groups.

	DYSPHAGIA	GERD MANIFESTATIONS	GASTRIC UPSETS	COLONIC SYMPTOMS
GROUP I (N=17)	0 (0%)	8 (47%)	7 (41.1%)	13 (76.5%)
GROUP II (N=19)	0 (0%)	6 (31.6%)	10 (52.6%)	12 (63.2%)
GROUP III (N=20)	2 (10%)	7 (35%)	14 (70%)	4 (20%)
GROUP IV (N=16)	0 (0%)	6 (37.5%)	9 (56.3%)	11 (68.8%)
P VALUE:	NA	0.185	0.258	0.209
I VS. II	0.143	0.241	0.047	0.342
I VS. III	NA	0.301	0.207	0.322
I VS. IV	0.128	0.416	0.145	0.334
II VS. III	NA	0.364	0.420	0.374
II VS. IV	0.151	0.441	0.211	0.468
III VS. IV				

Clinical history in individual study groups revealed that gastric upsets were common in group III (obese subjects) (70%) with significant differences in comparison to group I (normal subjects) (41.1%) (p-value < 0.05), with insignificant changes in other clinical history (p-value > 0.05) (Table 3).

Table 4. Abdominal examination in individual study groups.

	RT. LOBE HEPATOMEGALY	LT. LOBE HEPATOMEGALY	SPLENOMEGALY	+VE MURPHY'S SIGN	EPIGASTRIC TENDERNESS
GROUP I (N=17)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	8(47.1%)
GROUP II (N=19)	0(0%)	0(0%)	0(0%)	1(5.3%)	7(36.8%)
GROUP III (N=20)	1(5%)	1(5%)	1(5%)	3(15%)	10(50%)
GROUP IV (N=16)	5(31.3%)	5(31.3%)	1(6.3%)	1(6.3%)	8(50%)
P VALUE I VS. II	NA	NA	NA	0.264	0.279
I VS. III	0.271	0.277	0.474	0.073	0.562
I VS. IV	0.009	0.011	0.471	0.242	0.433
II VS. III	0.500	0.256	0.556	0.0191	0.216
II VS. IV	0.007	0.007	0.444	0.457	0.229
III VS. IV	0.027	0.026	0.444	0.237	0.500

Abdominal examinations in individual study groups addressed hepatomegaly in group IV (morbid obese) (31.3%) with high significance when compared to group III (obese) (5%) (p-value < 0.05). Murphy's sign was prevalent in group III (obese) (15%) with a high significance relative to other groups (p-value < 0.05). No significant changes detected among groups in other GIT examination findings (p-value > 0.05) (Table 4).

Table 5. Laboratory biochemical blood tests results.

STUDY GROUPS	MEAN ± STANDARD DEVIATION (SD) OF EACH LABORATORY BLOOD TEST								
	Serum uric acid	FBG	PPBG	Blood Cholesterol	Blood TG	LDL	VLDL	HDL	WBCs count
GROUP I (N=17)	4.70 ± 1.30	92.3 ± 14.2	122.8 ± 15.2	179.7 ± 18.6	123.3 ± 41.1	99.4 ± 23.6	25.80 ± 7.9	51.5 ± 12.9	4.44 ± 0.58
GROUP II (N=19)	4.20 ± 0.90	104.8 ± 20.2	133.0 ± 101.8	199.5 ± 45.3	130.2 ± 70.4	109.3 ± 36.0	25.6 ± 14.4	48.0 ± 7.60	5.87 ± 0.60
GROUP III (N=20)	4.50 ± 1.70	116.8 ± 37.9	190.4 ± 65.8	210.1 ± 36.2	206.9 ± 139.3	126.2 ± 43.6	42.2 ± 28.8	43.3 ± 8.50	7.88 ± 0.50
GROUP IV (N=16)	6.00 ± 1.40	142.3 ± 49.2	205.2 ± 97.9	208.3 ± 36.4	168.0 ± 73.4	124.8 ± 39.9	33.6 ± 14.6	45.6 ± 9.50	10.36 ± 0.92
P VALUE I VS. II	0.132	0.114	0.455	0.046	0.359	0.165	0.479	0.171	<0.001
I VS. III	0.36	0.035	0.041	0.001	0.009	0.012	0.019	0.016	<0.001
I VS. IV	0.004	0.027	0.047	0.005	0.022	0.024	0.039	0.077	<0.001
II VS. III	0.279	0.206	0.284	0.215	0.019	0.098	0.023	0.037	<0.001
II VS. IV	<0.001	0.065	0.244	0.265	0.066	0.131	0.065	0.220	<0.001
III VS. IV	0.003	0.15	0.387	0.443	0.145	0.463	0.144	0.234	<0.001

Regarding laboratory biochemical blood test results, higher levels of serum uric acid were seen more often in group IV (morbid obese) compared to other groups (p-value < 0.05). Both groups III (obese) and IV (morbid obese) showed a significantly increased fasting and post prandial blood glucose, cholesterol, triglycerides compared to other study groups (p-value < 0.05). High-density lipoproteins had a higher frequency in group I (normal BMI) compared to other study groups (p-value < 0.05) (Table 5).

