



Body Imaging

Gastric wall fat halo sign in patients without intestinal disease

Ali Kupeli^{a,*}, Gurkan Danisan^a, Mehmet Kocak^a, Ismail Taskent^a, Isa Gokturk Balci^a, Eser Bulut^b^a Mus State Hospital, Department of Radiology, 49000 Mus, Turkey^b Trabzon Kanuni Education and Research Hospital, Department of Radiology, 61080 Trabzon, Turkey

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ABSTRACT

Purpose: To investigate the relationship between gastric wall fat halo sign and visceral obesity with potentially associated diseases.

Materials and methods: Between September 2015 and April 2017, 90 patients with gastric wall fat halo signs and 130 controls were prospectively evaluated. Patient height, weight, body mass index (BMI), sex, age, subcutaneous fat area (SFA), visceral fat area (VFA), total fat area (TFA), percentage of visceral fat (VF%) and the presence of colic or ileal fat halo signs, hepatic steatosis and aortic calcified plaques were recorded for the two groups. Cut-off values for the VFA, TFA, and VF% were determined and the diagnostic efficacy was calculated using receiver operating characteristic (ROC) curve analysis.

Results: No significant differences were found in age, BMI and SFA, but the VFA, VF%, TFA and frequencies of colic or ileal fat halo signs, hepatic steatosis and aortic calcified plaques were significantly higher in the patient group. The areas under the ROC curve (AUCs) were 0.803, 0.770 and 0.596 for VFA, VF% and TFA, respectively. The diagnostic efficacies of VFA and VF% were significantly higher than those of the TFA.

Conclusion: Gastric wall fat halo signs may be observed in overweight people, especially those with increased VFA and VF%. Additionally, these signs are usually observed along with fat halo signs of the colon or terminal ileum. However, extensive studies are needed to clarify the relationship between gastric wall fat halo signs and type 2 diabetes, cardiovascular diseases and metabolic syndrome.

1. Introduction

The fat halo sign is described as infiltration of the submucosa with fat, between the muscularis propria and the mucosa on abdominopelvic computed tomographic (CT) scans [1]. The fat halo sign is characterized by an inner (mucosa) layer, and an outer (muscularis propria and serosa) layer of soft-tissue attenuation with a fatty attenuation middle layer (submucosa) [2,3]. The fatty submucosa layer typically ranges between −10 and −64 Hounsfield units (HU) [4].

Fat halo signs were initially associated with CT scans of chronic inflammatory bowel disease patients [5–8]. Additionally, these signs suggest ulcerative colitis when the colon is affected and Crohn's disease when the fat halo sign is observed on the small intestine. The fat halo sign can be observed in patients who have undergone cytoreduction therapy and in graft versus host disease [9,10]. However, submucosal fat deposition may also be visible in the colon and small bowel of patients without gastrointestinal disease. Therefore, it is speculated that fat halo signs may be a normal finding related to obesity, especially visceral obesity [9].

Only a small number of studies have investigated fatty infiltration of

the submucosal layer of the gastric wall [9,10]. However, these studies have not clarified the relationship between gastric wall fat halo signs and other diseases or obesity. In this study, we aimed to investigate the relationship between gastric wall fat halo signs and visceral obesity as well as other potentially associated diseases.

2. Materials and methods

2.1. Study population

Between September 2015 and April 2017, among patients who underwent unenhanced abdominopelvic CT due to clinical suspicion of ureterorenal stones, 94 patients with gastric wall fat halo sign were prospectively recorded. The patients' CT images were retrospectively re-evaluated by two radiologists (A.K. 8 years experience and G.D. 5 years experience) to reach a consensus. Four patients were excluded from the study due to chronic inflammatory bowel disease. Therefore, 90 patients were included in the final study.

The control group comprised 130 patients from the clinical database who underwent unenhanced abdominopelvic CT due to clinical

* Corresponding author.

E-mail address: dr.ali_3383@hotmail.com (A. Kupeli).

suspicion of ureterorenal stones and had no evidence of a gastric wall fat halo sign. Age, sex, body weight (kg) and height (m) were recorded for the patient and control groups. Additionally, the body mass index (BMI) was calculated as weight (kg) divided by the square of the height (m) for both groups. The study was approved by the Institutional Ethics Committee.

