

Associations of abdominal visceral and subcutaneous adipose tissue with clinical and computed tomography imaging markers of metabolic syndrome

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Abstract

Aim: Central obesity comprises visceral (VAT) and subcutaneous (SAT) adipose tissues, which were reported to have associations with metabolic syndrome. Abdominal computed tomography (CT) scan allows evaluation of VAT, SAT, hepatosteatosi s, pancreatic steatosis, and aortoiliac atherosclerosis. We aim to investigate the relationship between central obesity components (VAT, SAT) and metabolic syndrome imaging markers that are detectable on abdominal CT and the predictive power of these parameters for detecting hypertension (HT), diabetes mellitus (DM), and dyslipidemia (DL).

Materials and Methods: Abdominal CT scans of 321 patients were evaluated for VAT, SAT volumes, hepatosteatosi s, pancreatic steatosis, and aortoiliac atherosclerosis in this retrospective cross-sectional study. The associations of VAT, SAT, and visceral-to-subcutaneous fat (V/S) ratio with HT, DM, DL, hepatosteatosi s, pancreatic steatosis, and aortoiliac atherosclerosis were investigated by Mann-Whitney U test, Kendall's Tau-b test, and boosting linear regression analysis. Cut-off values of VAT, SAT, and V/S ratio were calculated to predict HT, DM, DL, hepatosteatosi s, pancreatic steatosis, and aortoiliac atherosclerosis by receiver operating characteristic (ROC) curve analysis.

Results: Increased VAT volume and V/S ratio were significantly related to HT, DM, DL, hepatosteatosi s, pancreatic steatosis, and aortoiliac atherosclerosis ($p < 0.001$). Increased SAT volume showed significant correlations to HT, iliac atherosclerosis, hepatosteatosi s, and pancreatic steatosis. VAT volume and V/S ratio predicted HT, DM, and DL with diagnostic accuracy ranging from sufficient to good. Specifically, VAT volume predicted hepatosteatosi s with a very good diagnostic accuracy.

Conclusion: VAT volume and V/S ratio are related to clinical manifestations and abdominal CT markers of the metabolic syndrome with a more substantial relationship present with VAT. Reported cut-off values could be utilized to detect metabolic syndrome earlier, which would provide early lifestyle alterations aiming at a lesser fat percentage in body weight, leading to decreased morbidity and mortality rates.

Keywords: Atherosclerosis; computed tomography; fatty liver; metabolic syndrome; subcutaneous adipose tissue; visceral adipose tissue

INTRODUCTION

Obesity is a serious clinical condition that is on an increasing trend worldwide, especially in developed countries (1). Abdominal fat accumulation which is also referred to as central obesity is considered a risk factor for metabolic syndrome. Subjects at normal body mass index (BMI) with increased waist-to-hip ratio indicating central obesity were found to have elevated mortality risk for cardiometabolic disease (2). Increased visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) contribute to central obesity but differ in their structural composition, metabolic activity and functional significance (3). VAT rather than SAT is shown to be closely associated with metabolic syndrome related clinical manifestations

such as atherosclerosis, hypertension, dyslipidemia, and diabetes (4-6).

Fatty infiltration of abdominal solid organs such as the liver and pancreas could have serious clinical presentations. Fatty liver (hepatosteatosi s) is associated with nonalcoholic steatohepatitis, which could lead to fibrosis and cirrhosis, presenting with a risk of hepatocellular carcinoma (7). Pancreatic steatosis causes an increase of cytokines and inflammation, which may cause diabetes and/or malignancy (8,9). Abdominal aortic wall calcification is an indicator of atherosclerosis, which is an inflammatory process related to visceral fat accumulation and dyslipidemia (10).

