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Associations of MRI-derived adrenal lipid content with hepatic and pancreatic steatosis and insulin resistance

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MAIN POINTS

- MRI-derived adrenal lipid indices show modest but significant associations with hepatic and pancreatic fat content.
- Higher adrenal lipid content is associated with increased ectopic fat accumulation in these organs.
- These relationships persist after adjustment for age and sex.
- No meaningful association was observed between adrenal lipid indices and the TyG index.
- These findings suggest that adrenal lipid content may reflect regional ectopic fat distribution rather than systemic insulin resistance.

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ABSTRACT

Aim: Adrenal adenomas are frequent incidental findings, and even non-functioning adrenal adenomas (NFAAs) may be linked to subtle metabolic alterations. According to the lipid overflow hypothesis, excess lipids accumulate in non-adipose organs, such as the liver, pancreas, and adrenal glands, potentially reflecting systemic metabolic stress. This study aimed to investigate the association between MRI-derived adrenal lipid indices and hepatic and pancreatic fat and to assess their correlation with the triglyceride-glucose (TyG) index as a marker of insulin resistance.

Materials and Methods: This retrospective study included 201 patients with lipid-rich adrenal adenomas [Adrenal Signal Intensity Index (ASII) $\geq$ 20%] identified on 1.5T MRI. Signal intensities were measured in adrenal lesions, liver, pancreas, and spleen using dual-echo sequences. ASII and adrenal-to-spleen chemical-shift imaging (CSI) ratios were calculated. Hepatic and pancreatic fat fractions were derived from signal intensity differences. The TyG index was computed from fasting triglyceride and glucose levels. Correlation and multivariable regression analyses were performed.

Results: Adrenal lipid indices showed significant correlations with hepatic (r = 0.32 and –0.30) and pancreatic fat fractions (r = 0.19 and –0.18) (p<0.01). These associations remained significant after adjustment for age and sex. However, no meaningful correlation was found between adrenal lipid indices and the TyG index; regression models explained only a minimal amount of the variation in TyG values (R^2 = 0.025).

Conclusion: MRI-derived adrenal lipid indices are associated with ectopic fat accumulation in the liver and pancreas but do not reflect systemic insulin resistance. These findings suggest that adrenal lipid content may serve as a marker of regional fat redistribution rather than global metabolic dysfunction.

Keywords: Adrenal adenoma, Chemical shift imaging, Ectopic fat, Hepatic steatosis, MRI, Insulin resistance

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INTRODUCTION

Adrenal adenomas are among the most commonly encountered incidental findings on abdominal imaging, and their detection has increased substantially with the widespread use of cross-sectional modalities such as CT and MRI [1,2]. Most of these lesions are classified as non-functioning adrenal adenomas (NFAAs), as they do not cause overt hormonal excess. However, recent studies have challenged the notion that NFAAs are metabolically inert, suggesting instead that they may be associated with subclinical metabolic alterations, including hypertension, insulin resistance, and dyslipidemia [3–5]. These associations have primarily been explored through laboratory-based assessments, while imaging-derived correlates remain largely under-investigated. This is consistent with the lipid overflow hypothesis, which suggests that ectopic fat accumulation in non-adipose organs, such as the adrenal glands, may reflect systemic metabolic burden rather than primary organ dysfunction [6]. This hypothesis posits that surplus lipids, unable to be stored in adipose tissue, accumulate in non-adipose organs such as the liver, pancreas, and

adrenal glands, potentially serving as indicators of systemic metabolic stress [6–8].

Magnetic resonance imaging (MRI), particularly chemical shift imaging (CSI), enables non-invasive, quantitative evaluation of intracellular lipid content in adrenal lesions. Quantitative metrics such as the adrenal signal intensity index (ASII) and adrenal-to-spleen CSI ratio are widely used to characterize lipid-rich adenomas in clinical practice [9–11]. These indices exploit the signal intensity differences between in-phase and opposed-phase sequences to detect microscopic intracellular lipid, a key imaging hallmark of benign adrenal cortical adenomas [10,11]. However, beyond lesion characterization, these indices may serve as potential imaging biomarkers that reflect systemic metabolic changes or ectopic fat accumulation, although this hypothesis has not been sufficiently tested.

