

Ultrasound-detected hepatic steatosis in psoriatic disease: prevalence, cardiometabolic correlates, and biochemical discordance

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Abstract

Background

Hepatic steatosis is an important cardiometabolic manifestation of psoriatic disease, but routine aminotransferases may fail to identify affected patients.

Objective

To determine the frequency and clinical-cardiometabolic correlates of ultrasound-detected hepatic steatosis and examine biochemical discordance as a secondary objective.

Methods

This cross-sectional study included 572 psoriatic adults with or without psoriatic arthritis. Ultrasound data were available in 534 (93.4%). Sequential logistic models evaluated associations with steatosis, prioritizing waist circumference and using body mass index in sensitivity analysis. Secondary analyses examined steatosis with normal aminotransferases and gamma-glutamyl transferase (GGT).

Results

Hepatic steatosis was detected in 129/534 patients (24.2%). Waist circumference remained associated after cardiometabolic adjustment (odds ratio per 10 cm 1.34, 95% confidence interval 1.10–1.64), whereas psoriatic arthritis and severe skin disease were not. Among 408 patients with normal aspartate and alanine aminotransferases, 37 (9.1%) had steatosis. Elevated GGT identified 28/37 cases, with 75.7% sensitivity, 98.7% specificity, 84.8% positive predictive value and 97.6% negative predictive value.

Conclusion

Ultrasound-detected hepatic steatosis affected approximately one quarter of evaluable patients and was more closely associated with central adiposity and cardiometabolic factors than with articular or skin disease. Normal aminotransferases did not exclude steatosis; GGT warrants external evaluation as a secondary risk-enrichment marker.

Introduction

Psoriatic disease encompasses psoriasis and psoriatic arthritis (PsA) and is increasingly understood as a systemic inflammatory condition with substantial cardiometabolic burden. Obesity, insulin resistance, dyslipidemia and hypertension occur frequently across the psoriatic disease spectrum, reflecting an

interaction between traditional risk factors, altered adipose biology and chronic inflammation. Hepatic steatosis represents a clinically relevant organ-level manifestation of this immunometabolic phenotype [1–3].

Observational evidence indicates that patients with chronic plaque psoriasis have an increased frequency of steatotic liver disease and that the burden may be greater in those with PsA [4–6]. However, much of the available literature was developed under the former non-alcoholic fatty liver disease framework. The current steatotic liver disease nomenclature defines metabolic dysfunction-associated steatotic liver disease (MASLD) by hepatic steatosis together with at least one cardiometabolic criterion and recognizes a separate MetALD category for patients with metabolic dysfunction and alcohol exposure above specified thresholds [7, 8]. Retrospective application of these categories requires caution when alternative causes of steatosis have not been systematically adjudicated.

Conventional ultrasonography is imperfect for mild steatosis and cannot stage fibrosis, but remains widely used, accessible and suitable for detecting established steatosis in clinical and epidemiological settings [9, 10]. Within this setting, it is important to establish the relative contributions of central adiposity, overall adiposity, metabolic abnormalities, alcohol exposure and psoriatic disease phenotype. Serum aminotransferases reflect hepatocellular injury rather than fat accumulation and may remain normal in patients with steatosis, creating a clinically relevant secondary question.

We therefore evaluated the frequency of ultrasound-detected hepatic steatosis in a large real-world psoriatic disease cohort, examined its clinical and cardiometabolic correlates using sequential models, and characterized contemporary SLD-compatible phenotypes. As prespecified secondary analyses, we examined the subgroup with normal AST and ALT and evaluated whether GGT, a marker linked to hepatobiliary injury, alcohol exposure, oxidative stress and metabolic dysfunction [11–14], provided additional risk discrimination within that subgroup.

Methods

Study design and population

We conducted a cross-sectional observational study of consecutive adult patients with psoriatic disease evaluated in routine clinical practice at a tertiary referral center. Patients had dermatologist- or rheumatologist-confirmed psoriasis; PsA was defined as physician-confirmed inflammatory arthritis fulfilling the Classification Criteria for Psoriatic Arthritis (CASPAR) [15]. The source cohort comprised 572 participants.