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Abdominal computed tomography (CT) without administration of intravenous contrast media is commonly utilized for the investigation of urinary stone disease and related complications. Besides the urinary system, the imaging area includes the liver, pancreas, abdominal aorta, iliac arteries as well as visceral and subcutaneous fat tissue. It is possible for the radiologist to report metabolic syndrome related alterations on the abdominal CT scan such as; hepatosteatosis, pancreatic steatosis, calcified atherosclerotic plaques in the abdominal aorta and iliac arteries, and visceral obesity characterized with increased VAT deposition. There are a few reports in the literature that have separately investigated the relationship between VAT accumulation and hepatosteatosis, pancreatic steatosis, and abdominal larger vessel atherosclerosis (9,11,12). However, a study investigating the relationship between VAT, SAT and abdominal CT markers of the metabolic syndrome, combined with clinical metabolic syndrome manifestations (hypertension, dyslipidemia, and diabetes mellitus) is still lacking. Revealing such relationships and predictive power of visceral/subcutaneous fat accumulation and abdominal CT markers for the metabolic syndrome components could provide early diagnosis of such conditions, leading to lower rates of morbidity and mortality.

In this study, we aim to investigate the relationship between central obesity components (VAT, SAT) and metabolic syndrome imaging markers that are detectable on abdominal CT and the predictive power of these conditions for detecting hypertension, dyslipidemia, and abdominal large vessel atherosclerosis.

MATERIALS and METHODS

Study Design and Population

Institutional ethics review board approval was obtained prior to this retrospective cross-sectional study (project number: KA20/208). Abdominal CT examinations that were acquired for urolithiasis investigation without the administration of intravenous contrast media between January 2019 and January 2020 were considered. Exclusion criteria were; repeat studies, being younger than 18 years old, cancer diagnosis, chronic renal/hepatic disease, transplantation, and major abdominal surgery. CT studies with artifacts (motion, beam hardening, or photon starvation) or without complete inclusion of SAT into the field of view were also excluded. Following exclusion, abdominal CT studies of 321 patients constituted the study cohort. Electronic medical records of patients were reviewed for chronic diseases after the completion of measurements. Age, gender, presence of hypertension, diabetes mellitus, and dyslipidemia were recorded.

Data Acquisition and Analysis

All CT studies were acquired with a 3rd generation dual-source multidetector CT scanner (Somatom Force, Siemens Healthcare), operated in dual-energy mode (for urinary stone analysis) with the following parameters: 103 mAs effective at 100 kV and 54 mAs effective at 150kV with a tin filter, 0.7 pitch factor, 0.5-second rotation time and 3 mm slice thickness. Image acquisition commenced

just above the diaphragm dome and ceased at the greater trochanter. Image reconstruction was performed utilizing the Br40 kernel.

Measurements were performed on axial images, with the patient's name and age anonymized, using a dedicated workstation (Syngo.via, Version 3.0, Siemens Healthineers) by a radiologist with 8 years of experience in evaluating abdominal CT studies. Anatomy Visualizer tool of the Syngo.via workstation was utilized. First, multiplanar reconstructions were reviewed to establish mid-level of L4 vertebra body, followed by the manual marking of SAT, visceral space, and muscle tissue in the selected slice. Thresholds for tissue attenuation value in Hounsfield Units (HU) of fat and muscle tissue were established between -200 HU - -40 HU and -40 HU - 140 HU, respectively. SAT, VAT, and muscle tissue volumes were recorded in cm³, and visceral-to-subcutaneous fat volume ratio (V/S ratio) was calculated (Figure 1). The next step was to measure attenuation values of liver, pancreas, and spleen in HU, which is demonstrated in Figure 2. Attenuation values in HU were obtained by placing a region of interest (ROI), 1 cm² in size. Parenchymal lesions and vascular structures were avoided. Liver attenuation was measured by placing two ROIs on the right hepatic lobe, one on the left hepatic lobe, and one on the caudate lobe approximately at the level of the porta hepatis. Pancreas attenuation was measured by placing ROIs on the head, body, and tail segments. Spleen attenuation was measured by placing one ROI on the center part of the organ. Mean values were calculated for the liver and pancreas. Criteria for hepatosteatosis were liver-to-spleen attenuation ratio ≤ 0.9 or a liver attenuation value at least 10 HU lower than the splenic attenuation value (13). Pancreatic steatosis was defined as a pancreas-to-spleen attenuation ratio ≤ 0.8 or a pancreas attenuation value at least -9 HU lower than the splenic attenuation value (14). For the final step, the presence of calcified atherosclerotic plaques on the abdominal aorta and/or iliac arteries was noted.