2.2. CT protocol

All CT examinations were performed using 16 slices (Toshiba Aquilion, Toshiba Medical Systems, Japan) with axial 2-mm thick reconstructed images from the diaphragm to the pubic symphysis. All patients were examined using the standard scanning protocol without intravenous contrast due to a clinical suspicion of ureterorenal stones. None of the patients received oral contrast agents. The CT protocol was as follows: 120 kVp, tube current of 150–165 mAs, maximum 2.5 mm collimation, slice thickness of 2 mm and 0.5 s rotation time. The images were reconstructed into multiplanar reformations.

2.3. Analysis of CT images

The CT images were re-evaluated by two radiologists to reach a consensus. A gastric wall fat halo sign diagnosis was defined as linear fat accumulation in the submucosal layer of the gastric wall with attenuation of < -10 HU (Fig. 1). The specific part of the gastric wall where the fat halo sign was observed was recorded. These sites included the fundus, greater curvature, lesser curvature, antropyloric junction and diffuse areas. The presence of a fat halo sign on the colon wall or small intestine was also recorded.

The cross-sectional abdominal visceral (VFA) and subcutaneous fat areas (SFA) were evaluated by CT using commercially available software (Aquilion, Toshiba Medical Systems) at the umbilical level. The VFA and SFA were measured by setting the attenuation values for a region of interest between -150 and -30 HU. The intra-abdominal fat tissue between the parietal peritoneum or the transversalis fascia, not including the vertebral column and paraspinal muscles, was defined as the VFA. The area between the dermis and the abdominal fascia was defined as the subcutaneous fat area. The total fat area

(TFA = SFA + VFA) and the percentage of visceral fat (VF% = VFA / TFA \times 100) were obtained simultaneously.

The attenuation of the right hepatic lobe was measured using a standard ROI of approximately 200 mm², avoiding the biliary, vascular, and extrahepatic structures and focal liver lesions. Hepatic steatosis was then defined as a liver attenuation value of 40 HU or less [11–13].

Additionally, the presence of ureterorenal stones or calcified atherosclerotic plaques in the abdominal aorta was recorded from the aortic hiatus to the point of iliac bifurcation.

2.4. Statistical analyses

Data analyses were conducted using MedCalc Statistical Software version 16.8 (MedCalc Software bvba, Ostend, Belgium) and SPSS 13.0 Statistical Software (SPSS Inc., Chicago, IL, USA). Descriptive statistics, including the means and ranges, were calculated for the age, BMI, SFA, VFA, TFA, and VF% in the patient and control groups. Normal distributions were verified using the Kolmogorov-Smirnov test. The non-parametric Mann-Whitney *U* test and the parametric Student's *t*-test were used to compare the CT findings between groups. The Mann-Whitney *U* test was used to analyze BMI. Student's *t*-tests were used to analyze the age, SFA, VFA, TFA and VF%. Additionally, Chi-squared tests were used to examine differences in the frequencies of ureterorenal stones, colonic-ileal fat halo signs, hepatosteatosi, and aortic wall calcified plaques between groups. Also, 56 cases from patient group and 57 cases from control group with similar VFA were selected to determine the relationship between gastric wall fat halo sign and ureterorenal stones, aortic wall calcified plaques, hepatosteatosi. Then, chi-squared tests were performed again to determine differences in the frequencies of ureterorenal stones, hepatosteatosi, and aortic wall calcified plaques between these new groups. Optimal cut-off points for the VFA, TFA, VF% were determined via receiver operating characteristic (ROC) curve analysis. If the obtained value was less than a given cut-off value, the patient was classified as not having a gastric wall fat halo sign. If the value was above the cut-off value, the patient was considered to have a gastric wall fat halo sign. The sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV) and accuracy of these parameters were calculated. The area