The study was approved by the Research Ethics Committee of the Principality of Asturias, Oviedo, Spain (reference 2020.423) and was conducted in accordance with the Declaration of Helsinki. All participants provided informed consent for the use of anonymized clinical data.

Clinical, anthropometric and laboratory variables

Demographic characteristics, psoriasis duration, PsA status, nail involvement, systemic treatment and PASI were recorded. Severe skin involvement was represented by PASI ≥ 10 . BMI was calculated as weight in kilograms divided by height in meters squared. Waist circumference was measured at the midpoint between the lowest rib and the iliac crest with the patient standing, consistent with standardized clinical measurement principles [16]. Hypertension and diabetes were based on documented diagnoses and/or treatment. Dysglycemia was defined by fasting glucose ≥ 100 mg/dL or diabetes. Hypertriglyceridemia was defined as triglycerides ≥ 150 mg/dL; low HDL cholesterol was defined as < 40 mg/dL in men and < 50 mg/dL in women. The atherogenic lipid phenotype incorporated hypertriglyceridemia, low HDL cholesterol or documented dyslipidemia.

Alcohol was recorded as current consumption and, when available, as daily standard drink units (SDU; one SDU was considered approximately 10 g ethanol. Current alcohol consumption was used in the primary multivariable models because amount was incompletely recorded. Sex-specific MetALD-compatible thresholds were 2–5 SDU/day in women and 3–6 SDU/day in men, corresponding to the contemporary weekly thresholds.

Ultrasound and liver enzyme definitions

Abdominal ultrasonography was performed as part of routine clinical assessment in nearly all participants, irrespective of liver enzyme levels or clinical suspicion of liver disease. Ultrasound reports were used to classify hepatic steatosis as present or absent. Ultrasound data were available for 534/572 participants (93.4%); the outcome was not imputed when unavailable.

Sex-specific reference limits defined normal AST as ≤ 40 U/L in men and ≤ 34 U/L in women, normal ALT as ≤ 40 U/L in men and ≤ 35 U/L in women, and normal GGT as ≤ 60 U/L in men and ≤ 40 U/L in women. The normal-aminotransferase subgroup required both AST and ALT to be within these limits. GGT was also expressed relative to the corresponding sex-specific upper limit of normal (GGT/ULN).

Contemporary SLD-compatible classification

Among ultrasound-confirmed steatosis cases, MASLD-compatible status required at least one reconstructed cardiometabolic criterion and alcohol exposure below the MetALD threshold. MetALD-compatible status required a cardiometabolic criterion together with alcohol exposure within the MetALD range. Because viral, autoimmune, drug-related and other alternative causes were not systematically adjudicated, these categories were treated as phenotype-compatible classifications rather than definitive etiological diagnoses.

Statistical analysis

Continuous variables are summarized as mean (standard deviation) or median (interquartile range), and categorical variables as number (percentage). Groups were compared using Welch's t test, Mann-Whitney U test, chi-square test or Fisher's exact test, as appropriate. These comparisons were considered descriptive.

Multivariable logistic regression models examined associations with ultrasound-detected steatosis. Model 1 included PsA, age, sex, current alcohol consumption and PASI ≥ 10 . Model 2 added waist circumference. Model 3 additionally included dysglycemia/diabetes, an atherogenic lipid phenotype and hypertension. A sensitivity model replaced waist circumference with BMI. Because specific and cumulative methotrexate exposure was unavailable, a further sensitivity analysis added systemic treatment as a global treatment indicator to the waist-based models. Associations are presented as adjusted odds ratios (ORs) with 95% confidence intervals (CIs).

Secondary analyses were conducted within the normal AST/ALT subgroup. GGT threshold performance was summarized by sensitivity, specificity and predictive values with exact binomial 95% CIs, and by likelihood ratios with log-method 95% CIs. AUCs used GGT/ULN as the continuous marker and stratified bootstrap CIs. Because elevated GGT produced marked separation, its incremental contribution was explored with ridge-penalized logistic regression rather than interpreted through unstable adjusted ORs. Base models included age, sex, current alcohol consumption and either waist circumference or BMI; extended models additionally included elevated GGT. Internal validation used 100 repetitions of stratified five-fold cross-validation, reporting mean out-of-fold AUCs and paired changes in AUC. Analyses were performed in Python 3.12 using pandas, SciPy and scikit-learn. Two-sided $p < 0.05$ was considered statistically significant for prespecified comparisons; secondary and sensitivity analyses were interpreted cautiously.