Ectopic fat deposition in metabolically active organs, such as the liver and pancreas, plays a central role in the pathophysiology of insulin resistance and cardiometabolic risk. Hepatic steatosis—particularly in the form of non-alcoholic fatty liver disease (NAFLD)—has been well characterized and robustly linked to insulin resistance, type 2 diabetes, and cardiovascular disease [12,13]. The clinical relevance of pancreatic fat, on the other hand, remains more controversial, though emerging evidence suggests associations with beta-cell dysfunction, glucose dysregulation, and even pancreatic fibrosis [14–17].

Investigating whether adrenal lipid content, assessed by MRI, correlates with ectopic fat accumulation in these organs could provide new insights into metabolic imaging and its pathophysiology.

In addition, the extent to which adrenal lipid indices correlate with markers of systemic metabolic dysfunction remains unclear. The triglyceride-glucose (TyG) index has emerged as a simple, cost-effective, and reliable surrogate marker for insulin resistance. It has been validated against traditional methods such as the HOMA-IR and hyperinsulinemic-euglycemic clamp [18–20]. Despite its growing use in clinical and research settings, the relationship between TyG and adrenal lipid content has not been systematically examined.

This study aims to quantitatively evaluate associations of MRI-derived adrenal lipid indices with hepatic and pancreatic fat fractions and determine whether these indices correlate with systemic insulin resistance as measured by the TyG index. By addressing these questions, we seek to clarify whether adrenal lipid accumulation reflects localized metabolic alterations or broader systemic dysfunction.

MATERIALS AND METHODS

Study design and population

The Ethics Committee of the University of Health Sciences Ümraniye Training and Research Hospital approved this retrospective single-center study (Approval no: 2025/31, Date: 13.03.2025). Abdominal MRI studies performed between January 2014 and December 2023 were reviewed to identify patients with adrenal adenomas. Since this was a retrospective study and all data were anonymized, informed consent was not required. The case selection process is summarized in Figure 1.

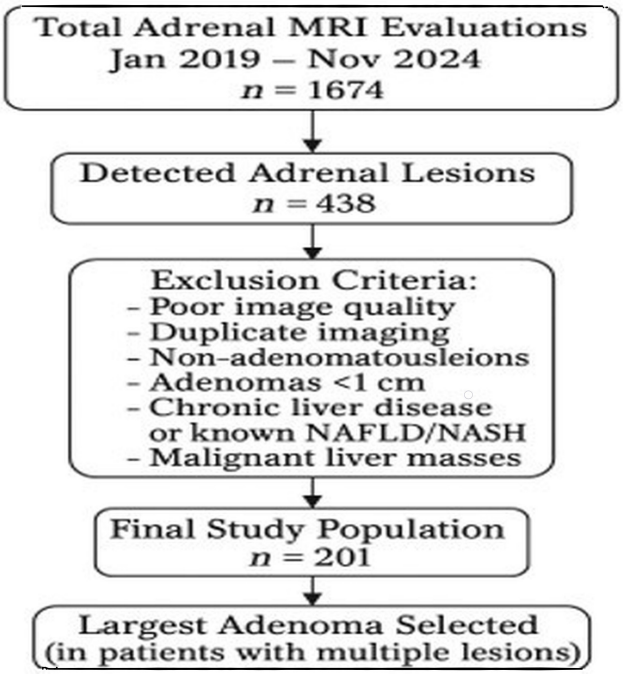

Figure 1. Flow diagram showing patient selection for the final study cohort. After exclusions, 201 patients with lipid-rich adrenal adenomas were included for analysis. In patients with multiple lesions, the largest adenoma was selected.