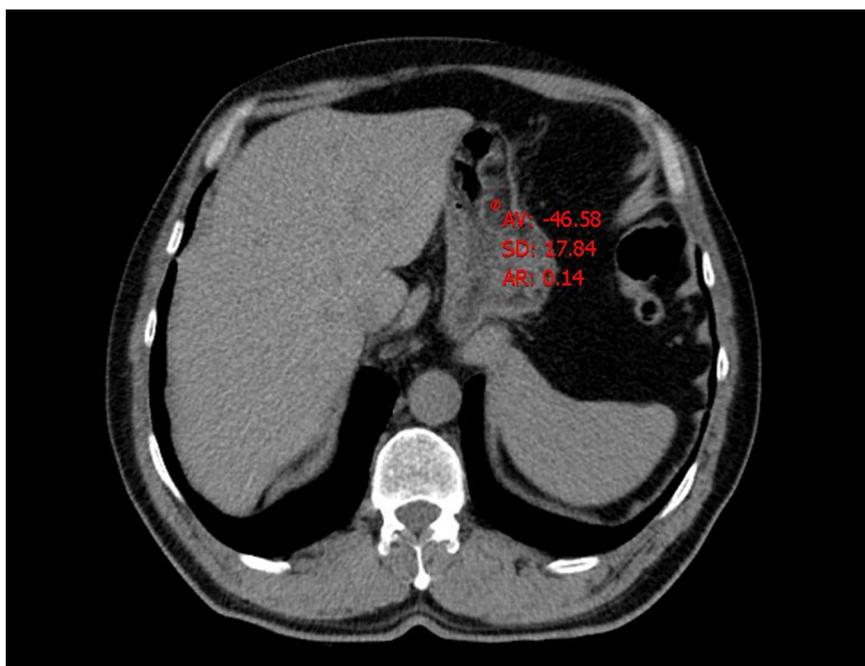


Fig. 1. 47 year-old man with renal colic. Unenhanced abdominal CT image shows thin linear fatty infiltration in submucosal layer of gastric wall with attenuation value < -10 HU.

beneath the fitted binormal ROC curve (AUC) was used to determine diagnostic efficacy. The AUC values were calculated and compared for selected parameters. Finally, odds ratios (ORs) were calculated in the univariate analysis. A p value < 0.05 indicated statistical significance.

3. Results

In the present study, 90 patients (55 male, 35 female) with gastric wall fat halo signs and 130 control patients (67 male, 63 female) were evaluated. The mean ages of the patient and control groups were 51.7 ± 14 and 49.5 ± 13 years, respectively. The mean height (m) and body weight (kg) of the patient and control groups were 1.67 ± 0.1 m and 1.68 ± 0.1 m and 83.3 ± 12.3 kg and 85.4 ± 10.6 kg, respectively. The mean BMI (kg/m^2) values of the patient and control groups were 30.1 ± 4.4 and 29.6 ± 3.8 , respectively. No statistically significant differences were observed in the mean height, body weight or BMI between the two groups ($p > 0.172$).

The mean SFA, VFA, TFA, and VF% values in the patient and control groups were as follows: SFA: 304.4 cm^2 and 312.1 cm^2 ; VFA: 208.7 cm^2 and 143.5 cm^2 ; TFA: 512.7 cm^2 and 455.6 cm^2 and VF%: 41 and 32, respectively. No significant difference was observed in the SFA ($p = 0.952$). However, significant differences were detected in the VFA, TFA, and VF% between the two groups ($p < 0.003$) (Figs. 2, 3) (Table 1).

The ROC curves for the VFA, VF% and TFA are shown in Fig. 4. The AUCs were 0.783, 0.770 and 0.596 for the VFA, VF% and TFA, respectively. The AUCs for the VFA and VF% were significantly higher than the AUC for the TFA ($p < 0.002$). The optimal cut-off values derived from the ROC analysis that provided the highest sensitivity and specificity for the VFA, VF% and TFA were 167.1 cm^2 , 35 and 475.9 cm^2 , respectively. The diagnostic values obtained with these cut-off values are presented in Table 2. The highest diagnostic values obtained for the VFA were 75.6% sensitivity, 73.1% specificity and 70.8% accuracy.

Gastric wall fat halo signs were most frequently observed in the greater curvature (96.7%), followed by the fundus (74.7%), lesser curvature (63.7%) and antropyloric region (29.7%). Additionally, diffuse fat halo signs were detected in 24.2% of cases.