As an exploratory, outcome-independent analysis of cardiometabolic structure, principal component analysis (PCA) was performed in complete cases using standardized waist circumference, BMI, log-transformed triglycerides, HDL cholesterol and log-transformed fasting glucose. Component retention and interpretation were based on explained variance and loading patterns. The association between the first component and steatosis was summarized per 1-SD increment with a bootstrap of 95% CI. PCA findings were considered supportive and were not substituted for the prespecified clinical models.

Results

Cohort and prevalence of hepatic steatosis

Of 572 participants, abdominal ultrasound data were available for 534 (93.4%); 38 (6.6%) had no available ultrasound result (Fig. 1 and Supplementary Table S1). Missing ultrasound information was more frequent in PsA than in psoriasis without PsA (12.9% vs 4.0%, $p < 0.001$). Hepatic steatosis was detected in 129/534 evaluable participants (24.2%).

Patients with steatosis were older, less frequently female, and more frequently reported current alcohol consumption (Table 1). They had higher BMI, waist circumference, triglycerides and fasting glucose. PsA frequency was similar in patients with and without steatosis (31.8% vs 26.7%, $p = 0.259$), as were psoriasis duration, PASI, PASI ≥ 10 , nail involvement and systemic treatment.

Table 1
Characteristics according to ultrasound-detected hepatic steatosis

Variable	No steatosis (n = 405)	Steatosis (n = 129)	p-value
Age, years	45.8 (15.0)	50.2 (12.9)	0.001
Female sex	216 (53.3%)	30 (23.3%)	< 0.001
Psoriatic arthritis	108 (26.7%)	41 (31.8%)	0.259
Psoriasis duration, years	18.8 (14.4)	20.0 (15.9)	0.451
PASI	8.0 (4.0–15.0)	10.0 (4.0–15.0)	0.674
PASI ≥ 10	199 (49.6%)	64 (51.2%)	0.759
Nail involvement	224 (57.0%)	80 (65.0%)	0.114
Systemic treatment	258 (63.9%)	82 (63.6%)	0.952
Current alcohol consumption	90 (22.8%)	61 (47.7%)	< 0.001
BMI, kg/m ²	27.2 (5.1)	29.2 (4.4)	< 0.001
Waist circumference, cm	94.6 (13.9)	102.9 (12.8)	< 0.001
Hypertension	75 (18.5%)	33 (25.6%)	0.082
Dysglycemia/diabetes	107 (26.4%)	51 (39.5%)	0.004
Triglycerides, mg/dL	93.0 (71.0–136.0)	132.0 (93.0–190.0)	< 0.001
Triglycerides ≥ 150 mg/dL	81 (20.0%)	58 (45.0%)	< 0.001
HDL cholesterol, mg/dL	53.8 (15.1)	51.8 (16.2)	0.222
Low HDL cholesterol	101 (24.9%)	33 (25.6%)	0.883
Fasting glucose, mg/dL	91.0 (84.0–100.0)	96.0 (89.0–110.0)	< 0.001
AST, UI/L	20.0 (17.0–24.0)	33.0 (26.0–42.0)	< 0.001
ALT, UI/L	20.0 (15.0–26.0)	48.0 (33.0–62.0)	< 0.001
GGT, UI/L	18.0 (12.0–26.0)	63.0 (39.0–123.0)	< 0.001
Values are mean (SD), median (IQR), or n (%). PsA, psoriatic arthritis; PASI, Psoriasis Area and Severity Index; BMI, body mass index; HDL, high-density lipoprotein. P values are descriptive.			