Inclusion criteria were: (1) radiologically confirmed adrenal adenoma based on MRI criteria, including signal drop on opposed-phase images and lesion size ≤4 cm; (2) availability of dual-echo chemical shift imaging sequences of diagnostic quality; and (3) laboratory data (fasting triglyceride and glucose levels) obtained within 7 days of MRI.

In this study, adrenal adenomas were diagnosed solely on the basis of imaging characteristics because histopathologic confirmation was not feasible, given the incidental nature of these lesions. Only lipid-rich adenomas—defined as lesions exhibiting significant intracellular fat content and an adrenal signal intensity index (ASII) greater than 20%—were included to ensure measurement reliability and biological homogeneity. This threshold was selected based on prior validation studies and reflects the sensitivity limit of chemical shift imaging for reliable fat quantification.

Lipid-poor adenomas (ASII < 20%) were excluded because they exhibit minimal signal drop on opposed-phase imaging, thereby reducing the accuracy of CSI-derived indices. Furthermore, lipid-poor lesions may exhibit distinct biological behavior, including an increased likelihood of subclinical cortisol secretion, which this study could not assess because hormonal profiling was not performed. Their inclusion would have introduced potential confounding effects and undermined the interpretability of adrenal lipid measurements.

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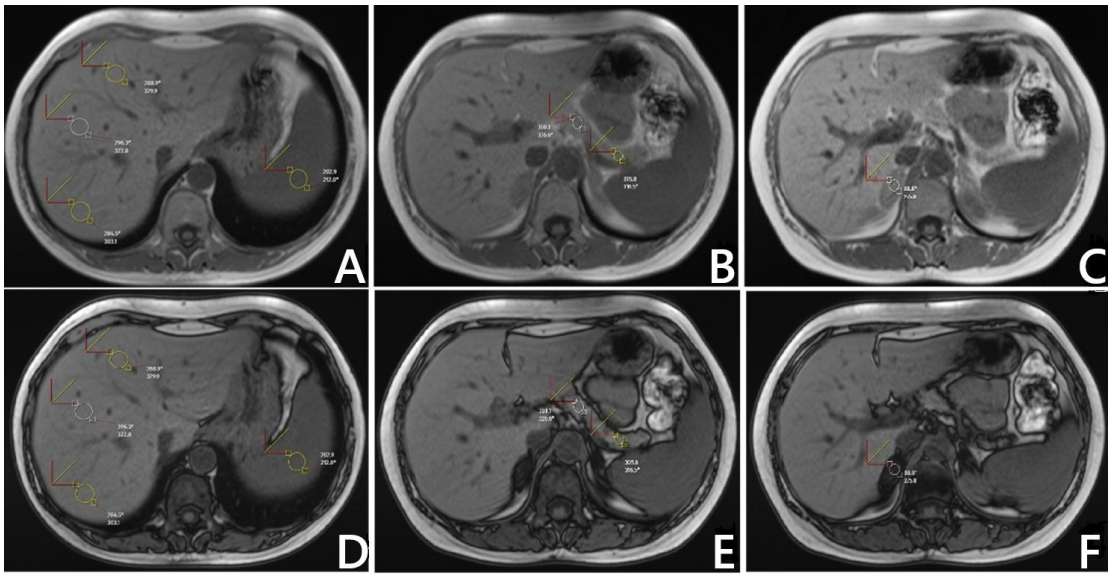

Figure 2. Axial gradient-echo T1-weighted chemical shift MRI images demonstrating ROI-based signal intensity measurements in representative cases. In-phase images are shown in panels A–C, and opposed-phase images in panels D–F. (A, D) illustrate ROI placements in the liver and spleen, (B, E) show measurements from the pancreatic head or body, and (C, F) display ROIs placed on the adrenal adenoma.