Values regarding the presence of colonic-ileal fat halo signs,

hepatosteatorosis, ureterorenal stones and aortic wall calcified plaques are shown in Table 1. Also, the frequencies of ureterorenal stones, aortic wall calcified plaques, and hepatosteatorosis in patient and control groups with similar VFA are shown in Table 3.

The OR analyses revealed that the presence of a fat halo sign in the terminal ileum (OR 12.11), VFA (OR 7.39), and VF% (OR 7.06) was associated with the presence of a gastric wall fat halo sign (Table 4).

4. Discussion

In the present study, we found that gastric wall fat halo signs are strongly associated with visceral obesity. Among the VFA, VF% and TFA, the VFA had the greatest predictive value and provided 70.8% accuracy. We also found that a fat halo sign in the terminal ileum had an OR of 12.11 and was the most strongly associated factor for the presence of a gastric wall fat halo sign.

Only a limited number of studies have investigated fat halo signs in the gastrointestinal system [4,6,9,10,14,15]. To the best of our knowledge, this is the first prospective study concerning the relationship between gastric wall fat halo signs and visceral obesity.

While the presence of a fat halo sign in the gastrointestinal system has been found to be an indication of inflammatory bowel disease, it can also be observed in healthy people and may be related to obesity [5–8]. Harisinghani et al. showed that fat halo signs were visible on the CT examinations of 21% of the patients they examined [4]. Gervaise et al. also noted that fat halo signs in the gastric wall could be detected in patients without gastrointestinal disease [10]. In the present study, we included patients who underwent unenhanced abdominopelvic CT scans due to clinical suspicion of ureterorenal stones, which helped reduce the possibility of including patients with chronic inflammatory bowel diseases. Three patients with known Crohn's disease and one patient with ulcerative colitis were excluded from the study.

Gervaise et al. reported that the greater curvature was the part of the stomach most often affected by infiltrated fat. They also suggested that fat halo signs can occur at more than one site on the gastric wall [10]. We found that gastric wall fat halo signs were observed in the greater curvature, fundus, lesser curvature and antropyloric region. While Gervaise et al. found diffuse fat halo signs in 48% of patients, we detected diffuse fat halo signs in 24.2% of patients [10]. Pickhardt et al.

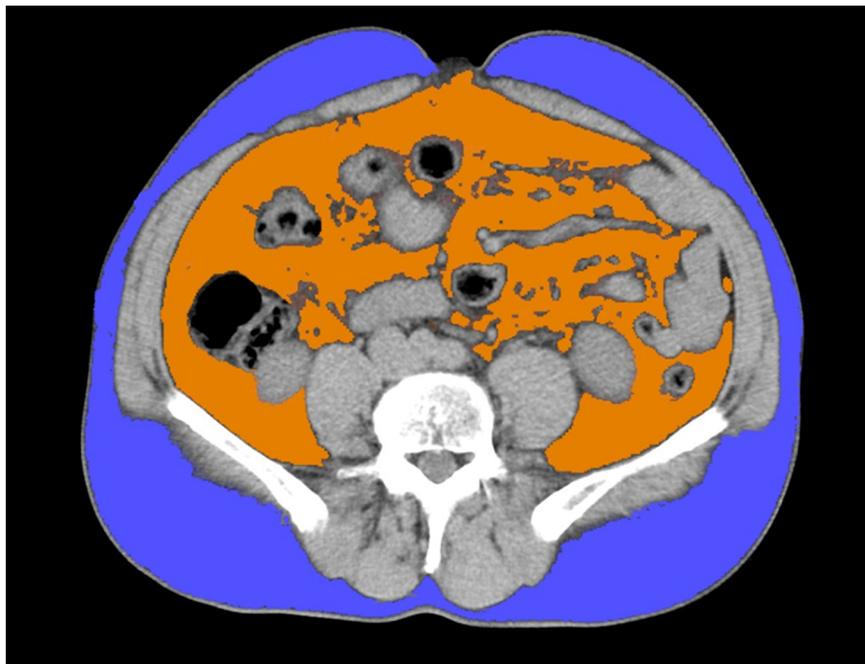


Fig. 2. 44-year-old woman with gastric wall fat halo sign shows increased VFA/TFA.