Multivariable cardiometabolic associations

In the clinical model, older age, male sex and current alcohol consumption were associated with steatosis, whereas PsA and PASI ≥ 10 were not (Table 2). After waist circumference was added, each 10-cm increment was associated with 38% higher odds of steatosis (OR 1.38, 95% CI 1.14–1.66). This association remained after additional cardiometabolic adjustment (OR 1.34, 95% CI 1.10–1.64). Current alcohol consumption also remained associated (OR 2.38, 95% CI 1.48–3.84), while PsA was not (OR 1.38, 95% CI 0.82–2.32). Further adjustment for systemic treatment did not materially alter the findings: systemic treatment was not independently associated with steatosis in either the clinical-waist model (OR 1.09, 95% CI 0.61–1.98; $p = 0.765$) or the fully adjusted model (OR 1.13, 95% CI 0.62–2.05; $p = 0.684$) (Supplementary Table S7).

Table 2
Multivariable associations with ultrasound-detected hepatic steatosis

Variable	Model 1: clinical	Model 2: + waist	Model 3: metabolic
Psoriatic arthritis	1.41 (0.86–2.31)	1.39 (0.83–2.32)	1.38 (0.82–2.32)
Age, per 10 years	1.20 (1.04–1.40)	1.13 (0.96–1.32)	1.16 (0.96–1.40)
Female sex	0.31 (0.19–0.51)	0.39 (0.23–0.66)	0.40 (0.23–0.67)
Current alcohol consumption	2.36 (1.49–3.72)	2.28 (1.43–3.66)	2.38 (1.48–3.84)
PASI ≥ 10	0.88 (0.57–1.36)	1.02 (0.65–1.61)	1.01 (0.64–1.59)
Waist circumference, per 10 cm	—	1.38 (1.14–1.66)	1.34 (1.10–1.64)
Dysglycemia/diabetes	—	—	1.13 (0.68–1.87)
Atherogenic lipid phenotype	—	—	1.42 (0.88–2.30)
Hypertension	—	—	0.70 (0.37–1.30)
N / events	516 / 124	492 / 120	492 / 120
Values are adjusted OR (95% CI). Model 1 includes age, sex, PsA, current alcohol consumption and PASI ≥ 10 . Model 2 adds waist circumference. Model 3 adds dysglycemia, atherogenic lipid phenotype and hypertension.			

SLD-compatible phenotypes

Among 129 steatosis cases, 121 (93.8%) had at least one reconstructed cardiometabolic criterion. Eighty-eight (68.2%) were MASLD-compatible and one (0.8%) was MetALD-compatible (Fig. 1 and

Supplementary Table S2). Alcohol amount (SDU) was unavailable in 31 metabolically positive cases and alcohol status was unavailable in one; eight cases had no reconstructed cardiometabolic criterion. These groups were retained as indeterminate rather than forced into an etiological category.

Exploratory PCA in 490 complete cases supported a shared adiposity-metabolic dimension (Supplementary Table S6). The first component explained 49.5% of total variance and loaded positively on waist circumference (0.87), BMI (0.82), log triglycerides (0.68) and log fasting glucose (0.45), and inversely on HDL cholesterol (-0.62). Each 1-SD increase in this component was associated with higher odds of steatosis (OR 1.99, bootstrap 95% CI 1.59–2.60). Its isolated discrimination was moderate (AUC 0.681, 95% CI 0.627–0.734), supporting its role as a structural sensitivity analysis rather than a diagnostic model.

Steatosis with normal aminotransferases

Both AST and ALT were within their sex-specific reference ranges in 408 participants. Nevertheless, 37/408 (9.1%) had ultrasound-detected steatosis. Within this subgroup, steatosis remained associated with older age, male sex, current alcohol consumption, higher BMI, larger waist circumference, higher triglycerides and fasting glucose (Table 3). PsA and PASI ≥ 10 again showed no material group differences.