Statistical Analysis

Statistical analyses were performed using SPSS 26.0 (IBM Corporation, Armonk, New York, United States) and Medcalc 14 (Acacialaan 22, B-8400 Ostend, Belgium). Power analysis was performed for the sample size of this study and was found 82.4%. Univariate data suitability for normal distribution was evaluated by Shapiro-Wilk and Shapiro-Francia tests. Mann-Whitney U test was used with Monte Carlo results for the comparison of two independent groups according to quantitative data. Kendall's Tau-b test was utilized in order to examine the correlations of variables. Boosting Linear Regression analysis, one of the ensemble machine learning methods, was used to reveal the causality between dependent and independent variables in the form of a mathematical model. Automatic data preparation steps (adjustment of measurement level, outlier and missing value handling, supervised merging, outlier and missing value handling and supervised merging) were applied to increase predictive power. Akaike information criterion (AICC) method was used in best subsets method, one of the model selection methods.

The sensitivity and specificity ratios for the relationship between the classification separated by the cut-off value calculated according to the variables of the groups and the actual classification were examined and expressed by ROC (Receiver Operating Curve) curve analysis. Quantitative variables were expressed as median (25th percentile/ 75th percentile) in the tables, while categorical variables were shown as n (%). Variables were examined at a 95% confidence level, and a p-value of less than 0.05 was considered significant.

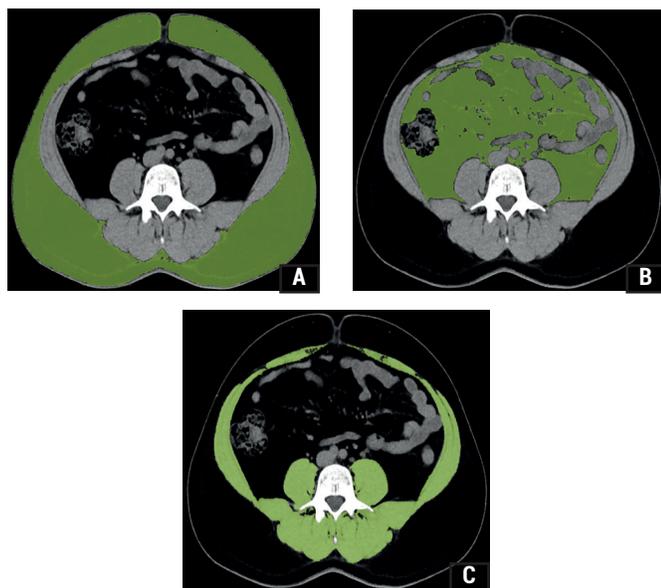


Figure 1. Axial unenhanced abdominal computed tomography images at the level of L4 demonstrate subcutaneous fat tissue (A), visceral fat tissue (B), and muscle tissue (C) following manual marking of these spaces and application of threshold values for fat and muscle