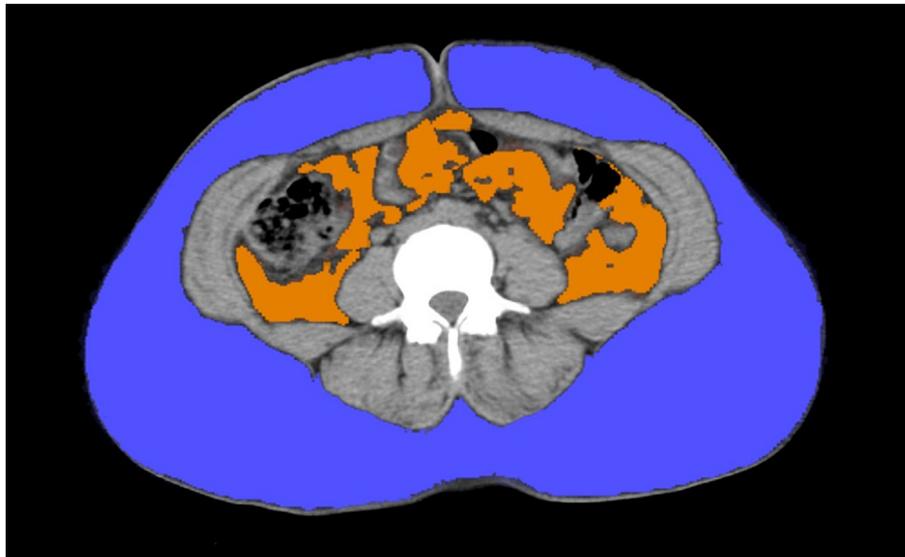


Fig. 3. 38 year-old woman without gastric wall fat halo sign shows decreased VFA/TFA.

reported that gastric wall fat halo signs were detected only on the antral part of the gastric wall [9]. Similar to Gervaise et al., we performed CT examinations without an intravenous contrast agent or gastric preparation [10]. This facilitated the assessment of fat density in the gastric wall.

This study found that gastric wall fat halo signs were more common among men than women, which is consistent with the results of Harisinghani et al. and Gervaise et al. [4,10].

Gervaise et al. showed significant differences in the mean height, body weight and BMI values between patients and controls [10]. A strong correlation exists between BMI and body fat accumulation. Therefore, we selected a control group without significant differences in

mean height, body weight or BMI values from the patient group [16].

Consistent with the findings of Gervaise et al., patients with gastric wall fat halo signs had significantly higher VFA, TFA, and VF% values than the control group, although no significant difference in the SFA was found between the two groups in our study [10]. However, Gervaise et al. did not evaluate the VF% and SFA in their study. The TFA exhibited a significantly lower AUC than the VFA and VF%. Although the VFA exhibited the highest AUC among the variables examined, no significant difference was observed between the AUCs of the VFA and VF%.

Previous studies have shown that fat halo signs can be observed in more than one segment of the gastrointestinal system [4,10]. Of the 90

Table 1
Patient characteristics.

	Total	Gastric wall fat halo sign		p value
		Present	Absent	
Number	220	90	130	
Age (year)	50.4 ± 14 (23–90)	51.7 ± 14 (27–90)	49.5 ± 13 (23–85)	0.245
Sex				
Men	122	55	67	
Women	98	35	63	
Height (cm)	167.4 ± 8.7 (150–195)	169.9 ± 8.1 (150–190)	166.6 ± 8.7 (150–195)	0.029
Weight (kg)	83.4 ± 11.9 (53–140)	85.3 ± 10.6 (62–110)	83.3 ± 12.2 (53–140)	0.327
Body mass index	29.9 ± 4.3 (20.7–54.6)	29.6 ± 3.8 (21.2–39.3)	30 ± 4.4 (20.7–54.6)	0.575
Hepatosteatois				< 0.001
Present	76	57	31	
Absent	144	33	99	
Ureterorenal stone				0.02
Present	80	41	39	
Absent	140	49	91	
Fat halo sign of colon				< 0.001
Present	88	57	31	
Absent	132	33	99	
Fat halo sign of ileum				< 0.001
Present	29	25	4	
Absent	191	65	126	
Aortic calcified plaque				0.002
Present	68	38	30	
Absent	152	52	100	
VFA (cm ²)	170 ± 64.6 (47–450.9)	208 ± 62(98.2–450.9)	143.5 ± 51.9 (47–327)	< 0.001
VF%	35 ± 1 (16–63)	41 ± 8 (25–63)	32 ± 8 (16–56)	< 0.001
SFA (cm ²)	308.8 ± 111 (104–694)	304 ± 115 (136–694)	312.1 ± 107 (104–637)	0.952
TFA (cm ²)	478 ± 139 (152–938)	512 ± 146 (243–938)	455 ± 130 (152–849)	0.003