Table 3
Characteristics with normal AST and ALT according to hepatic steatosis

Variable	No steatosis (n = 371)	Steatosis (n = 37)	p-value
Age, years	45.6 (15.1)	54.3 (13.9)	< 0.001
Female sex	200 (53.9%)	11 (29.7%)	0.005
Psoriatic arthritis	97 (26.1%)	11 (29.7%)	0.637
Psoriasis duration, years	18.7 (14.2)	23.6 (16.7)	0.096
PASI	8.0 (4.0–15.0)	10.0 (5.0–16.0)	0.314
PASI ≥ 10	180 (48.9%)	21 (56.8%)	0.363
Nail involvement	206 (56.3%)	22 (59.5%)	0.710
Systemic treatment	236 (63.8%)	25 (67.6%)	0.647
Current alcohol consumption	85 (23.2%)	17 (45.9%)	0.002
BMI, kg/m ²	26.9 (4.9)	28.9 (4.5)	0.015
Waist circumference, cm	94.0 (13.6)	101.6 (12.9)	0.001
Hypertension	68 (18.3%)	7 (18.9%)	0.930
Dysglycemia/diabetes	100 (27.0%)	14 (37.8%)	0.159
Triglycerides, mg/dL	92.0 (71.0–135.5)	144.0 (106.0–193.0)	< 0.001
Triglycerides ≥ 150 mg/dL	75 (20.2%)	18 (48.6%)	< 0.001
HDL cholesterol, mg/dL	54.3 (15.4)	56.1 (16.1)	0.506
Low HDL cholesterol	86 (23.2%)	6 (16.2%)	0.334
Fasting glucose, mg/dL	91.0 (84.0–100.0)	93.0 (87.0–110.0)	0.033
AST, UI/L	20.0 (17.0–23.0)	23.0 (21.0–28.0)	< 0.001
ALT, UI/L	19.0 (15.0–25.0)	28.0 (22.0–32.0)	< 0.001
GGT, UI/L	17.0 (12.0–25.0)	68.0 (54.0–128.0)	< 0.001
Restricted to patients with sex-specific normal AST and ALT. Values are mean (SD), median (IQR), or n (%). Comparisons are descriptive.			

Only 9/127 steatosis cases with complete AST, ALT and GGT data had all three enzymes within the reference range. Isolated GGT elevation occurred in 28 cases (22.0%), while other enzyme combinations were heterogeneous (Supplementary Table S3).

GGT performance and incremental discrimination

Among patients with normal AST/ALT, elevated GGT was present in 28/37 patients with steatosis and 5/371 without steatosis. Sensitivity was 75.7% (95% CI 58.8–88.2), specificity 98.7% (96.9–99.6), positive predictive value 84.8% (68.1–94.9), and negative predictive value 97.6% (95.5–98.9). The positive likelihood ratio was 56.15 (95% CI 23.07–136.68) and the negative likelihood ratio was 0.25 (95% CI 0.14–0.44). The AUC for continuous GGT/ULN was 0.989 (95% bootstrap CI 0.980–0.996) (Table 4 and Supplementary Figure S1). Performance was directionally consistent among abstainers, psoriasis without PsA, PsA and participants with PASI < 10 (Supplementary Table S4).

Table 4
Performance of GGT among patients with normal AST and ALT

Performance measure	Estimate	95% CI
Sensitivity	75.7%	58.8–88.2
Specificity	98.7%	96.9–99.6
Positive predictive value	84.8%	68.1–94.9
Negative predictive value	97.6%	95.5–98.9
Positive likelihood ratio	56.15	23.07–136.68
Negative likelihood ratio	0.25	0.14–0.44
AUC, GGT/ULN	0.989	0.980–0.996
<i>Sex-specific GGT elevation was defined as > 60 U/L in men or > 40 U/L in women. The 2×2 matrix was: 28 true positives, 9 false negatives, 366 true negatives and 5 false positives. AUC uses GGT divided by the sex-specific ULN with bootstrap confidence intervals.</i>		

In an exploratory incremental analysis, discrimination remained substantial under repeated cross-validation (Supplementary Table S5). In 389 complete cases, the clinical-waist model had a mean out-of-fold AUC of 0.719; adding elevated GGT increased it to 0.945 (mean Δ AUC 0.226; 2.5th–97.5th percentile range 0.201–0.253). In 396 complete cases, the corresponding BMI model increased from 0.713 to 0.936 (mean Δ AUC 0.223; range 0.198–0.248). Because of marked separation and the small number of events, adjusted GGT ORs were not interpreted.

Discussion

This study provides a clinically focused characterization of ultrasound-detected hepatic steatosis in a large real-world cohort spanning psoriasis and PsA. Three principal findings deserve emphasis. First, approximately one quarter of participants with available ultrasound had steatosis. Second, central adiposity remained independently associated after adjustment for demographic, inflammatory and cardiometabolic variables, while PsA and PASI $\geq$ 10 were not independently associated. Third, most cases showed a reconstructed metabolic phenotype, although incomplete alcohol quantification and the absence of systematic adjudication of alternative causes required cautious SLD-compatible terminology.