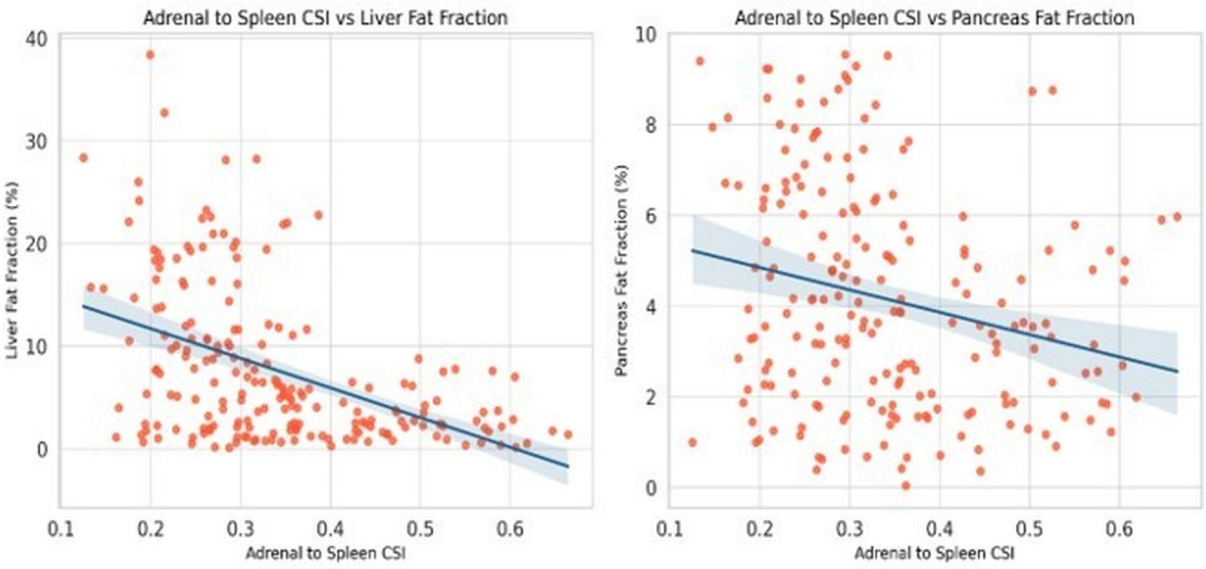

Figure 3. Correlation of adrenal to spleen CSI with liver and pancreas fat fractions.

Additional exclusion criteria were as follows: (1) known functional adrenal tumors (e.g., pheochromocytoma, cortisol-producing adenomas); (2) prior adrenal surgery or known adrenal malignancy; (3) chronic liver disease (defined as documented hepatitis, cirrhosis, or persistent liver enzyme elevation >3× upper limit of normal); (4) known NAFLD or NASH diagnosed by other modalities or by biopsy; (5) inadequate image quality; or (6) duplicate or follow-up MRI studies.

MRI acquisition and imaging analysis

All MRI examinations were performed using a 1.5T scanner (Optima MR450w, GE Healthcare). The standard abdominal MRI protocol included T2-weighted sequences, in-phase and opposed-phase dual-echo T1-weighted sequences, and diffusion-weighted imaging (DWI). Post-contrast imaging was not used for fat quantification. The dual-echo sequence parameters were as follows: TR = 165 ms; TE = 2.3/4.7 ms; slice thickness = 6 mm (Table 1).

Circular regions of interest (ROIs) with an area of 0.5–1 cm^2 were manually placed on the adrenal adenoma, liver parenchyma (segment VI), pancreatic head or body, and spleen. Care was taken to avoid visible vessels, lesions, or imaging artifacts. Three measurements were obtained for each organ and averaged for analysis (Figure 2A–F). Specifically,

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Table 1. MRI sequence parameters used in adrenal imaging.