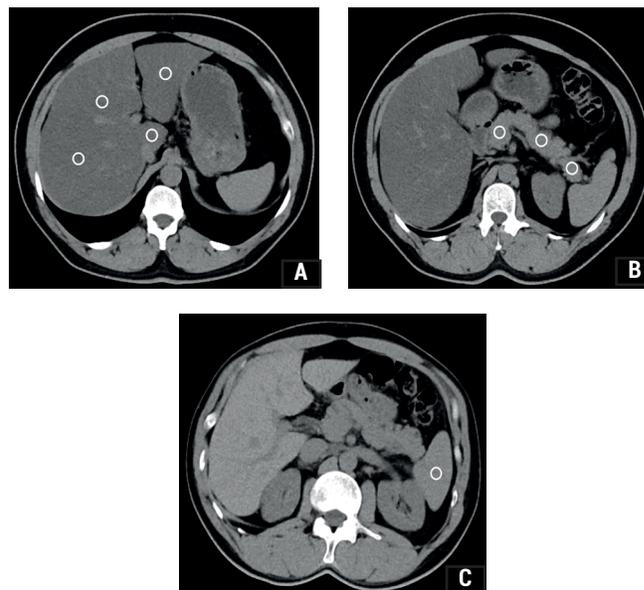


Figure 2. Axial unenhanced abdominal computed tomography images demonstrate region of interest (white circles, 1 cm² each) placement for determining attenuation values of the liver (A), pancreas (B), and spleen (C) in Hounsfield Units

RESULTS

Patient Characteristics and Abdominal CT Measurements

There were 191 males (59.5%) and 130 females (40.5%) with a mean age of 49.3 ± 15.6 (SD) years (range: 18 – 89 years). Mean age of males was 48.5 ± 14.1 (SD) years (range: 19 – 84 years) and females was 50.4 ± 17.6 (SD) years (range: 18 – 89 years). Patient characteristics and metabolic syndrome markers on abdominal CT were summarized in Table 1. Median values of VAT volume and V/S ratio were higher in males (p<0.001).

Table 1. Patient characteristics and metabolic syndrome markers on abdominal computed tomography						
	SAT Volume (cm ³) Median (Q1 / Q3)	p value	VAT Volume (cm ³) Median (Q1 / Q3)	p value	V/S Median (Q1 / Q3)	p value
Gender						
Female (n=130)	75.37 (56.01/105.24)	0.107 ^u	33.25 (16.85/55.17)	<0.001 ^u	0.45 (0.29/0.60)	<0.001 ^u
Male (n=191)	67.10 (52.72/89.08)		52.07 (38.72/72.87)		0.76 (0.56/1.03)	
Hypertension						
(-) (n=214)	67.42 (51.07/87.25)	<0.001 ^u	41.06 (22.89/54.46)	<0.001 ^u	0.54 (0.36/0.78)	<0.001 ^u
(+) (n=107)	80.50 (60.99/104.27)		63.13 (42.19/82.81)		0.75 (0.55/0.98)	
Diabetes Mellitus						
(-) (n=268)	68.00 (52.66/90.78)	0.214 ^u	43.48 (26.61/61.60)	<0.001 ^u	0.57 (0.39/0.81)	<0.001 ^u
(+) (n=53)	78.09 (58.02/96.91)		71.08 (49.41/95.40)		0.90 (0.69/1.20)	
Dyslipidemia						
(-) (n=280)	69.28 (52.66/92.35)	0.511 ^u	43.91 (26.71/64.72)	<0.001 ^u	0.60 (0.39/0.87)	<0.001 ^u
(+) (n=41)	74.30 (60.32/89.08)		55.47 (44.74/78.45)		0.79 (0.62/0.93)	
Iliac Atherosclerosis						
(-) (n=169)	66.60 (51.82/87.25)	0.028 ^u	38.24 (22.89/52.10)	<0.001 ^u	0.52 (0.35/0.74)	<0.001 ^u
(+) (n=152)	76.10 (57.50/96.02)		57.15 (39.17/77.90)		0.74 (0.49/1.02)	
Aorta Atherosclerosis						
(-) (n=191)	68.00 (52.86/92.17)	0.390 ^u	39.33 (24.04/54.45)	<0.001 ^u	0.52 (0.35/0.76)	<0.001 ^u
(+) (n=130)	72.88 (55.19/92.64)		60.16 (40.47/80.23)		0.76 (0.54/1.06)	
Pancreatic Steatosis						
(-) (n=137)	66.60 (48.27/80.87)	<0.001 ^u	31.85 (16.85/46.08)	<0.001 ^u	0.47 (0.32/0.68)	<0.001 ^u
(+) (n=184)	77.01 (58.52/101.20)		58.60 (42.04/77.92)		0.74 (0.53/1.00)	
Hepatosteatorosis						
(-) (n=244)	65.87 (50.35/87.27)	<0.001 ^u	40.27 (23.29/55.63)	<0.001 ^u	0.54 (0.37/0.82)	<0.001 ^u
(+) (n=77)	83.49 (66.85/105.94)		70.82 (50.84/90.09)		0.76 (0.63/1.04)	