In secondary analyses, normal AST and ALT did not exclude disease, and GGT identified a subgroup with a markedly higher probability of ultrasound-detected steatosis.

The observed prevalence is compatible with the recognized enrichment of hepatic steatosis in psoriasis [4–6]. Our results refined that evidence in two ways. The ultrasound assessment covered 93.4% of the source cohort and was not restricted to patients with abnormal liver enzymes or clinical suspicion, reducing an important form of indication bias. In addition, the absence of an adjusted PsA association suggests that, within this cohort, cardiometabolic and alcohol-related factors explained more of the cross-sectional steatosis distribution than articular status itself. This does not imply that PsA is irrelevant to long-term liver risk, particularly because treatment exposure, cumulative inflammation and physical activity were not fully captured.

Waist circumference was the most consistent anthropometric correlate. Each 10-cm increment remained associated with steatosis after adjustment for dysglycemia, lipid phenotype and hypertension. This finding aligns with a recent work showing that waist circumference improves cardiometabolic discrimination compared with BMI [17] and with broader evidence supporting waist circumference as a clinically informative measure of abdominal adiposity beyond BMI [16]. Visceral and ectopic fat are metabolically active compartments more closely connected to insulin resistance, altered lipid flux and hepatic fat deposition than body mass alone. Accordingly, routine waist measurement may provide clinically useful information even when BMI is below conventional obesity thresholds.

The normal-aminotransferase subgroup is particularly relevant. Aminotransferases reflect hepatocellular injury rather than fat accumulation itself, and their reference distributions are influenced by sex, adiposity and laboratory definitions. Normal values therefore cannot be interpreted as evidence against steatosis. Previous ultrasound and histology studies have shown that steatosis, steatohepatitis and even advanced fibrosis may occur despite modest or normal enzyme concentrations [9, 18–20]. In our cohort, 37 patients had steatosis despite normal AST/ALT, and their phenotype remained strongly cardiometabolic: older age, male sex, central adiposity, triglycerides and alcohol exposure were prominent.

As a focused secondary finding, GGT provided the clearest biochemical signal in the normal-aminotransferase subgroup. Its very high positive likelihood ratio indicates strong rule-in potential, whereas the negative likelihood ratio of 0.25 is insufficient to exclude steatosis when GGT is normal. The observation is biologically plausible because GGT is linked to glutathione metabolism, oxidative stress, alcohol exposure and hepatic metabolic dysfunction, and prospective population studies have associated higher GGT with metabolic syndrome and cardiovascular outcomes [11–14]. Importantly, GGT is not liver-specific and may be influenced by biliary disease, medication, alcohol and other systemic conditions. It should therefore be viewed as a candidate hepatometabolic risk-enrichment marker rather than a stand-alone diagnostic test.

Exploratory incremental modelling supports this interpretation but also illustrates the risk of overstatement. Adding elevated GGT increased cross-validated AUC by approximately 0.22 beyond clinical-adiposity models, with similar results using waist circumference or BMI. At the same time, the

2×2 distribution produced marked separation, making adjusted ORs extremely large and unstable. We therefore deliberately emphasize out-of-fold discrimination and diagnostic counts rather than dramatic coefficient. These analyses are reported in the supplement: even repeated cross-validation cannot substitute for testing the threshold and model in an independent cohort, and the very high AUC may partly reflect cohort-specific measurement and referral patterns.

Contemporary SLD analysis also requires nuance. Most steatosis cases had at least one reconstructed cardiometabolic criterion, supporting a predominantly metabolic phenotype. Nevertheless, incomplete alcohol quantification prevented definitive classification in a substantial minority, and other causes of steatosis were not systematically adjudicated. For that reason, MASLD-compatible and MetALD-compatible are the appropriate terms. This conservative approach avoids retrofitting a complete etiological diagnosis to variables collected for another purpose.