VFA: visceral fat area; VF%: percentage of visceral fat; SFA: subcutaneous fat area; TFA: total fat area.

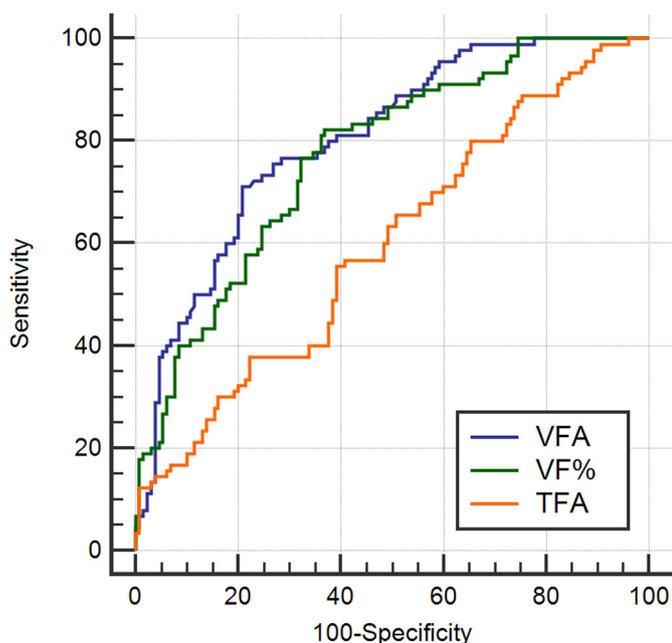


Fig. 4. Graph shows receiver operating characteristic curve for vascular VFA, TFA, VF%.

patients with gastric wall fat halo signs included in this study, 57 had a fat halo sign in the colon, and 25 had a fat halo sign in the ileum. However, no fat halo signs were observed in either the ileum or the colon in 30 patients. Of the 130 patients in the control group, 31 had a fat halo sign in the colon, and 4 had a fat halo sign in the ileum. No fat halo signs were present in either the ileum or the colon in 97 of the control patients. Fat halo signs in the ileum and colon were observed significantly more often in the patient group. The presence of a fat halo sign in the terminal ileum (OR 12.11), VFA (OR 7.39) and VF% (OR 7.06) were the factors most strongly associated with the presence of a gastric wall fat halo sign.

In our study, the prevalence of ureterorenal stones was significantly higher in patients with gastric wall fat halo signs than in control patients. Forty-one patients with gastric wall fat halo signs and 39 patients without gastric wall fat halo signs suffered from ureterorenal stones in the present study. Akarken et al. found that visceral obesity was an independent risk factor for urolithiasis, especially for uric acid kidney stones [17]. Similarly, we found that the VFA, VF% and TFA were higher in the patient group than in the control group.

In this study, patients with gastric wall fat halo signs had a significantly greater amount of calcified plaques on the aortic wall than patients without gastric wall fat halo signs. Previous studies have reported that aortic wall calcified plaques and VFA are significantly related [18]. We found that patients with gastric wall fat halo signs had hepatosteatorosis more often than patients without these signs, which is consistent with the findings of Gervaise et al. and Harisinghani et al. [4,10].

Visceral obesity has been associated with several pathological conditions such as abnormal glucose and lipid metabolism, insulin resistance and increased cardiovascular disease. It is also a risk factor for

Table 2 Results of Receiver Operating Characteristic (ROC) Analysis for VFA, VF% and TFA.