Sequence	TR (ms)	TE (ms)	Flip Angle (°)	Slice Thickness (mm)	Comments
Coronal & Axial T2-weighted	1000	81.3	-	6	Localization and lesion morphology
Axial T1-weighted Dual Echo (CSI)	165	TE1: 2.3 TE2: 4.7	60	6	In-phase and opposed-phase imaging
Axial T2-weighted FS (FIESTA)	4.4	1.5	70	6	Fat suppression
Diffusion-weighted Imaging (DWI)	-	-	-	6	b-values: 50, 500, 1000 s/mm²
ADC Mapping	-	-	-	6	Derived from DWI
Axial Dynamic Contrast-Enhanced (LAVA-F)	6.0	Min Full	15	5	Phases: 0, 8, 15, 30 seconds post-injection

Table 2. Quantitative MRI and laboratory formulas used in the study.

Parameter	Formula	Purpose
Fat Fraction (%)	(SIIP – SIOP) / (2 × SIIP) × 100	Quantification of hepatic and pancreatic fat
Adrenal Signal Intensity Index (ASII)	100 × [(SIIP – SIOP) / SIIP]	Measurement of adrenal lipid content
Adrenal-to-Spleen CSI Ratio	Lesion-to spleen SIIP / lesion-to-spleen SIOP	Fat sensitivity in adrenal lesions
TyG Index	Ln (2 x Triglycerides (mg/dL) × Glucose (mg/dL))	Surrogate marker of insulin resistance

(SIIP: Signal intensity in phase, SIOP: Signal intensity out-of-phase). (where SIIP and SIOP represent signal intensities in in-phase and opposed-phase images, respectively).

Table 3. Baseline characteristics of patients with adrenal adenomas.

Characteristic	Number (%)
Number of patients	201
Gender	
Female	165 (82.1%)
Male	36 (17.9%)
Mean age ± SD years	53.4 ± 9.2
Laboratory Findings	
Triglyceride (Median -- IQR)	146 - 91
Fasting Plasma Glucose (Median -- IQR)	98 - 26
TYG Index (Mean ± SD)	4.82 ± 0.39
Volume of adenoma (cm3)	
Mean ± SD	6.3 ± 5.3
Median -- IQR	4.46 - 6.05
Range	0.6 -- 25.8
Localisation of adenoma	
Right	99 (49.3%)
Left	102 (50.7%)

ROIs for the liver and spleen were placed as shown in Figures 2A and 2D; ROIs for the pancreatic head or body were placed as shown in Figures 2B and 2E; and ROIs for adrenal adenomas were placed as shown in Figures 2C and 2F. All measurements were performed jointly by two radiologists (with 5 and 10 years' experience in abdominal MRI) and recorded by consensus.

Quantitative indices, including the adrenal signal intensity index (ASII), adrenal-to-spleen chemical shift ratio (CSI ratio), liver and pancreas fat fractions, and TyG index, were calculated using standardized formulas summarized in Table 2.

Statistical analysis

All statistical analyses were performed using SPSS v24. Scatter plots were generated for visualization purposes using external software based on SPSS-derived outputs. Continuous variables were reported as mean ± standard deviation or median (interquartile range), as appropriate. The Kolmogorov–Smirnov test was used to assess the normality of the data. Univariable associations between adrenal lipid indices (CSI ratio, ASII) and hepatic and pancreatic fat content or the TyG index were evaluated using Spearman's rank correlation.

To account for potential confounding factors, separate multivariable linear regression models were constructed for each dependent variable (hepatic fat fraction, pancreatic fat fraction, and TyG index). In these models, the independent variables were the adrenal-to-spleen CSI ratio (the representative imaging-derived adrenal lipid parameter), age, and sex. The ASII was excluded from the multivariable models because it is derived from the same signal-intensity measurements as the CSI ratio; including both would violate the assumption of independence among predictors.

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A two-tailed p-value < 0.05 was considered statistically significant. As this was a retrospective observational study, no a priori sample size or power calculation was performed; instead, the study population consisted of all eligible patients who met the predefined inclusion criteria during the study period.