	r		r		r	
Age	0.089	0.018^b	0.322	<0.001^b	0.311	<0.001^b
Muscle Volume (cm ³)	0.137	<0.001^b	0.267	<0.001^b	0.226	<0.001^b
L (HU)	-0.258	<0.001^b	-0.348	<0.001^b	-0.208	<0.001^b
P (HU)	-0.195	<0.001^b	-0.371	<0.001^b	-0.274	<0.001^b
S (HU)	-0.039	0.296 ^b	0.086	<0.001^b	0.150	<0.001^b
L/S	-0.226	<0.001^b	-0.355	<0.001^b	-0.243	<0.001^b
L-S	-0.230	<0.001^b	-0.355	<0.001^b	-0.241	<0.001^b
P/S	-0.182	<0.001^b	-0.394	<0.001^b	-0.311	<0.001^b
P-S	-0.177	<0.001^b	-0.394	<0.001^b	-0.315	<0.001^b

^bKendall's tau-b test, HU: Hounsfield unit, L: liver attenuation, P: pancreas attenuation, Q1: 25th percentile, Q3: 75th percentile, r: correlation coefficient, S: spleen attenuation, SAT: subcutaneous adipose tissue, u: Mann-Whitney U Test (Monte Carlo), V/S: visceral-to-subcutaneous fat volume ratio, VAT: visceral adipose tissue

VAT volume and V/S ratio were significantly higher in patients with hypertension, diabetes mellitus, aortic and iliac atherosclerosis, pancreatic steatosis, and hepatosteatois (p<0.001). Also, there was a weak positive correlation between age vs. VAT volume and V/S ratio (p<0.001). SAT volume was significantly higher in patients with hypertension, iliac atherosclerosis, pancreatic steatosis, and hepatosteatois (p<0.005).

ROC Curve Analysis

ROC Curve analysis was performed to determine cut-off values for categorical variables that showed statistical significance with CT measurements and results are summarized in Table 2. SAT volume (cm³) cut-off values for adequate prediction of hypertension, pancreatic

steatosis and hepatosteatois were >78.06, >76.35 and >78.08 respectively (p values; 0.001, <0.001 and <0.001 respectively).

VAT volume (cm³) cut-off values for adequate prediction of dyslipidemia, iliac atherosclerosis, and aortic atherosclerosis were >44.46, >52.1 and >52.67 respectively (p values <0.001). VAT volume (cm³) cut-off values for good prediction of male gender, hypertension, diabetes mellitus, and pancreatic steatosis were >35.27, >52.1, >49.3 and >51.55 respectively (p values <0.001). VAT volume above 56.54 cm³ was found to have a very good diagnostic accuracy for predicting hepatosteatois (p<0.001).

Table 2. Prediction of statistically significant gender and metabolic syndrome components with receiver operating characteristic (ROC) curve analysis according to computed tomography measurements of subcutaneous and visceral fat tissue volumes