	AUC	Cutoff level	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
VFA	0.803	167.1	75.6	73.1	66.0	81.2	70.8
VF%	0.770	35	77.7	65.3	60.9	81.0	67.3
TFA	0.596	475.9	56.6	59.2	49.0	66.4	55.2

AUC: area under curve; PPV: positive predictive value; NPV: negative predictive value; VFA: visceral fat area; VF%: percentage of visceral fat; TFA: total fat area.

Table 3 Patient characteristics according to similar VFA.

	Total	Gastric wall fat halo sign		p value
		Present	Absent	
Number	113	56	57	
VFA (cm ²)	187.8 ± 37.8 (98.2–327.0)	187.6 ± 37.9 (98.2–255.6)	188.1 ± 38.1 (150.4–327.0)	0.942
Hepatosteatorosis				0.019
Present	50	31	19	
Absent	63	25	38	
Ureterorenal stone				0.017
Present	45	29	16	
Absent	68	27	41	
Aortic calcified plaque				0.028
Present	45	28	17	
Absent	68	28	40	

VFA: visceral fat area.

Table 4 Odds ratio of the presence of gastric wall fat halo sign.

Variable	Odds ratio	95% CI
Male sex	1.43	0.82–2.47
Age > 45 y	1.63	0.93–2.84
Body mass index (> 29.1)	0.96	0.48–1.91
Hepatosteatorosis	3.19	1.79–5.68
Fat halo sign of colon	5.51	3.06–9.93
Fat halo sign of ileum	12.11	4.04–36.29
VFA (> 166 cm ²)	7.39	3.99–13.66
VF% (> 35)	7.06	3.76–13.24
SFA (> 287 cm ²)	0.60	0.35–1.03
TFA (> 434 cm ²)	1.59	0.90–2.79
Aortic calcified plaque	2.43	1.35–4.36
Ureterorenal stone	1.95	1.11–3.41

VFA: visceral fat area; VF%: percentage of visceral fat; SFA: subcutaneous fat area; TFA: total fat area.

some cancers and surgical complications [19]. As an active hormonal tissue, visceral fat tissue releases atherogenic factors that increase atherosclerosis. This can accelerate vascular aging and the consequent risk of cardiovascular diseases and aortic atherosclerotic plaques [19]. Additionally, the increased visceral fat tissue may be an underlying cause of dysfunctional subcutaneous adipose tissue, which can lead to ectopic and excessive fat deposition in the heart, liver, skeletal muscle, pancreas and gastrointestinal system. This ectopic and excessive fat deposition could cause hepatosteatorosis and may be the reason why fat halo signs occur in more than one area of the gastrointestinal system [20]. Although strong associations exist between visceral adiposity and hepatic steatorosis and metabolic syndrome, visceral adiposity and hepatic steatorosis are not diagnostic criteria for metabolic syndrome.

In our study, we found that patient group with gastric wall fat halo sign had significantly higher frequencies of ureterorenal stones, aortic wall calcified plaques and hepatosteatorosis than control group with similar VFA. We thought that it might be the result of excessive fat deposition in patient group. But we could not confirm this hypothesis with laboratory parameters such as lipid profile.

This study has several limitations. First, we evaluated the CT images based on a consensus and did not evaluate the inter- and intra-observer variability. Second, gastric wall fat halo signs were diagnosed only by the CT findings. None of the patients were given an endoscopic examination. Third, the sample size was also relatively small. Fourth, we did not have access to laboratory parameters such as lipid panels. Fifth, the adipose tissue area measurements were determined from a single CT section obtained at the umbilicus level instead of via a volume calculation.

In conclusion, fat halo signs have been related to chronic inflammatory bowel disease. Fat halo signs in the gastric wall often occur in overweight people, especially those with increased VFA and VF%. Additionally, gastric wall fat halo signs are usually observed along with fat halo signs in the colon or terminal ileum. However, further studies are required to clarify the relationships of gastric wall fat halo signs with type 2 diabetes, cardiovascular diseases and metabolic syndrome.