RESULTS

The final analysis included 201 patients with lipid-rich adrenal adenomas (ASII ≥ 20%). The mean age was 53.4 ± 9.2 years (range: 30–75), and 82.1% were female (n = 165), while 17.9% were male (n = 36). Laboratory parameters showed median triglyceride and fasting plasma glucose levels of 146 mg/dL (IQR: 91) and 98 mg/dL (IQR: 26), respectively. The mean TyG index was 4.82 ± 0.39. The mean volume of adrenal adenomas was 6.3 ± 5.3 cm^3, with a median volume of 4.46 cm^3 (IQR: 6.05) and a range of 0.6 to 25.8 cm^3. Lesions were evenly distributed between the right (n = 99, 49.3%) and left (n = 102, 50.7%) adrenal glands (Table 3).

The median adrenal signal intensity index (ASII) was 68.9% (IQR: 16.5), and the adrenal-to-spleen CSI ratio was 0.316 (IQR: 0.167). The median hepatic fat fraction was 5.11% (IQR: 8.68), and the median pancreatic fat fraction was 3.67% (IQR: 3.94) (Table 4).

Descriptive and correlation analysis

Hepatic fat demonstrated wide interindividual variability, with 34.8% of patients exhibiting moderate to severe steatosis (fat fraction ≥ 5%). Pancreatic fat was more narrowly distributed, with 22.4% of individuals exceeding a 6% fat fraction.

Spearman correlation analysis revealed significant and directionally consistent associations between the adrenal-to-spleen CSI ratio and both hepatic and pancreatic fat fractions. Specifically, the CSI ratio was inversely correlated with hepatic fat ($r = -0.458$, $p<0.001$) and pancreatic fat ($r = -0.245$, $p<0.001$), indicating that lower adrenal CSI values (i.e., higher lipid content) were associated with increased ectopic fat accumulation in both organs (Figure 3). The TyG index did not correlate significantly with the CSI ratio ($r = -0.080$, $p = 0.258$) (Table 5).

Multivariable regression analysis

In multivariable linear regression analyses adjusting for age and sex, the adrenal-to-spleen CSI ratio demonstrated a statistically significant inverse association with both hepatic and pancreatic fat fractions. Specifically, lower CSI ratios, indicative of higher adrenal lipid content, were independently associated with increased hepatic fat accumulation ($\beta = -27.36$, $p<0.001$). The model explained approximately 25.3% of the variance in hepatic fat ($R^2 = 0.253$, $p<0.001$). A similar pattern was observed for pancreatic fat, where the CSI ratio again showed a strong inverse association ($\beta = -5.64$, $p<0.001$), with age emerging as a significant positive predictor ($\beta = 0.051$, $p=0.006$); this model accounted for 16.2% of the variance in pancreatic fat content ($R^2 = 0.162$, $p<0.001$).

When predictors of the TyG index were evaluated, the CSI ratio showed a small, marginally significant inverse association ($\beta = -1.97$, $p=0.043$). However, the overall model explained only a minimal proportion of the variance in the TyG index ($R^2 = 0.025$, $p=0.285$), indicating the limited explanatory value of adrenal lipid indices and demographic covariates (age and sex) for systemic insulin resistance (Table 6).

DISCUSSION

This study demonstrates that MRI-based adrenal lipid indices—specifically, the adrenal signal intensity index (ASII) and the adrenal-to-spleen chemical shift imaging (CSI) ratio—are independently associated with ectopic fat accumulation in the liver and pancreas, but not with systemic insulin resistance, as assessed by the triglyceride-glucose (TyG) index. These findings support the concept that adrenal lipid content may serve as an imaging correlate of localized ectopic fat redistribution rather than a marker of global metabolic dysfunction, particularly insulin resistance.