Several limitations should be acknowledged. The cross-sectional design precludes causal inference and cannot determine incident liver disease or progression. Conventional ultrasound has limited sensitivity for mild steatosis and cannot distinguish steatosis from steatohepatitis or quantify fibrosis; elastography, controlled attenuation parameter, magnetic resonance-based fat quantification and histology were unavailable. Ultrasound data were missing more frequently in PsA, creating potential selection bias despite high overall coverage. Alcohol quantity was incomplete and recorded in daily SDU, limiting contemporary etiological classification. Viral, autoimmune, medication-related and other liver diseases were not systematically excluded. Methotrexate exposure is particularly relevant in psoriatic disease because liver risk reflects an interplay between treatment and cardiometabolic susceptibility [21, 22]. The available variable did not distinguish methotrexate from other systemic therapies or capture previous exposure, duration, cumulative dose or its temporal relationship with ultrasonography. Although adjustment for systemic treatment did not modify the principal associations, a specific contribution of methotrexate cannot be excluded. We also lacked platelet-based fibrosis scores and longitudinal hepatic outcomes. Finally, the GGT analysis included only 37 events, was internally validated, and requires external replication.

Strengths include the large clinically characterized cohort, ultrasound coverage exceeding 90%, explicit handling of missing alcohol information, sequential multivariable modelling centered on waist circumference with BMI sensitivity analysis, and cautious reconstruction of contemporary SLD-compatible phenotypes. Sex-specific enzyme thresholds and the decision not to interpret separation-driven GGT ORs improve the transparency of the secondary biochemical analysis.

In conclusion, ultrasound-detected hepatic steatosis was frequent in psoriatic disease and was more closely related to central adiposity, cardiometabolic factors, sex and alcohol exposure than to PsA or skin severity. These findings support routine cardiometabolic assessment, including waist circumference, across the psoriatic disease spectrum. Normal AST and ALT did not exclude steatosis; within this secondary subgroup, GGT emerged as a candidate risk-enrichment marker requiring external validation.

Declarations

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Declaration of the use of Generative AI and AI-assisted technologies in the research and writing process

During the preparation of this work, the authors used OpenAI ChatGPT/Codex (GPT-5) to improve readability and language, harmonize terminology, organize references, check consistency across submission files, and assist with document formatting. Following use of this service, the authors reviewed and edited the content thoroughly and declare full responsibility for the authenticity and accuracy of this publication.

Ethics declarations and trial registry information

The study was approved by the Research Ethics Committee of the Principality of Asturias, Oviedo, Spain (reference 2020.423). Trial registration was not applicable.

Disclosure statement

Conflicts of interest: R.Q. has received institutional research support, project development funding, consultancy fees and speaker honoraria from AbbVie, Amgen, J&J Innovative Medicine, Lilly, Novartis, Pfizer and UCB. P.C.S. has received grants or honoraria from and/or conducted clinical trials for AbbVie, Amgen, LEO Pharma, Sanofi and UCB. I.M. and A.G.C declare no competing interests.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available because they contain information that could compromise participant privacy and are subject to ethical and institutional restrictions.

CRedit: Conceptualization, R.Q., P.C.S.; Formal analysis, R.Q., I.M., A.G.C., P.C.S.; Investigation, R.Q., I.M., A.G.C., P.C.S.; Resources, R.Q.; Supervision, R.Q., P.C.S.; Validation, R.Q., I.M., A.G.C., P.C.S.; Visualization, R.Q., I.M., A.G.C., P.C.S.; Writing—original draft, R.Q. P.C.S.; Writing—review and editing, R.Q., I.M., A.G.C., P.C.S, R.Q. All authors approved the final version of this manuscript.