Table 6. Liver function tests in individual study groups.

STUDY GROUPS	LIVER FUNCTION TESTS						
	ALT	AST	Total Bilirubin	Direct Bilirubin	Indirect Bilirubin	Serum Albumin	INR
GROUP I (N=17)	25.4±9.2	25.8±6.2	0.8±0.3	0.3±0.2	0.4±0.2	4.3±0.6	0.9±0.1
GROUP II (N=19)	25.2±15.4	23.8±9.0	0.8±0.2	0.4±0.2	0.4±0.1	4.1±0.5	0.9±0.1
GROUP III(N=20)	25.4±15.8	24.8±10.2	0.7±0.3	0.3±0.1	0.4±0.2	4.2±0.6	1.0±0.2
GROUP IV(N=16)	32.5±21.1	30.4±14.6	0.8±0.2	0.3±0.1	0.5±0.2	4.4±0.6	0.9±0.1
P VALUE: I VS. II	0.476	0.21	0.494	0.384	0.331	0.13	0.357
I VS. III	0.498	0.358	0.258	0.215	0.367	0.228	0.197
I VS. IV	0.116	0.128	0.334	0.32	0.18	0.441	0.37
II VS. III	0.478	0.362	0.237	0.138	0.489	0.356	0.112
II VS. IV	0.13	0.061	0.324	0.219	0.049*	0.102	0.496
III VS. IV	0.139	0.102	0.133	0.36	0.099	0.186	0.124

Liver function tests in individual study groups had no significant difference among groups (p-value > 0.05). These findings reflect that there was no steatohepatitis in all groups (Table 6).

Table 7. Correlation of BMI with liver tissues fibroscan results in studied groups.

STUDY GROUPS	FIBROSIS (F) SCORES IN KILOPASCAL (KP)				
	F0 (0-5 KP)	F1 (5.1-7KP)	F2 (7.1-10KP)	F3 (10.1-15KP)	F4 (>15 KP)
GROUP I (N=17)	15(88.2%)	1(5.9%)	1(5.9%)	0(0%)	0(0%)
GROUP II (N=19)	12(63.2%)	6(31.6%)	1(5.2%)	0(0%)	0(0%)
GROUP III (N=20)	9(45%)	3(15%)	8(40%)	0(0%)	0(0%)
GROUP IV (N=16)	4(25%)	9(56.2%)	2(12.5%)	1(6.3%)	0(0%)
P VALUE: I VS. II	0.051	0.033	0.472	NA	NA
I VS. III	0.003	0.220	0.009	NA	NA
I VS. IV	0.0001	0.001	0.289	0.242	NA
II VS. III	0.139	0.126	0.006	NA	NA
II VS. IV	0.015	0.082	0.259	0.228	NA
III VS. IV	0.121	0.006	0.041	0.222	NA

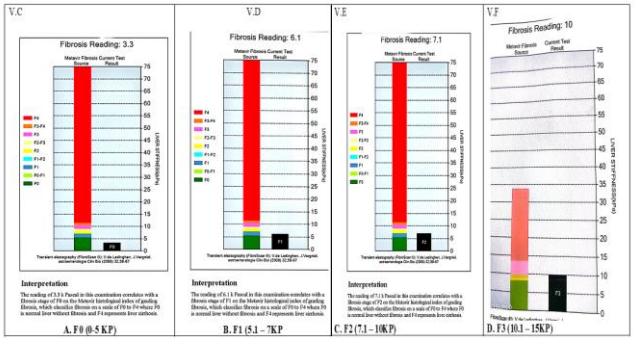

Figure 1. Fibrosis (F) grading in kilopascal (KP)

Correlation of BMI of each group with Fibroscan results of hepatic parenchymal changes evaluation revealed more frequent absence of hepatic fibrocirrhotic changes (F0) in group I (normal) (88.2%) and II (overweight) (63.2%) compared to other groups. Moderate hepatic fibrocirrhosis (F7.1-F10) was recognized in group III (obese) (40%) compared to the rest of study populations (p-value < 0.05) (Table 7) &(Figure 1).