Several studies have reported that non-functioning adrenal adenomas (NFAAs), though traditionally considered hormonally silent, are associated with metabolic syndrome components such as hypertension, visceral adiposity, and impaired glucose metabolism [3–5,21]. While subclinical hypercortisolism was previously thought to drive this association, recent data suggest that metabolic alterations may occur independently of hormonal secretion [22,23]. Our findings align with this paradigm shift, suggesting that adrenal lipid accumulation may reflect systemic metabolic stress, rather than direct endocrine involvement.

The significant association between adrenal lipid content and hepatic steatosis observed in our study reinforces the "lipid overflow" hypothesis, which posits that excess lipids not stored in adipose tissue are deposited in non-adipose organs such as the liver, pancreas, and adrenal glands [6–8]. This ectopic fat redistribution is a hallmark of metabolic stress and has been strongly linked to insulin resistance, particularly in the liver [12,13]. Previous imaging studies, including a recent chemical shift MRI analysis, have reported a positive correlation between adrenal and hepatic fat content [24]. Our study adds to this evidence by confirming the relationship using multivariable regression models adjusted for age and sex, thereby strengthening the generalizability of this association.

MRI-based fat quantification using CSI offers advantages over CT attenuation in detecting intracellular lipid content [9–11, 25–27]. While CT has limited sensitivity for detecting microscopic fat, CSI enables non-invasive and reproducible quantification of fat in adrenal lesions. Our study employed established indices, such as the ASII and the adrenal-to-spleen CSI ratio, to ensure methodological reliability. Notably, lipid-poor adenomas (ASII < 20%) were excluded to avoid biolog-

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Table 4. Quantitative measurements of liver, pancreas, spleen, and adrenal lesions.

	In Phase (Median–IQR)	Out-of-Phase (Median–IQR)	Fat Fraction (Median -- IQR)	
Liver Measurement	292.65-115.72	250.49-116.24	5.11-8.68	
Pancreas Measurement	323.79-156.44	297.88-141.74	3.67-3.94	
Spleen Measurement	191.79-61.87	186.21-57.13		
			ASII (Median-IQR)	Adrenal/Spleen CSI Ratio (Median -- IQR)
Adrenal Lesion Measurement	273.48-84.76	89.65-53.52	68.9-16.5	0.316-0.167

Table 5. Correlation of Adrenal/Spleen CSI ratio, ASII, and TyG index with liver fat fraction, pancreatic fat fraction, and inter-parameter relationships.

		Adrenal/Spleen CSI ratio	ASII
Liver Fat Fraction	Spearman correlation coefficient	**-0.439**	**0.452**
	p-value	**p<0.01**	**p<0.01**
Pancreatic Fat Fraction	Spearman correlation coefficient	**-0.245**	**0.220**
	p-value	**p=0.01**	**p=0.02**
Tyg Index	Spearman correlation coefficient	-0.079	0.078
	p-value	p=0.265	P=0.273

Table 6. Multivariable linear regression analysis of CSI ratio, age, and sex with liver fat fraction, pancreatic fat fraction, and TyG index.

		Intercept	Adrenal/Spleen CSI ratio	Age	Sex
Liver Fat Fraction	β	19.17	**-27.36**	-0.037	-2.25
	SE	3.05	**4.02**	0.053	1.27
	95% CI	13.14 to 25.19	**-34.43 to -18.55**	-0.13 to 0.07	-5.04 to 0.02
	p-value	<0.001	**<0.001**	0.479	0.076
Pancreatic Fat Fraction	β	2.98	**-5.64**	**0.051**	1.04
	SE	1.03	**1.36**	**0.018**	0.431
	95% CI	0.94 to 5.02	**-7.74 to -2.38**	**0.02 to 0.09**	0.93 to 1.78
	p-value	0.004	**<0.001**	**0.006**	0.141
Tyg Index	β	4.81	-1.97	-0.001	-0.135
	SE	0.21	0.28	0.004	0.091
	95% CI	4.38 to 5.24	-2.23 to 0.12	-0.007 to 0.009	-0.27 to 0.08
	p-value	<0.001	0.043	0.973	0.863

ical heterogeneity and minimize the inclusion of potentially functional tumors [22,23].