References

- [1] Wittenberg J, Harisinghani MG, Jhaveri K, Varghese J, Mueller PR. Algorithmic approach to CT diagnosis of the abnormal bowel wall. *Radiographics* 2002;22:1093–107.
- [2] Macari M, Balthazar EJ. CT of bowel wall thickening: significance and pitfalls of interpretation. *AJR Am J Roentgenol* 2001;176:1105–16.
- [3] Muldowney SM, Balfe DM, Hammerman A, Wick MR. “Acute” fat deposition in bowel wall submucosa: CT appearance. *J Comput Assist Tomogr* 1995;19:390–3.
- [4] Harisinghani MG, Wittenberg J, Lee W, Chen S, Gutierrez AL, Mueller PR. Bowel wall fat halo sign in patients without intestinal disease. *AJR Am J Roentgenol* 2003;181:781–4.
- [5] Gore RM, Balthazar EJ, Ghahremani GG, Miller FH. CT features of ulcerative colitis and Crohn's disease. *AJR* 1996;167:3–15.
- [6] Jones B, Fishman EK, Hamilton SR, Rubesin SE, Bayless TM, Cameron JC, et al. Submucosal accumulation of fat in inflammatory bowel disease: CT/pathologic correlation. *J Comput Assist Tomogr* 1986;10:759–63.
- [7] Philpotts LE, Heiken JP, Westcott MA, Gore RM. Colitis: use of CT findings in differential diagnosis. *Radiology* 1994;190:445–9.
- [8] Macari M, Balthazar EJ. CT of bowel wall thickening: significance and pitfalls of interpretation. *AJR* 2001;176:1105–16.
- [9] Pickhardt PJ, Asher DB. Wall thickening of the gastric antrum as a normal finding: multidetector CT with cadaveric comparison. *AJR* 2003;181:973–9.
- [10] Gervaise A, Naulet P, Gervaise-Henry C, Junca-Laplace C, Pernin M, Lapierre-Combes M. Gastric wall fatty infiltration in patients without overt gastrointestinal disease. *AJR Am J Roentgenol* 2016;206(4):734–9.
- [11] Park YS, Park SH, Lee SS, Kim DY, Shin YM, Lee W, et al. Biopsy-proven non-steatotic liver in adults: estimation of reference range for difference in attenuation between the liver and the spleen at nonenhanced CT. *Radiology* 2011;258:760–6.
- [12] Kodama Y, Ng CS, Wu TT, Ayers GD, Curley SA, Abdalla EK, et al. Comparison of CT methods for determining the fat content of the liver. *AJR* 2007;188:1307–12.
- [13] Park SH, Kim PN, Kim KW, Lee SW, Yoon SE, Park SW, et al. Macrovesicular hepatic steatosis in living liver donors: use of CT for quantitative and qualitative assessment. *Radiology* 2006;239:105–12.
- [14] Ahualli J. The fat halo sign. *Radiology* 2007;242:945–6.
- [15] Muldowney SM, Balfe DM, Hammerman A, Wick MR. Acute fat deposition in bowel wall submucosa: CT appearance. *J Comput Assist Tomogr* 1995;19:390–3.
- [16] Nuttall FQ. Body mass index: obesity, BMI, and health: a critical review. *Nutr Today* 2015;50(3):117–28.
- [17] Akarken I, Tarhan H, Ekin RG, Çakmak Ö, Koç G, İlbey YÖ, et al. Visceral obesity: a new risk factor for stone disease. *Can Urol Assoc J* 2015;9(11–12):795–9.
- [18] Efe D, Aygün F, Acar T, Yildiz M, Gemici K. Investigation of relation between visceral and subcutaneous abdominal fat volumes and calcified aortic plaques via multislice computed tomography. *Vascular* 2015;23(4):396–402.
- [19] Shuster A, Patlas M, Pinthus JH, Mourtzakis M. The clinical importance of visceral adiposity: a critical review of methods for visceral adipose tissue analysis. *Br J Radiol* 2012;85(1009):1–10.
- [20] Tchernof A, Després JP. Pathophysiology of human visceral obesity: an update. *Physiol Rev* 2013;93(1):359–404.