Previous dissemination and related work

The source cohort has supported previous cardiometabolic analyses, including the published article ‘Waist circumference provides improved cardiometabolic discrimination compared with body mass index in psoriatic disease: discrimination and reclassification analyses’ doi: 10.1080/00325481.2026.2727239. The present manuscript addresses a distinct hepatic outcome,

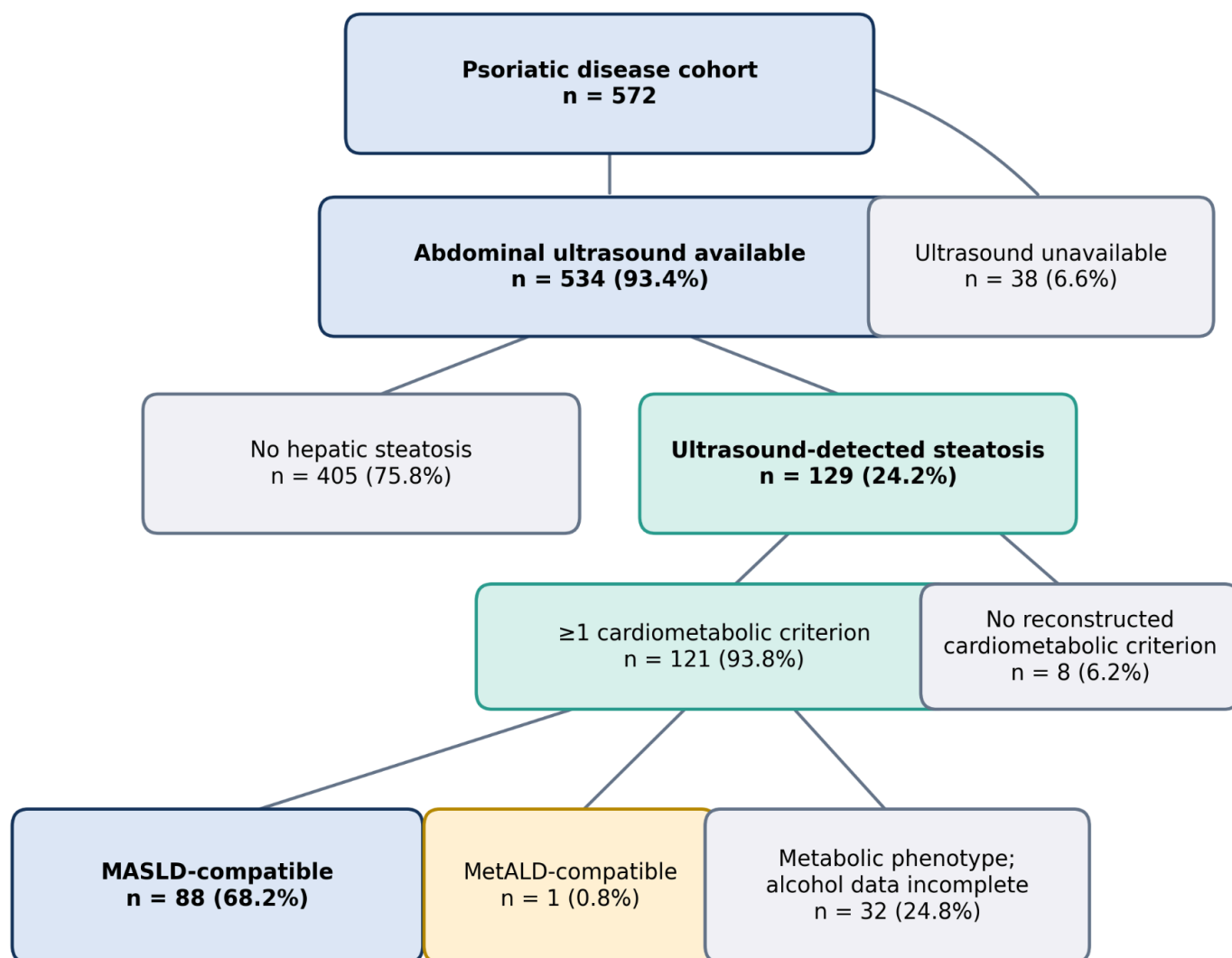
contemporary steatotic liver disease-compatible phenotyping, biochemical discordance and secondary GGT analyses. No part of the present manuscript is under consideration elsewhere.

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Figures



MASLD/MetALD categories are phenotype-compatible classifications.

Figure 1

Flow of the source cohort, ultrasound availability and phenotype-compatible contemporary SLD classification. Percentages for steatosis are calculated among participants with available ultrasound; classification percentages are calculated among the 129 steatosis cases. MASLD, metabolic dysfunction-associated steatotic liver disease; MetALD, metabolic dysfunction and alcohol-related liver disease.

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