	Cut-off	Sensitivity	Specificity	AUC±SE	Diagnostic Accuracy	p value
SAT Volume (cm³)						
Hypertension	>78.06	55.1%	68.7%	0.609 ± 0.033	Sufficient	0.001
Iliac Atherosclerosis	>78.06	48.0%	68.6%	0.570 ± 0.032	Bad	0.029
Pancreatic Steatosis	>76.35	51.1%	71.5%	0.628 ± 0.031	Sufficient	<0.001
Hepatosteatois	>78.08	70.1%	62.3%	0.692 ± 0.032	Sufficient	<0.001
VAT Volume (cm³)						
Gender (Male)	>35.27	80.6%	53.9%	0.701 ± 0.030	Good	<0.001
Hypertension	>52.1	64.5%	72.4%	0.723 ± 0.029	Good	<0.001
Diabetes Mellitus	>49.3	75.5%	61.9%	0.746 ± 0.038	Good	<0.001
Dyslipidemia	>44.46	78.1%	51.4%	0.656 ± 0.041	Sufficient	<0.001
Iliac Atherosclerosis	>52.1	56.6%	75.1%	0.694 ± 0.029	Sufficient	<0.001
Aorta Atherosclerosis	>52.67	57.7%	74.3%	0.690 ± 0.030	Sufficient	<0.001
Pancreatic Steatosis	>51.55	60.9%	85.4%	0.778 ± 0.026	Good	<0.001
Hepatosteatois	>56.54	71.4%	76.6%	0.802 ± 0.027	Very Good	<0.001
V/S Ratio						
Gender (Male)	>0.5526	76.4%	72.3%	0.795 ± 0.026	Good	<0.001
Hypertension	>0.5387	78.5%	50.9%	0.670 ± 0.031	Sufficient	<0.001
Diabetes Mellitus	>0.6848	75.5%	63.4%	0.734 ± 0.036	Good	<0.001
Dyslipidemia	>0.5109	87.8%	42.1%	0.663 ± 0.039	Sufficient	<0.001
Iliac Atherosclerosis	>0.6821	58.6%	69.2%	0.678 ± 0.029	Sufficient	<0.001
Aorta Atherosclerosis	>0.6927	60.0%	70.2%	0.696 ± 0.029	Sufficient	<0.001
Pancreatic Steatosis	>0.5079	77.7%	59.1%	0.729 ± 0.029	Good	<0.001
Hepatosteatois	>0.5571	85.7%	52.9%	0.698 ± 0.031	Sufficient	<0.001

AUC: area under receiver operating characteristic (ROC) curve, SE: standard error, SAT: subcutaneous adipose tissue, V/S: visceral-to-subcutaneous fat volume ratio, VAT: visceral adipose tissue

Table 3. Prediction of the causality between visceral adipose tissue volume and independent variables by boosting linear regression models

Dependent Variable; VAT Volume (cm ³)	Model 1 (Accuracy: 60%)		Model 2 (Accuracy: 59.1%)		Model 3 (Accuracy: 59.1%)	
	Included	Importance	Included	Importance	Included	Importance
Gender	+	6.0%	+	8.0%	+	8.0%
Hypertension	+	7.0%	+	10.0%	+	10.0%
Diabetes Mellitus	+	7.0%	+	10.0%	+	10.0%
Dyslipidemia	+	2.0%	+	3.0%	+	3.0%
Iliac Atherosclerosis	+	5.0%	+	8.0%	+	7.0%
Aortic Atherosclerosis	+	5.0%	+	1.0%	+	6.0%
Pancreatic Steatosis	+	9.0%	-	-	-	-
Hepatosteatorsis	+	14.0%	-	-	-	-
Age	+	7.0%	+	11.0%	+	10.0%
Muscle Volume (cm ³)	+	10.0%	+	13.0%	+	12.0%
Liver Attenuation (HU)	+	17.0%	-	-	-	-
Pancreas Attenuation (HU)	+	10.0%	-	-	-	-
Spleen Attenuation (HU)	+	1.0%	-	-	-	-
L/S	-	-	+	22.0%	-	-
L-S	-	-	-	-	+	21.0%
P/S	-	-	+	14.0%	-	-
P-S	-	-	-	-	+	13.0%

Ensemble learning method: boosting linear regression, model selection method: best subset (criteria: AICC).