The observed inverse relationship between adrenal lipid indices and pancreatic fat is notable. Although pancreatic fat is less well understood than hepatic steatosis, several studies have linked it to impaired insulin secretion, beta-cell dysfunction, and glucose dysregulation [12,13,15–17]. Our results suggest that adrenal lipid accumulation may parallel pancreatic fat deposition, implying a broader role for ectopic lipid redistribution across metabolically active organs. Whether this association reflects a shared pathogenic mechanism or is merely coincidental remains to be studied further.

In contrast to hepatic and pancreatic fat, adrenal lipid indices were not meaningfully associated with the TyG index. Although a marginally significant beta coefficient was observed in the regression analysis, the model demonstrated very low explanatory power (R^2 = 0.025), indicating limited clinical relevance. The temporal mismatch between static imaging-derived measures and dynamic metabolic indices may partly explain this discrepancy. While the TyG index is a validated surrogate marker of insulin resistance [18–20], it primarily reflects short-term metabolic fluctuations. It may not correspond to the slower, cumulative process of ectopic lipid deposition within tissues. Similar discrepancies between structural imaging biomarkers and functional metabolic markers have been reported previously [28].

Our findings underscore the need to interpret adrenal lipid content not as a marker of systemic insulin resistance, but rather as a regional indicator of ectopic fat burden, particularly in the liver and pancreas. The adrenal gland, due to its vascularity and lipid-handling capacity, may act as a passive depot for circulating lipids in individuals with excess visceral fat, in line with the lipid overflow hypothesis [6].

Limitations

This study has several limitations. The retrospective design limited access to comprehensive endocrine and anthropometric data, such as cortisol, insulin levels, BMI, and waist circumference. Consequently, the metabolic status of patients

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could not be fully characterized. Although we excluded individuals with known NAFLD or NASH to reduce confounding, this may have inadvertently introduced selection bias by excluding those with advanced hepatic fat accumulation. Furthermore, while the TyG index is practical and widely used, it does not accurately reflect organ-specific insulin sensitivity and may underestimate the complexity of metabolic relationships. Finally, although CSI-based fat quantification is reliable, can be affected by T2* decay or iron deposition. Newer techniques, such as proton density fat fraction (PDFF), may offer improved accuracy and cross-platform reproducibility, especially in longitudinal or multi-center studies [25,28].

CONCLUSION

In conclusion, MRI-derived adrenal lipid indices, particularly the adrenal-to-spleen CSI ratio and ASII, are associated with hepatic and pancreatic fat content but not with systemic insulin resistance as measured by the TyG index. These results suggest that adrenal lipid accumulation may reflect localized ectopic fat redistribution rather than generalized metabolic dysfunction. Future studies using advanced fat quantification methods and comprehensive metabolic profiling are warranted to clarify the potential role of adrenal fat content as an imaging biomarker of metabolic health.

Ethics Committee Approval: This study received approval from the Institutional Review Board of the University of Health Sciences, Ümraniye Training and Research Hospital (Approval no: 2025/31, Date: 13.03.2025).

Informed Consent: Informed consent was not obtained because of the study's retrospective design.

Peer-review: Externally peer-reviewed.

Conflict of Interest: The authors declare that they have no competing interests.

Author Contributions: Concept: Y.Y., S.N.E.; Design: Y.Y., S.N.E.; Supervision: Y.Y.; Materials: Y.Y., S.N.E., M.D., S.E.; Data Collection or Processing: Y.Y., S.N.E., M.D., S.E.; Analysis or Interpretation: Y.Y., M.D.; Literature Search: Y.Y., S.N.E., M.D., S.E.; Writing: Y.Y., S.N.E.; Critical Review: S.N.E., M.D., S.E.

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