Table 8. Ultrasound hepatic parenchymal changes in individual study groups.

STUDY GROUPS	HEPATIC PARENCHYMAL CHANGE GRADING		
	No abnormality detected	Cirrhotic liver	Fatty VS fibrotic liver
GROUP I (N=17)	17(100%)	0(0%)	0(0%)
GROUP II (N=19)	11(57.9%)	0(0%)	8(42.1%)
GROUP III (N=20)	12(60%)	0(0%)	8(40%)
GROUP IV (N=16)	3(18.8%)	1(6.2%)	12(75%)
P VALUE: I VS. II	0.001	NA	0.001
I VS. III	0.002	NA	0.002
I VS. IV	<0.001	0.250	0.0001
II VS. III	0.449	NA	NA
II VS. IV	0.012	0.250	0.031
III VS. IV	0.008	0.250	0.022

Ultrasound hepatic parenchymal changes in individual study groups detected that the frequency of no abnormality detected is the highest in group I (100%) and III (60%) compared to the rest of study population (p-value < 0.05), while bright fatty or fibrotic liver parenchyma was dominant in group IV (75%) compared to other groups (p-value < 0.05) (Table 8) (Figure 2).

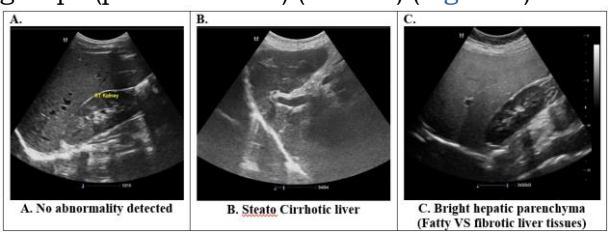

Figure 2. Grading of hepatic parenchymal changes of liver tissues by ultrasound

4. Discussion

Obesity refers to a failure of balance between the anabolic and catabolic processes involved in fat metabolism .[6] Excessive fat deposition can affect health and cause characteristic disorders that mandate medical and sometimes surgical intervention. Obesity is a leading cause of type 2 diabetes and cardiovascular diseases. Also, it can affect bone health and reproduction. Moreover, it may cause certain cancers. Obesity interferes with the quality of life, particularly sleep and movement. Obesity is a multifactorial disease resulting from psychological factors, obesogenic environments, and genetic predisposition. In some instances, other factors include (drugs, lack of mobilization and genetic susceptibility) .[7]

The current research included 72 subjects with different grades of BMI. They were selected from the outpatient clinic during assigned period of the study. Subjects were divided into normal BMI, overweight, obese, and morbid obese. This study was done to assess relationship between various grades of body mass index with the endoscopic changes in gastric and colonic mucosa, hepatic tissues fibrocirrhotic changes and blood chemistry abnormalities (routine blood tests).

In 2022, about 16% of adults aged 18 years and older were globally obese; the reported prevalence of obesity has more than doubled from 1990 to 2022 worldwide .[7] It is reported during the current research that the prevalence of high BMI (≥30 kg/m2) is found among older age subgroup (30-40 years old), while nonobese (BMI<30 kg/m2) in younger age subgroup (18–30 years old) without any variation in BMI after 40 years old. This finding is supported by Huang et al.[8] who concluded that there was an increasing prevalence of obesity in association with increasing age. Also, these results agree with Samper-Ternent and Al Snih [9], who concluded that obesity among older adults has increased noticeably during the last two decades worldwide.

The current study concluded that being overweight was more prevalent in women, while obesity was more prevalent in men. On the contrary, Cooper et al[10] reported that obesity is more prevalent in females than males due to sociocultural, environmental, and physiological differences. In 2022, 2.5 billion adults more than 18 years old were overweight, including more than 890 million adults who are obese. This includes 43% of adults aged 18 years and older, 43% of men, and 44% of women who were overweight .[7] Overweight changed from 31% in South-East Asia to 67% in Africa and America .[7] Future assessment of obesity indicates that eighteen percent of the male population and twenty-one percent of the female population will be obese by 2025 .[11]

In the current study, endoscopic screening of the stomach and colon of obese individuals revealed a normal appearance of mucosa despite reporting inflammatory and erosive lesions of the stomach and esophagus in obese dyspeptic patients by Basha et al[12]. So, the clinical values of recommending upper gastrointestinal endoscopy in the obese population should be related to the severity of symptoms and to the degree of response to medical management. Another contradiction with the current research data was reported by Huseini et al,[13] who declared an increased frequency of GI symptoms in obese patients, while only gastric upset (non-specific finding) was found by our team.