HU: Hounsfield unit, L: liver attenuation, P: pancreas attenuation, S: spleen attenuation, VAT: visceral adipose tissue

Table 4. Prediction of the causality between visceral-to-subcutaneous fat ratio and independent variables by boosting linear regression models

Dependent Variable: V/S Ratio	Model 1 (Accuracy: 43.6%)		Model 2 (Accuracy: 43.4%)		Model 3 (Accuracy: 43.4%)	
	Included	Importance	Included	Importance	Included	Importance
Gender	+	19.0%	+	21.0%	+	20.0%
Hypertension	+	6.0%	+	7.0%	+	7.0%
Diabetes Mellitus	+	6.0%	+	8.0%	+	8.0%
Dyslipidemia	+	2.0%	+	2.0%	+	2.0%
Iliac Atherosclerosis	+	8.0%	+	10.0%	+	10.0%
Aortic Atherosclerosis	+	8.0%	+	9.0%	+	9.0%
Pancreatic Steatosis	+	9.0%	-	-	-	-
Hepatosteatorsis	+	6.0%	-	-	-	-
Age	+	14.0%	+	18.0%	+	18.0%
Muscle Volume (cm ³)	+	6.0%	+	7.0%	+	7.0%
Liver Attenuation (HU)	+	7.0%	-	-	-	-
Pancreas Attenuation (HU)	+	7.0%	-	-	-	-
Spleen Attenuation (HU)	+	2.0%	-	-	-	-
L/S	-	-	+	9.0%	-	-
L-S	-	-	-	-	+	9.0%
P/S	-	-	+	9.0%	-	-
P-S	-	-	-	-	+	10.0%

Ensemble learning method: boosting linear regression, model selection method: best subset (criteria: AICC).

HU: Hounsfield unit, L: liver attenuation, P: pancreas attenuation, S: spleen attenuation, V/S: visceral-to-subcutaneous fat volume ratio

V/S ratio cut-off values for adequate prediction of hypertension, dyslipidemia, iliac atherosclerosis, aortic atherosclerosis, and hepatosteatosi were >0.5387 , >0.5109 , >0.6821 , >0.6927 , and >0.5571 respectively (p values <0.001). V/S ratio cut-off values for good prediction of male gender, diabetes mellitus, and pancreatic steatosis were >0.5526 , >0.6848 , and >0.5079 respectively (p values <0.001).

Boosting Linear Regression Analysis

Boosting linear regression models were created to determine and predict the causality between dependent (VAT and V/S ratio) and independent variables (age, gender, comorbidities and abdominal CT measurements) and results are given in Table 3 and Table 4.

Boosting linear regression analysis adjusted in model 1 showed all independent variables are related to VAT volume with 60% accuracy. Liver attenuation showed the highest importance (17%). In model 2, pancreatic steatosis and hepatosteatosi variables were replaced with liver attenuation (HU)/spleen attenuation (HU) ratio and pancreas attenuation (HU)/spleen attenuation (HU) ratio. In model 3 pancreatic steatosis and hepatosteatosi variables were replaced with liver attenuation (HU) – spleen attenuation (HU) and pancreas attenuation (HU) – spleen attenuation (HU) values. Both in model 2 and model 3 all independent variables are related to VAT volume with 59.1% accuracy (Table 3).

Similarly, boosting linear regression analysis adjusted in model 1 showed all independent variables are related to V/S ratio with 43.6% accuracy. Age showed the highest importance (19%). In model 2, pancreatic steatosis and hepatosteatosi variables were replaced with liver attenuation (HU)/spleen attenuation (HU) ratio and pancreas attenuation (HU)/spleen attenuation (HU) ratio. In model 3 pancreatic steatosis and hepatosteatosi variables were replaced with liver attenuation (HU) – spleen attenuation (HU) and pancreas attenuation (HU) – spleen attenuation (HU) values. Both in model 2 and model 3 all independent variables are related to V/S ratio with 43.4% accuracy (Table 4).

DISCUSSION

We found that VAT volume and V/S ratio are related to metabolic syndrome components (hypertension, diabetes mellitus, and dyslipidemia) as well as metabolic syndrome markers on abdominal CT, which are; hepatosteatosi, pancreatic steatosis, and aortoiliac atherosclerosis. On the other hand, SAT volume showed a significant correlation with some of these parameters, fewer compared to VAT volume and V/S ratio. VAT volume and V/S ratio could be used to predict hypertension, diabetes mellitus, and dyslipidemia with confidence. Specifically, VAT volume predicted hepatosteatosi with a very good diagnostic accuracy.

Our results are compatible with other studies in the literature regarding VAT and SAT are associated with cardiometabolic risk factors, with a stronger correlation reported for VAT (4,5,15,16). In addition to validating the

current literature, we have provided cut-off values for SAT volume, VAT volume, and V/S ratio for predicting hypertension, diabetes mellitus, and dyslipidemia in various diagnostic accuracy levels. These cut-off values could be utilized for reporting elevated risk of metabolic syndrome. Besides detecting central obesity as an incidental finding, implementing a low-dose CT scan limited to L3-L5 levels to a future diagnostic algorithm would allow accurate quantification of central obesity in risk groups.

Previous studies in literature showed that increased VAT volume is associated with hypertension (5,15,17,18), diabetes mellitus, prediabetes or insulin resistance (4,16,19), and dyslipidemia (4,16). In our study, VAT volume is a good predictor of hypertension and diabetes and could sufficiently predict dyslipidemia. On the other hand, the V/S ratio is a good predictor of diabetes mellitus and could sufficiently predict hypertension and dyslipidemia. Therefore, rather than the V/S ratio, we recommend VAT volume to predict clinical metabolic syndrome.

Both VAT volume and V/S ratio predicted pancreatic steatosis with good diagnostic accuracy and aortoiliac atherosclerosis with sufficient diagnostic accuracy. VAT volume performed superiorly while predicting hepatosteatosi. These results are also in line with previous studies (9,11,12).

Additionally, VAT volume was a good predictor of male gender in a lower cut-off value than clinical manifestations of the metabolic syndrome. This could indicate that increased VAT could account for the higher incidence of cardiovascular diseases among men (20).

Here, SAT volume showed a strong correlation to pancreatic steatosis and hepatosteatosi, a weaker correlation to hypertension and iliac atherosclerosis, and no correlation to diabetes mellitus, dyslipidemia, and aortic atherosclerosis. On the other hand, VAT volume and V/S ratio showed correlation to all of these parameters. These results are in line with previous studies from the literature (4,5,12). Increased SAT volume is reported to be associated with metabolic syndrome in non-obese subjects; on the other hand, no such association was reported in obese patients (15,21). This validates the argument that SAT is less contributing to the metabolic syndrome's pathophysiology but still important in early stages when the patient is still within normal BMI range.

LIMITATIONS

This study has some limitations. First, the current study's cross-sectional design is not appropriate to reveal the sequential relationship between central adiposity, abdominal CT findings, and clinical metabolic syndrome components. Second, the number of subjects is limited compared to some other studies in the literature. Also, the study cohort is comprised of patients with urolithiasis suspicion, which may be a limiting factor if there is an unknown relationship between urolithiasis and metabolic

syndrome or central obesity. Third, the presence of hypertension, diabetes mellitus, and dyslipidemia in the participants was acquired from the electronic medical charts. Up-to-date laboratory results and BMI values were not available due to the retrospective design of the study. Therefore, there may be subjects who have not yet been diagnosed.

CONCLUSION

In conclusion, this study showed that VAT volume and V/S ratio are related to clinical manifestations (hypertension, diabetes mellitus, and dyslipidemia) and abdominal CT markers (hepatosteatorosis, pancreatic steatorosis, and aortoiliac atherosclerosis) of the metabolic syndrome, with a more substantial relationship present with VAT. Reported cut-off values could be utilized to detect metabolic syndrome earlier, which would provide early lifestyle alterations aiming at a lesser fat percentage in body weight, leading to decreased morbidity and mortality rates.

Competing Interests: The authors declare that they have no competing interest.

Financial Disclosure: There are no financial supports.

Ethical Approval: Prior to the study, approval of the Institutional Ethics Review Board was obtained (project number: KA20/208, date: 01 June 2020).

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