By abdominal examination, the current research concluded that there were associations between high BMI and hepatomegaly and positive Murphy's signs. On the other hand, nonobese individuals (BMI <30 kg/m2) had normal liver size. These findings are supported by Dorostghol et al[14] who concluded that the size of the liver in obese persons was increased when compared with healthy subjects, and the liver size was correlated with age, gender, body mass index, liver steatosis and grades of hepatic steatosis. At the same time, Silva et al[15] concluded that there were significant changes in liver size detected only in persons with BMI < 25 kg/m² identified by clinical evaluation and ultrasonography.

The current study found a higher level of serum uric acid in morbidly obese subjects, unexplained findings that may need further exploration. Both obese and morbidly obese subjects showed an increased level of harmful lipids. Beneficial lipid (high-density lipoproteins) was frequently reported by the current research in subjects with normal BMI. Accordingly, it is reported in the text that obesity alone or with prediabetes has harmful impacts on male and female adolescents, while prediabetes alone independently influenced LDL-C and HDL-C .[16] Hussain et al[17] detected a negative correlation between HDL and BMI level and a little effect of BMI on lipid profile. On the contrary, Chinyere and Sola[18] reported an absence of correlation between central obesity and lipid profile. This contradiction may be due to the inappropriate sample size or the study's restriction on central obesity.

Both obese and morbid obese subjects in the current work reported frequently increased baseline mean plasma glucose levels compared to other study groups. These results are supported by Patel et al[19], who concluded that a significant correlation was detected between BMI and mean average plasma glucose levels.

All study populations had normal levels of liver function tests that reflect undetectable

steatohepatitis in all study populations, especially in those with overweight, obese and morbidly obese subjects. These findings agree with Al Akwaa et al[20] who concluded that the probability of abnormal liver function tests is minimal in obese and markedly obese patients.

Hepatic parenchymal tissues were evaluated in the current work using ultrasound images as a preliminary screening procedure, followed by using Fibroscan to grade fibrosis and calculate the F score. The absence of hepatic fibro cirrhotic changes (F0) was addressed in almost all subjects with normal BMI and in overweight subjects, but not in obese individuals. Moderate hepatic fibro cirrhotic changes (bright liver plus F7.1 to F10) were recognized in obese subjects, but not in the rest of the study populations. The above-mentioned findings agree with Gopalakrishna et al[21], Abdelfattah et al[22], Jaskiewicz et al[23], and Park et al[24]. There is a significant correlation between body mass index and liver stiffness measurement (LSM), and BMI correlates with the severity of LSM using transient elastography scores in patients with NAFLD .[21] Hepatic steatosis is common among obese patients who have undergone bariatric surgery and can be detected by abdominal ultrasonography and liver biopsy .[22] Obesity associated with type 2 diabetes develops more severe steatosis of the liver .[23] Correlation was detected between BMI and hepatic steatosis with increases in the BMI of .[24]

All participants in the current research underwent upper gastrointestinal endoscopy and colonoscopy for macroscopic evaluation of the GIT mucosa. The endoscopic screening did not reveal any lesion for specific GIT disease, despite the reported data by Islam et al[25] that recognized obesity as a cause for different GI diseases. At the same time, it is not clear whether dysbiosis is a reason or a result of obesity. Additionally, bacteria were detected in numerous adipose tissues that initiate and sustain adipose tissue asymptomatic inflammation and serious metabolic outcomes of the infected fatty tissues in obese subjects .[26] On the other hand, obesity has been linked with a change in the gut microbiome .[27]

Limitations of the study: This study has inherent limitations of a cross-sectional study design. It only studied the subjects with different BMIs at a specific time. It did not follow-up subjects, especially the obese and morbid obese subjects to document further development of symptoms, specific GIT disease or endoscopic changes. Another obstacle of the research is the small sample size of subjects with different BMI included in the research.

4. Conclusion

There is a considerable impact of various degrees of obesity on hepatic parenchyma and blood biochemistry. Obesity is associated with hepatic parenchymal fibrocirrhotic changes, increased levels of routine biochemical laboratory blood tests, and undetectable macroscopic lesions on gastro-colonic endoscopic screening.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

Funding

No Funds : Yes

Conflicts of interest

There are no conflicts of interest.

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